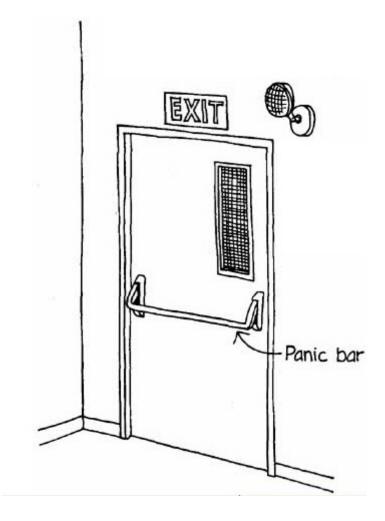
## Ascaris Fluke Tapeworm Formula (Round, Flat, and Oval)

Short and Sweet – How I did it.

## **ASCARIS 123 – How to escape Ascaris**



# By: Matt Kaltenbach mattk3@fuse.net mattk3 at the CureZone

This document can be found in the folder

http://www.curezone.org/ig/f.asp?f=3262

Ascaris 123 is my third attempt to find a formula to kill Ascaris, Flukes, and tapeworms

Ascaris 123 is a work in progress. I felt it so important that for years I spent on I knew not. I did not know what illness I had. After twirling like a compass, I passed through and got hits for **IBS**, **SIBO**, **SIYO**, **Leaky gut**, down the list of symptoms one at a time. Fast as I could. Learning every step along the way, how to throw anything against the wall and see if it sticks. Read Ascaris had been cured in two early teenage girls, hospital, one case, both cured. Else there is no cure for those over 60. Did not believe it.

Ascaris 123 is actually three different formula revisions:

- Ascaris 1 was a private attempt done with some early adopter CZ folks.
- Ascaris 2 was a public CZ forum formula, but had issues. So many messages and emails followed, I put the document into a text form.

Ascaris 3 (The CureZone forum Document folder) is the successful killing formula for me. I
cleared a systemic fluke infection, and stopped Ascaris damage. I have monitored the CZ
site, looking to update where I can, print the final document, with new information.

#### **Latest Document**

The CureZone folder now has an updated **Revision**, which expands on **Formula 3**, including my personal notes of what I experienced as I transitioned from killing Flukes and Ascaris, to parasite maintenance, to rehab, and **final thoughts**.

Red = Caution Green = Med Black Bold = attention

Blue = Information/Index/Subject/Find or Search in Document, Many links on subject

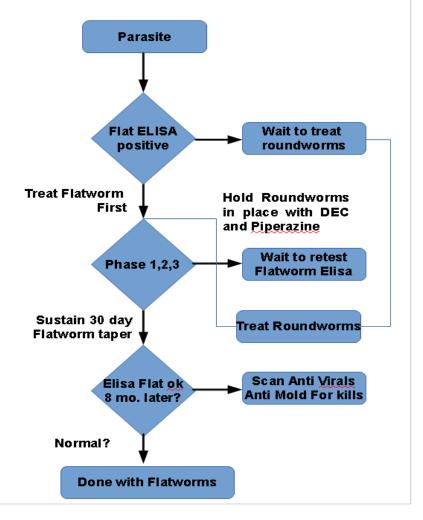
#### **Status:**

I no longer feel birthing of Ascaris baby worms, I no longer have sinus blood or little pricks in the feet or legs. I still carry the stench of dying worms from time to time, and still move toxins out of my body. My brain and body are healing, and I feel stronger every day.

I moved into the final push to kill Ascaris, and I landed on Alinia, keep DEC and PPZ dose at full 400x2, 750x2. OK Alinia at 25mg/kg 14D, 6pm, 6am DEC and PPZ baby, MBZ 100 mg at 6 AM, to ensure night birthing is killed ASAP, MBZ100 mg, and for those using Piperazine Phosphate, it does not seam to kill as well as

Piperazine Citrate.

#### **Parasites**



http://www.toolsforhealing.com/Health/Parasites/Parasites.htm

By **<u>Dr. Ross Anderson</u>** -the single most undiagnosed health challenge in the history of the human race is parasites.

A parasite is an organism that lives off of or inside of another organism, called a host, during all or part of its life.

If you were tested by a doctor for parasites, chances are the results would come back negative.

Does this mean you do not have parasites?

Unfortunately, medical testing procedures only catch about 20% of the actual cases of parasites. There are over a 1,000 species of parasites that can live in your body; tests are available for only approximately 40 to 50 types. This means doctors are only testing for about 5% of the parasites and missing 80% of those.

This brings the ability to clinically find parasites down to 1%.

Antibiotics of today as well as any other drug of tomorrow can have a slightly different than intended effect...again, I will **let Dr Andersen** explain:

o Once you have established that you have parasites, taking drugs to get rid of them may not work. A drug will often drive a parasite from one organ of the body to another.

It's like people **moving** to better climates to make their living conditions more pleasant, or birds flying south for the winter.

- So, if we aren't able to combat the rising number of parasitic infections with conventional medicine, it is even more important to understand just how we can go about that process.
- o First, let's look at what parasites do and how we sometimes unknowingly "help" them. Parasites tend to secrete **toxins** as they live within the human body, which, when coupled with other toxins (like alcohol, cigarettes, junk food, polluted air, etc) can lead to what is termed by many doctors as "toxic overload."

<u>Toxic overload comes about when the four primary cleaning systems of the body</u> <u>have been pushed too far</u> by an overload of toxins in the body.

- Within the four cleaning systems, the lungs, kidneys, skin and bowels, there are many types of overload that can occur. As an example, toxins will travel from one system to another as the current system gets overloaded.
- In toxic bowel syndrome, the excess of toxins in the bowels pass onto the liver and the liver becomes overclogged and the toxins begin to spill into the bloodstream.

This can take a long time or can occur very quickly, depending on how the body and its immune system handle(s) the overload.

- Parasites have an ability to cause a complete system breakdown, making them one of the most dangerous epidemics facing medicine today.
- o For the time being, the news isn't going to get much better, I'm afraid.

Not only do <u>80-85% of all American adults have some form of parasite</u>, not only are they hard to diagnose, not only can they cause serious damage internally (and often silently for a long period of time) they also come in many forms.

- From here, you can read about the different types of parasites, or you can skip to "How Do We Get Parasites?"
- Here are a few of the different types of parasitic worms the body can acquire and be plagued by - for more complete and in depth analysis of the types of worms found within the human body (complete with pictures,) please see the links section at the end of this article.
- The following section is taken from the article "Are you Clear of Parasites?" By Dr Andersen, wherein he cites "The Essentials of Medical Parasitology," by Dr. Thomas J. Brooks.

#### INTRODUCTION

During our relatively short history on Earth, humans have acquired an amazing number of parasites, about 300 species of helminth worms and over 70 species of protozoa (9). Many of these are rare and accidental parasites, but we still harbor about 90 relatively common species, of which a small proportion cause some of the most important diseases in the world, inevitably, these are the ones that have received the most attention. Since most of these parasitic diseases occur mainly in the tropics, the field of parasitology has tended to overlap with that of **tropical medicine**, and thus the histories of these two fields are intertwined. There is, however, much more to the history of human parasitology than this, and our understanding of parasites and parasitic infections cannot be separated from our knowledge of the history of the human race.

http://cmr.asm.org/content/15/4/595.full

#### **Parasite Classifications**

Parasites are generally classified into broad groups such as:

- Protozoa: amebae; flagellates; ciliates; coccidia, microsporidia that are all intestinal parasites; sporozoa and others that attack blood tissue, cells, and other body sites
- Nematodes: roundworms
- Cestodes: tapeworms
- Trematodes: flukes
- Arthropods all kinds of insects like scorpions, lice, and tongue worms

## <u>Parasites - http://patrickrambling-pb.blogspot.com/2012/12/lessons-from-parasites.html</u>

15/ Parasite species and 2/ host immunity (how strong your immune system is). People report a wide variety of symptoms from neurological problems, insomnia, fatigue, loose stools, constipation, muscle aches, joint pains, sore throat, memory loss, brain fog, concentration issues, cognitive problems, disorientation, gut pain, indigestion, nausea, IBS (irritable bowel syndrome), colitis, celiac, Crohn's, inflammatory bowel disease and many other symptoms.

**Practice hygiene** More than 72 species of protozoan and helminth parasites can reach humans

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#### 10 key signs a parasite is present in the body

Let us discuss in general the path of a parasite infection in the body before moving on to the distinct symptoms. When the parasite infection is acute, abdominal distress in varying degrees may be experienced by an individual. This distress includes diarrhea, fluid loss and burning sensations.

Visible evidence of an infection is seldom present at this stage of the problem. It is possible to go a step further and state that the parasites do not always appear in the specimens tested in laboratories. Guess they are pretty good at hiding themselves!

A parasitic infection will move from the acute to chronic stage. At this point apart from the diarrhea; constipation, intense burning, bloating of the abdomen, cramping and sudden urges to eliminate, become part of the symptoms.

There can be sudden food cravings, weight loss, irritable bowel syndrome, blood sugar fluctuations and malabsorption of nutrients from foods consumed. The problem when untreated can continue to manifest itself through acute itching of the body; sleep disorders, skin sensitivity, anxiety and depression.

#### **Symptoms**

Symptoms for worms vary depending on the species and where they are located in the body and are listed in detail below but here are some symptoms that can be caused by worm infestations:

- o Itchy nose, ears, anus
- o Feeling cold, chills, sweats, night sweats
- o Nosebleeds
- o Hand tremor
- Restless leg syndrome
- o Alzheimer's and Parkinson's symptoms
- o Sleep disturbances (especially between 2-3 am)
- o Headache
- o Congestion
- o Cannot get enough oxygen during exertion
- o Green or black stools
- o Slow reflexes, clumsiness
- o Foul breath, Sulfur smell of rotten eggs, greasy stools
- o Dark circles under the eyes
- o Yellowish face
- Lactose intolerance
- Fast heart beat
- Teeth grinding,
- o Nervousness, forgetfulness
- o Sleep disturbances, restlessness at night with bad dreams
- Nausea or vomiting
- o Sporadic constipation, diarrhea, unbelievable amount of gas and bloating
- o Irritable bowel syndrome, Crones, Celiac Disease

- o Nausea or vomiting
- o Numb hands
- o Stomach pain or tenderness
- o Aches in the joints and muscles,
- o Chronic fatigue or weakness
- o Allergies environmental and chemical sensitivities
- o Rashes
- o Low blood sugar (hypoglycaemia)
- o Anaemia
- o Flu-like symptoms
- o Long standing obesity
- o Depression
- o Changes in appetite
- Constant desire for food and or bouts of food cravings
- o Immune dysfunction
- o Weight loss, or Weight gain
- o Passing worms, their eggs or segments in the stool.
- o Wiggle behind eyes, in sinus, or ears
- o Red streaks in skin, with painful chewing
- o Ammonia smells
- o Clear Urine
- o Excessive constipation
- o Skin rashes with blisters
- o Worms in Humans depress the immune system by decreasing immunoglobulin A.
- o Gas and Stomach Bloating, Some parasites live in the upper intestine, which can cause both gas and stomach bloating.

- o **Nervousness**, The waste products from parasites irritate the nervous system, resulting in anxiety and restlessness.
- Dark black circles under the eyes
- o Toxic levels of aluminum and lead in hair/nails
- o No minerals or essential metals in hair/nails
- Acidosis
- o Malabsorption
- o SIBO
- o Steatorrhea- lack of <u>bile</u> acids, <u>Exocrine pancreatic</u> <u>insufficiency</u>
- o Parasitic worm infestations can cause total blockage of the intestinal tract causing severe constipation.
- o Parasitic worm infections speeding 'transit time' and causing diarrhoea and also reducing the body's opportunity to absorb the nutrients in food leading to malnutrition and weight loss
- o Worms can also interfere with normal digestive processes

#### **Tests**

- o If you were tested by a doctor for parasites, chances are the results would come back negative. Does this mean you do not have parasites? Unfortunately, medical testing procedures only catch about 20% of the actual cases of parasites. Over a 1,000 species of parasites can live in your body and tests are available for approximately 40 to 50 types. This means, doctors are only testing for about 5% of the parasites and missing 80% of those. This brings the clinically found parasites down to 1%. Now, if I had a 1% chance of winning in the stock market, I don't think I would invest. Only 1% of parasites are ever clinically found.
- o Parasites rob you off all your finest nutrients and you get the scraps and leftovers. They grow healthy and fat while your body starves for nutrition. And these visitors can subside and exist in the human body for anywhere in the upwards of 10, 20 or even 30 years.
- Tests are \$200-300 per parasite screening. There are thousands of possible tests. Computer PCR testing has been abandoned; it was to affordable and too large in scope.

- Let's now look at the way parasites reproduce this is the "lays eggs" part. To start, let's examine the two main types of parasites and then discuss how each reproduces: Large parasites are visible and are primarily worms and small parasites, which are mainly <u>microscopic</u> in size, include what are called <u>protozoa</u> and <u>amoebae</u>.
- o <u>http://www.metametrix.com/files/test-menu/interpretive-guides/GI-Effects-IG.pdf</u>

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http://www.vumicro.com/vumie/help/VUMICRO/Hydrogen\_Sulfide\_Production\_Test.htm

 http://www.authorstream.com/Presentation/doctorrao-1850545-enterobacteriaceae-basic-skills-identification/

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http://www.questdiagnostics.com/testcenter/BUOrderInfo.action?tc=36470X&labCode=QBA

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http://www.hardydiagnostics.com/pdf/sc\_posters/EnteroScreen4\_poster.pdf

- o <u>http://www.slideshare.net/doctorrao/biochemical-tests-in-enterobacteriaceae</u>
- http://www.gdx.net/core/supplemental-educationmaterials/Pathogenic-Organism-Chart.pdf
- o <a href="http://www.holisticheal.com/media/downloads/products/utm-uee-sample-test.pdf">http://www.holisticheal.com/media/downloads/products/utm-uee-sample-test.pdf</a>
- http://www.holisticheal.com/urine-toxic-metals-test-kit.html
- o 279 Walkers Mills Road, Bethel, Maine 04217, 1-800-768-8744

#### Size

- Large parasites, which are the worm type, can usually be seen by the naked eye. Sizes can exceed 15 inches long and normally these worms cannot/do not travel past the digestive tract.
- o The **smaller** organisms, the **protozoa** and **amoebas**, tend to act like a bacteria by traveling through the blood stream to virtually any part of the body. They reproduce without laying eggs and behave more like an infection in the body than do the larger parasites.
- o The **larger** parasites are worms which reproduce by laying eggs. Eggs are left in the intestinal tract, where they cling to the intestinal walls among the feces, and when they hatch, the young feed on the food that we eat and eventually grow into adults. The adults then repeat the process.
- A parasite eats, lays eggs and secretes. Ok? Let's break this down into the three parts: the first is "eats." Depending on the kind, parasites will eat different things. Many thrive off certain types of food,

from dairy to sugar to proteins. These parasites live off the food that goes into your body. Mainly found in the digestive tract, they can also be found in the liver as well as throughout the body. No organ within your body is immune to parasites, in reality. Some parasites actually get their nutrition directly from the cells of the body, and feed off those cells, thus making you sick. They can literally attach themselves anywhere and suck nutrition out of the cells. These parasites are perhaps the most dangerous because they can travel to places In the body where they can do a lot more damage than a parasite living solely in the digestive tract.

- Which is more dangerous? Despite their almost invisibility, small parasites can be extremely dangerous. Microscopic parasites can destroy calcium lining in your bones, eat the myecin lining off your nerve cells (causing breakdown of the brain-nerve connection) and even inhabit the liver, colon and other areas causing major discomforts and problems. The small parasites reproduce by duplicating themselves in a manner similar to bacteria or viral reproduction.
- o In extreme cases, parasitic worm infestations can cause total blockage of the intestinal tract causing **severe constipation**. The opposite is also true with parasitic worm infections speeding 'transit time' and causing **diarrhoea** and also reducing the body's opportunity to absorb the nutrients in food leading to **malnutrition** and weight loss.
- Various nutrient deficiencies can also be caused by parasitic worm infections as the parasites absorb vital vitamins and nutrients that would normally be absorbed by the host body.
- The actions of the worms can also interfere with normal digestive processes. For example, worms release protease inhibitors as a defense against the body's protein digesting enzymes and these may impair the breakdown of other proteins intended for use by the body.

#### **Lactose intolerance**

- o In addition, direct damage to the intestinal mucosa can cause impaired nutrient uptake, and the complex chemical imbalances caused by the body's reaction to the worms can also affect nutrient absorption. For example, roundworm (**ascaris**) infections have been linked to temporarily **induced lactose intolerance** and to the malabsorption of vitamin A, protein, and fat.
- o Parasitic **worms** also release chemicals that **suppress the host's immune system** in order to continue existing within the host. This, in combination with the long-term immune response triggered by worm infection may drain the body's ability to fight other diseases, making affected individuals more prone to viral, bacterial and fungal infections. The worms also excrete toxic waste products that continuously poison the host's body taxing detoxification processes.
- Worms and their larvae can also perforate organs and tissues as they migrate through the body causing inflammation, acute

symptoms and interfering with their functions. Finally, parasitic clumps are often mistaken for cancerous tumours with all that implies.

#### Next

This is a complex subject. The document continues to grow. I have more material than I have pages. I expect to continue to refine the document with some complexities of testing and start-up.

Questions I get a lot of, and I an attempt to answer what I went through, my perception, and aspects of how I adjusted the formula to keep my body functioning.

#### **Enzymes**

This research is a work in progress. The truth is that all parasite families share common enzymes, and toxin generating processes, making specific generic symptom identification in the medical literature, kind of way beyond most doctors.

#### **How do Anti-Parasitics Work?**

I attempted to see past the details of different parasite worm types, and instead focused on a way to treat the common weak points in the worm life cycle.

## **Keep it simple**

I started to use generic terms like round, flat, enzyme, toxins, to generalize the process. Turns out families of parasites have common enzyme toxins.

#### **Few Meds work**

There are only a few dozen meds that are available anyway, and possibly thousands of parasite types. Why look at detail of what species you have when the answer will always be A, B, or C Antiparasitic?

Invermectin
DEC
ALB
PIPERAZINE
PRAZIQUANTEL
BIOPERINE
FLAGYL
ALINIA
VERMOX – PULSE 3D.MAX? Skip 3D and pulse again 2X
FenBen? Caution?

#### **Treat**

When in doubt, treat. Several people have approached me with infections that span over a decade, and so many have family members that are infected, I can only assume these infections are beyond the current ability for medical science to identify.

I correspond with so many that have been wounded by this infection. While the symptoms vary, the treatment process from my perspective, does not.

I say this because my infection was life threatening.

Selecting an extreem process aimed at saving life is justified, in those situations where no parasite has been identified. The dammage that flukes and ascaris cause is significant and severe. Left untreated, physical damage to the DNA and the body result.

The meds will not kill you if you dose correctly. It made no sense to look at the parasite type in detail, Stool tests are useless, ELISA tests give confirmation of the direction, justify anti-parasitic meds, identify round and flat worm types.

For me it made sense to *treat first, ask questions later*.

Jumping into "<u>Treat</u>", not knowing the type of parasite, how to dose, or what time of day to take the meds, how to handle toxin load, degradation of the liver, kidneys, heart, brain is the real trick.

Approaching the parasite process using a process of <u>Treat</u>, Test, Identify the parasite type, is completely a backwards process. I propose that not treating causes more harm than treating.

I was able to get a confirmation ELISA test for Ascaris, during my treatment, attesting to the fact that ELISA is better than stool tests. I completely missed the fluke infection, which was even more destructive than Ascaris.

#### **ELISA**

Elisa is currently the test technology that is most current, and most accurate after an infection is present for a year, Besides, PCR research labs testing is no longer generally available.

It made every sense to look at the treatment, the dosing, the duration, and the formula, and look less at the specific parasite type.

Get Tested for Ascaris, Fluke, the most dangerous and distructive parasites first.

#### **Critical Accute Infection:**

- Feel like you are dying?
- PH Acid
- · Mineral and metal loss
- Malabsorption
- Maldigestion
- Gaining or Loosing Weight? A lot? How fast?
- Diarrhea for a prolonged period
- Skin issues

- Can't catch breath
- Can't think strait
- Heart Pain
- Circulation/Nerve issues
- Muscle, joint, cannot walk or function properly
- GERD, Reflux, Mucus, Acid pulse at 2-3AM, lung and sinus? Pulse Rate High.
- Ascaris ID

## Q&A

Q>

thank you I have'nt looked at it yet as my headache came back late last nite & my vision in right eye is barely there @%15 making it very hard to read.

i wanted too say i am a 50yrs old 220lb w/ male,

i wasn't sure if you new that or thought i was women because you sent me the womens formula , but that was good for me as thats were i am gonna start and move into the full dose male formula , so i can reduce their numbers and not get too sick from die off & i just <code>lost like 50</code>lbs since march , i had <code>gained all that weight back</code> in 2011 , i could'nt figure out why i was gaining all that weight! know it makes sense, when this started seems maybe <code>thats when i picked up the round worm infection</code>.

>question? is it better too take the doxycycline 100mg twice day or 200mg once daily? i was thinking if not getting much better by 3 -5 days of doxy then start the **Levoflaxcin**?

maybe the backslide is my gutz are blocked again!

i have already taken three potent laxatives & nothing is budging! this is so fustrating as it seems its either blocked or diearerhea

well crap my computer fried while typing this email to you so not sure if you getting more than one email!

i have to get replacement know power supply fried damn! man everything blows up at the same time! i still gotta order a bunch of stuff

so not sure if i am getting better or not, everytime i get feeling better bamn i get sick again! so i decided to dose Pinx as nothing is clearing my colon its blocked!.

so about hr after PinX i got some big ass something came out! and it burned bad on the way out, maybe a tapeworm! or fluke or ?? it floated on top and was long & light brown> my gutz felt better immediately after passing it@! so i am again inclined to think that dose of IVM knocked it loose from Liver and it re-attached on the left side of colon, and know i think that PinX killed it!

well i hope so! I am going to start Alba dosing tonite now that my colon is moving again

## **Get into the Critical Path Parasite treatment process:**

- If your parasite is critical infection, acute, chronic disease level, use **emergency** flat and roundworm treatments, high level, be save and understand dosing ramp, dosing, schedule, side effects, precautions, research this to be safe.
- Get tested for these two first.
- You can do this in parallel, just make sure to get tested, dont stop, get an actual worm id if at all possible.
- At the end when you are taking a lot of meds, knowing what you are fighting becomes very important!
- If one tests positive for a **flatworm**, this document has the supporting premise, supported by evidence that **Praziquantel** is the anti-parasitic of choice, especially in the clinic, where doctors actually treat parasite infections, for anyone with a brain infection.
- Test each med before ramping **dose**, understand dosing levels go by body weight
- <u>Support</u> Group,
- Testing Lab, ID TEST PCR, ELISA, 3D Pathologist microscopy
- Emergeny Contact, ER

#### **Severe Critical Parasite Infection**

#### **Severe – Critical Parasite Infection**

Because severe infections go to the lung, liver, kidneys, brain, and cause progressive damage. Treating a **flatworm** infection with a deep rapid treatment course that scales, can deeply migrate into tissues, and kills **flatworms** deeply, and finally.

## **Protect Organs**

- Liver
- Kidneys
- Brain
- Heart
- Circulation Capillaries keep open
- Platelett clumping thin blood
- Immune system, lower inflammation
- Toxins
- DNA
- Membrane osmotic stress
- Fluid retention

#### **BBB**

The Blood Brain Barrier

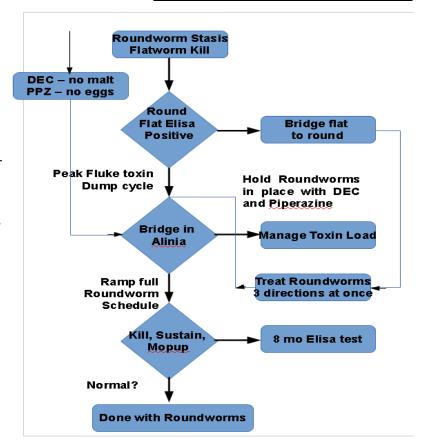
The **Praziquantel** molecule is

sucked past the BBB using <u>Albendazole</u>. Higher Doses of <u>Vitamin D3</u> produce a faster and deeper kill. <u>Calcium</u> in milk helps invade <u>fluke</u> shell and kill worms. Dodder seed moves out black stool massive toxin dump, near death experience. <u>Fluke</u> Kill.

#### **Large Round Worm**

#### Movement, older infection, or the very large roundworm 2B people have.

If Ascaris <u>ELISA</u> (roundworm) test is positive, then identifying the <u>flatworm</u> infection is the most important next step, because as worm infections go.., the common liver <u>fluke</u> is a very damaging worm. The enzymes it emits through its skin is caustic to human <u>DNA</u>. Treatment of Ascaris does



not work when one is infected with <u>flukes</u>. Treat the <u>flukes</u> first, put roundworms in Stasis, by killing offspring.

This strategy worked for me.

Remember, if you have more than one parasite in your body, It is best to know through ELISA.

The Good, The Bad, and The UGLY TRUTH

- 3.5 billion people have parasites.
- The major harmful parasites estimate is a billion or more for each of the major parasites!
- CFS, Lime, Flesh eating disease is everywhere
- The municipal water tests are showing positive for heavy egg counts around the globe.
- Currently there is no water treatment technology available to filter out 10 micron eggs.
- **Priority**: Clear your body of worms, bacterial load, viral load, fungal load, worm shit, nitrogen, parasitic worms, Larval Stages, and Eggs in real time, kill adults, and prevent inflamitory scaring, and toxin illness. Death!
- There are hundreds of standardized tests. There are hundreds of thousands of research reports. Doctors find fewer than 1% of parasites (by testing their accuracy of diagnosis of parasites).
- Hospitals identify fewer than 20% of parasites in pathology lab accuracy reports.
   Private test Labratories have higher accuracy. Autopsy identifies the majority of parasitic conditions.
- If you have a parasite and live in a third world country you have better odds than if you live in the United States, or any old world ,other city, location, around the Globes. Parasite warning maps are drawn every year.
- In the USA the common belief is that most parasitic conditions were eliminated in the 1930's. This is untrue, and sad.
- If you go to a doctor, as I did, my doctor told me "we do not have parasites in this country".
- O> He repeated it many times, and many places in the conversation, like I was a fool.
- Dentists had a probe a few decades ago, hospital treatment of worm infection has high mortality, do everything you can do to identify the worm type, detection always found in autopsy.
- A> He was foolish: This one is untrue...The USA is full of parasite infections, Doctors miss them!
- The Infectious Disease Doctor, number 1 and number 2, both missed it, or how to get it treated go elsewhere!
- If you find an "accidential" test positive, the hospital or doctor must report in many cases, many types of parasitic conditions, to the CDC, can only get medications from the ... CDC,

and their allocation of anti-parasitic allocation (number of pills) is so antiquated and out of date from the latest research, that you may never be cured. – this is true.

- The CDC is now in the parasite testing lab facility, it is active, and up, and the CDC is itself
  developing parasite tests!, The group is hidden, available for a doctors consultation fee, or
  hospital contact. Doctors can use as a second opinion.
- South Korea has opened up its research, doctors, universities and Associations to manage a multicountry effort.
- If you are of the unfortunate, have taken prescription anti-biotics, or are getting older, the path to curing yourself is not easy. You may want to give up now, because the path to health is difficult, complicated, costly, and requires a complete refocusing of your life to live parasite free.
- Parasites are everwhere True
- Everyone has parasites Nearly true
- Most Diseases are caused by parasites some mainstream researchers are starting to believe this
- Parasitic infections can never be cured just a few decades ago this was thought to be true
- Traditional one dose, or short dose antiparasitics cure most infections The CDC believes this, few others do at this point.
- Parasites is a third world illness absolutely untrue. It is a Global scurage!

## **Testing**

#### **Get a Flatworm Elisa, (<u>Highest Priority Test</u>)**

Schitzomiasis lung fluke liver fluke Antigen in 13KDA. Common most Playmouths https://en.wikipedia.org/wiki/Flatworm

#### **Get a Roundworm Elisa (Second priority test)**

Get Ascaris family of roundworms - Enzymes cause antigen 209 KDA on ELISA test for Ascaris – in human and other animals SUUM – in human and pig lumbricoides not an Ascarid

#### **Round to Flat to Round Elimination Process**

First, it is important to change those **things you can change**, (egg killing and Larvae in the GI tract, and in the system). Roundworms can be put into **Stasis** - by using **egg** and **larva** kill antiparasitics, and killing **larva migrans** and **eggs** in real time. This takes a **coctail** of meds within

doses, ranges, and times (a schedule). This is a complex schedule everyone should build for themselves. Minerals, Metals, PH, Hair Analysis, Std Blood test. Have some data. Get Data.

Many other tapeworms will be addressed by this formula, the pork one is bad. **PZQ** kills this one too, as well as the intestinal tapeworm.

**Killing Flatworms is a risk prone process**, that involves a systemic kill. If **flatworms** are in the organs and brains, this is a near death experience. It is possible to kill the worms with **PZQ**, but it took me 300 Grams to make sure the flatworms were dead.

After <u>Flatworm</u> black stool toxins are expelled, a bridge can be made to the kill phase by tapering <u>PZQ</u> and <u>ALB</u>, and ramping <u>Alinia</u>. Additional anti-parasitics may be needed.

#### **Bridge**

<u>Vitamin A</u> and <u>Zinc</u> are required to kill <u>flukes</u>.

After the **flukes** are killed, killing of roundworm adults can start.

This one simple step transitions to killing roundworms, exactly at the time the **Fluke** enzyme control is broken.

Flukes dump black toxins for about two weeks.

By halting <u>Vitamin A</u> and <u>Zinc after the fluke kill phase</u>, faster roundworm killing can be allowed to happen, studies support that <u>killing of Ascaris</u> is harder if the person is on <u>either Vitamins A or Zinc supplements.</u>

#### Q&A

#### Q> I cannot see out of my right eye.

A> parasites eat vitamin A,take rose hip seed tea, and pure vitamin a, not betacarotine, dose high enough, keep A under 60,000 IU/D

- How much do you take
- Eyes need billberry
- Eves need lutin

Go to local whole foods, get some Vitamin A, Billberry, Lutin.

Do not miss a dose of white willow bark, or Ginkgo, they open up capillaries as far as they can go.

If you are not on a, start now. Rosehip seed tea.

The next taper phase of flatworm meds, and increasing roundworm meds, should lead to freedom of parasites, with the body ready for movement.

#### **Keys**

Like curing a <u>fungal infection</u>, the parasite has many chemical process chain(s) that need to be broken.

- Energy
- Motion
- Iron
- Muscle
- Enzyme
- Calcium

It is at probing the <u>life cycle of the worm</u>, success may lie. There are known worm life cycle <u>choke points</u>, of iron enzyme, carbon, and carbohydrate feeding. Different Anti-Parasitics attack with these choke points. Malting can also be stopped, and the shell degraded.

Searching deeply on the internet, there are several widely held belief's that parasitologists discuss. A mind expanding concept is reading <a href="https://document.com/how-anti-parasitics-attack-their-pray">how anti-parasitics attack their pray</a>.

#### Fix the Body

There are some supplement and body process, vitamin progressions, like:

- Stabilize mineral flow
- Increase **Toxin Removal** Rate
- Increase Salt chemistries Magnesium, Zinc, Calcium
- Neutral (no change) in natural sources of **iron** only, supplements that are ionic pose risk.
- Pull on DNA and command full open throttle ALA
- Command DNA to dump toxins L Carnitine, CQ10, ALA, KGP Flush
- Feed DNA Amino acids full spectrum Algae Spirulina
- Gentle Chelation Chlorella Algae.
- Demand Maximum DNA Accelerator ALA, DNA Program set: forced Magnesium Regulation Protocol, Lipid A/C vitamin Channel, GLA, alpha tocopheroyl.
- **Electron Transport CQ10** between all four cellular process membranes
- ROS lost minerals and metals <u>Selenium</u>, <u>Zinc</u>, <u>Calcium Salt of Magnesium</u>, <u>Salt of Potassium</u>
   Kidney <u>regulation</u> Potassium
- Removal of kidney toxins KGP Flush
- Removal of Liver <u>toxins</u> <u>Magnesium Sulfate</u>
- Removal of Bacterial infection Herbs and copper metal that forces GI tract good/bad bacteria ratio
- Lymph stimulation Dodder Seed
- Clear the <u>Blood</u> System <u>Ginger</u>, <u>Tumeric</u>, <u>Oregon grape</u>, <u>Goldenseal</u>, <u>Barberry Bark</u>,
   <u>Saw Palmetto Berries</u>, <u>Bioperine</u>,
- Clear the GI tract: Oil of Oregano, Black Seed oil EFA'S, Fucoidan, LEM Powder, Chitosan, Caprillic Acid, Herbs, Berries, Bananas, yogurt, natural grains, flaxseed Ground, Spirulina, Chlorella,

#### **In General**

There are some common <u>beliefs</u>, like: <u>PinX</u> clears the GI tract of worms, <u>Invermectin IVM</u> <u>stops</u> roundworm muscle conduction, <u>Praziquantel PZQ</u> kills flukes, <u>ALB</u> transports <u>Praziquantel</u> deep into tissues. <u>DEC</u> attacks the Carbon chemistry and prevents larvae from malting. <u>Flagyl</u> blocks Luminal fermentation, bacteria enzymes, turns their innards black, <u>Alinia</u> attacks the iron chemistry of blood worms, and <u>Piperazine</u> kills babies in upper GI, and burns round adults in systemic infection.

#### **ELISA TEST**

It is not in the pattern match to a specific parasite, but in the specific physical symptoms, like organs being attacked, time cycles, and challenge tests that more precise identification of worm types can be surmised.

**ELISA** tests can discriminate between different worms or types (round or flat), but the science to identify specific species is still mostly in the lab and in the hands of the **WHO**. Truth be told PCR and DNA analysis is still mostly in research labs, it was available to the public, but now is very hard to find.

#### **IVM Test**

I found out I had parasites, by taking a single dose of an anti-parasitic **IVM.** I had tried everything else. The effect was so dramatic I knew at that moment what kind of illness I had.

The **IVM** challenge test is an example of a simple affordable test, where a single \$3.00 test dose of **IVM** is used to identify one has a "parasite infection". The affirmation test that has about 85% round worm specificity, and 15% flat worm specificity is a bold simple way to identify an underlying infection.

Simply stated if your symptoms significantly decline, think "parasite infection". A \$3.00 test dose is much more affordable as a gross parasite test, than the tens of thousands of dollars I spent for "examinations", which provided me with absolutely no diagnosis.

## **Nothing Worked**

To make a long story short, I tried every anti parasitic I would get my hands on. Nothing worked, naturals, pharmaceuticals, or combinations. I discovered I had an underlying <u>fluke</u> infection that prevented any treatment of Ascaris.

**The common practice** in parasitology research, is to **treat Ascaris first**. I found nothing to be further from the truth, and **flatworm** infection blocks roundworm treatment.

Treat the **flatworm** infections first.

#### Significance of Fluke infection

(Besides real and progressive organ damage)

Treating the underlying <u>fluke infection</u>, while holding the roundworm infection at bay, allowed me to progress from an untreatable infection, to a treatable one.

Now I have found a reaction to every anti-parasitic medication, several weeks after killing the **flukes**. A **fluke** infection kills.

## **Preamble Wrapup**

To wrap up this preamble, You will notice on day 68ish of my personal **log** section, I switched from **Albendazole** to **Alinia**. \* days later it seems to have done magic. The Ascaris seam to be no longer releasing eggs. The reprieve lasted for 6 days, until it returned. A single (One dose) of **Alinia**, and a day long kill again put Ascaris in reprieve. During this single dose, tiny sesame or caraway seeds (black) came out of the urine, with microfiliment things. **Flagyl** also seams to work

now as well, confirming to me that an underlying fluke infection, masked all my attempts at killing roundworms, even after I was tested ELISA positive.

## Log

It is a good idea to keep a daily journal.

I now keep a short form **log**, after getting used to the process.

Now the body responds correctly to medications, after the fluke infection is gone. At **Day 86** I am starting a 3 day course of **Flagyl** 500. Now I am in the final stretch. I am very hopeful that in a matter of days or weeks, the entire "parasite thing" will be a thing of the past. **Flagyl** is nasty, and tiny doses should be taken. I do **Alinia** for 9, **Flagyl** (2) doses spaced 12 hours apart. Then take a probiotic.

## Rehab

The battle of having no **immune system** function, massive toxin removals, and having a parasite **hyperinfection** seams to be over. Organs are in recovery, I am having to force my muscles to work again. Liver, **Kidney**, Sinuses, Lung all seam to be in recovery. The body is having problems moving.

I maintain the **<u>Day log</u>**, and help answer questions to those that need help digesting what I have learned.

It is in the **Log** section, that the challenge of my infection was finally won or at least put into stasis. I have been symptom free for weeks. **Lifetime** maintenance supplements of algae will be used to help to prevent cancer in my future.

Finally, feedback from others will help tune the process for specific parasites, identification of low cost home ELISA testing kits for specific parasites, and the confirmation of the minerals, metals, vitamins, and substances that were used during the parasite killing process.

Also to be determined is a list of qualified doctors, labs, and tests that one can use that do not break the piggy bank.

You will need to retrain your body to work and move again, after you have finished your final kill.

Feel free to keep your questions coming. I hope for you all it is just a matter of time for your total cure as well.

## **Revision Log:**

012416R240 100 page review in color markup information links 012016R218 Start release pdf update, document flow added 011016 Start final revision of notes, Bridge to Alinia Section added 010516R210 Start adding graphs, loading dose, current pattern 122615R195 Add Flowchart Diagrams 122615R194 After Fluke kill, Anti-parasitics now kill at will/work properly 122415R Add PinX Dosing Table 122114R184 Add Snake Oil section 12615R179 Quick Release to CZ 121615R174 Start cleanup 121215R163 Update LEM 120715R147 Include Stronglyloid section guick release of **SIBO** Release Date 120415 C133 Lower **Albendazole dose**, it clogs the kidneys. Release Date 112815 C121 added kidney section Release Date 112715 C112 Modified Ascaris dose sequence Release Date 112215 C100 Add PPZ dose PPM, update maintenance, test taper PZQ Release Date 112115 C79-91 Cleanup Release Date 112015 C74 Updated Weight based Dosing Tables added Release Date 111915 C72 Updated sequencing Release Date 111415 C59 Detailed Vitamin Section Revision 102615 - Staggered Praziquantel away from AM/PM to increase effectiveness, Ann Revision upd102615 P51 - General Public Release Revision 102015 c57 – 4th Release Private

## To Do:

\_Add <u>Alina</u> Dose \_Add **Flagy!** Dose Add MBZ dose Add **Ramp** graphics Add **Bridge** graphics Need to expand on **PZQ** timeline, ramp, graphics to show 3 tier/phase 3, 4, 5 PZQ total dose buy 300 Grams my weight-loss End of year emails Intro sect on PPZ and DEC Black pepper extract formula? \_Alinia plan for final kill, Detail again log 90 daily supplement extra liquid juices, vitamin C. Start recovery of old documents, add menu items Replace Vitamin A with Tomato Soup, Tomato Juice, Bananas, Apples Update Vitamin source on Dosing Schedule Create expected (I used) dose, buy quantity Calcium

### **Forward**

This is not medical advice, if you want medical advice ask a doctor, **I am not a doctor**. I am an unqualified electrical engineer, with a small amount of chemistry from 30 years ago. My thoughts are my own, and do not represent that of any recognized medical establishment. I assume no responsibility for the accuracy, or representations of others research, claims, or uses. My observations are mine, and mine alone.

## **Liability**

This document contains my process, formula, ramp sequence, dosing levels, dosing times, dosing durations, my taper <u>log</u>, maintenance, and research notes, personal notes, and excerpts from dialogs from others in the parasite self treatment process.

Much of the research may not be completely documented, have complete footnote or author assignments, and may include Research papers copied without permission, links that no longer exist, article excerpts, sources, and commentary that is incomplete.

## Q&A

This document also has **Q&A** commentary from private correspondence with CZ folks. I have acquired this information for informative purposes only.

I collected everything, and distilled the best of tens of thousands of papers into summary documents. These summary documents were distilled into plans and theory proposals, and were tested. These were processes and actions I performed for me, and me alone. This information is applicable to me only. I had a specific set of parasite infections.

This information may not apply to other kinds, types, or other infections. It is in the generalization of the health restoration process, techniques, and realizations I believe the value of this document resides. I reserve all rights to my deductions, formulations, and theories.

## This information is provided for information purposes only.

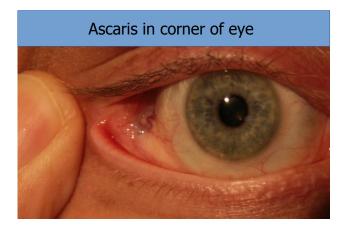
Any actions you take based upon this information is done at your own risk. Where ever possible safety information, and precautions are provided where information was provided by others. No liability or accuracy should be assumed by the reader or responsibility taken by the author, for actions you perform based on any of this information.



Migrans in the Right Hand-Picture presented to 2<sup>nd</sup> ID "Dr."

## **Ascaris 123**

**Ascaris 123** is my third attempt to find a formula to kill Ascaris (and as it turns out Flukes). There were two previous CZ formulas, and before that there were many unsuccessful attempts I made over the last year. I tried naturals, herbals, oils, leaves, roots, Chinese and global products, homeopathic substances, pharmaceuticals, combinations, and everything at once.



#### **Direction**

Nothing but <u>Invermectin</u> continued to remind me I was on the right track. Dose after Dose, and substance after substance gave me no relief. It was in understanding how high a dose was required, the sequence of parasite clearing, and in the identification of the parasitic families I was dealing with, that lead me to a cure.

I thought natural products were stronger or broader in spectrum, but all my tests show natual sources are too lame to be of any use, need stronger meds from natural or best science products list.

#### Research

I started pharmaceuticals with the standard doses and durations, then doubled them in dose and duration. I then tried combinations, natural and pharmaceuticals, and every permutation imaginable. I read what all the "people in the know" had to say. I found very little accurate information.

#### **Dose**

On the second CZ Formula attempt I took so much <u>Albendazole</u> (<u>42mg/kg</u>) that my hair fell out. I was unsuccessful. During that attempt, flat worm (flukes) came out.

- I adjusted formula number 3 to address a dual infection of flat worms and roundworms.
- Focusing on killing Flukes first became a stroke of genius.
- As it turns out, this was the key required to solve my puzzle. This dual kill formula worked.
- By combining non-traditional roundworm medications to control health and enzymes, and dosing long and tall for flatworms, I broke through the parasite infection log jam.

Never dose a medicine without knowing its effects, symptoms of overdose, and understand the long term risk.

#### **Some Success**

I was able to kill the <u>massive fluke infection</u>. This very aggressive and out of the box approach has changed everything. It appears now that the flukes are gone, all the traditional anti-parasitics are starting to work, the digestion system is returning to normal, and the body and the immune system are once again functional.

Formula #3 has **broken dam**, and things that should have worked initially now work as they should.

#### **NTD**

I read the other day what the definition of A Neglected Tropical Disease means. The definition is a Disease where less than 1% of the WHO budget is allocated towards the disease. If you read of someone using the term in your country, it holds no weight, and has no meaning within a civilized country, where government health officials frequently do not understand terminology.

What bearing does the WHO status have on the funding and recognition of a Disease within a civilized country?

None.

## Here is my Story:

In 2008 I had WHITE URINE, COLD HANDS, KNEE SWOLEN, NIGHT SWEATS, my Primary Care Physician (PCP) doctor #0 ran a physical, urine, ultrasound and many tests including Lyme and disease tests. I went to a stream of non-specific PCP doctors. Theory being one of these doctors would be able to identify a cause. Doctor after Doctor could not identify a cause. I got extremely cold in the winter (Nose and hands), and had wounds that would not heal, a lump in my throat that came and went. In October 2012 I got nosebleeds. They would not stop. By December my sleep was down to 4 hours, I was blathering. I had Alzheimer's like memory. I experienced a lot of acid reflux and GERD. I started taking high doses of vitamins. By January the world started to spin. Sleep fell to zero. My vision went dim, my senses numb, I shook like I had Parkinson's.

2010 - I started heavy physical activity, the more I worked, the sicker I got. I knew something was wrong, but I passed every physical. By 2012 diarrhea started, which took over a year and a half of

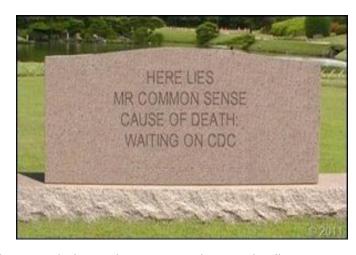
**SIBO SIYO** attempts to stop. By January 2013 I had red urine, orange urine, white urine, and I made another doctor appointment. I had blood in the stool, white stool, grey stool, shakes, muscle vibrations and could not sleep. Mucus covered stools freaked me out. I made an appointment with my primary care physician. By February I had electrical shocks in my head, burning around my mouth, restless leg syndrome, and started to fall to the floor. I was shaking badly. I kissed my wife goodbye and it felt like I had only a few days left to live. I smelled like a sewer pipe, I smelled CO2 and **ammonia**, and had black was coming out of every orifice. I thought I would get a diagnosis, I was wrong.

## **Stay Alive Formula**

I kept myself alive by taking 2 grams of **ALA**, 2 grams of **CQ10**, and 2 grams of **L Carnitine per day**. I took 8000 IU of **B12**. This simple formula commands the mitochondria to dump toxins.

Finally I got in to my primary care physician. He said he had to see 200 patients today; I had 5 minutes, Go! I told him my story and he offered me a prescription for a sleeping pill.

I saw in total 22 doctors, some of them better than others. I saw specialists,



integrated health centers, local herbalists, homeopath, hospital PCP, Researchers, and Collegiate experts. I researched non-stop for 3 years, corresponding directly with researchers, and university PHD doctors. I spent a year attempting to develop a formula to kill the parasites. Each formula failed.

I reached deeper and deeper into finding a handle on this thing.

I spent tens of thousands of dollars, bought every vitamin, herb, metal, amino acid, and substance, trying each one at a time, documenting the effects of dose, combination, developing another theory, and trying the next.

I developed and documented hundreds of combinations. Slowly but surely I identified key consistent common elements, minerals and metals, that helped and worked again and again. The same elements helped for bacterial, fungal, viral, and parasite infections.

#### SIBO and SIYO

Then I killed yeasts and bacteria overgrowth's. Finally I made significant progress.

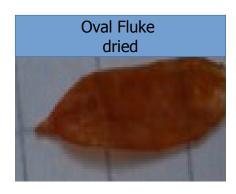
Only the vitamins I was taking kept me alive. It seamed that nothing helped clear the infection completely. At one point I took a cereal bowl full of vitamins twice a day to try to stay alive. Progress stalled.

Finally in August of 2014 I took one dose of **Invermectin (IVM)**. The symptoms abated for almost a day. I finally knew what I had. Parasites. I asked The Mayo Clinic in Rochester MN, Duke in Durham NC, and called 50 Tropical Disease doctors that are on the CDC list of care referrals. All of this effort was a waste of time. No one indicated they would help.

- I went to **PCP Dr #1** he offered me a script for a sleeping pill
- I went to the local **CFS guru, MD**, he measured my PH, tested thyroid, adrenal, put me on detox program.
- I located 3 local health food stores.
- I got local university research hospital, <u>Dr PCP # 2</u>
- I went to the local university hospital, ran tens of thousands of dollars of tests, GI and colonoscopy, Brain MRI, hearing (refused offer for catscan#1, refused offer for ear surgery), blood, nerve conduction study, (Refused offer for catscan#2). No problems found. <a href="PCP#2">PCP#2</a> recommended I see a Psychiatrist.
- Lungs filled with fluid, circulation stopped in arms and legs. Could not walk or drive. Took MSM, Baking soda, Stinging nettle root, magnolia bark and rose hip seed tea to stay alive.
- I demanded a visit to the ID **Dr #1**, found no fungal or AZ desert parasite infection, Immune panels showed nothing. (I appear to have eliminated SIYO infection at this point.)
- I took a single dose of <u>IVM</u>, I concluded I had a parasite infection.
- I went to <u>PCP #3</u> and got a Gastrointestinal GI Dr turned white, sat down, said he would not treat me, ran a test for Strongyloides and filiaria, he suggested I go to a teaching hospital.
- Took <u>PinX</u>, took pictures of my parasites, vomited for 3 weeks. Pics of round, tape, flukes.
- I went to my PCP, Dr #3
- I asked to see **ID Dr #2**, Took one look at the parasite pictures, he declined to treat me, and suggested I go to Mayo.
- I was refused by Mayo, PCP consultant said she needed a biopsy report from a pathologist.
- I was refused by Duke, said they were no longer treat parasite cases.
- I emailed the CDC who provided me list of both parasite associations ... A list I had already emailed and called to no avail.
- I called every USA based tropical medicine doctor I could fine, called research facilities, emailed university and parasite researchers, If I was not a child, they would not treat me.
- I became unstable, <u>PCP #3</u> wanted to medicate me with blood pressure medicine, I refused. BP 200/150, Pulse 120.
- Forced <u>PCP #3</u> to run a Ascaris test, came back positive, <u>PCP #3</u> told me and my wife I did not have Ascaris, even though the test result in his hand was positive and above the threshold. Offered me a one-way trip to the Psyc ward.
- I was on mv own
- I concluded that the medical establishment was a waste of time, and if I continued to pursue the establishment, it would lead to my death. I stopped all contact with Doctors, and cured myself.

## **Weight**

The attached formula is for a 113 kgs 258 lb male (Me). A lot of dosing of substances (especially antiparasitic substances) are meds tied to weight, and can be influenced by sex, and age to some degree. Most research has huge cautions when dosing these substances for persons under the age of 18. Since weight is used to calculate the dose of anti-parasitics, it is necessary to ascertain an exact weight figure.



I used my body in an experiment. Everything everyone else said should work, did not work. Step by step, process-by-process, test-by-test, I was in the slow lane, when I needed to be in the fast lane. I had to learn and relearn everything, even things I thought I knew about health, body processes, process loops, and methods to extract more performance out of the human body.

- Vitamin <u>supplementation</u>, to preserve the body and health while I did diagnostics, and problem identification.
- Metals and their function within the body
- Body chemistry, process loops, waste elimination.
- How to get <u>more performance out of the body DNA</u> and the bodies ability to respond to amino acids.
- Parasites
- Anti-parasitic treatments

There were no easy answers. I decided to do anything, I could do, I would do, short of killing myself, to find a cure. Research became my education, my mind focused on reason; my pain became an improvement level indicator. It was a crude, cumbersome, and slow process.

After 3 years of trying everything, I finally developed a formula that could kill parasites at will. I had a lot of help and questions from the CureZone, whose constant questions helped me move this crude formula and vitamin research into a successful, workable and repeatable healing process (according to others) from critically ill email correspondance. I had so much time invested in the research, I had to document my impressions of the kill process in more detail.



## **Dosing for Women**

Since <u>women are different</u>, I asked for and delighted for a fellow CureZone gal (I'll call Ann). Ann so kindly provided me the following guidance (feedback) for women. Ann has helped dozens of folks start the mineral, metal, and vitamin start-up sequence. Ann has gone through the <u>IVM</u> test, <u>PinX</u> clearing, and Anti-parasitic clearing of flat and oval worms. She asked more questions than anyone I corresponded with. She is a natural.

In total it appears that the starting formula for anti-parasitics are way to strong for women. Also I have been doing this for a while, and had several kills under my belt. I had cleared SIBO and SIYO infections, and was getting tough. Ann was hitting this process cold, and for the first time.

The dosing modifications for **Women's dosing** of start-up supplements and anti-parasitics are provided, so that two different viewpoints are represented.



Tapeworm Segments

## **Body in Balance**

No cure is possible unless your body is working properly, that is why the minerals, metals, and herbs and vitamins are provided.

I did not pull this out of my hat, I have 3 years of constant research, 80 gigabytes of stored research, I have read tens of thousands of research papers, tested everything in this formula to extreme dosing levels, and finally arrived at what I think was the proper and safe mix for me.

99% of the doctors do not have a clue. I am sorry this falls to you, but ultimately you are responsible for your health and life.

I had to figure this thing out on my own. There were only very few clues.

I found so many inconsistencies in the research, looked at history, associations, conference reports, looked at the people who were doing the research, the papers, and documenting the subject. I started to believe it was one big lie! So I researched parasites back up to to 3000 years ago.



Parasitologists treat children. I called 50 of them, they had one question, are you a child? Only children get a "cocktail". I read in a

pediatric journal, where the author substituted the word and in leau of the word or, when he discussed the way to defeat Ascaris. That was the vital clue that confirmed my suspicions. A cocktail is a mix of substances, and several meds taken together would be required to quell the infection.

If you read enough parasite **history**, you will realize treating parasites in the early days was using this root, or this or bush, and entire families were treated by this tea or by eating that plant. Hundreds of years were documented in books, that describes a simple process.

By the late 1800's and into the early 1920's, many of these natural substances were identified, researched, and



refined into repeatable chemical killing processes. Doctors equipped with microscope and a few books became the early parasitologists. They went around the world testing and curing tropical diseases. Later there were oils, and more powerful extracted substances, combined with extracts of black pepper, pumpkin, trees, roots, and berries, became the early medicines.

#### What is a Medicine?

Truth be told, most patent medicines at that time were derived from natural substances. A medicine is a dose duration of a specific molecule or coctail of substances that generates a desired effect.

The amplitude of a single molecule type can have a high level, making it a **dose**.

Some medicines have a group of chemicals, like white willow bark, that unlike asprin, has a spectrum of substances to perform all the roles of the natural compound, towards disease, and not just a headache.

A medicine is typically only a single molecule type extracted from a broad spectrum natural compounds, or a chemical substance. Early medicines contained many substances of a natural plant.

As testing identified the single active substance in a plant, extraction and chemical processes replaced a simple extraction with chemical processes which produced higher concentrations of a single active ingredient. Many studies show these simple single molecule medicines may not perform as well as the extractions of the original natural medicine.

#### **Trials and Studies**

With patent medicines came trials and science. Then came the mass deployment of anti-parasitics made by pharma companies into the herds of animals. This science developed from the simple early days of herb teas, to become a chemical, then medical industry.

#### **Field Research**

Human anti parasitic patent medicine treatment progress came at a slower pace, with early stories of cures, and deaths. This evolved into today's politically correct view, which is to ignore disease, or labeling it as a "neglected tropical disease". Sad since we have the technology, just not the will. Slurs such as using the Poor, under developed countries, or certain races of people are so often used in mainstream research by so called "professionals" it makes me physically sick to read this slander. It is intended to convince the public they are not at risk, which is a centuries old thinking which serves no one, and is a huge dis service to everyone. Such narrow minds in the year 2015. If it were up to me, anyone issuing this slander would be instantly fired.

#### **Myths**

Typically all the modern books and documents of today call for 3 capsules of this or that. Even the CDC is so misinformed they repeat the same garbage. The typical definition of a "Cure" means that a doctor scripts 3 pills, the <u>stool is tested and shows no eggs</u>, and the patient is declared cured. **This is total parasitology bullshit**. Clearing the GI tract of parasite eggs is only the first step of a cure.

## **Stool Sample Test**

Let me repeat Parasitologists that I have read base all treatment, identification, and proclamation of a cure based on a simple optical stool sample test.

The stool sample only tells you what is going on in the GI tract. This is a useless metric set up by doctors a hundred years ago. This negates the ELISA and PCR test technologies that are used by modern medicine researchers, and several parasite test labs.

I started looking at the science, the process, and the assumptions. The entire discipline started to look like a house of cards. Testing the stool and declaring some one well or cured is total CRAP!

## The 3 pill theory

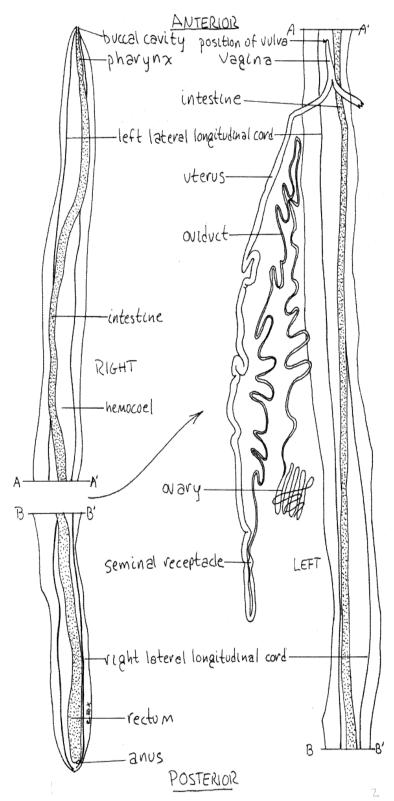
An infection in the GI tract is completely different than a systemic infection. The 3 pill theory only works to clear eggs from the GI trac.

Again this treatment was based on a 100 year old metric, and a 100 year old cure criteria. Only a retarded doctor would read and do this process.

## **Systemic Infection**

After a few hours or days parasite infections disseminate. The dosing and duration for systemic infections are much higher, or longer, or both.

The art of parasitology is so backwards, that resistant strains have been put in place, making the treatment of a parasite by 3 pills of anything like putting 3 drops of water on a house that is on fire!.



When ever you see the claims for treatment for 3 pills of this or that, keep in mind, **they are talking about An infection that has not yet disseminated**. This is parasitology fantasy land.

For disseminated infections, the protocol, formula, and plan are completely not covered by any study, plan, or research paper you read.

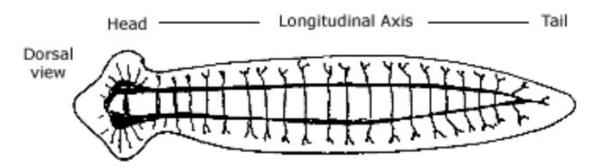
They know not what they preach. They have no field experience. Only 0.1% of the papers I read, and acknowledge the truth. Infections in the GI are easy to treat, once worms travel, to organs, tissues, and systems, they are hard to treat.

Early success from the 1600's and 1700's were refined by 1880, and 1890. This knowledge was again refined to attempt to successfully treat parasitic infections.

These early approaches and methods were replaced by Oils (Oil of chenopodium), Cina, Santolina, and substances that had quite a kick. By 1915, metrics, and success analysis were in place. After several public studies and deaths, Parasitology were relegated to the scientific field of zoology.

Noted Parasitologists wrote papers stating it was not necessary to treat parasite infections. When actually tested, newer substances did not really work safely, and killed many documented cases. The CDC removed the words "'Parasite" and "'bug" in 1938, and by 1945 doctors were forgetting the knowledge of previous generations. Now the CDC uses a "'cover story" of it's own history, calling parasites "Neglected Tropical Infections". The CDC is part of the same medical establishment that uses sick people as piggy banks, prolonging the pain, misery, torture, and death that parasite infections afflict. Shame, Shame, Shame.

Newer Meds finally started to emerge in the 1970's, and were sold to the tune of millions of tons for use on farm animals, cattle, sheep, chickens, goats. Medicines also were released for Vets to treat cats, dogs, and fish. Human trials ended in failure or death. The field of Parasitology never recovered. Failures and success stories were removed from most of the literature (including the NIH database), and were replaced by the 3 pill dosing cover story.



3 pills? Hell, I could do the same thing with a single dose of Reese's **PinX** and bypass all of the medical yahoo's. This "Cure" metric of stool eggs is the most stupid measure of success I have ever heard of. Repeated ELISA or PCR tests being negative means you are cured. Optical stool tests are useless.

Once parasites move into the body, doctors are so ignorant and clueless of the systemic cure processes you might as well ask a veterinarian, CATTLE RAISERS ASSOCIATION, U.S. Poultry & Egg Association, Dairy Association, North American Native Fishes Association, American Water Works Association, or an old fashioned farmer - on how to treat an infection, their answers will be more accurate and truthful than the medical profession in the United States of America.

The medical system is so dysfunctional, and information over the Internet is so easily available it falls again to the sick individual to determine their own diagnosis, write their own prescriptions,

procure their own substances, and administer a cure to easily treatable conditions. The medical system is so broken.

I suggest a consultation with a unicorn, snake oil salesman, or blood letter would be vastly more educational and cheaper than anyone employed in the medical profession.

Going to your doctor, local university hospital, or expert specialists will usually result in blank stares, statements like there are no parasites in the United States, or I am not qualified to diagnose you, find a doctor that treats tropical disease parasitic infections, a teaching hospital, or ask the CDC.

So it is left up to the sick, infected, the dying, and barely functional patient to be knowledgeable about an extremely complex and mystic subject as parasites.

It is left up to the sick person to determine whether a parasitic infection is likely the cause of illness.

It is left up to the sick person to self-administer some remedy or allopathic medicine.

It is left up to you to cure yourself. Why do we have medical insurance?

## **Emergency Fluke Treatment**

The combination treatment using **Albendazole** (**ALB**) and **Praziquantel** (**PZQ** or **Prazi**) appears in very few research papers, but I, and others have found it very very effective for flatworms. A parasite infection is the result of an exposure and a depressed immune system. Parasites are experts in altering your immune system. One parasite dramatically changes the immune system. Boosting the immune system during treatment is mandatory. Smaller roundworms may be excited by this treatment, and an additional low level anti-parasitic may be required to control acid reflux births. Excessive GI pain may be noted, that poorly improves with GI oils, butter, calcium, flax, spirulina supplements.

#### Q&A

**Q>** The flukes have to be the most awful of them all, theyre like cactus inside me. My hair is coming out alot also. I guess tea tree oil is best for the scalp ones? i poured so much on my head my scalp burned and peeled. I pray your protocol ends this nightmare.

**Q>** Hi matt, several people on cz say theres no info on a cloris fluke on the web, i googled it and it was all in german.do u happen to have a pic of this fluke? Mine arent coming out my scalp, atleast not yet, but they are very prickly and ive lost half my hair. Being female the hair loss is devastating. Ive attached a photo of a cz new girl that is also dealing with massive hair loss. Any discoveries you might have on regrowth is appreciated, the pic is not me, it is a friend also battling parasites and hair came out b4 taking any meds.

**A>** <a href="https://en.wikipedia.org/wiki/Clonorchis\_sinensis">https://en.wikipedia.org/wiki/Clonorchis\_sinensis</a>

sorry for the mis spelling

## **Anti-parasitics in General**

<u>Albendazole</u> slowly puts worms (round worms primarily) to sleep. Research shows it blocks sugar from feeding the worms. **MBZ** blocks carbs. **Praziquantel** tends to paralyze worm suckers (Round

or Flat). <u>DEC</u> causes internal worm damage. <u>Piperazine</u> (<u>PPZ</u>) paints the parasites for your immune system to see small worms, cysts and eggs. <u>PPZ</u> damages the innards, <u>and aids in the</u> <u>removal of uric acid - nitrogen wastes.</u> <u>Alinia</u> damages internal worm organs, and appears to effect the reproductive function of Ascaris.

#### **Q>** I cannot get Piperazine citrate in my country.

A> If you cannot find **Piperazine citrate**, try finding bulk black pepper or black pepper oil. You can make your own "essential oil" grind up Organic un radiated **black pepper seeds**. If you run the seeds through a steam oil distiller you will make Piperazine oil. The reason I use Piperazine citrate is that it is nearly impossible to get enough of the natural compound. If you get a herbal oil still, you can make your own. Dosing has yet to be determined.

Whole spectrum can be made by setting the black pepper in glycol. Glycol is available by the gallon. The Citrate "salt" form is more stable, and maintains freshness.

#### **Nutraceutical brief**

#### Herbal medicine is the oldest form of medicine.

- Core minerals, metals, vitamins
- Herb, plant, Latin, area, anecdotal reports
- Studies, compounds, identification, test, trials
- Whole herbs, roots berries, bush, tree
- Parts, seed, leaf, pistol, root, bark, core
- Capsules, pills, liquid
- Extract Co2, distill oil, water, glycol, alcohol
- Spectrum of compounds in herb can be strengthened and selected by using different extraction method or base extracting compound.

#### Stabilizers:

Dry

Extract

Base

Glycol

Alcohol

Oil

Salt

#### **Piperazine**

https://en.wikipedia.org/wiki/Piperazine

Piperazine is freely soluble in water and ethylene glycol, but insoluble in diethyl ether. It is a weak base with two pK<sub>b</sub>s of 5.35 and 9.73 at 25°C.; the pH of a 10% aqueous solution of piperazine is 10.8-11.8. Piperazine readily absorbs water and carbon dioxide from the air.

Salt can be made by adding citric acid

Two common salts in the form of which piperazine is usually prepared for pharmaceutical or veterinary purposes are the citrate,  $3C_4H_{10}N_2.2C_6H_8O_7$  (i.e. containing 3 molecules of piperazine to 2 molecules of citric acid), and the adipate,  $C_4H_{10}N_2.C_6H_{10}O_4$  (containing 1 molecule each of piperazine and adipic acid).[2]

#### STOP!

**Invermectin (IVM)** 

temporally "stops" worms, and stops babies. Invermectin works against a high percentage of round worm species, and several flat worm species.

Invermectin should be used with restraint for large worm infections, when the worm stops, it ceases to absorb antiparasitics, making the elimination of worms more difficult. Invermectin does not kill parasite worms, it only affects babies, L1 larvae, but not adult or Juvie worms.

**Invermectin** kills virtually nothing but eggs larvae 1 babies, it does not kill a worm, infact it attenuates worm



killing. **IVM** essentially says Stop and Sleep to roundworms for up to 24 hours. **IVM** is useful system wide for these limited functions and roles of sweat. IVM may cause parasites from feeding.

If you are taking **DEC**, **ALB**, or **Prazi**, then they are being feed anti-parasitics. **IVM** makes them stop feeding anti-parasitics. **Piperazine** also stops parasites from feeding, makes them hungrier for blood, and hence when **Piperazine**, they ingest more anti-parasitic meds.

Taking **Invermectin** during a birth of baby worms can be quite useful.

Certain Species of Strongyloides respond to **Fenbendazole**, @ 2.5mg/kg/D. Its safety is still in question.

## Fixing your body first

Parasites change your body into a huge worm factory, change your lipids, PH, suck you of your metals, and minerals, overload you with toxins, and crush your immune system. If you have a runaway systemic infection, your immune system has been affected significantly; your minerals, calcium status, nitrogen loops, toxin levels, and chemistries have all been manipulated by the parasites. That is why antiparasitic Meds do not work without balance and the other co-factors.



#### **Anti-Parasitic Dosing**

Anti-parasitics are Meds, that are dosed by the measure of milligrams per kilogram of weight (mg/kg) per day. Milligrams to Kilograms ratio is a measure of drug weight/to the weight of a person. This ratio defines a dose. When this ratio is multiplied by a persons weight, it defines the amount of the substance. The administration of a dose is defined also over a period of time, such as a day, so dosing is defined to be how many milligrams per kilogram of body weight per day (mg/kg/D).

### **Deciphering Scripts**

Scripts can be written as instructions, such as TID, SID, BID or QID, Three times in a day, Three Dose in a Day, Twice half dose in a day, or four quarter doses in a day.

**q.i.d.** (on prescription): Seen on a prescription, q.i.d. (or qid) means 4 times a day (from the Latin quarter in die). The abbreviation q.i.d. is also sometimes written without a period in capital letters as "QID". However it is written, it is one of a number of hallowed abbreviations of Latin terms that have been traditionally used in prescriptions to specify the frequency with which medicines should be taken.

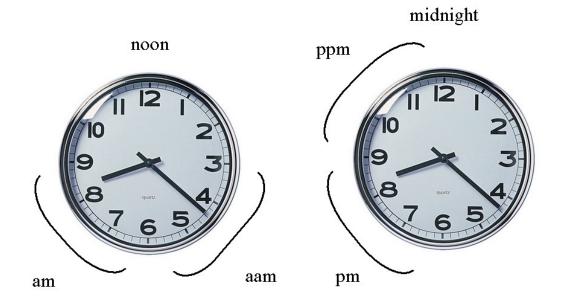
https://en.wikipedia.org/wiki/List of medical abbreviations: Latin abbreviations

Abbrev.	Meaning	Latin (or New Latin) origin
a.c.	before meals	ante cibum
a.m., am, AM	morning	ante meridiem
b.d.s, bds, BDS	2 times a day	bis die sumendum
b.i.d., bid, bd	twice a day / twice daily / 2 times daily	bis in die
gtt., gtt	drop(s)	gutta(e)
h., h	hour	<u>hora</u>
h.s., hs	at bedtime or half strength	hora somni
ii	two tablets	duos doses
iii	three tablets	trēs doses
n.p.o., npo, NPO	nothing by mouth / not by oral administration	nil per os
ad ad OD	once a day	omne in die
o.d., od, OD	<u>right eye</u>	oculus dexter
p.c.	after food	post cibum
p.m., pm, PM	afternoon or evening	post meridiem
p.o., po, PO	orally / by mouth / oral administration	per os / nonstandard form per orem
p.r., pr, PR	rectally	per rectum
p.r.n., prn, PRN	as needed, (also Pertactin - a key antigen of ac.Pertussis vaccine)	pro re nata
q.	every	<u>quaque</u>
q.1.d., q1d	every day	quaque die
q.1.h., q1h	every hour	quaque hora
q.2.h., q2h	every 2 hours	quaque secunda hora
q.4.h., q4h	every 4 hours	quaque quarta hora
q.6.h., q6h	every 6 hours	quaque sexta hora
q.8.h., q8h	every 8 hours	quaque octava hora
q.a.m., qAM, qam	every morning	quaque ante meridiem
q.d., qd	every day / daily	quaque die

q.h.s., qhs	every night at bedtime	guaguo hora compi
q.ii.s., qiis	every flight at bedtime	quaque hora somni
q.d.s, qds, QDS	4 times a day	quater die sumendum
q.i.d, qid	4 times a day	quater in die
q.h., qh	every hour, hourly	quaque hora
q.o.d., qod	every other day / alternate days	quaque otra die
q.p.m., qPM, qpm	every afternoon or evening	quaque post meridiem
q.s., qs	a sufficient quantity (enough)	quantum sufficiat
<b>x, Rx,</b> , R	prescription	recipe
Sig., S.	<u>directions</u>	<u>signa</u>
Stat.	immediately, with no delay, now	statim
t.d.s, tds, TDS	3 times a day	ter die sumendum
t.i.d., tid	3 times a day	ter in die
u.d., ud	as directed	ut dictum

#### **Time - For this document:**

PPM = Past 6 PM AAM = Before 6 AM



## **Supplement Dosing Schedule**

Supplements are Minerals, Metals, and Vitamins. Dosing of each supplement has a range (Minimum – Medium – Maximum) (SP = maximum "special purpose" dose in a day). Normally one starts low, and ramps up to the maximum dose, then after completing that **phase**, moves to the "medium" or "typical" dose, and stays there for another 30 days. Upon completing that **phase**, the dosing moves to the minimum level. Thereafter a **maintenance plan** is provided to continue the cellular repair process, and maintain damaged DNA in cells until programmed cell death, new cell generation, and for the amount of time it will take for cell replacement (up to 7 years!)

 After working up to the maximum dose, (days or weeks) every 30 days I would step down a level.

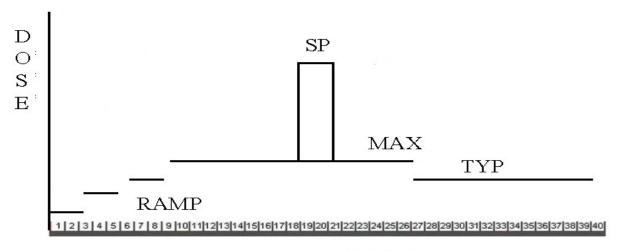
At the conclusion of  $\sim$ 90 days (everyone is a little different), I would go onto the supplement items listed in the **Lifetime Maintenance Section**.

Certain <u>elements</u> are pro human body, pro parasite. It is best to pulse these elements. Examples are <u>ALA</u>, <u>Vitamin A</u>, and <u>Zinc sulfate</u>. These are best done at a high level, over a short period of time. Later on, you will notice that a complete elimination of these supplements from the formula, this is done after the initial <u>fluke</u> kill. These are tricky supplements, that one needs to get the knack for. <u>Pulsing</u> means skipping day(s) and taking a high level of the supplement.

Long **term** supplements like **vitamin C**, **Spirulina**, and **LEM** extract will be used in the maintenance section, which will provide a lifetime of parasite immunity. These substances are approached differently.

#### **Dosing and Ramp**

Never take a medicine unless you understand that its effect may not be felt for 3 days after a dosing level is started. Steping up the level and pausing for 3 days is required to feel the effect. Do not ramp to quickly.



DAYS

## **Safety**

I have tested everything on this list at doses that way way exceed the levels in this paper, and what I took. Just because I tested these substances at 2-3X the documented levels, does not mean it will be <u>safe</u> for you.

It is still recommended that anyone pre-tests the introduction of each and every new substance or supplement, you incorporate into your daily routine. **One by one, and at low doses**, start each new substance in a **test dose**, to determine if there is an adverse **reaction**.

**Starting more than one substance or supplement at once**, prevents one from knowing which substance caused an **adverse reaction**. I suggest adding a small amount of each substance, one at a time, to determine if there is an **adverse reaction**.

I now (after concluding the big kills) dose much closer to the **minimums**, as I have killed just about everything, **Ascaris**, Fluke(s) (I had 3 types), tapeworms, etc.

Certain <u>vitamins like A, D and metal zinc</u> have cautions and <u>transitions and tempo</u>. I suggest you read all the information on these substances.

#### **Brute Force Scientific Method**

Early on I derived a set of assumptions, processes, methods to identify my disease. I started by scanning and testing the body, blood work, physicals (I had 8), and sought a number of medical processes and approaches. I eliminated, and fixed, fungal infections, bacterial overgrowth, mineral, and metal imbalances, and tried to heal the DNA using algae. I pushed the problem around, still I got no better. No matter what I tried, I failed to cure. As soon as I stopped vitamins, I got worse, and went down hill quickly. The scientific process works, sometimes just not work that quickly.

Process	Identification and Sensitivity Test of all the elements	Group the related elements into	Formula 1 Test the combinations together	Formula 2 Test the Combinations , revise the mixes	 Sustain the formula to ensure every virus cell is dead	Taper to 0 slowly ensureing the best outcome
Hypothesis	Test all the foods, vitamins, minerals, enzymes, drugs, in my inventory, one at a time, increasing and decreasing the dose to find out what lowers pain level	combinations (mixes) and determine what combinations and quantities are required to work together	Formulate – develop test formulas and try the entire coordinated formula at once		sustain the formula to ensure the virus has been completely eliminated	
Results						
Conclusions						

The <u>trial and error method</u> produced a number of red herring test results, including kills of <u>SIYO</u> yeast using <u>Caprylic acid</u>, <u>Beta Glucan</u>, and <u>Turkey Tail</u>, kills of bacterial overgrowth using <u>HUmineral</u>, <u>Chitosan</u>, <u>Deoxycycline</u>, and others. You may feel better but you know you are still sick.

This early progress stalled as I found no other bacteria or yeast infections to kill.

All of these early successes were misleading and confusing to me. I had yet to test and identify I had a parasite infection. A single dose of **Invermectin** informed me of what I had been avoiding and fighting, a parasite infection.

#### Research links

http://www.microbeworld.org/podcasts/this-week-in-parasitism

#### **The Final Formula**

In August of 2014 a **IVM** test dose proved I had a parasite infection. I took one "**Test Dose**" and all my symptoms abated for 24 hours. I ran doctors testing, and my own research in parallel. It was a long search that did not end with the knowledge that I had a roundworm infection. After trying everything I seam to have cleared my infection in a matter of weeks, after a year of testing antiparasitics, documenting the test effects, discussing this with a small circle of friends, and then reformulating the approach.

When your vision comes back, and you start dumping black, you know you are almost home free.

The **Dodder seed** (**Cuscuta**) stimulates your lymph system to help move out the black junk. When you finally generate a real kill, there is very little doubt as to the effectiveness of the formula.

Good Luck, <u>I'm finally back</u> to working, and wrapping the whole parasite thing up, finally. I am moving on with my life, and look forward to one day publishing what I have learned. I have several longer versions that explain what each mineral, metal, herb does, do not hesitate to ask questions if you need to know <u>how</u> this formula actually works.

I am in the process of moving all of my **research** documents into "Short and Sweetn – How I did it!", making it longer and more detailed, and incorporating research for different kind of parasite infections. This material may not be complete or up to date, new parasite research is being generated on a daily basis.

#### The Formula for Women

Ann needed to **keep working** so she did all three phases at once, start-up, minerals, metals, herbs, clearing the GI trac, and anti-parasitics. I normally

suggest starting with PH herbs, then clearing the GI with the **IVM/PinX**, then doing a tailored anti-parasitic dosing. Ann then did all four phases at once. These phases in parallel stressed her detox system to the max.

The GI system is calmed and made to run again using Magnesium Citrate, Spirulina, Chorella, Charcoal, Magnesium sulfate, Probiotics, and yeast or bacteria killers, like Caprylic acid, Chitosan, Saccharomyces boulardii, etc. Without any more of my preface, here is how Ann adjusted the formula for her:

### **Dosing for Women (Ann):**

I tried to do all 3 phases, start, minerals, metals, herbals, clear the GI tract, and start anti-parasitics at once. It resulted in a toxic overload to my system.



Also, it seems like such high doses **<u>Piperazine</u>** aren't as needed when you start **<u>Albendazole</u>** and **<u>Praziquantel</u>** together. I am not sure why that is possible.

I am now 127 lbs, 5'8

I cut down **Piperazine** even more yesterday. The brain feels much better today after 2 light days of meds.

AM Morning I am on 140mg Piperazine

**Noon** 200 mg <u>Albendazole</u> **Noon** 1/2 tsp <u>Praziquantel</u>

**Noonish** 3/4 wafer **DEC** (usually closer to one, I try to take w food)

PM 100 mg Albendazole

PM (before bed) 140 mg Piperazine

I took the 1/2 tsp **Praziquantel** once, at noon, instead of separating it into two doses of AM/PM.

I am still playing with this but will definitely hold for a while until I'm stronger. I'll let you know of any updates or thoughts I have.

Currently, am dosing **IVM** only when I need a break from **ALB** or if I feel crazy bursting, which I haven't felt for a while since (I think) I was out of zinc for a bit. I still take the starting dose of 600mg/day, which is high, and I still take the 4 teaspoons of **MSM** powder in water a day.

#### **Update 4 weeks:**

Q.>I am also dumping tons of black liquid after normal stool. Have to stay home til it stops around 5 every day. Bowels still somewhat locked, mornings difficult but lately it's been eventually moving then toxins. Can't believe how much. Every once at a while I see a tinge of green then its back to black.

My GI has been completely empty the last few evenings, its hard to eat its so raw and sensitive. Needs to heal, but its hard w all the shit continually coming out.

I have increased meds slowly am up to 280mg **PPZ** am/pm 300-400 mg **ALB** noon 1/2 tsp **Prazi** noon 3/4-1 wafer **DEC** (400mg)

#### **Update 6 weeks:**

Back at it today. 380 mg **PPZ** this morning, the max I handle at this phase.

Can still only handle once daily dose <u>ALB / Prazi</u>, as it can cause bowel unlock and dump and I need to be standing, moving around the house for that. Did 500mg <u>ALB</u> and just over 1/2 tsp <u>Prazi</u>, felt stronger, ready for the slight increase.

**DEC** still makes me feel messed up, freezing cold, tired, not thinking totally clear. I am still between 1/2 -3/4 (early eve, can't take in the middle of toxin dumping). But have gone up to

1 1/2 tsp **PPZ** am/pm

600 mg **ALB** afternoon w

3/4 tsp Prazi

Bowel regular, using **Iodine**, lugols. 8-10 drops, 3x per day

Backing down on **Zinc** and **MSM** 

Started grinding **Flaxseeds** for mixing with the yogurt.

I am still having at this crazy dumping. The **IVM** is effectively a pause that was my only day of not dumping, going through this in the last ten.

It's pretty crazy intense, dealing w this bowel lock and body dumping toxins at once. I wait to eat, usually at 6 or so til its done, but in the meantime am dosing lots, and lots of **Spirulina**.

#### **Update 8 weeks:**

So, **PinX** did a real number on my gut and, not surprisingly for me, the colon is really affected by trying to deal w the fallout/toxins.

Trying to go back on meds the next day was too soon, the day after that it was too much for my body trying to move out toxins. I feel my parasite load much decreased. Colon needs to move out toxins and heal. I have had to work the last couple days and have to be busy on Thursday, Friday so I devised a plan.

I have been dosing **IVM** starting yesterday going to do just **IVM/PPZ/DEC** to keep things at bay thru Friday, when I work, then hopefully back on **ALB** and **Prazi** Saturday. I need to let my body balance a bit before starting in on a serious kill again. Also I believe by letting things settle I'll get a sense of where I am.

I do not have such a need for supplements at the moment, I think my body is re learning how to balance and fight on its own. You said that once the bowel is so affected it can take a while to heal:

**Q.>** Are we talking years or months once I've moved the most serious bunch of toxins? It would be great to have some idea because this affects my life so much.

My GI tract continues to improve, I think the living and dying worm enzymes cause most of the problems, once they are dead, it takes a few days to weeks to clear out the debris.

 $\mathbf{Q}.\geq$  is this just because my body has been so severely sick and affected by this toxicity and my colon took much of the stress? I am assuming that it's a bit luck of the draw, ie you had to deal w your kidneys shutting down on and off, for me its my colon. >>>>am I correct in this assumption? Because I know not everyone dealing with parasites at this level is affected the same.

I had an infection for 8 years, it just took to so long for me, and I had to many kinds of parasites. I think this is a matter of severity. I have many people worse than you, Cannot walk, no vision, etc. I can only say that it passes with time. The vits are explained now in the document, and how they work.

Q.>So the plan is IVM am/pm
PPZ 1-2 tsp AM/PM
DEC 200-400mg mid point between IVM dosing
Doing this and less supplements to help my body balance and detox on its own Does this sound OK?

Yes it sounds like a time out, and will keep babies from growing/living, you have the hang with this thing!

**Q>** Then I will remove **IVM** and reintroduce **Prazi** 1/4 tsp AM, **alb** 200 noon and slowly increase from there as I can handle it.

> I would like to suggest using my latest second 30 day formula, it takes about 3 days to kill, I take <u>ALB/DEC/Piperazine</u> at AM/Noon and <u>Prazi</u> in yogurt PM and PPM. It was 3 days of hell, but it kills the fastest. <u>Prazi</u> pinches their mouth, they get hungry, then wham, scoot them the Meds, They ache, and twitch, then you stink, they die.

Parasite load definitely much better. But interestingly enough, brain and right foot where I feel most activity.

#### **Update 9 Weeks:**

Hey!:)

I feel absolutely incredible today, still colon issues, but I think that will be a bit slower healing as I've done so much in such a relatively short time. I am walking right now in the sun, been walking at a good pace almost an hour and not tired at all.

I feel more and more and more like myself again and have more and more moments of such joy to be alive.

The last few weeks have been the first time I can say 100% I am not going anywhere. It's a good feeling.

I am going back on stronger meds today. I will follow your new formula as close as I can (only first dose to begin **ALB and Prazi**)

I just took 4 days off <u>ALB/Prazi. PinX</u> even though still hard on me, showed me that my GI is relatively clear. I think you're right, at this stage it is most likely not necessary. I don't foresee having to take it again.

Break days I kept things at bay with **IVM PPZ and DEC** (as far away in time from **IVM** dose as possible)

My body is very very strong. After that crazy dumping phase I had my immune system is really kicking in.

I feel good to be alive.

When I started this and kept saying thank you, you told me not to thank you yet.

Even though it's not over I have survived and know how to continue to get better.

So, thank you.

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#### The Truth

Health Spending in U.S. Topped \$3 Trillion Last Year, an average of \$9,500 a person

http://www.nytimes.com/2015/12/03/us/politics/health-spending-in-us-topped-3-trillion-last-year.html?
mabReward=A7&module=WelcomeBackModal&contentCollection=Politics&region=FixedCenter&action=click&src=recg&pqtype=article&r=1

#### Water

http://www.disabled-world.com/health/water-diseases.php

Synopsis: Information regarding types of water-borne diseases caused by water people drink that is contaminated by animal or human feces.

#### **Definition: Water-borne Diseases**

Waterborne diseases are caused by pathogenic microorganisms that most commonly are transmitted in contaminated fresh water. Infection commonly results during bathing, washing, drinking, in the preparation of food, or the consumption of food thus infected. Various forms of waterborne diarrheal disease probably are the most prominent examples, and affect mainly children in developing countries; according to the World Health Organization, such diseases account for an estimated 4.1% of the total DALY global burden of disease, and cause about 1.8 million human deaths annually.

#### **Main Document**

Water-borne diseases are any illness caused by water people drink that is contaminated by animal or human feces, which contain pathogenic microorganisms. The complete picture of water-associated diseases is complex for some different reasons. Over a period of decades, the picture of water-related human health issues has become more and more comprehensive, with the emergence of new water-related infection diseases and the re-emergence of ones we already know about.

Data is available for some water, hygiene-related and sanitation diseases such as cholera, salmonellosis, or <a href="shigellosis">shigellosis</a>. Yet for others such as schistosomiasis, malaria, or more modern infections such as SARS CoV or legionellosis the analysis has yet to be performed. The burden of a number of disease groups may only partly be attributed to water determinants. Even where water has an essential role in the ecology of diseases, it might be difficult to pinpoint the relative importance of water components of the local ecosystem.

#### Water-borne diseases include the following:

- Polio
- Malaria
- Cholera
- Dengue
- Scabies
- Typhoid
- Anaemia
- Botulism
- Fluorosis

- Trachoma
- Hepatitis
- Diarrhea
- Giardiasis
- Ascariasis
- Trichuriasis
- Arsenicosis
- Malnutrition
- Legionellosis
- Leptospirosis
- Schistomiasis
- Dracunculiasis
- Onchocerciasis
- Lead poisoning
- Cryptosporiodiosis
- Campylobacteriosis
- Lymphatic filariasis
- Hookworm infection
- Ring Worm or Tinea
- Methaemoglobinemia
- Cyanobacterial toxins
- Japanese encephalitis

#### How Big is the Issue

In developing countries, four-fifths of all illnesses are caused by water-borne diseases, with diarrhea being the leading cause of death among children. The global picture of health and water has a strong local dimension for approximately 1.1 billion people who still lack access to improved drinking water sources. Around 2.4 billion people on Earth have inadequate sanitation. There is strong evidence that sanitation, water and hygiene-related diseases account for around 2,223,000 deaths each year, as well as an annual loss of 82,196,000 Disability Adjusted Life Years (DALY's).

The World Health Organization estimates indicate that worldwide, more than 2 billion people are infected with schistosomes and soil-transmitted helminthes. Approximately 300 million people experience serious illness due to this fact. Malaria; for example, kills more than a million people every single year and a large percentage of them are under the age of five - mainly in Africa south of the Sahara. In the year 2001, the estimated global burden of malaria amounted to 42.3 million DALY's, constituting 10% of the overall disease burden in Africa. Malaria causes at least 396.8 million instances of acute illness every year. Pregnant women are the main adult group at risk. As one of the major public health issues in tropical countries, it has been claimed that malaria has reduced economic growth in African countries by 1.3% every year for the past three decades.

Approximately 246.7 million people around the world are infected by schistomiasis and of this population, 20 million people experience the consequences of the infection, while 120 million people experience symptoms that are milder. An estimated 80% of transmission happens in Africa, south of the Sahara. Diarrhea occurs around the world and causes 4% of all deaths and 5% of the health loss due to disability.

Bangladesh alone finds around 35 million people being exposed on a daily basis to elevated levels of arsenic in the water they drink, which will eventually threaten their health while shortening their life expectancy. Following the tsunami in Asia on the 26th of December in 2004, people faced the threat of water-borne diseases linked to flooding such as:

- Malaria
- Cholera
- Shigellosis
- · Hepatitis A
- · Dengue fever
- Leptospirosis
- Typhoid Fever

#### **Transmission of Water-Borne Diseases**

Water-borne diseases spread by contaminating drinking water systems with feces and urine of infected animals or people. The spread of contaminated water is likely to happen where private and public drinking systems get their water such as surface waters - creeks, rivers, lakes, and rain. These sources of water may be contaminated by infected animals or people. Runoff from:

- Landfills
- Sewer pipes
- Septic fields
- Industrial or residential developments

May also spread contamination, which has been the cause of a number of dramatic outbreaks of fecal-oral diseases such as typhoid or cholera. There are a number of additional ways in which fecal material may reach a person's mouth such as in food that is contaminated, or the person's hands. Generally, food that is contaminated is the one most common way people become infected. The germs in feces may cause the diseases by even slight contact and transfer. The contamination might happen because of floodwaters, septic fields, water runoff from landfills, and sewer pipes.

The one way to break continued transmission of water-borne diseases is to improve the hygienic behavior of people and provide them with basic needs such as:

- Sanitation
- Drinking water
- Bathing facilities
- Washing facilities

Transmission of malaria is facilitated when large numbers of people sleep outside in hot weather, or sleep in homes that have no protection against mosquitoes. Malaria mosquitoes, bilharzias snails and tropical black flies can all be controlled with efficient drainage; they all depend on water to complete their respective life cycles.

#### **Preventing Water-Borne Diseases**

Clean water is a prerequisite for reducing the spread of water-borne diseases. It is well recognized that the prevalence of water-borne diseases may be greatly reduced by providing people with safe, sanitary disposal of feces and provision of clean drinking water. Water is disinfected to kill any pathogens that might be present in the water supply and to prevent them from growing again in distribution systems.

Disinfection is then used in order to prevent the growth of pathogenic organisms and to protect people's health. People need clean water and water supply systems. Without disinfection, the risk of water-borne disease increases. The 2 most common methods of killing microorganisms in the water supply are irradiation with ultra-violet radiation, or oxidation with chemicals like chlorine dioxide or ozone, or chlorine.

The Health Advisory Program, sponsored by the Office of Water (OW) provides information on the health effects, analytical methodology and treatment technology that would be useful in dealing with the contamination of drinking water. Most of the Health Advisories (HAs) prepared by the OW are for chemical substances. This HA is different in that it addresses contamination of drinking water by a microbial pathogen, examines pathogen control, and addresses the issue of an infective dose (i.e., the number of particles of a pathogen necessary to cause an infection in a host). Thus, for a variety of reasons, the format and contents necessarily vary somewhat from the standard HA document. HAs serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as new information becomes available.

This HA is based on information presented in the OW's Criteria Document (CD) for *Giardia*. Persons desiring further information should consult the CD. This document will be available from the U.S. Environmental Protection Agency, OW Resource Center, Room M6099; Mail Code: PC-4100, 401 M Street, S.W., Washington, D.C. 20460; the telephone number is (202) 260-7786. The document can also be obtained by calling the Safe Drinking Water Hotline at 1-800-426-4791.

# <u>Inactivation of Single-Celled Ascaris suum Eggs by Low-Pressure UV</u> Radiation

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**Next Section** 

#### **ABSTRACT**

Intact and decorticated single-celled Ascaris suum eggs were exposed to UV radiation from lowpressure, germicidal lamps at fluences (doses) ranging from 0 to 8,000 J/m<sup>2</sup> for intact eggs and from 0 to 500 J/m<sup>2</sup> for decorticated eggs. With a UV fluence of 500 J/m<sup>2</sup>, 0.44-  $\pm$  0.20-log inactivation (mean  $\pm$  95% confidence interval) (63.7%) of intact eggs was observed, while a fluence of 4,000 J/m<sup>2</sup> resulted in 2.23-  $\pm$  0.49-log inactivation (99.4%). (The maximum quantifiable inactivation was 2.5 log units.) Thus, according to the methods used here, Ascaris eggs are the most UV-resistant water-related pathogen identified to date. For the range of fluences recommended for disinfecting drinking water and wastewater (200 to 2,000 J/m<sup>2</sup>). from 0- to 1.5-log inactivation can be expected, although at typical fluences (less than 1,000 J/m<sup>2</sup>), the inactivation may be less than 1 log. When the eggs were decorticated (the outer egg shell layers were removed with sodium hypochlorite, leaving only the lipoprotein ascaroside layer) before exposure to UV, 1.80- ± 0.32-log reduction (98.4%) was achieved with a fluence of 500 J/m<sup>2</sup>, suggesting that the outer eggshell layers protected *A. suum* eggs from inactivation by UV radiation. This protection may have been due to UV absorption by proteins in the outer layers of the 3- to 4-µm-thick eggshell. Stirring alone (without UV exposure) also inactivated some of the Ascaris eggs (~20% after 75 min), which complicated determination of the inactivation caused by UV radiation alone.

Ascaris lumbricoides is the most prevalent of the parasitic intestinal worms; an estimated 1.4 billion people are infected worldwide, mostly in developing countries (7). Helminth infections like ascariasis lead to a host of physical and mental disabilities, including cognitive and societal impairment, higher susceptibility to infection, decreased responsiveness to vaccination, and malnutrition (6, 7), which impair the development of several hundred million children in developing countries (7). An adult female A. lumbricoides worm sheds up to 200,000 eggs daily (37); these eggs are passed in the feces of the infected individual and are thus present in wastewater, contaminated soil, and, in some cases, contaminated drinking water sources. Under favorable environmental conditions, the eggs may remain viable for up to 15 years (32).

Ascaris eggs have a 3- to 4- $\mu$ m thick, four-layer shell that consists of an inner lipoprotein layer (ascaroside layer), a thicker chitin/protein layer, a lipoprotein vitelline layer, and an outer acid mucopolysaccharide/protein uterine layer (43). The inner lipoprotein layer consists of a unique mixture of 25% protein and 75% lipid-containing ascarosides and is responsible for the impermeability of the shell (43). The chitinous layer, which provides structural strength, contains chitin spindles in a protein matrix (11). The compositions of the vitelline and uterine layers are not well characterized, but both contain protein (43). The outer three layers can be removed by soaking the eggs in a solution of hypochlorite, leaving only the inner lipoprotein layer (2, 19); this process is referred to as "decortication."

Ascaris eggs are more resistant than other water-related pathogens to most types of inactivation; the only exceptions are some viruses and bacterial endospores that are more resistant to very high temperatures. Ascaris eggs can be inactivated in minutes by temperatures above 60°C, but they can survive for more than 1 year at 40°C (10). Disinfection with chlorine at commonly applied doses is ineffectual (20). The impermeability of the inner ascaroside membrane also protects the eggs from a variety of strong acids, strong bases, oxidants, reductants, protein-disrupting agents, and surface-active agents (2). Although the use of UV disinfection is becoming more common in wastewater and drinking water treatment systems, the effect of UV radiation on Ascaris eggs has not been adequately studied yet.

Previous reports on UV irradiation of *Ascaris* eggs present conflicting evidence. Using data reported by de Lemos Chernicharo et al. (8), we calculated that 0.77-log inactivation was achieved for unembryonated (single-celled) *A. lumbricoides* eggs subjected to a 254-nm UV fluence (dose) of 200 J/m² in a small photoreactor. In contrast, based on the data reported by Tromba (40), we calculated that infective (containing a larva) *Ascaris suum* eggs exposed to UV fluences between 240 and 960 J/m² were inactivated by between 1.9 and 2.3 logs, although a fluence-response relationship was not observed. A complete fluence (dose)-response curve for *Ascaris* egg inactivation by low-pressure UV, using standardized procedures and a collimated light source, has not been reported. Such bench-scale experiments have become the standard for determining the fluence response of microorganisms to UV radiation (4, 29, 30).

The objectives of the research presented here were (i) to determine the inactivation of *A. suum* eggs exposed to various fluences of UV light in a bench-scale experiment and (ii) to determine the contribution of the outer layers of the eggshell to the resistance of the eggs to UV exposure.

#### **Beef**

Source: UNIV OF MINNESOTA: submitted to NATIONAL ANTHELMINTIC SURVEY

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Project End Date Sep 30, 2010
Grant Year(N/A)

Progress Report Objectives (from AD-416) The objective of this study is to identify the level of infection with gastrointestinal nematodes found in **American cow-calf operations** in the United States, and to assess the effectiveness of current parasite control programs and the efficacy of commonly used anthelminics in control of the parasites. Approach (from AD-416) As part of the USDA,APHIS, NAHMS 2008 cow-calf survey American cattle producers were given the opportunity to submit samples to participating laboratories (USDA, ARS, BFGL, University of Minnesota, and Colorado State University. Samples could be collected at any time from March to December, and usually coincided with a deworming treatment, or some other management practice. Samples were taken prior to deworming (Phase 1) or 2 weeks after deworming (Phase 2). Producers were given the option to participate in either Phase 1 only or both Phase 1 and Phase 2. Sampling consisted of taking a golf-ball sized sample from 20 fresh fecal pats per group of animals. Sampling is restricted to animals from 6 -18 months of age. Within 24 hours of sample collection samples are sent to one of the collaborating labs as determined randomly by APHIS. At the lab fecal samples for analyses by PCR for the parasite genera present.

This study is the first ever large scale comprehensive look at nematode parasitism in cattle in the U.S. and is the first attempt to define the extent of anthelmintic resistance in American cattle operations. This work was performed as part of the USDA National Animal Health Monitoring System ♦s (NAHMS) study to evaluate the presence of anthelmintic resistance in the U.S. beef industry. This project underwent an unfunded, 1-year extension and was closed out February 2011. During this study, 567 producers from 24 states were offered the opportunity to collect fecal samples from weaned calves for evaluation of the presence of parasite eggs (Phase 1). Producers choosing to participate were provided with instructions and materials to collect fecal samples. Fresh fecal samples were submitted to 1 of 3 randomly assigned laboratories for evaluation. In the laboratories all samples were processed in a similar manner for the enumeration of gastrointestinal nematode eggs, and the notation of the presence or absence of coccidian oocysts and tapeworm eggs. In submissions where the strongyle eggs were sufficiently high, aliquots were pooled for extraction of DNA.

Extracted DNA was subjected to PCR analysis for the presence of Ostertagia, Cooperia, Haemonchus, Oesophagostomum, and Trichostrongylus. In this study, 85.6% of the samples had GI nematode eggs.

Overall, 91% of animals had Cooperia (Order: Strongylida), **79% Ostertagia** (Family Trichostrongylidae), 53% Haemonchus (known as the <u>barber's pole</u> worm), 38% Oesophagostomum (<u>nematodes</u> of the family <u>Strongyloidae</u>.), 18% Nematodirus (zoonotic so **humans** can get it too!), 7% Trichuris, and 3% Trichostrongylus. The prevalence of coccidia and tapeworm eggs were 59.9% and 13.7% of samples, respectively. This collaboration was monitored by phone conversation, written communication, emails, and periodic interactions at society meetings. Project plans, goals, and accomplishments were discussed via conference calls and e-mail; technical advice was provided to the Cooperator in writing and by telephone.

#### **Discussion**

Nematodes, also known as round worms, are associated with parasitic infection of animals and humans.

Only eat beef that has been cooked to 160 degrees Fahrenheit.

http://www.foodsafety.gov/keep/charts/mintemp.html

## The Compass – Which way to Go?

#### Come one, come all.

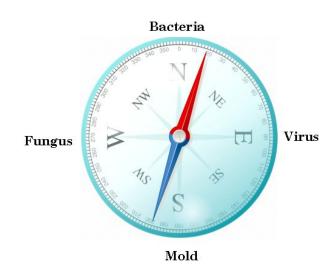
Parasites beget parasites.

Parasites weaken the immune system. Once the immune system is weakened, many types of eggs present in the water and food can infect you.

#1) Parasites beget parasites; it is like a golden rule number one ... or something. I had so many types of parasites when I scanned for them, it was scary. I actually believe this is why Parasitology has failed today.

If Doctors treat bacteria, viruses, and yeasts, (and I am stretching it a bit when I say treat), and they never treat

## Which direction do you go?



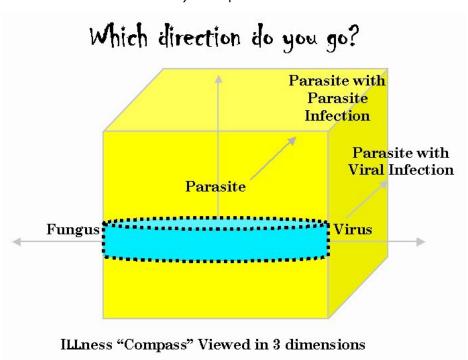
parasites, then how do they treat parasites with viruses? How do they treat parasites with yeast infections?

The short answer is they do not, because if they do not see it, it does not exist in their mind. They have missed the "compass"

#2) A compass exists in 3 dimensions.

At the base are directions, Mold, Yeast, Bacteria, Virus, (and maybe a few others). These are part of the human GI biodome.

Then as we go up, say one dimension, the Human biodome could have a parasite, and inside the gut of the parasite they have an infection, mold, yeast, bacteria, or virus. Now you have a sick parasite that generates the toxins



of an infection that you absorb, but when doctors treat it is is "one of those cases we can't seam to treat"

The great medical mystery is that at higher doses, or longer treatment durations, or different treatment more precise, the problem magically goes away. The reality is they just cured the infection inside the gut of another parasite, making your parasite healthier.

Well that makes you healthier, and makes the parasite easier to kill by your immune system.

That brings us to the third dimension 'prime' which is:

#3) What happens when your parasite is infected with a parasite?

Doctors can barely treat the directions of the compass, Doctors never see infections inside parasites, because parasites do not exist. Doctors will never see, and you will never be treated for a parasite infection, where that species is infected with yet another smaller species.

#### The Answer

You must go in all directions at once, if you want to cure a parasite infection. There is just no way to know what kind of infection you have.

If you have a fluke infection, your situation is critical

If you have a deseminated infection, a lot of skill is required

Without testing, you are flying blind. Tests are the only validation to take anti-parasitic Meds.

A body in balance is the first step at approaching a life threatening illness. Critical Metals, Minerals, Amino, detoxification, and vitamins can and do help.

The good news, is that several Nutraceuticals have bacterial, viral, fungal, and mold killing power, and are helpful to killing parasites.

By including these herbs into the formula, you can ensure a one method, one time kill.

Good Hunting.

### Its All Snake Oil Medicine

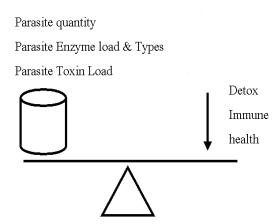
The tests, the medicines, the expense, the misinformation. It is all snake oil medicine designed to transfer benjamins and cure nothing. The less you know the more it costs, in a futile stuggle to regain your health.

Parasite quantity

- By the age of 40 everyone has at least one parasite
- Several round worms like Ascaris have no cure?
- It is a matter of parasite load vs health that determine health, nothing else.

### Parasite types, Load, Health

Each parasite Different enymes tax immune flat and round load tip spiral down health



### **Parasite Load**

Each parasite releases a mix of enzymes. The quantity and mix of enzymes make the immune system weaker. Each parasite releases a different mix of enzymes. Some are tough, i.e. they contain many enzymes, Some parasites are easy, the enzymes are easy to control, some parasites migrate fast, and multiply quickly, some parasites migrate slow, and multiply slow.

It is in the total weight of parasites, and in the techniques employed that determine if you are healthy or sick.

Some parasites are easy to remove, most Flat worms like tape worms are easily removed with PinX. Almost all tapeworms stay in the GI tract. A few tapeworms are small and travel to the brain, they are bad, but easily killed with Praziquantel.

Parasite Enzyme load & Types Parasite Toxin Load Detox Immune Ov<u>al</u> health Flat Round

Sick

Oval tapeworms like flukes are also toxing to the bodys health. They can be killed using <u>Praziquantel</u>. The small baby worms can travel to the brain, causing s significant and dramatic loss in brain power, nerve connections, Axiom loss, and cognitive decline.

Last but not least are round worms. Round worms are more difficult to control or eliminate, depending on the load of tape and oval worms. Most parasitologists for a hundred years state and believe that killing Ascaris first is the priority. I dispute that theory, because the medicines that killed Ascaris no longer exist in many of the world markets. Oil of chempotium was removed from the arsenal decades ago by large corporations that did not want cheep and effective control of parasites.

Removing Ascasis first is an impossible task, when the enzyme load, toxin load, and body health has been compermised. It is possible to remove tapeworms with <u>PinX</u>, Oval <u>flukes</u> with <u>Praziquantel</u>, and several easy speceis using traditional molicules.

Almost all medicines have been derived from natural products, that still work if they are available. Oil of chempotium can be derived from purple/yellow lavender. **Piperazine** can be derived from black pepper. Other equavelent medicine can be derived from pumpkin seeds.

It is in the sequence, strength and duration of anti-parasitics, and in the knowledge of how to treat parasites, that health can be restored.

The techniques, the technology, and the art are fraught with many details, technical jargon, and techniques, all of which must be maintained to successfully treat parasites.

This book proposes the removal of easy to eliminate parasites first, to lower the toxic load. The removal of systemic parasites generate a significant toxin load in the body, and can take weeks to remove.

When you shit black for days and weeks, it is the amount and quantity of accumulated toxins that make you ill. Killing flukes on a large scale causes black stool as well, you can loose so much iron your eyes dilate.

The removal of other parasite invaders, that also generate the black stool, once removed it can help your immune system to recover.

Finally the removal of round worm, and Ascaris load, when your immune system can generate chlorine and peroxide immune responses, that allow you to significantly reduce the burden. Ascaris sense the lower toxin levels and start to migrate to the lung, leaving the sinus, brain, throat. Ascaris make sacks of eggs, and acid. When they burst you need to dose **IVM** or **PPZ** or **DEC**. Eventually males die out, the populations thins, and you have them where you want them. Dose **Alinia** at 15 mg/kg/D for a couple of weeks and they fade into history. Then you can restore your health, and stay free of parasites.

### **Parasite Load**

Every parasite test has a scale, like 0.25, 0.5, 0.75 or 1.0 +. This scale indicates the load they put on the body. It is almost impossible in a world that refuses to use PCR analysis, DNA analysis, or even ELISA tests for humans, to determine which of 75000 known parasite types, which parasite is loading the human immune system to a load over 1.0.

ELISA tests cost about \$3.00 each or less for the WHO. Some tell me they want to be charged over 300 Euros for a test one could give to a dog for 20 dollars. I guess treating people have so much liability it is easier to treat a dog, horse, cattle, chickens, or fish farms, than it is a human. Guess we know how we rate in soceity.

las

t

### THE PLAN

### PH

Parasites can generate so much acid in a very short time, that without basic treatment, like **Baking soda** water, you could die from too much acid. After weeks of falling to the floor, being seen by 3 more doctors, and finally getting to see my selected diagnostician who previously worked at the Cleveland Clinic, he performed one astounding test, he put litmus paper on my tongue. It turned yellow, my peripheral PH was 4.5. He said he was surprised I was vertical, and not horizontal. When you get that disoriented, sipping **MSM** water with **Baking soda** can keep you alive. Keep in mind I passed my physical and saw 3 other doctors in the preceding weeks before this happened!

### **Parasite poisoning**

Parasite poisoning is the result of a worm's waste toxins. It produces symptoms such as dizzy', fuzzy vision, unclear thinking, high and low blood sugar, hunger pains, poor digestion or locked GI trac, and a range of physical ailments and disease. Detoxification is a never ending task until all of your parasites are dead.

### PH Emergency

Emergency treatment for a low body PH "acid" (potential of hydrogen) is essential to stay alive. PH essentially means does the water in your body have more acid hydrogen ions +, or alkaline hydroxyl (OH) ions -. Ions are electrical charges like positive and negative. When your body has to many hydrogen ions, the cells that make up your body have problems talking with each other. If these signaling messages between cells stop, you stop. This could mean death.

### **Death by Acid**

Electricity keeps us alive, and signaling between cells is an essential activity. The funny part in this is that you have very few clues that you are seriously ill. The body is so well designed many of us continue to function <u>right up to the second we die</u>. I find this almost unbelievable, that we can be so acid we are about to die, and do not have a clue. The reason this happens is that every cell has the ability to function without signaling, for a short period of time. This is an amazing capability built into every cell.

After that, even if your body is very acid, or the PH is extremely unhealthy (sick), the cell has the ability to use **ROS** signals. These are like primitive Indian smoke signals, which impart simple slow chemical messages of acid/alkaline/acid Morse code, which primitively signal basic messages between cells. The body is so well designed I find it funny that as complicated as we are, there is not a mechanism or clue to tell you how sick you really are. This is like leaving the keys in you car, or having your front door unlocked. You could be normally talking to your doctor one second, and be dead the next. It kind of makes no sense to be so clueless as to how unhealthy you are. But then I guess most of us do not really want to hear how bad someone feels, or hear bad news. Kind of Ironic.

### **Everyone should have Personal Emergency medicines.**

### **Essential Emergency Medicines**

Get the phone number of your local hospital, Emergency room address, or the local university teaching hospital emergency room phone number and address.

Write it down he	ere	

Go there, have someone drive you or get there safely if things start to go bad. Know how to get there.

List of current medicines and your doctor's names and phone numbers.

Keep a list of medicines and natural products you are taking, and itemize the dosing levels you are taking, and keep it with your emergency plan.

Everyone should have **Personal Emergency Medicines**.

Gather in one place or put into a box the following:

- A gallon of <u>Water</u>
- **<u>Baking soda</u>** (Bobs red mill (non-aluminum) baking soda)
- MSM Powder and or capsules
- Magnesium sulfate (Epsom salt) and or capsules
- Magnesium Citrate capsules
- Salicylic acid (Beyer aspirin or White Willow Bark) no blood clots.
- **Spirulina** Powder ( I have bought the 4 lb canister for the early days, but now use the capsules as the price came down)
- Bottle **CQ10** 400mg
- Bottle ALA 500mg
- Bottle **L Carnitine** 500mg
- Bottle Charcoal
- **Ginkgo** open capillaries.
- PH tape

# Rescue Minerals and detoxification Algae ... are life savers.

When the peripheral PH goes below 5, the body is so acid severe cellular stress is set up. The body dumps **calcium**, **magnesium**, **potassium**, and is unable to function almost on any level. Rescue Minerals can help raise the PH significantly. Initially more baking soda may be needed.



#### **Kickoff - Phase 0 Supplements**

- Take 133mg <u>Magnesium Citrate</u> per day, Initially 266 mg may be useful.
- Take 99 mg **Potassium citrate** per day.
- Take 4 glasses of distilled water, each with a teaspoon of <u>MSM</u> powder, and a large pinch (1/4 tea) of Bobs red mill <u>Baking soda</u>. Eventually ramp down to 2 grams of <u>MSM</u> in capsule form.
- Natures Plus, <u>Source Of Life</u> #3058, Two A Day With 9mg Kelp Iron, just enough, also available without iron.
- Take 5-6 G of **Spirulina** per day.

#### Q&A

**Q>** Do I really have to take all these supplements, I have sores, hurt, and while I now feel 80%, I am worried about my kidneys.

I have developed sore flank/lower back from all the supplements i take.

I think its my kidneys.

I am 80% full functioning i am not as sick as u were is it really that necessary for me to take in all those supplements?

**A>**My lower right back hurt for 8 years.

I have proved to my self, that worms return into the hip bone to hide. Especially the large ones.

The more I dosed Piperazine, or Mebendazole, or Alinia, the more the worms ache.

They are very hard to kill. They used to head right for the brain fluid, you could feel the burn.

Kidneys are damaged. That is a no brainer. Do not look at the pictures of what they do to the liver. It is not pretty.

As my infection stopped, feeling came back into my body. The actual damage they inflicted I now feel.

I am sorry to tell you but it takes a lot of healing before the body can deal with this damage.

It is hard for me to answer your question.

The battlefield of your body is so corrupted by parasites, I cannot say exactly.

I am thru it, and I can say once they are dead, you are left with a lump of a human body, that has to heal, heal it does, and the pain goes down day by day.

#### **Heavy Metal Load**

• If the hair analysis shows toxic levels of heavy metals like lead and or aluminum, take 1 Gram of <u>Chlorella</u> every evening. No more, no less, 1 gram. <u>Chlorella</u> can also pull out

of the body essential metals, so creating a conveyor, where you bring in essential metals, and remove toxic metals, is a process that cannot be done quickly.

- I you have a GI overgrowth, Heavy metals in the hair analysis test, or have a severely swollen intestine, an additional 1 gram of **Chlorella** may be required.
- Parasitic, Bacterial, Viral, and Fungal Infections: Generally, these conditions cannot be completely cleared until the toxic metals have been mostly eliminated from the body. However, if one reaches an acute stage, 2 grams of <u>Chlorella</u>, and 2 grams of <u>Charcoal</u> can be taken every evening. Too aggressive an approach can produce side effects, especially the treatment of candida[9]. Heavy metals may take one year to remove through this process.
- Take 2G <u>Charcoal</u> capsules at 6pm..every night, absorbs nasty alcohol and formaldehyde toxins from bile, preventing the colon from reabsorbing them. If toxic metal levels are present (nerve malfunctions, malabsorption, Acidosis) <u>charcoal</u> and a small amount of <u>Chlorella</u> will remove the toxins from the body. Removing toxins faster than healthy vitamins and minerals can be introduced into the body can make a ill person much sicker. Keep the ratio of <u>Spirulina</u> to <u>Chlorella</u> at 4:1.

#### **Relief from Hell**

- Hot weekly hot baths in <u>Epsom Salt</u> water. After a lymphatic drainage, it is important to drink plenty of water and take a detoxification bath. One of the easiest products to use is epsom salt. At least one pound of epsom salt is added to the bath water. Submerge the body as much as possible for 15 to 20 minutes. Use a loofah or vegetable brush to thoroughly brush the skin at the end of the bath. Baths may be incorporated into any protocol as it stimulates the elimination of toxins through the skin.
- Drink **Rosehip seed tea**, buy Rosehip seeds bulk from Mountain rose herbs.
- If your immune system is compromised, take 6 **Stinging nettle root** capsules per day. take 2,2 **Magnolia bark** capsules per day from Amermed.
- **Guaiaid** can be taken daily to force phosphate loops to suck down lactic acid, re establish G0 dna base pair, start the repair process, and get some flow of fluids.
- GUAIAID is ultrapure Guaifenesin, Typically available in 600 mg capsules.
- PH test paper, under the tongue AM first thing with no liquid or food, for 5 seconds. Keep PH above 6 or 6.5 if possible, 7.0+ is ideal.

#### Sepsis – call 911

Because sepsis can begin in different parts of the body, it can have many different symptoms. Rapid breathing and a change in mental status, such as reduced alertness or confusion, may be the first signs that sepsis is starting. Other common symptoms include:

#### Fever and shaking chills or, alternatively, a very cold - low body temperature

(Warning signs of shock include feeling "freezing" cold and uncontrollable shaking)

- Decreased urination
- Rapid pulse
- Rapid breathing
- Nausea and vomiting (from hypoxia, blue, ashen)

#### Diarrhea

http://www.annalsofintensivecare.com/content/1/1/37

Annals of Intensive Care 2011, 1:37 doi:10.1186/2110-5820-1-37

The electronic version of this article is the complete one and can be found online at: <a href="http://www.annalsofintensivecare.com/content/1/1/37">http://www.annalsofintensivecare.com/content/1/1/37</a>

#### **Blood Clot**

Swelling ache, throbbing, sensory changes, sharp stabbing pain, cannot breath, spacy confusion.

#### White Willow Bark natural full spectrum extract

White Willow Bark (2) AM and/or PM in advance thins the blood, reduces platelet clumping.

**Salicylic acid** (from Latin *salix*, *willow tree*, from the bark of which the substance used to be obtained) is a monohydroxybenzoic acid, a type of phenolic acid and a beta hydroxy acid. It has the formula C<sub>7</sub>H<sub>6</sub>O<sub>3</sub>. This colorless crystalline organic acid is widely used in organic synthesis and functions as a plant hormone. It is derived from the metabolism of salicin. In addition to being an important active metabolite of aspirin (acetylsalicylic acid), which acts in part as a prodrug to salicylic acid. The salts and esters of salicylic acid are known as **salicylates**. Hippocrates, Galen, Pliny the Elder and others knew willow bark could ease aches and pains and reduce fevers.[13] It has long been used in Europe and China for the treatment of these conditions.[14] This remedy is also mentioned in texts from ancient Egypt, Sumer, and Assyria.[15] The Cherokee and other Native Americans used an infusion of the bark for fever and other medicinal purposes.[16] The medicinal part of the plant is the inner bark and was used as a pain reliever for a variety of ailments. In 2014, archaeologists identified traces of salicylic acid on 7th century pottery fragments found in east central Colorado.[17] The Reverend Edward Stone, a vicar from Chipping Norton, Oxfordshire, England, noted in 1763 that the bark of the willow was effective in reducing a fever.[18]

The active extract of the bark, called <u>salicin</u>, after the <u>Latin</u> name for the white willow (<u>Salix alba</u>), was isolated and named by the <u>German</u> chemist <u>Johann Andreas Buchner</u> in 1828.[19] A larger amount of the substance was isolated in 1829 by <u>Henri Leroux</u>, a <u>French</u> pharmacist.[20] <u>Raffaele Piria</u>, an <u>Italian</u> chemist, was able to convert the substance into a sugar and a second component, which on oxidation becomes salicylic acid.[21][22]

Salicylic acid was also isolated from the herb <u>meadowsweet</u> (*Filipendula ulmaria*, formerly classified as *Spiraea ulmaria*) by German researchers in 1839.[23] While their extract was somewhat effective, it also caused digestive problems such as <u>gastric irritation</u>, <u>bleeding</u>, <u>diarrhea</u>, and even death when consumed in high doses.

Salicylic acid has been shown to work through several different pathways. It produces its anti-inflammatory effects via suppressing the activity of <a href="cyclooxygenase">cyclooxygenase</a> (COX), an <a href="enzyme">enzyme</a> that is responsible for the production of pro-inflammatory mediators such as the <a href="prostaglandins">prostaglandins</a>. It does this not by direct <a href="inhibition">inhibition</a> of COX like most other <a href="non-steroidal anti-inflammatory drugs">non-steroidal anti-inflammatory drugs</a> (NSAIDs) but instead by suppression of the <a href="expression">expression</a> of the enzyme (via a yet-unelucidated mechanism). [34] Salicylic acid has also been shown to activate <a href="adenosine monophosphate-activated protein kinase">adenosine monophosphate-activated protein kinase</a> (<a href="AMPK">AMPK</a>), and it is thought that this action may play a role in the <a href="anticancer">anticancer</a> effects of the compound and its prodrugs aspirin and <a href="salsalate">salsalate</a>. In addition, the <a href="anticancer">anticancer</a> effects of salicylic acid are likely mediated by AMPK activation primarily through allosteric conformational change that increases levels of phosphorylation. [35] Salicylic acid also uncouples oxidative phosphorylation, which leads to increased ADP:ATP and AMP:ATP ratios in the cell. As a consequence, salicylic acid may alter AMPK

activity and subsequently exert its anti-diabetic properties through altered energy status of the cell. Even in AMPK knock-out mice, however, there is an anti-diabetic effect, demonstrating that there is at least one additional, yet-unidentified action of the compound.[36] Salicylic acid has shown to regulate c-Myc level at both transcriptional and post-transcription levels. Inhibition of c-Myc may represent an important pathway by which aspirin exerts its anti-cancer effect and decrease the occurrence of cancer in epithelial tissues.[37]

#### Other uses

Salicylic acid is used as a <u>food preservative</u>, a bactericidal, and an antiseptic.[38] salicylic acid is capable of penetrating and breaking down fats and lipids.

**AMPK** activation is the stimulation of <a href="https://hepatic.com/hepatic">hepatic</a> fatty acid oxidation and <a href="https://ketogenesis">ketogenesis</a>, <a href="https://inhibition.organics.com/hepatic

Oral salicylic acid has not been associated with an increase in malformations if used during the first trimester, but use of aspirin in late pregnancy has been associated with bleeding, especially intracranial bleeding.[42]

**Ginkgo** opens the capillaries. Taking these meds (white willow bark and Ginkgo) taken daily **before** a clot happens, is essential.

### **Early Interventions**

200/400 mg/D **Magnesium Complex** Supplements may be initially required to re-establish brain balance.

Severe brain disturbance may require physician intervention. A typical physical checks most blood parameters. A local Clinic can run this test. Periodic blood tests may be useful, but the body is able to compensate for most illnesses, making blood tests less valuable than hair analysis.

#### **Magnesium**

<u>Magnesium</u> Plus 200 mG @ magnesium amino acid chelate, Neuron Salt Calcium Balance Nerve Cell, ProHealth, PH88

#### **Epsom Salt Baths**

Baths in **Epson salt** with **Baking soda** may be helpful. 45 minutes is required to extract toxins.

### **Impacted Bowel**

<u>Miralax</u> for the bowels, <u>Psyllium</u>, <u>Bladderwrack</u>, <u>Chorella</u>, <u>Charcoal</u>, and <u>Spirulina</u> can be used to start the bowels moving if they are impacted. <u>Magnesium citrate</u> about 133mg can be used daily, you may need more (266mg) for several days to get the bowel running.

If the bowel is impacted with worms, **PinX** dosing can cause a worm impaction, or blockage, requiring a hospital intervention. Low dose **PinX** is advisable. A soft diet can help until the blockage is resolved.

#### **Dried fruits**

Dried fruits like prunes, dates, figs, raisins and even apricots are a good source of fibre. Prunes in particular not only contain high amounts of fibre but it also contains sorbitol which acts like a natural laxative.

#### **Apples**

Apples, especially their skin have good amounts of fibre in them which can help ease out constipation.

#### **Plums**

This monsoon fruit also can help treat constipation.

#### **Pears**

Like apples, pear too has ample fibre especially in their skin. Eating this fruit will regulate your digestive system.

#### **Berries**

Strawberries, blackberries and raspberries have a good amount of fibre which can help ease out your constipation symptoms. These fruits are also low in calorie which makes them healthy anyway.

#### **Banana**

Bananas are also a good fruit to treat mild constipation but it is extremely important that you pick on ripe bananas only. Unripe bananas can worsen the situation.

### Keeping your organs alive

- <u>Magnesium sulfate</u> is essential. It causes the liver to dump toxins.
- KGP Flush keeps the kidney tubes clear.
- Milk Thistle keeps the liver and kidney membranes in tact.
- **CQ10** establishes the movement of alkaline/acid electron movements, which prevent cells from turning to a genetic alteration known as **ROS** signaling.
- Start <u>Algae</u> and <u>Charcoal</u> as early as possible to detoxify the body and DNA, and raise the PH, and force essential amino acids into the mitochondria of every cell.
- Maintain PH by drinking <u>MSM Baking soda</u> water in large quantities is not a good idea, but small quantities sipped throughout the day can be helpful.
- Test your PH regularly.
- Rosehips seed tea which contains a special Vitamin A molecule, should be taken away
  from vitamin or anti-parasitic dosing, do not mix it with milk. In the start-up and initial
  killing phase, it is essential that the body is as healthy as possible. After the initial killing
  phase, where a large amount of black stool is evidenced, discontinue All vitamin A products.

#### **MSM**

AM 3/4--1 1/2 TEA MSM with Baking soda in 8OZ of distilled water.

MSM contains the same molecular components of DMSO, however, MSM contains an extra oxygen atom. MSM (Methylsulfonylmethane or methyl-sulfonyl-methane) is a superb treatment for reducing and preventing swelling and inflammation. The use of at least 7 grams of MSM per day on ANY cancer treatment. That is 2-4 glasses of MSM water per day. Spirulina and MSM help get your ph up to 6.5.

Use PH paper under the tongue when you get up before water or food. Under the tongue for 5 seconds, the PH paper should read 6.5 for a string of days before you start treatment. 12-30 grams of **Spirulina** per day may be required for several weeks to budge your PH.

### **Getting Started Sequence:**

The ramp of vitamins, minerals and metals (Phase 0) should follow a sequence. Clearing of the GI tract (Phase 3), accelerated by the **SIBO** annd **SIYO** kills (Phase 2), the accelerated removal of toxins, and initial anti-parasitics like **DEC** and **Piperazine** (Phase 2/3/4). **DEC** and **Piperazine** are required to clear the blood supply. Clearing the heart is essential, prior to dosing **Albendazole** and **Praziquantel**. **Pulsing IVM keeps the birth rate down**.

- Minerals
- Metals
- Vitamins
- Anti toxin supplements and herbs
- SIBO
- SIYO
- Piperazine
- DEC
- Praziquantel
- Albendazole
- Alinia

Going after the actual parasites takes several stages of preparation. People should start slow on anti-parasitics.

Chitin molecules are long chains of nitrogen

#### Q/A Nitrogen in Parasites

# What is Nitrogen (them) vs Sulfur (us) based lifeform...

Chitin molecules are long chains of nitrogen-containing sugar. The chitin-degrading enzymes known as chitinases are not just important to insects with chitin shells and to their predators, they also seem to be involved in the establishment of parasites in the human body and in asthmatic diseases. Insect chitinases belong to family 18 of the glycoside hydrolase superfamily (GH18) and comprise endo-splitting enzymes that retain the anomeric b-(1,4) configuration of the cleavage products.

Chitin is a b-(1,4)-linked polymer of N-aceytylglucosamine moieties (NAG). Insects, spiders, scorpions, crabs -- many animals have a shell made of chitin. In addition, chitin is found in the cell walls of fungi, dust mites, and various parasites.

"Chitinase inhibitors are potential insecticides and fungicides," explains Withers. "They are also interesting as pharmaceuticals.

They could stop the transmission of the malaria parasite to humans and help to fight trichomoniasis infections." Furthermore, there seems to be a connection between asthma and an elevated level of chitinase-like enzymes in the lungs.

The core structural element is a ring-shaped sugar building block fused with a thiazoline, a five-membered ring made from one nitrogen, one sulfur, and three carbon atoms. "This arrangement imitates a cyclic intermediate formed in the enzymatic degradation of chitin, and docks to the binding sites on chitinase enzymes," explains Withers. "To augment the inhibitory effect, we added two or three additional sugar units that resemble those in chitin (chitobiose or chitotriose). Further modifications ensure that the inhibitors themselves cannot be degraded, so they remain effective for a long time." The inhibitors could be a good starting point for the development of novel medications and further research into the role of chitinases in biological systems.

### **Chitinase Enzymes**

Papaya seeds contain a good amount of chitinase

http://www.ctahr.hawaii.edu/seed/seeds.asp Seeds cost about \$8.00 per ounce in which you get 2,000 seeds per ounce. Shipping and handling makes the total \$14.50.

### **Lufenuron**

Chitosan doubles the enzyme activity that suppresses nematode activity

Metal ions are known to act **as activators or inhibitors of enzyme** activity.

The metal ions such as Co2+, Mn2+, Na+ or EDTA may act either as activators or inhibitors for CDA depending on their concentration. For instance, Zn2+ (1mM) slightly promoted the CDA activity in C. lindemuthianum but, increase in concentration to 10mM strongly inhibited activity of the enzyme [9]. In case of S. cerevisiae and C. lindemuthianum CDA was activated in presence of Co2+ but inhibition occurred in case of CDA from A. nidulans (Table 1). The different source of CDA and the varying concentration of metal ions used could be the possible reason for the different response to the presence of metal ions.

# Q/A - What is a Zoonis/Parasite/Nematode?

A-Nitrogen based life, they suck out sulfur, pee ammonia, and trash your health

### **Consume**

3 Meals and 3 very small meal/snacks

**Butter – Land of Lakes** 

Olive oil

**Fat** 

Cheese

**Garlic** 

- o Garlic: Garlic is used both medicinally and as a food spice. Preliminary research suggests that oral plus intravenous garlic may help manage symptoms of cryptococcal meningitis, a fungal infection that commonly occurs in HIV patients. Further research is needed before recommending for or against the use of garlic in the treatment of this potentially serious condition, for which other treatments are available. Several studies describe the use of garlic as a topical antifungal to treat fungal infections of the skin, including yeast infections. More research is needed in this area. Use cautiously as garlic can cause severe burns and rash when applied to the skin of sensitive individuals.
- O Avoid if allergic or hypersensitive to garlic or other members of the Lilaceae (lily) family (e.g. hyacinth, tulip, onion, leek, or chive). Avoid with a history of bleeding problems, asthma, diabetes, low blood pressure, or thyroid disorders. Stop using supplemental garlic two weeks before and immediately after dental/surgical/diagnostic procedures with bleeding risks.

Sea salt

**Double vegetable** 

4 oz meat

Salads, Raw Vegtables

**Cottage cheese** 

**Nuts** 

**Whole Oats, Whole Grain** 

**Fruit Juices** 

**Soups** 

**Cranberry juice** 

Reported beneficial for kidneys – no sugar

### 80% alkaline-forming foods - examples include:

#### Regularly consume and make it a habit to consume:

Lemons, watermelon, lentils, blackberries, raspberry, pineapple, strawberries, limes, grapefruit, mangoes, asparagus, onions, vegetable juices (excl tomato), broccoli, garlic, grapes, berries, apples, pears, almonds, radishes, yams, endives, beetroots, celery, lettuce, organic carrots, ginger, cantaloupe melon, bean sprouts, almonds, cashews, chestnuts, millet, quinoa, goats milk, apple cider vinegar, bananas, oranges, fresh ginger root, watermelon, avocados, mandarin oranges, tangerines, horse-radish root, pumpkin seeds, sunflower seeds, sweet potatoes, kiwi fruit, oats, wild rice, chestnuts, natural olives, green leafy vegetables, sea vegetables, sea salt, natural still mineral water, umeboshi vinegar.

Liquid Chlorophyll is a great tonic for alkalising the body and it also helps to cleanse and strengthen the blood. Another useful natural supplement that can help alkalise the body is Kelp & Hops Combination, Barley Grass and also Magnesium.

# 20% acidic-forming foods

(You should limit your intake (portions) of these. Portions means not taking to excess, or regularly or repeated in nature to become a habit. examples include:

Whole Cranberries, prunes, peanuts, fried potatoes, beans, seeds, walnuts, meat, fish, dairy, nuts, 1 egg, tea, coffee, vegetable oils and fats, fried foods, sugar, refined/processed foods eg. White bread, rice and pasta, fizzy soft drinks, fizzy water, processed meats, processed pies, cakes and pastries, ALL alcohol, soy milk, most grains, tomatoes, carrots, couscous, lobster, mussels, shrimps, hazelnuts, soy beans, red wine vinegar, white vinegar yeast, tofu.

**Maitake** (*Grifola frondosa*), shiitake (*Lentinula edodes*), and *Agaricus* (*Agaricus blazei*) are all delicious edible mushrooms that have immune-enhancing and anti-cancer effects and can be found at specialty grocery stores

# **Drink plenty of water**

**Eat more raw fruit on an empty stomach.** The enzymes and acids in fruit are powerful lymph cleansers

### **Intestinal Flora, Foods, and Other Measures**

Use of green juices, aloe juice, and a diet high in greens also helps as does supplemental **garlic** and asafoetida (in capsules or food.) In addition, one can nibble on pumpkin seeds and eat **fresh pineapple** and calmyrna figs. **Coconut** also has antiparasitic properties. According to some sources, sesame oil is somewhat antiparasitic, and black cumin seed, Nigella sativa, has significant anti-parasitic properties. **Many recommend drinking sesame oil**, a teaspoon or so at a time throughout the day. I personally would add clove oil and/or fennel seed oil to the sesame oil. Fennel seed tea, three cups per day, can be used, especially towards the end of the cleanse. Some authorities believe that fennel intoxicates parasites, making them less protective and easier to annihilate.

# Pumpkin Butter

http://www.kitchendoctor.com/recipes/pumpkin\_butter.php

Take herbs, minerals, and metals, and then clear the GI with **PinX** (Phase 2) prior to starting the (Phase 3) anti-parasitic operation to remove the worms.

<u>Sulfur needs to dominate Nitrogen</u>, Initially taking <u>MSM</u>, <u>Garlic</u>, Onions, <u>Magnesium Sulfate</u> helps infuse the body with Sulfur, and Sulfur compounds.

**PH dominates everything in the body**, an acidic body does not absorb minerals or metals. A acidic body has organs that malfunction. An acidic body has no operating or functional enzyme processes.

Balanced function of the body can not be accomplished without having the PH high enough, **Spirulina** and **MSM** with **Baking soda** help to quickly restore PH. Killing **SIBO** and **SIYO** also help raise PH.

The object is to feed your body, and deplete the energy and mischief of the parasite(s). No one has just one parasite, they would not go wild if you had only one kind.

#### Q&A

A quick example Email from the forum:

**Q>** Do you think it's a good idea to clean up my diet before starting?

**A>** No, Do not deprive yourself; one can sooner change the spots on a leopard.

**Q>** I have been binging like crazy these past 2 weeks. Gained a ton of weight.

**A>** It happens

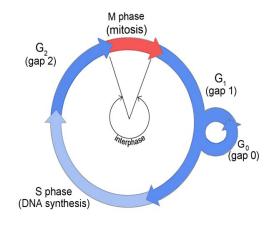
**Q>** I'm giving in to the parasite Sugar cravings.

**A>** Sugar cravings may be from cells that turned to sugar (Pre cancer). Parasites prefer simple carbs like Booze, or Pasta.

### Sugar

A> What is your PH?

A> Cells that burn sugar have the base pair of the DNA turned off, the cell has decided that the DNA damage is go great, and lost its DNA tail, Turns G0



and G1 to toggle. This signals the cell is burning sugar, not oxygen. The cell is in survival mode. Cells do not run well on sugar. They generate even more toxins that the parasites use to hide in.

#### **Dont's**

The use of caffeine, Cola, Coffee, Booze, and Pasta are not allowed.

### ROS, Oxygen, and DNA repair

If you take **Zinc** and **Selenium**, oxygen is transported to the cells. **Ginkgo** helps repair and build new capillaries. If the cells are exposed to **Guaifenesyn** (GuaiAid 6-8 per day), it pulls on the phosphate chemistry of the cell, the ATP receptor, which tells the mitochondria it need to do work, forcing the cell to return to burning lipids ( Lipid chemistry is where **Vitamin A** and **Vitamin C** meet, Lipids feed the cell), and once again the cell burns oxygen. If sufficient DNA repair molecules and amino food are present (**Spirulina** and **Sulfur**) the cell will flip itself into a normal and functioning cell, and start the DNA repair process. The cell no longer burns sugar.

>There also are some gut bacteria that eat sugar, <u>Chitosan</u> will kill these. A little copper forces them back under control. There is a section called <u>SIBO</u> in the back of this document, that addresses bacterial overgrowth.

Is it best to eat better for a couple days before starting treatment or should I just jump right into it?

### **Starting Supplements**

- > The starting protocol is **MSM** teaspoon powder in glass of non-chlorine water, with a pinch of **Baking soda** 4X/D. Distilled water accelerates the toxin removal process.
- > The starting protocol is <u>Magnesium Citrate, Magnesium sulfate, Potassium Citrate, Zinc</u> <u>Sulfate monohydrate, Selenium.</u>
- >The starting protocol is **Spirulina**, **Source of Life**, **and Charcoal**.
- > The starting process includes White willow bark, and Ginkgo.

These may make you feel better, do not be fooled. I had one such person who stated <u>MSM</u> cured him. This is not so, it only makes it harder for parasites to grab onto the lining of the intestine, and provides organic sulfur.

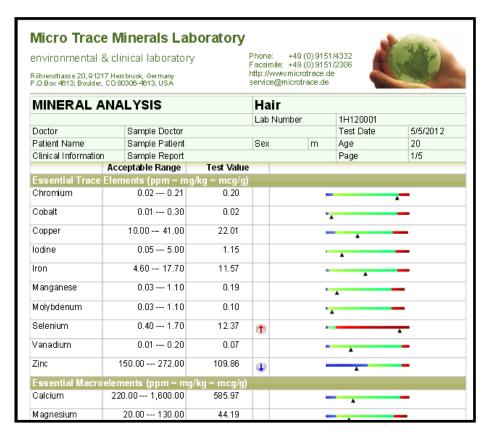
#### **PH Process:**

By the numbers, you need to: There can be no treatment without the proper PH.

#### PH

**Emergency** treatment for a low body PH "acid" (potential of hydrogen) is essential to stay alive. PH essentially means does the water in your body have more acid hydrogen ions, or alkaline hydroxyl (OH) ions. Ions are electrical charges like positive and negative.

When your body has to many hydrogen ions, the cells that make up your body have problems talking with each other. If these signaling messages stop, you stop. This could mean death.



Electricity keeps us alive, and signaling between cells is an essential activity. The funny part in this is that you have very few clues that you are seriously ill. The body is so well designed many of us continue to function right up to the second we die. I find this almost unbelievable, that we can be so acid we are about to die, and do not have a clue. The reason this happens is that every cell has the ability to function without signaling, for a short period of time. This is an amazing capability built into every cell.

### **Hair Analysis**

Get a hair analysis, don't guess, if you have a severe parasitic infestation you could have virtually no essential minerals and metals in your body.

#### Chelation

Parasitosis causes massive acidity, which mobilizes many ionic forms of metal ions within the body, causing a massive sickness. Forcing all types of supplement metals into the body does little to stem the tide.

These metals are moving, collected in bile, and will cause massive sickness until Acidosis can be brought under control.

Years of metal toxins will begin a feedback loop between bile, and the large intestine, unless the heavy metals are removed. Problem is good metals are also lost, just in the middle of a health crisis. Supplementing all essential metals, and gently removing the overload dump in bile at 3AM, is the only course to pursue.

Maintaining as many health practices, natural sources of chelation stabilizing, and using **Chlorella** and not cilantro is a much safer way to maintain **metal homeostasis**, in leau of such a huge body imbalance from **acidosis**.

http://www.earthclinic.com/CURES/fluoride\_questions.html

http://www.earthclinic.com/CURES/fluoride\_guestions.html#Question\_4039

Chelation is the process of removing heavy metals from the body. **Avoid chelation**. A single no **iron** multivitamin, taken with **Chlorella** 1G with **Spirulina** 4G and **Charcoal** 2G at 8 PM, will totally detox aluminum, and lead. Keep the ratio of 1:4 **Chlorella** to **Spirulina**. If necessary increase to 2 Grams **Chlorella** to 8 grams **Spirulina**. Dose algae powder at night, for maximum benefit to the intestine.

- Most of the heavy metal toxin patients are terribly deficient in <u>zinc</u>.
- **Zinc**, which is needed for vision, hearing, muscle function, and immune health, is often replaced by lead in the tissues, where it causes cellular malfunctions.

Medicinal foods are a much more useful method to detoxify the body

- <u>Cilantro</u> has been found to chelate (remove) heavy metals like mercury, aluminum, and lead from the body. In fact, it is believed to cross the blood-brain barrier and actually remove said metals from the brain.
- Soup, eggs and salads W cilantro
- Garden salads with <u>cilantro pesto</u> using olive oil and using either Walnuts or Brazialin nuts
- Pectin removes toxins and heavy metals, lowers cholesterol
- Methionine is a natural occurring chelating agent that supplies sulfur in the body. It helps
  in heavy metal detox by increasing the production cysteine and lecithin for the liver and
  protecting your kidney.

Heavy Metals will (re)attach to open mineral binding sites. To rebuild mineral stores from Heavy Metal toxification and chelation one must supply antagonized minerals to prevent Heavy Metals from binding and removing essential minerals.

- Heavy metals bind to empty mineral receptors
- Mineralize to prevent Heavy Metal binding).
- Electrolyte Balance
- Replacement- (K, Na, Ca, + Mg) is critical to reducing symptoms and ANS nerve function.

Supplement Minerals per mineral studies.

Magnesium, Potassium, Calcium Zinc, Selenium, Molybdenum, Chromium;

<u>Chlorella</u> food-oral chelators are essential for mobilizing the mercury from the deeper extra cellular tissues to be excreted. <u>Chlorella</u> binds Heavy Metal (especially Mercury salts) in the gut and in extracellular spaces. <u>Chlorella</u> does not cross brain barrier and can be used to remove mobilized metals, and for binding toxic chemicals and neurotoxins.

- 1).Low Dose <u>Chlorella</u> (2-4 caps with food) To bind Heavy Metals excreted from liver (bile), minimize GI re-absorption during chelation.
- 2). Mobilizing Dose: 3-5 g **Chlorella** once/day away from food better chelation effect, or 1-2 g 3x/day with food to bind Hg and neurotoxins from liver/bile.

To bind Heavy Metals in gut and reduce dysbiotic (fungus, bacteria, parasites); Stir-up/ mobilize Heavy Metals in extra-cellular spaces, which increases chelation yield of heavy metals.

#### **Chelation Strategy:**

Toxic levels of heavy metals are best removed in a gentle manner.

Mobilizing phase away from food, when not using **cilantro** or when using **garlic**, post chelation phase with food to bind heavy metals in bile.

- 3). High Dose **Chlorella**: 2-3 times mobilization dose, for 2 days. A chelation dose of chlorella: To excrete HM from extra cellular spaces and through the GI (mostly feces). Strategy: can be used with **Glutathione**
- 4) Sulfur Supplementation: Critical for heavy metal detox because sulfur in its various forms is the detox carrying element, and primary heavy metal binding site. (options for supplementation)
  - MSM 3-10g/day divide dose with meals
  - D.L. Methionine
  - NAC ( N-acetyl-cysteine) no more than 250 mg/day Divide dose with meals will cross the brain barrier;
  - Oral Glutathione (Ivsine-cysteine-alutatimine) 750-1000 mg /day away from meals.
  - Others: Garlic; Alpha Lipoic Acid (ALA)
  - Garlic is a good souce of N-Acetyl-L-Cysteine (NAC) which increases the production of
    cysteine and glutathaione which are both powerful antioxidants which lessen the effects of
    heavy metals.
  - Consider Brazil Nuts which contain one the highest amount of **zinc**, **selenium and magnesium** in a food. This seems like a perfect food for helping the body recover minerals during the chelation process.
  - Some patients require up to 2 years of iodine therapy to bring post loading urine bromide levels below 10 mg/24 hr, if chloride load is not included in the bromine detoxification program. Fluoride also antagonizes iodine levels. Consider natural toothpaste, or rinse repeatedly Rapid mobilization of bromine from storage sites with orthoiodo supplementation combined with increased renal clearance of bromide with a chloride load often causes side effects. Increasing fluid intake and adding a complete nutritional program minimizes these side effects. Doctors involved in the chelation of children with neurological conditions need to take notice and start treating them with iodine as a primary not secondary form of treatment

- Magnesium protects cells from aluminum, mercury, lead, cadmium, beryllium and nickel. Magnesium protects the cell against oxyradical damage and assists in the absorption and metabolism of B vitamins, vitamin C and E, which are anti-oxidants important in cell protection. Data demonstrates a direct action of glutathione both in vivo and in vitro to enhance intracellular magnesium and a clinical linkage between cellular magnesium, GSH/GSSG ratios, and tissue glucose metabolism.[xxviii] According to Dr. Russell Blaylock, low magnesium is associated with dramatic increases in free radical generation as well as glutathione depletion and this is vital since glutathione is one of the few antioxidant molecules known to neutralize mercury.[xxix]
- Baking soda 1/2 teaspoon taken twice a day is what was missing from the equation. TO
   PREVENT CALCIUM FLOURIDE IN PINEAL GLAND, OR SODIUM MAGNESIUM OR
   POTASSIUM CITRATES FROM REDUCING FLOURIDE, BUT IN BLOOD ITS ACID
   LEVEL LEADS TO its ACCUMULATION.

### The Healing Crisis

It is not appropriate to treat a chronic condition in the presence of an acute condition. When this basic rule of prioritization is broken, the health is further compromised by additional and unnecessary stress, thus aggravating the acute condition. Therefore, a 'healing crisis,' which is an acute condition, cannot be considered acceptable during heavy metal detoxification, a therapy which is used to address the underlying causes of chronic degenerative diseases.

The symptoms of a healing crisis can include: acute nausea, vomiting, fever, fatigue, muscle weakness, malaise, headache, body aches & pains, back pain, hair loss, flatulence, peripheral neuropathy, sore throat, constipation, abdominal pain, dizziness, confusion, irritability, emotional and mental instability.

One or more of the following causes a healing crisis:

- More toxins have been mobilized than the chelator can bind and or the patient's pathways of
  elimination can excrete, and that the resulting symptoms are caused by the redeposition of
  the remaining toxins in the body.
- Over dosage of a chelator, like <u>Cilantro</u>, <u>Chlorella</u>, or <u>ALA</u>
- An allergic reaction to mobilized metals.
- A low level of systemic reserves, health is to poor to withstand the stress.
- There is an unidentified focus with greater priority; i.e. a chronic condition is being treated
  in the presence of an acute condition. It is important that healing be done in a sequence
  that makes the body stronger, that underlying health issues are addressed, (infected teeth,
  sinus, appendix, emotional stress, etc.), and that the body is made stronger through the
  process.
- Blocked autonomic regulation (blood pressure, pulse, regular heart beat).
- Instability of the central nervous system (CNS).
- Inadequate or inappropriate toxin removal methods.
- Low urine output

### Malabsorption

https://en.wikipedia.org/wiki/Ascariasis

Malabsorption may be due to a loss of brush border (small intestine) **enzymes**, that occur with Ascaris/Small intestine infections.

Acidity can cause malabsorption, decreased mineral and metal status, and ultimately huge imbalances in body chemistry.

SIBO and SIYO can cause GI malfunction.

Worm enzymes alter the entire process within the GI trac. Acidosis, Malabsorption, and Dysbiosis are very common when the GI tract has been inundated by parasites.

A <u>Hair analysis</u> can evaluate cellular processing of minerals and metals, by actually recording the cellular utilization of these critical health elements. When compared to normal amounts of these critical elements, it is easy to see how much your cellular processes have been impacted, how sick you are, and which elements may need additional supplementation.

Each parasite creates a different signature, some elements are common, like <u>Sulfur</u> and <u>Selenium</u>. Get a hair analysis to see how bad your cellular processes are. My hair analysis showed no essential minerals, no essential metals, and toxic levels of aluminum and lead. My hair analysis report was not pretty, not pretty at all.

<u>Malabsorption</u> affects all macronutrients: carbohydrate, fat and protein.

<u>Carbohydrate</u> <u>malabsorption</u> is due to both fermentation of carbs in the small intestine and the damage done to the intestinal brush border caused by the byproducts generated by these same bacteria. Fermentation will contribute to diarrhea as the production of fatty acids is a result. Fats tend to speed up the plumbing.

Another way carbohydrate <u>malabsorption</u> causes diarrhea is because undigested carbohydrates enter the colon. This pulls water rapidly from the body leading to that oh-so-awful and potentially embarrassing dash to the nearest toilet.

<u>Fat</u> **malabsorption** will inevitably lead to malabsorption of all fat-soluble vitamins (A, D, E and K) and most minerals as both fat-soluble vitamins and minerals rely on proper fat digestion to pass through the intestinal wall and enter the portal vein leading to your liver.

The deconjugation of bile will also lead to the production of a substance called lithocholic acid that can be directly toxic to the cells (enterocytes) lining your digestive tract. Toxicity equals inflammation, and inflammation equals the very real possibility of developing an inflammatory bowel disorder if this goes on long enough.

Inflammed enterocytes in turn will contribute to increased intestinal permeability or "leaky gut" and all the negative consequences resulting from that. Increased intestinal permeability leads to more inflammation, which attracts more pathogenic bacteria resulting in a vicious feedback loop that can be difficult to resolve. Throw in *Candida* overgrowth for good measure and you'll wonder who you pissed off in a past life.

<u>Protein malabsorption</u> can lead to loss of muscle mass and low albumin levels in your blood as your body becomes protein starved. Normal repair and recovery of protein structures in your body are therefore impaired. Finally, maldigested protein will result in potentially toxic amounts of <u>ammonia</u> as <u>ammonia</u> is a byproduct of protein degradation caused by bacteria adding even more insult to already damaged enterocytes.

Abundant amounts of gas are produced by all this **fermentation** of food in the small intestine and by the increased amounts of undigested carbohydrate, fat and protein that now enter the colon. Here, colonic bacteria try to break it down adding further to the gas produced in the small intestine. You'll be producing so much gas Goodyear® could use you for their blimps.

If you're lucky, you'll pass plenty of wind, although those unfortunate enough to be near you will feel anything but lucky. If you're unlucky, (and boy was I ever) the ability to fart and move things along will be impaired as many of these pathogenic organisms negatively affect intestinal movement. Gas continues to build up but has no where to go resulting in painful bloating and the profile of a six-month pregnant woman. Fun times!

I do want to mention that gluten **opioids** will also slow intestinal movement so it can be hard to tell whether it's gluten or toxic bacterial metabolites causing the paralysis of the gut. For me, cutting gluten out of the diet resolved the bloating and constipation but did not resolve yeast and bacterial overgrowth. A very good case can be made that it's a combination of both leading to the inability to pass gas.

These pathogenic bacteria eat, and guess where they get their food? Yep, that food you just ate gets eaten by the pathogenic organisms residing in your small intestine and will have first shot at any nutrients. Hope you didn't spend good money at the local Whole Foods Store!

Pathogenic bacteria love, love <u>iron</u> so don't be surprised when your doctor tells you you're anemic even though there isn't any hint of bleeding anywhere in your body, and you eat plenty of iron-rich food. Vitamin B12 deficiencies are also a big problem in those with <u>SIBO</u>.

#### References:

Bures J., Cyrany J., Kohoutova D., et al. (2010) Small intestinal bacterial overgrowth syndrome. *World Journal of Gastroenterology*, 16(24): 2978-90.

Parodi A., Lauritano E.C., Nardone G., Fontana L., Savarino V., Gasbarrini A. (2009). Small intestinal bacterial overgrowth. *Digestive and Liver Disease*, (3), 44-49.

Quigley E. M. M., Quera R. (2006). Small Intestinal Bacterial Overgrowth: Roles of Antibiotics, Prebiotics and Probiotics. *Gastroenterology*, 130: S78-S90.

### **ROS** Signaling

Reactive oxygen species (ROS) are chemically reactive molecules containing oxygen. ROS signaling is present, when your body is extremely acid.

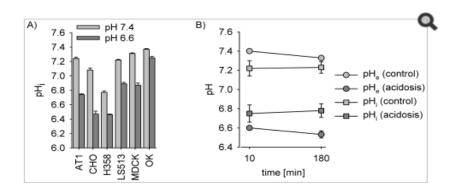
Your body relies on them as a survival mechanism to stay alive, in a <u>low enzyme</u> <u>environment</u>. Enzyme communication signals are complex and wholesome. <u>ROS</u> signaling is like a caveman's club, very basic, very crude.

**Reactive oxygen species** (**ROS**) can act as a intracellular signaling molecule ... confirming a strong impact of <u>acidic</u> pH on <u>ROS</u> production in mitochondria.

https://www.youtube.com/watch?v=7VoPE5bRV-U

#### Intracellular pH following extracellular acidosis

Fig. 1A shows the intracellular pH of the different cell types. Lowering extracellular pH always led to a decrease in pH, albeit the degree of this reduction differed



strongly between the cell lines. Under control conditions (pH 7.4) the intracellular pH was always lower than in the extracellular compartment.

However, under acidosis (pH 6.6) the intracellular pH was higher in most cell lines. An acidic tumor microenvironment can induce a longer lasting change in the transcriptional program, representing a "memory effect" which maintains the altered phenotype even when the cells leave the tumor environment.

After that, even if your body is very acid, or the PH is extremely unhealthy (sick), the cell has the ability to use **ROS** signals. These are like primitive Indian smoke signals, which impart simple slow chemical messages of acid/alkaline/acid Morse code, which primitively signal basic messages between cells. The body is so well designed I find it funny that as complicated as we are, there is not a mechanism or clue to tell you how sick you really are. This is like leaving the keys in you car, or having your front door unlocked. You could be normally talking to your doctor one second, and be dead the next second. It kind of makes no sense to be so clueless as to how unhealthy you are. But then I guess most of us do not really want to hear how bad someone feels, or hear bad news. Kind of Ironic.

<u>GuaiAid</u> helps convert the body to an alkaline environment, along with the other herbal <u>proton</u> <u>pump inhibitors (PPI)</u>. Once the cell has been changed by an acid environment, it is mandatory that the body be kept in an alkaline state. <u>Spirulina</u> does this, and should be done for life.

### **Constipation**

Parasite infections cause swollen intestines, congested lymph pathways, congested organs, all blocked with a high toxin loads.

Sufficient liquids must be ingested.

Initial treatment for toxins can be done by natural products like algae (green or blue-green) and or bulk psyllium products, **charcoal**, bladderwrack (brown algae).

Atonic constipation is associated with minimal or no abdominal discomfort. **Weakness in muscle** can result from riboflavin (**vitamin B2**) and/or thiamin (**vitamin B1**) deficiency, lack of fiber in the diet, lack of water or lack of exercise, ignoring the urge to defecate, or physical nerve damage. Clearing the GI tract of parasites is an essential first step, and a healthy dose of Enzyme **B50** vitamins, helps repair the processing of food. Avoid B2 for Protozoan Infections.

<u>Malabsorption</u> is the inadequate absorption of one or more nutrients. Acidosis frequently results in the deficiency nutrients needed for bowel health and function. Deficiency of <u>B1 thiamin</u> or <u>B2 riboflavin</u> or <u>magnesium</u> contribute to constipation.

**B1** Thiamin and **B2** riboflavin are vitamins that are important in maintaining muscle tone along the lining of the digestive tract and promoting the health of the mouth.

Other than low bile acids and salts, digestive problems usually include thiamin deficiency, which results in abdominal discomfort and anorexia.

Other digestive problems from <u>riboflavin</u> deficiency are sore, swollen, magenta-colored tongue with changes to the tongue papillae (atrophied or hypertrophied), and the development of cracks in the tongue, and sores at corners of mouth.

**Magnesium** is needed for **muscles** to relax, thus preventing spasms in the intestines.

Vitamin A is needed for mucous production and healthy cells.

The following foods and herbs can be used to correct or prevent constipation:

### Atonic constipation can be helped by the following foods:

- Carminatives are herbs or a preparation intended to either prevent formation of gas in the
  gastrointestinal tract or facilitate the expulsion of said gas, thereby combating flatulence and
  a sluggish bowel.
- Raspberry, celery and lemon are carminatives.
- Horseradish is a carminative and also a digestive stimulant in small amounts.
- Carrot is a carminative and also a cleansing digestive tonic.
- Grape is a carminative, bile stimulating and cleansing remedy for sluggish digestion and laxative.
- Rice bran acts as a laxative with 8 grams fiber in ¼ cup. Use as cereal or in cooking.
- Asparagus acts as a laxative and is also a liver stimulant.
- Figs are laxative and counter habitual constipation.
- Pear (ripe) is an excellent laxative and cleansing tonic.
- <u>Strawberry</u> is laxative and also a liver tonic.
- Onions and Garlic stimulate and improve digestion and also cleanse the gastrointestinal tract to help maintain healthy gut bacteria, prevent fermentation and tone the intestine.
- **<u>Pineapple juice before eating</u>** can help stimulate a sluggish digestion. It is cleansing and nutritious, rich in minerals.
- Red beets stimulate and improve digestion and are easily digested.
- Cabbage stimulates and improves digestion and is also a liver decongestant.
- <u>Coffee</u> stimulates and improves digestion and also increases gastrointestinal activity in many people.
- Artichoke stimulates and improves digestion and is also a laxative, bile stimulant and liver tonic.
- <u>Lettuce stimulates and improves digestion</u> and is also an alternative, meaning it improves the function of organs involved with the digestion and excretion of waste products

### **Substances that cause constipation:**

- Antacids (tums)
- Antidepressants
- Anticonvulsants
- Calcium channel blocking drugs (in heart disease)
- <u>Iron</u> supplements
- Laxative abuse
- Opiates, like morphine or codeine, also heroine, most other painkillers

### **Minerals and Metals**

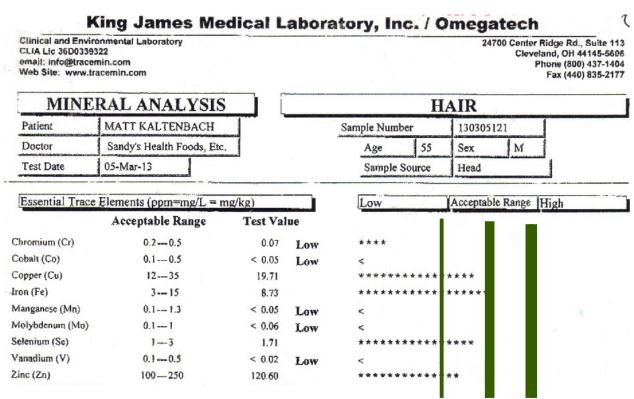
The plain fact is that **parasites absorb and steal minerals and metals**. When the body is under attack many many substances are lost. Acidosis also causes a loss of minerals and metals. The two

fisted punch depletes so many minerals and metals, the body becomes seriously unstable. I took repeated hair analysis tests every year, which showed no (**Below the measurable limits**) essential minerals or metals. Acidosis wipes out entire segments of essential elements the body needs to function.

#### Metals

I started taking supplements in 2012. A year later In 2013 many metals were low, including **Chromium**, **Cobalt, Manganese, Molybdenum**, and **Vanadium**. By low, I note, that the level of these metals is outside the acceptable range. Also noteworthy, are the transitional metals, in the center of the periodic table, metals which have open electron orbits in their outer band. These metals are reactive, and are capable of bonding with various substances to become compounds. The absence of these metals will hamper an effective and healthy body chemistry.

Attached below is a hair analysis of essential trace metals.



The need to rebuild the immune system is essential. The use of minerals to restore body processes, and metals to stimulate the immune system are essential steps in restoring the body to health. **Chromium**, **Vandium**, and **Molybdenum** are thought to be associated with immune system health. **Copper**, and **Zinc** are essential for digestive and mental health. **Selenium** is essential to get oxygen into the body.

**Molybdenum** helps the body maintain the zinc/ copper ratio.

The metals consumed by parasites include metals that are heavier than <u>Iron</u>. <u>Iron</u> in particular is consumed by parasites. They can steal so much iron, a person can become anemic. Supplementing <u>Iron</u> is thought to make worms stronger. Flukes in particular steal iron from the GI tract. Black stool may be loss of some blood and iron. Take spinach with <u>PZQ</u> to keep iron level up during a <u>fluke</u> <u>kill</u>.

**Zinc** is consumed, so much so that the body cannot make white blood cells. **Zinc** commands the body to build white blood cells. Together with **Selenium**, they make a powerful pair.

**Iodine** is consumed, as the body attempts to heal itself from thousands of little wounds. My initial reaction to Iodine was severe. I went back to the herbal store and complained it was to strong. I did not realize how sick I really was. It is seldom the supplement, it is the environment that the parasites create that makes the reaction so severe. Skip a few days between doses (Pulse) if you get a severe reaction to **iodine**.

**Copper** is essential for maintaining protection against bacteria in the gut. **Copper** is used for several key enzymes. **SIBO** bacteria overgrowth is the result in the loss of the immune system and **Copper**. **Copper** supports good bacteria, and interferes with bad bacteria types.

<u>Magnesium, Boron, Silver, Vanadium, Molybdenum,</u> and other metals can be used to bring the body back into balance.

It is critical that these metals are present, especially during the beginning phases of a parasite kill. Later, depending on the parasite(s) present, **the removal of Zinc** may be required in Phase 4 to complete the Ascaris kill. Guidance is provided later in this document to help transition through these phases.



I had no improvement with silver, that is why it did not make it into the final formula.

#### **Minerals**

Minerals are also consumed by the body in large amounts, especially during periods of stress.

To understand the role of vitamins and minerals, it helps to understand a little bit about the chemistry of your body. The body runs on highly complex processes to perform and regulate those internal functions that are necessary to maintain life. Processes are in place to:

- Build the cells that make up your body's structures
- Meet the needs of different types of cells
- Permit each of these different cell types to carry out its specific functions
- Help maintain cellular structure and performance
- Alter the type and degree of cellular activity in the face of changing circumstances
- Communicate each cell's activity with the activity of other cells and organs in the body
- Adjust cellular behavior, as needed, in ways that will permit a coordinated response of the whole organism to its environment
- Dispose of waste products
- Govern the disposal of cells when their job is done

The scientific term used to describe all of these processes is homeostasis.

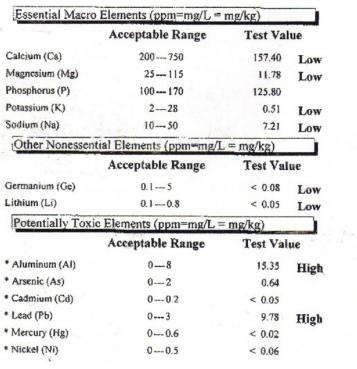
Homeostasis is defined, as the ability of a living organism to maintain its structure, separate from and invulnerable to the forces of its environment.

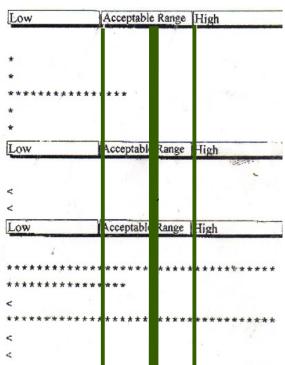
The maintenance of homeostasis is highly complex, requiring many series of chemical reactions, distinct and yet interrelated. Each step in each reaction requires the presence of specific substances. If these materials are not present, the reactions that require them cannot proceed correctly.

Vitamins, minerals, and trace elements are the necessary substances. Some are produced for us within the intestines, by bacteria that live there.

Higher amounts of some of these substances may actually improve our health even when we're not sick, by giving our bodies a richer source of materials to draw on in the face of normal stress.

In my case the hair analysis showed I had no minerals or essential metals. These results were confirmed year after year, despite my best efforts to supplement. Parasites cause depletion of these essential elements, unless the parasites are killed.





#### **RDA vitamin Dose**

The RDA, or recommended dietary allowance, is a baseline figure. It is used to suggest the lowest amount of a substance that we should take in, daily, from our diet. You'll see references to the RDA on labels for vitamins and for many prepared foods. With certain vitamins, the upper limit and lower limit are much closer together, such is the case with **Vitamin A**.

It's important to understand that <u>RDA</u> is not a recommended intake level. Rather, it represents the minimum amount required to prevent an overt, frank deficiency-in healthy people with good absorption and the ability to maintain normal nutritional status. Thus the RDA holds very little meaning for you.

<u>The RDA underestimates the requirements of an organism under stress</u>. It is not a good guide for your nutritional or dietary intake. For <u>vitamin C</u>, for example, the RDA is 60 milligrams a day-just enough to prevent scurvy in a healthy sailor.

The RDA also does not take into account specific actions of a given vitamin that can be of special value in illness, or may promote your overall good health.

### **Kidneys**

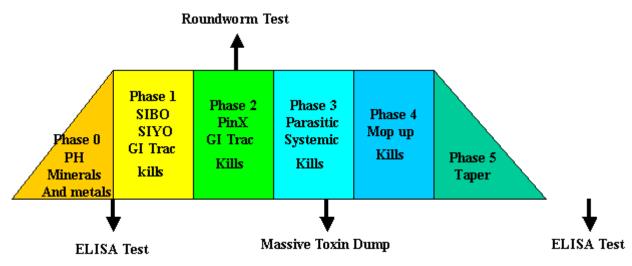
The kidneys are one of the first organs to bear the brunt of a parasitic infection. Daily tea's, Herbs, supplements like Dr Christophers kidney cleanse, and KGP flush, are required to keep the kidneys functioning. Milk thistle, CQ10, and Ginger/Golden seal may be required to keep them functioning. Keep vitamin D3 below 50,000 units, and confine this dose to the killing of flukes (flat) worms. As soon as these worms are eliminated, lowering the D3 level to the minimum is advised.

### **My standard Process:**

The standard process includes 6 stages, including stabilize health by ramping minerals and metals. It is essential to quickly balance the body, this is done by supplementing minerals, vitamins, metals, amino acids, and removing toxins.

- 1. Attempt to stabilize the digestive process by killing **SIBO** and **SIYO** GI tract infections.
- 2. Physically clearing the GI tract of parasites using (Takes about a month) PinX, then
- 3. Killing systemic parasites (Takes about 4 to 6 weeks),
- 4. Moping up the kill which takes another 4 to 6 weeks,
- 5. Tapering of the substances used.
- 6. Then moving to lifetime maintenance supplements.

#### **Standard Process Diagram**



### Phase 0 – Supplement, Balance, and ELISA Testing:

The body must work properly to kill parasites. Ramping supplements to get the body toxin level lower is essential to killing parasites safely.

Normally parasite infections are self limiting, which means they grow until they sense toxin levels, and slow their growth to match the environment. Many of these sensing regulations are nitrogen based. When the toxin level is lowered, the new cleaner environment encourage the parasites to grow.

It is possible that as soon as the toxin level is lowered, more parasites are born, or the parasite worsens. the introduction of anti-parasitics (**DEC** and **Piperazine**) may be required to limit larvae and egg growth of roundworms, and assist in clearing the GI trac.

It is possible that the removal of toxins causes an improvement in the parasite infection, weakening them, and making it easier to remove them.

Balance of the body also makes the body healthy enough to withstand the parasite killing process. Rushing the increase in PH, dosing of MSM and **Spirulina**, will cause a healing crisis. Take the time to conduct early testing of Roundworm (ELISA), Flatworm Fluke (ELISA) or other parasite tests can be performed during this phase. These ELISA tests will help to confirm a total kill at the end of the killing process. Vitamins, minerals and metals can also be introduced.



#### **Q&A 1**

**Q>** I am about to send email to internal medicine doc at hospital they assigned me to after I went to emergency last week to try to get someone to do tests.

They have stool in lab and tissue I gave them from lesions. Whether or not they will actually culture tissue is a question. He may have agreed only to humor my request to do so.

A> Canada has more parasitologists than the USA. I think 133 at my last count. If they have a hospital pathologist, your odds are greater. There is a blood lab in atlanta GA, and there may be a few underground PCR analysis labs setting up shop. There were high visability labs, but they shut off PCR a few months before I spent \$1500. having my stool tested. It is a crap shoot (pardon the pun)

Q> If I am going to add for ELISA what exactly do I ask for. How do I phrase it? We have NO private labs here in BC.

In a calm low voice request ELISA blood tests.

I had ELISA for Ascaris (which was positive). I had test for Strongyloides (negative), and Filariae (negative)

Each singular test is about \$50 - 300 depending on greed. Tell them you want a copy of the test report, and scale of infection(s). Many report 0.26, 0.48, 0.96 of full scale threshold 1.0. They do not consider you infected if you are below the

threshold, just managing it with your immune system.

I should have had ELISA for Flukes.



- <u>liver flukes</u>, <u>Clonorchis sinensis</u> (oval flat) and <u>Fasciola hepatica</u> (Oval fleshy peach pit).
- Blood flukes inhabit the <u>blood</u> in some stages of their <u>life cycle</u>. Blood flukes include <u>species</u> of the <u>genus <u>Schistosoma</u> (red bumps on skin).
  </u>

Of these three flukes, I had all 3, my initial dose of Praziquantel was to low, and I went another year in hell until I discovered the depth of my fluke infection went into the brain, and beyond.

Having a parasite is normal, everyone has one. It is when you have 2, or 3 you are in trouble, I had 5 (that I know of)

### **Q&A2**

Q> why was my stool test negative for Ascaris?

A> Stool samples are less than 8% accurate.

PCR test is more accurate, no one does this anymore, it identified problems that doctors were not interested in treating.

ELISA tests are proving to be the most accurate way of determining which parasite you have.

Ascaris is one of the hardest worms to get rid of. The reason is that:



- 1) No one has just one parasite
- 2) The Ascaris depresses the immune system which makes one more likely to catch an additional parasite that presses on the immune system further.

In my case, I had a severe fluke infection that made all of my attempts at clearing Ascaris impossible.

Finally I wiped out the flat worm flukes, and made real progress. I am still feeling my way through the Ascaris infection, down to a right sinus nest, and possible lower colon group.

#### Q&A

Q> hey matt. I've been waiting on the hair analysis forever.. Just got the doxycycline, getting hair analysis results tomorrow. Also i looked at old tests and turns out i made my doc give me iGg4 antibody test for filaria. It came back super low so thats negative. But I'm going to brg in to my appointment tomarow that i need test for <a href="Ascaris">Ascaris</a> 209 kda and fluke 13 kda as the dominant antigen detections, and angiostrongylus, cysticercosis, echinococcossis and hookworm if i can. lol. I have been taking the other medications as well but since i have been loosing weight since this started.

I'm thinking i have  $\underline{\text{Tapeworm}}$  and also i do see specs of white in stool. But there's something definitely, big moving around throughout my whole body which is the most unnerving. I was wondering if u recommend taking niclosamide to at least kill the tapeworms. ICU has it in his  $\underline{\text{Tapeworm}}$  protocol. Just wondering . thx

#### **Q&A - ELISA**

http://www.rapidtest.com/products-elisakits.php?product=Parasitology-ELISA-kits&cat=17

http://www.rapidtest.com/index.php?product=Rapid-Tests&cat=3

http://www.rapidtest.com/index.php?i=Ascaris-IgG-ELISA-kit-&id=751&cat=17

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### **Filaria**

# MAYO CLINIC Mayo Medical Laboratories

#### Laboratory Service Report

1-800-533-1710

Patient Name SAMPLEREPORT,FFAG4 NORMAL	Patient ID SA00067389	Age 43	Gender M	Order # SA00067389
Ordering Phys CLIENT, CLIENT			DOB 11/09/1970	
Client Order # SA00067389	Account Information		Report Notes	
Collected 05/07/2014 13:00	C7028846-DLMP Rochester SDSC 2 - Client Support Rochester, MN 55901			
Printed 05/09/2014 09:04				

Test Flag Results Unit Reference Value Site\*

Filaria IgG4 Antibody 1.49 Y03

REFERENCE RANGE: <1.50

INTERPRETIVE CRITERIA:
<1.50 NEGATIVE
1.50-3.00 EQUIVOCAL
>3.00 POSITIVE

This assay detects Filaria IgG4 associated with infections caused by the major filarial parasites, including Dirofilaria immitis, Wuchereria brancrofti, Brugia malayi, and Onchocerca volvulus. Detection of IgG4 subclass antibody offers enhanced specificity without sacrifice of sensitivity. Chronic filarial infections manifesting as elephantiasis may not show a significant IgG4 response, and cannot be ruled out by this assay. Equivocal results may represent cross-reactive antibodies induced by infection with other nematodes.

This test was developed and its performance characteristics have been determined by Focus Diagnostics. Performance characteristics refer to the analytical performance of the

RECEIVED: 05/08/2014 11:05 REPORTED: 05/08/2014 13:12

#### Filaria

James B. Peter, M.D., Ph.D.

Seven filarial organisms cause infections in man. Subsumed under the term "filarial infections" are lymphatic filariasis caused by *Wuchereria bancrofti* (>90%) and *Brugia* spp. (<10%), non-lymphatic, subcutaneous filariasis (*Loa loa, Onchocerca volvulus, Mansonella streptocerca*) and non-lymphatic, serous cavity filariasis (*Mansonella ozzardi, M. perstans*). Living, dead or degenerating worms (2-50 cm long) cause the inflammatory lesions of filariasis and ultimately result in lymphatic obstruction and fibrosis, which is at least partially reversible. Live worms produce swarms of microfilariae (immature larvae), which are transmitted by mosquitoes.

The most important advances in lymphatic filariasis stem from development of antigen detection tests, which are more sensitive diagnostically than microscopy. These tests have been the basis of a critical evaluation of promising new therapy and also have taught us a great deal about the natural history of lymphatic filariasis which was not readily defined by serology alone.

All filarial worms share some unique characteristics: (a) adult females produce a larva called microfilaria which shows a circadian rhythm or periodicity often synchronized with the biting behavior of their vectors; (b) all are carried by blood- sucking arthropods; and (c) have the same basic life cycle with five larval stages of development. The mosquitoborne lymphatic filariases (including asymptomatic microfilaremia, classical elephantiasis and tropical pulmonary eosinophilia) are caused by Wuchereria bancrofti, Brugia malayi and B. timori. The non-lymphatic filariases, manifested by cutaneous and subcutaneous infections include: (a) Onchocerca volvulus which causes cutaneous and subcutaneous nodules or onchocercomata and is transmitted by black flies; (b) Loa loa or the "eye worm" of Africa which moves freely through tissues and is carried by horseflies; and (c) Mansonella ozzardi, M. perstans and M. streptocerca, a comparatively innocuous group of filariae that cause little or no pathology in the human host and are vectored by biting midges. 1 Occasionally, one of the most prevalent animal filaria, *Dirofilaria immitis*, the heartworm of dogs, causes pulmonary and extrapulmonary infections in humans.<sup>2</sup> Detection of filaria-specific IgG and IgE antibodies by EIA with soluble adult filarial antigen from *B. malayi*<sup>3</sup> is hampered by cross-reactive antibodies found in sera of patients with closely related infections, including non-filarial nematodes (e.g., Strongyloides stercoralis, Trichinella spiralis and Necator americanus), trematodes or flukes (e.g., Schistosoma mansoni and Echinococcus granulosus) and surprisingly with some protozoan infections, including *Trypanosoma cruzi* and *Leishmania* spp. (cf. 3 for succinct review of the laboratory evaluation of the filariases). Both total IgM and filaria antigen-reactive IgG4 are greatly elevated in lymphatic filariasis. 4 Detection of B. malavireactive IgG4 improves the specificity of detection of filarial antibodies, <sup>5-6</sup> probably due to the exclusion of antibodies to phosphorylcholine (PC) from this subclass. PC is an immunodominant epitope in many infections, <sup>7</sup> and the development of assays that spotlight production of antibodies directed against non-PC-containing antigens marks a major advance.<sup>3</sup> Decreases of *B. malayi* IqG4 follow treatment.<sup>8</sup> Serum antibodies to a recombinant antigen (recSXP1) are found in 69% and 82% of patients with patent Malayan or Bancroftian filariasis, respectively. 9 Detection of filarial-specific IgE is enhanced by removal of IqG4 from the serum. 10 Antigen detection for diagnosis and monitoring of therapy is promising, 11,12 but workshops, standardization and exchange of specimens and reagents are urgently in need of expansion. 13 A specific, sensitive monoclonal antibody-based EIA, directed against antigens of *O. gibsoni* is useful to detect circulating *W. bancrofti* antigen in human serum. 14 The spectrum of Bancroftian filariasis is elucidated by detection of circulating antigen (reactive with Oq4C3) which is more sensitive than microfilaremia which misclassifies ~50% of those infected in an endemic area. <sup>15</sup> A similar assay works well for detection of filarial-specific IgG4 in urine. <sup>16</sup> Ultrasonography for detection of the filarial dance sign and subclinical hydrocele yields a powerful tool for epidemiology when combined with antigen detection for Bancroftian filariasis. <sup>17</sup> The combination of filarialspecific iaG4 in urine and filarial antigen detection in whole blood is also very effective. 18 The Brugian filariasis elimination program will be aided by a rapid dipstick test (Brugia Rapid) for detection of IgG4 antibodies to a recombinant antigen of *B. malayi*. <sup>19</sup> Crossreactivity in patients infected with *Brugia* spp., *O. volvulus* or *Loa loa* is absent. <sup>14</sup> Cloning of potentially protective antigens<sup>20</sup> from filarial parasites bodes well for improved laboratory diagnosis. Sensitivity of serodiagnosis for filariasis varies with the causative agent, the technique employed, the absence or presence of microfilaremia and with the clinical setting. Where appropriate, membrane filtration of blood, Giemsa-stained thick and thin smears or QBC® (Quantitative Buffy Coat) are the microscopic tests of choice, 3,6,21 Serology and skin tests cannot distinguish between heavy versus light

parasite load nor between past or present infections.<sup>22</sup> Among other important differences, parasitic helminths, in contrast to microparasites (including bacteria, protozoa and viruses), generally do not multiply in humans; this difference may be fundamental to the slowness with which acquired immunity to macroparasites (e.g., helminths) develops in humans.<sup>23</sup> The feasibility of using ultrasonography of the scrotum for detection of living adult worms and lymphatic dilation was demonstrated.<sup>24-26</sup> Vector infection rates tend to predict infection prevalence in humans.<sup>27</sup> PCR-based studies suggest that some individuals have circulating filariae DNA in the blood in the absence of microfilariae.<sup>28-30</sup> Similar studies on wild-caught anopheline mosquitoes will also be useful in detecting the parasitic load in the vector populations.<sup>31</sup> PCR-based evaluation of Bancroftian filariasis may also serve as a tool in detecting filaremia.<sup>32</sup> A PCR-RFLP technique to detect filarial parasite DNAs in blood and mosquito samples can be valuable to differentiate between species of filaria in humans, reservoir hosts and the vectors in endemic areas.<sup>33</sup>

#### See Also:

Brugia spp./Lymphatic filariasis

Dirofilaria immitis and D. (Nochtiella) repens

Loa loa

Mansonella ozzardi, M. perstans and M. streptocerca

Onchocerca volvulus

Wuchereria bancrofti

#### Filaria Manifestations can be protean and classified as:

**1) Acute** – Fever with chills and rigors, lymphedema with pain, lymphadenopathy (cervical, axillary, inguinal and generalised – Acute Filarial Lymphangitis/Acute Dermatolymphangioadenitis), chyluria, hematuria, inflammatory granuloma or abscesses, pain in testes, funiculitis, epididymoorchitis.

- 2) **Chronic** funiculitis, epididymoorchitis, hydrocele, breast edema, elephantiasis.
- 3) *Occult* Pulmonary eosinophilia, mono and polyarthritis, tenosynovitis, glomerulonephropathy, retroperitoneal lymphangitis (acute abdomen), central serous retinopathy, iridocyclitis, recurrent scleritis and macular oedema, endomyocardial fibrosis, urticaria, recurrent upper respiratory infections, asthmatic bronchitis. Any lymph node or any body part can be affected but commonly genital lymphatics are involved in males.
- 4) **Asymptomatic** Endemic normalsnegative for Mf but positive for antigens (pre-patency) and asymptomatic microfilaremic is characterised by the presence of microfilaria in peripheral blood during night but without any overt clinical manifestations of filariasis with or without antigens also known as Mf carriers (patency). 1-3,6,8

Factors affecting pathogenesis of filarial manifestations include the cumulative exposure to bites, quantity of accumulating adults, number of secondary infections, the degree and type of host immune response (Th1/Th2) and possibly genetic predisposition. 11-13

Hydrocele is less common in microfilaria carriers than endemic normals. Lymphatic filarial parasites also harbor an endosymbiont – Wolbachia - that contributes to inflammation. 14 Commonly, the diagnosis of lymphatic filariasis relies upon suggestive clinical and epidemiologic clues and supportive laboratory findings. The morbidity of human filariasis results mainly from the host reaction to microfilaria and adult worms in different areas of the body which increases with duration. 14-16

So, early diagnosis and treatment are the best options. The patients of acute and chronic manifestations usually come for medical attention while the very big pool of asymptomatic and occult infections are usually inaccessible individually, their coming to medical attention is primarily by mass community screening or as a chance finding.

#### **DIAGNOSIS**

Most of the clinicians rely on their clinical acumen in the diagnosis of clinical filariasis (low specificity and sensitivity for acute or asymptomatic active infections). 15, 16 Laboratory tests can be divided into nonspecific

and specific tests. Specific tests include - direct detection of microfilaria on blood smears, serologic tests, DNA PCR and radiology. Nonspecific tests are eosinophilia, high IgE levels and lymphoscintigraphy (that reveal dilated lymph channels or backflow even in the early stage of infection).

The direct methods include visualization of microfilaria (or the adult worm) - is made by microscopic examination of thick film of blood collected between 10:00 PM and 2:00 AM, with or without DEC provocation, stained by Geimsa or hematoxylineosin for the presence of microfilaria. Adult worm may be found in fluids drawn from swollen areas or serous collections.

X-ray tests can show calcified adult worms in lymphatics; ultrasonography can show the 'filarial dance'.1,2,7 Lymph node aspirate and chylus fluid may also yield microfilaria or worm.

Direct diagnosis, though definitive, is difficult, because of timing inconvenience of blood collection, long prepatency, low patency (<60%) and inadequate sensitivity. Reliance on microfilaria testing may lead to late as well as under diagnosis; it is necessary to develop diagnostics for early detection of the disease. 16-18

Early diagnosis of filarial infections is best possible with seromarkers. With the entry of the microfilaria there is a natural IgM response within a few weeks which slowly changes to an IgG response – initially IgG1 and IgG2 – which changes to IgG4 after some more time. 11-13 Antigens appear with development of adult worms from microfilaria. This appearance of antigenemia from both adults and the larvae leads to increased easily detectable levels of IgG4. Antibody detection has served as the basis for diagnostic assays for filariasis for many decades, especially with the native antigens. The best of these assays were sensitive for infection but cannot distinguish current infection from past infection or exposure to other parasite and there was significant degree of crossreactivity with other helminth infections – leading to poor specificity (~40%). 15-17

With the advent of recombinant antigens antibody tests have become refined. 19, 20, 21 Antibody-based diagnostic assays using four recombinant antigens, Bm14, WbSXP, BmSXP and BmR1 have become commercially available.22-26 They are based on the detection of antifilarial IgG4 antibodies. The BmR1 ELISA as well as dipstick (Brugia Rapid immunochromatography based) antibody tests have very high sensitivity for Brugia malayi (~100%), Bm14 ELISA is sensitive for both Wuchereria bancrofti and Brugia malayi (~91%-96%), and WbSXP was predominantly sensitive for Wuchereria bancrofti (91%) and somewhat less for Brugia malayi (40%). BmSXP is another recombinant antigen having high sensitivity for Wuchereria bancrofti (95%).25, 26 Both WbSXP and BmSXP(WB Rapid) have high specificity for Wuchereria bancrofti (96-99%). A combined test format using BmR1 and BmSXP has been advocated by some as a more comprehensive test of pan filariasis.26,27,28.

'Seva Filachek' is a dipstick based ELISA system which has been permitted by government of India (Signal MF) for microfilarial antigen(IC-Ag) as well as filarial antibodies (IgG4) in diagnosis of filarial infection in different clinical groups. The detection of IgG4 antibody titre of 1:300 and above against specific microfilarial antigen was found to be useful. Free as well as immune- complexed antigen could also be detected. Overall the test system has a sensitivity and specificity of around 80% for antibody detection and 88% Medicine Update 2012 

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for antigen detection.27-30. There was no interference from non-filarial helminths or other immune activation states like positive rheumatoid factor, ANF or high levels of IgE.

However the problem of specificity especially in the form of distinguishing past from present infection and rarely other helminths remains.<sup>30</sup> The rapid format assays, though more convenient can give indeterminate result in upto 30% cases, which is unusual in ELISA.<sup>27-30</sup>. The 'OnSite' Filariasis IgG/IgM Rapid Test uses conserved recombinant antigens to simultaneously detect IgG and IgM to the Wuchereria bancrofti and Brugia malayi parasites without the restriction on specimen collection. Recombinant multiple beaded antigens including Bm14 and Bm33 from Brugia malayi has been used to follow therapy response by determining IgG4 levels. Bronchoalveolar lavage fluid of patients with tropical pulmonary eosinophilia contains IgE antibodies that recognize Brugia malayi antigen, Bm 23-25.<sup>30,27</sup>

Filarial Antigen tests, available commercially, are probably better than antibody tests. They show little 24 hour variation in their positivity can detect active infection (better specificity) and have a significantly wider user experience and evidence base for monitoring therapy.31-33 This antigen test is available only for infection caused by Wuchereria bancrofti but not for Brugia. Immunochromatographic test (ICT) is a highly sensitive and specific circulating filarial antigen (CFA) detection assay, both as card test (AD-12.1 antibody) and in ELISA based format (Og4C3 antibody— 'Tropbio') are now available for the diagnosis of Wuchereria bancrofti infection.32-37 This test is positive in early stages of the disease when the adult worms are alive and becomes negative once they are dead. The Card test is a qualitative test with possibility of indeterminate results but very rapid; the Elisa format is a semi quantitative test that provides definite results.36-38 Filarial antigen detection has been found to be more useful in epididymoorchitis and allergic state such as tropical eosinophilia and in lymphedema. 'Binax Filariasis Now Test' is a variety of commercial card test for Wuchereria bancrofti antigen.37

DNA probes using Real-Time Polymerase Chain Reaction (RT-PCR) are of high specificity and sensitivity, but are not cost effective.<sup>39</sup> Assays for circulating immune complex of filarial antigen and antibodies are also used for serodiagnosis in microfilaria negative cases.<sup>40</sup>

#### CONCLUSION

With the knowledge of filarial endemicity, its associated morbidities and the national and international intervention strategies for its elimination, it nearly becomes obligatory for any practising doctor to have some working ideas on this disease. Serological tests are the tests of choice for early markers of infection, but they can be hardly used in individuals as the disease is most often asymptomatic - all age group mass screening is the preferred option.<sub>41,42</sub> Antibody detection provides an early means to detect filarial parasite infection.

Presence of IgM antibody to the parasite antigens suggest current infection, whereas, IgG corresponds to late stage or past infection. Utilization of recombinant proteins eliminates cross-reaction. 43,44 Furthermore, identification of conserved antigens allows 'pan-filaria' test to be applicable. However antibody testing is probably better in children. Antigen positivity indicates active disease and it can be used both in sera and body fluids and also in urine and it is the better test considering its specificity. In nearly all antigens positive cases the antibody will be positive and both will be positive in microfilaria positive cases. 45

Only antibody positivity without antigen or microfilaria may indicate early infection especially the IgM variety but commercial kits for its detection are hardly available

## Ascaris IgG ELISA kit description:

The Diagnostic Automation Ascaris ELISA test is a qualitative enzyme immunoassay for the detection of antibodies to Ascaris, in samples of human serum or plasma.

#### **Material Provided with Ascaris IgG ELISA Kit:**

- 1. Plate: 96 Microwells containing Ascaris antigens
- 2. Enzyme Conjugate: Protein A conjugated to peroxidase
- 3. Positive Control: diluted rabbit sera
- 4. Negative Control: diluted human sera.
- 5. Chromogen:TMB
- 6. Wash Concentrate 20X: buffer and surfactant
- 7. Dilution Buffer: buffered protein solution
- 8. Stop Solution: 1 M phosphoric acid

#### **Materials Required, not Provided:**

- 1. Freshly distilled or deionized water
- 2. Dispensing system and/or pipette
- 3. Microshaker for dissolving and mixing conjugate with samples
- 4. EIA kit Microplate washer
- 5. EIA kit Microplate Reader with 450nm or dual wavelength 620-650 nm filter

#### **Ascaris IgG ELISA Kit Background Information:**

Ascaris lumbricoides has probably been infecting humans for thousands of years. It is the most common nematode parasite infecting humans and over 1 billion people are believed to be infected. Children who live in moist, warm climates are the most at risk to become infected. Ingesting embryonated eggs from contaminated soil is the primary means of infection. The eggs will hatch in either the stomach or small intestine where the larvae penetrate through the intestine wall. Larvae are carried to the heart and then to the lungs, where they stay for approximately 10 days. Larvae will then go into the alveoli and migrate via the bronchi to the trachea and pharynx. The larvae are coughed up, swallowed, and returned to the intestine where they mature and mate, eventually producing eggs. This process occurs over 8-12 weeks. Eggs will get passed into the environment via feces. Fertilized eggs will become infective within 2 weeks if they are kept in warm, moist soil. The primary means of preventing the spread of Ascaris infection is through the use of appropriate sanitation facilities and practices, such as frequent washing of the hands.

#### **Ascaris IgG ELISA Test Principle:**

The microwells are coated with Ascaris antigen. During the first incubation with the diluted patients sera, any antibodies which are reactive with the antigen will bind to the coated wells. After washing to remove the rest of the sample, the Enzyme Conjugate is added. If antibodies have been bound to the wells, the Enzyme Conjugate will then bind to these antibodies. After another series of washes, a chromogen (TMB) is added. If the Enzyme Conjugate is present, the peroxidase will catalyze a reaction that consumes the peroxide and turns the chromogen from clear to blue. Addition of the Stop Solution ends the reaction and turns the blue color to a bright yellow color. The reaction may then be read visually or with an ELISA reader.For additional details please refer to the instructions for use.

Diagnostic Automation Inc. also provides other Parasitology ELISA Kits. For more information about ELISA Kits, Rapid Tests, IFA Kits, CLIA Test Kits, or Serology tests, please see our website home page, or contact our Customer Service Representatives at 818-591-3030

#### **Strongyloides**

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STRONGY		Semi-Quantitative Enzyme-Linked Immunosorbent Assay		Diarrhea
Performed 6		Reported 6	_	▶ Interface Map
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New York DOH Approval S	tatus 🛭		_	
This test is New York DOH appro	ved.			
Submit With Order 6			_	
Specimen Required 6				
	Patient Preparation:			
	Collect:	Serum separator tube or plain red.		
Spe	ecimen Preparation:	Transfer 1 mL serum to an ARUP Standard Transpor	t Tube. (Min. 0.05 mL)	
Storage/Tran	sport Temperature:	Refrigerated.		
Unac		Bacterially contaminated, heat-inactivated, hemolyzed	f, icteric, or lipemic specimens.	
	Remarks:			
	Stability:	After separation from cells: Ambient: 48 hours; Refrig	erated: 2 weeks; Frozen: 1 year	

#### Leishmania



### AccuDiag<sup>TM</sup> Leishmania **ELISA Kit**

Cat # 8203-35



Test	Leishmania ELISA		
Method	Enzyme Linked Immunosorbent Assay		
Principle	Sandwich Complex		
Detection Range	Qualitative Positive ; Negative Control		
Sample	10 μL serum		
Total Time	~ 25 min.		
Shelf Life	12 Months from the manufacturing date		
Specificity	84%		
Sensitivity	97%		

#### **INTENDED USE**

The Leishmania ELISA Kit is an enzyme-linked immunosorbent assay (ELISA) for the qualitative screening of IgG antibodies to visceral Leishmania in serum.

#### SUMMARY AND EXPLANATION

Leishmania is a widespread disease affecting millions of people around the world

to any antibodies present. Before the third incubation necessary. Then a chromogen (tetramethylbenzidine or TMB) is added. With the presence of Enzyme Conjugate and the peroxidase causing the consumption of peroxide, the chromogen changes to a blue color. The blue color turns to a bright yellow color after the addition of the stop solution, which ends the reaction. ELISA readers can be used to obtain results

#### SPECIMEN COLLECTION AND PREPARATION

Coagulate blood and remove serum. Freeze sample at -20 °C or lower if not used

Do not heat inactivate serum

Avoid repeated freezing and thawing of samples

#### MATERIALS AND COMPONENTS

#### Materials provided with the test kits

- Plate: Microwells containing Leishmania antigens 96 test wells in a test strip
- Enzyme Conjugate: One (1) bottle containing 11 ml of anti-human Ig-Peroxidase (HRP) in a stabilizing buffer with Thimerosal.
- Positive Control: One (1) vial containing 1 ml of diluted Leishmania-positive human sera in buffer with Thimerosal.
- Negative Control: One (1) vial containing 1 ml of diluted Leishmania negative human sera in buffer with Thimerosal.
- TMB Substrate Solution: One (1) bottle containing 11 ml of the chromogen tetramethylbenzidine (TMB). Wash Concentrate 20X: One (1) bottle containing 25 ml of concentrated
- buffer and surfactant
- Dilution Buffer: Two (2) bottles containing 30 ml of buffered protein solution
- Stop Solution: One (1) bottle containing 11 ml of 1 M phosphoric acid.

#### Materials required but not provided

- Squeeze bottle for washing strips
- ELISA plate reader with a 450 nm and a 620-650 nm filter(optional if results
- Tubes for serum dilutions

#### Preparation

#### **Q> I hear Elisa tests are not Accurate.**

#### Α

Many emails have I replied to, where the sender tested positive for Strongyloides.

;;

There are a few WHO approved, developed by the Bill and Melinda Gates foundation, to the tune of many tens of millions of dollars. They cost \$1 to 3 dollars in a stick form, 2 bottles fluid.

The test is accurate for the high visibility parasite infections, and was developed using international standards practices.

The research ELISA array has less that such a stellar reputation.

The 62, 72, 93 well test looks more like a dna test, and the results are up to interpretation.

Seams like if you have multiple parasite infections, some enzyme antigens dont read correctly, the accuracy for single test that are more species specific, can fall from 85-95% down to 40%.

The major worms, flukes, ascaris, strongyloides have a higher accuracy, since the antigen they represent, are also common with other species.

If the other species testing needs to be done, in the event of primary care practices, then and only then can PCR analysis be done, in facilities like Duke, but they do not accept patients or referrals. I tried.

Even Mayo sends samples to Duke.

They turned me down too.

#### **Q&A3**

Q> How can I start ?

A> Get a Hair analysis and get PH strips. A physical to test your blood is also a good idea.

...Hey Matt,

So, I'm trying to be optimistic but feel pretty scared. I'm fairly certain they are in my brain, should I be worried?

...in your brain,

...or in the lymph in your skull, up down throat

...or in your sinus, ears,

...or embedded into the BBB

These are all different.

ONLY ONE do you worry about.

IF you have unsymmetrical neural symptoms,

I.E. your right elbow shakes every afternoon between 2 to 4 pm, or your left leg walks left when you ask it to go right, in the morning,

These are unsymmetrical neural symptoms, are abnormal, and need to be seen by a doctor, and a MRI is ordered up.

Symmetrical symptoms are normal.

Double vision is ok.

Headaches are ok.

Burning in the back of the skull, electrical zapps, electroshock, white flashes in the eyes are normal. Ear ringing is normal.

Falling to the floor is normal.

Huge rashes are normal.

Bumps (cysts) under the skin or in a muscle or two is normal.

Cysts on your chest, back of the neck, are normal, if there are only a few.

Brain Fog is ok.

Cold nose, face, skin.

Whipping in sinus or on ear drum is ok.

Worms traveling around the body is normal.

Worms trying to get into your eye is normal.

Worms getting into you eye is abnormal....

Numb skin, burning lips, poor vision, hearing, memory, problems walking or doing tasks, these are all normal symmetrical neural disturbance.

Unsymmetrical neural symptoms are caused by a pork or beef tape worm, which can cause focal

(local specific and repeating) deficits (holes in the brain). This guy you worry about, the rest of the guys, it is just a game of time until they are dead.

If you have unsymmetrical neural symptoms get a MRI, and if abnormal, get a CAT scan. Its worth the radiation if you truly have this one kind of parasite. Thank god it is a rare parasite.

I believe you have a situation of round worms and rope worms. Rope worms are congested lymph matter. Dodder seed extract can help to flush this out of your body, stay tough with magnesium sulfate, that will keep you alive, and liver filtering.

Q> Have you looked at my stool pictures? What do you think?

A> You seem to have a significant infection, where large fluke like (stubby) worms are migrating out of the skin. These may appear to be short round worms, but they are actually flukes.

I believe by looking at the stool, you may have a co infection of roundish worms, it is easy to tell. A single dose of **Invermectin** calms down most round worms.

Flukes are a different matter, having them come out of the hand indicates a tough course of **praziquantel**, and drinking quinine tonic tea may be required.

Flukes are flat and oval. Round worms are well kind of round, and rope worms are not actually worms at all, just kind of blobs of junk that is the body dumping worm shit through the lymph system, which is actually 6 quarts of fluid, more than the blood system has in circulation which is 2 quarts.

#### Q&A

#### O> How should I start?

A> I suggest you balance your body with magnesium, potassium, sulfur(s), etc first, establish zinc, selenium, chromium, copper sources. I suspect your amino levels are low, Spirulina helps bring this up, and you are probably highly toxic. I use a batch of detox herbs and things I will attach.

One person wanted to dose kill drugs in parallel with ph, minerals, and metals. I can tell you that at the first doses of **PinX**, I thought they would wind up in the ER.

Always have emergency drugs, White willow bark, bobs redmill baking soda, a bottle of water, powdered Spirulina, Magnesium sulfate usp, MSM, Magnesium citrate capsules, Potassium citrate capsules. Keep your tongue PH higher than 6.

The earlier you start on alkaline minerals, the better. Oxygen is essential, **Zinc sulfate and Selenium chelate** move oxygen into the body, I would ramp **Zinc sulfate** to 400 mg/D. I would ramp **Selenium** to 600 ucg/D, **Copper** to 4 mg/D, Start **Magnesium sulfate** at 2060 mg per day, then taper to 1030 as the stool turns brown, not green or black. If the stool does not turn brown again suspect SIBO or SIYO infection.

Initially 133 to 266 mg <u>Magnesium citrate</u> per day, 99 mg of <u>Potassium</u>. Try to get the stool loose, but not so loose that you loose bile salt.

The goal is keep a brown stool. Green is liver junk, you want to keep your liver as clear as possible.

**KGP flush**, cranberry juice, distilled water help move stuff out of the kidneys, if the kidneys slow or

stop use high dose CQ10, and do 3 doses (droppers full) of KGP flush per day to keep the kidneys working. New studies show P5P and piridoxamine help kidneys function in the presence of severe damage.

In Phase 2, I would start with **PinX** dose to box, if you vomit for days, you may want to start again in a week with drops of **PinX**. I vomited every day at 3 am for 3 weeks. My gut was infested.

Once you have one solid dose of PinX, repeat the "box" dose - once every 3 weeks - until you no longer get a reaction, your GI trac is clear.

Now I would initially suggest taking up to 750 mg of Piperazine citrate (50mg/ml) per day, this helps dump ammonia, uric acids, and alike.

Taking a high dose initially can cause a severe die off event. The maximum dose is specified as 3 grams for 3 days. If you dose to guickly you can get a die off event in the brain. This is very painful.

If your brain burns, back down a little, it can cause egg antigen responses that make you shake. It fades in about 3 weeks.

**Zinc** tells your body to make white blood cells. If you need some help with your immune system take some Magnolia bark.

Later in Phase 3 To kill the flukes you will need to bring out Praziguantel. I used a level teaspoon total per day, for 113kgs, for 2 weeks, and after the kill stops maint 3 weeks, for several months. It will drive things right out of your skin.

If you have round worms, I would treat these last, using Albendazole, puts them to sleep in high dose, 10 mg/kg for 14-28 days, then repeat. This will take out 90 % of some species the round worms or more if you flunk the **Invermectin** test.

Then stand back and keep minerals and metals until you see what is left.

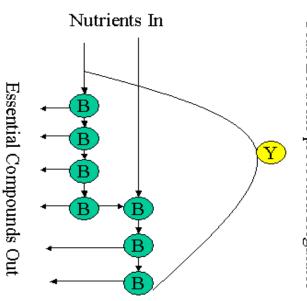
Iodine may help.



The GI tract must process food properly to make the body healthy enough to kill parasites.

#### **Good Bacteria**

Healthy bacteria are normally present in the large intestine. A good biodome of bacteria break down Nutrients, into thousands, or hundreds of thousands of essential compounds that the body cells and organs use every day to stay healthy.



Treatment For Pinworms

**Pyrantel Pamoate Suspension** FOR THE ENTIRE FAMILY

Single Dose Effectiveness

Measuring Cup Included

Doctor Recommended

Yeast Biodome protection Regulator

Normally some Immune system, and good yeasts help protect and regulate the population of good bacteria.

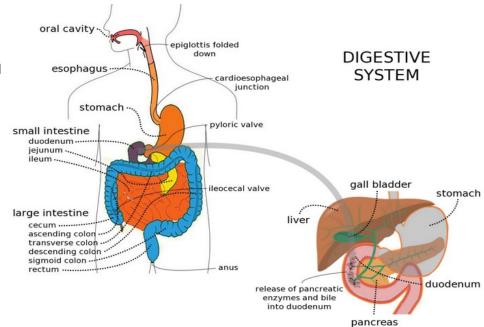
The digestive tract has an acid stomach, the first protection against bad bacteria and yeasts. The broken up food is mixed with pancrease enzymes and gallbladder bile to start the digestion process. The small intestine is mostly bacteria free, and can directly absorb simple nutritional elements. The small intestine is separated from the large concentration of bacteria that resides in the large intestine by the ileocec valve. Here bacteria create more complex essential vitamins, substances, and compounds.

#### **Normal Digestion Process**

Digestion is the process of turning large pieces of food into its molecular components.

The digestive system is a group of organs working together to convert food into energy and basic nutrients to feed the entire body.

Food enters your stomach through a muscular ring, or sphincter, that



closes to keep the food in your stomach and stomach acid out of your esophagus. As you continue eating, your food's mixed with gastric acid and other digestive juices in your stomach. Then the stomach empties this mixture into the small intestine.

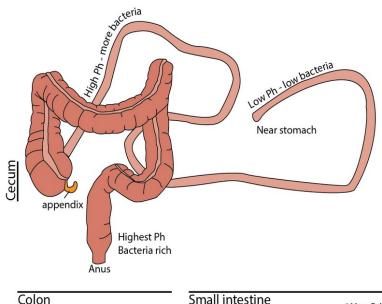
Your food is digested more thoroughly in your small intestine, also known as the duodenum. The small intestine, as well as the liver and pancreas, produces digestive juices and enzymes that separate out the nutrients in food. Among those enzymes are lipase and amylase from the pancreas.

Bile produced by the liver is also used to mechanically break fats into smaller globules. While food is being mechanically digested it is also being chemically digested as larger and more complex molecules are being broken down into smaller molecules that are easier to absorb. Chemical digestion begins in the mouth with salivary amylase in saliva splitting complex carbohydrates into simple carbohydrates. The enzymes and acid in the stomach continue chemical digestion, but the bulk of chemical digestion takes place in the small intestine thanks to the action of the pancreas.

The pancreas secretes an incredibly strong digestive cocktail known as pancreatic juice, which is capable of digesting lipids, carbohydrates, proteins and nucleic acids. By the time food has left the **duodenum**, it has been reduced to its chemical building blocks—fatty acids, amino acids, monosaccharides, and nucleotides.

The <u>small intestine</u> is a long, thin tube about 1 inch in diameter and about 10 feet long that is part of the <u>lower</u> <u>gastrointestinal tract</u>. It is

molecules and carry them to the rest of the body.



located just inferior to the stomach and takes up most of the space in the abdominal cavity. The entire small intestine is coiled like a hose and the inside surface is full of many ridges and folds. These folds are used to maximize the digestion of food and absorption of nutrients. Most absorption takes place in the walls of the small intestine, which are densely folded to maximize the surface area in contact with digested food. Small blood and lymphatic vessels in the intestinal wall pick up the

©Mary E. Morgan

By the time food leaves the small intestine, around 90% of all nutrients have been extracted from the food that entered it. *Muscular contractions keep the food moving along toward the large intestine.* 

The large intestine takes about 16 hours to finish the digestion of the food. It removes water and any remaining absorbable nutrients from the food before sending the indigestible matter to the rectum. The colon absorbs vitamins that are created by the colonic bacteria, such as <u>vitamin K</u> (especially important as the daily ingestion of vitamin K is not normally enough to maintain adequate <u>blood coagulation</u>), <u>vitamin B12</u>, <u>thiamine</u> and <u>riboflavin</u>. The large intestine also secretes K+ and Cl-. Chloride secretion, Recycles various nutrients used in colon. And processes include fermentation of carbohydrates, short chain fatty acids, and urea cycling.

The large intestine manufactures many of the vitamin like substances used by the body. The section of intestine between the small intestine, the valve, and the first part of the large intestine is responsible for a large amount of the immune system regulation processes. Having a good gut biodome therefore helps lead to a healthy immune system.

#### Flora Biodome

The large intestine houses over 700 species of <u>bacteria</u> that perform a variety of functions, as well as <u>fungi</u>, <u>protozoa</u>, and <u>archaea</u>. Species diversity varies by geography and diet.[30] The microbes in a human distal gut often number in the vicinity of 100 trillion, and can weigh around 200 grams (0.44 pounds). This mass of mostly symbiotic microbes has recently been called the latest human organ to be "discovered" or in other words, the "forgotten organ".[31]

The large intestine absorbs some of the products formed by the bacteria inhabiting this region. Undigested polysaccharides (fiber) are metabolized to short-chain fatty acids by bacteria in the large

intestine and absorbed by <u>passive diffusion</u>. The bicarbonate that the large intestine secretes helps to neutralize the increased acidity resulting from the formation of these fatty acids.[<u>citation needed</u>]

These bacteria also produce large amounts of <u>vitamins</u>, especially <u>vitamin K</u> and <u>biotin</u> (a <u>B vitamin</u>), for absorption into the blood. Although this source of vitamins, in general, provides only a small part of the daily requirement, it makes a significant contribution when dietary vitamin intake is low. An individual who depends on absorption of vitamins formed by bacteria in the large intestine may become vitamin-deficient if treated with <u>antibiotics</u> that inhibit other species of bacteria as well as the disease-causing bacteria.[<u>citation needed</u>]

Other bacterial products include gas (flatus), which is a mixture of <u>nitrogen</u> and <u>carbon dioxide</u>, with small amounts of the gases <u>hydrogen</u>, <u>methane</u>, and <u>hydrogen sulfide</u>. Bacterial <u>fermentation</u> of undigested <u>polysaccharides</u> produces these. Some of the fecal odor is due to <u>indoles</u>, metabolized from the amino acid tryptophan. The normal flora is also essential in the development of certain tissues, including the cecum and <u>lymphatics</u>.[citation needed]

They are also involved in the production of cross-reactive antibodies. These are antibodies produced by the immune system against the normal flora, that are also effective against related pathogens, thereby preventing infection or invasion.

The most prevalent bacteria are the <u>bacteroides</u>, which have been implicated in the initiation of <u>colitis</u> and <u>colon cancer</u>. <u>Bifidobacteria</u> are also abundant, and are often described as 'friendly bacteria'. [citation needed]

A <u>mucus</u> layer protects the large intestine from attacks from colonic <u>commensal bacteria</u>.[32]

#### **Gut Regulation of the bodies Immune system**

It is now evident that the gut microbiota has a profound effect on the host immune system and can affect autoimmune-related diseases both within and outside the gut.

Keeping a delicate balance in the immune system by eliminating invading pathogens, while still maintaining self-tolerance to avoid autoimmunity, is critical for the body's health.

The gut microbiota that resides in the gastrointestinal tract provides essential health benefits to its host, particularly by regulating immune homeostasis.

Moreover, it has recently become obvious that alterations of these gut microbial communities can cause immune dysregulation, leading to autoimmune disorders.

The mammalian gastrointestinal (GI) tract is home to an enormous and complex community of commensal bacteria.  $\frac{1-3}{3}$  This gut microbial community (microbiota) has co-evolved with its host over millennia and provides benefits to its host in many ways, including, but not limited to, digestion, production of nutrients, detoxification, protection against pathogens and regulation of immune system.  $\frac{1-5}{3}$ 

The immune system plays a vital role in keeping the body healthy by providing a fine balance between the elimination of invading pathogens and the maintenance of tolerance to healthy self-tissue.

However, in the case of patients with autoimmune disorders, the mechanism to maintain self-tolerance fails and the result is that the immune system mistakenly attacks and destroys healthy self-tissue.<sup>6,7</sup>

Given the intimate interplay between gut microbiota and the host immune system, it is not surprising that some members of the gut microbiota have been linked to autoimmune diseases. However, only

recently has the study of the gut microbiota and autoimmunity become a more navigable field, owing to the ground-breaking advances in "next-generation" sequencing technology, which have now provided culture-independent microbial analysis that greatly facilitates the characterization of these complex commensal communities. 8-11

#### T cells

CD4<sup>+</sup> T cells are a key component of the adaptive immune system. Intestinal CD4<sup>+</sup> T cells are mostly located in the LP of the intestine. Upon stimulation, naive CD4<sup>+</sup> T cells can differentiate into four major subtypes: T helper 1 (Th1), Th2, Th17, or regulatory T cell (Treg). These various CD4<sup>+</sup> T cell subtypes are distinguished by their expression of various transcription factors and cytokines (Fig. 1). The proper regulation and balance of T-cell subtypes is a crucial factor in determining one's health status.

#### Th1 cells are critical for the host defense

against intracellular microbial infection, while Th2 cells play an important role in eliminating parasite infections. Uncontrolled Th responses can be pathological, as the Th1 and Th17

Th17 IL-17(A), IL-17F, Control of Th1 Th2 Gata-3 Naïve IFN-y CD4+T IL-4, IL-5, IL-13 Protection Protection cell against intracellular microbes against parasites Clostridium Foxp3 Treg IL-10, IL-35, Regulation of

responses have been linked to autoimmune diseases while the Th2 response has been associated with allergic reactions. Treg is a key mediator of immune tolerance; its dysfunction can lead to autoimmune disorders.

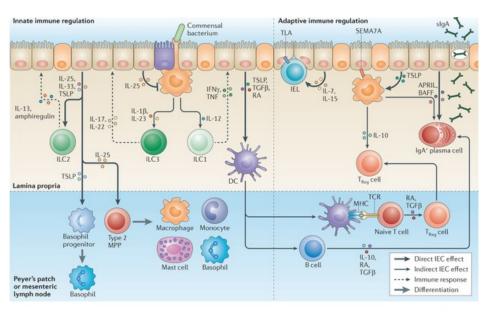
Not surprisingly, there are also "beneficial" commensal bacteria that can ameliorate disease. For example, *B. fragilis* can reduce the colitis induced by *Helicobacter hepaticus* in immunocompromised mice through its production of PSA, which suppresses disease by both stimulating the anti-inflammatory IL-10 production from CD4<sup>+</sup> T cells and downregulating the pro-inflammatory IL-17 production in the colonic tissue.<sup>73</sup>

Bacteroides thetaiotaomicron was also demonstrated to attenuate Salmonella enterica-induced inflammation by enhancing the nuclear export of peroxisome proliferator activated receptor- $\gamma$  (PPAR- $\gamma$ ), a transcription factor that plays key roles in the regulation of lipid metabolism and inflammation. 74,75

Short-chain fatty acids (SCFAs) produced by the gut microbiota have also been shown to reduce inflammation in the dextran sulfate sodium (DSS)-induced colitis model. This anti-inflammatory effect required the interaction of SCFAs with G-protein-coupled receptor 43 expressed on immune cells.  $\frac{76}{}$ 

The introduction of Clostridium upregulated the colon Treg population and coincided with the reduction of DSS-induced colitis, suggesting that Tregs might be responsible for the anti-inflammatory effects mediated by Clostridium. 43

#### Immune system regulation in the gut



Nature Reviews | Immunology

#### **Bad Bacteria in Malabsorbtion**

Malabsorbtion occurs when pathogens invade the gi tract, bacteria overgrowth or yeast overgrowth occur, or when so many digestive processes are disrupted, the gut can no longer spend enough energy to digest food. Parasites force a larger and larger drain on the immune system.

Parasites disrupt human enzyme systems. Parasites spread gut bacteria into the body where increased immune response is required. As the immune system falters, overgrowth, bad bacteria, and yeast overgrowth drain even more health processes. The house of good health starts to collapse.

# 

#### **Starvation**

The body can actually go into **starvation** to support essential

Bad GUT Bacteria Chemical Factory

body processes. Organs and muscles waste, the body cannibalizes anything and everything to keep the boat afloat. These situations have been programmed into the DNA by millions of years of evolution.

If Diarrhea causes a loss of <u>bile salt</u>, fat soluble vitamins cease to be absorbed. You do not feel how sick you are. You are in a critical situation. Reserves of <u>Sulfur</u> and <u>Selenium</u> start to deplete.

The body stops removing wastes, and focuses all its attention towards survival, immune response, healing wounds, the prevention of blood loss, keeping essential organs functioning. <a href="Magnesium">Magnesium</a>, potassium, and calcium levels fall. The body is now stressing the Design and integrity of the DNA program to its maximum extent.

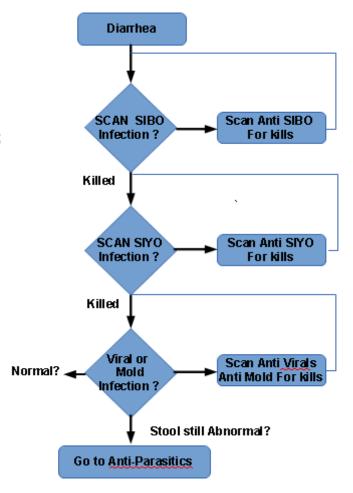
What is required is bypassing the conditions created by the parasite infection to enable a kill of the parasite. Enough reserve energy must be created to survive the parasite kill.

Balance of the body makes the body healthy enough to withstand the parasite killing process.

#### Avoiding Death, Cancer, and bring the body into Equilibrium

To be healthy enough to kill parasites you need to:

- Kill SIBO and SIVO overgrowth.
- <u>Replace</u> stores of essential minerals, fat, lipids, and metals
- Provide <u>direct replacement</u> <u>of vitamins</u> not being manufactured in the gut
- · Reduce inflammation.
- Aid in toxin removal from the organs, kidneys, liver, blood, lymph
- force the immune system to stop fighting shadows and inflammation
- Balance White blood cell production TL1/TL2.
- Pull on <u>PPAR gamma</u> receptor, preventing immune skew
- Pull on RXR 3T3, preventing cytome twist, Pull on FXR.
- Supplement the DNA directly with Amino food
- Remove DNA toxins
- Balance PH
- Restore flora with probiotics
- Set D3, 1-25



Clearing the GI tract of <u>SIBO</u> and <u>SIYO</u> overgrowth is an essential step towards PH and enzyme recovery. <u>SIBO and SIYO</u> are typically found in the <u>Small intestine</u>, hence the SI letters in the abbreviation. In general the digestion process has an acid stomach to kill bad things, the small intestine is where pancreas enzymes are added to the mix and bacteria count is low, the mixed mash is dumped into the large intestine trough a valve, which keeps higher bacteria digestion into the large intestine, where most vitamin substances are removed.

Bacteria and healthy yeasts are like little chemical factories, normally taking in a few substances, and building hundreds of healthy nutrients, that the body needs.

The disruption of the digestive chemical factory process is felt as poor health.

<u>During Phase 1</u> one needs to improve digestive health. I started by incorporating yogurt, probiotics, healthy butter, oils, fats, foods, and vitamins. I usually suggest that everyone tries a two week dose of <u>Deoxycycline</u> antibiotic to assist in this process. For me Deoxycycline did not appear to help.

**During Phase 1** the use of natural killing substances are used to scan for invaders. Each invader that overtakes you GI tract, bypasses digestion stages, and results in poor nutrition, irregardless of how healthy you eat. Each invader my jump over several bacterial processes, making healthy bacteria weaker, providing you fewer digested nutrients. The sign of a poorly working digestive system are numerous, including gas, acid stool, poorly formed stool, diarrhea, undigested food, cramps, pains, food sensitivities, allergies, and more.

There are large sections for <u>SIBO</u> and <u>SIYO</u>, which can be found in later sections of this document.

#### There are also suggestions of things that breakup tough Biofilms (SIBO And SIYO)

**Phase 1** requires one clear the GI tract of intestinal infections. A couple of weeks of **Deoxycycline**, a few key anti-bacterial natural herbs should be tried, and a few key anti-fungal natural substances should be tried. I tried everyone I could find and detailed the test results. I found several that were able to cut through the layers of infection, and once you do a few of the most powerful herbs, you may want to move quickly on to the next phase.

If you have **SIBO**, or froth at the mouth, **humic fulvic (www.humineral.com) may help break up the biofilm.** 

Taking a probiotic may also help turn your stool from green to brown. I would relapse to green, foul stools, even after killing many bacteria, fungal, and viral components.

Constantly doing a few <u>SIBO</u> herbs, while you determine if you have a **SIYO** film in the GI tract, may be a good idea. I found magnesium and copper constantly helped me during my search. I would dump mucus, blobs of foul smelling stool products, and had a bad biofilm problem.

Biofilms actually exist due to the ability of an infection to create a voltage potential, isolating it from the human immune system. Shorting out this potential using **charcoal**, metals, and **humic fulvic** is partially effective in eliminating **biofilms**. Probiotics can help as well. Copper can be used.

The gluten-free diet must exclude carbohydrates (sugars and starches) as much as possible while taking the anti-fungal medication. Sugars include jelly, maple syrup, table sugar, honey, molasses, fructose, soda, fruit and fruit juices. Starches include gluten-free flours, bread, bagel, pizza, pasta, bakery products (cookies, cakes, pies, muffins, brownies), cereals, granolas, energy/ breakfast bars, and chips of any kind.

This removes the type of food needed by the yeast to thrive. Other exclusions are mushrooms and other fungi, yeast-raised bread, vinegar, cheese and milk products.

Afterwards you can test milkshakes, pancakes to ensure your GI is free of yeast.

#### **Phase 2 - GI tract Parasite Clearing:**

Clearing the GI Tract in **Phase 2** is performed after Supplements, Balance, And Elisa testing have been accomplished.

I researched every anti-parasitic, and found **PinX** is the broadest spectrum antiparasitic on the market, that is unmatched in survival and safety. It is an over the counter medication.

Balance of the body makes the body healthy enough to withstand the parasite killing process. The detoxification pathways are stimulated, the sulfurization cycles are accelerated, and the GI tract has been inundated with <u>MSM</u> and <u>baking soda</u> to re-establish alkaline processes. <u>ALA</u> and <u>CQ10</u> helps detoxify the DNA. <u>Spirulina</u> helps detoxify the GI trac. <u>Magnesium</u> helps stimulate the adrenal glands, many body systems start to ramp up to detoxify. After a few weeks, the body may be ready for Phase 2, clearing the GI trac.

**Phase 2** requires the small intestine to be cleared of parasites, in that the majority of critical absorption processes are conducted there. The function of the large intestine processes many vitamin processes, and therefore many vitamins are taken, assuming that the large intestine, and body processes are compromised by the parasite infection. There yet may be several large worms that hide in the GI trac. These will be killed with time, or by the final mop up phases. Eventually as these worms sense a lower toxin and worm enzyme level, they migrate to reproduce. As they migrate, later phases deal with these worms, when they leave the ball of worm nests.

**Phase 2** clears the <u>majority</u> of the intestinal trac, and is performed using **PinX**. When parasites are removed, the GI tract can absorb Vitamins, and Anti-parasitics. Repeat **PinX** dosing may be required after the body has been given several weeks to recover.

The swelling of the intestine needs to be reduced, and the entire bowel will need to be working prior to the next phase. It is necessary to determine if a round worm infection is present. A single test dose of **Invermectin** is used determine if this is so. If roundworms are present, they are a blood worm, with the ability to absorb certain substances like zinc and **vitamin A**.

Introduction of low dose **DEC** is advisable for worms in the eyes, or other sensitive organs, to drive larvae from the blood supply. **DEC** and **Piperazine** stops Ascaris Malting of the worms.

Nitazoxanida helps clear L3 cysts and Ascaris worms.

Knowledge of which types of parasites is helpful in identifying special anti-parasitics, and in the final taper phase, to verify a complete kill has been accomplished.

#### Q&A

- ..."What is your PH
- ...What did your hair analysis say?
- ...Are you on "minerals, metals, vitamins, herbs"?

 $\mathbf{Q} \geq \mathbf{I}$  didn't do any of that. I am dependent on my parents for money so that's why I am going to take the  $\mathbf{PinX}$  test to show them that I have parasites. Once they see the worms then I can have the money to start with your parasite protocol.

**Q>** I do not have access to ELISA testing.

**A> Magnesium Citrate** and **Magnesium Sulfate** (Epsom Salt) is essential.

This was true a hundred years ago, as it is today.

<u>Magnesium Citrate</u> bottle from pharmacy may still be found around. It is \$3.00 Use of a Tablespoon or two is dramatic. Start with a teaspoon in a glass of water per day. Do a little before <u>PinX</u> and during, to improve your magnesium status. It also helps in the flushing time.

Magnesium Sulfate is essential for sulfur. a bag of USP is about \$3.00. Take 1/4 teaspoon (1000

mg of the salt) dissolved in a glass of distilled water with a pinch of baking soda. It also can be taken for several days ahead and after the <a href="Pinx">Pinx</a> dose, it also will help flush the liver. Do not be surprised when your stool turns green, or black, flushing out toxins.

Dose **PinX** per your weight, on the box

Have a macro zoom camera and some white paper ready for pictures. Post them on the CureZone for identification.

Good Luck.

You know what they say

#### Phase 3 - Systemic Killing:

Systemic parasite kills must be done once parasites migrate through the intestine, and enter the blood and lymph systems, organs, and bones. It is imperative to do kills of flukes before roundworms, in that the enzymes and damage they cause is higher than that of most round worms, and their enzyme effects are higher. By dosing all roundworm antiparasitics at once, their effect is superficial. Zinc can be ramped, and has a deeper roundworm killing, especially for smaller worms. Large worms actually will become slightly more aggressive when you are on zinc.

**Systemic** parasite kills take longer, and increase in duration depending on the immune system status, age of the individual, and the bodies ability to recover.

Smaller molecule anti-parasitics penetrate deeper into tissues than larger anti-parasitics. Few anti-parasitics can be passed through the blood brain barrier. This is not so essential, since parasites that enter into the blood supply, feed from the blood supply. They regularly feed on blood, and absorb **iron** from the blood, and if not fed every 3 days, get very hungry.

Kills require someone to ramp anti-parasitic meds, such as **Albendazole** for roundworms, and **praziquantel** for flat worms, and maintain the dosing for weeks.

In truth, <u>Albendazole</u> is not strictly a roundworm medicine, and <u>praziquantel</u> is not strictly a flatworm medicine. Each anti-parasitic medicine is capable of some effectiveness on many parasite types. They have been stereotyped by doctors because certain medicines work better on certain types of worms.

Singling out one medicine for a parasite is a very narrow view of the process. Phase 3 is the most critical phase of the parasite killing. The combination of Albendazole and Praziquantel are used to create a broad spectrum treatment.

Eventually a mass die-off event is generated.

- See Zinc Sulfate Kill
- See Alinia Kill
- See Praziguantel
- See DEC
- See Mebendazole
- See Flagyl

Fluke kill using Albendazole and Praziquantel

#### Q&A

- Q> How does the formula work and why D3?
- A> The obscure study indicated Albendazole low dose helped Praziquantel cross the blood brain barrier. Further studies indicate Praziquantel can be totally effective in system wide kills.
- A> I kind stumbled across the D3 protocol. I have seen some animal studies. It has worked for two others besides myself, with a half a dozen in the process now.
- Q> But, I am interested in what you find in the obscure study to cause you to take the vitamin D3 and the dairy products. Can you share what the study indicated? My vitamin D is always super low, rickets range actually. But I do ingest enough fats. I wonder if it was the calcium you were after?
- A> The theory of how Praziquantel works, is it makes cracks in the worm exterior, and these small cracks let calcium into the worm. Vitamin D3 simply accelerates the process.
- Q> Also, I know I have a fluke in my brain as well. So I'm interested in learning from you how it felt when it died.
- A> I had hundreds or thousands, they were in the process of consuming my brain, I had severe memory issues in 2012. I progressed to falling to the floor, massive tremor, restless leg syndrome, and no sleep, I felt electrical zaps in the brain as they consumed it.
- Q≥ I am also interested in how you "felt" it in your head. I know that the brain has no nerves and I don't feel any pain in my head. But I feel pressure and movement after taking flukicide substances that reach my brain.
- A> Movement is a key issue, The flukes die in about 4 days to 7 days, the toxin dump causes black stool for 10-14 days. The experience I would not wish for my worst enemy, but is a ray of sunlight for those who have systemic fluke infections. It saved my life, and that of others. So far about 50% of the people that try the formula express fluke die off experience in various degrees. My reaction was severe, and near death.
- **Q>** Sometimes I feel like the fluke is contracting and it feels like someone is patting my brain. Like someone pats your hand. It is like a sudden awareness of pressure that immediately stops, then repeats. But it also feels like a force with movement. Sometimes I feel something behind my eyes. I'm well beyond fear of dying now. It is more a search for a path to living well.
- A>Sounds like a larger fluke species. I had a few come out of the GI tract.
- Q> So how did the flukes in your brain feel? How were you aware of their presence? How did you fair / feel as they died?
- A> Don't ask, experience it.

#### **Q&A 1**

**Q>** The reason I was asking about mebendazole vs prazi was this: If you have orange beefy chunks of large oval Cestode Flatworms) they respond better to

Fenbendazole and Mebendazole.

A> MEB and FENBEN are more caustic to the kidneys, liver, and do not kill flukes in the brain, tend to kill faster, causing more damage to the organs and brain. I read many brain studies that indicated successful treatments with PZQ, I saw none for FenBen. My kill took 4-7 days with 14 day recovery time, my brain felt like Swiss cheese. I have one gal with brain bleeding at the moment. She found that 50,000 IU D3 caused a bleed, and now is down to 20,000 IU.

Look, there is risk either way, I chose one that had a little wiggle room. Once they start to die, to a certain extent, you are on autopiliot, and very little can be done to stop the death. The total formula of blood thinning, supplements, detox removal, black stool dumping is a very scary event, but I and several others have made it through alive. There is risk.

Since the large oval part applies to me I was wondering if mebendazole or prazi would be better?

The formula is ALB, DEC, PPZ, and PZQ. I have had 2 reports that DEC flushed large flukes by itself. The goal is to create a broad spectrum of killing, that does so in a gentle fashion. Could it be better? You tell me.

#### **Q&A 2**

Q> Ok so think there was some bleeding in the centre of my brain last night. Has this ever happened to you?

**A>** Yes, Especially when the blood pressure is high.

**Q>** Is there anything I can do that will help?

**A>** Occasionally a worm leaves a hole, they should heal in a day. Lay down. I had many of these.

- I would cut high doses of A, Zinc
- Keep everything else at typical or lower dose. Lower White Willow Bark and Ginkgo to the minimum. Increase Horse Chestnut.
- Vitamin D3 over 20,000 IU is a risk. I say in the document 50,000 IU kills so fast it is a near death experience. I was not kidding. After 4 days of killing, I just wanted to be over the brain killing phase.
- After the kill, It took over 3 weeks for the brain to normalize (Heal). There were many neural issues that faded. I know it is scary

Q> I think people need to be **gravely warned** about vitamin **D3**. I was feeling very safe, didn't even know I was in danger last night. Yesterday was my 50,000 IU day but I felt totally fine. I had been doing 20-30 000 IU on off days and kept feeling OK.

**A**≥ Vitamin **D3** has about 4 warnings in the document. I tested it at 5,000, 10,000, 20,000, and 50,000. I described the rate of killing using 50,000 units as a near death experience. I did research afterwords that indicates 50,000 units is safe, therefore the risk comes from killing to quickly. You are free to adjust the D3 to the level that kills at a rate you can handle.

Q> I didn't notice til early this morning, do you think I'll be OK?

**A>** If you get symptoms of stroke, frozen arm, leg, focal deficits, you may want to to to the ER.

Object to the state of the s

<u>A></u> My brain is much improved. I cannot say perfect because I have years of parasite damage. I can say I am in a much much better place. If they do not dissolve, I suspect the body will put calcium around them.

It's hard for me to feel that (I will be ok) because I feel like there's blood everywhere.

#### Phase 4 - Mop up:

Phase 4 involves mopping up of the parasite infection. Continued Minerals, Metals, And vitamins are tapered back,

 Metals like <u>Zinc</u> and Vitamins like <u>Vitamin A</u> are removed from the formula, after flukes are killed, to avoid keeping Ascaris worms alive. The anti-parasitic schedule, and dosing are slightly changed to kill deeper into the body. The mop-up killing is continued until and beyond all signs of the parasite infection are removed.

Taking a day or two off of supplements, will show the body is still dependent on supplements for some 2-3 weeks later. After a while, lowering the level of magnesium, minerals, metals, and certain vitamins may make you feel better. It is at this point transitioning to the maintenance supplements may make sense.

Later it is expected that supplements are moved to a short list, detailed later in the document.

It is expected that several substances, like **Piperazine**, and certain antiparasitics are maintained during this period.

## Phase 5 Taper

The final process step is to taper the dosing of anti-parasitics, to determine if a successful clearing of the system has been accomplished.

• If yes, the process is over, if after 8 months, a repeat ELISA test shows a continued infection, one must revisit the systemic clearing phase, and attempt again to remove parasites from the system.

The taper period is typically one month.

If your first kill happened with **PZQ**, move on to **ALINIA** kill. (See the log)

#### Start-up

The start up phase is detailed earlier in the early interventions and start-up section.

## Phase 2 Antiparasitic meds dosing

## The Single Dose IVM Test

1) Dose **IVM**, if symptoms subside, you have a runaway round worm infection. 1) **Invermectin** single STD dose, 200ucg/kg. If your symptoms abate, think round worm, **IVM** is about 85% coverage for most roundworm types.

If you are positive, you will have to lower the <u>vitamin A</u> and <u>Zinc</u> supplements, immediately after the first kill in Phase 3. Round worm infections also require periodic pulse doses of IVM to kill babies. Set the test result aside, and move on.

DURVET APPLE - Apple flavor (ain't that sweet!) is the same strength and you can get it online for less than \$3. Available at most US feed stores, or internet animal stores. The Durvet plunger has markings for every 250 pounds of body weight and has 5 notches between each marking. Each notch equals a dosage for 50 pounds of body weight. Mix the Paste in a tablespoon of **Greek Yogurt** to kill the taste. **IVM** should start working in about 30 minutes.



#### **0&A**

A> (Flash Forward) One of the biggest regrets I have had, is that I was never able to integrate ALA into the formula, or take ALA long term in the formula, it seamed to make large Ascaris more active, which was ok in the early days during killing, but I miss it now.

I had brain fog yesterday, which I wanted to clear it away. Since I am on nothing but Vitamin C juice, I thought a minute, and grabbed the only two things I could think of, IVM and ALA.

Well the neural fog not only disappeared, but in 4 hours I felt pretty good. The next morning I felt fantastic.

I tried many times to put **ALA** into the formula, it speeds brain and nerve healing. I am going to double down on finding ways to use it.

I do not know why I had fog. I may still have a worm that can either dump toxins or eggs, but I have been worm free, and baby free for almost a week, after my 9th dose of 500mg Alina. I plan to redose Alinia after a 21 day gap, to mop up any Ascaris that pop up.

Currently I take nothing but OJ, Cranberry, and Tomato Juice, but now I think finding ways to speed brain healing is a priority. I am afraid to take any supplements at all, for fear of assisting any missed eggs, or cysts.

I may also be able to have my favorite drink, Coffee. Oh how I miss Coffee.

Mattk3

#### Clear the GI Trac

2) Clear the GI tract using **Reeses PinX**, one standard dose every 3 weeks until GI tract clear. GI tract must be clear and functional to absorb anything oral. Back in the 1800's doctors would administer some kind of **magnesium**, say 2-3 grams or more, with the purgative, to balance the

release of toxins, acids, and speed the process of dumping parasites from the GI trac. I still think this is a good idea.

#### Q&A

**Q>** The concentration of the product I have is 250 mg/5 ml. I just don't know how much am I supposed to take according to my weight, like IVM should be taken 0.2mg/kg.

#### A> Your pamoate, is 50mg/ml, that is about 1/3 the strength of Reeses.

Also, how are you doing? Free of the parasites? Or still fighting?

A> What is your PH?

What did your hair analysis say?

Are you on minerals, metals, vitamins, herbs?

Reeses PinX: in each 1mL: Pyrantel Pamoate - 144 mg/mL Anthelmintic

Less than 25 lbs. or under 2 years old: Do not use unless directed by a doctor

25-37 lbs.: 1/2 teaspoonful (as a single dose)
38-62 lbs: 1 teaspoonful (as a single dose)
63-87 lbs: 1 1/2 teaspoonfuls (as a single dose)
88-112 lbs: 2 teaspoonfuls (as a single dose)
113-137 lbs: 2 1/2 teaspoonfuls (as a single dose)
138-162 lbs: 3 teaspoonfuls (as a single dose)
163-187 lbs: 3 1/2 teaspoonfuls (as a single dose)
188 lbs and over: 4 teaspoonfuls (as a single dose)

#### Again: Steps 1 and 2 with even more detail...

If your PH is less than 6.5, if your hair test shows mineral and <a href="metal">metal</a> profile errors, ...start supplementing minerals, metals, <a href="metals">Magnesium</a>, <a href="metals">Potassium</a>, non-ionic <a href="metals">calcium</a>, <a href="metals">Magnesium</a>, <a href="metals">Sulfate</a>, <a href="metals">Zinc Sulfate</a>, <a href="metals">Copper</a>, <a href="metals">Selenium</a>, non ionic <a href="metals">iron —</a> spinach for iron, kelp for iron, etc, etc, etc.

#### **PinX**

>Dose <u>PinX</u> per your weight, the dose is on the box. Dose <u>Invermectin</u> at 200 ucg/kg (0.2mg/kg) If you dose in 1 day, the GI tract is cleared. If you still have an infection, the worms are not in the GI trac. A good mop up for eggs is <u>Piperazine citrate</u> or <u>Pripsen</u>. I vomited because it took 8 years to figure out what I had, Doctors are useless. I had to request the test, going against the doctor's advice (doctor #19).

By the time I figured it out, they have gone everywhere. I killed them in my spine, in my ear, in my sinus, and in my brain, heart, lung, you name it, worms are there. **Magnesium** dosing during **PinX** is essential, it helps clear the GI trac, 2 Grams + is about right.



2) <u>PinX</u> standard dose: While taking a standard dose of <u>PinX</u> when you have a systemic parasite infection is aggressive, it provides you a gauge of how bad you really are, <u>Pyrantel Palmoate</u> is very broad spectrum, and it is virtually unmatched in its ability to clear the GI trac. I now suggest that taking <u>IVM</u> along with <u>PinX</u> will paralyze worms.

If you are systemically infected, the GI tract must be clear to recover your immune system, which is regulated by the intestine, and you need the intestine to absorb the minerals and metals. Clear the intestine first before doing any anti-parasitic treatment.

Do not take **PinX** with anti-parasitics like Albendazole or Praziquantel, the combination could create a toxic sickness. Light dosing of **IVM** or **Piperazine** is ok.

**Get a macro zoom camera**, wash the round, flat, tape worms and take pictures for identification by the cure zone forum. I had round (Ascaris ELISA) positive, Tapeworms, and Flukes of 3 kinds.

<u>Magnesium Sulfate</u> is a decades old cofactor in clearing the GI trac. <u>Magnesium Citrate</u> liquid has been used as well. <u>Magnesium Citrate</u> can cause a overload of toxins, so lower dosing than the bottle is advised.

Getting parasites out, without giving them a chance to scatter, is essential unless you want to deal with problems for years or decades. At the conclusion of Step 2, your GI tract is now clear. The recommended time between **PinX** re-dosing is published to be 6 weeks, I use 3 weeks.

#### **PinX Dosing**

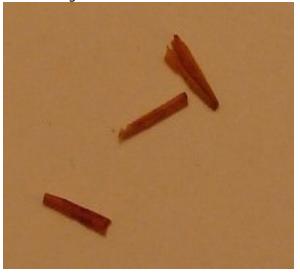
What I found was that I vomited worms for 3 weeks after trying **PinX**. I was so infested I took drops per day for weeks, until I was able to take a standard dose. I was also in denial. I still thought, it is ok; maybe I just had a severe GI tract infection. Wrong, they were everywhere. I was in denial. I dosed **PinX** every 3 weeks for several months, and would from time to time get hits, indicating worms were trying to re-infect my GI trac. I was again in denial.

## **Stool Visual Inspection**

Worms beget worms, and more parasite types. One does not get sick from one kind as bad as you are. Keep a camera ready, wash and put them using a wood depressor or something on white paper, use macro zoom camera. Parasites are made of cellulose, and when dried turn into translucent cellophane.

#### **Look For**

- Look for white seeds 1mm x 2mm
- Look for red rolled up tomato skins (2mm)
- Look for brown rolled up cinnamon sticks 1/16 inch in diameter (1mm)
- Look for cone heads, 1mm-2mm cone
- Look for blobs of orange or pink fleshy rolled up with black edge, (large fluke).
- Look for oval 8mm flat flukes or broken in half with sucker at one end.
- PinX may break free some of these, but will not kill them.
   Do not dose more than one dose of PinX at a time.
   Use my starting formula, MSM, magnesium sulfate, zinc, selenium, Spirulina, etc.



- If you have Tapeworms (Flat tubes with segments) (<u>Praziquantel</u>) or round worms (Flat tubes without segments) (<u>Albendazole</u>).
- If you have flukes (Flat oval with triangle sucker, or tomato skins rolled up, cinnamon sticks rolled up 1/16 inch diameter, pink flesh rolls (¼ inch) with black edges Praziquantel or/and **Triclabendazole**,
- If you have orange beefy chunks of large oval Cestode Flatworms) they respond better to **Fenbendazole** and **Mebendazole**.
- You need to figure this out. Round worms you gain weight, tape worms you loose weight,
  Flukes cause extreme liver stress, and can migrate as well. You could have a mix, I had all
  three (Tape, Flukes, Ascaris), and I did a progression of ELISA tests and dosed
  Antiparasitics, when doctors refused to treat me. Stool tests looking for worms are useless,
  ELISA tests work almost all the time, and do not rely on stool tests, that are seldom 8%
  accurate.

#### Q&A

Q>Hi guys, I took a full dose of PinX 2 days back but I didn't find any parasites in my stools.

I don't have any major parasite symptoms except that I feel some wriggling sensations in the same 3-4 points of my body but I don't think they are parasites because it usually occurs only when I sit in a particular position and they are gone as soon as I move my body. I think they are muscle twitches or something else. I don't feel any biting sensations in my body either.

My main concern is that my Eosinophils have been rising since September 2014. They are:

1% (47/cmm) in September 2014

3% (198/cmm) in April 2015

4% (256/cmm) in December 2015.

I do suffer from candida and bad digestion if that's related to Eosinophil count in any way.

I will be doing another **PinX** flush one day before the next full moon since I have read that parasite migrate back to the GI track around the full moon.

No lab in my country offers ELISA tests so I am stuck to diagnose it on my own. I can't conclude if I have parasites or not.

**A>PinX** flushes Flat worms (flukes), Tapeworms, Pinworms, and about 6 kinds of worms. It is broad spectrum.

I had Ascaris (Elisa positive).

I saw plenty of flat cones, seeds, segments, heads, black oval. never saw Ascaris. I vomited round worms for weeks though.

My Eosinophils were up in 2009 and the dr diagnosed pneumonia. - wrong!

Round worms of the larger types, hide in nests, bones, Lymph, years before they go hyper.

The only way to eliminate Roundworms, is to do the ELISA test.

I have found out that labs can send out kits. Learned this from fellow CZ folks working in teams in the same Provence.

They go to lab, have blood drawn, pay to have box shipped.

Try:

http://www.parasitic.com/test.htm

Also, **PinX** does not work on **Protozoa/Amoeba**.

Alinia and Flagyl do these.

### **Nitrogen Quick Fix**

I would take low dose <u>Piperazine</u> citrate every day. I tried <u>ornithine</u>, <u>arginine</u>, and every conceivable mixture of amino acid, and nitrogen loop supplement, none worked. When I tried <u>piperazine</u> it became apparent that the only way to process enough <u>ammonia</u>, and uric acid waste was to take <u>piperazine</u>, it quickly became a staple. <u>Low dose PPZ</u> done twice a day is essential to removing any parasite that dumps nitrogen waste, totally overwhelming your body's ability to remove the waste products on a daily basis.

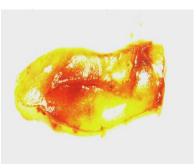
**Piperazine** is also dangerous at their recommended 3 grams per day, one cannot go more than 3 days, I massively burned my brain as cysts, eggs, and larvae burned up. I needed to keep the dose more like 750 mg (1 tbs/day) and take a few months to clear out the stuff, I had 8 years of cysts accumulated all over, and in the last 2 years the double vision was terrible.

<u>Piperazine</u> is an incredible medicine, it is the one medicine that I will probably credit for my cure, but it is a very risky, hard to dose, hard to stay safe with medicine. Any Nitrogen based Organism will die in its presence. I think it is one of the most important medicines of our time, I just wish I had done more dosing research on it before I dove into the deep end of the pool.

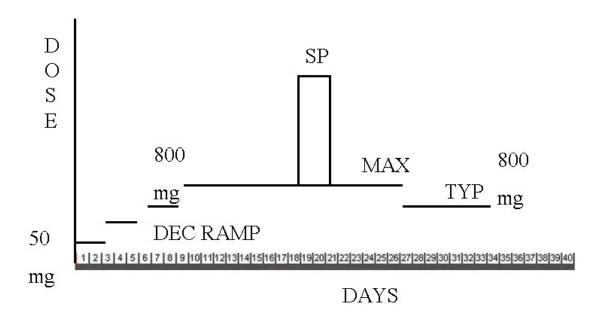
## How to use Invermectin and Piperazine to kill baby worms

- I would dose <u>Invermectin</u> when you get GERD, sweats, acid reflux, or you feel worms burst and release babies. One standard dose of IVM is usually all that is needed to kill babies for almost 24 hours. <u>IVM</u> tells the worm to stop eating, shitting, or moving.
- <u>Piperazine</u> Citrate also kills baby larvae L1, by removing worm enzymes that protect the worm from the immune system.
- Start <u>piperazine</u> slowly, and drink plenty of water, <u>piperazine</u> depletes your body of water when it removes <u>ammonia</u> waste.
- Do not exceed 2 TBS a Day of <u>piperazine citrate</u> (50 mg/ml). If your brain burns from a baby killing, slow down to 2 teaspoons per day.
- Note that <u>DEC</u> also kills baby worms, this is discussed in detail later.

## Worm larvae in Arteries, heart, and eyes



After Low dose **Piperazine**, Start Low dose **DEC first**, say 200 mg twice a day to start to clear the blood system. **If you have round worms, worms in the heart**, brain, or other vital areas (eyes) this step must be performed very very slowly. I **started with 50 mG of DEC**, and ramped over two weeks to about 400mg of DEC. When larvae are in critical areas, they will start to die, and the toxins can harm tissue. Go Slowly.



**DEC** helps clear the Blood Stream, where the circulation system could be blocked (eyes), clearing the circulation system is mandatory. **If you have an eye infection**, external, **DEC** is the best route. Enzymes emitted from worms behind the eye, when dosing **Piperazine** can be very irritating. If the worm is in the eye, it can cause blindness, temporary or permanent. Use **DEC** first if you have worms in the eye, and start very very slowly. Later start Dosing **GuaiAid** with **Piperazine** may also reduce eye inflammation. **White Willow bark** and **Ginkgo** make the tiny blood vesssels expand, making a blockage and tissue death a lower risk.

There are few studies of <u>DEC</u> in humans, many studies in Dogs. Most of the research of the first Bendazole on the market were done in France who developed the drug shortly after world war II.

The American drug companies initially tested, then ignored the drug, and started to develop its own version, which could be used to generate profits.

Studies show the survival rate for heart worm was 90 percent in dogs. They started slow, with low dose **DEC**. I could feel the worms being born, and clogging the heart. I found



that the combination of white willow bark and **ginkgo** greatly lowered the time of the pain, and the duration of the heart pain.

When I started taking **DEC**, you must remember my arms and legs were asleep, my lungs were full of fluid, I was dying. **DEC** at low dose, and gradually raised, will clear the circulation system, there really is no other drug like it. It was the first drug I actually found that worked for its intended purpose. I had huge migrans problems in my skin after **PinX**. I could actually see worms move through the skin.

**DEC** stopped migrans, I mean now.

I started like a chicken, DEC at 50 mg, then 100mg. Later the next day I went up to 200mg, and held that a few days till going to 400mg. The next week I did 800mg, and then 1200 and more. I slept on my left side. Sleep is a misnomer back then, there was no sleep. Anyway It proved safe for me, in that I am still here. It did not touch the adult worms, but really knocked the shit out of the babies. My next target became the adults, which proved to be a tall order.



The heart would stab with pain when there were bursts or baby worms were birthed. **White** willow bark and ginkgo helped reduce the pain, and keep the capillaries open.

#### **Avoid Blood Clots**

I suggest starting White Willow bark (natural aspirin) and Ginkgo (capillary expansion with growth) as soon as possible. A blood blockage causes tissue loss, and this can be minimized by using DEC. DEC is used as a heart worm medicine, and for those who have worms in the heart, this risky phase of clearing the circulation system can be fraught with stabbing heart pain, neurological, or increased acidity. DEC will kill small worms, and Larvae. In conjunction with pulsed IVM, small migrans worms can be cleared, with relative safety. Studies I have seen for dogs lead me to believe that 90 % of dogs infected with heart worm, survive this phase. I slept on my left side during this initial kill, in an effort to keep critical circulation paths clear of clogging.

Don't worry about blocked capillaries, **Ginkgo** helps grow 2 miles of capillaries for every mile you walk.

## How to ramp meds

I then suggest slowly ramping up the formula, and the antiparasitics. Keep in mind that it takes 2 weeks at full strength to generate a kill, and that any dosing level you start at, will only react with a 3 day delay. So the dosing level you take today, will only show its effects in 3 days.



I therefore suggest starting low, step up slowly, pausing for 3 days, before you increase to the next level. This will prevent any toxic overloads, mass migrations, or massive die-off events.

This formula is capable of treating systemic infections, and massive die-off events are possible (HERX Reactions). Test dose **chitosan**, **caprillic acid**, and alike to ensure you do not have yeast, or bacterial **SIBO** or SIYO conditions.

You must be able to process calcium to complete the anti-parasitic killing process. Make sure SIYO and SIBO infections are clear prior to ramping any anti-parasitic.

I suggest starting the anti-parasitics formula by ramping up Minerals, Metals, Herbs. The first anti-parasitic in the formula is **MSM**, beyond **MSM** are **IVM**, and **PinX**, followed by **DEC**, then followed by **selenium**, and **zinc sulfate**. **Zinc sulfate** is in itself a very powerful anti-parasitic, capable of killing protozoa, and worms.

Zinc-sulfate was the first thing I discovered would cause worm bursts. I started a chain reaction of worm bursting that would not stop.

I ramped <u>zinc</u> up to the standard dose of 600 mg/D TID. (Total in Day), I then went to 9.32mg/kg and worms started to explode. After the initial kill, I went on to discover <u>Piperazine</u>, <u>Albendazole</u>, and <u>Praziquantel</u> effects.

The actual start-up sequence is altered, because ramping **Praziquantel** needs to be before **Albendazole**, in that if you have a fluke infection, there is no way to kill Ascaris, until the flukes are killed first. Ramp **Praziquantel** for two weeks to determine if you have a fluke infection, if you have not performed the ELISA test for flukes. If they start to die, you may be in for a rough ride. Flukes are easy too kill at 20-25mg/kg/day, but if they are in your brain expect to be in for a bad ride for 4 days.

## **Phase 3 and Phase 4 Dosing**

The following

- Daily time line, and
- typical Dosing tables

are provided as quidance, based upon the levels I determined based upon my systemic infection.

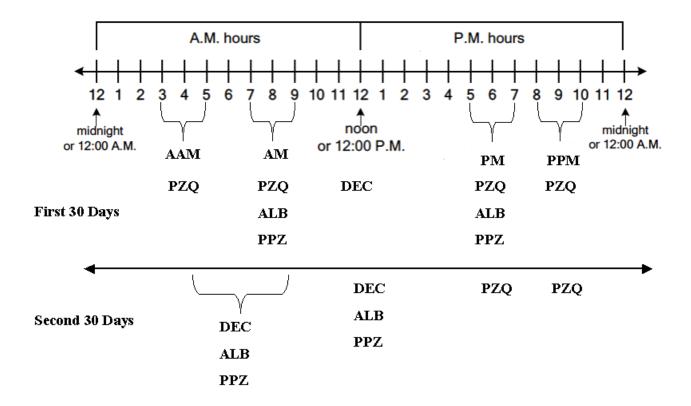
The dosing level kills parasites slowly, over a period of days, taking about 14 days to start killing, and within 21 days, getting to flukes in the brain. Ascaris are much more difficult, at Day 48 the worms are still decaying, and dying, but the majority of the worms seam to be delt with. Removal of Vitamin A and Zinc-sulfate after the initial kill, is advised, in that these vitamins are able to keep Ascaris alive.

After both Flukes and Ascaris are dying, change the vitamin dosing to maintenance dosing for flukes, and taper. Maintain Ascaris formula (**Piperazine**, **DEC**, **IVM** pulse, and wait about 4-6 weeks at this taper mode, for your body to recover from the fluke kill. When the immune system has recovered, Ascaris killing is made possible. This should accelerate the mop-up of the Ascaris worms.

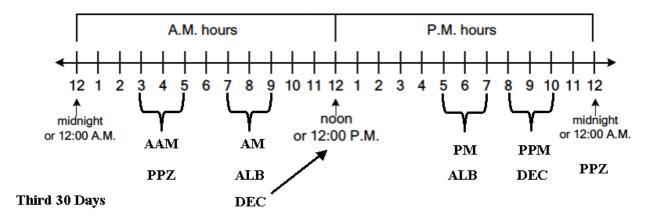
First 30 days of Antiparasitcs = Phase 3 (Fluke and Flatworm kill)

Second 30 days of Antiparasitics = Phase 4. (Immune system recovery, taper Fluke, Status Quo for round worms. ) Spreading out round worm meds increases pressure on roundworm infections, while taper of **PZQ** allows immune recovery, and ensures flatworm kill.

## 24-Hour Timeline



#### 24-Hour Timeline



After tape worms and flukes are killed, moving PPZ around going to sleep, maximizes rest. Since ALB has less kick than DEC, flattening the formula increases the movement of anti-parasitics into the remaining roundworms.

## **Dosing Timeline**

## Anti-parasitic dosing Total in Day (Q1D)

	Total Dose in a Day (Typical)										
		ALB	PPZ	PZQ	IVM	MnSo4	ZNS 04-H20	Vit A			
250 lbs	113 kgs	800 mg	1500 mg	5000 mg	22.6 mg	1030 mg	600 mg	60,000 IU			
225 lbs	102 kgs	720 mg	1350 mg	4500 mg	20.4 mg						
200 lbs	91 kgs	640 mg	1200 mg	4000 mg	18.2 mg						
175 lbs	75.5 kgs	560 mg	1050 mg	3500 kg	15.1 mg						
150 lbs	68 kgs	480 mg	900 mg	3000 mg	13.6 mg						
125lbs	57 kgs	400 mg	750 mg	2500 mg	11.3 mg	? Skip a day	300 mg?	30,000 IU?			
100 lbs	45.5 kgs	320 mg	600 mg	2000 mg	9.1 mg						
75 lbs	34 kgs	240 mg	450 mg	1500 mg	6.8 mg						
50 lbs	23 kgs	160 mg	300 mg	1000 mg	4.6 mg						

Q1D quantity taken over one day, Total daily dose maximum.

MBZ Maximum 600mg/D Flagyl Maximum 800mg/D Alinia tested to 25mg/kg/D

Maximum for 238 lb 113 kgs male (250 lbs row in table)

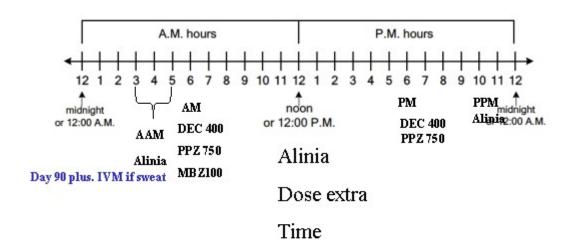
#### **Do Not Exceed**

- Maximum 400 mg ALB per single dose
- Maximum 400 mg DEC per single dose
- Maximum 750 mg PPZ per single dose

PPZ taken with (mixed) Greek yogurt (Thick), these essential fats are essential. IVM taken with (mixed) Greek yogurt (Thick), these essential fats are essential.

#### **Final Formula**

#### 24-Hour Timeline



# **ASCARIS AND FLUKE FORMULA 10/20/15**

# (min-typ-max)

ALBENDAZOLE/PRAZIQUANTEL Combination Formula

## AAM (2-4 AM) - if awake, if not dose on rising

(1/2) teaspoon <u>Praziquantel powder</u> (measuring spoon) in <u>Greek Gods Greek Yogurt</u> or 4% cottage cheese, try to take <u>Praziquantel</u> several hours away from <u>Albendazole</u>. <u>Praziquantel</u> keeps worms from feeding on blood, making them hungry. Later when you dose <u>Albendazole</u>, they are ready to feed, absorbing the anti-parasitic drug.

## **AM Kickoff Minerals, Metals, Liver Stimulation**

(I dose anti-parasitics between 3 AM to 9AM, to optimize sleep)

- <u>Magnesium citrate</u> (1-1-1) 133mg, Citrate loop, source of <u>Magnesium</u> for all cellular processes (2SP)
- Potassium citrate (1-2-2) 99mg, Primary to balance kidney toxin removal controls
- Magnesium Sulfate (1-1-1) 1030mg, Primary Liver flush, Magnesium and Sulfur Source (2SP)
- MSM (2-3-4) 1000mg, Organic Sulfur- Increases cell fluid movement, protects GI tract (6SP), Sulfer Consumable 450CYP ROS Leak
- Malic acid (0-1-2) Solaray w HTP5 b1 b6 Magnesium chelate Malate loop, gi sickness
- ALA (1-2) (may overstimulate worms) DNA accelerator gas petal (4SP) Recycles <u>vitamin A/C</u> in mitochondria 10X, Recycling Vitamin A after the initial kill, and the discontinuation of Vitamin A supplements, may be offset by <u>ALA</u>. There is no research that directly indicates that <u>ALA</u> increases the survivability of Ascaris. Continued research will be required to determine if <u>ALA</u> helps or hinders the Ascaris killing process. <u>R FRACTION</u> SWANSON SWU250 may be safer alternative, Swanson (1)
- CQ10 (1-2-2) 400mg, Travels through all 4 layers of cellular transport, moving electron ionic force. (4 SP)
- L Carnitine (2-3-2) 500mg/cap, DNA detox Instruction, tells mitochondria to dump toxins
- **Source of life (1-2-2)** Source Naturals, #3058, protein, amino source vitamin, with or without kelp, used to repair DNA with amino food. (8SP any time) Essential DNA balance, broad spectrum vitamin.
- LEM Mushroom Extract (4)
- **Zinc Sulfate Monohydrate (0-2-6)** 50mg yield per capsule, WBC production, Suck **Iron** from Parasite, O2 (9.32mg/Kg SP) Zinc is essential, only this exact kind of **zinc** can be taken in these levels, "Rising". Studies show 600mg of Zinc Sulfate Monohydrate can be safely taken for a month. My worms started to burst at 9.32mg/Kg/D).
- After 30 days it is essential to discontinue ZINC supplementation, to complete the killing of Ascaris alone, Flukes should be dead

http://www.ncbi.nlm.nih.gov/pubmed/17908741

#### **CONCLUSIONS:**

We found that vitamin A and zinc supplementation was associated with distinct parasite-specific health outcomes. Vitamin A plus zinc reduces G. lamblia incidence, whereas zinc supplementation increases A. lumbricoides incidence but decreases E. histolytica-associated diarrhea.

- Selenium (2-3-4) 200ucg/cap, Offset <u>ROS</u> loop stress, delivers O2 to cells, WBC production, 450CYP ROS Consumable under Ox stress
- Copper Citrate (1-2-3) or Chelate 2mg/cap, force gut bacteria profile, balances Zinc
- **Chaparral (1-2-3)** fixes genetic psora DNA error of immune system 33% of people have, FXR bottleneck.
- Tri Iodine (1-1-1) lowers inflammation, heals parasite wounds, alternate with chromium (below)
- Optional **Stinging nettle root** (4), Immune ligand 26R, helps with lung fluids should they appear.
- **Ginkgo Giloba** (2-3-3) keeps capillaries open and unblocked, **very important for startup**!
- **Spirulina (4-4-10) capsules** 500mg, tablets, or optional teaspoon (5 grams) Spirulina powder in yogurt
- Optional **Chromium** (0-1). Get up and go, will cause a headache over 3 capsules, CQ10 helps.

## AM Liver, Supplement, Sustain, Flush formula

#### **Fat soluble Vitamins**

- 1 Glass <u>orange juice</u> with orange peel zest.
- With few exceptions, like some vitamins from B complex, and fat-soluble vitamins (D, E, K and A or 'DEKA'), which are stored in the liver and fatty tissues of the body. These vitamins build up and remain for a longer time in the body than water soluble vitamins.[1] To prevent overload, the dose should start high, then diminish with time.
- Start high dosage <u>vitamin A</u>; high dosage, slow release <u>vitamin B3</u> (30mg); and very high dosage <u>vitamin B6</u> alone (i.e. with or without vitamin B complex) are sometimes associated with vitamin side effects that usually rapidly cease with supplement reduction or cessation.
- Vitamin D3-1 (1000IU) long term D3-5(5000IU) D3 typical D3-50 (50,000IU) max). D3-50 will accelerate the killing of worms in the brain) to start, ramp to D3-50/D D3 prevents Calcium in suspension. I found killing brain flukes on a D3-50/D to be intense. After 14 days I can only say this is a near death experience, and spent many days in bed, as they died. Much black stool was evidenced. By day 21, on the full strength formula, the killing is mostly gone. I have since backed down to D3-5 (5000IU), after about 38 days.
- Vitamin E 800 IU (1) E2000 IU maximum Helps pull Lipid toxins out of the cells.
- Vitamin A 45,000 IU ((0-3)\*15000 IU) Palmitate Accelerate Liver function (6 SP) Vitamin A is
  essential for the human body, but also allows Ascaris worms to survive. It is essential to discontinue
  Dosing of vitamin A after the infection has been brought under control.
  http://www.ncbi.nlm.nih.gov/pubmed/17908741

A. lumbricoides infections increased among children in the combined vitamin A and zinc group or the zinc alone group, respectively.

- <u>Silymarin</u> (<u>milk thistle</u>) (3\*300mg) 1000mg, urine production, protect liver membranes from toxin purge, Protects Organs Ox 450CYP ROS
- **Choline (0-1-1)** offload Liver function
- Taurine (0-1-1) Accelerate Liver
- Bentaine (0-1-1) Protects Liver cells from toxins, taken with milk thistle.
- **2G** Ester C (1-2-3) Detox Liver, calcium source.
- **B25 B50 (1-1-1)** Supply complement of Liver consumables, The liver needs B vitamins.
- <u>Boron</u> (0-1-1) Metal used by Liver, helps regenerate liver, It enhances the body's ability to use calcium, <u>magnesium</u>, as well as vitamin D. It also seems to assist in brain functioning and recognition. <u>Boron</u> seems to prevent calcium and <u>magnesium</u> from being lost in the urine and may help with decreasing menstrual pain by increasing the oestradiol level, which is a very active type of estrogen.
- Biotin (0-2-2) 5mg, Intestine under stress fails to get this essential gateway molecule
- **Eggs** (1-2-2) 33 vitamins and minerals
- Valerian Root (0-2-3) Stimulates Bile production.

## AM Nitrogen Loop Stress Reduction

- Piperazine citrate (1 Teaspoon (Measuring kitchen spoon, level) 1 Tablespoon)
- (250 mg 750mg) Removes Uric and <u>Ammonia</u> compounds, helps kidneys at low dose, Provides stability to nitrogen NH3, NH4 processes. Dosing more than 1.5 grams per day can cause egg, larvae, and cyst die off in brain, CNS symptoms. <u>Use the lowest dose of Piperazine</u> that removes <u>ammonia</u> smell from stool and urine. Caution: <u>Piperazine</u> can numb the brain to pain, if you dose anti-parasitics to hard, or kill to fast, you may not feel the brain pain. Be aware of this numbing side effect of <u>Piperazine</u>. If you smell fixer fluid <u>ammonium thiosulfate</u> smell, it is <u>protein</u> <u>breakdown</u>.
- The kind of round worm liquid I used had 50 mg/ml. This is equal to 250 mg/teaspoon/ or 5ml for metric folks. Peperazine phosphate may have a higher base absorption ratio. Taper slowly after 90 days?, significant brain pain may be evidenced if a significant brain kill was accomplished.

## **AM Antiparasitics**

- Albendazole 200mg (2-2-2) (~10mg/kg/D). Persons who weigh 125 lbs should lower dose to (1-1-1) Albendazole 200mg
- First 30 days 1/2 teaspoon <u>Praziquantel powder</u> (measuring kind of kitchen spoon) in cottage cheese or Greek yogurt.
- Second 30 days DEC (1 or ½ tablet) 400mg 200mg, Kills Deeper into the body.

## **AM Immune System**

- Danish Rose Hips (2-2-8) 9 cis trans retinolic A (Take Tea or capsules if you cant make tea)
  Only molecule that binds 3T3 in DNA Helix, Source of broad spectrum C and A vitamins. (6 SP)
- Magnolia Extract (2-3-3) capsules, Amermed (4SP)
- Fruits, Bananas, Oranges, Apples

## (Noon) Blood Flush Substances

- 1 glass orange juice
- Ginger (1) Blood Detox
- Barberry bark (0-1-1) Detox
- GoldenSeal (1) Kidney Detox
- Oregon Grape Root (0-1-1) Detox, bacteria fighting properties of Oregon grape root in the treatment of bacterial diarrhea, intestinal parasites, can be used intermittently.
- Berberine (1) isoquinoline alkaloids
- MSM (2) (6 SP)

## (Noon) Circulation health

- Horse Chestnut (1) heal circulation tissue, patch leaks in arteries and veins
- White willow bark (1-2-3) no clot, pain reliever, blood thinner, platelet control (3SP)
- Antibiotics to capture fluke bacteria in CNS, Optional Deoxycycline (1) if kills cause brain swelling, if you get fever during heavy kill, deoxy stops bacteria
- Spirulina (4-10) capsules 500mg or optional teaspoon (5 grams) Spirulina powder in yogurt
- Vitamin K Salads, raw vegetables

## **Noon Antiparasitics**

- First 30 days <u>Albendazole</u> **200mg (0-0-2)** (10mg/kg/D max) for the first 30 days, Persons who weigh 125 lbs should lower dose to (1) Albendazole 200mg.
- Second 30-60 days ½ teaspoon <u>Praziquantel powder</u> (measuring spoon) in cottage cheese or Greek yogurt. (¼ teaspoon for persons of 125 lbs) for deeper killing power

- **Second 30 days** teaspoon **piperazine** citrate.
- **DEC** (1 or ½) 400mg 200mg

## PM Lymph Flush substances

- **Dodder Seed Extract** (2), Dodder at night can make your lymph system gurgle. No kidding.
- GuaiAid (2-3) 600mg
- Water

#### PM

- 1 glass orange juice
- Cod liver oil (1)
- MSM (2) 1000 mg (6SP)
- Source of life (2)
- <u>Selenium</u> (2-3-3) 200 ucg ea.
- Zinc Sulfate Monohydrate (2-4-6) 50mg yield, dose for 113 kgs person
- Copper (1-2-2) 2 mg
- L Carnitine (1-2-2) 500 mg, I like to keep L Carnitine mostly in the morning, powerful detox instruction.
- Berberine Sulfate (1-2-2)
- Chaparral (1)
- KGP flush (1) full dropper in Cranberry Juice to keep kidney tubes clear, optional P5P and piridoxamine
- Spirulina (4-4-10) capsules 500mg or optional teaspoon (5 grams) Spirulina powder in yogurt

## PM Antiparasitics

- First 30 days (2) <u>Albendazole</u> **200mg** (10mg/kg/D) up to 6 months for persons over 50 years old
- first 30 days Piperazine citrate 1 teaspoon 1 tablespoon PPZ, taper after 90 days?
- 30-60 days Praziquantel powder 1/2 teaspoon (measuring spoon) in cottage cheese or Greek vogurt

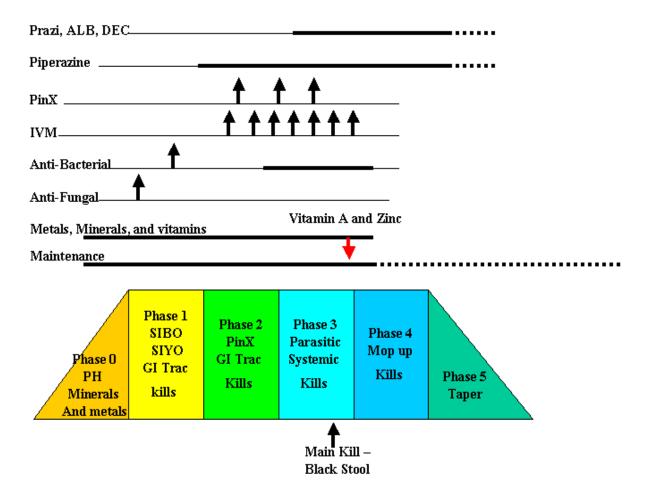
#### PPM

- Borage seed oil (2)- GLA is DNA antioxidant G=no replication errors in DNA, no cancer please.
- <u>Ester C (2)</u> 1000mg tabs
- St John's Wart (1) optional for sleep
- **GuaiAid** (2-3-3) (Guaifensyn) 600mg, Cut mucin, repairs DNA base pair, stops cells from burning sugar.
- <u>Dodder Seed Extract</u> (1-2-2) 20-1, 30-1 strength Bartlow
- White willow bark (2) (3SP)
- Milk fats, cottage cheese, milkshake, butter, cream, any milk fat product.

## **PPM Anti-parasitics at bedtime**

- Praziquantel powder ½ teaspoon (measuring spoon) in cottage cheese or Greek yogurt
- 30-60 days Piperazine citrate 1 teaspoon 1 tablespoon PPZ, taper after 90 days

### **Time Line**



Each phase may take about a month. My phase 4 took 6 weeks because I maintained Vitamin A and Zinc to long, keeping the Ascaris worms alive. The Flukes were dead in about 3 weeks. I am maintaining the anti-parasitics for an additional 4 weeks, to ensure I never have to do this again. I am at 6.8 weeks, and I have transitioned to Maintenance, discontinuing all metals, minerals, and vitamins. My personal log tracts my progress in eliminating the round worm Ascaris infection. I will be putting in additional effort into DNA repair, and memory supplements some time in the future.

## **Conversion Factors**

To understand the quantities that are needed, you have to know the relevant units of measure:

Kg = Lbs/2.2 28 grams = 1 ounce of liquid Teaspoon = 5 grams, 5ml of liquid (powder it is  $\sim 1/2$  less) Tablespoon = 15 grams, 15 ml of liquid 1,000 milligrams = 1 gram 1,000 micrograms (ug or ucg) = 1 milligram

## **Shopping Sources 051515**

#### (nominal-typical-maximum)



## **Water**

**Drink pure water,** or drinks that are mostly water (tea, very diluted fruit juice, sparkling water with lemon) throughout the day.

The following drinking water treatment is recommended at a minimum.

- 1) Oversize Carbon filter on primary faucet water tap. (GE or equiv)
- 2) Water basin filter, with essential minerals (brand XYZ)

## **Nutrient destroyers:**

- Aspirin--destroys vitamin A, calcium, potassium, B complex and C.
- Caffeine--destroys vitamin B1, inositol and biotin, potassium and zinc and prevents calcium and **iron** assimilation.
- · Chlorine in water--destroys vitamin E.
- Chocolate--contains caffeine and it is very irritating to the kidneys. High in fat, which can cause indigestion.
- · Fluoride--destroys vitamin C
- Sleeping Pills--destroy folic acid and vitamin D.
- Menstruation--requires extra iron, vitamin B12, calcium and magnesium.
- Nitrates/Nitrites--Destroys vitamins A, C and E
- Stress--Physical, emotional and mental all vitamins are depleted.

### **Interventionalists**

These substances can help keep you alive.

- MSM and baking soda in distilled water
- Adding ¼ of a teaspoon of baking soda into the MSM water helps in the early weeks.
- <u>Selenium</u>, <u>CQ10</u>, <u>ALA</u>, <u>I-carnitine</u>, and <u>Milk Thistle</u> are early interventionists, but they do not come close to solving the toxin problem.
- 2Grams CQ10 in d alpha E oil
- 2Grams **L Carnitine** (Acytl or Chelated forms.
- 2Grams ALA

#### **HOW TO USE A DRUG / SUPPLEMENT**

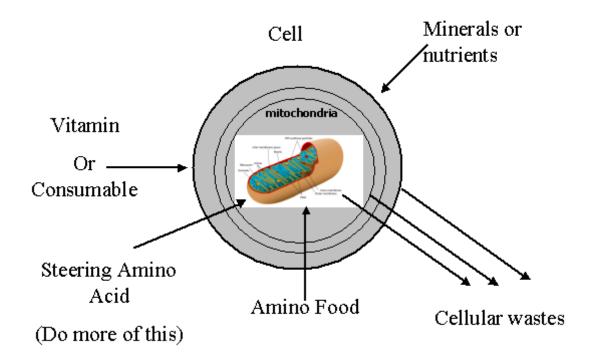
## Vitamin process ...factor, cofactors, steering amino acid: MAK

Each drug or vitamin supplement usually has a base (supply of material the drug/supplement uses during its activity, like oxygen is required for a flame, the supplement provides an essential element(s) that is/are consumed during the activity process.

Vitamin + consumed mineral + amino acid (steering acids)= result

Each drug or vitamin have co-factors. These co-factors can take the form(s) of genetic steering acids, or complementary nutrition process elements or substances, catalysis's, minerals, or process consumables, which assist or direct the drug or vitamin to do a specific task, do it with more intensity, or behave in the presence of other body processes. Like an instruction to go here and do that it is the entire group of substances, collectively, that enable a function.

It is in this formulation and support of a drug or vitamin that one needs to understand fully to use the chemical process to accomplish a specific purpose, task, or result. The formulation of how one uses a drug or vitamin should always be shaped or customized so that a specific end result is achieved.



Since parasites disturb many body processes, and the formulations become quite long and extensive, the vitamin list provided was reduced from long form, by selectively incorporating substances which were capable of performing many simultaneous roles, withing a reduced framework. This reduction reiterative process took quite some time, and was performed while the process, and anti-parasitic substance evaluations were being performed. The omission of even a single substance could have a profound effect, so the vitamin list may not be exactly optimized, but is the closest formula I could develop.

I have found that the formulation (kind or base) of a vitamin makes a huge difference if it has any positive effect on the body. The formulation has everything to do with its effect, if the vitamin will perform its intended task. Different kinds of copper, i.e. Chelate, citrate, metalic fraction, each go to different areas of the body or cells. Do not deviate from the specified vitamin type (form).

A perfect example of a vitamin not doing its function properly is Rosehip C, a chemically created vitamin tablet. Rosehips are a natural seed product. If taken in the chemically reduced form, like a pill, it has the same name but a different function. If taken in large pill form concentrations can generate crystals in the kidneys. If the natural rosehip seeds are ground, and put into tea, a much higher level of C and A can be obtained, with no harmful side effects.

This is of particular importance to me since I damaged my kidneys during several bouts with staff infection as a child. Thankfully the doctors saved me after my kidneys shut down, and they were restored to function.

#### **Vitamin Links**

http://en.wikipedia.org/wiki/Vitamin

http://www.raysahelian.com/

www.peacefulmind.com/anti-aging.htm

http://www.lef.org/anti-aging/

http://ods.od.nih.gov/factsheets/list-all/

http://www.ifnh.org/

http://www.herbs2000.com/

https://www.rainpharm.com/

## A primer on free radicals, pro-oxidants, and antioxidants.

## Complete antioxidant protection, how

Simply defined, a free radical is a highly reactive atom that can destroy body tissues. Normally, an atom's electrons come in pairs. If one of the electrons gets stripped away, the atom--now a free radical--becomes unstable. It sets off on a frantic search to find another electron to complete its set, grabbing onto any electron it can find. But by stealing electrons, free radicals destroy those other molecules. Because the oxygen atom is most often involved as the donor of the electron, this damaging process is known as oxidation, and is similar to the process that causes sliced apples to turn brown or cars to rust. Compounds that promote oxidative damage are referred to as prooxidants.

Free radicals come from our environment, in pollutants such as chemicals or cigarette smoke; in our diet in the form of fats damaged by frying or the presence of nitrates in smoked or cured meats. Even sunlight produces free radical damage. But free radicals also result from the cell's own metabolic activity.

Free radicals shoot through the cell's membranes, tearing gaping holes, damaging the cell's delicate structures, including DNA, (the cumulative damage they cause leads to cellular aging). This, in turn, contributes to heart disease and cancer. Carcinogens (cancer-causing compounds or virus, mold, bacteria infections) cause severe free radical or oxidative damage to cell structures.

<u>Antioxidants</u> quench the unpaired electron by donating one of its own electrons, effectively "calming down" the free radical, resulting in help for aging, cancer and other degenerative diseases. Because they protect cell integrity, <u>antioxidants</u> slow down the aging process, enhance immune function, reduce inflammation, and fight allergies.

The ultimate combination of **antioxidants** requires both Part A and Part B

**Antioxidants** vitamin C, zinc, selenium, beta-carotene, and vitamin E. are useless without enablers

Most **antioxidants** require some sort of "partner" antioxidant that allows it to work more efficiently. And scientists have discovered that it is quite easy for one antioxidant nutrient like beta-carotene to become damaged if it's used alone ( that is, without its partner **antioxidants** vitamin C, vitamin E, and selenium ).

Similarly, selenium functions primarily as a component of the antioxidant enzyme **glutathione** peroxidase. This enzyme works closely with **vitamin E** to prevent free radical damage to cell membranes. Studies looking only at **vitamin E**'s ability to reduce cancer and heart disease are often

faulty because they failed to factor in the critical partnership between selenium and **vitamin E**, not to mention the interrelationship between **vitamin E** and coenzyme Q10

## **Vitamin Classification: MAK**

Vitamins are classified as either <u>water</u>-soluble or fat-soluble. In humans there are 13 vitamins: 4 fat-soluble (A, D, E, and K) and 9 water-soluble (8 B vitamins and vitamin C). Water-soluble vitamins dissolve easily in water and, in general, are readily excreted from the body, to the degree that urinary output is a strong predictor of vitamin consumption. [13] Because they are not readily stored, consistent and daily intake is important. [14]

B-complex vitamins and vitamin C are **water-soluble vitamins** that are not stored in the body and must be replaced each day. These vitamins are easily destroyed or washed out during food storage and preparation. Acid supplements: Indeed, there may be a role for vitamin C as ascorbic acid.

Ascorbic acid is acid and so improves digestion of protein. It is also toxic to all microbes including bacteria, yeast and viruses as well as being an important anti-oxidant - indeed the eventual receiver of most electrons from free radicals. Humans, guinea pigs and fruit bats are the only mammal species which cannot make their own vitamin C and we have to get it in food. Scaling up from other mammals we should be consuming 2-6 grams daily (a hundred fold more than the government RDA of 30mgs daily!).

The B-complex group is found in a variety of foods: cereal grains, meat, poultry, eggs, fish, milk, legumes and fresh vegetables.

### Dr Earl Mindell's Vitamin Bible

Later I find many people raving about Dr. Earl Mendel's Vitamin Bible revision 7 on the internet. To date I have purchased four copies of this book. Whenever I lend it out, it never comes back. I guess that says a lot. I guess that says it all.

EARL MINDELLS NEW VITAMIN BIBLE ISBN 0-446-614089-2

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### **Dr Royal Lee**

We will only discuss two fractions that are lost by this processing and substitution. These are vitamin E and the enzyme, phosphatase. The bleach and chemicals used to keep bugs out of flour destroy both. Pasteurization destroys phosphatase in milk. Oleomargarine contains no vitamin E as does butter. It also contains a poison, sodium benzoate, as oleo cannot be made to keep without a chemical preservative.

Why do we need phosphatase? Simply because without it, we fail to split and assimilate the mineral salts in our foods that are in the form of phytates. No enzyme is naturally secreted in the human intestinal tract that splits phytates although many other animals, including the rat, do have such an enzyme

(Cereal germ and bran are the highest common sources of phosphatase, other than raw milk

Watch against the use of white flour, refined sugar, pasteurized milk, and imitation butter.

One way this can be done is by increasing the intake of fluid which is acid in reaction, such as apple juice, cranberry, or grape juice; for Vermont folk medicine knows that acid thins the body fluids, keeping them liquid, while alkaline fluids thicken them, impeding circulation."

Apple Cider Vinegar
Eat potatoes instead of bread
Eliminate white bread as much as possible
Under cook or eat fresh fruits and vegetables as much as possible

Many Functions

Vitamin F was first discovered as a part of the wheat germ oil vitamin complex; at least the term vitamin F was first used to designate the essential fatty acid fraction.)

### Water

Short Name: water

Color: Clear

Date Discovered: Millions of years ago

Properties:Since the water molecule is not linear and the oxygen atom has a higher electronegativity than hydrogen atoms, it carries a slight negative charge, whereas the hydrogen atoms are slightly positive. As a result, water is a polar molecule with an electrical dipole moment. Water also can form an unusually large number of intermolecular hydrogen bonds (four) for a molecule of its size. These factors lead to strong attractive forces between molecules of water, giving rise to water's high surface tension[15] and capillary forces. The capillary action refers to the tendency of water to move up a narrow tube against the force of gravity. This property is relied upon by all vascular plants, such as trees.

Water is a good solvent and is often referred to as the universal solvent. Substances that dissolve in water, e.g., salts, sugars, acids, alkalis, and some gases – especially oxygen, carbon dioxide (carbonation) are known as hydrophilic (water-loving) substances, while those that do not mix well with water (e.g., fats and oils), are known as hydrophobic (water-fearing) substances. All the major components in cells (proteins, DNA and polysaccharides) are also dissolved in water.

The human body contains from 55% to 78% water, depending on body size.[46] To function properly, the body requires between one and seven liters of water per day to avoid dehydration; the precise amount depends on the level of activity, temperature, humidity, and other factors. Most of this is ingested through foods or beverages other than drinking straight water. It is not clear how much water intake is needed by healthy people, though most advocates agree that approximately 2 liters (6 to 7 glasses) of water daily is the minimum to maintain proper hydration.[47]

Medical literature favors a lower consumption, typically 1 liter of water for an average male, excluding extra requirements due to fluid loss from exercise or warm weather.[48] For those who have healthy kidneys, it is rather difficult to drink too much water, but (especially in warm humid weather and while exercising) it is dangerous to drink too little. People can drink far more water than necessary while exercising, however, putting them at risk of water intoxication (hyperhydration), which can be fatal.[49][50]

The popular claim that "a person should consume eight glasses of water per day" seems to have no real basis in science.[51] Similar misconceptions concerning the effect of water on weight loss and constipation have also been dispelled.[52] Hazard symbol for non-potable water. An original recommendation for water intake in 1945 by the Food and Nutrition Board of the United States National Research Council read: "An ordinary standard for diverse persons is 1 milliliter for each calorie of food. Most of this quantity is contained in prepared foods."[53] The latest dietary reference intake report by the United States National Research Council in general recommended (including food sources): 3.7 liters for men and 2.7 liters of water total for women.[54] Specifically, pregnant and breastfeeding women need additional fluids to stay hydrated. The Institute of Medicine (U.S.) recommends that, on average, men consume 3.0 liters and women 2.2 liters; pregnant women should increase intake to 2.4 liters (10 cups) and breastfeeding women should get 3 liters (12 cups), since an especially large amount of fluid is lost during nursing.[55] Also noted is that normally, about 20% of water intake comes from food, while the rest comes from drinking water and beverages (caffeinated included).

Water is excreted from the body in multiple forms; through urine and feces, through sweating, and by exhalation of water vapor in the breath. With physical exertion and heat exposure, water loss will increase and daily fluid needs may increase as well. Humans require water with few impurities. Common impurities include metal salts and oxides, including **copper**, iron, calcium and lead,[56] and/or harmful bacteria, such as Vibrio. Some solutes are acceptable and even desirable for taste enhancement and to provide needed electrolytes.[57]

Forms: RAW, Water,

"H2O" and "HOH"USE: Essential for Life. Functions as a solvent for a wide variety of chemical substances.

Contents: Water

## **Herbal Medicine Store**

I asked my wife to take a road trip with me to visit the Timberlake Herb Store. It was in the middle of nowhere, on a small road between two small towns north of Raleigh North Carolina. It was an eye-opening trip. On the shelves were raw herbs and roots of every kind in large gallon glass apothecary containers. Each one labeled with common and Latin names. The storeowner and herbal helper were very knowledgeable. They also had prepackaged mixes, teas, and capsules. The number one issue of the day was help with my digestion and constipation.

Timberlake Herb Store

1776 Highway 401 South
Louisburg NC 27549
1-800-213-0666
http://timberlakeherbstore.com/
We discussed Chinese medicine, and digestive issues. They carried Nature's Sunshine Products.

Nature's Sunshine Products, Inc. Spanish Fork, Utah 84660 1-800-223-8225

## **AMINO ACIDS, FOOD, SUPPLEMENTS**

Life began in the sea 4 billion years ago. Fossil records indicate that the blue-green Algae's are the most primitive of the 1,000 or so different algae varieties on earth, dating back over four billion years. The blue-green Algae's have managed to survive all that time while millions of other plant and animal species became extinct. What makes blue-green Algae's so powerful and amazing for human health is the same super-dense nutrition and energy that we can now harvest and enjoy for maximizing our human potential.

Proteins are made up of amino acids, which are the building blocks of the body.

- Our DNA is made of them.
- Our Mitochondria is packed full of them.
- The cell core process, decisions, human process, and life comes from them
- The human cell after billions of years of evolution is still able to absorb amino acids directly.

Humans have evolved to eat food, higher orders of life, but after 4 billion years, just like our appendix, we have left over functions, gates, and secrets inside our body. Algae and Amino acids have a back door into our cell, and are directly absorbed, bypassing or so called evolution, and can operate in a primitive molecular code, similar to binary inside a computer, amino acids are the basis of human function.

#### http://www.skinnybits.com/index.php/history-algae

- It was the first plant life on earth.
- Algae was forgotten about until WW II, when the Hiroshima bomb left the Japanese without food. The USA Government was committed to helping Japan get back on their feet and remembered that years earlier, Germany had used algae to avoid mass starvation. So once again, algae came to the rescue Asia much needed protein source to avert mass starvation.
- Interestingly, the algae given to the Japanese was <u>Chlorella</u> algae. And not only did it solve their protein needs, it also seemed to cure their radiation poisoning (from the nuclear bomb). This stunning discovery along with remarkable ability of algae to feed large numbers of people led the Americans (Carnegie Institute and Rockefeller Institute) to research <u>chlorella</u> algae extensively. At the time there was a growing nutrition crisis in America. Large numbers of military personnel were returning to America after the war to start families and there was an urgent need for fast production of healthy food to feed them. Algae looked like it could be the answer.
- It may take up to four weeks to see results with Spirulina, so be patient and stick with it!

http://www.algaeindustrymagazine.com/department/features/special-report-spirulina/

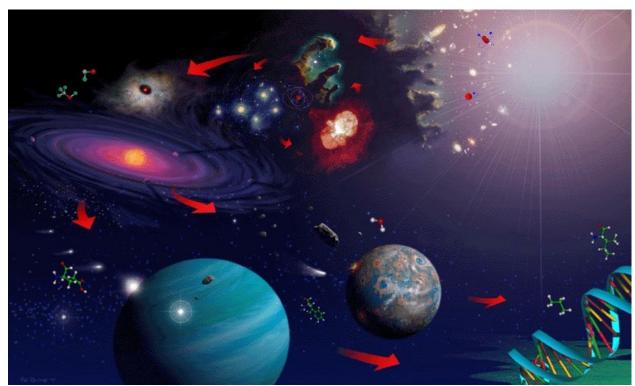
## Understanding the role of microscopic algae

http://www.superfoodsforlife.com/page/103407

- The foundation of life
- The Bible describes, when the Israelites were starving in the wilderness, God provided 'manna'...dried algae on the rocks.

- Spirulina is the immortal descendent of the first photosynthetic life form. Beginning 3.5 billion years ago, blue-green algae created our oxygen atmosphere so other life could evolve.
- **Spirulina** is 60-70% protein and 83 95% digestible
- All 10 Dietary-Essential Amino Acids 20 amino acids total, in synergistic balance
- The building blocks for over 2,000 enzymes with 2-3 times the energy of any other 'live food'
- Spirulina cleanses and nourishes, balances and energizes, strengthens and protects, regenerates and rejuvenates
- Spirulina has everything you need to live on

http://www.indiana.edu/~geol105b/1425chap10.htm



When the Earth formed at about 4.6 to 4.5 billion years ago, it was initially a cold place bombarded by meteorites, and later on it probably got so hot that the surface may even have been in a molten state (the magma ocean). Neither condition was very conducive to life as we know it. Yet a billion years later we have rocks that contain evidence (fossils, stromatolites) of microorganisms such as bacteria and blue-green algae. Organic life probably existed even earlier than that, based on carbon isotope data from the 3.8 billion year old Isua Formation of Greenland. The Isua Formation consists of metamorphosed sediments that contain graphite with an unusual carbon isotope ratio. They are enriched in Carbon 12 (C12) over C13 when compared to carbon isotope ratios in meteorites (presumed to record the primordial carbon isotope ratio of the solar system). Preferential uptake of light carbon (C12) is something that microorganisms routinely do when they absorb carbon from their environment, either by photosynthesis or by metabolizing other carbon compounds. Thus, C12 enrichment in these rocks suggests the presence of living organisms as early as 3.8 billion years ago.

Much of what our own DNA is also found in the DNA of other organisms, all the way down to simple bacteria. The common thread that runs through all life is the use of the DNA molecule to store and transmit information. Thus, in order to find out about the origins of life we have to go back to bacteria and beyond. Thanks to the advances of cell biology, biochemistry, and molecular biology, scien-

tists are becoming increasingly adept at reading the DNA record, and also at decoding the relative antiquity of various cell components and metabolic functions (such as oxygen based metabolism, photosynthesis, etc.). As we try to reconstruct the earliest events in life's history we have to assume that all that went on happened without the benefit of foresight (just as our daisies did not purposely cooperate to stabilize Earth's climate). Every step must be accounted for in terms of antecedent and concomitant events, and must obey the laws of physics and chemistry. Each must stand on its own and cannot be viewed as a preparation for things to come.

Actually, as early as the 1930s, Alexander I. Oparin in Russia and J.B.S. Haldane in England had pointed out that the organic compounds needed for life could not have formed on the Earth if the atmosphere was as rich in oxygen (oxidizing) as it is today. Oxygen, which takes hydrogen atoms from other compounds, interferes with the reactions that transform simple organic molecules into complex ones. Oparin and Haldane proposed, therefore, that the atmosphere of the young Earth, like that of the outer planets (Jupiter, Saturn, etc.), was reducing: it contained very little oxygen and was rich in hydrogen (H2) and compounds that can donate hydrogen atoms to other substances. Such gases were presumed to include methane (CH4) and ammonia (NH3). Oparin's and Haldane's ideas inspired the famous Miller-Urey experiment, which in 1953 began the era of experimental prebiotic chemistry. Harold C. Urey of the University of Chicago and Stanley L. Miller, a graduate student in Urey's laboratory, wondered about the kinds of reactions that might have occurred when the Earth was still enveloped in a reducing atmosphere. In a self-contained apparatus, Miller created such an "atmosphere." It consisted of methane, ammonia, water and hydrogen above an "ocean" of water. Then he subjected the gases to "lightning" in the form of a continuous electrical discharge. After a few days, he analyzed the contents of the mock ocean.

Miller found that as much as 10 percent of the carbon in the system was converted to a relatively small number of identifiable organic compounds, and up to 2 percent of the carbon went to making amino acids of the kinds that serve as constituents of proteins. This last discovery was particularly exciting because it suggested that the amino acids needed for the construction of proteins - and for life itself.

*Never Underestimate the Power of Soup,* Assuming that there is a way to RNA world, it very likely was the foundation for protein synthesis, DNA formation, and the emergence of the first cells.

#### http://superfood-spirulina.weebly.com/spirulem.html

Spirulina is 60-70% vegetable protein; it contains 12x digestable protein than beef. Spirulina is that it has the highest **GLA** (gamma-linolenic acid) next to mother's milk http://umm.edu/health/medical/altmed/supplement/spirulina

#### Folks,

We are no longer fish.

We are no longer amoeba.

We do however have one genetic trick that has not been silenced.

Amino Food (algae) will still **nourish our DNA directly**, bypassing everything, and needs no processing, 4 Billion years later.

Better than breathing with Gills.

http://blog.radiantlifecatalog.com/bid/59541/Chlorella-vs-Alga-Spirulina-which-algae-is-best

## **Chlorophyll**

Composing nearly 7% of the biomass of **chlorella**, the pigment chlorophyll is a powerful cleansing agent for the body. it aids in the processing of oxygen, helps to prime the key elimination system of the body and promotes the growth and repair of your tissues.

### Chlorella

Yaeyama Chlorella (2) 400mg Capsules Jarrow Formulas, Inc. 117003

https://www.swansonvitamins.com/jarrow-formulas-inc-yaeyama-chlorella-400-mg-150-caps http://www.jarrow.com/product/146/Yaeyama\_Chlorella

Detox, **GLA**, Amino mitochondrial nutrient

**Powerful Heavy Metal and Pesticide Detoxification- Chlorella** is a powerful detoxification aid, strongly binding to heavy metals such as cadmium, uranium, mercury and even radioactive materials to usher their prompt removal. Studies have indicated that it has the unique potential to remove toxins while simultaneously strengthening the immune system response.

**Ultra-Rich in Nucleic Aids- Chlorella** is extremely rich in nucleic acids, key factors for RNA and DNA that protect cells and raise energy levels.

**Bountiful in Beta Carotene-** This influential carotenoid acts as a powerful antioxidant to reduce the harmful effects of free radicals and minimize the damage generated from oxidative stress. A fat soluble provitamin.

**Improves Bowel Function-** Chlorella stimulates the growth of friendly aerobic bacteria, has a profound, beneficial effect on bowel health, chlorella's cell walls to absorb toxic compounds within the intestines, restoring proper gastrointestinal pH and helping to promote normal peristalsis. Chlorella:

Is a food-like all purpose mild chelator of heavy metals; it is a specially processed green-algae type of food that is taken with meals and is quite tolerable and pleasant for many. But since **chlorella** is so easily contaminated, the manufacturer's quality control is important. detox The detoxification capability of **Chlorella** is due to its unique cell wall and the material associated with it. The cell walls of **Chlorella** have been shown to have three layers of which the thicker middle layer contains cellulose microfibrils. Atkinson et all found a 14nm thick trilaminar layer outside the cell wall proper which was extremely resistant to breakage and thought to be composed of a polymerised carotene like material.....Laboratory studies showed that there were two active absorbing substances - sporopollenin (a naturally occurring carotene like polymer which is resistant to degradation) and the algae cell walls." **Chlorella**'s ability to detoxify the body is very significant because of the large amount of chemicals we are exposed to in today's modern world. This ability to detoxify chemicals is also one of the important differences between **Chlorella** and other "green" products."

## **Spirulina**

### **Source of Life Multivitamin Spirulina Tablets**

Source of Life — (4-8) Source Naturals with/without Iron Swanson NTP035 http://www.swansonvitamins.com/natures-plus-source-of-life-no-iron-multi-vitamin-180-tabs

Product ID 3058

#### **Spirulina Capsules**

## Spirulina Capsules, (4-30) 500mg, Swanson SWR030

http://www.swansonvitamins.com/swanson-greenfoods-formulas-spirulina-500-mg-360-tabs

### **Bulk Spirulina Powder**

## **Now Foods 1 LB Spirulina powder**

https://www.swansonvitamins.com/now-foods-spirulina-1-lb-pwdr

Spirulina is a close cousin of **Chlorella** and related to other robust sea vegetables including kelp, dulse, nori, arame and wakame. Spirulina however, is a unique blue-green algae, meaning it is not a *true* algae in biological terms but rather a cyanobacteria. This distinctive classification is given because its genetic material is not organized in a membrane-bound nucleus, yet it still uses the sun as a source of energy the way that plants do.

**Easily Digested**- Because the cell wall of spirulina is composed of mucopolysaccharides instead of indigestible cellulose, it is easily broken down by the human digestive system. Nutrients are thus highly bioavailable and become quickly assimilated into the biochemistry of the body.

**Rich in Complete Protein and B12**- Slightly higher in content than <a href="chlorella">chlorella</a> (which contains about 50% protein), Spirulina is 70% full protein by biomass. Often used as a supplement for those who consume poor quality or limited amounts of meat in their diet, Spirulina is also one of the few plant sources of essential vitamin B12. Because of it's highly digestible composition, the hardy protein found in this algae is high in net protein utilization and efficiency ratio, allowing all of the amino acids to be readily utilized without cooking.

**High in Fatty Gamma Linolenic Acid (GLA)-** Few foods contain this substance, an omega-6 fatty acid that plays a crucial role in brain function, reproductive health, growth and development, skin and hair growth, bone health, and metabolism regulation. While most commonly consumed in the form of linoleic acid and then converted into **GLA** within the body, spirulina contains the full form of this fatty acid allowing for efficient absorption and potent effect.

**Unique Phytochemical Phycocyanin-** This outstanding phytochemical is the pigment which gives spirulina it's blue hue. It is unique to spirulina and studies have found it to be useful to brain function, heart health, immune system strengthening, and in supporting bone marrow function for building blood cells. In mammals phycocyanin is converted into phycocyanorubin, an antioxidant that is helpful in protecting the tissues from free radicals.

### **ALA**

**(1-2-4) Alpha Lipoic Acid (ALA) 600 mg** Now Foods 3045B Capsules <a href="http://www.swansonvitamins.com/now-foods-alpha-lipoic-acid-600-mg-600-mg-60-vcaps">http://www.swansonvitamins.com/now-foods-alpha-lipoic-acid-600-mg-600-mg-60-vcaps</a> \$ 18.59

Swanson SWF282 SKU: 733739030450

**Swanson SWU167** 

https://www.swansonvitamins.com/swanson-ultra-alpha-lipoic-acid-600-mg-60-caps \$8.47

a Lipoic Acid: Enhances action of all other anti-oxidants, supplies sulfur, weak chelator. Three dosages with different actions: a) dose (50mg/d)-protects mitochondria and enhances ATP; b) dose (100-200mg/d)-potent antioxidant; c) dose (400-600 mg/d)- will open the brain barrier

**Lipoic acid.** Lipoic acid is known as the "recycler" antioxidant because it can restore the antioxidant properties of vitamins C and E after they have been neutralized by free radicals. It also stimulates the production of glutathione and helps in the absorption of CoQ10 (Balch PA et al 2000; Hendler SS et al 2001; Jamison JR 2003). The body produces this antioxidant (glutathione) in limited amounts.

ALA decelerates the loss of Vitamin s in the mitochondria

**ALA** helps maintain Long Term Memory

**ALA** is well known as a powerful antioxidant Command to RNA.Alpha Lipoic Acid is one of the most powerful antioxidants known to man. It is found in several foods, yeast and liver, kidney, spinach, broccoli and potatoes. It acts both intra- and extra-cellularly while fighting oxidative damage in general, and also helps maintain a normal blood sugar metabolism.

A breakthrough study on Anti-Aging was profiled in Readers Digest some years ago. Conducted by Dr Ames at the University of California at Berkeley, on an element know as L-Carnitine, it found that the most potent effect was arrived at by combining the powerful antioxidant and "free radicals-fighter" Alpha Lipoic Acid with Acetyl-L-Carnitine.

"Preliminary data suggests that Alpha Lipoic Acid can protect against damage to the brain or neural tissue as well. This is generally thought to be related to potent antioxidant properties, which reduce oxidative damage."

### ALC and ALA:

ALC is the acetyl ester of carnitine, an amino acid derivative, and is distributed throughout the central and peripheral nervous system. Both <u>ALA</u> and ALC are known as mitochondrial antioxidants that can provide anti-aging nutrition.\*

How does CoO10 + Alpha Lipoic Acid + Acetyl L-Carnitine HCl support health?

- All three nutrients help provide antioxidant protection against free radical damage.\*
- All three nutrients have been shown to promote cardiovascular and cognitive health.\*
- All three nutrients have been shown to be effective in maintaining blood pressure levels already within the normal range.\*

### ALA with C, A, CQ10:

ALA enhances the antioxidant activity of vitamins C and E and coenzyme Q10.

**ALC** is a precursor to the important brain neurotransmitter acetylcholine, which contributes to the support of cognitive function and memory.\*

**ALA** is a mitochondria amino acid, it works directly with the DNA in the cell.

#### Metabolic syndrome use ALA

- LA aka Lopoic Acid contains two <u>vicinal</u> sulfur atoms (at C6 and C8) attached by a <u>disulfide</u> <u>bond</u> and is thus considered to be oxidized (although either sulfur atom can exist in higher oxidation states).
- The carbon atom at C6 is <u>chiral</u> and the molecule exists as two <u>enantiomers</u> R-(+)-lipoic acid (RLA) and S-(-)-lipoic acid (SLA) and as a racemic mixture R/S-lipoic acid (R/S-LA). Only the R-(+)-enantiomer exists in nature and is an essential cofactor of four mitochondrial enzyme complexes.(alpha version) [4]

### ALA is the perfect antioxidant

Alpha lipoic acid is regarded by many as the "perfect" antioxidant. Scientists have known about alpha lipoic acid, a vitamin-like substance, since the 1930's when it was isolated from potatoes. Alpha lipoic acid is a very small molecule that is efficiently absorbed and easily crosses cell membranes, quenching either water- or fat-soluble free radicals both inside the cell and outside in the intracellular spaces, extending the biochemical life of vitamin C and E as well as other antioxidants.

- The "R" form is the biologically active component (native to the body) that is responsible for lipoic acid's phenomenal antioxidant effect.
- **ALA** directly reverses brain damage.
- Alpha lipoic acid is an approved drug in Germany, used for the treatment of diabetic neuropathy ( nerve disease ) for over 30 years. The value in diabetic neuropathy has been confirmed in several double-blind studies. In studies alpha-lipoic acid:
- Improves long term memory
- Prevents cataract formation
- Workes in aerobatic chemistries involved in breaking down lipoids into simplier acids, it aids the enzyme system in: Digesting DNA and cellular processes Phase 1.
- The release free R-lipoic acid and either L-lysine or <u>ammonia</u> into the bloodstream. By adeing
  the digestive anti-oxidant like chemistries, it works in oil and mylin production which makes tissue from fatty tissues and membranes.\* distributed throughout the central and peripheral nervous system

#### **ALA Studies**

- It has been the subject of many studies including Prevent organ dysfunction [36]
- Reduce endothelial dysfunction and improve albuminuria [37][38]
- Treat or prevent cardiovascular disease<sup>[39]</sup>
- Accelerate chronic wound healing [40]
- Reduce levels of ADMA in diabetic end-stage renal disease patients on hemodialysis<sup>[41]</sup>
- Management of burning mouth syndrome<sup>[42][43][44]</sup>
- Treat metabolic syndrome [46][47][48]
- Improve or prevent age-related cognitive dysfunction [49][50]
- Prevent or slow the progression of Alzheimer's Disease [51][52][53]
- Prevent erectile dysfunction (animal models but anecdotally applies to humans as well)<sup>[54]</sup>
- Prevent migraines<sup>[56]</sup>
- Treat multiple sclerosis<sup>[57][58][59]</sup>
- Treat chronic diseases associated with oxidative stress<sup>[60]</sup>
- Reduce inflammation<sup>[61]</sup>
- Inhibit advanced glycation end products (AGE)<sup>[62]</sup>
- Treat peripheral artery disease. [63]

#### **Antioxidant stress Cocktail**

This suggests **antioxidants** may be beneficial in management of specific DNA stress. **Glutathione** (GSH), N-acetylcysteine (NAC), a-lipoic acid, oligomeric proanthocyanidins (OPCs), **Ginkgo** biloba, and Vaccinium myrtillus (bilberry) would therefore be dietary supplements with potential therapeutic benefit. Oxidative stress can be caused by an increase in the generation of reactive oxygen species, of which mitochondrial dysfunction. Mitochondrial dysfunction is believed to be a main source, or it can be caused by a decline in the efficiency of antioxidant enzyme systems. elevated peroxynitrite causes mitochondrial dysfunction, lipid peroxidation, and, by way of positive feedback, elevated cytokine levels. The cytokines, in turn, cause the formation of nitric oxide that combines with superoxide to form the potent oxidant peroxynitrite, thus continuing the cycle. Peroxynitrite targets the mitochondria and Pall notes this may help explain mitochondrial dysfunction. As support for the peroxynitrite theory, Pall cites evidence that the mitochondrial enzymes succinic dehydrogenase and cis-aconitase are inactivated by peroxynitrite.22,23 This makes for an interesting finding because decreased succinic dehydrogenase activity has been found.

Pall proposes a number of nutritional and botanical interventions that may reduce peroxynitrite and cytokine levels; among them, the soy isoflavone genistein, **epigallocatechin-3-gallate** from **green tea, and vitamins C and E**. Keenoy et al found impaired antioxidant capacity in a sample of patients with "subclinical" or moderate **magnesium** deficiency.

28 The impaired capacity involved both the total antioxidative capacity of plasma, as measured by Trolox Equivalents Antioxidant Capacity (TEAC), and the antioxidant component dependent on albumin. While no improvement was observed in these parameters after oral or intravenous **magnesium** supplementation, some patients demonstrated increased serum **vitamin E** and an associated decrease in lipid peroxidation. This finding, according to the authors, is likely due to the sparing effect of **magnesium on vitamin E** by preventing its *in vivo* oxidation. In addition, the researchers postulated that an elevated concentration of inflammatory cytokines might indirectly cause diminished antioxidant capacity by inhibiting albumin transcription in the liver.

The above findings on oxidative stress suggest that supplementing with certain antioxidants, in addition to <u>vitamins C and E</u>, may be valuable in a treatment protocol (Table 1). A number of supplements should be considered for potential therapeutic intervention, including selenium (necessary to support <u>glutathione</u> peroxidase activity),34 <u>GSH</u>, <u>NAC</u>, and α-lipoic acid. Although there is conflicting evidence, a number of studies have shown oral administration of GSH can directly increase plasma and tissue <u>GSH</u> concentration.35- 37 Alternately, <u>NAC</u> and <u>α-lipoic acid (ALA)</u> can increase <u>GSH</u> concentration <u>indirectly</u>;38,39 <u>NAC</u> provides cysteine for <u>GSH</u> synthesis, and <u>α-lipoic acid (ALA)</u> is believed to increase intracellular <u>GSH</u> levels by reducing extracellular cystine to cysteine, bypassing the cystine transporter.40

**GSH** is neuroprotective and may play a role in preventing additional CNS lesions.41 **a-Lipoic acid** is also neuroprotective, scavenges nitric oxide and peroxynitrite, and may be especially promising as an antioxidant against mitochondrial dysfunction.40 The supplement **coenzyme Q10** has similar neuroprotective qualities and has the ability to improve mitochondrial function.42

The botanical antioxidants OPCs and <u>Ginkgo biloba</u> should also be considered. Bagchi et al found that OPCs are highly bioavailable and provide significantly greater protection against free radical damage than <u>beta carotene and vitamins C and E</u>.43 These authors also reported the ability of OPCs to provide protection from radical-induced lipid peroxidation and DNA damage, which is of particular importance.

Antioxidant	Mechanism of Action	
Selenium	Supports glutathione peroxidase activity, a Se- dependant antioxidant system <sup>34</sup>	
Glutathione	Reduced glutathione (GSH) directly increases glutathione levels <sup>35-37</sup>	
N-acetylcysteine	Provides cysteine for GSH synthesis <sup>38</sup>	
α-Lipoic acid	Increases intracellular GSH by reducing extracellula cystine to cysteine <sup>40</sup>	
CoQ10	Improves mitochondrial function; neuroprotective <sup>42</sup>	
Oligomeric proanthocyanidins	Protects against radical-induced lipid peroxidation and DNA damage <sup>43</sup>	
Ginkgo biloba	Powerful antioxidant; increases cerebral perfusion and associated memory and cognitive deficits; neuroprotective <sup>45-47</sup>	
Vaccinium myrtillus (bilberry)	Neuroprotective; <sup>52</sup> protects RBCs from in vivo oxidative damage <sup>53</sup>	

### Alternative Medicine Review Volume 6, Number 5 2001

As an antioxidant and anti age stress chemistry player it: The antioxidant effects of lipoic acid (LA) were demonstrated when it was found to prevent the symptoms of vitamin C and vitamin E deficiency. [93] LA is reduced intracellularly to dihydrolipoic acid, which in cell culture regenerates by reduction of antioxidant radicals, such as vitamin C and vitamin E.[90] LA is able to scavenge reactive oxygen and reactive nitrogen species in vitro due to long incubation times, but there is little evidence this occurs in vivo or that radical scavenging contributes to the primary mechanisms of action of LA.[31][2] The relatively good scavenging activity of LA toward hypochlorous acid (a bactericidal produced by neutrophils that may produce inflammation and tissue damage) is due to the strained conformation of the 5-membered dithiolane ring, which is lost upon reduction to DHLA. In cells, LA is reduced to dihydrolipoic acid, which is generally regarded as the more bioactive form of LA and the form responsible for most of the antioxidant effects. [94] This theory has been challenged due to the high level of reactivity of the two free sulfhydryls, low intracellular concentrations of DHLA as well as the rapid methylation of one or both sulfhydryls, rapid side chain oxidation to shorter metabolites and rapid efflux from the cell. Although both DHLA and LA have been found inside cells after administration, most intracellular DHLA probably exists as mixed disulfides with various cysteine residues from cytosolic and mitochondrial proteins. [95] Recent findings suggest therapeutic and anti-aging effects are due to modulation of signal transduction and gene transcription, which improve the antioxidant status of the cell. Paradoxically, this likely occurs via pro-oxidant mechanisms, not by radical scavenging or reducing effects. [31][2][77]

Protects the liver

### L-Carnitine

L-Carnitine (1-5) Swanson 500mg capsule SW1000

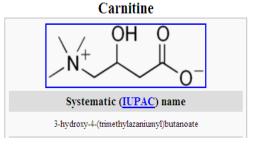
### Acetyl L-Carnitine (ALC) 500 mg Capsule GNC 37631, Now Foods

http://www.swansonvitamins.com/swanson-premium-acetyl-l-carnitine-500-mg-240-veg-caps? otherSize=SW1649

(0-2 Grams) W ALA and CQ10 to dump Toxins from DNA

Builds amino acids lysine and methionine
(3) <u>L-Carnitine</u>: GNC 37631 or NOW Foods work equally as well

<u>Jarrow Formulas' CarnitAll 600 contains all 4 forms of Carnitine.</u>



- L-Carnitine is an amino acid which nourishes the heart, nourishes and strengthens muscles, and nutritionally supports the circulatory system. L-Carnitine is considered to be a "carrier" of fat to the mitochondria or "fatburning" area of the cell. This remarkable amino acid-like substance is not only necessary for the metabolism of fat at the cellular level; it is also essential in the forming of firm, lean muscle tissue in the body. Recent studies support earlier research which shows that the heart has the greatest amount of L-Carnitine of any muscle in the body. L-Carnitine has also shown to be instrumental in the metabolism of cholesterol. Some overweight people may lack L-Carnitine in their bodies. The heart produces most of its energy from fats; thus is dependent upon L-carnitine. An L-Carnitine deficiency causes extreme metabolic impairment to heart tissue. On the other hand, supplemental L-Carnitine has proved to be beneficial to heart patients.
- Carnitine is a <u>quaternary ammonium compound</u> biosynthesized from the <u>amino acids</u> <u>lysine</u> and <u>methionine.[1]</u> In living cells, it is required for the transport of <u>fatty acids</u> from the <u>cytosol</u> into the <u>mitochondria</u> during the breakdown of <u>lipids</u> (fats) for the generation of metabolic energy. It is widely available as a <u>nutritional supplement</u>. Carnitine was originally found as a <u>growth factor</u> for <u>mealworms</u> and labeled <u>vitamin Bt</u>. Carnitine exists in two <u>stereoisomers</u>: Its biologically active form is L-carnitine, whereas its <u>enantiomer</u>, <u>D-carnitine</u>, is biologically inactive.[2]

School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, M.

### Friday, 27 June 2014

## Revision: Lipid Metabolism 1 July 2014

#### INTRODUCTION

LIPIDS topics are covered by 2 lecturers - Dr Win Mar Kyi (WMK) and myself (FAR). The topics have to fit into the number of slots allocated by the Phase I Committee, based on the Objectives submitted by the Department at Curriculum Reviews in the past. I only attended one Curriculum Review.

Lipid Structure & Function (FAR)

Lipid Degradation (Fat Mobilisation); covered under Lipid Metabolism (FAR)

Ketogenesis (WMK)

Fatty Acid Synthesis; covered under Lipid Metabolism (FAR)

Triglyceride & Phospholipid Biosynthesis; covered under Lipid Metabolism (FAR)

Cholesterol (Steroid) Metabolism (FAR)

Lipoprotein Metabolism (FAR)

Integration of Metabolism

Lipid Metabolism is covered in 2 blocks in Phase 1 - Foundation and Endocrine,

In Foundation Black, I covered one topic: Structure and Function of Lipids (1 Oct 2013).

In Endocrine Block, I covered 4 topics:

- 1. Lipid Metabolism (30 April 2014)
- 2. Regulation of Lipid Metabolism (30 April 2014)
- 3. Lipoprotein Metabolism (4 May 2014)
- 4. Cholesterol Metabolism (4 May 2014)

From an applied standpoint, lipids are important as pesticides are fat-soluble and can accumulate if fat tissues of the body (referred to as biological magnification). http://www.rsc.org/education/eic/issues/2007May/PesticidesKeepingOneStepAhead.asp

The vast majority of people with CFIDS have low levels of acetylcarnitine, although their levels of free carnitine are normal. Carnitine, vital for the conversion of fats into energy, also plays some role in detoxification and is believed to be essential for heart function. Finally, carnitine helps transport long-chain fatty acids into the mitochondria. Carnitine deficiencies result in muscle weakness, aches, and poor tone. When people were

supplemented with 4 grams daily of <u>acetylcarnitine</u>, half showed improvement in their symptoms. Dosage: 4 grams <u>acetyl-carnitine daily.</u>

- Acetyl L-Carnitine (ALC). ALC is the acetyl ester of <u>Carnitine</u>, an amino acid derivative, i.e. it supplements RNA processes of amino acid uses in the core of the cell, and will be consumed especially when <u>GLA</u> is on.
- Core of the Cell Nerves: Distributed throughout the central and peripheral nervous system
- The amino acid Acetyl-L-Carnitine boosts mitochondrial energy production through its ability to facilitate fatty acid transport and oxidation in the cell. Since 1995, Life Extension members have been supplementing with Acetyl-L-Carnitine and deriving the many benefits this form of Carnitine has shown in published studies. With the discovery of Acetyl-L-Carnitine Arginate the benefits of Acetyl-L-Carnitine can now be greatly augmented. Acetyl-L-Carnitine Arginate is a patented form of carnitine that stimulates the growth of neurites in the brain. Studies show that acetyl-l-carnitine-arginate stimulates the growth of new neurites by an astounding 19.5% (as much as Nerve Growth Factor itself). Acetyl-l-carnitine-arginate acts together with acetyl-l-carnitine to increase neurite outgrowth. Mitochondrial antioxidants that can provide anti-aging nutrition associated with RNA Waste products, production in the cell when it is turned full on with ALA and supported all phases with CQ10 turn cell full on, Go to the highest volume level Cellular process connections from core to outer process loops, support ion phosephate chemistry out the door, from the cell to waste, turn on antioxidant removal of waste process, than feed the need good.
- <u>Acetyl L-Carnitine</u> (ALC). <u>ALC</u> is the acetyl ester of <u>Carnitine</u>, an amino acid derivative, and is distributed throughout the central and peripheral nervous system. Both <u>ALA</u> and <u>ALC</u> are known as mitochondrial antioxidants that can provide anti-aging nutrition.\*
- Early evidence suggests that <u>L-carnitine</u> may effectively treat cirrhosis, further research is needed to confirm these results. Preliminary evidence also suggests that <u>L-carnitine</u> may be of benefit to individuals with hepatic encephalopathy, in terms of <u>ammonia</u> levels and psychometric functioning. Use: An active inhibitor of intestinal protozoa. Used to prevent and alleviate amoebic dysentery and giardia. Also good for use after antibiotic therapy and for lingering gastroenteritis. Do not use if pregnant.

Having low levels of <u>acety L Carnitine</u> (ALC), although their levels of free <u>carnitine</u> are normal. <u>Carnitine</u>, vital for the conversion of fats into energy, also plays some role in detoxification and is believed to be essential for heart function. Finally, <u>carnitine</u> helps transport long-chain fatty acids into the mitochondria. <u>Carnitine</u> deficiencies result in muscle weakness, aches, and poor tone. When people were supplemented with 4 grams daily of acetylcarnitine, half showed improvement in their symptoms.

Dosage maximum: 4 grams acetyl-carnitine daily.

In animals, carnitine is biosynthesized primarily in the liver and kidneys from the amino acids lysine (via trimethyllysine) or methionine.[3] Vitamin C (ascorbic acid) is essential to the synthesis of carnitine. During growth[4] or pregnancy,[5] the requirement of carnitine might exceed its natural production.

## Role in fatty acid metabolism

Carnitine transports long-chain acyl groups from fatty acids into the <u>mitochondrial matrix</u>, so they can be broken down through  $\beta$ -oxidation to Acetyl CoA to obtain usable energy via the <u>citric acid cycle</u>. In some organisms such as fungi, the acetate is used in the <u>glyoxylate cycle</u> for <u>gluconeogenesis</u> and formation of <u>carbohydrates</u>. Fatty acids must be activated before binding to the carnitine molecule to form *acylcarnitine*. The free fatty acid in the cytosol is attached with a <u>thioester</u> bond to <u>coenzyme A</u> (CoA). This reaction is catalyzed by the enzyme <u>fatty acyl-CoA synthetase</u> and driven to completion by <u>inorganic pyrophosphatase</u>.

The acyl group on CoA can now be transferred to carnitine and the resulting acylcarnitine transported into the <u>mitochondrial matrix</u>. This occurs via a series of similar steps:

- 1. Acyl CoA is conjugated to carnitine by <u>carnitine acyltransferase I</u> (palmitoyltransferase) located on the outer mitochondrial membrane
- 2. Acylcarnitine is shuttled inside by a <u>carnitine-acylcarnitine translocase</u>
- 3. Acylcarnitine is converted to acyl CoA by <u>carnitine acyltransferase II</u> (palmitoyltransferase) located on the inner mitochondrial membrane. The liberated carnitine returns to the cytosol.

Human genetic disorders such as <u>primary carnitine deficiency</u>, <u>carnitine palmitoyltransferase I deficiency</u>, <u>carnitine palmitoyltransferase II deficiency</u> and <u>carnitine-acylcarnitine translocase deficiency</u> affect different steps of this process.[6]

Carnitine acyltransferase I undergoes <u>allosteric</u> inhibition as a result of <u>malonyl-CoA</u>, an intermediate in fatty acid biosynthesis, to prevent futile cycling between  $\beta$ -oxidation and <u>fatty acid synthesis</u>.

Physiological effects

### **Effects on bone mass**

In the course of human aging, carnitine concentration in cells diminishes, affecting fatty acid metabolism in various tissues. Particularly adversely affected are bones, which require continuous reconstructive and metabolic functions of osteoblasts for maintenance of bone mass. There is a close correlation between changes in plasma levels of <u>osteocalcin</u> and <u>osteoblast</u> activity and a reduction in osteocalcin plasma levels is an indicator of reduced osteoblast activity,[7] which appears to underlie <u>osteoporosis</u> in elderly subjects and in postmenopausal women. Administration of a carnitine mixture or <u>propionyl-L-carnitine</u> is capable of increasing serum osteocalcin concentrations of animals thus treated, whereas serum osteocalcin levels tend to decrease with age in control animals.[8]

### **Antioxidant effects**

The carnitines exert a substantial <u>antioxidant</u> action, thereby providing a protective effect against <u>lipid peroxidation</u> of phospholipid membranes and against <u>oxidative stress</u> induced at the myocardial and endothelial cell level.[9]

Potential uses as a pharmaceutical

#### **Heart conditions**

Carnitine is primarily used for heart-related conditions. Several clinical trials show that L-carnitine and propionyl-L-carnitine can be used along with conventional treatment for angina to reduce medication needs and improve the ability of those with angina to exercise without chest pain. [10][11] There is little evidence about a positive effect of the use of carnitine after a heart attack. Some studies suggest that people taking L-carnitine may be less likely to suffer a subsequent heart attack

or experience chest pain and abnormal heart rhythms.[12] However, other studies have not found similar benefits.[13] Further research on this subject is needed.

## Kidney disease and dialysis

Because kidneys produce carnitine, kidney disease may lead to the deficiency of carnitine in the body. Thus, carnitine may be prescribed to those with kidney disease.[14]

## **Effect in male infertility**

The use of carnitine showed some promise in a controlled trial in selected cases of <u>male infertility</u> by improving sperm quality.[15] L-carnitine supplementation has also shown to have beneficial effects in the treatment of <u>varicocele</u>, a major cause of male infertility. [16]

## As a weight loss supplement

"Although L-carnitine has been marketed as a weight-loss supplement, there is no scientific evidence to show that it improves weight loss; however, some studies show that oral carnitine reduces fat mass, increases muscle mass, and reduces fatigue. All of these effects may contribute to weight loss." [17] Furthermore, whereas researchers in the 20th century failed to show that muscle carnitine content could be increased by dietary supplementation, this may have been in part due to inadequate lengths of the supplementation periods. [18] In 2011, researchers using L-carnitine L-tartrate supplementation for 6 months in a well controlled study demonstrated not only increased muscle carnitine in subjects without carnitine deficiencies, but also an impact on muscle metabolism and performance; however, measurements of lipid oxidation were not taken in this study, and further research is needed. [19]
Regular supplements of L-carnitine, however, contribute to energy metabolism and improved neurotransmitter function in the brain in elderly patients. [20]

## As an antidote in valproic acid poisoning

"[In the treatment of valproate toxicity] L-carnitine supplementation ...is thought to provide benefit, particularly in patients with concomitant hyperammonemia, encephalopathy, and/or hepatotoxicity."[21] Further trials are warranted, as benefit is largely theoretical, rather than proven at this stage.

### **Sources**

#### **Food**

#### **Vitamin A foods**

The highest concentrations of carnitine are found in <u>red meat</u> and dairy products. Other natural sources of carnitine include <u>nuts</u> and <u>seeds</u> (e.g. <u>pumpkin</u>, <u>sunflower</u>, <u>sesame</u>), <u>legumes</u> or <u>pulses</u> (<u>beans</u>, <u>peas</u>, <u>lentils</u>, <u>peanuts</u>), <u>vegetables</u> (<u>artichokes</u>, <u>asparagus</u>, <u>beet greens</u>, <u>broccoli</u>, <u>brussels sprouts</u>, <u>collard greens</u>, <u>garlic</u>, <u>mustard greens</u>, <u>okra</u>, <u>parsley</u>, <u>kale</u>), <u>fruits</u> (<u>apricots</u>, <u>bananas</u>), <u>cereals</u> (<u>buckwheat</u>, <u>corn</u>, <u>millet</u>, <u>oatmeal</u>, rice <u>bran</u>, rye, <u>whole wheat</u>, wheat <u>bran</u>, <u>wheat germ</u>) and other "health" foods (<u>bee pollen</u>, <u>brewer's yeast</u>, <u>carob</u>). [citation needed]

Product	Quantity	Carnitine
Beef steak	100 g	95 mg
Ground beef	100 g	94 mg

100 g	27.7 mg
100 g	23.3 mg
100 g	19.5 mg
100 g	5.6 mg
100 g	3.9 mg
100 g	3.7 mg
100 ml	3.7 mg
100 ml	3.3 mg
one medium	2 mg[22]
100 g	1.1 mg
100 g	0.36 mg
100 g	0.195 mg
100 g	0.147 mg
100 g	0.126 mg
100 g	0.083 mg
100 g	0.0449 mg
100 g	0.0121 mg
100 ml	0.0019 mg
	100 g 100 g 100 g 100 g 100 ml 100 ml 100 ml one medium 100 g 100 g 100 g 100 g 100 g 100 g 100 g 100 g 100 g

In general, 20 to 200 mg are ingested per day by those on an <u>omnivorous</u> diet, whereas those on a strict <u>vegetarian</u> or <u>vegan</u> diet may ingest as little as 1 mg/day. [citation needed] No advantage appears to exist in giving an oral dose greater than 2 g at one time, since <u>absorption</u> studies indicate <u>saturation</u> at this dose. [23]

## Minerals and Compounds

The regulation of mineral balance in the body is essential to survival. Like the body itself, each cell is a living organism and must maintain its internal environment. And it must interact with its surroundings in order to perform the functions assigned to it. The movement of minerals across cell membranes, between the extracellular and intracellular fluid, forms the basis for the body's most primary functions. Electrical activity is initiated; hearts beat, nerve cells signal. Muscles respond. Blood vessels tighten or relax. Water balance is maintained. important minerals include calcium, phosphorous, sodium and **potassium**, while trace minerals like **iodine**, **iron** and **zinc** appear in smaller quantities in the body. Each of these minerals has had documented benefits. For example, **potassium** works in conjunction with sodium to regulate the water balance in the body, and has been proven effective in reducing blood pressure. **Zinc** has proven to be effective in helping proper brain function and ensuring mental alertness. Some of the minerals also act like antioxidants, fighting off those dangerous free radicals that threaten cardiovascular health through oxidation. **Selenium** has been shown to be particularly effective in this regard. Each mineral performs a vital function, and a mineral supplement will guarantee that you are receiving all of their health benefits.

Mineral supplements should be part of a complete solution that includes vitamins as well. Research has shown that certain vitamins and minerals work well together. For example, the minerals **calcium**, **magnesium**, phosphorous, **selenium and zinc** have been shown to enhance the effectiveness of **vitamin A** in the body. **Calcium**, cobalt, **copper**, **iron** and sodium have been shown to help **vitamin D**. These are just a few examples. A total solution of vitamins and minerals such as those found in a vitamin and mineral supplement should therefore be part of your regimen, rather than taking a mineral supplement alone.

In theory, an individual can gain the necessary supply of minerals through diet alone, however this is no longer as feasible as it used to be. Modern farming methods have bleached a significant amount of minerals from fruits and vegetables, making it necessary for individuals to take mineral supplements to compensate for the loss.

### **Calcium**

<u>Calcium salts</u> are calcium in an inorganic form that bound to chemical salts. Since these forms are inorganic the body does not readily absorb these salts and utilize them efficiently.

Their low cost makes them a bad, and common choice for formulating products. These minerals are often too large to pass through the intestinal walls intact so they must be restructured for absorption. These forms of calcium are responsible for the mass publication that calcium must be formulated with a specific ratio of **magnesium** for absorption.

### The most natural non-ionic form of calcium is yogurt, milk, cream, butter, cheese.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3635625/

Yonsei Med J. 2013 May 1; 54(3): 626-636.

Published online 2013 Mar 19. doi: 10.3349/ymj.2013.54.3.626

PMCID: PMC3635625

## Relationship between Milk and Calcium Intake

and Lipid Metabolism in Female Patients with Type 2 Diabetes

JaeHee Kim,<sup>1,\*</sup> Ji-Yun Hwang,<sup>2,\*</sup> Ki Nam Kim,<sup>1</sup> Young-Ju Choi,<sup>3</sup> Namsoo Chang,<sup>1</sup> and Kap-Bum Huh<sup>3</sup>

<u>Author information</u> ► <u>Article notes</u> ► <u>Copyright and License information</u> ►

#### **Abstract**

#### **Purpose**

This study was conducted to determine the association between intake of milk and dairy products as well as calcium and biomarkers related to lipid metabolism in Korean female patients with type 2 diabetes.

### **Materials and Methods**

A cohort of 509 female subjects (mean age: 59.0 years; range: 35-80 years) was recruited from Huh's Diabetes Clinic in Seoul between 2005 and 2010. Dietary intake was assessed using a validated food-frequency questionnaire. Subjects were divided into three groups on the basis of their daily intake of milk and dairy products [<50 g/day (0<50 g/day), 50-200 g/day, and >200 g/day (>200-1201 g/day)] and then further divided into two groups according to their daily calcium intake: below and above the estimated average requirement (EAR).

#### **Results**

After adjustment for age, body mass index, energy intake, exercise, use of nutritional supplements and cholesterol medication, the level of serum high-density lipoprotein (HDL)-cholesterol was significantly higher in subjects with milk and dairy products consumption of >200 g/day than in subjects in the other two groups. Those subjects with a milk and dairy products consumption of >200 g/day had significantly higher levels of apolipoprotein A-1 and a

significantly lower atherogenic index than the other two groups. Patients with a calcium intake above the EAR exhibited a significantly greater serum HDL-cholesterol level than those with a calcium intake below the EAR.

#### Conclusion

Milk and dairy products, good sources of calcium, play a positive role in lipid profiles in female patients with type 2 diabetes.

Amino acid chelated calcium is a calcium molecule bound by two amino acids making this form of calcium organic just as minerals are found in plants. This form of calcium does NOT need to be restructured for absorption does NOT need to be formulated with <a href="Magnesium">Magnesium</a> for absorption. The patented technology that is used to produce amino acid chelated minerals used in OHS's products creates molecules with molecular weight small enough to pass easily through the intestinal wall. This patented chelation process also guarantees stability, purity, neutrality (no electrical charge).

Optimal Calcium only contains calcium in the form of amino acid chelate, an organic form of calcium. You will notice that OHS has also included amino acid chelated <a href="magnesium">magnesium</a> in the formulation. It was added to this product for two reasons: (1) growing evidence shows that <a href="magnesium">magnesium</a> is just as important as calcium in the role of preventing and treating osteoporosis in fact, 60% of the body's <a href="magnesium">magnesium</a> is stored in bone - and (2) the overall benefits of supplementing with <a href="magnesium">magnesium</a>, including energy production, muscle relaxation, prevention of tooth decay, and helps to prevent atherosclerosis.

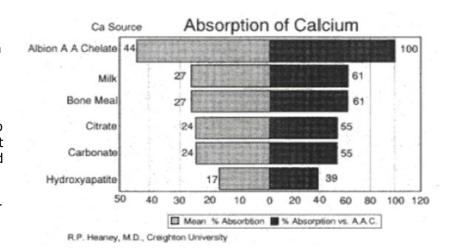
3. Is the patented amino acid chelated form of calcium safe to take long term?

Safety patented amino acid chelated calcium has demonstrated long and short term safety. Other forms of calcium, such as calcium carbonate, calcium chloride, and other non-chelated calcium salts have created symptoms that include constipation, diarrhea, and gastrointestinal irritation. Continued use of calcium carbonate has also been associated with milk-alkali syndrome (hypercalcemia, metabolic alkalosis, and renal failure).

4. What form of calcium has the highest absorption rate and best tissue retention?

As seen the graph below, amino acid chelated calcium has the highest absorption rate. Its bioavailability is even higher than milk.

Bioavailability refers to the amount of nutrient ingested and absorbed into the body to be used by the individual cells. It doesn't matter how much of a nutrient is in a supplement, if the



nutrients are not made available for the body to use then you are wasting your money.

### **Activated charcoal**

# (2-4) PPM activated charcoal heavy metal detox Solaray/GNC Solaray/GNC, 4+ initial CAPSULES EVENING

Avoid dosing charcoal when you take other substances, the charcoal will absorb them, take charcoal at least 2 hours before or after other dosing.

The third thing that parasites do is secrete toxins. Simply put, the secretions from parasites in our bodies are poisons and toxins that our bodies are forced to deal with by increasing the process of detoxification. Anyone who has experienced food poisoning or dysentery will tell you how debilitating these toxins can be. These are intense and very high levels of toxins being released into the body at once. On the other end, a chronic parasitic infection that secretes low levels of toxins can eventually create an extremely stressed immune system. When the immune system is stressed over a long period of time, it weakens. When the immune system "goes off line," our bodies become susceptible to infections of other kinds. This can be extremely dangerous in this day and age because we are more exposed to more viruses than ever before. Also, the viruses are changing and adapting at a very fast rate as are the bacteria, many of which are now resistant to antibiotics and other artificial measures that are used to combat them. Detoxing these volatile alcohol based molecules is essential for health recovery.

<u>Activated Charcoal</u> powder is used medicinally as well as in air purifiers and <u>water purifiers</u>. Its recorded use dates back to 1550 BC and in modern times is most widely used medicinally as a detoxifier and poison antidote. I always keep it on hand for spider bites, accidental ingestion of toxins or stomach bugs.

<u>Activated charcoal</u> is well known as a antidote as it adsorbs most organic toxins, chemicals and poisons before they can harm the body. Some Emergency Rooms administer large doses of activated charcoal for certain types of poisoning.

"It was 1831. In front of his distinguished colleagues at the French Academy of Medicine, Professor Touery drank a lethal dose of strychnine and lived to tell the tale. He had combined the deadly poison with <u>activated charcoal</u>.

**Charcoal** is not known to be toxic, though it should not be taken within two hours of vitamins or medications because it will keep the body from adsorbing them.

<u>Activated Charcoal</u> will chelate Aluminum, Arsenic, Cadmium, Chromium, Copper, Iron, Manganese, Platinum, Tin, Thorium, Tungsten. It **will NOT** for Beryllium, Cobalt, Nickel, some Lead, Rubidium, Silver, Uranium and Mercury.

### **Magnesium CITRATE**

Magnesium CITRATE - (1-2) AM 133mg Source Naturals Product#02100 Swanson SN536

http://www.swansonvitamins.com/source-naturals-magnesium-citrate-133-mg-180-caps

Magnesium helps with the absorption of calcium, phosphorus, sodium, potassium, B complex, C and F

Magnesium, however, is a different matter. It plays an active role in the metabolism of sodium, potassium, and calcium. It acts on your heart and blood vessels, your nerves and muscles, and your gut. Most of it is concentrated in tissue, so levels in the blood don't tell us much.

### **The Best Brain Supplement**

The two most common forms of magnesium are Citrate and Sulfate. These are primarily not used for the brain but for cleansing the body of toxins, and also for digestion.

- -Within our brain there is a fine balance of sodium and potassium. Magnesium activates an enzyme that serves to balance the levels of those key chemicals.
- One of the toxins which magnesium takes care of is called ammonia
- Magnesium helps to activate "glutamine synthetase this substance converts ammonia into urea
- Other enzymes magnesium activates serve your body by improving the effectiveness of the energy glucose gives us
- which indirectly improves the amount of energy which can be used by the brain

Kidneys spill magnesium. Losses also occur in the stool and through the skin. Malabsorption can reduce its availability from the diet. High alcohol use reduces it. Diuretics and some antibiotics deplete it.

Levels are often low in states of severe infection, and often then rise in that setting, suggesting that tissue damage and cell death cause its release. Then it's carried out of the body and lost. Thus illness can deplete you rapidly; and your doctor may not know it, even if she or he tests you. Calcium deficiency results from magnesium depletion, and may not respond to treatment without magnesium supplementation.

Unlike calcium, there are no reserves in bone to draw on if magnesium supplies get low. But also unlike calcium, you cannot get too much.

Recent studies show that magnesium supplementation can reduce lung injury from oxygen toxicity. It blocks blood vessel constriction, so it can augment blood flow. It has been shown to increase the speed of recovery from open heart surgery, and to improve the likelihood of recovery from severe, life-threatening infection.

Since it can be hard to get magnesium in and easy to lose it, since it's so important, since you can't get too much, and since too much can't hurt you, it makes sense to supplement.

The only time you shouldn't supplement your magnesium is in the case of kidney failure. If you can't excrete it in the urine, it can build up. Otherwise, you're safe.

### **Magnesium Sulfate**

Magnesium Sulfate (1-2) AM 1030mg CAPSULE ( Epsom Salt USP) Dr Hula Clark cap 100 ct 10552-100

http://www.drclarkstore.com/magnesium-sulfate-usp-epsom-salt-1030-mg-100-capsules/

I heard of a PHD at MIT and struck up a dialog with her. I watched her videos, and listened to her reasoning of how the body works. She theorized that Nitrogen loop stress depleted Sulfur, When Nitrogen stores were depleted, the body and DNA would kludge in Carbon atoms. The body is designed to survive. I tested the theory with a combination of Nitrogen and Sulfur Amino Acids, and found she was right. Sulfur must dominate any plan to dominate the parasite, and Nitrogen loop performance can only be achieved through the use of **Piperazine**. The actual amino acids cannot be used, you feel better for a week, then slide back as the toxins pile up.

<u>Piperazine</u> removes the parasite compounds from the <u>Ammonia</u> and Urea, allowing them to pass the kidneys. Magnesium Sulfate supplies the primary detox agent for the liver, and the primary sulfur source for dominating the nitrogen loop chemistries in the body.

Sulfur must dominate Nitrogen, parasites inject so many nitrogen process stresses, the body quickly depletes **ROS** elements of **Sulfur**, **Selenium**, Zinc, and **copper**. Magnesium Sulfate keeps the liver flushed daily, establishes and maintains sulfur dominance, and provides an available source of Magnesium. The Magnesium channel in the body has a maximum capacity of 700 mg per day. 50% of the magnesium you take, or virtually anything you take orally, only enters the body with an efficiency of 50%. So a total maximum magnesium supply is established with one or two **magnesium citrate** 133mg, and a single 1030mg **magnesium sulfate** capsule, (266 +1030mg) =1299/2 = 650 mg of magnesium in the form of citrate and sulfate. This leaves 50 mg for other forms, and a little more margin if only one magnesium citrate capsule is taken. After the maximum is achieved, the channel is maxed out, and body chemistry could be degraded. Dose magnesium as stated, try not to deviate unless the body PH goes very acid. Magnesium citrate is the fastest acting, and highly absorbed, and works in the citrate cycle to maintain balance.

Magnesium Sulfate used for liver cleanse

Chemical name: Magnesium Sulfate (Heptahydrate) or (Hydrated)

Chemical Formula: MgSO4 + 7H2O, Hydrated Magnesium Sulfate

Mineral: EPSOMITE (MgSO4 + 7H2O)

Other minerals: KIESERITE (MgSO4 + H2O, Hydrated Magnesium Sulfate)

Hexahydrite (MgSO4 + 6H2O)

#### **MAGNESIUM SULFATE**

Description

http://eilat.sci.brooklyn.cuny.edu/newnyc/drugs/magnesiu.htm

Magnesium sulfate reduces striated muscle contractions and blocks peripheral neuromuscular transmission by reducing acetylcholine release at the myoneural junction. In emergency care, magnesium sulfate is used to manage seizures associated with toxemia of pregnancy. Other uses include uterine relaxation (to inhibit contractions of premature labor), as a bronchodilator after beta-agonist and anticholinergic agents have been used, replacement therapy for magnesium deficiency, as a cathartic to reduce the absorption of poisons from the Gl tract, and in the initial therapy for convulsions. Magnesium sulfate is gaining popularity as an initial treatment in the management of various dysrhythmias, particularly torsades de pointes, and dysrhythmias secondary to a tricyclic antidepressant overdose or digitalis toxicity. The drug is also considered as a class Ila agent (probably helpful) for refractory ventricular fibrillation and ventricular tachycardia after administration of lidocaine or bretylium doses.

Magnesium sulfate is effective for severe acute asthma treated in the emergency department

http://www.acponline.org/journals/ebm/sepoct99/rowe.htm

Intravenous **magnesium sulfate** reduces the rate of hospital admissions and improves pulmonary function in patients with severe acute asthma treated in the emergency department.

Sources of funding: Canadian Association of Emergency Physicians and National Institutes of Health.

#### 5.8.4.1 Liver Toxin Flush

http://curezone.com/cleanse/liver/epsom\_salt\_and\_liver\_flush.asp

Magnesium sulfate reduces striated muscle contractions and blocks peripheral neuromuscular transmission by reducing acetylcholine release at the myoneural junction. In emergency care, magnesium sulfate is used to manage seizures associated with toxemia of pregnancy. Other uses include uterine relaxation (to inhibit contractions of premature labor), as a bronchodilator after beta-agonist and anticholinergic agents have been used, replacement therapy for magnesium deficiency, as a cathartic to reduce the absorption of poisons from the Gl tract, and in the initial therapy for convulsions. Magnesium sulfate is gaining popularity as an initial treatment in the management of various dysrhythmias, particularly torsades de pointes, and dysrhythmias secondary to a tricyclic antidepressant overdose or digitalis toxicity. The drug is also considered as a class Ila agent (probably helpful) for refractory ventricular fibrillation and ventricular tachycardia after administration of lidocaine or bretylium doses.

Bryant died because she got too much of the drug too fast. She might have given too much of the drug to start with, or incorrectly programmed the pump that controls the IV solution. Bryant's lawyer has said she was given a 40-gram solution, while the safety group recommends a 20-gram solution. The dangers of magnesium sulfate led the Institute for Safe Medication Practices to classify it as one of 25 "high-alert" medicines. Others include chemotherapy drugs, insulin and warfarin, a blood thinner.

### **MSM - Organic Sulfur**

**MSM** Powder Swanson SW851 – "Organic Sulfer" (initially teaspoon AAM - first thing of day, teaspoon PPM- last thing in day)

https://www.swansonvitamins.com/swanson-premium-msm-powder-16-oz-454-grams-pwdr www.healthtalkhawaii.com

MSM (1-8-16) Swanson SWU656 1000mg cap — Methylsulphonylmethane http://www.swansonvitamins.com/swanson-ultra-msm-1000-mg-240-caps

**MSM** is simply an acronym for methylsulfonylmethane, a dietary source of sulfur that naturally supports the immune system and plays a major role in the formation of enzymes and hormones that control body activities.

Helps manage High Homocysteine Levels

Organic Sulfur is supposed to be prominent in all living organisms; however, for various reasons it is only present in very minute/trace quantities—not enough to be of benefit. That fact, combined with the horrendous increase of stress and the toxins our bodies must contend with (some estimates are over 400 or 500 times more than two generations ago) make organic sulfur crucial to our health.

**MSM** supplementation can significantly help lower high homocysteine (an amino acid) levels in the body. High homocysteine levels have been associated with cardiovascular disease, rheumatoid arthritis, renal failure, and type II diabetes.

My current guidance is to consider optimizing your sulfur intake through dietary sources. Onions and **Garlic** are good if they are grown in sulfur replete soils but most soils are actually deficient in sulfur. So animal-based proteins seem to be one of your best bets. Whey protein concentrate is particularly high in cysteine, one of the two sulfur-bearing amino acids that is a direct precursor to **glutathione**.

More recently, an in vitro study demonstrated both <u>MSM</u> and aspirin induce terminal differentiation, utilizing COX-independent mechanisms. 10 Although aspirin was used at a low dose in this study, <u>MSM</u> was used in a much higher concentration to achieve a higher level of differentiation. The COX-independent reaction is presumed to be chemopreventive by invoking the activation of gene functions that lead to differentiation, thereby dismantling the cellular capacity for proliferation.

Methylsulfonylmethane suppresses breast cancer growth by down-regulating STAT3 and STAT5b pathways.Methylsulfonylmethane (MSM) is a widely available 'alternative' medicine.

In vivo magnetic resonance spectroscopy (MRS) was used to detect and quantify <u>MSM</u> in the brains of four patients with memory loss and in three normal volunteers all of who had ingested <u>MSM</u> at the recommended doses of 1-3 g daily. <u>MSM</u> was detected in all subjects at concentrations of 0.42-3.40 mmole/kg brain and was equally distributed between gray and white matter. No adverse clinical or neurochemical effects were observed.

Combined effects of **silymarin** and **methylsulfonylmethane(MSM)** in the management of rosacea: clinical and instrumental evaluation.

- Skin Conditions: especially acne, psoriasis, rosacea (red skin splotches), liver spots, and disorders associated with Lupus Erythematosus have been greatly reduced or eliminated. With mild rosacea and acne, sometimes in as little as two weeks. Skin tone, color, softness, pliability and appearance improve after 6 8 months of taking this organic sulfur according to the established guidelines.
- Allergies, Asthma, and Emphysema: some people with more serious conditions stopped depending on the bottled oxygen they had been toting around in spite of the fact that they continued smoking. (When combined with related EFT/energy work most mild and moderate cases reported 100% resolution for allergies and asthma.)
- Addictions and Food Cravings: Detoxification and reduction of addictions cravings and food cravings appears to be accelerated and reduced when people are maintaining abstinence from addictive substances and junk food. We have reports that the detoxifying phase of recovery is shorter and less intense.
- Autism: About 12 parents have placed their children and young adults on <u>organic sulfur</u>.
   In 9 of those cases the parents report noticeable improvement. In two cases there was dramatic improvement. The reported results varied. One parent reported their child was now enrolled for regular school. Two parents reported their child was now talking and participating in their environment. There is still too little information to make any solid reports on this.
- Cancer and Chemotherapy: Study members undergoing cancer treatment who took 30 40 g of Organic Sulfur (15 20 g twice a day) had no nausea, diarrhea, or hair loss during chemotherapy. There was also a surprisingly noticeable reduction of cancer cell counts as reported by their oncologists.
- Lymphomas appear to consistently reduce in response to <u>Organic Sulfur</u>—both in the associated pain and in decreased size of tumors. When combined with EFT/body energy work, several cases of cervical, breast, and prostate cancer have been resolved without any medical involvement (sometimes in two weeks).
- NAC forms <u>L-cysteine</u>, <u>cystine</u>, <u>L-methionine</u>, <u>glutathione</u> (<u>GSH</u>), and mixed disulfides. Stimulates the body to produce large amounts of cysteine and <u>glutathione</u>, thus greatly augmenting plasma and red blood cell content of both cysteine and <u>glutathione</u>;

Methylsulfonylmethane (<u>MSM</u>): <u>MSM</u>, like fresh <u>Garlic</u>, provides a bioavailable dietary source of sulfur. <u>MSM</u> exerts a direct beneficial effect in ameliorating a variety of allergic responsees and pain associated with systemic inflammatory disorders.

### **Potassium**

### Potassium citrate (1-2)99mg capsule NOW 01448

https://www.swansonvitamins.com/swanson-ultra-potassium-citrate-99-mg-120-caps

Kidney regulation

Cardiovascular health, energy production

PH Balance

Potassium nourishes the heart, kidneys, pancreas, muscles and the nerves. It assists in the growth and repair of body tissues, and also helps conduct messages in the body through the nervous system. Potassium helps regulate blood pressure. There must be a balanced potassium-to-sodium ratio in the body for all systems to function at their optimum level.

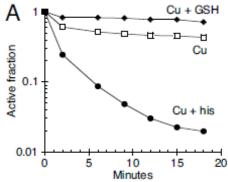
### Metals

## Copper

<u>Copper Citrate</u> (1-2-6) 2mg – used to keep zinc balance, bacteria balance <u>BioCitrate Copper</u> 2 mg Solaray P#04596 / or... <u>Copper Chelate</u> (2-6) 2 mg Twin lab <u>copper caps</u> 01017, Swanson Item: SW223

Copper is a necessary part of some of the enzymes which help inactivate free radicals. Thus it plays a part in antioxidant protection. It is also used for making blood cells. It is active in the metabolism of iron. Coppercontaining enzymes are involved in immune function.

Absorption can be reduced by critical illness, by highdose zinc supplementation, and by antacids. Deficiencies are rare. Measurement is difficult. Excesses can be harmful and can lead to liver failure.



Copper supplementation is not normally recommended, except for those on TPN. However, copper deficiencies have been reported in people with HIV, and it is further reduced with AZT treatment. One study, though, showed higher levels of copper in people with HIV.

There is no RDA for copper. Two to 3 milligrams are generally found in daily multivitamins. **toxicity** involves the action of reactive oxygen species. Low micromolar levels of copper were sufficient to inhibit the growth of both WT and mutant strains

### **Bio Enzyme forms of Copper**

Copper blocks their biosynthesis. Indeed, copper treatment rapidly inactivated isopropylmalate dehydratase, an iron-sulfur cluster enzyme in this pathway suggesting that Cu(I) damages these proteins by liganding to the coordinating sulfur atoms.

Microbes are particularly vulnerable to metal poisoning because they cannot control their extracellular environment

The mechanism by which copper poisons cells has been elusive. A long-standing hypothesis is that copper reacts with endogenous H2O2 to generate hydroxyl radicals in a process analogous to the Fenton reaction:

Indeed, we found that copper-stressed cells produced endogenous H2O2 at a rate several fold higher than did untreated cells (data not shown). Excessive levels of superoxide (O2\_) and H2O2 can disrupt several amino acid biosynthetic pathways (18–20); therefore, we tested whether copper creates similar blocks. (This experiment was complicated by the fact that \_-amino acids chelate copper and thereby increase the dose necessary for toxicity; to correct for this effect, alanine was added in equivalent doses to cultures from which amino acids of interest were withheld.) The *copA cueO cusCFBA* mutant could not grow aerobically in glucose/alanine medium to which 10 \_M copper was added (Fig. 2A). The addition of branched-chain amino acids partially restored growth, indicating that this pathway is a primary target of copper toxicity. The failure to fully restore growth indicates that additional growthlimiting damage also occurred outside of this pathway.

Copper Inactivated Iron-Sulfur Cluster Dehydratases Inside Aerobic Cells. Reactive oxygen species block branched-chain biosynthesis because they directly damage the iron-sulfur clusters of 2 dehydratases: dihydroxy-acid dehydratase in the common branchedchain pathway and isopropylmalate isomerase (IPMI) in the leucine-specific branch (18, 21–23). In conformity with the **The Mechanism of Copper Toxicity Is Independent of Oxygen.** Other investigators have noted that copper can poison bacterial cells in anaerobic medium (12, 17, 24), and we observed, in fact, that both WT cells and the *copA cueO cusCFBA* mutant were much more sensitive to growth inhibition by copper when oxygen was absent (Fig. S2). The anaerobic growth of the WT and mutant strains was effectively blocked by 1 \_M and 125 nM copper, respectively. At the very least, this means that copper has an acute mechanism of toxicity that does not involve reactive oxygen species.

Surprisingly, experiments suggested that the primary target of anaerobic toxicity was no different from that in aerobic cells. Growth resumed when branched-chain amino acids were added to anaerobic medium (Fig. 3). This restoration of growth was not caused by chelation of copper, because the D-enantiomers of the amino acids failed to restore growth (data not shown). Further, an overexpression plasmid carrying *leuCD* fully suppressed the growth defect, indicating that it arose from the lack of IPMI activity (Fig. S1B). Indeed, during anaerobic exposure to 4 \_M copper, the IPMI activities of bothWT cells and *copA cueO cusCFBA* mutant cells decreased to less than 15% of the activity of untreated cells (Fig. 4A). Copper also damaged fumarase B, the anaerobic isozyme of fumarase A (Fig. 4B). These results indicated that the poisoning of these enzymes by copper occurred through a non-oxidative mechanism.

It is likely that the same was true in aerobic media **Copper Damaged the Iron-Sulfur Cluster of Fumarase A.** Saturating concentrations of malate, a substrate of fumarase A, fully protected the enzyme activity from copper (Fig. 5*B*). This effect was probably mediated by its shielding the the enzyme active site, as the protective effect tracked closely to the dose needed to form an ES complex.

#### **Discussion**

Life evolved in an anaerobic world that was rich in iron. Evolution exploited the versatile surface chemistry of this metal by integrating it into iron-sulfur clusters that serve to bind substrates into enzyme active sites. This arrangement succeeded for 2 billion years, until the emergence of photosystem 2 caused the gradual oxygenation of the environment. As molecular oxygen

accumulated, it oxidized dissolved sulfur compounds, which until that time had sequestered soft metals into mineral precipitates. The subsequent solubilization of these metals evidently posed a new problem for extant organisms. The present study reveals that one of these metals, copper, rapidly inactivates the catalytic clusters of dehydratases. This enzyme family has representatives in central catabolic and biosynthetic pathways, which therefore become dysfunctional in copper-exposed cells.

These enzymes are uniquely vulnerable to chemical damage because their clusters are substantially exposed to solvent (31). Previous studies show that O2 \_ and H2O2 are small enough to invade the active site, where they coordinate and oxidize the iron-sulfur cluster to an unstable valence (18, 21, 32). In this work, we found that these clusters are also the primary targets of **copper**, which evidently displaces their **iron** atoms when it coordinates their thiolate or inorganic sulfur ligands. The extreme avidity with which it does so means that cellular environments that contain such enzymes cannot tolerate even modest levels of active **copper**.

#### **Iodine**

### Tri-iodine (1) Terry Naturally 12500mcg #18299

Swanson Ultra **Triple Iodine Complex** SWU809, 12.5 mg 60 Veg Caps <a href="http://www.iherb.com/EuroPharma-Terry-Naturally-Tri-Iodine-12-5-mg-90-Capsules/23508">http://www.iherb.com/EuroPharma-Terry-Naturally-Tri-Iodine-12-5-mg-90-Capsules/23508</a>

<u>Lugol's Solution Iodine:</u> Contains 6.3 mg of molecular iodine/iodide per drop. Useful where the GI tract cannot be regulated (Hard/loose stool)

<u>Iodine</u> - a Pillar Against Infections

**Iodine** offers a serious and potent replacement for many antibiotics. Though it kills 90 percent of bacteria on the skin within 90 seconds, its use as an antibiotic has been ignored. **Iodine** exhibits activity against bacteria, molds, yeasts, protozoa, and many viruses; indeed, of all antiseptic preparations suitable for direct use on humans and animals and upon tissues, only iodine is capable of killing all classes of pathogens: gram-positive and gram-negative bacteria, mycobacteria, fungi, yeasts, viruses and protozoa. Iodine is by far the best antibiotic, antiviral and antiseptic of all time - Dr. David Derry

Dr. Derry says that **iodine** is effective "for standard pathogens such as Staphylococcus, but also iodine has the broadest range of action, fewest side effects and no development of bacterial resistance." There is a world of difference between using an antibiotic – anti-life substance – and an antibiotic, antiviral and antifungal substance like iodine, which is life serving because it is a basic and most necessary nutritional substance.

<u>Iodine</u> kills single celled organisms by combining with the amino acids tyrosine or histidine when they are exposed to the extra-cellular environment. All single cells showing tyrosine on their outer cell membranes are killed instantly by a simple chemical reaction with iodine that denatures proteins. Nature and evolution have given us an important mechanism to control pathogenic life forms and we should use it and trust it to protect us in ways that antibiotics can't.

In Africa iodine (in the atomic or detoxified form) to successfully treat malaria.

### **Review Of Atomidine (Iodine)**

www.Iodinesource.com.

Iodine is toxic. What you get in the store I would NOT put in my mouth. Having a friend who owed me a favor (who just happens to be a chemist), I sent him a CC of the Atomidine in a bottle for a toxicology reading. He reported back, "Hey, this is some good stuff." I asked how much could the average adult take in a week. His response was, "A heck of a lot more than any other iodine out there." He seemed to feel that one or two CCs a week would not be harmful, but that side effects should be noted if they occurred. We both agreed that it was powerful stuff and should be used with caution. Someone once asked me, "Why do you take all of your discoveries and put them in your mouth?"

Now I put a drop of Atomidine on the cut and it heals in less than three days. Stings, but it heals.

Cayce Concept's Atomidine is 1% iodine in alcohol. As you can guess, or if you have a degree in chemistry, the second is stronger (and more expensive). Both have been magnetically charged in the manner prescribed by the Cayce readings. Baar's product tastes salty, and does not burn much when applied.

Phil at Cayce Concepts on the web: http://www.iodinesource.com/. He doesn't have much of a store online, but you can many of Cayce's therapies (equipment) there to try on your own. Phil is very dedicated and sincere about his work and wants to bring Cayce's healing remedies back into the mainstream.

Baar Products can be found at www.baar.com Though, I must rank Cayce Concepts's Atomidine a giant step above Baar's product. My medicine chest will not be without a bottle at all times. Put one in your First Aid kit too.

Additionally, Barr's product, Iodine Trichloride, should NEVER be used internally. Here is the Material Safety Data Sheet on Iodine Trichloride; #2

CAUTION: According to Phil Thomas, all iodine has a poisonous effect upon the body unless it is first detoxified, and even then it's a powerful element to contend with. As is the case with everything, the body and it's reaction are specific to that given situation and/or individual. Iodine Source

### **PRODUCT: Detoxified Iodine**

IodineSource.com is your source for detoxified iodine. Unlike commercially available products, which use iodine bonded with chlorine, we use only naturally occurring iodine derived from organic Asian seaweed, as was specified in the Cayce readings. This pure crystalline iodine is then reduced to a 1% concentration in 100% ethyl alcohol and electro-magnetically transmuted (while being suspended in a wet bath containing a mild acid solution) into the Atomic state. This is the form which Mr. Cayce suggested the body could recognize and fully assimilate, and indeed was his primary contribution to the concept. From there it is bottled in 1/2 oz. (amber) dropper bottles and unless otherwise indicated, labeled and sealed with a full bottle heat shrink. For more information about detoxified iodine, go to Cayce Concepts. \$20.00/bottle. 6 bottles/\$60.00

### The History of Iodine

Iodine And The Halogen Revolution, Halogen is defined in chemistry as any of the electronegative elements, including iodine, fluorine, chlorine, bromine and astatine, that form binary salts by direct union with metals. The term was coined in 1842 by the Swedish chemist Baron Jöns Jakob Berzelius. It's from the Greek words halos (meaning salt) and gen (to produce). This term is used because a salt is formed in reactions involving these elements. In my many years of testing people, iodine has always shown up as a very important and primary nutrient in regards to people's health

and healing. Testing has always shown an iodine deficient person to be unable to utilize their proteins properly and an iodine deficient person will remain protein deficient.

In the past I have seen that a couple of milligrams of iodine daily will cure iodine deficiency in the person with adequate gut absorption powers. This will allow them to utilize their proteins, however it will not mean that they have full body iodine sufficiency (iodine receptors) in tissues such as breast, uterus, prostate, skin, salivary glands, stomach, colon, choroid plexus, and eye. It will also not assure that a thyroid whose receptor space is taken up with another halogen, such as bromine or fluorine, will have a full sufficiency of iodine for hormone production.

There are four halogens: iodine, bromine, fluorine and chlorine. Only iodine and chlorine are necessary to the body. We need iodine in all the aforementioned tissues (probably all tissues, but it is most concentrated in the thyroid). We need chlorine in the stomach for secretion of hydrochloric acid. Chloride ion is also an important part of the blood's regulation of its acid-base balance. You need sufficient chloride to breathe.

All these halogens use the same receptors in the body. Therefore if a person's diet is deficient in iodine, the iodine receptors in the thyroid and stomach, may fill up with bromine, which is commonly present in grains, bleached flour, sodas, nuts and oils as well as several plant foods. This person's thyroid function is deficient and the iodide-pump in the stomach will not work efficiently either.

A person whose sweat glands are low in iodine will suffer from dry skin. It is important to note here that the present "low salt" regimes pushed by the mainstream medicine leave us chloride deficient as well.

Fluorine from sources such as toothpaste, certain teas, and fluoridated water will also take up receptor space.

Research shows me that once this iodine receptor space is taken up by another halogen, it takes a certain level of iodine loading to replace the unwanted halogens with iodine. This point seems to be especially pronounced for bromine. While I have seen that 1 to 2 milligrams of iodine daily will allow protein metabolism to normalize, It takes much more to remove unwanted halogens from the system.

### **Iodine From Sea Plant Minerals**

Full body iodine sufficiency has become an item of interest to several pioneering researchers such as Dr. Abraham and Dr. Brownstein. Both of them have done thorough and credible research into the methods and efficiency of iodine loading in the body. Their research shows that 12.5 milligrams of iodine daily is the minimum requirement for full body iodine sufficiency.

### **Iodine and Vitamin D**

An apparent connection between bringing sufficient iodine into a bromine plugged thyroid and the vitamin D metabolism of the body. It seems clear that the calcitonin-parathyroid hormone-vitamin D-calcium balance in the body changes as people on iodine loading programs often register as vitamin D deficient when they did not previously. It is interesting to note that when people are given calcitonin as part of an osteoporosis program, one of the side effects mentioned is a "flushing sensation of the skin". "A sunburn flushed feeling on the skin of their faces, arms and back." This flushing disappeared with vitamin D3 supplementation at 2000 iu daily for a limited period of time.

The protein that the body uses for iodine transport (sodium iodide symporter) needs to be sufficient in quantity also in order to deal with the influx of iodine now available for utilization as the bromine and fluorine are being pushed out. A protein deficient person will not be able to do this, so it is wise not to begin iodine loading until your patient is no longer protein deficient.

Of course, the application of both the Sea Plant Minerals and Amino Acids together will also cause a very rapid and thorough flushing of heavy metals such as aluminum, mercury, cadmium and lead.

#### **Iodine**

While the liquid iodine tincture (Lugol's) tests well for people, it is hard to regulate its dosage. Drs Abraham and Brownstein promote the iodine tablet Iodoral. Iodine loading on the Vickery Sea Plant Minerals, she replied, "It's like coming home."

I find the Vickery Sea Plant Minerals, which contain a high iodine form of organic kelp powder, to be very gentle yet effective in iodine loading and supplementation as well as supplying ionic trace minerals and plant processed vitamins. Kelp is also high in the essential sugars, which are an integral part of our immune proteins (gamma globulins) and are also very important in intercellular communication. Essential sugars are involved in red blood cell metabolism (although the mechanism is unclear, their presence is apparent).

People in the U.S. consume an average 240 micrograms ( $\mu$ g) of iodine a day. In contrast, people in Japan consume more than 12 milligrams (mg) of iodine a day (12,000  $\mu$ g), a 50-fold greater amount. They eat seaweed, which include brown algae (kelp), red algae (nori sheets, with sushi), and green algae (chlorella).

Now iodized table salt is the chief source of iodine in a Western diet. But 45 percent of American households buy much higher concentrations of chloride in salt (NaCl) which inhibits absorption of its sister halogen iodine (the intestines absorb only 10 percent of the iodine present in iodized table salt). As a result, 15 percent of the U.S. adult female population suffers from moderate to severe iodine deficiency. A high intake of iodine is associated with a low incidence breast cancer, and a low intake with a high incidence of breast cancer.

Russian researchers first showed, in 1966, that iodine effectively relieves signs and symptoms of fibrocystic breast disease, iodine relieves signs and symptoms of fibrocystic breast disease in 70 percent of their patients.

It declined to approve the study, telling its lead investigator, Dr. Donald Low, "iodine is a natural substance, not a drug." But the FDA has now decided to approve a similar trial sponsored by Symbollon Pharmaceuticals. This company is enrolling 175 women in a phase III trial, registered on clinicaltrials.gov. (Any women with fibrocystic disease reading this who might be interested in participating in this study should call its sponsor, Jack Kessler, Ph.D., at 508-620-7676, Ext. 201.)

Most physicians and surgeons view iodine from a narrow perspective. It is an antiseptic that disinfects drinking water and prevents surgical wound infections, and the thyroid gland needs it to make thyroid hormones — and that's it. (When painted on the skin prior to surgery, tincture of iodine kills 90 percent of bacteria present within 90 seconds.) The thyroid gland needs iodine to synthesize thyroxine (T4) and triiodothyronine (T3), hormones that regulate metabolism and steer growth and development. T4 contains four iodine atoms combined with 27 other atoms of carbon, hydrogen, oxygen, and nitrogen, but owing to its large size accounts for 65 percent of the molecule's weight. (T3 has three iodine atoms.) The thyroid needs only a trace amount of iodine, 70  $\mu$ g a day, to produce the requisite amount of T4 and T3. For that reason thyroidologists say that iodine is best taken just in microgram amounts. They consider consuming more than 1 to 2 mg of iodine a day to be excessive and potentially harmful.

Expert opinion on iodine is now the purview of thyroidologists. Mainstream physicians and surgeons accept their thyroid-only view of iodine and either ignore or discount studies that show iodine in larger amounts provides extrathyroidal benefits, particularly for women's breasts. Thus a leading textbook on breast disease, Bland and Copeland's The Breast: Comprehensive Management of Benign and Malignant Disorders (2003), fails to mention iodine anywhere in its 1,766 pages.

Iodine has an important and little understood history. This relatively scarce element has played a pivotal role in the formation of our planet's atmosphere and in the evolution of life. For more than two billion years there was no oxygen in the atmosphere until a new kind of bacteria, cyanobacteria (blue-green algae), began producing oxygen as a byproduct of photosynthesis. Cyanobacteria also developed an affinity for iodine. The most likely reason is that these organisms used iodine as an antioxidant to protect themselves against the free radicals that oxygen breeds (superoxide anion, hydrogen peroxide, and hydroxyl radical). Studying kelp, researchers have shown how iodine does this and have found that kelp will absorb increased amounts of iodine when placed under oxidative stress. Other researchers have shown that iodine increases the antioxidant status of human serum similar to that of vitamin C.

Iodine also induces apoptosis, programmed cell death. This process is essential to growth and development (fingers form in the fetus by apoptosis of the tissue between them) and for destroying cells that represent a threat to the integrity of the organism, like cancer cells and cells infected with viruses. Human lung cancer cells with genes spliced into them that enhance iodine uptake and utilization undergo apoptosis and shrink when given iodine, both when grown in vitro outside the body and implanted in mice. Its anti-cancer function may well prove to be iodine's most important extrathyroidal benefit.

Iodine has other extrathyroidal functions that require more study. It removes toxic chemicals — fluoride, bromide, lead, aluminum, mercury — and biological toxins, suppresses auto-immunity, strengthens the T-cell adaptive immune system, and protects against abnormal growth of bacteria in the stomach.

In addition to the thyroid and mammary glands, other tissues possess an iodine pump (the sodium/iodine symporter). Stomach mucosa, the salivary glands, and lactating mammary glands can concentrate iodine almost to the same degree as the thyroid gland (40-fold greater than its concentration in blood). Other tissues that have this pump include the ovaries; thymus gland, seat of the adaptive immune system; skin; choroid plexus in the brain, which makes cerebrospinal fluid; and joints, arteries and bone.

Today's medical establishment is wary of iodine (as they are of most naturally occurring, nonpatentable, nonpharmaceutical agents). Thyroidologists cite the Wolff-Chaikoff effect and warn that TSH (thyroid stimulating hormone) blood levels can rise with an iodine intake of a milligram or more. The Wolff-Chaikoff effect, a temporary inhibition of thyroid hormone synthesis that supposedly occurs with increased iodine intake, is of no clinical significance. And an elevated TSH, when it occurs, is "subclinical." This means that no signs or symptoms of hypothyroidism accompany its rise. Some people taking milligram doses of iodine, usually more than 50 mg a day, develop mild swelling of the thyroid gland without symptoms. The vast majority of people, 98 to 99 percent, can take iodine in doses ranging from 10 to 200 mg a day without any clinically adverse affects on thyroid function. The prevalence of thyroid diseases in the 127 million people in Japan who consume high amounts of iodine is not much different than that in the U.S.

Everyone agrees that a lack of iodine in the diet causes a spectrum of disorders that includes, in increasing order of severity, goiter and hypothyroidism, mental retardation, and cretinism (severe mental retardation accompanied by physical deformities). Health authorities in the U.S. and Europe have agreed upon a Reference Daily Intake (RDI), formerly called the Recommended Dietary

Allowance (RDA), for iodine designed to prevent these disorders, which the World Health Organization (WHO) estimates afflicts 30 percent of the world's population. The RDI for iodine, first proposed in 1980, is  $100-150~\mu g/day$ . Organizations advocating this amount include the American Medical Association, National Institutes of Health's National Research Council, Institute of Medicine, United Nations Food and Agricultural Organization, WHO Expert Committee, and the European Union International Programme on Chemical Safety. These health authorities consider an RDI of  $100-150~\mu g/day$  of iodine sufficient to meet the requirements of nearly all (97–98%) healthy individuals.

This consensus on iodine intake flies in the face of evidence justifying a higher amount. This evidence includes animal studies, in vitro studies on human cancer cell lines, clinical trials of iodine for fibrocystic breast disease, and epidemiological data. An intake of 150  $\mu$ g/day of iodine will prevent goiters and the other recognized iodine deficiency disorders, but not breast disease. Prevention of breast disease requires higher doses of iodine. Indeed, a reasonable hypothesis is that, like goiters and cretinism, fibrocystic disease of the breast and breast cancer are iodine deficiency disorders (also uterine fibroids).

What Albert Guérard writes about new truths applies especially to iodine: "When you seek a new path to truth, you must expect to find it blocked by expert opinion." The reigning truth on iodine is that the thyroid gland is the only organ in the body that requires this micronutrient, and a daily intake considerably more than what the thyroid gland needs is potentially harmful. The new truth is that the rest of the body also needs iodine, in milligram, not microgram amounts. Tell that to a thyroidologist and her response will call to mind this admonition on new truths.

Iodine was used for a wide variety of ailments after its discovery in 1811 up until the mid-1900s, when thyroidologists warned that "excess" amounts of iodine might adversely affect thyroid function. It is effective in gram amounts for treating various dermatologic conditions, chronic lung disease, fungal infestations, tertiary syphilis, and even arteriosclerosis. The Nobel laureate Dr. Albert Szent Györgi (1893—1986), the physician who discovered vitamin C, writes: "When I was a medical student, iodine in the form of KI was the universal medicine. Nobody knew what it did, but it did something and did something good. We students used to sum up the situation in this little rhyme:

If ye don't know where, what, and why Prescribe ye then K and I"

The standard dose of potassium iodide given was 1 gram, which contains 770 mg of iodine.

Regarding KI and other iodine salts (like sodium iodide), the venerated 11th edition of the Encyclopedia Britannica, published in 1911, states, "Their pharmacological action is as obscure as their effects in certain diseased conditions are consistently brilliant. Our ignorance of their mode of action is cloaked by the term deobstruent, which implies that they possess the power of driving out impurities from the blood and tissues. Most notably is this the case with the poisonous products of syphilis. In its tertiary stage — and also earlier — this disease yields in the most rapid and unmistakable fashion to iodides, so much so that the administration of these salts is at present the best means of determining whether, for instance, a cranial tumor be syphilitic or not."

This 19th and early 20th century medicine continues to be used in gram amounts in the 21st century by dermatologists. They treat inflammatory dermatoses, like nodular vasculitis and pyoderma gangrenosum (shown here), with SSKI, beginning with an iodine dose of 900 mg a day, followed by weekly increases up to 6 grams a day as tolerated. Fungal eruptions, like sporotrichosis, are treated initially in gram amounts with great success. These lesions can disappear within two weeks after treatment with iodine.

For many years physicians used potassium iodide in doses starting at 1.5 to 3 gm and up to more than 10 grams a day, on and off, to treat bronchial asthma and chronic obstructive pulmonary disease with good results and surprisingly few side effects.

There is a case report in the medical literature of a 54-year-old man who, thinking it was iced tea, drank a "home preparation" of SSKI in water that his aunt kept in the refrigerator for her rheumatism. Over a short period of time he consumed 600 ml of this solution, which contained 15 gm of iodide, an amount 100,000 times more than the RDI. He developed swelling of the face, neck, and mouth, had transient cardiac arrhythmias and made an uneventful recovery.

Dr. Guy Abraham, a former professor of obstetrics and gynecology at UCLA, mounted what he calls "The Iodine Project" in 1997 after he read the Ghent paper on iodine for fibrocystic disease. He had his company, Optimox Corp., make Iodoral, the tablet form of Lugol's solution, and he engaged two family practice physicians, Dr. Jorge Flechas (in 2000) in North Carolina and Dr. David Brownstein (in 2003) in Michigan to carry out clinical studies with it.

The project's hypothesis is that maintaining whole body sufficiency of iodine requires 12.5 mg a day, an amount similar to what the Japanese consume. The conventional view is that the body contains 25—50 mg of iodine, of which 70—80 percent resides in the thyroid gland. Dr. Abraham concluded that whole body sufficiency exists when a person excretes 90 percent of the iodine ingested. He devised an iodine-loading test where one takes 50 mg and measures the amount excreted in the urine over the next 24 hours. He found that the vast majority of people retain a substantial amount of the 50 mg dose. Many require 50 mg a day for several months before they will excrete 90 percent of it. His studies indicate that, given a sufficient amount, the body will retain much more iodine than originally thought, 1,500 mg, with only 3 percent of that amount held in the thyroid gland.

More than 4,000 patients in this project take iodine in daily doses ranging from 12.5 to 50 mg, and in those with diabetes, up to 100 mg a day. These investigators have found that iodine does indeed reverse fibrocystic disease; their diabetic patients require less insulin; hypothyroid patients, less thyroid medication; symptoms of fibromyalgia resolve, and patients with migraine headaches stop having them. To paraphrase Dr. Szent-Györgi, these investigators aren't sure how iodine does it, but it does something good.

Thyroid function remains unchanged in 99 percent of patients. Untoward effects of iodine, allergies, swelling of the salivary glands and thyroid, and iodism, occur rarely, in less than 1 percent. Iodine removes the toxic halogens fluoride and bromide from the body. Iodism, an unpleasant brassy taste, runny nose, and acne-like skin lesions, is caused by the bromide that iodine extracts from the tissues. Symptoms subside on a lesser dose of iodine.

As these physicians point out, consuming iodine in milligram doses should, of course, be coupled with a complete nutritional program that includes adequate amounts of **selenium**, **magnesium**, **and Omega-3 fatty acids**. Done this way, an iodine intake 100 times the reference daily intake is "the simplest, safest, most effective and least expensive way to help solve the health care crisis crippling our nation," as the leader of The Iodine Project, Dr. Abraham, puts it.

People who take iodine in these amounts report that they have a greater sense of well-being, increased energy, and a lifting of brain fog. They feel warmer in cold environments, need somewhat less sleep, improved skin complexion, and have more regular bowel movements. These purported health benefits need to be studied more thoroughly, as do those with regard to fibrocystic breast disease and cancer.

Meanwhile, perhaps we should emulate the Japanese and substantially increase our iodine intake, if not with seaweed, then with two drops of Lugol's Solution (or one Iodoral tablet) a day.

Recommended Reading:

Miller DW. Iodine in Health and Civil Defense. Presented at the 24th Annual Meeting of Doctors for Disaster Preparedness in Portland, Oregon, August 6, 2006. The text for this talk, with 68 references, can be found here, and the PowerPoint slides I used for it, here.

Abraham GE. The safe and effective implementation of orthoiodosupplementation in medical practice. The Original Internist 2004;11:17—36. Available online here. This is a good introduction to The Iodine Project. His other research studies are online here.

Flechas, JD. Orthoiodosupplementation in a primary care practice. The Original Internist 2005;12(2):89—96. Available online here.

Brownstein D. Clinical experience with inorganic, non-radioactive iodine/iodide. The Original Internist 2005;12(3):105—108. Available online here.

Derry D. Breast cancer and iodine: How to prevent and how to survive breast cancer. Victoria, B.C.: Trafford Publishing; 2002. The book is a bit disorganized, has references at the end of each chapter not cited in the text, and no index; but it is an eye-opener nonetheless.

Brownstein D. Iodine: why you need it why you can't live without it. West Bloomfield, Michigan: Medical Alternatives Press; 2004. Well-written and referenced, with case histories.

Low DE, Ghent WR, Hill LD. Diatomic iodine treatment for fibrocystic disease: special report of efficacy and safety results. [Submitted to the FDA] 1995:1—38. Available online here. This study makes a strong case for iodine as the preferred treatment for fibrocystic disease.

#### Selenium

# <u>Selenium</u> (2-4-8) CAP. 200 mcg 120ct NOW FOODS P#01486 SKU: 733739014856 , Swanson SWU171

https://www.swansonvitamins.com/swanson-ultra-semsc-selenium-200-mcg-120-caps

Selenium 200 mcg @ L-Selenomethionine

<u>Selenium</u> deficiency that results in an increase in expression of adhesion molecules, which causes greater adhesion of neutrophils.[viii]

Immune system booster, 450CYP ROS Consumable under Ox stress, CYP450 deficiency leak, Beneficial in the prevention of several types of infection, Selenium, which acts both as a free radical scavenger, and as a mineral that helps prevent cells from turning cancerous, boosts white blood cell production.

### Avoid with a history of non-melanoma skin cancer

**Selenium** influences both the innate, "nonadaptive" and the acquired, "adaptive" immune systems[iii]-[iv]-[vi]-[vii] The innate immune system includes barriers to infection and nonspecific effector cells such as macrophages. Both the T and B-lymphocytes form the major effector cells of the acquired system that mature with exposure to immune challenges. Selenium-deficient lymphocytes are less able to proliferate in response to mitogen, and in macrophages, leukotriene B4 synthesis, which is essential for neutrophil chemotaxis, is impaired by this deficiency. These processes can be improved by selenium supplementation. The humoral system is also affected by selenium deficiency; for example, IgM, IgG and IgA titers are decreased in rats, and IgG and IgM titers are decreased in humans. In endothelial cells from asthmatics, there is a marked selenium deficiency that results in an increase in expression of adhesion molecules, which causes greater adhesion of neutrophils.[viii]

**Selenium** is also involved in several key metabolic activities through its selenoprotein enzymes that protect against oxidative damage.[ix] Further, selenium deficiency may allow invading viruses to mutate and cause longer-lasting, more severe illness.[x] Animal research has shown selenium and **vitamin E** have synergistic effects, enhancing the body's response to bacterial[xi] and parasitic infections.[xii]

The use of selenium compounds as a cancer treatment predates most conventional treatments currently in use. [91] In spite of this, comparatively little is known regarding the use of selenium as a cancer therapy in living systems. Subcutaneous injection of 2 mcg/g selenium into tumor-bearing mice led to a 75-percent reduction in tumor mass compared to controls. [92] This inhibitory effect of selenium was confirmed in human breast cancer cells in vitro. [93] In an open trial of 32 patients with treatment refractory brain tumors, intravenous infusion of selenium (1000 mcg/day for 4-8 weeks) was associated with a slight to definite improvement in all participants. Symptomatic decrease was seen in nausea, emesis, headache, vertigo, and seizure activity. Although the results are largely credited to the selenium treatment, it should be noted these patients were concurrently receiving chemotherapy, oxygen therapy, vitamins E and A, dietary changes, and psychotherapy. [94] Unpublished research from the 1950s outlines the treatment of over 1000 malignancies with selenium compounds, reportedly with beneficial results. [95] Unfortunately, a study of this magnitude has yet to appear in the peer-reviewed literature.

### http://www.nlm.nih.gov/medlineplus/druginfo/natural/1003.html

Clinical research shows that taking a combination of selenium 100 mcg along with zinc 20 mg, vitamin C 120 mg, vitamin E 30 mg, and beta-carotene 6 mg/day once daily for 7.5 years does not lower the overall chance of developing cancer of any type.

#### **Selenium and iodine**

<u>Selenium</u> is a mineral. It is taken into the body in water and foods. People use it for medicine.

Most of the selenium in the body comes from the diet. The amount of **selenium** in food depends on where it is grown or raised. Crab, liver, fish, poultry, and wheat are generally good selenium sources. The amount of **selenium** in soils varies a lot around the world, which means that the foods grown in these soils also have differing **selenium** levels. In the U.S., the Eastern Coastal Plain and the Pacific Northwest have the lowest selenium levels. People in these regions naturally take in about 60 to 90 mcg of selenium per day from their diet. Although this amount of **selenium** is adequate, it is below the average daily intake in the U.S., which is 125 mcg.

<u>Selenium</u> is used for diseases of the heart and blood vessels, including stroke and "hardening of the arteries" (atherosclerosis). It is also used for preventing various cancers including cancer of the prostate, stomach, lung, and skin.

Some people use <u>selenium</u> for under-active thyroid, osteoarthritis, rheumatoid arthritis (RA), an eye disease called macular degeneration, hay fever, infertility, cataracts, gray hair, abnormal pap smears, chronic fatigue syndrome (CFS), mood disorders, arsenic poisoning, and preventing miscarriage.

<u>Selenium</u> is also used for preventing serious complications and death from critical illnesses such as head injury and burns. It is also used for preventing bird flu, treating HIV/AIDS, and reducing side effects from cancer chemotherapy.

The effectiveness ratings for SELENIUM are as follows:

- •Preventing lower than normal levels of selenium (selenium deficiency).
- •Autoimmune thyroiditis (Hashimoto's thyroiditis). Taking selenium 200 mcg daily along with thyroid hormone might decrease antibodies in the body that contribute to this condition. Selenium might also help improve mood and general feelings of well-being in people with thyroiditis.
- •High cholesterol. Some research shows that taking a specific selenium supplement (SelenoPrecise, Pharma Nord, Denmark) 100-200 mcg daily for 6 months can modestly reduce cholesterol levels. Many people in this study had low levels of selenium in their body before the start of the study. It is not clear if taking extra selenium would have any benefit on cholesterol levels in people with normal selenium levels in the body.
- •Diabetes. Some research has shown that people with low selenium levels in the body have a higher chance of getting type 2 diabetes. But other research shows that people who have high amounts of selenium in the body also have increased chance of getting type 2 diabetes. Additionally, more reliable research shows that people who take a selenium supplement 200 mcg daily for an average of 7.7 years actually have a significantly increased chance of getting type 2 diabetes.

**Selenium** is important for making many body processes work correctly. It seems to increase the action of antioxidants.

Higher doses are POSSIBLY UNSAFE. They can cause significant side effects including nausea, vomiting, nail changes, loss of energy, and irritability. Poisoning from long-term use is similar to arsenic poisoning, with symptoms including hair loss, white horizontal streaking on fingernails, nail inflammation, fatigue, irritability, nausea, vomiting, **garlic** breath odor, and a metallic taste.

<u>Selenium</u> can also cause muscle tenderness, tremor, lightheadedness, facial flushing, blood clotting problems, liver and kidney problems, and other side effects.

Symptoms usually reverse when selenium is removed.

There is concern that taking selenium for a long time high dose levels might not be safe. Long-term consumption of **selenium** supplements appears to increase the chance of getting type 2 diabetes. It also seems to increase the risk of skin cancer recurrence. There is also some concern that having too much selenium in the body might increase the risk of overall death as well as death from cancer.

**Selenium** use is POSSIBLY SAFE during pregnancy and breast-feeding when used short-term in amounts that are not larger than 400 mcg per day.

Avoid long-term use of selenium supplements if you have ever had skin cancer.

Under-active thyroid (hypothyroidism): Taking <u>selenium</u> can worsen hypothyroidism especially in people with <u>iodine</u> deficiency. In this case, you should take <u>iodine</u> along with <u>selenium</u>. Check with your healthcare provider.

Surgery: Selenium might increase the risk of bleeding during and after surgery. Stop taking selenium at least 2 weeks before a scheduled surgery.

<u>Selenium</u> might slow blood clotting. Taking <u>selenium</u> along with medications that also slow blood clotting might increase the chances of bruising and bleeding.

Some medications that slow blood clotting include aspirin, clopidogrel (Plavix), dalteparin (Fragmin), enoxaparin (Lovenox), heparin, ticlopidine (Ticlid), warfarin (Coumadin), and others.

Medications used for lowering cholesterol include atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Mevacor), and pravastatin (Pravachol).

Taking <u>selenium</u> along with vitamin E, vitamin C, and beta-carotene might decrease some of the beneficial effects of niacin. Niacin can increase the good cholesterol. Taking selenium along with these other vitamins might decrease how well niacin works for increasing good cholesterol.

The body breaks down medications to get rid of them. Selenium might slow how fast the body breaks down sedative medications (barbiturates). Taking selenium with these medications might increase the effects and side effects of these medications.

#### **Astragalus**

Some species of astragalus accumulate large amounts of selenium, especially when grown in selenium-rich soils. Taking products made from these plants along with selenium supplements could cause selenium poisoning. However, most astragalus supplements contain Astragalus membranaceus, which is not a selenium accumulator.

#### **Zinc**

Zinc might make it more difficult for the body to absorb selenium.

Dose RDA: BY MOUTH:

- •Autoimmune thyroiditis (Hashimoto's thyroiditis): 200 mcg daily.
- •High cholesterol: 100-200 mcg daily of a specific selenium product (SelenoPrecise, Pharma Nord, Denmark).

The daily recommended dietary allowances (RDAs) of selenium are: •Children 1-3 years, 20 mcg; children 4-8 years, 30 mcg; children 9-13 years, 40 mcg;

- •People over 13 years, 55 mcg;
- •Pregnant women, 60 mcg; and lactating women, 70 mcg. Due to the demands of the fetus on the mother, the dietary need for selenium increases during pregnancy.

The tolerable upper limit is: •Adults, 400 mcg per day for adults and adolescents 14 years and older.

- •The tolerable upper intake level (UL) for infants up to age 6 months is 45 mcg per day;
- •Infants 7 to 12 months, 60 mcg per day;
- •Children 1 to 3 years, 90 mcg per day;
- •Children 4 to 8 years, 150 mcg per day;
- •Children 9 to 13 years, 280 mcg per day.

Atomic number 34, Dioxyde de Sélénium, Ebselen, L-Selenomethionine, L-Sélénométhionine, Levure Sélénisée, Numéro Atomique 34, Se, Selenio, Selenite, Sélénite de Sodium, Sélénium, Selenium Ascorbate, Selenium Dioxide, Selenized Yeast, Selenomethionine, Sélénométhionine, Sodium Selenite.

Please see the Natural Medicines Comprehensive Database methodology.

#### **Boron**

### **Boron (1) 3mg Now 01410**

Boron may retard bone loss. There are indications that boron increases the absorption of calcium, magnesium, and phosphorus, as well as controls the urinary loss of these minerals. The ability to use energy and to think may also be related to boron levels, but this is not yet confirmed. Boron be be nutritionally supportive in conditions of osteoarthritis, osteoporosis, and rheumatoid arthritis.

#### **Vandium**

### Vandium - (1) KAL vandyl comples 70311

http://www.kalvitamins.com

### **Chromium**

### Chromium Picolinate - (1-3 in am - 0 in pm) 200 MCG NOW FOODS 1422 01422

Chromium Picolinate 200mcg 200ct. Swanson Premium SW923

Chromium is necessary for the body to convert glucose to energy. A U.S. Department of Agriculture study has shown that nine out of ten Americans get less than the low end range of chromium recommended by the National Academy of Sciences. Biologically-active chromium (as found in ChromaTone), helps maximize the body's efficient use of insulin. Insulin regulates the metabolism of blood sugar, lipids (including cholesterol) and protein. Studies also indicate that chromium, when combined with appropriate exercise, has very positive muscle-tissue building and toning properties.

<u>Chromium</u> helps insulin perform, so it's needed by your cells to take up glucose. Thus when it is deficient, blood sugar levels can be elevated. Cholesterol and triglyceride levels rise too. Peripheral neuropathy has been reported, as has weight loss. Heavy exercise, infection, and injury increase its use, and hence its loss.

**Chromium** is found in good supply in brewer's yeast and in meats and cheeses.

The normal American diet is said to contain only about half of what we should have, but deficiencies have rarely been reported. And the above effects are the only ones we've seen.

The RDA, which is all we have to go by here, is fifty to two hundred micrograms. There are no studies reported on its importance in HIV.

Multivitamins contain from 15 to 100 micrograms of chromium. Trace element supplements can add another 100 micrograms or so.

### **ZINC SULFATE**

**ZINC SULFATE (4 – 12) 600mg maximum for 30 days.** Peak 9.32mg/kg/D max causes WORM EXPLOSION, 50 mg zinc sulfate monohydrate in each 220 capsule, Item No: 0802

http://www.Wonderlabs.com

http://www.ebay.com/itm/Rising-Zinc-Sulfate-220-mg-100-Capsules-Pack-of-3-/261936684663?hash=item3cfca49e77:g:O6EAAOSwyQtVhGGu

Zinc has been used since ancient Egyptian times to enhance wound healing, although the usefulness of this approach is only partially confirmed by the clinical data of today.

Zinc can be shuttled from blood to tissue in times of stress or illness. Thus plasma levels may not reflect its true concentration in the body. Zinc is absorbed in the small intestine. High-fiber diets and the presence of parasites can limit its absorption. Only 25 percent of what's ingested is absorbed, at best; with poor intestinal absorption, the amount can be even less.

Wound healing and the maintenance of membranes are among its tasks. It also plays a role in antibody production, and other aspects of immune response.

With zinc deficiency, immune response is impaired. Hair loss can result. Night vision is lost. We may think less clearly. Wound healing is slowed, and protein metabolism impaired.

Diarrhea can be both a cause and a result of zinc deficiency, and thus can compound the problem. Zinc should always be supplemented in people with severe, chronic diarrhea.

A reduction or change in our sense of taste can also result from zinc deficiency. This can be especially disturbing, as it affects both appetite and absorption.

**Zinc** is necessary for the functioning of more than 300 different enzymes and plays a vital role in an enormous number of biological processes. **Zinc** is a cofactor for the antioxidant enzyme superoxide dismutase (SOD) and is in a number of enzymatic reactions involved in carbohydrate and protein metabolism.

Its immune-enhancing activities include regulation of T lymphocytes, CD4, natural killer cells, and interleukin II. In addition, zinc has been claimed to possess antiviral activity. It has been shown to play a role in wound healing, especially following burns or surgical incisions. **Zinc** is necessary for the maturation of sperm and normal fetal development. It is involved in sensory perception (taste, smell, and vision) and controls the release of stored **vitamin A** from the liver.

**ZINC SULFATE** (4-5 typical TID) - this is nothing, you can take 12 capsules a day for a month easily, and according to the study, 85% of the subjects made it to the end. Within the endocrine system, **Zinc** has been shown to regulate insulin activity and promote the conversion thyroid hormones thyroxine to triiodothyronine. Many diseases have been cured with **Zinc** alone. It tells the immune system to crank out more white blood cells, it antagonizes iron, that parasites love to steal, and it helps transport oxygen. Short term use of high doses should be kept under a month or two, an overdose is evidenced by a **Zinc** taste in the mouth. My hair analysis shows I still have no zinc in my body, after 3 years of incredible dosing levels. This trick is a keeper for parasite infections.

**Zinc** is a double edged sword. **Zinc** helps the human body in crisis, it also helps some worms survive in the long term, it kills others. Why this happens is not exactly clear, but in the case of Ascaris, there is a study that indicates **Zinc and Vitamin A** need to be discontinued, to ultimately kill that specific parasite.

### <u>Manganese</u>

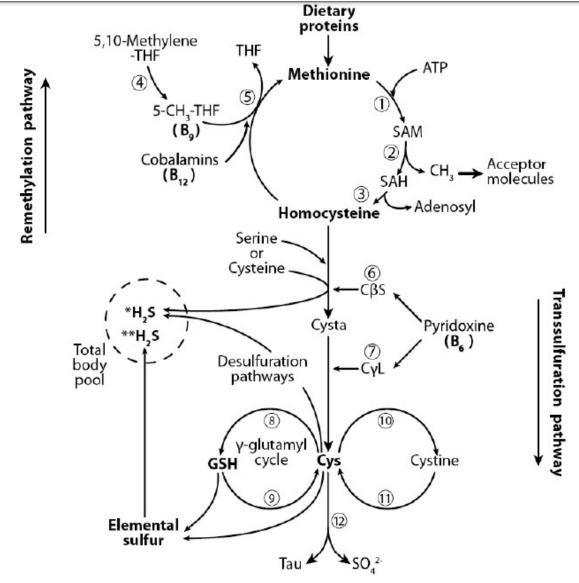
When manganese is bound or "chelated" with specific amino acids, it targets parts of the body to help metabolism. When manganese is chelated with the amino acids proline, histidine, and glutamic acid, it travels through the blood to the hypothalamus. The hypothalamus is the organ below the

brain which is responsible for regulating many body functions, including temperature, appetite and sleep. The chelated manganese increases the natural thyrotropin-releasing hormone secreted by the hypothalamus. This hormone, known as T.R.H., has a direct effect on the master gland (pituitary).

### Germanium

#### **Germanium**

This trace mineral is a potent antioxidant. It improves oxygen flow to cells, and strengthens the immune system. It has pain-relieving properties and studies have shown that organic germanium (sesquioxide) can nutritionally support the body's defense system. Many people have attributed remarkable benefits with the intake of Germanium. One woman had suffered many years with arthritis, nervous problems and a painful muscle disease called "fibrositis." She could not stand to even barely be touched because it caused her such agony. This person began taking one capsule daily of 30 mg. of germanium sesquioxide, which also had odorless **garlic** added. She said, "Now I'm on 'cloud nine' since taking Germanium ... and I have so much energy, I don't know what to do with it all!"



#### **Glutathione, Sulfur, Sulfur Amino antioxidants**

#### **How Antioxidants Work**

The body is made up of molecules, combinations of atoms constructed in a particular way to do a specific job.

Sulfur amino-acids (which command DNA actions) perform different roles in parasitic infections. Cysteine and Methionine can supplement parasites. The loops are best regulated by organic sulfur, I cysteine, and other techniques.

The molecules that take part in or regulate processes in your body are held together by the forces between their electrons, which have a sort of magnetic attraction to one another, and fit together based on that attraction.

Many molecules within your body exist periodically in what is called an oxidized state. This means that they have one or more unpaired electrons. They are looking for molecular partners, often at the

expense of other important molecular relationships. When they are in this state, they are called free radicals.

These free radicals can react much more with their surroundings, often in ways that are damaging. They can interact with and disrupt many finely tuned processes which are needed to maintain the body.

In some cases, the presence of these active molecules, or free radicals, can be helpful. Cells of your immune system, for example, rely on the destructive power of free radicals and use them as ammunition, discharging them where infection is present in order to kill the invaders. But along with the destruction of unwelcome cells or organisms, there can be damage to nearby tissues and processes your body needs.

In other cases, however, free radicals are simply an unavoidable by-product of body processes -- the leftovers, so to speak, of necessary reactions -- and serve no useful purpose. In these cases they are like static on the radio: they mess things up a little, but the music keeps on playing.

All of this is true for every living body.

Our bodies contain natural substances, called antioxidants, which can gather up and neutralize free radicals, limiting their capacity for destruction. Often there are not enough to do the whole job.

There are stages or circumstances in our lives in which free radicals and their damage are increased. Much of the deterioration we see in aging, for example, results from the presence of an increased amount of these **reactive molecules**. Our natural stores of antioxidants cannot successfully overcome the increase. In states of illness or infection, free radical presence is also increased and results in many of our symptoms.

Superfruits such as Mangosteen and Pomegranate that are valued for their very high

concentration of natural plant **antioxidant**s that may help boost and protect the body's immune system. Every day you're exposed to free radicals. Some are from outside factors, like pollution and radiation; and some from inside, like stress and hormones. Fight back with pomegranate shots, a super-concentrated punch of an ultra-potent natural

**antioxidant**. It has more free-radical- fighting power than red wine, grape seed and <u>acai</u> extracts.

**MANGOSTEEN**, known throughout Asia as the 'Queen of Fruits', is highly valued for it's **antioxidant** benefits. It's juice and rind contain a very high concentration of natural plant **antioxidant**s, called Xanthones, which may have numerous

health benefits and are richer in potency than both Vitamin  ${\bf C}$  and Vitamin  ${\bf E}$ .

<u>POMEGRANATE</u>, is loaded with Anthocyanins and polyphenol **antioxidant**s which may help boost and **protect** the body's immune system. Recent studies have suggested that pomegranate contains 3 times the **antioxidant** potency then the same amount of green tea.

POMx Shot (6 Bottles) 3 Ounces \$39.99

Shipping Weight: 5.1 ounces

ASIN: B0047CSB8U UPC: 824150464334 3 Ounces Liquid

Serving Size: 1 bottle (3 fl oz)

# ESTER C

https://www.prohealth.com/shop/product.cfm?product\_\_code=PH319
PRO-C COMPLEX™

We've created our **own-buffered vitamin** C, with **calcium for increased assimilation** and retention. In fact, research shows that buffered C is absorbed 400% faster than regular vitamin C and remains in the body as much as three-times longer. Its neutral pH makes it easy on the stomach, too!

PRO-C COMPLEX™ is synergistic blend of bioflavonoids, acerola, rose hips and rutin.\*

### **Citrus Bioflavoids Complex contains:**

Flavones
Herperidin
Eriocitrin
Naringin, Narirutin & other Flavones
Flavonols

#### **Fat based Vitamins**

Fat Malabsorption occurs as a result of bacterial deconjugation of **bile salts**. In addition, free bile acids are toxic to the intestinal mucosa, resulting in mucosal inflammation and malabsorption.63,64 Deconjugated **bile salts** are reabsorbed in the jejunum rather than the ileum, leading to impaired micelle formation, fat Malabsorption, and deficiencies in fat-soluble vitamins (A, D, E, and K). Fortunately, symptoms rarely develop; however, in severe cases night blindness (vitamin A), osteomalacia and tetany due to hypocalcemia (vitamin D), prolonged prothrombin times (vitamin K), or neuropathy, retinopathy, and impairments in T-cell function can occur.

#### Vitamin A

# Vitamin A - (0-2-6) 15000 IU Retinyl Palmitate, Carlson Labs P# 01102 Swanson CSN155 \$5.99

http://www.swansonvitamins.com/carlson-vitamin-a-palmitate-15000-iu-240-sgels?otherSize=CSN155

Lifetime maximum xxx Million IU?, one week up to 10X, can kill humans at 100X

Dry eyes, night blindness, and other eye conditions are symptoms of vitamin A deficiency. In extreme cases, blindness can result. Also, white cells can be reduced, as can red blood cells. Resistance to infection is impaired. Thus vitamin A deficiency can result in more, and more severe,

diseases of many types. Even a mild deficiency has been shown to increase the risk of pneumonia, diarrhea, and even death in children.

- Vitamin A is a subclass of a family of lipid-soluble compounds referred to as retinoic acids.
- Supports enzymes that catalyze retinyl esters.
- Help heal brush border membrane of small intestine.
- Prevent chronic liver disease, reverse portal hypertension
- Help prevent hearing loss, blindness
- Help prevent morbidity
- Help prevent mental illness.
- Retinoids, such as retinal and retinoic acid, are found in animal sources like liver, kidney, eggs, and dairy produce. Carotenoids like beta-carotene (which has the highest vitamin A activity) are found in plants such as dark or yellow vegetables and carrots.
- Research suggests that vitamin A may reduce fever, morbidity, and certain parasite blood levels in patients with malaria (Plasmodium falciparum infection).
- Vitamin A helps cell reproduction. It also stimulates immunity and is needed for formation of some hormones. Many research reports on the anti-cancer properties of vitamin A and the related retinoids have been published over the last 20 years. Most of these studies examined all-trans retinoic acid (RA). RA is formed in human tissues from beta-carotene and retinol, does not accumulate in the liver, thus it is not associated with significant hepatotoxicity. [24] Treatment with RA is associated with many side effects, including headache, lethargy, anorexia, vomiting, and visual disturbance. [24] Another retinoid used in cancer treatment is 13-Cis-retinoic acid (cRA), also known as isotretinoin. [25]
- RA in vitro demonstrates growth inhibitory activity against at least 14 types of human cancers. [24] Acute promyelocytic leukemia (APL) has been shown to respond well to RA, but not to cRA. [26] In one study, nine of 11 patients with APL entered complete remission after treatment with 45 mg/m2 daily oral dose of RA. [27] Similar results are reported elsewhere, [28,29] and have been confirmed in vitro. [30]
- Local application of an RA-containing cream demonstrated low toxicity and some histological improvement of cervical intraepithelial neoplasia II (CIN II) in a phase I study. [31]In a phase III trial, RA led to complete regression of CIN II in 42 percent of women compared with 27 percent in the placebo group. [32] No significant effect was noted in severe cervical dysplasia. [32] After remission induced by conventional therapy, treatment with cRA is associated with fewer second primary tumors in head and neck squamous-cell carcinoma. [33]
- Retinoic acid decreased the growth rate and increased differentiation of human small cell lung cancer lines in vitro. [34] Daily oral administration of 300,000 IU vitamin A as retinol palmitate led to a significant reduction in second primary tumors and an increase in diseasefree survival post-surgery in stage I lung cancer.

#### Carrot oil

Contents Oil: Carrot Seed is a highly nutritious plant, containing substantial amounts of Vitamins A, C, B1, and B2.

 Carrot seed oil also assists in removing toxin and water build

Research: Wikipedia1, Organic Vitamins and Supplements Kingdom:

**Organic Vitamins and Supplements** 



Kingdom: Plantae

Family: Apiaceae

Genus: Daucus

Latin Name: <u>Daucus carota</u> (Wikipedia)

Family Members: : angelica, anise, arracacha, asafoetida, caraway, carrot, celery, centella asiatica, chervil, cicely, coriander/cilantro, cumin, dill, fennel, hemlock, lovage, Queen Anne's Lace, parsley, parsnip, sea holly, and the now extinct silphium.

Preperation into product How it is madeBases:

Raw RootBases: Oil from seeds

Bases: DNA use D alpha e base for cq10 Ryboflavin: Phase 4 Dependant Antioxidant Chords: B, C, E

PhasesLong NameAKA Name: known as carrot or parsley family

Antioxidant Base for processes: Carrot seed oil removes toxin and water build-upBio Activators:Base:Oil: Not to be confused with the base carrot oil which should be blended with other base oils at 10%. The base oil contains many of the same properties. Base carrot oil is orange and processed from the root, carrot seed essential oil is yellowShort Name: Carrot Color: Yellow or amberDate

Discovered:Origon/Region: Plant Seeds, HungaryYear of Discovery: best technology: Best cultivation: seed HungaryMedical Properties: Antiseptic, diuretic, settle digestion, detoxifying, stimulant, tonic, smooth muscle relaxantClassification: VegtableForms: RAW, Water, Oil Contents: Raw roots have a strong tonic action on the liver and gall bladder, good for treatment of jaundice [ ] http://www.personalhealthfacts.com/aromatherapy/carrot-seed.htmlUSE: Physical Benefits:, Asthma, Bronchitis, Colds, Colitis, Emphysema, Flu, Gastric spasms, Gout, Hair loss, Headaches, Herpes, Hiccups, Migraines, Mouth ulcers, Muscle aches, Pains, Sinus congestion, Skin toner, Stress-related allergies, Varicose veins

**Hypervitaminosis A** refers to the effects of excessive vitamin A (specifically retinoid) intake. Effects include birth defects, liver problems, reduced bone mineral density that may result in osteoporosis, coarse bone growths, skin discoloration, hair loss, excessive skin dryness/peeling, a inflammatory lesion at the corner of the mouth, and often presents as deep cracks or splits in the skin.

### **Hypervitaminosis A**

**Hypervitaminosis A** occurs when the maximum limit for liver stores of <u>retinoids</u> is exceeded. The excess vitamin A enters the circulation causing systemic toxicity. **Betacarotene**, a <u>precursor</u> of vitamin A, is selectively converted into retinoids, so it does not cause toxicity.

Although **hypervitaminosis A** can occur when large amounts of liver (including cod liver oil and other fish oils) are regularly consumed, most cases of vitamin A toxicity result from an excess intake of vitamin A in the form of vitamin supplements. Toxic symptoms can also arise after consuming very large amounts of preformed vitamin A over a short period of time. The U.S. Institute of Medicine says that the Lowest Observed Adverse Effect Level (LOAEL) for vitamin A, when taken over an extended period of time is 21,600 IU.[2] Most

multivitamins contain vitamin A doses below 10,000 IU, therefore multi-vitamins are unlikely to cause vitamin A toxicity when taken at their recommended dosages. But in high doses, its central nervous system toxicity can be enhanced by its lipid solubility because it is readily transported across the blood brain barrier and concentrated in the brain.

Vitamin A causes cells to swell with fluid; too much vitamin A causes them to rupture in hyposmotic environments, hence the toxicity. Toxicity has been shown to be mitigated through vitamin E (tocopherol), cholesterol, zinc, taurine, and calcium.

The U.S. Institute of Medicine has established Daily Tolerable Upper Levels (UL) of intake for vitamin A from supplements that apply to **healthy populations**, in order to help prevent the risk of vitamin A toxicity. These levels for preformed vitamin A in micrograms (µg) and International Units (IU) are:

0–3 years: 600 μg or 2000 IU 4–8 years: 900 μg or 3000 IU 9–13 years: 1700 μg or 5665 IU 14–18 years: 2800 μg or 9335 IU 19+ years: 3000 μg or 10,000 IU

### **B** complex **B50**

### B50 (1) or B25 vitamin B multi B Complex - NOW or GNC 017913, Swanson

https://www.swansonvitamins.com/swanson-premium-balance-b-50-complex-250-caps? otherSize=SW057

Will turn urine yellow or orange when B12 saturates, take to keep urine yellow.

#### The "Enzyme Vitamin" - B

#### Vitamin B1 (Thiamine)

**Thiamine** is needed for conversion of carbohydrates into energy, for transmission of signals from your nerves to your muscles, and for maintaining the structure of membranes in the nervous system.

It is absorbed high in the small intestine and stored mainly in muscle tissue. Raw fish, **coffee**, and tea can break it down. Deficiency can occur quickly, as the body can't store this vitamin for long. Malabsorption, malnutrition, alcohol, diarrhea, and low folate levels can all contribute to **B1** deficiency. Antacids and other medicines that reduce stomach acidity can destroy it.

Need is increased with fever, heavy exercise, or high caloric intake.

Deficiency can result in weight loss, irritability, poor appetite, and paresthesias-the burning or prickling sensations we associate with peripheral neuropathy-especially in the feet and lower legs. When deficiency is more severe there can be weakness and changes in mental status.

One to two milligrams a day is said to prevent frank deficiency. Excess thiamine goes out in the urine, so too much can't hurt you.

Most multivitamins contain 1.5 to 3 milligrams of thiamine. High-potency pills and B-complex supplements contain 50 to 100 milligrams.

Dietary sources of thiamine include red meat, whole grains, potatoes, peas, beans, nuts, and yeast. As this nutrient is water-soluble, it can be lost when food is cooked in liquids.

### Vitamin B2 (Riboflavin)

**Riboflavin** is also absorbed in the small intestine. It is needed for many reactions in the body, and particularly for the metabolism-the energy economy-of amino acids, the basic units from which proteins are constructed. It is also needed to convert dietary vitamin B6 to its active form in the body.

Deficiency can result in burning and itching eyes, painful sensitivity to light, tongue and mouth pain, anemia, and personality changes. In addition, metabolism of drugs can be altered.

Three milligrams a day are recommended to prevent riboflavin deficiency. Again, if you don't need it, you excrete it.

Supplementation in standard multivitamins is in the range of 1.5 to 3.5 milligrams. High-potency sources and B-complex preparations contain 75 to 100 milligrams.

People who supplement their B2 notice that their urine shines a bright, fluorescent yellow. That's why you see this now, if you're taking a B-complex supplement.

Natural sources of riboflavin include dairy products, meat, fish, and green leafy vegetables. Whole grain cereals are also good sources, as are egg whites. Riboflavin is broken down by light, so exposure while cooking (by broiling, for instance) can deplete it.

#### Vitamin B3 (Niacin)

**Niacin** is needed for the metabolism of proteins, carbohydrates, and fats. It is absorbed throughout the intestine and excreted in the urine. In large doses, it can have beneficial effects on cholesterol and triglyceride levels.

Without serious malnutrition, we rarely see nutritionally based niacin deficiency. Classically, deficiency has been found only in people who eat a corn-based and otherwise unbalanced diet.

Five to 20 milligrams a day are enough to protect against niacin deficiency, and large amounts may not be good for you. What you get in your diet should go a long way toward meeting your needs. Beyond that, my patients stick to what they get in their multivitamin pills.

Symptoms of niacin deficiency include generalized weakness and indigestion. Headaches and insomnia can follow. Severe deficiency can cause characteristic skin rashes, massive and bloody diarrhea, and even dementia.

Symptoms of supplementation include flushing and temporary tingling and burning sensations. Over supplementation can cause vomiting, diarrhea, and even fainting, due to a fast heart rate and low blood pressure. At very high doses ulcers, liver damage, and high blood sugar can also result. This is not a vitamin to play around with.

Standard preparations contain 20 to 30 milligrams, which is a minimal amount. B-complex supplements can contain up to 100 milligrams. Even at that dose you may get some flushing and a characteristic prickling feeling.

### Vitamin B6 (Pyridoxine)

**Pyridoxine** is also absorbed in the small intestine, and any excess is excreted in the urine. Deficiency can develop in two or three weeks. Like vitamin B2, it is active in the metabolism of amino acids. It also plays a role in making neurotransmitters-the chemicals that brain cells use to communicate with one another. It is essential to many enzyme reactions.

Symptoms include irritability and depression, followed by skin rashes and tongue and mouth tenderness. Nausea and vomiting, as well as seizures, are late manifestations. B6 deficiency can also cause anemia, and it has been shown to further impair immune function.

Standard multivitamin preparations contain 2 to 5 milligrams. B-complex pills can have 5 to 100 milligrams.

Foods rich in B6 include meat, fish, egg yolks, beans, fruits, and vegetables. Liver is a good source, as are whole grain cereals. Losses occur during cooking.

#### **Biotin B7**

#### Biotin (2-4) 5mg Swanson sw877

Biotin - AM/PM 2 Biotin 5000 mcg Bluebonnet 00447/Swanson SW877 • 5 mg 100 Caps

<u>Biotin</u>, also known as **vitamin H** or **coenzyme R**,[2] is a water-soluble <u>B-vitamin</u> (**vitamin B**<sub>7</sub>).

It is composed of a ureido (tetrahydroimidizalone) ring fused with a <u>tetrahydrothiophene</u> ring. A <u>valeric acid</u> substituent is attached to one of the carbon atoms of the tetrahydrothiophene ring. Biotin is a <u>coenzyme</u> for <u>carboxylase</u> enzymes, involved in the synthesis of <u>fatty acids</u>, <u>isoleucine</u>, and <u>valine</u>, and in <u>gluconeogenesis</u>.

The only human health condition for which there is strong evidence of **biotin's** potential benefit as a treatment is biotin deficiency.[3]

All oxidation from gut inflammation has the potential to affect proteins. Oxidation is a normal process of cells that use oxygen to produce energy from various substrates, including those cells lining the intestinal tract. This process is called oxidative phosphorylation.

**Biotin** is an essential water-soluble B vitamin. The name <u>Biotin</u> is taken from the Greek word *bios* meaning "life." Without biotin, certain enzymes, including acetyl-CoA carboxylase and pyruvate carboxylase, do not work properly, and complications can occur involving the skin, intestinal tract, and nervous system. Metabolic problems including very low blood sugars between meals, high blood <u>ammonia</u>, or acidic blood (acidosis) can occur. Death is theoretically possible, although no clear cases have been reported. Recent studies suggest that biotin is also necessary for processes on the genetic level in cells (DNA replication and gene expression).

#### **Folate B9**

There is an epidemic of **folic acid** deficiency. **Folic acid** deficient people are extremely susceptible to viral, **fungal** and bacterial infections. Alcohol destroys folic acid stores in the

body. **Folic acid** has been shown to slow the onset of Alzheimer's and minimize the severity of symptoms. Mentally disturbed individuals show definite benefit by the addition of folic acid to their treatment. Folic acid also helps combat the circulation problem so common in our aging population; it acts as an effective dilator of small vessels.

Folic acid is a vitamin that stimulates the formation of normal red blood cells. Folic acid helps nourish the skin and nervous system

**Folate** changes into its active form after it has been absorbed by the body. It is excreted through the gastrointestinal tract. It is necessary for making red blood cells and for neurological function. Thus deficiencies in **folate**, as with vitamin **B12** deficiencies, are associated with neurologic symptoms. This can be particularly important to people with HIV. More **folate** is needed in the presence of severe infection, cancer, and pregnancy.

One report measured **folate** in the cerebrospinal fluid -- the fluid that bathes the brain and spinal cord -- in children infected with HIV. Results showed a lower level of folate in that fluid than in the blood. Thus we may be folate-deficient where we need it most, even when we test in the normal range. And, as is found with vitamin B12 deficiencies, the red-blood-cell changes normally associated with **folate** deficiency are often not seen in the presence of HIV.

Alcohol is a special villain. It also blocks folate absorption, and people who regularly use substantial amounts of alcohol are often seriously deficient. Vitamin B12 deficiency can also lower available folate levels, as it is needed to change folate into its active form.

Loss of appetite, nausea, diarrhea, hair loss, and mouth and tongue pain can all be symptoms of folate deficiency. Fatigue is common too. As things get worse, changes in blood cells may be seen.

**Folate** deficiencies are treated with 1 to 2 milligrams a day. One milligram or less a day is given thereafter for maintenance. Over supplementation is not thought to be dangerous. Leafy vegetables, organ meats, and yeast are good dietary sources.

Multivitamin <u>folate</u> levels are usually set at 4 milligrams. Folate is not normally included in B-complex preparations.

### Vitamin B12 (Cobalamin)

Vitamin B 12 is an Nitric Oxide scavenger and may be useful in detoxification.

Methylcobalamin B12 are nothing short of astounding. Research has shown it to provide significant protection for everything from Neuropathy (even restoration of feeling in feet and legs after years of total lack of feeling), to dramatic reversal of Bell's palsy, to improvements of chronic depression and a remarkable reduction in the frequency/severity of disabling headaches and improvements in neurological conditions.

Benefits in prevention of Alzheimer's, aging, cancer and arteriosclerosis have been realized. The most important of the B Vitamins, Studies have found that some people committed for life to mental institutions could be restored to full functioning simply by correcting their Vitamin **B-12** deficiency, which are associated with mild disorders of mood, Mental Changes from B12 Deficiency, to severe psychotic symptoms.

Absorption of vitamin **B12** is more difficult than that of the other B vitamins. Cells in the stomach produce a factor which binds to **B12** and permits it to be absorbed in the small intestine. Thus there are two points in its journey at which oral absorption can be impaired.

On the other hand, the body can recycle some of what gets in, shuttling it back and forth between the intestine and the liver, for reuptake and reuse. It is stored in the liver in ample quantities, so B12 deficiency takes longer to occur than other B-vitamin deficiencies.

Doctors may also not consider the enormous contribution of malabsorption to nutritional status and disease progression, which might otherwise encourage them to check **B12** levels automatically. Thus, when not checked routinely, **B12** deficiency can often be missed.

A low level of vitamin **B12** is especially because of its potential role in problems with nerve conduction or function (neuropathy) and spinal cord abnormalities (myelopathy). they have substantial impact on quality of life. In Studies, people presenting with neuropathy or myelopathy were low in B12. Of those who had both conditions, vitamin B12 was found to be low in more than half.

Vitamin **B12** deficiency has also been associated with early, subtle changes in mental function in people. Those changes include the speed with which we process information, and our performance on tasks requiring visual-spatial coordination. Because they are subtle, we may not pick these changes up.

Vitamin **B12** is provided in the diet by meat, fish, and eggs, so vegetarians are particularly at risk. It can be obtained in lesser amounts from milk and milk products. Generally, it is not destroyed by cooking.

Your basic multivitamin has 6 to 18 micrograms of **B12**. B-complex preparations include from 12 to 500 micrograms. Separate B12 oral supplements are also available, in doses from 25 micrograms to 1 milligram. An excess of B12 won't hurt you, but it may not be necessary.

We'll talk more about vitamin **B12** supplementation later. Because absorption is so frequently a factor in B12 deficiency, you may need some help from your doctor. Mushrooms, Tomatoes, Artichokes, Lima beans, Onions, yellow snap Beans, raw Parsnips, green peas, carrots, Brussels sprouts, Cabbage, Cauliflower, raw skin Potatoes, yellow sweat Corn, Potatoes O'Brian home prepared, Cowpeas,

Shellfish are a great source of vitamin **B12** and can be eaten raw, baked, steamed, fried, or made into chowder.

Popular in Mediterranean, Japanese, and Hawaiian cuisine, octopus is a vitamin **b12** rich food.

Known for their omega 3 fats and for being a high protein food, fish are also a good source of vitamin **B12**. Mackerel provides the most vitamin **B-12** with 19μg per 100g serving (317% RDA), followed by Herring (312% RDA), Salmon (302%), Tuna (181%), Cod (167%), Sardines (149%), Trout (130%), and Bluefish (104%).

Crab and lobster are most commonly served baked, steamed, or in bisque. A 100g serving of crab contains 11.5µg of vitamin **B12** (192% of the RDA),

Beef is also a good source of protein, zinc, and heme-iron. The amount of vitamin  $\underline{\textbf{B-12}}$  in beef depends on the cut, lean fat-trimmed chuck contains the most vitamin  $\underline{\textbf{B12}}$  with 6.18µg (103% RDA) per 100g serving, 11.49µg (103% RDA) in a chuck steak, and 5.25µg (88% RDA) in a 3 ounce serving. Chuck is followed by sirloin (62% RDA), rib-eye (60% RDA), and ribs (58% RDA).

Cheese is a good source of calcium, protein, and Riboflavin (Vitamin B2).

When it comes to chicken eggs the raw yellow has most of the vitamin **B-12** with 1.95µg per 100g serving (33%), however, this equates to 0.33µg per yolk or just 6% of the RDA.

#### **Ester C**

**Ester C** - (1-2-6-8) 1000mg with Citrus Bioflavonoids, American Health P#16984 Swanson AM123, AM122 AM091

https://www.swansonvitamins.com/american-health-ester-c-citrus-bioflavonoids-1000-mg-120-veg-tab

- We do not produce our own vitamin C it must be obtained from our diet. Science
  continues to confirm, with ever increasing evidence, the benefits of the C vitamin
  family to especially nourish the body's structural and defense systems. Vitamin C
  strengthens cells and tissues and helps build the body's defense system.
- Excellent food sources are oranges, grapefruits, lemon and lime juice, tomatoes, and strawberries. Potatoes, broccoli, cabbage also have some vitamin C. And, green chillies, if you can take them, are rich, rich, rich in Vitamin C!
- To ensure you're getting the right amount of C, take a nutritional supplement.

  It's a wise move for your overall health but particularly for your heart. We don't take any chances with this one!
- Recent studies on vitamin C at the National Institutes of Health and the Harvard School of Public Health,
- found that vitamin C supplementation appears to protect against heart disease.
- The American Journal of Clinical Nutrition reported that an adequate intake of vitamin C might protect against stroke and heart attack by lowering blood pressure and LDL ("bad cholesterol") levels, and helping to thin the blood to protect against clots. It also helps prevent atherosclerosis (commonly known as hardening of the arteries) by strengthening the artery walls as it manufactures collagen, the protein that gives shape to connective tissues and strength to skin and blood vessels.
- **Vitamin C** is one of the most important of all vitamins. It plays a significant role as an antioxidant, thereby protecting body tissue from the damage of oxidation. Antioxidants act to protect your cells against the effects of free radicals, which are potentially damaging byproducts of the body's metabolism. Free radicals can cause cell damage that may contribute to the development of cardiovascular disease and cancer. Vitamin C has also been found by scientists to be an effective antiviral agent.
- **<u>Buffered vitamin C</u>**, with <u>calcium</u> for increased assimilation and retention. In fact, research shows that buffered C is absorbed 400% faster than regular <u>vitamin C</u> and remains in the body as much as three-times longer. Its neutral pH makes it easy on the stomach, too!
- Research has demonstrated that this combination of non-acidic C with metabolites is well
  absorbed and retained in blood cells and tissues. <u>Ester C</u> is enhanced by bioflavonoids,
  acerola, rose hips and rutin.\*

Citrus Bioflavoids Complex contains:

- Flavones
- Herperidin
- Eriocitrin
- Naringin, Narirutin & other Flavones
- Flavonols

### Vitamin D3

D3 - (1) 1000 IU Vitamin D Swanson SW1030 D3-5 5000 IU NOW 3072 D3-50 (1) 50,000 IU Bioteh Fayettville AK

12,500 times daily value, can be done for a few weeks strait, thereafter lower dose.

https://www.biotechpharmacal.com/catalog/d3-50-50000-iu/

Immune system support, helps flush out infections, excessive D can suppress immune function, best taken in pulsed high dosing, daily long term level should not exceed 1000 iu.

#### Vitamin D is an oil-soluble vitamin

- This substance seems to act more like a hormone than a vitamin. Hormones affect our various systems, making those systems run better. Vitamin D regulates and enhances the body's production of certain antimicrobial peptides called "bacterioncins." These peptides attack bacteria, mold, fungi, and viruses by dissolving their cell walls. At this same time, the body responds with inflammation, and vitamin D takes a roll in this process by preventing the immune system from releasing too many inflammatory agents (cytokines) especially into the lung area.
- It helps to absorb dietary calcium and phosphorus from the intestines.
- It suppresses the release of parathyroid hormone, a hormone that causes bone resorption.
- Helps prevent Calcium dumping, maintains PH stores of Calcium
- Responsible for intestinal absorption of calcium and phosphate.
- Essential for good health, helps prevent premature aging, prevention of cancer
- Essential for endocrine system, intestine, cardiovascular, nerve function, tyrosine expression, adrenal gland function, bile acid production,
- **<u>Vitamin D</u>** supplements both innate and adaptive immunity, and hormone function.
- Antiviral supplement.
- Insufficiency leads to genome wide degradation.
- Increased Brain Healing, and Memory

Toxic

100,000 IU

300,000 IU

30,000 IU

30,000 IU

30,000 IU

3 10 30 100

Weeks of daily dose

Researchers have discovered that Vitamin D is required to metabolize stored fat and that more than 60% of people tested are severely deficient. You can help your body burn abdominal fat and lose weight naturally by regulating your vitamin D intake. The results of a study published in the *Journal of Clinical Endocrinology and Metabolism* reveal that 59% of young women tested had too little

circulating vitamin D and nearly one-quarter were grossly deficient in the active form of the vitamin. Vitamin D insufficiency has been shown to be a factor in the accumulation of excess body fat as the fat-soluble vitamin becomes locked away in adipocytes (fat cells). In the absence of sufficient vitamin D, the body increases the number and size of newly formed fat cells that promote and accelerate abdominal obesity.

It's been discovered that people lacking in vitamin D catch more upper respiratory illnesses than those with sufficient vitamin D. This is another reason that the cold and flu season hits in the winter months when there is less sun to make vitamin D and we simply do not spend as much time out of doors.

**Vitamin D** appears to have effects on immune function.[32] It has been postulated to play a <u>role in influenza</u> with lack of vitamin D synthesis during the winter as one explanation for high rates of influenza infection during the winter.[33] For viral infections, other implicated factors include low relative humidities produced by indoor heating and low temperatures that favor virus spread.[34] <u>In healthy adults</u>, sustained intake of more than 1250 micrograms/day (50,000 IU) can produce overtoxicity after several months;[79] those with certain medical conditions such as primaryhyperparathyroidism[80] are far more sensitive to vitamin D and develop <u>hypercalcemia</u> in response to any increase in vitamin D nutrition. main symptoms of vitamin D overdose are those of hypercalcemia: <u>anorexia</u>, nausea, and vomiting can occur, frequently followed by <u>polyuria</u>, <u>polydipsia</u>, weakness, insomnia, nervousness, <u>pruritus</u>, and, ultimately, <u>renal failure</u>. <u>Proteinuria</u>, <u>urinary casts</u>, <u>azotemia</u>, and <u>metastatic calcification</u> (especially in the kidneys) may develop.

• Vitamin D2 may be useful in **fungal** infections.

### Vitamin E

### Vitamin E - AM (1-3) D alpha e 800 IU CAP.CARLSON 00381, SWANSON SW145,

4 or more may cause shakes.

Deactivates wormwood effectiveness on parasites

Vitamin E is a powerful antioxidant and helps the body cope with toxic substances. It also helps protect the cells and increase oxygen to them.

#### http://lpi.oregonstate.edu/infocenter/vitamins/vitaminE/

During feeding experiments with rats <a href="Herbert McLean Evans">Herbert McLean Evans</a> concluded in 1922 that besides vitamins B and C, an unknown vitamin existed. <a href="[11]">[11]</a> Although every other nutrition was present, the rats were not fertile. This condition could be changed by additional feeding with wheat germ. It took several years until 1936 when the substance was isolated from wheat germ and the formula C29H50O2 was determined. Evans also found that the compound reacted like an <a href="alcohol">alcohol</a> and concluded that one of the oxygen atoms was part of an OH (hydroxyl) group. As noted in the introduction, the vitamin was given its name by Evans from Greek words meaning "to bear young" with the addition of the -ol as an alcohol. <a href="[12]">[12]</a> The structure was determined shortly thereafter in 1938. <a href="[13]">[13]</a>

Vitamin E deficiency causes neurological problems due to poor nerve conduction. These include neuromuscular problems such as <u>spinocerebellar ataxia</u> and <u>myopathies</u>.[8] Deficiency can also cause <u>anemia</u>, due to oxidative damage to red blood cells.

**Vitamin E** is proberbly the most misunderstood vitamin. **Vitamin E** exists in eight different forms, four tocopherols and four tocotrienols. All feature a chromanol ring, with a hydroxyl group that can donate a hydrogen atom to reduce free radicals and a hydrophobic side chain which allows for penetration into biological membranes.

Both the tocopherols and tocotrienols occur in alpha, beta, gamma and delta forms, determined by the number and position of methyl groups on the chromanol ring.

<u>Natural</u> alpha-tocopherol is the <u>RRR-alpha</u> (or ddd-alpha) form. The synthetic dl,dl,dl-alpha ("dl-alpha") form is not as active as the natural ddd-alpha ("d-alpha") tocopherol form.

### Mixed tocopherols

"Mixed tocopherols" in the US contain at least 20% w/w other natural R, R,R- tocopherols, i.e. R, R,R-alpha-tocopherol content plus at least 25% R, R,R-beta-, R, R,R-gamma-, R, R,R-delta-tocopherols.

Some brands may contain 200% w/w or more of the other tocopherols and measurable tocotrienols. Some mixed tocopherols with higher gamma-tocopherol content are marketed as "High Gamma-Tocopherol." The label should report each component in milligrams, except R, R,R-alpha-tocopherol may still be reported in IU. Mixed tocopherols can also be found in other nutritional supplements.

Vitamin E is absorbed in and with fat; it requires pancreatic and biliary enzymes for absorption. Its antioxidant properties serve to protect and stabilize cell membranes. It is found in vegetable oils, and to a lesser degree in eggs, whole grain cereals, and butter. You can get a little from vegetables. Frank deficiency is rare and takes a long time to occur, but can be seen when there is lasting, severe fat malabsorption. It can also be seen after long-term TPN administration. Effects of vitamin E deficiency include peripheral neuropathy, poor position sense and balance, and a reduction in kneejerk and other reflexes.

Cell membranes have a fat, or lipid, layer. Free radicals in these membranes react with oxygen and initiate a chain reaction forming new free radicals at every step in the chain. Vitamin E counters this process by entering the lipid membrane and uniting with the free radical. The molecule which results has a different shape; it sticks its head out of the membrane, where it becomes visible to vitamin C. When attacked by vitamin C, it can be reduced back to a stable molecule, and the chain of damage is halted.

It's important to know what kind of vitamin E you're taking. If its not alpha tocopherol, which is naturally produced, Vitamin C won't recognize it, and you won't get all of this effect. If it's gamma tocopherol, such as is found in soybean oil, it will be excreted quickly. So you should try to find supplements which contain alpha tocopherols. These have the most activity. Most vitamin E is sold in the alpha form.

### **Vitamin E protection**

Vitamin E is a fat-soluble compound and an antioxidant and protects cell membranes from oxidation and destruction. Approximately eight naturally occurring vitamin E compounds have been described, including alpha-, beta-, gamma-, and delta- tocopherol. The only forms of tocopherol that are efficiently maintained in human plasma are four of the many isomers of alpha-tocopherol (RRR-, RSR-, RRS-, and RSS- alpha-tocopherol, which are present in "all racemic" synthetic vitamin E; only the RRR-form is present in foods). These are the forms of vitamin E that are most biologically active.

- Lowers LDL oxidation rate
- Prevention of cardiovascular disease, blood cell deformities, colitis, digestion loss, vision loss,
- Resist cyst formation

- Resist billiary cirrhosis, malabsorbtion, maintains balance of lipid soluble vitamins
- Acute Anterior Uveitis (in Combination With Vitamin C )
- Alzheimer's Disease
- Cancer Treatment Support
- Cataracts
- Cyclical Mastalgia
- Deep Venous Thrombosis (Prevention)
- Diabetic Neuropathy and Other Complications of Diabetes
- Diabetic Neuropathy
- Epilepsy
- Immune Support
- Macular Degeneration
- Male Infertility
- Menopausal Symptoms
- Menstrual Pain
- Premenstrual Syndrome (PMS)
- Preeclampsia (Prevention)
- Restless Legs Syndrome
- Rheumatoid Arthritis
- Sports Performance
- Tardive Dyskinesia
- Vascular Dementia In general, food sources with the highest concentrations of vitamin E are vegetable oils, followed by nuts and seeds including whole grains. Adjusting for typical portion sizes, however, for many people in the United States the most important sources of vitamin E include commercial breakfast cereal and tomato sauce. [17] Although originally extracted from wheat germ oil, most natural vitamin E supplements are now derived from vegetable oils, usually soybean oil.

#### **Vitamin E content**

In general, food sources with the highest concentrations of vitamin E are vegetable oils, followed by nuts and seeds including whole grains. Adjusting for typical portion sizes, however, for many people in the United States the most important sources of vitamin E include commercial breakfast cereal and tomato sauce.[16] Although originally extracted from wheat germ oil, most natural vitamin E supplements are now derived from vegetable oils, usually soybean oil.

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per 100 g of source include: [18][19]
Wheat germ oil (215.4 mg)
Sunflower oil (55.8 mg)
Almond oil (39.2 mg)
Sunflower seed (35.17 mg)
<u>Almond</u> (26.2 mg)
Hazelnut (26.0 mg)
Walnut oil (20.0 mg)
Peanut oil (17.2 mg)
Olive oil (12.0 mg)
Poppyseed oil (11.4 mg)
<u>Peanut</u> (9.0 mg)
Pollard (2.4 mg)
Maize (2.0 mg)
Poppy seed (1.8 mg)
Asparagus (1.5 mg)
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Oats (1.5 mg)
Chestnut (1.2 mg)
Coconut (1.0 mg)
Tomatoes (0.9 mg)
Walnut (0.7 mg)
Carrots (0.6 mg)
Goat's milk (0.1 mg)

### **Tocopherol**

Tocopherols (or TCP) are a class of chemical compounds of which many have vitamin E activity. It is a series of organic compounds consisting of various methylated phenols. Because the vitamin activity was first identified in 1936 from a dietary fertility factor in rats, it was given the name "tocopherol" from the Greek words "τόκος" [birth], and "φέρειν", [to bear or carry] meaning in sum "to carry a pregnancy," with the ending "-ol" signifying its status as a chemical alcohol.

Tocopherols are fat-soluble <u>antioxidants</u>.

http://en.wikipedia.org/wiki/Tocopherol

<u>alpha-Tocopherol</u> is the main source found in supplements and in the European diet, where the main dietary sources are olive and sunflower oils, while *gamma*-tocopherol is the most common form in the American diet due to a higher intake of soybean and corn oil., [1][2]

alpha-Tocopherol is the main source found in supplements and in the European diet, [citation needed] while gamma-tocopherol is the most common form in the American diet. [1] The compound a-tocopherol, a common form of tocopherol added to food products, is denoted by the E number E307.

### **Vitamin E Forms**

<u>Vitamin E</u> exists in eight different forms, four tocopherols and four <u>tocotrienols</u>. All feature a chromanol ring, with a <u>hydroxyl</u> group that can donate a <u>hydrogen</u> atom to <u>reduce free</u> <u>radicals</u> and a <u>hydrophobic side chain</u> which allows for penetration into <u>biological</u> membranes.

Both the tocopherols and <u>tocotrienols</u> occur in alpha, beta, gamma and delta forms, determined by the number and position of <u>methyl</u> groups on the chromanol ring.

### **Alpha-tocopherol**

Main article: alpha-Tocopherol

Alpha-tocopherol is the form of vitamin E that is preferentially absorbed and accumulated in humans. [7] The measurement of "vitamin E" activity in <u>international units</u> (IU) was based on fertility enhancement by the prevention of miscarriages in pregnant rats relative to alphatocopherol.

Although the mono-methylated form ddd-gamma-tocopherol is the most prevalent form of vitamin E in oils, there is evidence that rats can methylate this form to the preferred alphatocopherol, since several generations of rats retained alpha-tocopherol tissue levels, even when fed only gamma-tocopherol through their lives.

There are three <u>stereocenters</u> in alpha-tocopherol, so this is a <u>chiral</u> molecule. [8] The eight <u>stereoisomers</u> of alpha-tocopherol differ in the arrangement of groups around these stereocenters. In the image of *RRR*-alpha-tocopherol below, all three stereocenters are in the *R* form. However, if the middle of the three stereocenters were changed (so the hydrogen was now pointing down and the <u>methyl group</u> pointing up), this would become the structure of *RSR*-alpha-tocopherol. These stereoisomers can also be named in an alternative older nomenclature, where the stereocenters are either in the *d* or *l* form. [9]

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*RRR* <u>stereoisomer</u> of alpha-tocopherol, bonds around the <u>stereocenters</u> are shown as dashed lines (pointing down) or wedges (pointing up).

1 IU of tocopherol is defined as  $\frac{2}{3}$  milligrams of RRR-alpha-tocopherol (formerly named d-alpha-tocopherol or sometimes ddd-alpha-tocopherol). 1 IU is also defined as 1 milligram of an equal mix of the eight stereoisomers, which is a <u>racemic mixture</u> called <u>all-rac-alpha-tocopheryl acetate</u>. This mix of stereoisomers is often called dl-alpha-tocopheryl acetate, even though it is more precisely dl,dl,dl-alpha-tocopheryl acetate). However, 1 IU of this racemic mixture is not now considered equivalent to 1 IU of natural (RRR) a-tocopherol, and the <u>Institute of Medicine</u> and the <u>USDA</u> now convert IU's of the racemic mixture to milligrams of equivalent RRR using 1 IU racemic mixture = 0.45 "milligrams a-tocopherol". [10]

### **Tocotrienols**

Tocotrienols, although less commonly known, also belong to the vitamin E family. Tocotrienols have four natural 2' d-isomers (they have a stereoisomeric carbon only at the 2' ring-tail position). The four tocotrienols (in order of decreasing methylation: d-alpha, d-beta, d-gamma, and d-delta-tocotrienol) have structures corresponding to the four tocopherols, except with an unsaturated bond in each of the three <a href="isoprene">isoprene</a> units that form the hydrocarbon tail, whereas tocopherols have a saturated phytyl tail (the phytyl tail of tocopherols gives the possibility for 2 more stereoisomeric sites in these molecules that tocotrienols do not have). Tocotrienol has been subject to fewer clinical studies and seen less research as compared to tocopherol. However, there is growing interest in the health effects of these compounds. [11]

# d-alpha tocopherol (FOR cq10)

### **Alpha-tocopherol**

In the U.S., the average intake of alpha-tocopherol from food (including enriched and fortified sources) for individuals 2 years and older is 6.9 mg/day (14); this level is well below the RDA of 15 mg/day of *RRR*-alpha-tocopherol (4). Many scientists believe it is difficult for an individual to consume more than 15 mg/day of alpha-tocopherol from food alone without increasing fat intake above recommended levels. All alpha-tocopherol in food is the form of the <u>isomer</u> *RRR*-alpha-tocopherol. The same is not always true for supplements. Vitamin E supplements generally contain 100 IU to 1,000 IU of alpha-tocopherol. Supplements made from entirely natural sources contain only *RRR*-alpha-tocopherol (also labeled *d*-alpha-tocopherol). *RRR*-alpha-tocopherol is the isomer preferred for use by the body, making it the

most <u>bioavailable</u> form of alpha-tocopherol. Synthetic alpha-tocopherol, which is often found in fortified foods and nutritional supplements, is usually labeled *all-rac-*alpha-tocopherol or *dl-*alpha-tocopherol, meaning that all eight isomers of alpha-tocopherol are present in the mixture. Because half of the isomers of alpha-tocopherol present in *all-rac-*alpha-tocopherol are not usable by the body, synthetic alpha-tocopherol is less bioavailable and only half as potent. To calculate the number of mg of bioavailable alpha-tocopherol present in a supplement, use the following formulas:

### RRR-alpha-tocopherol (natural or d-alpha-tocopherol):

IU x 0.67 = mg RRR-alpha-tocopherol.

Example: 100 IU = 67 mg

all-rac-alpha-tocopherol (synthetic or dl-alpha-tocopherol):

IU x 0.45 = mg RRR-alpha-tocopherol.

Example: 100 IU = 45 mg

#### **Tocotrienols**

Tocotrienols, which are related compounds, also have vitamin E activity. All of these various derivatives with vitamin activity may correctly be referred to as "**vitamin E.**" Tocopherols and tocotrienols are fat-soluble antioxidants but also seem to have many other functions in the body.

The tocotrienols have the same methyl structure at the ring and the same Greek letter-methyl-notation, but differ from the analogous tocopherols by the presence of three double bonds in the hydrophobic side chain. The unsaturation of the tails gives tocotrienols only a single stereoisomeric carbon (and thus two possible isomers per structural formula, one of which occurs naturally), whereas tocopherols have 3 centers (and eight possible stereoisomers per structural formula, again, only one of which occurs naturally).

Each form has slightly different biological activity.[2] In general, the unnatural l-isomers of tocotrienols lack almost all vitamin activity, and half of the possible 8 isomers of the tocopherols (those with 2S chirality at the ring-tail junction) also lack vitamin activity. Of the stereoisomers which retain activity, increasing methylation, especially full methylation to the alpha-form, increases vitamin activity. In tocopherols, this is due to the preference of the tocophrol binding protein for the alpha-tocopherol form of the vitamin.

As a food additive, tocopherol is labeled with these E numbers:

E306 (tocopherol),

E307 (a-tocopherol/alpha-tocopherol),

E308 (y-tocopherol/gamma-tocopherol), and

E309 ( $\delta$ -tocopherol/delta-tocopherol).

These are all approved in the USA,[3] EU[4] and Australia and New Zealand[5] for use as antioxidants.

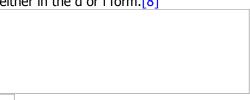
### **Vitamin E Alpha-tocopherol**

Alpha-tocopherol is the form of <u>vitamin E</u> that is preferentially <u>absorbed and accumulated in humans</u>.[6] The measurement of "<u>vitamin E</u>" activity in international units (IU) was based on fertility enhancement by the prevention of miscarriages in pregnant rats relative to alpha-tocopherol.

Although the mono-methylated form ddd-gamma-tocopherol is the most prevalent form of **vitamin E** in oils, there is evidence that rats can methylate this form to the preferred alpha-tocopherol, since

several generations of rats retained alpha-tocopherol tissue levels, even when fed only gamma-tocopherol through their lives.

There are three stereocenters in alpha-tocopherol, so this is a chiral molecule.[7] The eight stereoisomers of alpha-tocopherol differ in the arrangement of groups around these stereocenters. In the image of RRR-alpha-tocopherol below, all three stereocenters are in the R form. However, if the middle of the three stereocenters were changed (so the hydrogen was now pointing down and the methyl group pointing up), this would become the structure of RSR-alpha-tocopherol. These stereoisomers can also be named in an alternative older nomenclature, where the stereocenters are either in the d or I form.[8]



RRR stereoisomer of alpha-tocopherol, bonds around the stereocenters are shown as dashed lines (pointing down) or wedges (pointing up).

1 IU of tocopherol is defined as ¾ milligrams of RRR-alpha-tocopherol (formerly named d-alpha-tocopherol or sometimes ddd-alpha-tocopherol). 1 IU is also defined as 1 milligram of an equal mix of the eight stereoisomers, which is a racemic mixture called all-rac-alpha-tocopheryl acetate. This mix of stereoisomers is often called dl-alpha-tocopheryl acetate, even though it is more precisely dl,dl,dl-alpha-tocopheryl acetate). However, 1 IU of this racemic mixture is not now considered equivalent to 1 IU of natural (RRR) α-tocopherol, and the Institute of Medicine and the USDA now convert IU's of the racemic mixture to milligrams of equivalent RRR using 1 IU racemic mixture = 0.45 "milligrams α-tocopherol".[9]

#### Vitamin E Tocotrienols

Tocotrienols, although less commonly known, also belong to the <u>vitamin E</u> family. Tocotrienols have four natural 2' d-isomers (they have a stereoisomeric carbon only at the 2' ring-tail position). The four

### Vitamin F

#### **NOW Foods EFA DHA**

Vitamin F, also known as Essential Fatty Acids (EFA), cannot be made from the body - they must be supplied in the diet. Essential fatty acids nourish the body at the very foundation of health . . . at the cellular level. They strengthen cell membranes to fortify against the invasion of harmful microorganisms. These nutrients also help dissolve body fat and increase metabolism and energy production. Thus, they are also very helpful in a comprehensive weight management program.

### Vitamin K

#### Vitamin K K2 PM 1-3 K2 100 mcg NOW 0990

Salicylates can also block Vitamin K. This may be important for some people with fibromyalgia, as easy bruising is a common symptom in some people with fibromyalgia, and a vitamin K deficiency could cause this. However, Vitamin K does much more than this. It is also a significant antioxidant, controls insulin release, and is important in protecting

osteoporosis. Additionally, vitamin K reduces IL-6, an inflammatory cytokine, which some people theorize places a role in creating fibromyalgia pain. Vitamin K deficiencies have also been linked to mitral valve prolapse and hypermobility, both conditions which also commonly overlap in some people with fibromyalgia. Therefore, reducing salicylates may be helping some people with fibromyalgia by increasing levels of vitamin K.

The best source of <u>Vitamin K</u> is found in Salads and Green Vegetables. <u>Vitamin K</u> is fatsoluble and plays a critical role in blood clotting. It regulates blood calcium levels and activates at least 3 proteins involved in bone health.

**Vitamin K** occurs in two forms. It can be obtained in the diet from green leafy vegetables and liver. Bacteria in the gut provide another form, which is less active. Vitamin K is stored in limited amounts in the liver, where it is used to make factors that promote blood clotting. Thus a **vitamin K** deficiency can result in anticoagulation. If the liver is damaged, it may not be able to use vitamin K, even when present or provided in adequate amounts.

Malabsorption or the long-term use of powerful antibiotics, which sterilize the gut, can lead to a **vitamin K** deficiency. So can long-term use of TPN. Such deficiencies respond to injections of vitamin K, if the liver is healthy.

When a deficiency is documented in people with long-term malabsorption and diarrhea.

#### **QGEL** water-soluble CQ10 for the kidneys

https://www.google.com/search?q=Qgel+for+kidneys&ie=utf-8&oe=utf-8

### **CQ10**

CO10 (1-4) 400 mg with Vitamin E (as d-alpha Tocopherol), NOW Foods 3198

1-2 CQ10 400 mg with Vitamin E (as d-alpha Tocopherol 30 IU)

http://www.swansonvitamins.com/now-foods-coq10-400-mg-60-sgels

NOW Foods 400 mg 60 Sgels Item: NWF739 Swanson \$41.99

Cofactor: Works with <u>vitamin e</u> and **ALA**, original Japanese formulation, most studied form of **CO10**.

**CoQ10** is notoriously difficult to absorb. When it comes to choosing a **CoQ10** supplement, the primary factor is how many swallowed milligrams actually make it into your bloodstream.

#### **CQ10** Activity

Coenzyme Q10 (CoQ10) is an essential component of healthy mitochondrial function. It is incorporated into cells' mitochondria throughout the body where it facilitates and regulates the oxidation of fats and sugars into energy. Aging humans have been found to have over 50% less CoQ10 on average compared to that of young adults. This finding makes CoQ10 one of the most important nutrients for people over 30 to supplement with. About 95% of cellular energy is produced in the mitochondria. The mitochondria are the cells "energy powerhouses" and many maladies have been referred to as "mitochondrial disorders." A growing body of scientific research links a deficiency of CoQ10 to age-related mitochondrial disorders.

The biosynthesis of **CoQ10** from the amino acid tyrosine is a multistage process requiring at least eight vitamins and several trace elements. Coenzymes are cofactors upon which the comparatively large and complex enzymes absolutely depend for their function. Coenzyme Q10 is the coenzyme for at least three mitochondrial enzymes

The mitochondria are the cells' energy powerhouses, and **coenzyme Q10** (CoQ10) is an essential component of healthy mitochondrial function.16 CoQ10 is required to convert the energy from fats and sugars into usable cellular energy. Yet, the body's production of CoQ10 declines significantly with advancing age.17 With an ample amount of CoQ10, mitochondria can work most efficiently throughout the entire body—including the most densely populated area, the heart.18 CoQ10 is also a potent antioxidant, helping protect proteins, and DNA of mitochondria from oxidation and supporting mitochondrial function.16

**Coenzyme Q10** is the coenzyme for at least three mitochondrial enzymes (complexes I, II and III) as well as enzymes in other parts of the cell. Mitochondrial enzymes of the oxidative phosphorylation pathway are essential for the production of the high-energy phosphate, adenosine triphosphate (ATP), upon which all cellular functions depend.

**Coenzyme Q10** (CoQ10). CoQ10 is a fat-soluble, vitamin-like compound found in every human cell. It's used by the mitochondria ("power plants") of cells to produce energy. CoQ10 is found in highest concentration in cells of organs that require large amounts of energy, such as the heart. It also functions as an antioxidant. While the body produces **CoQ10** on its own, levels decline over time, with a steady decrease beginning after age 30. Factors such as aging, genetics, and cholesterollowering statins can lead to a **CoQ10** deficiency

#### COENZYME Q10 By PETER H. LANGSJOEN, M.D., F.A.C.C.

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#### **CQ10 DEFINITION**

Coenzyme Q10 (CoQ 10) (CQ10) or ubiquinone is essentially a vitamin or vitamin-like substance. Disagreements on nomenclature notwithstanding, vitamins are defined as organic compounds essential in minute amounts for normal body function acting as coenzymes or precursors to coenzymes. They are present naturally in foods and sometimes are also synthesized in the body. CoQ10 likewise is found in small amounts in a wide variety of foods and is synthesized in all tissues. The biosynthesis of CoO10 from the amino acid tyrosine is a multistage process requiring at least eight vitamins and several trace elements. Coenzymes are cofactors upon which the comparatively large and complex enzymes absolutely depend for their function. Coenzyme Q10 is the coenzyme for at least three mitochondrial enzymes (complexes I, II and III) as well as enzymes in other parts of the cell. Mitochondrial enzymes of the oxidative phosphorylation pathway are essential for the production of the high-energy phosphate, adenosine triphosphate (ATP), upon which all cellular functions depend. The electron and proton transfer functions of the guinone ring are of fundamental importance to all life forms; ubiquinone in the mitochondria of animals, plastoquinone in the chloroplast of plants, and menaguinone in bacteria. The term "bioenergetics" has been used to describe the field of biochemistry looking specifically at cellular energy production. In the related field of free radical chemistry, CoQ10 has been studied in its reduced form (Fig. 1) as a potent antioxidant. The bioenergetics and free radical chemistry of **CoQ10** are reviewed in Gian Paolo Littarru's book, Energy and Defense, published in 1994(1).

### **CQ10 HISTORY**

**CoO10** was first isolated from beef heart mitochondria by Dr. Frederick Crane of Wisconsin, U.S.A., in 1957 (2). The same year, Professor Morton of England defined a compound obtained from **vitamin A** deficient rat liver to be the same as **CoQ10**(3). Professor Morton introduced the name ubiquinone, meaning the ubiquitous quinone. In 1958, Professor Karl Folkers and coworkers at Merck, Inc., determined the precise chemical structure of CoQ10: 2,3 dimethoxy-5 methyl-6 decaprenyl benzoquinone (Fig. 1), synthesized it, and were the first to produce it by fermentation. In the mid-1960's, Professor Yamamura of Japan became the first in the world to use **coenzyme Q7** (a related compound) in the treatment of human disease: congestive heart failure. In 1966, Mellors and Tappel showed that reduced CoO6 was an effective antioxidant (4.5). In 1972 Gian Paolo Littarru of Italy along with Professor Karl Folkers documented a deficiency of CoQ10 in human heart disease (6). By the mid-1970's, the Japanese perfected the industrial technology to produce pure **CoQ10** in quantities sufficient for larger clinical trials. Peter Mitchell received the Nobel Prize in 1978 for his contribution to the understanding of biological energy transfer through the formulation of the chemiosmotic theory, which includes the vital protonmotive role of **CoQ10** in energy transfer systems (7,8,9,10).

In the early 1980's, there was a considerable acceleration in the number and size of clinical trials. These resulted in part from the availability of pure **CoQ10** in large quantities from pharmaceutical companies in Japan and from the capacity to directly measure **CoQ10** in blood and tissue by high performance liquid chromatography. Lars Ernster of Sweden, enlarged upon **CoQ10's** importance as an antioxidant and free radical scavenger (11). Professor Karl Folkers went on to receive the Priestly Medal from the American Chemical Society in 1986 and the National Medal of Science from President Bush in 1990 for his work with **CoQ10** and other vitamins.

#### **COENZYME Q10 DEFICIENCY**

Normal blood and tissue levels of **CoQ10** have been well established by numerous investigators around the world. Significantly decreased levels of **CoQ10** have been noted in a wide variety of diseases in both animal and human studies. CoQ10 deficiency may be caused by insufficient dietary **CoQ10**, impairment in **CoQ10** biosynthesis, excessive utilization of **CoQ10** by the body, or any combination of the three. Decreased dietary intake is presumed in chronic malnutrition and cachexia(12).

The relative contribution of **CoQ10** biosynthesis versus dietary **CoQ10** is under investigation. Karl Folkers takes the position that the dominant source of **CoQ10** in man is biosynthesis. This complex, 17 step process, requiring at least seven vitamins (**vitamin B2** - **riboflavin**, **vitamin B3** - **niacinamide**, **vitamin B6**, **folic acid**, **vitamin B12**, **vitamin C**, and **pantothenic acid**) and several trace elements, is, by its nature, highly vulnerable. Karl Folkers argues that suboptimal nutrient intake in man is almost universal and that there is subsequent secondary impairment in **CoQ10** biosynthesis. This would mean that average or "normal" levels of **CoQ10** are really suboptimal and the very low levels observed in advanced disease states represent only the tip of a deficiency "ice berg".

HMG-CoA reductase inhibitors used to treat elevated blood cholesterol levels by blocking cholesterol biosynthesis also block **CoQ10** biosynthesis(13). The resulting lowering of blood **CoQ10** level is due to the partially shared biosynthetic pathway of **CoQ10** and cholesterol. In patients with heart failure this is more than a laboratory observation. It has a significant harmful effect which can be negated by oral **CoQ10** supplementation(14).

Increased body consumption of <u>CoQ10</u> is the presumed cause of low blood <u>CoQ10</u> levels seen in excessive exertion, hypermetabolism, and acute shock states. It is likely that all three mechanisms (insufficient dietary <u>CoQ10</u>, impaired <u>CoQ10</u> biosynthesis, and excessive utilization of <u>CoQ10</u>) are operable to varying degrees in most cases of observed <u>CoQ10</u> deficiency.

### TREATMENT OF HEART DISEASE WITH COENZYME Q10

**CoQ10** is known to be highly concentrated in heart muscle cells due to the high energy requirements of this cell type. For the past 14 years, the great bulk of clinical work with **CoQ10** has focused on heart disease. Specifically, congestive heart failure (from a wide variety of causes) has been strongly correlated with significantly low blood and tissue levels of **CoQ10** (15). The severity of heart failure correlates with the severity of **CoQ10** deficiency may well be a primary etiologic factor in some types of heart muscle dysfunction while in others it may be a secondary phenomenon. Whether primary, secondary or both, this deficiency of **CoQ10** appears to be a major treatable factor in the otherwise inexorable progression of heart failure.

Pioneering trials of **CoQ10** in heart failure involved primarily patients with dilated weak heart muscle of unknown cause (idiopathic dilated cardiomyopathy). **CoQ10** was added to standard treatments for heart failure such as fluid pills (diuretics), digitalis preparations (Lanoxin), and ACE inhibitors. Several trials involved the comparison between supplemental **CoQ10** and placebo on heart function as measured by echocardiography. **CoQ10** was given orally in divided doses as a dry tablet chewed with a fat containing food or an oil based gel cap swallowed at mealtime. Heart function, as indicated by the fraction of blood pumped out of the heart with each beat (the ejection fraction), showed a gradual and sustained improvement in tempo with a gradual and sustained improvement in patients' symptoms of fatigue, dyspnea, chest pain, and palpitations. The degree of improvement was occasionally dramatic with some patients developing a normal heart size and function on **CoQ10** alone. Most of these dramatic cases were patients who began **CoQ10** shortly after the onset of congestive heart failure. Patients with more established disease frequently showed clear improvement but not a return to normal heart size and function.

Internationally, there have been at least nine placebo controlled studies on the treatment of heart disease with <u>CoQ10</u>:two in Japan,two in the United States, two in Italy, two in Germany, and one in Sweden (17,18,19,20,21,22,23,24,25). All nine of these studies have confirmed the effectiveness of CoQ10 as well as its remarkable safety. There have now been eight international symposia on the biomedical and clinical aspects of <u>CoQ10</u> (from 1976 through 1993 (26,27,28,29,30,31,32,33)). These eight symposia comprised over 300 papers presented by approximately 200 different physicians and scientists from 18 different countries.

It should be mentioned that a slight decrease in the effectiveness of the blood thinner, coumadin, was noted in a case by a Norwegian clinician (34). This possible drug - CoQ10 interaction has not been observed by other investigators even when using much higher doses of CoQ10 for up to seven years and involving 25 patients treated with coumadin concomitantly with CoQ10 (this is still, as of this date, unpublished data).

#### 1. What is CoQ10?

It is a fat-soluble vitamin-like substance present in every cell of the body and serves as a coenzyme for several of the key enzymatic steps in the production of energy within the cell. It also functions as an antioxidant which is important in its clinical effects. It is naturally

present in small amounts in a wide variety of foods but is particularly high in organ meats such as heart, liver and kidney, as well as beef, soy oil, sardines, mackerel, and peanuts. To put dietary **CoQ10** intake into perspective, one pound of sardines, two pounds of beef, or two and one half pounds of peanuts, provide 30 mg of **CoQ10**. **CoQ10** is also synthesized in all tissues and in healthy individuals normal levels are maintained both by **CoQ10** intake and by the body's synthesis of **CoQ10**. It has no known toxicity or side effects.

### 2. Should I take CoQ10?

This question can be asked in two ways. First, should a reasonably healthy person take **CoQ10** to stay healthy or to become more robust? At present I do not believe anyone knows the answer to this question. Second, should a person with an illness such as congestive heart failure take **CoQ10**? As with any change in nutrition, diet, medication, or even activity, **CoQ10** should be discussed with one's physician. An simprovement in heart function occurs, a patient should have regular medical follow up with particular attention to concomitant drug therapy. The attached references will provide detailed information on the clinical use of **CoQ10** and can be obtained from any good medical library.

### 3. What is the dosage of CoQ10?

The dosage of **CoQ10** used in clinical trials has evolved over the past 20 years. Initially, doses as small as 30 to 45 mg per day were associated with measurable clinical responses in patients with heart failure. More recent studies have used higher doses with improved clinical response, again in patients with heart failure. Most studies with **CoQ10** involve the measurement of the level of **CoQ10** in blood. **CoQ10** shows a moderate variability in its absorption, with some patients attaining good blood levels of **CoQ10** on 100 mg per day while others require two or three times this amount to attain the same blood level. All **CoQ10** available today in the United States is manufactured in Japan and is distributed by a number of companies who place the **CoQ10** either in pressed tablets, powder-filled capsules, or oil-based gelcaps. **CoQ10** is fat-soluble and absorption is significantly improved when it is chewed with a fat-containing food. Published data on the dosage of **CoQ10** relates almost exclusively to the treatment of disease states. There is no information on the use of **CoQ10** for prevention of illness. This is an extremely important question which, to date, does not have an answer.

Since **CoQ10** is essential to the optimal function of all celltypes,it is not surprising to find a seemingly diverse number of disease states which respond favorably to **CoQ10** supplementation. All metabolically active tissues are highly sensitive to a deficiency of **CoQ10**. **CoQ10**'s function as a free radical scavenger only adds to the protean manifestations of **CoQ10** deficiency. Preliminary observations in a wide variety of disease states have already been published (48,49,50,51,52,53,54,55,56,57,58).

Another interesting topic is the relationship between the immune system and **CoQ10**. Immune function is extraordinarily complex and undoubtedly is influenced by numerous nutritional variables. There are some encouraging preliminary data from the study of AIDS patients (50,51). End stage AIDS, like other overwhelming illnesses, has been associated with a significant deficiency in **CoQ10**. Regarding AIDS and cancer, it would be foolish to make premature statements about future utility of **CoQ10**, but it is even more foolish to ignore the importance of adequate **CoQ10** levels in these disease states. Adequate **CoQ10** supplementation (with close attention to plasma **CoQ10** levels) is analogous to adequate hydration, and any treatment of critically ill patients should not ignore this easily measured and correctable deficiency.

The antioxidant or free radical quenching properties of CoQ10 serve to greatly reduce oxidative damage to tissues as well as significantly inhibit the oxidation of LDL cholesterol (much more efficiently than **vitamin E**) (60,61). This has great implications in the treatment of ischemia and reperfusion injury as well as the potential for slowing the development of atherosclerosis. In keeping with the free radical theory of aging, these antioxidant properties of CoQ10 have clear implications in the slowing of aging and age related degenerative diseases. There is epidemiologic evidence in humans that uniformly shows a gradual decline in **CoQ10** levels after the age of twenty.

Until recently, attention has been focused on requirements for CoQ10 in energy conversion in the mitochondrial compartment of cells or on the antioxidant properties of CoQ10. New evidence shows that CoQ10 is present in other cell membranes. In the outer membrane it may contribute to the control of cell growth, especially in lymphocytes (the implications are far reaching (62,63,64,65)). The clinical experience with CoQ10 in heart failure is nothing short of dramatic, and it is reasonable to believe that the entire field of medicine should be re-evaluated in light of this growing knowledge. We have only scratched the surface of the biomedical and clinical applications of CoQ10 and the associated fields of bioenergetics and free radical chemistry.

### **Herbs and Herb extracts**

http://www.all-natural.com/herbguid.html

#### **Borage Seed oil**

Borage Seed oil - (1-2-4) (GLA) 1000 mg, NOW P#01722, SUN 05051 60CT 1500MG

**Borage seed oil** is neither a vitamin or an amino acid, but it has characteristics of both. Its most useful aspect, is that of Being one of the highest natural forms of Gamma Liolenic Acid (GLA), a substance that can directly help the DNA make additional genetic error check sum processes, ensuring accurate genetic replication within the mitochondria.

#### **Natural GLA Sources:**

Gamma linolenic acid (**GLA**) is an omega-6 unsaturated fatty acid made in the human body from linoleic acid, an essential fatty acid found in vegetable oils and egg yolks. The main supplemental sources of GLA are oils of the seeds of evening primrose, borage, and black **currant** plants.

#### **Black Currant Oil**

**Black currant oil** is rich in linoleic acid and gamma-linolenic acid (GLA). This substance supports the body's manufacture of hormone-like substances known as prostaglandins which help regulate functions of the circulatory system. **GLA** assists the body with its energy processes and is a structural component of the brain, bone marrow, muscles and cell membranes.

GLA Complex encourages the growth of healthy cells and stops the growth of cancer cells, but interferes with the ability of cisplatin to kill cancer cells. (Super gla ingredients - 130 mg gamma-linolenic acid), Evening Primrose Oil (9% GLA)†, Black Currant Oil (13% GLA)†, Borage Oil (21% GLA)†)

#### **USES**

Synthesis of prostaglandins and thromboxanes 109 from arachidonic acid9.3. Most NSAIDs inhibit both COX-1 and COX-2 1129.4. Metabolism of arachidonic acid and EPA 114by aspirin-modified COX-29.5. Physiological actions of prostaglandin E2. 1159.6. Steroids inhibit release of arachidonic acid 120 from membrane phospholipids10.1. Carcinogenesis 12410.2. Angiogenesis can be inhibited by NSAIDs 128and COX-2 inhibitors10.3. NSAIDs can promote apoptosis (cell death) by suppression 129 of prostaglandin E2 production10.4. Structures of vaccenic acid, linoleic acid, and two 130of its conjugated isomers10.5.

Anticancer effects of conjugated linoleic acid 13210.6. Production of mevalonate is inhibited by statin drugs 13614.1. Relationship of body mass index to hypertension 184and high blood cholesterol14.2. Metabolic consequences of lack of exercise and high sugar 197consumptionA.1. Normal bonding in organic chemistry 215A.2. Saturated and unsaturated molecules 216A.3. Neutral molecules 216A.4. Molecular polarity 217C.1. Eicosanoids synthesized from arachidonic acid 222C.2. Eicosanoids synthesized from eicosapentaenoic acid 223C.3. Eicosanoids synthesized from dihomo-gamma-linolenic acid 224D.1. Schematic illustration of a lipoprotein particle 226E.1. Enzymatic conversion of glutamate to GABA 228E.2. Neuronal excitation 229E.3. Basic structure of a neuron 230E.4.

Ions and depolarization

#### **Valerian root extract**

Valerian root extract (2) (Swanson 200mg 0.8%) at PM very good for sleep

http://www.vitacost.com/solgar-valerian-root

Solgar Valerian Root –300mg - *Valeriana officinalis* Valerian Extract (4:1) [root] 100ct <u>Planetary Herbals</u> Full Spectrum Valerian Extract PF063

http://www.swansonvitamins.com/planetary-herbals-full-spectrum-valerian-extract-650-mg-60-tabs 500 mg Swanson SWR047 69ct \$8,50,

Valerian root extract (6-4, 12, 20) (Swanson 200mg 0.8%) at PM very good for sleep

http://www.newswithviews.com/Howenstine/james16.htm

Two of the drugs used to treat psychosis and bipolar disorder (Haldol and **Valproic A**cid) inhibit the growth of t. gondii in cerebrospinal fluid and blood at concentrations below that being treated with these therapies suggesting that *improved mental status might actually be due to killing t. gondii not anti-psychotic effects*.

over its 128 year history valproic acid has periodically risen in importance from the ashes like the phoenix bird\* as new properties of it were discovered and new important applications found for it. In 1962 it was found to be a powerful anticonvulsant and it soon evolved to become a favorite mood stabilizer. Currently valproic acid seems to have strong potential applications for treating cancers and Alzheimer's disease, and for guiding stem cell regeneration of nerves in cases of spinal cord injuries.

<u>Valerian</u> (*Valeriana officinalis*) is a perennial plant of European and Asian origin. Dietary supplements have traditionally been made from its roots and have been used as a sedative for dealing with insomnia(<u>ref</u>). My wife tells me that back in the 60s valerian was often found in hippy cookbooks, and has been thought to have magical powers. "Its magical reputation is Evil and Protective, and it is used to **Force Love**. It is burned in <u>Black Arts Incense</u> for hexing, but added to <u>Uncrossing Incense</u> to destroy jinxes if burned with a yellow

candle(ref)." Magic apart, research in the 1990s suggests that valerian achieves its effects through acting on the GABAA (gamma-aminobutyric acid) receptor, promoting the expression of GABA(ref). "In conclusion, our data show that the extent of GABAA receptor modulation by Valerian extracts is related to the content of valeric acid(ref)." Valproic acid (also known as valproate and abbreviated VPA) is a synthetic substance, not present in the valerian plant. "Valproic acid (by its official name 2-propylvaleric acid) was first synthesized in 1882 by Burton as an analogue of valeric acid, found naturally in valerian.[1] (ref)" As we shall see, VPA too is a strong modulator of the GABA receptor. Although mostly not known to be evil, it is also strongly protective.

#### Valeriana wallichii root extract

http://www.amazon.com/Valeriana-wallichii-improves-modulates-monoamine/dp/B00AJFDGHU

http://link.springer.com/article/10.1007%2Fs00436-010-2127-0

#### **Abstract**

Leishmanial diseases, posing a public health problem worldwide, are caused by Leishmania parasites with a dimorphic life cycle alternating between the promastigate and amastigate forms. Promastigates transmitted by the vector are transformed into amastigotes residing in the host tissue macrophages. Presently, new antiparasitic agents are needed against Leishmania donovani and Leishmania major, the respective organisms causing visceral and cutaneous leishmaniasis, since the available treatments are unsatisfactory due to toxicity, high cost, and emerging drug resistance. Over the years, traditional medicinal flora throughout the world enriched the modern pharmacopeia. Hence, roots of 'Indian Valerian' (Valeriana wallichii DC) were studied for its antileishmanial activity for the first time. The methanol and chloroform extracts showed activity against L. donovanipromastigotes and both promastigotes and amastigotes of L. major. The most active fraction, F3, obtained from the chloroform extract, showed IC<sub>50</sub> at  $\sim$ 3–7 µg/ml against both the promastigotes and 0.3 µg/ml against *L. major* amastigotes. On investigation of the mechanism of cytotoxicity in L. donovani promastigotes, the 'hall-mark' events of morphological degeneration, DNA fragmentation, externalization of **phosphatidyl** serine, and mitochondrial membrane depolarization indicated that F3 could induce apoptotic death in leishmanial cells. Therefore, the present study revealed a novel and unconventional property of V. wallichii root as a prospective source of effective antileishmanial

http://www.herbalsafety.utep.edu/herbs-pdfs/valerian.pdf

## **Active Principles**

- Valepotriates (which decompose to form Baldrinals)
- Sesquiterpenes
- Sesquiterpene carboxylic acids (valerenic acid and others)
- GABA (gamma- amino-butyric acid)
- Lignans (hydroxypinoresinol)
- Monoterpenes (pinenes and camphene)
- Flavonoids

#### **Valerian**

http://www.amazon.com/Valerian-Root-Powder-Valeriana-Botanicals/dp/B002DXVJP2

Valerian Root Powder - Valeriana wallichii, 1 lb,

http://healthyeating.sfgate.com/medicinal-uses-benefits-valerian-7793.html

Valeriana, a genus of perennial flowering plant native to Europe, North America and South America, contains several species with potentially medicinal benefits, collectively referred to as valerian. Herbalists have used valerian, a mainstay in traditional medicine, since 200 A.D., according to the University of Maryland Medical Center. Consult your doctor about the appropriate use of valerian.

http://www.nutragreenbio.com/product/valerian-root-extract-powder

http://www.jenabioscience.com/images/b3e879b381/Ghosh\_2011\_antileishmnial\_Valeriana\_extracts.pdf

## **Treatment of Human Parasitosis in Traditional Chinese Medicine**

By Heinz Mehlhorn, Zhongdao Wu, Bin Ye

http://smithspharmacy.com/ns/DisplayMonograph.asp? storeID=A2CA0A52C8E242409B8D577B01E035A1&DocID=valerian

(1R.3R.5R.7S.8S,9S)-3.8-epoxy-1-O-ethyl-5-hydroxyvalechlorine, (1S.3R.5R.7S.8S,9S)-3.8-epoxy-1-O-ethyl-5-hydroxyvalechlorine, (1S,3R,5R,7S,8S,9S)-3,8-epoxy-1,5-dihydroxyvalechlorine, 2S(-)hesperidin, (5S,6S,8S,9R)-1,3-isovaleroxy-?4,11-1,3-diol, (5S,6S,8S,9R)-3-isovaleroxy-6isovaleroyloxy-?4,11-1,3-diol, (5S,6S,8S,9R)-6-isovaleroyloxy-?4,11-1,3-diol, (5S,7S,8S,9S)-7hydroxy-8-isovaleroyloxy-?4,11-dihyronepetalactone, (5S,7S,8S,9S)-7-hydroxy-10-isovaleroyloxy-? 4,11-dihyronepetalactone, (5S,8S,9S)-10-isovaleroyloxy-?4,11-dihyronepetalactone, 6methylapigenin, 6'-O-acyl-beta-D-glucosyl-clionasterols, 14-methylpentadecanoyl, actinidine, all-heal, amantilla, Balderbrackenwurzel (German), Baldisedron®, Baldrian (German), Baldrian-Dispert, Baldrianwurzel (German), baldrinal, baldrion, Belgian valerian, blessed herb, capon's tail, chlorogenic acid, chlorovaltrates A-O, clionasterol-3-O-beta-D-glucopyranoside, common valerian, English valerian, Euvegal® forte, flavonoids, fragrant valerian, garden heliotrope, garden valerian, German valerian, great wild valerian, Harmonicum Much®, heliptrope, herba benedicta, hexadecanoyl 8E, hexadecanoyl, 8E,11E-octadecadienoyl, homobaldrinal, Indian valerian, irioids, isovaleric acid, Jacob's ladder, Japanese valerian, jatadoids A-B, jatairidoids A-C, Katzenwurzel (German), kessanes, laege-baldrian (Danish), Li 156, lignans, Mexican valerian, monoterpenes, Nature Made®, Nature's Resource®, Nature's Way Valerian, Nervex®, Neurapas® balance, Neurol®, Orasedon®, Pacific valerian, phu, phu germanicum, phu parvum, pinnis dentatis, racine de valériane (French), radix valerian, red valerian, rupesin B, Sanox-N®, Seda-Kneipp, Sedamine, Sedonium®, sesquiterpenes, setewale capon's tail, setwall, setwell, tagara (Sanskrit), terpenoids (valepotriates), theriacaria, Ticalma®, vaimane, valariana, Valdispert, Valdispert forte, valepotriates, valeranone, valerenal, valeriana (Italian), Valeriana edulis, Valeriana edulis Nutt., Valeriana faurieri, Valeriana foliis pinnatis, Valeriana jatamansi, Valeriana jatamansi Jones, Valeriana officinalis L., Valeriana officinalis var. latifolia, Valeriana procera Kunth (Mexican valeriana), Valeriana radix, Valeriana sitchensis, Valeriana sitchensis Bong., Valeriana wallichii, Valeriana wallichii DC., Valerianaceae (family), Valerianae radix, Valerianaheel®, valériane (French), Valerina Forte®, Valerina Natt®, valerinic acid, Valmane®, valtrate, Valverde®, Valverde Sleeping Syrup, vandal root, Vermont valerian, volatile oils, volvaltrate B, wild valerian, Ze 185, Ze 91019, Ze 911.

· Note: Valeriana procera Kunth (Mexican valerian) is sometimes used as a substitute for Valeriana officinalis L. Other valerian species used in commercial preparations include Valeriana jatamansi Jones, Valeriana edulis Nutt., and V. sitchensis Bong. Although the primary focus of this bottom line

is on Valeriana officinalis, information pertaining to other Valeriana species has been identified whenever possible.

## Background

- Limited evidence suggests that <u>valerian</u> may benefit women with moderate-to-severe menstrual cramps by relieving pain and reducing the need for pain relievers.
- Valerian is often used to treat sleep disorders and anxiety. Early studies suggest that valerian may help improve sleep quality. Ongoing use may be more effective than single-dose use. However, more high-quality research is needed before firm conclusions can be made.
- Studies report that valerian is generally well tolerated for up to 4-6 weeks. It may rarely cause mild side effects such as dizziness, hangover, or headache. Early research suggests that small doses of <a href="walerian">walerian</a> may lack effect on alertness, concentration, coordination, and reaction time. However, other studies report that valerian may slow the processing of complex thoughts for a few hours after use.
- Acne, amenorrhea (lack of menstrual period), anorexia, anti-spasm, antiviral, arthritis, asthma, bloating, chest pain, colic, constipation, cough, cramping, cyanosis (blue skin due to lack of oxygen), digestive problems, dizziness, epilepsy, fatigue, fever, gas, hangovers, headache, heartburn, heart disease, heart failure, high blood pressure, HIV, hypnotic, hypochondria, improving urine flow, irritable bowel syndrome (IBS), kidney stones, liver disorders, measles, memory enhancement, menstrual flow stimulant, migraine, musculoskeletal conditions, nausea, nerve pain, nervous system disorders, pain relief, peptic ulcer disease, premenstrual syndrome (PMS), respiratory disorders (lung spasms), restless legs syndrome, rheumatic pain, seizures, skin disorders, skin sores, stomach disorders, stomach upset, sweating, urinary tract disorders, vaginal yeast infections, vision enhancement, withdrawal from tranquilizers.
- <u>Valerian</u> is likely safe when taken by mouth in doses of 400-600 milligrams of root extract
  daily by otherwise healthy people for the short term. <u>Valerian root</u> is widely thought to be
  safe in recommended doses for 4-6 weeks.
- <u>Valerian</u> may cause changes in heart rate, delirium, diarrhea, dizziness, excitability, headache, heartburn, heart failure, hypothermia (low body temperature), impaired concentration or thinking, increased breast cancer risk, insomnia, liver toxicity, movement problems, muscle fatigue, nausea, shaky hands, skin rash, sperm abnormalities, stomach pain, uneasiness, upset stomach, valerian withdrawal, and vomiting.
- Drowsiness or sedation may occur. Use caution if driving or operating heavy machinery.
- <u>Valerian</u> may increase the risk of bleeding. Caution is advised in people with bleeding
  disorders or taking drugs that may increase the risk of bleeding. Dosing adjustments may be
  necessary
- Use caution when taking preparations that have high concentrations of valepotriates and baldrinals.
- Use cautiously in people who have breast cancer, heart disorders, liver dysfunction, and stomach or intestine disorders, or those taking agents that affect gamma-aminobutyric-acid

A (GABA[A]) receptors, agents that are toxic to the liver, antidepressants, central nervous system (CNS) agents, sedatives, and sleep-inducing agents.

- Use cautiously in children, especially those under three years of age.
- Avoid two weeks before and during surgery, and in pregnant or breastfeeding women.
- Avoid in people with known allergy or sensitivity to <u>valerian</u>, its parts, or members of the same family.

## **Interactions with Drugs**

- <u>Valerian</u> may increase the risk of bleeding when taken with drugs that increase the risk of bleeding. Some examples include aspirin, anticoagulants ("blood thinners") such as warfarin (Coumadin®) or heparin, anti-platelet drugs such as clopidogrel (Plavix®), and non-steroidal anti-inflammatory drugs such as ibuprofen (Motrin®, Advil®) or naproxen (Naprosyn®, Aleve®).
- <u>Valerian</u> may cause low blood pressure. Caution is advised in people taking drugs that lower blood pressure.
- Valerian may interfere with the way the body processes certain drugs using the liver's
   "cytochrome P450" enzyme system. As a result, the levels of these drugs may be increased
   in the blood, and may cause increased effects or potentially serious adverse reactions.
   People using any medications should check the package insert, and speak with a qualified
   healthcare professional, including a pharmacist, about possible interactions.
- <u>Valerian</u> may increase the amount of drowsiness caused by some drugs. Examples include benzodiazepines such as lorazepam (Ativan®) or diazepam (Valium®), barbiturates such as phenobarbital, narcotics such as codeine, some antidepressants, and alcohol. <u>Caution is advised while driving or operating machinery</u>.
- <u>Valerian</u> may also interact with acetaminophen, agents for the heart, agents that affect blood vessel width, agents that affect the immune system, agents that affect the lungs, agents that affect the nervous system, agents that harm the liver, alcohol, anti-anxiety agents, anticancer agents, antidepressants (including monoamine oxidase inhibitors [MAOIs] and selective serotonin reuptake inhibitors [SSRIs]), anti-diarrhea agents, antihistamines, anti-seizure agents, anti-spasm agents, aromatase inhibitors, barbiturates, benzodiazepines, beta-blockers, caffeine, codeine, diphenhydramine (Benadryl®), disulfiram (Antabuse®), flunitrazepam, hormonal agents, loperamide (Imodium®), lorazepam, loreclezole, metronidazole (Flagyl®), mood stabilizers, morphine, pain relievers, pentobarbital, skin agents, stomach agents, tamoxifen, and vasopressin.

## **Interactions with Herbs and Dietary Supplements**

- •Valerian may increase the risk of bleeding when taken with herbs and supplements that are believed to increase the risk of bleeding. Multiple cases of bleeding have been reported with the use of *Ginkgo biloba*, and fewer cases with garlic and saw palmetto. Numerous other agents may theoretically increase the risk of bleeding, although this has not been proven in most cases.
- <u>Valerian</u> may interfere with the way the body processes certain herbs or supplements using the liver's "cytochrome P450" enzyme system. As a result, the levels of other herbs or

supplements may become too high in the blood. It may also alter the effects that other herbs or supplements possibly have on the P450 system.

- Valerian may cause low blood pressure. Caution is advised in people taking herbs or supplements that lower blood pressure.
- Valerian may increase the amount of drowsiness caused by some herbs or supplements.
- Valerian may also interact with anti-anxiety herbs and supplements, anticancer herbs and supplements, antidepressants (including monoamine oxidase inhibitors [MAOIs] and selective serotonin reuptake inhibitors [SSRIs]), anti-diarrhea herbs and supplements, antihistamines, anti-seizure herbs and supplements, anti-spasm herbs and supplements, caffeine, herbs and supplements for the heart, herbs and supplements for the skin, herbs and supplements for the stomach and intestines, herbs and supplements that affect blood vessel width, herbs and supplements that affect the immune system, herbs and supplements that affect the lungs, herbs and supplements that affect the nervous system, herbs and supplements that harm the liver, hops, hormonal herbs and supplements, kava, lemon balm, melatonin, mood stabilizers, motherwort, pain relievers, passionflower, and St. John's wort.

## **Evening Primrose Oil**

Evening Primrose Oil Seed 500 mg Cis-Linoelic LA <u>CLA</u>, Gamma <u>GLA</u> Royal Brittany

http://www.americanhealthus.com/

## **Silymarin**

Silymarin (4 per day - 2 (AM) 2 (PM)

Protects liver and kidneys as toxins leave the body <u>Silymarin</u> provides support and protection against liver toxins which can cause free-radical-mediated oxidative damage. <u>Silymarin</u> is many times more potent in antioxidant activity than <u>vitamin E</u>. In addition, it increases liver production of <u>glutathione</u> and protects red blood cell membranes against lipid peroxidation and hemolysis.

## **NAC**

And **NAC** –"Amino Sulfer" (use **NAC** with caution).

(1) AM NAC 600mg - w molydbenum amino acid chelate and selenomethionine Sulphur amino Acid protein cell 4 processes/GLUtathone NOW Foods #0086

Why N-Acetyl-Cysteine (NAC)?

NAC is a derivative of the naturally occurring amino acid, cysteine. It boosts tissue levels of glutathione—a small protein composed of three amino acids—cysteine, glutamic acid, and glycine. Glutathione is involved in detoxification mechanisms, binding to fat-soluble toxins Useful in situations like heavy metals, solvents, and pesticides to transform them into a water-soluble form allowing more efficient excretion via the kidneys.

**NAC** is readily turned into **glutathione** by your body. **Glutathione** is essential for normal phase two detoxification in your liver. You can buy oral **glutathione**, but it is considerably

more expensive than **NAC** and there is some controversy among biochemists and clinical nutritionists as to whether it passes through your digestive system intact.

## **Glutathione**

<u>Glutathione's</u> combination of detoxification and free radical protection makes it one of the most important cancer and aging fighters in our cells. Without the protection of <u>glutathione</u>, your cells die at a faster rate, making you age more quickly and putting you at risk for toxin-induced diseases including cancer. People who smoke, who are chronically exposed to toxins, who suffer from inflammatory conditions such as rheumatoid arthritis or chronic conditions such as diabetes, AIDS, or cancer typically have lower levels of <u>glutathione</u>.

\*\*\* **Glutathione** – is the most important antioxidant in the body, and gamma-aminobutyric acid, **GABA**- **GABA** prevents cell oxidation, or damage from accumulated toxins and cellular waste products. Toxin overload occurs when cells become overworked. **Glutathione** and **GABA** are two important molecules your body manufactures from amino acids.

**Both <u>GABA</u> and <u>Glutathione</u>** molecules use the amino acid <u>glutamine</u> as the base molecule, their final chemical structures are quite different. <u>Glutathione</u>, is most important antioxidant of your brain and of your liver. <u>Glutathione</u> keeps your body toxin-free and is part of an enzyme known as <u>glutathione</u> peroxidase loop. <u>GABA</u>, a neurotransmitter, resides in your brain, where it helps process aspects of cognitive, adrenal, antioxidant, and emotional function.

Parasites destroy stores of <u>Glutathione</u> and <u>GABA</u>. <u>It is not possible to supplement the construction of these molecules directly by supplementation</u>. Offloading of the process loops is possible indirectly by supplementing the sulfur supply, and <u>GABA</u> production, but the <u>Glutathione</u> molecule will always be in short supply. Maintaining <u>MSM</u> dosing every day is essential to preserving any assemblage of <u>Glutathione</u> status and supply.

## **Toxin Removing Herbs**

Goldenseal Root and Oregon Grape Root turn urine dark yellow and remove smelly toxins

Common Causes of Urine Discolouration		
Colour	Pathological causes	Food and drug causes
Brown	Bile pigments, myoglobin	Levodopa, metronidazole, nitrofurantoin, some antimalarial agents, fava beans
Brownish- black	Bile pigments, melanin, methaemoglobin	Cascara, levodopa, methyldopa, senna
Green or blue	Pseudomonal urinary tract infection (UTI), biliverdin	Amitriptyline, indigo carmine, IV cimetidine, IV promethazine, methylthioninium chloride, triamterene
Orange	Bile pigments	Phenothiazines, phenazopyridine, rifampicin, hydroxocobalamin
Red	Haematuria, haemoglobinuria, myoglobinuria, porphyria	Beets, blackberries, rhubarb, phenolphthalein, rifampicin
Yellow	Concentrated urine (orange to gold in dehydration)	Carrots, cascara

While plants like <u>Goldenseal</u> (Hydrastis canadensis), <u>Barberry</u> (Berberis vulgaris), <u>Goldthread</u> (Coptis trifolia), and Amur cork tree (Phellodendron amurense) <u>all contain Berberine</u>, <u>they aren't</u> <u>necessarily used in the exact same manner</u>. The differentiation is evident.

## **Scientific Name(s):**

Berberis vulgaris L. and Mahonia aquifolium Nutt. Family: Berberidaceae Common Name(s): Barberry , Oregon grape , Oregon barberry , Oregon grapeholly , trailing Mahonia , Berberis , jaundice berry , woodsour , sowberry , Pepperidge bush , sour-spine 1 , 2

#### Uses

The fruits have been used in jams, jellies, and juices. Plant alkaloids have been found to be antibacterial, antifungal, anti-inflammatory, antioxidant, and antidiarrheal. **Berberine** is a uterine stimulant.

## **Dosing**

Barberry berries and root bark have been used as a source of **Berberine**. Daily doses of 2 g of the berries have been used, but there are no clinical studies to substantiate **barberry**'s varied uses.

## Chemistry

The root and wood are rich in protoberberines (**Berberine**, palmatine, jatorrhizine) and bisbenzylisoquinoline derivatives (oxyacanthine, berbamine) as well as other alkaloids such as bervulcine, magnoflorine, and columbamine. 2 , 3 , 8 , 9 The root may contain as much as 3% alkaloids, which impart a yellow color to the wood. **Berberine**, berbamine, and oxyacanthine are considered the 3 most important alkaloids. 10 The edible berries are rich in vitamin C, sugars, and pectin.

## **Anti-inflammatory**

Products of lipoxygenase metabolism enhance the pathophysiology of psoriasis. Each of the 6 bisbenzylisoquinoline alkaloids (oxyacanthine, armoline, baluchistine, berbamine, obamegine, aquifoline) isolated from M. aquifolium exhibited various lipoxygenase inhibitory activity resulting in an anti-inflammatory and antioxidant effect. 11

#### **Antibacterial**

**Berberine** and several related alkaloids are bactericidal, in 1 study exceeding chloramphenicol (e.g., Chloromycetin) against Staphylococcus epidermidis, Neisseria meningitidis, Escherichia coli, and other bacteria. 2 Another study reported that amethanolic extract (containing 80 mg of dried plant material) from the root of M. aquifolium exhibited antifungal activity against Trichoderma viridae and was considered more effective than nystatin. 13

### **Anti-diarrheal**

**Berberine** does not appear to exert its antidiarrheal effect by astringency.

## Goldenseal

## Goldenseal Root (1-2) 570 mg Natures Way 13900

http://www.IHERB.com (taper quickly as infection under control)

http://ntp.niehs.nih.gov/ntp/htdocs/lt rpts/tr562.pdf

We conclude that goldenseal root powder caused cancer in the liver of male and female rats and male mice. There was no effect of goldenseal root powder on female mice.

Because goldenseal is a mixture of several alkaloids, it is not possible to clearly attribute the effects noted in this study to any one of the constituent alkaloids.

http://www.swansonvitamins.com/natures-way-goldenseal-root-570-mg-100-caps

Dislodges mucus, moderates anti inflammatory response, a powerful antimicrobial, and a digestive tonic The rhizomes and roots of this herb contain isoquinoline alkaloids, such as hydrastine, canadine, Berberine, I-hydrastine, canadaline, and traces of fatty oil, essential oil, and resin. High content of alkaloids is responsible for the anti-infective, antibiotic, and immune stimulating properties of **goldenseal**.

Used for 3 months to detoxify the physique. The way **goldenseal** helps the body is by flushing illness and toxins outside of your body. This has many advantages for the digestive tract, the lymphatic system, and the cardiovascular system. **Goldenseal** also is thought to help support the immune system, helps in curing ear infections, calming the central nervous system

**Goldenseal** helps the body is by flushing illness and toxins outside of your body. Digestion, such a gas, indigestion, irritable bowel, and trouble with not absorbing nutrients correctly, herbalists suggest a mix of cayenne pepper and equivalent parts goldenseal, in tincture or capsules, right before/after you eat.

The **goldenseal** herb and its extracts help in preventing and treating heartburn, particularly caused by emotional stress. It helps in lowering the levels of acids, thus soothing the digestive system and reducing heartburn.

Manages several kinds of diarrhea from "travelers diarrhea" to the severe kind

The alkaloid **Berberine** can be extracted from the roots of several plant species, notably Berberis aquifolium (**Oregon grape**), Hydrastis Canadensis (**goldenseal**) root, and Coptis chinensis (**goldthread**). **Berberine** has protostatic and protocial activity against E. histolytica, G. lamblia and B. hominis.i[57] ii[58]iii[59]

Like pokeroot, yellow dock (Rumex crispus) can compromise red blood cells if improperly overused. Or in other words, unless used properly, it can have toxic properties. But also like pokeroot, if used properly, studies have shown that it has strong anticancer properties.

**Goldenseal** is one of the five top-selling herbal products in the United States. A small amount of research reports that **Berberine**, a chemical found in **goldenseal**, may be beneficial in the treatment of chloroquine-resistant malaria when used in combination with pyrimethamine. Due to the very small amount of **Berberine** found in most **goldenseal** preparations, it is unclear whether **goldenseal** contains enough **Berberine** to have these effects. More research is needed before a recommendation can be made.

Avoid if allergic or hypersensitive to **goldenseal** or any of its constituents, like **Berberine** and hydrastine. Use cautiously with bleeding disorders, diabetes, or low blood sugar.

As your body responds to the toxic characteristics, getting large doses can be poisonous for one's body and trigger nausea. Taking too high of a dosage of **goldenseal** can also result in a shortness of breath and trouble breathing. Overdosing could lead to severe muscle spasms and potentially paralyze the nervous system.

Taking too much **goldenseal** for a lengthy amount of time may lead to problems of the digestive tract, including constipation and diarrhea. It may also trigger extreme states of stress, and in some cases delirium hallucinations. It could cause some of the issues that it is intended to aid with if taken for too long a time.

\* Antibacterial \* Antifungal \* AntiViral \* Bitter \* Cholagogue \* Hepatic \* Immunostimulant \* Vermifuge

**Goldenseal** is also a <u>bitter herb</u> that <u>stimulates bile</u> and can be used to improve digestive problems from peptic <u>ulcers</u> to colitis. It works well against <u>diarrhea</u> caused by intestinal bugs and can be used in formulas to treat intestinal <u>parasites</u>. Hoffman

MAK - blood cleanser and, as its extremely bitter taste would suggest, to stimulate the liver and gallbladder. It purifies the blood and cleanses the liver by helping to stimulate bile flow and releasing toxins and helping purge the spleen. It also helps the liver metabolize wastes and toxins and, because of its anti-pathogenic properties used by natural healers in the treatment of chronic hepatitis-B.

## **Oregon Grape Root**

Oregon Grape Root (1) 500mg Natures way 14159

http://www.IHERB.com

PM 2 Oregon Grape Root 500mg Natures way 14159 IHERB

<u>Oregon grape root</u> is strong cooling, draining and detoxifying herb. Helps with digestion of fats, and in elimination of a congested Liver

Ability to fight infection as well as its affinity for the liver, effects the liver and the gallbladder. The bitter taste on the tongue stimulates saliva, which then creates a whole cascade of digestive functions and digestive enzymatic secretions.

Heat in the body is characterized by redness, inflammation, yellow secretions (or a yellow coating of the tongue) and, of course, heat. Moistness in the body is just that: weepy or infections that seep pus or leak fluids, especially fluids that are yellow.

Contains a specific multidrug resistance pump inhibitor (MDR Inhibitor) named 5′-methoxyhydnocarpin (5′-MHC)3

Strongly potentates the action of Berberine by disabling the bacterial resistance mechanism against both synthetic and natural antimicrobials.

Eye infections, vaginal infections, wounds on the skin, mouth infections, inflammatory bowel conditions, infectious diarrhea (e.g., giardia and other parasites), infections in the upper digestive tract (h. pylori), urinary tract infections, and sore throats

<u>Goldenseal</u>: Berberine, an alkaloid that may act complementary to proanthocyanidins in inhibiting bacteria from adhering to the walls of the bladder, is present in Goldenseal, as well as Oregon grape and other plants.

#### **BARBERRY**

Barberry bark (1-2) 500mg Berberis Vulgaris 4:1 Swanson sw1150

The ingredients of Barberry - Berberine, columbamine, and oxyacanthine have anti-bacterial and anti-viral properties with some suggestion that Berberine sulfate might be amebicidal and trypanocida.

Berberine Sulfate trihydrate (1) 500mg www.Nutriguard.com Natural AMPK activator

Research indicates that **Berberine** is specifically effective against cholera, giardia, shigella, salmonella and E. coli The Berberine, aids in the secretion of bile and is good for liver problems, that is also a mild laxative, and helps the digestive processes.

**Berberine** also fights Staphylococcus, Streptococcus, Salmonella, Shigella and Eschorichia Coli, cholera and chronic candidiasis (yeast). Berberine is highly bactericidal, amoeboidal and trypanocidal. Barberry is antihelicobacter, fungicidal and anti-parasitic Barberry acts in the same way as chloramphenicol, a commonly prescribed antibiotic drug.

**Barberry** helps to purify the respiratory and digestive systems and also has an anti-parasitic effect.

The potential for an increase in free bilirubin, jaundice. Use cautiously with cardiovascular disease, gastrointestinal disorders, hematologic disorders, leukopenia, kidney disease, liver disease, respiratory disorders, cancer, hypertyraminemia, diabetes, or low blood pressure.

Berberine suppresses MEK/ERK-dependent EGR-1 signaling pathway and inhibits vascular smooth muscle cell regrowth after in vitro mechanical injury. Biochem Pharmacol 2006;71:806-817.

Tumor metastasis is the major barrier for tumor treatment. Some metastases occur in 5 or 10 years and some even in 20 years after tumor is controlled, but the metastases are impossible to defend effectively till now.

Damage - Deficit in connectivity among cells, enhancement of matrix catabolic enzymes activity, increasing level of variant surface glycoprotein, reduction of cell immunogenicity, lack of hormone, growth factors and vitamin D receptors on tumor cells surface, and modification of cellular morphology (noncircular and irregular). [4],[5] All the above processes are correlated with cell metastasis potential. We will mainly overview tumor metastasis related molecules in this part.

Metastasis is regulated and controlled by many related genes, such as cancer gene, metastasis-related gene and relevant metastasis suppressor gene. [81] When these related genes were modified in some conditions, either activated or inactivated, tumor cells metastases would be induced. It is found that some TCM can inhibit tumor metastases through affecting these genes. Arsenic trioxide up regulated the expression of metastasis suppressor gene mn23 and down regulate the expression of metastasis-related gene N-myc so as to inhibit tumor metastasis. [82] **Berberine** strengthened the inhibiting effects of arsenic trioxide on the expression of tumor metastasis-related genes myc and jun, consequently inhibiting the invasion and metastasis of glioma cells. [83] Norcantharidin facilitated the expression of tumor cell metastasis suppressor gene mn23 and inhibit the metastasis of human gallbladder carcinoma. [84] Zhong et al., reported that allicin decreased the expression of VEGF, uPAR, and HPA, and inhibited the invasion and metastasis of human colon cancer cells in vitro.[85]

## **Fucoidan Extract**

## Fucoidan Extract (2-12) Swanson Green Foods 500mg SWR047

- "full spectrum" Brown Seaweed (Wakame) Undaria pinnatifida extract sw1504 with Fucoxanthin, \$\$
- (2) 250mg "Natures Vision Brown Seaweed 08375" fights Flagellated Promastigote, forces TH1 immune dominance— 4wks, \$\$
- <u>Bladderwack</u> Fucus vesiculous thallus Swanson SW1399 (antiviral cold sore zoonosis) mild relief of constipation.
- **Kelp** Kelp contains nearly thirty minerals which nourish the glands (especially the thyroid and pituitary). By enhancing the action of the glandular system, it helps balance the body's metabolism and rate at which it burns calories. Kelp, also known as seaweed, grows in the rich ocean beds, far below surface pollution levels. Because of its high nutrient content, this herb is reputedly beneficial for a wide range of applications. It is known to nourish the sensory nerves, brain membranes, also spinal cord and brain tissue.

## **Turkey Tail**

## Turkey Tail - Antibacterial, AntiViral, Antifungal

http://www.iherb.com/Fungi-Perfecti-Host-Defense-Turkey-Tail-60-NP-Caps/21457?at=0

In traditional Chinese medicine, Turkey Tail Mushroom extract is used to treat liver cancer and some types of jaundice. 160 In modern medicine, the best known and most researched medicinal extract of Turkey Tail Mushroom is PSK. It is used in Asia as an anti-cancer drug under the brand name Krestin. 188

Two Japanese studies in the 1990's encompassing a total of 486 patients showed an increased survival rate from gastric cancer when PSK was added to conventional chemotherapy treatment. 161, 162, 163 It's also been found that PSK reduces cancer metastasis and recurrence. 161, 162, 164

Two other polysaccharides from <u>Turkey Tail Mushroom</u> extract have been found to have an inhibitory effect on leukemia. The polysaccharide CVP was shown to inhibit leukemia cell proliferation without any negative effect on normal lymphocytes. 165, 166, 167 Another smaller polysaccharide named SPCV also had an inhibitory effect on leukemia cells. 168

The compounds of <u>Turkey Tail Mushroom extract</u> appear to work in two ways to combat cancer: 1) They demonstrate a direct inhibitory effect on the proliferation of cancer cells, and 2) They stimulate the activity of NK (Natural Killer) cells in the patient. 169, 170, 171 NK cells are an important part of the immune response to fight cancer.

Though the effectiveness against different forms of cancer varies, PSK is currently used to treat cervical cancer in conjunction with radiation. Studies have linked it to increased survival rate from cervical cancer. 134 It also decreases tumor cell regeneration in hormone responsive prostate cancer, as demonstrated in a 2001 study at New York Medical College. 172

Other forms of cancer that PSK has been tested against with promising results include certain forms of sarcoma, carcinoma, breast, lung and colon cancer. 25, 173 However, it appears ineffective against Sarcoma 180. 174

PSK has also been proven as a potent antibiotic, in particular against strains of Staphylococcus aureus, Pseudomonas aeruginosa, Candida albicans, Cryptococcus neoformans, Escherichia coli and Listeria monocytogenes. 175, 176, 177, 178, 179

Tests in vitro (test tubes / Petri dishes - not live subjects) have even shown a compound (PSP) from **Turkey Tail Mushroom extract** to inhibit the replication 180 and infection of HIV. 181

In a study conducted in 1995, PSK demonstrated effectiveness at normalizing the immune function of people with chronic rheumatoid arthritis. 182

Paul Stamets, in his book Growing Gourmet and Medicinal Mushrooms, says he's aware of several individuals with Hepatitis C who have reported relief of symptoms on a regimen of medicinal mushroom tea. In particular, he mentions a man with a swollen spleen and liver. Every day, he would drink a tea made from Reishi and Turkey Tail. The swelling disappeared after two weeks. His liver enzymes normalized as HCV was reduced from 1.3 million to 140,000. 183

In conclusion, <u>Turkey Tail Mushroom extracts</u> also contain many antioxidant compounds 184,185, 186, 187, and may assist the recovery of the spleen after radiation. 189

In his book "Mycelium Running," medicinal mushroom expert Paul Stamets also lists the following areas where research has shown <u>Turkey Tail extract</u> to have a beneficial effect: Kidney Tonic; Uterine Cancer. 134

## Ginkgo

# Ginkgo Biloba (2-4-6) 60mg Swanson sw893 /01893-

BRAIN CAPILLARIES, prevents blood clots.

**Ginkgo biloba** seeds are crushed and believed under TCM to treat asthma.[62] Ginkgo has been used in TCM for nearly 5,000 years.[63] Further studies to establish the efficacy both as used under TCM beliefs and for proposed uses as an evidence based medicine are required.[63]

<u>Ginkgo Biloba</u> extract from the ginkgo tree has been shown to benefit visual function by improving microcirculation to the eyes especially among patients suffering from senile macular degeneration, a common condition thought to involve free radical damage, says Steven Schechter, N.D., author of Fighting Radiation & Chemical Pollutants With Foods, Herbs &Vitamins (Vitality, Ink).

More than 280 scientific studies indicate standardized **ginkgo** extract prevents and/or benefits ailments such as vertigo, tinnitus, inner ear disturbances, memory impairment, ability to concentrate, anxiety, depression, neurological disorders, senility, circulatory disorders, edema and Raynaud's disease (a vascular disorder). Ginkgo extract improves the quality and increases the quantity of capillary circulation, thus increasing blood flow to the brain, heart and tissues in organs and glands, Schechter says. In addition, he notes, the flavonoids in **ginkgo** are potent free radical scavengers.

# **Ginkgo Biloba**

 Experts at the Mayo Clinic state some patients have success when taking the herbal supplement "ginko biloba". The herb has been reported to relieve leg pain in some PAD patients by thinning the blood.

The experts at the Mayo Clinic caution people who are considering "ginko biloba" as an alternative treatment. The herb can cause severe amounts of bleeding when taken in *large* doses. Many PAD patients are prescribed anticoagulant and anti-platelet therapy, such as aspirin and other medications; adding ginkgo biloba, also an anticoagulant, may increase bleeding to dangerous levels. A *standard dose* of ginkgo

biloba is 120 mg/day; however, a 1999 **Study found** a dose double the standard dose was more effective in treating symptoms of leg pain.

## **Ginger**

## Ginger Root (1) 550 mg Nature's Way 13108

- **Ginger root** Zingiber officinale) has been used in China for over 2,000 years under a belief that it aids digestion and treats uspet stomach, diarrhea, and nausea. TCM also teaches that it helps treat arthritis, colic, diarrhea, and heart conditions. Traditional Chinese medicine believes that it treats the common cold, flu-like symptoms, headaches, and menstrual cramps. Today, health care professionals commonly recommend to help prevent or treat nausea and vomiting associated with motion sickness, pregnancy, and cancer chemotherapy. It is also used as a digestive aid for mild stomach upset, as support in inflammatory conditions such as arthritis, and may even be used in heart disease or cancer. [61]
- <u>Ginger</u> has been used as a stimulant of the peripheral circulation in cases of bad circulation, chilblains and cramps. In cases of fever, <u>ginger</u> may act as a diaphoretic to promote perspiration. It may also be effective, as a gargle, in relieving sore throats.
- The second spice that should be on everyone's mind (pun intended) is **Ginger**. It was reported several years ago that ginger might indirectly lower the risk of Alzheimer's via its anti-inflammatory properties.
- It turns out that ginger decreases prostaglandins. These are the body's chemicals that lead to inflammation and perhaps other chronic diseases. Thus, ginger may ease those minor aches and pains in much the same way as aspirin (without the side effects, such as upset stomach), while simultaneously helping maintain brainpower.
- **Ginger** pens enzymes , help with dissolving unsoluables, along with enzyme 15 Serrapeptase
- The second spice that should be on everyone's mind (pun intended) is <u>Ginger</u>. It was reported several years ago that <u>ginger</u> might indirectly lower the risk of Alzheimer's via its anti-inflammatory properties. It turns out that <u>ginger</u> decreases prostaglandins. These are the body's chemicals that lead to inflammation and perhaps other chronic diseases. Thus, <u>ginger</u> may ease those minor aches and pains in much the same way as aspirin (without the side effects, such as upset stomach), while simultaneously helping maintain brainpower.
- Take for stomach discomfort
- <u>Ginger</u> may darken the color of your urine, Alkalizing herbs alkalize the urine, lessen inflammation caused by bruising or trauma, or from infection, Streptococcus faecalis, Candida, Klebsiella, E. Coli
- Chinese medicine Beets, blackberries and rhubarb can turn your urine red or pink, Antimicrobial UTI use **ginger**, the most common cause of bacterial urinary tract infections, from adhering to the inner walls of the bladder.
- Dark circles around eyes, puffy eyes and frequent urination
- Alkaline cell performs its task of respiration, it continues to secrete metabolic wastes that are acidic in nature.

- Remove lactic acid, healthy pH levels, increase oxygen affinity of haemoglobin, and protect minerals including calcium, sodium, potassium and magnesium
- Symptoms of a weakened urinary system include aching teeth, brittle bones and joints
  (primarily lower back, knees and ankles), hair loss, poor circulation, fluid retention, swollen
  tongue, dark circles around eyes, puffy eyes, and frequent urination. Nature's Sunshine's
  Juniper and Parsley Combination is the key product for strengthening the urinary system.

#### **Tumeric**

<u>Tumeric</u> is a member of the <u>Ginger</u> family Natures Bounty Inc. Turmeric Capsules

Natures Bounty 450 mg

NIH/ Ultimate Herb/DNA

This is the spice that makes Indian food yellow and is responsible for curry's delectable flavor. It is also the spice that gives mustard its bright yellow color. Interestingly, in Biblical times, the smell of **turmeric** was so loved that it was used in perfume making. Its unique and quite pleasant scent makes this spice one of my all-time favorite aromas. Be careful though, some people use the term curcumin interchangeably with turmeric. But curcumin is just an extract of turmeric. So be sure to buy and use the whole spice **tumeric**, because it contains all of the ingredients and micronutrients. Studies have shown that turmeric activates the expression of a gene that is a powerful antioxidant in the brain. Moreover, researchers at UCLA reported five years ago that one of the ingredients in turmeric actually inhibited the production of amyloid plaques in the brains of rats. Guess what? These plaques are the same plaques implicated in Alzheimer's disease. In fact, not only did the spice inhibit the NEW production of these plaques, the OLD plaques actually decreased! This is something no drug has ever done! Eat the spice **turmeric** whenever you can. It is a powerful chemical that probably deactivates the plaque-forming process due to its similarity to the plaque's structure (just like ginger). More science is forthcoming, but it is pretty clear that this spice is another winner if you're looking to improve your brainpower.

Curcumin, the Indian Spice **Turmeric** (Zingiberaceae). Curcumin is an antioxidant, anti-inflammatory, active principal ingredient of the curry spice, turmeric. The compound is marketed as a dietary supplement [140] and has attracted interest as a cancer-preventive agent [57]. It is well known that curcumin prevents the onset of inflammation by inhibiting the activation of nuclear factorkappa beta (NF-κB), the production of TNF-α, interferongamma (IFN-γ), and NO, and the gene expression of inducible nitric oxide synthase (iNOS) [141–143]. It acts by transrepressing NF-κB, activating protein-1, and the signal transducer and activator of transcription-1 [144–148]. Curcumin activates PPAR-γ in Moser cells, a human colon cancer cell line [145], and is able to suppress sepsis through PPAR-γ [146]. In addition, it increased PPAR and decreased iNOS gene expression in infected macrophages, as well as downregulated IFN-γ production by primed lymphocytes [147]. Curcumin action on PPAR could involve a curcumin-responsive element that resides in the PPAR-γ gene regulatory region [148].

## The value of Curcumin

Curcumin is the yellow pigment of turmeric ( curcuma longa )--the chief ingredient in curry, with beneficial antioxidant and anti-inflammatory effects. One of the advantages of curcumin over vitamins C and E is that while these nutritional antioxidants are effective against only water- and fat-soluble pro-oxidants, respectively, curcumin is effective in protecting against both. Curcumin enhances the body's levels of antioxidant compounds such as glutathione and superoxide dismutase, promoting the proper detoxification of cancer-causing compounds by the liver. Cigarette smokers receiving curcumin demonstrate significant reduction in the level of urinary-excreted

mutagens--an indication of the ability of the body to rid cancer-causing compounds via detoxification mechanisms.

## Guaifenesin

## Guaifenesin (2-4-8) 600 mg GuaiAID 00026

http://www.guai-aid.com/

<u>Guaifenesin</u>: Derived from a tree bark extract called guaiacum, guaifenesin was first used during the 16th century. It has been found to be very effective in the treatment of colds and flu.as it helps loosen phlegm (mucus) and thins bronchial secretions to rid the respiratory passageways of mucus.

<u>Guaifenesin</u> reversal process, which rids the body of "metabolic debris", Lumps decrease in size, <u>Guaifenesin</u> increases urinary excretion of 5HIAA, a serotonin metabolite, Sodium-phosphate cotransporters. Guaifenesin pulls ATP to convert to a simple form, pulling the Mitochondria into G0.

## **Dodder seed**

<u>Dodder seed (2) extract 600mg 20:1 60-Cap, Barlowes Herbal Elixars – Tu Si Zi, \$12.95</u> <a href="https://barlowesherbalelixirs.com/search?">https://barlowesherbalelixirs.com/search?</a> orderby=position&controller=search&orderway=desc&search query=dodder

http://www-personal.umich.edu/~rburnham/SpeciesAccountspdfs/CuscepitCONVFINAL.pdf

Background (herbs2000,com)

- AKA (Latin) <u>Cuscuta epithymum</u> (Cuscuta epithymum extract) Etymology: In Latin, Cuscuta means Dodder. However, Cuscuta is thought by some to have Arabic origins in the word "Kushkut." The specific epithet suggests the plant that this dodder was found growing on: Thyme. The Greek prefix "epi" means upon or over, and "thymum" is Latin for thyme
- <u>Cuscuta epithymum</u> Sievers ex Ledeb., Cuscuta epithymum Webb & Berthel., Cuscuta epithymum Thuill., Cuscuta epithymum Bové ex Choisy
- Yellow-orange, spaghetti-like vine
- Dodder contains flavonoids (including kaempferol and quercitin) glycoside, saponins, and hydroxycinnamic acid.

http://en.wikipedia.org/wiki/Cuscuta

- The methanol extract of the plant showed moderate antibacterial activity against four bacterial strains such as Bacillus megaterium (11.63±0.10 mm), Pseudomonas aeruginosa (6.13±0.10 mm), Escherichia coli (9.72±0.21 mm) and Salmonella typhi (9.95±0.07 mm)
- Previous studies have indicated that some species of Cuscuta possess anticancer activity on various cell lines.
  - $\circ$  Volatiles, including 2-carene, α-pinene, limonene, and β-phellandrene.
- <u>Dodder</u> posses medicinally properties, mentioned in the "Materia Medica" written by the Greek physician Dioscorides around the 1st century AD, the physician states dodder was used in an herbal combination with honey to purge "<u>black bile</u>" from the body.
- In the Middle Ages in Europe, the European herbalist Nicholas Culpeper in his writing in 1652 recommended plucking Dodder from the thyme it was parasitizing, according to Culpeper it was effective in treating parasite problems. This early herbalist thus makes an interesting inference the use of the dodder "to purge black or burnt choler" from the body.

- Free radical scavenging properties (Middleton and Kandaswami, 1992; Okwu and Orji, 2007).
- Carminative prevent formation of gas
- · Anti-bilious Countering liver disorder
- Cuscuta epithymum possesses mild laxative and diuretic properties.
- LC50 and LC90 values of 36.31 μg mL-1 and 83.18 μg mL-1 by brine shrimp lethality
- Test shows yellow urine removal ammonia only happens when 2 dodder capsules taken.

#### **White Willow Bark**

## White Willow Bark (1-2-6) 500mg Swanson SWH148

AM/PM 2-1 White Willow bark 400mg NOW 04775

(natural aspirin - blood clot probability reduction)?

http://www.swansonvitamins.com/swanson-superior-herbs-maximum-strength-white-willow-bark-500-mg-60-veg-caps

Salix genus plants were used since the time of Hippocrates (400 BC) when patients were advised to chew on the bark to reduce fever and inflammation. Willow bark has been used throughout the centuries in China and Europe to the present for the treatment of pain (particularly low back pain and osteoarthritis), headache, and inflammatory conditions such as bursitis and tendinitis. The bark of **white willow contains salicin**, which is a chemical similar to aspirin (acetylsalicylic acid). It is thought to be responsible for the pain-relieving and anti-inflammatory effects of the herb. In the 1829, salicin was used to develop aspirin. White willow appears to be slower than aspirin to bring pain relief, but its effects may last longer.[90]

Promotes joint and muscle comfort, Without stomach irritation Reduce blood clotting and reduces stroke potential

#### Horse chestnut

## Horse chestnut (1-2) 300mg NOW 4713 std. 20% Aescin

http://www.swansonvitamins.com/now-foods-horse-chestnut-300-mg-90-caps

NOW Horse Chestnut Extract 300 mg 90 Caps Swanson \$8.99 Item: NWF553

Horse chestnut may be useful against edema, inflammation, and venous insufficiency, especially with circulation in the legs

Heal veins, stop lymph fluid leaks, Repair vein weaknesses and holes, Horse chestnut: Vein strengthener

Horse chestnut (Aesculus hippocastanum) products (including both oral and topical dosage forms) are the single most widely prescribed remedy in Germany for edema with chronic venous insufficiency (CVI)—a condition sometimes associated with varicose veins. At least thirteen placebocontrolled, double-blind studies published between 1973 and 1996 show that oral standardized horse chestnut relieves CVI.

Horse chestnut extracts help veins withstand damage, reduce capillary-wall permeability, and prevent absorption of damaging UV radiation. A compound in the extracts called aescin (or escin) helps seal tiny openings in capillary walls, reducing the outflow of fluid into surrounding tissue

http://www.motherearthliving.com/health-and-wellness/treat-varicose-veins-naturaly.aspx#ixzz2lstfk3Vm

Drives parasites from body by means of suppression.

https://books.google.com/books?

 $\frac{id=XNwDq7hpo28C\&pg=PA215\&lpg=PA215\&dq=parasites+niacin\&source=bl\&ots=2779kYOy6v\&sing=tMn03PDGuUFh7jxCAtesGvX99pE\&hl=en\&sa=X\&ei=8d2MVI\_8MpT-$ 

yQTw1ILoCw&ved=0CEoQ6AEwCA#v=onepage&q=parasites%20niacin&f=false

## Milk Thistle

Milk Thistle (2-4-8) 300mg CAP.- Silymarin 80% w dandelion, NOW FOODS P#0476(5)3,SKU: 733739047533

#### 4753B/ Swanson NWF092

# AM/PM 2 Silymarin 300mg 80% w dandelion NOW P#0476(5)3, 4753B/ Swanson NWF092

https://www.swansonvitamins.com/now-foods-silymarin-2x-300-mg-100-vcaps

At first, in my naive mind I thought all I had to do was take my milk thistle along with lecithin (which contains high levels of PC) and I would enjoy this valuable benefit of more milk thistle getting to my liver. Researching further I discovered that the process was much more sophisticated than that. The researchers had actually bound one molecule from the milk thistle extract to two molecules of PC on a molecular level. The resulting compound was called Silybin Phytosome. The end result was more like a medicine and less like a supplement. I think of it as a cofactor delivery system. Your body soaks up PC like a sponge soaks up water. The milk thistle extract gets to go along for the ride through your bloodstream to where it does the most good, your liver.

Commission E Monograph (EU) has this to say, "The therapeutic activity of silymarin is based on two sites or mechanisms of action: (a) it alters the structure of the outer cell membrane of the hepatocytes in such a way as to prevent penetration of the liver toxin into the interior of the cell; (b) it stimulates the action of nucleolar polymerase A, resulting in an increase in ribosomal protein synthesis, and thus stimulates the regenerative ability of the liver and the formation of new hepatocytes." This means it both helps to protect and regenerate healthy liver cells.

#### **Dandelion root:**

<u>Dandelion root</u>: Contains bitter compounds that enhance efficiency of elimination and detoxification functions. Dandelion root also restores normal liver function and increases the flow of urine. It is used as a strong diuretic and relieves false sensations of urgency.

Protects liver - 4 Milk thistle capsules every day Milk Thistle 300mg CAP.- Silymarin 80% w dandelion,

NOW P#0476(5)3, 4753B/ Swanson NWF092

**Milk Thistle** is the #1 recommended natural herb for liver health, confirmed by hundreds of clinical studies to protect the liver against virtually all types of damage. Many years of research shows the active flavanoid-lignan (flavanolignan) group of constituents, called silymarin, contained only in the seed shell has liver-protective and regenerative properties, as well as antioxidant effects. The liver protective effects were known and written about in ancient times, leading to the active chemical, pharmacological, and safety research beginning in Germany in the 1950s

"I consider standardized milk thistle seed extract the most beneficial herbal product for liver detoxification, regeneration and protection, and, in general, one of the most universally necessary herbal products for the 1990s," says Schechter. He notes that the stress of toxins from chemical pollutants, pharmaceuticals, alcohol, tobacco smoke, drugs and different forms of radiation have cumulative side effects that need to be addressed.

More than 120 scientific studies have shown that <u>milk thistle</u> extract regenerates, regulates and strengthens liver functions. Because free radicals attack the liver, primarily the fat tissue in the liver, the antioxidant qualities of milk thistle are extremely beneficial. Milk thistle stimulates your body to produce superoxide dismutase, which is one of two primary antioxidants the body can manufacture.

## **Piperine**

Piperine (3-6) - 10mg Bioperine Nutrient Absorption Enhancer, 10 mg 60 Caps, Swanson Item #: SWU308

http://www.swansonvitamins.com/swanson-ultra-bioperine-nutrient-absorption-enhancer-10-mg-60-caps

Supplementation of piperine with coenzyme Q 10 for long time or at a high dose only can increase the bioavailability. It is assumed that piperine follows nonspecific thermogenic or bioenergetics properties for augmentation. [31].

## **Malic Acid**

Solaray

<u>Malic acid</u> comes from apples and is important in energy production at a cellular level. Several physicians have found malic acid supplementation reduces fatigue and pain. <u>Malic acid</u> helps to chelate excess aluminum.

## **SOD**

# SOD (1-2) 250mg SOD SWANSON Blend 60CT SW157 02157, Source naturals SN519 90ct \$6.19

#### **Elecampane Root**

Elecampane Root (4) 675mg Inula helenium Natures Wonderland 15400 Elecampane Capsules 2@ \$8.99 ea. P154x http://www.PennHerb.com
Or Swanson full spectrum 4:1 root 100mg SW1297
Firms stool, helps turn stool brown, dried up oil in stool, bulk up the stool.

#### **Blessed Thistle**

Blessed Thistle (2-4-6) 400mg Cnicus benedictus Natures Wonderland 05400

## **Black Cumin Seed**

# **Black Cumin Seed Oil** (1-2-3) Swanson EFAs Liq Vegcap 60 500 mg Liq Vegcap Item # SWE063

Many of black cumin's traditionally ascribed health benefits have been thoroughly confirmed in the biomedical literature. In fact, since 1964, there have been <u>458 published, peer-reviewed studies referencing it</u>.

We have indexed salient research, available to view on GreenMedInfo.com on our <u>Black Seed</u> (Nigella Sativa) page, on well over 40 health conditions that may be benefited from the use of the herb, including over 20 distinct pharmacological actions it expresses, such as:

- Analgesic (Pain-Killing)
- Anti-Bacterial
- Anti-Inflammatory
- Anti-Ulcer
- Anti-Cholinergic
- Anti-Fungal
- Ant-Hypertensive
- Antioxidant
- Antispasmodic
- Antiviral
- Bronchodilator
- Gluconeogenesis Inhibitor (Anti-Diabetic)
- Hepatoprotective (Liver Protecting)
- Hypotensive
- Insulin Sensitizing
- Interferon Inducer
- Leukotriene Antagonist
- Renoprotective (Kidney Protecting)
- Tumor Necrosis Factor Alpha Inhibitor

## **Oregano**

## Oregano Oil (0-3) Swanson Premium Sgels 120 150 mg Sgels Item # SW1016

\* Limit to 3 weeks duration, to prevent toxic buildup. **SIBO** 

**Oregano**: Early study shows that taking oregano by mouth may help get rid of parasites. Further research is needed to confirm these results. Research suggests that oregano is well tolerated in recommended doses. Avoid if allergic or hypersensitive to oregano or to other herbs from the Lamiaceae family including hyssop, basil marjoram, mint, sage and <u>lavender</u>. Use cautiously with diabetes and bleeding disorders because oregano may increase the risk of bleeding or decrease blood sugar levels. Pregnant or breastfeeding women should not consume oregano at doses above those normally found in food.

**Oregano**: Early study shows that taking oregano by mouth may help treat parasites. Further research is needed to confirm these results. Avoid in individuals with a known allergy or hypersensitivity to oregano. Based on historical use, it appears that oregano is well tolerated in recommended doses. However, reliable clinical trials demonstrating safety or efficacy of a particular dose or for a recommended treatment duration are currently lacking in the available literature. Oregano may lower blood sugar levels. Caution is advised in patients with diabetes or hypoglycemia,

and in those taking drugs, herbs, or supplements that affect blood sugar. Serum glucose levels may need to be monitored by a healthcare provider, and medication adjustments may be necessary. Oregano is not recommended at doses above those normally found in food during pregnancy and lactation due to a lack of available scientific evidence.

## **Pumpkin Seed**

Pumpkin Seed Oil (0-2) Swanson Premium Sgels 100 1,000 mg Sgels Item # SW364

http://www.amazon.com/Pumpkin-Seed-Oil-000-Sgels/dp/B00068U44S/ref=sr\_1\_1/188-1732236-2714103?s=hpc&ie=UTF8&gid=1449945957&sr=1-1&keywords=swanson+pumpkin+seed+oil

Unfortunately One needs to eat pounds of pumpkin seeds a day to equal 1 TBS of <u>Piperazine</u> citrate.

<u>Pumpkin seed</u>: Able to kill eggs, contains a natural fat that is toxic to parasite eggs. Curcurbitin in pumpkin seeds has shown anti-parasitic activity since it has the ability to paralyze worms so they drop off the intestinal walls Chinese scientists used pumpkin seeds to treat acute schistosomiasis and <u>Tapeworm</u> infestations. OIL

## Nutmeg with Buttermilk for Amoebiasis

Nutmeg is the seed of the nutmeg tree that is a source of essential oils and is known for its health benefits. One of the health benefits is the ability of nutmeg to detoxify the liver and kidney, which plays a particularly important role when it comes to home remedies for amoebiasis. When there is excessive strain on the liver on account of accumulation of toxin, it would help to take in a tonic of nutmeg towards detoxification. You could add freshly grated nutmeg to a glass of fresh buttermilk and enjoy the drink as a delicious home remedy for amoebiasis

## **Chaparral**

#### Chaparral (2-4) 500 mg Arizona Naturals 20201

Chaparral is essential for balancing Psora body types against a critical pathway in FXR Farnesoid chemistry where the uric acid and Bile acid are regulated by Ornathine chemistry. This critical missing acid at FXR can completely throttle your bodies ability to rid itself of Ammonia, and Uric Acid. I smelled like an Ammonia bottle for months. Got a crusty patch of skin on your knee or elbow? that is a genetic reversal of DNA that causes an immune response to a infection to attack your body, not the invader, causing local area of excess white blood cells that try to overwhelm the invader by mass movement of fluid and other T factors, which fights your body, not your infection, causing a real strain on your white blood cell chemistry. 30% of the world has a Psora body type, known for 5000 years, but medicine today is clueless about Psora. Chaparral deals with this reversal of genetic glue, to lower the inflammation of certain Tcell Thelper responses, preserving more IL6 IL10, IL17 function for your parasites, not destroying your own immune system.

## **Taurine**

**Taurine**: **Taurine**, or 2-aminoethanesulfonic acid, was originally discovered in ox (*Bos taurus*) bile and was named after taurus, or bull. A nonessential amino acid-like compound, **taurine**, is found in high abundance in the tissues of many animals, especially sea animals, and in much lower concentrations in plants, fungi, and some bacteria. As an amine, **taurine** is important in several metabolic processes of the body, including stabilizing cell membranes in electrically active tissues, such as the brain and heart. It also has functions in the gallbladder, eyes, and blood vessels, and it may have some antioxidant and detoxifying properties.

## Magnolia bark extract

## 2-3 magnolia bark 600 mg

CO2 extract (shield white blood T4 cells against microbes, levels IG and IL balance) http://www.amermed.com/magnoliabark.htm

Magnolia Extract (0-4) capsules, Amermed note: 12 capsules will stop the heart Magnolia Bark extract 600 mg CO2 extract method 600mg Magnolia Bark extract 4:1 Amermed, PPAR Gamma Interluken 17 antagonist, forces Tilter 1

## **Magnolia Bark**

### Magnolia bark

neo-ligand binds to site PPAR $\gamma$  that mytotoxin grabs, preventing immune system alteration. Plant lectins that can bind to sialylated glycans are from the leguminous tree Maackia amurensis. Although these lectins are discussed here, they do not show a "classical" R-type domain but instead have an L-type lectin domain. The cysteine-rich R-type domain of the MR binds other sulfated glycans and also N-glycans on pituitary glycoprotein hormones containing 4-SO4-GalNAc $\beta$ 1-4GlcNAc $\beta$ 1-2Mand1-R.nutraceutics that are reported to be able to modulate PPAR- $\gamma$ 9 expression or action.

There are other R-type plant lectins in the RIP-II class that are not toxic, and these include several proteins from the genus Sambucus (elderberry), such as nigrin-b, sieboldin-b, ebulin-f, and ebulin-r. All of the B subunits of these proteins appear to bind Gal/GalNAc, but they may have some differences in affinity and may recognize different Gal/GalNAc-containing glycoconjugates. Cell lines selected for resistance to killing by modeccin are not resistant to abrin and ricin, and vice versa. The glycan-binding specificity of these lectins should be explored more fully in the future using glycan microarrays and related screening approaches.

Previous studies have shown that MR expression (Yeast Response) can be positively modulated in vitro by many agents, in particular by 1,25-dihydroxyvitamin D3 (8), prostaglandin E2 (Standard Process Cataplex E2 90 Tablets) (9), IL-4, and IL-13 (10, 11).

## **Tunjuk Langit (Magnolia seeds)**

#### Sweetina Magnolia seeds (1) chopped in yogart Tunjuk Langit - rasha herbal

Used for: Severe immune malfunctions, very useful in blood toxin poisoning, near death recoveries <a href="http://rahsiaherbal.com/Herbal/TunjukLangit.aspx">http://rahsiaherbal.com/Herbal/TunjukLangit.aspx</a>

http://www.amazon.com/Natural-Herbal-Fructus-Swietenia-Macrophylla/dp/B00C4QBYUI/ref=sr 1 1?ie=UTF8&qid=1387469365&sr=8-1&keywords=All+Natural+Asian+Herbal+Remedy%2C+Sky+Fruits+%5BTunjuk+Langit+ %2F+Fructus+Swietenia+Macrophylla%5D+%2850g%29

Magnolia bark neo-ligand binds to site PPARy that mytotoxin grabs, preventing immune system alteration.

Once activated by their ligands, the PPARs translocate into the nucleus, form heterodimers with the retinoid X receptor (RXR), and subsequently bind to PPAR response elements (PPREs) that are located in the promoter regions of PPAR-responsive target genes (Bardot et al., 1993).

Binding of the PPAR-RXR heterodimers to the PPREs triggers further recruitment of diverse nuclear receptor coactivators (SRC-1, TRAP220, cAMP response element-binding binding protein, p300, PGS-1, and/or others), contributing to the transcriptional regulation of the target genes (Yu and Reddy, 2007). A combination of PPAR-γ ligands with a retinoid X receptor agonist (i.e., LG100268) or a retinoic acid receptor agonist (i.e., all trans-retinoic acid) enhanced immune differentiating effects.

## **FlaxSeed**

**Flaxseed** — one of the most concentrated vegetarian source of Omega-3 fatty acids found in nature — contributes to heart and while providing an energy source for the body. High-quality natural **Flaxseed** Oil is dispensed and processed without solvents, under a nitrogen blanket and special yellow lights, then encased in a protective amber-colored gelatin shell involved in cardiovascular disease, as well as inflammatory and immune disorders.

<u>Flaxseed</u> may have anti-inflammatory and anti-thrombotic activities. Development of atherosclerosis has been reduced by up to 69% in some studies using diets enriched with flaxseed. Flaxseed has been recommended by some for hyperlipidemia, to decrease platelet aggregation, and to help prevent heart attack and stroke. It may have some ability to lower blood pressure and to have anti-inflammatory effects in persons with arthritis, as well as assist in fighting breast cancer.

## **Cod Liver oil**

### **DHA** (Docosahexaenoic Acid)

Essential fatty acids are dietary fats required for a healthy nervous and immune system. For example, docosahexaenoic acid (**DHA**) is an important constituent of brain cell membranes. Many fats can be synthesized by the body, but some, like DHA, must be obtained through the diet. **DHA** is an omega-3 polyunsaturated fat, and the omega-3-type fats must be kept in balance with omega-6 fats to insure that proper physiological functioning can be maintained. While omega-6's are abundant in the typical American diet - occurring in most vegetable oils - omega-3's are harder to come by. One good source, however, is fish.

Long term use of <u>Fish Oil</u> help prevent aging skin, menopausal symptoms, promote better circulation, lower cholesterol, prevent blood clots, reduce heart related risk, and the pain of arthritis.

Omega-3 and polyunsaturated fatty acids found in fish and Alaska Deep Sea Fish Oil help to protect against heart and blood vessel disease. Natural, essential fatty acids are essential to normal human cell and tissue growth and maintenance. If not found regularly in the diet, the diet must be supplemented. These fatty acids are especially abundant in brain cells, nerve relay stations (synapses), visual receptors (retinas), adrenal glands, and sex glands. The most biologically active tissues in the body

## **GABA relaxer**, Country Life

B6, GABA, ...
GABA helps force sleep pattern, offloads Gluthione loop

## Melatonin

Melatonin w P5P Take with GABA relaxer above to sleep, ProHealth PH65

Stop ROS/ PLS talking from cell

Melatonin is produced in the body by the pineal gland in the brain.

Tryptophan, an amino acid found in food, is taken in by the body and made into serotonin, a neurotransmitter (conductor of nerve signals).

The pineal gland takes the serotonin and makes it into <u>melatonin</u>, but only during the night. (The enzymes in the brain which change serotonin into melatonin are inactivated by light). Norepinephrine is another neurotransmitter which assists in melatonin production.

It acts as a catalyst to melatonin production by stimulating cells in the pineal gland to begin making melatonin in the absence of light. Sometimes, for one reason or another, the body does not produce adequate amounts of melatonin for its needs. This can result in insomnia and depression, among other symptoms. The body's ability to synthesize **melatonin** may decrease with age. Melatonin is a highly potent antioxidant, which has been described as the pacemaker of the aging clock in humans. It is released every night as part of our time-dependent biorhythms to help induce sleep and recuperation from fatigue. Published studies indicate the importance of maintaining youthful levels of melatonin to help protect against age-related degenerative diseases.

#### St. John's Wort

St. John's Wort (Hypericum) has been studied for its potential antidepressant and antiviral effects. (Caution: Dr. Donald Brown of Bastyr University recommends that persons with fair skin avoid exposure to strong sunlight and other sources of ultraviolet light when taking St. John's Wort because of some cases of photosensitivity that have been reported. He also advises avoiding foods that contain tyramine, alcoholic beverages, and medications such as tyrosine, narcotics, amphetamines, and over-the-counter cold and flu remedies while taking St. John's Wort. St. John's Wort should not be taken while also taking prescription antidepressants. It is also Dr. Brown's opinion that St. John's Wort should not be used during pregnancy or lactation.)

## **Yogurt**

**Greek Gods Plain Greek Yogurt** 

## **DHEA**

DHEA 25 mg - w calcium carbonate, Adrenal exhaustion, chronic infection with NAC
Natrol DHEA 50 # 16106
DHEA (Dehydroepiandrosterone)

25 mg, 100 capsules

Item Catalog Number: 00335

**<u>DHEA</u>** promotes Th1 immune response.

It has been shown that the serum hormone **DHEA** often declines by 75%–80% from peak levels by age 70, leading to hormonal imbalances that can affect one's quality of life. Peak blood levels of **DHEA** occur at approximately age 25, decreasing progressively thereafter.

Since 1981, several thousand studies have been published on **DHEA**'s various benefits, including immunomodulatory properties as well as positive effects on mood, quality of life, and body composition. It has been proposed that restoring the circulating levels of **DHEA** to those found in young people may improve well-being and sexual function. The studies of **DHEA** therapy in women with adrenal insufficiency also suggest beneficial effects on well-being, mood, and sexuality. **DHEA** could be of benefit to the normal aging brain.6-8 Some studies have reported **DHEA** may improve mood and alleviate melancholy. In addition, recent studies have shown that **DHEA** has the capacity to improve endothelial function by a number of complex processes. One of them is increasing nitric oxide synthesis.

In a randomized, double-blind, controlled trial, **DHEA** replacement therapy for one year helped protect hip bone mineral density in older adults and spine bone mineral density in older women. **DHEA** has also been shown to support a healthy circulatory system and joint/bone health.

## **DHEA** usage and safety precautions

Because of the overwhelming evidence connecting low levels of **DHEA** to problems associated with aging, Life Extension suggests that all people over age 40 begin **DHEA** therapy. For most people, the starting dose of **DHEA** is between 15–75 mg, taken in one daily dose. Many studies have used a daily dose of 50 mg.

Since almost everyone over age 35–40 has lower than optimal levels of **DHEA**, most people begin supplementation and then test their blood **DHEA** levels later to make sure they are taking the proper dose. People over age 40 who do not supplement with **DHEA** usually have serum levels well below optimal since a steady decline takes place after the third decade in life.

#### **DHEA** precautions for men

Before attempting to restore **DHEA**, men should know their serum PSA (prostate specific antigen) level. Men with prostate cancer or severe benign prostate disease are advised to avoid **DHEA** since it can be converted into testosterone (and estrogen). Therefore, men are advised to have a PSA and digital rectal exam before initiating **DHEA** to rule out existing prostate disease.

Other daily nutrients:

- •Vitamin D 5,000 10,000 IU
- •Vitamin E (D-tocopheryl succinate) 400 IU
- Selenium 200 mcg
- •Gamma E Tocopherol with Sesame Lignans 200 mg

- •Lycopene Extract 10 40 mg
- •Boron 3 10 mg
- •Cruciferous Vegetable Extracts 500 1000 mg

## **DHEA** precautions for women

Women should consider estrogen and testosterone testing when they take their **DHEA** blood test in order to evaluate **DHEA**'s effect on their blood levels of these hormones.

Women who have been diagnosed with an estrogen-dependent cancer should consult their physicians before beginning the **DHEA** restoration process.

Taking the following other nutrients to maintain a healthy balance each day:

- •Melatonin 300 mcg 3 mg (nightly)
- •Vitamin E (D-tocopheryl succinate) 400 IU
- •Broccoli Extract (as found in Triple Action Cruciferous Vegetable Extract) 400 mg
- •Indole-3-carbinol (as found in Triple Action Cruciferous Vegetable Extract) 80 160 mg
- •Vitamin D3 5,000 10,000 IU
- •Gamma Tocopherol with Sesame Lignans 200 mg

## **NADA**

NADH: The object of new and ongoing research is an element, a co-enzyme called NADH, short for Nicotinamide Adenine Dinucleotide, with the 'H' standing for hydrogen. On the basis of such research, NADH is considered by many to be amongst the most biological potent elements found in the body.

" Science is learning that NADH may have a direct positive involvement in enhancing the immune system, fighting disease and in repairing the body damage caused by disease."

NADH is also a potent energy producer that works directly with your metabolism to create more energy for every cell in your body, including muscle, nerve cells and the brain cells. While creating energy, it helps your body to burn all foods. And, it has a potent effect on the memory cells, too, in addition to being a mood enhancer (as it has a role in the development of serotonin and dopamine, that cause a feeling of well being).

## Cranberry

Cranberry contains a compound that prevents bacteria from adhering to the walls of the bladder and rest of the urinary tract. This prevents the bacteria from spreading and eventually results in the halt of infection. Using cranberry on a regular basis may help prevent the formation of kidney stones.

### 5.5.15 Nutmeg with Buttermilk for Amoebiasis

Nutmeg is the seed of the nutmeg tree that is a source of essential oils and is known for its health benefits. One of the health benefits is the ability of nutmeg to detoxify the liver and kidney, which plays a particularly important role when it comes to home remedies for amoebiasis.

When there is excessive strain on the liver on account of accumulation of toxin, it would

help to take in a tonic of nutmeg towards detoxification. You could add freshly grated nutmeg to a glass of fresh buttermilk and enjoy the drink as a delicious home remedy for amoebiasis

#### 5.5.15.1 Amoebiasis and Giardiasis: Fennel Seed

Amoebiasis and Giardiasis, the following have been found to be effective; goldenseal or barberry or oregon grape, together with garlic, wormwood and / or grapefruit seed extract. Foeniculum vulgare and Anethum Foeniculum, Bari-Sanuf, Bitter Fennel, Carosella, Common Fennel, Fennel Oil, Fennel Seed, Finnochio, Florence Fennel, Foeniculi Antheroleum, Foeniculum Officinale, Foeniculum Capillaceum, Garden Fennel, Large Fennel, Sanuf, Shatapuspha, Sweet Fennel, Wild Fennel.

#### **CONSTITUENTS**

The essential oil contains anethole (50 to 80%), limonene (5%), fenchone (5%), estragole (methyl-chavicol), safrole, a-pinene (0.5%), camphene, b-pinene, b-myrcene and p-cymene. The seed also contains fiber and complex carbohydrates.

Fennel seed teas are helpful for colicky infants, but fennel seed oil should never be given to infants or young children because of the danger of spasms of the throat.

Weight loss lost an average of 19.3 pounds in 28 days

Give her fennel extract or boil 1 spoon of fennel seeds in 1 glass water, when water reduced to 1/4 th ,cool it at room temp and give 5 spoons in every 2 hours. Avoid dairy products, spicy things, raw veggies and salads. Can give her isabgol husk mixed with yogurt and roasted cumin seeds. Powdered nutmeg consumed with buttermilk every day, is also an effective cure for amoebiasis.

In cases of Blastocystis, consider using oregano, wormwood, black walnut, cloves, quassia, and goldenseal.

In cases of Cryptosporidiosis, consider using products containing garlic with oils of coconut, oregano, clove and cinnamon.

A number of products contain a variety of anti-parasitic agents. I find that most products from well-known suppliers are very effective. It is best to follow the suggested dose on the container and to treat for approximately one month, although recommendations vary from two weeks to three months.

The duration of treatment really depends on the severity of the problem, the speed with which your client responds to treatment and whether conventional antibiotics, such as Metronidazole, Tinidazole, Paromomycin or Doxycycline, have been prescribed. Some practitioners give high doses for the first two weeks then reduce to recommended doses.

Remember that these herbs can themselves cause nausea and vomiting and can aggravate stomach ulcers and inflammatory bowel conditions such as Crohn so Disease. Care should be taken to check the suitability of your chosen product in pregnancy and lactation, and in children.

All cases should be followed-up for a month after symptoms have disappeared.

## 5.5.16 Picrorhiza kurroa

#### http://www.nutripeople.co.uk/print/692

Background & objectives: Picroliv, isolated from the root and rhizome of Picrorhiza kurroa, is known to have significant hepatoprotective activity. Its effects against Entamoeba histolytica induced liver damage are not studied. This study aims to evaluate the hepatoprotective action of picroliv against the hepatotoxic changes induced by carbon tetrachloride (CCl4) and E.histolytica infection in three animal models.

picroliv isolated from the root and rhizome of Picrorhiza kurroa (Scrophulariaceae)

Thyme leaves
Thymus vulgaris
See Tomillo

#### ALSO KNOWN AS

Thymus vulgaris, Creeping Thyme, French Thyme, Garden Thyme, Common Thyme, Mountain Thyme. Botanists refer to the species of the herb used in cooking as garden thyme and to other species as simply "thyme" As with other mints, there are many thyme variants with interesting tastes and fragrances, including lemon thyme, orange thyme, and caraway thyme.

#### **CONSTITUENTS**

Alpha-linolenic acid, anethole, apigenin, borneol, caffeic acid, calcium, chromium, eugenol, ferulic acid, geraniol, kaempferol, limonene, lithium, luteolin, magnesium, manganese, methionine, p-coumaric acid, potassium, rosmarinic acid, selenium, thymol, tryptophan, ursolic acid.

Bulk mountain rose herbs 4 0z 3.00

Do not take thyme as a medicine if you have a duodenal ulcer or if you have thyroid disease.

#### THYMUS SERPYLLUM

Action ► Antiseptic, antibacterial, antifungal, antiviral, antispasmodic, mild sedative, expectorant. T. serpyllum and T. vulgaris L. are used for coughs and common cold. Key application ► German Commission E approved T. vulgaris for symptoms of bronchitis, whooping cough and catarrhs of the upper respiratory tracts. Also to treat stomatitis. (ESCOP.)

The British Herbal Pharmacopoeia recognizes expectorant activity of T. serpyllum.

T. serpyllum contains more linalool and p-cymol than Garden Thyme (T. vulgaris). Major constituent of the volatile oil of both the species (highly variable) is thymol; with carvacrol (lesser amount in T. serpyllum, higher in T. vulgaris), 1,8-cineole, borneol, geraniol, linalool. bornyl and lina- lyl acetate, thymol methyl ether and alpha-pinene. Flavonoids include apigenin, lute- olin, thymonin, naringenin; other constituents include

labiatic acid, caffeic acid, tannins.

The flavonoid fraction has shown to have a potent effect on smooth muscle on guinea-pig trachea and ileum.

Thymol is expectorant and antiseptic. Thymol and carvacrol are spasmolytic. Thymol is also urinary tract antiseptic and anthelmintic.

Dosage ► Whole plant—3–5 g powder. (CCRAS.)

https://www.google.com/search?

hl=en&source=hp&q=mountain+rose+herbs&gbv=2&oq=mountain+rose+herbs&gs\_l=heirloom-hp.3..5j0l9.3125.9813.0.9969.19.15.0.4.4.0.188.2045.0j13.13.0....0...1ac.1.34.heirloom-hp..2.17.2232.kAW1yjv5lul

5.5.17 Culantro

http://www.caribbeanseeds.com/culantro.htm

Culantro (Eryngium Foetidum) is a flavorful herb used in caribbean cooking. Puerto Rico uses it extensively in all kinds of stews, soups, beans, asopao, etc. It is a more flavorful substitute for Cilantro for all your culinary creations. It is definitely NOT EASY to come by the seeds for this fragrant herb until now. Another name for this herb in Puerto Rico is RECAO. In Asia it is also known as Long Coriander. Culantro is also known as: ngo-gai, spirit weed, long coriander, false coriander, shadon beni, black benny, recao de monte, Mexican coriander, and well over 65 more names in different parts

#### 5.5.18 Clove Buds

## CLOVES (Syzygium aromaticum)

Syzygium aromaticum. They are native to the Maluku Islands in Indonesia, and are commonly used as a spice. Cloves are commercially harvested primarily in Indonesia, India, Madagascar, Zanzibar, Pakistan, Sri Lanka—and the largest producer, Pemba Island, just off the coast of Tanzania.

The clove tree is an evergreen that grows up to 8–12 m tall, with large leaves and sanguine flowers grouped in terminal clusters. The flower buds initially have a pale hue, gradually turn green, then transition to a bright red when ready for harvest. Cloves are harvested at 1.5–2.0 cm long, and consist of a long calyx that terminates in four spreading sepals, and four unopened petals that form a small central ball.

Active compounds[edit]

### https://en.wikipedia.org/wiki/File:Eugenol\_acsv.svg

The compound eugenol is responsible for most of the characteristic aroma of cloves. Eugenol comprises 72-90% of the essential oil extracted from cloves, and is the compound most responsible for the cloves' aroma. Other important essential oil constituents of clove oil include acetyl eugenol, beta-caryophyllene and vanillin, crategolic acid, tannins such as bicornin,[22] gallotannic acid, methyl salicylate (painkiller), the flavonoids eugenin, kaempferol, rhamnetin, and eugenitin, triterpenoids such as oleanolic acid, stigmasterol, and campesterol, and several sesquiterpenes.[23][24]

Eugenol can be toxic in relatively small quantities; with a dose of 5 - 10 ml severely affecting a 2 year old child.[25]

cloves remove the parasite eggs.

on the labeling of blood constituents with technetium-99m and on the morphology of red blood cells

Modulatory influences of clove (Caryophyllus aromaticus, L) on hepatic detoxification systems and bone marrow genotoxicity

Today in America Clove is used to treat worms, viruses, candida, and various bacterial and protozoan infections

2-methoxy-4-(2-propenyl)-phenol

Caryophyllus Aromaticus night sweats pungent heat upon the

skin; on which account I have discarded them from all of my sweating mixtures Eugenia ... It also promotes sweating with fevers, colds, and flu, which is very healing. A strong antiparasitic and digestive oil of ... cramps, hot flashes, night sweats, water retention, cellulite, and headaches.

https://www.google.com/search?

hl=en&source=hp&q=Caryophyllus+Aromaticus+night+sweats&gbv=2&oq=Caryophyllus+Aromaticus+night+sweats&gs l=heirloom-

hp.3...59796.65687.0.65890.14.3.0.11.0.0.141.376.0j3.3.0....0...1ac.1.34.heirloom-hp..12.2.266.ROil2K2pajl

http://www.essential-oils-farmacy.com/there-is-an-oil-for-that.html

http://www.scortishealthcare.com/productinfo.asp?itemno=Gynex-Forte

http://www.hogwartsishere.com/library/book/2026/read/?chapter=1

http://everveda.com/library/

http://www.potentiallifeinstitute.com/essential-oils-and-herbs

http://www.herbalremedies.com/clove.html

Ingredients Clove Powder. Directions

Take 1/2 to 1 Teaspoons in juice or tea. Or as directed by recipe.

Our Low price: \$8.99

#### Product Details:

Product Name: Starwest Botanicals Organic Cloves WholeSize: 1 LbUPC: 767963025470 Whole cloves come from a tree with the Latin botanical name of Syzygium aromaticum, where the flower buds are dried to produce this popular spice. The tree is native to India and Indonesia, but is also found in Sri Lanka, Pakistan, Zanzibar and Madagascar. We offer this spice in powder and whole forms, as well as organic and kosher varieties. When you buy cloves from Starwest Botanicals, you will notice that we directly source our dried spices and herbs from our producers worldwide, and then process them all in-house in order to bring you the finest quality dried herbs and spices available today.

Mountain rose herbs 4 oz \$6.50

https://www.mountainroseherbs.com/search?page=1&g=clove&utf8=%E2%9C%93

Monterey Bay Spice company 1 pound 14.00

http://www.herbco.com/p-555-cloves-whole.aspx

Cloves: This magical flower bud is a powerhouse of goodness when it comes to clearing your gut of creepy crawlies! From the plant Syzygium aromaticum, clove has also been used since ancient times in both Ayurvedic and Western herbal medicine. Originally used as a carminative (preventer of intestinal gas formation/expulsion), cloves have also been proven to work for indigestion, abdominal cramping and pain, nausea, and even as a topical application for toothaches! For our purposes, the key constituents in clove that pertain to its anti-parasitic activity are eugenol, eugenol acetate, beta-caryophyllene, flavonoids, tannins and phenolic acids, with EUGENOL being the main medicinal property. Both water and alcohol preparations of clove bud have shown to be nematocidal and particularly effective against Anisakis spp. Larva, Caenorhabditis elegans, Rhabditis macrocerca and Ascaris suum. Perhaps most important though is that clove doesn't differentiate between the eggs of different species and can be instrumental in preventing recurrent infections by dislodging eggs from the intestinal wall and killing them.

There is a particular phenomenon outlined in the above-mentioned Townsend Letter article that illustrates this concept (however gruesomely). It is called "Larva Bursting" and involves tearing the larva's outer covering, exposing its intestines. Eugenol in cloves has been shown again and again to cause this bursting of worm larvae, especially when synergistically combined with tannins which are normally not larvicidal on their own. Together, the two chemicals double the killing capacity of both substances. To sum up, "the synergistic action of tannins and an anthelmintic not only damages the worms irreversibly, but also, in some instances markedly reduced the required amount of the anthelmintic."(pp.4) Take less of both together to increase your killing capacity!

5.6 Oil of Oregano

- · Oregano oil package: The European herb Oregano is one of the most nutritious substances on earth, with 42 times more antioxidants than apples and 30 times more than potatoes.
- · Additionally, it's loaded with antimicrobial compounds that fight cold-causing bacteria and researchers found that it destroys giardia.
- · Giardia is an amoeba, common throughout the world. There are more than 200 million people infected with Giardia each year. Research studies indicate that oregano is effective against Giardia.[8] In one study, oregano oil proved to be more effective than the commonly prescribed drug, tinidazol.[9]
- · Oregano oil even protects against parasitic infiltration both within the body, as well as the physical environment. This includes round worms, tape worms, bed bugs, lice, fleas and mosquitoes.
- · Follow package directions, each manufacturer may have different strength.
- In vitro studies have demonstrated antibacterial activity of the essential oils against some of the most common food-borne pathogens including Listeria, Salmonella, E-Coli, and Shigella dysenteria (causes dysentery).[10,11] In fact, you can add oregano oil to your eggs, meat, seafood, salad, or other dishes to actually prevent food poisoning microbes from growing. You can also add a few drops to your left-overs to prevent food spoilage.
- · Effective against and inhibits growth of E. Coli (Escherichia coli O157:H7)
- · Effective against and inhibits growth of Proteus, bacteria that also cause UTIs.[5,6]
- · Oregano oil may be a more powerful anti-fungal agent than many commercial products on the market
- · Oregano oil is effective in eliminating the fungus that causes athlete's foot as well as treating fungal infections on the skin and nails.
- · Oregano oil benefits also include inhibiting the Candida fungi (yeast). It is more effective than other natural supplements in cases of yeast infections resistant to the conventional drug fluconazole (Diflucan), according to research published in the Canadian Journal of Microbiology.[7]

#### 5.7 Make your own capsules

- take enzymes containing large amounts of Protease (which digests protein and parasites, bacteria and viruses are protein)

#### Household herbs:

Nutmeg, Bay Leaves and Cloves contain Eugenol, a powerful killer of parasite larva and eggs

Turmeric- anti-inflammatory wound healing worm-expelling body purifier Dosing

Take once a day before lunch

First day- 1 cap

2nd day- 3caps

3rd day- 6 caps

4th day- 10 caps if on food, 5 caps if on total liquid cleanse

days 5 to 90-10 caps, 1 day of rest per week

STAY ON THIS FOR 3 MONTHS !!! Adult parasites leave eggs behind when they die. It takes time to rid your body of all stages.

Take probiotics at the end of the day because Parasite-Free™ is powerful and knocks out everything

Take some sort of fiber during cleanse, also colon cleansing is recommended to flush out the dead parasites.

Do NOT feed the parasites! This means NO bread, pasta, sugar, dairy, meat, sushi, cereal, baked, processed or fast foods

Scrub your hands, keep fingernails clean, Wash all produce, pet owners wear slippers, practice impeccable hygiene

5.8 Critical Organs and Systems

5.8.1 Blood PH

1 tea. Bob's Red Mill Baking Soda in water

5.8.2 Circulation Veins

Ginkgo Boblica, White Willow bark, Asprin in a pinch

5.8.3 Magnesium

Magnesium Citrate, Potassium Citrate, Magnesium Sulfate in water

## **5.8.4 Liver Protection and Detoxification Sulfates**

1 tea. MSM in water

Clevland Clinic formula

- · Milk Thissle
- · CQ10
- · ALA
- · L-Carnitine

#### 5.8.4.1 Liver Toxin Flush

http://curezone.com/cleanse/liver/epsom\_salt\_and\_liver\_flush.asp

Magnesium sulfate reduces striated muscle contractions and blocks peripheral neuromuscular transmission by reducing acetylcholine release at the myoneural junction. In emergency care, magnesium sulfate is used to manage seizures associated with toxemia of pregnancy. Other uses include uterine relaxation (to inhibit contractions of premature labor), as a bronchodilator after beta-agonist and anticholinergic agents have been used, replacement therapy for magnesium deficiency, as a cathartic to reduce the absorption of poisons from the Gl tract, and in the initial therapy for convulsions. Magnesium sulfate is gaining popularity as an initial treatment in the management of various dysrhythmias, particularly torsades de pointes, and dysrhythmias secondary to a tricyclic antidepressant overdose or digitalis toxicity. The drug is also considered as a class Ila agent (probably helpful) for refractory ventricular fibrillation and ventricular tachycardia after administration of lidocaine or bretylium doses.

Bryant died because she got too much of the drug too fast. She might have given too much of the drug to start with, or incorrectly programmed the pump that controls the IV solution. Bryant's lawyer has said she was given a 40-gram solution, while the safety group recommends a 20-gram solution. The dangers of magnesium sulfate led the Institute for Safe Medication Practices to classify it as one of 25 "high-alert" medicines. Others include chemotherapy drugs, insulin and warfarin, a blood thinner.

# Rosehip seed

## 3-4 cups of rosehip seed tea

(restores IL17 T helper cells, prevents D3 twist in DNA helix) http://www.mountainroseherbs.com Rosa spp. Origin- Chile https://www.mountainroseherbs.com/search/search.php? page=3&refine=y&keywords=Rosehips

If your immune system is shot, start by buying Rosehip seeds from Mountain Rose Herbs, buy 4 pounds of seeds, get a krupps grinder, put the seeds in the grinder and make a powder, takes about 16 seconds. I put in enough seeds to cover the blade. Put the seeds in a coffee machine, do not use chlorine water. drink several coups a day. The **vitamin A and C** from chilean rosehip seeds is the best in the world. Take a little **stinging** nettle root, say 3-4 per day. Start with 4 zinc sulfates per day. Start with 3-4 selenium chelate per day. Take magnolia bark extract 4:1 Ameramed, take 2-3 per day.

Rosehip seed tea. Mountain rose herb, bulk rosehip buds, grind in coffee maker.

https://www.mountainroseherbs.com/products/rosehips/profile

Rosehip seeds grabs Vitamin D3 ( Primary Disease adaptogen 3T3 receptor, preventing white

blood cells from inhibiting learned immune response T helper cells- invader infection signals. Immune system is no longer controlled by mytotoxin. Rosehip seed is an excellent source of topical trans-retinoic acid (vitamin A) in a natural form. Retinoic acid, found in Tretinoin, Steroid receptor superfamily has identified certain members as molecular targets for cancer therapy (1). They include estrogen receptors, retinoic acid receptors, retinoid X receptors (RXR; the RXR-specific ligands are termed "rexinoids"), and the vitamin D receptor (the vitamin D-specific ligands are termed "deltanoids"). These nuclear receptors are putative cancer therapy targets because they function as transcription factors that control the expression of many genes related to cell differentiation (1, 2). The strongest evidence for the therapeutic potential of this approach comes from the

efficacy of retinoic acid receptor a activation in the treatment of acute promyelocytic leukemia (3). Over 90% of patients with acute promyelocytic leukemia achieve complete remission following treatment with the naturally occurring retinoid all trans-retinoic acid (ATRA; ref. 4).

# **Glass Jug**

Luminarc® Quadro 1.7-Liter Glass Jug with Infuser

# Krups grinder

Krups 203-42 Grinder, Fast Touch

## Rosehip seed tea recipe





**Orange Juicer and Zester** 

**Metrokane juicer from Target** 

IQ citrus zester from williams and sonoma

Metrokane Mighty OJ - Orange/ Chrome



**Orange Juice Recipe C with Bioflavinoids** 

**Lemon Juice PH Bioflavinoid Drink** 

**Tomato Juice Recipe C** 



# Stinging nettle

<u>Stinging nettle root capsules 600mg Swanson sw968, 0-2.4G + TID</u> Stinging nettle robs fungus of hard outer shell - "chitinin"

http://www.swansonvitamins.com/swanson-premium-stinging-nettle-root-500-mg-100-caps

# **Danish RoseHips CAPSULES**

Danish RoseHips Swanson SWU424 https://www.swansonvitamins.com/swanson-ultra-pure-danish-rose-hips-750-mg-60-caps

## Choline

# (1) AM CHOLINE 293mg - CHOLINE Bitartrate Builds membranes back - Country Life Choline:

- Choline is an essential nutrient related to the water-soluble B-complex vitamins, folate, pyridoxine, and B12, and to the essential amino acid, methionine. It is synthesized in the body as well as consumed in the diet. The largest dietary source of choline is egg yolk. Choline can also be found in high amounts in liver, peanuts, fish, milk, brewer's yeast, wheat germ, soy beans, bottle gourd fruit, fenugreek leaves, shepherd's purse herb, Brazil nuts, dandelion flowers, poppy seeds, mung and other beans, and a variety of meats and vegetables, including cabbage and cauliflower.
- <u>Choline</u> is a major building block of <u>lecithin</u>. Choline is a precursor to
  acetylcholine, a chemical used to transfer nerve impulses. Therefore, choline is
  believed to have neurological effects.
- <u>Choline</u> is a constituent of <u>phosphatidylcholine (PC)</u>, which is a component of cell walls and membranes. It is involved in fat and cholesterol metabolism and transport. In this form, <u>choline</u> aids in fat metabolism and transport away from the liver.
- <u>Choline</u> is likely effective when used orally as a nutritional supplement in infant formula. Also, <u>choline</u> is likely effective when used intravenously to treat total parenteral nutrition associated liver dysfunction.
- The liver uses **choline** to break down cholesterols in order to create hormones and the like. In a body that is too acidic (precisely the environment where candida thrives), cholesterols get oxidized and there you get the beginning of bile stones.

<u>Phosphatidylcholine</u> (supplemental) has been quoted as helping a great deal in liver flushing, together with ox bile (bile is primarily cholieric materials).

#### **Bromelain**

Fresh raw pineapple, pineapple enzyme **Bromelain** helps clear and counteract parasite enzyme changes in the gut **Bromelain** is a pineapple extract thought to be effective for reducing swelling (inflammation), especially of the nose and sinuses, after surgery or injury. It may also be used for a variety of other effects that remain scientifically unconfirmed and not authorized by regulatory authorities like the Food and Drug Administration. These may include: hay fever, treating a bowel condition that includes swelling and ulcers ulcerative colitis, removing dead and damaged tissue after a burn debridement, preventing the collection of water in the lung pulmonary edema, relaxing muscles, stimulating muscle contractions, slowing clotting, improving the absorption of antibiotics, preventing cancer, shortening labor, and helping the body get rid of fat. **Bromelain** also contains chemicals that might interfere with the growth of tumor cells and slow blood clotting, according to laboratory research only.[1]

"Bromelain" is also used as a culinary ingredient, primarily as a tenderizer. The term "bromelain" can refer to either of two protease enzymes extracted from the plant family Bromeliaceae, or it can refer to a combination of those enzymes along with other compounds produced in an extract.

<u>Bromelain</u> extract is a mixture of protein-digesting enzymes—called proteolytic enzymes or proteases and several other substances in smaller quantities. The proteolytic enzymes are

referred to as sulfhydryl proteases, since a free sulfhydryl group of a cysteine side-chain is required for function.

## The two main enzymes are:

Stem bromelain - EC 3.4.22.32 Fruit bromelain - EC 3.4.22.33

The other substances typically include peroxidase, acid phosphatase, protease inhibitors, and calcium

### **History**

The first isolation of bromelain was recorded by the Venezuelan chemist Vicente Marcano[2] (BU1 1.Phar. 5,77) in 1891 from the fruit of pineapple. In 1892, Chittenden, assisted by Joslin and Meara, investigated the matter fully (Trans. Conn. Acad. Arts Sci. 8, 281-308), and called it 'bromelin'. Later, the term 'bromelain' was introduced and originally applied to any protease from any plant member of the plant family Bromeliaceae.

Bromelain was first introduced as a therapeutic supplement in 1957. Research on bromelain was first conducted in Hawaii but more recently has been conducted in countries in Asia, Europe, and Latin America. Germany has recently taken a great interest in bromelain research; bromelain is currently the thirteenth-most-widely-used herbal medicine in Germany.

### **Source**

Bromelain is present in all parts of the pineapple plant (Ananas sp.).[citation needed] However, the stem is the most common commercial source,[citation needed] presumably because it is readily available after the fruit has been harvested. Pineapples have had a long tradition as a medicinal plant among the natives of South and Central America.

- 2 <u>Bromelain</u>/4800 mg GDU with water on empty stomach (anti-inflammatory, reduces tumor invasion/migration, boosts immunity, blocks the production of PGE2, reduces radiation side effects)
- The maximum recommended dosage of **Bromelain** is 1000mg 3x daily.
- Jarrow Fibrinogen (Blood Clots) Support -- **Bromelain** Enzymes
- Helps improve removal of waste from body
- · Dissolve chitin and moves the shit out

Bromelain enzyme supplementation has also been shown to be effective for sport injuries and traumatic injuries. It helps to break down the clots that form as a result of the trauma. Here is how it works. The Bromelain enzymes tend to promote the synthesis of anti-inflammatory hormones such as the prostatagland series one. As you promote the prostatagland in series one, you tend to have an anti-inflammatory effect. It also inhibits the release of inflammatory kinnins that are part of the inflammatory process in most inflammatory conditions. It then helps to break down abnormal clots from traumatic injuries, sport injuries, car accidents as well as other types of trauma that we might incur. Bromelain does this by encouraging activation of what is called plasmin, found in the blood vessel wall, which helps to break the clot down.

As you can see, **Bromelain** enzymes can be very helpful at reducing inflammation and helping patient's speed up the recovery of traumatic injuries. However, you have to know

how much to take. Also, there are some very important drug nutrient interactions to know about with respect to using **Bromelain** so that it does not become a dangerous supplement for you.

Drink homemade pineapple fruit juice recipes. The importance here is that the pineapple juice is taken fresh before its natural enzymes die off. One such enzyme is called **Bromelain** and is believed to kill off tapeworms. Only use organic pineapples as others have almost zero **Bromelain** content.

## **Probiotic 225**

#### **Probiotic 225 Brand: Ortho Molecular Products**

Item Number: OM1219

http://www.iherb.com/ortho-molecular-products-probiotic-225-gastrointestinal-support-15-packets-3-q-each/45207

https://www.pureformulas.com/probiotic-225-15-sachets-by-ortho-molecular-products.html

MFG # 470015 UPC # 615033004705

Probiotic 225 is a high potency formula delivering substantial amounts of beneficial bacteria to support gastrointestinal health and proper micro flora balance. Disruption of normal flora, called dysbiosis, by reducing beneficial bacteria colonies and populations, allows space for the yeast colonies to grow. Normally, beneficial bacteria colonies cover the intestinal surface, crowding out disease-producing organisms like yeast. In this way, yeast though present, are kept to a minimum.

## **Select Organisms**

Ortho Molecular (Gut Repair Kit) Potential Game Changer Anyone who has used VSL#3 or Mutaflor has spent enough on Probiotic to make a car payment. It's not just the cost of Clinical grade Probiotic; but, the price of all the testing, Upper GI, Stool samples, Bla, Bla

Probiotic 225 contains a combination of six unique strains of live organisms, scientifically selected for their superior resilience, endurance and effectiveness. Mix with yogurt

## **Activia Yogurt by Dannon**

Active strain of Bifidobacterium lactis DN-173 010. Researchers assembled a study group of people diagnosed with active IBS. Reporting its findings in the January 2009 issue of "Alimentary Pharmacology and Therapeutics," the team said test subjects who were regularly fed yogurt containing the probiotic over the course of four weeks showed a significant reduction in bloating and stomach distension.

## **Kidney Flushing**

## **KGP Flush**

#### **KGP Flush**

https://www.baselinenutritionals.com/products/kidney-liver-detox-package.php
https://www.baselinenutritionals.com/products/KGP-Flush.php
PM KGP Flush For Optimum Kidney Health Baseline Nutritionals
Certified medicine (NDC 75830-002-04) to relieve kidney stones and gallstones. \$49.95
https://www.baselinenutritionals.com/products/KGP-Flush.php
KGP Flush<sup>TM</sup> 4 fl oz \$49.95

Hydrangea arborescens, Eupatorium purpureum - gravelroot , Althea officinalis - marshmellow, Black Cherry Concentrate, Solidago virgaurea- goldenrod, Zingiber officinale rosc – Ginger, B6, Magnesium oxide, Uva ursi- bearberry, Parsley – Dr Clark

### **Enzymes**

Enzyme Supplements did little for me. Serrapeptase helped circulation ache, Bromelain (pineapple and pineapple juice) helped digestion. When you have a paraste infection you essentially have no enzymes, and almost no amount of supplementation helped me, excepting the two above. I looked elsewhere for feeling and getting better.

### Serrapeptase

https://www.herbspro.com/default.asp https://www.herbspro.com customerservice@herbspro.com 33453 Western Ave. Union City, CA-94587. Order Date: 9/30/2011 6:07:00 AM

Item: Best Serrapeptase 40,000 units

Item no: 69142 UPC: 753950001497 type: 90vc quan 2 \$12.71 \$25.42

### Dosage:

Directions: Take 1 to 2 capsules twice a day bu itself. Take on an empty stomach. Take capsules at least 30 to 60 minutes before or 2 hours after a meal with 8 oz. of water.

Do not take during a time where you take other vitamins.

If you are taking any medication or under medical supervision, consult your doctor before use. Discontinue use and consult your doctor if any adverse reactions occur.

Store in cool, dry place with the lid tightly closed. Avoid excessive heat. Keep out of the reach of children. Not intended for use by persons under the age of 18.

### **Supplement enzymes**

#### CoFactors:

Papain, Bromelain, Amylase, Lipase, Rutin and Amla.

Elimination of potentially toxic substances from the body is a crucial part in the metabolic processes for optimal health. A variety of health issues can develop without a proper store of enzymes or if toxins within the body impair enzyme function or availability.

When sulfur and vitamin A are combined in the body with all the other required nutrients including minerals (like zinc and silica) and amino acids (including lysine and proline), the body <u>immediately</u> starts producing enzymes.

Zinc is required to produce most collagen and keratin enzymes, which in turn initiate the chemical reaction that produces keratins (combining the nutrients chemically to form the many unique types of this protein) and deposits them where they are needed in the body – this is simply how DNA works! In fact, thousands of the genes in human DNA are identified as being keratin and collagen genes. The various types and amounts of keratins produced depend on the types and amounts of amino acids and minerals present and how DNA naturally "directs" the production of these proteins. The production of collagens works the same way except that it is sulfur and vitamin C that are combined with the other nutrients.

### **Enteric Coated Serrapeptase**

Used by silkworms to digest and emerge from their silken cocoons; studied extensively for more than 50 years; an excellent fibrinolytic (fibrin digesting) and powerful anti-inflammatory enzyme\*

#### **Protease**

Hydrolyze (break-down) proteins like casein, steak, gelatin, soy, fish and other plant and animal proteins to smaller chains of polypeptides (small proteins) and amino acids for easier uptake throughout the body

#### **Bromelain\***

A collective name for the proteolytic (protein-digesting) enzymes found in the pineapple plant (Ananas comosus); known for its anti-inflammatory properties. A good substitute for animal-based enzymes a good substitute for pepsin and trypsin for digestive support\*

#### Papain\*

Obtained from the latex of the fruit of the Papaya tree; used for centuries as an effective digestive aid\*

Papain; Papain is a protein digestive enzyme that really works synergistically with herbs to further break down material. This Digestive enzyme will help restore your intestinal tract to its normal state, which makes it inhospitable to parasites. Papain taken 30 minutes before or after meals helps kill worms.

#### **Amylase**

Breaks down carbohydrates, or more specifically, starches, into smaller dextrins and sugars; produced naturally by humans, microorganisms and plants

### Lipase

Catalyzes the break-down of fats into essential fatty acids needed for healthy tissues and cells\*

#### **Amla**

Also known as Indian gooseberry (Emblica officinalis); a natural, efficacious antioxidant, and one of the richest sources of absorbable vitamin C

#### **Rutin**

Bioflavonoid; strengthens and tones arteries and veins; provides antioxidant support against free-radicals and inflammation\*

### Serrapeptase actions

Serrapeptase is a proteolytic enzyme isolated from the non-pathogenic bacteria Serratia species found in the digestive tract of the Japanese silkworm. The enzyme is used by the worms to digest their cocoons. Serrapeptase has been used as a nutritional supplement in Europe and Asia for nearly three decades. Each vegetarian capsule of Best Serrapeptase contains enteric-coated pure serrapeptase designed for optimal absorption in the intestinal tract.

\* Dietary supplement. Serrapeptase has been used widely in clinical practice in Japan and Europe for decades. Dr. Hans Nieper, a legendary medical doctor known for his extensive use of proteolytic enzymes, named it the Miracle Enzyme because of its unique abilities. Serrapeptase is made by the bacteria Serratia E15 found in the digestive tract of silkworms, which harness the serrapeptase enzyme to break down food and the walls of their silk cocoons as they emerge in their moth state.

Serrapeptase is a powerful systemic enzyme that supports the body's response to inflammation and the respiratory and sinus systems. The bacteria Serratia mercesans produces Serrapeptase, an enzyme that enables the silkworm to dissolve its silken cocoon and emerge after metamorphosis. Serratia is now grown in cultures to produce serrapeptase by fermentation. Numerous research and clinical studies have demonstrated serrapeptase has anti-inflammatory, proteolytic (protein dissolving) and fibrinolytic (fibrin dissolving) properties. Studies also show serrapeptase promotes respiratory and sinus health, improves tissue healing and significantly reduces inflammation.

<u>Serrapeptase</u> also has the unique ability to digest non-living tissue that is a by-product of the healing response without harming living tissue. Fibrin is a tough protein arranged in long fibrous chains. It is formed from fibrinogen, a soluble protein produced by the liver and

found in blood plasma. Fibrin tends to form circulating complexes that create barriers against the absorption of healing nutrients in and around areas of inflammation.

Serrapeptase supports normal fibrin metabolism, blood viscosity and blood flow. In addition, serrapeptase supports healthy inflammatory response by reducing metabolic inflammation, usually an asymptomatic inflammatory process in response to stress, improper nutrition and other environmental issues. There is also evidence of inhibition of C-Reactive Protein, a marker of inflammation that has been linked to cardiovascular health.\*

Many people reach for NSAIDS (non-steroid anti-inflammation drugs) such as aspirin, ibuprofen and naproxen to address pain and inflammation. They are effective but come with serious side effects such as gastric bleeding, joint/cartilage damage, disrupted metabolism, increased blood sugar, loss of bone and emotional disorders, among others. Serrapeptase can significantly reduce inflammation when taken alone and provide a synergistic affect when taken in low doses with aspirin.

For instance, the immune system can be overwhelmed with bacteria, fungi, toxins and even food particles that doesn't get completely digested in the gastro-intestinal tract. In many instances, proteins the body detects as foreign initiate an immune response. Recognized as antigens, the immune system produces antibodies to start the process of their degradation and removal. After coupling with one another, these antigens and antibodies form circulating immune complexes (CICs), which induce attack and destruction by the immune system's macrophages (cells specifically designed to destroy CICs). In cases where inflammation is prolonged or when CICs are predominant within the body's tissues and organs, the body may not have enough enzyme potential to begin the next process of renewal and recovery. Moreover, research has demonstrated that a lack of proteolytic enzymes may not adequately address a large accumulation of CIC's in the body.

To address this problem, Exclzyme has been formulated with an ample supply of various proteases to facilitate the body's immune defenses and breakdown free-roaming CICs which can keep the body in a constant state of inflammation. Exclzyme also contains **papain** and bromelain, enzymes known for their potent systemic properties. Enteric-coated serrapeptase highlights this powerful mix of proteolytic enzymes with its scientifically proven fibrinolytic power.

In addition to these systemic and digestive proteases, lipase and amylase also offer their own enzyme activity to bolster the body with extra catalytic energy, especially for the complete digestion of fats, sugar and protein complexes. And for extra synergy, Exclzyme is rounded with anti-oxidant support in the form of rutin and amla, herbs well known for their unique bioflavanoid content, especially useful for tissue rejuvenation and recovery.

### **Systemic Enzyme Therapy for Men and Women**

For nearly 40 years, serrapeptasehas been at the heart of systemic enzyme therapy. Systemic enzymes such as serrapeptase have been used for optimal cell, tissue, organ and metabolic function. Likewise, companies, health practitioners and consumers have turned to the original Exclzyme formulation for their systemic enzyme therapy needs.

The reality is that inflammation affects everyone, men and women alike. However, along with nearly all other tissue and organs, inflammation can affect reproductive tissue and organs particularly. For instance, many women experience painful swelling of the breast as

well as uterine fibroids. In men, prostate health is a major concern. Although inflammation in each case may manifest differently in both men and women, inflammation has one thing in common: fibrin.

Fibrin is an essential protein that is produced during inflammation. As a blood clotting protein, the body utilizes fibrin to contain blood loss and keep infection at bay after injury. It is the initial first step in the body's attempts at recovery. However, when inflammation has become prolonged, fibrin along with other proteins such as collagen can begin to transform original tissue into a tough fibrous matrix. Differentiated and sometimes sequestered apart from healthy tissue, this fibrous matrix still has the biological markers of inflammation. Nutrients and other building blocks are still needed for healthy and normal tissue.

Systemic and proteolytic enzyme research over the years suggests that fibroids in women and swollen prostrate glands in men are indications that the body has not been able to get past inflammation. The resulting fibrous tissue usually accumulates toxins that have not been adequately removed by the body.

Enzymes are energetic catalysts that push essential chemical reactions in the body in the right direction. In its quest for internal balance, the body always turns to enzymes for help. In this way, Exclzyme helps get inflamed tissue over the hump.

Exclzyme's superior proteolytic action assists the body in removing those larger protein molecules from damaged cells and tissues as well as the inflammatory response itself. In helping breakdown these compounds, the impediments for sustaining healthy cell and tissue growth are removed and the processes for rejuvenation and recovery can move forward. Exclzyme is the #1, non-animal derived systemic formulation on the market today. Why not use it as a part of your continual systemic enzyme therapy?

#### **USES:**

Researchers in India conducted a study to assess the response of serrapeptase in patients with carpal tunnel syndrome (CTS). They wanted to determine if a conservative, non-surgical approach would be beneficial. Twenty patients with CTS were evaluated clinically after 6 weeks taking serrapeptase. Sixty five percent showed significant clinical improvement, which was supported by improvement in electrophysiological parameters. No significant side effects were observed. The doctors concluded that serrapeptase therapy may prove to be a useful alternative conservative treatment.3

Another study was conducted comparing the efficacy of two proteolytic enzymes in the treatment of venous inflammatory disease. The efficacy of Serrapeptase and Seaprose S was assessed using good or excellent results as the measure of effective treatment. Serrapeptase was effective in 65% of the cases compared to 85% for Seaprose S. Though Seaprose S had better overall results, both enzymes were effective. It can thus be confirmed that both enzymes were effective in patients with inflammatory venous disease.4 (Note: Seaprose S has since been withdrawn from the market).

A clinical evaluation of serrapeptase was conducted to determine its efficacy in reducing inflammation in patients with breast engorgement. Serrapeptase was noted to be superior to placebo for improvement of breast pain, breast swelling and induration and while 85.7% of the patients receiving serrapeptase had "Moderate to Marked" improvement. No adverse reactions were reported with the use of serrapeptase. The researchers conclude that serrapeptase is a safe and effective method for the treatment of breast engorgement.5

A prospective study was conducted on the effect of serrapeptase on post-operative swelling and pain of the ankle. In the serrapeptase group, the swelling decreased by 50% on the third post-operative day, while in the control groups (no treatment and treatment with ice) no reduction in swelling occurred. A decrease in pain correlated for the most part with the reduction in swelling. On the basis of these results, serrapeptase would appear to be an effective preparation for the post-operative reduction of swelling, in comparison with the classical conservative measures, for example, the application of ice.6

Moving to another part of the body, a study was conducted in men with amicrobial prostatovesiculitis (APV is a non-infectious inflammation of the prostate). The researchers wanted to determine if treatment with nonsteroidal anti-inflammatory (NSAIDS) drugs, including serrapeptase, could reduce inflammation and swelling of the prostate. The doctors conclude that in APV patients, the treatment with NSAIDS, including serrapeptase is an effective therapy, producing multiple positive effects.7

The efficacy of serrapeptase was evaluated in a multicentre, double-blind, placebo-controlled study of 193 subjects suffering from acute or chronic ear, nose or throat disorders. After 3-4 days' treatment, significant symptom regression was observed in serrapeptase treated patients. Statistical comparison confirmed the greater efficacy of serrapeptase against all the symptoms examined. It was concluded that serrapeptase has anti-inflammatory, anti-edemic and fibrinolytic activity and acts rapidly on localized inflammation.8

A multi-centre, double-blind, placebo-controlled trial was carried out to investigate the clinical efficacy of the anti-inflammatory enzyme Serratiopeptidase in 174 patients who underwent Caldwell-Luc antrotomy for chronic empyema(puncturing and draining pus from the nasal maxillary sinus). This puncture is placed under the top lip and above the gum line. Changes in swelling at the puncture sites after the procedure were observed. The degree of swelling in the Serratiopeptidase-treated patients was significantly less than that in the placebo-treated patients at every point of observation. No side-effects were reported.9

### **Other Applications**

Serrapeptase is widely used in clinical practice in Japan. One research trial in Japan investigated the effect of serrapeptase on sputum properties and symptoms in patients with chronic airway diseases. After 4 weeks of serrapeptase treatment, sputum output, viscosity and sputum neutrophil count decreased significantly. In addition, the frequency of coughing and of expectoration also decreased. The researchers concluded serrapeptase may exert a beneficial effect on mucus clearance by reducing neutrophil numbers and altering the viscoelasticity of sputum in patients with chronic airway diseases.10

Another clinical study evaluated the effect of serrapeptase on the elasticity and viscosity of the nasal mucus in adult patients with chronic sinusitis. Serrapeptase was administered orally for 4 weeks. The dynamic viscosity of the mucus at week 4 was significantly lower than that at week 0. The authors conclude that serrapeptase may have some application in patients with chronic sinusitis.11

An unusual clinical trial evaluated the effectiveness of serratiopeptidase in the eradication of a periprosthetic infection (an infection at the site of an implanted orthopedic device) in an in vivo animal model. Infections of slime-forming bacteria are especially difficult at these sites. Rats were inoculated with Staphylococcus epidermidis at the prosthetic site. After two weeks, infection persisted in 63.2% of animals in the no-treatmentgroup; 37.5% of

animalsin an antibiotic-only group; and only 5.6% of animals in the serratiopeptidase-and-antibioticgroup. The authors conclude that serratiopeptidase was effective at eradicating infection this experimental animal model and may enhance antibioticefficacy in the treatment of staphylococcal infections.12

### **Food**

### **Grandparents**

My grandpa Welker was a farmer, in eastern Ohio. My family went out almost every summer to visit my mom's parents and then my dad's parents. Grandma Welker was a little deaf, and so was grandpa. They used to holler at each other in different rooms, each one not hearing the other. It made us kids laugh when they hollered at each other. It was even funnier when they swore in Dutch under their breath. Grandpa used to take me through the fields, show me the apple trees he had, he would pick one, cut it up with his pocketknife, and give me a slice. We went out in the woods to pull down trees, cut wood, or just generally take care of the land. I loved driving that old Farmall tractor. At night, we would sit out on the patio and eat watermelon, spitting the seeds on the ground.

A couple of summers grandpa and grandma drove out from Ohio to where we lived in New Jersey, in an old 1960's Chevy. It was loaded stem to stern and up to the ceiling with wooden bushel baskets of fresh produce, grown on the Welker farm in Ohio. We would cut, and peel, can and jar, tomatoes, corn, beans, peas, carrots, and all sorts of vegetables. The process would go on for days.

On the summers we kids spent in Ohio we got to experience the farm. Grandpa had retired years ago but he still helped run the hardware store, made furniture and picture frames, sold cider and apples on the road, and had a 10-acre garden. In the morning the breakfast table was a full out chow down spread. Grandma would put on the **coffee** and start setting the breakfast table. There was always grapefruit sliced and sectioned, and cantaloupe (orange or green honey dew) sliced into sections, with a bowl of fruit, homemade bread, browned toast, sliced tomatoes, OJ, cereals of every type, wheat germ, homemade apple sauce with cinnamon on top, homemade apple butter jelly, butter, cottage cheese, milk, fresh honey, sugar, salt, pepper and what-not. The whatnot could be almost anything. I was always amazed to sit down to my grandparent's breakfast table, looking at all the stuff put out on the table. It was very unusual, I thought, I had never seen such a breakfast table spread like this, and to this day I have not seen one quite like it.

As kids we would sit down at the table and look out at the table in disbelief. I got used to eating grapefruit and cantaloupe. I guess I rotated cereals, as I never had Cheerios, Wheaties, or Shredded Wheat at home. I was a Captain Crunch guy. There was always a box of Total on the table, but I never tried that one. I learned to enjoy sugar on my grapefruit; it was too strong a taste to take without sugar. Fill your tank, Grandpa would say at the table, your tank is only half-full, Slop Nye ... he would say, (an old Pennsylvania Dutch saying I guess. I decided to look up the translation).

Slop (n.)

**Dutch Translation:** 

Food, nutriment, eat, feed, expression, formulation, sentimentalism, overemotional, eat up, nutrient, nourishment; sustenance; aliment; fare; eats; food; solid food; Making up; preparation; the art of cooking;

Alimentation: The act or process of giving or receiving nourishment. Support; sustenance.

Webster's:

Nye (n) a brood or flock of pheasants

The workday started at nine AM. After grinding up apples or corn meal, and a few trips out to the barn or the fields working on this or that, Grandpa would talk while he worked about his life, the jobs he had, the war, about the depression, and how little food there was. He told us of the places he lived, and where he had traveled, and how he came to buy and then build the farm during the depression so he could provide the family with plenty of food. Grandpa loved to eat. It was good he bought a farm...I thought to myself... as he loved to eat! He told the same stories many times, including his first cow that he bought for the farm had no teeth. I always showed respect and looked like I was listening even if I was not. I wish now I had listened more.

Coming in from the fields for lunch was also a special treat. Grandma would start setting the lunch table about 11:30. There was always something hot, and something cold. Grandma would cook ham, beef, or homemade vegetable beef soup. There was always a plate of thick sliced baloney, beef salami, and cheese. There was always a plate of sliced lettuce, tomatoes, and onions. There were jars of homemade butter pickles, homemade red beets, and of course out came the breads, rye, wheat, and white. Bowls were filled with applesauce, and cottage cheese. There was a pitcher of milk and cans of soda. Grandpa would say that you are what you eat, and you should eat all kinds of food, a little bit of everything to be sure. This was a dining room table that proudly displayed Pennsylvania Dutch cooking. Grandma would make buttermilk lettuce in a pan as I watched in fascination. Her scratch gravy and sauces were masterful. The dining room was a place for conversation and good food. Grandpa's philosophy: there are good habits and bad habits; we get to choose which habits we are stuck with through life. Eat a bit of everything; it is all good for you. Keep it moving, (keep the bowels moving). Work hard, eat well. Grandpa's home spun philosophy of life. Both my grandparents lived well into their late 80's and I was sorry to lose them.

The Dinner setting – or was it called supper?, anyway there would be a chicken or pot-roast, at least three cooked vegetables, a full chopped salad, and you guessed it, a ton of stuff from breakfast and lunch to clean-up. Along with the applesauce, cottage cheese, a pitcher of ice water, and **coffee** again. Let's not discuss dessert, the endless rotation of homemade pies (apple, rhubarb, peach, blueberry, lemon, or mince), cakes, and sweet tarts. I have never been at a dinner table that served so much good food. In total there were 6 of us at the table, a table that could serve many more. On occasion, I remember two or three joining us for dinner, and there was always plenty of food. Grandma cooked for an army from habit. When the farm was in business, the farm help and field workers would have always come in for a meal, and Grandma was always prepared.

### **Micronutrients by Food Groups**

#### Dairy:

- 1. The primary source of calcium in your diet.
- 2. Heavy in saturated fat
- 3. Calcium, potassium, phosphorus, magnesium, and iodine [10]
- 4. Protein [13]

Eggs Yogurt

Kefir



- What is Kefir?
- · How to make Kefir
- How to Second Ferment Kefir
- How to Make Kefir Cheese and Whey

### **Fatty Fish:**

- 1. Omega-3 fatty acids (eicosapentaenoic acid [**EPA**] & docosahexaenoic acid [**DHA**]) [12]
- 2. Protein [13]
- 3. **Iron**

#### Fruits:

- 1. Because fruits are normally eaten raw, they are the primary source of many water-soluble / living nutrients your diet.
- 2. Soluble dietary fiber (such as pectin) [6]
- 3. Folic acid
- 4. 57.9% of the total vitamin C intake [7]
- 5. 40.6% of the total beta-carotene intake [7]

#### 6.Plant families of Fruits:

- A.Citrus Fruits (orange, tangelos, tangerine)
- •Monoterpenes (inhibits carcinogen activation) [18]
- Vitamin C
- B.Carotene rich fruits (raw tomatoes, cantaloupe, mango, papaya, dried apricots & peaches)

### **Whole-Grains / Cereal:**

- 1. Bran, which may contain the most important bioactive nutrients.[20]
- 2. Insoluble fiber (such as cellulose) [6]
- 3. Sodium, iron, magnesium, manganese, and iodine [9],[10]
- 4. "The long list of cereal antioxidants includes **vitamin E**, tocotrieonols, selenium, phenolic acids, and phytic acid. These multifunctional antioxidants come in immediate-release to slow-release forms and thus are available throughout the gastrointestinal tract over a long period after being consumed."[20]
- 5. B vitamins, **vitamin E**, selenium, zinc, copper, and magnesium.[8]
- 6. Protein [13]
- 7. Lignans [20]
- 8. See Health Benefits of Whole-Grains for additional information.

Flax Seeds

### **Legumes:**

- Soluble dietary fiber (such as pectin) [6]
- Insoluble fiber (such as cellulose)
- Protein [13]
- 4. "Legumes are rich sources of slowly digestible starch promoting moderate postprandial glycernic and insulinemic responses." [14]

Limas

Green Beans

#### Meat:

- 1. Saturated fat
- 2. The primary source of iron, zinc, and vitamin B-12 in your diet. [11]
- 3. Protein [13]
- 4. Copper, phosphorus, selenium, and zinc [9]

Grass Fed Beef Salmon

#### **Nuts:**

- 1. Low in saturated fatty acids and high in monounsaturated and polyunsaturated fatty acids [16]
- 2. Protein, copper, magnesium [16]
- 3. Best natural source of **vitamin E** and are relatively concentrated repositories of magnesium, potassium, and arginine [17]
- 4. Protein [13]

**Almonds** 

### **Vegetables:**

- 1. Vegetables are normally eaten cooked in order to break down fiber and to destroy toxins. Thus, they are the primary source of many nutrients that are improved by the cooking process.
- 2. Soluble dietary fiber (such as pectin) [6]
- 3. Insoluble fiber (such as cellulose)
- 4. Magnesium, potassium [9]
- 5. Folic acid
- 6. Greens (calcium and magnesium)
- 7. 34.8% of the total beta-carotene intake [7]
- 8. 17.3 % of the total vitamin C intake [7]

### 9. Plant families of Vegetables:

A.Brassica or Cruciferous vegetables



- (broccoli, cabbage, kale, Brussels sprouts, cauliflower)
- •Aromatic isothiocyanates (inhibits carcinogen activation) [18]
- •Glucobrassicin & indoles (inhibits carcinogen activation)[18]
- . Beta Carotene rich vegetables

([dark-green leafy vegetables (kale, collard greens, spinach, greens in general) and deep-yellow-orange vegetables (cooked tomatoes, carrots, sweet potato, pumpkin)

•Approximately 80-90% of the carotenoids present in green, leafy vegetables such as

broccoli, kale, spinach and brussel sprouts are xanthophylls, whereas 10-20% are carotenes. Conversely, yellow and orange vegetables including carrots, sweet potatoes and squash contain predominantly carotenes. [15]



C.Allium vegetables (onion, **garlic**, leek, chive).

•Organosulphur compounds (inhibits carcinogen activation ) [18]

Carrots

### **Special Nutrition Foods**

#### Spinach - Source of Iron

http://www.healthaliciousness.com/nutritionfacts/nutrition-comparison.php?o=11458&t=11458&h=11148&s=100&e=180.000&r=175.000



# #3: Dark Green Leafy Vegetables (Spinach, cooked)

Iron 100g	Per cup (180g)	Per 1/2 cup (90g)	
3.57mg (20% DV)	6.43mg (36% DV)	3.22mg (18% DV)	

#### Other Dark Green Leafy Vegetables High in Iron (%DV per cup, cooked):

Swiss Chard (22%), Spinach (20%), Beet Greens (15%), Scotch Kale (14%), Dandelion Greens (11%), Pak Choi (10%), and Kale (7%). Click to see complete

Bananas - Source of Potassium

Oranges – Source of Bioflavinoids

http://www.healthaliciousness.com/nutritionfacts/nutrition\_facts.php?id=Oranges%20Raw%20With%20Peel&idn=9205

#### Lemons - Source of Limonoids

http://www.healthaliciousness.com/nutritionfacts/nutrition\_facts.php?id=Lemons%20Raw%20With%20Peel&idn=9151

### Blueberries - Source of Lutin

http://www.healthaliciousness.com/nutritionfacts/nutrition\_facts.php?id=Blueberries %20Raw&idn=9050

Manganese

Olives

Green Onion

**Tomatoes** 

**Sweet Potatoes** 

**Bell Peppers** 

Swiss Chard

We've been reminding you about the importance of <u>vitamin K</u> lately. Swiss chard happens to be one of the best sources. This powerhouse green is chewy, substantial and richly flavored. You'll enjoy this pungent leaf's nutritional benefits, too: fiber, manganese, magnesium, vitamin A, vitamin C, iron, vitamin E, potassium, and plenty of other nutrients. It's one of the most comprehensive greens, in terms of vitamins and minerals, so eat it regularly.

Read more: http://www.marksdailyapple.com/power-foods/#ixzz3wDzE7uEl

Olive oil – the healthiest oil on earth, Scilian is world renown for its C22 C23 carbon chains.

### **Garlic**

<u>Garlic</u> provides nourishment for the circulatory, immune and urinary systems. It aids in supporting with normal circulation, nourishing stomach tissues, maintaining normal blood pressure and aids the body's natural ability to resist disease. <u>Garlic</u> is a natural antibiotic and fungicide.

#### Garlic:

**Garlic** is able to slow and kill over 60 types of fungus and 20 types of bacteria, as well as some of the most potent viruses. **Garlic** has a history of killing parasites and controlling secondary fungal infections, detoxifying while gently stimulating elimination, and has antioxidant properties to protect against oxidation caused by parasite toxins.

The active components in **garlic** that kill parasites are Allicin and Ajoene. These compounds can kill ameba's including one-cell varieties, as well as pinworms and hookworms. Allicin is not present in **garlic** in its natural state. When **garlic** is chopped or otherwise damaged, the enzyme alliinase acts on the chemical alliin converting it into allicin, the active component contributing for its success for killing parasites.

**Garlic**, Allium sativum: Poor Man's Treacle, Bawang, Bauang Monocot Perennial: Readily available and inexpensive **garlic** may be the first line of defense in treating high blood pressure, and dealing with common skin and fungal infections....

### Cayenne

Cayenne is a pepper well known for its benefits to the circulatory system. It aids the body to balance pressure levels and resist abnormal bleeding. Cayenne also nourishes the digestive system. This plant assists in the body's utilization of other herbs, when used in an herbal combination. When applied topically, it helps relieve minor discomfort.

### **Celery Seed**

Celery seeds contain vitamins A, C and B-complex. Use a Krups coffee grinder.

#### Cinnamon

Cinnamon has received much publicity since early 2004 about its effectiveness in reducing high cholesterol and triglycerides. Cinnamon bark or oil has been used to fight microorganisms, diarrhea and other gastro-intestinal disorders, and dysmennorhea.

### Cranberry

Cranberry contains a compound that prevents bacteria from adhering to the walls of the bladder and rest of the urinary tract. This prevents the bacteria from spreading and eventually results in the halt of infection. Using cranberry on a regular basis may help prevent the formation of kidney stones.

### **Red Raspberry**

Red Raspberry Seed (Ellagitannin): Raspberry seeds usually pass through our bodies, but when ground up, contain one of the most powerful antioxidants known- Ellagitannin (Ellagic acid). Use a Krups coffee grinder. Aside from being used very successfully in cancer treatment, Ellagitannin has also been found to be a powerful destroyer of parasites. It's a very strong anti-bacterial, anti-fungal, anti-viral that lowers cholesterol and protects our DNA. This is very expensive and hard to get but worth every molecule.

#### **Goats Rue**

Goats Rue; Goat's rue is a wild legume used during the Middle Ages to treat the plague. It was also used to induce sweating to break fevers and to treat infections with parasitic worms and snakebite. This herb can help balance blood <u>Sugar</u> levels, help women balance hormones and The plant has no odor unless a stem or leaf is bruised, causing the release of a stench, hence the name "goat's rue." Effective in both humans and animals alike.

### **Cruciferous Vegetables**

Research conducted by Dr. Jon Michnovicz of the Institute for Hormone Research and Dr. Leon Bradlow of the Strang Institute, suggests that increased consumption of cruciferous vegetables such as broccoli, cabbage and Brussels sprouts may reduce the risk of certain cancers, particularly breast cancer. Cruciferous vegetables contain indoles - a unique class of phytonutrients that have been scientifically shown to balance hormone levels, detoxify the intestines and liver and reinforce the body's immune system.

### Rosemary

#### Rosemary extract standardized for carnosic acid

Natural polyphenols such as carnosic acid found in rosemary have potent antioxidant activities. Unlike vitamin C or E, which have little impact on protecting against damage to DNA, rosemary extract and carnosic acid have been shown to offer significant protection against DNA damage. Rosemary extract also works synergistically with lycopene ( see below ) in protecting against free radical damage to LDL cholesterol. When lycopene is combined with rosemary extract there is significant protection against LDL damage.

Rosemary has been reported to decrease capillary permeability and fragility. Extracts have been used in insect repellents. The plant may have anticancer properties and has spasmolytic actions, liver and immune effects, and other various actions from asthma treatment to aromatherapy. It has antimicrobial actions against a variety of bacteria, fungi, mold, and viruses. (*Caution*: Taking large quantities of rosemary internally can result in stomach and intestinal irritation as well as kidney damage. Allergic contact dermatitis has been associated with the plant, but rosemary is not generally considered to be a human skin sensitizer. Rosemary's constituents, monoterpene ketones, are convulsants, and have caused seizures in large doses. Rosemary is also an abortifacient.)

**Rosemary** (*Rosmarinus officinalis*) is a woody, <u>perennial herb</u> with fragrant evergreen needle-like <u>leaves</u>. It is native to the <u>Mediterranean region</u>. It is a member of the mint family <u>Lamiaceae</u>, which also includes many other herbs.

Rosemary contains a number of biologiccompounds, including antioxidants such as carnosic acid and <u>rosmarinic acid</u>. Other bioactive compounds include <u>camphor</u> (up to 20% in dry rosemary leaves), <u>caffeic acid</u>, <u>ursolic acid</u>, <u>betulinic acid</u>, <u>rosmaridiphenol</u>, and <u>rosmanol</u>.

Natural polyphenols such as carnosic acid found in rosemary have potent antioxidant activities. Unlike vitamin C or E, which have little impact on protecting against damage to DNA, rosemary extract and carnosic acid have been shown to offer significant protection against DNA damage.

**Rosemary has a very old reputation for improving** memory, and has been used as a symbol for remembrance (during weddings, war commemorations and funerals) in Europe and Australia

The results of a study suggest that <u>carnosic acid</u>, found in rosemary, may shield the brain from free radicals, lowering the risk of strokes and neurodegenerative diseases like <u>Alzheimer's</u> and <u>Lou Gehrig</u>'s. [9]

Carnosic acid is a phytochemical that occurs naturally in rosemary, According to studies conducted by U.S. and Japanese medical researchers, carnosic acid has powerful antioxidant properties that protect the brain from free radical damage. Carnosic acid seems to assist in preventing damage from neurodegenerative conditions and can also help to boost brain functioning by increasing blood circulation in the brain. Naturally-occurring phenolic compound with antioxidant properties. Inhibits lipid peroxidation induced by NADH or NADPH oxidation. Isolated from Rosmarinus officinalis.

### **Thyme**

Thyme has been used internally for respiratory and digestive infections. It has also been used as a gargle in laryngitis and tonsillitis - to soothe sore throats and irritable coughs. As a cough remedy, it assists in producing expectoration and reducing unnecessary spasm. It may be helpful in bronchitis, whooping cough and asthma.

### Small Red potatoes Mashed With B bio Recipe DNA

Garlic Mashed Red potatoes optional whole food Mashed with Skin
Bio available B and C
Boil for till hard soft
Salt kosher
Boil with herb like thyme if you like
Boil with Sicilian olive oil
Drain
Salt - kosher sea salt
Pepper Cracked
Paprika Lite

Chopped raw rosemary
Land of lakes butter – ¼ stick
½ Cup ½ and ½ Cream Land of Lakes
Fresh Basil Leaf (optional)
Vermont Maple syrup (optional)
Roasted Garlic (optional)
Upper State NY. Or Wisconson Chedder Cheese Orange-Yellow)

Mash Rough With skin and butter salt. Wait cream mash to smooth, chicken Broth, or with some more butter, or olive oil Sicily, or roasted **garlic**, with raw herb, dr Mash Rough With skin and butter salt. Wait cream mash to smooth, chicken Broth, or with some more butter, or olive oil Sicily, or roasted **garlic**, with raw herb, dry Sherry(optional with **garlic** or olive oil only) Fresh basil, red onion, **Garlic** shop fine, Vermont Maple syrup complex carbon sugars are ok.

M Recipe C @ copyright

### **4 Ounce Meat Dinner**

4 oz meat grilled chicken or steak Baked patoe with chives, butter, sour cream, cheddar, scallions Raw carrot chopped Fresh pressed <u>Garlic</u> veggies dip Glass <u>cranberry</u>, or oj

### alkaline-forming foods

### **Habit foods:**

Lemons, watermelon, lentils, blackberries, <u>raspberry</u>, pineapple, strawberries, limes, <u>grapefruit</u>, mangoes, asparagus, onions, vegetable juices (excl tomato), broccoli, garlic, grapes, berries, apples, pears, almonds, radishes, yams, endives, beetroots, celery, lettuce, organic carrots, ginger, cantaloupe melon, bean sprouts, almonds, cashews, chestnuts, millet, quinoa, goats milk, apple cider vinegar, bananas, oranges, fresh ginger root, watermelon, avocados, mandarin oranges, tangerines, horse-radish root, pumpkin seeds, sunflower seeds, sweet potatoes, kiwi fruit, oats, wild rice, chestnuts, natural olives, green leafy vegetables, sea vegetables, sea salt, natural still mineral water, umeboshi vinegar.

# **Antiparasitics**

**DEC**=Dimmitrol for dogs \$13.00/100 **Piperazine** = Liquid dog dewormer 250mg/teaspoon **Piperazine** Citrate PPZ 4 oz \$4.00 ea IVM = Durvet 1.87% horse paste tube Invermectin \$3.00 ea Albendazole = fish tablet 200mg 100ct. ALB \$35.00

Praziquantel = powdered fish Praziquantel 50 Grams \$55.00

Fenbendazole = powdered fish 250 mg x 3 for \$5.95

Alinia = pharma

#### **PinX**

### **PAMOATE**

# AM 4 TEA Pyrantel PAMOATE 200LBS + Reeses Pinworm Medicine 1-Oz., NDC 10956-618 01

WITH some FOOD

If vomit, take DROPS Pyrantel PAMOATE and work up for 14 days, then try a standard box label dose.

Dose every 3 weeks, until GI is clear for 2 or 3 more 3week doses. Parasites could come back at 6 weeks, or 9 weeks later.

It is intended that once the GI tract is clear, anti-parasitics will maintain and keep the GI tract clear, with no further need for PinX. if GI tract parasites are not gone after 3 weeks,

o Take PinX at 5am 5 hours minimum before L Cysteine dose The natural alternative is ozonated olive oil.

### State of the art

Anthelmintics are drugs that are used to treat infections with parasitic worms. This includes both flat worms, e.g., flukes and tapeworms and round worms, i.e., nematodes. They are of huge importance for human tropical medicine and for veterinary medicine. The World Health Organization estimates that a staggering 2 billion people harbour parasitic worm infections

#### http://www.who.int/wormcontrol/statistics/

Parasitic worms also infect livestock and crops, affecting food production with a resultant economic impact. Also of importance is the infection of domestic pets. Indeed, the companion animal market is a major economic consideration for animal health companies undertaking drug discovery programmes. Despite the prevalence of parasitic worms, anthelmintic drug discovery is the poor relation of the pharmaceutical industry. The simple reason is that the nations which suffer most from these tropical diseases have little money to invest in drug discovery or therapy. It comes as no surprise therefore that the drugs available for human treatment were first developed as veterinary medicines. There is thus a pitifully small repertoire of chemotherapeutic agents available for treatment (see Table 1). In some respects, this situation has been exacerbated by the remarkable success of **ivermectin** over the last twenty years (Geary, 2005), which has decreased motivation for anthelmintic drug discovery programmes (Geary, Sangster and Thompson, 1999). This prompts concern, as anthelmintic resistance has been widely reported in livestock and it may also only be a matter of time before this phenomenon occurs in parasites of humans.

Table 1. Key drugs registered for the treatment of parasitic worms in humans.

Schistosomiasis (blood fluke)	Intestinal round worms	
Antimonials	Piperazine	
Metrifonate	Benzimidazoles	
Oxamnaquine	Morantel	
Praziquantel	Pyrantel	
	Levamisole	
Cestodiasis (tape worm)	Avermectins and milbemycins	
Niclosamide	Closantel (and halogenated salicylamides)	
Benzimidazoles	Emodepside	
Praziquantel		
Fasciolasis (liver fluke)	Filariasis (tissue round worms)	
Praziquantel	Diethylcarbmazine	
Closantel	Suramin	
(and halogenated salicylamides)	Ivermectin	

### **Albendaz**ole

### A weak anti-parasitic

Test Dose

**Albendazole (ALB) start 200mg,** after 12 hours increase. If no reaction go to 600mg or 800mg loading for over 150 lbs. maintain for minimum of 3 days no reaction. If worm kill starts, increase to 1200-1600 mg for 7 days. Repeat every 1-3 months for a year to ensure kill and worms sterile.

### Co Factor

Albendazole has been studied at lower doses in combination with other anti-parasitic meds. It appears that if albendazole does not work directly, it has the potential to assist other anti-parasitic meds.

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TOLERANCE AND EFFICACY OF COMBINED DIETHYLCARBAMAZINE AND ALBENDAZOLE FOR TREATMENT OF WUCHERERIA BANCROFTI AND INTESTINAL HELMINTH INFECTIONS IN HAITIAN CHILDREN

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Abstract. This randomized, placebo-controlled trial investigated the tolerance, efficacy, and nutritional benefit of combining chemotherapeutic treatment of intestinal helminths and lymphatic filariasis. Children were infected with Ascaris (30.7%), Trichuris (53.4%), and hookworm (9.7%) with 69.9% having more than one of these parasites. A total of 15.8% of the children had Wuchereria bancrofti microfilariae. Children were randomly assigned treatment with placebo, albendazole (ALB), diethylcarbamazine (DEC), or combined therapy. The combination of DEC/ALB reduced microfilarial density compared with placebo, ALB, or DEC (P # 0.03). Albendazole and DEC/ALB reduced the prevalence of Ascaris, Trichuris, and hookworm more than placebo or DEC (P # 0.03). Among Trichurisinfected children, those receiving ALB and DEC/ALB demonstrated greater gains in weight compared with placebo (P # 0.05).

Albendazole and DEC/ALB were equally efficacious in treating intestinal helminths and for children with W. bancroftimicrofilaremia, DEC/ALB was more effective than DEC, with no increase in severity of adverse reactions.

RANDOMIZED TRIAL OF COMBINED HELMINTH TREATMENT

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TABLE 3 Helminth prevalence and intensity in Leogane, Haiti\*

	Treatment group	Pretreatment % prevalence†	Post-treatment % prevalence 5 weeks	Post-treatment % prevalence reduction 5 weeks	Pretreatment intensity, geometric mean, eggs/gram (arithmetic range)	Geometric mean % intensity reduction (paired) 5 weeks
Ascaris‡	Placebo	31.7 (97/306)	30.1 (92/306)	5.0	393 (40-24,0000)	11.7
	Albendazole	28.4 (91/320)	0.9 (3/320)	96.88	535 (40-34,800)	99.88
	DEC	28.1 (88/313)	23.3 (73/313)	17.1	617 (40-40,760)	18.0
	Combination	34.5 (107/310)	2.3 (7/310)	93.38	564 (40-36,000)	93.08
Hookworm‡	Placebo	10.1 (31/306)	7.2 (22/306)	28.7	84 (40-440)	48.8
	Albendazole	8.1 (26/320)	1.3 (4/320)	84.08	66 (40-320)	82.79
	DEC	10.2 (32/313)	8.0 (25/313)	21.6	59 (40-400)#	19.1
	Combination	10.3 (32/310)	1.9 (6/310)	81.68	83 (40-1,840)	99.28
Trichuris‡	Placebo	51.0 (156/306)	55.9 (171/306)	-9.6	153 (40-7,520)	9.6
	Albendazole	51.9 (166/320)	31.9 (102/320)	38.58	134 (40-6,600)	29.28
	DEC	55.3 (173/313)	58.1 (182/313)	-5.1	105 (40-6,480)**	9.8
	Combination	55.5 (172/310)	40.3 (125/310)	27.48	160 (40-30,840)	26.8§

Data from this study indicate that combination therapy was significantly more effective at suppressing microfilaremia when compared with placebo, ALB, or DEC alone at the

six-month assessment. Although both combination therapy and DEC alone demonstrated a decrease in the prevalence of microfilaremia, combination therapy decreased the geometric mean MF density to a greater extent than did DEC alone, resulting in a geometric mean reduction of 80.4% in MF density six-months post-treatment. Although this study was not designed to detect the macrofilaricidal effects of DEC or ALB, particularly since there was no ultrasonographic assessment of adult worms, DEC and combination therapy both demonstrated modest, but significant reductions in filarial antigen densities six-months post-treatment when compared with placebo or ALB alone, which is suggestive of a macrofilaricidal effect of DEC.

Additionally, this study suggests a microfilaricidal role for ALB.34 Although there was no difference in prevalence of microfilaremia, at the six-month follow-up, a significant geometric mean percent reduction in MF density was seen between ALB and placebo (34.7% versus 10.3%, respectively).

DEC = diethylcarbamazine. No significant differences between treatment groups. Non-exclusive infection; could have other intestinal helminth or filarial coinfections Significantly different from placebo and DEC (P < 0.05). Significantly different from DEC (P < 0.05). Significantly different from DEC (P < 0.05). Significantly different from placebo, (P < 0.05). Significantly different from placebo, albendazole, and combination.

However, this microfilaricidal effect is less than has been seen in studies of ivermectin.30,35 Moreover, this data differs from previous trials where no statistically significant differences have been seen in either MF prevalence or density between ALB and placebo.12,30

Compared with the ivermectin/ALB combination, for which reductions in geometric mean MF density of between 98.9% at four months and 88.6% at 12 months, respectively, have been shown, the DEC/ALB combination exhibited a slightly lower MF density reduction of 80.4% at six months, suggesting the enhanced microfilaricidal properties of the ivermectin/ALB combination.10,30 Since we re-treated the children in this study at six months, we were unable to determine if MF prevalence and density would continue to decrease after six months, as suggested by others.36,37 In this study, ALB and combination therapy successfully reduced both the prevalence and intensity of intestinal helminths, although less of a decrease was seen with Trichuris than with Ascaris or hookworm (Table 3). Similar to previous studies with children in Haiti, our study population was parasitized to a lesser degree (the prevalence of each helminth infection was less than 55%) and the intensity of helminth infection in this population was comparatively low (> 90% were light infections).11

### **Benzimidazoles**

### **thiabendazole**

**Brand Name: Mintezol** 

Take with food

tell your doctor about any other medical conditions that you have, especially liver or kidney disease. You may not be able to take thiabendazole, or you may require a dosage adjustment or special monitoring during treatment if you have any other medical conditions.

#### **Usual Adult Dose for Angiostrongylosis:**

25 mg/kg (up to 1 g) orally 2 to 3 times a day for 3 days.

The recommended maximum daily dose is 3000 mg.

### **Usual Adult Dose for Capillariasis:**

25 mg/kg (up to 3 g) orally once a day for 30 days.

The recommended maximum daily dose is 3000 mg.

### **Usual Adult Dose for Ascariasis:**

orally 2 times a day for 2 successive days. Alternatively, a single dose of 20 mg/lbs (50 mg/kg) may be used.

Dosage calculated using patient's weight:

If 30 lbs, give 250 mg
If 50 lbs, give 500 mg
If 75 lbs, give 750 mg
If 100 lbs, give 1000 mg
If 125 lbs, give 1250 mg
If greater than or equal to 150 lbs, give 1500 mg

The recommended maximum daily dose is 3000 mg

Thiabendazole (thye" a ben' da zole) is a benzimidazole antihelmintic agent similar in structure and mechanism of action to albendazole and mebendazole. The benzimidazoles act by selective binding to beta-tubulin of parasitic worms, causing their immobilization and death. Thiabendazole was approved in the United States in 1967 but has subsequently been withdrawn because of the availability of other, better tolerated antihelmintic agents, such as ivermectin, albendazole and mebendazole. Thiabendazole is, however, still available in other countries and is used in veterinary medicine in the United States. Its major indication is strongyloidiasis infestation. Thiabendazole was formerly available in chewable tablets of 500 mg under the trade name of Mintezol and as an oral suspension. The typical dose in adults was 1500 mg orally daily for 1 to 3 days.

### Hepatotoxicity

Thiabendazole therapy is associated with serum aminotransferase elevations in up to 36% of patients, but it is usually given for a brief period only and its effects on serum enzyme levels after single dose administration has not been systematically evaluated. Importantly, thiabendazole therapy has also been associated with clinically apparent liver injury which can be prolonged and severe. The onset of injury is usually within 1 to 2 weeks of finishing a 1 to 5 day course of therapy. The pattern of serum enzyme elevations is typically cholestatic. Autoantibodies are usually negative and fever, arthralgias and rash are uncommon. Several reported cases have been associated with sicca complex marked by parotid enlargement and tenderness, dry eyes and dry mouth arising before the onset of jaundice. The cholestatic injury can be associated with damage to small bile ducts and with prolonged jaundice and/or pruritus and alkaline phosphatase elevations. Several instances of prolonged cholestasis and chronic vanishing bile duct syndrome and end stage liver disease has been reported even after a single dose of thiabendazole.

#### **Mechanism of Injury**

Thiabendazole hepatotoxicity appears to be due to an immunological reaction to the drug, which is directed largely at bile, lacrimal, and salivary gland ducts.

#### **Outcome and Management**

While most reported cases of thiabendazole induced liver injury were self-limited, many were marked by severe and prolonged cholestasis. Several cases of prolonged jaundice with vanishing bile duct syndrome and evolution to cirrhosis leading to liver transplantation have been reported. Patients with acute liver injury attributed to thiabendazole should avoid repeat exposure. It is unknown whether there is cross sensitivity with other benzimidazoles (such as mebendazole), but there probably is and switching to another class of antihelmintic agents is appropriate if therapy is still needed.

The first of this class, **thiabendazole**, was discovered in 1961 and subsequently a number of further benzamidazoles were introduced as broad spectrum anthelmintics. There is an extensive literature on these compounds reporting a number of different biochemical effects.

Nonetheless, it is clear that their anthelmintic efficacy is due to their ability to compromise the cytoskeleton through a selective interaction with b-tubulin (Borgers and de Nollin, 1975; for review see Lacey, 1990). The effects of benzimidazoles on C. elegans, which include impaired locomotion, reproduction and a detrimental effect on oocytes, are consistent with disruption of processes requiring integral microtubules. The sensitivity of C. elegans to benzimidazoles is mediated by a single gene, ben-1, which encodes b-tubulin (Driscoll et al., 1989). This has provided a platform to investigate the molecular basis of benzimidazole resistance in parasitic nematodes. It has been noted that benzimidazole resistance in Haemonchus contortus seems to be associated with the presence of specific alleles for btubulin in the drug resistant isolates (Kwa et al., 1994). Whether or not a specific b-tubulin isoform could confer resistance to the drug was tested by experiments which showed that the sensitivity of C. elegans ben-1 mutants to benzimidazole can be rescued by expressing a H. contortus allele of b-tubulin from benzimidazole susceptible isolates but cannot be rescued by the allele present in the resistant isolates (Kwa et al., 1995). This unequivocally demonstrated that a single amino acid sustitution, Y for F, in b-tubulin, can confer anthelmintic resistance.

### Praziquantel - Prazi - PZQ

Praziquantel = powdered fish Praziquantel 50grams PZQ \$50.00 Powdered Fish Praziquantel normally comes in a 4 ounce bottle = 112 ml 1 teaspoon = 5 ml, ½ teaspoon = 2.5ml 2.5ml X 50G/112ml = 1.12 Grams Praziquantel per level teaspoon. Heaping ½ teaspoon = 1.5 Grams. Average slightly domed = 1.31 Grams 2 doses per day = 2.62 Grams 3 doses per day = 3.93 Grams. For 113 kgs, the daily 3 dose is 3.93/113=35mg/kg

### **Initial test**

Praziquantel (PZQ) or oxamniquine try 6mg/kg test, in 12 hrs move to 40mg/kg one day.

Prazi works for about 6 hours +/ at 1/2 teaspoon, I mix in Greek Gods Greek Yogurt.

Each 1/2 teaspoon of compacted <u>Prazi</u> powder contains about 1650mg of powder, when the kitchen measuring spoon is just slightly domed. Keeping the lid on tightly prevents powder compaction. Gently use the kitchen measuring spoon while removing the powder. A single 4 ounce bottle of <u>Prazi</u> lasts a little longer than a week.

The compacted dose produces a yield of 1650/113 kgs (my weight) or about 15 mg/kg dose.

Most research papers indicate the maximum dose is 100 mg/kg. The nominal dose is like 40 mg/kg, and the minimum dose is about 20 mg/kg. I take **Prazi** at 6PM, and at bedtime, for a much improved sleep. If I awaken at 3AM, I may dose again.

My total dose is 30 - 45 mg/kg/D.

If you kill to quickly, it is just as bad as not killing quickly enough.

I read several studies that indicated <u>Albendazole</u> actually makes the transport of <u>Prazi</u> deep into the body, possible.

I dose <u>Prazi</u> mostly at night, and <u>Albendazole</u> during the day, which appears to perform a very deep kill. Dosing <u>Prazi</u> with <u>Albendazole</u> appears to kill slower, probably due to the <u>Prazi</u> paralyzing the mouths of the worms.**Praziquantel** 

http://www.labome.org/expert/korea/seoul/chai/jong-yil-chai-883442.html

...The efficacy of praziquantel on the blood, liver, and lung fluke infections is considered to be due to the polarized metabolite form, which constitutes the majority of the praziquantel in the plasma; this form has only slightly lower anthelmintic efficacy compared with its unmetabolized form [1, 2].

The efficacy of praziquantel against cerebral cysticercosis, in which the larval cestode is located in the brain parenchyme or ventricle, is probably due to the passing of the metabolized form of the drug through the blood-brain barrier, despite the drug's overall low concentration in the CSF (only 1/7-1/5 of the plasma concentration) [2].

... <u>Praziquantel</u> is rapidly taken up through the gut (in humans more than 80% of the orally administered dose is absorbed from the gastrointestinal tract) and reaches its maximal plasma concentration within 1-2 hours [2].

If a person was orally administered 20-50 mg/kg praziquantel, the peak plasma concentration would become  $0.2-1.0 \mu g/mL$  [2].

The cerebrospinal fluid (CSF) concentration can be 1/7-1/5 of the plasma concentration [1].

Most of the praziquantel absorbed is metabolized in the liver by the cytochrome P450 system and becomes a hydroxylated polar metabolite form which has a lower protein binding capacity (lower helminthotoxic activity) than the unmetabolized form [1, 2]. The excretion of praziquantel occurs mainly through urine (60-80%) as well as bile and feces (15-35%) and is completed within 24 hours [1].

The elimination half-life of praziquantel in the human body is 1-2 hours [2], and after 24 hours, only a trace amount remains in the human body.

http://www.sciencedirect.com/science/article/pii/S0090301907004028

**FLUKE** Fascioliasis can be successfully treated using Praziquantel.

http://www.parasitetesting.com/Praziquantel.cfm

**Praziquantel increases the permeability of the membranes of schistosome cells towards calcium ions**. The drug thereby induces contraction of the parasites, resulting in paralysis in the contracted state. The dying parasites are dislodged from their site of action in the host organism and may enter systemic circulation or may be destroyed by host immune reaction (phagocytosis). Additional mechanisms including focal disintegrations and disturbances of oviposition (laying of eggs) are seen in other types of sensitive parasites. Another hypothesis concerning the mechanism of action of praziquantel has been recently reported. The drug seems to interfere with adenosine uptake in cultured worms. This effect may have therapeutical relevance given that the schistosome, as the taenia and the echinococcus (other praziquantel sensitive parasites), is unable to synthesize purines such as adenosine de novo. Bayer's Animal Health Division website states, "Praziquantel is active

against cestodes (tapeworms). Praziquantel is absorbed, metabolized in the liver and excreted in the bile. Upon entering the digestive tract from the bile, cestocidal activity is exhibited. Following exposure to praziquantel, the tapeworm loses its ability to resist digestion by the mammalian host. Because of this, whole tapeworms, including the scolices (plural of "scolex"), are very rarely passed after administration of praziquantel. In many instances only disintegrated and partially digested pieces of tapeworms will be seen in the stool. The majority of tapeworms are digested and are not found in the feces." Praziquantel is administered as a racemate, but only the (R)-enantiomer is biologically active; the enantiomers may be separated using a resolution of an amine obtained from praziquantel.

### **Praziquantel side effects**

The majority of side effects develop due to the release of the contents of the parasites as they are killed and the consequent host immune reaction. The heavier the parasite burden, the heavier and more frequent the side effects normally are. Central nervous system: Frequently occurring side effects are dizziness, headache, and malaise. Drowsiness, somnolence, fatigue, and vertigo have also been seen. Almost all patients with cerebral cysticercosis experience CNS side effects related to the cell-death of the parasites (headache, worsening of pre-existing neurological problems, seizures, arachnoiditis, and meningism). These side effects may be life-threatening and can be reduced by coadministration of corticosteroids. It is strongly recommended that all patients with cerebral cysticercosis are hospitalized during treatment. GI Tract: Approximately 90% of all patients have abdominal pain or cramps with or without nausea and vomiting. Diarrhea may develop and may be severe with colic. Sweating, fever, and sometimes bloody stools may occur together with diarrhea. Liver: Asymptomatic and transient increases of liver enzymes (AST and ALT) are noted frequently (up to 27%). No case of symptomatic liver damage has ever been seen so far. Sensitivity reactions: Urticaria, rash, pruritus and eosinophilia in white blood cell counts Other locations/body as a whole: Lower back pain, myalqia, arthralqia, fever, sweating, various cardiac arrhythmias, and hypotension.

### **TCBZ**

Triclabendazole sheep TCBZ, if they ask, enter any number for animal and flock

### **Piperazine Citrate**

http://www.sciencedirect.com/science/article/pii/S0001706X06001392 http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1980175/?page=2 http://wormbook.org/chapters/www\_anthelminticdrugs/anthelminticdrugs.pdf

<u>Piperazine</u> was first used as an anthelmintic in the 1950s and it is still the active constituent of over the counter remedies for thread worm infection in children. Its mode of action has primarily been studied in A. suum. There is surprisingly no literature on its action in C. elegans though there is no indication that it acts differently from its effects in A. suum. In A. suum it acts as a weak GABA-mimetic and causes a flaccid, reversible paralysis of body wall muscle. Single channel recordings provide evidence that it is a low efficacy, partial agonist at GABA-gated chloride channels (Martin, 1985).

<u>Piperazine</u> <u>Causes parasites to detach from the blood supply</u>. Natural body processes will remove the worm debris, if the worm is detached for a period of time. A flaccid paralyzing agent that causes a blocking response of ascaris muscle to acetylcholine.

**Piperazine** blocks *Ascaris suum* larval moulting and development processes and affects larval stages. **PPZ potently inhibited moulting of** *A. suum* **LL3** in a dose-dependent manner and that moulting was completely blocked (100%) at 50 mM concentrations. PPZ exposure also inhibited expression of 13 immunogenic protein spots in unmoulted LL3. More importantly, **PPZ exposure inhibited activity of a moulting-specific enzyme**, inorganic pyrophosphatase of *A. suum* (AsPPase), by 26%. PPZ interfered with growth and ecdysis of the cuticle and **caused damage to gut tissues of the larvae**.

<u>Piperazine</u> phosphate treated worms seamed a little less active than other salts of Piperazine.

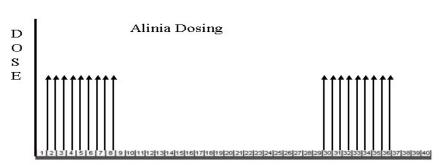
<u>Dosing of piperazine falls within a very narrow window, and must be accurately dosed.</u>

Piperazine has a low acute toxicity in animals and man. However there had been occasional reports of neurotoxicity when piperazine was administered to individuals with intestinal worm infections. It has been suggested that the mechanism of neurotoxicity involves GABA receptors antagonism. Piperazine base is strongly alkaline and therefore is an irritant; however its salts have a much lower irritant potential. The above findings were supported by the findings in the present paper, as treated pigeons at various dosage levels shows evidence of neurotoxicity supported by the findings of degenerate vacuolated nerve fibers mainly in white matter of sciatic nerve and spinal cord with dose related effects

Piperazine is a mild hepatotoxin and neurotoxin. In dogs which appeared to be among the more sensitive laboratory animal NOEL values of 25 mg/kg body weight day<sup>-1</sup> and 50 mg/kg body weight day<sup>-1</sup> have been identified. Neurotoxic effects had also been observed in man generally at relatively high levels. However some individuals do appear to be rather more sensitive than the general population to the neurotoxic effects. Consequently a NOEL for humans has not been established satisfactorily. It is likely to be of the order of 30 mg/kg body weight day.

Can cause liver damage at levels at or above 50mg/kg/D NOEL = no observable damage

Advise: max level less than 90 days at 14 mg/kg/D



277

**Dosing** 

Single dose 9 days on and 21 days off, 9pm to 9am, tested 3AM

Pulse probiotic every week for 4 weeks to ensure good gut bacteria.

### **Black Pepper (Piperazine) Background**

https://www.mountainroseherbs.com/products/peppercorn-black/profile

### **Common Name**

Standardized: pepper Other: maricha

### **Botanical Name**

Piper nigrum L.1

Plant Family: Piperaceae

### **Parts Used**

Dried fruits often referred to as 'peppercorns'

#### **Overview**

Black pepper is one of the most commonly used spices in the world, adding warmth and zest to savory dishes. It has been popular in India for thousands of years and is now easy to find almost anywhere on the planet. It has been employed medicinally in both Ayurveda and TCM and more recently in herbal folk healing practices. In modern times, numerous studies have confirmed its healing properties. Several derivatives of black pepper, such as piperine, have shown promise in supporting the health of the digestive system and as an antioxidant. Further, it has demonstrated that it can enhance the absorption of other nutrients when taken simultaneously. <sup>3-10</sup>

### **Botany**

A member of the *Piperaceace* family alongside long pepper (*Piper longum*), and kava kava (*Piper methysticum*), black pepper is native to southern India and Sri Lanka and widely cultivated in the tropics. <sup>1,11</sup> The pepper plant is a tropical perennial vine requiring a trellis or some other support such as a tree to grow along. <sup>11,12</sup> It has aromatic, green, ovate leaves that give off a strong fragrance and greenish yellow flower spikes. <sup>12</sup> The generic name *Piper* is Latin in origin and is derived from the Sanskrit name for long pepper 'pippali. <sup>13</sup>

### **Cultivation And Harvesting**

Black pepper is the most traded cultivated spice<sup>12</sup> and is propagated by cuttings that are grown by the base of trees.<sup>14</sup> They bear fruit three to four years after planting and cease around the fifteenth year. The black peppercorns and ground pepper of commerce are actually immature fruits that are collected as soon as they turn red and dried in the sun.<sup>14</sup> The peppercorns turn black after three days of drying, and when ground, produce black pepper powder.<sup>11</sup> When the fruits are left to ripen and the red outer covering is removed, then white pepper is obtained. Malabar produces the highest quality commercial pepper and Sumatra and Java supply much of the U.S. demand.<sup>11</sup> Some of the main pepper producing countries include: Brazil, India, Indonesia, Madagascar, and Malaysia.<sup>15</sup>

### **History And Folklore**

Black pepper has been grown in Southern India for over two thousand years. <sup>13</sup> Next to salt, pepper is the most popular spice being used continually for thousands of years, and in fact, used in Indian cooking since at least 2,000 BCE. <sup>11</sup> Alexander the Great fought his way through central Asia making way for new trade routes which led to the eventual availability of black pepper in the West. It became increasingly popular and was highly traded by the Arabic spice merchants. <sup>13</sup> Pliny the Elder (23-79 CE), the Roman naturalist, wrote in his book *Natural History:* 

It is quite surprising that the use of pepper has come so much into fashion, seeing that in other substances which we use, it is sometimes their sweetness, and sometimes their appearance that has attracted our notice; whereas, pepper has nothing in it that can plead as a recommendation to either fruit or berry, its only desirable quality being a certain pungency; and yet it is for this that we import it all the way from India! 16

And, as the story goes, during the 5th century CE, Attila the Hun demanded 3,000 pounds of pepper as a ransom for the city of Rome. <sup>14</sup> Although pepper was very expensive, it was still utilized often in cooking by the ancient Romans, and by the Middle Ages, became a symbol of fine cuisine. <sup>13</sup> Due to the high prices of pepper, Europeans often used pepper substitutes such as chaste tree (*Vitex sp.*), grains of paradise (*Aframomum melegueta*), and myrtle berries (*Myrtus communis*) amongst others. <sup>13</sup> However, by the 15th century Europeans ventured out to regions where spices where grown so that they could trade directly with growers, therefore cutting out the middlemen who had driven up the prices. This venture was led by the Portuguese, British, Spanish, and Dutch. <sup>13</sup>

Black pepper was believed to be imbued with magical qualities, to be ruled by the planet Mars, and represented by the fire element. It was thought to protect against the 'evil eye.' Further, it was said that wearing a peppercorn would free one of envious thoughts.<sup>17</sup> In Asia, black pepper is considered a powerful medicinal plant useful in supporting healthy aging, liver detoxification, circulation, and digestion and is thus a prominent healing herb in TCM (traditional Chinese medicine) and Ayurveda (traditional healing system from India).<sup>2</sup> In both TCM and Ayurveda pepper is considered to be a pungent tasting and energetically heating spice (khalsa/TCM wiki). In Ayurveda, it is often found in the formula called 'Trikatu' which is a combination of black pepper (*Piper nigrum*) and also its relative long pepper (*Piper longum*) and ginger (*Zingiber officinale*), and is used to support digestion.<sup>2,5</sup> It effects the stomach and large intestine meridians in TCM and is thus employed in digestive complaints and in cases where qi (energy) needs to be directed downward.<sup>18</sup>

### **Flavor Notes And Energetics**

Pungent tasting and energetically heating  $^{2,18}$ 

### **Herbal Actions**

Alterative, expectorant, energizer, carminative

#### **Constituents**

Essential oil containing the monoterpenes sabinene, pinene, limonene, terpinene, pinene, myrcene, carene and mono¬terpene derivatives (borneol, carvone, carvacrol, 1,8-cineol, linalool), sesqui¬terpenes such as caryophyllene, humulene, bisabolone and caryophyllene oxide and ketone, <sup>13</sup> alkaloids such as piperine, chavicina, piperic acid, piperidine, various vitamins and minerals. <sup>12</sup>

### **Scientific Research**

The alkaloid derived from black pepper, **piperazine**, is used as an anthelmintic drug called **Entacyl**. The derivative piperine has been shown to be useful in increasing bioavailability and the absorption of nutrients.<sup>3,4,5</sup> Piperine has also demonstrated monoamine oxidase (MAO) inhibitory activity. Certain derivatives from black pepper have proven to be useful insecticides against various mosquito species as well.<sup>7</sup>

Used as alternative treatment for ascariasis caused by *Ascaris lumbricoides* (roundworm) and enterobiasis (oxyuriasis) caused by *Enterobius vermicularis* (pinworm). It is also used to treat partial intestinal obstruction by the common roundworm, a condition primarily occurring in children.

 $LD_{50} = 5$  g/kg (Human, oral). Symptoms of overdose include muscle fatigue, seizures, and difficulty breathing.

https://pharmacycode.com/Entacyl.html

### **Entacyl Brand Names Mixture**

- Canoids Cap (Aloin + Areca Catechu + Arecoline HBr + Benzocaine + Piperazine Citrate + Santonin)
- Dyrex T F (Phenothiazine + Piperazine + Trichlorfon)
- Feloids Tab (Aloin + Areca Catechu + Arecoline HBr + Benzocaine + Piperazine Citrate + Santonin)
- Multi Wormer for Cats (Dichlorophene + Piperazine (Piperazine Citrate))
- Multi Wormer for Dogs (Dichlorophene + Piperazine (Piperazine Citrate))
- Ripercol Horse Wormer (Piperazine (Piperazine Hydrochloride) + Tetramisole HCl)

### **Entacyl**

### http://edudrugs.com/E/Entacyl.html

Entacyl is an organic compound that consists of a six-membered ring containing two opposing nitrogen atoms. Entacyl exists as small alkaline deliquescent crystals with a saline taste.

Entacyl was introduced to medicine as a solvent for uric acid. When taken into the body the drug is partly oxidized and partly eliminated unchanged. Outside the body, piperazine has a remarkable power to dissolve uric acid and producing a soluble urate, but in clinical experience it has not proved equally successful.

Entacyl was first introduced as an anthelmintic in 1953. A large number of piperazine compounds have anthelmintic action. Their mode of action is generally by paralysing parasites, which allows the host body to easily remove or expel the invading organism.

Entacyl is a GABA receptor agonist. Piperzine binds directly and selectively to muscle membrane GABA receptors, presumably causing hyperpolarization of nerve endings, resulting in flaccid paralysis of the worm. While the worm is paralyzed, it is dislodged from the intestinal lumen and expelled live from the body by normal intestinal peristalsis.

Phenothiazines - Taking piperazine and a phenothiazine together may increase the risk of convulsions (seizures).

Pyrantel (e.g., Antiminth) - Taking piperazine and pyrantel together may decrease the effects of piperazine.

Patients with hypersensitivity to piperazine salts or a history of renal function impairment should avoid this medication

### **Invermectin**

Ivermectin (macrocylic lactones and milbemycins)

Ivermectin was introduced as an anthelmintic in the 1980s by Merck. It is a semi-synthetic derivative of avermectin which is a large macrocyclic lactone fermentation product of the micro-organism Streptomyces avermitilis. It is remarkably potent (~1nM) and persistent in its effect and its discovery enthused other companies to invest in the development of ivermectin analogues which include moxidectin, milbemycin oxime, doramectin, selamectin, abamectin and eprinomectin. Here C. elegans played a role as it was employed in a screen for further macrocyclic lactones with ivermectin-like activity (Haber et al., 1991). Ivermectin elicits a potent and persistent paralysis of nematode pharyngeal (Brownlee, Holden-Dye and Walker, 1997; Pemberton et al., 2001) and body wall musculature (Kass et al., 1980; Kass et al., 1982). It has been shown to interact with a range of ligand-gated ion channels including a7 nACh receptors (Krause et al., 1998), acetylcholine-gated chloride channels (Bokisch and Walker, 1986), GABA-gated chloride channels (Robertson, 1989; Holden-Dve and Walker, 1990), histamine-gated chloride channels (Zheng et al., 2002), glycine receptors (Shan et al., 2001) and P2X4 receptors (Khakh et al., 1999). However, it is its high affinity for nematode glutamate-gated chloride channels (GluCl) that correlates with its potent anthelmintic activity.

### Latrotoxin

Latrotoxin paralyses mammals by triggering neurotransmitter release, and thus the identification of

latrophilin as an emodepside receptor raised the intriguing possibility that emodepside may cause paralysis of nematodes by stimulating excessive neurotransmitter release at neuromuscular sites. Studies in *A. suum* have highlighted muscle paralysis and point to a

calcium- and potassium-dependent mechanism of action (Willson et al., 2003). C. elegans is very susceptible to the effects of emodepside at nanomolar concentrations (Bull et al., 2007). The effects include slowed development, inhibition of pharyngeal pumping, decreased locomotion (forward movement is most affected at low concentrations), and inhibition of eag-laying leading to 'bagging' in adult hermaphrodites. Thus C, elegans has provided an excellent model in which to define the molecular target, or targets, through which emodepside exerts its pleiotropic actions and indeed, to test whether latrophilins are involved.SLO-1 is a calcium-activated potassium channel (Wang et al. 2001) homologous to the mammalian BK channels. Thus the discovery of SLO-1 as an important effector for emodepside resonates with earlier work on Ascaris muscle which showed a calcium and potassium-dependent hyperpolarisation (Willson et al., 2003). This channel is highly conserved throughout the animal phyla and plays a pivotal role in regulating neuronal and muscle cell excitability (for review, Salkoff et al., 2006). In C. elegans it is widely expressed in the nervous system and body wall muscle, but not in pharyngeal muscle. By expressing a wild-type copy of slo-1 in specific subsets of cells in a slo-1 null background it has been shown that emodepside can inhibit locomotor activity when SLO-1 is present in neurones or body wall muscle but it can only inhibit feeding if SLO-1 is present in neurones (Guest et al., 2007). Earlier studies showed that mutations in a number of genes encoding synaptic proteins confer altered sensitivity to the effect of emodepside on feeding consistent with the idea that it acts in the neuronal network to disrupt rhythmic feeding behaviour (Willson et al., 2004).

### **Nitazoxanide**

#### Alinia generic - 500 mg 7 tablet Rosanil Nitzzoxanida \$6.50/box of 7

Nitazoxanide, a pyruvate ferredoxin oxidoreductase inhibitor, acts against a broad spectrum of protozoa and helminths that occur in the intestinal tract. It is currently used for the treatment of protozoal infections (and is therefore not listed in Table 1). The site of action of this compound has not been established in nematodes although anaerobic electron transport enzymes may be a potential target (Gilles and Hoffman 2002). The effect of nitazoxanide has been examined on growth and development of C. elegans (Fonseca-Salamanca et al., 2003). After seven days culture, nitazoxanide 100  $\mu$ M, only reduced population growth by 33%. In contrast mebendazole,  $5\mu$ M, and albendazole,  $1\mu$ M, reduced growth by over 90%. Nitazoxanide, 100  $\mu$ M, had no effect on either embryonation or hatching in Heligmosomoides polygyrus. Therefore the efficacy of this compound is relatively low compared to other anthelmintic agents.

Uh-oh.... are you taking a probiotic? Alinia will wipe out your intestinal bacteria (which you need). Lactobacillus GG should be taken concurrently. I don't know if this the cause of your problem though. Hopefully others will weigh in soon.

It is hard to say if Alinia is the culprit of the nausea and diarrhea. It is a broad acting antibiotic and can reduce the intestinal flora, so using antibiotic resistant Lactobacillus from the start seems like a good idea.

MAK - Dosing a single pill for more than 9 days seams to be counter productive.

### Levamisole, pyrantel and morantel

These anthelmintics are nicotinic receptor agonists (Aceves et al., 1970; Aubry et al., 1970) and elicit spastic muscle paralysis due to prolonged activation of the excitatory nicotinic acetylcholine (nACh) receptors on body wall muscle. Their precise mode of action has been carefully studied at the single-channel level on the body wall muscle preparation of A. suum (see Martin et al., 2005 for review). Pharmacological analysis has provided evidence for subtypes of nACh receptor (Qian et al., 2006), an N-type (preferentially activated by nicotine), a B-type (preferentially activated by bephenium) and an L-type (preferentially activated by levamisole and associated with

levamisole resistance). Levamisole, and related compounds, also cause spastic paralysis and egg-laying in C.elegans. Indeed, recordings from C. elegans body wall muscle using levamisole and nicotine as agonists have provided further evidence that there are muscle subtypes of nACh receptor and that these subtypes have different nACh receptor subunit compositions. At least four subunits, unc-38, unc-29, unc-63 and lev-1 contribute to the levamisole receptor (Culetto et al., 2004; for further information on the nACh receptor subunit family see Rand, 2007). Thus, these anthelmintics are providing pharmacological tools to dissect subtypes and stoichiometries of native nematode nicotinic receptors.

Perhaps more importantly, levamisole has been extremely productive in forward genetic screens. In the earliest studies tetramisole was used (Brenner, 1974) and later this was replaced by the more active isomer, levamisole (Lewis et al., 1980). These screens have provided a resource of mutants that have been used over the last two decades to assign function to genes expressed at the neuromuscular junction.

#### **DEC**

**DEC-C - PM (4TID) TAB. 400 mg** DEC-c (Diethylcarbamazine citrate), (Dimmitrol) 9 wks or until Ascaris dead or wounded reproduction

**DEC-C start slow provocation test, 5, 10, 25mg.** If a reaction go to DEC-C progressive start dose, start with 5mg, 10mg, 25mg, 50mg, 100mg, 200mg, load to 6mg/km with 12-24 mg/kg peak, increase from first level if there is a reaction every 12 hours. If no reaction, double dose every 12 hours till tolerance. If reaction maintain 6 mg/kg for a month with 100-200 mg DEC level for 8 weeks.

Lymphatic filariasis causes a severe social burden because infected individuals, both men and women, often are social outcasts and have physical limitations. Opportunities for employment and marriage are severely reduced.

#### **Treatment**

For effective control of the disease in a community, infected individuals must have the microfilariae completely removed (eradicated) from their blood. Until recently, a 12-day course of an antiparasitic drug called diethycarbamazine (DEC) was given to infected individuals. Following treatment, microfilariae are eradicated for a full year. Recent studies have shown that a single dose of DEC is equally effective for treatment.

The most effective treatment currently available is a single dose of two drugs administered at the same time. This two-drug treatment may include albendazole with either DEC or ivermectin and is 99% effective in removing microfilariae from the blood for one year.

Current antiparisitic drug therapy is approaching 100% effectiveness. However, some drugs have unpleasant and sometimes serious side effects. Common side effects of albendazole, DEC, and ivermectin include chills, fever, and muscle pain (myalgia). The intensity of side effects is significantly

higher with ivermectin than with DEC. Side effects usually are most severe 24 hours after taking the medicine. Serious side effects are rare and usually occur with prolonged usage. They include loss of vision, tunnel vision, and night blindness.

**DEC-C** 400 mg DEC-c (Diethylcarbamazine citrate), (Dimmitrol)

- oUpsets and slows Nematodes and Roundworms
- o DEC strips the parasite shell off on some specis like w.b.
- TAKE WITH CALCIUM

http://www.vetshopmax.com/Dimmitrol-Tabs-for-Large-Dogs-67-132lbs-400mg-P274C105.aspx

o Antihelmith, Derivative of piperazine, Hetrazan trade name

http://www.ncbi.nlm.nih.gov/pubmed/7573715

- Diethylcarbamazine is a drug that is used for the treatment of filariasis in humans which has been used since 1947, and animals;
- o **It also has effects on intestinal nematodes**, but its mechanism of action remains unclear, which in an unknown way
- o It leads to immobilization and sequestration of the microfilariae.
- o Emodepside is a resistance-busting anthelmintic approved for treating intestinal parasitic nematodes in animals. The novel mode of action and resistance-breaking properties of emodepside has led to its use against **intestinal nematodes** of animals, and as a candidate drug for treating **filarial parasites**. We have previously demonstrated effects of emodepside on SLO-1 K<sup>+</sup>-like currents in *Ascaris suum*. Here, we demonstrate that diethylcarbamazine, which has been proposed to work through host-mediated effects, has direct effects on a nematode parasite, *Ascaris suum*. It increases activation of SLO-1 K<sup>+</sup> currents and potentiates effects of emodepside. Our results suggest consideration of the combination of emodepside and diethylcarbamazine for therapy, which is predicted to be synergistic. The mode of action of diethylcarbamazine may involve effects on parasite signaling pathways (including nitric oxide) as well as effects mediated by host inflammatory mediators.
- o Is more effective when combined with ivermectin or albendazole [21]. Although diethylcarbamazine is a piperazine derivative, diethylcarbamazine does not mimic the effects of piperazine by acting as a GABA agonist on parasite muscles [22]. Here we observed that diethylcarbamazine, in the presence of sufficient calcium, had a direct effect on the worm preparation and increased activation of the SLO-1 K+ channel currents by shifting the *V50* in the hyperpolarizing direction. SLO-1 channels are calciumdependent K+ channels that are pharmacologically different from delayed rectifier K+ channels. In low-calcium, even high concentrations of diethylcarbamazine (mM) do not activate SLO-1 K+ currents showing that this action requires calcium [22]. High, mM concentrations of diethylcarbamazine, in low-calcium conditions, inhibits nicotinic acetylcholine currents (b3????) and a delayed rectifier K+ current [22] but these high-concentration effects are non-selective.

### **Flagyl**

The effect of metronidazole in treating human fascioliasis.

http://www.ncbi.nlm.nih.gov/pubmed/14523339

#### Abstract

#### BACKGROUND:

The aim of this study was to determine the effect of metronidazole in patients who did not cure after treatment with triclabendazole, in Guilan (Northern Province of Iran).

### MATERIAL/METHODS:

Patients, who passed fasciola egg in stool and had positive serum anti fasciola antibody (ELISA), at least three months after treatment with triclabendazole, were enrolled and received 1.5 g/day metronidazole orally for three weeks. Two months and 12 months after end of therapy, stool examination in 3 consecutive days and serum anti fasciola antibody were performed. Frequency of patients with negative serology for fasciola and/or absence of fasciola egg in stool were determined. Chi-square test was used and P value <0.05 was considered significant.

#### **RESULTS:**

Forty-six patients, 26 females and 20 males, were enrolled with mean (+/-SD) age of 34.6(+/-9.8) years. Three patients excluded because of drug side effect and poor compliance. Two months after end of therapy, stool exam became negative in 35 patients and in 31 patients became negative both in serology and stool examination. (Difference in response to treat between age groups and genders was not significant). All patients with abdominal pain became pain free after therapy. Most frequent side effects were metallic taste in 14 (30.4%), headache in 8 (17.4%) and nausea in 6 (13%). 12 months after end of therapy, 28 out of 35 patients were examined again and all were negative both in serology and egg in stool examination.

### **CONCLUSIONS:**

Metronidazole, 1.5 g/day for 3 weeks, seems to be an effective, available, well-tolerated alternative for treatment of human fascioliasis.

#### METRONIDAZOLE General Monograph

http://www.rxmed.com/b.main/b2.pharmaceutical/b2.1.monographs/CPS-%20Monographs/CPS-%20(General%20Monographs-%20M)/METRONIDAZOLE.html

#### Extract

Metronidazole is less than 20% bound to serum proteins and is widely distributed in the body. It reaches all tissues and fluids, with CSF concentrations reaching approximately 43% of serum concentrations. The drug crosses the placenta and is distributed into breast milk. Metronidazole is metabolized in the liver. It is excreted primarily in the urine as metabolites, with 20% of a dose excreted as unchanged drug. The half-life of metronidazole in adults ranges between 6 and 12 hours. Accumulation may occur in patients with severely impaired hepatic function; dosage reduction may be indicated. Dosage adjustment is generally unnecessary in patients with decreased renal function.

### Mebendazole (Vermox)

Mebendazole is used to treat:

- Common roundworms (ascariasis);
- Hookworm infections (uncinariasis);

- Pinworms (enterobiasis; oxyuriasis);
- · Whipworms (trichuriasis); and
- · More than one worm infection at a time.

This medicine may also be used for other worm infections as determined by your doctor.

Mebendazole works by keeping the worm from absorbing sugar (glucose). This gradually causes loss of energy and death of the worm.

Mebendazole is an antihelmintic agent used commonly for roundworm (pinworm and hookworm) infections, trichinosis, capillariasis and toxocariasis and other parasitic worm infections. Mebendazole when given for prolonged periods in high doses has been associated with elevations in serum enzyme levels, and rare instances of acute, clinically apparent liver injury have been linked to its use.

Mebendazole was approved for use in the United States in 1974 and is indicated for therapy of common parasitic worm infections. Mebendazole is available generically in 100 mg chewable tablets. The usual dose is 100 to 500 mg once (pinworm) or varying doses for 3 days (whipworm, hookworm and roundworm infections), or varying doses for up to 28 days, depending upon the indication. Side effects are uncommon, but can include gastrointestinal upset, fever and diarrhea.

Mebendazole when given in typical doses has not been associated with serum enzyme elevations, although the duration of therapy is usually short and monitoring for enzyme elevations has rarely been reported. With high dose therapy (which is now rarely used with the availability of albendazole), elevations in serum aminotransferase levels (2 to 10 times normal) can occur, but are usually well tolerated. There have been rare reports of acute liver injury due to mebenazole, particularly when it is given repeatedly or in higher doses. The onset is usually with fever and malaise within days of starting or restarting therapy. The pattern of serum enzyme elevations is typically hepatocellular and jaundice is uncommon. The abnormalities usually resolve rapidly with stopping therapy. Signs of hypersensitivity (rash, fever and eosinophilia) are typical and liver biopsy may show granulomas.

### Mattk3 Personal Log - Formula #3

This log starts at Day 0 of the combined Ascaris/Fluke formula. The weeks of trying things is over, previous kills found in **SIBO**, **SIYO**, Virus. Zinc kill of worms found at 9,32mg/kg. Previous attempts at **DEC**, **Praziquantel**, **Albendazole**, **Piperazine** taken alone did not produce a substantive kill. This formula combines all elements, after a high dose of ALB discovered an underlying Fluke infection. The theory being tested here is that there can be no Ascaris kill, without first eliminating Flukes.

Early Ascaris kills were made with Zinc-Sulfate @9.32mg/kg (11/2014), I killed hundreds, then dosed Magnesium Citrate, MSM, <u>Magnesium sulfate</u>, and Potassium Citrate to try to stop the bursting. I then dosed IVM and <u>DEC</u> to kill the babies. The bursting continued until I took a second dose of IVM. I dosed IVM and <u>DEC</u> for 2 days to ensure a total baby kill of worms.

Day 0 Attempted Albendazole and Prazi kill, totally different.

Doing full formula.

Day 14 Dumping black

Day 15 Kill starts in brain, lasts for 4 days in bed

# Day 15 Massive Fluke kill

Day 15 Dumping black, killing in brain, discovered relationship between D3 and kill rate in brain. 50,000 IU D3.

Day 16 Massive stink, toxin, ammonia dumping, hundreds of flukes visible in loose stool.

Day 18, Stinky stinky stinky stools.

Day 20 Sleep over 8 hours.

Day 24 Brain clearing, 3 flights of stairs no problem.

Day 28 Stool returns to brown.

Day 30

I think I am down to 2 or one worm.

I made my throat/brain fluke sick on **Prazi**. I think Ascaris are long gone.

The **Prazi** paralyzes the mouth, and the worm just did not move, and burned.

I think it may be a matter of days, that brings is close to the 35 days I read about.

Sleep is good, so much brain to repair. Skin looking better. Finally loosing that "parasite tan".

### **Day 34 Anti-Parasitics ASCARIS FLUKE**

Crazy buzzing in body, Sore throat, then Acid Reflux and GERD, Sweats, Worm burst in lung, Ascaris giving birth, Dosed **IVM**. Decided to throw the alphabet at the babies, **DEC**, **ALB**, **Piperazine** tablespoon, then later, second dose of **IVM**. The Acid kept coming up the throat for several hours, like a burn. Then 24 hours later, at 6PM a little more birthing, Mrs worm must not be done, did a second round to kill the babies in the upper GI to keep them from traveling.

### **Day 35**

Headache at 2 AM, toxin sick feeling, Dosed Prazi, The brain sucking fluke stopped almost in minutes, went back to sleep. Strange that this re-birthing came at day 35, Must be straggler worms not killed in the first 30 days. I will have to keep the formula up for another 30 days to make sure they are all dead, really dead, certainly dead. I will get the fluke ELISA test so I can retest at 9 months to ensure a total kill. **TCBZ** still on B/O.

I am not sure how long to taper.

I will review my file server and write a paper that addresses the following topics:

### **Day 36**

I backed off meds at 30 days. I knew it was a mistake after 8 years of mischief. I am back on the protocol, and within hours everything snaps back into view. **DEC** is essential to the protocol, after several days off of the **DEC**, babies emerged.

I also suspect after testing <u>Prazi</u> at off hours, that it may work better separated in time from <u>Piperazine</u>, <u>Albendazole</u>, and <u>DEC</u>. I think <u>Prazi</u> paralyzes the worms mouths, slowing the ingestion of anti-parasitics. I also discovered that dosing all at once gives about 8 hours of coverage. I still have a few things to learn I now can tell the moment some guy starts to feed, cause I sense the toxins. They get meaner and meaner, when you deprive them of food. I think there are a few large ones left, they almost make me sweat, but not quite.

Day 35, 36, 37 test new time separation between Albendazole and **Praziquantel**, works 4x better. Day 38 – was a perfect day.

Day 43 Acid Ammonia stool

Day 44 45 smell of dead worms.

Day 49 urine, brain returning to more normal, sleep > 8 hours, reek of dead worms.

Day 50 dead worm stench subsiding

### Tweak 1

? Worms die faster without Vitamin A, etc,

Day 47 Toxins and worms diminish, acid mouth skin

worms are smaller, focus is better, return of cold nose says missed a birth of small worms.

Day 46 – Dosed IVM, **DEC** AM Noon, ALB AM NOON, PIPERAZINE AM/PM

Day 54 changed up sequence of ppz, alb, **dec** 

Day 55 stopped praziquantel Day 58 new sequence works, move Cq10 to AM, etc

### **Day 59 Massive FLUKE? Ascaris Kill Stench**

Need more vitamin C, lot of OJ, Cranberry

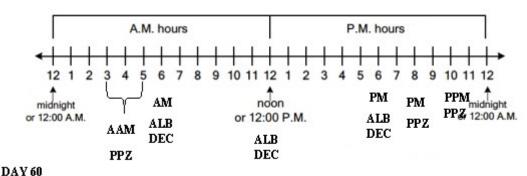
### **TWEAK 2 – Day 60**

Sinuses cleared in hours, dead worms slid down lymph,

Worm stench is nauseating, moved **dec** alb to 6 pm, 6 am, 3 doses of ppz, 8pm 1030 pm, 3AM Extreme sodium (salt taste) in mouth, Ascaris dying

No supplements, orange juice, **cranberry** juice, distilled water, after double dose of kgp the kidneys are moving fluid.

#### 24-Hour Timeline



### Day 60 FLUKE? Ascaris Killing Formula

**If after 60 days**, Stronger antiparasitics should be considered. Several species do not respond fully, Fenbendazole is a stronger alternative, at 2.5mg/kg for 3 days, 6 days max for stronogyloides.

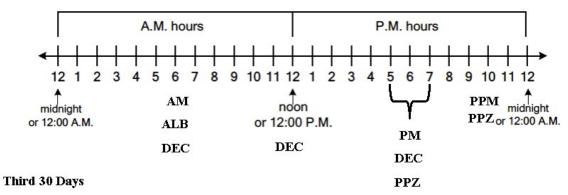
<u>If after 60 days</u> of a systemic infection consider an additional dose of alb and <u>DEC</u> at noon to 3 PM to finish them off. Even one large worm can try to multiply with fervor. At day 60, being off of Zinc and Vitamin A, and reducing supplements, the body should start to return to normal.

### Day 63 Lower Albendazole, Lets get roundworms

Liver congestion, vision shows fluid retention, even with removal of vits, better kidney flow, salt taste in mouth, cannot process even one piece of rum cake. Knocked ALBENDAZOLE down to single pill, or 200mg. Suspect it no longer has a function. Knocked it down last night, dosing **DEC** alone. AM started one **DEC** 400mg, and one ALB 200 mg. Will do noon **DEC**, PM **DEC** with PPZ?

No, move to final formula, dose 6 am, 6 pm PPZ, and DEC, kill GERD.

#### 24-Hour Timeline



Ascaris only

Sleeping through the night

### **Day 64**

Huge vitamin C craving, Smells, Fluid flowing through lymph, body detoxing in mass. Feeling better every day. Minimal parasite activity. Lowering ALB shows kidney sensitivity to prolonged high dose ALB. Lowered dose in main table to reflect the maximum I took in a prolonged fashion, add caution and notes on kidney flow and ALB dosing.

Add OJ to AM/PM, the vitamin C from OJ has huge difference at this stage, not enough C in the plan.

A banana is an excellent source of potassium.

Day 66 air burns, massive clarity increase from pulsing vitamins, add ed ALA, BAM ivm dose

(1-2) Chlorella

Testing (4-2) Spirulona tablets

(1-2) milk thistle

(0-1) ginger

- (1) B50
- (1) CQ10 testing 400mg, 200mg, 100mg
- (0) ALA develop a pulse protocol?
- (2) Ginkgo

(0-1) borage oil

OJ

**Cranberry** Juice

Distilled h20

Milk, Greek yogurt, cream cheese, butter, half and half cream.

- (1) egg pulsed
- (0-1-2) ALB am
- (1) DEC am/noon
- (1) tbs **PPZ** pm,ppm,

green vegi pm

soup salad noon

fruit am, ppm

#### **DAY 67**

I am still on DEC at day 64. I expected to take it a total of 60 days ish. It helps suppress new born worms, till the immune system takes over.

The formula kills flukes first, so you can get to the Ascaris.

The second kill, (taper Prazi) (lower alb) maintain Piperazine and DEC till the round worms are gone. The DEC and Piperazine are actually related to each other, they prevent worm malting. This prevents worms from shedding their skin, and going to the next level. While great for decreasing the population, it takes a while before the large worms go sterile from the ALB.

The process takes as little time as possible to wipe out the worms in sequence, and totally.

**Day 67**, time between birth > 12 hours.

**Day 70** extreme gut kill, worm smell, chlorine and peroxide smell in nose, immune system kicking back? Leg wiggles, L3?, dosed Alinia 3PM

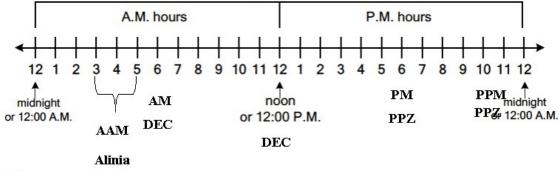
#### **Day 72**

<u>Day 72</u> took Alinia 3AM, DEC at 6AM, Noon, Awoke to huge chlorine peroxide right sinus, old people smell - killing of worms, worms deep in sinus came out to feed while I slept, immune system now fully working, worms cannot feed, they are dying. The entire escapade was depressed immune from fluke infection, that allowed Ascaris infection to go wild. Expect Alinia will take me to the finish line.

<u>Day 73</u> dead worm smell, Alinia appears to work best 3-6 AM, Pill slides into pharynx pouch. May slow absorption and go directly into blood stream. Sub-lingual Alinia may work better than taking oral.

Pills sticking in throat, Left arm circulation collapse, stench of dying worms is sickening Start drinking Tomato Juice –more vitamin C.

#### 24-Hour Timeline



**DAY 70** 

**Day 75** 

**Day 75** old person stench, dead worms in nose, clearing sinus, air burns as it enters the nose. Neural recovery, healing.

**Day 76** sinus clear day2, sinus clearing deeper, worm in throat/palate not moving. Smell subsiding, healing pain in knees? Back, knuckles, etc. Neural issues subsiding. Light ache back of neck. Light gut nausea, Urine flow better. Vision good. Slept, Large sinus worm causes brain stem shaking at wake-up. Smell of dying worm (Alinia?) no babies, only a matter of time.

Day # 8 of Alinia AAM. Working Day 77 Senses coming back, sleeping 10 hours

**Day 78** single dose piperazine 9 pm, no Alinia.

**Day 79** dose AAM alinia, single dose DEC 6pm, Transition to herbs in process, Stay on Vitamin C. **Day 80** take probiotic 225, discontinue alinia, no babies for days. See if it holds. Can see traffic lights ½ mile away.

**Day 82** no symptoms, brain healing

Day 84 Drank Booze, took PPZ PPM at bedtime

Day 85 Worm latched on to back of brain, huge pain. Took 1 Alinia 400mg AAM, Took 1 DEC AM, bad gut hip ache all day, black tiny carroway seeds in urine, tiny filaments floating in bowl. No energy, body in pain.

**Day 86** Drank coffee. Good day, Dosed PPZ at PPM at bedtime, Dosed Flagyl 400at AAM, Dose single DEC 400at AM, If Alinia worked post fluke kill, maybe Flagyl will kill as well. Testing Flagyl for 3 days. Urine output increasing. Body pain decreasing. Sense to touch improving all over body, fewer dead spots. Can see 50 feet brick joints.

**Day 87** Drank coffee. Good day dosed PPZ at 6pm, Alinia at bedtime, 5AM burst of sac in lung, smell of dead worms in fart, 8 AM GERD babies, Dosed 1 std 750 mg PPZ, ½ DEC 200mg, 1 STD IVM.

Alternating Flagyl and Alinia seams to have killed adults, wor sac cyst in lung was unexpected. The large quanity of fluid kind of indicates at it for a while, the massive lung pain a few days ago must have been a small birth?

Back in Kill babies mode for 48 hours, will reorder stock, since out of almost everything.

Noon, full dose PPZ, 200mg DEC, Std dose IVM in Greek Yogurt

**Day 88** Dosed PPZ at 6pm, Dosed Alinia at 9pm, slept, Smell dead worms. Woke 1AM, Flem in lungs, fluid, no acid. dosed PPZ and ½ dec. 5AM-830AM slept, dosed ½ dec.

**Day 89** Drank coffee. Dead worm smell till noon. Vision Very Very Good. So much rest, spent most of day with healing brain. Dosed PPZ at 6PM. Worm in left lung, worm behind voice box. Dosed Alinia at 6 PM to try to move kill earlier into evening. Rough night, Woke at 2AM, Dosed PPZ, Acid Reflux Lung, ½ DEC (200mg) IVM std dose @430AM. Nose worm burn. ½ DEC AM.

#### **Day 90**

**Day 90**, Appears that last few days worms hanging exclusivly around lung. Bursts of GERD and acid is worm attempt to multiply. No worms in other locations. Worms are exclusivly trying to multiply by making sac of babies that burst. Dead worm smell AM, Coffee AM. ½ DEC AM. Quiet Day. PM TBS PPZ, Alinia 9 PM, Slept to 2 AM. Dosed ½ DEC. Flow of non acid fluid from lung. No bites or wiggles. AM ½ DEC. Worm migrated below sinus, above palate. Virtually no energy,

heading back to lung? No tremor for days, Do not know if any males in sinus are alive at this point. Feel as if it is only a matter of days at this point.

PM Tbs Piperazine 6 PM, Alina at 9 PM, ½ DEC at 2 AM, ½ DEC AM. Lung pain less, Worms slowing down.

I started Alinia. It has been 2 weeks since I started, 6-8 weeks since I finished Praziquantel. I stayed on Prazi for 3-4 weeks after they were dead, just to make sure they were dead.

Then I started Alinia for 18 pills worth, I skipped 7 days, worms came back into nose. I take Alinia at 9 PM. I take Piperazine at 4-6 pm,

If there is GERD, I dose Piperazine, IVM, and 200 mg of DEC. I take 200mg of DEC at 6-9AM. Good all day.

Last 4 days, worms don't move from lung, 4 days IVM. When the worms GERD continuous, they are almost dead.

Their last gasp I hope.

#### Q&A

**Q>** How are you doing now?

A> It has been over a month since I finished killing Flukes.

I took on Ascaris.

I took 9 days of Alinia, @ 500mg 9PM, which is 1/4 the patent documented dose of 15mg/kg/D to kill <u>Ascaris</u> (90%).

I then ran out of Alinia. I had Flagyl. I took 400mg spaced at 12 hours (6 AM, 6PM) for 3 days. This is 4X the CDC dose of 100mg. I weigh 113 kgs.

Ascaris influence on the body is gone.

Is it painful?

Well Flagyl in the body kills deep, the first 2 days were large GI reaction until I learned ... and ate food with the Mebendazole. As usual the French Drugs work. The CDC level is all wet, and it is left to the user to figure this stuff out.

I started recovery about 2 weeks ago. After the Alinia. Getting on my feet and working took quite a toll on me. Actual physical work after being sick for 3 years really brings the amount of physical decline into focus. The nerves, muscles, and entire body and back bones ache. I climb stairs, 3 stories, 10 times per day. The last areas the worms were defending, center chest bone, right hip, anus, skull, were holdout positions for the worms. Ache seams to be gone, and is replaced with the pain of making the body work again. Quite a rehab is in store for me.

I am taking no supplements now, just to make sure I do not feed the <u>Ascaris</u> worm, that seams to feed on everything. Anyway Liver is recovering, and kidneys working without using any supplement to increase urine output. I continue to use pulsed Algae, and distilled water, to eliminate toxins. I still am doing a high vitamin C juice habit. I plan ALA, and Algae after a period of time to verify the worms are gone.

I had my doubts, but after 3 days of Flagyl, I can say I have no symptoms of Ascaris. I tested them with a -OH liquid, and absolutely no ill effects.

I may add one more day (total of 4 days). I will have to say, that the adverse reaction seams to be more of parasite reaction making one feel ill, and hurt, than one of the med itself. I cannot say it hurt the body in any way. I was in lots of pain, but it appears I did not hurt the organs. I am really busted up inside from all the worm damage, so I really do not want to do any more injury to my kidneys. It appears that high dose, 3 day did not cause any additional injury to the body.

The documented Flagyl dose duration is 3 days, I was skeptical, but it seams to work now.

I tried **Alinia** 3X before, **Flagy!** 3X before. Nothing worked until I killed the flukes first. Now everything seams too work.

I now have hope for the future.

Thanks to all who helped me on my journey.

I now enter the recovery phase of my strength and memory, which will be a large challenge.

I am happy to be able to say Ascaris are behind me.

I have to wait 8 more months to test their death with the Blood FLISA Ascaris test.

Mattk3

**Day 93** ran out of Alinia Sub Mebendazole

**Day 94** huge smell stool, killed good gut bacteria at 2. ¼ inch black nasal worm. Alinia order on its way. Suspect Mebendazole needs probiotic 225at 6 hours later? Space 12 hours a part, 100mg X2 is optimal, interesting but not a star, put comments under MBZ in Shopping, and move on to the new 15mg/kg 25mg/kg/D Alinia formula.

Day 94, Back ache, problem working, muscles, Lumbar, Ass, Knees hurt,

**Day 95**, Smelly stool from Flagyl and MBZ pulse 3 days. , Die off of good bacteria, dosing MEB 1x 4 hoours is no good seperation, huge die off, gas gone in 24 hours, need to seperate MBZ by 12 hours? , 24 hours?

Need 2, dead 1/4 worm black streek out sinus.

Mucus flow huge, Last night day off of dosing Alinia or MEB Slept till 5 AM

#### Q&A

Q:> Great! Glad to hear you are doing better, How are you really doing? Does it work?

A:>How are you?
The crazy email traffic...

I seem to be in ok liver zone, kidneys recovering weeks later, finally pee is colored dark, Muscles and bones in recovery Wow back and veins in ass real painful, ouch, ouch, ouch

Walking and moving, retraining the body from all the worm invasion Memory still not good.

Guess this is better than expected recovery Will take real sweat to rebuild capillaries.

#### Q&A:

Q: Maybe I should Take Alinia, I have heard it is good.

Q:>Oh wow, should i order it? Ive heard alinia is awesome for <u>Tapeworms</u> and giardia, crypto also.

I have 500 mg tablet.

http://www.meds.com.mx/product\_info.php/products\_id/1779

I take 1 tablet at 6-9 pm. roundworms inc. large come out and feed mostly at 1-3 AM, My worms now feed at 5AM.

- I still have several that have escaped my rounds.
- I take 9 days on, 1-2 days off, I bought 100.
- .....g... = = ..., = = ..., = ..., = ....g...
- I tried high dose when I had flukes, it did nothing.
- I tried low dose and in 2 days sinuses were clear.
- Now that the **flukes** are gone, it works.

It really cleared out my tissues. Still have bone worms, hips, and nests. Maybe 1 large one left, several smaller.

Mucus is starting out of lung, and sinus. Was birth a lot, after starting alinia, the worms went onto reproduction overtime. Head worms gone, foot worms gone, muscles clear, liver healing, kidneys still working, dumping dark urine for first time in 3 years, they filter again!

I take 9 days of <u>Alinia</u>, I take 2 Mebendazole (Flagyl) seperated by 12 hours, and a day off. This is the first day off in the pattern since I do not know when.

I just got more Alinia. I am going up to the dose specified in the patent, 15 mg/kg/D.

I will hold this for I guess 9-10 days, and repeat the cycle 2-3 times to try to take out the remaining adults.

While the worms were dying, they constantly gave birth in lungs, I dosed IVM, DEC, PPZ.

The discharges from the worms are now clear, my lungs and sinuses are clearing. The worm discharge must be free of babies, or the low dose DEC, PPZ hold the babies in check. The Mebendazole kills good bacteria, so a probiotic after, on the day off, is a good idea.

My stomach ache after Mebendazole, single pill 2X/D at 12 hour space, really rips up the gut, huge kill of good bacteria, bad stool smell, ache in stomach. It is the ache in the stomach that leads me to believe the Ascaris burning fluid sac;s that burst in the lung, lead to a population into the stomach. It is in the killing in the stomach, by MBZ, Flagyl, that I believe the killing of Ascaris lie.

I will graph more in the document, next revision.

**Day 98**, finished 3 day **flagy!** for 2x per day, seperated 12 days. Started final pass of Alina 9 days 15mg/kg/D and **flagy!** on days 4, 5, 6

>Dosed some Algae

Should I take any DEC or PPZ? Is the question....

**Day99** headache no energy, pm fluid balls up throat, fluid up lungs no burn, chlorine in back of sinus 2am, some odor

**Day 100**, Skull ache, eyes want to pop, sinus pressure, Took 1 Flagyl at noon, still doing Alinia @ 1500mg/D for 113KGS. Day 3 of the plan, decided to try a MBZ early, since it can be used as a test to see if there are any births in gut from sinus lung drainage.

Result: No killing in GI, reproduction loop is broken using Alinia and Flagyl. The exact sequence I used, singular Alinia, Singular Flagyl, Now MBZ inside Alinia zone seams to be plausable.

No real wiggles excepting chlorine sinus @ 2AM. Think last worm may be in the skull. Slept a lot last night.

**Day 101** add 400mg **dec** aam or am ascaris formula 3-111 to date, what I have been taking. 3 **alinia** 500, pm, ppm, am – iron (111) **Flagy!** 400mg 12 hr sep, 2am, 2pm - carbs **DEC** 400mg 2am – 9am for malting

**<u>Day 105</u>** massive smelly toxin dump soft stool, <u>Alinia</u> at 20 mg/kg/D for second day. Symptom free, High level of chlorine smell. Staying horizontal all day. Working and Lifting still has difficulty.

<u>Day 107</u>, still some small population, wake up with short ache in stomach. After 2 days of 10-12 hours sleep mild vibration, neurons still healing. Running out of <u>Alinia</u>, backed down to 15mg/kg/D. Circulation issues, cold hands, etc still remain.

**Day 111**. smelled iodine last night. Down to 6AM, 6 PM Alinia and 200mg DEC.

Semi active day, Cocoa not Coffee. Headache PM,

took B50,

Chlorella,

Spirulina.

Back of neck, Base of skull, Back of brain, eye splitting, Suspect reconnecting neurons causing excessive blood flow to head, very hot, cold arms, cold feet. Could be circulation imbalance, continue to wait on horse chestnut and ginkgo during the final kill, all vitamins for that matter, due to concern they may feed Ascaris.

9pm alinia

10 pm dec 400

- 2am headache gone
- left lung rib ache
- need dec 200?
- 15mg/kg/d <u>alinia</u>
- · horse chestnut for leaky veins

2am lung birth take **piperazine** 8 am nose birth tiny worms take alinia almost gone

maybe can use 25 to 15mg kg d drop to sync mbz feeding

11 runny nose burn throat **DEC** 400 IVM?

#### Day 114,

Rt sinus and lung keep birthing AAM, feels like post nasal drip, babies giving birth to babies, Adults gone. Key is to put **DEC** 400 and Piperazine 750 at 6 PM, <u>6 AM to stop any acid fluid baby stream from multiplying in GI</u>.

On Jan 23, 2016, at 4:38 PM, "Matt Kaltenbach" < mattk3@fuse.net > wrote:

I did a run of 9 days 1 pill per day
I ran out. It took a week to get more, I ordered 56 pills. I was shorted 8 pills on the order.
I did a run of 4 days at 3 pills per day
I did a run of 4 days at 4 pills per day
I did a run of 2 days at 5 pills per day
Then 2 days of 3 pills
2 days of 2 pills
1 day of 1 pill
I ran out.

I have on order MBZ (I was out)
I have on order Alinia (140-160 pills?)
I received Piperazine, Ivermectin
I have Flagyl, DEC.

I have a few babies left, not much else.
I never ran a strait line, in that my research did not show dose.
Truth is it, they stopped giving birth at a number closer to 25 mg/kg/D I found tests on animals at 50mg/kg/D and 100mg/kg/D

I am going to do an Alinia run at 25mg/kg/D with DEC 400mg, Piperazine 750mg, and MBZ on days 4, 5, 6 at 400-600 mg.

Then I will do a low dose Alinia for a couple of more weeks.

1/24/16 final update

For the last few weeks I have sent out updates on Alinia, Test doses, Results.

This is an update.
I have completed two short test runs,
The first using low dose Alinia with Piperazine citrate and DEC,
The second using a ramp of Alinia dose without Piperazine Citrate and DEC.
>Piperazine citrate and DEC are intended to kill eggs and birth larvae in blood, lungs and sinus.
> Alinia is intended to kill adults L3, L4 Ascaris.
It appears that all the adult Ascaris are now dead, even using my two preliminary Alinia trials.
It appears that babies are birthing babies, I wake up with sinus drip, and lung secretions while I sleep.
It now appears that both simultaneous dosing of DEC, Piperazine Citrate, and Alinia will be required to kill the rapidly reproducing Ascaris offspring.
I have backordered Vermox, and Alinia on the way for a final run.
It appears that stopping babies in their track is essential to winning the battle.
Either 100mg MBZ at 6 AM, or dosing a second Piperazine and DEC at 6 AM will be required to break the loop.
Breaking the baby loop is essential to winning the battle with Ascaris.
Several people have reported this never ending GERD, Acid Reflux, birthing situation.
I weigh 113 KGS, and I dose 400mg DEC at 6PM, 6 AM, and 750 mg Piperazine citrate at 6 PM, 6 AM.

I will try 100 mg of MBZ at 6 AM to break the baby loop, which should provide final victory against my roundworm infection.

Sorry this takes so long, but the data the test process provides is very valuable.

Hope this email finds you all on the path to recovery

١

Matt

Matt Kaltenbach

**USA** 

Mattk3@fuse.net (Personal)

Mattk3 at the CureZone

PS, The document is still being updated, a final release should appear shortly.

To: Matt

**Subject:** Re: The final solution

Mattk,

Hi mattk, how are you?

Sleeping, Large worm free, a few straglers, cold hands, learning how to move the body, getting my

well I am starting to get unpacked and everything is at new place, prolly take 1 more week to get everything unboxed and start too get my plan ready , togo on the attack.

so what happened with the user name & email address thing?

A large portion, like 50%, wanted privacy. There are still sourcing problems in various countries.

#### I never published your hopeful message to the group, you never gave me permission.

so I did have to stop my course of Alba , and the worms are back full force & piss'd off! omg I blew out so many from nose tonite they filled my palm of hand was pretty yuky, so far the only way i can get them out of nose is to use a 50/50 mix water & peroxide and pour 1/2 tsp up nose and let soak for couple minutes, the they die! and can rinse out with regular warm water and they come out, look like the 1/4in white spagehtti worms (females?) and the 1/4in white cotton threads (males?) the most times are smaller the biggest ones are about 1/4 in, that doctor said he used a sniff of ethanol

vapor too knock them unconscious and remove live ones like that, the peroxide kills them dead! they are all over me know I can see white threads in my skin most every where I look know, either just under the skin or in the skin and i also see squiggly line tracks on skin I believe from the Larve crawling across skin, i'll have to cut it open and try too pull one out, that'll tell the tale. they have severely infested my ears know also , my ears look real bad with white threads all over and red bumps marks most likely were the Larve is going back into me thru skin.

Are you taking DEC every day?

Are you still on Piperazine every day?

Alinia is the most powerful, and main roundworm knockout med. I have come through 2 courses, the first course of 9 days I did at 500mg with Piperazine and DEC. I followed that with 3 days of Flagyl. I did no meds for 6 days, worms returned.

The second coarse of Alinia I did @ 15mg/kg/D to start, Did 3 days of 200mg of Vermox at days 4, 5, 6, and then ramped Alinia to 20 mg/kg/D for a few days, then ramped to 25mg/kg/D. Worms ache, fade, die. Without DEC they seam to have a few babies through, but mostly they birth clear non-acid fluid, which I assume have no viable larvae. They went nuts trying to make babies, and I found at 15mg/kg/D they were trying to reproduce like mad. At 20 mg/kg/D they were kind of unable to move, and gave little birth. At 25mg/kg/D there were virtually no worm symptoms at all, and in less than 3 days. After 3 days at this level, with virtually no side effects, I had some GI discomfort, leading me to believe the correct dose may be in the 20 mg/kg area. I have virtually run out of meds (all of them), my order is still being processed, they were out of Vermox I guess.

I may make a seperate order for just Alinia, and order twice as much to have stock. I am very near the finish, so I am trying to figure out how much I really need.

Alinia is a designer drug, with so few side effects, and such a benifit to the gentle killing, that it is a huge keeper, by itself, or in a coctail, Alinia has turned out to be the biggest player on the table.

I rank the coctail as follows:

- 1) Alinia stops worms from absorbing and processing Iron
- 2) Vermox stops worms from processing Carbohydrates
- 3) DEC stops Larvae from Malting (sheding their skin)
- 4) (not using for a month) Piperazine, kills birth, eggs, larvae, and processes nitrogen uric acid for removal

I have one final course of the formula planned, in an attempt to kill Ascaris, the toughest worm on the planet to kill.

My plan is:

Alinia 20 mg/kg/D for 9-14 days Vermox 400 - 600 mg a day for 3 days, at days 4, 5, 6 DEC 400 mg at 6 AM Piperazine citrate 750 mg at 9PM

There you have it. The final formula. I have had emails from folks that take Vermox yearly to keep Ascaris in check for life. I am unsure I will have to do this, my goal is to kill the Cysts, eggs, Adults, L4, L3, L2, L1 larvae, and the eggs. Ascaris is one of the most complex life cycle worms one can get. The formula is a bit complex, but I have a high degree of confidence that this final formula will wipe out the systemic infection once and for all. While I am 99.5% better, I am still looking for 100%.

I still have to order Alinia & Vermox & Flagyl how much did you end up needing ? 30 days? 60days?

Given your state, having visual migrans, you will need to do the formula from the beginning, need to have prolonged doses of DEC and Piperazine, And plan on at least 3 courses of the final formula.

I would interject PZQ course, you may or may not need, for 21 days at 25mg/kg/d with 50,000 IU of D3, and a lot of calcium. If you kill flukes, you will need another month of PZQ to make sure they are wiped out of your body. My fluke kill seams to have worked, I bought 300 grams of PZQ, but I had several false starts, and I weigh 238 lbs. I am guessing this preventive phase will take you some 150 - 200 grams, and it cost about a dollar per gram. This PZQ kills flat worms of all types, and is necessary to pre-treat for flatworms prior to killing roundworms.

First things first, we need to stop migrans, that means 2 doses of DEC per day, say 6 am, 6 pm. Piperazine at 9pm(bed time) is critical. Piperazine additional dose (1 TBS) can be taken anytime.

Expect to need 10 mg/kg/D of Albendazole for 45 days. Expect to need 20 mg/kg/D of Alinia, for 45 days

If you buy a couple of bottles of DEC 100ct for \$10 ea that should due.

If you buy 20 + bottles of Piperazine Citrate (2.79 - 4.00 ea) from Drs Foster and Smith, you should be set for most of the trial. Expect to use more upfront, less to none towards the end.

# <u>Taking IVM and ALA together causes a dramatic reduction in brain Fog</u>

hi friedegg,

You are the first person to offer any ideas on Ascaris.

Early on I tried several black nigra, pepper, oil. I also tried every tumeric/curcumin/nano/lipid

I found Natures bounty tumeric curcumin 450mg was the best, but I rated it at 2%. This was way before I found piperazine/ and now Alinia, or id/d the fluke infection.

Killing the larvae is easy, Killing the adults is hard. I think my adults are no longer producing babies, and they just kind of move and lay there, no real energy. It is taking way to long. I tried Alinia 3times, to no avail. Now that flukes are gone, I see such a great improvement, I would call them almost frozen.

I have about 12 day supply left of Alinia, do not know where I jump after that. I may want to try your combination tricks, I need the last adult or two to go.

I have a crate full of stuff to retry and ponder my next approach if Alinia does not deal them a knockout punch. I will take your information seriously, since it sounds like the edge I am looking for.

My senses, smell, feeling, and brain are all in massive recovery mode. I am just now feeling the pain the worm nests have put into my body. This experience is like a train wreck in slow motion. I cannot believe how much effort is required to kill these guys. Thanks for the heads up.

Matt

hi mattk3. was reading your idea on Ascaris and how they use enzymes to avoid or alter our immune response.

upon looking up the mechanism of action of dec and that it inhibits the synthesis of arachnid acid --- which decloaks the worm so our immune system can then kill.

I wanted to let you know that for 2 days of using organic turmeric with black pepper which enhances the active medicinal ingrediant in turmeric curcumin. energy levels,pain,brain fog and happiness came back to my body.

- >Piperine has been shown to increase Tumeric curcumin effect like 100X
- >SOD increases the NO burst that helps immune system kill invaders.

the parasites figured out in 48 hours how to negate the benefit of turmeric.

turmeric with blackpepper I believe inhibited the worms ability to produce these enzymes by inhibiting that arachnid acid they need.

holy basil which contains ursolic acid I believe and also boswhelia extact which contains its own acids and also ursolic acid which bodybuilders are using might inhibit the arachnid acid and slow down the worms ability to make enymes.

> I have holy thistle, and several others I have yet to document, which I also tried to no avail. I have so many things to retest, I will add these to the list.

if it worked like turmeric did there may be hope as well as using your plan as well.

the more things the better to throw. them off guard

#### Q&A

**Q>**If I stay on my current regime this should eventually clear the gi as well?

A≥ Once you hit a tipping point, the anti-parasitics seam to work. I do not fully understand what I could have done differently. I wrote it down to help others in the same dire condition I was in. It is part magic, part daily routine, part knowledge in what each piece does, and how much to take. I wrote down each formula, and never followed it exactly, it was always a little more of this, a little less of that, daily. I wrote down the range, to reflect what I was doing. I think moving the body over a mountain, takes a constant attention to good in, bad out. I knew when I saw the explosion of black, I had moved my body to the peak of the mountain. I suspect it is all down hill from here. I hope to be back to work shortly, a true vindication of the sequence, approach, and elements. I did it without LEM, which I tried earlier, but just did not do enough. I also underestimated the power of OJ, but with the fear of all that natural sugar, and my bouts with SIBO and SIYO, I never felt the risk was justified. I also was afraid that more vitamin C would make the loose stool looser. It may not have been a justified fear. I guess I will never know if there was a more direct pathway, I was just trying to keep the car on the course, and not wreck the car. Me.

#### **Q>** My colon is locked again.

<u>A></u> Keep Spirulina at 9 grams, Keep <u>Chlorella</u> at 2 - 2.5 grams. Eventually your body will get to the top of the mountain.

They fight back with toxins. Try crushing cloves garlic, mix it with yogurt, and swallow in less than a few minutes, the compounds in **Garlic** only works for 10 minutes.

### **TAPER PLAN – first try**

<u>Distilled water</u> is very important when you want to normalize the body after discontinuing heavy vitamin dosing.

I guess a month of continued anti-parasitics to mop up any Ascaris eggs/cysts. **Piperazine** taper?

Attempts to remove Supplement dosing, starting a week after the large parasite kill, after 3 weeks, a maximum of 2 days off can be tolerated.

I think it is best to keep a months supply on hand, in case they pop up again.

During the second month, Dosing of **ALB, DEC, and Piperazine** were moved to AM and Noon, **Praziquantel** was moved to PM and PPM.

#### P>I forgot to mention that some times the tremors are worse than at other times.

Take **Piperazine citrate**, and later **DEC** with ALB

This migration of worms through the base of the brain causes tremor. The worms are looking to get into CNF fluid to detox, or go to the brain to feed.

In either case, taking Piperazine just before bed, and just as you wake, keeps them from feeding, making them hungry.

I am trying **DEC and ALB** at 1-3AM and at noon.

Piperazine makes them hungry, **DEC** and **ALB** give them the juice as they start to feed.

- 1) if you have flukes, you cannot kill Ascaris. That is why Praziquantel.
- 2) Ascaris feed at night, mostly, into the AM. <u>Piperazine</u> makes them not feed, get hungry, <u>ALB</u> and <u>DEC</u> kills them, slowly, at 8 years old, less that 2 weeks, at 60 it takes 6 months. Immune system dominates the equation.

I originally took everything at once, alb, **dec**, ppz, pzq, to kill slowly, that worked.

Now I am free of **flukes** at day 55, so I stopped **PZQ**. I am now moving **ALB, DEC,** to 6 PM, Noon, with **PPZ** at bedtime, and 3-4 AM. I keep playing with this, but I now think flat dosing will kill Ascaris faster, that is 6 pm, 9pm, 1-3AM 6-8 AM Noon. Which ones I take, and when is the question.

When you dose **PPZ**, and a worm is feeding from your blood, it gets very sick and burns for days. When It returns to feeding, it is weaker. Give them a snoot full of **DEC**, they belly ache for hours.

Once you reduce their numbers, you can just tell.

I would do the plan, by you get to the second 30 days of anti-parasitics, I should have a better plan.

Start with **Zinc**, **Dec**, etc. If you get bursting think flukes, small Ascaris.

When you finish getting rid of fluke risk, and eye worms, then move on to the full plan, and spread out the anti-ascaris meds.

I however, some 60 days, still have a few worms left, after destroying hundreds or thousands.

I have heard that it may take 6 months to kill Ascaris on <u>Albendazole</u>. <u>Alinia</u> was supposed to work in weeks, I have tried this twice. My body is still recovering from the massive doses of stuff. I stopped <u>PZQ</u>, my flukes are gone. I still sense an enzyme emission, and some brief booze smells, leading me to believe there is more yet to discover.

I suspect <u>Pineapple</u> enzymes, or such may be the magic ingredient to bring the whole formula into focus.

I am more than 99% better. But I know, every last one must die.

I released revision 110 on the CureZone.

I am going to go deeper into the enzyme relationships of Ascaris, before I can put this one to bed. The movement of Piperazine to last in day, first in day (3AM-6AM) has been a revelation. Ascaris are nocturnal.

The movement of <u>ALB</u> and <u>DEC</u> to 9AM-Noon and 6PM also is showing effectiveness, just not enough.

Enzymes are released by the worm, when they clearly ingest ALB and DEC. Rotting fruit is what I smell.

It is what is in the worm digestive tract that alters ours, and it must be in their enzymes where the answer lies.

### **HAIR Regrowth plan**

I found a herb to regrow hair. I was a hairy guy, now I look like a cue ball. http://www.maxnature.com/oxtati.html

#### **Maintenance**

This is the initial formula, that is being tested. This formula is preliminary and still under development. It is intended that this repair and immune system formula be maintained for 7 years.

I will be doing a shutdown formula (liver repair, brain repair, etc)

I am currently working on kidneys, as they were bad years ago before I did this formula, and it needs to be made stronger. The use of KGP flush on a continuous basis is not a good idea, in that it depletes certain substances. It will be replaced with a mixture of herbs and substances on a regular an or periodic basis.

Currently I am pulsing (skipping every other day) the following supplement list, to determine if anything in the formula feeds the worms. Judging by the continued dead worm smell, I believe everything but ALA is acceptable. ALA is essential to healing the brain. Combination of ALA and IVM seams to show no side effects for Ascaris. It may be possible to develop natural anti parasitics that enable the use of ALA.

#### **AM Pulse**

1 CQ10 400mg AM

1 Glass OJ

(0-2) Ester C

(2) LEM extract

Rosemary for memory (TBD)

1-2 Dropper full KGP flush for kidney function in juice

0-2 Dr Christophers Kidney formula? TBD

1-2 Milk Thistle

- (1) B50 complex for enzymes
- (2) **Ginkgo** for brain capillaries PM
- (0) low dose **ALA**? AM Pulse 4 every 4 days?

#### **PM Pulse**

1 glass pineapple or pineapple juice

(4) Spirulina caps 500 mg (2Grams total) PM

(2) Chlorella

(0-1) Source of life

(1) Borage oil GLA PM

(0-1-2) GuAIAid PM

(0-1-2) Dodder Seed PM

Anti-parasitics if you are still in taper mode

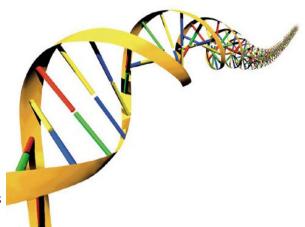
### **DNA and DNA Repair Primer**

www.yourgenome.org

I will be doing a 7 year no cancer formula, to cleanup/mopop, and fix DNA using the 4 repair molecules I researched some 3 years ago. Need to look them up.

The DNA Instruction Set

DNA is in every cell of your body, the nucleus contains DNA, the instruction set for your body. The information in the DNA can be used for many purposes. Tiny building blocks called cells make up your entire body. There is DNA in nearly every cell, within a sack called the



nucleus. The nucleus is like the brain of the cell. DNA is an endless march of characters in a 4-letter alphabet. If you pull it apart ( the double helix), you would see the exposed ends of the strands, thefour different chemicals in the air. Those four chemicals, called "bases" carry the information used to make a body and to keep it running.

Scientists named each of the four bases with a letter, G, A, T or C. All of the letters in one cell make up the human genome, a complete instruction set for making a person. If you wrote down all of the bases in one cell, you could fill a 1,000 phone books with A's, T's, G's and C's.

Scientists are working on reading and manipulating the information in DNA. DNA is constantly subject to mutations, accidental changes in its code. Mutations can lead to missing or malformed proteins, and that can lead to disease.

We all start out our lives with some mutations. These mutations inherited from your parents are called germ-line mutations. However, you can also acquire mutations during your lifetime. Some mutations happen during cell division, when DNA is duplicated. Still other mutations are caused when DNA gets damaged by viruses.

Few mutations are really bad for you. In fact, some mutations can be beneficial. Many mutations have no effect, these are called silent mutations.

The mutations we hear about most often are the ones that cause disease. Diseases can be caused by just one copy of a single defective gene. Thanks to probability and chemistry, the body has a number of processes that weed out cells with defective genetic diseases over time. Cells renew themselves quite often, and therefore some diseases can be reversed if the weakened cell dies, and does not replicate. Sometimes it takes a little help to get a cell to die before reproducing.

Scientists estimate that every one of us has between 5 and 10 potentially deadly mutations in our genes-the good news is that because there's usually only one copy of the bad gene, these diseases don't multiply.

Many of the worst diseases around are caused by glitches in our genes, and the therapies for these diseases often involve a lifetime of drugs (and their nasty side effects) that help but don't really solve the problem. Wouldn't it be great if you could just "fix" this faulty DNA? DNA can be damaged by many sorts of mutagens, which change the DNA sequence. Mutagens include oxidizing agents, alkylating agents. oxidants such as free radicals or hydrogen peroxide produce multiple forms of damage, including base modifications, particularly of guanosine, and double-strand breaks. [64] A typical human cell contains about 150,000 bases that have suffered oxidative damage. [65] Of these oxidative lesions, the most dangerous are double-strand breaks, as these are difficult to repair and can produce point mutations, insertions and deletions from the DNA sequence, as well as chromosomal translocations. Many mutagens fit into the space between two adjacent base pairs, this is called intercalation. Most intercalators are aromatic and planar molecules; examples include ethidium bromide, acridines, daunomycin, and doxorubicin. In order for an intercalator to fit between base pairs, the bases must separate, distorting the DNA strands by unwinding of the double helix. This inhibits both transcription and DNA replication, causing toxicity and mutations.[67] As a result, DNA intercalators may be carcinogens, and in the case of thalidomide, a teratogen.[68] Others such as benzo[a]pyrene diol epoxide and aflatoxin form DNA adducts which induce errors in replication.[69

To interpret the DNA code, the cell first makes a copy of the DNA segment to be read. The copy then travels to another part of the cell, where the code is used to assemble a chain of protein subunits. RNA has a different sugar in its sugar-phosphate backbone; it uses ribose, rather than deoxyribose.

RNA also uses a different base from the thymine (T) that DNA uses; The four bases found in DNA are adenine (abbreviated A), cytosine (C), guanine (G) and thymine (T). These four bases are attached to the sugar/phosphate to form the complete nucleotide, as shown for adenosine monophosphate.

The enzyme RNA polymerase makes the RNA copy, recognizing the 'start here' and 'stop here' signals that appear in the DNA code. It uses available bases, sugars and phosphate molecules. The RNA molecule that is made - called 'messenger RNA' (mRNA) - then carries its 'message' out of the nucleus to the outer part of the cell (the cytoplasm). The mRNA passes through the pores in the nuclear membrane, and makes its way to cellular components called the rough endoplasmic reticulum (ER), where proteins are made.

At the ribosomes, the mRNA is used as a template for assembling a protein molecule from its building blocks (amino acids). This process is called translation.

There are 20 different types of amino acids; biologists have given each a code letter. For example, M is methionine, L is leucine, F is phenylalanine (because P is proline). Translation at the ribosomes is very similar to translating from one language to another. In this case, the translation is from the four-letter language of DNA into the 20-letter language of proteins.

The DNA code is read three letters at a time: these DNA triplets are called codons. Since there are four different RNA letters (A, G, C and U), there are  $4 \times 4 \times 4 = 64$  different codon combinations. Most of the codons correspond to a specific amino acid. However, as there are only 20 different types of amino acid, some of the 64 codons code for the same amino acid. Three of the codons are used as 'stop' signals - telling the cell to end the transcript checksum.

Replication the DNA sequence is copied into a complementary RNA sequence. Cell division is essential for an organism to grow, but, when a cell divides, it must replicate the DNA in its genome so that the two daughter cells have the same genetic information as their parent. Helicases are proteins that are a type of molecular motor. They use the chemical energy in nucleoside triphosphates, predominantly ATP, to break hydrogen bonds between bases and unwind the DNA double helix into single strands.[97] These enzymes are essential for most processes where enzymes need to access the DNA bases. In DNA replication, a DNA-dependent DNA polymerase makes a copy of a DNA sequence. Accuracy is vital in this process, so many of these polymerases have a proofreading activity. Here, the polymerase recognizes the occasional mistakes in the synthesis reaction by the lack of base pairing between the mismatched nucleotides. If a mismatch is detected, a 3' to 5' exonuclease activity is activated and the incorrect base removed.[99] In most organisms, DNA polymerases function in a large complex called the replisome that contains multiple accessory subunits, such as the DNA clamp or helicases.[100]

RNA-dependent DNA polymerases are a specialized class of polymerases that copy the sequence of an RNA strand into DNA. They include reverse transcriptase, which is a viral enzyme involved in the infection of cells by retroviruses, and telomerase, which is required for the replication of telomeres. [44][101] Telomerase is an unusual polymerase because it contains its own RNA template as part of its structure.[45]

Transcription is carried out by a DNA-dependent RNA polymerase that copies the sequence of a DNA strand into RNA. To begin transcribing a gene, the RNA polymerase binds to a sequence of DNA called a promoter and separates the DNA strands. It then copies the gene sequence into a messenger RNA transcript until it reaches a region of DNA called the terminator, where it halts and detaches from the DNA. As with human DNA-dependent DNA polymerases, RNA polymerase II, the enzyme that transcribes most of the genes in the human genome, operates as part of a large protein complex with multiple regulatory and accessory subunits.[102]

To kill a cell this checksum must = defec protien 84 generated, cell dies. There are two ways in which cells die:

- •They are killed by injurious agents.
- •They are induced to commit suicide.

### **Cell Death by suicide**

Cells that are induced to commit suicide:

- shrink;
- develop bubble-like blebs on their surface;
- have the chromatin (DNA and protein) in their nucleus degraded;
- have their mitochondria break down with the release of cytochrome c;
- break into small, membrane-wrapped, fragments;
- release (at least in mammalian cells) ATP and UTP.
- These nucleotides bind to receptors on wandering phagocytic cells like macrophages and dendritic cells and attract them to the dying cells (a "find-me" signal").
- The phospholipid phosphatidylserine, which is normally hidden within the plasma membrane, is exposed on the surface.
- This "eat me and die" signal is bound by other receptors on the phagocytes which then engulf the cell fragments.
- The phagocytic cells secrete cytokines that inhibit inflammation (e.g., IL-10 and TGF-β)

The pattern of events in death by suicide is so orderly that the process is often called programmed cell death or PCD. The cellular machinery of programmed cell death turns out to be as intrinsic to the cell as, say, mitosis.

One of the methods by which cytotoxic T lymphocytes (CTLs) kill virus-infected cells is by inducing apoptosis [diagram of the mechanism]. (And some viruses mount countermeasures to thwart it. Cells respond to DNA damage by increasing their production of p53. Programmed cell death is needed to destroy cells that represent a threat to the integrity of the organism.

There are 3 different mechanisms by which a cell commits suicide by apoptosis.

- 1. One generated by signals arising within the cell;
- 2. Another triggered by death activators binding to receptors at the cell surface:
  - ∘TNF-a
  - Lymphotoxin
  - ∘Fas ligand (FasL)
- 3. A third that may be triggered by dangerous reactive oxygen species.
- Several human papilloma viruses (HPV) have been implicated in causing cervical cancer. One of them produces a protein (E6) that binds and inactivates the apoptosis promoter p53.
- Epstein-Barr Virus (EBV), the cause of mononucleosis and associated with some lymphomas
- produces a protein similar to Bcl-2
- produces another protein that causes the cell to increase its own production of Bcl-2. Both these actions make the cell more resistant to apoptosis (thus enabling a cancer cell to continue to proliferate).

Even cancer cells produced without the participation of viruses may have tricks to avoid apoptosis.

- Some B-cell leukemias and lymphomas express high levels of Bcl-2, thus blocking apoptotic signals they may receive. The high levels result from a translocation of the BCL-2 gene into an enhancer region for antibody production.
- Melanoma (the most dangerous type of skin cancer) cells avoid apoptosis by inhibiting the expression of the gene encoding Apaf-1.
- Some cancer cells, especially lung and colon cancer cells, secrete elevated levels of a soluble "decoy" molecule that binds to FasL, plugging it up so it cannot bind Fas. Thus, cytotoxic T cells (CTL) cannot kill the cancer cells by the mechanism shown above.
- Other cancer cells express high levels of FasL, and can kill any cytotoxic T cells (CTL) that try to kill them because CTL also express Fas (but are protected from their own FasL).

http://users.rcn.com/jkimball.ma.ultranet/BiologyPages/A/Apoptosis.html

http://128.121.104.17/cfs-inform/Virus/krueger.etal01.pdf

P53: A specific protein with a mass of 53 kilodaltons that is produced by a tumor-suppressor gene. Like other tumor-suppressor genes, the p53 gene normally controls cell growth. If p53 is physically lost or functionally inactivated, cells can grow without restraint. Many human tumors have mutations in the gene coding for the p53 protein.

STAT1-alpha and p53 Deficiencies are Found in Patients with Chronic Fatigue Syndrome ( http://www.ncf-net.org/forum/P1-STAT1.htm )

The genetic code is read by 'adaptor' molecules called transfer RNAs (tRNAs). These deliver amino acids to the ribosomes according to the sequence of the mRNA.

Each tRNA molecule is attached specifically to one of the 20 amino acids and three critical bases recognize the complementary codon in the mRNA. As the tRNAs bind and release, the amino acids on adjacent tRNAs are joined to form a growing amino acid chain.

Each codon on the mRNA molecule is read, one at a time. For each codon, the tRNA molecule with the complementary anticodon temporarily binds to the mRNA. The amino acid that is joined to the end of the tRNA molecule is brought in line with the growing polypeptide chain, and the amino acid links to the end of that chain. Once their amino acid is added, the tRNAs disengage from the mRNA molecule, leaving the next codons on the mRNA molecule to be 'read'.

Proteins carry out most of active functions of a cell. From the DNA, we can recognize and read off the amino acids that make up the proteins in our bodies. However, we still only understand a little about how the chain of amino acids becomes a working protein. Proteins have three dimensional (3D) structures, which are difficult to predict just from the DNA sequence.

Many proteins are enzymes; these biological catalysts enable or speed up chemical reactions in the cell. They can act as enzymes because of their unique and complicated 3D shapes (like the structure of insulin at right). Each enzyme has a region into which two or more chemicals fit snugly. This region is called the 'active site'. While the appropriate substances are attached to the active site, they react with each other. The reaction means that they are no longer fitted to the active site, so they vacate it, leaving the enzyme free to catalyse another reaction.

The backbone of the DNA strand is made from alternating phosphate and sugar residues.[10] The sugar in DNA is 2-deoxyribose, which is a pentose (five-carbon) sugar. The sugars are joined together by phosphate groups that form phosphodiester bonds between the third and fifth carbon atoms of adjacent sugar rings.

Sugar influences Phosphate influences Acid instructions

#### No Cancer

I will probably do Spirulina for life. MSM Borage oil Dodder?

Many other cancer researchers, starting over 100 years ago in the 1880s, have isolated the cause of cancer to be microbes, though they did not understand the mechanism inside the cell which caused a microbe to make a cell cancerous.

Today the main mechanism inside the cell which allows microbes to cause cancer is understood. We now know that a microbe which is able to get inside of a normal cell blocks glucose from being used to create pyruvate, which in turn blocks the Citric Acid Cycle and in turn the Electron Transport Chain, both in the mitochondria. Blocking these two chemical chain-reactions cause the number of ATP molecules in a cancer cell to plummet!!

The MSM/D3 Protocol for cancer includes four substances: 1) MSM (Methyl Sulfonyl Methane),

- 2) Vitamin D3
- 3) Colloidal Silver,
- 4) Ionic Magnesium

http://www.cdc.gov/parasites/fasciola/health\_professionals/index.html

### **Fluke Treatment News**

https://en.wikipedia.org/wiki/Flatworm

Alpha-tocopherol has been reported to have beneficial effects in minimizing hepatic and pancreatic damage associated with fluke migration

### <u>Triclabendazole – CDC - Update</u>

<u>Triclabendazole</u> is the drug of choice for treatment of fascioliasis. It is the medication recommended by the World Health Organization. It is not yet widely available to treat people. In the United States, it is not approved by the Food and Drug Administration. However, it is available through CDC, under an investigational protocol.

As with all medications, use of **triclabendazole** should be individualized. It is a benzimidazole compound that is active against immature and adult *Fasciola* parasites. The therapy usually is effective and safe. **Triclabendazole** is given orally, with food, to improve absorption.

The medication comes in scored tablets. The dosage is calculated on the basis of the patient's weight. The typical regimen is a single oral dose of 10 mg of **triclabendazole** per kilogram of body weight (10 mg/kg).

Two-dose (double-dose) **triclabendazole** therapy can be given to patients who have severe or heavy *Fasciola* infections (many parasites) or who did not respond to single-dose therapy. Of note, some experts routinely use 2-dose therapy, which might have a higher response rate, on the basis of limited data.

Two-dose therapy means that the patient is given 2 individual doses of 10 mg/kg, separated in time by 12 to 24 hours. In other words, the patient receives a total dose of 20 mg/kg, given in 2 divided doses, 12 to 24 hours apart.

### **Additional Perspective About Fasciola Therapy**

*Fasciola* serologic testing is one of the means that can be used to assess the response to therapy. Seroreversion (loss of detectable antibodies) usually is noted 6 to 12 months after curative therapy.

On the basis of limited data, **nitazoxanide** might be effective therapy in some patients. The drug is given orally, with food. The dosage regimen for adults is 500 mg po bid (twice a day) for 7 days. **Praziquantel**, which is active against most trematodes (flukes), typically is not active against *Fasciola* parasites. Therefore, **praziquantel** therapy is not recommended for fascioliasis.

### **Vitamin D3 and Praziquantel Discovery**

I have found that Prazi and D3 causes accelerated Death of flukes. I currently have to take some pictures of mine. I then have to research D3 and Prazi, it takes the back of my brain off at D3-50(50,000 IU). Today I tested D3-5 and the pain was lower than yesterday.

I may have found a way to get around the triclabendazole problem, take D3 with Prazi?

**Triclabendazole** kills flukes in 2 days, unknown is how painful it is to the brain.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3780935/

#### **Trematode and Cestode Abstract**

Status and emerging issues in the use of **praziquantel** for treatment of human trematode and cestode infections are briefly reviewed. Since praziquantel was first introduced as a broadspectrum anthelmintic in 1975, innumerable articles describing its successful use in the treatment of the majority of human-infecting trematodes and cestodes have been published. The target trematode and cestode diseases include schistosomiasis, clonorchiasis and opisthorchiasis, paragonimiasis, heterophyidiasis, echinostomiasis, fasciolopsiasis, neodiplostomiasis, gymnophalloidiasis, taeniases, diphyllobothriasis, hymenolepiasis, and cysticercosis. However, *Fasciola hepatica* and *Fasciola gigantica* infections are refractory to **praziquantel**, for which **triclabendazole**, an alternative drug, is necessary.

In addition, larval cestode infections, particularly hydatid disease and sparganosis, are not successfully treated by **praziquantel**. The precise mechanism of action of praziquantel is still poorly understood. There are also emerging problems with praziquantel treatment, which include the appearance of drug resistance in the treatment of *Schistosoma mansoni* and possibly *Schistosoma japonicum*, along with allergic or hypersensitivity reactions against **praziquantel** treatment. To cope with and overcome these problems, combined use of drugs, i.e., **praziquantel** and other newly introduced compounds such as triclabendazole, artemisinins, and tribendimidine, is being tried.

### **Metabolic and Systemic Issues**

#### **Acidosis**

http://www.vaclib.org/basic/health/pHandCancerAcidosisConnection.pdf

http://en.wikipedia.org/wiki/Acidosis

**Acidosis** is an increased <u>acidity</u> in the blood and other body tissue (i.e., an increased <u>hydrogen ion concentration</u>). If not further qualified, it usually refers to acidity of the <u>blood plasma</u>.

Acidosis is said to occur when arterial <u>pH</u> falls below 7.35 (except in the fetus- see below), while its counterpart (<u>alkalosis</u>) occurs at a pH over 7.45. <u>Arterial blood gas</u> analysis and other tests are required to separate the main causes.

The term *acidemia* describes the state of low blood pH, while *acidosis* is used to describe the processes leading to these states. Nevertheless, the terms are sometimes used interchangeably. The distinction may be relevant where a patient has factors causing both acidosis and alkalosis, wherein the relative severity of both determines whether the result is a high or a low pH.

The rate of cellular metabolic activity affects and, at the same time, is affected by the pH of the body fluids. In mammals, the normal pH of arterial blood lies between 7.35 and 7.50 depending on

the species (e.g., healthy human-arterial blood pH varies between 7.35 and 7.45). Blood pH values compatible with life in mammals are limited to a pH range between 6.8 and 7.8. Changes in the pH of arterial blood (and therefore the extracellular fluid) outside this range result in irreversible cell damage. [1]

<u>Metabolic acidosis</u> may result from increased production of metabolic acids or disturbances in the ability to excrete acid via the <u>kidneys</u>. Renal acidosis is associated with an accumulation of <u>urea</u> and <u>creatinine</u> as well as metabolic acid residues of protein catabolism.

An increase in the production of other acids may also produce metabolic acidosis. For example, <u>lactic</u> <u>acidosis</u> may occur from:

- 1. severe (PaO<sub>2</sub> <36mm Hg) <u>hypoxemia</u> causing a fall in the rate of oxygen diffusion from arterial blood to tissues
- 2. <u>hypoperfusion</u> (e.g., hypovolemic shock) causing an inadequate blood delivery of oxygen to tissues.

A rise in lactate out of proportion to the level of pyruvate, e.g., in mixed venous blood, is termed "excess lactate", and may also be an indicator of fermentation due to anaerobic metabolism occurring in muscle cells, as seen during strenuous exercise. Once oxygenation is restored, the acidosis clears quickly. Another example of increased production of acids occurs in starvation and diabetic acidosis. It is due to the accumulation of ketoacids (ketosis) and reflects a severe shift from glycolysis to lipolysis for energy needs.

Acid consumption from <u>poisoning</u> such as <u>hypercapnia</u>, elevated levels of <u>iron</u> in the blood, and chronically decreased production of <u>bicarbonate</u> may also produce metabolic acidosis.

One key to distinguish between respiratory and metabolic acidosis is that in respiratory acidosis, the CO<sub>2</sub> is increased while the <u>bicarbonate</u> is either normal (uncompensated) or increased (compensated). Compensation occurs if respiratory acidosis is present, and a chronic phase is entered with partial buffering of the acidosis through renal bicarbonate retention.

However, in cases where chronic illnesses that compromise pulmonary function persist, such as late-stage emphysema and certain types of <u>muscular dystrophy</u>, compensatory mechanisms will be unable to reverse this acidotic condition. As metabolic bicarbonate production becomes exhausted, and extraneous bicarbonate infusion can no longer reverse the extreme buildup of carbon dioxide associated with uncompensated respiratory acidosis, <u>mechanical ventilation</u> will usually be applied. [6][7]

### The Acidosis/Hypoxia Cancer Link

#### **Body Acidity**

Through normal day to day activity in the body, acids are formed as waste products that need to be neutralized or eliminated. Some of the acids are released with carbon dioxide (CO<sub>2</sub>) from exhaling and others are excreted from the blood via the kidneys. But the body can only excrete a limited amount and can easily be overburdened by an acidic diet (meat/milk/grains, no vegetables or fruit). And an <u>acidic diet</u> with excess animal protein (especially milk-derived products) causes congestion in the lymph fluid which makes acidic cellular waste removal more difficult. According to the research paper *Metabolic Acidosis due to Drugs and Toxins* several drugs and toxins have been implicated as a cause of

metabolic acidosis including isopropyl alcohol which is in many cosmetic products that the skin readily absorbs into the body.

#### **Automatic Hyperventilation as a Correction of Acidosis**

To try to correct the imbalance of an over-acid body condition the bodies respiratory center will automatically increase respiration rate to lower the acidity. Recently the Journal of Applied Physiology published a study done in the Department of Medicine, Queen's University (Kingston, Ontario, Canada). It was found that 40 patients with cancer breathed about 12 litres of air per minute at rest (Travers et al, 2008), instead of 6 litre/min, which is the medical norm. Breathing frequency of these cancer patients was 20 breaths per minute (the norm is only 12 breaths per minute at rest). What this over-breathing does is to increase exhaled carbon dioxide ( $\mathrm{CO}_2$ ) which removes it from the blood and therefore from all the body. Carbon dioxide is a crucial variable in pH balance. Its reduction shifts the body towards greater alkalinity (increased pH) and less acidity. But additional  $\mathrm{CO}_2$  exhalation is often not enough to make a complete pH correction and the body is left with excess acid despite all efforts at rebalancing.

### Hyperventilation Causes Low Body CO<sub>2</sub>

The respiratory center, situated in the brain stem, increases or lowers breathing in order to maintain pH according to the Henderson-Hasselbach equation. Hence, to maintain pH, the ratio of acidic  $\mathrm{CO}_2$  to alkaline bicarbonate in the cerebral-spinal fluid needs to remain constant. Since the blood-brain barrier is extremely permeable to  $\mathrm{CO}_2$ , this is readily accomplished by regulation of breathing. To compensate for high acidity the  $\mathrm{CO}_2$  has to be kept low. This leaves the body tissues with low  $\mathrm{CO}_2$  levels.

### Low CO<sub>2</sub> Causes Vascular Restriction and Low Tissue Oxygen

The low  $\mathrm{CO}_2$  results in a profound derangement of normal body chemistry. Carbon dioxide is a known muscle relaxant. Smooth muscle in the walls of some arteries and bronchioles is affected by low carbon dioxide. The smooth muscle in the blood vessels going to the brain contract during hyperventilation which is what causes the resultant dizziness. There are many other effects of hyperventilation on the body. One of the most significant is poor oxygenation of the cells. Apart from constriction of blood vessels causing a reduction in blood flow, low  $\mathrm{CO}_2$  decreases the ability of hemoglobin to release its carried oxygen to body cells. This is know as the Bohr effect (the higher the  $\mathrm{CO2}$  in tissues, the more oxygen is released to them, an exchange of one for the other). This results in low tissue oxygen.

#### Hypoxia (low oxygen in body tissues) as Cancer Cause

Otto Warburg discovered one of the main causes of cancer when he won two Nobel Prizes fifty years ago for his work on proving that cancer is caused by a lack of oxygen respiration in cells. He stated in *The Prime Cause and Prevention of Cancer* that: "We find by such experiments that 35 percent inhibition of oxygen respiration already suffices to bring about such a transformation during cell growth. Oxygen pressures that inhibit respiration 35 percent can occur at the end of blood capillaries in living animals, so that the possibility arises that cancer may result when too low oxygen pressures occur during cell growth in animal bodies." Also; "tumors live in the body almost anaerobically [w/o oxygen]"

#### The Vicious Cycle Leading to Cancer

So, in conclusion, it can be seen that acidity causes a chain of events which disallows normal oxygen levels in body tissues. The acidity causes a higher respiration rate, lowering body  ${\rm CO}_2$  which causes the blood cells to deliver less oxygen to tissues. Oxygen normally has a cleansing effect in cells, and the lack of it allows toxicity to increase. So greater acidity causes oxygen deprivation which permits greater toxicity, including toxicity of cancercausing toxins. The end result is a toxic low-oxygen body which is a perfect state for normal cells to become cancerous and multiply to form tumors.

#### **Low Immunity Allows Cancer Growth**

Of course there can be many reasons for low immunity which results in a limited response to the presence of cancer cells by the immune system. General bad habits such as a diet poor in fresh fruits and vegetables, lack of pure water and air, smoking, drinking, lack of deep sleep, and others can cause your immunity to be limited. Also any dental infection with anaerobic bacteria (non-oxygen dependent bacteria) causes a lack of response by the immune system to cancer cells. This tidbit of knowledge comes from 30 years of research by Dr. Dowling in his North Carolina Institute of Technology.

#### **Stopping the Cancer Process**

Stopping the growth of existing tumors and the development of new cancer tumors is as easy as cleansing the body of its excess acid and alkalinizing the body with **baking soda** (sodium bicarbonate), minerals and vegetable juice. The most time tested method of cleansing out toxins is water fasting. Fruit juice fasting would be easier but cancer thrives on sugar, even when its natural. The only exception allowed for this is using red grapes because of their many cancer fighting natural compounds. Once the toxins leave the tissues and start circulating in the bloodstream the patient will feel worse with headaches and tiredness but it is an expected part of the cleansing process. Once the toxins are in the bloodstream the liver and kidneys will work to eliminate them. So it will be better to beforehand cleanse the gallbladder with **olive oil**, cleanse the liver with **Dandelion Root** extract (capsules or liquid), and cleanse the kidneys with kidney cleansing herbal/vitamin formulas. That way the body cleansing process can be done with maximum speed without hindrance.

#### Gauging Return to Normal Body pH

The return of a normal respiration rate can be used to gauge the return of normal body pH from being over acid. Using urine pH may not be a reliable guide because the body robs minerals from bones and tissues to maintain blood pH balance when the body is acidic. And urine is filtered out of the pH balanced blood by the kidneys which means that it will maintain a relative balance. Also the amount of seconds a person can hold their breath can be used as an indication of body oxygenation. Healthy oxygen levels in the body can allow a person to hold their breath 40 seconds. Less than 20 seconds is an indication of acidity/hypoxia, with more imbalance indicated with less breath-holding time.

#### **Destroying Tumors**

It is best to wait until after the body returns to normal pH before you start to burden the body with the toxic waste of dead cancer tissue. Then you can start with anti-tumor electrotherapy to acidify the tumors to death. Please click onto the last source listed below for more info. Otto Warburg said "If one then brings such [cancer] cells, in which during their growth under reduced oxygen pressure a cancer cell metabolism has been produced, back under the original high oxygen pressure, and allows the cell to grow further, the cancer

metabolism *remains*." So the assumption that returning the body to normal oxygen levels will destroy cancer cells has already been disproved by Otto's experiments.

#### **PH Measurement**

http://www.WakeupGethealthy.com

Herman Aihara, in his book entitled "Acid & Alkaline" states that: "If the condition of our extra cellular fluids, especially the blood, becomes acidic, our physical condition will first manifest tiredness, proneness to catching colds, etc. When these fluids become more acidic, our condition then manifests pains and suffering such as headaches, chest pains, stomach aches, etc. According to Keiichi Morishita in his Hidden Truth of Cancer, If the Blood develops a more acidic condition, then our body inevitably deposits these excess acidic substances in some area of the body such so that the blood will not be able to maintain an alkaline condition which causes the cells to become acidic and lowers in oxygen.

As this tendency continues, such areas increase in acidity and some cells die; then these dead cells themselves turn into acids. However, some other cells may adapt in that environment. In other words, instead of dying - as normal cells do in an acid environment - some cells survive by becoming abnormal cells. These abnormal cells are called malignant cells. Malignant cells do not correspond with brain function nor with our own DNS memory code. Therefore, malignant cells grow indefinitely and without order. This is cancer."

One of the least understood concepts of nutrition is understanding what acid and alkaline balance is. The cells of the human body depend on a balanced acid-alkaline pH. If any body fluids are abnormal, digestive enzymes are rendered inactive, food does not digest properly, and allergic reactions can result. Food-bound microorganisms such as yeast, bacteria, parasites, molds, viruses, etc. breed in the body, which puts stress on the immune system.

The body is largely made up of water, a medium which is biologically useful in allowing nutrients, oxygen and bio-chemicals to be transported from place to place. This water-based medium can have either acid or alkaline properties that are measured by a graduated scale called pH (for potential hydrogen), wherein 1.0 to 6.9 is considered acidic, 7.0 is neutral and 7.1 to 14.0 is alkaline. The lower the pH number, the greater the acidity, and the higher the pH number, the greater the alkalinity.

Optimally, we want the fluids in our bodies to have a neutral or 7.0-7.2 pH level. Under 5.3 the body can not assimilate vitamins or minerals, it must be above 6.4 for maximum utilization and weight loss.

Urine or saliva pH levels should be tested in A.M. prior to eating, drinking, or exercising.

#### Why should we be concerned about pH levels?

Since most of the body is water-based (50-60%), the pH level has profound effects on all body chemistry, health and disease. All regulatory mechanisms (including breathing, circulation, digestion, hormonal production) serve the purpose of balancing pH, by removing caustic metabolized acid residues from body tissues without damaging living cells. If the pH deviates too far to the acid side or too far to the alkaline side, cells become poisoned by their own toxic waste and die. Just as acid rain can destroy a forest and alkaline wastes can pollute a lake, an imbalanced pH corrodes body tissue, slowly eating into the 60,000 miles of veins and arteries like corrosives eating into marble. If left unchecked, an imbalanced pH will

interrupt cellular activities and functions, from the beating of your heart to the neural firing of your brain. Our bodies contain many toxins, chemicals, parasites, fungus, bacteria and yeast that, if not cleansed from our system, tend to develop into major illnesses. Most people use soaps that contain animal-fat that may cause the pores of the skin to clog, thereby trapping chemicals and toxins in the body.

#### **Understanding pH Level and Why Many People Develop Cancer**

According to the research of the world famous Dr. Enderlein, <u>total healing of chronic illness</u> <u>ONLY takes place when and if the blood is restored to a normal, slightly alkaline pH.</u>

#### pH: what does it mean?

pH is the abbreviation for *Potential Hydrogen* or the measurement of hydrogen-ion concentration of any solution. The higher the pH reading, the more alkaline and oxygen rich the fluid is. The lower the reading, the more acidic and oxygen deprived the fluid is. The pH scale is from 0 to 14 with 7.0 being neutral. Anything above 7.0 is alkaline; anything below 7.0 is acid.

To be considered healthy, human blood must maintain a narrow pH range of 7.365. Any slight variation means symptoms and disease. If **blood** pH drops below 6.8 or increases above 7.8, cells stop functioning and the patient dies. Blood pH is difficult to test, however, home test kits are available to test urine and saliva pH. Optimum urine and saliva pH is 7.0 to 7.4. Test your pH each morning before food, drink or exercise.

#### If you have health problems, this is a sign that you are acidic.

When the body goes into extreme acidosis, the kidneys start producing ammonia, which may cause the pH to test too alkaline (low pH). This condition is frequently found in elderly people and is the cause of the unpleasant odor in senior citizen homes. Treating for acidosis will help the kidneys to stop producing ammonia.

In 1964, only 1 person in 214 contracted Cancer. Today it is 1 in 3 females and 1 in 2 males. The determining factor between health and disease is pH. It is not uncommon for the average American to test between 4 pH and 5 pH.

- Oxygen levels in the body are directly related to pH.
- Increasing pH from 4 pH to 5 pH increases oxygen to the cells by ten fold.
- Increasing pH from 4 to 6 increases oxygen by 100 times and raising pH from 4 pH to 7 pH increases oxygen levels by 1,000 times.
- CANCER CELLS HAVE AN EXTREME ACID pH AND ARE OXYGEN DEPLETED while HEALTHY CELLS HAVE A SLIGHTLY ALKALINE pH WITH A HIGH OXYGEN CONTENT.

Research shows that unless the body's pH level is slightly alkaline, the body cannot heal itself. So, no matter what type of modality you use to improve your health problem, the modality won't be effective until the pH level comes up.

Drugs, medications and toxic chemicals have the effect of lowering the pH of the body, that is the reason why there are side effects to drugs and none of them effect a cure.

When body pH drops below 6.4, enzymes are deactivated, digestion does not work properly; vitamins, minerals and food supplements cannot effectively assimilate. Acid decreases energy production in the cells, the ability to repair damaged cells, the ability to detoxify heavy metals and makes the body more susceptible to fatigue and illness.

#### Your body pH affects everything

Research has proven that disease cannot survive in an alkaline state, and that, viruses, bacteria, yeast, mold, fungus, Candida and Cancer cells thrive in an acidic, low oxygen / low pH environment. An acid pH can result from an acid forming diet, emotional stress, toxic overload, and/or immune reactions or any process that deprives the cells of oxygen and other nutrients. The body will try to compensate for acid by using alkaline minerals, like sodium from the stomach and calcium from the bones. This is the cause of Osteoporosis and a number of other diseases. If there are not enough minerals in the diet to compensate, a build up of acids in the cells will occur, resulting in symptoms like pain, Arthritis, Fibromyalgia, MS, Lupus, etc.

There are two factors that are ALWAYS present with Cancer no matter what else may be present.

Those two factors are: 1. Acidic pH, and 2. Lack of Oxygen
Can we manipulate those two factors that always have to be present for Cancer
to develop? If we can, will we be able to reverse the Cancer? If so, we need to learn how
to manipulate pH and Oxygen.

Remember, the pH number is an exponent number of 10; therefore, a small difference in pH translates to a BIG difference in the number of oxygen or OHions. In other words, blood with a pH value of 7.45 contains 64.9% more oxygen than blood with a pH value of 7.3.

Cancer needs an acid / low oxygen environment to survive and flourish. The bodies of terminal cancer patients are approximately 1000 times more acidic than they should be. This equates to dangerously low amounts of oxygen at the cellular level.

#### Cancer is not compatible in a healthy pH environment full of oxygen.

For example, CANCER OF THE HEART DOESN'T EXIST. This is because, blood flowing from the lungs into the heart, are at the highest pH and oxygen levels within the entire body. As the blood travels threw the lungs, acidic toxins are thrown out of the system leaving it rich with oxygen and a high blood pH.

In the absence of oxygen, glucose undergoes fermentation to lactic acid. This causes the pH of the cell to drop even lower. Urine and saliva pH of terminal cancer patients almost always runs between 4.0 and 5.5. When the cancer goes into metastases the pH drops even lower.

Our bodies simply cannot effectively fight disease if our body pH is not properly balanced. In other words, it's either alkalize or die. It's that important!

### **Minerals**

Trace minerals? Well, low back pain is one good reason. This pain often arises from a deficiency of trace minerals needed for the enzyme activity necessary for the formation and repair of ligaments, tendons, and bones.

Everyone knows how important vitamins are to good health, but it is easy to overlook the importance of minerals in a healthy diet. When taking mineral supplements, however, it is important to remember that food also contains many essential trace elements, including some trace elements that are not present in multivitamin and mineral supplements. These essential trace elements are present in extremely tiny amounts in the foods we eat. These trace minerals are everywhere, including in the soil, in foods, and in the body, but it is

important that you get the necessary amounts of these elements from your diet or a supplement.

Important minerals include **calcium**, phosphorous, sodium and **potassium**, while trace minerals like **iodine**, **iron** and **zinc** appear in smaller quantities in the body. Each of these minerals has had documented benefits. For example, potassium works in conjunction with sodium to regulate the water balance in the body, and has been proven effective in reducing blood pressure. Zinc has proven to be effective in helping proper brain function and ensuring mental alertness. Some of the minerals also act like antioxidants, fighting off those dangerous free radicals that threaten cardiovascular health through oxidation. Selenium has been shown to be particularly effective in this regard. Each mineral performs a vital function, and a mineral supplement will guarantee that you are receiving all of their health benefits.

Mineral supplements should be part of a complete solution that includes vitamins as well. Research has shown that certain vitamins and minerals work well together. For example, the minerals calcium, magnesium, phosphorous, selenium and zinc have been shown to enhance the effectiveness of vitamin A in the body. Calcium, cobalt, copper, iron and sodium have been shown to help vitamin D. These are just a few examples. A total solution of vitamins and minerals such as those found in a vitamin and mineral supplement should therefore be part of your regimen, rather than taking a mineral supplement alone.

In theory, an individual can gain the necessary supply of minerals through diet alone, however this is no longer as feasible as it used to be. Modern farming methods have bleached a significant amount of minerals from fruits and vegetables, making it necessary for individuals to take mineral supplements to compensate for the loss. It is recommended that you take a complete vitamin and mineral supplement in which the minerals come in chelated form. Chelation binds minerals to amino acids, ensuring adequate absorption in the bloodstream. Also, choose a mineral supplement that has been scientifically formulated for the best blend, and labeled pharmaceutical GMP compliance to ensure it meets the most exacting standards of manufacturing.

Chromium Benefits - Chromium is an important trace mineral whose primary purpose is to aid in the synthesis and transport of protein throughout your body, and to help metabolize glucose for energy. Because of its ability to work with insulin in managing blood sugar, it has been thought by some to be

Selenium Benefits - Selenium is trace mineral found in the soil, and is needed in only small amounts by the body. The mineral, through a series of processes, converts into a potent antioxidant enzyme and plays an important role in the functioning of vitamin E. The vitamin and the mineral, in tandem, represent a powerful combination.

Zinc Benefits - Zinc is an important mineral present in every part of the body and is involved in the management and direction of numerous bodily processes. It's involved in the manufacture of DNA—specifically, in growth and cell division.

#### **Trace Metals**

Some of the most important essential trace elements include:

**Boron** Chromium Copper

Fluoride

**Iodine** 

Iron

Lithium

**Manganese** 

<u>Molybdenum</u>

Selenium

Vanadium

Zinc

#### **Vitamin Links**

www.peacefulmind.com/anti-aging.htm

http://www.lef.org/anti-aging/

http://ods.od.nih.gov/factsheets/list-all/

http://www.ifnh.org/

### What do adrenal hormones do?

The adrenals are your "life saving" organs because they control your body's hormones and help you survive in stressful situations. They act as control organs for your "fight or flight" response and secrete many of our most important hormones including: pregnenolone, adrenaline, estrogen, progesterone, testosterone, **DHEA** and cortisol.

When your adrenals are constantly stressed this sets off an autoimmune, inflammatory response in your entire body. The adrenal-hypothalamus-pituitary feedback loop regulates the secretion of cortisol.4 All of your organs AND your immunity are impacted negatively by the resulting constant assault of cortisol. Cortisol suppresses **DHEA** the" youth hormone."

#### http://bodyecology.com/

Your brain obtains this hormone from your adrenals. **DHEA** is the most abundant hormone in the body and your brain has an enormous "appetite" for **DHEA**. Common symptoms of adrenal fatigue are:

- ■Cravings for sugar
- ■Cravings for salt on food when you eat
- ■Feel dehydrated and thirsty and require plenty of water
- ■Difficulty falling asleep at night, sleep lightly or wake early or often
- ■Difficulty relaxing, nervous, anxious or hyperactive
- ■Often spacey, or foggy thinking, even memory loss
- ■Lack willpower to accomplish
- ■General exhaustion
- ■Hormone imbalances
- ■Low libido
- ■Weight gain, especially in abdomen and waist area
- ■Losing muscle tone
- ■Sagging skin, dry, yellow or pale in color
- ■Hair starting to gray, thin out and become dry
- ■Lips losing their color
- ■Loss of appetite
- ■Anorexia
- ■Weight loss
- ■High blood sugar

Certain nutrients, especially **B-Vitamins, Vitamin C** and minerals are essential for feeding your adrenals. Of these, perhaps most important are minerals (sodium, potassium, magnesium, zinc, copper, manganese, etc). Here are some common foods, drinks and lifestyle habits that contribute to adrenal fatigue AND what to choose instead:1. Sugar

- Instead: Use stevia - an all-natural herbal sweetener that gives you a sweet taste without feeding candida, raising your blood sugar or robbing your body of minerals. Learn more by reading: The One Supplement that Should be in Everyone's Cooking Cupboard.

#### 2. Coffee

Coffee depletes B vitamins and destroys the immune system.

- Instead: Replace your morning cup of adrenaline boosting **coffee** with far superior options. Vitality SuperGreen this gut-healing green drink is full of the nutrients that your adrenals and thyroid love. On top of that, it provides a natural source of alkalizing energy for your body. For more information, read: Are You at Peak Vitality? Answer these Key Questions. A shot glass of fermented spirulina will get your energy up and almost instantly. One ounce of black currant juice in 4 6 ounces of young coconut kefir is another classic Body Ecology adrenal tonic.
- 3. Too much animal protein most people don't have enough stomach acid to digest animal protein properly, creating an acidic condition in their blood.
- Instead: follow the 80/20 rule and enjoy meals with 80% vegetables and 20% protein.
- Also use Body Ecology's ASSIST for Protein
- Fermented foods and liquids are a must for digestion of proteins. That means animal proteins and vegetable proteins.
- 4. Processed foods strip your body of minerals making your blood acidic and your adrenals weaken quickly. Over time you will look and feel older than your age. This is one of the most important anti-aging secrets to delay and even reverse old age.
- Instead: change your diet step-by-step focus on Body Ecology's 7 key healthy eating habits and immediately introduce at least one of our fermented foods and liquids into your diet. These are the new and true "fast foods" of today. They are quickly becoming the new "stars" in the natural foods industry. But no one knows fermented foods like we do here at Body Ecology. Why would we call them a fast food? How much time does it take to open a jar of cultured veggies or a bottle of CocoBiotic or BE Wholegrain Liquid?

#### 5. Soda pop

- Instead: try Body Ecology's probiotic liquids for their immune-boosting and adrenal nourishing properties. To make the new "soda pop" once again we have a great solution. Pour a small amount of your favorite fermented liquid (like BE WholeGrain) into a glass. Add sparkling mineral water and stevia to taste. If you are a parent or a grandparent please introduce your own little ones to this great alternative. Then watch their health and their behavior change for the better.

6.Drugs - this includes over the counter, recreational and prescription drugs. For more information, read: How this All-Too-Common Habit is Making Your Blood Dangerously Acidic.

7.Stress

- Instead - Get plenty of rest and heal negative emotions. take time for self-care routines like meditation, yoga, deep breathing and getting plenty of sleep. Negative thoughts and emotions can sabotage your best efforts at relaxing. Try effective emotional clearing approaches like the Emotional Freedom Technique (EFT). Just like diet, thoughts and emotions become habits. EFT is a great way to easily change these thought habits and start healing your emotional health.

#### **Cortisol**

Cortisol, often referred to as the death or stress hormone, does have some life-giving tasks. For example, cortisol helps: prepare your body for survival in stressful situations, metabolize carbohydrates, protein and fats into energy, slow the immune system's inflammatory response, regulate the balance of converting insulin in breaking down sugar for energy and helps maintain your cardiovascular health and blood pressure.3

Cortisol becomes the "death hormone" when levels remain too high - which it does under chronic stress. The human body was designed to respond effectively to stress and then have periods of rest in between. Unfortunately, our modern day society is so full of constant stressors that we are no longer having that time of rest.

There is an eloquent connection between your adrenals and your brain. These organs are constantly in communication with each other using chemical messengers (or hormones) that flow between the adrenals, the hypothalamus and the pituitary.

#### **DHEA**

When your adrenals are constantly stressed this sets off an autoimmune, inflammatory response in your entire body. The adrenal-hypothalamus-pituitary feedback loop regulates the secretion of cortisol.4 All of your organs AND your immunity are impacted negatively by the resulting constant assault of cortisol. Cortisol suppresses **DHEA** the" youth hormone."

Your brain obtains this hormone from your adrenals. **DHEA** is the most abundant hormone in the body and your brain has an enormous "appetite" for **DHEA**.

#### **Cortisol Functions**

Cortisol belongs to a class of hormones called glucocorticoids, which affect almost every organ and tissue in the body. Cortisol's most important job is to help the body respond to stress. Among its many vital tasks, cortisol helps

- maintain blood pressure and cardiovascular function
- · slow the immune system's inflammatory response
- · maintain levels of glucose-a form of sugar used for energy-in the blood
- · regulate the metabolism of proteins, carbohydrates, and fats

#### **Pituitary**

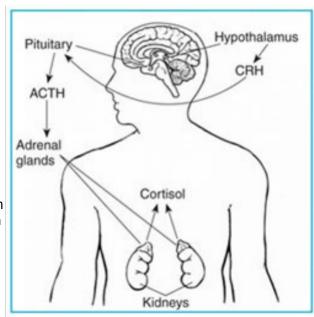
The amount of cortisol produced by the adrenals is precisely balanced. Like many other hormones, cortisol is regulated by the brain's hypothalamus and the pituitary gland. First, the hypothalamus releases a "trigger" hormone called corticotropin-releasing hormone (CRH) that signals the pituitary gland. The pituitary responds by sending out ACTH, which in

turn stimulates the adrenal glands. The adrenal glands respond by producing cortisol. Completing the cycle, cortisol then signals back to both the pituitary and hypothalamus to decrease these trigger hormones.

The hypothalamus sends CRH to the pituitary, which responds by sending out ACTH. ACTH then causes the adrenals to release cortisol into the bloodstream.

#### **Aldosterone**

Aldosterone belongs to a class of hormones called mineralocorticoids, also produced by the adrenal glands. Aldosterone helps maintain blood pressure and water and salt balance in the body by helping the kidneys retain sodium and excrete potassium. When aldosterone production falls too low, the kidneys are not able to regulate water and salt balance, leading to a drop in both blood volume and blood pressure.



### What are the symptoms of adrenal insufficiency?

The symptoms of adrenal insufficiency usually begin gradually. The most common symptoms are

- chronic, worsening fatigue
- muscle weakness
- loss of appetite
- weight loss

#### Other symptoms can include:

- nausea
- vomiting
- diarrhea
- low blood pressure that falls further when standing, causing dizziness or fainting
- irritability and depression
- a craving for salty foods due to salt loss
- · hypoglycemia, or low blood glucose
- headache
- sweating
- in women, irregular or absent menstrual periods

#### Hyperpigmentation

Parasite infections can cause hyperpigmentation, like having a tan in the winter.

Hyperpigmentation, or darkening of the skin, can occur in Addison's disease but not in secondary adrenal insufficiency. This darkening is most visible on scars; skin folds; pressure points such as the elbows, knees, knuckles, and toes; lips; and mucous membranes such as the lining of the cheek.

Because the symptoms progress slowly, they are often ignored until a stressful event like an illness or accident causes them to worsen. Sudden, severe worsening of symptoms is called an Addisonian crisis, or acute adrenal insufficiency. In most cases, symptoms of adrenal insufficiency become serious enough that people seek medical treatment before a crisis occurs. However, sometimes symptoms first appear during an Addisonian crisis.

Symptoms of an Addisonian or "adrenal" crisis include

- sudden, penetrating pain in the lower back, abdomen, or legs
- · severe vomiting and diarrhea
- dehydration
- · low blood pressure
- loss of consciousness

If not treated, an Addisonian crisis can be fatal.

#### What causes Addison's disease?

#### **Autoimmune Disorders**

The gradual destruction of the adrenal cortex, the outer layer of the adrenal glands, by the body's immune system causes up to 80 percent of Addison's disease cases.<sup>2</sup> In autoimmune disorders, the immune system makes antibodies that attack the body's own tissues or organs and slowly destroy them.

Adrenal insufficiency occurs when at least 90 percent of the adrenal cortex has been destroyed. As a result, often both cortisol and aldosterone are lacking. Sometimes only the adrenal glands are affected. Sometimes other endocrine glands are affected as well, as in polyendocrine deficiency syndrome.

Polyendocrine deficiency syndrome is classified into two separate forms, type 1 and type 2. Type 1 is inherited and occurs in children. In addition to adrenal insufficiency, these children may have

- underactive parathyroid glands, which produce a hormone that regulates calcium and phosphorus balance in the body
- slow sexual development
- pernicious anemia, a severe type of anemia
- · chronic candida infections, a type of fungal infection
- · chronic active hepatitis, a liver disease

Type 2, sometimes called Schmidt's syndrome, usually affects young adults and may include

- an underactive thyroid gland, which produces hormones that regulate metabolism
- slow sexual development
- diabetes

vitiligo, a loss of pigment on areas of the skin
 Scientists think type 2 polyendocrine deficiency syndrome is also inherited because often more than one family member has one or more endocrine deficiencies.

<sup>2</sup>Martorell PM, Roep BO, Smit JWA. Autoimmunity in Addison's disease. *The Netherlands Journal of Medicine*. 2002;60(7):269-275

### What causes secondary adrenal insufficiency?

Secondary adrenal insufficiency can be traced to a lack of ACTH. Without ACTH to stimulate the adrenal glands, the adrenals' production of cortisol drops. Aldosterone production is not usually affected.

Less commonly, adrenal insufficiency occurs when the pituitary gland either decreases in size or stops producing ACTH. These events can result from

- tumors or infections of the area
- loss of blood flow to the pituitary
- · damage to parts of the hypothalamus
- · damage to the pituitary gland

#### **Adrenal Stimulation**

- Magnesium Oxide
- Ester C
- Ashwaghanda
- Ginkgo

### **Ashwaghanda**

Ashwaghanda is known as an adaptogen. It helps your body adapt.

Ashwaghanda helps the adrenals adjust to stress. It helps you to produce stress hormones when you need them and stop producing them when you don't.



**Studies show** Ashwaghanda decreases anxiety, improves immune function, and improves sleep. It also functions as an antioxidant. Many people report a calming effect when they take ashwagandha, but even if you feel you have adrenal fatigue and are tired and dragging most of the time, this herb can help you as well due to its adoptogenic properties. It will help you to produce more epi, norepi, and cortisol if that's what you need.

There are many other adaptogenic herbs, <u>Ashwaghanda</u> has the most research behind it and the least number of potential side effects. The recommended dose is around 500mg per day. **No loose stool with this one.** Adrenal burnout

These individuals exhibit a mineral pattern referred to as slow oxidation, and/or a sodium/potassium ratio less than 2.3:1, which is indicative of **adrenal burnout**. Hormone boosing **DHEA** 50 mg, or **ENADA** 10 mg are usually prescribed.

NADH is known to trigger energy production by generating ATP (adenosine triphosphate) which stores energy in cells. If cellular levels of NADH are depleted, brain and muscle cells lose their ability to function effectively.

Suppression of the hypothalamic pituitary adrenal axis (HPA axis). As a result of adrenal suppression, sufferers tend to put out less cortisol, and also less of another adrenal hormone called **DHEA**.

It seems that, if you try to replace the cortisol, it doesn't work. But replacing the **DHEA** seems to bring about a small improvement in some sufferers. The dose of **DHEA** is 25-50 mg for women, 50-100 mg for men, daily. It is best taken in the morning. **DHEA** is freely available without prescription.

### **Neurotoxic Syndromes**

The presentation of biotoxin exposure often parallels neurological and psychological impairment due to the interrelationship between the ENS (Enteral Nervous System) and the CNS. The biliary tree, gall bladder, and bile formation within the liver serve in the vital processes of detoxication (disposal of waste products bilirubin, heavy metals, biotoxins, xenobiotics), lipid metabolism, transport and digestion (bile acids). Abnormalities of the hepatobiliary system may involve biliary stasis whereby infectious material or biotoxins reside within the liver, biliary tree and gall bladder, as a viscous suspension in biliary sludge. Biotoxins as bacteria, viruses, parasites, spirochetes, dinoflagelletes, and fungus may be within biliary sludge often creating neurotoxins that may then impact the CNS via the ENS, or the gut (The Second Brain). The occurrence of biliary sludge may be due to prolonged fasting, low fat intake, high carbohydrate diets or exposure to pathogens. Restriction of dietary fat and the subsequent lack of bile production may impair biliary flow which would be contraindicated in attempting to clear toxicity as bile is paramount to cleansing the body and getting biotoxins and heavy metals excreted into the fecal matter.

Neurotoxins are minute compounds between 200-1000 KD (kilodaltons) that are comprised of oxygen, nitrogen and sulfate atoms arranged in such a way as to make the outside of the molecule fat loving and water hating. As such, once it enters the body, it tends to bind to structures that are rich in fat such as most of our cells, especially the liver, kidney, and brain. Neurotoxins are capable of dissolving in fatty tissue and moving through it, crossing cell membranes (transporting against a gradient, particularly with potassium) and disrupting the electrical balance of the cell itself.

As fat soluble neurotoxins move through the cells of the body from the GI tract to sinus, to lung, to eye, to muscle, to joint, to nerve, they eventually enter the liver and the bile. Once -neurotoxins bind with bile they have access to the liver; the body is then poisoned over and over again as the bile is re-circulated (first released into the intestine to digest fats, and then reabsorbed).

Neurotoxins cause damage by disrupting sodium and calcium channel receptors, attacking enzyme reactions involved in glucose production thereby disrupting energy metabolism in the cell, manufacturing renegade fatty acids as saturated very long chain, odd chain and branched chain fatty acids impairing membrane function, stimulating enzymes (PLA2) which uncouple essential fatty acids from the cell membrane and impairing the function of the nuclear receptor PPAR gamma which partially controls transcription (the conversion of instructions held in our DNA to RNA which then impacts protein production in the cell).

Arachidonic acid (AA) has been given a negative association, it is the most prominent essential fatty acid in the red cell and comprises 12% of the total brain and 15.5% of the body lipid content. If AA is depleted, the balance of the EFAs is profoundly impaired.

The increases in PLA2 activity result in premature uncoupling of the essential fatty acids (EFAs) from phospholipids in the cell membrane. The promiscuous release of AA (it's the highest concentrated

HUFA in the membrane) in the presence of an over expression of PLA2 results in a severely compromised increase in inflammation. Carbohydrate consumption, as one of the most profound stimulators of PLA2, must be restricted to control the insulin response and the subsequent loss of those essential EFAs and the eventual metabolic distortion.

Unhealthy bacteria have been known to colonize the liver and its biliary system. These bacteria as well as viruses, spirochetes, dinoflagellates, and the like can synthesize very long chain saturated or renegade fats (Harrington et al 1968, Carballerira et al 1998) that lead to liver toxicity, biliary congestion, impairment of prostaglandin synthesis and the release of glutathione (Ballatori et al 1990). Lipids vibrate in the cell at millions of times/second. The double bonds of the omega 6 and omega 3 lipids are the singing backbone of life expressed through their high energy level. These bonds are their vibratory song, and they absolutely carry a tune befitting every act and function in the exercise of life, providing all 70 trillion of our cells their flexible nature. When renegade fats are over represented in the cell membrane they result in off key expression, and if strong enough, may spell cellular death and apoptosis. Healing the outer leaflet of the membrane (Schachter et al 1983), comprised primarily of **phosphatidylcholine**, with phospholipid therapy, is one of our highest priorities in addressing chronic illness and hypercoagulation.

Once neurotoxins enter the cell they move toward the nucleus turning on indirectly the production of cytokines such as TNF alpha, IL6, and IL-1Beta (Shrief and Thompson 1993, Tsukamoto 1995, Abordo et al 1997,Rajora et al 1997, Brettelal 1989, Hassen et al 1999, Davidson 2001). TNF alpha will stimulate macrophages in the body to become active. The white cells are also induced to gather in the area of cytokine (TNF alpha) release. In addition, TNF alpha induces endothelial cell adhesion. Endothelial cells which line the blood vessels of the body become "sticky" in conjunction with the increase in white cells. The increased blood viscosity results in restricted blood flow in neurotoxic patients leading to fatigue and discomfort, and quite possibly disturbed toxic photoreceptor lipid structures that become compromised with subsequent reduction in visual performance.

The cellular impact of biotoxin and heavy metal burdens results in disturbed prostaglandin synthesis, poor cellular integrity, and decreased GSH levels (DeLeve and Kaplowitz 1990, Dentico et al 1995, Hayter et al 2001, Miles et al 2000, Nagai et al 2002, Zalups and Barfuss 1995, Watanabe et al 1988, Fernandez-Checa et al 1996), with significant suppression of omega 6 arachidonic acid, marked elevation of Renegade fats and ultimately with demyelination (depressed DMAs). The presence of VLCFAs are evidence of peroxisomal dysfunction and suppression of the beta oxidation of lipids and cellular respiration. Renegade fats (VLCFAs, Odd Chains, Branched Chains) are represented as an increase in fat content in the brain as discovered in stroke patients examined by Stanley Rapoport, Chief of the Laboratory of Neuroscience at the NIH. Biotoxins and heavy metals are lipid soluble thus the effect upon cellular processes and hepatobiliary function is often gravely deranged.

Peroxisomes, most prevalent in the liver and kidney, are organelles within the cell that play a crucial role in clearing xenobiotics and the third phase of detoxification. Peroxisomes are intimately involved in cellular lipid metabolism (Bentley et al 1993, Mannaerts and Van Veldhoven 1992, Luers et al 1990, Leiper 1995) **as in the biosynthesis of fatty acids via <u>B-oxidation</u>** involving physiologically important substrates for thromboxanes, leukotrienes and prostaglandins. The creation of a prostaglandin is an oxidative event (Diczfalusy 1994). **Inappropriate use of antioxidants** (mega-dosing) will inhibit **B-oxidation**, the production of prostaglandins and cellular metabolism, thus the liberal use of potent antioxidants might be contraindicated in the buildup of Renegade fats (Akasaka et al 2000) which are the hallmark of toxicity (Kane and Kane 1997, Kane 1999, Kane 2000, Roels et al 1993, Rustan et al 1992). Peroxisomal oxidation enzymes are suppressed by elevation of cytokines such as TNFalpha (Beier et al 1992).

Individuals with immune, CNS, cardiac, GI and endocrine disorders often present with complex xenobiotics involving <u>disturbances in the cytochrome P450 superfamily</u> (hepatic detoxification difficulties) which parallels disturbances in peroxisomal function. The cytochrome P450's are

responsible for the biotransformation of endogenous compounds including fatty acids, steroids, prostaglandins, leukotrienes and vitamins as well as the detoxification of exogenous compounds resulting in substantial alterations of P450s (Guengerich 1991) as xenobiotics may turn off or greatly reduce the expression of constitutive isoenzymes (Sharma et al 1988). Inadequate stores of arachidonic acid can compromise P450 function (McGiff 1991). Oral application of hormones such as pregnenolone, **DHEA** (Di Santo et al 1996, Ram et al 1994, Rao et al 1993) or thyroid **stimulate** ppar peroxisomal proliferation and the β-oxidation of Renegade fats as would nutrients (riboflavin, pyruvate, manganese) and oxidative therapies. Anti-oxidants slow cellular metabolism and must remain in the proper balance with all the essential nutrients and substrates (lipids, protein) to maintain metabolic equilibrium. Removal of renegade fats in the diet is accomplished by the avoidance of mustard, canola oil (Naito et al 2000), peanuts and peanut oil which contain VLCFAs that can challenge patients with liver and CNS toxicity. The oral **use of butyrate, a short 4-carbon chain fatty** acid, is of striking benefit (Fusunyan et al 1998, Segain et al 1983, Yin et al 2001) in mobilizing renegade fats, lowering TNFalpha, sequestering **ammonia**, and clearing biotoxins.

In states of toxicity it is paramount to stabilize omega 6 fatty acids and the lead eicosanoid (Attwell et al 1993) Arachidonic acid (AA) before introducing omega 3 lipids. There exists a crucial balance between omega 6 and omega 3 fatty acids in human lipid metabolism which has only recently been brought into clearer focus through the work of Yehuda (1993, 1994, 1995, 1998, 2000, 2002). His extensive research has revealed that the optimum ratio is 4:1 of omega 6 to omega 3 FAs. AA, the lead eicosanoid, as well as the other w6 EFAs must be stable (in sufficient amounts) before w3 fatty acids are introduced. Clinicians are often met with poor patient outcomes when merely administering omega 3 lipids without first checking on the omega 6 fatty acid content. Low w6s call for dietary additions of animal protein as eggs, butter and meat, which then permit the addition of controlled addition of w3s. The preferred marine oils are generally those higher in EPA and lower in DHA (EPA:DHA), such as Kirunol 3:1, Nordic Naturals 4.5:1, or Omega **Brite 7:1).** The fear of w6s has been grossly overemphasized by focusing on AA and inflammation. It is true that our modern diet is currently flooded with w6 oils. However, there is currently an over expression of marine oils in a majority of our patients that easily distorts the balance of w6s and w3s with the subsequent suppression in many patients of the all important w6s. We see this distortion daily through the examination of their lipid profiles from Johns Hopkins U. The solution lies in dietary control of carbohydrates which directly lowers PLA2 and inflammation, and the correct administration of a true balance of w6s and w3s with adequate testing information. The balance of lipids is far too important for quesswork, we must be more precise before we can impact toxic body syndromes.

The body loses its ability to metabolize fats in states of toxicity and therefore becomes depleted in the eicosanoids and prostaglandins. Essential fatty acids are the precursors to the regulatory prostaglandins which are "local hormones" providing the communication controlling all cell to cell interactions. An optimum balance of fatty acids make up the dynamic membrane. The membrane of every living cell and organelle is composed of two fatty acid tails facing each other. This bilipid layer is so minute (3.5 nanometers) that it would take 10,000 membranes layered on top of each other to make up the thickness of this paper. Yet the dynamics that occur within this tiny envelope is a microcosm that is a challenge for the human mind to envision. Mercury toxicity damages the microtubule structure of the cell, actually dissolves them (Boyd Hailey, lecture 1997). All cells must synthesize molecules and expel waste. All cells must create, through gene expression, the proteins needed for cellular gates embedded in the membrane as ion channels and receptors. The ultimate control of how those peptides behave rests with the high degree of fluidity and the balance of omega 6, omega 3 lipds in the membrane, while the strength of the membrane rests with the structural fats (oleic, stearic, palmitic, cholesterol). Without control of membrane function through lipid manipulation, detoxication is compromised. In essence, the life of the cell is intimately tied to the health of the membrane and the health of the entire organism.

By stabilizing lipid status with intravenous Phospholipid exchange and oral EFA supplementation we have remarkable tools to unload the body burden of neurotoxins (Jenkins et al 1982, Cariso et al 1983, Jaeschke et al 1987, Kolde et al 1992) in both pediatric and adult populations, without side effects. Oral use of phospholipids in a Liver Flush is also an effective intervention in addressing neurotoxic syndromes.

### **Immune System manipulation**

4<- 6<-9 cups of **rose hip seed tea** (9 trans cis retinolic acid)

Throughout the day

**Rose hip seeds** (Rosa rubiginosa) are the only natural source of 9 cis trans retinoic acid. Sweet briar (*Rosa rubiginosa*) is a native of Europe that now grows throughout the world. The fruit or hips are rich in vitamin C and have been used in making syrups.

Rose hip seeds (*Rosa rubiginosa*) contain the natural form of all-trans-Retinoic acid; Vitamin A acid C<sub>20</sub>H<sub>27</sub>O<sub>2</sub> (9cis)-retinoic acid "Tretinoin".(the active ingredient in Retin-A), In Chile, Spain and Argentina, where it is known as "Rosa Mosqueta", it can be found in the wild around the Andes range and is also cultivated to produce marmalades and cosmetic products. Orally, rose hip is used as a supplemental source of dietary vitamin C, for preventing and treating colds, influenza-like infections, infectious diseases, vitamin C deficiencies, fever, increasing immune function during exhaustion, gastric spasms, gastric acid deficiency, preventing gastric mucosal inflammation and gastric ulcers, and as a "stomach tonic" for intestinal diseases. It is also used orally for diarrhea, gallstones, gallbladder ailments, lower urinary tract and kidney disorders, dropsy (edema), gout, aging skin, disorders of uric acid metabolism, arthritis, sciatica, diabetes, weight loss, hyperlipidemia, hypertension, increasing peripheral circulation, for reducing thirst, as a laxative and diuretic, and to treat chest ailments. In foods and in manufacturing, it is used for rose hip tea, jam and soup, and as a natural source of vitamin C.

Steroid receptor superfamily has identified certain members as molecular targets for cancer therapy (1). They include **estrogen receptors**, **retinoic acid receptors**, **retinoid X receptors** (**RXR**; the RXR-specific ligands are termed "rexinoids"), **and the vitamin D receptor** (the vitamin D—specific ligands are termed "deltanoids"). These nuclear receptors are putative cancer therapy targets because they function as transcription factors that control the expression of many genes related to cell differentiation (1, 2). The strongest evidence for the therapeutic potential of this approach comes from the efficacy of retinoic acid receptor a activation in the treatment of acute promyelocytic leukemia (3). Over 90% of patients with acute promyelocytic leukemia achieve complete remission following treatment with the naturally occurring retinoid all trans-retinoic acid

(Restores IL17 T helper cells, prevents D3 twist in DNA helix)

Much of the action of 1,25D can be explained by its binding to and activation of the VDR. The VDR is a nuclear receptor and ligand-activated transcription factor (20, 49) composed of a highly conserved DNA binding domain and an a-helical ligand binding domain (72). The ligand-bound VDR activates transcription by heterodimerization with retinoid X receptors (RXRs), which is essential for high-affinity DNA binding to cognate vitamin D response elements (VDREs) located in the regulatory regions of 1,25D target genes. VDREs are composed of direct repeats of PuG(G/T)TCA motifs separated by 3 bp (DR3) or

everted repeats with 6-bp spacing (ER6) (20, 26, 49, 89, 90). (Note that everted repeats are palindromic but have symmetry [toes pointing out] opposite that of the so-called inverted repeats [toes pointing in] originally identified as response elements for steroid receptors.) ER8 motifs can also function as response elements for the VDR and related retinoic acid receptors (86), thus partially integrating 1,25D and retinoid signaling. DNA-bound VDR/RXR heterodimers recruit numerous so-called coregulatory proteins, which control histone modifications, chromatin remodeling, and RNA polymerase II binding and transcriptional initiation (24, 31, 56, 70, 74). The ligand-bound VDR can also repress transcription. For example, in the presence of 1,25D, VDR/RXR heterodimers can displace DNA-bound nuclear factor of activated T cells, thus repressing cytokine gene expression (5, 85).

#### Retinoic Acid and 1,25-Dihydroxy Vitamin D3 (1,25 = (OH)2=20 D3)

Retinoic acid (RA) influences adipocyte differentiation [75, = 76] = and fat=20 deposition [77] = and the=20 expression of adipokines such as leptin, resistin, and the serum = retinol-binding=20 protein [78=E2=80==9381]. = Part of=20 these effects is mediated via RAR [82], = which can=20 interfere with the activity of PPAR-=CE=B3 [76, = 77]. = In=20 addition, RA may influence PPAR-mediated responses by activating the RXR = moiety=20 of permissive PPAR:RXR heterodimers [83] = and,=20 possibly, by serving as an agonist of PPAR-=CE=B2/=CE=B4 = [49, = 84].

RA and the 1,25-dihydroxy vitamin D3 (1,25 (OH)2 D3) inhibited = adipocyte=20 differentiation of 3T3-L1 preadipocytes by repressing the upregulated = protein=20 expression of PPAR-=CE=B32 [85]. = The active=20 form of vitamin D, (1,25 (OH)2 D3), inhibits adipogenesis in the bone = marrow of=20 SAM-P/6 mice and is associated with reduction in PPAR-=CE=B32 = expression [86].

www.mountainroseherbs.com Rosa spp. Origin- Chile

The rose hip genius from Chile is the only tested source in NIH database for trans Retinoic acid. **RXR**. Grind rose hip seeds in a coffee grinder for 15-20 seconds, put in coffee maker. Use carbon filtered water.

https://www.mountainroseherbs.com/search/search.php? page=3&refine=y&keywords=Rosehips

Rosehips

Rosa canina Origin- Chile

#### **Formula PM**

1X<- 2X/D 1 teaspoon **MSM** (Organic Sulphur) 10 drops **DMSO** in 8 oz water ¼ teaspoon Bobs Red Mill non-aluminum **Baking Soda**.

Because of it, **DMSO** solvent substitutes for water as it moves through the cell membranes; pulling substances through cells that ordinarily would not penetrate them. Its basic therapeutic principle is its ability to alter and restore damaged cells by hydrating them, changing their water structure, and increasing cellular permeability—which allows cells to nourish themselves and dispose of wastes more easily.

https://www.herbspro.com/login?sef\_rewrite=1

DMSO Liquid, Glass 70% CONC / 30% WATER, 8 OZ

1 Prohealth **Guaifenesin** 600 (otc Mucinex) (proton pump, phosphate inhibitor)

2<- 3<- 6 Natures Plus Source of Life 3058 - Spirulina Algae 2000 Amino Acid complex food

- or -  $\frac{3}{4}$  teaspoon in the yogurt at night

1<- 2 daily vitamin NOW 3770T

2<- 3<- 4 stinging nettle leaf 400mg Swanson SW1346

2<- 3<- 4 stinging nettle root 500 mg Swanson SW968

Stinging nettle robs fungus of hard outer shell - "chitin"

Rhizomes of **stinging nettle** contain a small-sized lectin that exhibits binding specificity toward chitin. This lectin inhibits growth of several phytopathogenic and saprophytic chitin-containing fungi in vitro. The antifungal action of the **nettle** lectin differs from the action of chitinases, which are a ubiquitous class of antifungal plant proteins. Moreover, the **nettle** lectin acts synergistically with chitinase in inhibiting fungal growth. The **nettle** lectin may be a promising candidate for possible applications in the genetic engineering of disease-resistant crops.

http://www.swansonvitamins.com/?SourceCode=INTCON001

#### 2<- 3<-4 magnolia bark 600 mg 4:1 CO2 extract

(PPAR neoligand shields white blood T4 cells against microbes, levels IG and IL balance) do not exceed 6 of these capsules a day, water may build up in lungs/heart/veins, if ascites fluid or fluid builds up in any organ or lymph tissue, increase **Guaifenesin** to 2 or 4 pills, take **willow bark** if clots or blood flow issues show up. If legs or arms fall asleep stand up and walk around a while. Monitor for circulation issues.

Caution: the use of PPAR-*=CE=B3* ligands is associated with an increased = risk of=20 cardiovascular ischemic events.

In mammals, three isoforms have been identified (=CE=B1, = =CE=B2/=CE=B4,=20 and =CE=B3), which are encoded by separate genes. All PPAR = isotypes form a=20 heterodimeric complex with the retinoid X receptor (RXR), and the = complex binds=20 to the PPAR response element, which functions as the central regulator = of=20 cellular differentiation [5], = apoptosis=20 [6],=20 inflammatory responses [7, 8], = lipid=20 metabolism, and metabolic disease [9]. = Also, the=20 farnesoid X receptor (FXR) is involved in adipogenesis, adipocyte=20 differentiation, and lipid storage, increasing adiponectin through a = mechanism=20 partially mediated by PPAR-gamma (PPAR==CE=B3) [10, = 11].

Recently, in a computer-assisted search for PPAR-=CE=B3 = agonists by=20 three-dimensional (3D) structure homology, some neolignans emerge as = strong=20 candidates. Two of these, dieugenol and tetrahydrodieugenol, can be = isolated=20 from Magnolia officinalis Rehd. et Wils. bark with the use of = different=20 chromatographic methods [163].= These=20 naturally derived compounds act as partial PPAR-=CE=B3 agonists; = both=20 exhibited higher affinity for PPAR-=CE=B3 than for the clinically = used agonist=20 pioglitazone (Actos) and were identified as selective activators of=20 PPAR-=CE=B3, but not of PPAR-=CE=B1 or of = PPAR-=CE=B2/=CE=B4. In=20 addition, these compounds induced adipocyte differentiation in 3T3-L1 = cells in a=20 PPAR-=CE=B3-dependent manner. The activation pattern exhibited = from 1 and 2=20 renders them highly interesting, novel PPAR-=CE=B3 agonists that = have the=20 potential to be explored further which may lead to the development of = novel=20 pharmaceuticals

Neutrophil-derived IL-17 was also reported in LPS-induced lung inflammation [97], kidney ischemia-reperfusion injury [98], and systemic vasculitis [99]. TLR2 stimulation induced IL-

17 in macrophages [100]. Subsets of mucosal NK cells have been found to produce IL-22. ). Over 90% of patients with acute **promyelocytic leukemia** <u>achieve complete remission</u> <u>following treatment with the naturally occurring retinoid all trans-retinoic acid</u> (ATRA; ref. 4).

The combination of the PPAR- $\gamma$  ligand **troglitazone** and **certain retinoids** reportedly **suppressed colony formation of leukemic cells** in an additive fashion (44). Because PPAR- $\gamma$  and RXR are known to function as heterodimers, we hypothesized that combined treatment with PPAR- $\gamma$  and RXR-specific ligands would significantly potentate the antileukemic effect of PPAR- $\gamma$  ligands alone. Indeed, the combination of PPAR- $\gamma$  and RXR activation (using the specific ligand LG100268) profoundly induced differentiation in HL-60 cells. Furthermore, LG100268 enhanced CDDO-induced apoptosis in an additive fashion in primary AML and CLL samples.

http://www.amermed.com/magnoliabark.htm

http://www.amazon.com/Magnolia-Bark-Extract-Capsules-2400mg/dp/B000VG60DY/ref=sr 1 3?s=hpc&ie=UTF8&gid=1389796657&sr=1-3

1X-3X/D (throughout day) 25 drops magnolia extract

**PPAR gamma IL23 tilter restoration** 

#### Throughout the day

http://www.betterhealthherbs.com/product/3564.html

http://www.betterhealthherbs.com/

John — "I'm taking 20 to 30 drops of the <u>Magnolia Glycerite Extract</u> 4 to 5 times a day in less than an ounce of water per day. (I've not detected any side effects, so would conclude that the drop count and/or frequency could be increased without discomfort). When taking the extract keep it under the tongue for as many minutes as comfortable; because it's sublingual and is absorbed through oral tissue. When ordering the <u>Magnolia Glycerite</u> Extract select the non alcohol option which is the Glycerite based product."

### The 4 horsemen final - ROSE HIP, Stinging Nettle, Magnolia, Spirulina

2/26/14

#### AM – before anything else

### Hydrogen peroxide (TNF alpha stimulant)

50 drops H2O2 6 oz water

4 cups of rose hip seed tea (9 trans cis retinolic acid) Throughout the day

#### **Formula PM**

- 1X- 2X/D 1 teaspoon MSM (Organic Sulphur)10 drops DMSO in 8 oz water ¼ teaspoon Bobs Red Mill non-aluminum Baking Soda.
- 1 Prohealth Guaifenesin 600 (otc Mucinex) (proton pump, phosphate inhibitor)
- 2 Natures Plus Source of Life 3058 Spirulina Algae 2000 Amino Acid complex
- 1 daily vitamin NOW 3770T
- 2 stinging nettle leaf 400mg Swanson SW1346
- 2 stinging nettle root 500 mg Swanson SW968

- 2 magnolia bark 600 mg 4:1 CO2 extract
- 1 B complex 50 mg GNC 00018
- 1 Selenium 200mg NOW 1486
- 3 **Charcoal** 260 mg Source Naturals 00136
- 1 black currant oil (GLA) 1000 mg now 1717
- 1 Silymarin 300 mg now 4753
- 1 Ginko Biloba 24% 120mg NOW 4683
- 1 HORSE CHESTNUT 300 mg NOW 4713
- 0<-1 Willow Bark to prevent blood clots NOW

#### **Bed Time**

2 <u>Magnolia Seeds</u> per day chopped with shell (do not remove the shell!), chopped in yogurt. This is a whole natural seed of the Swietenia Magnolia King tree grown in Malaysia.

#### Sleeping pill

3 Acidophilus Strain LA5 CHR Hanson with a 1/2 glass of milk

http://www.pureencapsulations.com/media/Lacto Acidophilus.pdf

### **LEAKY GUT & IMMUNE SYSTEM**

A talk given by Tessa Jupp RN in June 2003 has led to a new addition to our book range - \$5.

Here is a summary of the book.

The information for this topic came mainly from a very interesting document by Willis S Langford entitled "Autism - a comprehensive guide" which is available on the internet. Although this article is aimed at treating autistic children, a lot of this information gleaned from world-wide research, is as valuable to the "normal" population as well.

There is an integral link between the functioning of the gut and the immune system and what affects one has a bearing on the other. In order to fix something, we need to understand how it works properly in the first place. The overheads from the talk which explain all this in simple language, have been put together into booklet form and are available at \$5 each.

The important message is that there are 2 types of Helper T-cells in the immune system called Th1 and Th2. The Th1 cells deal with acute infection - like colds and flu's, infected cuts etc. The Th2 cells are responsible for antibody production so that the body can overcome a repeat infection much faster. In simple terms - Th1 are the soldiers fighting in hand to hand combat and the Th2 cells are those on guard duty to sound the alarm and repel invaders.

Problems occur when the Th2 cells become dominant unbalancing the Th1 cells. When this occurs we tend to develop chronic allergies, depressive psychological disturbances, auto-immune diseases, leaky gut and constant infections that are difficult to resolve.

The pattern can be set in childhood as vaccination stimulates Th2 production (can be further stimulated by fluvac etc) and lowers Th1 cells. So a pattern is set up with frequent colds, flu's and ear infections etc that are hard to throw off. We may then end up on antibiotics which kill off the good gut flora, leading to overgrowth of candida and causing damage to the gut lining. This results in decreased nutrient absorption, indigestion, gut ulceration, leaky gut, food intolerances allergic reactions, chronic illness like asthma etc, build-up of toxic metals, chronic ill health and auto immune

disease such as Chronic Fatigue, diabetes, Lupus, rheumatoid arthritis, cancer and autism in susceptible individuals as the Th2 climbs higher. So at any point in our lives we can be thrown into this vicious cycle of ill-health that is self perpetuating.

T Helper 2 Cells are raised by infection, vaccines, stress, faulty digestion, leaky gut, candida, eating processed sugars & flours, trans-fats like margarine, omega 6 fatty acids like canola oil - all of which can lead to adrenal exhaustion, a further complication.

#### SIGNS Th2 IMMUNE SYSTEM is HIGH

- allergies
- muscle aches/headaches
- poor sleep/anxiety/tantrums
- leaky gut
- candida
- grain &/or dairy intolerances
- stressed, depressed, withdrawn

### **CAUSES of LEAKY GUT**

- stress & lifestyle factors immune dysfunction,
- poor diet high in refined foods low in fruit & veg
- nutritional deficiencies poor digestion & absorption
- intestinal invaders candida, heliobacter, parasites
- environmental toxins chemicals & heavy metals
- medicinal drug overuse antibiotics, NSAIDs, antacids

### **SIGNS of LEAKY GUT**

- · abdominal discomfit
- bloating, distension
- reflux, indigestion
- burping, flatulence, gaseousness
- diarrhoea &/or constipation
- problems sleeping
- irritability or weird behaviour
- allergy prone eg hayfever, sinus
- skin eruptions rashes, acne, boils, eczema

### SIGNS of LOW STOMACH ACID

- · stomach uncomfortable after a meal
- burping, flatulence
- bloating, distension
- reflux, indigestion
- diarrhea &/or constipation
- undigested food in faeces
- coated tongue
- fatigue

### **CAUSES of LOW STOMACH ACID**

- antacids acid production shuts down for 9 days
- stress physical & emotional
- bacterial overgrowth candida, heliobacter
- grain or dairy intolerances
- inflamed irritated gut
- low zinc, magnesium, Vits B1, B3, B6
- · chronic pain or illness
- surgery or trauma
- depression
- chemotherapy
- blood type A has lower levels of stomach acid

### DO YOU SEE SIMILARITIES OCCURRING HERE?

The same things keep coming up as happening with leaky gut and low stomach acid. And there is an inter-relationship between the effect the immune system and digestive system have on each other.

Lowered resistance to infection, by removing natural defenses, allows yeast to take hold and multiply more freely. Stomach acid is a first line of defense against ingested organisms like yeast. Low acid is common in celiac disease and cannot properly kill yeast to prevent them from passing into the intestine. Lack of appropriate nutrients diminish or disable resistance of the intestinal lining to infection. Low spleen function, also common in celiac disease, may not produce adequate antibodies and white blood cells to fight infection.

It is important for all parts and systems of the body to get the appropriate nutrients - vitamins, minerals, amino acids as well as the carbohydrates, proteins and fats that are needed for normal body metabolism. Otherwise they cannot function as they are designed to and we recognize this as disease rather than as nutritional dysfunction and therefore correctable.

Many so called "diseases" could be addressed in this fashion. Stomach acid is necessary to break down the foods we eat, particularly proteins before they pass through to the next part of the gut to an alkaline environment and where the doorways to absorption from the gut are like border guards where you have to have the right passport to enter or need a sponsor to accompany your admission.

Just as stomach acid is dependent on the necessary nutrients esp **zinc**, **B1**, **B6** so digestive enzymes, bile and insulin rely on the message for release sent by stomach acidity and need certain nutrients also for production. Each system is reliant on another working well too. For Bile to emulsify dietary fat we need cholesterol, taurine, Vit C, **manganese** and sulphur to make the bile.

### **Estrogen and T1 T2 Balance**

High estrogen levels shift the immune system from T1 to T2 allowing pathogens to grow inside of cells. Infections that are eliminated slowly from the body can produce a shift from T1 to T2 balance. Some people may just have a T2 dominant immune system which will predispose them to intracellular infections.

To reiterate, there are many symptoms of intracellular infection in CFS. Some of these symptoms are increases in T1 cytokines causing flue-like symptoms and tender lymph nodes. Other symptoms of increase RNase L and dysfunctional RNase L activity are: hypoglycemia, night seats, low pain threshold, chemical sensitivity, drug sensitivity, depression due to poor tryptophan uptake, loss of potassium, visual problems, loss of urine concentration during the night, sodium retention, fatigue and sleep disturbance, low magnesium levels leading to muscle weakness, weakness of respiratory muscles leading to respiratory problems, hyperventilation, abnormal response to exercise, low blood volume, ectopic heart beats, prominent U-wave on ECG, bladder problems, premenstrual syndrome, slow wound healing, resistance to hormone uptake in cells.

#### SIGNS of LOW BILE PRODUCTION

dietary fats are not being digested well if:

- · faeces float in toilet
- faeces colour is light tan or grey
- faeces are bulky and shiny
- · bowel action is foul smelling

If you have problems with fatty liver or gallstones, taking <u>taurine</u>, <u>carnitine</u>, <u>Vit E and lemon</u> <u>juice</u> can alleviate. Causes include high copper levels, excessive sugar or fats in the diet, obesity, low stomach acid, low intake of <u>Vit C & E, taurine</u>, <u>manganese</u>

#### **HEALING LEAKY GUT**

So to fix leaky gut we need to address a lot of the causes. We need to -

- 1 Increase stomach acid by taking lemon juice, apple cider vinegar or hydrochloric acid tablets immediately before a meal.
- 2 Avoid processed foods esp grains & sugars (includes flours, breads, pasta cereals, rice)
- 3 Take glutamine and Vitamin A to help repair the damaged villi (gut mucosa absorption sites) and reduce gut hyper-permeability (inappropriate absorption)

  4 Increase hile and pancreatic enymes production take tauring. Vit E & C R3
- 4 <u>Increase bile and pancreatic enymes production take taurine, Vit E & C, B3, chromium, zinc, manganese</u>
- 5 Restore Gut Flora with acidophilus, biodynamic yoghurt, or probiotics on an empty stomach
- 6 Eat organic fruit and veg
- 7 Use blood group diet
- 8 Drink more water spring or filtered
- 9 Avoid constipation take magnesium and Vit C to bowel tolerance (twice a day on empty stomach)
- 10 Boost the immune system get more sleep, gentle exercise,

### Boost Th1 and balance Th2 inflammatory cytokines

http://forums.phoenixrising.me/index.php?threads/article-on-boosting-th1-surpressing-th2.17673/

- \* take **glutamine**, **Vit A**, **C**, **E**, **zinc**, **B Vitamins**, essential fatty acids according to blood group
- \* boost friendly gut flora
- \* improve digestion stomach acid, digestive enzymes, bile, insulin
- \* detox any heavy metals eg copper, mercury, lead
- \* reduce stress relax, sleep, massage, meditation, gentle exercise, laugh, have fun

So we keep coming up with the same things still.

https://www.jakpathways.com/cytokines

https://www.jakpathways.com/jakpathwaysresources

### **JAK Pathways in Depth**

JAK is a family of nonreceptor protein tyrosine kinases (PTKs) located in the cytoplasm of cells, rather than on the cell surface. When activated, JAKs stimulate a cascade involved in the production of pro-inflammatory cytokines. Overactivation of JAK can lead to inflammation and tissue destruction. <sup>1,2</sup>

The JAK family consists of 4 members: JAK 1, JAK 2, JAK 3, and TYK 2 (tyrosine kinase 2)<sup>1,3</sup>:

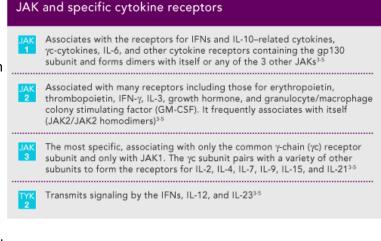
- JAK 1, JAK 2, and TYK 2 are ubiquitously expressed
- JAK 3 (which pairs with JAK 1) is predominately expressed in hematopoietic cells

Each JAK protein has specificity for a different set of cytokine receptors.<sup>3</sup>

 The function of the JAK protein is linked to the function of the cytokines that bind the receptors

JAK Pairings and Associated Cytokines

JAKs do not work alone—each cytokine receptor requires at least 2 associated JAKs in order to signal. <sup>4,6</sup>



JAK pairings and associated cytokines <sup>1,7</sup>					
JAK 1	JAK JAK TYK	JAK TYK 2	JAK JAK	JAK 2	JAK TYK 2
IL-2 IL-4 IL-7 IL-9 IL-15 IL-21	IL-6* IL-11* IL-27 G-CSF OSM LIF CTF	IFN-α IFN-β IL-19* IL-20* IL-22*	IFN-γ	IL-3 IL-5 GM-CSF EPO TPO GH Leptin Prolactin	IL-12 IL-23

<sup>\*</sup>Type II cytokine receptors such as those for IL-10, IL-19, IL-20, and IL-22 as well as gp130 subunit sharing receptors for IL-6 and IL-11 mainly signal through JAK 1, but also associate with JAK 2 and TYK 2.9

JAKs may work in pairs of identical JAKs—homodimers (eg, JAK 2/JAK 2)—or as heterodimers (eg, JAK 1/JAK 3)

Each JAK pairing has specificity for a different set of cytokines. The function of JAK pairings is linked to the function of the cytokines that bind to the receptors.<sup>3,4</sup>

Cytokines That Signal Through

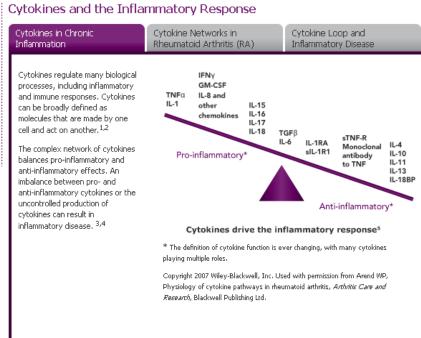
In diseases such as RA, many proinflammatory cytokines are produced in excess at sites of pathologic inflammation.<sup>8-10</sup>

JAK pathways are utilized by an important subset of cytokines to regulate immune and inflammatory response.1,11

Cytokines that utilize the JAK pathways historically have been grouped into two main categories<sup>5,12</sup>:

> Type I includes many interleukins (ILs), both common cytokine receptor y-chain and non-y-chain, and some growth and hematopoietic factors

Type II includes interferons (IFNs) and IL-10



#### **How about CONSTIPATION?**

#### **CAUSES of CONSTIPATION**

- dehydration need 8-10 glasses of water per day
- low digestive enzymes
- low nutrient levels esp Vit C & magnesium
- low protein and fibre intake
- parasites incl candida, intestinal worms
- excessive dairy intake
- low thyroid

### OVERCOMING CONSTIPATION

- Drink more water
- take magnesium and Vit C to bowel tolerance
- increase stomach acid & digestive enzymes
- take supplements essential fatty acids, zinc, chromium, B1, B6 etc
- eat according to blood group

- eliminate problem grains & dairy
- eat balanced diet meat, veg, fruit
- exercise walk, swim, bounce, yoga

### SIGNS OF COLON DYSFUNCTION

- lower back pain (also neck & shoulder pain)
- pain in lower stomach particularly left side
- bloating, flatulence, gas
- fullness under diaphragm can cause
- shortness of breath & discomfit
- fatigue, sluggishness, brain fog
- · constipation or diarrhoea
- · digestive problems
- leaky gut
- Irritable Bowel Syndrome, colitis
- Diverticulitis, Ulcerative Colitis, Crohn's

# Anti inflammatory diet

http://www.drweil.com/drw/u/ART02012/anti-inflammatory-diet?print=1

### **Kidneys**

http://jonbarron.org/program/full-body-detox-program http://jonbarron.org/detox/kidney-gallbladder-flush-natural-health-newsletter-1 http://www.curezone.org/forums/am.asp?i=1052823

# Kidney, Gallbladder and Pancreas Care by Jon Barron

Kidney detox and maintenance has always been part of the Baseline of Health Program. So far, I have not used a dedicated formula for this purpose, because if you're doing the full Program, you're covering the kidneys – particularly when doing the liver detox. As it turns out, a number of herbs (chanca piedra, juniper berry, uva ursi, parsley root, dandelion root, and horsetail) are common to both kidney and liver maintenance. Nevertheless, as the incidence of kidney problems has soared to epidemic levels over the last ten years, the need for a stronger, dedicated formula for doing a regular kidney flush has become paramount. In addition, as you will see, such a formula also can play a major role in eliminating gallstones and, amazingly, even helping with arthritis.

But first, let's explore some background on the kidneys and the extent of the problem in the world today.

### The kidneys

The kidneys are bean-shaped organs located on either side of the lower back, just below the rib cage. Their function is to:

· Keep the composition of your blood balanced.

- Regulate the amount of fluid in the body.
- Control the balance of electrolytes in your blood.
- Help to control blood pressure.
- Produce hormones that are crucial for blood and bone formation.

After blood is filtered through the kidneys, the primary byproduct is urine. The production of urine is a complex process. Far from being a simple removal of water from the body, it is, rather, a process of selective filtration that not only removes waste and potential toxins from the blood while retaining essential molecules, but that also serves to balance key biochemicals and hormones in the blood. Blood enters the kidneys by way of the renal artery and is processed in tiny tubes called nephrons and returned to circulation through the renal veins. The substances that are filtered are turned into urine, composed of 95% water, 2.5% urea, 2.5% mixture of minerals, salt, hormones and enzymes. Urine is then collected in the central part of the kidney and passes through the ureters to the bladder. When the bladder is full of urine, it is emptied from the body through the urethra. Approximately 180 liters of blood move through the two kidneys every day, about 1.5 liters of urine are produced.

### **Other Functions of the Kidneys**

In addition to cleaning the blood, the kidneys regulate the amount of water contained in the blood. ADH (Vasopressin) is an anti-diuretic hormone produced in the hypothalamus and stored in the pituitary gland. When the amount of salt and other substances in the blood becomes too high, the pituitary glands release ADH into the bloodstream and to the kidneys. This increases the permeability of the walls of the renal tubules, helping to reabsorb more water into the blood stream. **The kidneys also adjust the body's acid-base balance to prevent acidosis and alkalosis.** 

Another function of the kidneys is the processing of vitamin D. The kidneys convert this vitamin to an active form that stimulates bone development.

#### The nephrons

As mentioned above, the processing (or filtering) in the kidneys is done in the nephrons. Not surprisingly then, most kidney problems are focused in the nephrons, and correspondingly, most kidney drugs target the nephrons. For example, most diuretics inhibit the ability of the nephrons to retain water, thereby increasing the amount of urine produced.

The important thing to understand about the nephrons is that they are very, very tiny – and therefore easily plugged. When plugged, they become inflamed and infected and die. Also, because they are so small they are easily damaged by chemical imbalances in the blood that attack protein – specifically, high blood sugar and high insulin – which is why diabetes and kidney disease go hand in hand. The bottom line is that over time, when enough nephrons die, kidney function is compromised to the point that the kidneys can no longer do their job. At that point they require outside support (i.e. dialysis) or outright replacement (a kidney transplant).

For a full explanation of the anatomy of nephrons and how they work, check out <a href="http://en.wikipedia.org/wiki/Nephron">http://en.wikipedia.org/wiki/Nephron</a>.

### **Kidney Diseases**

Diseases of the kidneys range from mild infection to life-threatening kidney failure. The most common form of kidney disease is an inflammation of the kidneys. Kidney sludge is the result of the

accumulated crystallized minerals that sometimes obstruct the flow of urine and damage the kidneys. If the minerals accumulate to a sufficient degree, the sludge actually forms into rough surfaced stones that actually rip and tear at the ureters on their way out of the kidneys.

Anyone who suffers from kidney stones knows the pain involved in passing a stone, but keep in mind that a kidney stone is only the extreme manifestation of sludge. Just because you don't suffer from kidney stones doesn't mean you don't have a problem. Kidney sludge may not be painful passing out the ureter, but it is nevertheless deadly over time as it slowly chokes off kidney function nephron by nephron.

How extensive is the problem? **Virtually, every living person has some degree of sludge build up and some loss of kidney function over time.** The only question is how much. Does it reach the point where it causes painful kidney stones to form or the point where it chokes off a critical mass of kidney tissue, ultimately leading to kidney failure.

It should be noted that although kidney stones and gallstones are not identical, the mechanisms involved in their formation are remarkably similar. This means that the same formulas used for eliminating kidney sludge and kidney stones will also help remove gallstones – and for that matter, even help with removing pancreatic sludge and arthritic calcium deposits in joints.

Kidney failure occurs when the kidneys are no longer able to keep the blood balanced. In acute kidney failure, symptoms include swelling, drowsiness, and irregular heartbeat. In chronic kidney failure, symptoms include fatigue, loss of appetite, headaches, cramps and thirst.

In addition to sludge, the other major kidney problems are connected to infection, inflammation, and direct damage to the protein that makes up the kidney tissue as a result of high sugar and high insulin levels.

### A growing epidemic

The National Kidney and Urologic Diseases Information Clearinghouse estimates that each year, nearly 100,000 Americans are newly diagnosed with kidney failure. More than 100,000 currently have End Stage Renal Disease (ESRD) due to diabetes, and **an astounding 7.7 million have physiological evidence of chronic kidney disease**. (1) According to the U.S. Health and Human Services Agency for Healthcare Research and Quality, an estimated 650,000 Americans will have kidney failure by 2010 and will require renal replacement therapy, either ongoing renal dialysis or a kidney transplant. (2) Without one of these therapies, ESRD is fatal. Yikes!

Fortunately, it's good to know there are ways to keep your kidneys healthy to avoid going down that road.

# Taking care of your kidneys

Paramount to good care of the kidneys is reducing the toxic load they have to deal with, especially proteins and chemical contaminants which can build up in the kidneys, slowing their function, increasing acidity and raising blood pressure. So, consider lightening the diet--instead of eating meat every day, try going vegetarian for a day or so a week. A vegetable/fruit-based diet allows the body system to alkalinize via the kidneys, lowering blood pressure, and contributing to a sense of well-being. Also, drinking enough water so that the urine is a light color of yellow. A whole lot of water is not necessarily good for the kidneys, and it is always better to drink small amounts of water throughout the day, rather than gulping down a quart or two because you're thirsty. This just creates kidney stress.

The regular use of a kidney cleansing and rebuilding formula is now mandatory considering the stresses we put our kidneys under thanks to our "modern" lifestyles. A good kidney formula/s will include most of the following properties and ingredients.

#### What the formula needs to do

- 1. Anti-lithic (stone breaking)
- 2. Diuretic (water removing)
- 3. Antiseptic (infection killing)
- 4. Anti-nephrotoxic and anti-hepatotoxic
- 5. Soothing to urinary tract tissue
- 6. Anti-inflammatory
- 7. Stimulating to renal tissue

#### A note on stones

Different stones in the body have different chemical make-ups.

For example, gallstones are primarily formed from cholesterol, **bile salt**s, and proteins. The more protein, the harder the stones. Think of it like the protein used to make fingernails. Incidentally, this protein primarily comes from the lining of the gallbladder. In other words, although stones get their start in the liver, they turn problematic in the gallbladder – which is why removing the gallbladder gets rid of symptoms, but not necessarily the underlying problem, which starts in the liver.

Pancreatic stones are formed from fatty acids, calcium, and proteins.

And kidney stones themselves vary significantly. There are four types.

- **Calcium stones** are composed of calcium that is chemically bound to <u>oxalate</u> (calcium oxalate) or phosphate (calcium phosphate).
- Uric acid stones. If the acid level in the urine is high or too much acid is excreted, the uric acid may not dissolve and uric acid stones may form.
   Struvite or infection stones develop when a urinary tract infection alters the chemical balance of the urine causing stones to form from ammonium, magnesium, phosphate (aka struvite).
- **Cysteine stones**. Some people inherit a rare condition that results in large amounts of cystine in the urine, which causes the formation of cystine stones that are difficult to treat.

The important thing to understand is that although all of the above types of stones have different chemical compositions, most of them can be dissolved by the right combination of herbs in a single formula.

#### What to look for in a formula

#### Chanca piedra (Phyllanthus niruri)

For a number of years now, I have recommended using chanca piedra before liver detoxing to soften gallstones before trying to pass them during the detox. Chanca piedra works equally well on gallstones, kidney stones, and kidney sludge. In fact, the name chanca piedra, as it is known in Peru, comes from its effect on kidney stones and gallstones. The literal translation is "stone breaker." It effectively softens both kidney stones and gallstones for easy passage out of the body. It is also renowned for its diuretic qualities and has been shown effective at helping relieve edema

and urine retention. It also works as an anti-inflammatory agent in the kidneys and as an antihepatotoxic in the liver. That is to say, it counters the effects of toxins in the liver.

References (3, 4, 5, 6, 7, 8, 9)

### Hydrangea root (Hydrangea Arborescens)

The most common use for hydrangea is for the kidneys and bladder because of its effective diuretic property which helps increase the flow of urine. This removes impurities from the system and lessens the likelihood of infection along the entire urinary tract, which includes the kidneys, bladder, prostate (in men) and urethra. <a href="Hydrangea">Hydrangea</a>, like chanca piedra, is also considered an anti-lithic herb, which prevents stones or gravel from forming in the kidneys and bladder. As an anti-lithic herb, it can also assist the body in removing stones and gravel from these organs. This was a primary use of <a href="hydrangea">hydrangea</a> by Native Americans.

Like most diuretic herbs, hydrangea is an excellent choice for treating inflamed or enlarged prostate glands. It is commonly combined with horsetail for this purpose. Maintaining healthy urine flow keeps the prostate less likely to constrict around the urethra, which prevents stagnant urine from causing more infection. This can also reduce inflammation by eliminating impurities from the prostate.

A scientific study published in Bioscience, Biotechnology, and Biochemistry in 2003 noted that hydrangea root extracts have greater antioxidant power in liver tissue than **milk thistle** and turmeric combined. The findings of Japanese researchers amplify observations of nineteenth-century American physicians who used hydrangea primarily as a treatment for "kidney gravel," small stones in the kidneys that could be passed with a minimum of pain after treatment with the herb. Physicians of the time also used hydrangea as a treatment for chronic chest pain caused by bronchitis. **Hydrangea root powder** has a greater diuretic effect than other preparations of the herb , but it has less of an effect on pain.

References (10, 11, 12)

### **Gravel root (***Eupatorium purpureum***)**

Like chanca piedra and hydrangea, gravel root also exhibits both diuretic and anti-lithic properties. Used primarily for kidney stones or gravel (which accounts for its name), it also helps with cystitis, dysuria, urethritis, and pelvic inflammatory disease. It can also play a role in the systemic treatment of rheumatism and gout as it encourages excretion of excess uric acid. And finally, it tones the reproductive tract and is used to treat inflammation of the prostate.

References (13, 14, 15, 16, 17, 18, 19)

#### Marshmallow root (Althaea Officinalis)

<u>Marshmallow</u>'s highest medicinal acclaim is as a demulcent. Internally it has a soothing effect on inflamed and irritated tissues of the alimentary canal, and urinary and respiratory organs. It aids in the passage of kidney stones and is used in combination with other diuretic herbs for kidney treatments which assist in the release of gravel and stones. It works very well for urinary problems.

<u>Marshmallow</u> has factors which combine with and eliminate toxins, helping the body to cleanse. This makes <u>marshmallow</u> an excellent herb to add to other formulas to help neutralize toxins that are the causative factors of arthritis.

<u>Marshmallow</u> is also very soothing to any sore or inflamed part(s) of the body. As well as the urinary tract, this herb will soothe an irritated digestive tract and help with diarrhea or dysentery.

References (20, 21)

### Juniper berry (Juniperus communis)

Juniper Berries are used to treat infections, especially within the urinary tract, bladder, kidneys, and prostate. Their antiseptic properties help remove waste and acidic toxins from the body, stimulating a fighting action against bacterial and yeast infections. Juniper Berries also help increase the flow of digestive fluids, improving digestion and eliminating gas and stomach cramping. As a diuretic, Juniper Berries eliminate excess water retention contributing to weight loss. Juniper Berries' anti-inflammatory properties are ideal for relieving pain and inflammation related to rheumatism and arthritis. In addition, Juniper Berries are beneficial in reducing congestion, as well as treating asthma and colds. Juniper Berries make an excellent antiseptic in conditions such as cystitis. But the essential oil present in this herb is quite stimulating to the kidney nephrons. Some texts warn that juniper oil may be a kidney irritant at higher doses, but there is no real evidence that this is the case, and the dosage in this formula is quite low. Nonetheless, people with serious kidney disease probably shouldn't take juniper.

Contemporary herbalists primarily use **juniper** as a diuretic ("water pill") component of herbal formulas designed to treat bladder infections. The volatile oils of juniper reportedly increase the rate of kidney filtration, thereby increasing urine flow and perhaps helping to "wash out" offending bacteria. The volatile oils, particularly terpinen-4-ol, may cause an increase in urine volume. According to some sources, juniper increases urine volume without a loss of electrolytes such as potassium. It is recommended by the German Commission E for kidney ailments.

References (22, 23, 24, 25, 26)

### Corn silk (Zea mays)

Corn silk is a soothing diuretic and works as an excellent remedy for urinary conditions such as retained urine, burning urine, kidney stones, bladder infections, gonorrhea, and as a lymphatic system cleanser. Corn Silk is used to treat bladder infections, kidney stones, infections of the prostate gland, and urinary infections.

References (27, 28, 29)

#### Uva ursi (Arctosyaphylos uva ursi)

The chief constituent of <u>Uva Ursi</u> is a glycoside called arbutin. This is what is responsible for its diuretic action. During its excretion arbutin produces an antiseptic effect on the urinary mucous membrane and can therefore help eliminate urinary tract infections. Tannic acid is also contained in the leaves. This herb also helps to keep the pH balance of urine from being too acid. It actually strengthens the lining of the urinary tract and helps to relieve any inflammation in the system. It has a direct sedative effect on the bladder walls. Allantoin, also found in Uva Ursi spurs the healing of wounds. For chronic inflammation of the bladder or kidneys <u>Uva Ursi</u> has no equal. Two studies report that urine from individuals given uva ursi is active against the most commonly involved bacteria in bladder and urinary tract infection.

This study supports the results of a double blind study of 57 women with recurrent cystitis. After one year, the placebo group had 20% incidence of recurring cystitis, whereas the **uva ursi** group had no recurring infection.

In addition it has anti-lithic properties that help in dissolving crystals not just in the kidneys, but throughout the body as well. It has, therefore, been used for arthritis and other painful joint problems.

References (30, 31, 32, 33)

#### Parsley root (Petroselinum crispum)

An important diuretic, <u>parsley root</u> also helps clear uric acid from the urinary tract and helps dissolve and expel gallstones and gravel – and prevent their future formation. It also inhibits the secretion of histamine and is therefore useful in treating hives and relieving other allergy symptoms. A decoction of <u>parsley root</u> can help eliminate bloating and reduce weight by eliminating excess water gain. Note: the German Commission E, an advisory panel on herbal medicines, has approved parsley for use in the prevention and treatment of kidney stones.

References (34, 35)

#### Carrot tops (Daucus carota)

<u>Carrot tops</u> are an under-appreciated source of relief for all sorts of urinary tract problems and symptoms. The Amish swear by it, dedicating significant acreage to carrots just for this reason. <u>Carrot Top Tea</u> can clear up skin blemishes, flush the kidneys and bladder, and clean the blood of toxins.

<u>Carrot tops</u> are also helpful in clearing the kidneys and urinary tract (as well as the prostate). They are also highly alkalizing to the blood, taking stress off the kidneys.

#### Dandelion leaf (Taraxacum officinale)

<u>Dandelion leaves and roots</u> have been used for centuries to treat liver, gall bladder, kidney, and joint problems. In some countries, <u>Dandelion</u> is considered a blood purifier and is used for ailments such as eczema and cancer. It has also been used to treat poor digestion, water retention, and diseases of the liver such as hepatitis.

<u>Dandelion leaf</u> is also a good natural source of potassium, and will replenish any potassium that may be lost due to the diuretic action of the other herbs in this formula.

Studies show beneficial effects of **dandelion** on reducing urinary tract gravel, attributed to disinfectant action and possibly the presence of saponins. Dandelion has also been used traditionally to treat respiratory disorders. Dr. James Duke notes in his book, The Green Pharmacy, that numerous clinical trials have demonstrated the efficacy of **dandelion leaves** and root for treating pneumonia, bronchitis and upper respiratory infections. Dr. Duke recommends drinking the juice that remains after the greens have been cooked. The German Pharmacopoeia lists **dandelion leaf and root** for treating gastrointestinal complaints stemming from bile deficiency, as well as to stimulate appetite and diuresis. **Dandelion** was also used in folk medicine to ease painful joint and bone conditions. The tea reduces water retention and is considered a traditional blood purifier. The diuretic effect is also useful for reducing swelling.

References (36, 37, 38)

#### Horsetail (Equisetum arvense)

Horsetail has not been extensively studied in people, but professional herbalists recognize that the herb has diuretic (promotes the excretion of urine) properties that may be useful for the following health problems:

- Urinary tract infections
- Kidney stones

References (39, 40, 41, 42, 43)

#### Orange peel - detox

Limonene and flavonoids found in <u>orange peel</u> seem to have anti-carcinogenic properties. They can block the carcinogenesis by acting as a blocking agent. Studies have shown that limonin and limonene can induce the enzyme activity of <u>glutathione</u> S-transferase, <u>which is an important</u> <u>detoxifying enzyme.</u>

In addition, orange peel has antiseptic, bactericidal, and fungicidal properties.

References (44, 45, 46)

#### Peppermint (Mentha piperita)

<u>Peppermint</u> has a relaxing effect on the muscles of the digestive and urinary system. It is useful for treating spasm problems in the urinary tract. It also has strong antibacterial and anti-fungal properties which help rid the kidneys of bacteria.

Three double-blind trials found that enteric-coated peppermint oil reduced the pain associated with intestinal spasms, commonly experienced in IBS.

References (47, 48, 49)

### Goldenrod (Solidago virguarea)

**Goldenrod** is used as an aquaretic agent, meaning that it promotes the loss of water from the body (as compared to a diuretic, which promotes the loss of both water and electrolytes such as salt). It is used frequently in Europe to treat urinary tract inflammation and to prevent or treat kidney stones. In fact, goldenrod has received official recognition in Germany for its effectiveness in getting rid of kidney stones, and itis commonly found in teas to help "flush out" kidney stones and stop inflammatory diseases of the urinary tract. Goldenrod is said to wash out bacteria and kidney stones by increasing the flow of urine, and also, soothe inflamed tissues and calm muscle spasms in the urinary tract. It isn't used as a cure in itself, but rather as an adjunct to other, more definitive treatments such as (in the case of bladder infections) antibiotics.

Several studies have found that goldenrod does in fact increase urine flow.

*References* (50, 51, 52, 53, 54, 55)

#### Who should use this formula and when

Since everyone accumulates sludge in their kidneys and livers, everyone is a candidate for regular use of this formula. Even people who have had their gallbladders removed will benefit. Regular softening and flushing of stones and gravel will keep your kidneys, liver, and gallbladder functioning at optimum levels and, more importantly, keep areas of those organs from choking to death and becoming non-functional. The sooner you start in life the better, but certainly the older you get, the more mandatory regular use becomes.

And as for anyone already suffering from painful kidney stones or gallstones, this formula can be a godsend. Whereas medical doctors can offer only surgery or expensive <u>lithotripsy</u> procedures (which are also not without risk), this formula offers a safe, highly effective alternative – that can work with remarkable speed. Painful kidney stones and gallstones can usually soften enough for easy passage in as little as 2-8 days. And regular use of the formula can prevent any recurrence.

#### How to use

Simple

Use 4-8 droppers in diluted juice three times a day until bottle is gone.

(MAK I use a lower amount of any treatment, and take longer, to prevent any disturbance in the overall formula, I would take a lower amount!!)

#### Better

Take 4 ounces of this formula and mix with a quart of fresh squeezed apple juice (not bottled) and a quart of water. Drink a pint each day over 4 days.

For most people, doing this program twice a year should be enough to keep the kidneys functioning properly. An ideal time to do this program is shortly before doing a liver detox. Again, the same herbs that soften kidney stones for easy passage will also soften gallbladder stones. Using this formula shortly before doing the liver detox will greatly reduce the likelihood of discomfort when doing the liver detox.

For those who have a predilection to getting kidney stones or gallstones, this program can be done once a month to minimize the chances of any future occurrence.

If you have currently existing painful kidney or gallstones, you probably will want to mix up two batches and drink it for 8 straight days.

Do not do more than once a month on a regular basis as the diuretic effect may deplete the body of essential water soluble vitamins and minerals over time.

Note: If using in preparation for the liver detox, make sure you use within 30 days of starting the detox so the gallstones don't get a chance to reharden before flushing.

**Warning**: The diuretic effects of this formula may enhance the toxic effects of certain medications, such as digoxin (used to treat congestive heart failure), phenytoin (for seizures), anticoagulants, and others. For this reason, people taking prescription medications should not use this formula without first consulting a health care provider. Also, anyone with severe kidney problems should not use this formula without first consulting their physician.

#### **Testing the formula**

There was never any question that this formula would work for many people. I've used variations and pieces of it for years, and some of the herbs have been time tested over decades, if not several centuries. But I wanted to see if this version of the formula was indeed stronger than anything I had put together before – after all, that was the intent. I needed an extreme test case. I decided to test it with Patty C., someone with a known, long-standing kidney condition, to see if it would help...and do so quickly. If you're interested in reading her experiences with the formula, click here.

#### Introduction

The functional tissue of the kidneys is delicate and can be damaged not only by disease, but also by drugs, especially anti-cancer chemotherapy (notoriously the platinum compounds) as well as some immunosuppressant and antibiotic agents. Some herbal ingredients are also documented as nephrotoxic.

Medically, laboratory values of creatinine in a blood sample are used to check kidney function; supranormal creatinine values reflect impaired kidney function due to damage to the renal tissue. Short term, recovery of levels can occur if the cause is rapidly rectified, but persistently elevated creatinine is considered indicative of lasting mechanical damage and does not usually return the previous normal range.

**Stinging nettle \*seed \*** is not at all known outside the herbalist community. Trained herbalists regularly work with hundreds of herbal remedies, the majority of which lack mainstream scientific or clinical data. Restoration of kidney function due to mechanical damage is traditionally not considered possible, either by either conventional or herbal medicine. The following case reports show normalization of elevated creatine levels using **nettle seed** as the primary herbal remedy.

Employing beneficial herb-drug interactions is a consistent theme of my practice that is well illustrated by incorporating **nettle seed** into renal-protective protocols for cancer patients undergoing chemotherapy with platinum drugs. Monitoring creatinine before during and after platinum chemotherapy has consistently demonstrated the value of this approach, often to the surprise of the oncologists involved.

Urtica semen reduces serum creatinine levels.

### **Case History Nettle Seed & Kidney Function Jonathan Treasure**

Case History: Nettle Seed & Kidney Function

#### Introduction

### **Stinging Nettle Seed Extract**

Urtica dioica L. and Urtica urens L., (**stinging nettles**) have a long history of use in folkloric and science based herbal medicine. Traditionally used as a nutritive and "blood cleanser" or alterative agent, a substantial pharmacological and clinical literature supports its use for arthritic and allergic conditions (leaf/herb) and improving urological symptoms of benign prostatic hyperplasia (root). The available literature is based on either the aerial parts (folia/herba), or the root (radix). The root is monographed by WHO, ESCOP and Commission E, and the herb/leaf is monographed by ESCOP and Commission E.1-4 However none of these, or other pharmacopoeial sources identify the seed (semen) as a medicinally distinct plant part.

The literature on constituents or pharmacology of nettle seeds is sparse. An HPLC analysis of the lipid fraction indicates the presence of a high proportion of unsaturated fatty acids, especially palmitic, and a small amount of omega-3 unsaturated fatty acids.5 Of interest is the presence of a lectin in the seeds of U. pilulifera, a Turkish stinging nettle.6 A lectin from the roots of U. dioica known as Urtica dioica agglutinin (UDA) is known as a novel T-Cell mitogen with superantigenic properties.7 UDA has an unique pattern of T-cell activation and cytokine induction which has led to its use as a probe in investigations of superantigen activity.8

Whether the seeds of U. dioica also contain a lectin with UDA superantigenic properties is unknown due to the absence of research.

The clinical use of nettle seed extracts for treatment of renal dysfunction represents a novel indication for the herb, first suggested by North American herbalist David Winston and to date unsupported by published reports.9 The following two cases involve the use of nettle seed extracts in patients with serious renal challenges, and utilize serum creatinine laboratory values data to serially monitor the effects of the extract.

#### **Serum Creatinine**

Serum creatinine is commonly measured as an index of glomerular function. Broadly speaking serum creatinine varies inversely with the glomerular filtration rate (GFR), other things being equal. A number of non-renal factors may affect creatinine levels, ranging from heavy dietary meat ingestion, medications including glucocorticoids and cimetidine, and muscle mass decrease in chronic illness. There is also a small but variable degree of proximal tubule secretion of creatinine, which declines as GFR decreases and which may confound interpretations of creatinine values. In cases where renal function and GFR require more accurate evaluation, inulin, or radionuclide labelled molecules such as 125I-iothalamate are used. Nonetheless, creatinine remains a simple single measure test of nephron function. Units of measurement vary in laboratory reports, upper limits for of normal serum concentration in men are 1.2mg/dL (conventional units), or 110 micromol/L (SI units). Values for women are ~85% men's values.

#### Postscript 2004

Both patients were impressed with the effects of the Urtica semen. Both continued to do well with apparently stable renal function unaided by continued herbal support. The nephrectomy patient recently experienced an increase in creatinine levels following major surgical procedure. The allograft patient continues to pursue an active career in performing arts in the Pacific Northwest.

#### **Nettle seed extract**

The extract of nettle seed used in the above case series was a standard tincture 1:5, 30% EtOH, supplied by Herbalist & Alchemist Inc. 51 S Wandling Avenue, Washington, NJ. 07882-3537.

#### **American Journal of Kidney Diseases**

Volume 61, Issue 3, Pages 501–513, March 2013

Reviewing the evolution of human knowledge for these parasites discloses a lot of similarities regarding their discovery, patterns of kidney injury, and pathogenic mechanisms. From a historical perspective, these parasites discloses a lot of similarities regarding their discovery, patterns of kidney injury, and pathogenic mechanisms. From a historical perspective, our relevant information may be classified into 4 phases: (1) disease documentation in ancient and medieval scripts as far back as 2000-3000 bce; (2) discovery of the parasites, their life cycles, and clinical correlates by European clinicians working in African and Asian colonies during the second half of the 19th century; (3) discovery and characterization of the renal manifestations of monoparasitic infections during the second half of the 20th century; and (4) recognition of the confounding effects of coinfection with bacteria, viruses, or other parasites. The spectrum of respective kidney diseases extends all the way from acute kidney injury to glomerulonephritis, amyloidosis, urologic disorders, and malignancy. Discovery of the common immunopathogenetic host response to parasitic infections has provided a knowledge core that explains the similarities, diversities, and interactions with regard to kidney injury.

#### **First Line of Defense**

The first line is to maintain a constant state of over-hydration from morning to 6 PM. Good water is the best detox agent available and cannot be underemphasized.

Ganoderma extract has been found to be useful in **detoxifying the kidneys** and improving its overall function.

#### **Current Kidney Support Formula**

CQ10

#### Milk thistle

KGP flush

New studies show P5P and piridoxamine help kidneys function in the presence of severe damage. Chinese medicine - Beets, blackberries and rhubarb can turn your urine red or pink,

Keep us safe from the harmful effects of internal (and external) toxins by filtering blood of the natural by-products of metabolism, drugs, and other toxins;

**B6** 

B6 move to b section, add section to remove b2 for protozoan infection, P5P not really required, test this version of B6

### PYRIDOXAMINE | C8H12N2O2

http://www.amazon.com/PrimAGE-pyridoxamine-200-60-capsules/product-reviews/B009JMF000 http://www.ncbi.nlm.nih.gov/pubmed/17224327

Pyridoxamine, especially at 400 mg/kg per day, improved the levels of urinary ACR, fasting serum TG, and 3DG. CML and nitrotyrosine accumulations in glomeruli were decreased.

**Piridoxamine** 

Synonyms: PM

Superclasses: <u>a vitamin</u> → <u>a vitamin B6</u>

Chemical Formula: C<sub>8</sub>H<sub>13</sub>N<sub>2</sub>O<sub>2</sub>

Molecular Weight: 169.2 Daltons

Monoisotopic Molecular Weight: 169.0977026719 Daltons

SMILES: CC1(=NC=C(CO)C(C[N+])=C(O)1)

InChI: InChI=1S/C8H12N2O2/c1-5-8(12)7(2-9)6(4-11)3-10-5/h3,11-12H,2,4,9H2,1H3/p+1

InChIKey: InChIKey=NHZMQXZHNVQTQA-UHFFFAOYSA-O

Unification Links: CAS:85-87-0, ChEBI:57761, HMDB:HMDB01431, IAF1260:35277,

KEGG:C00534, MetaboLights:MTBLC57761, PubChem:25245492

Standard Gibbs Free Energy of Change Formation ( $\Delta_f G^{\circ}$  in kcal/mol): 78.50251 $\boxed{\text{Latendresse13}}$ 

Reactions known to consume the compound:

pyridoxal 5'-phosphate salvage I, pyridoxal 5'-phosphate salvage II (plants) :  $ATP + pyridoxamine \rightarrow ADP + pyridoxamine 5'-phosphate + H^+$ 

Not in pathways:

<u>pyridoxamine + oxygen + H2O → pyridoxal + ammonium + hydrogen peroxide</u>

Reactions known to produce the compound:

pyridoxal 5'-phosphate salvage II (plants) : pyridoxamine 5'-phosphate +  $H_2O \rightarrow pyridoxamine + phosphate$ 

Reactions known to both consume and produce the compound:

vitamin B<sub>6</sub> degradation :

<u>pyruvate + pyridoxamine ↔ L-alanine + pyridoxal</u>

Not in pathways:

<u>pyridoxamine</u> + oxaloacetate ↔ pyridoxal + L-aspartate

In Transport reactions:

<u>pyridoxamine</u>[periplasm] → <u>pyridoxamine</u>[cytosol]

Enzymes inhibited by pyridoxamine, sorted by the type of inhibition, are:

Inhibitor (Competitive) of: <a href="mailto:pyridoxal kinase">pyridoxal kinase</a> [White70]

This compound has been characterized as an alternative substrate of the following enzymes: pyridoxal kinase, hydroxymethylpyrimidine kinase

http://biocyc.org/META/NEW-IMAGE?type=COMPOUND&object=PYRIDOXAMINE

http://www.iherb.com/Nature-s-Way-Vitamin-B-6-100-Capsules/1823

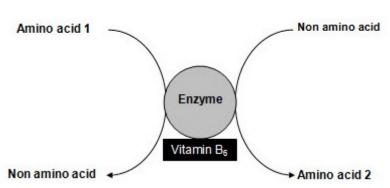
http://www.vitaminshoppe.com/p/allergy-research-nutricology-pyridoxine-p5p-vitamin-b6-275-mg-60-capsules/ar-1051#.VnoW4k\_VRqA

https://en.wikipedia.org/wiki/Pyridoxine

http://www.thenutritiondr.com/vitamin-b6-or-pyridoxal-pyridoxine-pyridoxamine-phosphatesfood-sources-of-vitamin-b6roles-of-vitamin-b6rdas/

HO OH

Vitamin B<sub>6</sub> is the general name for six compounds pyridoxal (PL), pyridoxine (PN), pyridoxamine (PM), and their phosphate derivatives including



pyridoxal 5'-phosphate (PLP), pyridoxine 5'-phosphate (PNP), and pyridoxamine 5'-phosphate (PMP). It is the PLP form that is the most significant in human operations. While long recognized for its pivotal role in the processing of amino acids, vitamin B6 has received attention for its role in homocysteine metabolism and reducing cardiovascular disease risk.

Vitamin  $B_6$  can be found in nearly if not all cells throughout the body with higher concentrations found in muscle and liver tissue. Similar to most of its water-soluble vitamin siblings, vitamin  $B_6$  is primarily lost from the body in urine. Once inside the cells, vitamin  $B_6$  forms can be converted to the active forms of vitamin  $B_6$ , PLP (pyridoxal phosphate) and PMP (pyridoxamine phosphate). PLP and PMP are key participants in many cell reactions. By and large the most significant roles of vitamin  $B_6$  are:

# VITAMIN B6(91% AS PYRIDOXINE HYDROCHLORIDE AND 9% AS PYRIDOXAL-5'-PHOSPHATE)

This formula is a combination of pyridoxine hydrochloride and pyridoxal-5-phosphate (P-5-P, the coenzyme or "activated" form of B6). As P5P, vitamin B6 is extensively involved in the metabolism of amino acids, lipids and nucleic acids. Supplementation with vitamin B6 as P5P can be helpful for those unable to produce it efficiently from pyridoxine in the liver.\* Excessive protein intake, alcohol or contraceptive use also indicates a greater need for vitamin B6. Vitamin B6 plays a crucial role in the multiplication of cells.\*

Amino acid metabolism- Vitamin  $B_6$  is crucial for the processing of amino acids including the production of nonessential amino acids made from other amino acids. During this process, the nitrogen-containing amine portion of an amino acid is transferred to a specific molecule (see Vitamin  $B_6$  and Amino Acids Figure), which creates a nonessential amino acid. In fact, if an individual developed a vitamin  $B_6$  deficiency, most of the nonessential amino acids would actually become dietary essentials.

- Glycogen breakdown Glycogen breakdown in muscle requires vitamin B<sub>6</sub>. Glycogen is stored glucose and the breakdown of this complex provides invaluable fuel during exercise and work.
- Neurotransmitter production Vitamin B<sub>6</sub> is also necessary to convert certain amino acids into neurotransmitters gama-amino-butyric acid (GABA) and serotonin.
- Hemoglobin- Vitamin B<sub>6</sub> is crucial for the normal production of hemoglobin, the O<sub>2</sub> carrying protein found in RBCs.

Immunity- In addition, vitamin B<sub>6</sub> is essential in the formation of hemoglobin and white blood cells. Finally, vitamin B<sub>6</sub> also seems to be necessary to break down glycogen stores during exercise and fasting.

P5P does not have this issue. Pyridoxine actually has this issue because it is similar to P5P and binds at the receptor at higher doses blocking endogenous p5p... ironically. Very high doses of P5P have demonstrated zero toxicity and it is the coenzymated form. Moral of the story synthetic, crap versions of vitamins in your total cereal are not the same as what's found in food or your body. They can not and should not be lumped in together and to do so is quite erroneous. Pyridoxamine is the other coenzyme form available besides p5p. Both should have no neurotoxicity associated with them.

https://en.wikipedia.org/wiki/Pyridoxamine

http://www.longecity.org/forum/topic/38112-vitamin-b6-pyridoxamine-neurotoxicity/

#### What's the difference between vitamin B6 and its vitamers? Here's a quick overview:

- Pyridoxine is the "storage form" of vitamin B6, the kind that is found in whole grains, seeds, and other plant foods. Pyridoxine is the most shelf-stable form of the vitamin.
  The body activates it by changing it to pyridoxal 5-phosphate.
- **Pyridoxal** is another "storage form" of vitamin B6, the kind that is found in meat, fish, and dairy products. The human body can also change this form of vitamin B6 into the active form of the vitamin, pyridoxal 5-phosphate.
- **Pyridoxamine** is a chemical combination of pyridoxine and pyridoxal. The body can break it down it into pyridoxine and pyridoxal and then into the active form of B6.
- **Pyridoxal 5-phosphate** is the form of B6 the body actually uses. Strictly speaking, there is no such thing as a recommended pyridoxal 5-phosphate dosage or a pyridoxal 5-phosphate deficiency, since our bodies make this specific chemical. Dosages are measured for the pyridoxine that is most commonly found in food.

Pyridoxamine, however, has some functions in the body that pyridoxine and pyridoxal do not. Pyridoxamine is a chelating agent. It can interact with and trap heavy metals. And, more importantly, it can block the aging effects of sugar.

# **Pyridoxamine and Reversing the Aging Process**

One of the causes of aging, especially in the skin, is the formation of "advanced glycation end-products." Any cell in the body can become essentially sugar-coated by interaction with glucose from the bloodstream. In diabetics, of course, the problem is much worse, since there is more sugar in the bloodstream to "caramelize" cells.

The sugar on these cells is not a problem until it begins to break down. The advanced glycation endproducts release tremendous amounts of free radicals, so many that the cell's energy center, the mitochondria, "burn out" when they use oxygen to make energy. The cell dies, and when enough cells die, tissues and organs don't function as well.

This is the underlying chemical process beneath wrinkling, cataracts, atherosclerosis, certain kinds of kidney failure, the complications of diabetes, and possibly Alzheimer's disease (although there are other kinds of cell destruction also involved in Alzheimer's). Stopping the formation of advanced glycation end products, if started soon enough, stops these diseases of aging.

That's where pyridoxamine comes in. It stops a kind of chemical reaction known as the Maillard reaction. You may not know the chemistry, but you have definitely seen a Maillard reaction in the real world. This combination of sugars and amino acids is what makes toast brown, or tater tots crunchy when they've been fried, or puts the "burn" on roasted meat or barbecue, or gives maple syrup its brown color. At a cellular level, pyridoxamine keeps your cells from becoming toast.

It's so useful you would think someone might try to patent it. And someone did.

First, P5P = PLP is converted to pyridoxal (PL) in the gut before absorption. So what is interesting is the differences between pyridoxal (PL), pyridoxine (PN), and pyridoxamine (PM).

Institute of Medicine: Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline (1998): "PN at high doses raises the plasma PLP concentration and is retained more effectively than is PL (Shane, 1978). Similarly, dietary pyridoxamine (PM) and PL are about 10 percent less effective than PN in raising the plasma PLP concentration, and slightly more of these vitamins is excreted in the urine as 4-pyridoxic acid (4-PA) (Wozenski et al., 1980). Most controlled B6 studies have used PN as the added B6 source, but requirements calculated from these studies would underestimate the B6 requirement by only 5 percent or less for individuals deriving most of their B6 as PLP and PMP from animal sources."

So PN is actually slightly better than the other forms at raising plasma PLP (P5P) in a normal population. That may be different in patients with severe disease, especially liver disease, as suggested by the studies mentioned in the Thorne report.

Pyridoxamine (PM) has some interesting research and looks better than the other forms at least in vitro. However, it has a rather short half-life before conversion to PLP. Not sure how much in vivo research, where it is taken intermittently, there are showing it is better than the other forms.

Now regarding the central question of toxicity. As far as I know there is no research showing that the non-pyridoxine forms are less toxic. That pyridoxine itself should be damaging as compared to PLP or its metabolities seems just to be an unproven speculation. That most cases of neurotoxicity have been seen from pyridoxine is only to be expected since it the commonly used form. Does anyone have an actual study showing less neurotoxicity for any of the non-pyridoxine forms?

**Pyridoxamine** is one form of <u>vitamin B</u><sub>6</sub>. Chemically it is based on a <u>pyridine</u> ring structure, with <u>hydroxyl</u>, <u>methyl</u>, <u>aminomethyl</u>, and <u>hydroxymethyl</u> <u>substituents</u>. It differs from pyridoxine by the substituent at the 4-position. The <u>phenol</u> at position 3 and aminomethyl group at position 4 of its ring endow pyridoxamine with a variety of chemical properties, including the <u>scavenging</u> of <u>free radical</u> species and carbonyl species formed in sugar and lipid degradation and <u>chelation</u> of metal ions that catalyze <u>Amadori reactions</u>.[1]

Pyridoxamine can form fairly weak complexes with a number of transition metal ions, with a preference for  $\underline{\text{Cu}}^{2+}$  and  $\underline{\text{Fe}}^{3+}$ .[2] The 3'-hydroxyl group of pyridoxamine allows for efficient <u>hydroxyl radical</u> scavenging.[2]

Pyridoxamine inhibits the <u>Maillard reaction</u> and can block the formation of <u>advanced glycation</u> <u>endproducts,[3]</u> which are associated with medical complications of <u>diabetes</u>.[4] Pyridoxamine is

hypothesized to trap intermediates in the formation of <u>Amadori products</u> released from <u>glycated proteins</u>, possibly preventing the breakdown of glycated proteins by disrupting the <u>catalysis</u> of this process through disruptive interactions with the metal ions crucial to the <u>redox reaction.[5]</u> One research study found that pyridoxamine specifically reacts with the <u>carbonyl</u> group in Amadori products, but inhibition of post-Amadori reactions (that can lead to advanced glycation endproducts) is due in much greater part to the metal <u>chelation</u> effects of pyridoxamine.[1]

A variety of preclinical studies in <u>animal models</u> of diabetes indicated that pyridoxamine improved <u>kidney histology</u> comparable or superior to <u>aminoguanidine</u>.[5] Because of these results, pyridoxamine has been investigated for clinical utility in the treatment of <u>diabetic nephropathy</u>.[5][6]

Pyridoxamine also inhibits the formation of advanced lipoxidation endproducts during <u>lipid</u> <u>peroxidation</u> reactions by reaction with di<u>carbonyl</u> intermediates.[7] In other preclinical research, pyridoxamine may be efficacious in treating <u>diabetic neuropathy</u> and <u>retinopathy</u> associated with diabetes[5][7] and <u>kidney stone</u> disease.[2] In one study, pyridoxamine was more effective at protecting from <u>ionizing radiation</u>-induced <u>gastrointestinal</u> <u>epithelial</u> <u>apoptosis</u> than <u>amifostine</u> (the only <u>radioprotector</u> currently <u>Food and Drug Administration</u> (FDA)-approved) due to pyridoxamine reactive oxygen species and reactive carbonyl species scavenging profile.[8]

### **Kidney functions:**

- Keep the blood balanced so that it is not too acidic or too basic;
- Balance water and electrolyte concentrations (minerals such as potassium, magnesium, sodium, and others), and control the amount of blood volume;
- Are one of the main controllers of blood pressure;
- Produce the hormone renin, which is responsible for helping to maintain proper blood pressure;
- Produce the hormone erythropoietin, which is responsible for creating new red blood cells; and partly manage bone health – vitamin D is changed from the inactive to active form within the kidneys, and is then able to signal the intestines to absorb more calcium, ensuring the availability of adequate amounts of bone-building calcium.

### **CQ10 Prevents Kidney Failure**

81% Positive Response to Coenzyme Q10 Treatment for Chronic Kidney Failure

Ninety-seven patients (mean age, 48 years) with chronic renal failure (serum creatinine > 5 mg/dl), with a history of declining renal function for at least 12 weeks, were randomly assigned to receive, in double-blind fashion, water-soluble coenzyme Q10 (CoQ10; 60 mg, 3 times per day orally) (Q-Gel) or placebo for 12 weeks.

The 45 patients who were receiving hemodialysis at the start of the study were encouraged to decrease the frequency or stop dialysis if there was an increase in urine output and a decrease in serum creatinine of more than 2 mg/dl. In the patients receiving hemodialysis and CoQ10, the mean serum creatinine concentration decreased from 9.5 to 6.7 mg/dl; mean BUN decreased from 88.2 to 79.8 mg/dl; mean creatinine clearance increased from 40 to 54.9 ml/min; and 24-hour urine output increased from 1,300 to 1,920 ml. Renal function tended to worsen in hemodialysis patients receiving placebo, and the differences in the changes between groups were significant (p < 0.01 to p < 0.001).

Significant improvements in each of these parameters relative to the placebo group were also seen in the non-dialysis patients treated with CoQ10. The number of patients receiving dialysis decreased from 21 to 12 in the CoQ10 group, and remained unchanged at 24 in the placebo group (p < 0.02). Eighty-one percent of the patients receiving CoQ10 had a positive response to treatment.

Comment by Alan R. Gaby MD:

These results suggest that hydrosoluble CoenzymeQ10 (Q-Gel) can improve renal function and reduce the need for dialysis in patients with chronic renal failure. The public-health implications of this study are enormous, considering that chronic renal failure is a serious and debilitating disease and that the annual cost of dialysis in the United States is more than \$22 billion.

According to Dr. Singh, lead author of this study (Interview with Kirk Hamilton; Clinical Pearls News, August, 2001, pp. 128-9), CoQ10 is usually effective if pre-treatment urine output, with or without furosemide, is at least 1,000 ml/day. However, if urine output is less than 500 ml/day, then CoQ10 usually does not work, presumably because the kidney has been irreversibly damaged.

Dr. Singh recommends that all patients with renal failure take 180 mg/day of water-soluble CoQ10 (Q-Gel) if their urine output is greater than 500 ml/day on dialysis. If urine output increases to 1,000 ml/day within 12 weeks, then CoQ10 is likely to be effective. Patients should be able to stop dialysis within 12-48 weeks if the urine output goes above 1,500 ml/day. If urine output does not increase in 12 weeks, then CoQ10 is unlikely to be effective.

While the mechanism by which CoQ10 improves renal function is not clear, it may work by improving cellular bioenergetics. Large controlled trials are urgently needed.

[Thus we hypothesize that using the full Metabolic Optimizer protocol with Ubiquinol instead of standard CoQ10 may have an even greater effect.]

### Comment by LEF

In a randomized, double-blind, placebo-controlled trial, the researchers found CoQ10 treatment decreased progression and reversed renal dysfunction in a majority of patients with end-stage disease, many of whom were able to discontinue dialysis over the course of the 12-week trial. The report followed up on a pilot study the scientists published in 2000 involving a smaller number of subjects.

End-stage kidney disease produces marked organ contraction and progressive dysfunction, with corresponding increases in levels of serum creatinine and blood urea nitrogen. Levels of toxic waste products accumulate in the blood because the kidneys cannot clear them from the body.

Dr. Singh and his colleagues documented significantly lower levels of serum creatinine and blood urea nitrogen in the CoQ10-treated patients, with increases in creatinine clearance and urine output regardless of patient dialysis or baseline status. More significantly, only half the number of CoQ10 patients required dialysis at the end of the study when compared to subjects receiving placebo.

The researchers also reported considerable increases in the antioxidant vitamins E and C and beta-carotene in treated subjects, while plasma levels of oxidative stress such as

thiobarbituric acid reactive substances, diene conjugates, and malondialdehyde all fell dramatically.

Although one in five patients did not respond, the researchers concluded that CoQ10 Q-Gel supplementation improves renal function in end-stage patients regardless of dialysis status, and can delay or avert the need for dialysis. They suggested that higher doses than those used in their study (180 mg per day) might result in even greater improvement and response in others.

#### Reference:

Singh RB, et al. Randomized, double-blind, placebo-controlled trial of coenzyme CoQ10 in patients with end-stage renal failure. J Nutr Environ Med 2003;13:13-22.

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### Scott Olson, ND

According to Scott Olson, ND and author of Sugarettes, there are various herbs that can help cleanse the kidneys.

#### **Healing Herbs:**

Your kidneys don't have to go it alone. There are many natural herbs and vitamins that can work to help your kidneys negotiate our toxic world. Below are some examples.

- Tumeric A root that many of us know as a spice used in cooking, research on this powerful herb has blossomed in the last few years, and it has been discovered that tumeric aids the liver, helps ease the symptoms of arthritis and heart disease, and prevents and treats certain cancers. Tumeric also protects the kidneys from being damaged by drugs and other chemicals.<sup>2</sup>
- Ginger This herb is well known for calming the stomach, and being good for the heart. Ginger acts as an anti-inflammatory, and it protects the kidneys from damage by a variety of drugs and chemicals.<sup>3</sup>
- Garlic –The number of studies demonstrating the way in which Garlic is a boon to health could fill a small room. While most beneficial for the heart, Garlic is also great for the digestive system. It is both an antioxidant and anti-inflammatory.
   Garlic aids the kidneys by protecting them from damage at the hands of heavy metals such as lead and cadmium, and it helps reduce kidney-damaging high blood pressure. 4,5
- Aloe This herb is great for both digestive problems, and for offering the kidneys protection from the damage normally caused by diabetes.<sup>6</sup>
- <u>Milk Thistle</u> There is likely no better herb for the liver than <u>milk thistle</u>. And current research is beginning to illustrate how <u>milk thistle</u> helps the kidneys

function better,<sup>7</sup> and protects them from the damaging effects of a wide variety of insults.

- Glutathione If you want your kidneys to last a long time, make sure that you have enough glutathione in your body. Glutathione is the all body superantioxidant. It can be supplemented directly, or can be recharged in the body by taking enough vitamin C. Glutathione helps the kidneys detoxify many chemicals, and may even help to reverse chronic kidney disease.<sup>8,9</sup>
- Methionine The kidneys can be damaged by oxidants; the solution to this problem
  is to ensure there are enough <u>antioxidants</u> in the blood by supplementing with
  methionine, which also helps to lessen the effects of the overburdening of toxins on
  the kidney.<sup>10</sup>

### **Dr Clark's Kidney program**

http://www.drclarkstore.com/kidney-cleanse-by-dr-hulda-clark/?
hstc=753710.2660ad2a2ef8d10f2549b89b87e9481e.1448728649482.1448728649482.14
48728649482.1& hssc=753710.1.1448728649483& hsfp=2245633810

Dr. Clark Kidney Cleanse contains a synergistic blend of natural herbs: Hydrangea Root, Marshmallow Root, Gravel Root, Ginger Root and Uva Ursi. These carefully selected herbs provide a natural cleansing action. By combining several different complementary herbs we greatly enhance each ingredient's effectiveness. This cleanse also contains Magnesium Oxide which inhibits calcium from binding with oxalate and forming stones, Vitamin B6 to help maintain healthy kidneys, and Freeze-Dried Parsley to help flush out toxins by increasing urine flow.\*

Black Cherry Concentrate does more than just improve the taste of the Kidney Cleanse Tea (Hydrangea/Marshmallow/Gravel Root). While cherries provide a nutrient profile rich in vitamins and **antioxidants** that support the health of the whole body, they are also a good source of citrate, a derivative of citric acid. Citrate reduces levels of uric acid in the blood. High levels of circulating uric acid can contribute to kidney stone formation.\*

- dried hydrangea root (Hydrangea arborescens)
- gravel root (Eupatorium purpureum)
- marshmallow root (Althea officinallis)
- Black Cherry Concentrate, 8 oz. bottles are cheap
- Pinch vitamin B2 powder
- fresh parsley
- Goldenrod
- Ginger
- Uva Ursi: one capsule in the morning and 2 capsules in the evening.
- Vitamin B6 (250mg): one a day.
- Magnesium oxide, 300mg caps
- **Ginger** capsules, **ginger**, the most common cause of bacterial urinary tract infections, from adhering to the inner walls of the bladder.
- Magnesium oxide (300mg): one a day.

### **Dr Christopher's**

http://www.iherb.com/Christopher-s-Original-Formulas-Kidney-Formula-500-mg-Each-100-Veggie-Caps/9192

Christopher's Original Formulas, Kidney Formula, 500 mg

**Serving Size:** 2 Capsules **Servings per container:** 50

Amount Per % Daily Value

Serving

Proprietary Blend
Organic juniper berry, parsley root, organic & wildcrafted
marshmallow root, wildcrafted goldenseal root, organic uva ursi

leaf, wildcrafted lobelia herb, & organic **ginger** root.

https://www.herbdoc.com/k-b-formula-kidney-bladder.html

PMC3072992

# Modified Citrus Pectin Reduces Galectin-3 Expression and Disease Severity in Experimental Acute Kidney Injury

we experimentally modulated galectin-3 in folic acid (FA)-induced acute kidney injury utilising modified citrus pectin (MCP), a derivative of pectin which can bind to the galectin-3 carbohydrate recognition domain thereby predominantly antagonising functions linked to this role. MCP clearly reduced renal cell proliferation but did not affect apoptosis. Later, during the recovery phase at two weeks, MCP-treated mice demonstrated reduced galectin-3 in association with decreased renal fibrosis, macrophages, pro-inflammatory cytokine expression and apoptosis. Other renal galectins, galectin-1 and -9, were unchanged. Our data indicates that MCP is protective in experimental nephropathy with modulation of early proliferation and later galectin-3 expression, apoptosis and fibrosis. This raises the possibility that MCP may be a novel strategy to reduce renal injury in the long term, perhaps via carbohydrate binding-related functions of galectin-3.

MCP decreased renal mRNA and protein levels of galectin-3 at 14 days after FA injection, in concert with significantly improved renal fibrosis as assessed by reduced expression of multiple fibrotic genes. MCP had no effect on galectin-1 and galectin-9, which are also expressed in the kidney [33], [34], suggesting these effects do not result from MCP interactions with other galectins. The direct correlation of less galectin-3 with less renal injury is consistent with studies by Henderson and colleagues [35] wherein mice lacking galectin-3 have less fibrosis and decreased collagen and a-SMA expression seven days after UUO. In contrast, a recent paper reported that fibrosis severity was increased by day 14 post UUO in galectin-3 deficient mice [11].

We also observed a small but highly significant reduction in apoptosis with MCP in the later disease phase. This difference is likely to be biologically important because Coles and colleagues [45] previously demonstrated that small changes in measured apoptosis in the kidney actually reflect larger overall changes in cell death due to apoptotic cells being cleared so quickly. Galectin-3 and MCP have different effects on apoptosis: galectin-3 has a BH1 domain of BCL2 that can protect cells from apoptosis [46], [47] and transfection with the lectin makes T-cells resistant to this type of cell death [48]; MCP, in contrast, promotes apoptosis in angiosarcoma [49] and prostate cancer cell lines [31]. This pro-apoptotic effect of MCP is formulation-dependent, however, because alterations in pH and heat-treatment (as we used here to prepare the MCP) can abrogate these pro-apoptotic effects [50]. We suspect that our observed reduction in apoptosis is not directly related to MCP actions on galectin-3 but simply reflects the reduction in disease severity with the modified pectin leading to

less remodelling being required; in this case, decreased early proliferation might generate less 'unwanted' cells that need to be deleted by apoptosis later.

Oxidative Medicine and Cellular LongevityVolume 2013 (2013), Article ID 367040, 9 pageshttp://dx.doi.org/10.1155/2013/367040Research Article

Magnolia Extract (BL153) Ameliorates Kidney Damage

The results showed that inflammation markers (tumor necrosis factor-α and plasminogen activator inhibitor-1) and oxidative stress markers (3-nitrotyrosine and 4-hydroxy-2-nonenal) were all significantly increased in the kidney of HFD-fed mice compared to mice fed with a low fat diet (LFD). Additionally, proteinuria and renal structure changes in HFD-fed mice were much more severe than that in LFD-fed mice. However, all these alterations were attenuated by BL153 treatment, accompanied by upregulation of peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α) and hexokinase II (HK II) expression in the kidney. The present study indicates that BL153 administration may be a novel approach for renoprotection in obese individuals by antiinflammation and anti-oxidative stress most likely via upregulation of PGC-1α and HK II signal in the kidney.

Our data demonstrated for the first time that these alterations could be reversed by BL153 administration. Several previous studies about magnolia extract obtained similar conclusions. Purified from the Magnolia officinalis, magnolol was found to exert an anti-inflammatory property via repressing lipopolysaccharide-induced Toll-like receptor 4 expression, subsequent nuclear factor kappa-B (NF-κB), and MAPK signaling pathway in uterine epithelial cells [23]. Furthermore, in vivo study indicated that honokiol, another compound isolated from the Magnolia herb, modulated inflammation-associated cytokines, such as interleukin-1β, interleukin-6, TNF-α, and monocyte chemoattractant protein-1, through activation of NF-κB [24]. Besides anti-inflammation properties, Magnolia extract has also been found to take part in ameliorating oxidative stress through different pathways. For example, 4-O-methylhonokiol, a novel compound isolated from Magnolia officinalis, prevents the development and progression of Alzheimer's disease by improving oxidative stress through a p38 MAPK-dependent pathway [25]. The beneficial effects of magnolol on learning and memory abilities were reported to be associated with superoxide dismutase (SOD) restoration in a scopolamine-induced mouse model [26]. However, how does magnolia extract modulate kidney inflammation and oxidative stress in obesity status is still unclear.

Phytotherapy has been used for human disease as an alternative or complement to allopathic medicines for several centuries [8, 9]. One of the representative medicinal plants is the Magnolia genus, which is mainly distributed in East and Southeast Asia. Up till now, more than 250 kinds of ingredients have been isolated from the cones, bark, and leaves of the Magnolia genus, such as magnolol, honokiol, 4-O-methylhonokiol, and obovatol [10]. The medicinal use of this species is attributed to its different pharmacological effects, including anti-inflammation [11, 12] and antioxidative stress [13, 14].

Uva Ursi Leaf, Juniper Berry, Horsetail Herb, Burdock Root, Corn Silk, Parsley Root

# Knowledge Seeker:

You want these four products asap: <u>Dr. Schulze</u> s Kidney Tincture and his Kidney tea Cayenne Tincture & powder and Superfood

You can find them at Schulzes website here:

http://www.herbdoc.com/home\_1024x768.asp

DR. Richard Schulze 'S OFFICIAL WEBSITE Or you can find the equivalent products here:

### http://healthfree.com/index.html

https://www.herbdoc.com/5-day-kidney-detox-program.html

The herbs in Dr. Schulze's K-B Formula have been shown to have three useful actions to promote health—healthy urine flow, cleansing functions and dissolving calcium deposits.

Uva Ursi Leaf, Juniper Berry, Corn Silk, Horsetail Herb, Burdock, Parsley Leaf and Root and others in this formula have been used for centuries as diuretics, helping to create a clean and healthy environment within the urinary tract.

Some of these herbs, like Juniper Berry and Uva Ursi Leaf, are also powerful cleansers for your kidneys and bladder. Uva Ursi Leaf contains powerful phytochemicals, such as volatile oils, arbutin, **quercetin**, and mallic and gallic acids.

Arbutin supports a healthy and clean urinary tract. **Quercetin** protects the trillions of capillaries in the delicate kidney filtering system.

Mallic and gallic acids (the same as found in apples and apple cider vinegar) have long been used for kidney and bladder health.

The herbs in the K-B Tea have the identical action as the K-B Formula with the addition of Hydrangea Root and Gravel Root, which are specifics for dissolving excess minerals in the urinary system.

Cayenne Tincture contains the powerful phytochemicals Capsaicin and Oleoresin. Cayenne stimulates the blood flow so rapidly, powerfully and completely. It has been used to improve heart health and brain function, and to maintain healthy joints.

Your kidneys clean ALL of the blood in your body EVERY HOUR!

- 2 Your kidneys balance your body's fluid levels, stopping water retention, as well as dehydration.
- 3 Hypertension is the #1 reason Americans die of heart attacks and strokes. Your kidneys regulate your blood pressure!
- 4 Your kidneys are responsible for the assimilation of vitamins into your bloodstream.
- 5 Your kidneys regulate and control the production of red blood cells, which carry oxygen to every cell in your body.

do the ones you can but especially the 5 at the top cayenne etc)

especially do bean pod tea, juniper berry tea, parsley tea, and plantain --

#15 use at least one of these teas daily..maybe two

#16 very ill --juice fast

somewhat ill mostly raw diet -vegan healthy vegan diet..include as many of these foods as

possible..definitely use the recommended juices in 10,11, and 12

http://curezone.com/forums/fm.asp?i=1224134#i

Although he does not mention anything new, he did repost some of what he said about-it is possible to get off dialysis in his book *25 ways to have the cleanest kidneys* found here

http://issuu.com/gordonmorton/docs/dr.\_schulze\_25\_ways\_cleanest\_kidneys?e=1017808/2739059

on page 24 (subheading #14)

If you have not read this yet, please do It will give you hope.

http://naturalhealing.wikispaces.com/Dr.+Richard+Schulze

https://www.herbdoc.com/index.php/Publication.html?pgtotal=16&pubpage=09&pubtitle=2013-06 Publication

booklet https://www.herbdoc.com/index.php/Publication.html?pgtotal=16&pubpage=01&pubtitle=2013-06\_Publication

how to access his newsletters:

https://www.herbdoc.com/index.php/Education get on new publication

For his full word on -can one get off dialysis- see the book linked above 25 ways to have the cleanest kidneys. for an excerpt of that or for a hard copy of something to give someone, request or see his current newsletter links above called attack of the mechanical vampires page 9 '

http://www.curezone.org/forums/fm.asp?i=1884321#i

http://www.curezone.org/forums/fm.asp?i=1884325# ihttp://www.curezone.org/forums/fm.asp?i=1638651#i http://www.curezone.org/forums/fm.asp?i=1884331#i http://www.curezone.org/forums/am.asp?i=2044478&s=7#i121

#### UTI

Hydrangea, parsley, and dandelion leaves are diuretics. Parsley's seeds also contain a substance that is sometimes used to treat UTIs; it also helps reduce inflammation.

Horsetail is an astringent diuretic encouraging urine flow and halting bleeding from the urinary tract.

Marshmallow soothes inflamed areas and enhances immune function to help fight off unwanted bacteria. Mullein also is known for reducing urinary tract inflammation.

The leaves, flowers, and especially the seeds of nasturtium contain natural antibiotics that may be helpful in preventing UTIs. **Horseradish root** also contains an antibiotic substance along with a good dose of **vitamin C**, both of which may be helpful in treating this condition.

<u>Vitamins A and C</u>, richly found in the produce from your healing garden, are good for preventing UTIs. The vitamin A, usually in the form of beta-carotene in plants, helps cells form properly and maintains their integrity, making them more resistant to invasion from unfriendly bacteria. <u>Vitamin</u>

<u>C</u> not only helps the immune system with production of certain infection-fighters, but in large amounts it can increase urine's acidity. The bacteria that cause this infection do not survive well under acidic conditions, so <u>vitamin C</u> tends to inhibit them.

However, it takes more <u>vitamin C</u> than can be obtained from foods. At least 5,000 mg of this nutrient are needed to significantly acidify the urine; one <u>orange</u> contains about 60 mg.

**Do not use goldenseal, Oregon grape**, or **uva ursi** if you are pregnant or nursing. Also, although a UTI may feel better, it's important to proceed cautiously. Unless you are quickly successful with herbal remedies, it's very important to seek medical care promptly.

### **DNA flush formula:**

DNA is in every cell of your body, the nucleus contains DNA, the instruction set for your body. The information in the DNA can be used for many purposes. Tiny building blocks called cells make up your entire body. There is DNA in nearly every cell, within a sack called the nucleus. The nucleus is like the brain of the cell. DNA is an endless march of characters in a 4-letter alphabet. If you pull it apart (the double helix), you would see the exposed ends of the strands, thefour different chemicals in the air. Those four chemicals, called "bases" carry the information used to make a body and to keep it running.

Weekly (unless more urgent) take in the AM:

2G **ALA** (4) alpha Lipoic acid 400-600mg NOW 3045B, 2G **CQ10** (4) CQ10 in D alpha E oil 400mg NOW 3198 2G **L Carnitine** (4) 500 mg Swanson SW1001

Few amino acids work for parasite infections, **I cysteine**, **taurine** with caution, not for continuous use.

>>> **Dangerous ornathine, arginine**. Temporary fix to a long-term problem. Will unbalance loops eventually and stimulate worm growth. Piperazine does a better job in low dose, along with **chaparral** for psora body types.

### **SIYO**

It's unwise to start fungal treatment until minerals areormalized; peripheral Body PH is 7.0++, and normal body enzymes in place. Failure of kidneys, liver, sepsis, or death could result. Initial 2004 trials in hospitals had fatality rates as high as 20% or higher when IV yeast/fungus kills were done in a few days in controlled environments. 2004 Dentistry association says survival rate of stage 4 Candidiasis is 50%. In 2007 the survival rate fell to 30%. Failure to kill candida in the brain was the primary cause of fatality. Starting a Yeast/Fungi kill inside your body without a full understanding of what you are doing is foolish. Seek out competent help.

### **Excessive Alkalinity Of The Intestinal Tract**

Individuals with candida overgrowth frequently have impaired digestive enzyme production.

It is not easy to identify Candida albicans overgrowth in the intestine. Proliferation of candida reportedly causes sugar and starch cravings because carbohydrates are the foods yeast ferment (feeds on). Fermentation causes bloating as the yeast rapidly give off gases. Diarrhea and/or constipation, abdominal pain and possible food intolerance's occur. If the yeast invades the

bloodstream, fatigue, anxiety, irritability, depression, difficulty concentrating and lethargy develop that can be extreme and life-threatening.

### Fungus:

Current pharmaceutical treatments meet with about a 50% success rate, usually done in combination. Esterol disruption, Beta Glucan therapy, and PH oxygen treatment strategies exist for killing fungus. No success or failure studies have been performed.

Once fungus is in check, DNA disruption and Immune system recovery can be attempted....

Chitin inhibitors show promise in changing the picture as well. There are several organizations that are studying natural plant and herb treatments around the globe. Contrast in vetro effectiveness and minimum dose requirements are being performed for a vast array of strains, to provide alternatives to physicians. <a href="Stinging nettle leaf">Stinging nettle leaf</a> AND STINGING NETTLE ROOT ARE EFFECTIVE in disrupting Chitin. There are many other effective substances. Fungal infections push TL2 immune regulators. <a href="LEM extract">LEM extract</a> is effective in restoring TL1 normal immune regulation.

#### IV

IV treatments antagonize the various structures within the yeast. Beta Glucan treatment does the opposite and stimulates the immune system, helping your own immune system to kill the yeast and fungus naturally. A combination of chitin inhibition, and beta Glucan supplementation show in a May 2013 study very promising results. The 1920,s Hydrogen Peroxide treatments have also shown promise.

Sacromeyces cerveste

Killing one fungus by using a better one has shown rapid alteration of the gut biodome. While it is possible to get a good fungus infection, it is rare. 5 million units for several weeks starves the food consumed by bad fungal infections, and can assist in clearing the GI tract.

**ROS** 

ROS production is initiated through assembly and activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase in phagocytes (Babior, 2004). This triggers the respiratory burst by generating superoxide anions ( $O_2^-$ ) (Schrenzel *et al.*, 1998), which are subsequently converted to hydrogen peroxide ( $H_2O_2$ ), hydroxyl radical (OH°) and hypochlorous acid, the latter conversion only taking place in neutrophils. In *C. albicans*, the Cat1 catalase has been implicated in counteracting the respiratory burst by protecting cells from killing by  $H_2O_2$  stress. Cells lacking Cat1 also display attenuated virulence in an invasive mouse virulence model as reviewed in Chauhan *et al.* (2006). Furthermore, the *C. albicans* genome harbours six genes encoding putative superoxide dismutases (SOD), four of which are copper-zinc (CuZn)-dependent, namely the cytoplasmic Sod1 and the cell surface Sod4, Sod5 and Sod6; two SODs, the mitochondrial Sod2 and cytoplasmic Sod3, are manganese-dependent (Chauhan *et al.*, 2006). SODs convert  $O_2^-$  into molecular oxygen and hydrogen peroxide, thereby scavenging the toxic effects of  $O_2^-$  and preventing higher  $H_2O_2$  levels by other downstream reactions (Teixeira *et al.*, 1998).

Like most organisms, fungi possess various antioxidant enzymes to counteract oxidative damage, including thioredoxin, glutathione reductase, catalase, gluthathione peroxidase as well as SODs. The genome of *C. albicans* encodes six putative SODs (*SOD1–6*, reviewed in <u>Chauhan et al.</u>, 2006).

The SODs are believed to destroy harmful superoxides produced by converting them first to  $H_2O_2$ ; subsequently catalase converts  $H_2O_2$  into harmless  $H_2O$  and  $O_2$ . We therefore hypothesized that deletion of an SOD gene should increase superoxide levels. Because the main

type of ROS detected by the luminol assay is peroxide but not superoxide, we measured superoxide levels using lucigenin as a luminescence probe (Li et al., 1998). Superoxide accumulation in BMDMs cocultured with the wild-type strain, as well as the  $sod4\Delta/\Delta$  strain, was similar. By contrast, the  $sod5\Delta/\Delta$  mutant showed a more than threefold superoxide

### **Hydrogen Peroxide**

http://www.ncbi.nlm.nih.gov/pubmed/18562577

Hydrogen peroxide induces the production of tumor necrosis factor-alpha in RAW 264.7 macrophage cells via activation of p38 and stress-activated protein kinase.

The effect of hydrogen peroxide (H(2)O(2)) on production of tumor necrosis factor (TNF)-alpha was examined in RAW 264.7 murine macrophage cells. H(2)O(2) led to production of TNF-alpha up to 24 h after the treatment, but not nitric oxide in RAW 264.7 cells. H(2)O(2) induced TNF-alpha production in mouse peritoneal macrophages as well as RAW 264.7 cells. The H(2)O(2)induced TNF-alpha production was prevented by inhibitors of p38 and stress-activated protein kinase (SAPK/JNK), and H(2)O(2) induced the phosphorylation of p38 and SAPK. Further, H(2)O(2) significantly augmented the AP-1 activity, but not nuclear factor (NF)-kappaB activity in RAW 264.7 cells. A high level of intracellular reactive oxygen radicals (ROS) was detected in H(2)O(2)-exposed RAW 264.7 cells. Ebselen, a cell permeable antioxidant, prevented the H(2)O(2)-induced TNFalpha production. H(2)O(2) significantly enhanced lipopolysaccharide (LPS)-induced TNF-alpha production. Therefore, H(2)O(2) was suggested to induce TNF-alpha production in macrophages via activating p38 and SAPK/JNK as oxidative stress-related signal pathways.

http://www.ncbi.nlm.nih.gov/pubmed/16790636

A small dose of hydrogen peroxide enhances tumor necrosis factor-alpha toxicity

Increases in apoptosis, Bax, lipid peroxidation product malondialdehyde, LDH, and decreases in Bcl-2, superoxide dismutase, and glutathione peroxidase were observed in TNF-alpha-treated cells. H2O2 10 microM did not cause significant lipid peroxidation (0.75 +/- 0.03 nmol/mg of malondialdehyde protein) as compared with control (0.70 +/- 0.04 nmol/mg of malondialdehyde protein) (P > 0.05) but further enhanced TNF-alpha-induced lipid peroxidation, upregulated Bax, and down-regulated Bcl-2 expression and enhanced TNF-alpha-induced cell apoptosis (P < 0.05). Propofol 50 microM attenuated TNF-alpha and H2O2-induced cell apoptosis, accompanied by decreases in malondialdehyde and LDH production and restoration of Bcl-2 expression.

H2O2 "Cute" - "John Hopkins "observations - ( ~ guidelines)

Legitimate medical health agencies are obliged to embrace the widespread scientific supportive findings regarding prooxidant hydrogen peroxide existent in the literature and encourage in depth scientific investigation into prooxidant EMOD disease protection and prevention. Scientific fact must supplant unsupported skepticism or flawed conclusions based on outdated data. While emphasizing hydrogen peroxide, this review is aimed at presenting the scientific facts regarding prooxidant EMOD health applications and cancer therapy. Based on the scientific literature currently available, EMOD use has significant potential benefits in the treatment of a wide range of human pathophysiologies. To deny the crucial role of hydrogen peroxide and prooxidant EMODs in normal metabolic processes and disease protection is to deny scientific truth.

- A retrospective review of all 95,052 exposures reported, 325 (.34%) were due to hydrogen peroxide. (- *safe*)
- Ingestion was the most common route of exposure (83%). (- drink it)
- 3% H2O2 has resulted in very few patients who developed serious complications or severe outcomes. (- food grade is not what you buy in the store!)
- There was a trend toward more severe outcomes in those who ingested a concentration greater than 10% (p = 0.011). (- dilute the 35% food grade to be less than 10 %)
- The level of lipid peroxidation in patients with cancer was significantly reduced.
- Proton Pumps"" Cytosolic acidification is an early event in apoptosis and provides an
  intracellular milieu permissive for efficient death execution. In this regard, exposure of
  cells to H2O2 or drugs that trigger intracellular increase in H2O2 results in a significant
  drop in cytosolic pH (- body should become alkaline, enzyme systems should recover).

Significant in vitro data exists showing that antioxidants can block EMOD-induced apoptosis for a wide variety of cancerous cell types, such as leukemia, lymphoma, retinoblastoma, myeloma, pheochromocytoma and human cancers of the breast, lung, pancreas, liver, colon, rectum and endometrium. 204 This data can not be ignored. However, it has recently been shown that EMODs may have an alternative activity, by modulating tumor cell signaling and that tumor cell signaling mediated by EMODs are readily reversible upon treatment with antioxidants. - "If you suppress free radicals, you suppress programmed cell death." ( - H2O2 balances antioxidant downsides, preventing vitamin supplementation from keeping mutated c DNA cells alive, increasing healing rate - many times X )

#### Hydrogen Peroxide and Flax Seed (Linseed): H2O2 and Linseed oil:

> Killing of intracellular fungi. The stimulation of iNOS depends on a balance of cytokines that are involved in the immunological response, such as T helper (Th)1, IFN- $\gamma$  and TNF- $\alpha$ , together with products released by the fungus. The variations observed in the immune response during PCM are dependent on the level of fungal suppression mediated by macrophages. There is little modulation of Th-1 type cytokines during the immune response to acute phase disease and Th-2 type cytokines are predominantly expressed during this phase, leading to the activation and differentiation of B lymphocytes into plasma cells and an increase in the levels of immunoglobulins, IL-4, IL-5 and IL-10. However, during chronic and disseminated PCM, there are high levels of TNF- $\alpha$ , IL-1 and IL-6 and a normal level of IL-4 (Sadahiro et al. 2007, Bernard 2008, Ramos & Saraiva 2008).

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3356939/

Sesamin: A Naturally Occurring Lignan Inhibits CYP3A4 by Antagonizing the Pregnane X Receptor Activation

An important cytochrome, cytochrome P450 3A4 (CYP3A4), is predominantly regulated by a nuclear receptor, pregnane X receptor (PXR). Sesamin, a major lignan constituent in **sesame seeds and oil**, exhibits a variety of biological functions; however, the effect of sesamin on the modulation of CYP3A4 is not well understood. In this study, the effects of sesamin on the PXR-CYP3A4 pathway were characterized, as well as the underlying mechanisms of those effects. Sesamin potently attenuated CYP3A4 induction in a dose-dependent manner by blocking the activation of PXR. The PXR inducer-mediated inhibition of CYP3A4 was further evidenced by the ability of sesamin to attenuate the effects of several PXR ligands in the CYP3A4 reporter assay. Further mechanistic studies showed that sesamin inhibited PXR by interrupting the interacting with coregulators. These results may lead to the development of new therapeutic and dietary approaches to reduce the frequency of inducer-drug interaction.

### https://en.wikipedia.org/wiki/CYP3A4

**Cytochrome P450 3A4** (abbreviated **CYP3A4**) (<u>EC 1.14.13.97</u>), is an important <u>enzyme</u> in the body, mainly found in the liver and in the intestine. Its purpose is to <u>oxidize</u> small foreign organic molecules (<u>xenobiotics</u>), such as <u>toxins</u> or drugs, so that they can be removed from the body.

While many drugs are deactivated by CYP3A4, there are also some drugs which are *activated* by the enzyme. Some substances, such as grapefruit juice and some drugs, interfere with the action of CYP3A4. These substances will therefore either amplify or weaken the action of those drugs that are modified by CYP3A4.

CYP3A4 is a member of the <u>cytochrome P450</u> family of oxidizing enzymes. Several other members of this family are also involved in drug metabolism, but CYP3A4 is the most common and the most versatile one. Like all members of this family, it is a <u>hemoprotein</u>, i.e. a <u>protein</u> containing a <u>heme</u> group with an <u>iron</u> atom. In humans, the CYP3A4 protein is encoded by the CYP3A4 <u>gene.[1]</u> This gene is part of a cluster of cytochrome  $P_{450}$  genes on <u>chromosome</u>

7q21.1 [2]

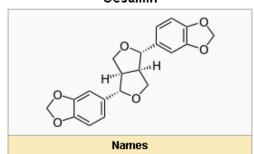
The CYP3A4 gene exhibits a much more complicated upstream regulatory region in comparison with its <a href="mailto:paralogs.[4">paralogs.[4]</a>. This increased complexity renders the CYP3A4 gene more sensitive to endogenous and exogenous PXR and CAR ligands, instead of relying on gene variants for wider specificity. [4] Chimpanzee and human CYP3A4 are highly conserved in metabolism of many <a href="mailto:ligands">ligands</a>, although four amino acids positively selected in humans led to a 5-fold <a href="mailto:benzylation">benzylation</a> of <a href="mailto:7-BFC">7-BFC</a> in the presence of the <a href="hepatotoxic">hepatotoxic</a> secondary <a href="mailto:bile-acid-lithocholic acid-lithocholic acid-lith

Sesame seeds (Sesamum indicum) and their oil have been used in human diets for thousands of years and are believed to provide health benefits. A major constituent of sesame is a lignan called sesamin. Sesamin has a variety of biological functions including reduction of serum and hepatic cholesterol levels [1–5] as well as serum triglycerides [6] by increasing hepatic fatty acid oxidation [1, 2]. Sesamin's involvement in the suppression of hypertension [7, 8] and stroke prevention has been extensively studied by many researchers [9]. Moreover, sesamin has been shown to elevate the levels of y-tocopherol [10];

decrease production of endotoxin-induced interleukin (IL)- $1\beta$ , prostaglandin E2 (PGE2), and thromboxane B2 [11]; elevate the production of IL-6 [12], thus inhibiting endotoxin-mediated shock [11].

Some studies also suggest that sesamin has an inhibitory effect on 7,12-dimethylbenz[a]anthracene-induced mammary carcinogenesis [13] and inhibits the growth of a

#### Sesamin



#### IUPAC name

5,5'-(1*S*,3a*R*,4*S*,6a*R*)-tetrahydro-1*H*,3*H*furo[3,4-c]furan-1,4-diylbis(1,3-benzodioxole)

variety of neoplastic cells through different mechanisms [14–18]. Thus, sesamin is believed to protect against cancer and other chronic diseases.

### https://en.wikipedia.org/wiki/Sesamin

**Sesamin** is a <u>lignan</u> isolated from the bark of <u>Fagara</u> plants and from <u>sesame oil</u>. It has been used as a dietary fat-reduction supplement, although no controlled studies on this application have been performed.[1] Its major metabolite is <u>enterolactone</u>, which has an elimination half life of less than 6 hours.[2] Sesamin and <u>sesamolin</u> are minor components of sesame oil,[3] on average comprising only 0.14% of the oil by mass.[4]

### **Killing Fungus**

### Caprylic acid

**Caprylic acid** is a natural substance, a fatty acid, but it is totally lethal to *most fungus*.

The success of Caprylic acid products in controlling intestinal yeast indicates another nutritional problem area for many individuals. Many people today suffer from poor fat digestion and utilization. This is related to a deficiency of digestive enzymes, but may also be adversely influenced by an inadequate diet, and inadequate fat metabolism. The underlying causes must go back to impaired liver function, low energy production, deficiency of essential minerals and disabling of critical enzymes due to toxic metals, etc.

Caprylic acid is actually a coconut oil extraction with 8 carbon atoms

Usually die off lasts only a few hours, though it can last several days. It can usually be controlled almost entirely by the amount of ingestion of the agent and the rate or frequency it is taken.

### **Undecenoic acid**

**Undecenoic** acid is actually caster bean oil extraction.

**Signs of Herxheimer reaction** can be many and varied but generally involve such discomfort as aching, bloating, dizzines, nausea, and overall "goopy sick" feeling, or a worsening of original symptoms. Fortunately, die off is generally short in duration, and although uncomfortable, is at least a confirmation of the presence of *candida* and that something "good" is happening.

#### **Yeast Strains**

There are over 2700 identified species, and 100 families of yeast.

http://www.annalsofintensivecare.com/content/1/1/37

Hospitalized Candidemia is associated with significant morbidity, which is reflected by a long ICU and hospital stay, ranging between one and several weeks [7,14]. The overall mortality in patients with invasive Candida infections is high: 42.6% in the EPIC II study [22]; 35.2% at 12 weeks in the PATH study [12]; 37.9% in the ECMM study [8]; and 53.4% in non-ICU vs. 85.9% in ICU patients in the Brazilian SCOPE study [11]. In the PATH study, the highest mortality has been reported in C. krusei infections (52.9%) and the lowest in C. parapsilosis infections (23.7%), whereas intermediate rates were reported for C. albicans (35.6%), C. glabrata (38.1%), and C. tropicalis (41.1%) [12]. Similar

differences were found in the ECMM and the French surveys [7,8]. Significant differences in mortality in age groups also were reported: 16.8% in patients 0-19 years of age, 31.3% in 19-65 years of age, and 52.7% in > 65 years of age [12]. Mortality higher than 80% was reported in candidemic patients with septic shock [23].

Delayed detection, or hospital admission for Candida infection statistics are not promising. Early detection and treatment are essential for survival.

### http://www.ncbi.nlm.nih.gov/pmc/articles/PMC387410/

Infections by pathogenic fungi, particularly Candida species, are both widespread and increasing in frequency (68, 276). Oral colonization by Candida albicans has been reported at 17.7% in the healthy population (40). Among hospitalized patients, oral carriage of Candida albicans rises to 40.6%. Healthy, asymptomatic women demonstrate an incidence of vaginal colonization by Candida of 15 to 20%. This percentage rises to 20 to 40% in healthy pregnant women and to 40 to 60% in human immunodeficiency virus-infected pregnant women (26, 209, 254, 273). Hospitals in the United States participating in the National Nosocomial Infection Survey System reported a nosocomial fungal infection frequency of 3.8 per 1,000 discharges in 1990, an increase from 2.0 per 1,000 discharges in 1980 (17). Candida species accounted for 78.3% of all such nosocomial fungal infections, followed by Torulopsis (now Candida) glabrata and Aspergillus species. C. albicans was the most frequently isolated of all the Candida species (17, 219). Furthermore, a European study reported that fungal species in general accounted for 17.1% of intensive care unit-acquired infections (306)

### Yeast (fungus) mutates the DNA of cells.

Cells start to burn sugar and not oxygen. This is called cellular ROS signaling. It is the last and most protective mode of a cell to maintain life. Once Redox signaling has started, the cell is virtually on its own, and has very few communications channels, enzyme pathways, or interactions with the outside world. It is very sick. It may be pre-cancerous or cancerous.

#### Salt

It is essential to ingest sufficient salt every day, it helps maintain signalling, membrane potential, processes.

#### Garlic

It is essential to ingest sufficient number of cloves of garlic every day. Garlic is one of the only sources of Germanium 132 required for white blood cell production.

#### Minerals

It is essential to ingest a number of multivitamin tablets from a quality supplier (NOW) every day.

### Copper

Bio-unavailable copper is indicated on a tissue mineral test by a copper level above 3.0 mg/% or below 1.0 mgs/%. Other mineral indicators of a candida overgrowth are an elevated calcium level, elevated calcium/magnesium ratio, (greater than 10/1) or a low sodium/potassium ratio (less than 2.3/1). When

copper is bio-unavailable, it cannot serve its normal function as a fungicide. Copper is involved in enzymes in cellular oxidative (aerobic) metabolism, and this appears to be the reason for its antifungal action. When copper is bio-unavailable, it cannot serve its normal function as a fungicide. Copper is involved in enzymes in cellular oxidative (aerobic) metabolism, and this appears to be the reason for its anti-fungal action.

#### **Zinc Chelate**

Zinc Deficiency -Zinc metabolism is closely related to Candida because 1) the zinc/copper

Balance is critical, and 2) zinc is required for many essential enzyme systems, including production of digestive enzymes and synthesis of all body proteins. A zinc imbalance is indicated on a tissue mineral chart by a zinc level less than 12.0 mgs/% or greater than 20.0 mgs/%, or a zinc/copper ratio greater than 12.0 mgs/%. A phosphorus level greater than 16 or less than 12 may also indicate a zinc imbalance. 30 mg zinc chelate supplement is normally recommended. Zinc is essential for white blood cell production.

Selenium Chelate

Brazil nuts and supplements

# **Chemistries**

**Thyroid supplementation** - several studies show that thyroid therapies can be very helpful. The main risks of thyroid treatment are:

- Triggering caffeine-like anxiety or palpitations. If this happens cut back the dose and increase by ½ to 1 tablet each 6 to 8 weeks (as is comfortable) or slower. Sometimes taking vitamin B1 (thiamine) 500 mg 1-3x day a day will also help after about a week. If you have severe, persistent racing heart, call your family doctor and/or go to the emergency room.
- 2. Like exercise (e.g., climbing steps), if one is on the edge of having a heart attack or severe "racing heart" (atrial fibrillation), thyroid hormone can trigger it. In the long run though, I suspect thyroid may decrease the risk of heart disease. If you have chest pain, go to the emergency room and/or call your family doctor. It will likely be chest muscle pain (not dangerous) but better safe than sorry. Increasing your thyroid dose to levels above the upper limit of the normal range may accelerate Osteoporosis. Fungus can accumulate on the heart valves, and small heart attacks are possible. Do not agressivly exersize. Walking for 45 minutes is best.

These individuals exhibit a mineral pattern referred to as slow oxidation, and/or a sodium/potassium ratio less than 2.3:1, which is indicative of adrenal burnout. Hormone boosing DHEA 50 mg, or **ENADA** 10 mg are usually prescribed.

**NADH** is known to trigger energy production by generating ATP (adenosine triphosphate) which stores energy in cells. If cellular levels of NADH are depleted, brain and muscle cells lose their ability to function effectively.

#### **Glutathione**

**Glutathione** is an amino acid and is available in vitamins. Unfortunately, free **glutathione** as a supplement is not absorbed by your cells.

### **Enzyme and low sodium potassium**

Individuals with **candida** overgrowth frequently have impaired digestive enzyme production.

This is indicated on the tissue mineral test by <u>low sodium and potassium</u> levels relative to calcium and magnesium levels, or a sodium-to-potassium ratio less than 2.3/1. Sodium and potassium are required in optimal amounts for production of hydrochloric acid in the parietal cells of the stomach. HCL production is impaired when tissue levels of these minerals are low. A 300 mg magnesium complex supplement is normally recommended.

A low sodium/potassium ratio is associated with a liver dysfunction, often accompanied by an inadequate secretion of bile acids. Bile acids also aid in maintaining intestinal pH and keep yeast organisms in check. Using salt (NaCl is usually recommended)

### **Excessive Systemic Alkalinity**

<u>Excessive systemic alkalinity is present in many Candida Albicans patients</u>. Increased systemic alkalinity <u>favors the spreading of the yeast infection from the intestines to other body tissues</u>.

Feeling tired and chilly, having constipation or losing your hair, can mean your thyroid is underactive-but these could also come from overwork, or aging, or a dozen other causes. Feeling totally tense and overworked could mean an overactive thyroid-or just plain stress. (see Thyroid above)

#### **HPA Axis**

Suppression of the hypothalamic pituitary adrenal axis (HPA axis). As a result of adrenal suppression, sufferers tend to put out less cortisol, and also less of another adrenal hormone called DHEA.

It seems that, if you try to replace the cortisol, it doesn't work. But replacing the DHEA seems to bring about a small improvement in some sufferers. The dose of DHEA is 25-50 mg for women, 50-100 mg for men, daily. It is best taken in the morning. DHEA is freely available without prescription.

### **Avoid B2**

Among the different eicosanoids, <u>both vitamins prostaglandin E<sub>2</sub> and thromboxane B<sub>2</sub></u> significantly <u>enhanced</u> serum-induced germination (30% more growth) and mutations (300%) of C. albicans.

### Lipid (oil and water)

### Impaired Short-Chain Fatty Acid Metabolism

The body converts short-chain omega-3 fatty acids to long-chain forms (EPA, DHA) with an efficiency below 5%

Alibicans sufferers usually are deficent in short chain fatty acids.

The Study of the effects of host long chain fatty acids, eicosanoids, and bacterial short chain fatty acids on control of germination.

None of the  $C_{18}$  or  $C_{20}$  fatty acids tested had an effect on enhancing germ tube formation (arachidonic acid, oleic acid, linolenic acid, or y-linolenic acid).

Among the different eicosanoids, both prostaglandin  $E_2$  and thromboxane  $B_2$  significantly enhanced serum-induced germination (30%) and mutations (300%) of *C. albicans*.

#### **Butaric Acid**

Addition of antiprostaglandin or antithromboxane antibodies to serum alone inhibited germ tube formation by almost 30%, while control antibody had no effect, indicating that these eicosanoids are major morphogenic factors in the serum. Since these molecules also bind to albumin, this may also explain the hyphal transforming activity in serum that associates with albumin. Interestingly, short chain fatty acids (**butyric acid**), the product of lactic acid bacteria (LAB), **inhibited germination**.

Acidolophilus testing showed a benifitial effect on fatty chain acid balance.

In addition, LAB culture supernatants as well as live LAB also inhibited **C. albicans** morphogenesis. Overall, these results indicate that fatty acid metabolites and fatty acid pathways can up-regulate and down-regulate germination in **C. albicans**.

C. albicans growth requires both oleic acid and nicotinic acid.

In general Animal source(s) of oils/fats can insite C18-C20 eicosanoids, both prostaglandin sources. Limiting consumption to 4 ounces of animal meat is the optimum ideal. A University of Minnesota research study of cattle says that "oil and subsequent animal feed meal with high levels of erucic acid reduces palatability and/or are toxic to some animals, and erucic acid has been shown to be a potential health hazard to humans.

# **Differentiating SIYO from the other symptoms**

Relationships between Candida, Leaky Gut, Parasites and Heavy Metals

#### **Symptoms**

Common Symptoms shared by all 4 - Candida, Heavy Metals, Parasites and Leaky Gut:
☐ Bloating
☐ Constipation and/or diarrhea
☐ Painful gas/abdominal bloating
☐ Irritable bowel
☐ Stomach pain
☐ Concentration problems/ foggy thinking
☐ Memory Problems
☐ Feeling like you're in a fog
☐ Personality changes
☐ Mood swings
☐ Irritability
☐ Anxiety
☐ Panic attacks
☐ Fatique, Chronic fatique syndrome,

Inca	pacitating fatigue
	<ul> <li>□ Concentration/focus problems</li> <li>□ Hyperactivity, ADD, ADHD, Autism</li> <li>□ Anemia</li> <li>□ Rashes</li> <li>□ Dry flaky Skin</li> <li>□ Headaches including migraines</li> <li>□ Ringing in the ears</li> <li>□ Tremors</li> <li>□ Candida</li> <li>□ Food/sweet cravings</li> <li>□ Weight changes without changes in diet</li> <li>□ Low sex Drive</li> </ul>
Cano	dida & Heavy metal shared Symptoms:
	□ Painful joints, muscle aches □ Vomiting □ Sweating/night sweats □ Numbness □ Burning/tingling sensation in the skin □ Dizziness □ High blood pressure □ Visual problems □ Kidney problems □ Sensitivity to heat/cold □ Skin discoloration/blotchiness Parasite and Heavy Metal Shared Symptoms: □ Constipation or diarrhea □ Kidney problems – inability to, or frequent urination □ Malabsorption
Leak	xy Gut Symptoms, Candida & Parasites Symptoms:
	<ul> <li>☐ Skin rashes</li> <li>☐ Head sores</li> <li>☐ Scalp or body itchiness</li> <li>☐ Food allergies</li> <li>☐ Chemical sensitivities</li> <li>☐ Nutritional deficiencies</li> <li>☐ Anxiety</li> <li>☐ Impaired immune system</li> <li>☐ Impaired memory</li> <li>☐ Moodiness or irritability</li> </ul>

☐ Bloating☐ Flatulence☐ Brain fog

□ Fatigue□ Headaches

☐ Diarrhea or constipation

### **Heavy Metal Precursors and symptoms:**

□ Nausea
☐ Metallic taste in the mouth
☐ Immune suppression
☐ Receiving vaccinations that contain Thimerosal (mercury preservative)
☐ Mishandled metals at a job site
☐ Chemical and heavy metal spills—even from a broken mercury thermometer
☐ Having mercury amalgams ("silver fillings") in teeth
☐ Living in a home built prior to 1978 that has lead-based paint
☐ Smoking and/or inhaling second-hand smoke
☐ Eating foods (such as contaminated fish) that contain high levels of heavy metals
☐ Living near a landfill
☐ Working in an environment where exposure to metals is prevalent (such as working in a factory
or at a dentist's office where amalgam is used to fill cavities)

# **Parasite Precursors & Symptoms:**

Mild Nagging Headache
Bronchitis
Coughing
Tiny Red Abrasions That Itch
Pet Bird
Eat Sushi, Pate, Snails, Squab, Wild Animals
Pet Cat
Pet Dog
Pet Ferret
Headaches
Brain fog –Poor concentration
Fatigue
Aches or pains
Constipation
Diarrhea
Skin rashes & itching
Rectal/vaginal itching
Irritability

### Candida

Candida is the one common factor shared by all 3 of these other systemic conditions. Deciding which of these problems you are actually experiencing and needing to treat can be difficult. Often we hear from our customers a few months after completing a Candida cleanse that they are again experiencing Candida symptoms or "the Candida has come back, why".

It's not until we get the one common denominator(Candida) eliminated and the digestion tract repopulated with the good flora, Acidophilus & Bifidum, that we are then able to narrow things down a bit and see if the Candida has actually retuned, or if there is yet another underlying issue. Leaky Gut can occur as a result of not having enough good floras in our intestines. The cells in most areas of the body are very close to one another. The cells in the intestinal tract are farther apart, leaving more room for Candida, parasites, heavy metal deposits or other bad bacteria's. Candida grows rhizoids, finger like protrusions, which burrow into the intestinal walls. Parasites will often do the same kind of damage. Once these conditions perforate the wall of the intestine, their toxins, along with decaying food and other bacteria can enter the bloodstream. When this happens we have

what is known as Leaky Gut Syndrome. Toxicity symptoms continue to plague us, even though we may be doing all kinds of cleansing to feel better. L-Glutamine is an amino acid (a protein building block) whose main function is to support cellular growth, energy and repair. It plays a role in the health of the immune system, the digestive tract and the muscles. L-glutamine is very helpful in repairing leaky gut and restoring intestinal health. Probiotics such as Acidophilus & Bifidum live in the spaces between the cells leaving less room for those pesky invaders to set up house. Probiotics also create Vitamin K, another common deficiency found in those with systemic Candida. That's why taking a probiotic supplement like Probiotic 11 is so important to your maintenance, after you've completed a Candida cleanse like Candida Clear. It ensures the healthy environment you've worked so hard to establish.

### **Parasites and Candida**

Parasitic infestations are more common than we realize. Candida and heavy metals create acidity and an anaerobic (lacking oxygen) environment they thrive in. Many Parasite symptoms are confused with Candida. For instance, brain fog, fatigue, memory problems, intestinal & digestive complaints can be parasites rather than Candida. Parasites also inhabit dark warm moist places, especially where there is mucus, such as in the intestines, and respiratory system. Chronic coughing up phlegm, or a nagging dull headache are often indicators of a parasitic infestation. Para Pak is a great parasite cleanse.

# **Heavy Metals, Candida, and Parasites**

Heavy Metals create an acidic, oxygen lacking environment in the body. This is the perfect living conditions for parasites and Candida. We all have Candida in our intestinal flora. It's there for a reason. One benefit it gives, when it's not overgrown and causing us problems, is it binds to heavy metals, especially mercury. As the Candida dies, it then releases Mercury and other heavy metal toxins into the body, thus we experience this strange mirage of symptoms and struggle to understand why we aren't getting better if we are cleansing. Heavy Metal Detox is an excellent herbal remedy for this condition. So you can see how treating Candida alone may not relieve you of all your symptoms and discomforts. We have personal experience with these conditions and have successfully treated them with products we've found of high quality. We would very much like to hear about your conditions and help you to a "Renewed State of Health and Well Being". Below are lists of symptoms caused by these systemic invasions. These lists are not all inclusive, but generally are the most common complaints.

http://candidablog4u.com/symptoms-of-candida-leaky-gut-heavy-metals-and-parasites-are-all-toxicity-symptoms/

#### **SIYO Yeasts**

SIYO can be caused by other yeasts besides candida. SIYO can also be caused by *Saccharomyces boulardii* if opportunities arise. *Boulardii* is a great probiotic yeast and is very useful in certain medical conditions, but caution should be used when supplementing if a patient has a severely compromised immune system. *Boulardii* can become opportunistic if the conditions are right and cause a condition called fungemia. If you are suffering from an opportunistic *boulardii* or any yeast infection, follow my candida protocols.14

The two tests that are used to diagnose SIYO are the alcohol challenge test and an ELISA test. During the alcohol challenge test, you ingest a lot of sugar that will feed the yeast and then you

measure how much alcohol is produced from the fermentation. 15 An ELISA test is a test of the antibodies that the body's immune system produces when it is trying to fight the yeast. 16 If you read about a *Candida* "spit" test online, it is a fake and doesn't prove if you are suffering from a systemic yeast infection. 17

If someone is suffering from SIYO then, the patient should try any of my candida protocols and then after they have completed the protocols they should rebuild their gut for a month.

# SIYO Symptoms

Excessive bleeding may experience a nearly 3/4 reduction in Vitamin K Wounds that do not heal Food reactions

White Rice gas pain, bloating like pregnant www.siboibs.org

Gas bloating diarrhea Vomiting Green stool White tongue Aches pains arthritis

I have a friend who has suffered for years, following a hospital stay.

He tried everything, he reported a fungal infection years ago.

After years of searching, we determined that he has some form of viral systemic fungal infection, which responds to high dose Fucoidan and Tiger Tail in combination. He also does Dr Christopher's parasite liquid. which he reports a definite benefit.

His GI tract has been so disrupted, what he reports is nothing but astounding. while the disbiosis is high, gas is high, and food allergies is high, the oil in his skin is returning, and oil in the stool has all but stopped. I really suspected a protozoan, but nothing has budged the infection.

Fucoidan at about 9 grams per day, and turkey tail about the same, has led to a transformation in about 2-3 weeks. He does my vitamin, metals, and mineral formulation, but the discovery and dosing is all his.

I requested his exact formula.

He has tri-chitosan on order to try a knockout punch.

There are several seaweed groups on the east coast, that carry bladderwrack, and alike. They appear to have been in business for decades, with many customers. We are exploring alternative products, which cost much less than fucoidan and turkey tail sources, which currently costs a premium due to its use by the cancer community.

#### **SIYO Treatment**

### Kill the yeast

- caprilic acid
- GSE
- Fucoidan
- Beta Glucan
- Turkey Tail
- LEM Extract

### Anti-fungal formula Day 90 10/03/13

#### AM

(morning routine) PH and Mineral Treatment

- 1 teaspoon MSM 10 drops DMSO in 8 oz water
- 1 NOW 4753 Silymarin 300
- 1 Prohealth Guaifenesin 600 (otc Mucinex)
- 3 Natures Plus Source of Life 3058
- 3 Spirulina 1000 NOW 2702C
- 1 Selenium 200 NOW 1486
- 1 Zinc 30 GNC255412
- 1 Black currant oil (GLA) NOW 1717
- 1 Potassium 99 GNC 256714
- 1 Solaray Enzyme 14801
- 1 **DHEA** 50 Natrol 16106

### AM 10//03/13

(morning routine) Fungus Treatment

- 5 10-undecenoic acid 250 Thorne SF722
- +3 Natures Plus Source of Life 3058
- 1 Pau D'Arco 500 NOW 4726
- 1 Burdock Root 500 Wholistic Botanicals 86712
- 3 Saccharomyces Boulardi NOW 2934
- 3 Serrapeptase 90,000 IU Puritans Pride 17605
- 2 Caprilic Acid 600 NOW 3347
- 4 Gymnema Sylvestre 250 Pure Encapsulations 00535
- 1 Mega Quercetin Solaray 44686
- 1 GNC B-COMPLEX 50 code 017913
- 1>3 Fungus Treatment Day 14+ immune system strong enough to eat the hyphae. Start at 1 per day NOW® Foods Beta-1,3/1,6-D-Glucan 100 mg 100 mg / 90 Vegi Caps / Item #021993. and ramp to 3 per day.

### PM 10//03/13

(evening routine) PH and Mineral Treatment

- 1 teaspoon MSM 10 drops DMSO in 8 oz water
- 1 NOW 4753 Silymarin 300
- 1 prohealth guaifenesin 600 (mucinex)
- 3 Spirulina 1000 NOW 2702C
- 3 chlorella 1000 NOW 2632
- 1 Borage Oil 1000 NOW 1722
- 3 Yaeyama Chlorella 400 Jarrow 17003
- 4 Charcoal 260 Source Naturals 00136
- 1 GABA 500 Swanson 01872

#### **PM** 10//03/13

(evening routine) Fungus Treatment

- 5 10-undecenoic acid 250 Thorne SF722
- 2 Caprilic Acid 600 NOW 3347
- 4 Gymnema Sylvestre 250 Pure Encapsulations 00535

0>2 Fungus Treatment Day 14+ immune system strong enough to eat the hyphae. Start at 0 per day and ramp to 2X per day NOW® Foods Beta-1,3/1,6-D-Glucan 100 mg 100 mg / 90 Vegi Caps / Item #021993

### 3 Acidophilus Strain LA5 CHR Hanson with a tall glass of milk

http://www.pureencapsulations.com/media/Lacto\_Acidophilus.pdf Sleeping pill

1 (sublingual) Melatonin 2.5mg with 500mcg P5p (Pro Health PH65)

# Fungus Formula - Day 21 july 21 2013

### PH-Mineral & Fungus kill formula Version #3 (day 30+)

### AM (morning routine) PH and Mineral

# 1 teaspoon MSM 10 drops DMSO in 8 oz water ¼ teaspoon Bobs Red Mill Baking Soda 2 NOW 4753 Silymarin 300

2 Prohealth Guaifenesin 600 (otc Mucinex)

6-12 Natures Plus Source of Life 3058

### 6-12 Spirulina 1000 NOW 2702C

- 1-2 Selenium 200 NOW 1486
- 1-2 Zinc 30 GNC255412
- 1-2 Black currant oil (GLA) NOW 1717

### 1 d3 extreem 20000 pro health ph324

- 1 Potassium 99 GNC 256714
- 1 Solaray Enzyme 14801

### 2 plant enzymes 530 now 2967

1 **DHEA** 50mg Natrol 16106

### AM (morning routine) Fungus

### 5 10-undecenoic acid 250 Thorne SF722

- 1 Pau D'Arco 500 NOW 4726
- 1 Burdock Root 500 Wholistic Botanicals 86712
- 3 Saccharomyces Boulardi NOW 2934
- 2 Stinging Nettle 300 mg Puritans Pride 6041

### 3<6 Serrapeptase 90,000 IU Puritans Pride 17605

3<6 Boiron of manganese 30c Manganum Metallicum http://www.hylands.com/

2 Caprylic Acid 600 NOW 3347

### 4-5 Gymnema Sylvestre 250 Pure Encapsulations 00535

- 1 Mega Quercetin Solaray 44686
- 2g (cc) "50 drops" olive leaf Glycerite now 04898
- 1 GNC B-COMPLEX 50 code 017913
- 2<3 Fungus Treatment NOW® Foods Beta-1,3/1,6-D-Glucan 100 mg Item #021993.
- \*0 Host Defense® Turkey Tail Item #: NCTV60 *Trametes versicolor, Coriolus versicolor*
- 1 GNC Magnesium 250 GNC 254213
- 1 Ferosol Iron 65 mg14067 Ionic form, will boost iron and feed parasites.

### 2 turmeric curcumin 450 natures bounty 15417

1>2 daily vitamin NOW 3770T

### PM (evening routine) PH and Mineral

1 teaspoon MSM 10 drops DMSO in 8 oz water ¼ teaspoon Bobs Red Mill Baking Soda 2 NOW 4753 Silymarin 300

2 Prohealth Guaifenesin 600 (otc Mucinex)

4 Spirulina 1000 NOW 2702C

4<7 Chlorella 1000 NOW 2632

1 Borage Oil 1000 NOW 1722

4 Yaeyama Chlorella 400 Jarrow 17003

4-5 Charcoal 260 Source Naturals 00136

PM (evening routine) Fungus

- 5 10-undecenoic acid 250 Thorne SF722
- 2 Caprylic Acid 600 NOW 3347
- 4 Gymnema Sylvestre 250 Pure Encapsulations 00535

0.1 Fungus Treatment NOW® Foods Beta-1,3/1,6-D-Glucan 100 mg / Item #021993 2 white willow bark 400 now 4775b – prevent blood clots

2>4 St Johns wart Whole Foods 450 28087 – numb brain for pain

### 3 Acidophilus Strain LA5 CHR Hanson with a 1/2 glass of milk http://www.pureencapsulations.com/media/Lacto\_Acidophilus.pdf

Sleeping pill

1-2 (sublingual) Melatonin 2.5mg with 500mcg P5p (Pro Health PH65)

1 GABA 500 Swanson 01872 (2 for severe neural stress)

Caprylic acid is a nutrient from coconuts which helps burst yeast cells and hinder their reproduction. Beta-glucans stimulate the activation of NK cells and therefore are helpful against tumors. Has also been used to stop certain bacterial infections.

Optional fungal- Quercetin dehydrate (0-2) 800 mg NOW 3070, Sweats or worm fungus

<u>Optional fungal - Caprylic Aid (0-2) NOW 3347,</u> Swanson Ultra Caprylic Acid, SWU096, 600 mg 60 Sgels, *Sweats or worm fungus* 

### **Stop Biofilm Formation**

The gluten-free diet must exclude carbohydrates (sugars and starches) as much as possible while taking the anti-fungal medication. Sugars include jelly, maple syrup, table sugar, honey, molasses, fructose, soda, fruit and fruit juices. Starches include gluten-free flours, bread, bagel, pizza, pasta, bakery products (cookies, cakes, pies, muffins, brownies), cereals, granolas, energy/ breakfast bars, and chips of any kind. This removes the type of food needed by the yeast to thrive. Other exclusions are mushrooms and other fungi, yeast-raised bread, vinegar, cheese and milk products.

#### **LEM Mushroom extract**

https://en.wikipedia.org/wiki/Shiitake

Christopher Hobbs L.Ac., A.H.G. web: <a href="http://www.mdidea.com">http://www.mdidea.com</a>

http://www.amazon.com/Solaray-Shiitake-Mushroom-Capsules-Count/dp/B00020HYKM

Brand: Solaray 600mg 100ct , Item Number: 15805, 2 or 3 capsules two times a day with meals or a glass of water. 875 mg of a 4:1 extract daily, between meals. Estimate for therapeutic use: 5 to 15 grams 4:1 extract daily for several months, then taper back to 1/4 to 1/2 that amount daily

Known alternatively as Black Mushroom or Chinese Mushroom, wild **Shiitake** grows in Asia only. The name derives from the Shii Tree, its preferred host, although **Shiitake** may also grow on oaks and beeches. **Shiitake** mushrooms have been cultivated in China and Japan for a thousand years.

Lentinan is a mentinus edodes mushroom abstract. It is being studied in Japan as a cancer treatment because it can correct the balance between T1 and T2 in the immune system.

Japanese products containing LEM, a polysaccharide-rich extract from the **shiitake** mushroom and similar extracts from maitake are currently undergoing trials to test their effectiveness in treating various forms of cancer.

The b-1,3-glucan Lentinan reversed tumor growth when injected in mice. It acts by stimulating the immune system, rather than by direct action on the tumor. Because of its large molecular size, Lentinan is not absorbed efficiently when taken orally, but some is absorbed. Lentinan activates the alternative complement pathway, stimulating the macrophages, thus inhibiting tumor growth. It also may activate interleukin-1 secretion, which helps trigger T lymphocytes. **Shiitake** is believed to stimulate interferon production. **Shiitake** significantly inhibited the toxic immunosuppressive effects of cancer drugs such as cyclocy-tidine, when taken with them. Lentinan restores impaired enzyme activity of X-proline-dipeptidyl-aminopeptidase in the serum of mice with tumors. Eritadenine, a purine alkaloid from **shiitake**, is similar to nucleotides in structure, and lowers cholesterol in animal studies

Lentinula edodes extract Mountainrose herbs

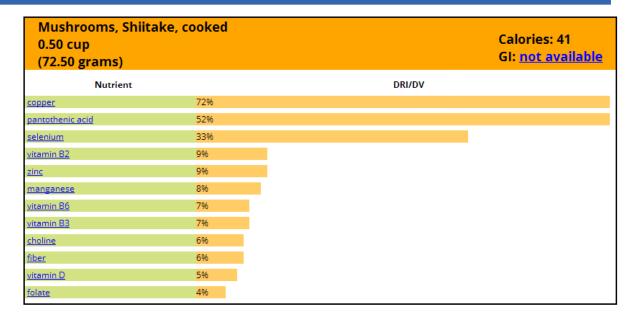
https://www.mountainroseherbs.com/products/shiitake-mushroom-powder/profile

http://www.whfoods.com/genpage.php?dbid=122&tname=foodspice

Active hexose correlated compound (AHCC) is an a-glucan-rich compound isolated from **shiitake**.[22] In Japan, AHCC is the second most popular complementary and alternative medicine used by cancer patients.[23] AHCC is a well tolerated compound[22] and is metabolized via the CYP450 2D6 pathway.[24]

In addition, animal research has shown that AHCC may increase the body's resistance to <u>pathogens</u> as shown in experiments with the influenza virus, [25][26] West Nile encephalitis virus, [27] and bacterial infection. [28][29][30] Animal research has shown AHCC may enhance immune function. [31] [32] A double-blind, placebo-controlled trial of 21 people supported the idea that AHCC may enhance immune function. [33] Clinical research has shown AHCC may benefit patients with hepatocellular carcinoma. [34][35] A published case study reported AHCC benefited a patient with prostate cancer. [36]

The Korea Food and Drug Administration approved on January 2000 that the extracts of the mycelium of **shiitake** mushrooms can protect and help the liver recover from substances such as alcohol.[citation needed] The main chemical for this effect is the beta-glucan. The research showed injecting the extracts of the mycelium in vitro raised the survival rates of liver cells and increased protein synthesis.



The most famous immune-supportive components in **shiitake** mushrooms are its polysaccharides. (Polysaccharides are large-sized carbohydrate molecules composed of many different sugars arranged in chains and branches.) Although many fungi are well-known for their polysaccharides, no single fungus has been more carefully studied than the **shiitake** mushroom.

We know that this fungus is unique in its variety of polysaccharides, and especially its polysaccharide glucans. (Glucans are polysaccharides in which all of the sugar components involve the simple sugar glucose.) Among the glucans contained in <a href="mailto:shiitake">shiitake</a> mushroom are alpha-1,6 glucan, alpha-1,4 glucan, beta-1,6 glucan, polysaccharides, glucan, polysaccharides, including fucoidans and galactomannins. The immune-related effects of polysaccharides in <a href="mailto:shiitake">shiitake</a> mushrooms have been studied on laboratory animals under a wide variety of circumstances, including exercise stress, exposure to inflammation-producing toxins, radiation exposure, and immunodeficiency. Under all of these circumstances, the polysaccharides in <a href="mailto:shiitake">shiitake</a> mushrooms have been shown to lessen problems. There is also some evidence that <a href="mailto:shiitake">shiitake</a> mushrooms' polysaccharides can help lower total cholesterol levels.

### CAS.NO:95009-14-6

**Shiitake** Mushroom Extract.10:1.Lentinus edodes.lentinan 25%.CAS.NO:95009-14-6.Synonyms:Mushroom Extract. Botanical Source:Lentinus edodes (Berk.)Sing.Armillaria mellea Common Names&Synonyms:Lentinus edodes (Berk.) Singer, Lentinula edodes (Berkeley) Pegler;Shii-ta-ke,Lentinus edodes,Filamentous Fungi,Lentinula edodes, Lentinus **shiitake**, Cortinelus edodes,Armillaria edodes.

#### Phytochemicals and constituents:

**Shiitake** mushrooms(lentinus edodes) contains multiple nutrition such as rich protein, fats, amino acids,carbohydrates,trace element and soluble dietary fiber that are indispensable for human health.Lentinan can produce the action of curing and preventing tumour. **shiitake** mushrooms (lentinus edodes) is a kind of natural green health food ,high content of protein and rich nutrition. Lentinus edodes is a delicious, nutritious, and completely edible fungus. The optimum conditions for growth are 80 - 90% relative humidity. a 5 - 15oC in temperature range for the low temperature

type and 10 - 20oC for the medium temperature type. It is one of the most popular cultivated mushrooms with an annual yield of 14% of the total edible mushrooms (15 million tons) cultivated in the world.

Active Substances: Peptidomsannan, KS-2 (a water solubilized lignin derivative), EPS-3 **Shiitake** is a popular edible mushroom from the Far East, for example served in miso soup. It is cultured on logs of the shii tree, Castanopsis (Querqus) cuspidata (Fagaceae), and exported in the dry state or pickled. An important odoriferous principle of **shiitake** is 1,2,3,5,6-pentathiepane, called lenthionine. In the pure state it is a crystalline substance **Shiitake** Mushroom contains 18 amino acids (7-8 of which are essential) and over 30 enzymes. Eritadenine is a unique amino acid believed to lower cholesterol. **Shiitake** Mushroom is high in B vitamins, especially B1, B2 and niacin; and in its sun-dried form, it provides vitamin D (found in very few foods). Oriental herbalists have used wild **Shiitake** Mushroom medicinally for many years; Oriental folklore recommends its use for tumors, flu, heart disease, high blood pressure, obesity, sexual dysfunction, and aging. The past two decades have provided well documented clinical studies showing that **Shiitake** Mushroom helps to decrease cholesterol; 3 ounces of **shiitake** per day can decrease cholesterol by as much as 12% per week. **Shiitake** Mushroom is an enhancer of the immune system, and it may stimulate production of interferon.

According to Kisaka Mori, Ph.D., **Shiitake** Mushroom is high in enzymes and vitamins not usually found in plant foods. In studies, extract form has helped to prevent transplanted tumors from taking hold. Possible indications for use of **Shiitake** Mushroom include: heart disease, cancer, AIDS, high cholesterol, gallstones, stomach disorders, ulcers, diabetes, vitamin deficiency, anemia, common cold, allergies, insomnia, and neuromuscular disorders. It contains lots of bio-active substances such as polysaccharide protein, nucleic acid derivatives, vitamin B group, ergosterol, erytadenine, and various minerals. **L.E.M.** extract improves and activates the organ'''s defence functions\*\*.

Especially, the action of erytadenine, very unique to <a href="Lentinus edodes">Lentinus edodes</a> is quite marked in preventing the accumulation of cholesterol and protecting from arteriosclerosis. Active constituents: <a href="Shiitake">Shiitake</a> contains proteins, fiber, vitamins, and minerals. In addition, <a href="shiitake">shiitake</a> is key ingredientiafound in the fruiting bodyiais a polysaccharide called lentinan. Commercial preparations employ the powdered mycelium of the mushroom before the cap and stem grow. This preparation is called lentinus edodes mycelium extract (LEM). LEM is rich in polysaccharides and lignans. One preliminary trial suggested that oral <a href="shiitake">shiitake</a> may be useful for people with hepatitis B. A highly purified, intravenous form of lentinan is used in China and has been reported to increase survival in people with recurrent stomach cancer, particularly when used in combination with chemotherapy. Similar findings have been found in one small clinical trial with people suffering from pancreatic cancer. Case reports from China suggest that intravenous lentinan may be helpful in treating people with HIV infection. However, large-scale clinical trials to confirm this action have not yet been performed. Oral supplementation of lentinan from <a href="shiitake">shiitake</a> has been shown to significantly reduce the recurrence rate of genital warts (condyloma acuminata).

A preliminary trial involving a group of men and women with genital warts found that those who took 12.5 mg of lentinan twice a day for two months after laser surgery had significantly fewer recurrences (10.53% recurrence rate) compared to those who only had the laser surgery (47.06% recurrence rate).6 Key constituents are the polysaccharied protein complex, a new peptidomannan, KS-2, and a water solubilized lignin derivative, EPS-3 Bioactive constituent: Beta-glucan, Heteroglucan, Adenine derivative, Guanosine 5"" - monophosphate, polyacelylene Bioactivities and Organ"'s Defence Funtions:

- 1.) Anti-tumor effect: Immune enhancement, Activating macrophage, T-cell and NK-cell, Increasing production of TNF-a, interleukins, interferon and complement C3
- 2.) Anti-HIV action: Producing synergistic effect with azidothymidine (AZT)

- 3.) Anti-hyperlipemia: Promoting the metabolism and excretion of the eaten cholesterol
- 4.) Anti-thrombogenicity: Inhibiting platelet aggregation
- 5.) Natural antidote: Strengthening liver function of detoxication
- 6.) Medicational effect against woodchuck hepatitis
- 7.) Protection against bacterial infection
- 8.) Lymphocyte blastogenic activity
- 9.) Promotion of chemotaxis in macrophages
- 10.) Influence on reverse transcriptase activity
- 11.) Inhibitive effect on proliferation of rat ascites hepatoma
- 12.) L929 cells cytotoxicity
- 13.) In vivo neutrophil conglomerating activity
- 14). Expelling micro-organisms of external origin including bacteria, fungi, viruses and protozoa.
- 15).expelling harmful chemial substances (residual agricultural chemicals, environmental pollutants, food additives), pollen and dust.
- 16).expelling abnormal cells (mutant cells, cancer cells)
- 17).expelling worn out autocells (old erythrocytes, etc.), excessively produced substances (antigen antibody complexes), etc.

#### How it Works:

**Shiitake** Mushroom has a polysaccharide compound called lentinan which helps produce T-cells to destroy bacteria and viruses and has anti-cancer, anti-tumor effects. It contains other nutrients helpful in strengthening the immune system and fighting disease-causing organisms. **Shiitake** Mushroom works to prevent heart disease by lowering blood pressure and cholesterol levels, helping pull fat from the system, and working as an anticoagulant. How much is usually taken? The traditional intake of the whole, dried **shiitake** mushroom is 6i§C16 grams per day. The mushroom is typically eaten in soups or taken as a decoction (i.e., boiled for 10i§C20 minutes, cooled, strained, and drunk). Recommended intake of LEM is 1i§C3 grams two to three times per day. Purified lentinan is considered a drug in China and is available as an herbal supplement in North America. Directions For extracts:100mg-400mg of extract. 3 times a day, with food. Before taking any herbal product, you are advised to consult with a trained health care professional.

Reference Link: Click for more Details Product Specifications and Supplying Conditions: Description: **Shiitake** Mushroom PE. Plant Part Used: **Shiitake** Mycelia Extract Ratio: 10:1 Serie Code: R-59 Expiration Date: 18~24Months in Good Condition Storage Stock: Bulk in Stock Pricing Terms: C&F;CIF;DDU;DDP. Delivery Arrange: Soonest on the Day Confirmed Appearance Showing: Yellow Brown

### Wei Qi Soup for Building Immune Strength

Excerpted from Herbs for Health Magazine, Christopher Hobbs L.Ac., A.H.G.

#### Directions:

Fill a pot 2/3 full with purified or spring water, then add:

- Astragalus membranaceus(5-7 sticks)
- Ganoderma lucidum(reishi) (1 medium)

- any other tonifying mushrooms 2-3
- Slightly sprouted beans (1/4-1/2 cup)
- (aduki, black, etc.)

Bring water to boil, simmer for 20 minutes, then add:

- Organic barley (1/2-1 cup)
- (choose amount depending on thickness desired)

Simmer another 20 minutes, then add favorite vegetables such as:

- carrots & celery
- beet tops (or chard, collards, mustard greens, etc.)
- cabbage
- potatoes (optional)
- sea vegetables (nori, kelp, wakame, etc.)
- gobo (i.e., burdock root)
- nettles or other wild greens (when available)
- garlic & onions

Simmer until the vegetables are tender, then add miso and spices such as ginger, celery, or fennel seed. Make enough for a few days and store it in the refrigerator.

Indications and Dosage: During illness, when solid food is not desirable, drink 3-4 cups of the warm broth (add less barley and more water to make broth). For degenerative immune conditions, eat 1-2 small bowls per day, and drink the broth as desired. For autoimmune diseases such as allergies, lupus, diabetes, and hepatitis accompanied by fatigue, weakness, or autoimmune conditions, eat the soup when desired, or drink the broth. This soup can be used upon occasion (1-2 times per week) for general tonification and may help to increase stamina.

#### Reishi

Ganoderma lucidum

The Latin wordlucidummeans ``shiny" or ``brilliant" and refers to the varnished surface of reishi's cap, which is reddish orange to black. The stalk usually is attached to the cap at the side. InJapan, 99 percent of reishi growing in the wild are found on old plum trees, although wild reishi are rare.

Medical uses: For 4,000 years, the Chinese and Japanese have called upon reishi to treat liver disorders, hypertension, arthritis, and other ailments.

Recent test-tube and human studies have demonstrated antiallergic, anti-inflammatory, antibacterial, and antioxidant effects. When more than 2,000 Chinese patients with chronic bronchitis were given reishi syrup in tablet form during the 1970s, 60 to 90 percent showed a marked improvement in health, including increased appetite, within two weeks.

### Maitake

Grifola frondosa

Maitakemeans "dancing mushroom" in Japanese; in ancient times, people who found the mushroom were said to dance with joy because it could be exchanged for its weight in silver. Alternatively, the name may derive from the way in which the small, fan-shaped fruiting bodies overlap like butterflies in a wild dance. In theUnited States, they also are known as hen-of-the-woods because the mass of mushrooms looks like fluffed-up feathers. The stalks are often fused, massed at the base of stumps and on roots. They are common in easternNorth America, Europe, and Asia. Maitake collectors always forage alone and never divulge the location of their treasure, even to their own families. In Japan,

they traditionally mark their hunting grounds with hatch marks on trees bordering the trove and keep others out of their hunting areas.

Until cultivation techniques were devised in 1979, maitake was harvested from the wild. In 1990, Japanese cultivators produced nearly 8,000 tons of maitake, and production is expected to increase with expanding exports to the West.

Medical uses: Laboratory studies have shown that maitake extract can inhibit the growth of tumors and stimulate the immune system of cancerous mice. Human clinical studies of patients with breast and colorectal cancers are under way in the United States. In China, sixty-three patients with lung, stomach, or liver cancers or leukemia who took four capsules of maitake extract three times daily before meals for one to three months showed an ``anticancer" effect. **Shiitake** 

#### **Shiitake**

**Lentinula edodes** In the wild, this light amber fungus is found on fallen hardwood trees. The caps have nearly ragged gills and an inrolled margin when young, and they are covered with a delicate white flocking. The stem may be central or off center. Indigenous to temperate Asia, they are not found in the wild in the United States but are widely cultivated. A similar species occurs wild inCosta Rica.

Medical uses: A vast amount of research into **shiitake**'s medicinal properties has been completed and shows that it has the ability to fight tumors and viruses and enhance the immune system. For more details, refer to the accompanying story.

Precautions: **Shiitake** is nonpoisonous, but researchers have observed cases of **shiitake**-induced skin rashes, and some people who work indoors cultivating **shiitake** experience ``mushroom worker's lung", an immune reaction to **shiitake** spores. A watery extract of the whole mushroom is reported to hinder blood coagulation, so people who bleed easily or who are taking blood thinners should check with their health-care provider before using **shiitake** or its derivatives for a long period.

LEM has shown no evidence of acute toxicity in more than seventeen years of use inJapan, even in massive doses (more than 50 mg a day for one week), though mild side effects such as diarrhea and skin rashes have been reported. Likewise, lentinan has no known serious side effects. People with allergies may experience adverse reactions due to its histamine-sensitizing properties.

Taking **shiitake**: The traditional dose is 1 or 2 fresh **shiitake** mushrooms daily for preventive care or 6 to 16 g of dried **shiitake** in tea, soup, or other dishes. Commercial preparations (extracts in capsule form) of **shiitake** are available in the United States in health-food stores but may be expensive. Dried **shiitake** mushrooms are available in Asian food stores in the United States, usually at more affordable prices. To avert possible digestive upset from eating large quantities of fresh **shiitake**, LEM, which is concentrated and easily absorbed, is preferred as medicine.

### **Recipe: Stuffed Shiitake**

The rich taste of **shiitake** makes this recipe a perfect one to serve as an appetizer or offer as a light evening meal.

- 1 dozen fresh shiitake
- 1 onion, finely chopped
- 1/2 cup celery, finely chopped
- 2 tablespoons olive oil

- 2 cloves garlic, minced
- 1/2 teaspoon tamari
- 1/2 dozen wild mushrooms, such as oyster, chopped
- 1 cup bread crumbs
- 1/3 to 2/3 cup Parmesan cheese
- · Chopped parsley to taste
- Paprika to taste

Cut the stems off the **shiitake**s and chop them finely. Reserve the caps. Saut, the onion, celery, and garlic in the olive oil. When the onion is transparent, add the **shiitake** stems, tamari, oyster mushrooms, bread crumbs, and Parmesan cheese, and saute for 3 to 4 minutes longer. Stuff the **shiitake** caps with the filling, sprinkle them with chopped parsley and paprika, and place them on a cookie sheet. Bake the **shiitake**s at 375øF for 15 minutes, broil for a minute longer to brown the cheese and serve.

### **Preparations**

Powdered extracts and capsules: Because the scientific literature indicates that whole mushrooms are especially active antitumor agents and immune-system enhancers, I recommend taking dried and powdered mushrooms by the teaspoon, either in a cup of ginger tea or sprinkled into soup or on stir-fry and rice. Mushrooms that are too tough and fibrous to powder can be sliced thinly and dried for use in teas and tinctures. Softer and thinner mushrooms can be easily powdered and put into capsules. A size 00 capsule holds about 400 mg of powdered mushroom. For mild to moderate immune-system support, I recommend taking two capsules morning and evening and, for specific immune-suppressed conditions, two to three capsules three times daily.

### **Mushroom Teas and Soups**

Teas and soups: Teas of medicinal mushrooms should be simmered for 40 minutes to an hour, or until they are dark and taste strong. You may add one part ginger to every eight parts mushrooms and one part licorice to every sixteen parts mushrooms to mask any bitterness.

To make a soup, begin with the mushroom tea, to which you may add broccoli, carrots, potatoes, beets, greens, garlic, onions, and/or a little seaweed. Thicken it with a little barley. Fish, chicken, or a little red meat can be added. Simmer for about fifteen minutes. Drink 1 to 3 cups of the soup a day. Tender, fleshy fungi, such as **shiitake** and oyster mushrooms, can be eaten with enthusiasm, but push fibrous chunks of reishi aside--the essence has already permeated the broth, and they are far too tough to chew, even after boiling.

Christopher Hobbs is a member of the Herbs for Health Editorial Advisory Board. He is author of Medicinal Mushrooms: An Exploration of Tradition, Healing, and Culture (Botanica Press, 1995) and many other books. He is a fourth-generation herbalist and botanist with more than twenty years of experience.

### Shiitake - Medicine in a Mushroom

by Kenneth Jones

Mushrooms are a fascinating class of life form: vast underground mycelia that push up strange, fruiting bodies when climatic conditions are right. They have a east range of forms, from common fairy rings and puffballs to strange, unearthly, pale violet discs; from tiny pinheads to hubcap-sized

saucers. Of an estimated 100,000 species of mushrooms, most are edible and very nutritious, containing large amounts of protein, fiber, minerals (including calcium), B vitamins, and vitamin C.

**Shiitake** (shee-TAH-kee) mushrooms are a culinary rage in the United States today (you can even buy kits for growing your own), but have been important in Chinese culture for thousands of years. One of the earliest recorded uses of **shiitake** (Lentinula edodes) dates as far back as the 141 century, when the Chinese physician Wu- Rui described it as a food that accelerates vital or "spirit" energy (known as Qi in Chinese), staves off hunger, "cures cold, and penetrates into the blood circulatory system."

Today, these attributes are collectively taken to mean that **shiitake** makes a person more lively. Wu also stated that **shiitake** was "good for treatment of Heart Troubles... beneficial to [all forms of] Malignancy, likewise certainly [good for] Snake's poison." For the past thirty years, scientists have been investigating some of these uses and have amassed evidence that **shiitake** can help the body fight heart disease, cancer, and viral diseases. Most of the research has been carried out in Japan. Some of the studies are discussed below.

#### **LEM for Heart Disease**

The body could not function without cholesterol, which helps break down fats, or lipids, in the small intestine so that they can be absorbed into the bloodstream. In the liver, cholesterol combines with lipids and proteins in the blood to form various complexes called lipoproteins (LDL or "bad" cholesterol). It has been linked to clogged artery walls, which can lead to heart attack or stroke. High-density lipo-proteins (HDL or "good" cholesterol), on the other hand, have been shown to scavenge excess LDL from the bloodstream and carry it to the liver for excretion or processing into good cholesterol.

According to studies performed in Japan during the 1970s, **shiitake** contains an amino acid called eritadenine that accelerates cholesterol's processing in the liver. In addition, **shiitake**'s high dietary fiber helps the body process cholesterol.

In a 1974 study, 40 elderly people and 420 young women ate 9 grams of dried **shiitake** or the equivalent amount of fresh **shiitake** (90 grams) daily. After seven days, total cholesterol level (the types of cholesterol affected were not distinguished) had decreased 7 to 15% in the elderly and 6 to 12% in the young women.

Another 1974 study involved 30 young women. Ten added 90 grams of fresh **shiitake** and 60 grams of butter to their daily diet, ten added only the butter, and ten added only the **shiitake**. After seven days, the total cholesterol level of the **shiitake** and butter group decreased an average of 4%, while that of the butter group increased an average of 14% and that of the **shiitake** group declined an average of 12%. The researchers concluded that **shiitake** had "completely nullified" the effect of the butter on the cholesterol level of the first group of participants.

### **LEM for Cancer**

In 1969, researchers at Tokyo's National Cancer Center Research Institute isolated a polysaccharide compound from **shiitake** that they named lentinan. Doses of 0.5 to I mg lentinan per kilogram of body weight caused tumors in laboratory mice to regress or disappear in 80 to 100% of the subjects. Researchers have since demonstrated that lentinan works by stimulating immune system cells to rid the body of tumor cells. In clinical trials, lentinan administered with chemotherapy has increased the life span of cancer patients, improved the effectiveness of chemotherapy and kept tumors from growing. In Japan, lentinan is approved for use as a drug to prolong the lives of patients undergoing chemotherapy for stomach cancer.

Additional studies have shown that when **shiitake** mushrooms make up 10% of the daily diet of cancerous mice, tumor growth is inhibited by nearly 40%. When **shiitake** is increased to 30% of the diet, tumor growth is inhibited by nearly 78%.

Researchers have since concluded that the entire mushroom stimulates immune cells such as macrophages and T cells, as well as natural killer cells, and contains compounds that block the formation of carcinogens from nitrates that are found in many processed meats and some vegetables.

### **Aloe Vera**

Aloe vera; This plant called herb of immortality by the Egyptians, contains a soothing gel that helps peristalsis. Aloe vera have antibacterial and antifungal activities. Also aloe destroys bacteria more powerful than any other hypoallergenic plant known.

#### **K Vitamin Diet**

So the diet will consist of meat, fish, seafood, poultry, eggs, nuts, fats, seeds and non-starchy vegetables such as lettuce, endive, escarole, kale, asparagus, Jerusalem artichoke, broccoli, cauliflower, Brussels sprouts, cabbage, bok choi, turnip greens, collards, onions, **Garlic**, celery, tomatoes, peppers, fennel, summer squash and cucumbers. These vegetables along with cinnamon, oregano, basil and mint help in the fight against **yeast**. Lemon and lime juices are OK.

#### **Probiotic Balance in the Gut Flora**

Probiotics foods or supplements with living microbes are needed to restore flora. A study in mice demonstrated that probiotic bacteria can affect the capacity of mice to form antibodies to Candida albicans and showed the usefulness of different probiotic bacteria to produce beneficial health effects in mice.2 Prebiotic foods such as Jerusalem artichokes, asparagus, onion, burdock root, and Chinese chives stimulate the growth of beneficial bacterial species.

#### **Essential Fatty Acids (oils and butter)**

- Fats have received a bad reputation, primarily from research studies done by Ancel Benjamin Keys in the late 1950's.
- They are essential for all body tissues
- Fats and oils are composed of chains of molecules, mostly carbon.
- High-quality fats and oils are one of the most important foods we consume every day.
- The idea to avoid fats because they may make you fat, or clog your arteries, is a lie, and is one of the worst nutritional misconceptions of our time.
- Fats should be consumed in the natural unmodified version whenever possible, and should not be overheated during cooking, Most fats and oils are quite damaged when overheated.

Oils and fats used by mankind are diverse in their origins and composition. The distinction between fats and oils are that the physical state is either liquid or solid at the arbitrary room temperature value of 20 °C . This is not scientifically meaningful to processes in the body, which buy the way is 32 C. Oils and Fats are therefore to be used interchangeably.

Interestingly enough:

There are two exception fatty oils, fish oils and cocoa butter, which contain a mixture of oils and fats.

Most animal sources (Bacon, Steak, Poultry) yield fats. Most vegetable sources happen to yield oils.

#### Meat Fat:

- hormone-free and preferably grass-fed meats, especially lamb, and healthful poultry such as dark meat free-range chicken and turkey.
- Fish. Oily fish such as sardines contain a lot of the omega-3 fatty acids. Salmon and tuna also have them, but can contain mercury. Fish Farm Shrimp and

#### Oil sources

Main sources of oil for humanity are in constant change as new vegetable cultures and cultivars evolve replacing older ones, and as diverse consumer needs and industrial purification technologies are developed. Soyabean is the main source of vegetable oil nowadays, but fragile in a market acception, inasmuch as only ca. 19% by weight of this seed is extractable as oil, whereas oil constitutes 50% of peanut or ca. 47% of sunflowerseed.

Vegetable sources of oils are both annual cultures (such as sunflower or soyabean) and perenial (such as palm and olive trees), and oil accumulates both in seeds (as palm kernel and cottonseed) and in fruits (such as avocado and coconut). Animal sources range from the menhaden fish caught off the US Southeaster cost, to lard hogg, bovine, buffalo or other domesticated animals around the world, and also to seals caught in the artic areas.

The physiological necessity of keeping lipids liquid, and the absence of internal temperature regulation mechanisms except for warm blooded animals may mean that more unsaturated fats are obtained in colder climates, even using the same cultivar. This characteristic is especially noticeable with sunflower which may be predominantly oleic when subject to high temperature cultivation, or predominantly linoleic under more usual temperature conditions.

Annual per capita consumption of oils and fats exhibits a pronounced statistical dependence from the affluence level, with variable dependence from specific cultural or regional pReferences. Though nutritional factors are complex and much controversy still surrounds the consumption of fats, it may be conservatively stated, as the World Health Organisation does, that no more than 30% of total energy be obtained from fatty components of the diet (which means no more than ca. 12% of total dry weight), of which roughly one each should be saturated, monounsaturated and polyunsaturated (and of these, roughly half should be 3 and another half 6).

When annual yearly production of oils is examined, it is apparent that an increase of annual consumption is occurring, together with population increase, that palm oil is becoming increasingly important, having overtaken sunflowerseed in 1981, and that rapeseed production presents the maximum overall growth rate, having overtaken sunflowerseed in 1987. Rapeseed, the major cold climate oilseed benefited from strongly motivated breeding and genetic improvement work performed both in Canada (responsible for the CanolaTM cultivars) and in Northern Europe (yielding the "0" and "00" cultivars, the numerals referring to low erucic acid in oil and to additional low glucosinolate level in the oil extraction presscake).

### **Impaired Short-Chain Fatty Acid Metabolism**

The body converts short-chain omega-3 fatty acids to long-chain forms (EPA, DHA) with an efficiency below 5%

Alibicans sufferers usually are deficient in short chain fatty acids.

The Study of the effects of host long chain fatty acids, eicosanoids, and bacterial short chain fatty acids on control of germination.

None of the  $C_{18}$  or  $C_{20}$  fatty acids tested had an effect on enhancing germ tube formation (arachidonic acid, oleic acid, linolenic acid, or y-linolenic acid).

Among the different eicosanoids, both prostaglandin  $\rm E_2$  and thromboxane  $\rm B_2$  significantly enhanced serum-induced germination (30%) and mutations (300%) of *C. albicans*.

Addition of antiprostaglandin or antithromboxane antibodies to serum alone inhibited germ tube formation by almost 30%, while control antibody had no effect, indicating that these eicosanoids are major morphogenic factors in the serum. Since these molecules also bind to albumin, this may also explain the hyphal transforming activity in serum that associates with albumin. Interestingly, short chain fatty acids (butyric acid), the product of lactic acid bacteria (LAB), inhibited germination.

Acidolophilus testing showed a benifitial effect on fatty chain acid balance.

In addition, LAB culture supernatants as well as live LAB also inhibited **C. albicans** morphogenesis. Overall, these results indicate that fatty acid metabolites and fatty acid pathways can up-regulate and down-regulate germination in **C. albicans**.

C. albicans growth requires both oleic acid and nicotinic acid.

In general Animal source(s) of oils/fats can insite C18-C20 eicosanoids, both prostaglandin sources. Limiting consumption to 4 ounces of animal meat is the optimum ideal. A University of Minnesota research study of cattle says that "oil and subsequent animal feed meal with high levels of erucic acid reduces palatability and/or are toxic to some animals, and erucic acid has been shown to be a potential health hazard to humans.

### **Carbon Oils**

C4 Butter Fat Butyric acid- butanoic acid, Interestingly, SCFA (butyric acid) inhibit germination.

C6 Butter Fat

- 3. Caprylic Acid Methyl Ester (C8:0) at 4 wt %
- 4. Capric Acid Methyl Ester (C10:0) at 4 wt %
- 5. Undecanoic Acid Methyl Ester (C11:0) at 2 wt %
- 6. Lauric Acid Methyl Ester (C12:0) at 4 wt %
- 7. Tridecanoic Acid Methyl Ester (C13:0) at 2 wt %
- 8. Myristic Acid Methyl Ester (C14:0) at 4 wt %
- 9. Myristoleic Acid Methyl Ester (C14:1) at 2 wt %
- 10. Pentadecanoic Acid Methyl Ester (C15:0) at 2 wt %
- 11. cis-10-Pentadecenoic Acid Methyl Ester (C15:1) at 2 wt %
- 12. Palmitic Acid Methyl Ester (C16:0) at 6 wt %
- 13. Palmitoleic Acid Methyl Ester (C16:1) at 2 wt %
- 14. Heptadecanoic Acid Methyl Ester (C17:0) at 2 wt %
- 15. cis-10-Heptadecenoic Acid Methyl Ester (C17:1) at 2 wt %

- 16. Stearic Acid Methyl Ester (C18:0) at 4 wt %
- 17. Oleic Acid Methyl Ester (C18:1n9c) at 4 wt %
- 18. Elaidic Acid Methyl Ester (C18:1n9t) at 2 wt %
- 19. Linoleic Acid Methyl Ester (C18:2n6c) at 2 wt %
- 20. Linolelaidic Acid Methyl Ester (C18:2n6t) at 2 wt %
- 21. □-Linolenic Acid Methyl Ester (C18:3n6) at 2 wt %
- 22. □-Linolenic Acid Methyl Ester (C18:3n3) at 2 wt %
- 23. Arachidic Acid Methyl Ester (C20:0) at 4 wt %
- 24. cis-11-Eicosenoic Acid Methyl Ester (C20:1n9) at 2 wt %
- 25. cis-11,14-Eicosadienoic Acid Methyl Ester (C20:2) at 2 wt %
- 26. cis-8,11,14-Eicosatrienoic Acid Methyl Ester (C20:3n6) at 2 wt %
- 27. cis-11,14,17-Eicosatrienoic Acid Methyl Ester (C20:3n3) at 2 wt %
- 28. Arachidonic Acid Methyl Ester (C20:4n6) at 2 wt %
- 29. cis-5,8,11,14,17-Eicosapentaenoic Acid Methyl Ester

(C20:5n3) at 2 wt %

#### **Good Oils**

C4 Butter Fat Butyric acid- butanoic acid, Interestingly, SCFA (butyric acid) inhibit germination.

C6 Butter Fat

C8 Capronic Acid - hexanoic acid

C8 Coconut Oil

C8 Caprylic Acid

C8 octanoic Acid

C10 Coconut Oil Capric Acid

C10 decanoic Acid

C11 Undecanoic Acid

C12 Coconut oil

C12 Lauric Acid

C12 Dodecanoic Acid

Alpha-Linolenic Acid (LNA) GLA Gamma Linolenic Acid

Olive Oil

#### **Good Oil Sources**

Flax Seed Oil (often called Linseed Oil) (50-57%)

Flax Seeds

Hemp Seed Oil (19%)

Sesame seeds

Canola Oil (10%)

**Almonds** 

Almond Oil

Non gmo - Soy Bean Oil (5-7%)

Walnuts (3-11% of their oil)

Oil Of Dark Green Leaves (50%) omega 3 - but the leaves are low in overall fat

levels

Pumpkin Seeds (0-15%)

Coconut meat

Hazelnuts

Pecans

Sunflower seeds

### **Linoleic Acid (LA) Content**

Safflower Seed Oil (78%)

Sunflower Seed Oil (68%) Wheat Germ Oil (60%) Corn Oil (57%) Hemp Seed Oil (57%) Soy Bean Oil (53%) Walnuts (54-62%) Sesame Oil (43%)

Pumpkin Seeds

### **Gamma-Linoleic Acid (GLA) Content**

Borage Oil (23%)
Black Current Seed Oil (15-19%)
Evening Primrose Oil (7-10%)
Hemp Seed Oil (2%)
Cod Oil
DHA Fish oil - 4,7,10,13,16,19-docosahexaenoic acid

### Inhibiting E2 and B2 fatty oil conversions

Mangosteen is a fruit and its extract is sold as a dietary supplement. Inhibitions of histamine release and prostaglandin E2 synthesis by mangosteen, a Thai medicinal plant. Biol Pharm Bull. 2002. The fruit hull of mangosteen, Garcinia mangostana has been used as a Thai indigenous medicine for many years. These results suggest that the 40% ethanol extract of mangosteen has potent inhibitory activities of both histamine release and prostaglandin E2 synthesis

<u>Curcumin</u> is an extract from turmeric. Phase I clinical trial of oral curcumin: biomarkers of systemic activity and compliance. Clin Cancer Res. 2004. Curcumin, a polyphenolic antioxidant derived from a dietary spice, exhibits anticancer activity in rodents and in humans. Its efficacy appears to be related to induction of glutathione S-transferase enzymes, inhibition of prostaglandin E(2) (PGE(2)) production, or suppression of oxidative DNA adduct (M(1)G) formation.

3.4. Curcumin, the Indian Spice Turmeric (Zingiberaceae). Curcumin is an antioxidant, anti-inflammatory, active principal ingredient of the curry spice, turmeric. The compound is marketed as a dietary supplement [140] and has attracted interest as a cancer-preventive agent [57]. It is well known that curcumin prevents the onset of inflammation by inhibiting the activation of nuclear factorkappa beta (NF-κB), the production of TNF-a, interferongamma (IFN-γ), and NO, and the gene expression of inducible nitric oxide synthase (iNOS) [141–143]. It acts by transrepressing NF-κB, activating protein-1, and the signal transducer and activator of transcription-1 [144–148]. Curcumin activates PPAR-γ in Moser cells, a human colon cancer cell line [145], and is able to suppress sepsis through PPAR-γ [146]. In addition, it increased PPAR and decreased iNOS gene expression in infected macrophages, as well as downregulated IFN-γ production by primed lymphocytes [147]. Curcumin action on PPAR could involve a curcumin-responsive element that resides in the PPAR-γ gene regulatory region [148].

An enzyme called cyclo-oxegenase-2 (COX-2) converts the omega-6 fatty acid, arachidonic acid (AA) to PGD2. If that conversion can be stopped or reduced anywhere along the chain it will stop or reduce the effects of PGD2

#### **Quercetin in tea**

Quercetin was found to inhibit the enzymatic activity of COX-2(2) and to reduce its production (3). Quercetin is a flavonoid found in many plants and foods. Tea contains very high amounts compared with other foods (9). Green and black tea leaves when dry contain a mean average of between 204 and 264 mg/100g. Decaffeinated tea contains more than ordinary. But Oolong contains hardly any less than 2mg/100g. Use as little water as possible when infusing the tea or other substances. When tea is brewed normally, with lots of water, 100g only contains about 2mg of quercetin. Naturally, most of the cup of tea is water. So just use enough water to cover the leaves and mash them and squeeze them to get as much out as possible and then you'll approach the 200-odd mg/100g found in dry tea leaves.

### **Ginger**

Ginger inhibited both COX-2 (4) and PGD2 (5). Ginger contains many constituents. The main one, gingerol, is transformed into shogaols when it is dried or cooked. The study referred to as number 4 used a methanol (alcohol) extract of ginger. This contained various gingerols, shogaols, gingerdiones and other constituents of ginger. One form of gingerol and two of shogaols inhibited COX-2. Another thing to be aware of is that COX-2 is also involved in the production of another prostaglandin called PGE2, and this is necessary for skin health. Something that interferes with the production or activity of COX-2 might also interfere with PGE2.

### **Aspirin**

is the drug form of salicylic acid - plants such as meadowsweet or willow bark - Aspirin can inhibit the activity or suppress the production of COX-2 (6) (7) (8). But, at least in cultures of smooth muscle cells or endothelial cells (reference 8), its inhibition of COX-2 is transient, and prostaglandin production begins again within 2-5 hours.

Tocopherols also inhibited COX-2, as was found in the study in reference 3, above.

Both alphaand gammatocopherol showed this ability. They are two of the forms of vitamin E.

Olive oil is one of the main sources of alphatocopherol.

# **Immune System Herbs**

 Magnolia bark neoligand binds to site PPARy that mytotoxin

grabs, preventing immune system alteration. Plant lectins that can bind to sialylated glycans are from the leguminous tree Maackia amurensis. Although these lectins are discussed here, they do not show a "classical" R-type domain but instead have an L-type lectin domain. The cysteine-rich R-type domain of the MR binds other sulfated glycans and also N-glycans on pituitary glycoprotein hormones containing 4-SO4-GalNAc $\beta$ 1-4GlcNAc $\beta$ 1-2Mana1-R.nutraceutics that are reported to be able to modulate PPAR- $\gamma$  expression or action.

### R-type plant lectins

- oThere are other R-type plant lectins in the RIP-II class that are not toxic, and these include several proteins from the genus **Sambucus** (**elderberry**), such as nigrin-b, sieboldin-b, ebulin-f, and ebulin-r. All of the B subunits of these proteins appear to **bind** Gal/GalNAc, but they may have some differences in affinity and may recognize different Gal/GalNAc-containing glycoconjugates. Cell lines selected for resistance to killing by modeccin are not resistant to abrin and ricin, and vice versa. The glycan-binding specificity of these lectins should be explored more fully in the future using glycan microarrays and related screening approaches.
- Previous studies have shown that MR expression can be positively modulated in vitro by many agents, in particular by 1,25-dihydroxyvitamin <u>D3</u> (8), prostaglandin <u>E2</u> (Standard Process Cataplex E2 90 Tablets) (9), IL-4, and IL-13 (10, 11).
- CHITOSAN IS Anticholesterolemic, Mucoadhesion COFACTOR, Haemostatic anticoagulant activity, Antitumor Activity, Analgesic Effect, produces w/o/w emulsions without adding any surfactant. Chitosan as a preservative coating in reducing or preventing moisture loss, lipid oxidation, and microbial growth. Chitosan support adhesion and differentiation of primary chick dorsal root ganglion neurons. Chitosan oligomers have also exhibited wound-healing
- Properties, it is suggested that their wound-healing properties are due to their ability to stimulate fibroblast production by affecting the fibroblast growth factor.. Chitosan has shown a significant scavenging capacity against different radical species, the results being comparable to those obtained with commercial antioxidants. The interaction between chitosan and anionic surfaceactive materials (phospholipids, bile acids) depends on its three types of reactive functional groups: the amino group at the C2 position and primary and secondary hydroxyl groups at the C-3 and C-6 positions, respectively. Thongngam et al.have demonstrated the formation of micelle-like clusters within the chitosan structure in its interactions with a model <a href="mailto:bile salt">bile salt</a> [93,94]. Another mechanism accounts for the adsorption of chitosan to the surface of the emulsified lipid and
- The formation of a protective coating that might prevent the lipase/co-lipase from adsorbing to the droplet surfaces and gaining access to the lipids inside the droplets [95]

The immune system is critical when fighting cancer. While some treatments kill cancer cells and other treatments revert the cancer cells into normal cells, the immune system still has to be fixed before the patient is whole. STEPS INCLUDE liver flush, microbes in the bloodstream, proteolytic enzymes needed to digest protein, lipases needed to digest fat, and amylases needed to digest carbohydrates or proteolytic enzymes (i.e. proteases) that we are interested in here, and particularly pancreatic enzymes trypsin and chymotrypsin, zinc, Vitamin C, manganese, magnesium, selenium, Vitamin B, and Vitamin A. Especially zinc and Vitamin C., Herbs (e.g. echinacea). Transfer Point Beta 1,3-D Glucan works by activating the macrophages, or immune cells, which trap and engulf foreign substances. Derived from broken cell walls brewer's yeast (Saccharomyces cerevisiae), Beta Glucan is a powerful immune stimulator

http://www.dramyyasko.com/resources/autism-pathways-to-recovery/chapter-6/

The best defense is to maintain a strict nutritious gluten-free diet bolstered with probiotic and prebiotic food and a daily 100% vitamin/mineral supplement.

# **Vision Issues**

Vitamin A for the retina Bilberry for the eyes

### **Bilberry**

#### **Overview:**

**Bilberry** has been used for centuries, both medicinally and as a food in jams and pies. It is related to the blueberry and is native to Northern Europe. **Bilberry** fruit contains chemicals known as anthocyanosides, plant pigments that have excellent antioxidant properties. They scavenge damaging particles in the body known as free radicals, helping prevent or reverse damage to cells. **Antioxidants** have been shown to help prevent a number of long term illnesses such as heart disease, cancer, and an eye disorder called macular degeneration. **Bilberry** also contains vitamin C, which is another antioxidant.

A strong antioxidant, **bilberry** benefits your circulatory system, eyes, heart and brain, and helps generate overall good health, says Schechter. **Bilberry** fruit contains a type of flavonoid called anthocyanosides, which are responsible for increasing flexibility of capillaries and increasing blood flow.

Research shows that standardized extract of **bilberry** can enlarge range of vision and improve sharpness of images, enhance ability to focus, and improve blurred vision, eyestrain and nearsightedness. **Bilberry** extract also helps strengthen coronary arteries and helps prevent atherosclerosis and venous insufficiency, which causes swollen ankles and feet. "Since adding **bilberry** to my own health program, I've noticed my muscles seem to recover slightly faster, I experience less muscular pain and my vision has improved from 20/100 to approximately 20/50," says Schechter.

Bilberry (Vaccinium myrtillus) contains nutrients that protect eyes from eyestrain or fatigue, and can improve circulation to the eyes. When British Royal Air Force pilots During World War II ate Bilberry preserves before night missions and discovered that their night vision improved afterwards, this herb was investigated and found to be beneficial for the eyes. Bilberry works by improving the microcirculation and regeneration of retinal purple, a substance required for good eyesight. It is believed that this property is related to the high amount of proanthocyanidins, a type of flavonoid that tends to prevent capillary fragility and strengthen the capillaries which nourish the eyes. Other properties appear to assist in thinning the blood and stimulating the release of vasodilators. Anthocyanin, a natural antioxidant, also lowers blood pressure, reduces clotting and improves blood supply to the nervous system. Anthocyanosides support and enhance the health of collagen structures in the blood vessels of the eyes, thus aiding in the development of strong healthy capillaries that can carry vital nutrients to eye muscles and nerves. Bilberry has long been a remedy for poor vision and "night blindness." Clinical tests have indicated that oral administration of bilberry tends to improve visual accuracy in healthy people and can help those with eye disorders such as pigmentosa, retinitis, glaucoma, and myopia.

Not many studies have been done to examine **bilberry** specifically. Even fewer studies have been done in humans. Most of the suggestions about bilberry's effectiveness come from research on similar **antioxidants**, or from test tube and animal studies.

# **Chronic venous insufficiency**

**<u>Bilberry</u>** extracts are used in Europe to treat this condition, which occurs when valves in veins in the legs that carry blood to the heart are damaged.

Read more: http://www.umm.edu/altmed/articles/bilberry-000225.htm#ixzz1h4raECpE

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# **Luten for the eyes**

# **Ginkgo for the eyes**

Ginkgo Biloba extract from the ginkgo tree has been shown to benefit visual function by improving microcirculation to the eyes especially among patients suffering from senile macular degeneration, a common condition thought to involve free radical damage, says Steven Schechter, N.D., author of Fighting Radiation & Chemical Pollutants With Foods, Herbs & Vitamins (Vitality, Ink).

# **Antiparasitic dosing and Vision**

Always take **DEC** first to clear the blood vessels of worm larvae first Pulse **IVM**, never take it continuously, an overdose will lower your vision Worm toxins cause your vision to dim maintain daily willow bark and **ginkgo**, to ensure adequate capillary flow.

### Q&A

Q> On page 66 of printout says: Dosing GuaiAid with Piparazine can help reduce eye inflammation. Also says use DEC if eye problem instead of Piperazine. Still debating about DEC.

Started PinX and Ivermectin today.

Will

A) continue this Pinx for 9 days then start Pip if no eye complications.

Or

B) continue this Pinx for 9 days and then start DEC instead of Pip? Or with it?

Will wait and see what's up in 9 days?

(Nausea on first very SMALL dose of 25 MG PinX) I think u said u threw up a lot when u took it.

2) have hardened whitish cellophane like scabs/flakes covering lesions. Is this Ascaris in your opinion?

No \$ for macro zoom camera yet or would show you pic.

Of interest: Psora on face and eyebrows melting off with application of Ume plum vinegar.

3) Have 10 days 750 mg dose metrodizanole for blastocystis. Wait and take later?

A> Do you have the impression you have filiarae?

Have you had the Elisa test?

I had tons of worms around the eye, not in it, I had Ascaris.

I cannot eliminate risk of a filiarae infection causing blindness.

If you have a positive ELISA test, call head of ophthalmology Duke University Dr Vann <a href="http://www.vitals.com/doctors/Dr\_Robin\_Vann.html">http://www.vitals.com/doctors/Dr\_Robin\_Vann.html</a>

Bilberry, Lutein help

Willow bark and Ginkgo open capillaries.

Tiny worms exposed to low level DEC will stop moving. If they are in the blood supply they will eventually dissolve. Low dose Piperazine helps remove toxins, and provides a minimal immune activation. Time to dissolve microscopic larvae is about 2 days.

When worms are outside the blood supply, long duration albendazole is required, it slowly goes into ears, lymph, CNS fluid, and eye fluid. The effect on very small worms is to choke off the sugar supply, they die slowly over a period of weeks, and cause minimal damage.

Taking to much DEC, Piperazine, or albendazole, can cause a migration and tissue damage.

Taking more caustic antiparasitics are known to cause vision loss.

Usually worms in the eye leave visual artifacts, so if they are in the fluid, you should have some idea, especially if you look into a bright light.

## **SIBO**

Parasites bring infected guts, that bring you toxins from their illnesses. Sick parasites are mean, nasty, and harder to kill. Fixing their guts, and treating their infections, makes them easier to kill inside your body.

Parasites come with infected guts. Parasites move bacteria and yeast into organs. Killing bacteria, and yeast infections are primary to making you stronger, parasites weaker.

## **Deoxycycline**

Take a single-dose of Deoxycycline for 30 Days. It is broad spectrum, and kills nastier bacteria.

## SIBO Scan

Q> I have tried everything, what should I do?

A> Have you dosed the following?

- Have you tried Caprillic acid?? (Fungal)
- Have you tried Xylitol (Sugar Bacteria)
- Have you tried Sorbitol
- Have you tried Lactulose (Lactase abnormal)
- Have you tried Fucoidan (Virus)
- Have you tried Glucomannan (Mold)
- Have you tried Liposan? (Biofilm)
- Beta Glucan?
- Humineral?
- Have you tried Probiotic 225
- Tried Listerine Natural Mint
- LEM extract (shiitake mushroom)
- Turkey Tail?
- · Dr Christophers parasite liquid
- Corriander oil
- Copper?
- \* Hemp Oil Nutiva
- Organic virgin coconut oil Nutiva
- Have you been tested for CDIFF?
- Entrobacterialecese nickel bacteria?"
- \*Kombucha TEA (Kombucha beverages from Camellia sinensis L. (black tea) and Satureja montana L. (winter savory tea))

Q> If I don't feel better by tomorrow I'm going back to the ER. My small intestine is now tight and feels blocked. Stomach hurts, still can't eat. I'm really scared. Not really sure what to do. I can't stop meds but I keep getting worse. I am doing the formula as close as I can but keep getting worse. Can't tell if toxins are just slowed all through my small intestine or if I'm actually blocked.

A> I think I know why blockage occurs.

I think worms separate the intestine from the muscle tube, and it slides down to block the valve. I have given this a great deal of thought.

The ER may order a barium Xray, dont know if they will or can do much, Removing this section of intestine is a bad idea. It is crucial for oils, fats, and many chemistries. Spending several hours lying down per day may help relax that section.

I suggest you crank olive oil, pour it over your salads with a little apple cider vinegar. Use coconut oil as much as possible.

Ose coconut on as much as pos

The intestine will re-attach. Try more bran, flax, germ (wheat) in your soft food diet.

Q> My tongue has been coated kind of black almost 2 weeks.

A> Yea, I had every color tongue possible, biofilm, try humic fulvic. I thought you rushed in, SIBO steps are important to kill, makes you stronger, your worms weaker.

Q> I am taking all the vitamins, minerals, zinc sulfate plus other supplements.

I got extremely sick last week, taking the zinc sulfate, I probably raised it too quickly, or I took too much by mistake (I have the powder, not the capsules, and I use the gold scale), but anyway I got so sick that I vomited twice, even the water with the baking soda and the emergency pills half digested, I wasn't able to keep anything in my body, deep stomach pain, I turned white, not able to stand up or think straight.

A> Zinc antagonizes Iron, that parasites steal. Copper antagonizes **iron** as well. While Zinc makes it impossible to kill Ascaris, It brings your enzyme system back to life, and forces the body to build white blood cells. It is important to stop all zinc and vitamin A after the PRAZI kill of flukes. Get a hair analysis, and see how bad your minerals and metals are. Then you can gauge how far gone you are. I was practically dead. My case was extreme, so the plan is based on an extreme case (me).

Q> Quite scared, and the fact is that I was working too, I need to understand if that was the result of a massive die off or zinc overdose, maybe both.

A> What you felt was an enzyme release of the worms. They do not like their food supply poisoned. They feed on blood.

Q> Unfortunately I cannot stop to work. I finished all my savings for the supplements. I just had a couple of questions for you, I need to know how to move in every step to avoid public problems, especially at work, I am pretty sure I am infested with Ascaris in my GI Tract. do you think that my first dose of IVM may cause any scattering?

A> I have copied \_\_\_\_ frrom the CZ. She is an expert in managing work and killing. I will bow to her experience. I have not worked in 3 years, and just now am getting close to getting back to work. IVM does not cause scattering. In less than 6 weeks, they have scattered. Doctors that say take 3 of these or 6 of that are a cure are fools. Only oil of chenopodium kills worms. They removed it from most major markets.

Q> I've been reading a few experiences about that from curezone. And also how do you suggest to take the Pinx + Ivm? Both in the same time or first one and after 30minutes the other?

A> IVM puts the worm to sleep, so taking both at once may have some reaction, but after they attach it will keep the toxins inside the worm. Magnesium Citrate, 2 Grams, will cause a rapid flush of the worms out of the GI so they cannot grab on to anything on the way out, nor wake up before they are flushed.

Q> Will it be so devastating about the die off?

A> Lets keep the nasty talk for later. If they have spread, you will be in for the fight of your life, but it is a life worth living.

Q> And the prazi and albenza? will be possible keep working during that protocol or I may get very sick when using those two medicines?

A>I will let \_\_\_\_ from the CZ answer that one.

Q> I always have my emergency supplements handy, in my car and at home. But work while doing this stuff is not very easy for me (I am a chef), and last week I had to leave the place to run in the toilet and vomit a bunch of pills twice.

A> Back down a bit, it takes months to eliminate, there is no hurry. Do not eat raw meat or fish.

Q> I actually am at the phase 2, Still trying to raise the PH, which is at 6, sometimes 6.5. But having difficult on the biofilm+sibo.

A> The next version will have ore SIBO SIVO sections. I cannot find them yet, I have years of files, and over 80 Gigs of data to crawl through.

Q> I've been reading a lot about that too. The symptoms are soo similar that sometimes I don't understand if my gas and lot of bloating is caused more from the sibo or from the parasites.

A> Both, dying worms make foul smell, SIBO dying is sweet, like rotting apples, grapes, leaves.

Q>My girlfriend must have some candida too. I am trying to eat as healthy as I can, my diet is 90% plant based, mostly organic, with no processed food.

A> Shiitake mushroom will keep her immune system up. Parasites are in the produce as well, it is really a function of clean water, good immune system, balanced body, and then anti-parasitics work. It is like they only work in a test tube or something. Fix the body, then kill the parasites. Costs a whole lot less.

Q>Though sometimes I HAVE TO eat something dirty or after 3-4 days with a perfect diet my parasites starts to drive me crazy, I am addicted mostly to refined flours, being an Italian, son of a baker, and being eating white flour every day for most of my life I thing is what fucked up most my intestines and immune system.

Q> I found I have to eliminate not just the sugars, but every carbs source for at least 2-3 weeks to eliminate the sibo, even the healty ones like quinoa, millet, ecc..., Food Map Diet. Did you follow any particular diet when eliminating the biofilm?

A> Slow down, so many questions, first parasites eat pasta for lunch. Quit. Bread is not as healthy as potatoes. Olive oil and butter are better than cheaper vegetable oils. Sugar feeds SIBO and SIYO. That tells you that you have a dual infection. There is a crappy section in the latest revision, it deals briefly with yeast and bacteria. I took <a href="https://www.humineral.com">www.humineral.com</a> humic fulvic to short out the voltage potential a biofilm generates. High pulses of chitosan, LEM, beta glucan, Turkey tail, Caprillic acid will knock down your infection, expect a 4 day down, minimum. The toxins and smell will be very bad. My head came off.

Q> And how much time did it take for you to accomplish this step?

A> SIYO took 21 days to kill.

Q> Now I get bloated and my abdomen is very distended most of the time, whatever I eat, except when I fast.

A> No fasting, make you strong, them weak, eat veggies K vitamins, Salads, Small portion of meat (cooked) potatoes not bread, go to good fats, coconut oil (butter) if you must. Use only olive oil from Sicily, the oil there has 22-23 carbon chains.

Q> I am keeping with the oregano oil, chitosan, caprilic acid, garlic coconut oil, echinacea, cloves, peppermint oil, zeolite, colostrum, echinacea ecc... plus a biofilm disruptor in caps every morning on a empty stomach. But how do you know when you are really killing the sibo?

A> Toxins are released, Pulse higher dose better than every day for some things. You are doing to many things at once, how about splitting it up into 2 different formulas, and alternating. It goes as fast as it goes, doing everything all at once is a bit much.

Q> Can you see it in the stool? I just need a few word from you because I am feeling stucked and not going anywhere, thanks a lot for your help, which is always very appreciated.

## https://en.wikipedia.org/wiki/Enterobacteriaceae

## 3/14 killed acetic acid bacteria with Chitosan

03/14 Killed acetic acid bacteria Using 6/6/6 /D Chitosan? Smelled like vinegar, large gassy painful stools, very acid stool, vision much improved.

## 3/28/14 (Disbiosis Formula)

## AM - PM Formula

- 2 cups of **rose hip seed tea** (9 trans cis retinolic acid)
- 1 magnesium citrate 250mg nature made 02896
- 1 magnesium plus 200mg prohealth ph88 Glycinate form
- 2 malic acid w magnesium soloray 46355 w B1 B6 griffonia simplicfolia 5HTP
- 4 copper caps 2 mg 01017 twinlab
- 1 Magnesium Citrate 133mg Source Naturals- highly absorbable w dibasic calcium phosphate, colloidal silicon dioxide (33% DV)
- 1 Chromium Picolinate 200 mcg NOW1422
- 1 Vandyl Complex KAL 70311 C100MG- VANDIUM SULFATE 10MG
- 1 Taurine 500 GNC 02615
- 1 Magnesium Citrate 200mg NOW 1292
- 1 Magnesium, AAC Solaray 4630 chelated with whole rice concintrate,w magnesium oxide
- 1 K2 100mcg NOW 0990
- 10 drops liquid Molybdenum NutriCology50700
- 1 Magnesium and Potassium Aspirate chelated with L aspartic Acid w Taurine NOW1320
- 25 drops Bartlow LDM100 Lomatium Dissectum

## 3/24/14 (Disbiosis and Mold Formula)

## AM - PM Formula

- 1 cups of rose hip seed tea (9 trans cis retinolic acid) 4X/D
- 1 barleans flax oil 1000 mg 10007
- 2<1 very **cranberry** Irwin naturals 57740
- 2<4<8<4 magnesium citrate 250mg nature made 02896
- 0<2<4<12<4 monolauren 300mg professional health products 19009
- 0<1 glucomannan 575 now 6512
- 2<1 magnesium plus 200mg prohealth ph88 Glycinate form
- 0<2<1 coconut oil natures answer 26131
- 4<4<2 malic acid w magnesium soloray 46355
- 2<8<4<2 copper caps 2 mg 01017 twinlab
- 0<1<2 Magnesium Citrate 133mg Source Naturals- highly absorbable w dibasic calcium phosphate, colloidal silicon dioxide (33% DV)
- 1 Chromium Picolinate 200 mcg NOW1422
- 1 Vandyl Complex KAL 70311 C100MG- VANDIUMSULFATE10MG
- 0 Taurine 500 GNC 02615
- 1<0 Magnesium Citrate 200mg NOW 1292
- 1 Magnesium, AAC Solaray 4630 chelated with whole rice concentrate w magnesium oxide
- 1 K2 100mcg NOW 0990

10 drops liquid Molybdenum NutriCology 50700

2<4<4<2 Malic Acid with Magnesium Solaray 46355 w B1 B6 griffonia simplicfolia 5HTP

1 Magnesium and Potassium Aspirate chelated with L aspartic Acid w Taurine NOW1320

25 drops Bartlow LDM100 Lomatium Dissectum

## 03/14/14 SIBO small intestine bacterial overgrowth

## 3/16/14

(Formula add, Magnesium Citrate,

Glucomannan,

monolauren,

copper chelate 2 mg.)

PS: Doctor \$250/2105 test from metametrix \$380

http://www.metametrix.com/files/test-menu/interpretive-quides/GI-Effects-IG.pdf

Tests for the entire NYC super bug strains, and more.\

http://www.slideshare.net/MMASSY/drugs-acting-on-the-gastrointestinal-tract

Antibiotics deplete copper reserves

The age-old folk remedy of wearing copper bracelets for rheumatoid arthritis

http://www.journalofanimalscience.org/content/20/1/163.abstract

Copper sulfate significantly increased lactobacilli, total aerobes, total anaerobes and streptococci organisms.

The antibiotics significantly increased the fecal count of coliforms and molds and yeasts and decreased the streptococci organisms.

## **Heavy Hitting SIBO formula**

## Shiitakai extract

Shiitakai extract Planetary Herbals

http://www.swansonvitamins.com/planetary-herbals-full-spectrum-shiitake-extract-2-fl-oz-liquid? SourceCode=INTL415&CAWELAID=530002460000015001

## Reishi extract

Reishi extract 40drops 2X/D

http://www.swansonvitamins.com/planetary-herbals-full-spectrum-reishi-extract-2-fl-oz-liquid

### **Samentos**

http://www.nutramedix.ec/ns/samento

http://www.newswithviews.com/Howenstine/james26.htm

http://www.livestrong.com/article/470251-benefits-of-samento/

## **Immune System Benefits**

Both Drugs.com and the Memorial Sloan-Kettering Cancer Center report that studies of cat's claw confirm the alkaloids present in the herb stimulate the immune system in various ways, including increasing the destruction of foreign bodies, increasing production of white blood cells and enhancing the action of other immune system cells.

- See more at: <a href="http://www.livestrong.com/article/470251-benefits-of-samento/#sthash.GhANm1nQ.dpuf">http://www.livestrong.com/article/470251-benefits-of-samento/#sthash.GhANm1nQ.dpuf</a>

Some of the beneficial properties of Samento are attributed to the pentacyclic oxindole alkaloids (POAs) that are found in the plant that act on the cellular immune system and demonstrate powerful immune system modulating properties. Samento does not contain the tetracyclic oxindole alkaloids (TOAs) that are found in traditional Cat's Claw. TOAs disrupt central nervous system function and greatly inhibit the effects of the POAs.

Some researchers believe that Samento may be as much as 1,000 times more effective than Cat's Claw. One difference between Samento and Cat's Claw is that Cat's Claw is an immune system stimulant and Samento is an immune system modulator. Also, Samento can be used to treat all autoimmune disorders.

In May 2005, pharmacological studies were conducted in laboratory rodents at the University of Guayaquil, Ecuador. The anti-inflammatory effect study showed that Nutramedix Samento extract inhibits inflammation by 83.8%.

There are no known contraindications, no known side effects and no known interactions with other drugs when using Samento. In May 2005, toxicology studies were conducted on Nutramedix Samento at the University of Guayaquil, Ecuador. No toxic effects were reported even when laboratory rodents received 120,000 times the equivalent human dose.

Some other medicinal properties that have been reported:

- ANTIBACTERIAL
- ANTICANCEROUS
- ANTIDEPRESSANT
- ANTIFUNGAL
- ANTI-HYPERTENSIVE
- ANTILEUKEMIC
- ANTIMUTAGENIC
- ANTI-OXIDANT
- ANTIPARASITIC
- ANTIPARKINSONISM
- ANTI-ULCEROUS
- ANTIVIRAL
- CYTOSTATIC
- DEPURATIVE
- DIURETIC
- VERMIFUGE

## Copper-soy

http://japr.oxfordjournals.org/content/20/1/21.abstract

Effects of supplemental copper-methionine chelate and copper-soy proteinate on the performance, blood parameters, liver mineral content, and intestinal microflora of broiler chickens G.-B. Kim\*, Y. M. Seo\*, K. S. Shin\*, A. R. Rhee\*, J. Han† and I. K. Paik\*

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### **SUMMARY**

In this study, we investigated the effect of dietary supplementation of Cu-Met chelate (Cu-Met) and Cu-soy proteinate (Cu-SP) on the performance, blood parameters, liver mineral content, and intestinal microflora in broiler chickens. A total of 1,008 hatched Ross broiler chickens were randomly assigned to 1 of 6 dietary treatments (T): T1, control; T2, antibiotic (6 ppm of avilamycin); T3, 50 ppm of Cu as Cu-Met; T4, 100 ppm of Cu as Cu-Met; T5, 50 ppm of Cu as Cu-SP; and T6, 100 ppm of Cu as Cu-SP. Each treatment had 3 replicates of 56 birds (28 birds of each sex). During the 4-wk feeding period, the BW increase of birds in the antibiotic treatment was 3.25% and those of birds in the 100 ppm of Cu treatments were 2.67% on average compared with the control group. The production efficiency factor {[livability (%)  $\times$  live weight (kg)/age (d)  $\times$  FCR]  $\times$  100} was increased by 5.23% for birds in the antibiotic treatment and by 0.7 to 7.8% for birds in Cu treatments, among which the treatment with 100 ppm of Cu as Cu-SP was highest. The red blood cell level, hematocrit level, and mean corpuscular volume of birds in the Cu treatments were lower than were those of birds in the control group. Copper concentration in the liver increased as the level of Cu supplementation increased. The populations of lactobacilli and total bacteria increased, and that of Escherichia coli decreased as the level of Cu increased, whereas all microbes, including Clostridium perfringens, decreased in the antibiotic treatment.

Key words:

antibioticbroilercopper

## http://ps.oxfordjournals.org/content/86/3/531.abstract

Effects of Dietary Copper Supplementation and Copper Source on Digesta pH, Calcium, Zinc, and Copper Complex Size in the Gastrointestinal Tract of the Broiler Chicken

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Received September 7, 2006. Accepted November 27, 2006.

### **Abstract**

An experiment was conducted to study the effects of high dietary Cu and Cu source on the pH of digesta from the gizzard, duodenum + jejunum, ileum, and complex size of Ca, Zn, and Cu in the duodenum + jejunum digesta of broiler chickens. Ross 308 male broiler chicks were randomly assigned to 32 cages and fed 1 of 4 treatments: control, 250 ppm Cu from sulfate, 250 ppm Cu from lysinate, and 250 ppm tribasic Cu from chloride from 15 to 21 d of age. Copper supplementation and Cu source had no effects on pH of gizzard or duodenum + jejunum contents.

Copper supplementation, however, increased the pH of the ileal contents (P < 0.05) but was not affected by Cu source. Neither Cu supplementation nor Cu source had significant effects on the solubility of Ca in the duodenum + jejunum contents, and the portions of Ca existing in different soluble complex sizes: >100,000, 100,000 to 30,000, 30,000 to 5,000, and <5,000 molecular weight (MW) in the duodenum + jejunum digesta. About 80% of soluble Ca, Cu, and Zn was associated with either large complexes (>100,000 MW) or small complexes (<5,000 MW). The solubility of supplemental Cu in digesta was from 59 to 61% (P < 0.05), but solubility was not affected by Cu source. No effects on portions of Cu existing in different sizes of complexes in the supernatant were noted. Copper lysinate decreased the Zn solubility in the digesta (P < 0.05), but Cu sulfate and tribasic Cu chloride supplementation did not. Copper supplementation increased (P < 0.05) the

percentage of Zn associated with large complexes (>100,000 MW) and decreased (P < 0.05) the percentage of Zn associated with small complexes (<5,000 MW; P < 0.05), thereby suggesting an antagonism between Cu and Zn.

## Cranberries

Another study has been performed where the effectiveness at preventing UTIs by antibiotics (TMP-SMX) was compared to that of **cranberry** extract (Cranmax®; Proprietary Nutritionals, USA)

- •Vitamin C. Vitamin C's role in preventing and treating infectious disease is well established. Intravenous vitamin C is an option, but if you don't have access to a practitioner who can administer it, liposomal vitamin C is the most potent oral form. For more information on vitamin C, listen to my interview with Dr. Ronald Hunninghake, an internationally recognized vitamin C expert. If you choose to supplement with vitamin C, liposomal C seems to be the best form to use.
- •Garlic. Garlic is a powerful antibacterial, antiviral and antifungal. It can stimulate your immune system, help wounds heal, and kill antibiotic-resistant bacteria (including MRSA and multi-drug resistant tuberculosis), plus it has shown more than 100 other health promoting properties.9 For highest potency, the garlic should be eaten fresh and raw (chopped or smashed.)
- •Olive leaf extract. In vitro studies show olive leaf extract is effective against Klebsiella, a gramnegative bacteria, inhibiting its replication, in addition to being toxic to other pathogenic microbes.
- •Manuka honey. Manuka honey, made from the flowers and pollen of the Manuka bush, has been shown to be more effective than antibiotics in the treatment of serious, hard-to-heal skin infections. Clinical trials have found Manuka honey can effectively eradicate more than 250 clinical strains of bacteria, including resistant varieties such as MRSA.
- •**Tea tree oil.** Tea tree oil is a natural antiseptic proven to kill many bacterial strains (including MRSA).10
- •Colloidal silver. Colloidal silver has been regarded as an effective natural antibiotic for centuries, and recent research shows it can even help eradicate antibiotic-resistant pathogens. If you are interested in this treatment, make sure you read the latest guidelines for safe usage of colloidal silver as there are risks with using it improperly.
- •Copper. Replacing fixtures with certain copper alloys can help kill bacteria, even superbugs. Installing copper faucets, light switches, toilet seats and push plates in germ-infested areas such as hospitals and nursing homes could potentially save thousands of lives each year.

In its most common form, the **potassium salt, potassium clavulanate** 

Some cations, such as **Ca2+**, **Mg2+**, **and Na+**, can inhibit the antimicrobial activity of cationic peptides (18, 32).

## Citrate salts

## Carbonate salts

Supplement Recommendations based on UTM/UEE Test Results

## **Increasing Calcium**

### Nettle

!Chamomile

**!Chervil** 

|Cal/Mag/Vit D/Vit K (Calcium & Magnesium Citrates)

Bone Support RNA

**Increasing Other Essential Minerals** 

|Sodium : Aerobic O7 | Potassium: Aerobic KO7

!Phosphorus (Complexed Phosphorus)

¦Magnesium ?Mag O7

## ?Magnesium Citrate

?Krebs Magnesium-Potassium Chelates

?Magnesium Drops

## ?Magnesium Malate Forte

Zinc

?Zinc Lozenges

?Zinc Drops

?Krebs Zinc (only if MAP or OAT tests also indicate a need for Krebs support)

## Copper

?BioThyro

?Cell food

## **|Manganese**

**| Molybdenum** 

Boron

### **!Chromium Picolinate**

!Lithium Orotate

## **Selenium Drops or tablets**

Strontium Support II

## |Vanadyl (Vanadyl Sulfate)

- <u>Oregano</u>: Early research shows that taking oregano by mouth may help treat parasites. Further research is needed to confirm these results.
- Oregano: Early study shows that taking oregano by mouth may help get rid of parasites. Further research is needed to confirm these results. Research suggests that oregano is well tolerated in recommended doses. Avoid if allergic or hypersensitive to oregano or to other herbs from the Lamiaceae family including hyssop, basil marjoram, mint, sage and <a href="lavender">lavender</a>. Use cautiously with diabetes and bleeding disorders because oregano may increase the risk of bleeding or decrease blood sugar levels. Pregnant or breastfeeding women should not consume oregano at doses above those normally found in food.
- Oregano: Early study shows that taking oregano by mouth may help treat parasites. Further research is needed to confirm these results. Avoid in individuals with a known allergy or hypersensitivity to oregano. Based on historical use, it appears that oregano is well tolerated in recommended doses. However, reliable clinical trials demonstrating safety or efficacy of a particular dose or for a recommended treatment duration are currently lacking in the available literature. Oregano may lower blood sugar levels. Caution is advised in patients with diabetes or hypoglycemia, and in those taking drugs, herbs, or supplements that affect blood sugar. Serum glucose levels may need to be monitored by a healthcare provider, and medication adjustments may be necessary. Oregano is not recommended at doses above those normally found in food during pregnancy and lactation due to a lack of available scientific evidence.

Carbapenem Resistant Enterobacteriaceae

http://www.liofilchem.net/en/mov\_enteropluritest.php

BETA-LAC CONFIRM

Health care professionals may recommend herbs such as DGL-licorice (Glycyrrhiza glabra)

## Copper chelate malic acid

-Escherichia coli

toxicity involves the action of reactive oxygen species. Low micromolar levels of copper were sufficient to inhibit the growth of both WT and mutant strains

## copper

blocks their biosynthesis. Indeed, copper treatment rapidly inactivated isopropylmalate

dehydratase, an iron-sulfur cluster enzyme in this pathway suggesting that Cu(I) damages these proteins by liganding to the coordinating sulfur atoms.

Microbes are particularly vulnerable to metal poisoning because they cannot control their extracellular environment

The mechanism by which copper poisons cells has been elusive. A long-standing hypothesis is that copper reacts with endogenous H2O2 to generate hydroxyl radicals in a process analogous to the Fenton reaction:

Indeed, we found that copper-stressed cells produced endogenous H2O2 at a rate several fold higher than did untreated cells (data not shown). Excessive levels of superoxide (O2\_) and H2O2 can disrupt several amino acid biosynthetic pathways (18–20); therefore, we tested whether copper creates similar blocks. (This experiment was complicated

by the fact that \_-amino acids chelate copper and thereby increase the dose necessary for toxicity; to correct for this effect, alanine was added in equivalent doses to cultures from which amino acids of interest were withheld.) The *copA cueO cusCFBA* mutant could not grow aerobically in glucose/alanine medium to which 10 \_M copper was added (Fig. 2A). The addition of branched-chain amino acids partially restored growth, indicating that this pathway is a primary target of copper toxicity. The failure to fully restore growth indicates that additional growthlimiting damage also occurred outside of this pathway.

**Copper Inactivated Iron-Sulfur Cluster Dehydratases Inside Aerobic Cells.** Reactive oxygen species block branched-chain biosynthesis because they directly damage the iron-sulfur clusters of 2 dehydratases: dihydroxy-acid dehydratase in the common branchedchain pathway and isopropylmalate isomerase (IPMI) in the leucine-specific branch (18, 21–23). In conformity with the

**The Mechanism of Copper Toxicity Is Independent of Oxygen.** Other investigators have noted that copper can poison bacterial cells in anaerobic medium (12, 17, 24), and we observed, in fact, that both WT cells and the *copA cueO cusCFBA* mutant were much more sensitive to growth inhibition by copper when oxygen was absent (Fig. S2). The anaerobic growth of the WT and mutant strains was effectively blocked by 1 M and 125 nM copper, respectively. At the very least,

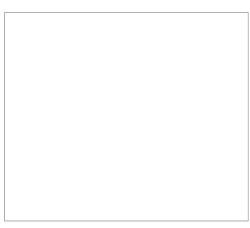
this means that copper has an acute mechanism of toxicity that does not involve reactive oxygen species.

Surprisingly, experiments suggested that the primary target of anaerobic toxicity was no different from that in aerobic cells. Growth resumed when branched-chain amino acids were added to anaerobic medium (Fig. 3). This restoration

of growth was not caused by chelation of copper, because the D-enantiomers of the amino acids failed to restore growth (data not shown). Further, an overexpression plasmid carrying *leuCD* fully suppressed the growth defect, indicating that it arose from the lack of IPMI activity (Fig. S1B). Indeed, during anaerobic exposure to 4 \_M copper, the IPMI

activities of bothWT cells and *copA cueO cusCFBA* mutant cells decreased to less than 15% of the activity of untreated cells (Fig. 4A). Copper also damaged fumarase B, the anaerobic isozyme of fumarase A (Fig. 4B). These results indicated that the poisoning of these enzymes by copper occurred through a non-oxidative mechanism. It is likely that the same was true in aerobic media

**Copper Damaged the Iron-Sulfur Cluster of Fumarase A.** Saturating concentrations of malate, a substrate of fumarase A, fully protected the enzyme activity from copper (Fig. 5*B*). This effect was probably mediated by its shielding the enzyme active site, as the protective effect tracked closely to the dose needed to form an ES complex.



**Fig. 7.** The ability of Cu(I) to damage iron-sulfur clusters is modulated by copper-binding metabolites and requires that clusters be solvent-exposed. (*A*) Fumarase A (4 \_M) was anaerobically challenged with 10 \_M Cu(I) at 27 °C without supplementation (open squares) or in the presence of 100 M histidine (*his*) (closed circles) or 100 M reduced glutathione (*GSH*) (closed diamonds).

The data are a representative of 3 independent experiments. (*B*) Fumarase A (0.1 \_M) was mixed with sulfite reductase (*SiRase*), and the

enzyme mixture was then challenged anaerobically with  $0 \, \underline{\hspace{0.1cm}}$  M(open bars) or  $10 \, \underline{\hspace{0.1cm}}$  M Cu(I) (gray bars) for 3 min at 27 °C. The data are the average of 3 independent experiments, and the error bars represent SD.

## **Discussion**

Life evolved in an anaerobic world that was rich in iron. Evolution exploited the versatile surface chemistry of this metal by integrating it into iron-sulfur clusters that serve to bind substrates into enzyme active sites. This arrangement succeeded for 2 billion years, until the emergence of photosystem 2 caused the gradual oxygenation of the environment. As molecular oxygen accumulated, it oxidized dissolved sulfur compounds, which until that time had sequestered soft metals into mineral precipitates. The subsequent solubilization of these

metals evidently posed a new problem for extant organisms. The present study reveals that one of these metals, copper, rapidly inactivates the catalytic clusters of dehydratases. This enzyme family has representatives in central catabolic and biosynthetic pathways, which therefore become dysfunctional in copper-exposed cells.

These enzymes are uniquely vulnerable to chemical damage because their clusters are substantially exposed to solvent (31). Previous studies show that O2 \_ and H2O2 are small enough to invade the active site, where they coordinate and oxidize the iron-sulfur cluster to an unstable valence (18, 21, 32). In this work, we found that these clusters are also the primary targets of copper, which evidently displaces their <u>iron</u> atoms when it coordinates their thiolate or inorganic sulfur ligands. The extreme avidity with which it does so means that cellular environments that contain such enzymes cannot tolerate even modest levels of active copper.

<u>Activated Charcoal</u> will chelate Aluminum, Arsenic, Cadmium, Chromium, Copper, Iron, Manganese, Platinum, Tin, Thorium, Tungsten. It **will NOT** for Beryllium, Cobalt, Nickel, some Lead, Rubidium, Silver, Uranium and Mercury.

## Molybdenum helps the body maintain the zinc/ copper

I recommend regularly testing both essential minerals (the Urine Essential Elements or UEE test)

**Increasing Calcium** 

Nettle

Chamomile

Chervil

Cal/Mag/Vit D/Vit K (Calcium & Magnesium Citrates)

Bone Support RNA

**Increasing Other Essential Minerals** 

Sodium: Aerobic O7 Potassium: Aerobic KO7

Phosphorus (Complexed Phosphorus)

Magnesium Mag O7

Magnesium Citrate

Krebs Magnesium-Potassium Chelates

Magnesium Drops

Magnesium Malate Forte

Zinc

Zinc Lozenges

Zinc Drops

Krebs Zinc (only if MAP or OAT tests also indicate a need for Krebs support)

Copper BioThyro

Cell food

Manganese - Detoxify excess ammonia

Molybdenum

Boron

Chromium Picolinate

Lithium Orotate

Selenium Drops or tablets

Strontium Support II

Vanadyl (Vanadyl Sulfate)

## **Dr. Larry Wilson**

### Introduction

When medical <u>Science</u> comes to under-stand the implications of a copper imbalance, it may be referred to as the scourge of the late 20th century. It is one of the most com-monly encountered imbalances that we find on tissue mineral tests today. Many of the most prevalent metabolic dysfunctions of our time are related in some way to a copper im-balance.

Copper toxicity is a much overlooked con-tributor to many health problems; including anorexia, fatigue, premenstrual syndrome, depression, anxiety, migraine headaches, al-lergies, childhood hyperactivity and learning disorders.

The involvement of copper toxicity and biounavailability in such a wide range of health conditions may seem unusual. It is our intent in this paper to show how copper is regulated in the body and why it is such a key mineral in so many metabolic dysfunctions.

## Source Naturals Ultra-Mag

**High-Efficiency Magnesium Complex** 

Vitamin B-6 (as pyridoxine HCl)
Magnesium (as magnesium citrate,
taurinate,
malate,
glycinate,
and succinate)

50 mg 2,500%

## **Magnesium taurinate**

- is an essential mineral that is used to treat individuals with low naturally occurring levels of magnesium
- It is used specifically to treat individuals with a natural magnesium deficiency
- Magnesium Taurate: The Best Brain Supplement

The two most common forms of magnesium are Citrate and Sulfate. These are primarily not used for the brain but for cleansing the body of toxins, and also for digestion.

- -Within our brain there is a fine balance of sodium and potassium. Magnesium activates an enzyme that serves to balance the levels of those key chemicals.
- One of the toxins which magnesium takes care of is called ammonia
- Magnesium helps to activate <u>glutamine synthetase</u> this substance converts ammonia into urea
- Other enzymes magnesium activates serve your body by improving the effectiveness of the energy glucose gives us
- which indirectly improves the amount of energy which can be used by the brain
- magnesium taurate contains an <u>amino acid</u> called taurine. Among the many benefits of taurine are the heightened control of neurotransmitters in the brain which can prevent the death of brain cells (and consequently the onset of brain dysfunction and even brain damage).

http://george-eby-research.com/html/depression-anxiety.html

Magnesium must be done cautiously, perhaps with no more than 800 mg daily of magnesium,

## **Xylitol**

## Chitosan

CHITOSAN, 8-24 PER DAY IN THE AM, EVERY third DAY for a month

Parasitol Res. 2013 Aug;112(8):2933-43. doi: 10.1007/s00436-013-3466-4. Epub 2013 Jul 5.

Improved antifilarial activity of ivermectin in chitosan-alginate nanoparticles against human lymphatic filarial parasite, Brugia malayi.

The current antifilarial treatments are not up to the mark partly due to deep location of filarial parasites in the human lymphatic system. We report here on the improvement in the antifilarial activity of ivermectin (IVM) using chitosan-alginate nanoparticles prepared by modified complex coacervation method. The nanoparticles were spherical having 155 nm size and 4.56 and 75.67% loading and entrapment efficiency respectively for IVM. The delivery system maintained the sustained release and significantly augmented the microfilaricidal (MIF) activity at a single low dose (200  $\mu$ g/kg body weight, subcutaneously) in contrast to much higher dose of free ivermectin (400  $\mu$ g/kg body weight, subcutaneously) against human lymphatic filariid, Brugia malayi in rodent host, Mastomys coucha. To substantiate increase in MIF activity, pharmacokinetics study was designed on Wistar rats which revealed a greater peak plasma concentration (45.3  $\pm$  1.79 ng/ml), area under the concentration curve (298  $\pm$  38.7 ng d/ml) and extended mean residence time (23.4  $\pm$  8.56 days)of IVM in chitosan-alginate nanoparticles. Administration of 25 mg/kg of diethylcarbamazine following nanoparticle therapy significantly improved the MIF and macrofilaricidal action of encapsulated drug and was considered superior in this study.

PMID:

## **HUMineral**

**<u>HUMineral</u>** will break up biofilm in gut

## **Probiotic 225**

Probiotic 225 will re establish bacteria biodome.

### Garlic

Chew 1/4 clove fresh chopped **garlic**, let set under tongue for 10 minutes, 153 sulfur compounds, has been shown to release chemicals that regulate body metal and mineral processes, regulate immune chemistries, and restore body health.

## **GuaiAid**

Www.guaiaid.com

(2-4-6) 600mg guaiaid caps

<u>Fibromyalgia Solutions</u> http://www.nevdgp.org.au/info/ArthritisF/management/herbal.htm

http://www.holistic-online.com/Herbal-Med/\_Herbs/h244.htm http://www.nlm.nih.gov/cgi/mesh/2K/MB\_cgi?term=Guaiacol+glyceryl+ether

A LITTLE **GUAI-AID**, AND FUCOIDAN IS HELPFUL AS WELL. HUMINERAL ALSO MAKES BIOFILMS DIE, shorts out the voltage potential of the biofilm.

## Mark London

Please report any links that are not working to mrl@psfc.mit.edu

In the early 1990s, I belonged to the first fibromyalgia discussion group on the internet, the FIBROM-L mailing list. We were probably the first people on the internet to hear about the guaifenesin treatment. Dr. St. Amand claimed that the drug guaifenesin could treat fibromyalgia symptoms by removing excess phosphate from the body, which he believes to be the cause of fibromyalgia. The removal of the phosphate would supposedly lead to a reversal of all fibromyalgia symptoms, which would essentially be as close to a cure as possible. Dr. St. Amand claims that he has successfully reversed all fibromyalgia symptoms in 90% of his patients.

In 1996, before the study was published, I went to a library, and quickly discovered that guaifenesin has a skeletal muscle relaxant property, a fact that people in the fibromyalgia community were not aware of. Surprisingly, anyone could have easily discovered this fact if they looked up guaifenesin in the Merck Index, a drug handbook, which lists guaifenesin as having this effect. Guaifenesin has known neurological effects, but most doctors are unaware of this, because it is no longer used in humans for this effect. However, it is used for this effect in veterinary medicine. And a slightly different form of guaifenesin, guaifenesin carbamate, is used in humans as a muscle relaxant, and is sold under the name Robaxin.

Additionally, any worsening of fibromyalgia symptoms during the treatment, is also a good sign, since these symptoms are attributed to guaifenesin's reversal process, that rids the body of "metabolic debris". Thus, even feeling worse, could make a person feel more optimistic. Plus, many patients are "mapped", i.e. their bodies are examined for lumps, and if the lumps decrease in size, this is also supposedly a sign that the guaifenesin is working by removing the phosphate deposits from the body.

Patients on guaifenesin for fibromyalgia take anywhere from 600 to 3600mg per day. Dr. St. Amand's own wife takes as much as 4800mg per day. So this effect would likely be significant in these people.

Guaifenesin was being used as an expectorant, well before propanediols were discovered, as it can be derived from the bark of the guaiac tree. However, as shall be shown later, guaifenesin doesn't appear to have a direct effect on mucus. Instead, it's possible that its expectorant ability is actually due to its muscle relaxant effect. Some types of expectorants are known to act via a relaxant effect, as the effect helps to soothe spasms and allow mucus to flow easier. Two common herbal remedies that are known to act both as relaxants and expectorants are kava kava and peppermint oil. Some relaxants, like pepperment oil, are also useful for digestion problems such as Irritable Bowel Syndrome, so it's not surprising that some people have reported quaifenesin to be useful for IBS

(although IBS is a multifaceted problem, so relaxants don't work for everyone.) In any event, this shows how a single property can have widespread and diverse effects on the body.

## **Guaifenesin's Analgesic Effect**

But guaifenesin's effect on the nervous system is not simply limited to acting as a muscle relaxant. By the way, guaifenesin is a centuries old remedy, as Dr. St. Amand himself notes. He points out that extracts of the guaiac tree, have a long history of being used for rheumatism. I assume that he mentions this, as a possible proof that it can treat fibromyalgia. However the specific history, is that in the 1500s, explorers to the new world of North America became aware of the guaiac tree, due to the fact that they were looking for remedies to treat untreatable diseases, such as syphilis. Basically, they were looking for a way to make money. They discovered that the local natives were using extracts of the guaiac tree for medicinal properties, and so they tested it on syphilis. The extracts were able to treat the back pain related to syphilis, so they believed that it could therefore treat syphilis. Thus, for a long time, it was more well known for treating syphilis, than rheumatism. The fact that guaifenesin has a history of treating various ailments, supports the theory that it has a more general analgesic effect, rather than a specific effect that only treats fibromyalgia. So it's no wonder that people such as Gregory Penniston, a chiropractor who designed the GuaiLife form of guaifenesin, markets it for a very wide range of pain conditions, such as pelvic pain, Ehlers-Danlos Syndrome and restless leg syndrome, among others.

Dr. St. Amand believes that the increased phosphate excretion is the reason for guaifenesin's benefit

It's not surprising that uricosuric agents cannot affect phosphate excretion. The process in the proximal tubule of the kidneys, where most of the phosphate reabsorption occurs, is highly controlled and specific to phosphate. In that area of the kidneys, there exist "type II sodium-phosphate cotransporters", which control phosphate reabsorption, and they are very specific for phosphate. They are controlled by several mediators of phosphate homeostasis (eg, parathyroid hormone [PTH], dopamine, dietary phosphate). If a drug could simply affect phosphate excretion, and not other minerals, then that would be of remarkable help for many hyperphosphate disorders. Right now, the way to treat such disorders, is via a low phosphate diet, combined with using phosphate binders that block the absorption of dietary phosphate. In severe case, diuretics are also used. However, these methods are not always very successful, or can create side effects. A drug that could remove only phosphate, without affecting other minerals, and without the need to change one's diet, would be a great discovery.

The reason for any possible initial increase in mineral excretion, that is seen from guaifenesin, might be due to the fact that guafenesin is metabolized by the liver into an acid, which is then excreted into the urine. In theory, this could increase urinary acidity, and increased urinary acidity has been associated with increased calcium excretion. This might explain why Dr. Bennett's study showed a small but significant increase in urinary calcium. However, when initially starting guaifenesin, there might be large increases in mineral excretion, until the body adapts to the changes. For example, high protein diets that increase urinary acidity, can initially increase mineral excretion, especially calcium However, a recent long term study on such a diet, has shown that such effects disappear after several weeks. Thus, only long term studies show the true effects.

Phosphorus, commonly referred to as phosphate, is one of the most common and most necessary minerals in the body. Phosphate is used everywhere, from the building of bones, to balancing the body's PH, and most important, for providing energy to run the body, via the formation of ATP. However, since phosphate is so common in the foods we eat, a phosphate deficiency is rare. And so is an excess of phosphate.

This is because the kidneys are the main factor in regulating proper phosphate levels in the body. And the kidneys are well able to excrete very large amounts of excess phosphate, up to several times the amount normally found in one's diet. Kidney functioning must fail by at least 50%, before they lose their ability to excrete the amount of excess phosphate that is ingested.

There are several factors that influence the rate of phosphate excretion by the kidneys. The main influence is the parathyroid glands, as they controls excretion rates via the production of parathyroid hormones, or PTH. Thus, phosphate problems mainly occur due to either kidney or parathyroid problems.

If phosphate excretion is too low, phosphate serum levels rise, resulting in the condition known as hyperphosphatemia This is normally due to either kidney failure, parathyroid deficiency (hypoparathyroidism), or due to the body not reacting properly to parathyroid hormone. The Merck Manual pages that relate to this condition are found here:

## http://www.merck.com/pubs/mmanual/section2/chapter12/12e.htm

Dr. St. Amand believes that the calcium phosphate deposits in cells is the cause of lower levels of ATP, which is found in fibromyalgia. ATP is a key chemical that the body creates for storing energy. However, studies have shown no relationship to the level of ATP and actual fibromyalgia symptoms. And there have been no published studies which have found that excess phosphate is associated with ATP depletion, or for that matter, any fibromyalgia symptoms. But there are studies which show that ATP deficiencies are found in people with phosphate deficiencies, which is not surprising, since ATP requires phosphate. In fact, one study has found that some people with chronic fatigue syndrome have phosphate diabetes, a condition caused by kidneys excreting too much phosphate.

If deposits in cells is the cause of fibromyalgia, then fibromyalgia should develop slowly, as the deposits slowly grow. And the disease should be progressive, i.e. the deposits would continue to keep growing, as in conditions such as hyperphosphatemia. This would then cause symptoms to constantly get worse. However, fibromyalgia is not believed to be a progessive disease, and many people with fibromyalgia develop it in a very short period. And if fibromyalgia was a truly a disease caused by ATP depletion from these deposits, then fibromyalgia symptoms and other ATP depletion symptoms, should overlap. For example, ATP depletion can cause muscle problems such as rhabdomyolysis. However, no such conditions are observed in fibromyalgia.

Bones are not only formed from calcium and phosphate, but also from magnesium. Without magnesium, the resulting formations will be soft. Teeth will have soft enamel, nails will be brittle, symptoms which match Dr. St. Amand's observations.

Magnesium is extremely necessary for proper ATP synthesis, because ATP is stored in the body as a combination of magnesium and ATP, which is known as MgATP. ATP requires magnesium in order to be stable. Without magnesium, ATP would easily break down into other components, ADP and inorganic phosphate.

Magnesium deficiency is very common in the general US population. Not only is our daily intake low, but we eat a diet which increases the demand for magnesium.

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=1384353&dopt=Abstract

Unfortunately, magnesium deficiency is not easily detected, as serum levels do not reflect the levels of magnesium in tissues. This is the reason why it is so overlooked and ignored, both by doctors and by studies. And unfortunately, oral magnesium supplementation can be difficult because of absorption problems. Digestion and diet play a key role in absorption. People with fibromyalgia often have conditions like Irritable Bowel System, gluten intolerance, or other problems that might limit absorption. Phosphate can bind to magnesium in the gut, creating magnesium phosphate, an insoluble salt that can't be utilized. Many forms of oral magnesium supplements are hard to assimilate. The most common, magnesium oxide and citrate, happen to be the worst to assimilate, which is why both have a strong laxative effect. If you suffer from that effect when you take magnesium, it is often not because you are taking too much, but because you are not assimilating it well. And it may take long term use of supplements before magnesium levels are raised in all the tissues, and for damaged cell functions to be restored.

Therefore, the symptoms which Dr. St. Amand has attributed to an excess of phosphate, would more likely be due to a magnesium deficiency.

in 1994 by a Dr. John Couvaras, an infertility doctor, who began using heparin for fertility problems, and discovered that it helped many symptoms of his patients who also had CFS and fibromyalgia. Perhaps not so coincidentally, guaifenesin is also known to have the ability to increase fertility (originally it was thought that this effect from guaifenesin was due to thinning of cervical mucus. But guaifenesin has not been found to have a direct effect on thinning mucus, but instead simply stimulates mucus glands to allow more mucus to flow, possibly by irritating gastric linings. This effect is not likely to occur in the cervix, because little if any guaifenesin could appear there. Plus, it's the thinning of the mucus which is important, not increased mucus flow. Thinning mucus occurs due to a raise in estrogen levels, and coincidentally estrogen inhibits platetlet aggregation.)

There are several reasons given as to possibly why anticoagulants have helped some people. But there is one specific effect that might be very relevant for fibromyalgia. In a recent study on fibromyalgia, it's been found that some fibromyalgia symptoms coorelate with lower levels of serum serotonin and higher levels of plasma serotonin. See:

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=11203736&dopt=Abstract

But platelet activation, which causes platelet aggregation, also causes the release of serotonin, resulting in high plasma serotonin In addition, only in the last few years has it been recognized that serotonin influences many other problems, such as migraines, hypoglycemia, asthma, Raynaud's, and IBS, all conditions which are associated with fibromyalgia. Some of these conditions are exacerbated due to serotonin's ability to cause constriction. However, Dr. Couvaras has said that migraines, irritable bowel syndrome, and pelvic pain, all went away when he put his patients on heparin. Interestingly, Dr. St. Amand has also claimed that guaifenesin is able to treat many different conditions. An imbalance of serotonin in the blood could be the link that connects all these conditions.

Hypoglycemia is one of the more interesting conditions related to serotonin, as it is especially common in fibromyalgia. It is so common, that Dr. St. Amand himself regularly prescribes a diet for hypoglycemia to many of his patients, and it is often an integral part of his treatment in combination with guaifenesin.

However, hypoglycemia can be influenced by a serotonin release, as serotonin has been shown to increase insulin levels. Not only that, but platelet aggregation sensitivity is increased due to hypoglycemia. Thus, this is one possible explanation of why hypoglycemia is so common. (As an aside, so many people have remarked how helpful the diet is, that anyone considering going on the guaifenesin protocol and the diet, might want to first try the diet alone, in order to be able to tell which effects are occurring from diet, and which effects are from the guaifenesin.)

Other commonly seen conditions also have a serotonin link. For example, plasma serotinin in celiac patients has been found to be elevated. Problems associated with blood pressure, such as Neurally Mediated Hypotension, are also influenced by serotonin.

In addition, platelet activity causes a release of other substances that might be affecting fibromyalgia. For example, ATP is also released, and this might be the cause of reduced levels of ATP found in red blood cells of people with fibromyalgia.

Both of the previous uricosuric drugs used by Dr. St. Amand, anturane and probenecid, also affect platelet activity. Anturane (sulphinpyrazone) is well known for having antiplatelet activity. Probenecid's effect is a bit different. It's able to block a number of different aggregating agents. And its main effect may be due to its ability to inactivate thrombin, which is the cause of platelet activation which leads to the secretion of serotonin from the serum to the plasma. See the following studies:

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=8865538&dopt=Abstract

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=23532&dopt=Abstract

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=2474820&dopt=Abstract

But what's more, is that an anticoagulant effect might be the reason for the increased phosphate excretion. The clue to this possibility is a recent report of a patient being treated with probenecid for calcinosis:

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=11361202&dopt=Abstract

According to most studies, probenecid does not cause phosphate excretion in either non-gout or gout patients. However, there are several reports in the medical literature of it occurring. But they are so rare, that whenever a case occurs, its reported in a medical journal. In the above report, the patient had Juvenile Dermatomyositis which led to calcinosis, a condition where calcium is abnormally deposited around bones, causing severely limited mobility. The patient also had hyperphosphatemia, and probenecid was able to reverse this condition by increasing phosphate excretion, and this led to reversing the calcinosis.

However, it's possible that the hyperphosphatemia seen in this patient was due to a drug that she was taking, which was Cyclosporin A. This drug is known to cause platelet aggregation and high plasma serotonin levels:

http://www.sums.ac.ir/~ijms/9834/fardaee9834.html

This paper mentions that impaired renal functioning and reduced renal plasma flow also occur with this drug. The impaired renal functioning could lead to phosphate retention. If an anticoagulant could improve renal flow, then theoretically this could cause increased phosphate excretion.

And there is possible proof that the phosphate excretion from probenecid is due to an effect other than the uricosuric effect. Here is a study of an earlier case of probenecid being successfully used for calcinosis:

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=9360503&dopt=Abstract

In that case, phoshate excretion occurred even without a significant increase in uric acid excretion. In other words, the two effects might be unrelated.

This would also explain the puzzle of why uricosuric drugs produce increased uric acid excretion in normal people, yet phosphate excretion does not occur. They are two separate effects, and the phosphate excretion would only occur in people who had impaired platelet functioning.

But it should be pointed out that not all anticoagulants affect the impaired renal functioning which is caused by platelet aggregation. This is not surprising, as there are several different pathways involved in platelet aggregation Thus, different drugs inhibit aggregation in different ways. In the following study, renal impairment was induced by endotoxin, a platelet growth factor which causes aggregation. In high enough doses, endotoxin is able to cause reduced phosphate excretion. Heparin had no effect on the renal impairment, while aspirin was able to restore proper renal functioning:

http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\_uids=1667536&dopt=Abstract

Heparin is strictly an anticoagulant, while aspirin is an antiplatelet drug. This study theorized that only antiplatelet drugs could reverse the renal impairment caused by platelet aggregation, If platelet activity is a factor in fibromyalgia, then how is it that other drugs and supplements for fibromyalgia are also effective, yet they don't have any effect on platelet activity? The answer is that coincidentally (or perhaps not), most other drugs and supplements for fibromyalgia do inhibit platelet aggregation.

All of the following have some affect on platelet activity:

Some antidepressants, especially tricyclics, benzodiazepines such as xanax and valium, antihistamines such as benadryl, anesthetics such as procaine, supplements such as MSM, ginko, pycnogenol, **quercetin**, and bromelain, magnesium, B12 (homocysteine increases platelet aggregation), whey (treats glutathione deficiency, which causes platelet aggregation), some amino acids such as taurine and arginine, and relaxin (presently experimentally used for fibromyalgia). Thus, like guaifenesin, all of these substances are recommended for use for fibromyalgia, but they also have some ability to inhibit platelet aggregation

Nattokinase blood clot dissolver.

<u>Lutein</u> is found is Eyesight Rx, a potent vision enhancer, this carotenoid, along with zeaxanthin, is found in the retina of the eye and helps with visual acuity Lycopene tomato antioxidant

Serrapeptase anti-inflammatory enzyme information and supplement

<u>St. John's wort</u> don't worry, be happy, how to use this natural antidepressant to lift mood and reduce the need for prescription antidepressants

### 5.5.2 Graviola

Graviola Bark: Graviola is a major component used in our formula because of the amazing results for purging parasites. Graviola has a great record in kiling intestinal parasites, calming nerves, reducing blood pressure and helping arthritis, heart and liver.http://www.graviolaleaves.com/ Some people saw parasites in their toilet's after just one day! Graviola has been researched in laboratory tests since the 1970s, where it's been shown to: Effectively target and kill malignant cells in 12 different types of cancer, including Colon, Breast, Prostrate, Lung and Pancreatic Cancer...10,000 times stronger in killing colon cancer cells than Adriamycin (a commonly used chemotherapeutic drug). Selectively hunt down and kill cancer cells without harming healthy cells, unlike chemotherapy. REFERANCE <a href="http://www.greenwoodhealth.net/np/graviola.htm">http://www.greenwoodhealth.net/np/graviola.htm</a> Graviola contains the chemical; Annonaceous acetogenins the active compound that is harmful to parasites.

## 5.5.3 Quassia

Quassia: a tree native to Jamaica and its neighboring islands, has traditionally been used as a remedy for roundworms and as an insecticide. It has also been used as a bitter digestive aid and a remedy for digestive disorders, parasites, and head lice.http://www.naturalstandard.com Quassia wood is very commonly used as a bitter tonic and anthelmintic.

Small cups known as "Bitter Cups" are sometimes made of the wood, and water standing in them soon acquires the medicinal properties of the wood. This water, or an infusion of 1 ounce of the chips in 1 pint of cold water is taken in wineglass doses as a remedy for indigestion and general debility of the digestive system. Quassia infusion is also given to children suffering from worms, in appropriate doses according to age. Midges, gnats, and other insect pests may be kept away by damping the hands and face with the liquid.

The history of Quassia wood as an agent in non-poisonous herbal medicine is interesting. The curative properties of the wood were first brought to general notice through a negro slave named Quassy, whose people in his native country of Surinam, used it as a remedy for the various fevers to which they were subject. Quassy communicated his knowledge of the tree's virtues to Daniel Rolander, a Swede, who brought specimens to Europe in 1755.

## 5.5.4 Butternut Bark

Butternut bark: used specifically for parasite cleansing. Butternut is a native of the midwestern and northeastern United States and been used since the 1800s as a laxative and in the elimination of parasites. Butternut is also used to support healthy liver function. Butternut, also called White Walnut, is used to expel, rather than kill, worms (vermifuge)

## **Alinia**

TRY <u>ALINIA</u> FOR <u>IRON</u> EATING GUYS. IT IS A REAL MIRACLE, EVEN PROTOZOA THAT EAT <u>IRON</u> ARE FLUSHED IN A FEW WEEKS. Alinia also kills Ascaris L3. Alinia at 15mg/kg/D kills a spectrum of roundworms

# How to Stop a Bacterial Herx Reaction

(All Must be Taken Together):

Jarrow Milk Thistle – Follow general supplementation recommendation on bottle.

Sun Chlorella - Follow box instructions 3,000 mg+

Vitamin C Ester C – Take vitamin C in divided doses two hours apart of 3,000 mg and don't take more than 30,000 mg a day.

Upgraded Activated <u>charcoal</u> – Follow general supplementation recommendation on bottle; don't take more than twelve capsules a day 4,000 mg L-glutamine
Daily Epsom Salt Bath Stay Hydrated

## **Heal the lining of the gut**

Flaxseed ground in yogurt GLA from evening primrose oil, zinc, vitamin A Probiotic – take every few days in yogurt for two months

## Gerd

Not to be confused with Ascaris birth, lung, acid throat, acid reflux. Gerd and Gerd symptoms can come from several sources, even when infected with parasites. I used Quercetain and caprilic acid (coconut or coconut derivative).

Common GERD treatment is:

For three months: 600 mg Magnesium glycinate – Take before bed

For a month: 8,000 Mg L-glutamine – Take in divided doses, 3x a day on an empty

stomach if possible.<sup>22</sup>

Life Extension Bio Curcumin – Follow general supplementation recommendation on bottle. 23

N-acetylglucosamine (Do not use if allergic to shellfish) –Follow general supplementation recommendation on bottle. <sup>24</sup>

GERD Protocol Two: H. pylori and Ulcers

GERD Protocols Three - Six: <u>Diet Modification</u>, <u>Clothing</u>, and <u>Posture</u>

GERD Protocols Seven - Eight: Strengthen LES, and Digestive Enzymes

GERD Protocol Nine: Candida

GERD Protocol Ten – Eleven: Constipation Relief and Salt!

## Diarrhea

Prolonged periods of Diarrhea are an indicator sign of parasitosis. The parasite infection can come with immune, bacteria, viral, mold, yeast, or other infections. Determining which of these are present, and how to attack each one is part of the parasitology cure process.

## Dysbiosis bacterial regulation imbalance

http://glutenfreeworks.com/blog/2010/07/20/probiotics-and-prebiotics-can-improve-health-of-celiacs/

Intestinal enzyme deficiencies, sugar intolerances and associated dysbiosis, or imbalance of intestinal microbes, called flora, appear commonly in persons with celiac disease. Research indicates that the metabolic activity of intestinal microbial flora in celiacs is

different from the general population and that it is a genuine phenomenon of celiac disease not affected by either the diet, the inflammation, or the autoimmune status of the patient. The severity of disturbances in intestinal balance of flora was found to depend on the gravity of the patients' state. 3

Celiac reactions work in the following way. Undigested fragments of gluten cause a "leaky gut syndrome" by relaxing the normally tight intercellular junctions that function to prevent large molecules from slipping through the lining. Penetrating gluten fragments become bound by the intestinal enzyme tissue transglutaminase (tTG) to form a molecule that triggers the development of antibodies. These antibodies then attack the altered gluten molecule within the lining. The immune reaction within the gut lining causes inflammation and damage to the delicate structures of the lining, leading to failure to digest and absorb nutrients.

# Malabsorption leads to malnutrition that then brings about malfunction of any or all body systems, depending on the nutrients that are missing.

Malnutrition, by depriving our cells of nutrients, encourages infection. Proteins are needed for tissue regeneration and repair and for producing enzymes needed to properly digest and metabolize food. Iron, zinc, copper, vitamin C and riboflavin are needed for proper blood cell formation and activity needed to fight infection. Vitamin D, omega-3 fatty acids and selenium also plays a role in immunity, while vitamin A and niacin are needed for the integrity of the intestinal lining itself.

In this way hundreds of diverse health problems may develop from celiac disease. Various microbial imbalances stem from the unnatural passage of undigested fat, carbohydrates and protein into the colon, poor gut motility, and certain nutrient deficiencies.

Passage of unabsorbed nutrients into the colon results from pancreatic insufficiency, bile insufficiency and digestive enzyme deficiencies.

- 1. **Pancreatic insufficiency** results in deficiency of pancreatic digestive enzymes lipase, amylase, and protease needed for the digestion of fat, carbohydrates, and protein.
- 2. **Bile insufficiency** results in impaired secretion of bile by the liver, obstruction of the bile ducts and abnormal circulation of **bile salts** that impairs the digestion of fats.
- 3. Deficiency of lactase, sucrase, maltase and proteases result in failure to finish the digestion of sugars and protein required for absorption. Undigested nutrients arriving in the colon cause excessive fermentation and drawing of water from the bloodstream into the colon. These conditions produce symptoms such as abdominal pain, watery diarrhea, IBS-like symptoms, bloating, and gas. Pale foul-smelling stool that float or stick to the toilet bowl results from fat malabsorption.
- 4. Altered gut motility and low stomach acidity commonly found in celiac disease promote fermentation in the large bowel and also overgrowth of yeast and bacteria in the small bowel, both serious conditions.

The health and integrity of intestinal cells depends on adequate availability of niacin, zinc and vitamin A. Deficiency of these nutrients sets the stage for inflammation, infection and diarrhea that worsen gut function and induce overpopulation by pathogens.

For these reasons, persons with celiac disease should learn about and properly use both probiotics and prebiotics to improve their overall health and specifically their intestinal health. This is especially important if they continue to experience fatigue,

weakness, achiness, depression, foggy thinking and digestive problems while maintaining a gluten-free diet.

**Probiotic supplements and fermented foods,** such as yogurt and unpasteurized apple cider vinegar, replace lost or reduced health-producing bacteria populations, such as lactobacillus and Bifidobacterium, in the colon.

**Prebiotics**, including flax, honey, greens (especially dandelion greens, but also spinach, collard greens, chard, kale, and mustard greens), berries, bananas, and other fruit, legumes (lentils, kidney beans, chickpeas, navy beans, white beans, black beans), will stimulate their growth.

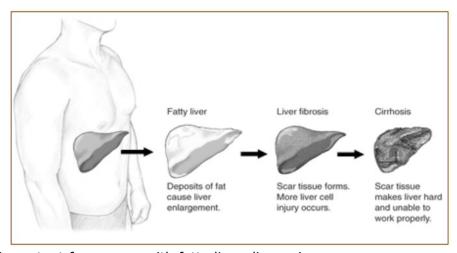
Indeed, good health depends on good balance of intestinal bacteria.

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## **NAFLD**

http://glutenfreeworks.com/blog/2007/11/16/non-alcoholic-fatty-liver-disease-and-gluten/



This is

super important for anyone with fatty liver disease!

While we were at Columbia University's Topics in Gastroenterology, Dr. Steven Lobritto talked about cirrhosis of the liver and how he has actually seen people who were on the liver transplant list heal enough to be taken off once they started a gluten-free diet. That's right. People who needed liver transplants – their liver's were basically done for – healed!

Non-alcoholic fatty liver is a non-inflammatory hepatic (liver) disorder characterized by degenerative changes in the liver secondary to excessive accumulation of lipid in hepatocytes.

According to research we found for our book, "Recognizing Celiac Disease" 3.4% of people with non-alcoholic fatty liver disease have SILENT Celiac Disease. That means they don't have symptoms. Most patients DO NOT have gastrointestinal symptoms.

The good news is that studies showed liver enzymes can normalize after 6 months on a gluten-free diet. If you or your family members have non-alcoholic fatty liver (cirrhosis), but have not been tested for celiac disease, get tested and give them this information so they can get tested. And if they test negative, try the diet anyway because we've seen time and time again that the test are NOT 100% accurate.

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## **IBS**

## **Hydrogen Sulfide**

Hydrogen Sulfide gas smells like rotten eggs.

ARTICLE SOURCE: http://www.webmd.com:80/ibs/news/20081219/new-ibs-quidelines-offer-treatment-ideas

### **New IBS Guidelines**

American College of Gastroenterology Updates Recommendations for Irritable Bowel Syndrome By Bill Hendrick

WebMD Health NewsReviewed by Louise Chang, MDDec. 19, 2008 — New guidelines have been issued by the nation's gastroenterologists that are aimed at easing the abdominal pain, diarrhea, and other symptoms of irritable bowel syndrome (IBS), which afflicts millions of Americans.

The guidelines, issued by the American College of Gastroenterology, also offer hope to patients who've struggled with the condition and found satisfactory treatments lacking.

IBS is diagnosed in people whose symptoms include abdominal pain, bloating, gas, diarrhea, and constipation, or a combination of these symptoms. Though sometimes confused with inflammatory bowel disease, which includes Crohn's disease and ulcerative colitis, IBS is a separate condition.

IBS care uses up more than \$20 billion a year in direct and indirect expenditures, according to William Chey, MD, professor of medicine and director of the Gastrointestinal Physiology Laboratory at the University of Michigan Health System. He developed the guidelines in conjunction with Philip Schoenfeld, MD.

"The last time the American College of Gastroenterology published guidelines for the management of IBS was in 2002, and the College recognized that in the span of five to six years there has been a remarkable explosion in knowledge that's become available that's helped us to understand the cause and management of IBS," Chey says in a news release.

### **Tests and Treatments for IBS**

## **According to the new guidelines:**

Patients with symptoms typical for IBS — and without alarm features like rectal bleeding, low blood count due to <u>iron</u> deficiency, weight loss, or a family history of colon cancer, IBD, or celiac disease — do not need extensive testing before being diagnosed.

IBS patients with diarrhea, or a combination of constipation and diarrhea, should be screened with blood tests for celiac disease, a disorder in which patients can't tolerate the gluten protein found in wheat or other grains.

When IBS patients have alarm features or are over 50 years old, they should have further tests (such as colonoscopy) to rule out other bowel disease such as IBD and colon cancer.

IBS patients and their doctors should consider treatments involving antidepressants, which have been shown to offer relief.

The drug Amitiza helps with women who have IBS with constipation; the non-absorbable antibiotic rifaximin can ease IBS and bloating as a short-term treatment. And Lotronex, a drug that affects serotonin receptors, can be considered for patients with severe IBS with diarrhea.

Certain anti-spasm treatments may offer short-term help with abdominal pain from IBS. These include hyoscine, cimetropium, and peppermint oil.

A probiotic called Bifidobacteria may help some IBS patients.

According to the guidelines, women are twice as likely as men to suffer from IBS, which often begins in young adulthood. Gastroenterologists have found that dietary changes have proved helpful, including the addition of dietary fiber supplements such as psyllium.

Chey says IBS can be managed in most patients with counseling, dietary and lifestyle interventions, and use of both over-the-counter and prescription medications.

The guidelines suggest many treatments might be tried, though the authors concede no single magical answer has yet been found to eliminate symptoms in IBS patients. But the guidelines offer hope for people with IBS that their doctors can try a number of methods to reduce discomfort, and that some of the steps that can be taken seem to work.

## **GDX Protocols**

Common symptoms of SIBO are gas, bloating, diarrhea, constipation, fat in the stool, anemia, B12 deficiencies and increased intestinal permeability. Do these symptoms strike any of you IBS (or gluten) sufferers as vaguely familiar? Yeah I thought so.

SIBO is a medical condition where a person has an opportunistic bacterial infection in the small intestine. 1 This situation is usually created by both a poor American diet or a long term use of a PPI medication (Proton Pump Inhibitor). Long term use of PPI's medications causes opportunistic bacteria that would normally be eliminated by stomach acid, to survive and flourish in the small intestine. Also, the standard American diet of fermenting carbohydrates cause the opportunistic bacteria to thrive, infect the small intestine, and produce excess gas. A lack of stomach acid also causes food proteins to become partially undigested. Allergies from the undigested proteins occur, and an increase of gas and inflammation also happen. 2

The opportunistic bacteria also make protective biofilms (one of the most common examples of a biofilm is the "film" on your teeth that appears when you don't brush for awhile) which make eradication with antibiotics very difficult. The biofilm protects the opportunistic bacteria from antibiotic treatment, bactericides, and probiotics. To eliminate the opportunistic bacteria you also have to disrupt the biofilm that protects the bacteria. Biofilm disruption can occur by either breaking down the biofilm itself using systemic enzymes or by chelating the **iron** out of the biofilm to break it up. To chelate the **iron** you can use either calcium disodium EDTA, 4 lactoferrin, 5 or **NAC**.6

The main symptoms of an SIBO infection are indigestion, increase of flatulence, and horrible smelling deification, burps, and flatulence. Most people with IBS are walking around with SIBO, and SIBO is their main cause of their digestive problems. Other symptoms of SIBO include abdominal pains, constipation, diarrhea, food allergies, and low-grade fever. There is also a big correlation between rosacea and SIBO.

## Rosacea - SIBO

In one study, the majority of patients with rosacea were in remission or cured for at least nine months after taking Rifaximin that was used to treat their SIBO.8

## **Symptoms**

Common symptoms of SIBO are gas, bloating, diarrhea, constipation, fat in the stool, anemia, B12 deficiencies and increased intestinal permeability. Do these symptoms strike any of you IBS (or gluten) sufferers as vaguely familiar? Yeah I thought so.

SIBO is a medical condition where a person has an opportunistic bacterial infection in the small intestine.

1 This situation is usually created by both a poor American diet or a long term use of a PPI medication (Proton Pump Inhibitor). Long term use of PPI's medications causes opportunistic bacteria that would normally be eliminated by stomach acid, to survive and flourish in the small intestine. Also, the standard American diet of fermenting carbohydrates cause the opportunistic bacteria to thrive, infect the small intestine, and produce excess gas. A lack of stomach acid also causes food proteins to become partially undigested. Allergies from the undigested proteins occur, and an increase of gas and inflammation also happen.

## **Main Symptoms of SIBO**

The main symptoms of an SIBO infection are indigestion, increase of flatulence, and horrible smelling deification, burps, and flatulence. Most people with IBS are walking around with SIBO, and SIBO is their main cause of their digestive problems. Other symptoms of SIBO include abdominal pains, constipation, diarrhea, food allergies, and low-grade fever.7 There is also a big correlation between rosacea and SIBO. In one study, the majority of patients with rosacea were in remission or cured for at least nine months after taking Rifaximin that was used to treat their SIBO.8

## What causes a leaky gut

Parasites chewing through your Organs, moves Gut bacteria and yeasts around, stressing your organs, immune system, and health.

Opportunistic bacteria in the small intestine can wreak havoc with your health and lead you intestinal health into a vicious cycle of destruction. The more opportunistic bacteria in your small intestine, the more food they consume, and the more gas products and toxins they will produce. This cycle can lead to nutrient deficiencies in vitamin B12 and iron, this then leads to the patient developing anemia. The opportunistic bacteria also consume more nutrients that are now unabsorbed by the gut which leads to both an increased flora of opportunistic bacteria and increased gas production. From this blooming of opportunistic bacteria in the small intestine, you might start having abdominal bloating, pain, and excessive flatulence.10

The opportunistic bacteria then begin to decrease fat absorption in the intestines which lead to stool problems with color / fat content. The decrease in fat absorption leads to deficiencies in the fat-soluble vitamins A and D. The intestinal lining further degrades and eventually can't digest larger food particles. These larger food particles start to cause food allergies and sensitivities (gluten first, then usually followed by fructose malabsorption, then lactose digestion problems).11 The opportunistic bacteria start to enter the bloodstream from the loss of integrity in the intestinal wall. Opportunistic bacteria in the bloodstream lead to an immune overreaction that causes fatigue, systemic pain, and elevated liver enzymes. Finally, the bacteria start to excrete acids that cause neurological and cognitive problems like, brain fog and memory problems. The vicious cycle continues as the body's immune system tries to eliminate the opportunistic bacteria, which at the same time poison the body with acids, toxins, and the opportunistic bacteria continue to flourish. The vicious cycle then repeats itself in the patient, who becomes ill for a very long time.12

## Is there a yeast infection?

In some patients, opportunistic Candida overgrowth can manifest itself in the small intestine and cause similar symptoms as if the patient is infected with SIBO. I do believe that **SIYO** is rarer condition than SIBO, but systemic yeast infection of the intestines can happen in some patients. The same vicious cycle that exists with SIBO occurs in patients with **SIYO**. The yeast in **SIYO** will rob nutrients from the body, decrease vitamin production and fat absorption (by eliminating probiotic bacteria), cause food allergies and sensitivities, as well as excrete their own set of toxic byproducts (antibodies, aldehydes) that destroys the immune system and causes similar joint pain and brain fog problems.13

## Stool analysis:

SIBO has some unique tests, new technologies, and methods to identify the basis of the problem(s) Faecal calprotectin test-= Calprotectin is a 36kDa calcium and zinc binding protein expressed by the gene S100 calcium-binding protein A8, S100A8. It accounts for 30 to 40% of neutrophils' cytosol. In vitro studies show it has bacteriostatic and fungistatic properties. It is resistant to enzymatic degradation, and can be easily measured in faeces.[1] The main diseases that cause an increased excretion of faecal calprotectin are infectious colitis,[3] Crohn's disease, ulcerative colitis, and neoplasms (cancer).

## **Cytochalasin D treatment**

SIBO...

## Predominant Bacteria - GDX

Microorganisms in the GI tract perform a host of useful functions, such as fermenting unused energy substances, communicating with the immune system, preventing growth of harmful species, regulating the development of the gut, producing vitamins for the host (such as biotin and vitamin K), and producing hormones to direct the host to store fats.[1]

Intestinal microflora are also thought to have many beneficial local and systemic roles such as improving lactose tolerance, supplying short chain fatty acids (SCFA) as an energy substrate for the host, anti-tumor properties, neutralizing certain toxins, stimulating the intestinal immune system, reducing blood lipid levels and preventing obesity and type II diabetes.[2] Under normal homeostatic conditions, the intestinal microflora are of central importance in preventing colonization by pathogens, termed "colonization resistance."[3] Predominant organisms are considered to be beneficial when they are in balance.

## Low Predominant Bacteria – GDX

## Significance:

- **Dysbiosis:** Predominant bacteria should be present at normal levels in the healthy gut. Bacteroides sp. And Bifidobacter sp. should be present in the greatest amounts.[4]
- Low levels of beneficial fecal bacteria such as Bifidobactersp., Lactobacillus sp. and E. coli have been associated with irritable bowel syndrome, characterized by alternating diarrhea, cramps, and food intolerance.[5]
- Low levels of predominant bacteria increase the likelihood of acquiring opportunistic and pathogenic organisms.[3]

## **Treatment Options:**

- Probiotics
- Prebiotics such as psyllium, oat bran, oligofructose, xylooligosaccharide, inulin, beta-glucan, and/or arabinogalactan[6]
- Increase intake of fresh vegetables and fibers
- · Address other GI Effects abnormalities

# <u>High Predominant Bacteria – GDX</u>

## Significance:

- Dysbiosis: Predominant bacteria should be present at normal levels in the healthy gut. Bacteroides sp.and Bifidobacter sp. should be present in the greatest amounts[4].
- Blood infections of Mycoplasma have been linked to chronic fatigue syndrome and fibromyalgia.[7]
- Fusobacterium increases putrification in the colon.

- Overgrowth of Lactobacillus sp. could produce D-lactic aciduria in those with short bowel syndrome. Limit intake of simple carbohydrates.[8]
- Overgrowth of certain Clostridia sp. clusters may play a role in certain cases of autism.[9, 10]
- If Prevotella sp. is in the 5th quintile suspect possible oral/ throat infection.[11] Treatment Options:
- Reduce poor quality fats, refined carbohydrates and sugars, and encourage intake of fresh vegetables. High fiber foods might exacerbate patient symptoms.
- For Lactobacillus sp. or Clostridia sp. overgrowth, supplement with Bifidobacter sp. or Saccharomyces boulardii probiotics, respectively.
- May need to use anti-microbial agents
- Address other GI Effects abnormalities
- Balance flora using appropriate probiotics

## <u>Pathogenic Bacteria present – GDX</u>

## Helicobacter pylori

Helicobacter pylori (H. pylori) bacterium causes peptic ulcer disease and has been associated with increased gastric cancer risk. H. pylori is a Type I carcinogen. It is estimated that 50% of the world's population is infected with H. pylori. Symptoms:

• Acute gastritis with abdominal pain, nausea and vomiting, usually within two weeks of infection. Recurrent abdominal symptoms (non-ulcer dyspepsia) without ulcer disease are common.

## Treatment Options (Adult Dosages):

- Standard treatment for H. pylori consists of a combination of 3 or 4 drugs, antibiotics, and proton pump inhibitors, for 7-14 days. Current recommendations can be found at www.acg.gi.org. Eradication does not generally exceed 80%.
- Supplementation with lactoferrin (200 mg/d), prebiotics, and vitamin C (up to 5 grams), may improve treatment efficacy, while reducing adverse reactions.[12][13] Botanical combination treatments have also been shown to be effective in eradicating H. pylori from the GI tract.

# Parasite present - GDX

Pharmaceutical recommendations for each parasite are from the 2007 publication in The Medical Letter, "Drugs for

Parasitic Infections."[14]

## Blastocystis sp. - GDX

In the GI trac.

Blastocystis sp. is transmitted via fecal-oral route or from contaminated food or water. Seven subspecies have been identified and Blastocystis sp. 4 infection has been correlated with disease. Blastocystis sp. 2 is considered to be asymptomatic.[15-17] Symptoms:

• May include diarrhea, cramps, nausea, fever, vomiting, abdominal pain or fatigue. Blastocystis sp. has been associated with irritable bowel syndrome, infective arthritis and intestinal obstruction. In certain cases, chronic fatigue may be the only complaint.

## **Treatment Options:**

- Blastocystis sp. can be prevented by personal hygiene and sanitary conditions
- Clinical significance of infection by these organisms is controversial

- Metronidazole 750 mg PO tid x 10d or iodoquinol 650 mg PO tid x 20d or trimethoprim/sulfamethoxazole 1 DS tab PO bid x 7d have been reported to be effective
- Infection is difficult to get rid of, botanicals may not be strong enough. Use of broad spectrum antiparasitic botanicals is most effective.\*
- Botanicals\*

## Clonorchis sinensis (Chinese Liver Fluke) – GDX

### In the GI trac.

Clonorchis sinensis is found in pickled, smoked, salted, imported, or undercooked freshwater fish.

## Symptoms:

• Frequently asymptomatic. Inflammation and intermittent obstruction of the biliary ducts. Acute abdominal pain, nausea, diarrhea and eosinophilia can occur. In long-standing infections, cholangitis, cholelithiasis, pancreatitis and cholangiocarcinoma can develop.

## **Treatment Options:**

- Praziquantel, 75 mg/kg/d PO in 3 doses x 2d
- Albendazole 10 mg/kg/d PO x 7d
- Botanicals\* (see page 7)

## **Cryptosporidium – GDX**

### In the GI trac.

Water, including swimming pools, is a common source of contamination as it is resistant to chlorine. Outbreaks are associated with raw milk and meat, and Cryptosporidium is a likely cause of traveler's diarrhea.

## Symptoms:

• Watery diarrhea is the most frequent symptom, and can be accompanied by dehydration, weight loss, abdominal pain, fever, nausea and vomiting. May be very severe in immunocompromised patients.

## **Treatment Options:**

- Usually self-limiting in an immunocompetent person, with symptoms lasting 1-2 weeks
- If symptoms persist look for possible water contamination
- Nitazoxanide, 500 mg PO bid x 3d for persistent infections
- Botanicals\* (see page 7)

## Dientamoeba fragilis - GDX

## In the GI trac.

Fecal-oral transmission and water contamination are common sources. Often accompanies pinworm.

## Symptoms:

• Diarrhea, fatigue and abdominal bloating, although often asymptomatic. In chronic infections, abdominal tenderness, nausea and weight loss may be present.

### Treatment Options:

- Iodoquinol, 650 mg PO tid x 20d; Paromomycin, 25-35 mg/kg/d PO in 3 doses x 7d; Tetracycline, 500 mg PO qid
- x 10d or Metronidazole, 500-750 mg PO tid x 10d
- Botanicals\* (see page 7)

## Endolimax nana or Entamoeba hartmanni – GDX

In the GI trac.

Endolimax nana and Entamoeba hartmanni are considered to be non-pathogenic amoeba. Detection is significant in that it means the patient has ingested something contaminated with fecal material. Increased personal hygiene is recommended.

## **Entamoeba histolytica - GDX**

In the GI trac.

Entamoeba histolytica is the only amoeba considered pathogenic. Contaminated food or water, pets, sexual contact, and fecal-oral route are possible sources of transmission. Cysts are sensitive to chlorinated water.

## Symptoms:

• Range from asymptomatic to fulminating colitis (resembling ulcerative colitis), dysentery, and extraintestinal lesions on the liver, lung, brain, skin and other tissues, to sugar sensitivities, and mild parasite symptoms. Other symptoms are (no libido, impotence), brain fog, anxiety issues, depression, 'emotional blunting,' and fatigue.

## Treatment Options:

- Asymptomatic carriers should be treated in order to avoid spread
- For asymptomatic patients: Iodoquinol, 650 mg PO tid x20d; Paromomycin, 25-35 mg/kg/d PO in 3 doses x 7d or Diloxanide furoate, 500 mg PO tid x 10d
- $\bullet$  For mild to moderate intestinal disease: Metronidazole, 500-750 mg PO tid x 7-10d or Tinidazole, 2 g once PO daily x 3d followed by either Iodoquinol, 650 mg PO tid x 20d or Paromomycin, 25-35 mg/kg/d PO in 3 doses x 7d
- For severe intestinal and extraintestinal disease: Metronidazole, 750 mg PO tid x 7-10d or Tinidazole, 2 g once PO daily x 5d followed by either Iodoquinol, 650 mg PO tid x 20d or Paromomycin, 25-35 mg/kg/d PO in 3 doses x 7d
- Botanicals\*

Relief from Lactoferrin Humaworm

Alinia?

## Enterobius vermicularis (pinworm) – GDX

In the GI trac.

Enterobius vermicularis is transmitted from fecal-oral route. Females emerge from the anus and lay eggs on the perianal surface. Eggs can survive on bed linens and fabrics for 2-3 weeks.

## Symptoms:

- Nocturnal perianal pruritus which can lead to skin bacterial infection, abdominal pain and anorexia. It may enter the vagina and has been associated with some cases of cystitis.
- **Treatment Options:** 
  - Mebendazole, 100 mg PO once, repeat in 2 weeks; Pyrantel pamoate, 11 mg/kg base PO once (max. 1 g), repeat in 2wks
  - Albendazole, 400 mg PO once; repeat in 2wks
- Botanicals\*

## **Giardia lamblia – GDX, Others**

In the GI trac.

Giardia lamblia is a flagellate considered to be a pathogen and the most common cause of diarrheal disease worldwide.

Transmitted via contaminated water, food or the fecal-oral route.

## makes Hydrogen Sulfide gas ... do the test

## http://www.thebigl.co/Interpreter/V2/Content/giardiasis.htm

Background

This disorder is the result of infection with the pathogen Giardia lamblia, (lambliasis) and it is one of the most common parasitic infections worldwide. As few as 10 cysts can cause an infection.

One in five people on the planet have the illness.

AKA Giardia intestinalis, Giardia duodenalis

Beaver Feavor as it is commonly known, is and has been associated with epidemics of water-borne diarrhea, for decades. Person-to-person, Animal to person, and food-borne transmission vectors may also occur.

## **Pathogenesis**

The Protozian organism infects the small intestine.

It can cause an enlarged liver.

## **Vectors**

Outbreaks/ infection have originated from:

- · Swimming in rivers and lakes
- · Other infected people or pets
- · Contaminated municipal water supplies,
- · infected animals,
- · swimming pools,
- · restaurants,
- · nursing homes,
- · and day-care centers.

The life cycle of G. lamblia entails two stages,

- · trophozoite
- · cyst.

Infection occurs when cysts are ingested (as few as 10 cysts may initiate infection) via contaminated food or water.

- · In the stomach, the cysts rupture releasing trophozoites which then migrate into the duodenum and jejunum.
- In these portions of the small intestine, the trophozoites attach to the brush border of the intestinal epithelial cells.
- · Cysts are then produced and excreted in the feces.
- · Cellular invasion does not occur.

## **Giardiasis Symptoms**

The majority of giardia infections are asymptomatic, meaning there are no obvious signs. When symptoms are present, the most common is diarrhea, which can be acute, chronic or fluctuant.

The mushy stool because often the stool improves on its own. Because of the on-again, offagain nature of loose stools associated with giardia, many assume its a meal that didn't agree with them.

That's why so many cases of giardia go undiagnosed.

After a week, month, or sometimes years of undiagnosed giardia infection, a giardia-positive person can experience an acute and very debilitating bout of bloody, dehydrating diarrhea.

with chronic cases – lose a lot of body weight.

This is because a giardia infection interferes with digestion and inhibits absorption of nutrients from the diet. It can also damage the lining of the intestine.

In fact, this particular parasite is at the root of many cases of chronic GI inflammation cases.

I see a lot of referrals for inflammatory bowel disease (IBD). Many of these patients have a history of being giardia-positive.

I also see a number with chronic diarrhea, malabsorption and other digestive issues who end up being giardia-positive.

## **Diagnosing Giardia**

Definitive diagnosis of giardia must be done using a special fecal test. Unfortunately, this test isn't used as often as it should be. This particular parasite is microscopic.

Secondly, in 2009 I read an article suggesting in-house parasite testing – which means stool tests that are analyzed at clinics rather than being sent out to laboratories – are yielding up to 30 percent false-negative results.

I decided to test this theory, so instead of running my patients' fecal samples at my clinic, I began sending them to a local laboratory for a more comprehensive analysis. And right away the number of giardia-positive in my practice began to increase.

I now believe national labs, which use standardized equipment that returns consistently reliable results, reduce the amount of fecal in-house testing errors.

Third, some parasites, and giardia is one of them, aren't consistently shed in every stool sample. So if a cyst-free stool sample is collected for analysis, it might not show any evidence of giardia infection.

This is why I recommend any patient with a history of bowel problems be tested for giardia with an ELISA test. A fecal ELISA test is different from a fecal flotation test in that it checks for giardia antigens present in the animal's body. A fecal float test only checks for evidence

of giardia cysts in a stool sample.

If you suffer from chronic diarrhea, make sure you ask for a fecal antigen test in addition to a regular fecal float.

## **Clearing a Giardia Infection**

Unfortunately, giardia is growing resistant to many common anti-protozoal medications.

The way I determine this is with monthly fecal float tests for the first 3 to 4 months after treatment is completed, followed by an ELISA test in some cases to make sure the infection is truly cleared.

ELISA tests can show positive for giardia for up to 6 months after treatment because it can take that long for the antigens to be cleared from the bloodstream, so I don't recommend them for immediate post-treatment follow-up to insure the infection is cleared.

Following up treatment with a few fecal floats will provide the best information about whether the infection is completely resolved. Remember giardia cysts aren't passed in every single stool sample, which is why multiple fecal float tests are necessary to insure the infection is completely resolved.

Investigate further if you have:

- · diarrhea,
- · Quick weight gain
- · nausea,
- · sometimes anorexia,
- · flatulence,
- · sometimes and weight loss.
- · Malabsorption. Blocking of the intestine processing fats, carbs, proteins.
- · Patients often complain of greasy, foul-smelling stools
- · (this is secondary to hydrogen sulfide production).

## Coarse

- Symptoms develop 1 to 2 weeks after ingestion,
- · Accute
- · Systemic
- · and if effective therapy is not administered, they may persist.
- · Associated laboratory abnormalities are uncommon. (you wont see it). The CBC usually does not show leukocytosis or other abnormalities; (your blood is normal), however, when children are infected, they may manifest a mild normocytic anemia.

Fecal specimens are without leukocytes or blood. (you cant see them in stool testing)

## **Amoebiasis and Giardiasis**

The following have been found to be effective;

- · goldenseal
- · barberry (mountain grape, Pepperidge, common grape)
- · >oregon grape,

- · together with garlic and or **magnesium sulfate** for systemic infection
- · wormwood and high dose grapefruit seed extract.

#### **Blastocystis**

Consider using oregano, wormwood, Black-Walnut, cloves, quassia, and goldenseal.

#### 5.4.3 Cryptosporidiosis

consider using products containing garlic with oils of coconut, oregano, clove and cinnamon. 5.4.4 Entamoeba bistolytica and Giadia lamblia herbs

### 5.4.4.1 Household infection

- · If one has it, all may have it
- · Household contacts of infected individuals and pets should all undergo evaluation for a possible infection.

#### Treatment

Oregon graperoot and goldenseal both contains berberine, which acts against the parasites.

- · goldenseal
- · barberry (mountain grape, Pepperidge, common grape)
- · >oregon grape,
- together with garlic and or **magnesium sulfate** for systemic infection
- · wormwood and high dose grapefruit seed extract.
- · Grapefruit
- · Beet juice
- · RAW Garlic and raw horseradish root
- · Raw coconut
- · My 9yo son has had giardia for about eight months, high numbers quantitatively. I treated him with Dr. Christophers Herbal Parasite Syrup.

#### 5.4.4.2 Pharma Angle

Antiprotozoal medications, such as Ronidazole, Flagyl, Ipronidazole, Metronidozole, are used to treat Giardiosis. Dimetridazole (Emtryl) is also said to be effective.

#### 5.4.4.3 Humans

Metronidazole (Flagyl, Prostat 250 mg PO TID x 14 days) are effective first-line therapy. Cure is established in 90-95% of patients who receive therapy. If initial therapy is ineffective, then therapy with the other first-line agent should be offered. If this is also ineffective, options for cure include administering both first-line agents together at full dose for 2 weeks (14 days), or another course of therapy with metropidate

together at full dose for 2 weeks (14 days), or another course of therapy with metronidazole may be administered concomitantly with propranolol hydrochloride (Inderal 10-40 mg PO BID).

Propranolol serves to inhibit the growth and motility of G. lamblia. If symptoms still persist, a second etiology (co infection) for the patients symptoms should be suspected.

#### Symptoms:

• Often asymptomatic. Incubation period is 1-3 weeks and symptoms range from acute diarrhea, to chronic diarrhea with bloating, intestinal malabsorption, steatorrhea (possibly due to <a href="bile salt">bile salt</a> deconjugation) and weight loss. Generally self-limiting, however 30-60% develop chronic giardiasis. Unusual presentations include allergic manifestations such as

urticaria, reactive arthritis, and biliary tract disease. May induce lactose intolerance, B12 deficiency and reduced sIgA.

#### **Treatment Options:**

- Metronidazole 250 mg PO tid x 5-7d
- Avoid fatty foods as giardia feeds on <u>bile salt</u>s
- Paromomycin, 25-35 mg/kg/d PO in 3 doses x 5-10d; or Furazolidone, 100 mg PO qid x 7-10d; or Quinacrine,
- 100 mg PO tid x 5d
- Botanicals\*

### Necator americanus and - GDX

Ancylostoma duodenale (hookworm) in the GI tract

Necator americanus and Ancylostoma duodenale are transmitted via skin contact with contaminated soil, or oral ingestion of the larvae. Worms can travel to the lungs or attach to the mucosa of the GI and suck blood.

#### Symptoms:

• Itching and a rash at the site of penetration. While a light infection may cause no symptoms, heavy infection can cause anemia, abdominal pain, diarrhea, loss of appetite and weight loss. Has been associated with reactive arthritis.

#### **Treatment Options:**

- Albendazole, 400 mg PO once; Mebendazole, 100 mg PO bid x 3d or 500 mg once, or Pyrantel pamoate, 11 mg/kg (max. 1g) PO x 3d
- Botanicals\*

#### **PSORA**

Psoriasis is a defect in the immune system.

The immune system attacks itself, instead of the invader, causing a strain on white blood supply, and greatly increases the inflammation. This can cause a GI tract infection that spreads. Routine Supplementation of Immune vitamins, Rosehip seed tea, magnolia bark extract, and Chaparral will stop this genetic transcription error in the DNA. Once your immune system is regulating correctly, fighting your infection is easier. I would take up to 4 capsules of magnolia bark, 4 cups of rosehip seed tea, and 2 capsules of Chaparral, twice a day. Very Very Powerful. Also keep in mind that Stinging Nettle root robs fungus of chitin. The stuff that makes the shell of the fungus, up to 6 caps per day really weakens fungal infections.

33% of people have the defective Gene.

Psora goes back thousands of years.

Look up Psora, it is just now receiving some attention.

One of Psora body types biggest problems is in the behavior of FXR, the regulation of ornathine, and the removal of **ammonia** from the body.

#### **FXR** Farnesoid x receptor:

The bile acid receptor (BAR), also known as farnesoid X receptor (FXR) or NR1H4 (nuclear receptor subfamily 1, group H, member 4) is a nuclear receptor that is encoded by the NR1H4 gene in humans.

It plays roles in:

**FXR** is expressed at high levels in the liver and intestine. Chenodeoxycholic acid and other bile acids are natural ligands for **FXR**. Similar to other nuclear receptors, when activated, **FXR** translocates to the cell nucleus, forms a dimer (in this case a heterodimer with RXR) and binds to hormone response elements on DNA, which up- or down-regulates the expression of certain genes.[2]

One of the primary functions of **FXR** activation is the suppression of cholesterol 7 alpha-hydroxylase (CYP7A1), the rate-limiting enzyme in bile acid synthesis from cholesterol. **FXR** does not directly bind to the CYP7A1 promoter. Rather, **FXR** induces expression of small heterodimer partner (SHP), which then functions to inhibit transcription of the CYP7A1 gene. In this way a negative feedback pathway is established in which synthesis of bile acids is inhibited when cellular levels are already high. **FXR** has also been found to be important in regulation of hepatic triglyceride levels.[3] Studies have also shown the **FXR** to regulate the expression and activity of epithelial transport proteins involved in fluid homestasis in the intestine, such as the cystic fibrosis conductance transmembrane regulator (CFTR)[4]

#### **Farnesoid X receptor** has been shown to interact with:

Peroxisome proliferator-activated receptor gamma (PPAR )coactivator 1-alpha[5] and **Retinoid X receptor** alpha.[6]

Demand	l for	immune	stabili	ity is	ent	nanced	usi	ng:	

>Magnolia bark extract (Amermed)

http://amermed.com/store/index.php?route=product/product&product\_id=94&search=magnolia+bark

>Rosehip seed tea (Mountain Rose Herbs from Chile) https://www.mountainroseherbs.com/products/rosehips/profile

### ><u>Chaparral</u> Capsules from Arizona Naturals

http://www.swansonvitamins.com/arizona-natural-chaparral-500-mg-180-caps

A little vitamin **D3**.

http://www.ncbi.nlm.nih.gov/pubmed/22648540

#### Abstract

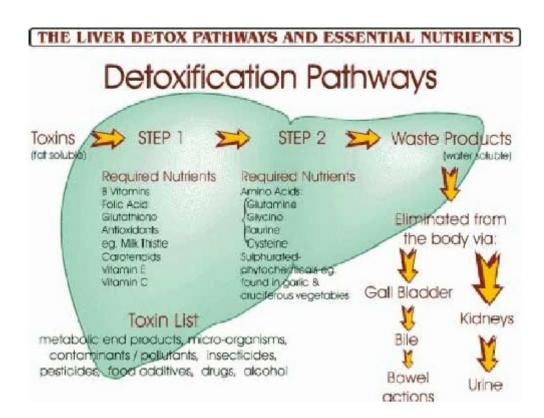
The **Farnesoid X Receptor (FXR)** is a member of the nuclear receptor superfamily of ligand-activated transcription factors, which plays crucial role in bile acid, cholesterol, lipid and glucose metabolism, as well as in the development of atherosclerosis, intestinal bacterial growth and liver regeneration. **FXR** is also involved in the pathogenesis of cholestatic diseases, non-alcoholic fatty

liver disease and inflammatory bowel disease. Recent evidence further suggests a key role for **FXR** in apoptosis and cancer. Notably, **FXR** deficiency promoted intestinal inflammation and tumorigenesis, suggesting that FXR activation might be a promising strategy in the treatment of colon cancer. **FXR** deficiency in mice led to the development of spontaneous hepatocarcinomas, while FXR inhibition might represent a novel therapeutic approach in Barett's esophagus. In **Breast Cancer** cell lines, **FXR** agonists down-regulated the **Breast Cancer** target gene aromatase. **FXR** inhibited Leydig tumor growth and progression, supporting evidence that **FXR** may be an important regulator of androgen homoeostasis. Further studies are required in order to establish possible antitumor effects of this nuclear receptor. Either reactivating or inhibiting **FXR** expression may represent promising therapeutic strategies in the treatment of certain types of human cancer.

http://www.news-medical.net/news/20150130/Farnesoid-X-receptor-could-play-key-role-in-hepatobiliary-and-gastrointestinal-disorders.aspx

The **farnesoid-X receptor (FXR)**, also known as the chief regulator of bile acid metabolism, is thought to play a role in some hepatobiliary and gastrointestinal disorders. In a study published in The American Journal of Pathology, researchers demonstrated dysfunctional intestinal FXR-signaling in a rat model of cholestatic liver injury, accompanied by intestinal bacterial translocation (BTL) and increased permeability and inflammation.

### Liver



http://thedetoxspecialist.com/blog/detox/how-liver-detox-diet-can-resore-your-health

Your liver is your best friend and superhero. Knowing how to look after it is the key to your health. Liver dysfunction plays a part in most diseases. Your liver is a multi-tasker performing over 500 functions which help keep your body in balance.

The modern stressful lifestyle, alcohol, environmental chemicals, medicinal drugs, processed foods and fats can all damage your liver. A liver detox can go a long way to help reduce symptoms of an overworked or damaged liver.

What does your liver do?

- Helps metabolize the fats, protein and carbohydrates from your diet.
- Balances blood sugar by releasing glycogen when blood sugar is low.
- Stores nutrients such as Vitamins A,D, E, K, B12 and the minerals **iron** and copper.
- Filters your blood removing harmful viruses, bacteria, yeasts, and foreign substances.
- Creates proteins needed for blood cells and the immune system.
- Breaks down and detoxifies excess and old hormones to maintain balance.
- Filters and breaks down unwanted compounds produced during metabolism.
- Detoxifies chemical toxins by converting them into substances that the body can eliminate in the bile and urine.
- Produces 1 quart of bile daily which enables you to digest fat, absorb the fat soluble vitamins and detox toxins.

Without it, cholesterol levels rise and many digestive disorders can result. Your liver relies on a number of nutrients to work efficiently. If you do not eat the right kinds of foods to provide the liver with everything it needs for the elimination of the extra toxins you are exposed to daily your health will suffer.

### Signs that your liver may be struggling:

- Yellow eyes
- White stool
- A slow digestive system
- Inability to digest fats
- Overweight
- Slow or sluggish metabolism
- Irritable bowel
- Abdominal bloating
- Food and chemical intolerance
- Feeling moody, depressed
- Foggy head
- Fatigue
- Frequent headaches
- Unstable blood sugar levels and sugar cravings
- Coated tongue and bad breath
- Immune system problems
- Dark circles under the eyes
- Premenstrual tension

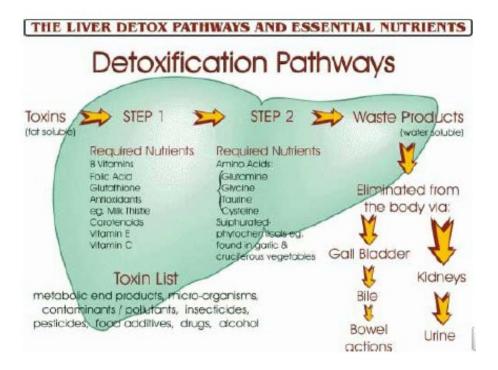
#### **Liver Tests**

Doctors often recommend a liver function blood test to see if your liver is damaged. It may show raised enzyme levels that could indicate a liver infection or alcoholic liver but it cannot tell you anything about your liver's detox function. There are however, specialized tests used by functional medicine or naturopathic doctors that can assess your liver's detox capacity. Problems in the liver detox pathways can show up long before the standard liver function blood tests show any abnormalities.

#### A two step detox system

Your liver has 2 main detox pathways or routes called Phase 1 and Phase 2. In Phase 1 a complicated process of enzymes breaks down toxins into forms that can be either be safely eliminated by your body or can go through Phase 2.

Here they are attached to other molecules that escort them out of the body through kidneys or bile.



Excessive amounts of toxic chemicals such as pesticides can disrupt the enzymes system of Phase 1 by causing over activity of it. This will result in high levels of damaging free radicals being produced which damage the liver cells.

Substances that may cause over activity during phase one are caffeine, dioxin, saturated fats, organophosphorus pesticides, paint fumes, sulfonamides, exhaust fumes and drugs. In order to get rid of or reduce these free radicals, you need foods that contain a lot of **antioxidants** and phytochemicals.

The **antioxidants** Vitamin A, C, E, selenium and many different phytochemicals are found in fresh fruits, vegetables and whole grains.

### A balancing act

There has to be a balance between the functions of Phase 1 liver detox and Phase 2 or a backlog of toxins can occur which also damage tissues. In some cases the chemical toxins modified by Phase 1 are more dangerous than the original if not dealt with by Phase 2. Researchers have discovered that your diet has a strong effect on liver detox and certain foods can help to balance Phase 1 and Phase 2.

### **How you can improve Liver Detox:**

- Eat plenty of fresh fruits and vegetables, especially cruciferous vegetables like broccoli, cabbage, Brussels sprouts kale and cauliflower. Eat a variety of orange, yellow, purple, and red colored fruits and vegetables.
- Asparagus, watermelon, broccoli are good sources of glutathione, an important substance involved
  in liver detox. Papayas and avocados help the body to produce glutathione. Other sources are
  Brussels sprouts, cauliflower, broccoli, cabbage, kale, bok choy, cress, mustard, horseradish, turnips,
  rutabagas (swede), kohlrabi, red beets.
- Include bitter foods like dandelion greens, mustard greens, bitter melon, Romaine lettuce and can help in cleansing the liver.
- Use dill, caraway seeds, tumeric, **Garlic** and onions in cooking.
- Drink at 6 8 x 8 oz glasses of filtered water a day.
- Try the <u>lemon liver detox drink</u> every morning
- Omega-3 fats are very helpful. These fats are found in cold water fish such as wild salmon, sardines, and halibut. (avoid high mercury fish) Other sources are flaxseeds/oil, hemp seeds/oil, <a href="mailto:pumpkin">pumpkin</a> seeds/oil and walnuts. Always make sure you buy very fresh nuts and seeds and store them in a cool place.
- Use cold pressed oils and do not use them for cooking. Only a little butter or coconut oil should be
  used for cooking. Cooking with olive oil is still controversial. If used then do not heat it to a high
  temperature.
- Avoid damaged fats such as hydrogenated fats found in many processed foods.
- Eat organically produced foods as much as possible to avoid toxic chemical residues.
- Avoid artificial flavorings and preservatives.
- Avoid excess saturated animal fat like fatty meat, sausages, bacon, salami, hot dogs, high fat dairy products like whole milk, ice cream and cheese.
- Alcohol is known to be a powerful toxin that will damage the liver.

Take Silymarin (<u>Milk thistle</u>) a potent antioxidant that protects the liver by increasing its ability to detoxify numerous toxic substances, including pesticides and heavy metals (lead, mercury, cadmium, arsenic). Help to regenerate liver cells.

 Take a good Multi Vitamin and Mineral supplement that contains specific nutrients and antioxidants that support liver detox. I recommend <u>Body Health Complete+ Detox.</u>

- Make sure your bowels move at least twice a day and do a colon cleanse if you have not done so already. Toxins from the bowel make the liver work harder and use up the nutrients that are needed to detox chemicals.
- Get some exercise. You liver depends on good circulation to function efficiently.

Proper functioning of the liver's detox systems is very important for the prevention of cancer. Up to 90% of all cancers are thought to be due to the effects of environmental pollutants from food, water, and air, combined with deficiencies of the nutrients the body needs for detox and immune system function. High levels of exposure to cancer causing substances together with slow detoxification enzymes significantly increases susceptibility to cancer. **Take care of your liver and it will take care of you.** 

### **Recommended Reading**

- detox diet plan
- Is The New 2 Day Diet Plan Really Healthy?
- Why You Must Avoid Toxins In The Home When You Detox

http://tuberose.com/Liver Detoxification.html

### **Liver Bile Salt**

http://www.naturalendocrinesolutions.com/archives/the-importance-of-bile-in-thyroid-health/

http://www.chamberlins.com/ns/DisplayMonograph.asp?
StoreID=2cb86c7b36be4cfd914079104818c49b&DocID=condition-nutritionaldeficiencies

#### **Bile Acid Diarrhea**

https://en.wikipedia.org/wiki/Bile acid malabsorption

Bile acid malabsorption, known also as Bile acid diarrhea, is a cause of several gut-related problems, the main one being chronic diarrhea. It has also been called Bile acid-induced diarrhea, Cholerheic or Choleretic enteropathy and Bile salt malabsorption. It can result from malabsorption secondary to gastrointestinal disease, or be a primary disorder, associated with excessive bile acid production. Treatment with bile acid sequestrants is often effective.

So...you have lost all your bile salt. You need to replenish your bile salt.

Taking some <u>Sea Salt</u> is important. No chelating Put butter on the bread

Of your liver's 500 functions; its production of bile is one of the most important. bile is an essential "de-greaser" and "emulsifier" of dietary fats. bile is also essential for the utilization of the fat soluble vitamins a, d, e and k. the bile that is produced by your liver also contains conjugated "already

used" hormones, toxins, foreign chemicals and heavy metals.

An insufficient amount of bile salts can prevent proper dietary fat utilization, cause acid indigestion as well as a backup of toxicity.

Bile insufficiency can also cause poor hormone synthesis because of the fact that all hormones are made from lipids.

### **Functions of bile**

Bile is produced by the hepatocyte cells of the liver from cholesterol. when acidified food enters into the small intestine from the stomach, bile salts alkalinize the food, preparing nutrients for assimilation in the small intestine.

Bile emulsifies fat, increasing fat absorption. Bile also contains the conjugated toxins from the 2 phases of liver detoxification. These toxins may include carcinogens, xenobiotic chemicals, pharmaceuticals and heavy metals like mercury, aluminum and lead. When hemoglobin is broken down in the liver, Bilirubin is conjugated and excreted through the bile.

#### Major problems arise when there is inadequate bile supply.

Heartburn can directly be caused if there is a bile salt insufficiency. One of the functions of the alkaline bile salts is to neutralize the food that has been acidified in the stomach by hydrochloric acid. Stomach acid that has not been neutralized is likely to cause heartburn.

A person who complains of feeling abdominal tightness, bloating and having a difficulty digesting fats may very well have a bile insufficiency.

Inadequate levels of bile can cause a build-up of toxins in the liver because of the fact that bile conjugates and carries out the body's burden of toxicity. Liver congestion can result in gall bladder stones and stagnation. If a person's cholesterol production is low, bile production is also likely to be low.

Another important component to fat digestion and utilization is hormone synthesis. All hormones are synthesized from fat. endocrine dysfunction may improve greatly with improving liver function.

### Helping the liver to detoxify & produce adequate bile

In order for the liver to function properly, adequate protein is essential. amino acids are primary constituents of phase i and ii of liver detoxification. The amino acid l-taurine is critical for bile formation, while the sulphur-bearing amino acid l-methionine is the primary methylating agent in the liver. cysteine, another sulphur-bearing amino acid is a precursor to the antioxidant glutathione, the body's most ubiquitous antioxidant. Glutathione scavenges free radicals and toxins, which end up excreted through bile.

Proper cellular hydration is also essential for liver detoxification. approximately 85% of bile is made

from water.

The nutrient betaine, also known as trimethylglycine (TMG) is one of the most powerful liver detoxificants. Betain is found in the highest concentrations in beets and beet greens.

Individuals with bile acid insufficiency can also benefit from taking bile salts derived from either ox bile or bovine bile salts.

### Bentane HCI

Premier HCL, (90 V-caps) by Premier Research Labs 021771

- O Digestive and Detox Support
- o Relieve heartburn
- o Betaine HCL without Pepsin
- o Other B-HCL suppliers add Pepsin
- o Three days until you develope acid stomach
- o More than just a digestion aid. This stuff here facilitates cell functions which can promote desirable health, both general and mental for people who have type 2 diabetes, people who have adhd, and also people such as body builders looking to put on muscle. Yes betaine, which is the same as trimethylglycine has more positive qualities to reap from than just digestion support, it's methylation support too. Ok, what methylation is basically the process of cell division where your cells actually use betaine

http://www.amazon.com/gp/product/B006OBVRMY/ref=oh\_aui\_detailpage\_o01\_s00?ie=UTF8&psc=1

#### **Identifying bile insufficiency**

On a blood test, if blood cholesterol levels are lower than 170, it is possible there is a bile acid insufficiency. increases or decreases in the liver enzymes alt (>30, <10) or ast (>30, <10), ggtp (>30) can indicate dysfunction and/or congestion in the liver.

http://metabolichealing.com/bile-deficiency-heartburn-poor-digestion-toxicity/

### Diet tips for low bile salts

- $\bullet$  begin each day with a glass of warm water with  $\frac{1}{2}$  lemon squeezed in it. Lemon juice stimulates digestive and liver function, including the gall bladder
- consume good raw oils in your diet. Foods which contain good oils are raw olive oil, fish oil capsules, flaxseed oil capsules, fish, nuts and seeds and avocados
- reduce saturated fats (animal fats and dairy foods), transfatty acids, processed foods and simple sugars. saturated fats and transfatty acids are commonly found in foods such as cakes, cookies, biscuits, bakery foods, margarine, donuts, processed and deep fried foods
- eat more bitter foods to stimulate liver and gall bladder function such as rocket, endive, raddichio and kale. also eating foods high in sulphur such as garlic, brussels sprouts, cabbage, onions,

broccoli, cauliflower and radish

- introduce herbal teas such as dandelion, burdock, peppermint, green tea, lemon and ginger to support liver detoxification, digestion and the production of bile
- increase omega 3 essential fatty acids in the form of deep sea oily fish (salmon, snapper, mackeral, anchovies, cod, sardines, halibut)
- lecithin sprinkled on your food, cereal or in a smoothie helps to emulsify fats, lipids and oils and the break down of cholesterol and bile in the digestion due to the phosphotidylcholine natural remedies for low bile salts
- Probiotics increase beneficial bacteria, as an imbalance of good vs bad bacteria in the bowels is a contributing factor to poor gall bladder function and limited synthesis of bile acids
- vitamin c and bioflavinoid help to stimulate bile movement
- liver herbs may help to encourage liver and gall bladder herbs due to their cholagogue (bile stimulant) effects such as globe artichoke, dandelion, st marys thistle, bupleurum, citrus peel, greater celandine, agrimony, golden seal, barberry, yellow dock, chamomile, ginger and turmeric
- the amino acids taurine, choline (lecithin) and methionine are beneficial for encouraging bile acid. they contain sulphur components which help to increase liver detoxification processes and stimulate the synthesis of bile and gall bladder function

#### Lifestyle factors for low bile salts

- •healthy bacteria in the digestive tract coverts bile from the gall bladder into bile acids, low gut bacteria may therefore be a contributing factor. you may consider introducing more probiotics into the diet, especially if he has had courses of anti-biotics in the past
- •low digestive ph levels
- •address poor gut bacteria
- •address any side effects of pharmaceutical medications
- address sluggish bowel function
- •it is also recommended to take a blood test to determine the functioning of the liver, as liver complications may be a related cause. elevated serum alt (liver enzymes) and ast should be tested as well as bilirubin, alkaline phosphate and ast/ alt levels
- •check gall bladder and liver function as a decline in the function of these can dramatically affect the storage of bile in the bile duct and the production of bile in the liver. Bile produced in the gall bladder ensures adequate break down of foods and also helps to lubricate the bowel

http://www.askanaturopath.com/faqs/low-bile-salts/p/712

#### Overproduction of bile acids

Primary bile acid diarrhea (Type 2 bile acid "malabsorption") may be caused by an overproduction of bile acids.[7][8] Several groups of workers have failed to show any defect in ileal bile acid absorption in these patients, and they have an enlarged bile acid pool, rather than the reduced pool expected with malabsorption.[9] The synthesis of bile acids in the liver is negatively regulated by the ileal hormone fibroblast growth factor 19 (FGF19), and lower levels of this hormone result in overproduction of bile acids, which are more than the ileum can absorb.[8]

#### Fat soluble vitamin breakdown

Bile problems will cause problems breaking down fat soluble vitamins such as vitamins A, E, K, and D. Their might be problems digesting fish oil supplements, as well as other types of fats, such as coconut oil. Hopefully you are beginning to understand how problems with bile production can lead to nutrient deficiencies. And since removing the gallbladder won't correct the bile problem, these people will still have problems breaking down fat soluble vitamins from the foods they eat.

There are three main components of bile, and this includes 1) bilirubin, 2) bile acids and salts, and 3) cholesterol. Most people reading this are familiar with cholesterol, but you might not be familiar with the other two main components of bile. Bilirubin is the toxic catabolic product of heme metabolism, and the goal of bile is to help with the excretion of bilirubin. However, certain factors can cause a disturbance of bilirubin transport, which causes increased levels of bilirubin in the blood (hyperbilirubinemia), which in turn can lead to jaundice if the levels become high enough.

### **Liver Support - The vitamin alphabet**

- . 4X Vitamin A palmitrate,
- . Vitamin A from RoseHip Seeds
- . B50
- . 4g Ester C
- . D3 5000
- . D alpha E 4000 IU
- . GLA from borage seed oil and spirulina
- . ALA
- . CQ10 in D alpha E oil
- . Chaparral by Arizona Naturals, essential for psora body types
- . Calcium Milk or Goat Cheese
- . Multivitamin per day
- . Boron
- . Use Sea Salt
- . Grape Juice
- . Eat beets
- . Taurine
- . Choline
- . NAC
- . Glycine
- . Vitamin A 10000 from cod liver oil
- . TAKE ONE CHOLINE.
- . Vitamin A PALMITRATE 15000 IU 60000 IU AM
- . COD LIVER IS GOOD IN PM

GLYCINE IS ANTI BACTERIA, TAKE A SMALL AMOUNT, OLIVE LEAF OIL IS BETTER.

Borage seed oil: Currently, there is insufficient available evidence evaluating the effectiveness of borage in the treatment of malnutrition.

- . Eat Carrots
- . Bannanas
- . Two cups of fruit and two and one-half cups of vegetables per day
- . Slow release neutral potassium phosphate
- . Saccharomyces boulardii
- . Spirulina

Choline: Choline is an essential nutrient related to the water-soluble B-complex vitamins, folate, pyridoxine, and B12, and to the essential amino acid, methionine. It is synthesized in the body as well as consumed in the diet. The largest dietary source of choline is egg yolk. Choline can also be found in high amounts in liver, peanuts, fish, milk, brewer's yeast, wheat germ, soy beans, bottle gourd fruit, fenugreek leaves, shepherd's purse herb, Brazil nuts, dandelion flowers, poppy seeds, mung and other beans, and a variety of meats and vegetables, including cabbage and cauliflower.

Choline is a major building block of lecithin. Choline is a precursor to acetylcholine, a chemical used to transfer nerve impulses. Therefore, choline is believed to have neurological effects.

Choline is a constituent of phosphatidylcholine (PC), which is a component of cell walls and membranes. It is involved in fat and cholesterol metabolism and transport. In this form, choline aids in fat metabolism and transport away from the liver.

Choline is likely effective when used orally as a nutritional supplement in infant formula. Also, choline is likely effective when used intravenously to treat total parenteral nutrition associated liver dysfunction.

Taurine: Taurine, or 2-aminoethanesulfonic acid, was originally discovered in ox (Bos taurus) bile and was named after taurus, or bull. A nonessential amino acid-like compound, taurine, is found in high abundance in the tissues of many animals, especially sea animals, and in much lower concentrations in plants, fungi, and some bacteria. As an amine, taurine is important in several metabolic processes of the body, including stabilizing cell membranes in electrically active tissues, such as the brain and heart. It also has functions in the gallbladder, eyes, and blood vessels, and it may have some antioxidant and detoxifying properties.

### Diarrhea causes a loss of liver salt that is poorly recycled

Parasites may start the process, but diarrhea will continue, and the body will go into liver crisis mode, increase the risk of developing a condition such as small intestinal bacterial overgrowth (**SIBO**) (Oregano Oil and Black seed Oil), low or absent production of gastric acid (Bentain HCL), bilirubin, which occurs when red blood cells are destroyed at to fast a rate, put **iron** into crisis, (Kelp)

B50 - The liver needs more than fat soluable vitamins, it needs B vitamins, and lots of them.

Biotin: Biotin is an essential water-soluble B vitamin. The name biotin is taken from the Greek word bios meaning "life." Without biotin, certain enzymes, including acetyl-CoA carboxylase and pyruvate carboxylase, do not work properly, and complications can occur involving the skin, intestinal tract, and nervous system. Metabolic problems including very low blood sugars between meals, high blood ammonia, or acidic blood (acidosis) can occur. Death is theoretically possible, although no clear cases have been reported. Recent studies suggest that biotin is also necessary for processes on the genetic level in cells (DNA replication and gene expression).

#### Diseases caused by bile salt regulation issues:

Achlorhydria, appetite stimulant, B12 absorption, beriberi, biotin deficiency, cachexia, copper deficiency, familial hypophosphatemia, folate deficiency, hypercalciuria, hypocalcemia, hypophosphatemia, <u>lodine</u> deficiency, <u>iron</u> absorption enhancement, <u>iron</u> deficiency, Korsakoff's

psychosis, Kwashiorkor, malnutrition, marasmus, nutritional deficiencies, nutritional support (TPN), pantothenic acid deficiency, pellagra, pyridoxine deficiency, riboflavin deficiency, rickets, scurvy, thiamin deficiency, total parenteral nutrition, TPN, vitamin A, vitamin B12, vitamin C, vitamin D, vitamin E, vitamin K, Wernicke-Korsakoff syndrome, Wernicke's encephalopathy.

http://en.wikipedia.org/wiki/Steatorrhea

# Steatorrhea

Steatorrhea (or steatorrhoea) is the presence of excess fat in feces. Stools may also float due to excess lipid, have an oily appearance and can be especially foul-smelling.[1] An oily anal leakage or some level of fecal incontinence may occur. There is increased fat excretion, which can be measured by determining the fecal fat level. The definition of how much fecal fat constitutes steatorrhea has not been standardized.

#### Cause

- . lack of bile acids
- . liver damage,
- . hypolipidemic drugs
- . gallbladder removal
- . pancreatic enzymes,
- . defective mucosal cells,
- . medicines that block fat absorption,

Consuming indigestible fat

The total absence of bile acids will cause the feces to turn gray or pale white. When this happens it is a little freaky. Parasites can so overtax the liver, it can happen. Ive experienced it

# **Hepatic encephalopathy**

Hepatic encephalopathy (brain swelling) is a worsening of brain function that occurs when the liver is no longer able to remove toxic substances in the blood.

Causes, incidence, and risk factors

Hepatic encephalopathy is caused by disorders that affect the liver. These include disorders that reduce liver function (such as <u>cirrhosis</u> or <u>hepatitis</u>) and conditions in which blood circulation does not enter the liver. The exact cause of hepatic encephalopathy is unknown.

An important job of the liver is to change toxic substances that are either made by the body or taken into the body (such as medicines) and make them harmless. However, when the liver is damaged, these "poisons" may build up in the bloodstream.

Ammonia, which is produced by the body when proteins are digested, is one of the harmful substances that is normally made harmless by the liver. Many other substances may also build up in the body if the liver is not working well. They can cause damage to the nervous system.

Hepatic encephalopathy may occur suddenly in people who previously had no liver problems when damage occurs to the liver. More often, the condition is seen in people with chronic liver disease.

Hepatic encephalopathy may be triggered by: (Parasite Toxins!)

- Dehydration
- Eating too much protein
- <u>Electrolyte</u> abnormalities (especially a decrease in potassium) from vomiting, or from treatments such as <u>paracentesis</u> or taking diuretics ("water pills")
- Bleeding from the intestines, stomach, or esophagus
- Infections
- Kidney problems
- Low oxygen levels in the body
- Shunt placement or complications (See: <u>Transjugular intrahepatic portosystemic shunt</u>)
- Surgery
- Use of medications that suppress the central nervous system (such as barbiturates or benzodiazepine tranquilizers)
- Disorders that can mimic or mask symptoms of hepatic encephalopathy include:
- Alcohol intoxication
- Complicated alcohol withdrawal
- Meningitis
- Metabolic abnormalities such as <u>low blood glucose</u>

Hepatic encephalopathy (also known as portosystemic encephalopathy) is the occurrence of confusion, altered level of consciousness, and coma as a result of liver failure. In the advanced stages it is called hepatic coma or coma hepaticum. It may ultimately lead to death. It is caused by accumulation in the bloodstream of toxic substances that are normally removed by the liver. The diagnosis of hepatic encephalopathy requires the presence of impaired liver function and the exclusion of an alternative explanation for the symptoms. Blood tests (ammonia levels) may assist in the diagnosis. Attacks are often precipitated by an intercurrent problem, such as infection or constipation. [1][2]

Hepatic encephalopathy is reversible with treatment. This relies on suppressing the production of the toxic substances in the <u>intestine</u> and is most commonly done with the laxative <u>lactulose</u> or with non-absorbable <u>antibiotics</u>. In addition, the treatment of any underlying condition may improve the symptoms. In particular settings, such as <u>acute liver failure</u>, the onset of encephalopathy may indicate the need for a <u>liver transplant</u>.[1][3]

Despite numerous studies demonstrating the central role of **ammonia**. **Ammonia** levels don't always correlate with the severity of the encephalopathy; it is suspected that this means that more **ammonia** has already been absorbed into the brain in those with severe symptoms whose serum levels are relatively low.[1][2] Other waste products implicated in hepatic encephalopathy include **mercaptans** (substances containing a thiol group), **short-chain fatty acids** and **phenol**.<sup>[2]</sup>

Numerous other abnormalities have been described in hepatic encephalopathy, although their relative contribution to the disease state is uncertain. Benzodiazepine-like compounds have been detected at increased levels as well as abnormalities in the GABA neurotransmission system. An imbalance between aromatic amino acids (phenylalanine, tryptophan and tyrosine) and branchedchain amino acids (leucine, isoleucine and valine) has been described; this would lead to the

generation of false <u>neurotransmitters</u> (such <u>octopamine</u> and <u>2-hydroxyphenethylamine</u>). Dysregulation of the <u>serotonin</u> system, too, has been reported. Depletion of <u>zinc</u> and accumulation of **manganese** may play a role.[1][2] Inflammation elsewhere in the body may precipitate encephalopathy through the action of <u>cytokines</u> and bacterial <u>lipopolysaccharide</u> on astrocytes.<sup>[4]</sup>

Lactulose and lactitol are disaccharides that are not absorbed from the digestive tract. They are thought to improve the generation of **ammonia** by bacteria, render the **ammonia** inabsorbable by converting it to ammonium (NH<sub>4</sub>), and increase transit of bowel content through the gut. Doses of 15-30 ml are administered three times a day; the result is aimed to be 3–5 soft stools a day, or (in some settings) a stool pH of <6.0.[1][2][4][10] Lactulose may also be given by enema, especially if encephalopathy is severe. [10] More commonly, phosphate enemas are used. This may relieve constipation, one of the causes of encephalopathy, and increase bowel transit. [1]

A preparation of <u>L-ornithine</u> and <u>L-aspartate</u> (LOLA) is used to increase the generation of <u>urea</u> through the <u>urea cycle</u>, a <u>metabolic pathway</u> that removes <u>ammonia</u> by turning it into the neutral substance <u>urea</u>. It may be combined with lactulose and/or rifaximin if these alone are ineffective at controlling symptoms. [1]

### **Dr Budwig**

http://www.healingcancernaturally.com/budwig\_protocol.html

Cancer: Essentially A Problem of Right and Wrong Fats & Lack of Sunlight?

### An important introduction to Dr. Budwig's protocol

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"What she (Dr. Johanna Budwig) has demonstrated to my initial disbelief but lately, to my complete satisfaction in my practice is: CANCER IS EASILY CURABLE, the treatment is dietary/lifestyle, the response is immediate; the cancer cell is weak and vulnerable; the precise biochemical breakdown point was identified by her in 1951 and is specifically correctable, in vitro (test tube) as well as in vivo (real)."

Dr. Dan C. Roehm M.D. FACP (oncologist & former cardiologist) in "Townsend Letter for Doctors", July 1990

What would you think if you heard someone claim that there is a diet-based protocol centered on the ingestion of two simple foodstuffs plus sunlight that has actually helped heal numerous cases of cancer including saving the so-called terminally ill - some given a few hours to live!? If you react like many, you will think that this sounds far too simplistic to be true.

And yet it is.

As Dr Willner, M.D., Ph.D. (The Cancer Solution) writes, "Numerous, independent clinical studies published in major medical journals world-wide confirm Dr. Budwig's findings.... Over 40 years ago Dr Budwig presented clear and convincing evidence, which has been confirmed by hundreds of other related scientific research papers since, that the essential fatty acids were at the core of the answer to the cancer problem...You will come to your own conclusions as to why this simple effective prevention and therapy has not only been ignored—it has been suppressed!". Or as someone

commented, "Over the years I have been given a lot of different advice as well, so when I heard about Dr Budwig's protocol I too was very sceptical, until I tried it."

Before you proceed to reading any further, please know that not everybody reacts the same to this or any other treatment, and that in any healing diet or protocol one may undertake, one should never underrate the often amazing power of the <a href="Mind">Mind</a>, i.e. the placebo or belief effect, as well as the influence of the <a href="emotions">emotions</a> and <a href="emotions">energetic</a> and <a href="emotions">spiritual factors</a> on both the disease and healing process.

So here are five important pieces of advice before we start:

1. If you do decide to follow the Budwig diet & protocol, give it a fair try, meaning that you follow Dr. Budwig's instructions to the letter for a while such as at least 1 to 3 months, and then see how you or the patient fares. Dr. Budwig's instructions are very strict and include no supplements apart from brewer's yeast flakes (and possibly plant-derived enzymes for fat/protein digestion, with herbs in their natural form also being ok), daily fresh ground flaxseeds etc. etc. You can read up on all the details on this site and her genuine books.

Apart from the possible placebo effect she exerted (as any healing professional who instills confidence in his patients), Dr. Budwig heavily insisted on her instructions being closely followed since she believed that only in this manner, success could be reached.

Don't get misled by self-proclaimed "Budwig diet experts" or a self-styled "Budwig center" who combine her basic flax oil/cottage cheese with a regime of their own making rather than with Dr. Budwig's real protocol, claiming to thus "reinforce" the Budwig diet. While such regimes may well be effective against cancer and other diseases, they are a far cry from what Dr. Budwig recommended (for more details and warnings see Oil-protein diet: on self-appointed "Budwig experts" and fake Budwig books misleading cancer patients). Remember that while it is true that much of the conventional cancer treatment approaches is about business (compare On Cancer Business & the Cancer Industry), so is much in the "alternative cancer treatment" field (compare Negative experiences in the alternative cancer treatment field).

- 2. Healing Cancer Naturally considers minerals and trace elements and the ubiquitous lack of them a major player in cancer genesis and healing, see Minerals. The fact that so relatively few of those who undertake the Budwig protocol today seem to report dramatic success when compared to the near 100% success rate claimed by Dr. Budwig who started treating patients decades ago, might (apart from non-compliance with her instructions) also be related to the reported significant decline in mineral and trace element (as well as vitamin) content in today's foods, caused by soil depletion and inorganic husbandry. So an important piece of advice would be to look for and incorporate naturally rich sources of mineral and trace elements in your diet. More inexpensive sources are, apart from organically grown foodstuffs, particularly seaweeds which I highly recommend adding. Seaweeds among other things contain high levels of iodine which reportedly has anticarcinogenic (toxic to cancer cells) and cancer-preventative properties (more at seaweed).
- 3. Detoxify in a healthy way (i.e. don't become obsessed with any uncleanliness or toxicity you may suspect in yourself while still striving to get rid of most obvious sources of "filth" in yourself and your environment). See <u>Detoxification</u>.
- 4. The Budwig diet already prescribes the daily ingestion of quark (cottage cheese) and sauerkraut juice both of which are lactic-acid fermented foods. Judging by another successful cancer researcher's results with cancer patients, the little-known but truly outstanding Dr. Dr. Johannes Kuhl who based his approach primarily on lactic-acid fermented foods and supplements (see <u>Lactic-acid-fermented food treatment for cancer according to Dr. Dr. Johannes Kuhl</u>), this part of the Budwig protocol may be one of the decisive pillars and reasons for its effects. So Healing Cancer Naturally advises the liberal addition of lactic-acid-fermented foods and drinks in the diet even

beyond what Dr. Budwig may have explicitly recommended to further enhance your chances of healing.

Incidentally, both Dr. Budwig's and Dr. Kuhl's approach are featured under <u>Greatest Hits</u>, and virtually all modalities listed there are fully compatible with Dr. Budwig's protocol while having obtained cancer remissions as a stand-alone treatment as well.

5. Lastly, but in fact most importantly, Healing Cancer Naturally advises following one's intuition, spiritual guidance or whatever name you may wish to call it as you travel along your journey to health and healing. A beautiful example has been set by Dr. Nancy Offenhauser who wrote a book about her experience called <a href="Healing Cancer Peacefully">Healing Cancer Peacefully</a> (Dr. Offenhauser did not use the Budwig approach - as you may or not be aware, there are numerous non-conventional ways which have been used to heal from cancer).

### Who Was Dr. Johanna Budwig?

Dr. Johanna Budwig (born 30 September 1908, died 19 May 2003, pronounced Yaw-hun-nah Boodvig), a seven-times Nobel prize nominee, was a qualified German pharmacologist, chemist and physicist with a doctorate in physics who worked as the chief expert-consultant for drugs and fats at the former Bundesanstalt für Fettforschung (Federal Institute for Fats Research). Described as "the world's leading authority on fats and oils", Dr. Budwig studied in-depth the effect of hydrogenated and other denatured fats upon human health and found it to be desastrous, while she discovered the truly "essential" and powerfully healing nature of essential fatty acids on all manner of degenerative diseases including cancer. She authored numerous books (among them Cancer - The Problem and The Solution, Das Fettsyndrom [The Fat Syndrome, discussing the links between fats and next to all diseases of the heart and lung as well as cancer], Krebs, ein Fettproblem [Cancer - A Fat Problem, on the right choice and use of fats], Der Tod des Tumors [The Death of the Tumor], as well as numerous scientific papers and treatises in which she published her findings on the critical importance of the right fatty acids and the deleterious effect of the wrong fats on human health (the use of "wrong fats" having become widespread since the invention and ubiquitous introduction of the hydrogenation process in order to extend the shelf life of fats and to create margarine).

Three of her works "FlaxOil As A True Aid Against Arthritis, Heart Infarction, Cancer, And Other Diseases" (read excerpts here), "The Oil Protein Diet Cookbook" and the above-mentioned Cancer - The Problem and The Solution (excerpts here) have been translated into English. The former, though a slender volume of only about 60 pages, is "packed with the most clear thinking on the genesis of serious diseases and health recovery" including healing cancer. The fact that so far solely three of Dr. Budwig's works are accessible to the English-speaking world makes it hard, however, to appreciate the true scope and breadth of her protocol and cancer treatment approach. While the situation is naturally better for German readers, even these have easy access only to parts of her work since a number of her books - in fact, the majority - are currently out of print and much determination is called for to find a copy of these books in second-hand stores.

As other researchers who have offered a non-profitable and effective way of healing cancer (or keeping it under control by preventing metastasis), Dr. Budwig and her work have been attacked and silenced by vested medical, industrial and pharmaceutical interests throughout her life (compare <a href="History of Cancer Treatment">History of Cancer Treatment</a>). As summarized by Cliff Beckwith who has kept his prostate cancer under control for over a decade: "Dr. Budwig to my knowledge had over 1000 documented successes. However, her work was not popular with the Oncology Industry in Europe. Her ideas would have meant a lot of losses in the Food Industry [too]; especially in the fats industry. My cousin, Richard Beckwith, called her probably eleven years ago and talked to her about forty five minutes. She told him that American doctors had come to Germany and been impressed with her work. Then they wanted to try to work out some way to have exclusive rights to her methods in the United States and make a lot of money and she wouldn't do that.

She believed her work was very important and was anxious to see it carried on but no one seemed interested unless they could make a lot of money.

I had Advanced [prostate] cancer and I could not wait for scientific confirmation and began to use these ideas immediately. One thing led to another, none of it planned, and we have seen many folks recover from cancer or have lives greatly extended. A lot of that information is at www.beckwithfamily.com.

That is all based on anecdotal evidence. Other than the evidence in Dr. Budwig's records [as far as they are available in English] that is all there is. No one is doing any scientific testing. That is done to prove the value of <u>drugs for profit</u> and one cannot patent Flaxseed Oil.

...In my view there should be enough on those lists [the evidence reported on two Budwig discussion lists he founded] and what we have seen to at least cause someone really interested in human welfare to be curious."

And it seems likely that apart from financial interests, human failings such as intellectual pride play a part in the silencing and ignoring of Dr. Budwig's apparently often life-saving discoveries. Cliff Beckwith again: "For the most part one cannot expect the doctors to place any real credibility to the use of FO/CC [flax oil and cottage cheese]. It seems incredible that anything could be successful other than what they are taught in Med school. Imagine the blow to one's ego if it became official that something with which he or she is not familiar would be found to be much better than the things that were studied 10 years to learn."

### Dr. Johanna Budwig's Major Discovery

In 1952, Dr. Budwig wrote in a paper entitled On Fat Biology V. Paper Chromatography of Blood Lipoids, the Tumour Problem and Fat Research: "It is basically proven that highly unsaturated fatty acids are the heretofore undiscovered decisive factor in respiratory enzyme function", i.e. constitute the second part of the "equation" that nobelist Otto Warburg [1] had been unable to find. What sounds insignificant to the layman's ears, is arguably one of the greatest breakthroughs in medicinal science: from that moment onward we have known that the highly unsaturated fatty acid is the decisive factor achieving the desired effect of cellular respiratory stimulation. Working in conjunction with sulfurated amino acids (protein), the highly unsaturated fatty acid plays a part, even the critical part, in the "bridging" taking place between fats and protein, in the absorption AND utilization of oxygen, in all growth processes, in the formation of blood and in many other processes. Working from this theory, Dr. Budwig was able to help a great many cancer patients with the scientific oilprotein diet of flaxoil plus cottage cheese she designed (the "Budwig diet"), which allows cancer cells to start "breathing" again. A few physicians followed in her footsteps, such as Dr. Dan C. Roehm from Florida or Dr. Robert E. Willner (Miami).

Unsolicited visitor's comment on Healing Cancer Naturally's Budwig Diet pages:

"I have been educating myself on the Budwig protocol and your site is by far the most informative." ~S. G.

Based upon her research findings, Dr. Budwig was not only against processed foods and supplements (no pills) but also against chemotherapy, radiation and drugs, and in a less categorical manner, surgery (see interview). And, rare as that may be, she also was aware of the critical importance of sunlight as well as the spiritual, mental and emotional factors in healing cancer and other illness. That said and as hinted at above, it is important to keep in mind that the fuller details of her published thought on healing cancer are not currently known/accessible in the English- and most of the German-speaking world, thus making all efforts presently undertaken at implementing and spreading the word about Dr. Budwig's discoveries in English a grassroots movement liable to be enlarged as more details become known to the general public via correctly translated editions of more of her books.

Basic Introduction to Dr. Budwig's Diet, Fats, Essential Fatty Acids and Related Subjects

The basis of Dr. Budwig's diet or protocol is the ingestion of a special oil-protein mixture in the form of organic cold-pressed flaxseed oil plus cottage cheese or "quark" (a dairy product readily available in German-speaking countries made from various types of milk and roughly similar to cottage cheese), to balance an oversupply of omega-6 fatty acids and hydrogenated fats in the Western diet and to provide an immediately available abundant supply of essential omega-3 fatty acids. Of all plant oils, flax oil is the richest source of these omega-3 fatty acids (naturally occurring variations not considered, 100 g of oil contain 72g of polyunsaturated fatty acids, 54g of which are omega-3 acids). This oil is combined with protein (or more precisely, sulphurated amino acids[2] such as liberally found in guark/cottage cheese) to allow the highly unsaturated fatty acids to become watersoluble, thus bypassing the need for an (often) diseased or impaired liver to break down the unsatured fat by its own efforts. Quote: "The lipotropic protein connections, e.g. Cystein, as they are found in ... cottage cheese or nuts are able to make water-soluble the ...highly unsaturated fatty acids in seed oils. And that is what matters. When you mix together ... cottage cheese and linseed oil in your blender the fat becomes water-soluble" and thereby immediately available for use by the body. In this manner, the necessary "spark plugs" are provided for cells to "breathe", optimally detoxify and function, even more so when additionally combining the flax oil cottage cheese mix with an optimised sugar-free diet devoid of respiratory poisons [substances which inhibit cellular respiration] but containing much raw organic food (compare excerpts from Dr. Budwig's Flax Oil As A True Aid and the Nutrition section).

Dr. Budwig's diet (which, when properly applied, is an entire protocol and involves not only ingestion of the above oil-protein mixture, but also a healthy minimally processed <u>vegetarian</u> diet, freshly ground flaxseeds, <u>sunlight</u>, <u>stress management</u>, <u>"Eldi" oils</u>, etc. [3], has literally pulled people back from death's doorstep. Based on this evidence and its ease of implementation, it may be the quickest and easiest move to take for many stricken with a cancer challenge and/or those who are looking for an often fast-working approach to health recovery. In fact, eminent <u>alternative & conventional cancer treatment researcher Lothar Hirneise</u> considers Dr. Johanna Budwig's protocol the indispensable nutritional basis of any healing plan for cancer patients.

Dr. Johanna Budwig's proposed diet and basic protocol IS easy to implement in daily life (see <u>Dr. Johanna Budwig: The practical implementation of my oil-protein diet</u>), and for "how-to" details on the Budwig diet, start with <u>Making the Flaxseed Oil plus Cottage Cheese or Quark Mixture. Budwig Linomel Breakfast Muesli Recipe</u>, <u>Oleolux Recipe</u>, <u>Quark-Flax Oil Mayonnaise Recipe</u> as well as the very extensive <u>Budwig Diet & Protocol FAQ</u> I have written and compiled from authentic Budwig sources including the 14 original German-language books by Dr. Budwig I own).

I'd also like to refer you to Dr. Johanna Budwig's available English-language books, with some people expressing particular appreciation for her oil-protein cookbook. One comment reads: "I have found that one of THE MOST VALUABLE, yea NECESSARY items one should get for following the Budwig diet is Dr. Budwig's 'Oil Protein Cookbook.' It demystifies all kinds of things for you, and has OVER 500 recipes in it to make sure [you] will be getting all the things Dr. Budwig wanted her clients to get in their diet." I too believe it is essential for anyone "serious" about implementing the Budwig diet with the aim of healing from serious disease such as cancer to at the very least own Dr. Budwig's inexpensive oil-protein cookbook. It would seem best to also read her book "Flax Oil as a True Aid" and to make sure to share these books with others, both to possibly help them prevent and heal from serious disease. More comments.

#### **Research Studies On Animals**

Effects of Flaxseed and Flaxseed Components (Lignan, Lignan Precursors & Oil) on Cancer and Tumor Growth

Please note Healing Cancer Naturally's stance on animal experimentation.

1 A 2002 animal experiment on the "[e]ffect of flaxseed supplementation on prostatic carcinoma in transgenic mice" found that a "diet supplemented with 5% flaxseed inhibits the growth and development of prostate cancer in the TRAMP model".

Aiming to "investigate the effects of flaxseed supplementation on prostatic neoplasia in the transgenic adenocarcinoma mouse prostate (TRAMP) model", a "total of 135 male TRAMP mice 5 to 6 weeks old were randomized to a control group (AIN-76A diet) or an experimental group (AIN-76A diet plus 5% flaxseed by weight). One half of the mice in each group were treated for 20 weeks and the remainder for 30 weeks. At autopsy, urogenital tissues (four prostatic lobes, seminal vesicles, and emptied bladder), lungs, lymph nodes, and grossly abnormal tissues were collected for histologic evaluation. RESULTS: Of the control mice, 100% developed prostate cancer versus 97% of the mice in the flaxseed group. The tumor/urogenital weight was 3.6 +/- 0.4 g in the controls versus  $1.9 + /- 0.2 \,\mathrm{g}$  in the flaxseed-treated mice (P = 0.0005). At 20 weeks, no significant difference in tumor grade was seen between the two groups; however, at 30 weeks, the flaxseed-treated mice had significantly less aggressive tumors than did the controls (P = 0.01). The prevalence of lung and lymph node metastases was 13% and 16%, respectively, in the control mice versus 5% and 12%, respectively, in the experimental group (difference not significant). After 20 weeks of treatment, cellular proliferation (Ki-67) differed significantly between the control and experimental groups (38.1 +/- 2.03 versus 26.2 +/- 2.03; P <0.0001), and the apoptotic index (deoxynucleotidyl transferasemediated dUTP-digoxigenin nick end labeling) was 1.45 +/- 0.14 versus 3.3 +/- 0.31 (P <0.0001). Similar differences were seen after 30 weeks of treatment.

The original NCBI report with references

2 A 2002 animal experiment on "the effect of flaxseed (FS), the richest source of lignans and alphalinolenic acid, on growth and metastasis of established human breast cancer in a nude mice model" concluded that "Dietary flaxseed inhibits human breast cancer growth and metastasis".

Dietary flaxseed inhibits human breast cancer growth and metastasis and downregulates expression of insulin-like growth factor and epidermal growth factor receptor.

Chen J, Stavro PM, Thompson LU. Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Toronto, ON, Canada M5S 3E2.

Recent studies indicate that diets rich in phytoestrogens and n-3 fatty acid have anticancer potential. This study determined the effect of flaxseed (FS), the richest source of lignans and alpha-linolenic acid, on growth and metastasis of established human breast cancer in a nude mice model. Estrogen receptor-negative human breast cancer cells, MDA-MB-435, were injected into the mammary fat pad of mice (Ncr nu/nu) fed a basal diet (BD). At Week 8, mice were randomized into two diet groups, such that the groups had similar tumor size and body weight. One continued on the BD, while the other was changed to BD supplemented with 10% FS, until sacrifice at Week 15. A significant reduction (P < 0.05) in tumor growth rate and a 45% reduction (P = 0.08) in total incidence of metastasis were observed in the FS group. Lung metastasis incidence was 55.6% in the BD group and 22.2% in the FS group, while the lymph node metastasis incidence was 88.9% in the BD group and 33.3% in the FS group (P < 0.05). Mean tumor number (tumor load) of total and lymph node metastasis was significantly lower in the FS than in the BD group (P < 0.05). Metastatic lung tumor number was reduced by 82%, and a significantly lower tumor trend (P < 0.05) was observed in the FS group. Lung weight, which also reflects metastatic tumor load, in the FS group was reduced by 20% (P < 0.05) compared with the BD group.

Immunohistochemical study showed that Ki-67 labeling index and expression of insulin-like growth factor I and epithelial growth factor receptor in the primary tumor were lower in the FS (P < 0.05) than in the BD group.

In conclusion, flaxseed inhibited the established human breast cancer growth and metastasis in a nude mice model, and this effect is partly due to its downregulation of insulin-like growth factor I and epidermal growth factor receptor expression.

The original NCBI report with references

More research studies incl. on humans.

### Why Use Organic Dairy in the Budwig Diet?

#### 1. Antibiotics

Conventional milk (and meat) products are very likely to contain antibiotics which harm and destroy the good (beneficial) intestinal flora. This apparently both impedes proper digestion/optimal mineral nutrient assimilation and immune system performance and contributes to the currently observed Candidiasis epidemic (Candidiasis being defined as "overgrowth in the gastrointestinal tract of the usually benign yeast [or fungus] Candida albicans"). Candida fungus overgrowth has become widespread apparently due to indiscriminate antibiotics use in the food chain.

Compare On food and supplement absorption, intestinal flora, cancer and immune system and Fungi producing mycotoxins: The Fungal/Mycotoxin Etiology of Human Disease (particularly CANCER).

### 2. Genetically engineered bovine growth hormone (Posilac)

Conventional dairy products in the US may or do contain bovine growth hormone. See for instance rense.com/general48/milk.htm ("cows treated with Posilac have been developing bone cancers").

Also compare Milk and the Cancer Connection I, Milk and the cancer connection II and Cancer Causes: Aflatoxins.

#### 3. Ethics

See Vegan Alternatives.

### **Budwig Testimonials**

See the Complete list of Johanna Budwig diet & protocol cancer healing testimonials.

# Support Groups, Flaxseed Oil/Cottage Cheese Forums, Budwig Forums & Other

Introductory note: As I know from owning 14 of Dr. Budwig's original German-language writings, Dr. Budwig's work contains many details and scientific intricacies. Browsing the internet for websites offering free Budwig information, alternative cancer treatment advisors who sell such information as well as for forums and groups discussing the Budwig diet/protocol made me keenly aware that there is a certain amount of (doubtless largely well-intentioned but) partially incorrect information on the Budwig Protocol offered to the English-speaking public. To help dispel the misinformation / misinterpretation / misrepresentation shrouding the genuine Budwig Protocol and to implement and spread the word about Dr. Budwig's authentic discoveries and "teachings", I have published the authentic central details (a number of which translated by me) gleaned from Dr. Budwig's original works as well as from my contacts with Budwig patients, Budwig cancer carers and Dr. Budwig's former associate, using the platform of Healing Cancer Naturally's Budwig pages.

It is to be hoped that eventually correctly translated editions of more of her <u>books</u> will allow all parts of Dr. Budwig's work and the full details of her thinking and experience with the healing of cancer to become known and accessible in the English-speaking world.

The following non-exhaustive list shows some "grassroots" forums discussing the use of Dr. Johanna Budwig's oil-protein protocol (or parts or "free-style versions" of it) to treat and control cancer as well as other diseases.

http://curezone.com/forums/f.asp?f=55

flaxseed Oil & Virgin Flax Oil Cottage cheese Forum - Dr. Budwig

http://groups.yahoo.com/group/flaxseedoil/

This flax oil/cottage cheese group is inactive but carries several thousand messages in its archives which are freely accessible without needing to subscribe.

http://groups.yahoo.com/group/flaxseedoil2/

Budwig approach for cancer healing discussion group (moderated)

http://health.groups.yahoo.com/group/flaxhealth/

"This group focus is to spread the good word about the postive benefits of utilizing flax in your diet" (The complete Budwig protocol for healing cancer and other diseases calls for the daily addition of freshly ground flaxseeds.)

### Flaxseed Oil Sources: Buying Quality Flax Oil

There are several companies and outlets which offer quality flax oil. Barlean's is known to offer a discount to (verified) cancer patients but you may also find bargains on quality flaxoil elsewhere including at Amazon. I'd make sure to go for **organic, fresh, cold-pressed and unadulterated flax oil** (no Vitamin E or other preservatives added) and read the customer reviews before deciding on any one brand. Also be aware that Dr. Budwig did not use <u>flaxseed oil with lignans</u> (in fact, lignan-"enriched" flax oil to this day is not even available in Germany). This factor may however be of little consequence.

To find flax oil on Amazon, you can enter Amazon via one of these links, then type "flax oil" or "flaxseed oil" into their search box:

- US visitors
- Canadian visitors
- UK visitors
- Amazon France
- Amazon Deutschland
- Amazon España
- Amazon Italia

By shopping (any items) via Healing Cancer Naturally's above Amazon partner links you help support its humanitarian work at no extra cost to you, for details see <u>Support this site</u>.

### Dr. Johanna Budwig's Works in English

Warning: since Dr. Budwig's death in 2003, books/CDs have been published which give misleading information on Dr. Budwig's approach, details under <u>Oil-Protein Diet: on self-appointed "Budwig experts" and fake Budwig books misleading cancer patients.</u>



# FlaxOil As A True Aid Against Arthritis, Heart Infarction, Cancer, And Other Diseases

by Dr. Johanna Budwig

"Every home library needs to have a copy of this book."

At the time of most recent publishing of this page, Amazon.com had the following offer: List Price: \$6.95, used & new from \$4.45

More Info (US visitors)

More Info (Canadian visitors)

More Info (UK visitors)

Read excerpts from FlaxOil As A True Aid.



#### **The Oil-Protein Diet Cookbook**

by Dr. Johanna Budwig

How to use flax, even including ice-cream! "[I]ndispensible to someone serious about closely following the Budwig Protocol." "I have found that

one of THE MOST VALUABLE, yea NECESSARY items one should get for following the Budwig diet is Dr. Budwig's 'Oil Protein Cookbook.' It demystifies all kinds of things for you, and has OVER 500 recipes in it to make sure [you] will oil plus cottage cheese in many varied tasty vegetarian dishesbe getting all the things Dr. Budwig wanted her clients to get in their diet." "I recently received my copy of the Oil Protein Diet Cookbook by Dr. Budwig. I am so glad I ordered it! It is much more than a cook book, it helped to really understand the use of flax oil and seeds, and how to portion our food intake throughout the day. The suggested ways of preparing food also were wonderful, and allow for using your imagination in ways to prepare the food to be tasty as well as healthful. This book is way [more] than a cook book....should be entitled more like, "practical applications of the oil protein diet in your everyday life". (Users' comments)

Read excerpts from her book FlaxOil As A True Aid.

More info (US visitors)

More info (Canadian visitors)

More info (UK visitors)



#### **Cancer - The Problem and The Solution**

by Dr Johanna Budwig

This is the last book Dr. Budwig published. You can read here an <u>Extract from Cancer - The Problem and The Solution</u>.

More Info (US visitors)
More Info (Canadian visitors)
More Info (UK visitors)

(The following title is not written by Dr. Budwig but on the subject of her healing diet & protocol for cancer and other disease:)



### **How to Fight Cancer & Win**

by William L. Fischer

William L. Fischer has been involved in medicine, health care, and natural healing for over 30 years. After working with several of the largest

pharmaceutical manufacturers in his native Germany, he moved to the United States and began publishing books on natural healing. For him to obtain the most comprehensive information available, his research has taken him around the world to such diverse places as Iran, the Far East, Europe, and Egypt to study natural healing techniques... Fischer, too, arrived at the conclusion that Dr. Johanna Budwig's easy-to-implement protocol is the number one choice in healing cancer... Read excerpts from Dr. Budwig's book FlaxOil As A True Aid.

More Info (US visitors)
More Info (Canadian visitors)

More Info (UK visitors)

#### **Cancer Formula**

I have attached the most powerful anticancer substances I know of. In high dose, with my lungs filling up with fluid, arms and legs a sleep with no circulation, and leukemia, rosehips and magnolia brought me back. Their effect on the immune system is unbelievable, RXR, RXD, and Chaparral FXR, forces the immune system back to action.

selenium can be taken in very high dose, almost 1200 ucg/day, Offset ROS loop stress, delivers O2 to cells, WBC production, 450CYP ROS Consumable under Ox stress

Selenium (2-4-8) CAP. 200 mcg 120ct NOW P#01486, Swanson SWU171

https://www.swansonvitamins.com/swanson-ultra-semsc-selenium-200-mcg-120-caps Selenium 200 mcg @ L-Selenomethionine

selenium deficiency that results in an increase in expression of adhesion molecules, which causes greater adhesion of neutrophils.[viii]

Immune system booster, 450CYP ROS Consumable under Ox stress, CYP450 deficiency leak, Beneficial in the prevention of several types of infection, Selenium, which acts both as a free radical scavenger, and as a mineral that helps prevent cells from turning cancerous, boosts white blood cell production.

Selenium influences both the innate, "nonadaptive" and the acquired, "adaptive" immune systems[iii]-[iv]-[vi]-[vii] The innate immune system includes barriers to infection and nonspecific effector cells such as macrophages. Both the T and B-lymphocytes form the major effector cells of the acquired system that mature with exposure to immune challenges. Selenium-deficient lymphocytes are less able to proliferate in response to mitogen, and in macrophages, leukotriene B4 synthesis, which is essential for neutrophil chemotaxis, is impaired by this deficiency. These processes can be improved by selenium supplementation. The humoral system is also affected by selenium deficiency; for example, IgM, IgG and IgA titers are decreased in rats, and IgG

and IgM titers are decreased in humans. In endothelial cells from asthmatics, there is a marked selenium deficiency that results in an increase in expression of adhesion molecules, which causes greater adhesion of neutrophils.[viii]

The use of selenium compounds as a cancer treatment predates most conventional treatments currently in use. [91] In spite of this, comparatively little is known regarding the use of selenium as a cancer therapy in living systems. Subcutaneous injection of 2 mcg/g selenium into tumor-bearing mice led to a 75-percent reduction in tumor mass compared to controls. [92] This inhibitory effect of selenium was confirmed in human Breast Cancer cells in vitro. [93] In an open trial of 32 patients with treatment refractory brain tumors, intravenous infusion of selenium (1000 mcg/day for 4-8 weeks) was associated with a slight to definite improvement in all participants. Symptomatic decrease was seen in nausea, emesis, headache, vertigo, and seizure activity. Although the results are largely credited to the selenium treatment, it should be noted these patients were concurrently receiving chemotherapy, oxygen therapy, vitamins E and A, dietary changes, and psychotherapy. [94] Unpublished research from the 1950s outlines the treatment of over 1000 malignancies with selenium compounds, reportedly with beneficial results. [95] Unfortunately, a study of this magnitude has yet to appear in the peer-reviewed literature.

Chromium Picolinate - (1-3 in am - 0 in pm) 200 MCG NOW FOODS 1422 01422 Chromium Picolinate 200mcg 200ct. Swanson Premium SW923

Chromium is necessary for the body to convert glucose to energy.

ZINC SULFATE?

ZINC SULFATE (4 - 12) 600mg maximum for 30 days. Peak 9.32mg/kg/D max causes WORM EXPLOSION, 50 mg zinc sulfate monohydrate in each 220 capsule,

Item No: 0802

8

http://www.Wonderlabs.com

http://www.ebay.com/itm/Rising-Zinc-Sulfate-220-mg-100-Capsules-Pack-of-3-/261936684663? hash=item3cfca49e77:g:O6EAAOSwyQtVhGGu

Zinc has been used since ancient Egyptian times to enhance wound healing, although the usefulness of this approach is only partially confirmed by the clinical data of today. Zinc Sulfate Monohydrate is a real kick at 600 mg (12 caps/day)Zinc Sulfate Monohydrate (0-2-6) -

50mg yield per capsule, WBC production, Suck Iron from Parasite, O2 (9.32mg/Kg SP) Zinc is essential, only this exact kind of zinc can be taken in these levels, "Rising". Studies show 600mg of Zinc Sulfate Monohydrate can be safely taken for a month. My worms started to burst at 9.32mg/Kg/D). Zinc is necessary for the functioning of more than 300 different enzymes and plays a vital role in an enormous number of biological processes. Zinc is a cofactor for the antioxidant enzyme superoxide dismutase (SOD) and is in a number of enzymatic reactions involved in carbohydrate and protein metabolism.

\*\*\*\*\*\*

CQ10

CQ10 (1-4) 400 mg with Vitamin E (as d-alpha Tocopherol), NOW Foods 3198 1-2 CQ10 400 mg with Vitamin E (as d-alpha Tocopherol 30 IU)

http://www.swansonvitamins.com/now-foods-cog10-400-mg-60-sgels

NOW Foods 400 mg 60 Sgels Item: NWF739 Swanson \$41.99

Cofactor: Works with vitamin e and ALA, original Japanese formulation, most studied form of CQ10.

Borage Seed oil - (1-2-4) (GLA) 1000 mg, NOW P#01722, SUN 05051 60CT 1500MG

Borage seed oil is neither a vitamin or an amino acid, but it has characteristics of both. Its most useful aspect, is that of Being one of the highest natural forms of Gamma Liolenic Acid (GLA), a substance that can directly help the DNA make additional genetic error check sum processes, ensuring accurate genetic replication within the mitochondria.

\*\*\*\*\*\*\*\*\*\*\*\*\*\*\*

Turkey Tail – Antibacterial, AntiViral, Antifungal

Up to 9 grams per day \$\$\$\$

http://www.iherb.com/Fungi-Perfecti-Host-Defense-Turkey-Tail-60-NP-Caps/21457?at=0

In traditional Chinese medicine, Turkey Tail Mushroom extract is used to treat liver cancer and some types of jaundice. 160 In modern medicine, the best known and most researched medicinal extract of Turkey Tail Mushroom is PSK. It is used in Asia as an anti-cancer drug under the brand name Krestin. 188

Two Japanese studies in the 1990's encompassing a total of 486 patients showed an increased survival rate from gastric cancer when PSK was added to conventional chemotherapy treatment. 161, 162, 163 It's also been found that PSK reduces cancer metastasis and recurrence. 161, 162, 164

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Chaparral (1-2-3) – fixes genetic psora DNA error of immune system 33% of people have, FXR bottleneck.

Chaparral (2-4) 500 mg Arizona Naturals 20201

Chaparral is essential for balancing Psora body types against a critical pathway in FXR Farnesoid chemistry where the uric acid and Bile acid are regulated by Ornathine chemistry. This critical missing acid at FXR can completely throttle your bodies ability to rid itself of Ammonia, and Uric Acid. I smelled like an Ammonia bottle for months. Got a crusty patch of skin on your knee or elbow? that is a genetic reversal of DNA that causes an immune response to a infection to attack your body, not the invader, causing local area of excess white blood cells that try to overwhelm the invader by mass movement of fluid and other T factors, which fights your body, not your infection, causing a real strain on your white blood cell chemistry. 30% of the world has a Psora body type, known for 5000 years, but medicine today is clueless about Psora. Chaparral deals with this reversal of genetic glue, to lower the inflammation of certain Tcell Thelper responses, preserving more IL6 IL10, IL17 function for your parasites, not destroying your own immune system.

Danish Rose Hips (2-2-8) - 9 cis trans retinolic A (Take Tea or capsules if you cant make tea) Only molecule that binds 3T3 in DNA Helix, Source of broad spectrum C and A vitamins. (6 SP)

8

Magnolia Extract (2-3-3) capsules, Amermed (4SP) (@ 12 the heart misses beats, keep under 6/Day. 2-3 magnolia bark 600 mg

CO2 extract (shield white blood T4 cells against microbes, levels IG and IL balance) http://www.amermed.com/magnoliabark.htm

Magnolia Extract (0-4) capsules, Amermed note: 12 capsules will stop the heart Magnolia Bark extract 600 mg CO2 extract method 600mg Magnolia Bark extract 4:1 Amermed, PPAR Gamma Interluken 17 antagonist, forces Tilter 1

### Magnolia bark

neo-ligand binds to site PPAR? that mytotoxin grabs, preventing immune system alteration. Plant lectins that can bind to sialylated glycans are from the leguminous tree Maackia amurensis. Although these lectins are discussed here, they do not show a "classical" R-type domain but instead have an L-type lectin domain. The cysteine-rich R-type domain of the MR binds other sulfated glycans and also N-glycans on pituitary glycoprotein hormones containing 4-SO4-GalNAcß1—4GlcNAcß1—2Mana1-R.nutraceutics that are reported to be able to modulate PPAR-? expression or action.

There are other R-type plant lectins in the RIP-II class that are not toxic, and these include several proteins from the genus Sambucus (elderberry), such as nigrin-b, sieboldin-b, ebulin-f, and ebulin-r. All of the B subunits of these proteins appear to bind Gal/GalNAc, but they may have some differences in affinity and may recognize different Gal/GalNAc-containing glycoconjugates. Cell lines selected for resistance to killing by modeccin are not resistant to abrin and ricin, and vice versa. The glycan-binding specificity of these lectins should be explored more fully in the future using glycan microarrays and related screening approaches.

Previous studies have shown that MR expression (Yeast Response) can be positively modulated in vitro by many agents, in particular by 1,25-dihydroxyvitamin D3 (8), prostaglandin E2 (Standard Process Cataplex E2 90 Tablets) (9), IL-4, and IL-13 (10, 11).

1 Tunjuk Langit

\*

\*\*\*\*\*

Sweetina Magnolia seeds (1)/ D very powerful. (2 SP) chopped in yogart Tunjuk Langit - rasha herbal

Used for: Severe immune malfunctions, very useful in blood toxin poisoning, near death recoveries http://rahsiaherbal.com/Herbal/TunjukLangit.aspx

http://www.amazon.com/Natural-Herbal-Fructus-Swietenia-Macrophylla/dp/B00C4QBYUI/ref=sr\_1\_1?ie=UTF8&qid=1387469365&sr=8-1&keywords=All+Natural+Asian+Herbal+Remedy%2C+Sky+Fruits+%5BTunjuk+Langit+%2F+Fructus+Swietenia+Macrophylla%5D+%2850g%29

Magnolia bark neo-ligand binds to site PPAR? that mytotoxin grabs, preventing immune system alteration.

Once activated by their ligands, the PPARs translocate into the nucleus, form heterodimers with the retinoid X receptor (RXR), and subsequently bind to PPAR response elements (PPREs) that are located in the promoter regions of PPAR-responsive target genes (Bardot et al., 1993).

Danish RoseHips CAPSULES

Danish RoseHips Swanson SWU424

https://www.swansonvitamins.com/swanson-ultra-pure-danish-rose-hips-750-mg-60-caps Rosehip seed

3-4 cups of rosehip seed tea

(restores IL17 T helper cells, prevents D3 twist in DNA helix)

http://www.mountainroseherbs.com Rosa spp. Origin- Chile

https://www.mountainroseherbs.com/search/search.php?page=3&refine=y&keywords=Rosehips

If your immune system is shot, start by buying Rosehip seeds from Mountain Rose Herbs, buy 4 pounds of seeds. get a krupps grinder. put the seeds in the grinder and make a powder, takes about 16 seconds. I put in enough seeds to cover the blade. Put the seeds in a coffee machine, do not use chlorine water. drink several coups a day. The vitamin A and C from chilean rosehip seeds is the best in the world. Take a little stinging nettle root, say 3-4 per day. Start with 4 zinc sulfates per day. Start with 3-4 selenium chelate per day. Take magnolia bark extract 4:1 Ameramed. take 2-3 per day.

Rosehip seed tea. Mountain rose herb, bulk rosehip buds, grind in coffee maker.

https://www.mountainroseherbs.com/products/rosehips/profile

Rosehip seeds grabs Vitamin D3 ( Primary Disease adaptogen 3T3 receptor, preventing white blood cells from inhibiting learned immune response T helper cells- invader infection signals.

• Immune system is no longer controlled by mytotoxin. Rosehip seed is an excellent source of topical trans-retinoic acid (vitamin A) in a natural form. Retinoic acid, found in Tretinoin, Steroid receptor superfamily has identified certain members as molecular targets for cancer therapy (1). They include estrogen receptors, retinoic acid receptors, retinoid X receptors (RXR; the RXR-specific ligands are termed "rexinoids"), and the vitamin D receptor (the vitamin D-specific ligands are termed "deltanoids"). These nuclear receptors are putative cancer therapy targets because they function as transcription factors that control the expression of many genes related to cell differentiation (1, 2). The strongest evidence for the therapeutic potential of this approach comes from the efficacy of retinoic acid receptor a activation in the treatment of acute promyelocytic leukemia (3). Over 90% of patients with acute promyelocytic leukemia achieve complete remission following treatment with the naturally occurring retinoid all trans-retinoic acid (ATRA; ref. 4).

\*\*\*\*\*\*\*\*\*\*\*

Dodder Seed Extract (2-4), Dodder at night can make your lymph system gurgle. No kidding. Dodder seed (2) extract 600mg 20:1 60-Cap, Barlowes Herbal Elixars – Tu Si Zi, \$12.95 https://barlowesherbalelixirs.com/search? orderby=position&controller=search&orderway=desc&search\_query=dodder

http://www-personal.umich.edu/~rburnham/SpeciesAccountspdfs/CuscepitCONVFINAL.pdf Background (herbs2000,com)

AKA (Latin) Cuscuta epithymum (Cuscuta epithymum extract) Etymology: In Latin, Cuscuta means Dodder. However, Cuscuta is thought by some to have Arabic origins in the word "Kushkut." The specific epithet suggests the plant that this dodder was found growing on: Thyme. The Greek prefix "epi" means upon or over, and "thymum" is Latin for thyme

Cuscuta epithymum Sievers ex Ledeb., Cuscuta epithymum Webb & Berthel., Cuscuta epithymum Thuill., Cuscuta epithymum Bové ex Choisy

Yellow-orange, spaghetti-like vine

Dodder contains flavonoids (including kaempferol and quercitin) glycoside, saponins, and hydroxycinnamic acid.

http://en.wikipedia.org/wiki/Cuscuta

odder posses medicinally properties, mentioned in the "Materia Medica" written by the Greek physician Dioscorides around the 1st century AD, the physician states dodder was used in an herbal combination with honey to purge "black bile" from the body.

\*\*\*\*\*\*\*\*\*\*\*\*\*\*\*\*\*\*

Guaifenesin (2-4-8)/D 600 mg GuaiAID 00026 http://www.guai-aid.com/

Guaifenesin reversal process, which rids the body of "metabolic debris", Lumps decrease in size, Guaifenesin increases urinary excretion of 5HIAA, a serotonin metabolite, Sodium-phosphate cotransporters. Guaifenesin pulls ATP to convert to a simple form, pulling the Mitochondria into G0.

metabolic syndrome - use ALA

LA aka Lopoic Acid contains two vicinal sulfur atoms (at C6 and C8) attached by a disulfide bond and is thus considered to be oxidized (although either sulfur atom can exist in higher oxidation states). The carbon atom at C6 is chiral and the molecule exists as two enantiomers R-(+)-lipoic acid (RLA) and S-(-)-lipoic acid (SLA) and as a racemic mixture R/S-lipoic acid (R/S-LA). Only the R-(+)-enantiomer exists in nature and is an essential cofactor of four mitochondrial enzyme complexes. (alpha version) [4]

L-Carnitine (1-5) Swanson 500mg capsule SW1000 http://www.swansonvitamins.com/swanson-premium-acetyl-l-carnitine-500-mg-240-veg-caps? otherSize=SW1649

(0-2Grams) W ALA and CQ10 to dump Toxins from DNA

(3) L-carnitine:

L-carnitine is an amino acid which nourishes the heart, nourishes and strengthens muscles, and nutritionally supports the circulatory system. L-Carnitine is considered to be a "carrier" of fat to the mitochondria or "fatburning" area of the cell. This remarkable amino acid-like substance is not only necessary for the metabolism of fat at the cellular level; it is also essential in the forming of firm, lean muscle tissue in the body. Recent studies support earlier research which shows that the heart has the greatest amount of L-Carnitine of any muscle in the body. L-Carnitine has also shown to be instrumental in the metabolism of cholesterol. Some overweight people may lack L-Carnitine in their bodies. The heart produces most of its energy from fats; thus is dependent upon L-carnitine. An L-Carnitine deficiency causes extreme metabolic impairment to heart tissue. On the other hand, supplemental L-Carnitine has proved to be beneficial to heart patients.

**19 SOD** 

SOD (1-2-4) 250mg SOD SWANSON Blend 60CT SW157 02157, Source naturals SN519 90ct \$6.19

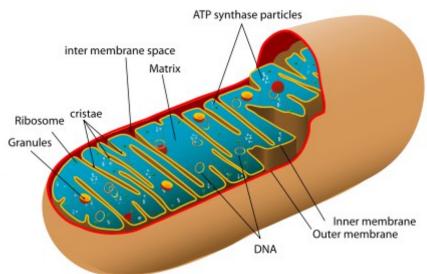
### Cellular core processes

#### Mitochondria:

Energy production (metabolic function) in the body is reliant on mitochondria in the cells (pictured above ).

The mitochondria are the body's furnaces, that are responsible for the production of energy inside

each cell. They take in oxygen, sugar and ADP (effectively spent energy) and produce energy, carbon dioxide and ATP (the currency of energy). A compelx variety of processes and compounds are involved in this process.



#### http://en.wikipedia.org/wiki/Mitochondria

http://users.rcn.com/jkimball.ma.ultranet/BiologyPages/C/CellularRespiration.html

www.immunesupport.com/library/showarticle.cfm/id/8799

An e-book (pdf) by Joseph L. Evans, PhD, of Xymogen, entitled 'The Secret Life of Mitochondria', can be read at the link below.

### www.xymogen.com/2008/xymo\_mitobook.pdf

The mitochondria are responsible for producing Adenosine-5'-triphosphate (ATP). The molecule with its three phosphate groups is pictured below.

www.biology-online.org/1/2\_ATP.htm http://en.wikipedia.org/wiki/Adenosine triphosphate

**ATP** is a multifunctional nucleotide, it's main role being a coenzyme responsible for intracellular energy transfer. That is to say, during cellular respiration, the mitochondria inside each cell produce **ATP**, a coenzyme, which acts to distribute chemical energy inside of that cell for metabolism. The **ATP** moves out of the mitochondrial membrane and float around inside the cell in the cytoplasm until it is used up in a variety of processes, described below.

Energy is released when <u>ATP</u> (adenosine triphosphate) is converted to ADP (adenosine diphosphate). This occurs by breaking off a phosphate molecule from the <u>ATP</u> molecule, the action of which actually releases energy which can be absorbed by a protein or enzyme as part of a cellular activity. It drives virtually every biochemical reaction in the body and is in constant demand by the cells of the body.

www.trueorigin.org/atp.asp http://users.rcn.com/jkimball.ma.ultranet/BiologyPages/D/Diffusion.html

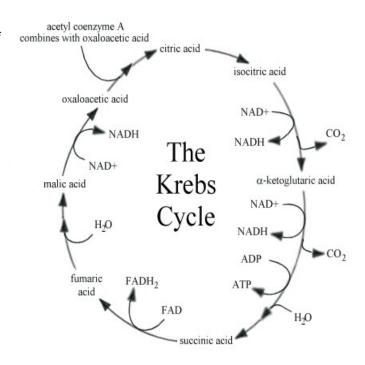
**ATP** is consumed by various enzymes and many cellular processes. These include the active transport of nutrient ions such as Sodium (Na+) or Potassim (K+) against the concentration gradients at the cell membrane, e.g. enabling transport proteins to push K+ into the cell and push Na+ out of the cell; other uses include mechanical work (e.g. muscle contraction - for skeletal movement and heart muscle to circulate blood around the body), motility (ability for a cell to move or 'swim' using its flagella - the tail like structures that protrude from the cell walls), biosynthetic/chemical reactions (i.e. conversion of chemical compounds and creation of macromolecules essential to life, e.g. conversion of amino acids from one form to another, creation of enzymes and coenzymes, etc.), act as a binary on/off control mechanism to cellular reactions by changing the shape of peptide chains (when energy is absorbed or released), and cell division.

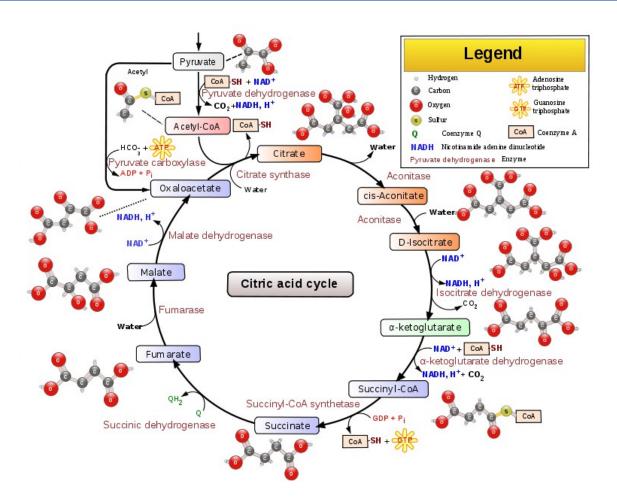
http://www.cliffsnotes.com/WileyCDA/CliffsReviewTopic/Mitochondrial-Transport-Systems.topicArticleId-24998,articleId-24992.html

The average person turns over approximately his or her own body weight in **ATP** each day. Studies show that a person of 68 produces approximately half the amount of **ATP** compared with a person of 39 years of age. An electron micrograph of a single mitochondrion showing the organised arrangement of the protein matrix and the inner mitochondrial membranes is shown above (Photo: U.S. Dept. of Health and Human Services/National Institutes of Health).

### **Krebs / Citric Acid Cycle:**

The main activity of the mitochondria is the recycling of ADP back to **ATP**, so that the **ATP** can again be used for energy release around the cell. The conversion of ADP back to **ATP** is achieved by a complex set of biochemical processes, which are part of the Krebs (citric acid) Cycle, and by the action of **ATP** Synthase enzymes. The Krebs Cycle, representing one form of aerobic metabolism, is pictured below





http://en.wikipedia.org/wiki/Citric\_acid\_cycle http://cellbio.utmb.edu/cellbio/mitochondria\_1.htm www.vrp.com/articles.aspx?ProdID=art868&zTYPE=2

'The citric acid cycle, also known as the tricarboxylic acid cycle (TCA cycle) or the Krebs cycle,...is a series of enzyme-catalysed chemical reactions of central importance in all living cells that use oxygen as part of cellular respiration. In eukaryotes, the citric acid cycle occurs in the matrix of the mitochondrion. The components and reactions of the citric acid cycle were established by seminal work from both Albert Szent-Gyšrgyi and Hans Krebs. In aerobic organisms, the citric acid cycle is part of a metabolic pathway involved in the chemical conversion of carbohydrates, fats and proteins into carbon dioxide and water to generate a form of usable energy. Other relevant reactions in the pathway include those in glycolysis and pyruvate oxidation before the citric acid cycle, and oxidative phosphorylation after it. In addition, it provides precursors for many compounds including some amino acids and is therefore functional even in cells performing fermentation.'

#### Inner mitochondrial membrane

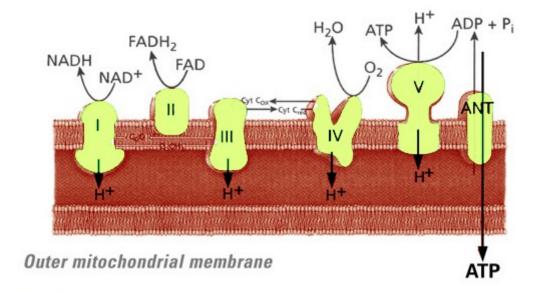


Fig. 2. Electron transport chain, by which oxidative phosphorylation takes place. Complex I (NADH dehydrogenase) accepts fuel from the citric acid cycle in the form of NADH, which donates electrons to the chain. Complex II (succinate dehydrogenase) accepts electrons from FADH2 and passes them to Complex III (cytochromecreductase) via CoQ 10. The electron is then passed to Complex IV (cytochromecoxidase) via cytochrome C. Complex V (ATP synthase) converts adenosine diphosphate (ADP) to adenosine triphosphate (ATP)—the chemical energy "currency" of the cell. ATP can then be transported to where work needs to be done.

#### http://en.wikipedia.org/wiki/Electron transport chain

'An electron transport chain (ETC) couples a reaction between an electron donor (such as NADH) and an electron acceptor (such as O2) to the transfer of H+ ions across a membrane, through a set of mediating biochemical reactions. These H+ ions are used to produce adenosine triphosphate (**ATP**), the main energy intermediate in living organisms, as they move back across the membrane.'

The electron transport chain is a very finely balanced system and may be easily disrupted by a number of processes described below, deficiencies, as well as a 'confused' and 'unbalanced' body being bombarded with various types of unwanted supplements.

### Inefficient Recycling of ADP back to ATP, and AMP Production:

In CFS patients, one of the main causative factors is inefficiency in recycling ADP back to **ATP** again. This pathway is often the bottleneck in energy production in such individuals. If the cell is not efficient at recyling ADP to **ATP**, then the cell runs out of energy very quickly, which causes the symptoms of weakness and poor stamina. The cell must then go into a 'rest' period until more **ATP** can be manufactured/recycled (from ADP). At any one time, the cells in the heart muscle only have enough **ATP** in reserve for around 10 contractions. If a cell is pushed to produce energy when no **ATP** is available, then it will use the ADP instead, and convert this into AMP (adenosine monophosphate). AMP consists of a phosphate group, the sugar ribose, and the nucleobase adenine. AMP cannot however be recycled, which is why the body does not normally use ADP to produce energy from. Any **ATP** which is converted to AMP is considered to be 'spent'. So any **ATP** 

must be recycled from any ADP that remains, and the rest must be created from scratch using fresh raw ingredients. To create **ATP** from scratch, the body must first breaking down the various proteins, triglycerides, fatty acids and sugars into their constituent parts, and then the mitochondria must build up **ATP** from these components using its enzymes **ATP** Synthase (see the Krebs Cycle above).

### http://en.wikipedia.org/wiki/Adenosine\_monophosphate

'The ratio between **ATP** and AMP is used as a way for a cell to sense how much energy is available and control the metabolic pathways that produce and consume **ATP**. Apart from its roles in energy metabolism and signaling, **ATP** is also incorporated into nucleic acids by polymerases in the processes of DNA replication and transcription.'

### www.cfids-cab.org/MESA/DrMyhill-373.pdf

The slow production of <u>ATP</u> by the body from scratch (when little or no ADP is available to convert) partly explains the delayed and prolonged fatigue that CFS patients experience after intensive activity, 'overdoing it' (more severe than their 'usual fatigue', i.e. a 'crash' or a 'flair') or even losing too much body heat (outside in the cold or at night with insufficient blankets). This is why CFS patients should try to pace themselves and take regular breaks and respect their limits so that they allow themselves a chance to regenerate <u>ATP</u> again rather than converting too much ADP to AMP, where they end up in the deficit part of the cycle (i.e. feeling much more fatigued than normal etc.)

This is further examined in the paper below, 'Chronic fatigue syndrome and mitochondrial dysfunction' (12 January 2009) by Sarah Myhill, Norman E. Booth, John McLaren-Howard.

#### http://www.ijcem.com/files/IJCEM812001.pdf

#### **Recycling AMP?**

Although Dr Sarah Myhill states in the above paper 'Chronic fatigue syndrome and mitochondrial dysfunction' that AMP cannot be recycled, she makes the further assertion that a small amount of AMP can actually be recycled:

Dr Sarah Myhill's book 'Diagnosing and Treating Chronic Fatigue Syndrome' 26th edition, January 2009:

### http://www.drmyhill.co.uk/cfs\_book.pdf

Dr Sarah Myhill's article: 'CFS - The Central Cause: Mitochondrial Failure' (January 2009):

#### www.drmyhill.co.uk/article.cfm?id=381

'Problems arise when the system is stressed. If the CFS sufferer asks for energy faster than he can supply it, (and actually most CFS sufferers are doing this most of the time!) **ATP** is converted to ADP faster than it can be recycled. This means there is a build up of ADP. Some ADP is inevitably shunted into adenosine monophosphate (AMP -1 phosphate). But this creates a real problem, indeed a metabolic disaster, because AMP, largely speaking, cannot be recycled and is lost in urine.'

'And now for a bit of good news! You will have read (and will read again) that AMP cannot be recycled. Actually, AMP can be recycled, but it happens very slowly. For practical purposes for patients who are very fatigued, this recycling is so slow that it is clinically insignificant. Interestingly, the enzyme which facilitates this recycling ("cyclic AMP") is activated by caffeine! So the perfect pick-me-up for CFS sufferers could be a real black organic **coffee** with a teaspoon of D-ribose! Not too much or one can run into calcium problems.'

I have not heard the above assertion regarding the ability to recycle AMP in very limited quantities, however am keen to find further sources that arrive at the same conclusion. However, it should be noted that the amount of additional **ATP** that can be recovered or recycled by consuming caffeine is relatively small, and one may wish to offset this against the negative effects of caffeine and/or **coffee** consumption (i.e. acidic pH, toxicity, diuretic qualities.)

#### **Anaerobic Respiration:**

Dr Sarah Myhill states in her article 'CFS - The Central Cause: Mitochondrial Failure' (January 2009) that anaerobic respiration is another mechanism used when insufficient **ATP** is available for requirements.

#### www.drmyhill.co.uk/article.cfm?id=381

'However there is another problem. If the body is very short of **ATP**, it can make a very small amount of **ATP** directly from glucose by converting it into lactic acid. This is exactly what many CFS sufferers do and indeed we know that CFS sufferers readily switch into anaerobic metabolism. However this results in two serious problems - lactic acid quickly builds up especially in muscles to cause pain, heaviness, aching and soreness ("lactic acid burn"), secondly no glucose is available in order to make D-ribose! So new **ATP** cannot be easily made when you are really run down. Recovery takes days! When mitochondria function well, as the person rests following exertion, lactic acid is quickly converted back to glucose (via-pyruvate) and the lactic burn disappears. But this is an energy requiring process! Glucose to lactic acid produces two molecules of **ATP** for the body to use, but the reverse process requires six molecules of **ATP**. If there is no **ATP** available, and this is of course what happens as mitochondria fail, then the lactic acid may persist for many minutes, or indeed hours causing great pain. (for the biochemists, this reverse process takes place in the liver and is called the Cori cycle).'

Anaerobic respiration is defined on Wikipedia at the link below.

#### http://en.wikipedia.org/wiki/Anaerobic respiration

Lactic acid build up (and improper breathing - i.e. CO2 build up) can contribute to <u>Acidosis</u> as well as muscle ache and 'burn' as described above.

#### According to Genova Diagnostics:

'Lactic acid, or lactate,...is formed from pyruvate in anaerobic or oxygen starved (hypoxic) circumstances to allow for ongoing production of **ATP** in these anaerobic conditions. There are no known clinical problems associated with low lactic acid. Low levels are usually a result of reduced amounts of its precursor, pyruvic acid.'

Please see the <u>Cardiac Insufficiency</u> page for more information.

N.B. Please note for Dutch readers that this section and all subsequent sections on this page have been translated into Dutch by a patient of Paul van Meerendonk of Biologisch Medisch Centrum in Utrecht, on his/her personal web site, with some removal of references to my personal experiences. The web site has no actual connection to van Meerendonk (nor myself).

www.biologischmedischcentrumbmc.nl/adp-atp-efficiency.htm

#### Reduced numbers of Mitochondria in each cell:

Another potential factor in explaining poor **ATP** availability is perhaps a lowered level of mitochondria in CFS patients or those with mitochondrial dysfunction in the first place. Mitochondria themselves have a very short life. In humans, it is estimated that they have a half life of 5-12 days (meaning half the mitochondria in the body will have 'died' after 5-12 days if no more were produced. In rat cardiac muscle, the half life is 18 days. If we were to assume the latter as a best case scenario, then the body would need to replace approximately 6% of its mitochondria every day. Mitochondria are recycled in the process called autophagy. This recycling of mitochondrial to produce new mitochondria requires energy, or ATP, which clearly if in deficit to start with, may be delayed or postponed, meaning that the resulting remaining functioning mitochondria may be somewhat less than it should be in a healthy organism. Fewer mitochondria means those that remain are put under more pressure to produce ATP and are thus depelted quicker than they would normally be. Most of this autophagy occurs during sleep when ATP demand is lowest, but ironically this is something that many CFS patients do not get enough of. This may be an important factor in explaining the low rate of mitochondrial regeneration (a catch 22 situation). Perhaps to some degree this is mitigated by having less mitochondria to recycle in the first place, so that an equilibrium is reached, somewhere below the amount that should be. How significant autophagy requirements are compared with cellular recycling in general (new cells) and muscle growth is not something I am an expert on, but indeed, all such functions seem to be impaired in CFS sufferers to varying degrees; exaccerbated by poor amino acid conversion.

#### 'An Engineering Perspective on CFS' (7 Nov. 2008) - by Dave Whitlock

In the above article (discussed by myself on the Peroxynitrite page), Dave Whitlock argues that low basal NO levels may explain low levels of mitochondrial regeneration, resulting in lower numbers of mitochondria per cell than a normal, healthy person. NO (Nitric Oxide) is a major regulator of ATP levels. Low NO levels causes low ATP levels, which thus disables autophagy, preventing recycling of mitochondria. There is more peroxynitrite damage observed not because peroxynitrite levels are high and NO levels are higher, but because there is less recycling of mitochondria occuring (less autophagy) and hence less repair of peoxynitrite-damaged proteins and lipids. In other words, there is a resulting accumulation of peroxynitrite-damaged proteins. Because of low NO levels, there is less synchronisation between cells in terms of their energy output (in a muscle group or particular organ), meaning some are overloaded and some are underloaded. According to Whitlock, techniques do not exist to measure if adjacent cells are working 'in sync'. Whitlock proposes a number of methods of boosting NO levels (or more specifically NO donors) in the body to allow the body to produce more mitochondria, which include (in no particular order and not necessarily recommended by me as this is a THEORY) taking Nitroglycerine, L-arginine, Viagra, eating more green leafy vegetables, and meditation.

I believe that in many cases, the actual integrity of the <u>mitochondrial membranes</u> is more of issue than their actual number, which may or may not be normal. This is discussed below. However, it may be worthwhile in looking at all aspects of mitochondrial function to identify where the bottleneck(s) is.

Paul Cheney and Martin Pall argue the exact opposite (as discussed on the <u>Nitric Oxide and Peroxynitrite</u> page), that NO levels and Peroxynitrite levels in CFS patients tend to be higher than normal, rather than lower, on account of the enzymatic activities associated with over-immune system activation, on account of prolonged exposure to viri or bacterial infections etc., amongst other factors. Cheney proposes a numbe of methods of reducing one's NO production. As to who is correct, I am not certain, and it presumably depends on the exact individual in question as to what is going on on a specific biochemical level and where. Everyone however is probably in agreement that poor mitochondrial function is behind cardiac insufficiency.

#### **Effect of Hydrogen Sulphide on the Mitochondria:**

As stated on the <u>Bacterial Overgrowth</u> page and above, Hydrogen Sulphide (H2S) is an endogenous toxin produced in the body by the action of bad bacteria (e.g. Prevotella) and fungi (such as Candida Albicans) fermenting sugar in the gastrointestinal tract. Elevated levels of H2S in the blood and tissues can result in mitochondrial dysfunction by their action on the Cytochrome C Oxidase enzyme which is involved in <u>ATP</u> production. Please see the <u>Toxicity</u> page for more information regarding H2S effects and treatment.

http://en.wikipedia.org/wiki/Hydrogen sulfide

<u>Hypothesis: Is ME/CFS caused by dysregulation of hydrogen sulfide metabolism? (2008) by Marian</u> Dix Lemle

#### **Effect of excessive D-Lactate on the Mitochondria:**

Streptococcus and Enterococcus bacteria ferment fibre to produce lactic acid. The two isomers of Lactic acid produced are L-Lactate and D-Lactate. Humans (and mammals in general) only produce L-lactate as part of anaerobic respiration and only possess the enzymes Lactate Dehydrogenase (LDH) for metabolising L-Lactate in any significant quantity. Mammals do not possess the D-Lactate Dehyrogenase enzyme in any significant quantity, and this is generally only found in plants and bacteria.

In humans, the two LDH enzymes act on L-Lactate to convert it into Pyruvate (and vice versa). One of these enzymes e.g. in Glycolysis in the NAD(P) dependent L-Lactate Dehydrogenase enzyme (EC.1.1.2.3). The other LDH enzyme is a Cytochrome c-enzyme found in the liver (EC.1.1.1.27). Mammals including humans however can metabolise D-Lactate using the D-alpha-hydroxy acid dehydrogenase enzyme found in the mitochondria (at 20% of the rate of a proper D-Lactate Dehydrogenase enzyme as found in plants).

#### http://en.wikipedia.org/wiki/Lactate\_dehydrogenase

If excessive conmensal Streptococcus and Enterococcus fermentation in the GI tract occurs, then D-Lactate levels tend to rise in th body, and <a href="mailto:acidosis">acidosis</a> (a drop in blood pH) occurs - known as D-Lactic Acidosis. D-Lactate can accumulate in the mitochondria and inhibit their proper function. The body then has two main methods available to eliminate D-Lactate are renal excretion (i.e. whatever is in the fluid filtered off by the kidneys into urine) and via faeces (excreting the D-Lactate remaining in the stool) - which is not particularly efficient in clearing the D-Lactate, especially if it is being produced continually in the GI tract. Recent studies however have claimed to show that humans do actually possess the D-Lactate Dehydrogenase enzyme on the inner mitochondrial membrane. Studies from the 1920s showed that D-Lactate was poorly metabolised compared with L-Lactate, whereas studies from the 1980-90s found that D-Lactate was actually readily metabolised, although most academic and medical sources still quote the 1920s results as fact. The area is still hotly debated.

D-Lactic Acidosis is rare in general terms and usually only occurs in the case of <a href="syndrome">syndrome</a> in humans (malabsorption disorder caused by surgical removal of the small intestine) and children with gastroenteritis. It can of course occur in patients who have markedly poor digestion with a large proportion of undigested carbohydrate in the GI tract. In animals, it can occur through excessive grain consumption by ruminants (e.g. cattle, goat, sheep etc.) or in cases of diarrhea in calves.

www.cfids-cab.org/rc/Sheedy.pdf http://jn.nutrition.org/cgi/content/full/135/7/1619

www.biolab.co.uk/docs/dlactate.pdf http://www.drmyhill.co.uk/wiki/Fermentation in the gut and CFS

Steptococcus and Enterococcus are types of lactic acid bacteria. There are many different species, some are probiotic, some are commensal and some are pathogenic. Probiotic strains include S.thermophilus, S.salivarius and S.faecium; and E.faecium and E.faecalis. The species most likely to be relevant in this instance are the commensal strains (i.e. imbalanced flora) that mke up the bulk of these species in the GI tract.

http://en.wikipedia.org/wiki/Streptococcus http://en.wikipedia.org/wiki/Enterococcus

Other pathogenic bacteria besides Steptococcus and Enterococcus also produce D-lactate, although these are probably not so likely to be the cause in most cases of D-Lactic Acidemia:

#### http://jn.nutrition.org/cgi/content/full/135/7/1619

'Various pathogenic bacteria produce D-lactate, including Bacteroides fragilis, Escherichia coli, Klebsiella pneumonia, and Staphylococcus aureus. The use of D-lactate as a marker for infection was proposed in 1986.'

It is possible that a disproportionately large amount of probiotic lactic acid producing species such as Strepococcus and Enterococcus can be responsible for D-Lactic acidemia. It is more likely that the imbalanced S. and E. flora species would be responsible (in instances of elevated undigested carbohydrates in the GI tract) and that repopulation with the relevant required numbers of probiotic species, both lactic and non-lactic acid producing species, would help to correct the problem. Some recommendations do include abstaining from taking additional lactic acid producing probiotic bacteria, and only consuming non-lactic bacteria and bacteria that consume D-Lactate.

Please see the <u>Bacterial</u> page for this topic and related areas.

D-Lactate levels can be measured in a blood test. Please see the <u>Tests</u> page for more information.

#### Excessive oxidative damage to Mitochondrial DNA:

In addition to excessive free radicals damaging the mitochondrial membranes, they may also cause damage to the actual mitochondrial DNA itself. Mitochondrial DNA is completely separate from nuclear DNA. Unlike Nuclear DNA, it is inherited solely from the mother in sexually reproducing organisms, e.g. humans. Mitochondrial DNA, because of its close proximity to the inner mitochondrial membrane's respiratory chain, a primary source of free radical production, and also their limited capacity for self-repair and self-protection, are particularly susceptible to free radical damage. General cell protection from damage by Superoxide is provided by intracellular Zinc:Copper SOD (Zn/Cu-SOD). Mitochondria are protected by Manganese-dependent SOD (Mn-SOD). Extracellular SOD (EC-SOD - another type of Zn/Cu SODase) protects the nitric oxide pathways that relax vascular smoother muscle tissue. For each form of SOD, genetic variations are known, and mutations and polymorphisms can occur during excessive oxidative stress placed on the DNA. DNA adducts (toxins that attach to DNA genes) can chemically block these genes however. Zinc, Copper and Manganese are extremely important elements for maintaining healthy SOD levels, and patients should ensure that these mineral levels are supplemented if they drop below their reference ranges.

http://en.wikipedia.org/wiki/Mitochondrial\_DNA http://qhr.nlm.nih.qov/chromosome=MT

In addition, free radicals can also be produced in excess by the liver. Liver function with regards to

clearing undesired compounds from the blood involves a two step process. This is described in detail on the <a href="Inefficient Liver function">Inefficient Liver function</a> page. The first step is Phase 1 Regulation, using the Cytochrome P450 enzymes, which are largely a set of oxidase reactions, producing a large number of free radicals. Sufficient antioxidant compounds and chemicals are required by the liver in order to keep these from causing too much damage within the liver and outside of the liver, these are both endogenously produced <a href="antioxidants">antioxidants</a> and dietary sources of <a href="antioxidants">antioxidants</a>. The second step of liver function is the Phase 2 Conjugation step, whereby molecules are added to the toxins in order to make them easier to remove from the body. These processes work in a perfect balance in a healthy liver. If antioxidant and conjugation steps are impaired, then a large number of free radicals will be produced which can cause oxidative damage within the liver and also spill out into the blood stream, flooding it with excessive free radicals.

The role of **antioxidants** such as Superoxide Dimutase and R-Lipoic Acid are discussed on the Cardiac and Nutritional pages.

#### Peroxynitrite-induced NADH deficiency:

Several types of DNA damage can be inflicted including the nicking of of the backbone of DNA chains. These nicks stimulate the poly (ADP-ribose) polymerase enzyem, which uses Active Vitamin B3 (NAD) as a substrate. NADH is the reduced form of Active B3 that is involved in the electron transport chain in mitochondria. Therefore elevated poly (ADP-ribose) polymerase enzyme production on account of the DNA damage caused by ONOO- can lead to a depletion of the pools of NADH/NAD that are normally used in mitochondrial function, thus heavily impacting **ATP** availability.

### **Mitochondrial Membrane Integrity:**

Factors affecting Mitochondrial Membrane Integrity:

Any factors that affect the mitochondrial membrane can severely impact the body's ability to aerobically respire and force it to use anaerobic respiration more, or to convert **ATP** to AMP, to produce energy, include some of the following:

#### Fatty Acid imbalances:

Essential Fatty Acids (EFAs), namely Omega 3 and 6 fatty acids, are a key component of cellular membranes and brain tissue, and with an insufficient intake, or excessive intake of bad saturated fats, rancid (heated polyunsaturated) fats or trans fats, our cellular membranes may experience inflammation and a loss of permeability essential to cellular and mitochondrial functioning. For more information see the <a href="Nutritional">Nutritional</a> page. If mitochondrial membranes are less permeable, it means that the requisite nutrients required to produce <a href="ATP">ATP</a> are not available quickly enough, and so it has a knock-on effect in terms of <a href="ATP">ATP</a> production in the cells of the body. The body is not able to produce energy as efficiently. Red blood cells are not able to oxygenate as efficiently either, nor to deliver their payload of oxygen to the cells of the body.

• Excessive free radical (oxidative) damage to the mitochondrial membrane:

Free radicals damage the integrity of the mitochondrial membrane by attacking/oxidising the actual phospholipids that make up the majority of the mitochondrial membrane material. Free radicals are produced inside the mitochondria as a byproduct of energy production and respiration. The more energy you produce, the more free radicals you

produce. The body has its own natural defence against such free radicals to prevent excessive free radical damage to the mitochondrial membranes. These are the antioxidant enzymes SOD and to a somewhat lesser extent **Glutathione**. Mitochondrial function is limited in a sense by the available of SOD as without it extensive mitochondrial damage would occur (with elevated respiration rates beyond the available SOD and **glutathione** that can be produced. However, if levels of the rogue oxidant peroxynitrite are elevated in the mitochondria, these may destroy the SOD enzymes, resulting in an increase in Sueroxide levels produced during respiration, resulting in more free radical damage to the mitochondrial membranes. Please see the Nitric Oxide and Peroyxnitrite Cycle page for more information on Peroxynitrite formation and damage.

A deficiency in the production of either of these primary antioxidant enzymes can of course result in excessive free radical damage to the mitochondrial membranes and excessive perforation and leaking of powerful free radicals like Superoxide out of the mitochondria, causing additional knock on problems. Damaged mitochondrial membranes are sometimes referred to as causing 'energy leaks', although this is rather a gross simplification.

The mitochondrial membranes are subject to wear and tear from the residual heat-induced damage from frequent discharge of its electric polarity.

http://www.lef.org/prod hp/abstracts/centrophenoxineabs.html#9

Two indicators (or downstream products) of excessive (phospho)lipid peroxidation (and mitochondrial membrane damage) are the aldehyde derivates <a href="Malondialdehyde">Malondialdehyde</a> (MDA) and <a href="Motoroaldehyde">Crotonaldehyde</a>, which can be found attached to damaged mitochondrial membranes. These factors are discussed in more detail in the next bullet point below.

A third indicator of peroxidation of the mitochondrial membranes is F2-Alpha Isoprostane (a.k.a. 8-iso-PGF2 alpha). It is a downstream oxidation production of membrane oxidation, specifically the Omega 6 Essential Fatty Acid (EFA) component of the phospholipid matrix. Isoprostanes as prostaglandin-like compounds created from the free radical attack of esterified of the Omega 6 EFA known as Arachidonic Acid (ARA) inside the membrane phospholipid. ARA is particularly sensitive to peroxidation by free radicals. 8-iso-PGF2 alpha is of course a very different molecule to a prostaglandin which is a lipid compound produced enyzmatically by the body using cyclooxygenase (Cox1-2) from EFAs and has nothing to do with free radical attack on cell membranes. Isoprostanes in general are valuable markers in clinical biology as they are found in all biological fluids and tissues and are stable in vivo and ex vivo. There are 10 times more isoprostanes in atherosclerotic plague compared with normal vascular tissue. They are also only dependent on their production (in this case free radical attack) rather than metabolism or excretion without intraindividual variability. There are many studies validating Isoprostanes as the most accurate and reliable indicator of oxidative stress in vitro and in vivo. F2-Alpha Isoprostane levels can be measured in one's urine and are a direct

measurement of the extent of free radical oxidation of the mitochondrial membranes. Please see the <u>Oxidative Stress Tests</u> section on the Tests page for more information.

• Compounds clogging up the Mitochondrial Membranes:

Toxins, waste products, partial detoxification products (failed detoxification attempts) or foreign/unwanted compounds cab clog up the mitochondrial membrane, in particular the translocator (TL) protein sites, thus reducing mitochondrial membrane permeability and **ATP** production. Mitochondria are the energy furnaces of the cells of the body, and it is essential for certain types of compounds, nutrients and minerals to be able to carried enzymatically in and out of the inner and outer membrane. If the mitochondrial membranes are clogged up, it can result in a bottleneck in the krebs cycle and thus a reduced capability to produce **ATP**, the energy currency of the cell. There should not be any such 'garbage' clogging up the mitochondrial membranes.

Translocator Protein (TL) scavenges ADP from the cytoplasm and returns **ATP** from re-conversion with 'new' **ATP** from oxidative phosphorylation. TL can be blocked by zenobiotics and/or partial detoxification products.

The presence of unwanted substances that can interfere with mitochondrial function contribute to a condition known as Neurotoxic Membrane Syndrome (a.k.a. Chronic Neurotoxic Syndrome). Please see the <a href="Inefficient Liver Function">Inefficient Liver Function</a> page for more information.

An article 'The Detoxx System: Detoxification of Biotoxins in Chronic Neurotoxic Syndrome' [a.k.a. Neurotoxic Membrane Syndrome] by John Foster, M.D., Patricia Kane, Ph.D., Neal Speight, M.D. is shown at the link below.

http://articles.mercola.com/sites/articles/archive/2003/08/09/detoxification-biotoxins.aspx

Examples of unwanted compounds on the mitochondrial membrane could include heavy metals, PCBs, PBBs, pesticides, dichlorobenzene, foreign DNA/RNA (probably viral), lactic acid,, keto-acids, lipofuscin and the body's partial detoxification products including peptide complexes, **glutathione** conjugates and organic sulphate conjugates (the body's detoxification defence molecules bonded with toxins for removal from the body). This may in turn be a result of impaired liver function and/or excessive toxin build up in the body.

Two indicators (or downstream products) of excessive (phospho)lipid peroxidation (and mitochondrial membrane damage), as discussed above, are the aldehyde derivates <a href="Malondialdehyde">Malondialdehyde</a> (MDA) and <a href="Crotonaldehyde">Crotonaldehyde</a>. MDA is reactive and potentially mutagenic (changing one's DNA). Crotonaldehyde is a known irritant.

According to John McLaren Howard of Acumen Laboratory, Aldehydes from lipid peroxidation (when it occurs) tend to accumulate in the mitochondrial membranes. Such aldehydes, usually in the form of

malondialdehyde or crotonaldehyde, can block some translocator (TL) sites in the inner mitochondrial membranes. It is rare for them to accumulate to very high levels on the inner mitochondrial membranes but moderate or trace accumulation on TL sites does occur. The accumulation of these aldehydes on the mitochondrial membrane can impact oxidative phosphorylation (i.e. **ATP** production) and energy production significantly. In other words, their presence not only tells us that the mitochondrial membranes themselves have been oxidised/damaged (as above) but their presence on the mitochondrial membrane itself may also may be actively preventing proper mitochondrial function.

However, according to John McLaren Howard, the body tends to clear MDA from the mitochondrial membranes, but if they are being produced as quickly as they are being removed, then they will remain on the mitocondrial membranes. If one stops or reduces the source of oxidative stress which resulted in the oxidation of the lipids and hence their production, then their levels will go down. The only exception to this is where MDA is adducted to the DNA (i.e. DNA Adducts) where it cannot be cleared very quickly.

It is possible to have clogged up/blocked TL sites AND damaged mitochondrial membranes; or fairly in fact mitochondrial membranes that are very clogged up.

Poor protein digestion can also result in peptide complexes (short chain proteins) attaching themselves to the mitochondrial membrane. In addition, poor immune function may result in cytokines attaching themselves to the mitochondrial membrane, and perhaps causing mitochondrial clumping (which in turn may disturb the function of the cytoskeleton within the cytoplasm of the cells). Cytokines are immune system messenger molecules (either up regulatory or down regulatory).

### **Cytokines**

are defined by Wikipedia <u>here</u>.

Such protein attachment to membranes does not normally affect translocator protein sites, but could affect **ATP** function in a more general way. More information on cell membrane congestion can be found in the <u>identification</u>, <u>nutrition</u> and <u>toxicity</u> pages. Cytoskeleton is defined by wikipedia below.

http://en.wikipedia.org/wiki/Cytoskeleton

• Elevated Hydrogen Sulphide levels:

Elevated H2S levels caused by the fermentation of sugar by bad bacteria and fungi in the digestive tract - H2S attaches to the mitochondrial enzyme Cytochrome C Oxidase and weakens oxidative phosphorylation and **ATP** production.

Too low a pH at the membrane (e.g. too acidic):

The Translocator (TL) Protein site on the inner mitochondrial membrane is extremely pH sensitive and can be affected by local or general acidosis, including the organic acid accumulation from over-dependence on anaerobic metabolism (the dominant form of metabolism where oxygen levels are too low).can affect the ability of the TL sites

• Elevated intracellular Calcium and reduced intracellular Magnesium:

The efficiency of TL site can also be compromised by increased intracellular Calcium or reduced intracellular Magnesium. Enzymatic reactions are required to transport minerals in and out of the cells of the body, which require **ATP**. **ATP** shortages can have knock on effects as above which further exacerbate the problem.

Mitochondrial dysfuction may in turn affect hypothalamic/hormonal dysfunction, poor liver and kidney functioning, cardiac capability and digestive efficiency. Mitochondrial function will impact all the cells of the body and their normal function to some degree, impacting all the organs and glands (some more than others), and their ability also to produce enzymes and hormones as they should etc. Symptoms of mitochondrial disfunction may include a lack of physical energy, lack of mental energy and ability to concentrate ('brain fog'), tendency to crash and burn, muscle and joint weakness, cardiac weakness/insufficiency, digestive inefficiency, and perhaps even muscular control. The exact effects varies according to the individual.

Getting sufficient oxygen to the mitochondria is key to enabling proper mitochondrial function. Low blood and body oxygen levels are frequently associated with excessive fat, insufficient cardiovascular exercise, slightly lowered blood/bodily pH (excessive acid producing food consumption), fatty acid imbalances and/or poor cell membrane permeability.

A high intake of essential minerals and krebs cycle metabolites, and sufficient levels of these in the blood, does not necessarily correlate to sufficient levels of these at the mitochondrial membrane (e.g. on account of toxin congestion and of displaced zinc from zinc finger proteins for example on account of the presence of heavy metals.)

#### Repairing oxidative damage to the Mitochondrial Membranes:

In order to repair the mitochondrial membranes, one must ingest or produce enough of the requisite Phospholipids and ingest sufficient Essential Fatty Acids. The problem that often occurs in CFS patients is that the body is unable to produce high volumes of Phospholipids on account of a blockage in the methylation pathway, which is dependent on the bio-availability of the correct amino acids as well as active forms of Folate and B-12. Thus, it is very important for those with damaged or leaking mitochondrial membranes to supplement sufficient Essential Fatty Acids (Omega 3 and 6) and also Phospholipids (especially Phosphatidyl Choline). This is discussed on the <a href="Nutritional">Nutritional</a> page. Phospholipid Therapy is discussed in detail on the <a href="Detoxification">Detoxification</a> page.

#### Clearing foreign/unwanted matter/waste from the mitochondrial membranes:

Treatments for removing heavy metals and partial detoxification products from the mitochondrial membrane, for example clathration, chelation, LED and Phospholipid supplementation or injections (PLX), are examined on the <a href="Detoxification">Detoxification</a> page.

Please note that heavy chelation may in the short term have a negative impact of mitochondrial function as more heavy metals are present in the blood, albeit usually bounded to a chelating agent, and detoxification requires **glutathione**, which is also needed as a protective measure to prevent oxidative damage during respiration (secondary to Superoxide Dismutase (SOD)), and if more is being used up for chelation, then less will be available for respiration. So a chelation programme must be offset against one's mitochondrial capability and levels of gluthatione production at any one moment in time.

Treatments for removing protein attachment (peptides from poor digestion or cytokines) to interand intra-cellular membranes include <u>FIR saunas</u> and <u>PLX (Phosphatidyl Choline/Glutathione)</u> <u>injections</u>, Phospholipid oral supplementation, as well as perhaps Zinc and Magnesium injections (in the case of cytokines). Phospholipid therapy is a type of detoxification protocol, to assist gallbladder function and to help clear the mitochondrial membrane TL sites of unwanted waste/toxin compounds, as well as protocol to repair mitochondrial membranes (as mentioned above), which is in a sense a type of nutritional therapy.

### **Important Cofactors and Coenzymes in Mitochondrial Function:**

There are therefore a number of goals when in comes to assisting a return to normal mitochondrial function in the body, with a positive knock on effect on many other systems of the body. As mentioned above, these are increasing the efficiency of <u>ATP</u> conversion and distribution (i.e. actual energy release), speeding up the rate of recycling of ADP back to <u>ATP</u> again (i.e. energy recovery times and energy reserve), and also providing the body with enough raw materials to produce new <u>ATP</u> (i.e. replenishing depleted energy reserves - having converted some of the ADP to non-recoverable AMP in lieu of any **ATP** being available).

A large number of different metabolites and co-factors are used by the body in the Krebs (citric acid) cycle, a series of chemical reactions that enable glucose to be used for energy production within the cells of the body. Some of these are discussed below.

#### Krebs Cycle Organic Acids

- Citric acid
- (Isocitric acid)
- Alpha-Ketoglutaric acid (AKG) the conversion from AKG to Succinic acid involves the reconversion of ADP to ATP
- · Succinic acid
- Fumaric acid oxidised form of Succinic acid
- Malic acid
- (Oxaloacetic acid)
- Pyruvate precursor to Acetyl Coenzyme A, which combines with Oxaloacetic acid (above) to form Citric acid

Those organic acids that are not significant supplementally are shown above in brackets. There are a number of possible organic acids that may not be converting properly in the

Krebs Cycle, as can be seen above.

High levels of Citric acid and Malic acid are available in fruits (e.g. apples - rich in both) and fermented foods (e.g. Kombucha - rich in Malic acid). Malic acid supplements are available in acid form (i.e. Malic acid) or salt form (e.g. Magnesium Malate); and in combination products such as Ultra Muscleze. Malic acid is frequently shown to be low in CFS sufferers (together with Magnesium).

Alpha-Ketoglutarate (AK) is the salt form of the acid Alpha-Ketoglutaric Acid (AKG). KA supplements are often found in the form of Potassium Magnesium Alpha-Ketaglutarate (K-Mag KG).

Another is Pyruvic acid which is produced from carbohydrates and is a precursor to Co-Enzyme A. Typically however, malic acid is most commonly deficient of all these acids.

- Magnesium is particularly important in regulating and stabilising ATP, and is often displaced by the presence of heavy metals. With a deficiency of Magnesium, the ATP becomes over-regulated or inhibited, resulting in low energy levels. Magnesium helps to fire up various essential enzymatic reactions. Nutritional elements such as Magnesium, Zinc and Selenium have an important role in protecting the body from the effects of heavy metals, and those individuals with elevated heavy metal levels have a greater requirement for these nutrients (and a corresponding great need to detoxify the heavy metals from their bodies). Magnesium is best taken in Chelated form, e.g. Citrate or Glycinate, together with the amino acid Taurine to help carry it into the cells.
- <u>Active B1</u> Thiamine PyroPhosphate (TPP), a.k.a. Thiamine Diphosphate (TDP) or Cocarboxylase, is the biologically active form of <u>Vitamin B1</u>, Thiamine, and is used in the efficient burning carbohyrate and removing excess lactic acid (a cause of muscle ache).
- Active B2 Riboflavin, Vitamin B2, is also involved in the Krebs Cycle, as
  a cofactor in Complex I and II. There are two active, coenzyme forms of
  B2, Flavin Mononucleotide (FMN) and Flavin Dinucleotide (FAD). FMN is
  the coenzyme form of B2 found in supplement form.

#### **Nicotinamide**

• Active B3 - A variant of Vitamin B3 (Niacin), Nicotinamide nourishes the digestive and circulatory systems. known as Nicotinamide Adenine Dinucleotide plus high-energy Hydrogen, or NADH for short, is also involved in the Krebs cycle. Niacin can be obtained from protein but it is dependent on efficient protein digestion and amino acid conversion in the body. NADH and NAD are an essential part of the ATP to ADP conversion. Active B3 probably has most immediate noticeable effect on energy levels, but all B-vitamins are important to some degree in energy production. B3 levels can drop significantly if there is Peroxynitrite-related oxidative damage occurring in the body (to poly (ADP-ribose) polymerase enzyme DNA), having a knock on effect on the mitochondrial electron transport chain. Please see the Peroxynitrite page for more information.

- <u>Vitamin B5</u> Pantethene (a biologicaly active form of <u>Vitamin B5</u>) is involved in the synthesis of Coenzyme-A (CoA). CoA is important in energy metabolism for pyruvate to enter the tricarboxylic acid cycle(TCA cycle) as acetyl-CoA, and for alpha-ketoglutarate to be transformed to succinyl-CoA in the cycle.
- Active B6 Pyridoxal-5-Phosphate the active form of <u>Vitamin B6</u> can catalyze transamination reactions that are essential for providing amino acids as a substrate for gluconeogenesis. P-5-P is also a required coenzyme of the glycogen phosphorylase enzyme, that allows glycogenolysis to occur.
- Active B12 The enyzme Methylmalonyl Coenzyme A mutase (MUT) requires vitamin B12 in the form <u>Adenosylcobalamin (AdoB12)</u>. MUT is involved in carbohydrate metabolism, converting Methylmalonyl-CoA (MMI-CoA), the coenzyme A link form of methylmalonic acid (MMA), into Succinic-CoA (Su-CoA), the conenzyme A link form of succinic acid. It forms part of the <u>Krebs cycle</u> for the production of energy.
- Acetyl-L-Carnitine (ALC or ALCAR) is an amino acid that and allows proper transport and burning of excess fat and prevents mitochondria from shutting down. It is the acetylated ester of the amino acid L-Carnitine. Carnitine helps to transport ATP and ADP across the mitochondrial membranes, and to transport activated ATP to where it is actually used. It is manufactured intracellularly in the body from the Essential Amino Acids L-lysine and L-methionine, by a process of methylation i.e. does not float around in the blood stream (and thus urine) and hence is usually not included as a parameter in amino acid analyses.

However the quantities manufactured endogenously are relatively low, especially if there is a bottleneck in the production of carnitine (i.e. impaired methylation, insufficient quantities or precursor(s), impaired enzymatic function, lack of availability of **ATP** for these enzymes, low levels of coenzymatic cofactors, etc.) Otherwise the body relies on external dietary sources of carnitine to maintain sufficient levels. External sources are limited to meat, i.e. other animal's cells. Vegetarians or vegans who do not supplement carnitine will not be ingesting any carnitine and will be relying solely on endogenously produced carnitine to enable them to fulfill their energy requirements. Dietary (meat) sources of L-Carnitine may also not cross the blood brain barrier efficiently in some individuals, or digestion and absorption may be impaired in those with digestive difficulties. A lack of carnitine, for any of the above reasons, may adversely affect the body's ability to produce energy on demand. In such cases, supplementation with Acetyl-L-Carnitine (and eating red meat if possible) will help.

A powerful antioxidant and a mitochondrial cofactor (transporter of **ATP**). It helps to lower the mitochondrial phase transition (which leads to apoptotic cell death), thus playing a protective role in mitochondria. It also helps to transport fatty acids into the mitochondria. The inner mitochondrial membranes' cardiolipin are made up predominantly of fatty acids. (90+% Omega 6 fatty acids).

www.renegadeneurologist.com/?s=acetyl-l-carnitine

http://en.wikipedia.org/wiki/Carnitine

http://en.wikipedia.org/wiki/Acetyl-l-Carnitine

The properties of Carnitine in its different forms is vastly different, and only one type may be absorbable by an individual at any given time. **L**-**Carnitine** is notoriously difficult to cross the blood-brain barrier. The body may only be able to utilise one form of Carnitine most effectively at a given time, and throwing the wrong kinds at it or too many different kinds may not necessarily increase the efficiency or absorption, ability to cross the blood-brain barrier or utilisation.

Acetyl-L-Carnitine is the form most widely recognised as being most appropriate and efficient. The Acetyl group allows it to more easily cross the blood-brain barrier, and the Acetyl group is broken off to be used to create Acetylcholine (the essential neurotransmitter), leaving the L-Carnitine to be utilised by the brain's cells. ALC also enahnces the release of dopamine from neurons and helps it bind to dopamine receptor sites. Acetyl-L-Carnitine has been noted to increase levels of neurite production (i.e. projections from the cell body of neurons) in the brain. For this reason, it is best to avoid taking ALC supplements in the evening, as it may interfere with one's ability to sleep. ALC is also a poweful antioxidant.

Besides Acetyl-L-Carnitine described above, Carnitine is also found in alternative forms, such as:

- L-Carnitine Fumarate a blend of L-Carnitine and Fumaric acid, a Krebs cycle organic acid. The Biosint licensed forms are known as CarniShield and DuraCarn which are reputed to be highly bioavailable. It is used by a number of supplement manufacturers.
- Acetyl L-Carnitine Arginate diHCl (ALCA) this is Acetyl-L-Carnitine blended with the amino acid Arginine. This is reputed to be four times more effective in stimulating neurite production in the brain than Acetyl-L-Carnitine.
- Acetyl L-Carnitine Taurinate HCl (a blend of Acetyl-L-Carnitine, Taurine and HCl).
- Glycine Propionyl L-Carnitine (GPLC).

Jarrow Formulas' CarnitAll 600 contains all of the above 4 forms. Jarrow states that the first three forms of Carnitine are for Cardiovascular function, whereas the GPLC is claimed to be a 'muscle-specific form of Carnitine that enhances endothelial function and promotes maximum muscular energetics'.

Life Extension's product 'Optimized Carnitine with GlycoCarn' contains Acetyl-L-Carnitine HCl and Acetyl-L-Carnitine Arginate Di-HCl (trademark - ArginoCarn), and a patented form of GPLC called Glycine Propionyl L-Carnitine HCl, known as GlycoCarn. The product does not contain any L-

Carnitine Fumarate however. GlycoCarn is also available (without the other forms of Carnitine) in the Life Extensions product Peak <u>ATP</u>, discussed below.

I was muscle tested for different forms of Carnitine in April and June 2009, and neither GlycoCarn (GPLC HCl) nor L-Carnitine Fumarate were not wanted by the body, whereas (Jarrow Formulas) Acetyl-L-Carnitine was.

http://en.wikipedia.org/wiki/Carnitine

- L-Lysine is the precursor to L-Carnitine, and is the amino acid found in all proteins and enzymes in the body. Lysine is metabolised to make Coenzyme A, a necessary fuel of the Krebs cycle for carbohydrate metabolism and energy production. Lysine is also used to make Collagen, the connective tissue in humans and other mammals. A deficiency in Lysine will have a serious impact on enzyme and protein production, as well as energy levels, and often results in weight loss. Determine whether you are actually deficient in Lysine as supplementing Lysine may well boost your Carnitine levels intracellularly more effectively than supplementing Carnitine as above.
- <u>Lipoic Acid</u> is a cofactor in energy production, helping to regulate glucose metabolism. Lipoic Acid is active in all the tissues of the body and in its cellular compartments. Lipoic acid recycles both water and fat soluble antioxidant vitamins (e.g. vitamins C and E) and Coenzyme Q10, reducing them back to their non-oxidised original forms. Lipoic acid also improves sugar metabolism and energy production. It is a cofactor in the multienzyme complex that catalyzes the last stage of the process called glycolysis. Glycolysis is the first step in converting blood sugar (glucose), which is obtained from carbohydrates and proteins, into energy in a form that the body can use. This involves Magnesium, Active Vitamin B1 (TPP or TDP) and also Biotin, and so Lipoic Acid is best taken in conjunction with these other three supplements. Lipoic acid promotes the incorporation of cysteine into **glutathione** and combines synergistically with other **antioxidants** for greatly increased benefits. It is therefore frequently referred to as the ideal or universal antioxidant and free radical scavenger. It is also an excellent chelation agent. Please see the <u>Detox</u> page for more information.
- <u>Coenzyme Q10</u> and also <u>Copper</u>, <u>Sulphur and <u>Iron</u> are vital parts of the Electron Transport System (ETS) that harvest <u>ATP</u> energy from our daily food intake. Q10 helps to transport electrons from one molecule to another, hence allowing for energy production and transfer to take place. Beta blockers, tricyclic antidepressants and phenothiazines actually block CoQ10 production in the body and way worsen the situation. Coenzyme Q10 is naturally produced by the body by a process of <u>methylation</u>. If methylation is impaired in an individual, then Q10 levels may be quite low. Coenzyme Q10, aka <u>Ubiquinone</u>, is the most common form of supplemental Q10. For the non-vegetarians, heart is the most concentrated naturally occurring nutritional source of Q10. Coenzyme Q10 is defined on Wikepedia at the following link. <a href="http://en.wikipedia.org/wiki/Coenzyme\_Q10">http://en.wikipedia.org/wiki/Coenzyme\_Q10</a></u>

CoQmax CF is a trademarked product by XYMOGEN. It is a proprietary,

patent-pending, crystal-free form of CoQ10 that is reputed to offer very high levels of absorption and bioavailability. It utilises a proprietary monoglyceride carrier. Each capsule contains 50mg of CoQ10 (as ubiquinone). Clinical trials have shown it to be over 8 times more absorbable than powdered CoQ10 and more than twice as bioavailable as other oil-based or 'nano'-dispersed formulas. An example dosage in extreme cases might be 2 capsules, twice a day or more.

Another readily absorbable form of Ubiquinone (Q10) is Jarrow Formulas' Q-absorb Co-Q10. This uses a form of Q10 called Kaneka Q10 which is under licence from the Kaneka Corporation of Japan, and uses the 'ProSome' Formula. It comes in 100mg capsules. It is reputed to be absorbed 3-4 times better than ordinary chewable Co-Q10 tablets. The best elevation of blood plasma Q10 levels with this product have resulted in exercise being performed at the time of taking it, according to studies. Jarrow also offer other Ubiquinone products, such as Qabsorb Co-Q10 Plus and Q-absorb Co-Q10 Ultra. Both of these products contain the Citrus extract Limolene, which is reputed to help prevent crystallisation of Co-Q10 and thus aid absorption. The 'Plus' version contains 300mg of Limolene per capsule, in addition to 600mg Lecithin. Jarrow claims Q-Absorb Ultra has been clinically demonstrated in humans to raise plasma Co-Q10 levels by 500% (a six-fold increase over baseline Co-Q10 levels). Whether Limolene is used in CoQMax CF by Xymogen above is uncertain at this point in time. Limolene, it should be noted, is also an antimicrobial oil used to kill intestinal parasites (protozoa or nematodes).

Ubiquinol is a bio-activated (un-oxidised), trans-isomer form of Co-Enzyme Q10 (ubiquinone) and is reputed to be much more readily absorbed or utilised, in studies up to twice as effectively. It can be found in a number of Ubiquinol supplements and also in Premier Research Labs 'CoQ10-Quinol' product or Healthy Origins Ubiquinol. It is manufactured under license from the Kaneka Corporation in Japan, under the trademark 'Kaneka QH'.

In April and June 2009, I was muscle tested for various forms of CoQ10 (Ubiquinone), also including a Ubiquinol supplement, and CoQmax was the only brand/form of CoQ10 that was found to be agreeable to his body (at those times).

An alternative to Coenzyme Q10 is Idebenone. It is a synthetic alternative to Coenzyme Q10, an organic acid. It is reputed to have many of the same chemical properties to Coenzyme Q10. From my understanding, it is really an 'optional' supplement, to be taken in addition to Coenzyme Q10 rather than instead of it, or not at all. According to Dr Ward Dean MD, Idebenone does offer some unique benefits over CoQ10, including superior ability electron transport. In low cellular oxygen environments, Idebenone is more efficient at preventing free radical damage than CoQ10, whilst helping cells maintain relatively normal **ATP** levels (particularly useful to tissues that are rapidly damaged by insufficient tissue oxygenation such as the brain and the cardiac muscle - although are we talking about states where the patient is not breathing here? Rather than CFS?).

#### http://en.wikipedia.org/wiki/Idebenone

<u>Creatine</u> - Creatine is a nitrogenous organic acid that helps to provide cells with energy until the Krebs cycle can start to produce energy. PhosphoCreatine is converted into <u>ATP</u>. It fills in the gap energetically between the 0.8s of energy (<u>ATP</u>) that cells have stored at any one time, and the 1.4 seconds it takes for the Krebs Cycle in the mitochondria to start producing more energy.

#### http://en.wikipedia.org/wiki/Creatine

By converting PhosphoCreatine into <u>ATP</u> during the initial stages of exercise, the body is able to provide energy before the body starts burn food to generate <u>ATP</u>. This can help to reduce the amount of <u>ATP</u> that is used up initially before more can be generated. Creatine helps to reduce lactic acid build up in the muscles and helps mitochondria to function efficiently at the start of a work out, and may also help with brain functioning.

Unlike the Krebs Cycle nutrients, Creatine is not stored in the mitochondria, but is found locally in the cell. 95% of the body's Creatine is found in the skeletal muscles.

Half of the Creatine in the body is derived from dietary sources, the other half is produced internally. Creatine is found in meat and fish, and vegetarians and vegans tend to have lower levels of Creatine unless they supplement it. Creatine tends to be denatured with heat in cooking. Creatine is manufactured by the body by a process of methylation, from the amino acids Arginine, Glycine and Methionine. However, if methylation is impaired, which it frequently is in patients with CFS and related conditions, and dietary sources are insufficient, then levels may be guite low. Those who are low in Creatine may benefit from taking Creatine Monohydrate or Creatine Ethyl Ester prior to a work out, or first thing in the morning. Bodybuilders are known to take creatine during high intensity weigh training workouts in order to perform addition reps at their maximum weight in order to build the maximum muscle mass in a session, and also to provide additional energy for intensive cardiovascular exercise (e.g. treadmills or jogging). Creatine is osmotically active and excessive supplementation, and in weight training applications, will result in increased water retention, especially in the muscles. This may result in a bloated or larger, ripped appearance, and may add as much as 1-2kg to one's normal weight. It has also been observed that regular creatine intake may encourage the liver to produce less creatine naturally, and so if supplemental creatine is then stopped, then the person may feel tired for a number of weeks before enzymatic creatine synthesis returns to normal to redress the balance. There are no known harmful effects from taking creatine, despite fears to the contrary. Creatine supplementation (and dosage recommendations) however should probably be based on ascertaining one's natural creatine levels and whether they require supplementation or not; and indeed whether the body reacts well to it using muscle testing, or other means, like any other form of supplementation. www.pponline.co.uk/encyc/0164.htm

- N-Acetyl Cysteine (NAC) is a powerful antioxidant and useful glutathione precursor, as has been discussed on the Nutritional page and Inefficiency Liver Function and Detox pages. It also promotes oxidative phosphorylation, key elements of the respiratory chain, mitochondrial membrane integrity and mitochondrial homeostasis, thereboy delaying apoptosis (Programmed Cell Death). L-Cysteine may also be supplemented instead, however the Acetyl group may make it more absorbable in the brain. Supplementing directly with Glutathione is also an alternative option. In addition, Superoxide Dismutase (SOD) enzyme production is probably even more important that Glutathione in terms of ability to assist in respiration, protecting against superoxide damage.
- <u>Guanosine Triphosphate (GTP)</u> is a purine nucleotide is produced by a
  Krebs cycle enzyme in the process of producing <u>ATP</u>. It consists of a
  Guanosine molecule attached to a Ribose ring. Increasing one's purine
  intake (within limits as one does not want Gout) may assist increasing
  <u>ATP</u> levels.
- Copper, Zinc, Manganese and Iron SOD enzymes are bound to a
  metal element, copper, zinc, manganese or iron, and therefore it is
  shrewd to maintain adequate levels of these mineral elements in one's
  diet or supplement regime (as mentioned above), and to ensure one's
  levels do not drop below normal (according to relevant test results).
- <u>Chromium (III)</u> is a trace element that enhances the action of insulin (involved with carbohydrate, fat and protein metabolism). Chromium is a trace element and only small amounts are required. It should only be supplemented if levels are low, as excessive supplementation may lead to detrimental effects. <a href="http://ods.od.nih.gov/factsheets/chromium.asp">http://ods.od.nih.gov/factsheets/chromium.asp</a>
- Vanadium is believed to be involved with glucose metabolism (enhancing, Na/K transport, adrenal catecholamine metabolism (oxidation), and inhibiting cholesterol synthesis (lowering total and LDL (bad) cholesterol levels). Vanadium is a trace element and only small amounts are required. It should only be supplemented if levels are low, as excessive supplementation may lead to detrimental effects such as decreased energy production.

www.umm.edu/altmed/articles/vanadium-000330.htm

Some of these cofactors are summarised and discussed in the article below by Ward Dean MD, entitled 'Restoring Mitochondrial Function and Bio-Energetics'. The mitochondria inner/outer membrane diagram above is also found here.

#### http://www.vrp.com/articleprinter.aspx?a=912

As one can see, there are a large number of bio-chemical compounds involved in energy production in the body. There are many more that have not been discussed here, including various organic acids and hormones. Any number of these may be deficient in the body in a sufferer of CFS or related conditions, and are required in the correct quantities and correct relative ratios for optimum metabolic function. Knowing exactly what is required and in what quantity is critical, and it is recommended to consult with your naturopath to address this area, rather than simply take every expensive supplement that is all the rage on the internet, as this approach is unlikely to be

completely effective. For example, you are unlikely to notice any benefit in taking high doses of Ubiquinol (CoQ10) if your levels of CoQ10 are actually satisfactory. The main effect will the antioxidant effect - and there are cheaper and more effective **antioxidants** - but mainly the effect on your wallet. Identifying what is actually in dire need by the body is therefore of paramount importance. It is tempting to simply take a long list of the above supplements expecting them to work, but one must consider what cofactors, minerals, vitamins and amino acids are actually deficient, and tackle those, rather than the arbitrary list above. Indeed, the actual problem may not be what you expect and may well be less obvious, relating to other krebs metabolites, dysbiosis, other amino acids being deficient, low hormone and neurotransmitter levels, amongst other things. A 'holistic' approach is therefore required to get to the bottom of why mitochondrial function is poor.

There are a number of combination mitochondrial formulas available by reputable suppliers, and many contain more than one of the cofactors and coenzymes listed above.

One can consider mitochondrial support and supplementation in two senses, for regular supplementation, usually twice or three times a day, and also ad hoc supplementation, when one's mitochondrial function is especially poor, i.e. dips during the day particularly when one has overdone things. There are various symptoms for this, which may vary from individual to individual, according to the exact mitochondrial and endocrine/neurotransmitter imbalance pattern. However, for 'emergency' or ad hoc supplementation, I have found personally that a combination of Acetyl-L-Carnitine (or whichever Carnitine works for you), Coenzyme Q10, and Active B3 help to relieve such symptoms most effectively. If cardiac symptoms have also arisen, then the above will also help to support more efficient energy production to the heart, but one should then also consider taking (additional) Hawthorn. This is described on the <u>Cardiac Insufficiency</u> page. If you are especially worn out in a mitochondrial sense, rather than sleep cycle sense, late in the evening, it may well be a good idea to take some extra ad hoc mitochondrial supplements so that the body can function properly, and thus allow you to fall asleep. Otherwise you may find your biochemistry is too chaotic to promote the correct neurotransmitter production pathways for sleep. Try experimenting if this is something that affects you to see what works best for you.

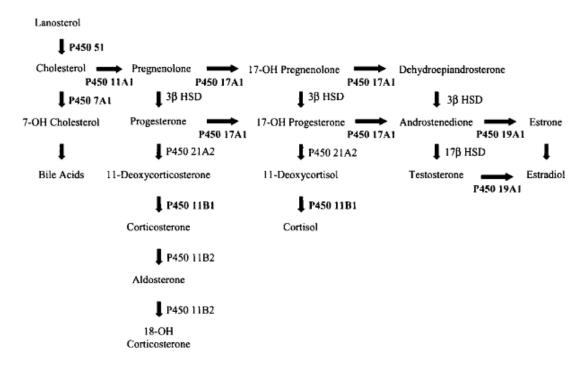
# Cytochrome P450

### **Background Information on P450**

Since the introduction of modern molecular biology techniques allowing for the sequencing of entire genomes, researchers have identified and continue to discover many P450 enzymes in a wide variety of organisms. In order to systematically identify and categorize this growing family of enzymes, a leading group of researchers in the field

established the current system of nomenclature (Nelson et al., 1996). P450s are named with the prefix CYP or P450 followed by an Arabic number defining the family, an uppercase letter defining the subfamily, and another Arabic number defining an individual enzyme (e.g., P450 3A4). In cases where only a single member exists in a given family, it may be identified simply as P450 followed only by the family number (e.g., P450 51). The reader may refer to the cytochrome P450 home page

(http://dmelson.utmem.edu/CytochromeP450.html) for the latest nomenclature updates and links to related sites.



**Figure 1.** Cholesterol, bile acid, and steroid hormone biosynthesis: phosphorylated P450 enzymes are indicated in boldface.

All mammalian tissues examined express some P450 enzyme system (Porter and Coon, 1991). In addition, mammals express multiple enzymes simultaneously in a variety of tissues, including liver, kidney, lung, and adrenal (Bhagwat et al., 1999a; Guengerich, 2001; Lohr et al., 1998; Parker and Schimmer, 1997). It is also appreciated that various enzymes are found not only in different cell and tissue types, but also in different subcellular compartments, such as the outer nuclear membrane, endoplasmic reticulum (ER), mitochondria, golgi, peroxisome, and plasma membrane. Certain enzymes are found in several different subcellular compartments simultaneously (Guengerich, 2001).

All cytochrome P450s, with the exception of bacterial enzymes, are membrane bound. Microsomal enzymes are tethered to the membrane through a hydrophobic transmembrane helix at the N-terminus of the protein, which also serves as a targeting sequence for the signal recognition particle dependent cotranslational incorporation of a nascent P450 into the ER membrane (Bar-Nun et al., 1980; Sakaguchi et al., 1984). Insertion continues until a halt-transfer signal is reached that effectively anchors the P450 to the membrane with the bulk of the protein exposed on the cytosolic side (Monier et al., 1988; Sakaguchi et al., 1987; Szczesna-Skorupa and Kemper, 1989; Szczesna-Skorupa et al., 1988). Because detergents are required for solubilizing membrane-bound P450s, all native mammalian P450s have evaded crystallization and x-ray crystallographic study. To avoid this problem, researchers have modified several P450s resulting in soluble mutants that can be crystallized and that have structures that were solved. The first mammalian P450 structure reported was that of rabbit P450 2C5 (Williams et al., 2000), which was also crystallized in complexes with two different substrates (Wester et al., 2003a,b). Interestingly, one of the two substrates binds in two different orientations (Wester et al., 2003a). These studies of P450 2C5 offer insightful information on the fiexibility of the enzyme and shed light on the ability of P450s to accommodate a variety of substrates with different shapes and sizes. The structure of human P450 2C8 was recently reported, revealing an active site volume twice that of P450 2C5, consistent with the size of its preferred substrates (Schoch et al., 2004). Human P450 2C9 has also been crystallized, with and without bound warfarin, a known

anticoagulant substrate (Williams et al., 2003). The structure reveals a new binding pocket that may accommodate several substrate molecules simultaneously and possibly

#### **REGULATION OF CYTOCHROME P450 381**

account for some complex drug-drug interactions. Another group has also reported a structure for P450 2C9 complexed with flurbiprofen (Wester et al., 2004), The structure was obtained without modifications to the catalytic domain, revealing some significant conformational differences. The more recent structure helps explain some experimental observations in terms of the substrate selectivity of the enzyme, which were not easily explained with the earlier structure, A crystal structure for rabbit P450 2B4, one of the first P450s to be purified and studied in detail, has been reported in a wide open conformation (Scott et al., 2003) and in a closed form complexed with the specific inhibitor 4-(4-chlorophenyl)-imidazole (Scott et al., 2004), These structures may help to understand how mammalian P450s "open/close," allowing substrate to access the buried active site (Poulos, 2003), Two groups have recently reported crystal structures for human P450 3A4: one unliganded structure from each group and two structures hound to different substrates (Williams et al. 2004; Yano et al., 2004a). The collection of structures should provide a better understanding of the substrate selectivity and unusual kinetics of this important enzyme. Finally, a structure for P450 2A6 has been reported in a meeting abstract (Yano et al., 2004b),

Humans express 57 putatively functional genes and 58 pseudogenes (Nelson et al., 2004), These may be divided roughly into two groups based on their substrate specificity, P450s involved in the metabolism of most drugs and carcinogens are derived from families 1-3, This group demonstrates wide substrate specificity, with certain enzymes acting on a large number of structurally varied substrates such as in the case of human P450 3A4.

The second group demonstrates high substrate specificity, catalyzing the biosynthesis and metabolism of endogenous substrates including cholesterol, steroids, vitamins, and eicosanoids. Several steroidogenic P450 enzymes are outlined in Fig, 1,P450s fall into three classes hased on the reduction system transferring electrons.

Class I P450s are associated with the inner mitochondrial memhrane and some hacterial systems. Reducing equivalents from NADPH or NADH are transferred two electrons at a time to redoxin reductase, which carries a flavin adenine dinucleotide (FAD) prosthetic group. The isoalloxazine ring of FAD may exist in several oxidation states, which allows for the subsequent transfer of electrons individually to a mobile Fe2S2-containing protein called redoxin. Reduced redoxin is thought to shuttle electrons to the P450, returning to the reductase in the oxidized form for additional cycles.

Class II P450s are those that reside in the ER and receive reducing equivalents from NADPH via P450 reductase or cytochrome  $b^{\wedge}$  (a heme protein associated with the ER), P450 reductase is a membrane-bound protein containing a FAD and a flavin mononucleotide (FMN) prosthetic group that also has an isoalloxazine ring with properties similar to FAD, An electron pair from NADPH is received by FAD, which relays the electrons to FMN, finally transferring electrons in single file to the P450, In certain reactions, the first electron is transferred from P450 reductase, while the second electron is transferred from cytochrome b<sup>^</sup>. Cytochrome b<sup>^</sup> may be reduced either by P450 reductase. or cytochrome  $b^{\wedge}$  reductase. Interestingly, apo- $^{\wedge}$ s (cytochrome  $^{\wedge}$ 5 devoid of heme), which cannot transfer electrons, is required for optimal activity in a number of P450 enzymes and only with certain substrates (Hlavica and Lewis, 2001; Yamazaki et al., 2001), Class III P450s are isomerases instead of monooxygenases. They do not require the participation of any redox partners or donors of reducing equivalents. Water is not produced, and therefore, molecular oxygen is not required for catalysis. Substrates of class III P450s are simply rearranged into products. Examples of P450s from this class include P450 8A1 [prostacyclin [PGI2] synthase] and P450 5 [thromboxane [TXA2] synthase].

#### POSTTRANSLATIOIMAL MODIFICATION

A posttranslational modification may be defined as "any difference between a functional protein and the linear polypeptide sequence encoded between the initiation and the termination codons of its structural gene" (Han and Martinage, 1992), Examples of noncovalent modifications include incorporation of cofactors such as heme, protein folding, and the association of subunits to form an oligomeric protein, Allosteric phenomena manifested as deviations from Michaelis-Menten kinetics have been demonstrated for numerous P450 enzymes. Various components known to interact with P450, including substrates, inhibitors, membrane lipids, and redox partners (such as the previously mentioned cytochrome ^5), have been shown to act as homotropic and heterotropic effectors (Hlavica and Lewis, 2001), P450 2E1 is stabilized by ethanol, leading to increased cellular levels of the P450 (Roberts et al., 1995), Covalent modifications, including cleavage of a signal peptide, formation of disulfide bonds, and an array of modifications to amino acid residues, including phosphorylation, nitration, glycosylation, methylation, sulfation, acetylation, and prenylation, provide another means of posttranslational modification. The remainder of the present article is dedicated to the identification and characterization of covalent P450 posttranslational modifications with an emphasis placed on describing the physiological role of each modification.

The first step in the conversion of cholesterol to steroids is the synthesis of pregnenolone. The reaction involves three separate hydroxylation steps, with three equivalents of O2 and NADPH. The enzyme that catalyzes this reaction (P450 llAl) is expressed in all tissues that synthesize steroids from cholesterol. P450 11 Al resides in the inner mitochondrial membrane. Transcriptional regulation of P450 llAl and other steroidogenic P450s is well documented (Parker and Schimmer, 1993, 1996, 1997; Stocco, 2000). Activity of P450 11 Al is also regulated by phosphorylation. Initial evidence for stimulation of P450 llAl activity by phosphorylation was reported several years ago (Caron et al., 1975). A crude preparation of P450 llAl was isolated from bovine corpus luteum mitochondria. Limiting P450 was reconstituted with excess redoxin and redoxin reductase, and the reconstituted system was subjected to treatment with PKA, cAMP, and ATP. The resulting system demonstrated increased P450 llAl activity. Decisive evidence for the posttranslational phosphorylation of P450 llAl came nearly a decade later (Vilgrain et al., 1984). A demonstration with purified bovine adrenocortical P450 llAl showed that the P450 is efficiently phosphorylated by Ca^"\^-activated phospholipid-sensitive protein kinase C (PKC). Four moles of phosphate were incorporated per mole of P450 11 Al, with serine and threonine as the target amino acids phosphorylated in a ratio of 1:1 as revealed by amino acid analysis. Interestingly, PKC activity is also found to be associated with bovine adrenocortical inner mitochondrial membrane (Vilgrain et al., 1984). Thus, it appears that phosphorylation of P450 llAl in a reconstituted system results in increased enzyme activity. Additional experiments would be required to demonstrate the physiological relevance of the phenomenon.

Reports in recent years have demonstrated the neuromodulatory effects of estrogenic metabolites [referenced in Balthazart et al. (2001a)]. A telling example comes from a study with castrated sexually experienced male rats that illustrates that estrogen administration rapidly activates male sexual behavior (within minutes), presumably by a nongenomic mechanism, as a genomic mechanism would require hours to days for an observable effect (Cross and Roselli, 1999). Rapid and pronounced changes in P450 19A1 activity of quail hypothalamic homogenates by pharmacological studies employing kinase activators and inhibitors have been described (Balthazart et al., 2001a,b). Stimulation of kinase by addition of nonnal intracellular concentrations of

Ca^"^, ATP, and Mg^"^ resulted in significant decreases in P450 19A1 activity. The decrease in activity could be completely abolished by the addition of ethylene glycolfc «(P-aminoethyl ether)-N,N,N',N'-tetraacetic acid (EGTA), which chelates free Ca^"^. The authors noted, however, that the studies were performed with total homogenates, thus the possibility remained that intermediary proteins are phosphorylated that interact with P450 19A1 to modulate its activity. Conclusive experiments confirming P450 19A1 phosphorylation have been reported (Balthazart et al, 2003). Aromatase from quail preoptic area homogenates was immunopurified, and phosphorylated Ser, Thr, and Tyr residues were detected by Western analysis employing phospho-amino acid specific antibodies. Together, these results demonstrate that the local production of estrogens in quail brain can be rapidly altered by calcium-dependent P450 19A1 phosphorylation. P450 19A1 activity is also a key factor in the progression of estrogen-dependent diseases such as breast, endometrial, and ovarian cancers. Like P450 17A1, P450 19A1 represents an important target in the treatment of such diseases.

### Vitamin D and Calcium Regulation

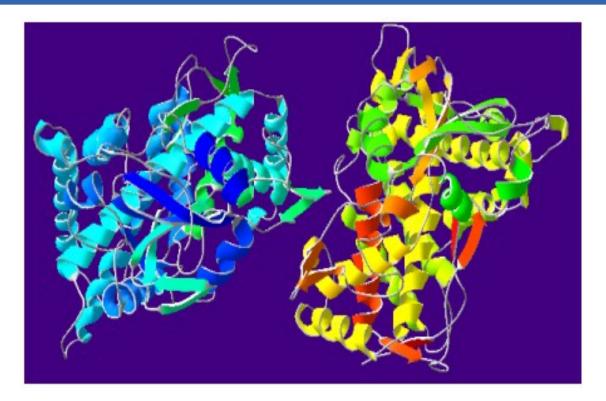
Vitamin D is a naturally occurring hormone involved in the regulation of calcium and phosphorus metabolism, affecting bone development. Requirements for vitamin D may be met by dietary intake or through biosynthesis. The first step in vitamin D biosynthesis is the photoactivation (by exposure of skin to sunlight) of 7-dehydrocholesterol to vitamin D3 by a nonenzymatic process. Hepatic vitamin D3 25-hydroxylase (P450 27A1) then acts on vitamin D3 to produce 25-hydroxycholecalciferol (25[OH]D3).

Further metabolism of vitamin D involves two renal mitochondrial P450 enzymes. The active form of vitamin D (la,25-dihydroxyvitamin D3) is preferentially formed under conditions of calcium deficiency. Biosynthesis of la,25-dihydroxyvitamin D3 is catalyzed by renal la-hydroxylase (P450 27B1). A second renal enzyme (P450 24) catalyzes the 24-hydroxylation of 25(OH)D3 as well as 1,25(OH)2D3. Vitamin D 24-hydroxylation results in increased susceptibility of the homione to oxidation and side-chain cleavage, providing a pathway for the degradation of the hormone.

When circulating levels of calcium become low, parathyroid hormone secretion is increased. This stimulates an increase in the biosynthesis of [1,25[OH]2D], which together with parathyroid hormone, signals the gastrointestinal absorption of calcium until bone requirements for calcium are met and circulating calcium levels return to

#### **REGULATION OF CYTOCHROME P450 391**

normal. This closes the endocrine loop by a feedback mechanism decreasing parathyroid hormone secretion (Hendy, 1997; Narbaitz et al., 1981). The mechanisms by which parathyroid hormone stimulates the biosynthesis of [1,25[OH]2D] are coming into focus. The emerging picture suggests a role for the reversible phosphorylation of ferredoxin.



The **cytochrome P450** superfamily (officially abbreviated as **CYP**) is a large and diverse group of <u>enzymes</u> that catalyze the <u>oxidation</u> of <u>organic substances</u>. The <u>substrates</u> of CYP enzymes include <u>metabolic</u> intermediates such as <u>lipids</u> and <u>steroidal</u> hormones, as well as <u>xenobiotic</u> substances such as drugs and other <u>toxic</u> chemicals. CYPs are the major enzymes involved in <u>drug metabolism</u> and bioactivation, accounting for about 75% of the total number of different metabolic reactions. [1]

The most common reaction catalyzed by cytochromes P450 is a <u>monooxygenase</u> reaction, e.g., insertion of one atom of oxygen into an organic substrate (RH) while the other oxygen atom is <u>reduced</u> to water:

$$RH + O_2 + NADPH + H^+ \rightarrow ROH + H_2O + NADP^+$$

Cytochromes P450 (CYPs) belong to the superfamily of proteins containing a <a href="hemograteins">hemograteins</a> CYPs use a variety of small and largemolecules as <a href="substrates">substrates</a> in enzymatic reactions. Often, they form part of multi-component <a href="electron transfer chains">electron transfer chains</a>, called <a href="P450-containing systems">P450-containing systems</a>

<u>P450-containing systems</u>. The letter in *P450*represents the word pigment as these enzymes are red because of their heme group. The number 450 reflects wavelength of the absorption maximum of the enzyme when it is in the reduced state and complexed with <u>CO</u>.

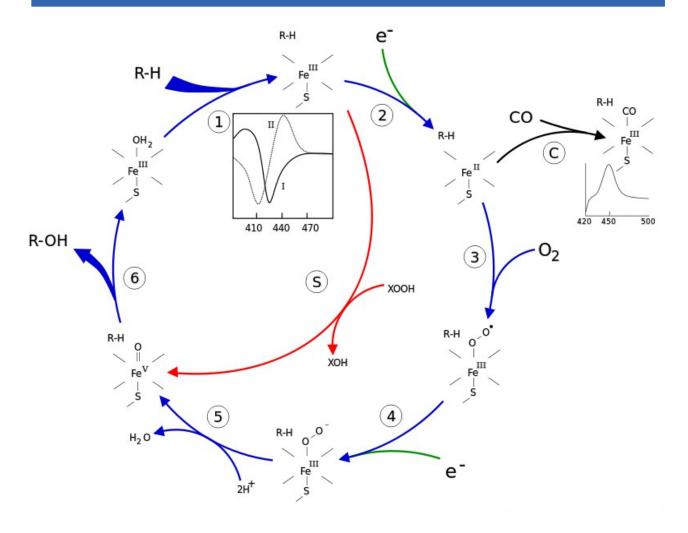
CYP <u>enzymes</u> have been identified in all <u>domains</u> of life, i.e., in <u>animals</u>, <u>plants</u>, <u>fungi</u>, <u>protists</u>, <u>bacteria</u>, <u>archaea</u>, and even <u>viruses</u>; although it is not found in <u>E. coli.[2][3]</u>More than 11,500 distinct CYP proteins are known.<sup>[4]</sup>

Most CYPs require a protein partner to deliver one or more electrons to reduce the <u>iron</u> (and eventually molecular oxygen). Based on the nature of the electron transfer proteins CYPs can be classified into several groups:<sup>[5]</sup>

- Microsomal P450 systems in which electrons are transferred from NADPH via <u>cytochrome</u>
   P450 reductase (variously CPR, POR, or CYPOR). <u>Cytochrome b5</u> (cyb5) can also contribute reducing power to this system after being reduced by <u>cytochrome b5 reductase</u> (CYB5R).
- Mitochondrial P450 systems, that employ adrenodoxin reductase and adrenodoxin to transfer electrons from NADPH to P450.
- Bacterial P450 systems, that employ a ferredoxin reductase and a ferredoxin to transfer electrons to P450.
- CYB5R/cyb5/P450 systems in which both electrons required by the CYP come from cytochrome b5.
- FMN/Fd/P450 systems originally found in Rhodococcus sp. in which a FMN-domaincontaining reductase is fused to the CYP.
- P450 only systems, which do not require external reducing power. Notable ones
  include <u>CYP5</u> (<u>thromboxane synthase</u>), <u>CYP8</u> (<u>prostacyclin synthase</u>), and CYP74A (<u>allene oxide synthase</u>).

Genes encoding CYP enzymes, and the enzymes themselves, are designated with the abbreviation CYP, followed by a number indicating the gene family, a capital letter indicating the subfamily, and another numeral for the individual gene. The convention is to *italicise* the name when referring to the gene. For example, CYP2E1 is the gene that encodes the enzyme CYP2E1 – one of the enzymes involved in paracetamol(acetaminophen) metabolism. The CYP nomenclature is the official naming convention, although occasionally (and incorrectly) CYP450 or CYP<sub>450</sub> is used. However, some gene or enzyme names for CYPs may differ from this nomenclature, denoting the catalytic activity and the name of the compound used as substrate. Examples include CYP5A1, thromboxane  $A_2$  synthase, abbreviated to CYP5A1 (ThromBoXane CYP5A1), and CYP51A1, lanosterol 14-α-demethylase, sometimes unofficially abbreviated to LDM according to its substrate (Lanosterol) and activity (DeMethylation).

The current nomenclature guidelines suggest that members of new CYP families share >40% amino acid identity, while members of subfamilies must share >55% amino acid identity. There are nomenclature committees that assign and track both base gene names (Cytochrome P450 Homepage) and allele names (CYP Allele Nomenclature Committee



The active site of cytochrome P450 contains a <u>heme **iron**</u> center. The **iron** is tethered to the P450 protein via a <u>thiolate</u> ligand derived from a <u>cysteine</u> residue. This cysteine and several flanking residues are highly conserved in known CYPs and have the formal <u>PROSITE</u> signature consensus pattern [FW] - [SGNH] - x - [GD] - {F} - [RKHPT] - {P} - C - [LIVMFAP] - [GAD]. Because of the vast variety of reactions catalyzed by CYPs, the activities and properties of the many CYPs differ in many aspects. In general, the P450 catalytic cycle proceeds as follows:

1. The substrate binds to the active site of the enzyme, in close proximity to the heme group, on the side opposite to the peptide chain. The bound substrate induces a change in the conformation of the active site, often displacing a water molecule from the distal axial coordination position of the heme iron, and sometimes changing the state of the heme iron from low-spin to high-spin. This gives rise to a change in the spectral properties of the enzyme, with an increase in absorbance at 390 nm and a decrease at 420 nm. This can be measured by difference spectrometry and is referred to as the "type I" difference spectrum (see inset graph in figure). Some substrates cause an opposite change in spectral properties, a "reverse type I" spectrum, by processes that are as yet unclear. Inhibitors and certain substrates that bind directly to the heme iron give rise to the type II difference spectrum, with a maximum at 430 nm and a minimum at 390 nm (see inset graph in figure). If no reducing equivalents are available, this complex may remain stable, allowing the degree of binding to be determined from absorbance measurements *in vitro*[10]

- 2. The change in the electronic state of the active site favors the transfer of an electron from NAD(P)H via <u>cytochrome P450 reductase</u> or another associated<u>reductase<sup>[11]</sup></u> This takes place by way of the electron transfer chain, as described above, reducing the ferric heme **iron** to the ferrous state.
- 3. Molecular oxygen binds covalently to the distal axial coordination position of the heme iron. The cysteine ligand is a better electron donor than histidine, which is normally found in heme-containing proteins. As a consequence, the oxygen is activated to a greater extent than in other heme proteins. However, this sometimes allows the iron-oxygen bond to dissociate, causing the so-called "uncoupling reaction", which releases a reactive superoxide radical and interrupts the catalytic cycle.
- 4. A second electron is transferred via the electron-transport system, from either <u>cytochrome P450</u> <u>reductase</u>, <u>ferredoxins</u>, or <u>cytochrome b5</u>, reducing the dioxygen adduct to a negatively charged peroxo group. This is a short-lived intermediate state.

The peroxo group formed in step 4 is rapidly protonated twice by local transfer from water or from surrounding amino-acid side-chains, releasing one water molecule, and forming a highly reactive species commonly referred to as **P450 Compound 1** ( or Compound I). This highly-reactive intermediate was not "seen in action" until 2010, [12] although it had been studied theoretically for many years. [8] P450

- 1. Compound 1 is most likely a iron(IV)oxo (or <u>ferryl</u>) species with an additional oxidizing equivalent <u>delocalized</u> over the <u>porphyrin</u> and thiolate ligands. Evidence for the alternative perferryl <u>iron(V)-oxo</u> [8] is lacking. [12]
- Depending on the substrate and enzyme involved, P450 enzymes can catalyze any of a
  wide variety of reactions. A hypothetical hydroxylation is shown in this illustration. After the
  product has been released from the active site, the enzyme returns to its original state, with
  a water molecule returning to occupy the distal coordination position of the <u>iron</u> nucleus.

S: An alternative route for mono-oxygenation is via the "peroxide shunt": Interaction with single-oxygen donors such as peroxides and hypochlorites can lead directly to the formation of the iron-oxo intermediate, allowing the catalytic cycle to be completed without going through steps 2, 3, 4, and 5. [10] A hypothetical peroxide "XOOH" is shown in the diagram.

C: If carbon monoxide (CO) binds to reduced P450, the catalytic cycle is interrupted. This reaction yields the classic CO difference spectrum with a maximum at 450 nm.

### P450s in humans

Human CYPs are primarily membrane-associated <u>proteins<sup>[13]</sup></u> located either in the inner membrane of <u>mitochondria</u> or in the <u>endoplasmic reticulum</u> of cells. CYPs metabolize thousands of <u>endogenous</u> and <u>exogenous</u>chemicals. Some CYPs metabolize only one (or a very few) substrates, such as *CYP19* (<u>aromatase</u>), while others may metabolize multiple <u>substrates</u>. Both of these characteristics account for their central importance in <u>medicine</u>. Cytochrome P450 enzymes are present in most tissues of the body, and play important roles in <u>hormone</u> synthesis and breakdown (including <u>estrogen</u> and <u>testosterone</u> synthesis and metabolism), <u>cholesterol</u> synthesis, and <u>vitamin D</u> metabolism. Cytochrome P450 enzymes also function to metabolize potentially toxic

compounds, including <u>drugs</u> and products of endogenous metabolism such as <u>bilirubin</u>, principally in the liver.

The <u>Human Genome Project</u> has identified 57 human <u>genes</u> coding for the various cytochrome P450 enzymes. [14]

CYPs are the major enzymes involved in <u>drug metabolism</u>, accounting for about 75% of the total metabolism. [1] Most drugs undergo deactivation by CYPs, either directly or by facilitated <u>excretion</u> from the body. Also, many substances are <u>bioactivated</u> by CYPs to form their active compounds.

### **Drug interaction**

Many drugs may increase or decrease the activity of various CYP isozymes either by inducing the biosynthesis of an isozyme (enzyme induction) or by directly inhibiting the activity of the CYP (enzyme inhibition). This is a major source of adverse drug interactions, since changes in CYP enzyme activity may affect the metabolism and clearance of various drugs. For example, if one drug inhibits the CYP-mediated metabolism of another drug, the second drug may accumulate within the body to toxic levels. Hence, these drug interactions may necessitate dosage adjustments or choosing drugs that do not interact with the CYP system. Such drug interactions are especially important to take into account when using drugs of vital importance to the patient, drugs with important side-effects and drugs with small therapeutic windows, but any drug may be subject to an altered plasma concentration due to altered drug metabolism.

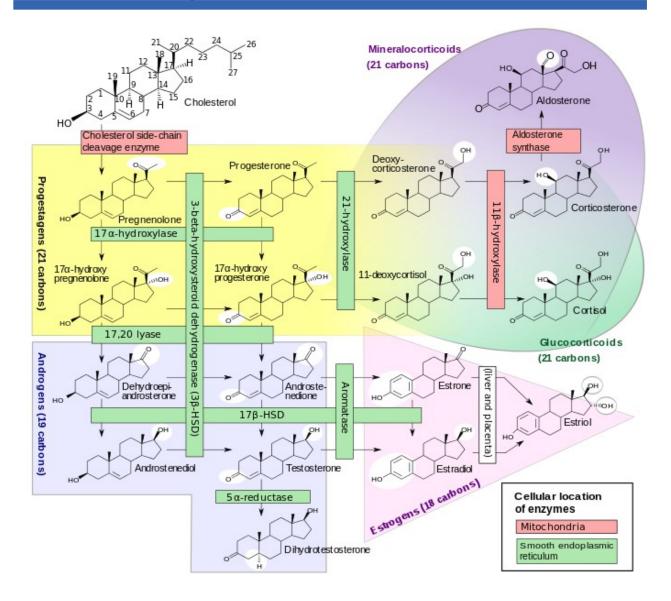
A classical example includes <u>anti-epileptic drugs</u>. <u>Phenytoin</u>, for example, induces <u>CYP1A2</u>, <u>CYP2C9</u>, <u>CYP2C19</u>, and <u>CYP3A4</u>. Substrates for the latter may be drugs with critical dosage, like <u>amiodarone</u> or <u>carbamazepine</u>, whose <u>blood plasma concentration</u> may either increase because of enzyme inhibition in the former, or decrease because of enzyme induction in the latter. <u>[citation needed]</u>

#### Interaction of other substances

Naturally occurring compounds may also induce or inhibit CYP activity. For example, <u>bioactive</u> compounds found in <u>grapefruit juice</u> and some other fruit juices, including <u>bergamottin</u>, <u>dihydroxybergamottin</u>, and <u>paradicin-A</u>, have been found to inhibit CYP3A4-mediated metabolism of <u>certain medications</u>, leading to increased <u>bioavailability</u> and, thus, the strong possibility of <u>overdosing</u>. [16] Because of this risk, avoiding grapefruit juice and fresh grapefruits entirely while on drugs is usually advised. [17]

#### Other examples:

- <u>Saint-John's wort</u>, a common <u>herbal remedy induces CYP3A4</u>, but also inhibits <u>CYP1A1</u>, <u>CYP1B1</u>, <u>CYP2D6</u>, and <u>CYP3A4</u>.[18][19]
- <u>Tobacco smoking</u> induces <u>CYP1A2</u> (example CYP1A2 substrates are <u>clozapine</u>, <u>olanzapine</u>, and <u>fluvoxamine</u>)<sup>[20]</sup>
- At relatively high concentrations, <u>starfruit</u> juice has also been shown to inhibit <u>CYP2A6</u> and other CYPs.<sup>[21]</sup> <u>Watercress</u> is also a known inhibitor of the Cytochrome P450 <u>CYP2E1</u>, which may result in altered drug metabolism for individuals on certain medications (ex., <u>chlorzoxazone</u>).<sup>[22]</sup>



<u>Steroidogenesis</u>, showing many of the enzyme activities that are performed by cytochrome P450 enzymes. HSD: Hydroxysteroid dehydrogenase

A subset of cytochrome P450 enzymes play important roles in the synthesis of <u>steroid</u> <u>hormones</u> (<u>steroidogenesis</u>) by the <u>adrenals</u>, <u>gonads</u>, and peripheral tissue:

- <u>CYP11A1</u> (also known as P450scc or P450c11a1) in adrenal <u>mitochondria</u> effects "the activity formerly known as 20,22-desmolase" (steroid 20α-hydroxylase, steroid 22-hydroxylase, cholesterol <u>sidechain</u> scission).
- <u>CYP11B1</u> (encoding the protein P450c11β) found in the <u>inner mitochondrial</u>
   <u>membrane</u> of <u>adrenal cortex</u> has steroid 11β-hydroxylase, steroid <u>18-hydroxylase</u>, and steroid 18 methyloxidase activities.
- <u>CYP11B2</u> (encoding the protein P450c11AS), found only in the mitochondria of the adrenal <u>zona glomerulosa</u>, has steroid 11β-hydroxylase, steroid 18-hydroxylase, and steroid 18-methyloxidase activities.
- <u>CYP17A1</u>, in endoplasmic reticulum of adrenal cortex has steroid 17α-hydroxylase and 17,20-lyase activities.
- CYP21A1 (P450c21) in adrenal cortex conducts <u>21-hydroxylase</u> activity.
- <u>CYP19A</u> (P450arom, <u>aromatase</u>) in <u>endoplasmic reticulum</u> of <u>gonads</u>, <u>brain</u>, <u>adipose tissue</u>, and elsewhere catalyzes aromatization of <u>androgens</u> to <u>estrogens</u>.

### **Other Parasites**

#### **Protozoan**

Alinia does not tear up the gut. I take 500 mg a day for 9 days in a row. Im going up to 1500 mg/D for Ascaris. Kills lesser worms easily.

Tinidazole is like Mebendazole, kills good gut bacteria, and protozoans.

Stool tests useless, PCR shut off years ago.

ELISA tests work, you need a spectrum of them, with Ascaris 209 KDA, and Fluke 13 KDA being the dominant Enzymes.

When you get to sub species, it gets harder to id the exact Antigens, but if you test positive for 209, and `13, rest assured you have round and flat worm infections. Treat the flat first, round second, not round first, this is a sequence that leads to disaster.

### **Leishmaniasis**

Zinc Sulfate

Sulfatrim

Mittlefosine

## **Strongyloides**

Merck Manual Wikipedia antimicrobe Papua New Guinea Medical Journal http://www.ards.com.au/StrongFront.pdf.

### **Centre for Disease Control in your region**

Alice Springs 8951 7540
Darwin 8922 8044
Katherine 8973 9049
Nhulunbuy 8987 0357
Tennant Creek 8962 4259
http://www.nt.gov.au/health/cdc/

The initial sign of acute strongyloidiasis, if noticed at all, is a localized pruritic, erythematous rash at the site of skin penetration. Patients may then develop tracheal irritation and a dry cough as the larvae migrate from the lungs up through the trachea. After the larvae are swallowed into the gastrointestinal tract, patients may experience diarrhea, constipation, abdominal pain, and anorexia.

#### 25 drops mimosa extract/D

Chemical constituents Mimosa pudica contains the toxic alkaloid mimosine, which has been found to also have antiproliferative and apoptotic effects.[17] The extracts of Mimosa pudica immobilize the filariform larvae of Strongyloides stercoralis in less than one hour.

**Fucoidan** 

**Rose Hips** 

**Magnolia** 

On the other hand, strongyloidiasis appears to be a relevant opportunistic infection in patients infected with human T-lymphotropic virus  $1 [\underline{1}, \underline{2}, \underline{36} - \underline{38}]$ .

Coinfection with *S.stercoralis* and HTLV-1 causes accelerated progression of both diseases People coinfected with HTLV-1 are more likely to return a positive direct faecal smear. In a study in Japan, 61% of people with both *S. stercoralis* and HTLV-1 had a positive direct faecal smear whereas only 18% of those with *S. stercoralis* and no HTLV-1 had a positive direct faecal smear (52).

HTLV-1 is associated with an exacerbated type 1 immune response. Coinfection with *S. stercoralis* or *Schistosoma mansoni* decreases the activation of type 1 cells, which may influence the outcome of HTLV-1 infection (79). Helminths including *S. stercoralis* induce a type 2 response. HTLV-1 decreases the type 2 immune response that is effective against *S. stercoralis* (10), leading to hyperinfective strongyloidiasis. HTLV-1 causes decreased levels of IgE and eosinophils, both of which are important in the immune response to *S. stercoralis* (74).

The high production of IFN-ganna observed in patients coinfected with HTLV-1 and S. stercoralis (73,80) decreases the production of IL-4, IL-5, IL-13 and IqE, molecules that participate in the host defence mechanism against helminths (80). Although coinfection with HTLV-1 was associated with a decrease in levels of IgE and skin sensitivity, it did not affect the levels of IgG (10). HTLV-1 patients are frequently refractory to anthelmintic treatment. Resistance to treatment with albendazole has been associated with high levels of serum IFN-ã and TGF-â-1 (73). Similarly, resistance to treatment with ivermectin by HTLV-1-positive patients has been demonstrated (11). This is probably due to impairment of immunity to S. stercoralis in people coinfected with HTLV-1.

Australia, in spite of having an excellent health system, has not come to grips with *S. stercoralis* in its midst. Although *S. stercoralis* is rare in mainstream Australia, it is hyperendemic throughout tropical and subtropical Indigenous Australia.

Estimates of prevalence in various Indigenous settlements are between 4.5% and 60% seropositive (19-25) and between 2% and 41% positive by stool testing (19,26,27). In central Australia, it coexists with HTLV-1 (28).

**Strongyloides stercoralis** is the <u>scientific name</u> of a <u>human parasitic roundworm</u> causing the disease of <u>strongyloidiasis</u>. Its common name is **threadworm**. In the UK and Australia, however, the term *threadworm* can also refer to <u>nematodes</u> of the genus <u>Enterobius</u>, otherwise known as pinworms.[1]

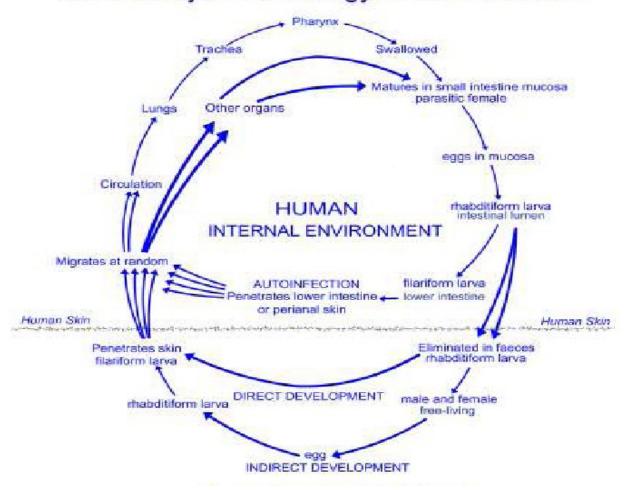
The Strongyloides stercoralis nematode can parasitize humans. The adult parasitic stage lives in tunnels in the <a href="mailto:mucosa">mucosa</a> of the small <a href="mailto:intestine">intestine</a>. The genus <a href="mailto:Strongyloides">Strongyloides</a> contains 53 species, <a href="mailto:[2][3]</a> and <a href="mailto:s.tercoralis">S. stercoralis</a> has been reported in other <a href="mailto:mammals">mammals</a>, including <a href="mailto:cats">cats</a> and <a href="mailto:dogs">dogs</a>. However, it seems that the species in dogs is typically not <a href="mailto:s.tercoralis">S. stercoralis</a>, but the related species <a href="mailto:s.tercoralis">S. canis</a>. <a href="mailto:Non-human primates">Non-human primates</a> are more commonly infected with <a href="mailto:s.tercoralis">S. fuelleborni</a> and <a href="mailto:s.tercoralis">S. cebus</a>, although <a href="mailto:s.tercoralis">S. stercoralis</a> has been reported in captive primates. Other species of <a href="mailto:strongyloides">Strongyloides</a>, naturally parasitic in humans, but with restricted distributions, are <a href="mailto:s.tercoralis">S. fuelleborni</a> in <a href="mailto:central Africa">central Africa</a> and <a href="mailto:s.tercoralis">S. kellyi</a> in <a href="mailto:Papua New Guinea</a>.

Strongyloidiasis is infection with Strongyloides stercoralis. Findings include rash and pulmonary symptoms (including cough and wheezing), eosinophilia, and abdominal pain with diarrhea. Diagnosis is by finding larvae in stool or small-bowel contents or occasionally in sputum or by detection of antibodies in blood. Treatment is with ivermectin or albendazole.

Strongyloidiasis is endemic throughout the tropics and subtropics, including rural areas of the southern US, at sites where bare skin is exposed to contaminated soil and conditions are unsanitary. Strongyloides fülleborni, which infects chimpanzees and baboons, can cause limited infections in humans.

### **Pathophysiology**

# The Life Cycle of Strongyloides stercoralis



Adult worms live in the mucosa and submucosa of the duodenum and jejunum. Released eggs hatch in the bowel lumen, liberating rhabditiform larvae. Most of the larvae are excreted in the stool. After a few days in soil, they develop into infectious filariform larvae. Like hookworms, Strongyloides larvae penetrate human skin, migrate via the bloodstream to the lungs, break through pulmonary capillaries, ascend the respiratory tract, are swallowed, and reach the intestine, where they mature in about 2 wk. In the soil, larvae that do not contact humans may develop into free-living adult worms that can reproduce for several generations before their larvae reenter a human host.

In the free-living cycle, the rhabditiform <u>larvae</u> passed in the <u>stool</u> can either molt twice and become infective filariform larvae (direct development) or molt four times and become free-living adult males and females that mate and produce <u>eggs</u> from which rhabditiform larvae hatch. In the direct development, first-stage larvae (L1) transform into infective larvae (IL) via three molts. The indirect route results first in the development of free-living adults that mate; the female lays eggs, which hatch and then develop into IL. The direct route gives IL faster (three days) versus the indirect route (seven to 10 days). However, the indirect route results in an increase in the number of IL produced. Speed of development of IL is traded for increased numbers. The free-living males and females of *S. stercoralis* die after one generation; they do not persist in the soil. The latter, in turn, can either develop into a new generation of free-living adults or develop into infective filariform larvae. The filariform larvae penetrate the human host <u>skin</u> to initiate the parasitic cycle.

PROGRESSION OF DISEASE DUE TO STRONGYLOIDES STERCORALIS: CHANGES IN BEHAVIOUR OF THE WORMS, LARVAL OUTPUT, EFFECTIVENESS OF THE DIAGNOSTIC IGG TEST, IMMUNE STATUS, AND SYMPTOMS AS THE DISEASE PROGRESSES\*

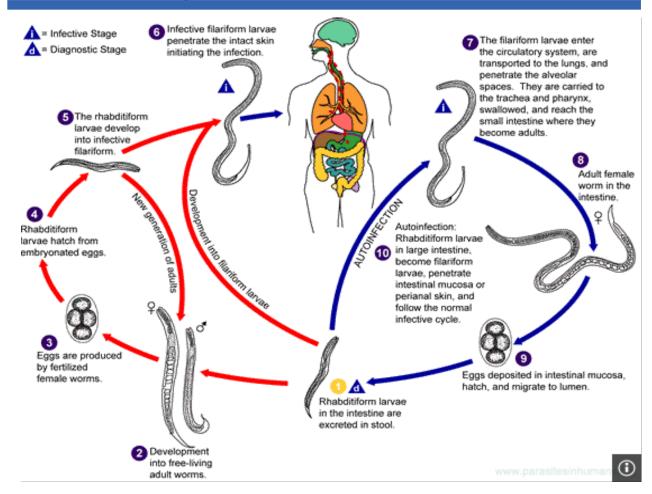
Papua New Guinea Medical Journal

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	Phase	Worms	Larvae/ml stool	Specific IgG test	Immunity	Symptoms
109	Acute	Number of females in gut increases via the autoinfective cycle, then larval output slows as immunity increases.	0 to >1000. Stool test negative or positive (35)	Negative, equivocal or positive	'Window period' before specific IgG levels become raised. Immune response increases, antibodies IgE, IgA, IgM and IgG increase, number of eosinophils increases.	Marked skin, lung and digestive system symptoms, severe diarrhoea, wasting and hypokalaemia in children; can be fatal (36).
	Chronic	Low larval output by stunted females reduces migration of larvae in tissues. Immunity keeps the number of worms low, but worms persist.	0 to 400. Stool test usually negative	Positive or equivocal	Immune response is strong, antibodies IgE, IgA, IgM and IgG are raised, eosinophilia is present in about 70%.	Skin, lung and digestive symptoms mild to moderate and may be intermittent; at least 70% have symptoms (37).
	Hyperinfective (Disseminated)	Females recover (38); numbers of females, migrating larvae and larvae in stools increase. The person will die unless they get effective treatment.	400 to >1000. Stool test usually positive	Positive, equivocal or negative	Immune suppression causes the antibodies IgE, IgA, IgM and IgG and the number of eosinophils to decrease (39).	Severe skin, lung and digestive system symptoms; other organs may be affected. Secondary infection with gut bacteria in 50%, which may present as pneumonia, meningitis or septicaemia. Case fatality rate 70% (8).

Some rhabditiform larvae convert within the intestine to infectious filariform larvae that immediately reenter the bowel wall, short-circuiting the life cycle (internal autoinfection). Sometimes filariform larvae are passed in stool and reenter through the skin of the buttocks and thighs (external autoinfection). Autoinfection explains why strongyloidiasis can persist for many decades and helps account for the extremely high worm burdens in the hyperinfection syndrome.

The infectious larvae penetrate the skin when it contacts soil. While S. stercoralis is attracted to chemicals such as carbon dioxide or sodium chloride, these chemicals are not specific. Larvae have been thought to locate their hosts via chemicals in the skin, the predominant one being urocanic acid, a histidine metabolite on the uppermost layer of skin that is removed by sweat or the daily skin-shedding cycle.[7] Urocanic acid concentrations can be up to five times greater in the foot than any other part of the human body. Some of them enter the superficial veins and ride the blood flow to the lungs, where they enter the alveoli. They are then coughed up and swallowed into the gut, where they parasitise the intestinal mucosa (duodenum and jejunum). In the small intestine, they molt twice and become adult female worms. The females live threaded in the epithelium of the small intestine and, by parthenogenesis, produce eggs, which yield rhabditiform larvae. Only females will reach reproductive adulthood in the intestine. Female strongyloids reproduce through parthenogenesis. The eggs hatch in the intestine and young larvae are then excreted in the feces. It takes about two weeks to reach egg development from the initial skin penetration. By this process, S. stercoralis can cause both respiratory and gastrointestinal symptoms. The worms also participate in autoinfection, in which the rhabditiform larvae become infective filariform larvae, which can penetrate either the intestinal mucosa (internal autoinfection) or the skin of the perianal area (external autoinfection); in either case, the filariform larvae may follow the previously described route, being carried successively to the lungs, the bronchial tree, the pharynx, and the small intestine, where they mature into adults; or they may disseminate widely in the body. To date, occurrence of autoinfection in humans with helminthic infections is recognized only in Strongyloides stercoralis and Capillaria philippinensis infections. In the case of Strongyloides, autoinfection may explain the possibility of persistent infections for many years in persons not having been in an endemic area and of hyperinfections in immunodepressed individuals.



Whereas males grow to only about 0.9 mm (0.04 in) in length, females can grow from 2.0 to 2.5 mm (0.08 to 0.10 in). Both genders also possess a tiny buccal capsule and cylindrical esophagus without a posterior bulb.[8] In the free-living stage, the esophagi of both sexes are rhabditiform. Males can be distinguished from females by two structures: the spicules and gubernaculum.

#### **Autoinfection**

An unusual feature of *S. stercoralis* is autoinfection. Only one other species in the *Strongyloides* genus, *S. felis*, has this trait. Autoinfection is the development of L1 into small infective larvae in the gut of the host. These autoinfective larvae penetrate the wall of the lower ileum or colon or the skin of the perianal region, enter the circulation again, travel to the lungs, and then to the small intestine, thus repeating the cycle. Autoinfection makes strongyloidiasis due to *S. stercoralis* an infection with several unusual features.

Persistence of infection is the first of these important features. Because of autoinfection, humans have been known to still be infected up to 65 years after they were first exposed to the parasite (e.g., <u>World War II</u> or <u>Vietnam War</u> veterans). Once a host is infected with *S. stercoralis*, infection is lifelong unless effective treatment eliminates all adult parasites and migrating autoinfective larvae.

### Hyperinfection

Hyperinfection may result from a newly acquired Strongyloides infection or from activation of a previously asymptomatic one. In either case, it can result in disseminated disease involving organs not usually part of the parasite's normal life cycle (eg, CNS, skin, liver, heart). Hyperinfection usually occurs in patients who are taking corticosteroids or who have impaired cell-mediated immunity, particularly those infected with the human T-lymphotropic virus 1 (HTLV-1). However, hyperinfection and disseminated strongyloidiasis are less common than might be predicted among patients with HIV/AIDS, even those living in areas where Strongyloides is highly endemic.

### Symptoms and Signs



Figure 3. Larva currens is pathognomonic for *Strongyloides stercoralis* infection and consists of itchy linear urticarial rashes that move at 2 to 10 cm per hour. Photo: W. Page.

Infection may be asymptomatic.

Larva currens (creeping infection) is a form of cutaneous larva migrans specific to Strongyloides infection; it results from autoinfection. The eruption begins in the perianal region and rapidly spreads, causing intense pruritus, but nonspecific maculopapular or urticarial eruptions may also occur.

Pulmonary symptoms are uncommon, although heavy infections may cause Löffler syndrome, with cough, wheezing, and eosinophilia. GI symptoms include anorexia, epigastric pain and tenderness, diarrhea, nausea, and vomiting. In heavy infections, malabsorption and protein-losing enteropathy may result in weight loss and cachexia.

Many people infected are usually asymptomatic at first. Symptoms include dermatitis: swelling, itching, <u>larva currens</u>, and mild hemorrhage at the site where the skin has been penetrated. If the parasite reaches the lungs, the chest may feel as if it is burning, and wheezing and coughing may result, along with pneumonia-like symptoms (<u>Löffler's syndrome</u>). The intestines could eventually be invaded, leading to burning pain, tissue damage, sepsis, and ulcers. Chronic

diarrhea can be a symptom. [9] In severe cases, edema may result in obstruction of the intestinal tract, as well as loss of peristaltic contractions. [10]

Strongyloidiasis in immunocompetent individuals is usually an indolent disease. However, in immunocompromised individuals, it can cause a hyperinfective syndrome (also called disseminated strongyloidiasis) due to the reproductive capacity of the parasite inside the host. This hyperinfective syndrome can have a mortality rate close to 90% if disseminated.[11][12][13]

Immunosuppressive drugs, such as those used for tissue transplantation (especially corticosteroids) can increase the rate of autoinfection to the point where an overwhelming number of larvae migrate through the lungs, which in many cases can prove fatal. In addition, diseases such as <a href="https://human.tr-lymphotropic.virus.1">human.tr-lymphotropic.virus.1</a>, which enhance the Th1 arm of the immune system and lessen the Th2 arm, increase the disease state. [12] Another consequence of autoinfection is the autoinfective larvae can carry gut bacteria back into the body. About 50% of people with hyperinfection present with bacterial disease due to enteric bacteria. Also, a unique effect of autoinfective larvae is larva currens due to the rapid migration of the larvae through the skin. Larva currens appears as a red line that moves rapidly (more than 5 cm or 2 in per day), and then quickly disappears. It is pathognomonic for autoinfective larvae and can be used as a diagnostic criterion for strongyloidiasis due to *S. stercoralis*.

#### **Hyperinfection syndrome:**

GI and pulmonary symptoms are often prominent. Ileus, obstruction, massive GI bleeding, severe malabsorption, and peritonitis may occur. Pulmonary symptoms include dyspnea, hemoptysis, and respiratory failure. Infiltrates may be seen on chest x-ray.

Other symptoms depend on the organs involved. CNS involvement includes parasitic meningitis, brain abscess, and diffuse invasion of the brain. Secondary gram-negative meningitis and bacteremia, which occurs with high frequency, probably reflect disruption of bowel mucosa, carriage of bacteria on migrating larvae, or both. Liver infection may result in cholestatic and granulomatous hepatitis. Infection may be fatal in immunocompromised patients, even with treatment.

### **Diagnosis**

- Identification of larvae by microscopic examination of stool or the agar plate method
- Enzyme immunoassay for antibodies

Microscopic examination of a single stool sample detects larvae in about 25% of uncomplicated infections. Repeated examination of concentrated stool samples raises the sensitivity; as many as 7 negative stool samples are recommended to exclude the diagnosis. The agar plate method has a sensitivity of > 85%. If the specimen stands at room temperature for several hours, rhabditiform larvae may transform into longer filariform larvae, leading to erroneous diagnosis of hyperinfection. Sampling of the proximal small bowel by aspiration may be positive in low-level infections and should be done endoscopically to permit biopsy of suspicious duodenal and jejunal lesions. In hyperinfection syndrome, filariform larvae may be found in stool, duodenal

contents, sputum, and bronchial washings and, uncommonly, in CSF, urine, or pleural or ascitic fluid. Chest x-rays may show diffuse interstitial infiltrates, consolidation, or abscess.

Several immunodiagnostic tests are available for strongyloidiasis. Enzyme immunoassay (EIA) is recommended because of its greater sensitivity (> 90%). IgG antibodies can usually be detected even in immunocompromised patients with disseminated strongyloidiasis, but the absence of detectable antibodies does not exclude infection. Cross-reactions in patients with filariasis or other nematode infections may result in false-positive tests. Antibody test results cannot be used to differentiate current from past infection. A positive test warrants continuing efforts to establish a parasitologic diagnosis. Serologic monitoring may be useful in follow-up because antibody levels decrease within 6 mo of successful chemotherapy. Sensitive and specific PCR-based methods for the diagnosis of S. stercoralis in stool samples are being developed and are available in research settings.

Eosinophilia is often present but can be suppressed by drugs such as corticosteroids or cytotoxic chemotherapeutic drugs.

### **Treatment**

Benzimidazole anthelmintics and ivermectin are the most commonly used agents for the treatment of strongyloidiasis. The modes of action of these drugs are poorly understood. A number of mechanisms have been described with regard to the action of benzimidazole agents on nematodes. The first of these is probably the most important. These drugs bind to the beta subunit of microtubule protein in the cytoplasm and prevent the assembly of microtubules (35). This leads to degenerative changes in the cells of the tegument and the gut which in turn leads to impaired digestion and absorption of nutrients. Some benzimidazoles also affect lipid membranes leading to bioenergetic disruption resulting from transmembrane proton discharge. Some benzimidazoles but not others interfere with glucose metabolism or glucose transport resulting the in the depletion of glycogen stores. Resistance to some benzimidazoles has been well-documented in some animal populations with some parasites but whether or not acquired resistance has developed in human strongyloidiasis is unknown. A number of mechanisms have been suggested for the anthelmintic activity of ivermectin. This drug interferes with transmission at the neuromuscular junctions and results in muscular paralysis including pharyngeal pumping (41).

#### **Ivermectin**

Ivermectin: there is dose-dependent eradication of adults in the gut and larvae in the tissues, and surviving larvae do not mature, in humans

Invermectin @ 200 mcg/kg po once/day for 2 days is used for uncomplicated infection and is generally well-tolerated. Albendazole 400 mg po bid for 7 days is an alternative. In immunocompromised patients, prolonged therapy or repeated courses may be needed. Combined therapy with albendazole and ivermectin has been used for hyperinfection. In severely ill patients who are unable to take oral drugs, rectal preparations of ivermectin

have been used.

Cure should be documented by repeated stool examinations.

Ivermectin is the drug of first choice for treatment because of higher tolerance in patients.[5] Thiabendazole was used previously, but, owing to its high prevalence of side effects (dizziness, vomiting, nausea, malaise) and lower efficacy, it has been superseded by ivermectin and as second-line albendazole. However, these drugs have little effect on the majority of these autoinfective larvae during their migration through the body. Hence, repeated treatments with ivermectin must be administered to kill adult parasites that develop from the autoinfective larvae.

In the UK, <u>mebendazole</u> and <u>piperazine</u> are currently (2007) preferred.[15] Mebendazole has a much higher failure rate in clinical practice than albendazole, thiabendazole, or ivermectin.[16]

This parasite depends on chemical cues to find a potential host. It uses sensor neurons of class AFD to identify cues excreted by the host. [17] *S. stercoralis* is attracted to nonspecific attractants of warmth, carbon dioxide, and sodium chloride. Urocanic acid, a component of skin secretions in mammals, is a major chemoattractant. Larvae of *S. stercoralis* are strongly attracted to this compound. [7] This compound can be suppressed by metal ions, suggesting a possible strategy for preventing infection.

### **In Vitro Susceptibility**

The effects of the benzimidazole agents, thiabendazole, mebendazole and cambendazole and albendazole on various stages of the environmental component of the life cycle of *S. ratti* have been reported (26, 27). Whereas *S. stercoralis* is found in stools in the form of first-stage larvae, *S. ratti* appears in faeces as eggs. None of these drugs inhibited the hatching of larvae from *S. ratti* eggs. When first-stage larvae are incubated in faeces in the laboratory, they moult twice after one and two days, respectively to become second-stage then third-stage larvae. All four drugs inhibited both of these moults. In addition, cambendazole but not thiabendazole or mebendazole impaired the viability (defined on the basis of structural integrity and movement) of first-, second-and third-stage larvae. Third-stage larvae are the infective form of the parasite. When such larvae were incubated with anthelmintics prior to being exposed to mice, cambendazole completely prevented the development of infection in exposed mice whereas thiabendazole, mebendazole and albendazole did not inhibit it at all. When parasitic adult *S. ratti* worms were removed from the intestines of mice and exposed to thiabendazole, mebendazole, there was no effect at all on viability *in vitro*.

Similar experiments were done with *S. stercoralis*. All four drugs completely prevented the transformation of first-stage larvae into infective third-stage larvae. It was possible to evaluate the infectivity of these latter larvae by examining mouse muscle after percutaneous infection with these parasites. Thiabendazole, mebendazole and albendazole had no effect on infectivity but cambendazole inhibited it completely. When parasitic adult *S. stercoralis* was removed from the intestines of dogs and exposed to the thiabendazole, mebendazole and cambendazole, there was no effect at all on viability.

These results suggest that cambendazole acts in a different manner to the other three drugs. They all inhibit moulting of worms but only cambendazole seems to impair the viability of worms in the periods between moults and to inhibit the infectivity of third-stage larvae.

The efficacy of thiabendazole and albendazole was compared with aqueous methanol extracts of various Jamaican plants in the ability to kill infective larvae of *Strongyloides stercoralis* (58). The inactivation for 50% of larvae was 35 and 74 hours for albendazole and thiabendazole, respectively. This compared with less than one hour for Mimosa pudica, 2 hours for love weed

(*Cuscuta americana*), 9.5 hours for breadfruit (*Artocarpus altilis*) and 20 hours for chicken weed (*Salvia serotina*). It is possible that these plants contain an active agent.

### In Vivo Susceptibility (Animal Models)

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The effects of the various benzimidazole agents on various stages of the *in vivo* component of the life cycle of *S. ratti* infections of mice and rats and *S. stercoralis* infections of dogs have been reported. Ivermectin has been investigated in *S. ratti* infections of mice, *S. venezuelensis* infections of rats and *S. stercoralis* infections of dogs and gerbils. Cyclosporine has been studied in rats infected with *S. ratti* and dogs infected with *S. stercoralis*. *S. stercoralis* completes its development in dogs and this provides the best model for investigating the precise effects of anthelmintic agents on the various stages of the life cycle. Furthermore, the intensity of infection and severity of disease is greatly increased in immunosuppressed dogs.

#### **Thiabendazole**

Thiabendazole: 50 mg/kg per day

Thiabendazole does not kill adult worms but reduces larval output; it has no effect on larvae in the tissues. Of these drugs, thiabendazole is no longer commonly used because of the side-effects of nausea and neuropsychiatric symptoms.

In mice, thiabendazole had no effect on *S. ratti* infective larvae in the skin or migrating through the lungs nor did it prevent their maturation into adult worms in the intestine. It did not expel adult worms from the gut but reduced their fecundity and therefore the numbers of eggs excreted in the faeces by 80-90%. Thiabendazole had no effect on the infectivity of *S. stercoralis* larvae as assessed by the involvement of the muscles of mice (21). The activity of thiabendazole has also been studied in rats infected with *S. ratti*; it was inactive against larvae in the tissue phase but was reported as being completely effective in the intestinal phase (44). However, only stools were tested for larvae and the intestines were not examined for the presence of adult worms or larvae. In a different study, a single dose of thiabendazole reduced the number of eggs in the stools by approximately 50% (2). Thiabendazole did not abrogate the development of patent infection with *S. stercoralis* when administered at the same time as infection to immunocompetent dogs. Nor did it eradicate the parasites when given after the onset of established infection (i.e. when larvae appeared in the stools) in immunosuppressed dogs (28).

#### Mebendazole

Mebendazole kills adults but not larvae, and is between 100 and 1000 times less effective than cambendazole, in humans.

In mice, mebendazole had no effect on the numbers of *S. ratti* larvae in the skin of mice but prevented most larvae from reaching the gut and developing into adult worms. In contrast to thiabendazole, mebendazole eliminated adult worms from the gut. Mebendazole had no effect on the infectivity of *S. stercoralis* larvae as assessed by the involvement of the muscles of mice (21). Mebendazole did not abrogate the development of patent infection with *S. stercoralis* when administered at the same time as infection to immunocompetent dogs. Nor did it eradicate the parasites when given after the onset of established infection (i.e. when larvae appeared in the stools) in immunosuppressed dogs (28).

### **Cambendazole**

Cambendazole eliminates adults and larvae in Humans.

In mice, cambendazole had no effect on the numbers of *S. ratti* larvae in the skin of mice but prevented most larvae from reaching the gut and developing into adult worms. In contrast to thiabendazole, cambendazole eliminated adult worms from the gut. Cambendazole was 100-1,000 times more active than mebendazole. Cambendazole was the most effective of all the benzimidazoles and completely eliminated *S. stercoralis* infective larvae from the muscles of mice (21). Cambendazole abrogated the development of patent infection with *S. stercoralis* when administered at the same time as infection to immunocompetent dogs. It did not eradicate the parasites when given after the onset of established infection (i.e. larvae appearing in the stools) although, in contrast to thiabendazole and mebendazole, worm burdens were greatly reduced in immunosuppressed dogs treated with cambendazole (28).

#### **Albendazole**

Albendazole: there is also dose dependent eradication of adults in the gut and larvae in the tissues, in humans.

Both albendazole and ivermectin are lipophilic and are best taken with a fatty food such as full-cream milk

Resistance of *S. stercoralis* to treatment with albendazole is associated with elevation of the *S. stercoralis*-specific IgG4 antibody titre at the expense of IgG1 (70). IgG4 is thought to block IgE-mediated responses in human strongyloidiasis (43).

Of these drugs, thiabendazole is no longer commonly used because of the side-effects of nausea and neuropsychiatric symptoms. There were a number of trials of cambendazole during the early 1980s, but it was withdrawn from the market by the manufacturer because of rare severe reactions in cattle (61). Mebendazole is completely unreliable (61).

Albendazole 400 mg for 3 days is commonly prescribed. The estimated efficacy of this regimen was 38% compared with 83% for ivermectin (65). This

In mice, albendazole had a dose-dependent inhibitory effect on *S. ratti* migratory larvae in the tissues and their development into adult worms. In contrast to thiabendazole, albendazole eliminated adult worms from the gut. Albendazole had a dose-dependent inhibitory effect on *S. stercoralis* in the muscles of mice (26). The activity of albendazole has also been studied in rats infected with *S. ratti*; it was inactive against larvae in the tissue phase but was reported as being completely effective in the intestinal phase (44). However, only stools were tested for larvae and the intestines were not examined for the presence of adult worms or larvae. Albendazole treatment for three days given at the same time as infection completely prevented the development of patent infections with *S. stercoralis* in immunocompetent dogs. When albendazole was given in a dose of 100 mg daily to immunosuppressed dogs with patent

infections, larvae disappeared from the stools transiently. When the dogs intestines were examined 7 weeks after cessation of treatment, small numbers of adult worms and rhabditiform larvae were found (26).

### **Ivermectin**

In studies of mice, ivermectin markedly reduced larval migration of *S. ratti*, prevented their development into adult worms in the gut and eradicated established adult worms from the intestinal tract. Furthermore, ivermectin eradicated *S. stercoralis* larvae from the muscles of mice (23). Rats were infected with *S. venezuelensis* and treated with a single oral dose of either a human or veterinary preparation of ivermectin (200  $\mu$ g/kg); 73-84% of tissue larvae and 59-98% of intestinal adult worms were cleared (1). Similar results were seen when ivermectin was given by injection (12). Two dogs naturally infected with *S. stercoralis* were given a single dose of ivermectin (200  $\mu$ g/kg); larvae disappeared from the faeces within a week but one dog had a recrudescence of infection and required a second course of treatment. Three dogs were infected experimentally and immunosuppressed with corticosteroids. A single dose of 800  $\mu$ g/kg was ineffective in removing infective larvae from the sites of infection but no parasites were found in the intestines (39). A single intraperitoneal injection of ivermectin given in the usual human dose (200  $\mu$ g/kg) to Mongolian gerbils (= jirds; Meriones unguiculatus) infected with *S. stercoralis* had no effect on the number of adult worms in the bowel or on their fecundity. When five times this dose was used, all worms were eradicated from the bowel (64).

### Cyclosporin

Cyclosporin, an immunosuppressant agent used to prevent rejection in organ transplantation, was serendipitously discovered to have an anti-*Strongyloides* effect when it was used to immunosuppressed dogs infected with *S. stercoralis*. Instead, the infection was eradicated. Subsequent studies showed that cyclosporin eradicated *S. ratti* from rats (<u>61</u>). A later study reported that a single dose given to rats reduced the number of larvae in the stools by approximately 50% (<u>2</u>).

### **Conclusions**

These studies suggest that cambendazole is the most active of the benzimidazole anthelmintics. It is the only one that kills first-, second-and third-stage larvae *in vitro* and prevents the development of patent infections by infective larvae exposed to drug *in vitro*. With regard to *S. ratti*, cambendazole is the most effective on a weight-for weight basis against larvae migrating through the tissues and in eliminating adult worms from the gut. Cambendazole and ivermectin were the only drugs which eradicated *S. stercoralis* larvae from the muscles of mice and the former drug produced the greatest reduction in worm burden in dogs infected with *S. stercoralis*. However, the availability of cambendazole is limited. Of the alternative drugs available, experimental studies indicate that albendazole and ivermectin are the next most effective and have similar efficacy.

### **ANTIPARASITIC THERAPY**

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The peculiar biological behavior of *Strongyloides stercoralis* is of critical importance in determining a successful outcome in the treatment of strongyloidiasis (25). The aim of treatment in most worm infections is to simply reduce the number of worms to the point at which the low intensity of infection is unlikely to cause disease. This is relatively easy to achieve in most intestinal nematode infections such as hookworm and roundworm as these parasites cannot reproduce within the human body. Recurrent infection with such parasites requires exposure to more larvae in the environment in the case of hookworm or the ingestion of eggs in food in the case of *Ascaris*. This simple approach is not applicable in strongyloidiasis as, in contrast to other worm infections, these parasites have the capacity to replicate within the

human host. Consequently, unless all worms are eradicated by treatment, those few remaining will multiply and build up the worm burden again. The problem is compounded by the fact that the diagnosis is often difficult to make in the first place because larvae are sometimes very sparse in the stools. It is therefore often impossible to be certain that all the worms have been eradicated. In some immunosuppressed patients in whom infection cannot be cleared, it is necessary to offer repeated courses of treatment.

*S. stercoralis* is relatively resistant to anthelmintic therapy when compared with other nematodes. Anti-*Strongyloides* activity is seen in two major classes of drugs, benzimidazoles and avermectins. Several benzimidazoles and one avermectin are available for human use. In addition, cyclosporin may have some effectiveness in strongyloidiasis. Unfortunately, the treatment of strongyloidiasis is often problematic.

### **Drug of Choice**

<u>Ivermectin:</u> Ivermectin is the drug of choice for initial therapy of all forms of strongyloidiasis. It is available in many countries and is probably the most effective drug. Unfortunately, a single course of treatment cannot always be relied upon to eradicate infection. Ivermectin is administered orally in the dosage indicated in <u>Table 1</u>.

Ivermectin was first used for the treatment of human strongyloidiasis in 1989. It is relatively well absorbed with a bioavailability of 50% after oral administration. A proportion of ivermectin is metabolized. The half-life of ivermectin is about 10 hours while that of the metabolites is about three days. Almost all of the drug, whether unchanged or a metabolite, is excreted in the faeces. Ivermectin is generally well tolerated. One or two percent of patients may complain of gastrointestinal symptoms or a rash. The product information provided by the manufacturer indicates that the safety of the drug has not been established in pregnancy. However, this is discussed in more detail in Section III. B. 5; ivermectin should not be used unless the mother's life is at risk. It is excreted in breast milk so should be used with caution in nursing mothers. Ivermectin is available as 3mg tablets. It is generally recommended that it be given in a single dose of 200  $\mu$ g/kg, or on two consecutive days.

There have now been a number of series investigating the efficacy of ivermectin in the treatment of strongyloidiasis. Reported cure rates vary between 67% and 100% (<u>Table 2</u>). In direct comparisons, it has been shown that ivermectin is more effective and better tolerated than thiabendazole and <u>albendazole</u> (16, 18, 40, 51, 65, 67). It must be remembered that follow-up undertaken shortly after administration of ivermectin may overestimate efficacy as the parasite may require a long time to build up worm numbers to a detectable level. The limited sensitivity of faecal analysis is underlined in the study of Toma and colleagues (67); those authors found that pyrvinium pamoate had an apparent cure rate of 23% even though this drug probably has no activity against *S. stercoralis*.

### **Special Situations**

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### Failure to Respond to Treatment in Uncomplicated Strongyloidiasis

If patients fail to respond to a single course of therapy, then repeated courses of treatment with <u>ivermectins</u>hould be offered. It infection still cannot be eradicated, then one of the alternative agents can be tried (<u>Table 1</u>).

Attempted Eradication of Infection in Immunosuppressed Patients Without Disseminated Infection

Every attempt should be made to eradicate infection in patients who have recently had or are about to have an organ transplant or are given corticosteroids for some other indication but have not yet developed disseminated infection. Similar regimens can be tried for these patients as well as for patients with HTLV-I infection who are prone to hyperinfection (Table 1).

### Initial Therapy of Disseminated Strongyloidiasis in Immunosuppressed Patients

Overwhelming strongyloidiasis occurs in patients with impaired defenses, particularly those who are receiving corticosteroid therapy. Most reported cases have been seen in patients with renal transplantation or lymphoma. Disseminated strongyloidiasis is sometimes seen in patients with AIDS but not as frequently as was originally predicted. Complications of disseminated infection include pneumonia and respiratory failure, intestinal obstruction, Gram-negative septicaemia and meningitis. All of these aspects may need attention. As with other forms of strongyloidiasis, ivermectin is the preferred initial therapy of overwhelming strongyloidiasis. The response to treatment with ivermectin in overwhelming strongyloidiasis is variable. Two patients with AIDS who were given a single dose of ivermectin relapsed whereas sustained clinical and parasitological responses were seen in seven such patients who were given four courses of the drug over 16 days (68). On the other hand, a patient with hypogammaglobulinaemia could not be cured despite repeated courses of treatment over 14 months (5). Some patients with disseminated strongyloidiasis have intestinal obstruction and cannot tolerate oral administration. Hyperinfection has been controlled in several patients who failed to respond to or could not tolerate oral therapy by subcutaneous administration of veterinary preparations of ivermectin (14, 42, 59, 69). Another patient has been treated successfully with rectal ivermectin (66). The favorable response to repeated doses of ivermectin reported in a small series by Torres and colleagues suggests that repeated courses or a long course of treatment may be required to eradicate infection in some patients (68).

Patients with disseminated strongyloidiasis and intestinal obstruction are difficult to treat. Such patients usually need suction and drainage as well as intravenous fluids. It may not be possible to administer anthelmintics orally in such situations. The options lie between subcutaneous injection of a veterinary preparation of ivermectin as discussed above, and rectal administration of thiabendazole as will be discussed later. Bacterial superinfections need treatment with appropriate antibiotics, usually those that cover coliforms. Many regimens would be appropriate until a specific organism is identified and its antibiotic susceptibilities determined; these include parenteral administration of a broad-spectrum penicillin, third-generation cephalosporin or quinolone together with an aminoglycoside. If possible, the intensity or immunosuppressive therapy should be reduced. Theoretically, changing the regimen to include cyclosporin might be helpful, in the hope that that agent will have some anti-*Strongyloides* effect (63). In desperate circumstances, cessation of immunosuppressive therapy may be the only means of saving a patient's life, even if means losing a renal graft. Despite intensive measures, many patients with severe, complicated strongyloidiasis still die.

### Suppression Therapy in Immunosuppressed Patients

In some immunosuppressed patients, it is impossible to eradicate infection. This may occur either before the onset of disseminated infection or be apparent as persistent faecal excretion of larvae after an initial response to treatment of a patient with overwhelming infection. In such circumstances, the best that can be hoped for is to keep the worm burden to a minimum by repeated administration of an anthelmintic. An appropriate initial regimen to try would be to give a single dose of ivermectin each month (Table 1).

### **Pregnancy**

The manufacturers advise caution in the treatment of strongyloidiasis with <u>ivermectin</u> and benzimidazole agents including <u>albendazole</u> and mebendazole. All of these drugs are said to have embryotoxic, fetotoxic, mutagenic and teratogenic potential (7). However, recent information indicates that hundreds of pregnant women have been given ivermectin without illeffect (11, 15). It appears that ivermectin does not easily cross the placenta (11). Similarly, WHO recommendations attest to the safety of single dose albendazole in pregnancy and so three-day courses are probably similarly safe (60). A decision on whether or not to treat a pregnant woman with strongyloidiasis should be made upon a balancing of the risks to the mother of not treating her with the small potential for damage to the unborn child. In women with uncomplicated strongyloidiasis, it is generally reasonable to defer treatment until after birth. However, if the mother is seriously ill with disseminated strongyloidiasis, then treatment in mandatory. In such circumstances, ivermectin is the drug of choice as it is more effective than albendazole in this life-threatening situation.

### **Alternative Therapy**

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The agent that is chosen to treat strongyloidiasis will depend upon availability which may vary from country to country, effectiveness, toxicity, cost and in some cases, governmental subsidy. In some countries such as Australia, ivermectin is subsidized, but only for a single course. Cambendazole is another extremely effective drug for the treatment of strongyloidiasis but its availability is limited to Brazil and possibly some other countries in South America. Albendazole is relatively reliable but needs to be given in repeated doses. Mebendazole is ineffective unless it is given for a three-week course which is cumbersome. Thiabendazole is the least effective and most toxic.

When ivermectin is unavailable, albendazole is the second choice although cambendazole may be a better option in those countries in which it is available (<u>Table 1</u>). Thiabendazole ought only to be used if the other drugs cannot be used, either because of limitation of supply or because of their costliness. If patients fail to respond to a single course of therapy, then a prolonged course with either mebendazole or albendazole should be offered. A similar regimen is appropriate for immunosuppressed patients with disseminated strongyloidiasis in whom an attempt is being made to eradicate infection.

### **Albendazole**

Albendazole is the benzimidazole agent most recently introduced into clinical practice and was first described in the treatment of strongyloidiasis in 1981. Albendazole is thought to be poorly absorbed although there is probably more absorption than with mebendazole; most of the drug is therefore active against intestinal forms of the parasite. Albendazole that is absorbed is metabolized rapidly into albendazole sulphoxide and this is probably active against tissue phases of the parasite. The half-life of the metabolite is about 8 hours; it is eliminated principally in the bile. Because of its poor absorption, albendazole has negligible toxicity when given in the doses used for strongyloidiasis. It has a remarkable safety record in several hundred million patient exposures over a 20 year period (33). It appears to be safe in pregnancy in single dose, and so a risk-benefit assessment could favour its use for symptomatic strongyloidiasis.

Contraindications are the same as for thiabendazole. Albendazole is generally available as 200 and 400 mg tablets and as a suspension of 100 mg/ml. It is usually given in a dose of 400mg once or twice daily irrespective of weight for persons two years of age and over for 3 days.

Analysis of the first 23 trials reported on the effectiveness of albendazole disclosed conflicting results (24). The doses varied but the claimed cure rates varied from 28% to 100%. Subsequent studies of treatment for several days have indicated cure rates of between 38% and 80% (4, 13, 29, 40, 43, 50, 67, 51, 65). Some authors have suggested that albendazole should be the treatment of choice for strongyloidiasis but there is considerable disagreement with the titles

of papers ranging from albendazole is effective treatment for chronic strongyloidiasis to the weak performance of albendazole in the treatment of strongyloidiasis ( $\frac{4}{}$ ).

A single repetition of the standard course after either one or two weeks made little difference to the cure rate (13, 50). Since albendazole has a greater larvicidal activity than mebendazole in experimental animals, it is likely that courses of treatment prolonged for three to four weeks are likely to be more effective. Unfortunately, no trials have yet been reported which examine this possibility.

### **Cambendazole**

Cambendazole was first used for the treatment of human strongyloidiasis in 1976. It is thought to be well absorbed from the gut and has a half-life of about 12 hours. Little is known about the pharmacokinetics of the drug. Limited studies in humans have found negligible severe toxicity although some patients complained of dizziness or diarrhea (3, 8). Unfortunately, several fatal idiosyncratic reactions occurred in cattle and this led to its withdrawal in most parts of the world (32, 38). It is not licensed for human use in the United States of America but is available in Brazil. It should not be used in pregnancy. Contraindications are the same as for thiabendazole. Cambendazole is available as cambendazol (uci-farma, Sao Paulo, Brazil) 180 mg tablets. The currently recommended therapy is a single dose of 5 mg/kg for immunocompetent patients.

Analysis of 8 trials undertaken in South America between 1976 and 1983 revealed cure rates of between 83% and 100% (24). A total of 380 patients were studied with an average cure rate of 94%. The dose of cambendazole given varied from 2.5 to 25 mg/kg. However, these patients were only followed up for several weeks and it is possible that a longer follow-up may have disclosed a higher relapse rate.

Although there have been no formal trials, cambendazole has been used with success in a patient with AIDS and disseminated strongyloidiasis (36). Repeated treatment for 12 months with thiabendazole failed to cure the patient. The patient was then given cambendazole 360 mg daily for 10 days then a single dose of 360 mg every two weeks. The patient died four months later from Pseudomonas aeruginosa pneumonia but no parasites were even seen in multiple faecal and sputum specimens, including broncho-alveolar lavage fluid obtained at bronchoscopy.

#### Mebendazole

Mebendazole was the second benzimidazole introduced for human use in 1973. It is poorly absorbed and most of the drug is active against intestinal forms of the parasite. The mebendazole that is absorbed is decarboxylated in the liver and has a half-life of about 6 hours. 90% of the orally administered drug is excreted unchanged in the faeces while the remaining 10% is excreted in the urine as the metabolite. Because of its poor absorption, mebendazole has negligible toxicity when given in the doses used for strongyloidiasis. It should not be used in pregnancy. Contraindications are the same as for thiabendazole. Mebendazole is generally available as 100 mg tablets and as a suspension of 100 mg/ml. It is usually given in a dose of 100 mg twice daily irrespective of weight for persons two years of age and over.

Mebendazole has had a disappointing record when given for only three days. Analysis of 9 trials suggested a cure rate of approximately 50% but this is probably an overestimate (24). In a comparative, trial, Beus found that mebendazole given for 5 days was only as half as effective as thiabendazole given for 2 days (6). There have, however, been suggestions that treatment for prolonged periods or repeated courses of mebendazole may be more effective. Wilson and Kaufman gave mebendazole in a dose of 1.5 g daily for 14 days and apparently cured a patient with an infected intestinal blind loop who had failed to respond to thiabendazole therapy (71). Mravak and colleagues gave mebendazole for three days to two patients without effect but cured them when 500 mg was given daily for 20 consecutive days (45). Shikiya and colleagues

studied various regimens (62). They claimed a 94% cure rate when mebendazole was given in a dose of 100 mg twice daily for 28 days to 16 patients; and 87% cure rate when given in the same dose for 5 days then the course repeated at weeks 1, 3 and 4 in 31 patients; and a 96% cure rate when given to 48 patients in a similar regimen except that each course was of only 4 days duration. The efficacy of these regimens was confirmed when patients were re-examined between 8 months and two years later but approximately half of the patients had abnormal liver function tests. The replication time for *S. stercoralis* to complete its development from first-stage larva to a gravid adult worm is between two and three weeks. It is likely that sequential therapy over several weeks is effective by eliminating adult worms in the gut initially and then removing those that develop over the next two to three weeks from autoinfecting larvae that migrate from the tissues back to the bowel.

#### **Thiabendazole**

Thiabendazole was the first benzimidazole introduced for the treatment of strongyloidiasis and has now been in use for forty years. It is rapidly absorbed from the gut then almost all the drug is metabolized in the liver by hydroxylation then conjugated to form inactive glucuronate and sulphonate esters. The half-life varies between one and two hours. More than 90% of the drug is excreted in the urine within 24 hours. It is probably widely distributed in the tissues and at least some drug enters the cerebrospinal fluid. Side-effects are common. Two-thirds of patients complain of nausea, which may be quite severe. Many patients will complain of malaise, dizziness, anorexia, smelly urine or a variety of neuropsychiatric disturbances. There may also be abdominal pain, vomiting, headache, facial flushing and pruritus. Thiabendazole is best avoided during pregnancy, especially in the first trimester, as there have been reports of teratogenic effects in experimental animals given benzimidazole agents early in pregnancy. It should not be given if there has been a prior hypersensitivity reaction to a benzimidazole agent. Thiabendazole is generally available as 500 mg tablets and as a suspension of 100 mg/ml. It is usually given in a dose of 25 mg/kg for 3 days.

Over 20 trials of the effectiveness of thiabendazole in uncomplicated strongyloidiasis were reported in the first decade after its introduction (24). The results seemed impressive with apparent efficacies ranging from 60-100%. However, they need to be viewed with some circumspection as it is extremely difficult to be certain that infection has been eradicated and that there are no worms remaining to multiply and cause persistent disease as a result of autoinfection. Further, many studies were carried out in endemic areas where reinfection was also possible. There are relatively few studies that have followed patients for a prolonged period in order to see whether there are any relapses. It is probable that infection persists in up to 20-30% of treated patients (17, 22, 49, 53). It has been suggested that cure rates may be improved by recurrent courses of treatment but the side-effect rate was high and nearly half of the patients dropped out in one trial (53).

The value of thiabendazole in patients with overwhelming strongyloidiasis due to disseminated infection has not been studied systematically in large series because of the sporadic occurrence of such infections. When those reports that were available were collated, it became apparent that of those patients who survived for at least three days after the initiation of thiabendazole therapy, more than one half of the patients failed to respond completely to treatment and many died (24). Some patients required repeated courses of treatment to suppress the infection. There is no intravenous preparation of thiabendazole but rectal administration of thiabendazole suspension (1.5g in 15 ml) as a retention enema once daily for 14 days was successful in one patient (9).

### **Prevention**

Prevention of primary infections is the same as for hookworms. To prevent potentially fatal hyperinfection syndrome, clinicians should do several stool examinations and serologic testing in patients with possible exposure to Strongyloides (even in the distant past), with unexplained eosinophilia, or with symptoms that suggest strongyloidiasis before corticosteroids or other immunosuppressants are used. If patients are infected, treatment for strongyloidiasis should be instituted and parasitologic cure should be documented before immunosuppression. Immunosuppressed people who have recurrent strongyloidiasis require additional courses of treatment until cured.

### **Key Points**

- Strongyloides larvae penetrate human skin when people walk barefoot on infested soil.
- Larvae travel through the bloodstream to the lungs, penetrate the alveoli, ascend the
  respiratory tract, are swallowed, and then mature in the intestines; adult worms produce
  ova that hatch in the intestines, releasing larvae; they can develop into infective
  filariform larvae, which may cause external or internal autoinfection, perpetuating the
  cycle.
- Patients who are taking corticosteroids or who have impaired cell-mediated immunity
  may develop potentially fatal hyperinfection syndrome—disseminated disease involving
  the lungs, intestines, skin, and organs that are not part of the parasite's normal life cycle
  (eg, CNS, liver, heart).
- Symptoms include rash, pulmonary symptoms (including cough and wheezing), and abdominal pain with diarrhea.
- Diagnose by microscopic examination of multiple stool samples, the agar plate method, or duodenal aspirate. Larvae may be identified in sputum in patients with hyperinfection.
- Treat with ivermectin

for 2 days or with albendazole

for 7 days; hyperinfection syndrome requires prolonged or repeated courses.

http://www.plosntds.org/article/info%3Adoi%2F10.1371%2Fjournal.pntd.0002640

Diagnostic Accuracy of Five Serologic Tests for Strongyloides stercoralis Infection

A brief description of all the methods follows:

- IFAT (CTD in house method): it detects IgG antibodies against S. stercoralis; for antigen preparation, intact S. stercoralis filariform larvae are obtained from a positive charcoal fecal culture, as it has been described previously [15]. Based on ROC analysis, samples with antibody titers ≥1:20 were considered positives.
- NIE refers to a 31-kDa recombinant antigen derived from a S. stercoralis L3 cDNA library. NIE-based assays used in this trial were NIE- ELISA [16] and NIE- LIPS (Luciferase Immunoprecipitation System) [17]. For the LIPS assay, all data were corrected for background reactivity. Cut offs for negatives and positives were based on

ROC analysis using sera from stool positive *Strongyloides*-infected patients and normal healthy controls as described [17]. For the NIE-ELISA, a standard curve was used and values (units/ml) interpolated from that standard curve [16]. ROC analyses performed previously were used to establish the negative and positive cutoffs for the NIE-ELISA. Cut-offs for NIE ELISA and NIE LIPS were  $\geq$ 24.13 Units/ml and  $\geq$ 1434 Relative Light Units (RLU), respectively.

Bordier ELISA [18]: it detects Strongyloides IgG antibodies by using somatic antigens from larvae of *Strongyloides ratti*. According to the manufacturer's instructions, the result is positive when the absorbance of the analyzed sample is higher than the absorbance of the weak positive control (provided in the kit). For the study purpose, in order to be able to compare results from different

sessions, we defined as positives samples with: absorbance of study sample/absorbance of weak positive serum≥1 (calculated value).

IVD ELISA [19]: it detects Strongyloides IgG antibodies by using somatic antigens from larvae of *Strongyloides stercoralis*. Positive samples are defined by absorbance greater than 0.2 OD units. For the study purpose, absorbance of study sample/0.2≥1 (calculated value) was used as the cutoff.

http://ltd.aruplab.com/Tests/Pub/0099564

http://www.questdiagnostics.com/testcenter/BUOrderInfo.action?tc=66324P&labCode=QBA

http://ltd.aruplab.com/Tests/Pub/0099564

http://www.inbios.com/elisas/Filariasis-ELISA

Filaria Detect™ IgG4 ELISA System

http://www.rapidtest.com/index.php?i=Strongyloides-IqG--ELISA-kit&id=168&cat=17

Strongyloides IgG ELISA kit

If ELISA shows the bug, on to DEC.

http://www.google.com/patents/US7709534

### Method of treating strongyloides infections and medicaments therefor

WM Forbes, PB Reese, RD Robinson - US Patent 7,709,534, 2010 - Google Patents

... 14, \*, Makarevich-Galperin et al, 49CA:74640, 1955. 15, \*, Manowitz et al, 60CA:15004, 1964. 16, \*, Mazza, Glacomo, "Minor volatile constituents of essential oil and extracts of coriander fruits" 2002, Sciences des Aliments, 22(5), 617-27

patentimages.storage.googleapis.com/pdfs/US7910114.pdf

#### SUMMARY OF THE INVENTION

The present invention meets this need by providing a method of treating a human or other mammal for a parasitic microorganism by administering an effective amount of a C8-Cl6-alpha, beta-unsaturated aliphatic aldehyde, prefer ably a ClO-C14-alpha, beta-unsaturated aliphatic aldehyde,

and most preferably trans-2-dodecenal (also referred to as "eryngial") to the human or other mammal. The method includes treating a human or other mammal Which is infected or is believed to be infected With the parasite. The method further includes administering the therapeutically effective amount of eryngial in unit dosage form. The trans-2-dodecenal compound can be isolated from

plant sources such as Eryngium foetidum and Coriandrum sativum. In an aspect of the present invention, there is provided a method of treating a mammal for a parasite of the genus Trypanosoma, comprising administering a therapeutically effective amount of a C8-Cl6-alpha, beta-unsaturated aliphatic aldehyde, preferably a ClO-Cl4-alpha, beta-unsaturated aliphatic aldehyde, and more preferably trans-2-dodecenal, to the mammal.

Regression equation Relative

TABLE 1
Immobilisation times (It<sub>50</sub> in hours) of Compound (1)
and commercial drugs (0.6% Active Ingredient (AI)) for
Strongyloides stercoralis infective larvae.

		regression equation relative		
Agent	lt <sub>50</sub> (hours) ±95%	(Y = a + bx)	activity	
(0.6% AI)	Fiducial limits (FL	) a	b	(SE) (RA)
Compound (1)	<1*			>1.00
lvermectin	<1*			>1.00
Albendazole**	73.7 (60.4-87.0)	-5.09	2.74	(0.36) 1.00
Thiabendazole	78.9 (65.5-92.8)	-12.02	6.27	(0.75) 0.93

<sup>\*</sup>Limits could not be calculated from data

<sup>\*\*</sup>RA of albendazole is used as a reference for other anthelmintic agents

TABLE 2

Mortality times (Lt<sub>50</sub> in hours) of Compound (1) and commercial

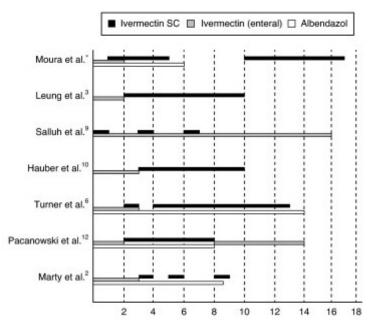
drugs (0.6% Al) for Strongyloides stercoralis infective larvae.

	Lt <sub>50</sub> (hours) Regressi		n Relative			
	±95%	equation				
Agent	Fiducial limits	(Y = a + bx)	activity			
0.6% AI)	(FL)	а	b	(SE)	(RA)	
Compound(1)	2.2 (1.2-3.3)	-10.62	4.92	(1.17)	1.00*	
Ivermectin	15.7 (12.5-18.1) -7.35		2.94	(1.72)	0.14	
Albendazole	145.3	-1.36	4.00	(0.37)	0.02	
	(127.5-194.9)					
Thiabendazole	178.9	-31.37	23.62	(1.2E+7	0.01	
	(155.9-229.0)					

<sup>\*</sup>RA of Compound (1) is used as a reference for other anthelmintic agents

http://www.sciencedirect.com/science/article/pii/S1413867012001225

The dosage of subcutaneous ivermectin used was 15 mg per day for the first four days (214  $\mu$ g/kg) and then, five days after discontinuation of the parenteral ivermectin, an additional seven-day therapy was initiated (20 mg per day; 285  $\mu$ g/kg). Daily monitoring of the ivermectin concentration in the serum was essential, as the drug can be toxic to the central nervous system. The patient's persistent coma could have been confused with symptoms of toxicity even though the dose (285  $\mu$ g/kg) was eight times lower than what can be tolerated by humans.12 It could also be due to herpetic encephalitis, but because the concentration of drug in the plasma could not be assessed,



Days of treatment

this hypothesis could not be confirmed.

Patients with hyperinfection and disseminated disease should be treated with ivermectin. For those too sick to tolerate or absorb oral (PO) ivermectin, rectal (PR) or subcutaneous (SC) dosing may be effective. [73] In these patients, ivermectin should be administered daily until symptoms have resolved and larvae have not been detected for at least 2 weeks.

### http://datasheets.scbt.com/sc-203609.pdf

Therapeutic doses of 0.2 mg/kg do not produce signs of toxicity in a variety of species including humans.

There were no gross or histological changes seen in dogs treated with ivermectin for 3 months (no-observed-adverse-effect-level

(NOAEL) = 0.5 mg/kg/day) or in monkeys treated for 2 weeks (NOAEL = 1.2 mg/kg/day).

Changes in the spleen, bone marrow and kidneys were reported in rats treated for 3 months (NOAEL = 0.4 mg/kg/day). Ivermectin produced developmental toxicity in mice, rats and rabbits at or near dosage levels that were maternally toxic (NOAEL = 0.1 mg/kg/day in mice, the most sensitive species). Neonatal rats are about 20 times more susceptible to ivermectin than adult rats because the blood brain barrier is not fully developed until after birth. There has been no evidence of teratogenicity in controlled studies in pregnant cattle, swine and dogs at up to three times the clinical dose nor has breeding performance been affected in various species.

Reproductive effects: Rats given 0.40 mg/kg/day of ivermectin had increased stillbirths, decreased pup viability, decreased lactation, and decreased pup weights. These data suggest that ivermectin may have the potential to cause reproductive effects at high enough doses.

Teratogenic effects: Ivermectin produced cleft palate in the offspring of treated mice and rabbits, but only at doses that were also toxic to the mothers. There were no birth defects in the offspring of rats given up to 1 mg/kg/day. Ivermectin is unlikely to cause teratogenic effects except at doses toxic to the mother.

The targeted clinical dosage of 0.15-0.2 mg/kg and doses in the range of 3 to 12 mg are given according to body weight.

# Higher dosages (0.4 mg/kg) have been given to patients with lymphatic filariasis.

For treatment of onchocerciasis caused by Onchcerca volvulus, a leading cause of river blindness in tropical areas), the drug is given only once every six or twelve months. Ivermectin is metabolised in the liver and excreted almost exclusively in the faeces over a period of twelve days. The plasma half-life in man is about 10-12 hours for ivermectin and 3 days for its metabolites. Side-effects are not considered to be due to the toxicity of ivermectin as such, but are attributed to hypersensitivity reactions resulting from the death of the microfilariae. In cases of accidental overdose with ivermectin, there have been no fatalities reported; however symptoms resemble those in animal studies.

Mutagenic effects: Ivermectin does not appear to be mutagenic. Mutagenicity tests in live rats and mice were negative. Ivermectin was shown to be nonmutagenic in the Ames test. Carcinogenic effects: Ivermectin is not carcinogenic in rats or mice.

The rats were fed dietary doses of up to 2 mg/kg/day for 24 months, and the mice were up to 8 mg/kg/day for 22 months. These represent the maximum tolerated doses.

## **Flagellated Promastigote**

Figure 3. Effect of fucoidan therapy on visceral infection in BALB/c mice. Various doses of fucoidan (25–250 mg/kg/day) were given orally (3 times weekly) for a period of 4 weeks

### 4-aminoquinaldine compounds

4-Aminoquinaldine compounds are effective against both SAG-sensitive and SAGresistant Leishmania parasites in culture. We determined the in vitro leishmanicidal activity of 4-aminoquinaldines (denoted PP) after 2 and 48 h exposure to L. donovani (AG83) promastigotes (Table 2). Of the series studies, PP-2, PP-8, PP-9, and PP-10 demonstrated potent activities, with half-maximal inhibition concentrations (IC<sub>50</sub>s for 2 h) ranging from 1.52 to 2.73  $\mu$ M. Growth inhibition studies (48 h) illustrated that PP-9 and PP-10 were stronger inhibitors of L. donovani promastigotes, with IC<sub>50</sub>s of 0.50 and 0.47 μM, respectively. Furthermore, exposure to intracellular amastigotes for 48 h demonstrated that 4-aminoquinaldine derivatives PP-2, PP-9, and PP-10 have significant  $IC_{50}$ s of 1.47, 1.08, and 0.94  $\mu$ M, respectively, without showing any toxicity on murine peritoneal macrophages (Table 2). PP-9 and PP-10 were also effective against intracellular amastigotes of SAG-resistant L. donovani strains exhibiting  $IC_{50}$ s of 6.44 and 3.91 μM, respectively, for GFP2039 and 7.7 and 5.84 μM, respectively, for GE1F8R ( $\underline{\text{Table 3}}$ ). The IC<sub>50</sub> of PP-10 is 1.54-fold less than that of miltefosine against GE1F8R strains. Moreover, a significant reduction in green fluorescence of intracellular GFP2039 L. donovani amastigotes after PP-10 treatment at the  $IC_{50}$  dose in contrast to that for untreated controls was observed

- Indolylquinoline derivatives are cytotoxic to Leishmania donovani promastigotes and amastigotes
- Ferroquine, an ingenious antimalarial drug:
- o mefloquine and aminosidine.
- Inhibitors of dihydrofolate reductase
- 2,4-diaminoquinazolines as inhibitors of trypanosomal and leishmanial dihydrofolate

1.

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#### CrossRefMedline

Cationic Liposomal Sodium Stibogluconate (SSG), a Potent Therapeutic Tool for Treatment of Infection by SSG-Sensitive and -Resistant Leishmania donovani *Antimicrob. Agents Chemother*. January 2015 59:1 344-355

### http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3256064/

Aspergillus japonicas in 1978

The epoxysuccinyl peptide l-*trans*-epoxysuccinyl-Leu-4-guanidinobutylamide (E64) was the first CP inhibitor to be discovered, with an epoxide being the essential structural feature (Fig. 1). This natural product was isolated from *Aspergillus japonicas* in 1978 (10). The broad-spectrum cysteine cathepsin inhibitor E64 selectively inhibited papainlike CPs (clan CA, family C1) and had little or no selectivity for different members of this proteinase family (27). Thus, E64 can be used as a broad-spectrum CAC1 inhibitor which does not discriminate between the CPA, CPB, and CPC enzymes.

Furthermore, many derivatives of E64 were developed to achieve selectivity within the papain family. I-trans-Epoxysuccinyl-Ile-Pro-OH propylamide (CA074), one of the first selective inactivators of cathepsin B and cathepsin B-like enzymes (e.g., CPC) (Fig. 1), was developed in 1991 (5, 34). Aziridinyl peptide inhibitors, a second class of cysteine cathepsin inhibitors, are aza analogues of epoxysuccinyl peptide inhibitors. The natural aziridinyl peptide miraziridine A was isolated from the marine sponge *Theonella* sp. aff. mirabilis (20). Derivatives containing aziridine-2-carboxylic acid and aziridine-2,3dicarboxylic acid as electrophilic building blocks were synthesized to develop new selective inhibitors (25, 26). In further investigations, aziridine-based inhibitors containing aziridine-2,3-dicarboxylates [Azi(OBn)<sub>2</sub>s] showed selective inhibition of cathepsin L-like enzymes, for instance, rhodesain, the major trypanosomal papain-like CP of the parasite Trypanosoma brucei rhodesiense (41), and falcipain-2 and falcipain-3 from the **malaria** parasite *Plasmodium falciparum* (29). In addition, aziridine-2,3dicarboxylate-based inhibitors had antiparasitic activity against Trypanosoma brucei brucei and P. falciparum in vitro (29, 41). Finally, this promising series of peptidomimetic aziridine-2,3-dicarboxylate inhibitors was demonstrated to exert significant antileishmanial activity. Two derivatives of this series, Boc-(S)-Leu-(R)-Pro-(S,S)-Azi $(OBn)_2$  (compound 13b) and Boc-(R)-Leu-(S)-Pro-(S,S)-Azi $(OBn)_2$  (compound 13e) (Fig. 1), reduced the growth and viability of Leishmania major promastigotes and the infection rate of macrophages with L. major amastigotes (23). The peptidomimetic inhibitors 13b and 13e were selectively active against L. major and did not display any cytotoxic effects against macrophages and fibroblasts (23).

Aziridine-2,3-dicarboxylate-based inhibitors 13b and 13e reduce activity of cathepsin B-like enzyme CPC in protein lysates of *L. major*. We recently demonstrated that aziridine-2,3-dicarboxylate-based CP inhibitors 13b and 13e exhibit highly significant leishmanicidal activity *in vitro* (23). Both compounds were developed to inhibit parasitic cathepsin L-like enzymes (38, 41). In the present study, we analyzed the ability of aziridine-2,3-dicarboxylate-based CP inhibitors 13b and 13e to affect the cysteine cathepsin activities in *L. major* promastigotes. The proteolytic activities of the cathepsin B-like and the cathepsin L-like enzymes could be detected by fluorescence proteinase

activity assays using the substrate Z-Phe-Arg-AMC (Fig. 2A). The detected proteolytic activity was E64 sensitive (inhibition of CPA, CPB, and CPC) and CA074 sensitive (selective inhibition of CPC) (Fig. 2A). However, minor residual proteinase activity remained after preincubation with E64 (100  $\mu$ M) and demonstrated that Z-Phe-Arg-AMC is also cleaved by additional non-papain-like proteinases (Fig. 2A). The leishmanicidal aziridine-2,3-dicarboxylate-based inhibitors 13b (200  $\mu$ M) and 13e (200  $\mu$ M) were also able to inhibit the proteolytic cleavage of substrate Z-Phe-Arg-AMC (Fig. 2A). No further reductions of proteinase activity by compounds 13b (200  $\mu$ M) and 13e (200  $\mu$ M) could be detected after preincubation of protein lysates with E64 (100  $\mu$ M) (Fig. 2B). Therefore, the Z-Phe-Arg-AMC-cleaving proteinases inhibited by compounds 13b and 13e were members of the papain-like cysteine cathepsins (clan CA, family C1). Preincubation of lysates with the CPC-specific CA074 (100  $\mu$ M), followed by a second incubation step with compound 13b (200  $\mu$ M) or 13e (200  $\mu$ M), also did not result in an additional reduction of the hydrolytic activity in

### **Ascaris**

http://cid.oxfordjournals.org/content/40/8/1173.full

### **Nitazoxanide: A New Thiazolide Antiparasitic Agent**

it has been >30 years since the introduction of any new innovative treatment and, for some pathogens (including *Cryptosporidium*), there is currently no accepted specific therapy [1]. Nitazoxanide, 2-acetyloxy-*N*-(5-nitro-2-thiazolyl) benzamide (Alinia; Romark Laboratories), is a new nitrothiazole benzamide compound notable for its activity in treating both intestinal protozoal and helminthic infections. It was first described in 1975 by Jean Francois Rossignol and was initially developed as a veterinary antihelminthic with activity against intestinal nematodes, cestodes, and liver trematodes [2]. In humans, nitazoxanide has been reported to be effective against a broad range of parasites, including *Giardia lamblia, Entamoeba histolytica, Cryptosporidium parvum, Cyclospora cayetanensis, Trichomonas vaginalis, Vittaforma corneae, Encephalitozoon intestinalis, Isospora belli, Blastocystis hominis, Balantidium coli, Enterocytozoon bieneusi, Ascaris lumbricoides, <i>Trichuris trichura, Taenia saginata, Hymenolepis nana,* and *Fasciola hepatica* [3–9]. In vitro studies have also shown antimicrobial activity against numerous gram-positive and gram-negative anaerobic bacteria, specifically *Bacteroides* species, *Clostridium* species, and *Helicobacter pylori,* and against aerobic gram-positive bacteria [10, 11].

https://en.wikipedia.org/wiki/Nitazoxanide

Nitazoxanide was originally discovered in the 1980s by <u>Jean-François Rossignol</u> at the <u>Pasteur Institute</u>. Initial studies demonstrated activity versus <u>tapeworms</u>. *In vitro* studies demonstrated much broader activity. Dr. Rossignol co-founded Romark Laboratories, with the goal of bringing nitazoxanide to market as an anti-parasitic drug. Initial studies in the USA were conducted in collaboration with Unimed Pharmaceuticals, Inc. (<u>Marietta, GA</u>) and focused on development of the drug for treatment of <u>cryptosporidiosis</u> in <u>AIDS</u>. Controlled trials began shortly after the advent of effective anti-retroviral therapies. The trials were abandoned due to poor enrollment and the FDA rejected an application based on uncontrolled studies.

Rather than abandon their efforts, Romark launched a series of controlled trials. A placebo-controlled study of nitazoxanide in cryptosporidiosis demonstrated significant clinical improvement in adults and children with mild illness. Among malnourished children in Zambia with chronic cryptosporidiosis, a three-day course of therapy led to clinical and parasitologic improvement and improved survival. In Zambia and in a study conducted in Mexico, nitazoxanide was not successful in the treatment of cryptosporidiosis in advanced infection with human immunodeficiency virus at the

doses used. However, it was effective in patients with higher CD4 counts. In treatment of giardiasis, nitazoxanide was superior to placebo and comparable to <a href="mailto:metronidazole">metronidazole</a>. Nitazoxanide was successful in the treatment of metronidazole-resistant giardiasis. Studies have suggested efficacy in the treatment of cyclosporiasis, isosporiasis, and amebiasis.[1]

The anti-protozoal activity of nitazoxanide is believed to be due to interference with the pyruvate:ferredoxin oxidoreductase (PFOR) enzyme dependent electron transfer reaction which is essential to anaerobic energy metabolism. This mechanism of action overlaps with the mechanism of action of the <u>nitrofurans</u> which is a class of antimicrobial drugs.[2]

Following oral administration, it is rapidly <u>hydrolyzed</u> to its active metabolite, <u>tizoxanide,[3]</u> which is 99% protein bound. Peak concentrations are observed 1–4 hours after administration. It is excreted in the <u>urine</u>, <u>bile</u> and <u>feces</u>.

It has also been shown to have activity against influenza A virus in vitro.[4] The mechanism appears to be by selectively blocking the maturation of the viral hemagglutinin at a stage preceding resistance to endoglycosidase H digestion. This impairs hemagglutinin intracellular trafficking and insertion of the protein into the host plasma membrane.

http://www.drugs.com/dosage/nitazoxanide.html

### **Usual Adult Dose for Cryptosporidiosis**

For diarrhea in immunocompetent patients: 500 mg twice daily with food for 3 days. For diarrhea in AIDS patients: 1000 mg twice daily with food for 14 days or until diarrhea resolves.

http://dailymed.nlm.nih.gov/dailymed/archives/fdaDrugInfo.cfm?archiveid=16374

Information on nitazoxanide overdosage is not available. In acute studies in rodents and dogs, the oral LD50 was higher than 10,000 mg/kg. Single oral doses of up to 4000 mg nitazoxanide have been administered to healthy adult volunteers without significant adverse effects.

http://www.ncbi.nlm.nih.gov/pubmed/19070525

#### **METHODS:**

Seventy-five children aged from 28 days to 24 months, with rotavirus diarrhea, were randomly assigned to receive either oral nitazoxanide (15 mg/kg/day) twice a day for three days, a combination of oral probiotics, 1 g twice a day for five days, or only oral or systemic rehydration solutions.

http://www.blastocystis.net/search/label/nitazoxanide n2x/d for 10 days at 500mg per

http://cc.bingj.com/cache.aspx?q=nitazoxanide+mg %2fkg+ascaris&d=4659074655654021&mkt=en-US&setlang=en-US&w=-YflujVeo\_1Gs6zxtTps-ENuRkm83rVs

Comparison of results of human clinical studies using compounds of

Formula I and Formula II having particle sizes ranging from 170  $\mu$ m to 520  $\mu$ m (mean = 352  $\mu$ m) with results obtained using Formula I and

Formula II having particle sizes ranging from 5  $\mu$ m to 200  $\mu$ m (mean = 34  $\mu$ m).

Compound of Formula I (98%) + Compound of Formula II (2%) Particle sizes 170 to 520  $\mu$ m Particle sizes 5 to 200  $\mu$ m

Dose = 15 to 50 mg/kg/day for 3 to 7 Dose = 15 mg/kg/day for days days Parasite No. Cured/Total = % Cure Rate No. Cured/Total = % Cure Rate

Blastocystis hominis 12/27 = 44% 10/10 = 100% Entamoeba histolytica 29/47 = 62% 106/133 = 80% Giardia lamblia 11/37 = 30% 50/73 = 68% **Ascaris** lumbricoides 3/69 = 4% 144/179 = 80% Trichuris trichiura 7/48 = 15% 58/79 = 73%

### http://eol.org/pages/3598509/details

Infections can be treated with drugs called <u>ascaricides</u>. The treatment of choice is <u>mebendazole</u>. The drug functions by binding to <u>tubulin</u> in the worms' intestinal cells and body-wall muscles. <u>Nitazoxanide</u> and <u>ivermectin</u> can also be used.[5]

^ a b c Roberts, Larry S.; Janovy, John Jr. *Foundations of Parasitology*, Eight Edition. United States: McGraw-Hill, 2009

### http://www.sciencedirect.com/science/article/pii/0035920354900401?np=v

The encouraging results obtained by Hewitt et al. (1948) in the treatment of canine ascariasis with hetrazan formed the basis of its trial in human beings. Oliver Gonzales et al. (1949) treated six patients with favourable results giving 2 mg. per kg. body weight three times daily for 24 hours, and followed 8 hours later by a purge. Ruiz-Reyes et al. (1950) administered it to children using a dose of 2 to 3 mg. per kg. body weight twice daily for 3 days, and concluded that although it was easy to administer hetrazan in palatable syrup, yet it is not the best drug for ascariasis. Etteledrof et al. (1950) treated 15 children with a dose of 6 mg. per kg. body weight daily for 7 days and reported that hetrazan is very effective for the treatment of ascariasis. Colbourne (1950) using the same dosage as Oliver-Gonzales in 24 patients, in comparison with oil of chenopodium, concluded that hetrazan was less effective. Loughlin et al. (1951) treated three groups of patients using higher doses or for longer periods, and considered hetrazan an effective anthelmintic in ascariasis; they believe that the less satisfactory results obtained by other workers were attributable to the insufficient dosage used. Thomson (1952) considered hetrazan as the most useful drug for the treatment of ascariasis especially in debilitated children. She treated 50 children with 20 mg. per kg. body weight daily for not less than 4 days and found this dose effective; yet she advised a course of 7 days.

http://www.neglecteddiseases.gov/target\_diseases/soil\_transmitted\_helminthiasis/roundworm/

Although ascariasis is not eradicable, it can be better controlled if the above measures are implemented in areas of high prevalence.

http://www.cdc.gov/mmwr/preview/mmwrhtml/00025967.htm

http://archsurg.jamanetwork.com/article.aspx?articleid=578981

The condition of a 2-year-old Chinese boy with hepatic ascariasis, proven by biopsy, was managed successfully with operative evacuation of the foci in the liver and piperazine citrate and diethylcarbamazine citrate treatment. Although he became well during the three weeks in which diethylcarbamazine therapy was given, this is not necessarily a claim for the specificity of this anthelmintic for hepatic ascariasis.

### **B** Hominis

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3745668/

Blastocystis is a protozoan parasite.

### **Anti Parasitics**

A number of antimicrobial agents have been used to treat *Blastocystis* infection. This includes metronidazole, nitazoxanide, trimethoprim-sulfamethoxazole (TMP-SMX), paramomycin, iodoquinol, ketoconazole, secnidazole, emetine, tinidazole, and the probiotic *Saccharomyces boulardii*.

#### Resistance

One clinic has reported successful treatment with a combination of secnidazole, nitazoxanide, and furazolidone. However, it is to be noted that all three drugs are not currently available in many countries. [3] A 2006 text described an USA patient returning from Nepal with chronic Blastocystosis who was treated without success over a period of 3 years with iodoquinol, paramomycin, doxycycline, albendazole, tinidazole, ornidazole, quinacrine, nitazoxanide, rifaximin, furazolidone, cotrimoxazole, itraconazole, ketoconazole, and various combinations of these drugs. [17] One recent study on metronidazole resistance and subtype dependent variation in drug susceptibilities of *Blastocystis* revealed that subtype 7 and subtype 4 are resistant to metronidazole

Nitazoxanide, a 5-nitro thiazole, broad spectrum antiparasitic agent is found to have potent activity against *Blastocystis*.[10,30] In children, the achievable clearance rates are 97-100% on treatment with this drug. It is well-tolerated with no serious adverse effects.[30] Metronidazole treatment failures in *Blastocystis* may well respond to nitazoxanide.[12]

nitazoxanide 500 mg twice daily for 3 days reported a clinical and parasitological cure of 86%.

### Quassa bark tea:

Other drugs that are found to have variable efficacy on *Blastocystis* are tinidazole, ornidazole, secnidazole, ketoconazole, pentamidine, furazolidone, quinine, iodoquinol, iodochlorhydroxyquin, and emetine. [10,12]

Cysteine proteases play an important role in the cell cycle and pathophysiology of *Blastocystis* and induce up regulation of pro-inflammatory cytokines.

*S. boulardii* is a non-pathogenic yeast and has proven effective in gastrointestinal diseases with predominant inflammatory component, indicating its role in interference with cellular signaling pathways

(*Brucea javanica* and *Coptis chinensis*). Their inhibitory activity was not as great as with similar concentrations of metronidazole.

genotype-1 have demonstrated increased susceptibility to garlic at 0.01 mg/ml. Other investigational agents such as ginger, black pepper, and white cumin did not have significant inhibitory effect in drug susceptibility assays.

### **CFS Chronic Fatigue Syndrome**

http://www.prohealth.com/library/showarticle.cfm?libid=3341

http://www.voiceamerica.com/episode/86639/the-discovery-of-a-new-worm-closely-linked-to-cfs-fibromyalgia-and-multiple-chemical-sensitivities

Cryptostrongylus

## Cryptostrongylus pulmoni

http://www.anapsid.org/cnd/diffdx/klapow.html CFS patients, estimated at 63%

http://lymebusters.proboards.com/thread/10080

polarizing microscope

### **CFS Radio Program**

Jan. 16th, 2000. Roger G. Mazlen, M.D. Host with Dr. Larry Klapow.

Dr. Mazlen: Dr. Larry Klapow was a guest on this past Sunday's CFS Radio Show and gave a very important and very informative report of the work he has been doing. Dr. Klapow has been finding a new roundworm type of parasite in CFS patients called "Cryptostrongylus pulmoni". I found this show to be of particular interest to me because I have been found to have this particular parasitic infection and was very interested to learn of the immunologic dysfunctions that it can cause. I truly think that this important work definitely plays a part in the complex puzzle that is known as Chronic Fatigue Syndrome.

- Dr. Mazlen To kick off the new millennium with a good show that you'll find of great interest and great importance, I'm talking today with Dr. Larry Klapow, a Ph.D. in Invertebrate Biology who's in Burlingame, California near San Francisco. Good morning Larry, welcome to our show.
- Dr. Klapow Good morning, Roger, I'm glad to be here.
- Dr. Mazlen Can you tell our audience something about the this suspected new parasite that you've found in a percentage of patients with Chronic Fatigue Syndrome? How'd you find it?
- Dr. Klapow Well, Roger, it came about as a result of a conversation I was having with an immunologist friend of mine, Dr. Vincent Marinkovich, here in Redwood City, California. He was treating a CFS patient we thought might have a roundworm infection. The patient had a low grade eosinophilia and some unusual rashes on the torso that suggested the possibility of threadworm disease. Antibody tests and stool tests were negative. I thought about this for a while and I know that some chronic parasites migrate between the digestive tract and the respiratory tract and some of them are coughed up in sputum. So I looked at the sputum and that's where I found it. I called the new parasite "Cryptostrongylus pulmoni", that's a provisional name and it means "the hidden lung worm".
- Dr. Mazlen That's pretty appropriate in terms of what you say.
- Dr. Klapow It definitely is, Roger. It's very difficult to find. And I hope other people will start looking for it. In fact, I've put together some material that I think can help them.
- Dr. Mazlen You recently completed a small blinded study in cooperation with a small number of CFS doctors including Dr. Anthony Komaroff in Harvard. You're now doing a larger blinded trial and you're also trying to develop a clinical test for the parasite. But for these other investigators and clinicians, can you tell us what does the parasite look like and how can they find it?
- Dr. Klapow You can identify the parasite, the female by its mouth parts and the male by its very intricate reproductive structure. This parasite is very small. The female is less than a millimeter long and the male is about a third that length. So, in addition to being small there's also a lot of difficulties. The specimens I usually pick up are naturally expelled in sputum and they're usually very decayed and rare and because of this you need very specialized imaging techniques to find them. They're not expensive techniques, they're just specialized. In any case I wanted to help people look for this parasite and so I put together a website which describes how to find it in great detail. It also includes anatomical drawings. I can give you the website address if you like. Dr. Mazlen Yes, give us the address right now, that'll be great.
- Dr. Klapow OK, I'll give you my own email address and then I can post the other rather longer address for people who contact me. My email address is lak123@gateway.net.
- Dr. Mazlen Now, this is really important because this introduces a whole new dimension about Chronic Fatigue Syndrome and its possible relationship to roundworm infestation. Can you tell us so far, at least, as you've been looking, what percentages of Chronic Fatigue Syndrome patients are turning out to be positive for this worm?
- Dr. Klapow Yes, I find the parasites in about 40% of three-day sputum samples from CFS patients. However, I have to tell you that yields are very low. In fact, they're so low that I think I'm probably missing as many positive patients as I'm finding. The problem here is that over 80% of the positives I get are represented by only one identifiable specimen. So just by chance it looks like I'm missing a fairly high percentage.
- Dr. Mazlen So, the prevalence can be a lot higher and this, of course, stirs some very great interest

in terms of causation and etiology which we'll go into later. Can you tell us anything about the blinded trials so far?

Dr. Klapow Yes, I did a small blinded trial in cooperation with a number of doctors including Dr. Anthony Komaroff at Harvard and here are the results. I think they're interesting but you judge for yourself. 5 of the 11 patients were positive while all 6 controls were negative. Now, it's a small trial but if you were trying to do as well by guessing, say by just tossing a coin, you'd only do as well as I report here in about 1 in a 100 tests, so it's a very hard thing to do by just guessing. The results of this small trial can be used to devise an experimental design for a large trial that could give a statistically significant result and going through that exercise suggests that between 50 and 80 samples will be needed. Now, it may take some time to process these samples microscopically. It's now taking me somewhere between 50 and 100 hours to find a single positive patient so I think the progress will be slow, that is unless we can get something more rapid going in the form of a PCR test.

Dr. Mazlen Well, I certainly congratulate you though for the effort that you're making because this is totally important to patients for their prognosis and recovery ultimately. There's a lot more to learn and, of course, we're going to go into that and you mentioned the PCR test and that you have some arrangements whereby this can be developed and hopefully you'll get some funding to help this along soon. If anybody in the audience is interested in helping in this regard they can reach me at rgm1@aol.com which is my email address and I'll forward it on to Dr. Klapow. Larry where do you think these parasites might be coming from?

Dr. Klapow Well, Roger, they have some specialized anatomical structures that suggest that they're related to parasites of animals that live in the jungles of Southeast Asia. In fact, there's been somewhat of a history of hard to diagnose parasites coming out of that area and being brought back to "Western" countries after periods of warfare. It happened in the Victorian era when French soldiers were returning from this area and brought back the chronic parasite Strongyloides stercoralis to Europe where it was first diagnosed in 1894. It also happened again in World War II. This time British soldiers became infected while they were imprisoned in Burma returned to England and 30 years later, in 1974 they were diagnosed with chronic parasites they had gotten while they were in prison. It's kind of a testament to how difficult some of these parasites are to find and treat. I would like to look at people who've been to Southeast Asia and I think I plan to do that as soon as I finish with the large trial I'm doing on CFS patients now.

Dr. Mazlen It's a natural sequitur because you're going to be having a chance to look at all of the veterans of the Viet Nam era who either served in Viet Nam or Cambodia or neighboring areas.

Dr. Klapow That will happen, I think, rather quickly if I can get the PCR test going.

Dr. Mazlen You say it might be coming from this source and that's a possibility. How is it contracted? How do you get it then?

Dr. Klapow I'm really not sure. What I can tell you is this. I've never seen a fresh transmissible stage of the parasite in any sputum sample I've seen so far. I've done a couple of hundred samples at this point. So I don't think there's any evidence right now of casual transmission. But roundworm parasites are typically acquired by eating contaminated food, but an outbreak of Cryptostrongylus infection, if it were transmitted in this way, would look very different then a typical food poisoning incident where people get sick within a couple of hours after eating.

Dr. Mazlen That's due to the long latency that you mentioned.

Dr. Klapow Cryptostrongylus is very small but it produces a larvae which is very large so there's an implication here that it must be reproducing very slowly and possibly has a very long latency time.

Of course, we know that the outbreak of Chronic Fatigue Syndrome usually take place over several months and in some cases a couple of years and that I think would be consistent with the possibility of a food borne infection with a very long latency period.

Dr. Mazlen Well, now we're going to turn to the clinical side. Most of the time that doctors are looking for parasitosis, they look to see elevated eosinophil and serum IgE, or immunoglobulin E, levels in patients. Isn't this usually the case?

Dr. Klapow Yes, but that's the first question that I get from doctors when I tell them that I found what I think is a new species of roundworm parasite. Where's the elevated IgE? And the answer is elevated IgE is mainly apparent in acute roundworm infections. With time, the chronic parasites are able to suppress the IgE response and many of them produce a clinical picture where the patients either have normal or lower than the normal average level of IgE and, in fact, that's the picture you see in CFS and in all the studies I've reviewed, IgE is lower in CFS patients than in healthy control populations.

Dr. Mazlen Here I want to interject that I'm part of the new study looking into C.pulmoni in CFS patients and one of the things that prompted me to call you and talk to you about getting involved is the fact that I had been seeing low IgE levels, low eosinophile counts in patients that I thought were inappropriate.

Dr. Klapow In fact, there was a paper that's a few years old in the Journal of Chronic Fatigue Syndrome that indicates that if you correlate IgE and eosinophil levels with the number of symptoms the patients report, the sicker they are the lower the IgE and eosinophil counts and that's a statistically significant relationship.

Dr. Mazlen And I see it and it seems to be borne out. Now, what do you think is suppressing IgE in this CFS or Chronic Fatigue Syndrome patients? What's the mechanism?

Dr. Klapow Well, I think the mechanism may involve the cell marker CD23 which suppresses IgE. There are a couple of other things that activate CD23, the IgE suppresser and those are active herpes viruses and some of the TH1 cytokines, particularly interferon-gamma and the 2'-5'A, the activator of the latent RNase enzyme. Both herpes viruses and 2'-5'A, as you know, are highly elevated in CFS patients. In fact, it looks like some roundworms may be using chronic viruses as cofactors to help perpetuate their own survival.

Dr. Mazlen That certainly rings true from what I've seen clinically and that leads us to another question. If a lot of Chronic Fatigue Syndrome patients have allergies, they should have elevated IgE levels but a lot of them, as we were just saying, don't. It seems to fit the model you propose of a suppresser.

Dr. Klapow Yes, there are some doctors, in fact, that think allergy is a risk factor for getting a roundworm infection and that's because patients who tend to produce too much IgE to non-specific stimuli, harmless things, may not have enough reserves left over to fight off the parasites so they get a foothold, and in fact, initially, you can even see patients who report increased allergies, but later on when they're diagnosed with CFS and the presumptive parasite, if we may go so far and speculate, has suppressed their IgE response and the values come out clinically low.

Dr. Mazlen Now, this brings us to a leading question, which, obviously is a speculation, but that's all right because that's what this show is about. We want to raise issues and have other people contribute to answering them as well. There seem to be many infectious agents that have been proposed as being possible etiological agents for Chronic Fatigue Syndrome. None of them have held up specifically as a single causative agent. What do you think about this roundworm infection, c.pulmoni, is it a primary infection or is it just another opportunistic organism?

Dr. Klapow Well, I don't know if it's a primary cause of CFS. We'll just have to have to go through the rules of Koch's postulates and see how far we can get. I think it's an interesting candidate for a possible primary agent. I don't think it's an opportunistic infection. Opportunistic infections are usually airborne and are present everywhere. They're just waiting for our immune systems to be weakened before they establish a chronic infection. Cryptostrongylus doesn't seem to be ubiquitous. If I'm right about the taxonomy, it looks like it's coming out of a particular geographic area. They're are also a number of things that I think can connect roundworm infection to the major physiological systems that malfunction in CFS. And they have to do with the wide variety of physiologically active agents roundworms are able to secrete.

Dr. Mazlen We're going back now and talking about the hormones that these roundworms secrete, namely vasoactive intestinal polypeptide, which is known as VIP, and hippocampal cholinergic neurostimulatory peptide which is known as HCNP, and what they do and Larry, what do these hormones cause? What do they do?

Dr. Klapow Well, VIP is involved in regulating blood pressure and blood flow. It's important in regulating blood flow to the brain. It's believed to be implicated in orthostatic intolerance from which a number of CFS patients suffer. And, it also controls hypothalmic CRH, a hormone that's ultimately responsible for the level of cortisol in the blood which is suppressed in CFS and it's also suppressed in chronic roundworm infections. And the other one, HCNP, is a limbic system neuropeptide and it's believed to be involved in memory and immune function. When it goes wrong in areas that have Alzheimer's lesions, there are cognitive symptoms. In fact, some doctors have suggested that CFS looks in some respects like a reversible form of Alzheimer's.

Dr. Mazlen It seems like that sometimes.

Dr. Klapow Well, the bad news is that it bare's any resemblance to that disease. \*What good news there is, is that the cognitive symptoms come and go, without apparently doing permanent damage. I think it is a reasonable hope that increasingly effective treatments for CFS will be found in time to substantially help most of those who now suffer from this difficult and often misunderstood disease.

Transcribed by Carolyn Viviani

\*Added to transcript by Dr. Klapow after the show.

Source: Transcribed by Carolyn Viviani; carolynv@inx.net.

(Note: The CFS Radio Show has once again lost its sponsor so this will be the last show until a new sponsor can be found.)

http://lymebusters.proboards.com/thread/10080

I looked in the forum archives and didn't see any reference to this in any previous threads/posts. If it is a repeat, then I apologize. I thought it was interesting and thought perhaps some of you might find it interesting as well.

members.aol.com/SynergyHN/roundworm

Click here to return to: CISRA--SynergyHN Home Page

### A Parasitic Roundworm Linked to Chronic Fatigue Syndrome

by J. C. Waterhouse, Ph.D.

(Disclaimer: This material is intended for information only and is not medical advice. Neither CISRA nor the editor receive funding from any doctor, lab or manufacturer of any medication or associated products.)

### **Abstract**

This article reports on the work of Lawrence Klapow, Ph.D., who has found evidence of a new roundworm species, provisionally named Cryptostrongylus pulmoni, in patients with chronic fatique syndrome (CFS). In a blinded, controlled study (1, 2), the roundworm adults and larvae were found in sputum of almost half of the CFS patients and none of the controls. This association with CFS was found to be highly statistically significant (P~0.001). Currently, commercial laboratory tests and drug treatment trials await the results of recent efforts to characterize the roundworm's DNA. It is thought that the parasite reproduces in the lungs, giving rise to larvae which migrate to the intestinal tract. Some roundworm larvae burrow through the intestinal walls into tissue and migrate back to the lungs to develop into adults, which then produce larvae to continue the cycle. Only 3 patients have attempted treatment with anti-roundworm drugs so far, and although symptom improvement appears promising, it appears that this proposed new roundworm may be difficult to completely eradicate. The most promising drugs based on this very limited anecdotal evidence appear to be ivermectin and inhaled low dose thiabendazole (now, it would have to be replaced by albendazole). Oral thiabendazole was too toxic and should not be used. The usefulness of oral albendazole remains to be further evaluated along with other drugs. The use of ivermectin was accompanied by itching and/or diarrhea, perhaps indicating a roundworm die-off reaction. Unlike many other helminthic parasites, this species does not appear to produce elevated eosinophils or IqE when the illness becomes chronic. Instead, it is thought to use strategies that certain other roundworm species have been found to use that elevate the TH1 immune response and inhibit a more effective anti-parasitic TH2 response. Klapow also proposes ways in which infection by this species may be able to explain a number of common CFS characteristics, like initial respiratory infection, low cortisol, frequent gastrointestinal complaints, leaky gut, low blood pressure and neurological symptoms. This author concludes with some speculations on alternative treatment and diagnostic possibilities.

Editorial Note (Added in 2006 and updated in April, 2008): For an update on my views of roundworm treatment at present and treatment of chronic fatigue syndrome in general, see the transcript of a talk I gave in 2005. I give an overview of what has helped me most, with an emphasis on a new approach, called the Marshall Protocol (MP). For many, the MP seems to be able to reverse the immune dysregulation that may account for susceptibility to multiple chronic infections. My own view is that there needs to be more research on treatment of this roundworm before I would recommend attempting it. Careful and thorough attention to identifying and controlling food sensitivities (see Food Allergy/Sensitivity Testing), followed by the use of the Marshall Protocol appears to be a good alternative approach at this time, while awaiting future research on this proposed new roundworm species.

#### **CFS Introduction**

Chronic fatigue syndrome (CFS) is a disease characterized by substantial fatigue of greater than 6 months duration, muscle aches and cognitive problems and frequently involves sore throat, tender lymph nodes and unrefreshing sleep. Currently no established cause has been found, but there are many proposed causes, some infectious and some not. Some have proposed there may be subgroups of CFS patients with different causes. One hypothesis is that many cases of CFS are caused by a new species of roundworm, provisionally named Cryptostrongylus pulmoni (C. pulmoni). An invertebrate zoologist, Lawrence Klapow, Ph.D. has found evidence for this roundworm during years of research, and his findings have been further supported by the results of a blinded controlled study (1). This article will first discuss Klapow's hypothesis that a chronic infection caused by C. pulmoni is the source of many of the prominent symptoms of CFS and then explore possibilities for treatment.

### **CFS Historical and Scientific Background**

Klapow's discovery began through a discussion with an immunologist regarding a CFS patient. The patient was thought to possibly have a roundworm infection, due to a slightly elevated level of eosinophils in the patient's blood and some unusual rashes (1, 3). A series of culture and stool tests for the known type of roundworm suspected to be the cause (a threadworm, Strongyloides stercoralis) came up negative. However, since Klapow was aware of chronic parasites that migrate between the digestive and respiratory tracts, he decided to do microscopic investigations of the sputum coughed up by the patient. He found what he believed to be a previously unknown roundworm and gave it a provisional name, Cryptostrongylus pulmoni, which means "hidden lung worm."

Roundworms (also called nematodes or helminthes) cause a variety of acute and chronic infections around the world, some serious and some mild and self limited (1, 4). There is a history of roundworms being introduced into Western countries following Asian wars, when soldiers and immigrants traveled between Europe and Asia (1). In the case of the proposed new roundworm, C. pulmoni, Klapow's study of the anatomy of C. pulmoni suggests it may be related to the trichostrongylid genus Nycteridostrongylus (1, 5, 6). This type of roundworm infects small mammal's intestines in Southeast Asian and Northern Australian rain forests. This resemblance, if confirmed, together with the increase in CFS cases in the 1980's (7) suggests that soldiers and immigrants from the East Asian Tropics might have introduced the disease into the U.S. and Europe following the French and American involvements in East Asia. The tendency to latency and slow development of symptoms would make it hard to clearly identify causal patterns. There is some support among data on soldiers returning from Southeast Asia developing CFS-like symptoms (8), but clearly much more research needs to be done on this. Taking a longer term historical view, in the Victorian era, there may have been a similar situation in which soldiers returning from Indochina brought a roundworm infection back to France (1). Many American women traveling to France in that era could then have brought back roundworm infections that had originated in Asia. This could possibly account for the "neurasthenia" observed in that era ( some have compared neurasthenia to CFS). Whether C. pulmoni was a source of infection at that time is unknown. For another example, in 1974, chronic cases of Strongyloides stercoralis, another roundworm, were diagnosed in some British soldiers who had been POWs in Southeast Asia in WWII (9).

### **Evidence From a Blinded, Controlled Study**

Evidence for the presence of this new roundworm has been reported in a blinded, controlled study, for which several prominent CFS researchers referred patients (e.g., Dr. Anthony Komaroff, Dr. David Bell and Dr. Roger Mazlen) (1, 2). In the first part of the study, 5 of the 11 CFS patients (45%) and none of the 6 controls were found to have the C. pulmoni larvae in a 3-day sputum sample (2). In the second larger blinded study that included additional CDC-defined CFS patients and controls, the same pattern was obtained. A total of 14 out of 30 (47%) of the CFS patients tested positive, while none of the 21 controls tested positive for the roundworm (1). The association between C. pulmoni infection and CFS was highly significant. It is extremely unlikely that the results could have been due to chance. The actual percentage who have the roundworm infection is likely to be somewhat higher, since the larvae are rare and hard to detect, requiring somewhat specialized techniques and considerable time at the microscope in distinguishing the adults and larvae from debris. However, despite difficulties, Klapow has succeeded in photographing C. pulmoni and distinguishing its anatomical characteristics (10).

In the larger study of the roundworm in CFS (1), questionnaires were filled out by the participants to see if any relationship could be found between the types of patients, their symptoms and whether or not their sputum tested positive for C. pulmoni. The data reveal no significant differences in symptoms between those patients who tested positive and those patients who tested negative. There was not a higher incidence of coughs (30-40%) or travel outside the U.S between C. pulmoni positive and negative patients. Patients showed highly statistically significant differences from controls in reported allergies, spotted rashes and bowel symptoms, as well as in the incidence of C. pulmoni (significance levels ranged from P-0.001 to P<0.001). The relatively low occurrence of significant coughing in C. pulmoni positive patients might relate to the lack of a very effective immune reaction to the roundworm, as discussed below.

Why Might a New Roundworm Species Be So Elusive and Chronically Debilitating?

There are a number of factor that might explain why this new roundworm has not been identified before. Among the reasons may be its rarity, small size, lack of an esophageal bulb (which makes it harder to identify) and its likely period of latency. There are also some very interesting characteristics of roundworm infections that have been discovered in other roundworm species that could contribute to a delay in discovery. For instance, C. pulmoni may be like other roundworms that have been found to have ways of manipulating the mammalian immune system to keep it from responding effectively to eliminate it (1). One consequence of these evasive tactics is the lack of much increase of the typical indicators of a TH2 anti-parasitic immune response in the blood of patients chronically infected with roundworms (e.g., elevated eosinophils and IgE). This may be explained by the fact that some roundworm species have been reported to produce a substance similar to interferon-gamma which might stimulate the TH1 anti-viral pathway (1, 11). The stimulation of the anti-viral pathway then suppresses the anti-parasitic pathway, thus suppressing IgE and eosinophils. This has been found to occur through soluble CD23, an IgE binding protein which reduces serum IqE (12). The consequence of these tactics is a picture fairly similar to what is observed in CFS. There is an activation of the anti-viral pathway (13), fairly low levels of increased eosinophils sometimes observed early in the illness, and an actual decrease in IqE relative to the normal population as the illness becomes chronic (14). The IqE levels are often reported as normal, however Klapow argues that they may really reflect a decline in IgE. A history of allergies is often reported by patients (1), which should raise IqE above normal. However the actual data indicate that IgE is lower than normal in most studies, according to Klapow, which suggests active IgE suppression.

There are also a number of characteristics of certain chronic roundworm infections that would result in fairly large effects from relatively low levels of organisms (1). And most of these effects correspond fairly closely with the situation in CFS. For instance, C. pulmoni might be like other roundworms that have been shown to secrete substances that could have quite negative effects on infected humans. The secreted substances include one that mimics the limbic system neuropeptide

HCNP (hippocampal cholinergic neurostimulatory polypeptide), which is thought to be involved in the immune system and memory (1, 16, 17, 18). Other potentially harmful substances include ones that are similar to the IgE inhibitor CD23 (19), acetylcholinesterase (20), VIP (vasoactive intestinal polypeptide) (1, 21), and some that are inducers of autoimmunity (22).

Substances secreted by the roundworms could act in a number of ways to affect the immune system, for instance, they could induce their host to have immune reactions to a wide variety of mostly harmless antigens (1, 23) or cause autoimmune reactions (1, 24). The VIP-like substances can cause MAST cells to become unstable and overreact to antigens (25). This might be involved in the food and chemical sensitivities reported in CFS patients. A VIP-like substance secreted by the roundworm might also influence autonomic cardiovascular reflexes and result in symptoms like those experienced in CFS involving abnormal regulation of blood pressure (orthostatic intolerance or POTS and related conditions). A sort of poisoning of cholinergic neurotransmission might occur through the roundworm's secretion of antigenic forms of acetylcholinesterase, and this might increase sensitivity to inhaled chemical irritants (1).

The probable life cycle characteristics of the roundworm also may be important in yielding clues to the nature of CFS. It is thought to enter the body through ingestion of food contaminated with feces containing C. pulmoni (1, 10). Then, the larvae pass through the intestinal tract, from which they burrow through the intestinal walls into tissue. Apparently, some larvae make it to the lungs to develop into adults and then produce another generation to continue the cycle. In the process of burrowing through tissue, the larvae create tiny holes in the gut, which could allow other infectious viruses, bacteria and fungi to enter the tissue more easily. Roundworms themselves can also serve as reservoirs for other microbes, such as viruses (1). This phenomenon, combined with the immunosuppressive effects already discussed, could help allow other infectious microbes, including opportunistic ones, to gain entry to the tissues, take hold and become more serious. This could help to explain the wide variety of infectious agents that have been identified in various subsets of CFS patients (26).

Further Possible Connections of Roundworms With CFS and Other Illnesses

It has been found that eosinophils are physiologically activated in CFS (1, 27). However, they are not much increased in number, being only marginally elevated in only about 20% of patients. This would be consistent with an immune suppressing roundworm infection.

CFS patients have considerable cognitive difficulties and these might be due primarily to cytokines or substances secreted by the proposed roundworm. But it should be noted that in other species, chronic disseminated roundworm infections can lead to roundworms infecting the brain (4). Although the postulated life cycle of C. pulmoni involves an ability to reproduce only in the lungs, there is also evidence of a migratory larval stage which could invade many different tissues, including the brain.

There is also an interesting way in which roundworms might interact with emotions and stress responses that some research has linked with CFS, as well as a variety of other illnesses. Klapow states that it "is known that roundworm infections can produce emotional symptoms, likely due to their cytokine and neurotransmitter secretions" (1, 18), and this may be due to secretion of HCNP (see above). In addition, elevated cortisol is known to potentially worsen roundworm infections (hyperinfection) (1, 4). Thus, a stressful event that produced a period of elevated cortisol might have seemed to initiate the CFS because it might have worsened an existing latent roundworm infection to the point where symptoms became obvious. In those with CFS, who have, by definition, been sick for more than six months, many patients have moved into another phase of illness where they have lower serum cortisol. This may be an attempt by the body to fight the roundworm infection, since it is known that animals with chronic roundworm infections have low cortisol (1, 28, 29). This low cortisol is thought to be an attempt by the body to eliminate the parasite by countering

the strategies used by the roundworm. In other words, high cortisol lowers the anti-parasitic immune response, so the low cortisol of the animal would increase the anti-parasitic response. According to this theory, long term use of cortisone in CFS, even at fairly low levels, might be counterproductive (1).

Whether or not C. pulmoni can account for CFS, roundworms of this sort might be a useful area for research in a variety of illnesses, especially given the recent increases in knowledge of their potential for evading and manipulating the immune system. Also, recent increases in international travel/immigration and the past history of introductions of roundworms into Western countries support the view that more research on helminthes is needed. The lower prevalence of the more obvious helminthic infections in wealthier countries might explain the slowness in research on these disease agents. There may well be other unknown roundworms that play roles in diseases that are not yet suspected.

### **Potentials for Treatment**

Only 3 patients are currently known to have attempted treatment of C. pulmoni with antiroundworm drugs. Improvement through drug treatment appears promising, however long term treatment in one patient indicates that the roundworm may be difficult to completely eradicate (5). The most promising drugs based on this very limited anecdotal evidence appear to be oral ivermectin and the inhalation of an amount of dissolved thiabendazole equal to approximately 5% to 7% of the oral dose. Now, **thiabendazole** would have to be replaced by the less toxic albendazole or another more soluble drug (inhaled forms of anti-parasitic drugs are not commercially available at present). Oral thiabendazole was too toxic and should not be used. Oral albendazole, while having significant side effects, might prove to be useful in combination with other drugs. Inhaled low dose thiabendazole in one patient seemed to have a delayed effect resulting in marked improvement after weekly use for 4 to 5 months. The delay in improvement could be due to the long life cycle of the roundworm, which means that larvae in the rest of the body can still cause symptoms. for a long time even if those in the lungs are killed. The use of oral ivermectin (brand name, Stromectol) was accompanied by itching and/or diarrhea, perhaps indicating a reaction to the die-off of the roundworm larvae. It might be that a combination of drugs used for a long time might be needed to eradicate the roundworm. However, based on the experience of two of the patients, it may be that treatment with ivermectin alone might reduce the severity of the illness significantly in only a few weeks or months.

There is not yet sufficient information to know what approach is best. Perhaps a weekly dose of oral **ivermectin** at the amount adjusted for weight (e.g., 6-21 mg) or perhaps several days in a row of **ivermectin** repeated every few weeks. Future research will have to determine what is most effective. The fairly low reproductive rate of the roundworm would probably mean that it would take intensive treatment for a fairly long time (perhaps a year or more) before drug resistance would be likely to develop. Two of the patients did take enough of the anti-roundworm drugs to feel they improved significantly (some improvement in one patient in a few weeks and in the other in a few months). One patient used several drugs and the other used only ivermectin. But, of course, this is only anecdotal information and must be interpreted very cautiously. Ivermectin has some potential side effects, but they are not as severe as some other drugs taken by CFS patients on an experimental basis. Inhalation of drugs other than **thiabendazole** might also have potential, however, one patient's trial of inhaled **ivermectin** did not seem to be very successful (30).

### Characterizing the Roundworm's DNA and the Development of a Commercial Test

The development of a commercially available test and a complete characterization of the DNA of C. pulmoni has been slowed during the first few years following Klapow's discovery by the poor quality of most of the specimens, which were often partial or decayed, and the long hours needed to find each specimen (sometimes as many as 200 hours per patient). Only very recently, Klapow was able to find several fresh C. pulmoni specimens, which lead to greater interest among roundworm specialists in academia. Now, specialized techniques can be employed to characterize specimens by DNA and electron microscope imaging. However, the availability of a commercial test for the roundworm may still be a year or more in the future. Those researchers interested in trying to find C. pulmoni in sputum might go to the web site (members.tripod.com/lak123), where there are diagrams of male and female C. pulmoni and detailed instructions on the techniques that might be used to process and examine samples from patients. Unfortunately, due to the many hours needed to use the microscope to find specimens and the time needed for his current research, Dr. Klapow can not test patients for C. pulmoni at the present time. When a test is available, the above web site will announce it. (Note: Doctors and researchers who would like to reach Dr. Klapow can do so through the author of this article at jcwat101@aol.com).

Some Speculations on Diagnosis and Treatment

So far, I have been summarizing the work of Dr. Klapow and the research on other roundworms that he describes. The current lack of a diagnostic test and the scarcity of information on the best treatment approach is disappointing for CFS patients. I have a number of speculative ideas for what doctors and patients might do if they want to try to diagnose, treat and help in research in this very experimental area. I am not advocating or recommending any treatment approaches, just sharing my own speculations on possibilities for what might be done while awaiting definitive answers.

First, in the area of diagnosis, those doctors and researchers with the abilities and supplies needed, might try to identify C. pulmoni microscopically according to the instructions at: www.members.tripod.com/lak123. In addition, the level of suspicion of a roundworm infection in a patient might be increased by a number of symptoms and signs. Although none of these signs and symptoms might prove to be necessary, and there is not yet sufficient data to prove they increase the likelihood, nevertheless, on theoretical grounds, they might increase the chances of a roundworm infection in a CFS patient. They include travel to, or direct or indirect contact with Southeast Asia or Northern Australia or their immigrants, spotted rashes, low grade eosinophilia at some point in the illness, pre-CFS IqE allergies that later developed into lower IqE levels, extensive non-IgE food and/or chemical sensitivities, gastrointestinal symptoms such as irritable bowel syndrome, a respiratory infection coinciding with the beginning of CFS symptoms, the role of high levels of stress in the beginning of the illness, or a persistent cough. Also, some of the substances that might be secreted by C. pulmoni might be investigated. This is particularly experimental, but a doctor might look for elevated CD23 lymphocyte markers. Or qualified researchers might look for substances that are similar in structure to HCNP, VIP or some of the other substances secreted by roundworms, as discussed above.

Another possibility for both diagnosis and treatment is the empirical therapeutic/diagnostic probe. If a doctor feels it is warranted in a particular CFS patient, he/she could prescribe a dose of ivermectin. If the patient has itching or diarrhea, or some other symptom that suggests a die-off reaction, soon after the ivermectin is given, then this might be evidence of a roundworm infection. The patients who have tried this drug indicate that no benefit is achieved by only one dose. If ivermectin was repeated on a number of occasions, and the die-off reaction became gradually less and/or the patient improved, this might support the role of a roundworm. For those using ivermectin intensively, it would probably be advisable to do periodic blood tests, in case side effects develop.

For other diseases, ivermectin is typically used at a rather low frequency and so there is not much data on more intensive use in humans, although use in animals has been fairly extensive.

There is also a way in which one could test the effect of herbal and other potential anti-roundworm agents that might have less side effects than ivermectin. If one gave ivemectin on 2 or 3 occasions and found a consistent die-off response, then one could give the alternative substance, and then by comparison with the effect of the ivermectin, one might attempt to judge whether the alternative substance had anti-roundworm effects. There is some research in the animal literature suggesting some herbs have an effect on roundworms (31). For instance, some dewormers used include some species of Artemesia (wormwood), as well as the herb, Chenopodium ambrosiodes (goosefoot). But of course, to establish the safety and efficacy of alternative agents against potential C. pulmoni infections in humans, much research would be needed.

It also may be that some dietary components of Asian food might have anti-roundworm effects. For instances marsala, a spice used in Southeast Asian cooking, contains catechin a condensed tannin (CT) also found in green tea. CT's have been reported to reduce nematode survival and reproduction (30). There is also some suggestive evidence that the alcohol extract of Pau D'arco may produce lowered body itching and might have a beneficial anti-roundworm effect (30). However, it may be difficult for some patients to tolerate herbs due to the tendency for food sensitivities in CFS (discussed below). For those interested in researching antihelminthic alternatives, further searching in the veterinary and herbal literature would probably be helpful.

Another potentially beneficial approach would be to focus on reducing the negative effects of immune overactivation that might be caused by the roundworm, which might be responsible for a large part of the symptoms experienced. This could be done by focusing on only consuming foods with the least potential for provoking sensitivity reactions, as well as avoiding exposure to chemical irritants. Most CFS patients do not have seem to have many IgE-mediated allergies, but they tend to have more non-IgE sensitivities that utilize Type II, Type III and Type IV immune reactions, sometimes called delayed-type hypersensitivities. The approach of minimizing these immune hypersensitivities has been helpful to many with CFS and a wide array of conditions (32, 33). It is likely that most CFS patients have more food and chemical sensitivities than they realize due to "masking" effects and could benefit greatly from going further in identifying their optimal diet and reducing chemical exposure (33, 34). A great deal can be done by the patient at home to reduce immune sensitivity reactions, by using the pulse test and other methods, in addition to laboratory and clinical methods (33).

For example, one CFS patient who tested positive for C. pulmoni has improved greatly over many years of reducing food and chemical immune hypersensitivities (this patient is still very far from well and must be on a very restrictive diet). It is also interesting that whenever a period of minimal food sensitivity exposure is achieved in this patient, with less anxiety, diarrhea and insomnia, there is a corresponding increase in sinus symptoms, such as congestion, sneezing and coughing. Thus, there is a tendency to alternate between types of symptoms. This occurs in the absence of any change in inhalant allergy exposure and in an environment in which inhalant exposures have been minimized with a good air filter (though admittedly, the environment is not as pure as a few chemically sensitive patients have attained). This type of alternation of symptom types has been described as alternating between symptom states defined by Randolph and Moss as alternating between +1 or +2 and -1 or -2 (34).

There may be other explanations for these alternating states, but it might be that in some patients, the alternation could be due to a C. pulmoni infection. The non-IgE food sensitivity reactions might be increasing both cortisol and TH1 immune reactions (with symptoms of anxiety, diarrhea and insomnia), which temporarily suppresses the TH2, anti-parasitic response. When the food sensitivity reactions and associated TH1 immune reactions are reduced, the TH2, anti-parasitic response can then increase and produce the sinus symptoms as part of the immune system's attempt to eradicate

the roundworm. The extreme difficulty this patient has experienced in completely eliminating the food sensitivities could be due to fighting an uphill battle in the context of the multiple effects on immune function of the roundworm infection (see above). The roundworm's proposed manipulation of the patient's immune system, as well as its burrowing through the gut wall making it truly a "leaky gut," may have made any fight to eliminate the sensitivities much harder and perhaps impossible with food/chemical sensitivity reduction techniques alone. One might speculate that some patients with milder or shorter-term C. pulmoni infection and few or no coinfections, perhaps could get well using the allergy/sensitivity reduction alone.

### Possible Relationship to a New Treatment Approach For TH1 Dominant Diseases

Now, I will briefly mention my own view on how research on this roundworm might relate to some recent research on autoimmune disease, Lyme disease, fibromyalgia and CFS that emphasizes treating the cause of excessive TH1 inflammation. This approach proposes the causative role of intracellular cell wall deficient (CWD) forms of bacteria that seem to use an excessive TH1 inflammatory response to hide from the immune system (35, 36). I would suggest the hypothesis that perhaps half of the CFS patients, possibly the sickest ones, may have a symbiotic situation in which the roundworm and CWD bacteria are able to benefit from each other's activities by promoting an excessive TH1 response The roundworm might directly cause a TH1 response through secreting substances that stimulate IFN gamma (11) or through other substances that stimulate autoimmunity and overreactions to antigens in general (23, 24). Indirectly, they may promote a TH1 response by burrowing through the intestinal wall, allowing other microbial pathogens (including CWD capable bacteria) and food and chemical antigens easier access to the bloodstream and tissues and thus provide even more antigens to stimulate and overactivate the immune system. By contributing to TH1 inflammation, C. pulmoni might be promoting the proliferation of CWD forms of bacteria, while itself benefiting from the TH1 response's suppression of the anti-parasitic TH2 response.

The research of Marshall et al (35, 36) has led to the development of the Marshall Protocol (MP) for diseases of excessive TH1 inflammation. The Marshall Protocol involves the use of immune modulation to reduce the excessive TH1 response, combined with a series of low dose antibiotics, introduced very gradually. After having good success in treating the autoimmune disease sarcoidosis, it has only recently begun to be used in CFS and Lyme patients and has shown some positive initial results. It is possible that integrating anti-roundworm treatment into the Marshall Protocol in some way might be worth considering at some point. Perhaps some of the sickest CFS patients might benefit from anti-roundworm treatment and/or reducing the food and chemical sensitivities it may be fostering, before trying the MP. The MP is quite difficult for some patients at the beginning and some patients have had to stop it and then try again later. The very sickest of the CFS population are, for the most part, probably not those who are currently trying the MP, as they are typically too ill to study it and participate in the free Internet discussion groups (marshallprotocol.com or sarcinfo.com).

The idea that some patients have both the proposed roundworm infection and CWD infection is not purely theoretical. A number of CFS patients that Klapow has found to be positive for C. pulmoni have also tested positive for Lyme disease (30). The Lyme disease spirochete is known to also exist in CWD forms, the form of bacteria that the Marshall Protocol was designed to treat. As is widely known from experience with HIV-AIDS, there are certain immune suppressing organisms that make it more likely for one to have multiple infections and it may be that this is a role that chronic infection with C. pulmoni plays in CFS. Whether it is involved in other diseases is an open possibility, as the only patients tested for this roundworm so far are CFS patients.

The way in which the MP is currently being used fits in some ways with the state of the roundworm diagnosis situation. This is because with the MP, the Jarisch-Herxheimer reaction (bacterial die-off reaction) in response to antibiotics is used as an indicator that a particular antibiotic is killing CWD bacteria. The use of ivermectin as a diagnostic/therapeutic probe (see above) to see if a die-off reaction occurs is somewhat similar. At present, it is unknown how ivermectin might interact with the immune modulation of the MP and it should not be combined with the MP until more is known. Caution is warranted because the MP intensifies the action and die-off reactions of certain antibiotics (thus requiring the use of quite low initial antibiotic dosages). However, prior treatment for those CFS patients who are too ill to begin the MP might be an option to consider. Or if it were found that a CFS patient was not progressing well, the patient might stop the MP temporarily and try an anti-roundworm therapeutic probe or treatment trial, and then resume the MP.

From the perspective of a hypothesized C. pulmoni infection, combining the MP with anti-roundworm treatment might get around the potential problem of anti-roundworm drug resistance or difficulty of complete eradication. These difficulties have been observed in animals with other chronic roundworm infections. Also, there is some indication that in the one patient who has attempted long term treatment of C. pulmoni with several anti-roundworm drugs, some drug resistance may have developed. It might be that the immune modulation of the MP and its reduction of the CWD bacteria, might help a promote a better immune response to the roundworm parasite that would aid the anti-roundworm drugs sufficiently to completely eradicate the roundworms.

The above sections have discussed a type of symbiotic relationship between CWD bacteria and C. pulmoni in which they each affect the human immune system in a way that helps themselves and each other. Another possibility in CFS is a sort of symbiotic relationship between certain viruses and roundworm infections. For example, there is evidence of a significant association between the roundworm infection strongylosis and HTLV1 viral infections (37).

#### **Conclusions**

In conclusion, for most patients, it would probably be wise to wait until DNA analysis is able to confirm the work on C. pulmoni done so far and a commercial test for it is available. Then, research trials will be done to try to determine what drug or combinations of drugs are most effective for treatment. But for those doctors, researchers and patients who want to contribute to research in this area and do not want to wait for several years, some speculations have been presented as a starting point. It is hoped that these research ideas will lead to information that will help in the process of determining what lab tests and anti-microbial agents would be most useful, as well as gain further evidence regarding the role of this "hidden lung worm" in chronic fatigue syndrome.

(Note: Any doctors, researchers or patients who would like to share any experiences they may have related to the above diagnostic and treatment possibilities are encouraged to send accounts to me at: jcwat101@aol.com), and if they seem significant, I may compile them for a future article for the purpose of advancing research in this area. All personal information will be kept confidential.)

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#### **Treatments**

http://forums.prohealth.com/forums/index.php?threads/cryptostrongylus-pulmonianyone.190413/page-3

Dr Wright found borrelia and cryptostrongylus pulmoni in my blood, so prescribed 3 months of Doxycycline and Metronidazole, plus 6 doses of Ivermectin(taken once a month over 6 months).

Well, I've finished the 3 months of **Doxycycline and Metronidazole**, still have 2 more doses of **Ivermectin** to take.

http://www.disabled-world.com/health/water-diseases.php

http://www.anapsid.org/cnd/diagnosis/index.html

http://forums.phoenixrising.me/index.php?threads/treatment-for-worms-has-helped-my-gut-a-bit-but-why.11327/page-5

She said **ivermectin and praziquantel** were most effective. She took them in sequence, not together.

<u>Mimosa pudica</u> powder is an Ayurvedic herb that is 30 times stronger than the best medical drug. Starting with 1/2 teaspoon twice a day two days a week and working up to 1 teaspoon daily for 3 months.

Can use rectal application of 1 capsule of BioPure Freeze-dried garlic, 100mg artemisinin, 1 teaspoon phospholipids with pure water in a bulb syringe from the local drugstore. Make the mix liposomal using the ultrasonic method used with the Klinghardt Cocktail. Hold it. This cleans the rectum and then gets absorbed such that many start to see visible evidence of worm elimination. A good first step in parasite elimination.

Parasite treatment is best done with concurrent colonic therapy. 2 colonics per week; 1 day after the other.

Nail biting is a sign of parasites.

Alinia and Albendazole both cross the blood-brain barrier.

Deworming is significant in ASD kids. Every biofilm has worm DNA in it.

The more you deworm, the longer you live and the healthier you are.

http://www.google.com/patents/US5645819

## **D. Comparison with Other Persistent Nematode Infections**

FIG. 2 illustrates the life cycle of human hookworms (Necator americanus and Ancylostoma duodenale) and threadworm (Strongyloides stercoralis) in comparison to the newly identified hidden lungworm (C. pulmoni (n.gen., n.sp.)). In hookworm disease, infective filariform larvae from contaminated soil penetrate the skin, most often the skin of bare feet. Upon finding a vein, they are carried by the venous circulation to the right side of the heart. They are then transported through the pulmonary circulation to the lungs, where they bore through the alveolar tissue and enter the bronchial airways. After climbing up the broncho-tracheal tree, the larvae are swallowed with sputum in the throat and enter the host's digestive system. Alternatively, larvae can directly enter the digestive track through an oral route following ingestion of contaminated food. Once in the intestine, the larvae attach to and feed on the intestine's epithelial surface and mature into sexual adults.

Sexually produced rhabditiform larvae, non-infective larvae that engage in a free-living cycle usually outside the host, are produced and discharged in the feces. After a few days in the soil environment, rhabditiform larvae molt into filariform larvae, which may then infect other hosts. Unless re-infection from the external environment occurs, infection will last no longer than a single generation. Hookworm infection has, for the most part, been effectively controlled in the United States with anthelmintic agents (e.g., pyrantel pamoate and mebendazole) and with public health education.

Threadworm infection is in many ways an extension and elaboration of the hookworm life cycle, further evolved to increase the duration of host infection. Initial infection follows the general pattern described for hookworms. However, unique adaptations in the threadworm life cycle allow it to reinfect the same host through the previously described autoinfection process. This process involves the production of larvae (through asexual parthenogenesis, a form of nonsexual reproduction, in this species) which mature into the infectious filariform stage while still in the intestine. These infective larvae either bore out of the host's intestine into the surrounding tissues (internal autoinfection) or, after passage through the rectum, bore into the perianal skin (external autoinfection). Once the larvae penetrate a vein, they are transported back into the digestive system via the heart, lungs,

tracheal, and esophageal circuit previously described. If the autoinfective route becomes established, multiple generations of threadworms can successfully infect the host. Threadworm infections of several decades and even lifelong infections have been reported (See generally, Liu & Weller, "Strongyloidiasis and Other Intestinal Nematode Infections," Infectious Disease Clinics of North America, Parasitic Diseases 7(3):655-82 (1993)]. As some larvae leave through the feces, stool and culture analysis can detect threadworm disease. However, single stool samples are often negative (approximately 80% of the time) due to the scarcity of fecal larvae in low and moderate level infections; such infections are often detected only after repeated attempts at diagnosis.

Intensive threadworm autoinfection can lead to widespread deposition of larvae, well beyond that of the "normal" respiratory and digestive system routes, into every part of the body. Disseminated infections of this sort occur when larvae enter the arterial circulation and are transported to all tissues via the systemic blood circulation. Intensive autoinfections that lead to widespread dissemination are very dangerous and often fatal. [See Genta, "Global Prevalence of Strongyloidiasis: Critical Review With Epidemiologic Insights into Prevention of Disseminated Disease," Rev Infect Dis 11(2):755-67 (1989)].

C. pulmoni (n.gen., n.sp.) has taken the autoinfective process to the extreme. All larvae apparently bore out of the intestine to re-infect the host and give rise to reproductive forms in the lungs. No larvae are found in the stool (as evidenced by the subject's autoinfective skin rashes and negative stool tests), the likely reason why this roundworm has not been discovered heretofore.

A possible rationale exists that explains the selective advantage of giving up fecal transmission as a means of perpetuating the species. This rationale relates to the energetic demands of reproduction. C. pulmoni (n.gen., n.sp.) may have taken an evolutionary course where survival was enhanced by conservation in the host (e.g., autoinfection), and highly precise oral transmission of a few larvae rather than wide dispersal of many more larvae in fetes. The end result is an exceedingly small adult form (both in absolute size and relative to the size of its larvae), implying low reproductive output, which has remained hidden to human observers until now. The other chronic human intestinal nematodes, decidedly more "visible" in terms of size and reproductive output, where discovered more than a century ago.

III. Pharmaceutical Preparation and Delivery

#### A. The Characteristics of Thiabendazole

Specimens of C. pulmoni (n.gen., n.sp.) were first recovered following treatment with oral thiabendazole. Thiabendazole is a benzimidazole-derivative anthelmintic agent that is structurally related to mebendazole. [See generally, AHFS Drug Information 94, supra]. Thiabendazole has the following chemical structure: ##STR1## Though not fully elucidated, thiabendazole's mechanism of action likely involves inhibition of the helminth-specific enzyme, fumarate reductase. The agent is effective against most intestinal roundworms that are pathogenic to humans, including hookworms (Necator americanus and Ancylostoma duodenale) and threadworms (Strongyloides stercoralis).

Thiabendazole is rapidly absorbed through the gastrointestinal tract and has also been applied topically to treat certain infections. Thiabendazole is subject to extensive hepatic metabolism, and most of the agent and its metabolites are excreted in urine and feces within 24 hours of administration. The drag has a  $pK_a$  of 4.7 and it is commercially available in both a tablet and a suspension; the suspension has a  $pK_a$  of 3.4-4.2. [See AHFS Drug Information 94, supra].

The adverse effects of thiabendazole are many, and up to one-third of patients receiving the drug in recommended doses experience at least one adverse effect. Thiabendazole's adverse effects are well known. [See, e.g., Physicians' Desk Reference, 45th Ed. (1991); AHFS Drug Information 94, supra]. The most common adverse effects include nausea, vomiting, and dizziness; less frequent adverse effects include paresthesia, psychic disturbances, and altered hepatic enzymes.

It is not uncommon for a recipient of an anthelmintic to expel roundworms from the nose and the mouth. For example, patients being treated with thiabendazole for ascariasis have experienced live worms in their mouths and noses. [See, e.g., AHFS Drug Information 94, supra]. Furthermore, even prior to administration of an anthelmintic, examination of the sputum of an individual suspected of having a roundworm infection may sometimes assist in diagnosis if that infection entails pulmonary manifestations; for instance, S. stercoralis larvae may sometimes be identified in one's sputum if the pulmonary system is involved. [See Medical Microbiology--An Introduction to Infectious Diseases, Ch. 49, supra]. However, there has never been a published report of a sputum sample containing C. pulmoni (n.gen., n.sp.).

#### **B.** Administration of Oral Thiabendazole

The subject undertook a therapeutic trial with oral thiabendazole that involved a dose of approximately 1.5 grams per day over a five-day period; this therapeutic trial was for the treatment of a presumptive S. stercoralis infection, the presence of which could subsequently not be confirmed. The subject reported several significant, and at times severe, responses to this treatment. In addition to the nausea and confusion which are common with thiabendazole, other symptoms suggested the presence of a disseminated roundworm infection. Such symptoms included strong focal itching in conjunction with the appearance of small blood spots on the face, legs, and neck. The blood spots suggest that oral drug therapy may have caused dispersed larvae to penetrate the host's skin.

Severe abdominal pain, thirst, and abdominal bloating on the fourth day of treatment was followed two days latter with the expulsion of dark brown, almost black, copious stools over the next several days. The tough elastic nature of the stool material, which resisted tearing when it was probed, along with the color of aged intestinal blood, suggests that it was actually part of the intestinal lining which was expelled as a result of the severe inflammatory reaction associated with oral thiabendazole treatment. Expulsion of portions of the epithelial lining of the gut has been reported for other roundworm infestations of the intestinal mucosa. [See generally, Klein The Parasites We Humans Harbor, Ch. 3 (1981)]. The subject also coughed up large amounts of frothy sputum during the last days of treatment, suggesting that the lungs were also infected. The sputum was not examined, as S. Stercoralis was believed to be the infective gent.

Following these expulsion type reactions, the subject was completely symptom-free until a minor relapse of fatigue two weeks later. A major attack involving prolonged fatigue and gastrointestinal pain and bloating occurred two weeks after the first minor re-occurrence.

An oral dosing regimen of 3 g of thiabendazole per day for 7 days (the recommended dose for treating disseminated strongyloidiasis) was eventually administered under the care of an infectious disease specialist. However, the subject experienced debilitating side effects following that oral regimen. In addition, orally administered thiabendazole did not effectively control the subject's infection, which was latter determined not to be caused by S. stercoralis (through Strongyloides IgG antibody testing). As a result, other means of identifying the subject's

infection were ultimately sought. These resulted in the aerosolized method, described infra, for the diagnosis of C. pulmoni (n.gen., n.sp.) infections.

It should be noted that C. pulmoni (n.gen., n.sp.) has also been recovered from the sputum of a member of the subject's household using the method described below. However, the household member had been virtually asymptomatic until the twelve months prior to isolation of C. pulmoni (n.gen., n.sp.) in the household member's sputum. All references to "the subject" are to the 50 year old male previously described.

Interpreted in the light of the now apparent C. pulmoni (n.gen., n.sp.) infection, the aforementioned observations offer important insights for directing further treatment. First, orally administered thiabendazole is potentially dangerous, and does not appear to kill all forms of C. pulmoni (n.gen., n.sp.). Second, re-infection is apparently possible within two weeks, which may represent the minimal interval between successive generations of the parasite.

#### **Fasciolis**

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3609237/

#### **Sharkman:**

Strongyloides is serious life and death stuff, see black in scars, on fore arms. Fenbendazole is the replacement for mebendazole in vet world years ago . I did moxi with Prazi premixed horse paste November and dec , it literally gave me a kidney infection from die off and had to go on doxy again .

This happened right b4 holidays, but I feel great now , wanted me to avoid Prazi until I was 99.9 percent symptom free . Her explanation was the kidneys are one of their safe zones but Prazi with moxi targets that area like a guided missile.

I am doing Fenben this month at 15 mg per kg for 5 days 2 weeks apart. She has read primates are quarantined when they are brought in this country and are treated with 50 mg per kg for 5 days , same as carnivores.

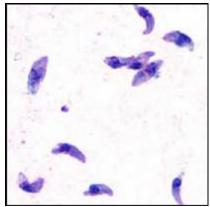
Fenbendazole according to her is the same as mebendazole except they added 1 compound of sulfur and adjusted 2 other compounds found in both by a slight margin. I only listen to her and her vet friends and backed up everything they provided me. As we seem to have both experienced severe infections , nematodes require lots of time with multi med treatments. Vets use Fenbendazole to treat ascariasis at same rate as strongyles. Vets have a new combo drug of Fenbendazole , ivermectin and Prazi for dogs and cats , it is the widest spectrum single monthly wormer developed.

never exceed canine doses of your meds , as she doesn't want anyone getting hurt. I will rotate back and forth each month from Fenben to moxi plus Prazi each month until 1000 percent healed and then I will worm bi monthly as a preventative from future infections from critters, bugs ,dirt ,food etc......

as they had severe strongyloides were afraid of dying and Drs couldn't get a handle on their infection , the morgellons myth. It was personnel for me to do that as I fell in that trap when I had no clue what the sores on my arms were with the threads.

scars from my severe stage 5 strongyloide parasitic infection

# **Toxoplasma gondii**



Toxoplasma gondii

http://www.bioone.org/doi/abs/10.1645/13-451.1

http://en.wikipedia.org/wiki/Toxoplasma\_gondii

http://www.naturecures.co.uk/toxoplasmosis.htm

## **CDC- Toxoplasmosis Frequently Asked Questions (FAQs)**

## What is toxoplasmosis?

A single-celled parasite called Toxoplasma gondii causes a disease known as toxoplasmosis. While the parasite is found throughout the world, more than 60 million people in the United States may be infected with the Toxoplasma parasite. Of those who are infected, very few have symptoms because a healthy person's immune system usually keeps the parasite from causing illness. However, pregnant women and individuals who have compromised immune systems should be cautious; for them, a Toxoplasma infection could cause serious health problems.

## What are the signs and symptoms of toxoplasmosis?

- Symptoms of the infection vary.
- Most people who become infected with Toxoplasma gondii are not aware of it.
- Some people who have toxoplasmosis may feel as if they have the "flu" with swollen lymph glands or muscle aches and pains that last for a month or more. Severe toxoplasmosis, causing damage to the

brain, eyes, or other organs, can develop from an acute Toxoplasma infection or one that had occurred earlier in life and is now reactivated. Severe cases are more likely in individuals who have weak immune systems, though occasionally, even persons with healthy immune systems may experience eye damage from toxoplasmosis. Signs and symptoms of ocular toxoplasmosis can include reduced vision, blurred vision, pain (often with bright light), redness of the eye, and sometimes tearing. Ophthalmologists sometimes prescribe medicine to treat active disease. Whether or not medication is recommended depends on the size of the eye lesion, the location, and the characteristics of the lesion (acute active, versus chronic not progressing). An **ophthalmologist** will provide the best care for ocular toxoplasmosis. Most infants who are infected while still in the womb have no symptoms at birth, but they may develop symptoms later in life. A small percentage of infected newborns have serious eye or brain damage at birth.

#### Intracellular

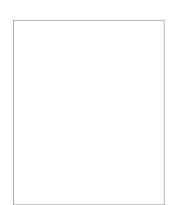
- Toxoplasma gondii (tŏk'sə-plāz'mə gŏn'dē-ī') is an obligate,
   intracellular, parasitic protozoan that causes the disease toxoplasmosis.
- Cellular stages During different periods of its lifecycle, individual parasites convert into various cellular stages, with each stage characterized by a distinct cellular morphology, biochemistry, and behavior. These stages include the tachyzoites, merozoites, bradyzoites (found in tissue cysts), and sporozoites (found in oocysts).

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 Description: "Toxoplasma gondii tachyzoites, stained with Giemsa, from a smear of peritoneal fluid

## **Tachyzoites**

Two tachyzoites, transmission electron microscopy[38]Motile, and quickly multiplying, tachyzoites are responsible for expanding the population of the parasite in the host.[39][38] When a host consumes a tissue cyst (containing bradyzoites) or an oocyst (containing sporozoites), the bradyzoites or sporozoites stage-convert into tachyzoites upon infecting the intestinal epithelium of the host.[40] During the initial, acute period of infection, tachyzoites spread throughout the body via the blood stream.[24] During the later, latent (chronic) stages of infection, tachyzoites stage-convert to bradyzoites to form tissue cysts.



Two
 apicomplexans,
 Toxoplasma gondii,
 within their host
 cell. Transmission
 electron microscopy

0

#### **Merozoites**

o An unstained T. gondii tissue cyst, bradyzoites can be seen withinLike tachyzoites, merozoites divide quickly, and are responsible for expanding the population of the parasite inside the cat intestine prior to sexual reproduction.[39] When a feline definitive host consumes a tissue cyst (containing bradyzoites), bradyzoites convert into merozoites inside intestinal epithelial cells. Following a brief period of rapid population growth in the intestinal epithelium, merozoites convert into the noninfectious sexual stages of the parasite to undergo sexual reproduction, eventually resulting in the formation of zygote-containing oocysts.[41]

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## **Bradyzoites**

o Bradyzoites are the slowly dividing stage of the parasite that make up tissue cysts. When an uninfected host consumes a tissue cyst, bradyzoites released from the cyst infect intestinal epithelial cells before converting to the proliferative tachyzoite stage.[40] Following the initial period of proliferation throughout the host body, tachyzoites then convert back to bradyzoites, which reproduce inside host cells to form tissue cysts in the new host.

## **Sporozoites**

Sporozoites are the stage of the parasite residing within oocysts.
 When a human or other warm-blooded host consumes an oocyst, sporozoites are released from it, infecting epithelial cells before converting to the proliferative tachyzoite stage.[40]

# Risk factors o human in have beer risk factor infection:

 Risk factors for human infectionThe following have been identified as being risk factors for T. gondii infection:

- 1.Exposure to or consumption of raw or undercooked meat[20][42][43] [44]
- 2.Drinking unpasteurized goat milk[42]
- 3.Contact with soil[7][43]
- 4.Eating unwashed raw vegetables or fruits[20]
- 5.Cleaning cat litter boxes[20]

- Sewage has been identified as a carriage medium for the organism.[45][46][47][48]
- Numerous studies have shown living in a household with a cat is not a significant risk factor for T. gondii infection,[20][43][49] though living with several kittens has some significance.

http://emedicine.medscape.com/article/229969-clinical#a0216

### **Symptoms**

# Only 10-20% of toxoplasmosis cases in adults and children are symptomatic.

- Toxoplasmosis is a serious and often lifethreatening disease in immunodeficient patients.
- Congenital toxoplasmosis may manifest as a mild or severe neonatal disease, with onset during the first month of life or with sequelae or relapse of a previously undiagnosed infection at any time during infancy or later in life. Congenital toxoplasmosis has a wide variety of manifestations.
- Patients may have cervical lymphadenopathy with discrete, usually nontender, nodes smaller than 3cm in diameter
- Fever, malaise, **night sweats**, and myalgias have been reported
- Patients may have a sore throat
- Retroperitoneal and mesenteric
   lymphadenopathy with abdominal pain may occur
- Retinochoroiditis is reported (an inflammation of the retina and choroid coat of the eye)

## The disease in these patients may be newly acquired or a reactivation. It may be characterized as follows:

- CNS toxoplasmosis occurs in 50% of patients Seizure, dysequilibrium, cranial nerve deficits, altered mental status, focal neurologic deficits, headache
- Patients may have **encephalitis**, meningoencephalitis, or mass lesions
- Hemiparesis and seizures have been reported

- Patients may report visual changes
- They may have signs and symptoms similar to those observed in immunocompetent hosts.
- Patients may have flu like symptoms and lymphadenopathy
- Myocarditis and pneumonitis are reported.
- •Toxoplasmic pneumonitis can occur Typical symptoms of a pulmonary infection, mirroring in particular P (carinii) jiroveci, including nonproductive cough, dyspnea, chest discomfort, and fever

Symptoms associated with reactivation toxoplasmosis are dependent on the tissue or organ affected.

## **Clinical findings include the following:**

- •Altered mental state
- Seizures
- •Weakness
- Cranial nerve disturbances
- Sensory abnormalities
- Cerebellar signs
- •Meningismus
- Movement disorders
- Neuropsychiatric manifestations

The characteristic presentation is usually a subacute onset, with **focal neurologic abnormalities** in 58-89% of cases. However, in 15-25% of cases, the clinical presentation is more abrupt, with seizures or cerebral hemorrhage. Most commonly, hemiparesis and/or speech abnormality is the major initial manifestation.

<u>Brainstem involvement</u> often produces cranial nerve lesions, and many patients exhibit cerebral dysfunction with disorientation, altered mental state, lethargy, and coma.

Less commonly, **parkinsonism**, focal dystonia, rubral tremor, hemichoreahemiballismus, panhypopituitarism, diabetes insipidus, or syndrome of inappropriate antidiuretic hormone secretion may dominate the clinical picture.

In some patients, **neuropsychiatric** symptoms such as paranoid psychosis, dementia, anxiety, and agitation may be the major manifestations.

Diffuse toxoplasmic **encephalitis** may develop acutely and can be rapidly fatal; generalized cerebral dysfunction without focal signs is the most common manifestation, and CT scan findings are normal or reveal cerebral atrophy.

**Spinal cord** involvement manifests as motor or sensory disturbances of single or multiple limbs, bladder or bowel dysfunctions, or both and local pain. Patients may present with clinical findings similar to those of a spinal cord tumor. Cervical myelopathy, thoracic myelopathy, and conus medullaris syndrome have been reported.

**Pulmonary toxoplasmosis** (pneumonitis) due to toxoplasmosis is increasingly recognized in patients with AIDS who are not receiving appropriate anti-HIV drugs or primary prophylaxis for toxoplasmosis. The diagnosis may be confirmed by demonstrating T gondii in bronchoalveolar lavage fluid.

**Pulmonary toxoplasmosis** occurs mainly in patients with advanced AIDS (mean CD4+ count of 40 cells/ $\mu$ L  $\pm$ 75 standard deviation) and primarily manifests as a prolonged febrile illness with cough and dyspnea. Pulmonary toxoplasmosis may be clinically indistinguishable from P (carinii) jiroveci pneumonia, and the mortality rate, even when treated appropriately, may be as high as 35%.

**Extrapulmonary** toxoplasmosis develops in approximately 54% of persons with toxoplasmic pneumonitis.

**Ocular toxoplasmosis**, ie, toxoplasmic retinochoroiditis, is relatively uncommon in patients with AIDS; it commonly manifests as ocular pain and loss of visual acuity. Funduscopic examination usually demonstrates necrotizing lesions, which may be multifocal or bilateral. Overlying vitreal inflammation is often present and may be extensive. The optic nerve is involved in as many as 10% of cases.

Other, uncommon manifestations of toxoplasmosis

- Panhypopituitarism and diabetes insipidus
- Multiple organ involvement, with the disease manifesting as acute respiratory failure and hemodynamic abnormalities similar to septic shock
- •Syndrome of inappropriate antidiuretic hormone secretion and possibly orchitis
- •Gastrointestinal system invasion of T gondii may result in abdominal pain, diarrhea, and/or ascites (due to involvement of the stomach, peritoneum, or pancreas)
- Acute hepatic failure
- Musculoskeletal involvement
- Parkinsonism
- Focal dystonia
- Rubral tremor
- Hemichorea-hemiballismus

#### **Congenital toxoplasmosis**

This is most severe when maternal infection occurs early in pregnancy. Approximately 15-55% of congenitally infected children do not have detectable T gondii —specific IgM antibodies at birth or early infancy. Approximately 67% of patients have no signs or symptoms of infection.

**Retinochoroiditis** occurs in about 15% of patients, and intracranial calcifications develop in about 10%. Cerebrospinal fluid (CSF) pleocytosis and elevated protein values are present in 20% of patients.

**Infected newborns have anemia,** thrombocytopenia, and jaundice at birth. Microcephaly has been reported. Affected survivors may have mental retardation, seizures, visual defects, spasticity, hearing loss or other severe neurologic sequelae.

**The prevalence of sensorineural hearing loss** is as high as 28% in children who do not receive treatment.[43]

## **Ocular toxoplasmosis**

Patients develop retinochoroiditis (focal necrotizing retinitis). They have a yellowish white, elevated cotton patch with indistinct margins. The lesions may occur in small clusters. Congenital disease is usually bilateral and acquired disease is usually unilateral.

## **Symptoms include the following:**

Swollen lymph glands, especially around the neck

Impaired vision - Either sudden or gradual

Muscle aches and pains

Blurred vision

Disorientation

Personality changes

Poor coordination

**Tremors** 

Seizures

Headache

Nausea

Fever

Generally feeling unwell

Tiredness and disturbed sleep patterns

Nerve damage

Inflammation of the brain (encephalitis)

Inflammation of the lungs

Inflammation of the heart muscle

- Scotoma
- Pain
- Photophobia
- Floaters
- •Red eye
- Metamorphopsia

Susceptibility

• People with T cell deficiencies are particularly susceptible to intracellular pathogens.

#### **Vectors**

- 1.by consuming raw or undercooked meat containing T. gondiitissue cysts[7] The most common threat to citizens in the united states it from eating raw or undercooked pork. It is possible to ingest the parasite through other products, but it is unlikely.[8]
- o2.by ingesting water, soil, vegetables, or anything contaminated with oocysts shed in the feces of an infected animal[7] Cat fecal matter is particularly dangerous. Just one cyst consumed by a cat will result in million of oocysts. This is why physicians recommend pregnant or ill persons do not clean the cat's litter box at home.[9]
- o 3.from a blood transfusion or organ transplant
- 4.or transplacental transmission from mother to fetus, particularly when T. gondii is contracted during pregnancy[7]
- Toxoplasma gondii- The parasite is able to run amok in the brain, creating circular abscesses visible on an MRI. Present in eyes, brain, heart, liver, Toxoplasmosis can cause headache, seizures, focal neurological deficits and mental status changes. Acquired from ingestion of uncooked/undercooked pork/lamb/goat with Toxoplasma bradyzoites, ingestion of raw milk with Toxoplasma tachyzoites, ingestion of contaminated water food or soil with oocysts in cat feces that is more than one day old.

#### History

O In 1908, while working at the Pasteur Institute in Tunis, Charles Nicolle and Louis Manceaux discovered a protozoan organism in the tissues of a hamster-like rodent known as the gundi, Ctenodactylus gundi.[10] Although Nicolle and Manceaux initially believed the organism to be a member of the genus Leishmania that they described as "Leishmania gondii", they soon realized they had discovered a new organism entirely. They named it Toxoplasma gondii, a reference to its morphology (Toxo, from Greek τόξον (toxon); arc, bow, and πλάσμα (plasma); i.e., anything shaped or molded) and the host in which it was discovered, the gundi (gondii). The same year Nicolle and Mancaeux discovered T. gondii, Alfonso Splendore identified the same organism in a rabbit in Brazil. However, he did not give it a name.[10]

- The first conclusive identification of T. gondii in humans was in an infant girl delivered full term by Caesarean section on May 23, 1938, at Babies' Hospital in New York City.[10] The girl began having seizures at three days of age, and doctors identified lesions in the maculae of both of her eyes. When she died at one month of age, an autopsy was performed. Lesions discovered in her brain and eye tissue were found to have both free and intracellular T. gondii'.[10] Infected tissue from the girl was homogenized and inoculated intracerebrally into rabbits and mice; the animals subsequently developed encephalitis. Later, congenital transmission was found to occur in numerous other species, particularly in sheep and rodents.
- The possibility of T. gondii transmission via consumption of undercooked meat was first proposed by D. Weinman and A.H Chandler in 1954.[10] In 1960, the cyst wall of tissue cysts was shown to dissolve in the proteolytic enzymes found in the stomach, releasing infectious bradyzoites into the stomach (and subsequently into the intestine). The hypothesis of transmission via consumption of undercooked meat was tested in an orphanage in Paris in 1965; yearly acquisition rates of T. gondii rose from 10% to 50% after adding two portions of barely cooked beef or horse meat to the orphans' daily diets, and to 100% after adding barely cooked lamb chops.[10]
- o In 1959, a study in Bombay found the prevalence of T. gondii in strict vegetarians to be similar to that found in nonvegetarians. This raised the possibility of a third major route of infection, beyond congenital and carnivorous transmission.[10] In 1970, the existence of oocysts was discovered in cat feces, and the fecal-oral route of infection via oocysts was demonstrated.[10]
- Throughout the 1970s and 1980s, a vast number of species were tested for the ability to shed oocysts upon infection. Whereas at least 17 different species of felids have been confirmed to shed oocysts, no nonfelid has been shown to be permissive for T. gondii sexual reproduction and subsequent oocyst shedding.

#### **TESTING**

- The only evidence of infection is detection of antibodies in the blood against the toxoplasmosis parasite.
- According to recent studies, the repetitive element of 529 bp in length has shown a sensitivity that is 10-times that of the sensitivity using the B1 gene. Real-time PCR detection of T gondii DNA based on the 529 bp repetitive element is the most frequently used molecular diagnostic approach for toxoplasmosis.
- Polymerase chain reaction (PCR) assay testing on body fluids, including CSF, amniotic fluid, bronchoalveolar lavage fluid, and blood, may be useful in the diagnosis. However, PCR assay is capable of

detecting T gondii deoxyribonucleic acid (DNA) in either an aqueous sample or a vitreous sample in only one third of patients with ocular toxoplasmosis.[46, 47]

Detection of immunoglobulin G (IgG) is possible within 2
 weeks of infection using the enzyme-linked immunosorbent assay (ELISA) test, the IgG avidity test, and the agglutination and differential agglutination tests. (Acute and convalescent sera have no role in the indirect detection of toxoplasmosis.)

#### **Procedures**

The following diagnostic procedures may be performed for toxoplasmosis:

- Lumbar puncture After imaging to identify evidence of increased intracranial pressure
- Brain biopsy
- Lymph node biopsy
- Amniocentesis Perform amniocentesis at 20-24 weeks'
   gestation if congenital disease is suggested
- Bronchoalveolar lavage
- Tachyzoites may be demonstrated in tissues or smears obtained from biopsy. They also can be seen in CSF. CSF also shows mononuclear pleocytosis and elevated protein level. Tachyzoites demonstrate acute infection, while tissue cysts and bradyzoites are seen in chronic/latent infection (although they may be present in acute infection/reactivation).

#### **Treatment**

http://www.lib.gdpu.edu.cn/cyzy/tssjk/CXXKNY/CXXKNY\_WWWX/201110/P020111020581912180349.pdf

- Current therapies for treating Toxoplasma infections show limited efficacy and are often associated with severe side effects. These therapies include **inhibition of folate metabolism**, of protein synthesis, and of electron transport. Antifolate combination therapies employing diaminopyrimidines, such as trimethoprim or pyrimethamine, combined with sulfonamides, such as sulfadiazine or sulfamethoxazole, act synergistically against various bacterial and parasitic microorganisms.
- Protein synthesis inhibitors, such as macrolide and lincosamide antibiotics, are a second class ofmedications with anti-Toxoplasma activity. Their mechanism of action in T. gondii is assumed to inhibit plastid or mitochondrial organellar protein synthesis. The third

class of anti-Toxoplasma drugs comprises the electron-transport inhibitors such as atovaquone. Atovaquone is occasionally used with pyrimethamine

Recently developed Toxoplasma inhibitors have shown a broad range of efficacy, toxicity, and therapeutic index (TIa).12-19

## **Artemisinin**

- Artemisinin is a sesquiterpene lactone that possesses a 1,2,4-trioxane moiety. Artemisinin was first identified as the active constituent in Qinghao (Artemisia annua) in 1972. The artemisinin class of drugs has shown a broad range of activity againstmany parasites in vitro including Plasmodium, Leishmania, Schistosoma, Trypanosoma, and Toxoplasma. 20,21
- While the 1,2,4-trioxane function has been shown to be the pharmacophore responsible for the potent activity against malaria parasites in vitro, 22,23 its role in anti-Toxoplasma activity is not clear.

## **Deoxyartemisinins**

- <u>Deoxyartemisinins</u> lacking the 1,2,4-trioxane moiety are unable to effectively block intracellular Toxoplasma tachyzoite replication but can moderately inhibit extracellular tachyzoite invastion of host cells.
- Thus the illumination of the exact mechanism of action of artemisinins against in vitro Toxoplasma is still an area of intense interest and investigation.25-28
- Thiazole-containing <u>artemisinins</u> compounds have been shown, in vitro, to possess antimicrobial 29 and antiparasitic properties.30,31
- Some <u>C9-C10 dehydroartemsinin (DART) derivatives</u>,
   <u>including a thiazole</u> moiety, inhibit multiple steps in the lytic cycle of T. <u>gondii</u>.24 This report describes results with novel <u>DART-thiazole</u>,
   DART-oxadiazole, and <u>DART-carboxamide</u> derivatives.

- Artemix Artemisinin Complex 140mg 30 capsules
   Artesunate 50mg Artemisinin 50mg, Artemether 40mg
- <a href="http://www.drclark.com/pictures/Artemix.jpg">http://www.drclark.com/pictures/Artemix.jpg</a>

## http://en.wikipedia.org/wiki/Dihydroartemisinin

<u>Dihydroartemisinin</u> (also known as **dihydroqinghaosu**, **artenimol** or **DHA**) is a drug used to treat <u>malaria</u>. Dihydroartemisinin is the active metabolite of all <u>artemisinin</u> compounds (artemisinin, <u>artesunate</u>, <u>artemether</u>, etc.) and is also available as a drug in itself. It is a semi-synthetic derivative of <u>artemisinin</u> and is widely used as an intermediate in the preparation of other artemisinin-derived antimalarial drugs. [1]

It is sold commercially in combination with <u>piperaquine</u> and has been shown to be equivalent to <u>artemether/lumefantrine</u>. [2]

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  - Correspondence should be sent to: vcarruth@umich.edu
  - o Print ISSN: 0022-3395
  - o Online ISSN: 1937-2345
  - o A Thiazole Derivative of Artemisinin Moderately Reduces Toxoplasma gondii Cyst Burden in Infected Mice
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#### **Abstract:**

Toxoplasmosis continues to be a public health problem, causing significant morbidity worldwide. Currently available medications, effective for acute toxoplasmosis, are nonetheless problematic due to adverse side effects in many patients. In addition, no medication is able to completely eradicate the parasite cysts, rendering infected individuals at risk for reactivation upon becoming immunocompromised. We examined the anti–T. gondii activity of 2 derivatives of artemisinin. In vitro metabolic stability tests revealed that both derivatives are stable in mouse plasma but only the thiazole CPH4-136 is stable in the presence of mouse microsomes. When tested in a mouse model of acute toxoplasmosis, both derivatives showed modest efficacy dependent upon the compound dose and the solvent vehicle. Finally, in a mouse model of chronic T. gondii infection, CPH4-136 at 3 mg/kg once per day for 32 days moderately but significantly decreased mouse brain cyst burden.

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• Collectively, our findings suggest that artemisinin derivatives are partially effective in treating experimental T. gondii infections.

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## **Neurocysticercosis**

## http://radiopaedia.org/articles/neurocysticercosis

#### Cause

o Infection which leads to extra-intestinal disease (including neurocysticercosis) usually occurs as a result of eating food or drinking water contaminated by human feces containing T. solium eggs. This is distinct from the 'normal' life cycle in which the undercooked pork is eaten and the larval cysts contained within mature into adult intestinal tape worm 3.

#### **Prevelance**

• The disease is endemic in Central and South America, Asia and Africa.

#### **Prevention**

Avoid products and meat from infected areas

## Clinical presentation includes:

- Seizures: most common cause of seizures in young adults in endemic areas 2
- headaches
- hydrocephalus
- •altered mental status
- neurological deficits

O Neurocysticercosis occurs when people ingest the eggs of Taenia solium, a pork tapeworm whose eggs are found in human feces. Ingesting the eggs leads to tapeworm larvae growing in many different human tissues, particularly brain and muscle. This leads to seizures and more. The disease is most common where pigs are raised and sanitation is poor, including much of South America and India.

## **Stages**

There are four main stages (also known as Escobar's pathological stages):

1. Vesicular: viable parasite with intact membrane and therefore no host reaction. 2.Colloidal vesicular: parasite dies within 4-5 years 1 untreated, or earlier with treatment and the cyst fluid becomes turbid. As the membrane becomes leaky oedema surrounds the cyst. This is the most symptomatic stage. 3. Granular nodular: 0 oedema decreases as the cyst retracts further; enhancement persists. 4. Nodular calcified: end-stage quiescent calcified cyst remnant; no oedema.

## **Progression**

- Infection can be both intra and extra axial. Commonest location is the subarachnoid space over the cerebral hemispheres. Other locations in order of decreasing frequency are:
- o 1.subarachnoid space over the cerebral hemispheres, ∘may be large
- 2.basal cisterns, ∘may be "grape like" (racemose): most lack an indentifiable scolex
- 3.parenchyma
- $\circ$  4.ventricles ,  $\circ$ usually solitary cysts,  $\circ$ 4th ventricle most frequent

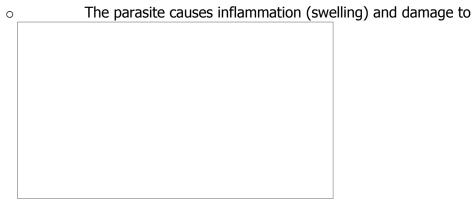
	the subarachnoid ones can be much bigger (up to 9cm): dif therefore being arachnoid cyst.	•
	<ul> <li>It is the most common cause of epilepsy in end (South-east Asia, South America) with progression through taking anywhere between 1 to 9 years.</li> </ul>	
Testing		
	o MRI	
	o CSF serology may be helpful with the initial diagence especially in cases of intraventricular/subarachnoid infection	
	0	
Treatment		
	<ul> <li>Albendazole and praziquantel</li> </ul>	
Radiograph	ic features- MRI	
Schistosomiasis		

Schistosoma

Intestinal schistosomia	asis			
		search?q=poly- isinin&hl=en&gbv=2&oq:	=&gs_l=	I
http://www.pa	arasitesandv	vectors.com/content/7/1/8	31	
	jellyfish (Li Seabather's clothing or	man disease schistosomia nuche unguiculata), which s eruption mostly occurs in hair. Schistosomiasis (also kr	, which infect humans and cause th sis, or with larval stages of thimble h give rise to seabather's eruption. in salt water, on skin covered by hown as bilharzia, bilharziosis or sna by several species of fluke of the	
	•	nistosoma".	by several species of make of the	
	fresh water	nistosomiasis is transmitter. Swimming, bathing, fisl	is a disease caused by parasitic ed by contact with contaminated ning and even domestic chores such n put people at risk of contracting th	
			ontaminated by Schistosoma eggs ecate in the water. The eggs hatch inside snails.	
	o called a blo	The state of the s	ed by infection with small, flatworm	
	of persons swimming,	Schistosoma an penetrate the skin who are wading, bathing, or washing in ted water. Within		

several weeks, worms grow inside the blood vessels of the body and produce eggs. Some of these eggs travel to the bladder or intestines and are passed into the urine or stool.

O Usually, these small, leaf-shaped worms cause peritoneum cavity, and live in tissues - intestinal, liver, kidney or bladder, not in the lumen of the digestive tract. Some Schistosoma eggs, however, remain trapped in the body and migrate to specific organs (depending on the type of parasite) where they can inflict major damage.



organs, particularly the liver. Urinary schistosomiasis causes scarring and tearing of the bladder and kidneys, and can lead to bladder cancer. Intestinal schistosomiasis develops slowly, causing abdominal bleeding; enlargement of the liver, lungs and spleen; and damage to the intestines. A major indicator of the disease is blood in the urine and/or feces.

Second most devastating parasitic disease, (after malaria)

Background

#### **Intestional**

o Given that the majority of basic schistosomiasis research is performed on Schistosoma mansoni, a major cause of the intestinal form of schistosomiasis, a relevant issue is whether the transcription of immune response- and fibrosis-related genes triggered by S. haematobium eggs in the bladder differ from those directed against S. mansoni eggs in other tissues. Perhaps the most appropriate comparisons can be made between our data and microarray analyses that have employed the S. mansoni egg-induced, synchronous lung granuloma model. These studies are methodologically analogous to this study's microarray analysis of our synchronous egg injection model. Our data indicates that although the S. haematobium egg-directed immune and fibrotic response in the bladder shares many similarities to the S. mansoni egg-triggered lung response, there are a number of potentially important disparities (see Venn diagram immediately following this paragraph). This

highlights the critical need to develop in vivo models which properly match schistosome species with their tropism for specific host organs.

o "we don't know...

#### Schistosoma haematobium

http://uti.stanford.edu/research/schistosomiasis.html

- o Chronic parasitic worm infection with *Schistosoma haematobium* **primarily affects the genitourinary** tract (i.e., urogenital schistosomiasis) and can result in hematuria (bloody urine), urinary symptoms, genital sores increasing HIV risk, **bladder** and ureteral fibrosis (scarring), and bladder cancer. Consequently, *S. haematobium* may be the most lethal worm on the planet. Our research group is particularly interested in combining microsurgical and *in vivo* imaging techniques with experimental models of urogenital schistosomiasis to better understand the immunology and epithelial and stromal biology of this terrible disease. *S. haematobium* is endemic in Africa and the Middle East, and relies on freshwater snails as an obligatory part of its life cycle. Controlling infestation has proven challenging due to the need to preserve local aquatic ecosystems and difficulties with public education. Currently, the only available medications to treat schistosomiasis are praziquantel and artemether. There is no approved vaccine.
- Due to technical issues with animal models, the majority of

basic schistosomiasis research is performed on S. mansoni and S. japonicum, the major causes of the intestinal form of schistosomiasis. However, over 112 million people are infected with S. haematobium, the most common schistosome species globally. Diagnosis of S. haematobium infection (urogenital schistosomiasis) is made by manual microscopic counting of eggs in urine. This method is expensive, labor-intensive, and

impractical in many endemic areas, given the lack of consistent electricity needed to power modern microscopes. Urogenital schistosomiasis-induced urinary tract fibrosis and cancer can require surgery, even after clearance of infection through medication. Coupled with the possibility of emerging schistosomal resistance to praziquantel, there is a need for vaccines, alternative diagnostic tests and medications, and a better understanding of the immunopathology associated with S. haematobium.

## Symptoms

- Frequent, painful or bloody urine
- •Abdominal pain and bloody diarrhea
- Anemia
- Fever, chills and muscle aches
- •Inflammation and scarring of the bladder
- Lymph node enlargement
- Enlargement of the liver or spleen
- Secondary blood disorders in cases of colon damage
- If infection persists, bladder cancer may eventually develop in some cases
- Children with repeated infection can develop anemia, malnutrition and learning disabilities

#### Disease Impact

- •Schistosomiasis is the most deadly NTD, **killing** an estimated 280,000 people each year
- •Twenty million schistosomiasis sufferers develop severe and sometimes disfiguring disabilities from complications from the disease, including kidney disease, liver disease and bladder cancer

#### Prevalance

o Globally, about one in thirty people (Around 200 million people) infected worldwide carries these schistosomes. Like many parasites, the life cycle of this organism is complex and involves many different stages. 10 percent are severely ill.

## Geography

- Liver fluke infections have been reported in Europe and the United States (The parasite is commonly found in fresh water lakes. Outbreaks are common in the upper Midwest, but the parasites have also been found in Delaware, New Mexico and Colorado) as well as the Middle East, China, Japan and Africa. Lung fluke infections are common in the Far East, South-east Asia, Africa, Central and South America, Indonesia and the Pacific Islands.
- Countries of Latin America, Africa, the Middle East, and Asia.
   The risk varies widely, however.
- o Intermediate-risk destinations include most Southern European countries and a few Caribbean islands.

Low-risk destinations include Canada, Northern Europe, Australia, New Zealand, the United States, and some Caribbean islands.

#### Risk

O Humans acquire the infection by contact with freshwater containing schistosomal larvae, which penetrate the skin and migrate into the blood vessels, which they use to travel through the body. The worms use suckers to adhere to the wall of the blood vessel, where they can live for up to 30 years.

#### **Food-borne diseases**

http://www.ncbi.nlm.nih.gov/pubmed/20153070? dopt=Abstract&holding=f1000,f1000m,isrctn

- The burden of diseases caused by food-borne pathogens remains largely unknown. Importantly data indicating trends in food-borne infectious intestinal disease is limited to a few industrialised countries, and even fewer pathogens. It has been predicted that the importance of diarrhoeal disease, mainly due to contaminated food and water, as a cause of death will decline worldwide. Evidence for such a downward trend is limited. This prediction presumes ...
- o Generating evidence, guiding policy sub-Saharan Africa regional edition. Seattle: Institute for Health Metrics and Evaluation; 2013.

Path

- The cercariae lose their tail upon entry and transform into schistosemulae that develop a double-lipid barrier that is resistant to human immune responses. They incorporate host proteins and major histocompatibility complexes and migrate through blood vessels. They enter pulmonary capillaries and eventually enter the portal veins where they mature into adult worms. Male and female worms attach together at the male's gynecophoric canal (Figure 4). Here they migrate into mesenteric veins (*S mansoni*, *S japonicum*) or vesicular veins (*S haematobium*). In the chronic stage, symptoms can present months or years later. They vary depending on the species that has infected the host. In general, the eggs induce a significant immune response and form granulomas. *S mansoni* and *S japonicum* cause abdominal pain, bloody diarrhea, and colonic polyposis. Eggs that remain in the portal system develop periportal fibrosis. Symptoms include portal hypertension, hematemesis, ascites, splenomegaly, and esophageal variceal bleeding. Granulomatosis in the pulmonary system leads to chronic coughs, palpitations, atypical chest pain, pulmonary hypertension, cor pulmonale, and ultimately death. 5
  - O Shaematobium deposit their eggs in the urinary tract system. Symptoms include dysuria, hematuria, bladder polyps, ulcers, obstructive uropathies, and squamous cell bladder cancer. Women manifest female genital schistosomiasis (discussion below). Eggs can end up in the skin, brain, muscle, adrenal glands, and eyes. In some cases, severe symptoms such as seizures, mental status changes, and even paralysis can occur. Coinfection of schistosomiasis along with other diseases such as hepatitis, human immunodeficiency virus, and malaria can raise the risk for hepatocellular carcinoma and increase the risk of mortality. 5

#### Types

There are several types of fluke - blood flukes, liver flukes, oriental lung flukes, sheep liver flukes and intestinal flukes, tissue flukes, zoonotic flukes, lancet flukes and a host of others. The differences vary in where and how a person has been infected and where and how they will damage the system internally. Liver, oriental lung, sheep liver, and intestinal flukes are transmitted via food; blood flukes are transmitted in swimming or bathing water. If living in the UK, Europe, North America or Canada it is likely that any fluke contamination will have been picked up on holiday in somewhere tropical. Areas such as Africa (especially Egypt), South America, the Caribbean, and China are worst affected. They can also be brought right to the table in food imported from abroad.

#### Sources

o Contracted by eating raw or undercooked fish, animals, or plants from fluke-infected fresh water.

#### **Symptoms**

 Is characterized by chronic inflammation of the intestines and urinary bladder.

- Most patients with this infection feel no symptoms at all. Sometimes, though, an acute infection can be seen one day after exposure with an itchy rash. Two to eight weeks later, a fever can develop. Later, as the schistosomes can spread to different organs, various symptoms can occur. Sometimes, the worms spread to the spinal cord, causing a myelopathy. This results in pain, bowel dysfunction, and weakness of the regions below the level of infection.
- O Diarrhea frequently occurs within the first week of travel, but may develop at any point, even after returning home. Traveler's diarrhea causes four or five loose or watery stools per day. Vomiting may also occur. It usually lasts 3 or 4 days, but about 14% of cases last longer. In rare cases, the diarrhea lasts more than 3 months. When Travelors diarrhea lasts a long time, it can cause post-infectious disease.
- Permanent paralysis can result. In other cases, the schistosomiasis can affect the brain, leading to epilepsy or elevated intracranial pressure.

## **Cerebral Schistosomiasis:**

- Three different schistosomal species (S haematobium, S japonicum, and S mansoni) can cause infection that involves the brain and spinal cord (3). Brain involvement is found in about 4% of all patients infected by S mansoni (4). Cerebral involvement is a rare ectopic manifestation of schistosomiasis.
- Although ova can be found in the brains in a majority of autopsied cases with proved schistosomiasis, only 2% of persons with acute Schistosomiasis incur complications of the central nervous system (1, 2). Clinical symptoms such as acute encephalopathy, seizure disorders, mass lesions, and paresis, as well as radiologic findings, can simulate a brain tumor, but in most cases thorough knowledge of the patient's history provides clues to the correct diagnosis.
- They migrate to the lungs and the liver. The organisms then mature into mating pairs of male and female worms which settle in the mesenteric veins (5). The adult worm lay eggs that are excreted with stool or urine. Different mechanisms of invasion of
- The brain have been discussed: the eggs may reach the brain through the valveless venous plexus of Batson, which joins the deep iliac veins and inferior vena cava with veins of spinal cord and brain, or eggs may migrate to the brain via pulmonary arteriovenous shunts, or portalpulmonary arteriovenous shunts (3, 6, 7). Finally, the worms themselves may enter the cerebral veins and place their eggs directly at the ectopic site, which could be the cerebrum, cerebellum, leptomeninges, brain stem, or choroids plexus (7, 8). The host then

develops an immunologic reaction to the deposited ova; the response may cover a wide spectrum, from a very intense reaction, resulting in granulomas or space-occupying mass lesions, to a minimal reaction with no clinical manifestation at all.

## **Testing**

- Epidemiologic history in combination with positive results of cerebrospinal fluid ELISA and the patient's recovery after chemotherapy can establish the diagnosis, although biopsy of the intracerebral lesions may be considered [9,10]. However, the history of exposure to schistosome infected water is important for the diagnosis. The prognosis of schistosomiasis depends on early diagnosis and good treatment.
- o If Laboratory findings revealed a leukocyte count of 11 000/mL, a normal red blood count and no eosinophilia, cerebrospinal fluid (CSF) with 7/3 cells, 0.81 g/dL proteins, and 3.7 mmol/L glucose. With the CSF ELISA, antibodies against Schistosoma organisms may detected.
- The most common brain sites involved, whereas the cerebellum, thalamus, hippocampus, midbrain, basal ganglia, choroid plexus, and white matter are less frequently involved. In the mass form of brain schistosomiasis, CT typically reveals a nodular, enhancing, space-occupying lesion with surrounding edema, and contrast-enhanced T1-weighted MR images in many patients revealed a central linear enhancement surrounded by multiple enhancing punctate nodules, forming an "arborized" appearance. Pathologically, this enhancement pattern correlated with a host granulomatous response to schistosome eggs. Although the pattern is not present in all cases of brain schistosomiasis, when it is observed, a diagnosis of brain schistosomiasis should be considered

#### **Treatment**

http://cid.oxfordjournals.org/content/50/9/1205.full.pdf

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3230106/

http://www.ncbi.nlm.nih.gov/pubmed/9114177

Morbidity control of schistosomiasis relies on a single drug, praziquantel. The antimalarial drug mefloquine possesses interesting antischistosomal properties, yet no clinical studies have been performed.

Artesunate in the treatment of urinary schistosomiasis

- Schistosoma mansoni praziquantel 40 mg/kg probably reduces parasitological treatment failure at one month post-treatment
- Schistosoma mansoni oxamniquine: Only one small study directly compared praziquantel 40 mg/kg
- Schistosoma haematobium Mefloquine, Artesunate, Mefloquine-Artesunate, and Praziquantel
- Artemether is effective against juvenile schistosomes during the first
   21 days of infection in animals and humans
   28 and, if given every two weeks,
   should kill all immature schistosomula.
- O Because these worms can live in the body for years, with potential for serious trouble at any time, infected persons should be treated regardless of whether they have serious symptoms.
- O Standard treatment with praziquantel was also more effective than artesunate with sulfalene plus pyrimethamine, My fever was highest (41.5°C) on the second day of my course of praziquantel. My stool, re-checked 10 and 20 days later, showed no more eggs. After three months, I feel well now.
- If the flukes have invaded the nervous system, <u>steroids</u> should be given as well in order to <u>reduce the inflammatory</u> <u>response</u>.
- O **Praziquantel (PZQ)** effectively treats all forms of schistosomiasis. It only requires 2 to 3 doses for 1 day. Although it has minimal side effects, it cannot prevent future infections. In the acute phase, especially in cases where the egg infestation is high, PZQ may worsen symptoms. Patients who become severely ill may need corticosteroids to decrease the inflammatory response. The recovery rate is as high as 98%.5
- Eighteen patients with acute Schistosoma mansoni infection were followed up for 2 years after treatment with praziguantel or oxamniquine. Cure rates, clinical features, abdominal ultrasonographic findings, and specific humoral responses were determined at 2-, 6-, and 24-month follow-ups. Fourteen patients (77.8%) were considered parasitologically cured. Levels of IgA antibody to soluble egg antigen (SEA) and IgM antibody to keyhole limpet hemocyanin (KLH) became negative or decreased to the cutoff level for chronic infection 2 months after treatment, while levels of IgG antibody to KLH declined between 12 and 24 months after treatment. Levels of IgM and IgG antibodies to saline worm adult protein as well as IgM and IgG antibodies to SEA remained positive during the follow-up period. Discrete lymph node enlargement and hepatomegaly were still present in six of the eight cured children 2 years after treatment, while complete regression was observed in adults. In our group of patients, in addition to presenting with more intense clinical manifestations, children were cured less often and had slower abatement of symptomatology after treatment than adults

**Natural Treatment** 

0 <u>black seed, cloves, gentian root, fennel seeds, green black walnut husks, hyssop</u> <u>leaves, oregano, peppermint leaves, pumpkin seeds, thyme leaves</u>

## **Echinococcosis**

 Echinococcus is a tapeworm that, in the early stage of life, can cause cysts in living human tissue, including the brain and spinal cord.

#### **Risk**

 Humans acquire the infection after eating contaminated food. The disease is rare in the United States, but is more common in Africa, Central Asia, Southern South America, the Mediterranean and the Middle East.

## **Symptoms**

The initial stages of infection are always asymptomatic, and it may be years before the cysts cause any problems. In the brain, the cysts can cause seizures or elevated intracranial pressure. In the spinal cord, the cysts can cause cord compression and paralysis. Such central nervous system infection is relatively rare, however—usually the cysts infect other organs, such as the lungs or liver.

#### **Tests**

O Cysts can be found with a CT scan, but they're usually found when an imaging test is done for some other reason.

#### **Treatment**

O Cysts may need surgical maneuver, often with additional medical treatment with a drug such as albendazole or praziquantel.

## **Trichenella**

This parasitic infection is caused by roundworms (nematodes), most commonly found in undercooked pig meat, though it can be found in other types of meat as well. The infection is relatively uncommon in the United States due to improvements in food preparation. Larvae invade the wall of the small bowel and develop into adult worms. Worms then go on to release eggs that grow into cysts in muscles. When the muscle is ingested by another animal, the cycle continues.

Headache is a common symptom. Swelling, strokes, and seizures can also occur. The CT can show small cystic lesions throughout the brain. Treatment is with albendazole or mebendazole, sometimes combined with prednisone in severe cases.	
O As unappealing as parasitic infections are, it's worth notin	ē
	i

that most of the time, these infections go unnoticed. A high percentage of people throughout the world live with a worm or other parasite. As close as we may be with these organisms, though, invasion of our central nervous systems is too close for comfort, and must always be taken seriously.

# Common types and causes of parasitic infections

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# **Roundworms**

o Roundworm infection comes from ingesting roundworm eggs via contaminated foods. Once in the small intestine the larvae are liberated and can even migrate through the intestinal wall to the lungs. About 10 days after ingesting the eggs, the larvae pass into the digestive tract and mature into egg-producing worms, which grow to 15-40 cm (6-16 inches) long. Adult roundworms are able to migrate into other parts of the body, where they produce abcesses and toxic manifestations.

# **Anisakids**

### http://en.wikipedia.org/wiki/Anisakis

Anisakis is a genus of parasitic nematodes, which have life cycles involving fish and marine mammals.[1] They are infective to humans and cause anisakiasis. People who produce immunoglobulin E in response to this parasite may subsequently have an allergic reaction, including anaphylaxis, after eating fish that have been infected with Anisakis species.

# Anisakids Dewsc

As with all parasites with a complex life cycle involving a number of hosts, details of the morphology vary depending on the host and life cycle stage. In the stage which infects fish, Anisakis species are found in a distinctive "watch-spring coil" shape. They are roughly 2 cm long when uncoiled. When in the final host, anisakids are longer, thicker and more sturdy, to deal with the hazardous environment of a mammalian gut.

# **Prevalance**

Fewer than ten cases occur annually in the United States.[9] Development of better diagnostic tools and greater awareness has led to more frequent reporting of anisakiasis.

\_

# **Vector**

**Anisakids** Contracted through consuming undercooked or raw fish such as sushi. The larval stage infects fish like Pacific salmon, red snapper, herring, cod and haddock and the mature anisakids infect sea mammals.

\_

### **Cause**

Anisakiasis is a human parasitic infection of the gastrointestinal tract caused by the consumption of raw or undercooked seafood containing larvae of the nematode Anisakis simplex. The first case of human infection by a member of the family Anisakidae was reported in the Netherlands by Van Thiel, who described the presence of a marine nematode in a patient suffering from acute abdominal pain.[8] It is frequently reported in areas of the world where fish is consumed raw, lightly pickled or salted. The areas of highest prevalence are Scandinavia (from cod livers), Japan (after eating sushi and sashimi), the Netherlands (by eating infected fermented herrings (maatjes)), and along the Pacific coast of South America (from eating ceviche). Fewer than ten cases occur annually in the United States.[9] Development of better diagnostic tools and greater awareness has led to more frequent reporting of anisakiasis.

-

# **Symptoms**

Acute abdominal pain.

\_

Appendicitis, Crohn's disease, and other forms of intestinal inflammation.

# **Symptoms**

- Within a few hours of ingestion, the parasitic worm tries to burrow though the intestinal wall, but since it cannot penetrate it, it gets stuck and dies. The presence of the parasite triggers an immune response; immune cells surround the worms, forming a ball-like structure that can block the digestive system, causing severe abdominal pain, malnutrition and vomiting.
- Occasionally, the larvae are regurgitated. If the larvae pass into the bowel or large intestine, a severe eosinophilic granulomatous

response may also occur one to two weeks following infection, causing symptoms mimicking Crohn's disease.

# **Diagnosis**

 Diagnosis can be made by gastroscopic examination, during which the 2-cm larvae are visually observed and removed, or by histopathologic examination of tissue removed at biopsy or during surgery.

### **Treatment**

o **For the worm, humans are a dead-end host.** Anisakis and Pseudoterranova larvae cannot survive in humans, and will eventually die. In some cases, the infection will resolve with only symptomatic treatment.[12] In other cases, however, infection can lead to small bowel obstruction, which may require surgery,[13] although treatment with albendazole alone (avoiding surgery) has been reported to be successful. Intestinal perforation (an emergency) is also possible.[14]

### **Prevention**

o Raising consumer and producer awareness about the existence of anisakid worms in fish is a critical and effective prevention strategy. Anisakiasis can be easily prevented by adequate cooking at temperatures greater than 60°C or freezing. The FDA recommends all shellfish and fish intended for raw consumption be blast frozen to -35°C or below for 15 hours or be regularly frozen to -20°C or below for seven days.[9] Salting and marinating will not necessarily kill the parasites. Humans are thought to be more at risk of anisakiasis from eating wild fish than farmed fish. Many countries require all types of fish with potential risk intended for raw consumption to be previously frozen to kill parasites. The mandate to freeze herring in the Netherlands has virtually eliminated human anisakiasis.[10]

# **Ascaris**

http://www.mayoclinic.org/diseases-conditions/ascariasis/basics/definition/con-20027084

https://web.stanford.edu/class/humbio103/ParaSites2005/Ascaris/#Hepato

http://web.stanford.edu/group/parasites/ParaSites2005/Ascaris/JLora\_ParaSite.htm

http://www.patient.co.uk/doctor/ascaris-lumbricoides

http://www.parasiteinfo.com/parasites/ascaris.htm

http://parasite.org.au/para-site/text/ascaris-text.html

https://books.google.com/books?

id=VirWpchAmyQC&pg=PA124&lpg=PA124&dq=ascaris+Behaviour&source=bl&ots=qHQm86mInD&sig=EnSQnl3SoQr7p0\_XO\_4e9ZNau8Y&hl=en&sa=X&ei=pOieVNDVJI6lyATQz4CYAw&ved=0CDwQ6AEwCQ#v=onepage&q=ascaris%20Behaviour&f=false

### **Ascariasis**

0	Ascaris lumbricoides is a nematode (roundworm), which
inhabits hu	nans.

0	Ascaris is a genus of	parasitic nematod	e worms	known	as t	:he
"giant	intestinal roundworms".					

- Ascariasis is an important infection in humans (*Ascaris lumbricoides*) and pigs (*Ascaris suum*)
- A suum is zoonotic, and adults are commonly found in preschool children in contact with swineherds.
   Visceral larva migrans due to migrating larvae has been described.
- Ascariasis (as-kuh-RIEuh-sis) is a type of roundworm infection that may live in the gut for 6-24 months..
- These worms are parasites that use your body as a host to mature from larvae or eggs to adult worms and reproduce.
- Adult worms can be more than a foot (30 centimeters) long.

- Adult worms inhabit the lumen of the small intestine, usually in the jejunum or ileum.
- They are passed in the stool.
- $\circ$  When both female and male worms are present in the intestine, each female worm produces approximately 200,000 fertilized ova per day. The ova are oval, have a thick shell, a mamillated outer coat, and measure 45 to 70 μm by 35 to 50 μm.
- O During the migratory phase, the larvae may penetrate into the tissues and circulate around the body via the blood and lymphatic systems, commonly to the lungs. In the lungs, the larvae penetrate the pulmonary capillaries to enter the alveoli, from where they ascend into the throat and descend back into the gut where they may grow
- The ova are passed out in the feces, and embryos develop into infective second-stage larvae in the environment in two to four weeks (depending upon environmental conditions). When ingested by humans, the ova hatch in the small intestine and release larvae, which penetrate the intestinal wall and migrate hematogenously or via lymphatics to the heart and lungs. Occasionally, larvae migrate to sites other than the lungs, including to the kidney or brain.

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### **Definition**

- Ascariasis is considered the most common type of roundworm infection in humans.
- Ascariasis are Nematodes, which can use humans as the destination host, and can reproduce entirely in a human host.
- They can infect a human host for over 30 years.

### **Prevalance**

- A. *lumbricoides* is endemic throughout the world.
- Researchers estimate that about 25% of the world's population (2 Billion People) are infected with the parasite.
- o In the United States, most infections occur in areas that have warm climates, such as the Southern United States.
- Ascarids have been epidemic in the recent past in the southeastern U.S.

- o Globally, 12 million acute cases and 10,000 deaths occur annually
- $_{\circ}$  It has been estimated that 9 x 10(14) eggs contaminate the soil per day worldwide [14].

### **Vector**

o Dirt, Fruit, Water Supply

### **Risk factors**

- Large numbers of *A. lumbricoides* eggs may be found in municipal domestic sewage and individuals eating unwashed fruit and vegetables, particularly those grown in, or near, soil fertilised with sewage are more likely to become infected.
- There is considerable biological and epidemiological evidence to suggest zoonotic transmission of *A. suum* to humans, although recent molecular studies have shown limited gene flow between human and pig ascarid populations.
- While the whole life-cycle of A. suum may not be completed in non-porcine hosts, their larvae can undergo extensive migration in a number of hosts (humans, cattle, sheep, etc) leading to manifestations.

### **Causes**

Ascariasis isn't spread directly from person to person.
 Instead, a person has to come into contact with soil mixed with human feces that contain ascaris eggs.

# Ingestion.

- o The microscopic ascariasis eggs can't become infective without coming into contact with soil. People can accidentally ingest contaminated soil through hand-to-mouth contact or by eating uncooked fruits or vegetables that have been grown in contaminated soil.
  - Humans become infected after they ingest the parasite's microscopic eggs.

- We all have **Ascariasis lumbricoides** tapeworm stages in our bodies, probably going back to childhood when we ate dirt.
- It is not normal for these stages to hatch and develop further. Their purpose is to stay dormant. And perhaps they do little harm this way.
- If stages are hatching. This spews the tiny larvae and cysts, plus unfertilized eggs and their bacteria all over the body, making you feel sick.
- Perhaps the large amounts of solvents accumulated in the body forces them to hatch; perhaps the lowered immunity allows them to hatch.
- Lowered immunity occurs when other parasitic infections persist for a period of time.

**Parasite morphology:** The parasite forms several different developmental stages: eggs, larvae [moult from first-stage (L1) through to fourth-stage (L4)], and adults (male and female).

Fertilized eggs appear as round-oval tan-coloured stages (45-75 $\mu$ m long by 35-50 $\mu$ m wide) surrounded by a thick albuminous mamillated (lumpy) outer coat. Before insemination or in early stages of oviposition, female worms may also excrete unfertilized eggs which are more elongate (85-95 x 45 $\mu$ m) and decorticated (not mamillated). Fertilized eggs are excreted unembryonated, but then develop first-stage then second-stage infective larvae.

The eggs of the roundworm have an incredibly thick shell and are very resistant to variations in temperature and humidity in the environment. They are coated with a jelly-like substance with a lumpy surface that allows the eggs to stick to almost anything. Although it takes about two to three weeks for them to become infective, roundworm eggs will remain infective in the soil for some time. The eggs hatch in the small intestine, the juvenile penetrates the small intestine and enters the circulatory system, and eventually the juvenile worm enters the lungs. In the lungs the juvenile worm leaves the circulatory system and enters the air passages of the lungs. The juvenile worm then migrates up the air passages into the pharynx where it is swallowed, and once in the small intestine the juvenile grows into an adult worm. Why Ascaris undergoes such a migration through the body to only end up where it started is unknown. Such a migration is not unique to Ascaris, as its close relatives undergo a similar migration in the bodies of their hosts.

When hatched in the host, these small larvae (juvenile) (1.2-1.8mm long) invade host tissues and undertake pulmonary migration in the circulatory system. This migration process takes about 8-12 weeks.

In the lungs the juvenile worm enters the air passages. It then migrates up the air passages into the pharynx where it is swallowed, and once in the small intestine the juvenile grows into an adult worm typically in four molts.

After about two months in the small intestine the roundworms mature into adults. The roundworm adults remain in the small intestine feeding on the contents of the intestine. They do not actually feed on the human host as many of the other nematodes do.

Large adult worms develop in the gut, female worms measuring 20-50cm long by 3-6mm wide, while males are smaller, measuring 15-30cm long by 2-4mm wide with two simple spicules 2.0-3.5mm long.

Adults have a striated cuticle and three small, but conspicuous, lips around the apical mouth. The hemocoel, or body cavity, is filled with fluid under exceptionally high pressure (higher than that of any other animal) and acts as a hydrostatic skeleton. All other worm's organ systems are affected by this pressure and function under its influence. The pressure maintains the body shape and acts as a hydrostatic skeleton against which the body wall muscles act to accomplish locomotion.

# Migration

- People accidentally ingest contaminated soil through handto-mouth contact or by eating uncooked fruits or vegetables that have been grown in contaminated soil.
- The microscopic ascariasis eggs travel to your small intestine where they attach.
- Once the eggs enter the body, they mature into larvae.
- Larvae hatch from the eggs in your small intestine and then penetrate the intestinal wall to travel to your lungs via your bloodstream or lymphatic system.
  - Larval migration may cause cough, dyspnoea, haemoptysis and eosinophilic pneumonitis (Löffler's syndrome).
  - During the larval phase, the larvae invade the intestinal mucosa and migrate through the circulation to the lungs. The larvae then break into the alveoli, ascend the bronchial tree, The larvae then enter the lungs.

- Larval invasion normally induces an immune response,
- Tissue damage can occur when the worms migrate. Ascaris pneumonitis may be caused the worms as the enter the lungs. On chest x-rays it may resemble viral pneumonia.
- After maturing for about a week in your lungs, the larvae break into your airway and travel up your throat, where they're coughed up and swallowed.
- Once back in the intestines, the parasites reattach and grow into male or female worms. Female worms can be more than 15 inches (40 centimeters) long and a little less than a quarter inch (6 millimeters) in diameter. Male worms are generally smaller.
- Reproduction. Male and female worms mate in the small intestine. And can initially produce up to 200,000 eggs a day.
- Treatment can cause adult worm migration
- Migrating adult worms can chew threw tissue and cause infections, and spread immature worms throughout tissue and the body.
  - Up throat
  - Behind tonsils
  - Behind sinuses
- o In the adult phase, the worms may migate to the appendix, hepatobiliary system, pancreatic ducts, and other organs such as kidneys or brain, Rarely, larvae lodge ectopically in the brain, kidney, eye, spinal cord, and other sites and may cause local symptoms
- Migrating adult worms may cause Visual, hearing, nervous,
   CNS, symptomatic occlusion of the biliary tract or oral expulsion.
- Ascarids can infect the gut and lungs in humans, or other locations, and the juvenille worms can crawl into the respiratory tract and out of the nose.
- Eventually, the larvae reach the throat, where they are coughed up and then swallowed. Once swallowed, the larvae enter the intestines, where they develop into adults and feed off of the food that enters the body. Adult worms can grow to be 15 inches long. Adult worms can live up to two years, and female worms can produce more than 200,000 eggs a day. Larvae penetrate through the skin of the feet and travel to the intestine where they reach maturity.
- It takes approximately 2-3 months from the initial infection for new egg release to take place.

Life Cycle Figure - Adult worms (1) live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces (2). Unfertilized eggs may be ingested but are not infective. Fertile eggs embryonate and become infective after 18 days to several weeks (3), depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed (4), the larvae hatch (5), invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs (6). The larvae mature further in the lungs (10 to 14 days), penetrate the alveolar walls, ascend the bronchial tree to the throat, and

are swallowed (7). Upon reaching the small intestine, they develop into adult worms (1). Between 2 and 3 months are required from ingestion of the infective eggs to oviposition by the adult female. Adult worms can live 1 to 2 years. Source: CDC's Parasite and Health Page about intestinal ascariasis.

Pathogenesis: Infections by small numbers of worms may remain asymptomatic, although some individuals may develop allergic reactions (urticaria, eosinophilia). Larger numbers of worms, however, can cause significant health problems for the host. Following infection, pulmonary migration by larvae may cause petechial haemorrhages, oedema, inflammation, and pulmonary congestion (pneumonitis, or Loeffler's pneumonia) with cough, chest pain and difficulty breathing. Migrating larvae lost or trapped in other tissues often die causing focal inflammation and vague symptoms difficult to diagnose. Adult worms developing in the gut feed on luminal content, they steal liquid nourishment from the host contributing to protein energy malnutrition and impaired carbohydrate absorption. Moderate-heavy infections may cause a variety of digestive disorders, poor growth and development in small children, abdominal pains, restlessness, insomnia and allergic responses (rashes, asthma). Heavy infections may also cause life-threatening gut obstructions where tangles of worms form a bolus mechanically blocking the gut. To the great consternation of their hosts, worms may also occasionally wander upstream (obstructing biliary or pancreatic ducts, sometimes even being regurgitated) or downstream (infecting the appendix, or being passed in faeces).

### **Symptoms**

- Most people infected with ascariasis have no symptoms.
   Moderate to heavy infestations cause symptoms that may vary, depending on which part of your body is affected.
- Ascaris are immunomodulatory infections, where the immune parameters associated with protective immunity mechanisms are geared towards parasite survival.
- When the larvae enter the lungs, patients may experience symptoms similar to pneumonia, such as persistent cough, shortness of breath, and wheezing.
- When the larvae reach the intestines and develop into adults, mild or moderate symptoms may include abdominal pain, nausea, diarrhea, and sometimes bloody stools.
- Severe infections may cause abdominal pain, fatigue, vomiting, weight loss, worm in vomit or stools, or worm emerging from the nose or mouth.

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# **General Roundworm Symptoms:**

 Many individuals are unaware of **roundworm** disease but some may show:

non-productive Cough

- nocturnal fever Low-grade fever
- Inflammation of the intestine and lungs,
- Nausea,
- Night sweats
- Tiredness,
- Allergic rash,
- Vomiting,
- Diarrhoea,
- Allergy
- Malnourishment
- Intestinal blockage Constipation
- "Flu like" symptoms

•	Restlessness
• 9	Sleep disturbances
•	Nervousness and irritability,
•	Nerve problems from toxic load,
•	Periodic Wheezing,
•	Periodic Coughing,
•	Vomiting worms
•	Passing of worm in stool
•	Gallstone formation
•	Liver abscesses
■ Reflux	Pancreatis, Gas, Indigestion, GERD, Acid
•	Pulmonary eosinophilia
• [	Pneumonia
•	Shortness of breath
•	Abdominal pain
•	Nausea and explosive watery diarrhea
•	Blood in the stool, White film Stool
•	Bloating of the adbdomen,
•	Greasy stools
•	Cardio and pulmonary disorders.
•	Reversible lactose intolerance
<ul><li>developed</li></ul>	Severe right hip and low back pain
•	Severe GI pain sharp
■ including Vitamin A,	Malabsorption of fat soluible vitamins
•	Malabsorption of protein,
•	Malabsortion of fat.

• After you ingest the microscopic ascariasis eggs, they hatch in your small intestine and the larvae migrate through your bloodstream or lymphatic system into your lungs.

# In the lungs

- o similar to asthma or pneumonia,
- At this stage, you may experience signs and symptoms similar to asthma or pneumonia, including:
- Persistent cough
- Shortness of breath
- •Wheezing
- After spending six to 10 days in the lungs, the larvae travel to your throat, where you cough them up and then swallow them.
- Worm burdens at key stages during infection leading up to the pulmonary stage of development.

### In the intestines

- The larvae mature into adult worms in your small intestine, and the adult worms typically live in the intestines until they die. In mild or moderate ascariasis, the intestinal infestation can cause:
- Vague abdominal pain
- •Nausea and vomiting
- Diarrhea or bloody stools
- If you have a heavy intestinal infestation a large number of worms — you may experience:
- Severe abdominal pain
- •Fatigue
- Vomiting
- A worm in vomit
- Obstruction of intestine

- Obstruction of liver
- Obstruction of Throat

# **Systemic**

- •
- Death

### **Behaviour**

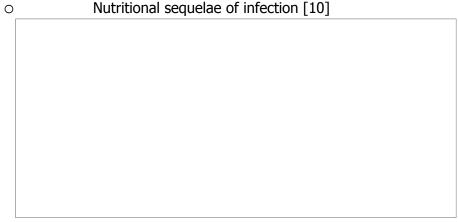
Ascaris behaviour pretty well... they tend to vibrate and can thrash around when threatened

# Pathophysiologic mechanisms include

- Direct tissue damage
- The immunologic response of the host to infection with larvae, eggs or adult worms [2]
- Obstruction of an orifice or the lumen of the gastrointestinal tract by an aggregation of worms
- O Nutritional sequelae of infection [10]

# **Complications**

- Clinical disease is largely restricted to individuals with a high worm load [1].
- When symptoms do occur, they relate either to the larval migration stage or to the adult worm intestinal stage. Pathophysiologic mechanisms include
- Direct tissue damage
- The immunologic response of the host to infection with larvae, eggs or adult worms [2]
- Obstruction of an orifice or the lumen of the gastrointestinal tract by an aggregation of worms



# 1. Pulmonary and hypersensitivity manifestations

# **Pulmonary and hypersensitivity manifestations**

- o Transient respiratory symptoms can occur in sensitized hosts during the stage of larval migration through the lungs.
- o Symptoms associated with the pneumonitis, which are known as Loffler's syndrome, tend to occur one to two weeks after ingestion of the eggs. The severity of symptoms tends to correlate with larval burden.
- o The migration of the larvae through the lungs causes the blood vessels of the lungs to hemorrhage, and there is an inflammatory response accompanied by edema. The resulting accumulation of fluids in the lungs results in "ascaris pneumonia," and this can be fatal.
- o Urticaria and other symptoms related to hypersensitivity usually occur toward the end of the period of migration through the lungs.

# 2. <u>Intestinal symptoms</u>

- o Larvae were recovered from the intestinal contents and also whilst actively migrating through the large intestinal wall.
- o Heavy infections with Ascaris are frequently believed to result in abdominal discomfort, anorexia, nausea and diarrhea. However, it has not been confirmed whether or not these non-specific symptoms can truly be attributed to ascariasis.
- o With relatively heavy infections, impaired absorption of dietary proteins, lactose and vitamin A has been noted, and steatorrhea may occur.
- o One review concluded that Ascaris-free or treated children showed better nutritional status in terms of growth, lactose tolerance, vitamins A and C, and albumin levels than Ascaris-infected children based upon almost 20 years of published cross-sectional and intervention studies. This review also found significant improvement in weight or height following therapy for

ascariasis. It has also been proposed that heavy infections may be associated with impaired cognitive development in school children [24,25].

- o Ascaris is notorious for its reputation to migrate within the small intestine, and when a large worm begins to migrate there is not much that can stop it.
- o Occasionally an adult will migrate to the stomach and cause nausea, and sometimes vomiting. This often occurs when chemotherapy is initially started, is started to slow, insufficient paralysis drugs are administered, or the infection is large.
- o In these cases, the human host can expel the worm in the vomit. (Expelled worms as big as 1 ft long have been reported.) Worms that reach the esophagus while a person is asleep can exit the body through the nose or mouth.
- o During treatment with DEC-C, Arthemeter, or mebendazole, worms can die, break open, expel infant worms, and can cause severe reactions, pain, sweating, toxic loads, Instances have been reported in which Ascaris have migrated into and blocked the bile or pancreatic duct or in which the worms have penetrated the small intestine resulting in acute (and fatal) peritonitis.
- o Ascaris seems to be especially sensitive to anesthetics, and numerous cases have been documented where patients in surgical recovery rooms have had worms migrate from the small intestine, through the stomach, and out the patient's nose or mouth.

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# 3. **3.** Intestinal obstruction

Intestinal blockage and perforation. In heavy ascariasis infestation, a mass of worms can block a portion of your intestine, causing severe abdominal cramping and vomiting. The blockage can even perforate the intestinal wall or appendix, causing internal bleeding (hemorrhage) or appendicitis.

- o A mass of worms can obstruct the bowel lumen in heavy Ascaris infection, leading to acute intestinal obstruction. The obstruction occurs most commonly at the ileocecal valve. Symptoms include colicky abdominal pain, vomiting and constipation. Vomitus may contain worms. Approximately 85 percent of obstructions occur in children between the ages of one and five years. Sometimes an abdominal mass that changes in size and location on serial examinations may be appreciated [10]. Complications including volvulus, ileocecal intussusception, gangrene, and intestinal perforation occasionally result.
- o The overall incidence of obstruction is approximately 1 in 500 children. It has been shown that between five and 35 percent of all cases of bowel obstruction are due to ascariasis [1]. One review estimated the worm burden with intestinal obstruction to be >60 (and ten times higher in fatal cases) [26].

o Ascariasis is said to be the most common cause of acute abdominal surgical emergencies in certain countries [8]. In a recent meta-analysis of morbidity and mortality related to ascariasis, intestinal obstruction accounted for a mean of 72 percent of complications of the infection [27].

# 4. 4. <u>Hepatobiliary and pancreatic symptoms</u>

https://web.stanford.edu/class/humbio103/ParaSites2005/Ascaris/#Hepato

- o Worms may block the narrow ducts of your liver or pancreas, causing severe pain.
- o Yellow and White Liver Stones about the size of a marble- may form, backing up the liver or pancreas causing an absess.
- o Hepatobiliary and pancreatic symptoms Symptoms related to the migration of adult worms into the biliary tree can cause abdominal pain, biliary colic, acalculous cholecystitis, ascending cholangitis, obstructive jaundice, or bile duct perforation with peritonitis.
- o Strictures of the biliary tree may occur [28]. Hepatic abscesses can also result [29].
- o Retained worm fragments can serve as a nidus for recurrent pyogenic cholangitis. The pancreatic duct may also be obstructed, leading to pancreatitis, and the appendix resulting in appendicitis.
- o Occasionally, migrating adult worms emerge from the mouth, nose, lacrimal ducts, umbilicus or inguinal canal. High fever, diarrhea, spicy foods, anesthesia and other stresses have all been associated with an increased likelihood of worm migration [10].
- 98 percent presented with abdominal pain,
- o 16 percent developed ascending cholangitis,
- 4 percent developed acute pancreatitis,
- 1 percent developed obstructive jaundice.
- A previous cholecystectomy or endoscopic sphincterotomy had been performed in 80 percent.
- Endoscopic extraction of the worms, successful in all but two cases, led to rapid resolution of symptoms.

# **Differential diagnosis**

The differential diagnosis will depend on the symptoms displayed, but will also include infection with *Trichuris trichiura*, another roundworm causing similar problems.

### **Test**

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In heavy infestations, it's possible to find worms after you cough or vomit, and the worms can come out of other body openings, such as your mouth or nostrils. If this happens to you, take the worm to your doctor so that he or she can identify it and prescribe the proper treatment.

### Stool tests

- o Diagnosis is usually made by identifying eggs in a stool sample.
- o The most common method for diagnosing intestinal ascariasis is light microscopy using direct wet mount examination or formalin-ethyl acetate sedimentation.
- o Fresh samples must be obtained, in that the eggs can degrade quicly
- Use a lab that can analyze samples in 24 hours.
- o Health care providers can diagnose ascariasis by taking a stool sample and using a microscope to look for the presence of eggs.
- o About two months after you ingest ascariasis eggs, the worms mature and begin laying thousands of eggs a day. These eggs travel through your digestive system and eventually can be found in your stool. To diagnose ascariasis, your doctor may examine your stool for the microscopic eggs and larvae.
- o Eggs won't appear in stool until at least 40 days after you're infected. And if you're infected with only male worms, you won't have any eggs at all.
- o Labs initially performed PCR analysis on stools. This practice has been all but discontinued. It proved to be to accurate.

### **Blood tests**

- o Your blood can be tested for the presence of an increased number of a certain type of white blood cell, called eosinophils. Elevations can occur during early infection stages
- o Ascariasis can elevate your eosinophils, but so can other types of health problems.
- o Blood count may reveal anaemia.
- o Late stage infections LFTs may reveal liver damage or low protein state.

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# **Imaging tests**

- o X-rays. If you're infested with a large number of worms, the mass of worms may be visible in an X-ray of your abdomen. In some cases, a chest X-ray can reveal the larvae in your lungs.
- o Ultrasound. An ultrasound may show if any worms are in your pancreas or liver. This technology uses sound waves to create images of internal organs.
- o CT scans or MRIs. Both types of tests create detailed images of your internal structures, which can help your doctor detect worms that are blocking ducts in your liver or pancreas. Computerized tomography (CT) combines X-ray images taken from many different angles, whereas magnetic resonance imaging (MRI) uses radio waves and a strong magnetic field.

### **Exploritary**

o Endoscopic retrograde cholangiopancreatography may be useful if biliary tree involvement is suspected.

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### **Prognosis**

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- o Early detection with GI involvement, leads to uncomplicated infections, which respond well to treatment with anthelmintics.
- o However, after dissemenation, and several maltings have occurred, migration to other locations such as the liver, lungs, heart, or genitourinary tract may cause severe acute problems and death.
- o Re-infections can occur rapidly after treatment and so there is a need for frequent anthelmintic drug administrations to maximise the benefit of preventive chemotherapy.[8]
- o Worm burdens in the lungs, Lung tissues
- o Leading up to the pulmonary stage
- The large intestine and rectum, liver and lungs
- o The large intestinal wall
- o The liver
- O The Pancreas
- o Ascariasis is an important infection in humans (*Ascaris lumbricoides*) and pigs (*Ascaris suum*) and individuals appear to be predisposed to either heavy or light worm burdens.
- o Mebendazole causes cytoplasmic microtubular degeneration and death of intestinal-dwelling adult Ascaris by selectively and irreversibly blocking their glucose uptake without affecting blood glucose levels in the host, including humans.2

### **Complications**

- o Poor nutritional state
- Failure to thrive
- Impaired cognition
- Nutritional deficiencies
- o Anaemia
- o Intestinal: intussusception, perforation, appendicitis, peritonitis, volvulus
- o Pancreatitis, cholangitis, jaundice, liver abscesses

Respiratory tract obstruction

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# **Treatment options**

- o Drugs available to treat Ascaris infections include pyrantel pamoate, <u>piperazine citrate</u>\* withdrawn, and mebendazole. Mebendazole is also the treatment ofchoice for trichuriasis.4
- o Physicians using mebendazole to treat ascariasis should inform patients and their families about the possibility of aberrant migration of Iwnbricoides from the nose or mouth.
- o Combination therapy with mebendazole, 150 mg, and pyrantel base, 34 mg, had the efficacy of mebendazole but did not cause erratic migration of A lumbricoides in more than 3,000 treated patients. Good to use irregardless of the worm type, just because it can kill the larvae.

Because of the risk of complications, patients with ascariasis who have other concomitant helminthic infections should always undergo treatment for ascariasis first.

Ascariasis commonly coexists with whipworm infection

Medical therapy is usually not indicated during active pulmonary infection because dying larvae are considered a higher risk for significant pneumonitis.

Anthelminth therapy is not usually given at the time of pulmonary symptoms because dying larvae may do more harm than migrating ones.

Standard therapy for intestinal ascariasis can be given after the worms have developed to maturity in the small intestine [6].

Single dose mebendazole, given either four times a year, decreased intensity of A. lumbricoides infection by 63 and 97 percent, respectively, it has a greater effect on the intensity of infection than on the overall prevalence [44-47].

**Follow-up** — All of these therapies act against the adult worm <u>but</u> <u>not the larvae.</u> Following therapy, patients should be reevaluated at two to three months to ensure that no eggs are detectable, either

because of inadequate elimination of adult worms or because of reinfection. Reinfection occurs frequently; more than 80 percent of individuals within six months [1]. Evaluation of other family members should be entertained whenever the diagnosis is made because of the propensity of the infection to cluster in families [10,12].

Treatment of Simple uncomplicated GI trac infection:

o therapy is mebendazole (100 mg bid for 3 d or 500 mg as a single dose). Mebendazole is not recommended during pregnancy;

### pyrantel pamoate

- o pyrantel pamoate is the drug of choice in most GI cases.
- o Severe disseminated infections should start with a single std dose of Pyrantel Pamoate, along with Nematode paralysis agent, to prevent migration into eyes, ears, nose, throat, larynx penetration, or scattering of worms.
- o **Pyrantel pamoate** Pyrantel pamoate (11 mg/kg up to a maximum of 1 g) is administered as a single dose. Adverse effects include gastrointestinal (GI) disturbances, headaches, rash, and fever. Parasite immobilization and death occur, although this happens slowly and complete clearance of the worm from the GI tract may take up to three days. Efficacy varies with worm load, but single dose therapy is approximately 90 percent effective in eradicating adult worms [6].

### Invermectin, DEC-C,

- o Invermectin, DEC-C, and others should be given at std dose levels concurantly to prevent migration
- o Paralyzing vermifuges (eg, pyrantel pamoate, piperazine, ivermectin) should be avoided in patients with complete or partial intestinal obstruction since the paralyzed worms may necessitate or further complicate surgery.
- o Drug therapy affects only adult worms. Larvae are not yet susceptible. Such patients should be re-treated every 3 months

o If stool ova persist beyond 2 years, additional exploration, coinfection enabling parasites should be considered.

### Mebendazole —

- o Mebendazole (100 mg BID for 3 days or 500 mg as a single dose) is an alternative. Adverse effects include transient GI discomfort, headache, and rarely leukopenia. The three-day regimen is approximately 95 percent effective, and the single dose seems to have similar results.
- \* **Albendazole** A single dose of albendazole (400 mg) is effective in almost 100 percent of cases(lie), although reinfection commonly occurs [39]. Albendazole causes the same adverse effects as mebendazole.
- >

o Nitazoxanide, a drug used primarily for protozoal infection, was shown to have 89% clinical efficacy for the treatment of ascariasis in rural Mexico and may offer a future alternative.

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o Vitamin A supplementation improved growth development of children in Zaire; deworming did not improve growth development in this study.[22]-

# **Management**

o Advise the patient to wash his or her hands thoroughly after using the toilet or changing nappies, and before eating or preparing food.[5]

### **Treatment Natural**

-

- o Ascaris eggs bring 3 very important pathogens that spread throughout your body: Rhizobium leguminosarum, Mycobacterium avum/intracellulare, and the common cold virus, **Adenovirus**. A flood of these are responsible for your **night sweats**! As soon as the last Ascaris egg is gone, these pathogens are gone, too, and the following night becomes free of sweating. **If your night sweats come back, you know Ascaris eggs are present again**. And in 24 hours, unless you kill them, they will hatch into larvae and start the whole cycle over again.
- o It takes about 3 weeks for large parasites like Ascaris and tapeworm larvae to disintegrate completely and be cleared from your tissues. If eggs or scolices are continually released during this time, the cycle of infection cannot be broken. Fortunately, the same two things that can penetrate tapeworm larvae can also penetrate Ascaris worms and mop up after them, whether dead or alive!
- o <u>L-cysteine</u>, 500mg, 2 capsules 3 times a day. After taking <u>6</u> <u>capsules daily</u> for 21days, go to Maintenance: 3 caps daily for 14 days
- o Ozonated olive oil, 1 tablespoon with food. Next day 2 tabsp, next day 3 tabsp. 400mg of VitC and Vit E two or more hours after the Olive Oil

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# **Prevention**

• Infection can be avoided by scrupulous attention to personal hygiene and the careful washing of all fruit and vegetables.

If the infected person defecates outside (near bushes, in a garden, or field) or if the feces of an infected person are used as fertilizer, eggs are deposited on soil. They can then mature into a form that is infective. Ascariasis is caused by ingesting eggs. This can happen when hands or fingers that have contaminated dirt on them are put in the mouth or by consuming vegetables or fruits that have not been carefully cooked, washed or peeled.

•

Dog and cat roundworm
 (toxocara canis, toxocara cati)
 Contracted through soil contaminated with dog faeces, cat or fox.

Humans are not intended hosts for the mature forms, but the immature forms can infect children causing <u>larva migrans -roundworm</u>, ground itch, creeping eruption.

Signs and symptoms: A high eosinophil count combined with anaemia as revealed by blood tests is typical. The larvae can hatch and travel to the lungs causing pneumonitis, to the intestines causing pain or to the liver causing enlargement in addition to the brain or eye. Dog heartworm (dirofilaria immitis) Contracted from a bite from an infected mosquito in tropical Africa, south-eastern Asia, South Pacific, Atlantic or the Gulf coast in the USA. Man is not a viable host for the mature worm, but larvae can remain in subcutaneous tissue or invade the lung. Signs and symptoms: If the If the larvae invade the lung, they can cause a cough and the lesions can be mistaken for lung cancer.

### TREATMENT FOR ROUNDWORMS

- Tapeworms, like **Echinococcus multiocularis**, have larvae inside their larvae! Streptomyces which accompanies the larvae makes ammonia out of your urea, two things that can penetrate a succession of membranes to kill the shielded larvae within, as well as any trapped eggs **are cysteine** and **ozonated olive oil**. When you kill Ascaris worms with the herbal recipe, they are mortally wounded. They are dying, but the eggs inside them are not. They were sheltered. Within a day these eggs begin to leave the dying worm. Soon hordes of eggs are dispersing in your body again! It takes about 3 weeks for large parasites like Ascaris and tapeworm larvae to disintegrate completely and be cleared from your tissues. If eggs or scolices are continually released during this time, the cycle of infection needs:
  - •Ozonated olive oil, 3 tbs. taken in the morning. (Pyrantel pamoate?)
  - •L-cysteine, 500mg, 3 capsules 2 times a day. Do not take within 5 hours of the ozonated oil. It would counteract the effect of the oil. Nevertheless, supplementing with cysteine should not be overdone. After taking 6 capsules daily for 3 weeks (plus baking soda at bedtime), go off it completely for one week.
- Rascal against tapeworms
  - "Kroeger Herb" product "Rascal"

<u>Co-enzyme Q10 should be done if parasite program not effective (for tapeworm and ascaris).</u>

 Co-enzyme Q10, take the large 3g definitive dose every fifth day until you are better

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# CoQ10, 100% Pure Powder bulk, Now Foods, 1 oz (28 g) (NOW-03158)

BulkSupplements Pure Coenzyme Q10 (COQ10) Powder (100 grams) http://www.bulksupplements.com/

- O **Pyrantel pamoate** Pyrantel pamoate (11 mg/kg up to a maximum of 1 g) is administered as a single dose. Adverse effects include gastrointestinal (GI) disturbances, headaches, rash, and fever. Parasite immobilization and death occur, although this happens slowly and complete clearance of the worm from the GI tract may take up to three days. Efficacy varies with worm load, but single dose therapy is approximately 90 percent effective in eradicating adult worms [6].
- Albendazole A single dose of albendazole (400 mg) is effective in almost 100 percent of cases, although reinfection commonly occurs [39]. Albendazole can causes the same adverse effects as mebendazole
- Ivermectin Ivermectin causes paralysis of adult worms and is approximately as effective as other available therapies but is not generally used.
- Paico Leaf can effectively eradicate roundworms in most cases. Pumpkin the seeds can be eaten whole or mashed and mixed with juice. Two or three hours after eating up to <u>25 ounces of seeds</u>, a laxative such as senna or prunes is recommended to purge the intestines.
- #Santonine. [Sant]
   The alkaloid of Cina is also a remedy for round worms. It is not a safe remedy as Cina and no more efficacious. The writer has observed convulsions produced by its use in too low potencies.
- Cascara this herb can be used after eating pumpkin seeds to help eliminate parasites from the body. It is milder than senna, but has a similar action. Wormwood can be taken as a tea. Do not take the pure oil as it is considered to be toxic. Wormseed can be taken as a tea. Do not use concentrated wormseed oil as it is too potent.
- Torrya seed can be used with chinaberry bark, quisqualis fruit and black plum

### **Protozoa**

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https://itunes.apple.com/us/app/solve-the-outbreak/id592485067?mt=8

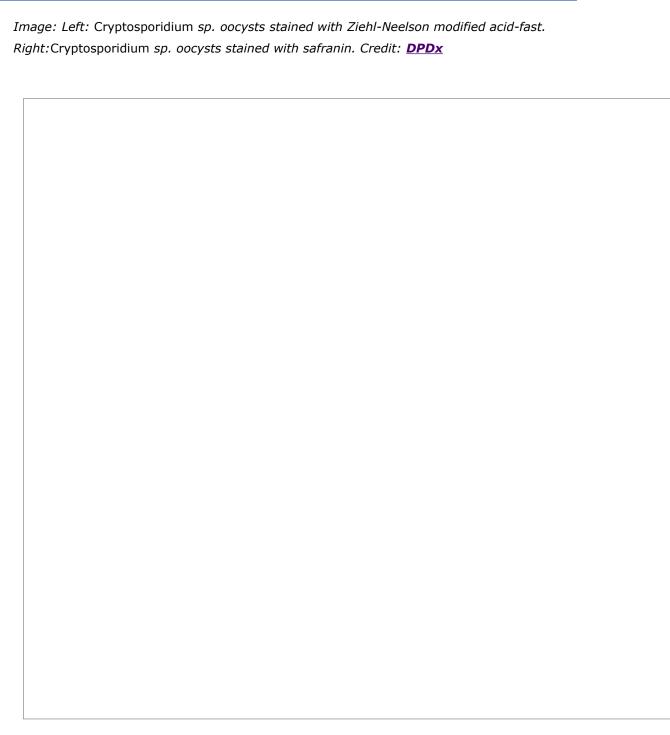
http://en.wikipedia.org/wiki/Cryptosporidium

# Parasites - <u>Cryptosporidium</u> (also known as "Crypto")

*Cryptosporidium* is a microscopic parasite that causes the diarrheal disease cryptosporidiosis. Both the parasite and the disease are commonly known as "Crypto."

There are many species of *Cryptosporidium* that infect humans and animals. The parasite is protected by an outer shell that allows it to survive outside the body for long periods of time and makes it very tolerant to chlorine disinfection.

While this parasite can be spread in several different ways, water (drinking water and recreational water) is the most common method of transmission. *Cryptosporidium* is one of the most frequent causes of waterborne disease among humans in the United States.



**Cryptosporidium** is a genus of apicomplexan protozoans that can cause gastrointestinal illness with <u>diarrhea</u> in humans. *Cryptosporidium* is the organism most commonly isolated in <u>HIV-positive</u> patients presenting with diarrhea. Treatment is symptomatic, with fluid rehydration, electrolyte correction and management of any pain. *Cryptosporidium* oocysts are 4-6 <u>µm</u> in diameter and exhibit partial <u>acid-fast</u> staining. They must be differentiated from other partially acid-fast organisms including <u>Cyclospora cayetanensis</u>.

# **General characteristics**[edit]

Cryptosporidium causes the diarrheal illness cryptosporidiosis.

Other <u>apicomplexan</u> pathogens include the<u>malaria</u> parasite <u>Plasmodium</u>, and <u>Toxoplasma</u>, the causative agent of <u>toxoplasmosis</u>. Unlike <u>Plasmodium</u>, which transmits via a <u>mosquito</u> vector, <u>Cryptosporidium</u> does not use an insect vector and is capable of completing its lifecycle within a single host, resulting in <u>cyst</u> stages that are excreted in feces and are capable of transmission to a new host.

A number of *Cryptosporidium* species infect mammals. In humans, the main causes of disease are *C. parvum* and *C. hominis* (previously *C. parvum* genotype 1). *C. canis, C. felis, C. meleagridis*, and *C. muris* can also cause disease in humans.

Cryptosporidiosis is typically an acute, short-term infection, but can become severe and nonresolving in children and immunocompromised individuals. In humans, it remains in the lower intestine and may remain for up to five weeks. [citation needed] The parasite is transmitted by environmentally hardy cysts (oocysts) that, once ingested, exist in the small intestine and result in an infection of intestinal epithelial tissue.

# <u>Treatment and detection[edit]</u>

Many <u>treatment plants</u> that take raw water from <u>rivers</u>, <u>lakes</u>, and <u>reservoirs</u> for public <u>drinking water</u> production use conventional filtration technologies. Direct filtration, which is typically used to treat water with low particulate levels, includes coagulation and filtration but not sedimentation. Other common filtration processes including <u>slow sand filters</u>, <u>diatomaceous earth filter</u>, and membranes will remove 99% of <u>Cryptosporidium</u>.

[4] Membranes and bag- and cartridge-filter products remove <u>Cryptosporidium</u> specifically.

*Cryptosporidium* is highly resistant to chlorine disinfection; <sup>[5]</sup> but with high enough concentrations and contact time, *Cryptosporidium* inactivation will occur with chlorine dioxide and ozone treatment. In general, the required levels of chlorine preclude the use of chlorine disinfection as a reliable method to control *Cryptosporidium* in drinking water. Ultraviolet light treatment at relatively low doses will inactivate *Cryptosporidium*. Water Research Foundation-funded research originally discovered UV's efficacy in inactivating *Cryptosporidium*. <sup>[6][7]</sup>

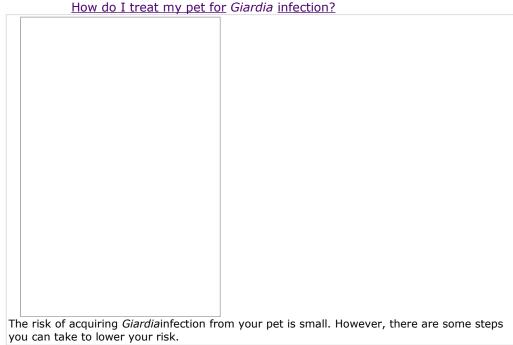
### Giardia and Pets

# On this Page

•	Can I get Giardia infection from my pet?	
•	How is Giardia spread?	
•	How does my dog or cat get infected with Giardia?	
•	How do I protect myself if my dog or cat has a Giardia infection?	
•	If my pet has a Giardia infection, how do I clean and disinfect my house?	
•	How do I reduce the amount of Giardia in my yard or outdoor	
environment?		
•	How long does Giardia survive in the environment?	
•	How often and for how long should I clean and disinfect my home after	

my dog or cat is diagnosed with Giardia infection?

• How do I prevent my dog or cat from getting re-infected, or sickening my other pets, during treatment?



Giardia intestinalis (aka: G.duodenalis, G.lamblia) is a common, microscopic (intestinal) parasite that commonly affects humans, dogs, and cats[1,2].

Common signs and symptoms of *Giardia* infection (in both humans and pets) are diarrhea, gas, abdominal discomfort, nausea, and vomiting. However, it is possible to be infected and have no signs or symptoms of illness[1,3,4].

Please visit our other web pages for a more detailed description of *Giardia*, including its <u>life</u> <u>cycle</u>, <u>prevention tips</u>, and <u>treatment information</u> for humans.

### Can I get Giardia infection from my pet?

The risk of humans acquiring *Giardia* infection from dogs or cats is small[4-7]. The exact type of *Giardia* that infects humans is usually not the same type that infects dogs and cats[5-7].

**Note:** If you own other household or exotic pets, please contact your veterinarian. Seek further information, as some rodents and other species can harbor human strains of *Giardia*.

### How is Giardia spread?

Anything that comes into contact with feces (poop) from infected humans or animals can become contaminated with the *Giardia* parasite. People and animals become infected when they swallow the parasite. It is not possible to become infected through contact with blood.

### How does my dog or cat get infected with Giardia?

Your dog or cat might get infected by:

- Being in contact with infected feces (poop) from another dog or cat
- Rolling and playing in contaminated soil
- Licking its body after contact with a contaminated surface (for example, a dirty litter box or dog cage or crate)
- Drinking water from a contaminated creek, pond, or other body of water

Young pets, like puppies and kittens, have a higher risk of infection than adult dogs and cats.

### How do I protect myself if my dog or cat has a Giardia infection?

The risk of acquiring *Giardia* infection from your dog or cat is small. However, there are some steps you can take to minimize your exposure to *Giardia* if you have dogs or cats:

- Wear gloves when gardening to reduce the risk of coming into contact with infected feces (poop) or soil.
- Clean household surfaces regularly.
- Clean and disinfect areas that your pet has access to—as well as items like toys, bedding, and water and food bowls—regularly.
- Wash hands frequently and properly:
- Wet your hands with clean, running water (warm or cold) and apply soap.
- o Rub your hands together to make a lather and scrub them well; be sure to scrub the backs of your hands, between your fingers, and under your nails.
- o Continue rubbing your hands for at least 20 seconds.
- o Rinse your hands well under running water.
- o Dry your hands using a clean towel or air dry them.

### If my pet has a Giardia infection, how do I clean and disinfect my house?

*Giardia* is hard to completely eliminate from the environment, but there are things you can do to decrease the risk of your pets' reinfection and of human infection.

• **Hard surfaces** (for example: cement and tile floors, crates, tables, trash cans, etc.)

### Cleaning

Wear gloves.

0

- Remove feces and discard in a plastic bag.
- Clean and scrub surfaces using soap. Rinse surface thoroughly until no obvious visible contamination is present.

### Disinfection

- Wear gloves.
- Disinfect according to manufacturer guidelines using **one** of the following:
  - Quaternary ammonium compound products (QATS)[4], which are found in some household cleaning products; the active ingredient may be listed as alkyl dimethyl ammonium chloride.
  - Bleach mixed with water (3/4 cup of bleach to 1 gallon of water)[8]
- Follow product instructions, ensuring the product stays in contact with the surface for the recommended amount of time.
- Rinse with clean water.
- Carpet / Upholstered Furniture
  - Cleaning
    - Wear gloves.

- If feces are on a carpet or upholstered furniture, remove them with absorbent material (for example, double layered paper towels).
- Place and discard the feces in a plastic bag.
- Clean the contaminated area with regular detergent or carpet cleaning agent.
- Allow carpet or upholstered furniture to fully dry.

### Disinfection

- Wear gloves.
- Steam clean the area at 158°F for 5 minutes or 212°F for 1 minute.
- QATS are found in some carpet cleaning products and can also be used after cleaning to disinfect. Read the product labels for specifications, and follow all instructions.

### • Other items (toys, clothing, pet bed, etc.)

 $\circ$  Household items should be cleaned and disinfected daily while a dog or cat is being treated for *Giardia* infection.

### Dishwasher

- Dishwasher-safe toys and water and food bowls can be disinfected in a dishwasher that has a dry cycle or a final rinse that exceeds **one** of the following:
- 113°F for 20 minutes
- 122°F for 5 minutes
- 162°F for 1 minute
- If a dishwasher is not available, submerge dishwasher-safe items in boiling water for at least 1 minute (at elevations above 6,500 feet, boil for 3 minutes).

### Washer and Dryer

- Clothing, some pet items (for example, bedding and cloth toys) and linens (sheets and towels) can be washed in the washing machine and then heat-dried on the highest heat setting for 30 minutes.
- If a clothes dryer is not available, allow clothes to thoroughly air dry under direct sunlight.

### How do I reduce the amount of Giardia in my yard or outdoor environment?

Giardia is hard to completely eliminate from the environment, but there are things you can do to help decrease the risk of pet reinfection and of human infection. Please remember that despite your best efforts to clean the environment, Giardia can persist in outdoor spaces and pet reinfection is possible[4].

Wear gloves when handling feces.

 $\circ$ 

- Remove feces promptly[4] and put them in a plastic bag.
- Limit access to common outdoor spaces, where possible, if pets have diarrhea or are being treated for *Giardia*.
- Eliminate any source of standing water (for example, puddles, containers with water, and fountains that are not in use).

- **Do not** attempt to use bleach or QATS in your soil or grass area, as they will be ineffective.
- **Do not** allow any new animals, especially young ones, to enter the yard or other outdoor space until advised by your veterinarian.

### How long does Giardia survive in the environment?

- In the soil[8,9]
  - o In cold temperatures (around 4°C/39.2°F), *Giardia* can survive for approximately 7 weeks (49 days).
  - O At room temperature (around 25°C/77°F), *Giardia* can survive for approximately 1 week (7 days).
- Dry vs. moist surface or environment
  - o In a dry, warm environment that experiences direct sunlight, *Giardia* can survive for only a few days[8,9].
  - o In a moist, cool environment, *Giardia* can survive for up to several weeks.
- Water[<u>10</u>]
  - o In water temperatures below 10°C/50°F (for example, lake water or puddle water during the winter, refrigerated water), *Giardia* can survive for 1–3 months.
  - o In water temperatures above 10°C/50°F (for example, river water during the fall, tap water, and puddles during the summer), *Giardia* can survive for less time than in colder temperatures. For example, in water above 37°C/98.6°F, *Giardia* can survive less than 4 days.

How often and for how long should I clean and disinfect my home after my dog or cat is diagnosed with *Giardia* infection?

- Clean and disinfect potentially contaminated items (toys, water bowls and food bowls, pet bedding, floors, dog crates, linens, towels, litter box, etc.) regularly for as long as your pet is sick.
- If your pet is taking medication, clean and disinfect frequently (daily if possible) until a few days after the last dose of medication is given.
- *Giardia* survival depends on many factors, so we recommend that you consult your veterinarian for further advice.

# How do I prevent my dog or cat from getting re-infected, or sickening my other pets, during treatment?

- If you have other dogs or cats, make sure you tell your veterinarian even if they are not showing signs of diarrhea. Other pets may also be put on medicine depending on the situation. Even animals showing no signs of *Giardia* infection could be infected and shedding *Giardia* into the environment [4].
- Bathe all household pets with pet shampoo following medical treatment to ensure no fecal residue is in the pet's coat[11].
- <u>Clean</u> dogs' and cats' environment[11] (holding areas, floors, crate, etc.) and wash water bowls daily with soap and water.
- Limit your dog's access to untreated surface water (creeks, ponds, lakes) to avoid re-infecting your animal and contaminating the water which could make other animals sick.

# How do I treat my pet for Giardia infection?

• If your pet has persistent diarrhea, seek veterinary care. Diarrhea has different causes and could result in dehydration or other serious complications.

- Diagnosis and treatment of Giardia infection must be done by a licensed veterinarian.
- No approved over-the-counter treatment is available for *Giardia* infection.
- *Giardia* can be passed in stool intermittently, and an animal may appear healthy or without signs of disease before it stops passing *Giardia*. Repeated fecal tests may be necessary[4].
- Follow your veterinarian's recommendations, and take your pet to all follow-up appointments.

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- and tap water. Appl Environ Microbiol. 1989;55(5):1223

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# **Ascaris Fluke Tapeworm Formula Giardia** infection (giardiasis): Giardia was originally observed by von Leeuwenhoek in 1681, in his own diarrheal stool, and was described by Vilem Dusan Lambl in 1859 and by Alfred Giard in

1895. The organism's previous name, honoring the contributions of Giard and Lambl, was bestowed in 1915.				
role as a pe community returning f	O Although <i>G intestinalis</i> was the first protozoan parasite described, its role as a pathogenic organism was not recognized until the 1970s, after community outbreaks and after the appearance of the disease in travelers returning from endemic regions. Prior to that time, the organism was thought to be a harmless commensal organism of the intestine.			
	to cause disease, and several different strains may be found in one host during			
o chronic dia	G intestinalis can cause asymptomatic colonization or acute or rrheal illness			
causative a	o Giardiasis is a major diarrheal disease found throughout the world. The flagellate protozoan <i>Giardia intestinalis</i> (previously known as <i>G lamblia</i> ), its causative agent, is the most commonly identified intestinal parasite in the United States and the most common protozoal intestinal parasite isolated worldwide. [1, 2, 3, 4] Infection is more common in children than in adults. [5, 6]			
o using unic force.	Giardia is a trophozoite to enterocytes in the small intestine, cellular flagellate motor			
o animal, w transmissi	Human human, Human ater, food based ion			
o <i>G. intestinalis</i> can cause asymptomatic colonization or acute or chronic diarrheal illness. The organism has been found in as many as 80% of raw water supplies from lakes, streams, and ponds and in as many as 15% of filtered water samples. [7.8] It is a common cause of chronic diarrhea and growth retardation in children in developing countries.				
<ul> <li>The organism has been found in as many as 80% of raw water supplies from lakes, streams, and ponds and in as many as 15% of filtered water samples.</li> </ul>				
0	Stomach acid triggers encapsulation to cyst			
0	Cysts survive for weeks			
o transmissi	Fecal oral infection is possible with 10-100 cysts required for ion			
0	Gene switching defense to the immune system			
o are Argir	4 proteins excreted that nine diminase adi, ornithine			

carbamoyl transferase oct alpha enolase and elongation factor 1a.

### adi and oct inhibits host innate immune response via interference of nitric oxide

- Subtracts Arginine from host in epithelial cells, <u>Giardia can</u> also prevent the formation of nitric oxide, a compound known to inhibit giardial growth, by consuming local arginine, which effectively removes the substrate needed by enterocytes to produce nitric oxide. This mechanism may contribute to <u>Giardia</u> -induced enterocyte apoptosis because arginine starvation in these cells is known to cause programmed cell death.[16, 25]
- Damage to the epithelium
- Kills small intestine cells through capase 3 and capase 9 expression
- Increased bax expression, and decrease in bcl-2
- Following apoptosis signaling, breakdown of tight junctions and loose gut starts
- T cell deficient agammaglobulinemia leads to chronic
   Giardiasis
- Mucosal surface absorption of nutrients, electrolytes and water are associated with CD8+ lymphocyte shortening of the microvilli.
- Malabsorptive diarrhea and lower weight gain are associated with chronic Giardiasis.
- Chronic diarrhea is the main clinical sign.
- o Giardia is recognized as a zoonosis by the WHO, which generated data as to the characterization of isolates

### **Symptoms**

- Giardiasis typically causes abdominal pain, nausea, bloating,
- Giardiasis typically causes diarrhea, soft or greasy stools,
- It can cause chronic diarrhea lasting for several weeks, in addition to vague pain, weight loss, excessive burping, bloating, and fatigue.

Risk

**Symptoms** 

apeworm rormana			
<ul> <li>Marked or moderate partial villous atrophy in the duodenum and jejunum can be observed in histologic sections from asymptomatic individuals who are infected.</li> </ul>			
O Disrupting the mucosal epithelium, effects in the intestinal lumen may contribute to malabsorption and the production of diarrhea.			
<ul> <li>Varying degrees of malabsorption of sugars (eg, xylose, disaccharides), fats, and fat-soluble vitamins (eg, vitamins A and E) may contribute to substantial weight loss.</li> </ul>			
O Giardia -induced disruption of epithelial tight junctions, which, in turn, increases intestinal permeability. Loss of epithelial barrier function is a result of Giardia -induced enterocyte apoptosis (chewing of cells)			
<ul> <li>Giardia is a parasite found in contaminated water in every country in the world.</li> </ul>			
o <u>Untreated, Giardia causes significant morbidity.</u>			
<ul> <li>Humans contract giardiasis after consuming food or water that is contaminated with Giardia lamblia.</li> </ul>			
between animals and humans. <i>Giardiaintestinalis</i> has been isolated from the stools of beavers, dogs, cats, and primates. Beavers may be an important reservoir host for <i>G intestinalis</i> . [9, 10, 11] Other <i>Giardia</i> species include <i>G muris</i> in rodents; <i>G agilis</i> in amphibians; <i>G psittaci</i> and <i>G ardeae</i> in birds; and <i>G microti</i> in voles and muskrats. [12, 13, 14]			
Once consumed, the parasitic eggs (called cysts) hatch in the stomach. The parasite then attaches to the small intestine where it feeds on the food that is consumed by its host.  O Giardia causes nausea, vomiting, malabsorption, diarrhea, and weight loss/Gain			
o <i>G intestinalis</i> has been implicated as the chief cause of growth retardation in infected children, even after other diarrhea-causing agents are controlled. <sup>[22, 15]</sup>			

Symptoms of giardiasis include the following<sup>[5, 6, 15]</sup>:

- Diarrhea
- Malaise, weakness
- Abdominal distention, bloating, pus, mucus in stool, blood in stool
- Flatulence
- Abdominal cramps, decreased stomach acid
- Nausea
- Stenorrhoea pale, foul smelly Malodorous, greasy stools
- Sulphuric-tasting belch
- Excessive gas
- Anorexia, loss in interest for food, can last for weeks or months
- Weight loss, Weight loss occurs in more than 50% of patients and averages 10 pounds.
- Vomiting, some unexpectily report previous episode of projectile vomiting for days or weeks
- Low-grade fever (infrequent)
- Various neurologic symptoms (eg, irritability, sleep disorder, mental depression, neuroasthenia)
- Urticaria
- Prolonged Chronic Gasteric or acute systemic infections in persons with lack of IgA response. (16% of the population)
- Lactase deficiency
- Precursor to Chrones, celiac disease
- The nature of the overall clinical manifestations in affected patients is influenced by numerous factors, including the parasite load, virulence of the isolate, and the host immune response.
- Diarrhea is the most common symptom of acute *Giardia* infection, occurring in 90% of symptomatic subjects. Abdominal cramping, bloating, and flatulence occur in 70-75% of symptomatic patients.
- Symptoms of chronic infection include chronic diarrhea, malaise, nausea, and anorexia. Weight loss, as extensive as 10-15 pounds in an adult, occurs in approximately 66% of symptomatic patients. Chronic sporadic diarrhea may continue for months.

Postinfection lactose deficiency also is a common finding, occurring in 2-40% of cases.

- Stools become malodorous, mushy, and greasy. Watery diarrhea may alternate with soft stools or even constipation. Upper GI symptoms, often exacerbated by eating, accompany stool changes or may be present in the absence of soft stools. These include upper and midabdominal cramping, nausea, early satiety, bloating, substernal burning, and acid indigestion
- Extraintestinal manifestations are rare and include allergic manifestations such as urticaria, erythema multiforme, bronchospasm, reactive arthritis, and biliary tract disease. The etiology of such extraintestinal symptoms is likely a result of host immune system activation and cross-reactivity/molecular mimicry.

Chronic illness may occur. Adults may present with long-standing malabsorption syndrome and children, with failure to thrive.

Complications of giardiasis may include the following:

- Development of chronic illness
- weight loss
- Malabsorption syndrome in adults
- Failure to thrive in children
- Disccharidase deficiency
- Giardiasis typically causes diarrhea, which causes the body to lose water and salts. As a result, patients may become dehydrated.
- Patients with giardiasis may become lactose intolerant. Individuals may continue to be lactose intolerant for several weeks after the infection is treated.
- Zinc deficiency in schoolchildren<sup>[47]</sup>
- Growth retardation<sup>[33]</sup>
- Persistent gastrointestinal symptoms<sup>[48]</sup>
- References

### **Differential Diagnoses**

- Amebiasis
- Crohn Disease
- Cryptosporidiosis

- Food Poisoning
- Gastroenteritis, Viral
- Irritable Bowel Syndrome
- <u>Lactose Intolerance</u>
- Sprue
- Strongyloidiasis

### **Prevelance**

 Giardiasis is common throughout the world, including the United States.

### **Tests**

Stool ova and parasite studies are diagnostic.

### **Stool Examination**

- Stool examination trophozoites or cysts is the traditional method for diagnosing giardiasis. At least 3 stools taken at 2-day intervals should be examined for ova and parasites. Trophozoites may be found in fresh, watery stools but disintegrate rapidly. If the stool is not fresh or is semiformed to formed, trophozoites will not be found.
- O The traditional basis of diagnosis is identification of *Giardia intestinalis* trophozoites or cysts in the stool of infected patients via a stool ova and parasite (O&P) examination. Stool antigen enzyme-linked immunosorbent assays also are available.
- The traditional basis of diagnosis is identification of *Giardia intestinalis* trophozoites or cysts in the stool of infected patients via a stool ova and parasite (O&P) examination. Stool examination may be performed on fresh specimens or after preservation with polyvinyl alcohol or 10% formalin (with appropriate staining).
- o Ideally, 3 specimens from different days should be examined because of potential variations in fecal excretion of cysts. *G intestinalis* is identified in 50-70% of patients after a single stool examination and in more than 90% after 3 stool examinations.
- Stool O&P testing aids in the diagnosis of giardiasis in 80-85% of patients. It remains the diagnostic method with which other tests are compared. Aspiration of duodenal contents and demonstration of trophozoites also have been used for diagnosis but this is more invasive than stool examination and, in direct comparison studies to stool microscopy, may have a lower diagnostic yield.

o If the results from 3 O&P tests are negative and giardiasis is still suspected, stool antigen enzyme-linked immunosorbent assay (ELISA) may be helpful. If both of these methods result in negative findings but the patient has symptoms consistent with small bowel diarrhea/malabsorption, upper endoscopy with biopsies and duodenal aspirate is a reasonable alternative.

### **Antigen**

Stool antigen enzyme-linked immunosorbent assays also are available.[49] These tests are similar to the stool O&P test in terms of cost and have a sensitivity of 88-98% and a specificity of 87-100%. These tests are best used as a screening test in high-incidence settings such as day-care centers or for identification of subjects during an epidemic, but they should not take the place of stool microscopy.

# **Stool Antigen Detection**

- Several tests to detect *Giardia* antigen in the stool are commercially available.[31, 12, 50] These utilize either an immunofluorescent antibody (IFA) assay or a capture enzyme-linked immunosorbent assay (ELISA) against cyst or trophozoite antigens. These tests have a sensitivity of 85-98% and a specificity of 90-100%.
- o Polymerase chain reaction (PCR) techniques may detect giardia in stool samples with parasites concentrations as low as 10 parasites/100 mcL. PCR may also be a valuable tool for screening of water supplies.[53] Real-time PCR has also the advantage of being able to detect both mild and asymptomatic infections.[54]
- While more sensitive than stool examination, these examinations are limited to the detection of *Giardia*; isolated use might result in missing an alternative or concurrent parasitic infection.
- A 2009 study evaluated a screening test for Giardia and Cryptosporidium on 136 fecal samples. The results showed the test to be 98.4% sensitive and 100% specific; the positive and negative predictive values were 98.7% and 99.3%, respectively.[51]

### **Culture**

- Stool culture is not routinely used because of the difficulty of reproducibly isolating *Giardia* from patient fecal samples. However, stool cultures are beneficial in ruling out other pathogens as the cause of a patient's symptoms.
- o Routine laboratory tests (eg, CBC count, electrolyte levels) usually show normal results. Eosinophilia is an uncommon feature of infection.
- Because immunoglobulin G (IgG) levels remain elevated for long periods, they are not beneficial in making the diagnosis of acute giardiasis.
   Serum anti-Giardia immunoglobulin M (IgM) can be beneficial in distinguishing between acute infections and past infections.

### **String Test**

- The string test (Entero-test) consists of a gelatin capsule containing a nylon string with a weight attached to it. The patient tapes one end of the string to his or her cheek and swallows the capsule. After the gelatin dissolves in the stomach, the weight carries the string into the duodenum.
- The string is left in place for 4-6 hours or overnight while the patient is fasting. After removal, it is examined for bilious staining, which indicates successful passage into the duodenum. The mucus from the string is examined for trophozoites in an iodine or saline wet mount or after fixation and staining.

### **Test-other**

 Smear of duodenal fluid aspirate is an examination of fluid taken from the duodenum to check for signs of a possible infection (such as giardia or strongyloides).

### **Alternative Test Names**

Duodenal aspirated fluid smear

### **How the Test Is Performed**

For information on how the sample is taken, see:
 Esophagogastroduodenoscopy (EGD)

### **How to Prepare for the Test**

 Do not eat or drink anything, even water, for 12 hours before the test.

### **What Abnormal Results Mean**

• The results may show the presence of giardia protozoa, the intestinal parasite strongyloides, or another infectious organism.

### **Blood**

- Fecal fat quantification or a qualitative fecal fat analysis with Sudan stain may confirm steatorrhea. Serum carotene, folate, and vitamin B-12 levels may be variably depressed as a result of malabsorption. The findings from D-xylose absorption tests may be abnormal.
- Disaccharidase deficiency is common during and after treatment and can be diagnosed with the aid of a lactose tolerance breath test. [52]

 Serum electrophoresis can help diagnose immunoglobulin A, immunoglobulin M, and, occasionally, immunoglobulin G deficiency states.

### **Treatment**

- The prognosis for patients with giardiasis is generally excellent.
- Metronidazole is the antimicrobial agent most commonly used in the treatment of giardiasis in the United States. Although most experts recommend metronidazole and tinidazole as the drugs of choice because the brief treatment periods encourage good patient adherence, treatment failures occur in as many as 20% of cases, probably because of resistance. Therefore, treatment with a second-line drug (eg, mepacrine) may be necessary. It has a cure rate of 85-90%. Metronidazole is a nitroimidazole that, once concentrated within the organism, is reduced by intracellular electron transport proteins. The formation of free radicals causes disruption of cellular elements and subsequent death of the organism. It is the most commonly prescribed antibiotic for giardiasis. The recommended adult dose is 250 mg PO tid for 5-7 days. (update 2004: Metronidazole (30 to 40 mg/kg per day divided into three doses for five to seven days) has an efficacy of 80 to 95 %.)
- Standard treatment for giardiasis consists of antibiotic therapy.
   Metronidazole is the most commonly prescribed antibiotic for this condition;
   however, tinidazole is now approved in the United States and is considered a first-line agent outside the United States. (See Treatment.)

### Albendazole (Albenza)

Dosing, Interactions, etc.

**Clinical Context:** This agent decreases adenosine triphosphate (ATP) production in worms, causing energy depletion, immobilization, and, finally, death. To avoid an inflammatory response in CNS, the patient also must be started on anticonvulsants and high-dose glucocorticoids.

- Works in children, may not work at all in adults
- Tinidazole is given in a single dose, the efficacy is reported at 90%, and it is believed to have fewer side effects than metronidazole. A common adverse effect is GI upset. Clinical Context: Tinidazole is a nitroimidazole antiprotozoal agent. The mechanism by which tinidazole exhibits activity against Giardia and Entamoeba species is not known. The recommended adult dose is 2 g PO once; for children, the recommended dose is 50 mg/kg PO once. (2004: A single 2 g dose (or 50 mg/kg for children) of tinidazole has an efficacy of more than 90 % with few associated side effects]. Tinidazole has been found to be safe in several pediatric studies but currently is only available in tablet form. Preliminary in vitro data suggest that newer nitroimidazole antibiotics in development may have even greater benefit. Furazolidone, which is available as a suspension, is 72 to 100 % effective when given for 7 to 10 days and is well-tolerated in children.)

- Paromomycin has been recommended for use in pregnancy because systemic absorption is low, but the cure rate is lower than with other agents.
- Quinacrine achieves a cure rate of 90-95% but is available as an orphan drug in the US. Clinical Context: This agent, available as an orphan drug in the US is indicated to treat giardiasis and cestodiasis. It is occasionally used to treat and suppress malaria. The recommended adult dose is 100 mg PO tid for 5-7 d; for children, the recommended dose is 2 mg/kg PO tid for 5-7 d. The effectiveness of quinacrine is similar to that of nitroimidazole derivatives; however, it is less tolerated because of its adverse effects. These include the following: mild and transient headache, dizziness, and GI complaints (diarrhea, anorexia, nausea, abdominal cramps, vomiting [rare]), pleomorphic skin eruptions, and neuropsychiatric disturbances (nervousness, vertigo, irritability, emotional change, nightmares, transient psychosis).

### **Nitazoxanide (Alinia)**

• <u>Dosing, Interactions, etc.</u>

**Clinical Context:** This agent inhibits growth of Cryptosporidium parvum sporozoites and oocysts and Giardia lamblia trophozoites. It elicits antiprotozoal activity by interfering with pyruvate-ferredoxin oxidoreductase (PFOR) enzyme-dependent electron transfer reaction, which is essential to anaerobic energy metabolism. It is available as a 20-mg/mL oral suspension.

References

### **Natural treatment**

- The parasite expert Dr Louis Parish has warned of the misconception that "treatment with a single course of metronidazole is 90% effective" Twenty five years that may have been the case but the protozoa rapidly became resistant.. Today the single cure rate is less than 5%. The ability of parasites to adapt is actually quite remarkable. Like bacteria parasites can use their encysted or resting periods to toughen their cell walls to a nearly impermeable state. Long term natural treatment may be the only successful way to prevent re-infection.
- The most beneficial way to treat giardiasis naturally may be through a combination approach, utilizing both nutritional interventions and phytotherapeutic agents. The main aims of nutritional intervention are to reduce the acute symptomatology of giardiasis, promote host defense mechanisms, and inhibit growth and replication of Giardia trophozoites. These aims can best be achieved by consuming a whole-foods, high fiber, low-fat, low simplecarbohydrate diet. Additionally, ingestion of wheat germ and probiotics can aid in parasite clearance.
- O The most promising phytotherapeutic agents in the treatment of giardiasis appear to be the berberine-containing herbs, garlic, and the Ayurvedic combination Pippali rasayana, although other medicinal herbs also show great potential. Blending nutritional interventions and phytotherapeutic agents should result in minimization of Giardia symptomatology and clearance of the parasite, without significant side effects.

### Artemisia Annua (sweet wormwood) Qing huo

- This herb is renowned for its use in treating malaria where its effectiveness is confirmed in both historical data and modern pharmacology. Accordint to WHO (World Health Organisation 1.5 million malaria patients in South East Asia and Latin America were treated with artemisin, a constituent of Qing Huo. In China Falciparum plasmodium resistance to chloroquine is 84.6% but only 2.2-2.4% to artemisinin. Thus a remisinin has completely replaced chloroquine and quinine as drugs of choice fror treatment of malaria. However, it is important to keep in mind that while it is effective as a drug for treatment it is not effective as a casual prophylactic agent.
- Qing huo has shown few adverse reactions in toxicology studies. In chronic toxicology studies no abnormalities were reported in heart, liver, kidneys, and other vital organs following long term administration. (Ref Chinese Herbology and Pharmacology by John and Tina Chen)
- Dose 3 10 grms in decoction. The herb should only be cooked for a short period of time as heat may make it less effective. i.e no more than 15 mins, Tincture: 7.5 10 ml per day 1.2 tincture

## Kidney herbs

### **Couch Grass**

• (agropyron repens) Parts used the rhizome, A good diuretic and its demulcent properties is good for irritation and inflammation of the kidneys. Useful in removing kidney stones and gravel

### **Gravel Root**

• (Eupatorium purpureum) Parts used Rhizome and root Primary use for kidney stones and gravel

### **Hydrangea**

• (Hydrangea arborescens) Parts used Dried roots and rhizome, Good in a kidney combination formulae

### **Parsley Piert**

• (Aphanes arvensis) Parts used Aerial parts A potent diuretic and good for painful urination. Commonly used for kidney and urinary stones and gravel. Also good for water retention due to kidney and liver problems.

### Alisma

• (Alisma orientalis) Parts used Rhizome Has a potent function to regulate water circulation and resolve fluid accumulation. A good diuretic

• In all kidney problems the above combination is a base formulae. The dose is 20ml per herb per week to equal 100ml and this can be doubled to give a stronger dose.

# Chologagues

### **BALMONY**

• (Chelone glabra) Parts used Dried aerial. From the North American Indians it has a long history as a curative remedy for liver and gallbladder problems. Used for gallstones and inflammation of gallbladder.

### **FRINGETREE**

• (Chionanthus virginicus) Parts used The root bark Specific for all liver and gallbladder problems. Gallstones and inflammation of gallbladder

### **BARBERRY**

(Berberis vulgaris) Parts used bark of root or stem.
 Excellent for stimulating bile flow and correcting liver function.
 Gallstones and inflammation of gallbladder

### **BOLDO**

• (Peumos boldo) Parts used Dried leaves. Specific for gallbladder problems. Gallstones and inflammation of gallbladder

### **WAHOO**

• (Euonymus atropurpureus) Part used Root bark. Primary liver herb and used for pain due to congestion of stones. Inflammation of gallbladder.

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Power vs Force The Hidden Determinants of Human Behavior bt David Hawkins

- O **Gastrointestinal infection** berberine hydrochloride (5 mg/kg/d), or metronidazole for six days. A longer duration of treatment would increase berberine's efficacy.
- Piper Longum extract 250 ug/mL water, 125ug ethanol –
   100% 5 days, systemic infection
- o Pippali rasayana (piper longum and Butea monosperma (polash) 900mg/kg 98% in 3 doses, significantly increased macrophage migration index and phagocytic activity, with 225mg/km showing the greatest migration activity. The increase in host immune response was the only means of clearance, in that the macrophage increased activity, but no toxic effectiveness against Giardia was noted.
- Epicatechin epigallocatechin, kaempferol, quercetin, and apigenin all were substantial anti-guiardial flavonoids herbs. Herbs rich in flavonoids and tannins such as oregano vulgare and guava psidium guajava both demonstrated activity superior to Tinidazole.
- Mango leaves mangifera indica and plantain leaves plantago major were also as effective as tindazole.
- Quercetin, apples, kale, French beans, parsley, black currants aid in Giardia clearance.
- Goldenseal 10mg/kg/D 1:1 extract, 20mg/kg/D capsule 5-10
   D 83% GI clearance sucess

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Toxoplasma gondii, A common, crescent-shaped parasite that invades the central nervous system. Humans become infected with this organism by eating undercooked meat or by handling infected cat litter or soil where cats have defecated. Most people have been exposed to this parasite and show antibodies for it, but few individuals show symptoms. Those with a compromised immune system are more susceptible, and foetuses can suffer serious or fatal effects from infection.

### **Amebia**

### **Amoebiasis**

### **Amoeba**

 Amoebae are microorganisms that infect the end of the smaller intestine and colon. They release an enzyme that causes ulcers

or abscesses where they can enter the bloodstream. They can eventually reach other organs like the brain or liver.

o Naegleria fowleri, Acanthamoeba spp., Balamuthia mandrillaris, and Sappinia sp. are pathogenic free-living amoebae.

### **Entamoeba histolytica**

http://www.nytimes.com/health/guides/specialtopic/travelers-guide-to-avoiding-infectious-diseases/traveler's-diarrhea.html?module=Search&mabReward=relbias%3Aw

### **Types**

- Single-celled organisms produce small stools that contain blood and mucus, pus, and open sores.
- o **Cutaneous amoebiasis** refers to a form of <u>amoebiasis</u> that presents primarily in the **skin**. It can be caused by <u>Acanthamoeba<sup>[1][2]</sup></u> or <u>Entamoeba histolytica</u>. [3] When associated with <u>Acanthamoeba</u>, it is also known as "**cutaneous acanthamoebiasis**". [4]

### **Symptoms**

as " <b>cutaneous acanthamoebiasis</b> ". E			
o fatal disease	<i>N. fowleri</i> causes Primary Amoebic Men of the <b>central nervous system</b>	ingoencephalitis, a <b>rapidly</b>	
o granulomato	Acanthamoeba spp. and B. mandrillaris us <b>encephalitis</b> .	cause <b>chronic</b>	
o as the cause encephaliti	Sappinia pedata has been identified of a nonlethal case of amoebic s.		
weight loss	E. histolytica can cause ulcerations, bloody diarrhea, fever, gastrointestinal and peritonitis.		
o into the ple	Amebas can cause abscesses in the ural space, peritoneum, or pericardiu	•	
0	Acanthamoeba spp. also can cause cut	aneous lesions	
o the skin, [6] a	Balamuthia mandrillaris has been descr nd it can have <b>cutaneous</b> open sore ex		
o is associated trauma.	Amoebic Keratitis, a sight-threatening in with contact lens use or corneal	nfection of the cornea that	
	Diarrhea frequently occurs within ek of travel, but may develop at even after returning home. Traveler's	diarrhea causes four or	

five loose or watery stools per day. Vomiting may also occur. It usually lasts 3 or 4 days, but about 14% of cases last longer. In rare cases, the diarrhea lasts more than 3 months. When TD lasts a long time, it can cause post-infectious disease.

		Cause		
			0	This
	single-celled organism causes a disease called <b>amoebiasis</b> .			
	<ul> <li>It predominantly infects humans and other primates.</li> </ul>			
RISK				
	<ul> <li>It can be found in water, damp environments and in soil, and can contaminate fruits and vegetables.</li> <li>Amoebia have been isolated from</li> </ul>			

o It can spread through faecal contamination. Other than the malarial parasite, it causes more deaths than any other protozoan.

freshwater lakes, thermally polluted waters, sediment, thermal springs, swimming pools, soil, air conditioning vents, air, and the domestic water

Amoebiasis can co-harbor intracellular pathogenic bacteria such as Legionella pneumophila and may serve as vectors of other bacterial infections in humans. Mycobacterium avium, Burkholderia spp., Escherichia coli O157:H7, and Vibrio cholerae, can survive and multiply in Amoebia [6, 21–26]. Intracellular growth of bacteria within amebae have been shown to increase resistance to antibiotics and to biocides, and to increase virulence [20, 23–26].

0

supply.

### **Locations**

- Entamoeba histolytica has a worldwide distribution, with a higher incidence of amebiasis in developing countries.
- Prevalent Mexico, India, Africa, and Central and South America.
- o Risk groups in industrialized countries include homosexual males, travellers and recent immigrants (although disease may develop months to years after exposure) and institutionalized populations.

### **Progression**

o Infection by E. histolytica typically occurs by ingestion of mature cysts in faecal contaminated food, water, or hands. Cysts can

survive for days to weeks in the external environment. Transmission can also occur through exposure to faecal matter during sexual contact (in this case not only cysts but also the far less durable trophozoite stage could prove infective).

### **Symptoms:**

- If the condition becomes chronic, it can resemble inflammatory bowel disease (IBD).
- Abdominal pain, weight loss, weakness, diarrhoea, liver abscess

### **Testing**

- Stool and serologic assays, biopsy, barium studies, and liver imaging have diagnostic merit.
- o In view of the potential health consequences due to infection with these amoebae, rapid diagnosis is critical for early treatment. Microscopic examination and culture of biopsy specimens, cerebral spinal fluid (CSF), and corneal scrapings have been used in the clinical laboratory. For amoebic keratitis, confocal microscopy has been used to successfully identify amoebae in corneal tissue. More recently, conventional and real-time PCR assays have been developed that are sensitive and specific for the amoebae. In addition, multiplex PCR assays are available for the rapid identification of these pathogens in biopsy tissue, CSF, and corneal specimens.

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### **TREATMENT FOR AMOEBIASIS.**

- Therapy includes luminal and tissue amebicides to attack both life-cycle stages. Metronidazole, <u>chloroquine</u>, and aspiration are treatments for liver abscess.
- Corticosteroids are used to treat IBD. <u>Steroids can</u>
   <u>cause death</u> in histolytica cases, and can have dangerous effects in people carrying the parasite.

### **Avoidance**

 Careful sanitation and use of peeled foods and bottled water are preventive.

### **Herbal Treatment**

 Black seed, cloves, gentian root, fennel seeds, green black walnut husks, hyssop leaves, oregano, peppermint leaves, pumpkin seeds, thyme leaves

- Cryptosporidiosis is a parasitic infection caused by Cryptosporidium parvum. Once a person is infected by the protozoan, the parasite resides in the intestine and then is passed into the stool of the infected person. "Crypto" as the parasite and disease are commonly known as, is a diarrheal disease; symptoms include watery diarrhea, dehydration, cramps and nausea.
- O Crusty area on the skin, nose, or foot. In other cases, skin problems which may start out small can progress and develop into more extensive lesions. They may open, drain, and then develop a crusty surface. In some instances there may be hair loss, and the surface of the skin can become red and oozing, and ulcers may develop
- Crypto has gained particularly notoriety during the past two decades as it has become one of the most common causes of waterborne diseases in the United States. It is spread easily by contaminated food and water thus making cleanliness vitally important in its prevention and control.
- 1. Abdominal cramps
- 2. Nausea
- 3. low-grade fever
- 4. Dehydration
- 5. Weight loss

# Treatment of Mild to moderate protozoan intestinal disease3

- o >no> Drug of choice:4 **Metronidazole** 500-750 mg tid x 7-10d 35-50 mg/kg/d in 3 doses x 7-10d
- $\circ$  >no> OR Tinidazole5 2 g once daily x 3d 50 mg/kg/d (max. 2g) in 1 dose x 3d
- Severe intestinal and extraintestinal disease3
- $_{\odot}$  Drug of choice: Metronidazole 750 mg tid x 7-10d 35-50 mg/kg/d in 3 doses x 7-10d
- OR Tinidazole5 2 g once daily x 5d 50 mg/kg/d (max. 2 g) x 5d

### **Trichomoniasis**

http://en.wikipedia.org/wiki/Trichomoniasis

"Trich" redirects here. For the hair-pulling disorder, see Trichotillomania.

Not to be confused with Trichinosis or Trichuriasis.

**Trichomoniasis**, is a common cause of <u>vaginitis</u>. It is a <u>sexually transmitted infection</u>, and is caused by the single-celled <u>protozoan</u> parasite <u>Trichomonas vaginalis</u> producing mechanical stress on host cells and then ingesting cell fragments after cell death. [1] Trichomoniasis is primarily an infection of the <u>urogenital tract</u>; the most common site of infection is the <u>urethra</u> and the <u>vagina</u> in women.

Trichomoniasis is a sexually transmitted disease (STD) caused by a small organism called *Trichomonas vaginalis*. Women are most often affected by this disease, although men can become infected and pass the infection to their partners through sexual contact.

Trichomonas vaginalis infection is the most common non-viral STI in the world with an estimated 248 million new cases per year. [22][23] It is more common in women (2.7%) than males (1.4%). [24]

Trichomoniasis is the most common curable STD in young, sexually active women. An estimated 7.4 million new cases occur each year in women and men worldwide.

It is also the most common non-viral STI in the U.S., with an estimated 3.7 million prevalent cases and 1.1 million new cases per year. [25][26] Recent studies have posited prevalence to be 3% of the general U.S. population, [8][27] and 7.5-32% of moderate-to-high risk (including incarcerated) populations. [28][29][30][31][32][33][34][35]

# Signs and symptoms[edit]

Symptoms experienced include pain, burning or itching in the penis, urethra (urethritis), or vagina (vaginitis). Discomfort for both sexes may increase during intercourse and urination. For women there may also be a yellow-green, itchy, frothy, foul-smelling ("fishy" smell) vaginal discharge. In rare cases, lower abdominal pain can occur. Symptoms usually appear within 5 to 28

days of exposure. [2] In many cases, men may hold the parasite for some years without any signs.

yellowish discharge accompanied by itching and burning

The **epididymis in** Males - It is a single, narrow, tightly-coiled tube (in adult humans, six to seven meters in length<sup>[1]</sup>) connecting the <u>efferent ducts</u> from the rear of each <u>testicle</u> to its <u>vas deferens</u>.

Men often do not have symptoms of trichomoniasis and usually do not know they are infected until their partners need treatment. But when symptoms do occur, they include:

Continue reading below...

- Irritation inside the penis
- Mild discharge
- Slight burning after urination or ejaculation

Many women do have signs or symptoms of infection. Symptoms in women can include:

- Greenish-yellow, frothy vaginal discharge with a strong odor
- Painful urination
- Vaginal itching and irritation
- Discomfort during intercourse
- Lower abdominal pain (rare)

Symptoms usually appear within five to 28 days of exposure in women.

### **Diagnosis**[edit]

1How Is Trichomoniasis Diagnosed?

To diagnose trichomoniasis, a doctor must perform a physical exam and lab test. Lab tests are performed on a sample of vaginal fluid or urethral fluid to look for the disease-causing parasite. The parasite is harder to detect in men than in women.

There are three main ways to test for Trichomoniasis: (1) Saline microscopy. This is the method most commonly used. It requires an endocervical, vaginal, or penile swab specimen for examination under a microscope. The presence of one or multiple trichomonads constitutes a positive result. This method is cheap but it has a low sensitivity (60-70%) often due to an inadequate sample, resulting in false negatives. [5] [6] (2) Culture, (InPouch TV culture test, BioMed Diagnostics, San Jose, CA) which has historically been the "gold standard" in infectious disease diagnosis. Trichomonas Vaginalis culture tests are relatively cheap however sensitivity is still somewhat low (70-89%)[7] (3) New, more sensitive tests including the nucleic acid amplification tests (NAATs). These new NAATs include the APTIMA Trichomonas assay (Gen-Probe Inc, San Diego, CA) and the AFFIRM VPIII (BD Diagnostics, Sparks, MD). [7] These tests are more costly than microscopy and culture, and are highly sensitive (80-90%). [8]

### **Treatment[edit]**

Usually an oral antibiotic called metronidazole (Flagyl) is given to treat trichomoniasis. Before taking this drug, it is very important to let your doctor know if there is any chance that you could be pregnant, because the drug could harm the baby.

Your partner should also be treated at the same time to prevent reinfection and further spread of the disease. In addition, persons being treated for trichomoniasis should avoid sex until they and their sex partners complete treatment and have no symptoms. It is important to take all of your antibiotics, even if you feel better.

Treatment for both pregnant and non-pregnant patients usually utilizes <u>metronidazole</u> (Flagyl), [15] but with caution especially in early stages of pregnancy [16]

**2000 mg by mouth once**. Sexual partners, even if asymptomatic, should be treated concurrently. [10]

For 95-97% of cases, infection is resolved after one dose of metronidazole. [13][17] Studies suggest that 4-5% of TV cases are resistant to metronidazole, which may account for some "repeat" cases. [18][19] Without treatment, trichomoniasis can persist for months to years in women, and is thought to typically "resolve itself" in men. [19]

### **Hookworm infection:**

http://www.pawnation.com/2012/07/12/15-diseases-you-can-catch-from-your-pet/5

http://www.cdc.gov/parasites/zoonotichookworm/

Zoonotic hookworms are hookworms that live in animals but can be transmitted to humans. Dogs and cats can become infected with several hookworm species, including *Ancylostoma brazilense*, *A. caninum*, *A. ceylanicum*, and *Uncinaria stenocephala*. The eggs of these parasites are shed in the feces of infected animals and can end up in the environment, contaminating the ground where the animal defecated. People become infected when the zoonotic hookworm larvae penetrate unprotected skin, especially when walking barefoot or sitting on contaminated soil or sand. This can result in a disease called cutaneous larva migrans (CLM), when the larvae migrate through the skin and cause inflammation.

http://www.nytimes.com/health/guides/disease/hookworm/overview.html?module=Search&mabReward=relbias%3Aw

 Hookworm is a condition caused by roundworms that affects the small intestine and lungs.

•	Parasites: Hookworm Vaccine Will Be Tried in Africa
•	An iPhone Jury-Rigged as a Microscope
•	<u>Parasites in Paradise</u>
• <u>Africa</u>	Hookworm Infection Linked to Anemia Among Pregnant Women in
•	The Worms Crawl In

- The Worms Crawl In
- <u>The Worm Turns</u>
- Health Risks Hiding in the Grass
- Attack of the Worms
- Gateses Give \$47 Million to Bolster Coordinated Assaults on Diseases
- Beyond Swollen Limbs, a Disease's Hidden Agony

### Reference from A.D.A.M.

### **Causes**

News & Features

- The disorder is caused by infestation with the roundworms:
  - Necator americanus
  - Ancylostoma duodenale

<ul> <li>Hookworm disease is common in the moist tropics and</li> </ul>
subtropics. It affects about 1 billion people worldwide. In developing nations, the disease leads to the death of many children by increasing their risk for infections that their bodies would normally fight off.
o 25% of the world's population has hookworms, and one
expert thinks that 50% of Americans have them. Hookworm infection
takes place by skin penetration, usually from walking with bare feet on
contaminated soil. The larvae enter the blood stream, from where they
are carried to the lungs.
o The larvae (immature form of the worm) get into the skin.
The larvae move to the lungs via the bloodstream and enter the airways
The worms are about 1/2 inch long.
o Once in the lungs, they burrow into the air spaces, migrate
upwards and are then swallowed. Once swallowed they pass into the
intestine and bury themselves in the intestinal wall, maturing over the
next four weeks to become egg-laying adults that suck blood from your
intestinal wall.
o This young man has a skin rash on his upper arm caused by
hookworm larvae.
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A bookwarm infaction is enused by one of two different
<ul> <li>A hookworm infection is caused by one of two different types of roundworms: Ancylostoma duodenale or Necator americanus. If</li> </ul>
not treated, Hookworm infections can lead to abdominal pain and iron
deficiency. Researchers estimate that about 20% of the world's

population is infected with hookworm. • Humans become infected with hookworm when they come into contact with contaminated soil or stool. The larvae enter through the skin and travel through the blood to the lungs. Eventually, the larvae reach the throat, where they are coughed up and swallowed. As the larvae enter the digestive tract, they attach themselves to the wall of the small intestine. Here they mature into adult worms and mate. The worms feed on the blood of the host, which may lead to iron deficiency. Adult hookworms may live up to ten years.

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### **Symptoms:**

- When hookworms reach adulthood, they can sap the victim's strength, vitality and overall well-being.
- Hookworm infection: Most patients with hookworm infections do not experience any symptoms.
- Some patients may develop an itchy skin rash where the worm entered the body.
- When the worm enters the lung, some patients may develop symptoms similar to asthma or pneumonia, such as persistent cough, wheezing, or difficulty breathing.
- When the worm enters the intestine, patients may experience abdominal pain, diarrhea, weight loss, decreased appetite, and excessive gas.

### **Complications**

- Long-term hookworm infections may cause the patient to become anemic because the worms feed on the patient's blood. Symptoms of anemia may include difficulty breathing, pale complexion, fatigue, weakness, fast heartbeat, generalized swelling, or bloating. Once the parasite is killed, symptoms of anemia will resolve.
- The first symptoms are itchy patches on the skin with pimples and blisters and itching at the site of entry. Migration of the Ascaris larvae through the body can create temporary severe tissue irritation and allergic reactions such as asthma. Thereafter symptoms such as dizziness, pneumonitis, anorexia, weakness, abdominal pain, nausea, diarrhoea, anaemia and nutritional disorders.
  - Abdominal discomfort

- Blood in the stool
- Bloody sputum
- Cough
- Diarrhea
- Fatigue
- Fever
- Gas
- Itchy rash
- Loss of appetite
- Nausea, vomiting
- Pale skin
- Most people have no symptoms once the worms enter the intestines.

### **Exams and Tests**

- Tests that can help diagnose the infection include:
- Complete blood count (CBC) with differential
- Stool ova and parasites exam
- This disease may also affect the results of a D-xylose absorption test.

### **Treatment**

- o The goals of treatment are to:
- Cure the infection
- Treat complications of anemia
- Improve nutrition
- Parasite-killing medications such as albendazole,
   mebendazole, or pyrantel pamoate are usually prescribed. Ivermectin,
   used for other worm infections, does not work for hookworm infections.

 $_{\odot}$  Symptoms and complications of anemia are treated as they arise. The doctor will likely recommend increasing the amount of protein in your diet.

### **Outlook (Prognosis)**

• You will have a complete recovery if you get treated before serious complications develop. Treatment gets rid of the infection.

### **Possible Complications**

- o Iron deficiency anemia caused by loss of blood
- Nutritional deficiencies
- $\circ$  Severe protein loss with fluid buildup in the abdomen (ascites)

### When to Contact a Medical Professional

 Call for an appointment with your health care provider if symptoms of a parasite, abdomen extension, or hookworm infection develop.

### References

- Kazura JW. Nematode infections. In: Goldman L, Ausiello D, eds. Cecil Medicine . 23rd ed. Philadelphia, Pa: Saunders Elsevier; 2007:chap 378.
- Maguire JH. Intestinal nematodes (roundworms). In:
   Mandell GL, Bennett JE, Dolin R, eds. Principles and Practice of Infectious Disease. 7th ed. Philadelphia, Pa: Elsevier Churchill Livingstone; 2009:chap 287.

### **Herbal TREATMENT FOR HOOKWORMS**

 Wormseed can be taken as a tea. Do not use concentrated wormseed oil as it is too potent. torrya seed is used with basket fern and betel nut See more natural cures for parasites and worms below



http://www.plosntds.org/article/info%3Adoi%2F10.1371%2Fjournal.pntd.0000480

http://www.patient.co.uk/doctor/leishmaniasis

http://emedicine.medscape.com/article/220298-clinical

http://www.drmhijazy.com/english/chapters/chapter12.htm#118

# **Protozoa Leishmaniasis:**

### Synonyms:

Cutaneous leishmaniasis, visceral leishmaniasis, kala azar, Dum Dum fever

### **Vector**

- Leishmaniasis is one of the most important vector-borne diseases of humans, many species of Leishmania are zoonotic.
- Leishmania, is a protozoan parasite of the family Trypanosomatidae (order Kinetoplastida).

o I ne leisnma	aniases are a complex of diseases caused by at
least 17 species of	
the protozoan	
parasite	
Leishmania. The	
parasite exists in	
two forms: the	
flagellated	
promastigote in the	
female	
phlebotomine	
sandfly vector; and	
the amastigote in	
the mammalian	
host.	

The genus <u>Leishmania</u> contains two subgenera, <u>Leishmania</u> and <u>Viannia</u>, which are differentiated by where they multiply in the digestive tract of the insect vector.

### **Cutaneous leishmaniasis**

**Most** Leishmania species cause symptoms of **cutaneous leishmaniasis** in people.

Leishmaniasis is divided into four main clinical forms and is caused by parasitic protozoa of the genus *Leishmania*. There are over 20 species and subspecies that infect humans via the bite of sandflies (subfamily *phlebotominae*) – tiny sand-coloured blood-feeding flies that breed in forest areas, caves and burrows in tropical and subtropical regions. The clinical features of the disease depend on the causative species and can range from simple, self-healing skin sores as found in cutaneous leishmaniasis (due to infection with *Leishmania major*), to severe, life-threatening disease of untreated visceral leishmaniasis caused by *Leishmania donovani*.

# There are three basic forms in which the disease presents: cutaneous, mucocutaneous and visceral, and 21 species of the genus are known to cause disease in humans. Leishmania spp., lives as an obligate intracellular parasite within mammalian hosts. The primary hosts are vertebrates - commonly humans, rodents,

Leishmania infections start in the skin

canids and hyraxes.

**Ascaris Fluke Tapeworm Formula** 

	inoculate a median of 5,000 to 10,000 parasites; with a minor proportion of flies able to deliver much higher doses of up to 100,000 parasites [4].  More than 90 of the 1,000 or so sandfly species are known to transmit the disease.  Different species cause different clinical forms of the disease in various parts of the world.  Tissue macrophages are major target cells for parasite replication.  The sand fly vector is an active participant in the infection event in mammals, especially in proxcimity to inflamed skin that can rapidly kill invading parasites.  Sand flies regurgitate a proteophosphoglycan gel synthesized by the parasites inside the fly midgut, termed promastigote secretory gel (PSG). Regurgitated PSG can exacerbate cutaneous leishmaniasis.
Immune system	<ul> <li>Outcome of infection depends largely on the activation status of macrophages</li> <li>The balance between T helper (Th) 1 and Th2 cell responses is a major determinant of the outcome of experimental leishmaniasis, but polarized Th1 or Th2 responses are not sufficient to account for healing or nonhealing.</li> <li>Healing, induced by chemotherapy, resulted in control of</li> </ul>
	<ul> <li>arginase activity and reversal of local immunosuppression.</li> <li>L-arginine, impairs the capacity of T cells in the lesion to proliferate and to produce interferon-γ</li> <li>L-arginine plays a crucial role in the regulation of immune responses.</li> </ul>

Leishmania major infected sand flies were found to

site of pathology correlates with **L-arginine** deprivation **L-arginine** deprivation changes antigen-specific T cells and Mphi activation. Extracellular **Arginine** improves the level of the magnitude and the quality of their responses in major-specific CD4(+) T cells, immune system can be partially rescued by addition of exogenous L-arginine to produce IL-4 and IL-10, and immune system is dose deprndant. **L-arginine** is a crucial amino acid required for both nitric oxide (NO)-mediated parasite killing and polyamine-mediated parasite replication. **Prevalance/Locations** Leishmaniasis is a severe, widespread zoonotic disease which occurs reported on every continent in the world. Prevelant in the Middle and Far East, the Meditterranean basin, South America, and in some states in the United States of America. The World Health Organization (WHO) reports an estimated 300,000 new cases of visceral leishmaniasis (VL) with 20,000 to 30,000 deaths annually.[1] Primarily found in southern hemisphere and in the med, it is found in warm climates, including USA south/rural areas. Sandflies are blood-sucking insects that are commonly found on beaches and marshes. They are especially common in Florida, south central Texas. Leishmania is primarily a parasite of rodents, carnivores, marsupials, edentates, insectivores, and secondarily of dogs and humans. Globally in 1990 there were 12 million "known to be infected cases" in 88 countries with 400 thousand new cases every year. Furthermore, reporting is far from complete. In Brazil, the "estimate" is

**Uncontrolled replication of Leishmania** parasites at the

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approximately 3000 cases per year. Leishmania is largely distributed in tropical and subtropical areas, extending from Central America to the Mediterranean countries, Africa, Central Asia, India, and China.

### **Typical Symptoms**

 Leishmaniasis: Patients with leishmaniasis typically develop skin sores weeks to months after the parasite enters the body. The skin may become red, ulcerated, or have lesions, blisters, or pimples.
 Smaller lesions may be present around one larger ulcer.

- Some patients may develop a stuffy or runny nose, nosebleeds, difficulty breathing, difficulty swallowing, as well as ulcers and sores in the mouth, tongue, gums, lips, nose, and the wall that separates the nostrils (called the nasal septum).
- The parasite may also enter the bloodstream and burrow into internal organs. If internal organs are involved, symptoms may include persistent fever, night sweats, fatigue, weakness, appetite loss, weight loss, vomiting (most common in children), abdominal pain, scaly skin, gray or dark skin, and thinning hair.

 The spectrum of illness ranges from asymptomatic infection or self-resolving disease to fulminant, severe, life-threatening infection; many subclinical cases occur and go unrecognized for each clinically recognized case.

Leishmaniasis can be a disfiguring and potentially fatal

parasitic infection that affect some 350 million people worldwide [1] are at risk (*have it*) and leishmaniases belong to the category of most "neglected tropical diseases"

- Syndromes range from mild self-limiting local skin lesions to systemic fatal diseases.
- The first sign of disease appears about 2-4 months after the initial infection.
- Symptoms range from Sores on the skin, peeling, ulcers, loss of weight, bald patches, conjunctivitis, blindness, nasal discharge, muscular atrophy, inflammation, swelling, and organ failure, including mild heart attacks.

### Cutaneous leishmaniasis

Cutaneous leishmaniasis presents as Skin boils.
 Cutaneous leishmaniasis can be simple or diffuse (disseminated).

### Diffuse cutaneous leishmaniasis

<u> </u>	onnanasis			
	o Diffuse cutaneous disease deve	Diffuse cutaneous disease develops in an anergic host with		
	poor immune response. This condition			
	is associated with a deficient cell-			

mediated immunity that enables the parasite to disseminate in the subcutaneous tissues

- Different species, as well as host factors, can also affect the clinical picture, in which some species cause "wet" ulcers and others "dry" ulcers. The hallmark of cutaneous leishmaniasis is skin lesions, which can spontaneously heal in 2-10 months.
- The lesions are usually without pain or pruritus, although secondary bacterial infection may complicate the wound (see the following image). Healing may occur spontaneously over 2-12 months and is followed by scarring and changes in pigmentation.
- O Infection is characterized by a primary lesion, which slowly spreads to involve multiple areas of the skin (face, ears, extremities, buttocks) until the whole body is affected. Plaques, ulcers, and nodules may form over the entire body, resembling lepromatous leprosy taking years to evolve. However, no neurologic or systemic invasion is involved; as a result, although the lesions are neither destructive nor erosive, they are disfiguring. The infections are chronic and may recur after treatment. Although diffuse disease is more common with New World species in Central and South America, Old World  $\it L$   $\it aethiopica$  may progress to diffuse disease.

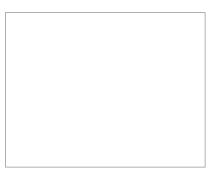
### Leishmaniasis recidivans:

o Presents as a recurrence of lesions at the site of apparently healed disease years after the original infection, typically on the face and often involving the cheek; manifests as an enlarging papule, plaque, or coalescence of papules that heals with central scarring (ie, lesions in the center or periphery of an old healed leishmaniasis scar); relentless expansion

at the periphery may cause significant facial destruction similar to the lupus vulgaris variant of cutaneous tuberculosis

- New World disease may progress to mucocutaneous leishmaniasis.
- Leishmaniases can present with a wide range of symptoms, ranging from the self healing cutaneous form, which produces skin ulcers; to the mucocutaneous form, which leads to the destruction of mucous membranes of the mouth, throat, nose and neighbouring tissue; to the visceral form, the most severe form of leishmaniasis, which leads to the distruction of immune system cells, in which the mortality rate can be as high as 100%.
- The cutaneous form presents with skin ulcers, and the mucocutaneous form with ulcers of the skin and also the mucous membranes of the mouth and nose.

**Mucocutaneous form** 



Mucocutaneous leishmaniasis (espundia) Infection by L (Viannia) braziliensis may lead to mucosal involvement in up to 10% of infections, depending on the region in which it was acquired. The incubation period is from 1 to 3 months. The initial infection is characterized by a persistent cutaneous lesion that eventually heals.

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- As many as 30% of patients report no prior evidence of leishmaniasis.
- o **Mucocutaneous leishmaniasis** tends to occur 1 to 5 years after cutaneous leishmaniasis caused by these organisms has healed, but it can also be seen while skin lesions are still present. The initial signs are erythema and ulcerations at the nares, followed by destructive inflammation that can spread to involve the nasal septum, and in some cases, the pharynx or larynx. Frequent nosebleeds can be an early sign. The inflammation may perforate the nasal septum, cause severe disfigurement of the face, or block the pharynx or larynx. In some cases, the genitalia may also be involved. Mucocutaneous leishmaniasis does not heal spontaneously.
- Ulcer progression is slow and steady. Several years later, oral and respiratory mucosal involvement occurs, causing inflammation and mutilation of the nose, mouth, oropharynx, and trachea (see the following image), resulting in symptoms of nasal obstruction and bleeding. These can become sites of infection, sometimes leading to sepsis. Cases in which the time between the primary lesion and the appearance of mucosal involvement is up to 2 decades have been reported.

#### **Visceral**

- O A variant of visceral leishmaniasis has been described in US soldiers who participated in the Gulf War. This is associated with light parasitic burden and mild symptoms including fever, malaise, and nausea.
  - http://www.gulflink.osd.mil/medical/med\_impact.htm
  - Sandfly fever visceral leishmaniasis due to *Leishmania tropica*.
  - Leishmania tropica, which causes cutaneous disease, would present with visceral infection without the classic severe symptoms and signs of kala-azar

Leishmania major in cutaneous cases in which parasites could be cultured and evaluated by isoenzyme analysis
 Predominantly fever, hepatosplenomegaly, and lymphadenopathy, but they did not have cutaneous manifestations
 Very mild anemia
 Modest aminotransferase
 All patients who had visceral leishmania

infection except one have had objective signs of disease

- Recently recognized infectious agents,

  Mycoplasma fermentans and M. penetrans are arthropod-borne viral diseases endemic in the Persian Gulf, such as sandfly fever, are not known to cause chronic infection and disease
- Thousands of cases of Leishmaniasis have been diagnosed in soldiers who have deployed to Iraq and/or Afghanistan.
  - Large percentage of leishmaniasis cases that have been diagnosed are of the cutaneous species L major. It is claimed that this species is of little concern as it only causes lesions on the skin and does not visceralize. Unfortunately this is not always true. AKA American troops in Iraq as "Baghdad boils")
  - Arvid unknowingly transmitted his Leishmaniasis to Janyce through personal and sexual contact, and Janyce, in turn, transmitted it to their children in utero. Arvid was diagnosed with the disease by a civilian doctor on October 1, 1998. His wife and children were diagnosed with the disease about two years later on October 4, 2000. his wife died. Janyce Brown developed a series of ailments and last year died at age 43 of a rare and inoperable form of liver cancer.
  - His head, muscles and bones ached, his strength was sapped; he was constantly exhausted but could not sleep. Through periods of disorientation, blackouts, extreme light sensitivity and almost unbearable pain. Chemotherapy put the disease into remission,
  - Some species of Leishmaniasis limit themselves to skin lesions and others migrate to the bones and organs
  - The more severe and deadly form, which Brown has, attacks blood cells and the body's internal organs. Like malaria, it is a chronic disease that can be controlled but not cured.
- North American sand flies carry another form of Leishmaniasis
  - Canine Leishmaniasis is a fatal zoonotic visceralizing disease usually associated with tropical areas

- In 1999, an outbreak of a canine leishmaniasis was reported in a Foxhound kennel in New York
- reported from dogs include L. mexicana, L. donovani, and
- o If visceral disease is left untreated, death frequently occurs within 2 years which may be due to hemorrhage (secondary to infiltration of the hematopoietic system), severe anemia, immunosuppression, and/or secondary infections.
- **Visceral disease**, <u>Visceral leishmaniasis</u>: A condition which is characterized by an infection of the viscera by leishmaniasis
  - The most devastating and fatal form of leishmaniasis is classically known as kala-azar or the Indian name for "black fever/disease," which is a reference to the characteristic darkening of the skin (hyperpigmentation) that is seen in patients with this condition.
  - The syndrome is characterized by the pentad of fever, weight loss, hepatosplenomegaly, pancytopenia, and hypergammaglobulinemia. The fever is continuous or remittent and becomes intermittent at a later stage. It is also characteristically described as a double rise in 24 hours, in which waves of pyrexia may be followed by a period without fever. Patients may also report night sweats, weakness, diarrhea, malaise, and anorexia. Melanocyte stimulation and xerosis can occur, causing characteristic skin\_hyperpigmentation.
  - Onset of visceral disease can be insidious or sudden. The incubation period varies after infection (usually 3-6 mo, but can be months or years) and may depend on the patient's age and immune status as well as the species of *Leishmania*.
  - The <u>visceral form</u> is more generalised, especially in the reticuloendothelial system (also called macrophage system or mononuclear phagocyte system, a class of cells that occur in widely separated parts and organs of the human body and that take up particular substances. These cells are part of the body's defense mechanisms.). Reticuloendothelial cells are derived from <u>precursor cells</u> in the <u>bone marrow</u>. These precursors develop into <u>monocytes</u>, phagocytic cells that are released into the bloodstream. Some monocytes remain in the general blood circulation, but most of them enter body tissues, where they develop into much larger phagocytic cells called macrophages. The great majority of macrophages remain as stationary cells within tissue, where they filter out and destroy foreign particles. Some of them break away, however, and wander through the circulation and within the intercellular spaces.
  - Disorders associated with the reticuloendothelial system include anemia caused by excessive destruction of red blood cells by reticulum cells. There are also malignant tumours related to reticuloendothelial cells that can be either localized or widespread throughout the body; reticulum-cell sarcoma is the most common such neoplasm and is usually located in the lymph nodes. Another condition, histiocytic medullary reticulosis, results from the diffuse proliferation of phagocytic cells. Niemann-Pick and Gauche's diseases are hereditary disorders characterized by abnormal products of lipid metabolism within the reticuloendothelial cells.

Tissue macrophages differ in appearance and name because of their various locations. For example, reticulum cells line the sinuses of the <u>lymph</u> nodes, spleen, and bone narrow, while

histiocytes are found in numerous subcutaneous tissues. Microglia occur in nervous tissue, alveolar macrophages in the air spaces of the lungs, and Kupffer cells in the liver.

#### **Visceral leishmaniasis Differential**

Visceral leishmaniasis may be confused with a variety of other infectious diseases or febrile systemic illnesses. In endemic areas, the diagnosis of visceral leishmaniasis is often made based on the history and physical examination.

Other conditions to consider in the differential diagnosis for visceral leishmaniasis include the following:

- Brucellosis
- Tropical splenomegaly syndrome
- Schistosomiasis
- African trypanosomiasis
- Sporotrichosis

#### **Description**

**Leishmaniasis** is a parasitic disease that is caused by protozoa called leishmania. Humans become infected with the parasite after they are bitten by a sandfly that is infected with the leishmania larvae. 0 The disease is caused by several species of protozoa in the genus Leishmania. Severe disease develops in the man and in the dog, which is characterized either by skin lesions or a general visceral involvement. This may be encountered in travellers and servicemen and servicewomen returning from the Middle East, especially the Persian Gulf. The protozoa is transmitted by a sandfly and has an average incubation period of 9 weeks. The key clinical finding is an erythematous papule 0 0

#### **Types**

• There are many different types of leishmaniasis.

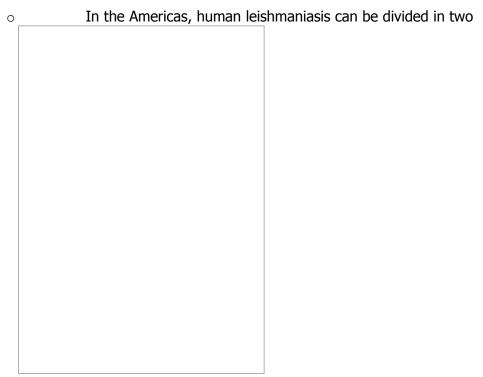
 The most common form is a skin disease called cutaneous leishmaniasis. This infection causes skin sores, which may or may not be painful. The sores have raised edges and a flat center.

**Visceral leishmaniasis** starts as a cutaneous lesion and the infection spreads systemically.

- Visceral forms of leishmania are caused by L.donovani and L.infantum/L.chagasi in the Old World and L.infantum/L.chagasi in the New World.
- Organs particularly affected are spleen, liver and bone marrow. VL is fatal if left untreated.
- Body temperature may fluctuate but is usually normal or subnormal. Immunosuppression may promote the occurrence of concomitant infections, hence the clinical picture may be complicated by demodicosis, pyoderma or pneumonia.

There is infection of liver, spleen and bone marrow, causing the classical features of:

- Night sweats, weakness and anorexia, which are typical.
- Fever.
- Weight loss.
- Hepatomegaly (can be marked).
- Splenomegaly (often enormous).
- Anaemia and pancytopenia (can lead to death from haemorrhage or infection).
- Hypergammaglobulinaemia.
- Dark pigmentation of the skin is uncommon but the name kala azar is Hindi for black fever.



broad categories: cutaneous and visceral leishmaniasis.

- Cutaneous leishmaniasis (self healing cutaneous form)
   has a large variety of forms which can be grouped in the following manner: cutaneous leishmaniasis, characterized by localized skin lesions which can heal spontaneously or become chronic lesions with disfiguring scars;
- Mucocutaneous leishmaniasis, characterized by ulcerative lesions and destruction of the mucosa; the mucocutaneous form, which leads to the destruction of mucous membranes of the mouth, throat, nose and neighbouring tissue;
- o **Diffuse cutaneous leishmaniasis**, characterized by nodular lesions that are not ulcerative ...and disseminated.
- The visceral form of the disease is chronic and progressive and affects various <u>organs</u>, including spleen, liver, bone marrow, lymph nodes, <u>and skin</u>.

http://www.emedicinehealth.com/drug-ketoconazole/article\_em.htm

http://www.selleckchem.com/products/Ketoconazole.html

http://www.ema.europa.eu/ema/index.jsp?

curl=pages/news and events/news/2013/07/news detail 001855.jsp&mid=WC0b01ac058004d5c

#### **Complications**

Leishmaniasis: Patients with leishmaniasis may develop allergic reactions when the larvae enter the muscle tissue. This typically happens when the dead or dying larvae release chemicals into the tissues. The body's immune system overreacts to these chemicals, and allergic symptoms, such as hives and itchy eyes, develop.

#### **Testing**

- O Diagnosis can be made by performing a punch biopsy and culturing tissue in a special medium.
- Molecular Methods (amplification/detection of Leishmania DNA)
- PCR uses primers from the rRNA gene to identify parasites from a variety of samples, including canine and human bone marrow, lymph nodes, skin biopsies, and heparinized whole blood. PCR is useful in the diagnosis of Leishmania, for follow-up patients pre and post

#### **Immunologic Diagnosis**

- This is the detection of antibodies (mainly IgG and especially IgG1) against Leishmania parasites or a specific cell-mediated response.
- The four main serological tests performed are IFAT, ELISA, DAT, and Western Blot. IFAT is considered the gold standard of tests, it has a high specificity and high sensitivity. IFAT uses the whole organism, which gives more repeatable and reliable results, rather than those using soluble Ag, such as complement fixation. ELISA is more sensitive, but less precise than IFAT. ELISA also cross reacts with Trypanosoma cruzi and Babesia.

#### **Treatment**

- Unfortunately, it now seems clear that the previous ambition to develop a single drug or drug formulation to be effective against all forms of leishmaniasis was too optimistic. Treatment of Leishmaniasis
- Most sores will heal spontaneously within one year .
- Treatment of cutaneous and muco cutaneous leishmaniasis is the same while the latter needs more intensive treatment due to the more severe and destructive complications.

disfiguring on the face, lower leg or over a joint; mucosa or cartilage, or sores that might be due to parasites of the L. braziliensis.
<ul> <li>Unfortunately some cases of leishmaniasis, may be seen treated by topical steroid preparation. This changes the clinical picture, deteriorates the lesion , which becomes later more chronic and decreases its response to the specific medications.</li> </ul>
<ul> <li>For adults, we give 6 cc of Pentostam I.M. daily for 10 days. This usually gives very good results, causing rapid healing of the ulcers. The dose is adjusted according to the age.</li> </ul>
<ul> <li>** El-Zawahry reported good results with dihydroemetine (Ciba) 2</li> <li>tablets daily for adult age for one month</li> </ul>
$^{\circ}$ ** Neostibosan (Bayer): is also an effective medication . The daily dose is 5mg./kg. body weight . A dose of 200-300 mg. can be given for older children and adults daily for 16 days is proved to be also effective .
Other medications such as Chloroquine , Fouadin and antibiotics such as Tetracycline have been found to be effective.
$^{\circ}$ ** Pentamidine isethionate can be used for Leishmania tropica in a dose of 4 mg/kg body weight once weekly for as long as necessary .
<ul> <li>Patients with diffuse cutaneous leishmaniasis require treatment for a longer time.</li> </ul>
<ul> <li>Leishmaniasis recidivans may respond to local infiltration, or systemic antimonies. antimonials, *must be given by injection and can cause damage to veins</li> </ul>
** Local infiltration with 1-2 ml sodium stibogluconate for solitary lesions. *twice-weekly injections of sodium stibogluconate, a commonly used compound for the skin disease, for six weeks. After 24 weeks, the leishmaniasis sores were still present.

\*\* Pentavalent antimony: used for sores that may cause scarring and

0		

- Cases occurring in other regions are due mainly to the migration of infected individuals to areas such as the United States, Canada, and many European and some Western Pacific countries (9). CD therapy is based on two nitroaromatic drugs, **nifurtimox** and **benznidazole** (Bz), that are recommended for all acute-stage, early-chronic-stage, and reactivated cases (2, 6). However, both drugs are far from ideal and give variable results, depending on the area where the disease is endemic; present considerable toxicity; are administered over 30 or more days; and are not very effective against the later chronic phase of CD or against naturally resistant strains (7, 21).
- Much effort has been put into the discovery of new drugs for the treatment of this pathology, but still the most widely used drugs remain the <u>pentavalent antimonials</u>, which were introduced 50 years ago.
- Treatment for extensive lesions is with high dosage ketoconazole for 1 month.
- Smaller lesions should be treated topically with 15% paromomycin and 12% methyl benzethonium chloride ointment applied bd for 10 days.9
- Few studies of phase II clinical trials mainly conducted in Kenya with another drug, sitamaquine or kalazaquine (WR 6026), an <u>8-aminoquinoline</u> has also shown promise as an orally effective agent (in a dose of 1 mg/kg/day for two weeks) for visceral leishmaniasis.

o **Arylimidamides** (AIAs) have shown outstanding *in vitro* potency against intracellular kinetoplastid parasites, and the AIA 2,5-bis[2-(2-propoxy)-4-(2-pyridylimino)aminophenyl]furan dihydrochloride (**DB766**) displayed good *in vivo* efficacy in rodent models of visceral leishmaniasis (VL) and Chagas' disease. In an attempt to further increase the solubility and *in vivo* antikinetoplastid potential of DB766, the mesylate salt of this compound and that of the closely related AIA 2,5-bis[2-(2-cyclopentyloxy)-4-(2-pyridylimino)aminophenyl]furan hydrochloride (**DB1852**) were prepared. These two mesylate salts, designated DB1960 and DB1955, respectively, exhibited dose-dependent activity in the murine model of VL, with DB1960 inhibiting liver parasitemia by 51% at an oral dose of 100 mg/kg/day × 5 and DB1955 reducing liver parasitemia by 57% when given by the same dosing regimen.

**Leishmania** resemble fungi in synthesizing 24-substituted sterols such as **ergosterol**, whereas mammals have just cholesterol.

Azoles, such as **ketoconazole**, inhibit 14a-demethylase, **a key enzyme** in this sterol biosynthesis pathway.

**Ketoconazole**, itraconazole and fluconazole have undergone several trials for CL and VL with **equivocal results**. In one controlled trial, ketoconazole was found to have some activity against L. mexicana, but not against L. braziliensis infections [25]. Some recent encouragement has been given by the oral activity of posoconazole in a Leishmania amazonensis experimental model [26]. Bisphosphonates, for example, risedronate and pamidronate, which are in widespread use in the treatment of bone disorders such as **osteoporosis**, have also shown activity against leishmaniasis in experimental models [27,28]. These studies followed the characterization of acidocalcisomes in trypanosomatids with high polyphosphate and pyrophosphate content, and the hypothesis that bisphosphonates could interfere with pyrophosphate metabolism, although it is now thought that the prime target might be **farnesyl pyrophosphate synthase** – a key enzyme in isoprenoid biosynthesis [29].

Other leads have come from plant products. <u>Licochalcone</u> A from the <u>Chinese liquorice plant Glycyrrhiza</u> has shown reasonable oral efficacy in experimental models <u>of VL and CL</u>; synthetic oxygenated derivatives are also active [30]. One derivative, 35 m4ac, resulted in 97% suppression of L. donovani liver amastigotes in a hamster model when given at **20 mg kg21 for six days intraperitoneally**. The

compounds appear to interfere with mitochondrial function. The 2-substituted quinoline alkaloids, from **the Bolivian plant Galipea longiflora**, have also shown oral activity in experimental <u>VL and CL</u> mouse models [31]. Saponins purified from the Vietnamese plant <u>Maesa balansae</u> and designated PX-6518 showed excellent activity after parenteral administration against <u>VL and CL</u> in rodent models [32]. However, the development of PX-6518 was halted as a result of unacceptable toxicity (L. Maes, 2003, PhD thesis, University of Antwerp, Belgium).

#### **Immunomodulation**

Cure of leishmaniasis, probably even during chemotherapy, appears to be dependent upon the development of an effective immune response that activates macrophages to produce toxic nitrogen and oxygen metabolites to kill the intracellular amastigotes [6,33,34]. This process is suppressed by the infection itself which downregulates the requisite signalling between macrophage and T cells, for example, the production of interleukin (IL)-12 or the presentation of major histocompatibility complex (MHC) and co-stimulatory molecules at the macrophage surface.

Studies in the 1980s showed that biological immunomodulators such as interferon (IFN)-g can provide a missing signal and enhance the activity of antimonials in the treatment of **VL and CL.** Recently, a new generation of

- Immunopotentiating drugs have shown potential for leishmaniasis treatment. The imidazoquinoline imiquimod, an ingredient of the topical cream for genital warts known as **Aladarae** cream (3M Pharmaceuticals; http://www.3m.com/), induces nitric oxide (NO) production in macrophages.
- <u>Imiquimod</u> cream was shown to have antileishmanial activity via macrophage activation in experimental models [35] and in clinical studies on CL in combination with antimonials [36]. This sensitivity of Leishmania amastigotes to NO was also exploited in a study using the NO generator nitroso-N-pencillamine (SNAP) topically on L. brazilienisis infections [37].

•

• In one approach to restore signalling, the substituted **benzaldehyde tucaresol**, which stimulates a signal to CD4p T cells and promotes T helper cell (Th) type 1 cytokine

production, showed activity in mouse VL models. A 5 mg/kg 21 oral dose for five days proved effective, resulting in a 60% reduction in the number of L. donovani liver amastigotes in mice. [38]. On a different pathway, anisomycin restores signalling via CD40 and activates p38 mitogen-activated protein (MAP) kinase thus killing parasites in mouse models [39]. These results indicate that immunomodulatory drugs show promise as an adjunct to chemotherapy.

• benzaldehydes" increases oxygen affinity ..

Tucaresol is an electron-rich benzaldehyde of moderate electrophilicity, which acts by forming an imine (a Schiff base) with an amino group of hemoglobin and

https://books.google.com/books?

id=YTeY9ZEfNccC&pg=PA138&lpg=PA138&dq=benzaldehyde+tucaresol&source=bl&ots=MYG 7NyeliS&sig=\_Onies0j\_59xBjubNG\_xhmDw6Ho&hl=en&sa=X&ei=pHajVMOdOliwyATol4CgAw &ved=0CDEQ6AEwCA#v=onepage&g=benzaldehyde%20tucaresol&f=false

• A special vaccine is available in some Middle Eastern countries (e.g. Israel).

#### Malaria:

Malaria is an infectious disease of the red blood cells that is caused by protozoan parasites called Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae, and Plasmodium ovale. The protozoa live the first part of their lives inside mosquitoes. When an infected mosquito bites a human, malaria can be transmitted. According to the U.S. Centers for Disease Control and Prevention (CDC), about 350-500 million patients become infected with malaria each year. • Malaria can be successfully treated with anti-malarial medications. However, drug resistance is a growing problem in many countries. • Studies on malaria causing parasites have revealed two new distinct forms different from the form already being studied in labs. Understanding the genome of the parasite and all the variations may help researchers discover new methods to combat the disease. • A mutation on the HLA-Bgene known as HLA-B53 has been shown to protect against malaria. Researchers believe that this mutation may help the immune system to respond better to the parasite that causes malaria. • A mutation in the glucose-6-phosphate dehydrogenase (G6PD) gene may also offer protection against malaria. The G6PD gene mutation causes glucose-6-phosphate dehydrogenase deficiency, which is a blood disorder that causes red blood cells to break down

prematurely. This blood disorder appears to make it difficult for the parasite to penetrate the blood cells. G6PD deficiency is most common in the world where malaria is also most common. • Another genetic mutation that protects against malaria is the gene mutation that causes sickle cell anemia, a blood disorder that causes red blood cells to be sickle shaped. As a result, the abnormally shaped blood cells may block narrow blood vessels potentially leading to tissue damage. Sickle cell anemia is caused by a mutation on the ? globin gene which leads to abnormal hemoglobin. There are two alleles, or variations, of the ? globin gene: A and S. ndividuals with two normal alleles (AA) have normal hemoglobin and normal RBCs. Individuals who have two mutated alleles (SS) produce abnormal hemoglobin and have sickle cell anemia, but individuals who only carry the allele (AS) produce both abnormal and normal hemoglobin.

#### **Sleeping sickness**

- Sleeping sickness is caused by two germs (protozoa),
   Trypanosoma brucei rhodesiense and Trypanosomoa brucei gambiense .
- "Sleeping sickness" is too benign a nickname for human African trypanosomiasis, which is caused by a protozoan spread by biting tsetse flies. When the parasites enter the brain, victims hallucinate wildly. They have been known to chase neighbors with machetes, throw themselves into latrines and scream with pain at the touch of water. Only at the end do they lapse into a lassitude so great that they cannot eat, followed by coma and death.

#### **Treatment**

- About 150,000 people contract the disease each year, but
   million people in 36 countries live in areas where they are at risk.
- The best treatment now is effornithine, sometimes called the resurrection drug because it can pull the dying out of comas.
- o It is almost a miracle that effornithine is available. It was discovered in 1980 at Pace University in New York. By early 2000, the last 7,500 doses in the world were running out. The patentholder, a precursor of the drug maker Sanofi-Aventis, abandoned it in 1995 because it had not lived up to its anticancer potential. Then, in late 2000, plans to make a topical form emerged. It was the key ingredient in Vaniqa, a cream to prevent facial hair in women.
- After critics accused Sanofi-Aventis of catering to vain rich women while letting poor Africans die, the company agreed to make an injectable form of the drug and now gives it free to the World Health Organization and Doctors Without Borders.
- Patients need intravenous infusions four times a day for two weeks.

- They still use the drug melarsoprol, which, Dr. Pécoul said,
   is not effective and sometimes kills."
- Melarsoprol, invented in the 1940s, is essentially arsenic dissolved in propylene glycol, the antifreeze ingredient. It can be given once a day for 10 days, which is easier on nurses. But it kills 5 percent of those who take it and burns survivors' veins.
- Dr. Pécoul hopes to have more countries switch to a mix of seven days of eflornithine twice a day — so that night nurses are not needed — plus seven days of nifurtimox, an oral drug that kills protozoa but is ineffective alone.
- Another drug, fexinidazole, is better. It can be taken orally, and in animal tests it cures even late-stage sleeping sickness in the brain within two weeks. But it has not been tested on humans. Phase 1 trials, small tests of its safety in humans, are to start late this year.
- Fexinidazole was developed by the German pharmaceutical company Hoechst, now part of Sanofi-Aventis, and abandoned in the 1980s when the company gave up its tropical disease programs, said Els Torreele, who directs the initiative's fexinidazole project. It is one of a class of drugs known as azoles, like fluconazole, that work against fungi and may work against cancer.
- o "We tested 500 different azoles," Dr. Pécoul said. The advantage of adopting an abandoned drug is that the former patentholder has usually done the chemical analyses, animal studies and, sometimes, early human trials, saving millions of dollars.
- The \$19 million from the Gates Foundation is not for that,
   but to begin the hunt for a completely new candidate.

#### The global distribution of clinical episodes of *Plasmodium falciparum*

Uncomplicated P. falciparum malaria

- artemether plus lumefantrine
- artesunate plus amodiaquine
- artesunate plus mefloquine
- artesunate plus sulfadoxine-pyrimethamine
- dihydroartemisinin plus piperaquine

The choice of ACT in a country or region will be based on the level of resistance to the constituents in the combination.[31] Artemisinin and its derivatives should not be used as monotherapy in uncomplicated falciparum malaria.[31] As second-line antimalarial treatment, when initial treatment does not work or stops working, an alternative ACT known to be effective in the region is recommended, such as:

- Artesunate plus tetracycline or doxycycline or clindamycin.[31]
- Quinine plus tetracycline or doxycycline or clindamycin[31]

Any of these combinations are to be given for 7 days.[31]

http://en.wikipedia.org/wiki/Plasmodium\_falciparum

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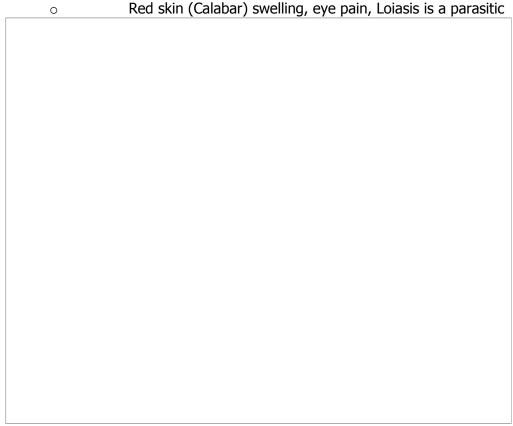
# <u>Filarioidea</u>

0	Mansonella species – nonperiodic anytime		
	■ Mansonella perstans		
	■ Mansonella streptocerca		
	■ Mansonella ozzardi		
	■ Dipetalonema perstans - africa		
	■ Dipetalonema streptocerca		
0	Loa loa- 1300 hours diurnal		
0	Onchocerca volvulus.		
0	Brugia timori- 2400 hours peak - nocturnal		
0	Brugia malayi- 2100 hours peak- Nocturnal subperiodic		
0	Wuchereria bancrofti – 2400 hours peak - nocturnal		

# **Ascaris Fluke Tapeworm Formula** Loiasis: Loiasis: 0 <u>Loaisis</u> Family Filarioidea When filariae that normally infect animals infect humans, the condition is termed zoonotic filariasis. Filarioidea - Bloodsucking arthropod vectors such as black flies and mosquitoes - bloodmeal 0 Reported worldwide Variable clinical presentations Asymptomatic state to a serious illness with widespread dissemination

Dirofilaria, Onchocerca, or Brugia genera.

Zoonotic filariasis of the skin is most commonly caused by filariae of the



infection that is caused by a roundworm called the African eye worm (Loa loa). Humans become infected with the parasite after they are bitten by the deer fly chrysops (typically found near the Congo River region, Sudan, and Ethiopia) that is carrying the immature African eye worm. Once the parasite enters the human host, it migrates toward the eyes, where it causes eye congestion and irritation. Sometimes the worms move to the brain, where it causes brain swelling, which is potentially fatal. • Loiasis is most prevalent in tropical areas of Africa.

#### **Prevalance**

Any patient in whom therapy for lymphatic filariasis or onchocerciasis is anticipated who comes from a region of Africa coendemic for loiasis should be screened for infection prior to initiating treatment because of the risk of fatal encephalopathy after treatment with diethylcarbamazine (DEC) or ivermectin.

#### **Symptoms**

Loiasis: Symptoms include irritated and watery itchy eyes,
 blurred vision, and eye discharge (called eye congestion). Patients may be able to see the thread-like worms move across their own eyeballs.

#### **Complications**

 Loaisis: If left untreated, the Loa loa worm may sometimes enter the brain, causing brain swelling (called encephalitis) and possibly brain damage.

http://jcm.asm.org/content/early/2014/01/30/JCM.03358-13.full.pdf

#### **Onchocerca:**

Family Filarioidea. - The Blood Nematodes Hypertension and hypercholesterolemia Chronic papular onchodermatitis – larger papules, resulting in hyperpigmentation 0 Inflammatory skin lesion caused by a zoonotic Onchocerca species - In humans, zoonotic nematodes have typically been found in the subcutaneous tissue, heart, lungs, eyes, lymphatic system, brain, and spinal cord (2). Skin swelling on his upper back Small, mildly tender pruritic papule one morning measuring less than 1 cm - resemble insect bites Travel to Florida onchocerciasis, also known as 'river blindness,' manifests as intense pruritis, skin depigmentation and ocular scarring due to migration of the microfilariae (3). The adult filariae are stationary in the human host and are found within a subcutaneous nodule. Clinically interpreted as a "cyst", the submitted sample consisted of an ellipse of skin and subcutaneous adipose tissue Cross sections of a nematode - 3 micrometer diameter = 0.1 mil Differential - organisms were excluded, 78 including: Strongyloides stercoralis, hookworm, Toxocara canis or T. cati, Gnathostoma spp., and Baylisascaris procyonis. Candidate organisms included Brugia spp., Mansonella spp., and Acanthocheilonema delicata. Mayo Clinic in Rochester, MN. There, a molecular 84 approach to identification of the organisms was undertaken. 174 base pair seq.

#### **SKIN**

- O Lesion measuring 1.4 x 0.3 x 1.3 cm.
- Molecular techniques
- Lesions become itchy, mostly around the head, neck, chest, shoulders, and underside of the belly. Onchocerca is what's known as a parasitic filarial worm (nematode). One reason these worms get relatively little attention is that they never live in the intestines. Aka: Sweet Itch, Summer Itch, certainly

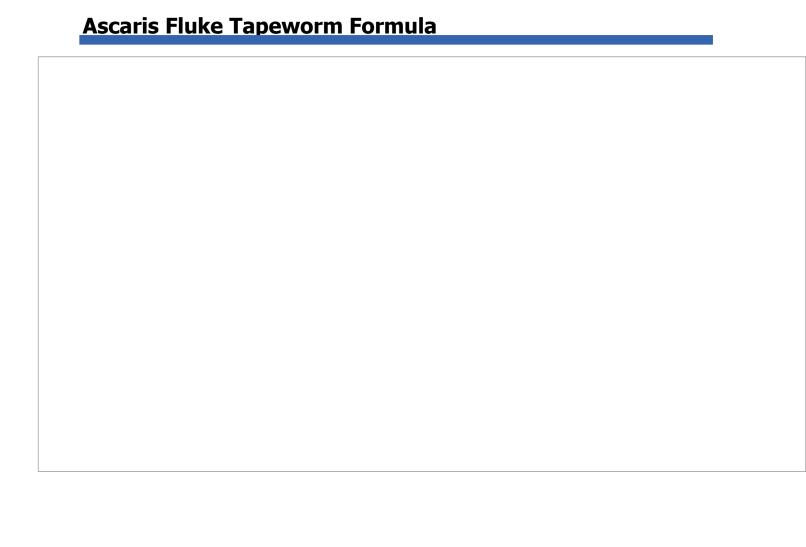
isn't to try and say that *all* itch cases are due to neck threadworms. Threadworms have a distinctive life cycle, but as is so often the case, the problem presents in different ways, depending on the individual. Weeping spots, and a scaly crest to an area. Ivermectin-based wormer is the quickest way to tell if you have them. If the microfilariae are present, they usually respond with intense itching. The adults live for 10-12 years. Better news it that the worms are so fine and the lumps so small. Apparently they intertwine and live in small clumps. Some say that an **ivermectin and praziquantel wormer** is more effective. research has shown that **moxidectin-based wormers** are equally as effective in addressing the microfilariae (but don't double-dose with this one – only with ivermectin). a single dose every 6-8 weeks. Research I've read shows onchecerca microfilariae in Canada, USA, Australia, France, Poland

The life cycle is similar to *W. bancrofti*, except that the intermediate hosts are various species from the genus *Simulium* (Black flies), the most important species is *Simulium damnosum*.

The microfilariae are ingested by a Black fly during a blood meal, from where they are carried to the midgut where they penetrate the epithelium and migrate, via the hemocoele, to the indirect flight muscles. Here they undergo two molts,  $L_1-L_3$  and develop into infective  $L_3$  larvae which move to the mouth parts. Development is completed in 6–9 days.

When the infected fly takes another blood meal the infective larvae are once again transmitted into another host (definitive host). The microfilariae are released from the mouth parts and transmitted directly into the hosts bloodstream. Molting takes place form  $\rm L_3$  -  $\rm L_4$  within 2-5 days and the larvae then migrate widely through the body under the skin and between muscles, ligaments and tendons. The final molt to  $\rm L_5$  occurs at 1.5–2.5 months after transmission. Male worms are known to mature in about four months later. Female worms initiate the formation of the nodules and the males may join later. The sexually mature female worms release microfilariae which migrate out from the nodules into the skin and other tissues, most significantly into the eye.

#### Morphology



#### **Clinical Disease**

Clinical manifestations are due to microfilariae in the epidermis.

Light infections may be asymptomatic or cause pruritis. This leads to scratching which can result in infection. Lyphadenopathy may also be a feature of early infection. After months or years, onchodermatitis results in secondary stage of thickening due to intradermal edema and pachydermis. There is a loss of elastic fibers resulting in hanging groin, hernias and elephantiasis of the scrotum. There is finally atrophy of the skin resulting in loss of elasticity. There is mottled depigmentation of the skin.

Ocular lesions are related to the intensity of the microfilariae in the skin. Ocular lesions include sclerosing keratitis, secondary glaucoma and cataract, coroidoretinitis and fluffy corneal opacities. The major complication of onchocerciasis is the development of lesions in the eye which may result in blindness or other distressing ocular diseases.

#### 2. Analysis of Biopsies

Biopsies of tissue nodules can be dabbed on to a slide to produce impression smears and then stained with Giemsa stain at pH 6.8 for the presence of microfilariae.

Recent advances in diagnostic methods includes and ELISA-based antibody detection assay which utilizes a cocktail of recombinant antigens. The advantages of using this test is that it is highly sensitive (almost 100% in onchocerciasis foci). It is also highly specific (100%), it also uses finger prick blood. Therefore, reducing the painful procedure of gaining a skin snip.

The disadvantages is that it requires advanced ELISA apparatus and reagents and cannot distinguish between past and present infections due to it detecting antibodies which stay present in the body for a long time after the infection. Another modern detection method is for Parasite DNA detection, which is based on the amplification of specific DNA sequences form microfilariae using molecular biology technology. The advantages of this technique is its exquisite sensitivity and detects active infections only. The disadvantages are that it requires specialized equipment and expensive reagents. Also it still requires a skin snip but a urine assay is a possibility for the future.



NAME: Onchocerca volvulus.

**SYNONYM OR CROSS REFERENCE**: Onchocerciasis, river blindness, and craw-craw Footnote 1-Footnote 3, Footnote 3-Footnote 12.

**CHARACTERISTICS**: *O. volvulus* is a filarial nematode with a five-stage life cycle: 4 larval stages (microfilariae), and an adult stage (macrofilariae) Footnote 9, Footnote 11. Females are 30 cm to 80 cm in length, whereas males are only 3 cm to 5 cm long appear to migrate from nodule to nodule to inseminate the females Footnote 2,

Footnote 11. Female macrofilariae can remain sessile and live coiled-up in subcutaneous tissue (forming a "nodule" or "onchocercoma") for up to 15 years, producing more than 700-1,500 microfilariae per day  $\frac{\text{Footnote }13}{\text{Footnote }5}$ . Microfilariae measure 220 to 360 µm in length and can survive in humans for 2 to 3 years  $\frac{\text{Footnote }5}{\text{Footnote }5}$ 

PATHOGENICITY/TOXICITY: Onchocerciasis is a chronic systemic illness associated with extensive and disfiguring skin changes, musculoskeletal complaints, weight loss, and changes in the immune system Footnote 3. The principal organ affected in onchocerciasis is the skin; however, infection with O. volvulus can lead to severe visual impairment and blindness Footnote 5, Footnote 7, Footnote 11. In Sub-Saharan Africa, onchocerciasis is the second most common cause of preventable blindness, and has caused visual impairments in 500,000, and blindness in 270,000 persons Footnote 3, Footnote 4. The pathogenesis of onchocerciasis is believed to be largely due to an immune reaction to dying or dead microfilariae that have localised in the skin and eyes Footnote 2, Footnote 3. In heavily infected persons, 100,000 or more microfilariae can die every day Footnote 3. There are 5 main categories of onchocercal skin disease: acute papular onchodermatitis, chronic papular onchodermatitis, lichenified onchodermatitis, atrophy, and depigmentation Footnote 7.

Acute papular onchodermatitis: Primarily affects the face, small pruritic papules may be scattered on limbs, trunk, shoulders and extremities. Lesions may progress to vesicles and pustules Footnote 7.

Chronic papular onchodermatitis: Often affects the shoulders, buttocks and extremities and consists of a severely itchy maculopapular rash containing scattered flat-topped papules and hyperpigmented macules Footnote 7

Lichenified onchodermatitis: Consists of hyperkeratotic and hyperpigmented confluent plaques most often affecting the lower extremities and associated with lymphadenopathy Footnote 7.

Atrophy: Typically affecting the buttocks and lower back, and consisting of large atrophic plaques with finely wrinkled inelastic skin resembling cigarette paper  $\frac{\text{Footnote 7}}{2}$ .

Depigmentation: Often referred to as leopard skin and consists of vitiligo-like lesions with hypopigmented patches containing perifollicular spots of normally pigmented skin. Onchocercal depigmentation often affects the shins and is rarely associated with itch and excoriations Footnote 7

Other clinical pictures include; "lizard skin" with dry ichthyoses-like lesions with a mosaic pattern resembling the scales of a lizard, and "hanging groin" with folds of atrophic inelastic skin in the inguinal region associated with lymphadenopathy Footnote 7. The different clinical

patterns are not mutually exclusive and may be present simultaneously or one pattern may evolve into another.

Onchocercal ocular disease: The disease ranges from mild symptoms such as itching, redness, pain, photophobia, diffuse keratitis, and blurred vision, to more severe symptoms such as corneal scarring, night blindness, intraocular inflammation, glaucoma, visual field loss, and, eventually, blindness Footnote 7. Ocular lesions are usually bilateral and can affect various structures of the anterior and posterior segments of the eye resulting in uveitis, iridocyclitis, conjunctivitis, chorioretinitis, cataracts and glaucoma Footnote 5, Footnote 11. Increasing corneal opacity is not caused by O. volvulus but is believed to be from a host inflammatory response to its endosymbiotic Wolbachia bacteria, which are released by dying microfilariae Footnote 14.

EPIDEMIOLOGY: O. volvulus infection is estimated to affect more than 17 million people worldwide in 34 countries in Africa, the Middle East, South America and Central America Footnote 2, Footnote 3, Footnote 5, Footnote 9. Countries with the highest prevalence of onchocerciasis include 11 sub-Saharan West African nations, including Ghana, Nigeria, Liberia, and parts of Mali Footnote 1, Footnote 9. The endemicity also extends latitudinally across the entire continent of Africa and into Southwest Asia, with foci in Yemen and Oman in the Arabian Peninsula Footnote 5, Footnote 9. Small foci also exist in Ecuador, Venezuela, Columbia, southern Mexico, and Guatemala. In West Africa, there are 3 major strains of O. volvulus: a forest strain with low ocular pathogenicity associated with high nodule numbers and severe skin disease, a dry savannah strain with high ocular pathogenesis and an associated high rate of blindness, and a humid savannah strain with an intermediate pattern Footnote 2, Footnote 3.

HOST RANGE: Humans are the sole definitive host; however, there is evidence of infection in gorillas Footnote 1, Footnote 2, Footnote 9.

INFECTIOUS DOSE: Unknown.

MODE OF TRANSMISSION: O. volvulus is spread by blackflies (genus Simulium), which breed in fast-flowing rivers Footnote 2, Footnote 8, Footnote 9, Footnote 15. Infection occurs during the bloodmeal of a blackfly, which results in the dissemination of an O. volvulus larva (at stage 3 of life cycle) into the host Footnote 9. Female worms stay permanently in a fibrous capsule, while males are able to move freely in skin tissues and subcutaneous regions.

INCUBATION PERIOD: Highly variable, but normally symptoms are developed within 1 to 2 years of infection Footnote 1-Footnote 5.

COMMUNICABILITY: No evidence for direct human-to-human transmission Footnote 1; however, infection can be spread indirectly between humans via blackfly bites Footnote 2, Footnote 3, Footnote 5, Footnote 9. People can infect flies as long as living microfilariae occur in the skin.

#### SECTION III - DISSEMINATION

RESERVOIR: Humans Footnote 1, Footnote 9.

ZOONOSIS: No, the Onchocerca species found in animals cannot infect humans Footnote 1

VECTORS: Blackfly (Simulium spp.) Footnote 1-Footnote 6, Footnote 8, Footnote 8, Footnote 11, Footnote 15. In Africa, the Simulium damnosus sensu lato (s.l.) species complex is the main vector and is responsible for more than 95% of onchocerciasis cases worldwide Footnote 3, Footnote 6. In Uganda, Tanzania, Ethiopia, and the Congo, the vectors are the S. neavei s.l. complex, and S. albivirgulatum s.l. is the vector that is only found in the Congo Basin Footnote 3. In Latin America, S. ochraceum s.l., S. exiguum s.l., S. metallicum s.l., and S. guianense s.l. are the main vectors Footnote 3, Footnote 6

#### SECTION IV - STABILITY AND VIABILITY

DRUG SUSCEPTIBILITY: Microfilariae are susceptible to ivermectin, diethylcarbamazine, and albendazole, although diethylcarbamazine is no longer used due to adverse side effects Footnote 1-Footnote 4, Footnote 6, Footnote 8, Footnote 9. Adult worms are susceptible to suramin, which is no longer used due to extreme toxicity, in favour of a new drug, moxidectin, which is safe for use in humans and has already undergone phase II trials Footnote 2, Footnote 9. Symbiotic Wolbachia spp. present within O. volvulus are sensitive to doxycycline treatment Footnote 15. Although the use of ivermectin against infection has proven to be effective, constant and regular treatment is necessary as studies have found live and possibly fertile worms present in patients even after 6 years of treatment Footnote 16.

SUSCEPTIBILITY TO DISINFECTANTS: Unknown. In a study testing the efficacy of Rifampin and Azithromycin on O.volvulus, the instruments used to obtain skin biopsies from patients were disinfected with a combination of disinfectants, which included the sequential use of full-strength bleach (30 seconds), 95% ethanol (30 seconds), distilled water (30 seconds), 95% ethanol (30 seconds), and then the instruments were air dried Footnote 17.

PHYSICAL INACTIVATION: Unknown; however, air drying, in combination with several common disinfectants, has been used clinically to disinfect instruments that have come into contact with O.volvulus Footnote 17.

SURVIVAL OUTSIDE HOST: Unknown.

#### SECTION V - FIRST AID / MEDICAL

SURVEILLANCE: Onchocerciasis should be considered in persons from endemic areas or expatriate visitors who present with itching with or without a rash Footnote 3. Onchocerciasis can be diagnosed based on the observation of living microfilaria in skin biopsies Footnote 2, Footnote 3, Footnote 5, Footnote 9, Footnote 10. Bloodless "skin snips" can be obtained from the shins, buttocks, and the iliac crests, and then placed in saline and examined microscopically for the presence of microfilariae Footnote 2, Footnote 3, Footnote 5, Footnote 9, Footnote 10. Microscopic demonstration of microfilaria may not be sensitive, and, therefore, methods such as PCR, ELISA, and immunolabelling are used to detect microfilaria in skin snips with greater sensitivity Footnote 1-Footnote 3, Footnote 9, Footnote 10. These methods still utilize skin snips which can be painful and increase the risk of blood borne contaminations, furthermore non-invasive tests, such as the dipstick assay, may prove to be more useful for the diagnosis of

onchocerciasis Footnote 2, Footnote 10. Microfilariae can also be detected in the anterior chamber of the eye with a slit lamp Footnote 3, Footnote 5. Adult worms may be found in excised nodules.

FIRST AID/TREATMENT: The drug of choice for the treatment of onchocerciasis is ivermectin (Stromectol, Mectizan) Footnote 1-Footnote 3, Footnote 5, Footnote 6, Footnote 8, Footnote 9, Footnote 12, Footnote 15. The drug is administered once per year and for the expected lifespan of the parasite (often more than 10 years) Footnote 2, Footnote 3, Footnote 5, Footnote 6. Co-treatment with ivermectin and doxycycline (for 6 weeks) has been shown to be more effective, since doxycycline will interrupt the embryogenesis of O. volvulus for a few months by depleting symbiotic Wolbachia endobacteria Footnote 15. Another treatment, based on removal of nodules (nodulectomy, particularly from the head) is available in Mexico and Guatemala and is thought to reduce the number of microfilariae that can enter the eye and hence limit the number of cases of blindness Footnote 2, Footnote 3.

IMMUNIZATION: None, although several strategies are currently being employed to develop a vaccine Footnote 2, Footnote 12.

PROPHYLAXIS: Although no chemoprophylaxis exists, measures such as insect nets and protective clothing, insecticide, and larvicides are used in endemic regions Footnote 1-Footnote 3, Footnote 6, Footnote 8, Footnote 10. Onchocerciasis Elimination Program for the Americas – ivermectin q6m to prevent disease and interrupt transmission.

#### 1Chemotherapy

Ivermectin (dihydroavermectin B1) is the drug of choice for the treatment of onchocerciasis. In 1996, ivermectin (Stromectol) was registered for human use in the United States; it was registered in France as Mectizan. (For animals in the United States, it was registered previously by Merck Sharpe & Dohme as Heartgard, a widely used veterinary gastrointestinal anthelmintic and ectoparasiticide.)

Ivermectin is a macrocyclic lactone derived from the actinomycete *Streptomyces avermitilis* found in a soil sample from a Japanese golf course and is produced by fermentation. [13, 14] It has structural similarities to the macrolide antibiotics but lacks antibacterial activity. Ivermectin enters the worm by the transcuticular route.

It functions as a single dose and is a rapidly effective microfilaricide for *O volvulus*; however, attempts to give a single dose of ivermectin (150 mcg/kg) on the preadult stages of *O volvulus* did not have any prophylactic effect.[15]

Unlike diethylcarbamazine (DEC), ivermectin does not produce a significant Mazzotti reaction in onchocerciasis,[16] most likely because it acts by paralyzing the microfilariae in the skin tissue spaces and lymphatics. They are then swept away into the local lymph nodes, which may swell up and only cause some local limb edema. On the other hand, DEC "unmasks" the microfilariae in the tissue spaces and the exposed *Wohlbachia* organisms within them. Ivermectin elicits a dose-dependent eosinophil sequestration, activation, and degranulation, but the duration apparently is very limited and resolution is quick.[17]-

Ivermectin kills microfilariae within female worms, as well as those in human tissues. Dead microfilariae within the uteri of the female worm degenerate and prevent further microfilarial production for 6-12 months. Ivermectin not only kills microfilariae, it is also a microfilarial suppressant. Two to 3 months after ivermectin therapy, microfilariae usually disappear

The addition of oral doxycycline (100 mg/d) given for 6 weeks from the start of ivermectin to kill off *Wolbachia* organisms helps to prevent treatment reactions.[29]-

Caution is needed in areas endemic for both loiasis and onchocerciasis because ivermectin can initiate serious adverse effects in patients with high  $L \log 1$  microfilarial loads. [27, 28]

Studies have demonstrated that after many rounds of ivermectin treatment, some patients continue to have surprisingly high microfilarial counts. This suboptimal response to ivermectin treatment may indicate a developing drug resistance.[30]

Because increasing numbers of parasite strains of importance are showing ivermectin resistance, a new interest has emerged in developing alternative drugs. The search for novel filaricides and **alternative medicines** includes a renewed interest in old herbicidal extracts. Extracts of *Margaritaria discoidea* (Bushveld Peacock-berry) and *Homalium africanum* (also called the elephant's legs tree) have demonstrated efficacy on *Onchocerca* and further testing is indicated. [31, 32]

**DEC** is a microfilaricide with no effect on the adult worm. It produces Mazzotti reactions that become severe in heavily infected persons. Eosinophilic degranulation is correlated strongly with the Mazzotti reaction. [33] A low dose of dexamethasone (3 mg/d), begun after onset of the Mazzotti reaction, modifies the progression of the Mazzotti reaction without interfering with the macrofilaricidal efficacy of DEC. [34] This suggests that the Mazzotti reaction is not directly caused by death of microfilariae, but rather is a direct effect of DEC on the immune system.

*Wolbachia* organisms appear to play a critical role in the biology and metabolism of filarial worms. The use of tetracycline to kill the *Wolbachia* organisms appears to be lethal to adult *O ochengi,* and recent evidence suggests it also is effective for *O volvulus* and perhaps other filarial worms. [38]

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#### **Diagnosis**

o The standard diagnostic test for the diagnosis of **loiasis** is demonstration of microfilariae on a daytime (10AM to 2PM) Giemsastained thin or thick blood smear. It is important to note that the timing of the blood smear should be adjusted to reflect local time at the point of origin of a traveler who is still experiencing jetlag. Concentration techniques such as Nuclepore™ filtration (Whatman Inc., Florham Park, NJ), Knott's concentration, or saponin lysis may increase the diagnostic

yield in persons with low numbers of circulating microfilariae. Identification of microfilariae on blood smear is sufficient for diagnosis of the infection. Quantification of the number of microfilariae per mL is needed to direct treatment.

#### **Differential**

 There is a general screen for any filarial infection (including Wuchereria, Brugia, Onchocerca, and Mansonella infections) that is available in some specialty diagnostic labs.

#### **Testing**

- Because the test is highly sensitive, it is useful in 0 determining if an individual has had filarial infection, but it is not specific enough to identify which filarial infection. As with any antibody test, the results indicate only that the patient has been exposed to the disease, but they do not indicate if the patient has an active infection. This distinction is less important in symptomatic travelers, but it limits the usefulness of the test in persons from endemic areas. One advantage of the test is that it can pick up evidence of infection in the pre-patent stage of infection. There are several *Loa*-specific serologic tests in existence, such as the tests for antibodies to the LISXP-1 recombinant antigen which can be used in both an ELISA and a luciferase immunoprecipitation systems (LIPS) assay, but these are currently available only in the research setting and are not approved for diagnosis in the United States. There is one polymerase chain reaction (PCR) test for loiasis approved for diagnosis in the United States.
- o In general the diagnosis of *L. loa* infection should be made with blood smear. However, when blood smears are negative and clinical suspicion of infection is high, the general antibody test could be used in an attempt to exclude infection. If the general antibody test were positive, then it might be necessary to consider seeking additional diagnostic information by enlisting the assistance of researchers who perform specific antibody and/or PCR tests.

#### treatment

Loa loa do not contain Wolbachia so doxycycline is not an effective treatment.

Treatment	Indication	Adult Dose	Pediatric Dose
Diethylcarbamazine (DEC)	Symptomatic loiasis with MF/mL <8,000	5. 5 ,	8–10 mg/kg orally in 3 divided doses daily for 21 days
Albendazole	Symptomatic loiasis, with MF/mL <8,000 and failed 2 rounds DEC OR Symptomatic loiasis, with MF/ml	200 mg orally twice daily for 21 days	200 mg orally twice daily for 21 days

Treatment	Indication	Adult Dose	Pediatric Dose
	≥8,000 to reduce level to <8,000 prior to treatment with DEC		
Apheresis* followed by DEC	Symptomatic loiasis, with MF/mL ≥8,000	N/A	N/A

MF = microfilariae of L. loa

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#### **Filariasis:**

- o A <u>filarial infection</u> (called filariasis) is a type of infection that is caused by any of **the round, thread-like parasitic nematode species worms**. Many develop **lymphodema. Symptoms start with** fever, Tropical pulmonary eosinophilia (TPE), digestion changes, GERD, dirreah, itches, and puritis. Later stages include •Testicular and/or inguinal pain, Limb or genital swelling Repeated episodes of inflammation and lymphedema lead to lymphatic damage, chronic swelling, and elephantiasis of the legs, arms, scrotum, vulva, and breasts.
- o Filarial infections (**microfilaria hatchlings**) can also be caused by common round worms, if sufficiently infested. Once the parasite enters the human, it migrates to the GI trac and lymph system. Females release larvae, which circulate in the patient's bloodstream. **Ascaris** adult worms typically live for two years, if not treated with antiparasitic medications. Rarely fatal, it can cause fevers, night sweats, frequent infections, and serious inflammation of the lymph system if it is not treated. **Cardio pulmonary** involvement is often felt, even if not diagnosed or noticed.
- Lymphatic filariasis Wuchereria bancrofti Mosquitoes carry this parasite, which they release into the bloodstream of a human host as they feed. Lymphatic filariasis, also called elephantiasis (even though few develop elephantiasis, many develop lymphodema), is caused by a nematode worm including severe cases of Wuchereria bancrofti or Brugia malayi. The infection is transmitted to humans when a mosquito infected with the larvae bites a person. In the lymph nodes it develops into an adult. Adult filarial worms typically live for about seven years. Over a period of years it can destroy the lymph system. The larvae move to the lymph nodes, which are predominantly in the legs and genital area and develop into an adult worm over the course of a

<sup>\*</sup> Apharesis should be performed at an institution with experience in using this therapeutic modality for loiasis.

year. They are commonly responsible for the tropical disease filariasis, but in extreme cases can cause elephantiasis.

#### **Symptoms:**

#### Filiariasis

 Fever, chills, skin infections, painful lymph nodes, thickened skin, swelling

#### bymphatic filariasis:

Symptoms of lymphatic filariasis generally develop 5-18 months after being bitten by an infected mosquito. Lymphatic filariasis causes tissue damage that limits the normal flow of lymph fluid through the body. As a result, patients typically experience swelling, scarring, and infections, especially of the legs and groin.

#### **Onchocerciasis**

 The so called "River blindness" (Onchocerciasis) has one in 3 varieties that cause eye infection and <u>blindness in untreated</u> cases:

#### **Symptoms**

- River blindness (onchocerciasis): Symptoms usually develop one to three years after the larvae enter the body. Patients may develop an itchy skin rash, skin lesions, loss of skin pigmentation (which causes the skin to become white), enlarged lymph nodes, visual impairment, and sometimes blindness.
- The most often displayed symptom is <u>Dermatitis</u> Skin lesions include edema, pruritus, erythema, papules, scablike eruptions, altered pigmentation, and lichenification.
- Itchy skin nodules, <u>de/pigmentations</u>, enlarged lymph nodes,
- River blindness, also called onchocerciasis, is a parasitic infection of the eyes that is caused by a worm called Onchocerca volvulu.
- Skin nodules (ie, onchocercomas) Skin <u>nodules</u> or large bumps, tend to be common over bony prominences,

#### **Vectors**

The disease is transmitted to humans by biting black flies
 (called Buffalo gnats). When these flies bite a human, they allow the

parasitic larvae to enter the human's body. Once inside the human, the larvae begin to mature into adults. Adults then produce millions of tiny worms, called **microfilaria**, which migrate throughout the body.

- River blindness often causes severe itching of the skin, and if left untreated, it may lead to blindness.
- o River blindness is considered an epidemic in more than 25 countries across the central part of Africa. According to the World Health Organizations' export committee on river blindness, about 18 million people are infected with the parasite each year worldwide. Of those infected, an estimated **6.5 million suffer from severe itching** or dermatitis, 500,000 suffer serious visual impairment, and 270,000 are blind.
- Scabies: Scabies is a contagious skin disease that is caused by microscopic mites that live three to four weeks in a person's skin. The female mite burrows into skin surface to lay her eggs. These eggs cause an inflammatory response in the host that causes itching, redness, and mild swelling. Scabies is often spread through direct or prolonged skin contact with an infected person or animal. It is easily spread through direct contact with sexual partners or family members. It may also be spread after sharing clothes, towels, bedding, or other linens, with an infected person. According to the American Academy of Dermatology, about 300 million cases of scabies are reported each year worldwide

#### **Cutaneous Filariasis**

Cutaneous Filariasis is a **parasitic** disease caused by thread-like filarial nematodes ... These worms occupy the subcutaneous layer of the **skin**, in the fat layer. .... Chronic papular lesions often with postinflammatory **hyperpigmentation**.

Filariasis is a parasitic disease caused by thread-like filarial nematodes (roundworms) in the family Filarioidea (also known as 'filariae').[1] Of the hundreds of described filarial parasites, only eight species cause natural infections in humans (see separate articlesLymphatic Filariasis and Body Cavity Filariasis).[1] Cutaneous filariasis may be caused by Loa loa (the African eye worm), Onchocerca volvulus and Mansonella streptocerca. These worms occupy the subcutaneous layer of the skin, in the fat layer.

• The larvae develop into adults in palpable subcutaneous nodules, usually found over bony prominences of the thorax, pelvic girdle or knees (also found on the head of children). Adults can live in the nodules for approximately 15 years. Some nodules may contain many male and female worms.

- In the subcutaneous nodules, the female worms produce microfilariae, which have a lifespan that may reach two years. Microfilariae are usually found in the skin and in the lymphatics of connective tissues.
- A blackfly ingests the microfilariae during a blood meal. After ingestion, the microfilariae migrate to the thoracic muscles, where they develop into third-stage infective larvae. The third-stage infective larvae migrate to the blackfly's proboscis and can then infect another human when the fly takes a blood meal.
- n the mild form there is localised maculopapular rash with itching. These may clear spontaneously or progress to a chronic and generalised form with severe itching.
- May heal with **hyperpigmentation**. Lichenified, hyperkeratotic lesions can be very distressing, as they are widespread and intensely itchy.
- A localised form in Arabia causes chronic **papular dermatitis**, often in one extremity only.
- In long-standing infection, destruction of elastic fibres in the skin makes it thin and wrinkled. The skin begins to sag and depigmentation of the pretibial areas is typical in older people living in endemic areas, (called 'leopard skin').
- Light-skinned patients infected on visiting a country may appear a year or so later with intensely itchy, red macular or maculopapular lesions that may be localised to one area of the body or be more generalised.
- There may also be fever, muscle or joint pain, weight loss and lymphadenitis.
- Rash sometimes lasts for several months after treatment.
- Ocular changes include intraocular microfilariae, punctate keratitis, sclerosing keratitis, anterior uveitis, chorioretinitis, optic neuritis, optic atrophy, glaucoma, and severe sight impairment (river blindness).

#### 1Treatment

- Ivermectin: a single dose clears microfilariae from the skin for several months. Repeating the dose every 6-12 months prevents progression.[6]
- Treatment is often associated with increased itching, **swelling of the face or extremities**, headache and body pains, which usually occur after the first treatment.

# 2Prevention

- Control of blackfly by spraying.
- Mass distribution of ivermectin.
- **Doxycycline** is effective at eliminating *W. pipientis* and its elimination may have a very important role in the treatment of onchocerciasis and other nematode infections in the future.[7]

# Mansonellosis (Mansonella streptocerca)

[8]

- M. streptocerca are found in Africa.
- Transmitted by *Culicoides* midges in tropical climates, they are of very limited clinical significance.
- Of the *Mansonella* species, only *M. streptocerca* causes recognised cutaneous symptoms.
- The females measure approximately 27 mm in length.

# 1Life cycle

- During a blood meal, an infected midge introduces third-stage filarial larvae on to the skin of the human host, where they penetrate into the bite wound.
- Larvae develop into adults in the dermis, close to the skin surface. Adults produce microfilariae, which live in the skin but can also reach the peripheral blood.
- A midge ingests the microfilariae during a blood meal. The microfilariae migrate to the thoracic muscles, where they develop into third-stage larvae. The third-stage larvae migrate to the midge's proboscis, and can then infect another human when the midge takes another blood meal.

#### 2Presentation

- Chronic papular lesions often with postinflammatory hyperpigmentation.
- Less commonly, it causes lichenification.

# 3Investigations

• Microfilariae shown in blood or skin (a distinctive 'walking stick' shape to the tail).

# 4Treatment

- If asymptomatic then no treatment is required.
- Otherwise, either diethylcarbamazine or ivermectin is effective.

#### **Tapeworm**

http://www.pawnation.com/2012/07/12/15-diseases-you-can-catch-from-your-pet/15

https://www.google.com/#g=flea+tapeworm

http://en.wikipedia.org/wiki/Dipylidium\_caninum

http://www.cdc.gov/parasites/dipylidium/faqs.html

**Dipylidium caninum,** also called the **flea tapeworm**, **double-pore tapeworm**, or **cucumber tapeworm** (in reference to the shape of its cucumber-seed-like <u>proglottids</u>), though these also resemble grains of rice or sesame seeds), is a <u>cyclophyllid cestode</u> that infects organisms afflicted with <u>fleas</u> and <u>canine chewing lice</u>, including <u>dogs,cats</u>, and sometimes human pet-owners, especially children. The adult worm is about 18 inches (46 cm) long. <u>Gravid</u> proglottids containing the worm's microscopic eggs are either passed in the definitive host's feces or may leave their host spontaneously and are then ingested by microscopic flea larvae (the intermediate hosts) in the surrounding environment. These larvae eventually <u>pupate</u> and transform into adult fleas still carrying the tape worm, which are then ingested by a dog or cat during grooming activity. From there, the worm enters the animal's digestive tract and anchors itself to the intestinal wall where it will soon begin generating <u>proglottids</u>, completing the life cycle. Examples of fleas that can spread *D. caninum* include <u>Ctenocephalides canis</u> and <u>Ctenocephalides felis</u>.

As in all members of family Dipylidiidae, p roglottids of the adult worm have genital pores on both sides (hence the name double-pore tapeworm). Each side has a set of male and female reproductive organs. The uterus is paired with 16 to 20 radial branches each. The scolex has a retractable rostellum wi th four rows of hooks. along with the four suckers that all cyclophyllid cestodes have.

<u>Cream Color Worms</u> <u>are plainly visable in</u> the stool.

Adult flea harbours Humans, normally children, the infective cysticercoid. acquire the infection by ingesting the infected flea. Host is infected Infected larval by ingesting fleas stage develop containing cysticercoid into adult flea Oncosphere Cysticercoi Scolex attaches Animals can transmit the Oncospheres hatch from in intestine the eggs and penetrate the infected fleas to humans. intestinal wall of the larvae. Cysticercoid to Gravid proglottids are develop in the body cavity. passed intact in the feces or emerge from perianal region of either anima or human hosts Adult in small intestine Δ Egg packets containing Each proglottid contains egg packets embryonated eggs that are held together by an outer are ingested by larval embryonic membrane (see 2). stage of flea. The proglottids disintegrate and release the egg packets. = Infective Stage A = Diagnostic Stage http://www.dpd.cdc.gov/dpdx

In children, infection

causes diarrhea and restlessness. As with most tapeworm infections, the drugs of choice are <u>niclosamide</u> or <u>praziquantel</u>. The best way to prevent human infection is to treat infected animals to kill fleas. Tapeworm infection usually does not cause pathology in the dog or cat, and most pets show no adverse reaction to infection other than increased appetite.

The other tapeworm infecting cats is <u>Taenia taeniaeformis</u>, though this form is much less commonly encountered that the one currently under discussion here.

#### Can humans be harmed by tapeworms?

Certain tapeworms found in dogs or cats may cause serious disease in humans. Fortunately, these tapeworms (*Echinococcus* species) are uncommon in the United States and are readily treated by prescriptions available from your veterinarian. There are rare reports of *Dipylidium*(a common tapeworm in pets) infections in children, but these infections are not associated with significant disease.

#### **Can Cat Worms Infect People?**

A disease which can be transmitted from animals to humans is known as a zoonotic disease. There are several <u>external and internal cat parasites</u> which are zoonotic, some of which can be more serious to humans than to their pets. This is because humans are an abnormal host for feline parasites, so the parasites become lost and confused in a human body and do some unusual things when they cannot find their way to a target feline organ.

Feline roundworms can also cause disease in humans. Eggs from this parasite are excreted in cat feces. After two weeks in the open they can become infectious to humans! If accidentally ingested, the worms can migrate to organs such as the liver, lungs, brain or eyes, where the human body generates an immune response to try to wall them off and prevent them moving any further.

- o **Tapeworm infection**: Fish tapeworm, beef tapeworm and pork tapeworm are obtained from eating raw or undercooked, infected meat. Adult worms can reach a length of more than 15 feet.
- o Pork tapeworms can enter the brain and cause seizures. Fish tapeworms can produce over one million eggs per day.
- The fish tapeworm is the largest of the human tapeworms, reaching the length of 33 feet or more. There can be 3,000 to 4,000 segments in one worm. It can produce more than 1,000,000 eggs a day. This type of infestation can cause anemia because of interference with vitamin B12, says Dr. Brooks in his book. Also, the weight challenges of some people can be directly attributed to tapeworms. This is especially true of weight loss programs that don't work. The person may be hosting a tapeworm which is eating all the food and making the person constantly hungry. Tapeworms can also cause water retention. Besides

tapeworms from beet, pork and fish, there is also a type of dog tapeworm you can get when dogs lick your face or hands.

- A **tapeworm** infection is a parasitic infection that affects the digestive tract.
- Humans become infected with tapeworms after they consume food or water that is contaminated with tapeworm larvae.
- Most tapeworm infections in humans are caused by the pork tapeworm (Taenia solium), the dwarf tapeworm (Hymenolepis nana), the beef tapeworm (Taenia saginata), or the fish tapeworm (diphyllobothrium latum). Tapeworm infections typically occur when a person consumes food, water, or soil that is contaminated with human or animal feces. • Most tapeworm infections cause no symptoms.

#### **Symptoms**

- Tapeworm infections do not always cause symptoms. The most common symptoms include weakness, nausea, decreased appetite, diarrhea, weight loss, and abdominal pain. Some patients may be able to see small white tapeworm eggs in their stools.
- However, if symptoms do occur, patients typically experience abdominal pain, bloating, nausea, and diarrhea.
- Can cause great harm when the immature <u>larvae invade the</u> <u>muscles, heart, eye or brain</u> when infection is often misdiagnosed as epilepsy.
- Signs and symptoms: Can consume over 80% of its host's vitamin B12 producing pernicious anaemia. Other common symptoms include digestive disturbances, pain and fullness in the upper abdomen, nausea and anorexia.

#### **Diagnosis**

- Stool Passing of white round ¼ inch or larger Segments,
- Flat ribbons, or sectioned worms are tapeworms.

#### **Differential**

 These worms don't appear to be doing that... just moving around and bunkering in in small hidy holes (under my tonsils) and small spaces at the back of my neck.

#### **Behaviour**

o Only <u>Tapeworms</u> are so <u>aggressive</u>

### **Complications**

Tapeworm infections may lead to malnutrition. The parasites absorb many of the nutrients from the food its host eats before the patient is able to do so. As a result, the patient may not get the necessary vitamins and minerals to stay healthy. This may also lead to weight loss. Once the parasite is killed, the patient will be able to absorb nutrients and will gain back lost weight.

#### **Treatment**

 $_{\odot}$   $\,$  Tapeworm infections that are limited to the intestines can be successfully treated with anti-helminthic medications.

#### **Prevention**

• Cooking meat thoroughly kills the parasites and prevents an infection from occurring. The meat must reach at least at least 150 degrees Fahrenheit in order to ensure that any tapeworm eggs and larvae have been killed.

#### 0

#### **TREATMENT**

### Natural treatment FOR TAPEWORMS

- Asafoetida Dissolve a small piece of asafoetida in water and drink it on an empty stomach once a day for 3 days. Ash Gourd Seeds Take a handful of ash gourd seeds and grind them. Eat it in the morning on an empty stomach. Two hours later, take two teaspoons of castor oil.

  Betel nut Grind one betel nut with a small glass of milk. Take this early in the morning on an empty stomach. NOTE: Do not give to children.

  Avoid: if suffering with cardiovascular disease or pregnant.
- Black Walnut Hulls can be taken as an extract. NOTE
   This herb is not recommended for infants or children.
- Coconut and Castor Oil. A tablespoon of the freshly ground coconut should be taken at breakfast followed by a dose of castor oil after three hours. The process may be repeated till the cure is complete.
- Paico Leaf can effectively eradicate tapeworms in most cases.
- Pumpkin the seeds of this herb can be eaten whole or mashed and mixed with juice. Two or three hours after eating up to 25 ounces of seeds, a laxative such as **senna** or **prunes** is recommended to purge the intestines.

- o **Cascara sagrada** this herb can be used after eating pumpkin seeds to help eliminate parasites from the body. It is milder than **senna**, but has a similar action.
- Wormseed can be taken as a tea. Do not use concentrated wormseed oil as it is too potent.
- Torrya seed can be used with pumpkin seed and betel nut

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# PYR-P **Pyrantel Pamoate** 1 oz Reeses Pinworm medicine liquid 144mg/ml NDC 10956-618-01

- o <u>www.drugs.com</u>
- Effective against most nematode infections
- clinical pharmacology Pin-X® has demonstrated anthelmintic activity against Enterobius vermicularis (pinworm) and Ascaris lumbricoides (common roundworm). The anthelmintic action is probably due to the neuromuscular blocking property of the drug. Pin-X® is partially absorbed after an oral dose. Plasma levels of unchanged drug are low. Peak levels (0.05 0.13 micrograms per milliliter) are reached in 1-3 hours. Quantities greater than 50% of administered drug are excreted in feces as the unchanged form, whereas only 7% or less of the dose is found in urine as the unchanged form of the drug and its metabolites.
- Symptoms suggestive of pinworm infestation: Pruritis Ani (itching in the anal area), insomnia, gastrointestinal distress, irritability, enuresis (bed wetting) and secondary infection due to localized scratching are symptoms of pinworms. However, you should make a visual inspection and confirmation of the pinworms before using this product.
- o pinworms all areas should be as clean as possible.
- Abdominal cramps, nausea, vomiting, diarrhea, headache, or dizziness sometimes occur
- 11 mg/kg (maximum: 1 g/dose) administered once daily for
   3 days
- Ancylostoma duodenale (hookworm), Ascariasis lumbricoides (roundworm), Necator americanus (hookworm) (unlabeled use):
- 11 mg/kg administered as a single dose; repeat twice 2 weeks apart

http://research.universityofcalifornia.edu/stories/2012/07/orphan\_drug.html

- What's more the UC researchers have already shown that auranofin is far more potent than the best drug currently used against the tropical infections an antibiotic called metronidazole, known to cause side effects such as nausea, vomiting and dizziness. If clinical trials confirm its effectiveness in infected people, the arthritis drug could be used at a lower dose and at a much lower cost per person, they said.
- Last year, they hit gold. They showed in lab and animal studies that an arthritis drug called auranofin can knock out a particularly aggressive parasite that disables hundreds of thousands and kills 70,000 people a year. The drug also cripples a second protozoan parasite responsible for nearly 300 million infections a year. Auranofin has been identified in a high-throughput drug screen as 10 times more potent than metronidazole on Entamoeba histolytica, the protozoan agent of human amebiasis. Assays of thioredoxin reductase and transcriptional profiling suggest that the effect of auranofin on the enzyme enhances the sensitivity of the trophozoites to reactive oxygen-mediated killing in mouse and hamster models; the results are markedly reductions of the number of parasites, the inflammatory reaction to the infestation and the damage to the liver.[5][6][7]
- Metronidazole Flagyl is a nitroimidazole antibiotic medication used particularly for anaerobic bacteria and protozoa.
- Metronidazole is primarily used to treat: bacterial vaginosis, pelvic inflammatory disease (along with other antibacterials like ceftriaxone), pseudomembranous colitis, aspiration pneumonia, rosacea (topical), fungating wounds (topical), intra-abdominal infections, lung abscess, gingivitis, amoebiasis, giardiasis, trichomoniasis, and infections caused by susceptible anaerobic organisms such as Bacteroides, Fusobacterium, Clostridium, Peptostreptococcus, and Prevotella species.[6] It is also often used to eradicate Helicobacter pylori along with other drugs and to prevent infection in people recovering from surgery.[6]
- Metronidazole is listed by the US <u>National Toxicology Program</u> (NTP) as reasonably anticipated to be a human <u>carcinogen</u>. [15] Although some of the testing methods have been questioned, oral exposure has been shown to cause cancer in experimental animals and has also demonstrated some mutagenic effects in bacterial cultures
- o Interaction with alcohol Consuming alcohol while taking metronidazole has long been thought to have a disulfiram-like reaction with effects that can include nausea, vomiting, flushing of the skin, tachycardia, and shortness of breath.[24] Consumption of alcohol is typically advised against by patients during systemic metronidazole therapy and for at least 48 hours after completion of treatment.[6] However, some studies call into question the mechanism of the interaction of alcohol and metronidazole,[25],[26][27] and a possible central toxic serotonin reaction for the alcohol intolerance is suggested. [13] Metronidazole is also generally thought to inhibit the liver metabolism of propylene glycol (found in some foods, medicines, and in

many electronic cigarette e-liquids), thus propylene glycol may potentially have similar interaction effects with metronidazole.[citation needed]

Piperazine Phosphate powder (Pripsen)

This works differently to the Mebendazole which deprives them of glucose.

Piperazine Phosphate by Pripsen paralyzes the worms

the Pripsen tablets also contain Senna, a herbal mild laxative which helps expel them.

It seemed to work well for us but will not get rid of the worms in other parts of the body (eyes, nose, ears, vagina), for that I recommend Mebendazole. After taking the Mebendazole you might feel tired and headachey as the dead and decaying worms in your guts release toxins before you're able to expel them, nice huh?

#### **Nitazoxanide**

http://www.ncbi.nlm.nih.gov/pubmed/17594741

http://www.intramed.net/userfiles/1.pdf

Alinia (Pro, More...)

generic name: nitazoxanide class: amebicides

First, names. Nitazoxanide comes in so many different brand names that I can only list a few. Alinia was the first, and is the drug name given to nitazoxanide by its discoverer, Dr. Jean-Francois Rossignol, who works at the Pasteur Institute in Paris.

Dr. Rossignol is part of a company called Romark Laboratories, in Tampa, Florida, which manufactures Alinia and is running the current trials for its use as an adjunct therapy with SOC.

Romark has licensed certain other manufacturers to produce nitazoxanide. These are the less-expensive equivalents that can be found, for example, in Europe and Canada and a few other places that still respect patents and copyrights. In places that don't, nitazoxanide is produced by a plethora of small-ish pharmaceutical companies under a myriad of brand names. A few of them are: Daxon, Dexidex, Pacovanton, Paramix, Nitax, Zox, Nitazox, Toza, Zóntricon, Celectan, Nixoran, Dexidex, Kidonax, Nitanid, Colufase, Nodik, Paraxin, Rosanil, Nitaxom, Noxolin, Uniplus7, Nitaxin, Nitapax, Zoxanid, Nitarid, Bionit, Rosanil, Parsenida, Zotanixin.

Prices vary widely, as one would imagine. And since the Internet has spawned huge industries of online wholesalers, retailers, and geographically-unidentifiable resellers of everything under the sun including pharmaceuticals, there are places (I use this term in its broadest virtual sense) where you can buy nitazoxanide, or something that looks very much like it, for a small fraction of what the name brand companies sell it for. Here's a list of the ones I've had time to look at, together with price data:

http://www.wisemeds.net/

http://www.freedom-pharmacy.com/Products2.asp?ID=4120&T=

\_\_\_\_\_\_ http://www.wisemeds.net/buy\_online/Nitarid.shtml \*\*\*@\*\*\* Nitarid Glenmark Ltd. 500mg x 60 Tablets \$55.42 (\$0.92/tablet) \_\_\_\_\_\_ http://www.brandprescriptiondrugs.com/Nitazoxanide.htm (Int'l comparison site) Nitarid Cipla Ltd. (India) 2 x 60 tabs x 500mg \$ 99.65 (\$0.83/tablet) \_\_\_\_\_ http://www.generics.ws/shopping\_carts/show/ Nizonide Lupin Pharm.  $12 \times 2 = 24 \text{ for } \$18.96 = (\$0.79/\text{tablet})$ 

Nitazoxanide is an antiparasitic agent. It works by interfering with the production of certain substances that are needed by the parasite to live.

Take nitazoxanide with food.

Diarrhea; headache; nausea; stomach pain.

Seek medical attention right away if any of these SEVERE side effects occur:

Severe allergic reactions (rash; hives; difficulty breathing; tightness in the chest; swelling of the mouth, face, lips, or tongue); bone pain; fainting; fast heartbeat; severe or persistent dizziness; shortness of breath; unusual tiredness.

#### 1Usual Adult Dose for:

- Amebiasis
- Cryptosporidiosis
- Giardiasis

### **Usual Adult Dose for Giardiasis**

For diarrhea in immunocompetent patients: 500 mg twice daily with food for 3 days. For diarrhea in AIDS patients: 1000 mg twice daily with food for 14 days or until diarrhea resolves.

### <u>Usual Pediatric Dose for Cryptosporidiosis</u>

For diarrhea in immunocompetent patients:

12 to 47 months: 100 mg (5 mL) by mouth with food every 12 hours for 3 days.

4 to 11 years: 200 mg (10 mL) with food every 12 hours for 3 days.

Greater than or equal to 12 years: 500 mg twice daily with food for 3 days.

### **Usual Pediatric Dose for Giardiasis**

#### For diarrhea in immunocompetent patients:

12 to 47 months: 100 mg (5 mL) by mouth with food every 12 hours for 3 days.

4 to 11 years: 200 mg (10 mL) with food every 12 hours for 3 days.

Greater than or equal to 12 years: 500 mg twice daily with food for 3 days.

### **Usual Pediatric Dose for Ascariasis**

Study (n=105)

Dosage given orally morning and evening for 3 consecutive days.

2 to 3 years: 100 mg/5 mL 4 to 11 years: 200 mg/10 mL

### <u>Usual Pediatric Dose for Hymenolepis nana (Dwarf Tapeworm)</u>

Study (n=105)

Dosage given orally morning and evening for 3 consecutive days.

2 to 3 years: 100 mg/5 mL 4 to 11 years: 200 mg/10 mL

### **Usual Pediatric Dose for Amebiasis**

Study (n=53):

Greater than or equal to 12 years:

For diarrhea in immunocompetent patients: 500 mg twice daily with food for 3 days.

# Renal Dose Adjustments

Data not available

# **Liver Dose Adjustments**

Data not available

# **Dose Adjustments**

Data not available. Caution recommended.

### **Precautions**

Caution is recommended in patients with renal and/or hepatic disease.

Nitazoxanide should be taken with food.

#### http://cid.oxfordjournals.org/content/40/8/1173.full

Intestinal parasitic infections rank among the most significant causes of morbidity and mortality in the world today. Nevertheless, it has been >30 years since the introduction of any new innovative treatment and, for some pathogens (including Cryptosporidium), there is currently no accepted specific therapy [1]. Nitazoxanide, 2-acetyloxy-N-(5-nitro-2-thiazolyl) benzamide (Alinia: Romark Laboratories), is a new nitrothiazole benzamide compound notable for its activity in treating both intestinal protozoal and helminthic infections. It was first described in 1975 by Jean Francois Rossignol and was initially developed as a veterinary antihelminthic with activity against intestinal nematodes, cestodes, and liver trematodes [2]. In humans, nitazoxanide has been reported to be effective against a broad range of parasites, including Giardia lamblia, Entamoeba histolytica, Cryptosporidium parvum, Cyclospora cayetanensis, Trichomonas vaginalis, Vittaforma corneae, Encephalitozoon intestinalis, Isospora belli, Blastocystis hominis, Balantidium coli, Enterocytozoon bieneusi, Ascaris lumbricoides, Trichuris trichura, Taenia saginata, Hymenolepis nana, and Fasciola hepatica [3-9]. In vitro studies have also shown antimicrobial activity against numerous gram-positive and gram-negative anaerobic bacteria, specifically Bacteroides species, Clostridium species, and Helicobacter pylori, and against aerobic gram-positive bacteria [10, 11].

Studies of protozoa and anaerobic bacteria have shown that nitazoxanide inhibits pyruvate-ferredoxin oxidoreductase (PFOR), an enzyme essential to anaerobic energy metabolism [12]. However, interference with the PFOR enzyme-dependent electron transfer reaction may not be the only pathway by which nitazoxanide exhibits antiprotozoal activity, and the mechanism of nitazoxanide's activity against helminths is unknown.

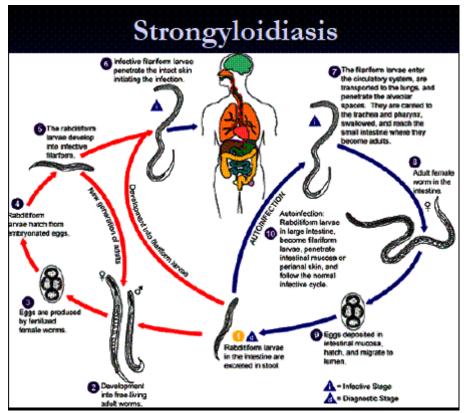
Nitazoxanide has demonstrated in vitro activity against *C. parvum* and *G. intestinalis*. It has been shown to inhibit the growth of sporozoites of *C. parvum* on its own [13, 14], and has also demonstrated combined in vitro activity with both azithromycin and rifampin, suppressing growth of *C. parvum* by 83.9% and 79.8%, respectively, compared with 56.1% when used alone [15]. Similarly, in vitro studies of nitazoxanide and its derivative, tizoxanide, have shown greater efficacy than metronidazole against *G. intestinalis* [16]. Specifically, tizoxanide was demonstrated to be 8 times more active than metronidazole against metronidazole-susceptible isolates of *G. intestinalis* and twice as active against resistant isolates [17].

Nitazoxanide has also shown broad in vitro activity against numerous other parasitic and microbial pathogens, including *E. intestinalis* [18], *V. corneae* [18], *E. histolytica* [16], *T. vaginalis* [16], *B. hominis* [19], *Echinococcus multilocularis* [20], *Echinococcus granulosus* [21], and *F. hepatica* [22]. The antimicrobial properties of nitazoxanide and tizoxanide have been tested against 241 anaerobes, the majority of which were inhibited in vitro, with an MIC<sub>90</sub> between 0.06 mg/L and 4 mg/L [10].

Nitazoxanide has also shown in vitro and in vivo antimicrobial activity against *Clostridium difficile* [23] and both metronidazole-susceptible and metronidazole-resistant strains of *H. pylori* [11, 24].

Nitazoxanide is available in oral suspension at a dose of 100 mg per 5 mL or in tablet formulation at a dose of 500 mg. The oral suspension, when reconstituted with water, has a pink color and a strawberry flavor. The recommended dosage for children aged 12–47 months is 100 mg b.i.d. for 3 days, and for children aged 4–11 years, the recommended dosage is 200 mg b.i.d. for 3 days. The recommended adult dosage is 500 mg b.i.d. for 3 days. The bioavailability of nitazoxanide is nearly doubled by administration with food [25].

Studies of pharmacokinetics in humans have shown that nitazoxanide is absorbed from the gastrointestinal tract, with approximately one-third of the oral dose excreted in urine and two-thirds excreted in feces [26]. In blood, nitazoxanide is rapidly hydrolyzed by plasma esterases into its desacetyl derivative, tizoxanide (desacetyl-nitazoxanide) [27]. Tizoxanide is the active metabolite in vivo and the only measurable species in plasma. Following oral administration of nitazoxanide, a maximum tizoxanide plasma concentration of 2 mg/L is observed within 1–4 h [27]. Tizoxanide is extensively bound to plasma proteins (>99%), and its urinary elimination half-life is 7.3 h [27]. Tizoxanide then undergoes glucuronidation to form tizoxanide glucuronide (figure 1). The parent drug, nitazoxanide, is not detected in plasma, urine, bile, or feces. Tizoxanide is found in plasma, urine, bile, and feces, and tizoxanide glucuronide is found in plasma, urine, and bile [27].



# **Strongyloides Protocol**

- Nosebleeds subsided and a worm ejected itself from each nostril, at different times. No nosebleeds since then.
- Continually suffer with the sores, crawling, biting, pricking, and itching

The name *threadworm* comes from the fact that although it is long in parasite standards (2 mm), **it is only 0.035 mm wide** – a thread. It is unusual in several respects. It has two forms: a parasitic form, and a form that is called 'free-living,' meaning it can live and reproduce just fine outside of a host. Another unique aspect of *Strongyloides* is that the parasitic worms are only females.

The female worm lives in the intestine of the host where it lays eggs. Remarkably, the eggs can develop even though they have not been fertilized by a male. In fact, there are no adult male worms. The eggs hatch into larvae in the intestine and are passed out in the feces. These larvae can either develop into infective parasitic larvae or into free-living worms of either sex. The parasitic larvae enter a new host by penetrating the skin. They then migrate to the lungs, travel up the trachea and are swallowed. The free-living larvae mate, but do not produce more free-living larvae, only infective larvae that must enter a

host to survive.

thread. It is unusual in several respects. It has two forms: a parasitic form, and a form that is called 'free-living,' meaning it can live and reproduce just fine outside of a host. Another unique aspect of *Strongyloides* is that the parasitic worms are only females.

The female worm lives in the intestine of the host where it lays eggs. Remarkably, the eggs can develop even though they have not been fertilized by a male. In fact, there are no adult male worms. The eggs hatch into larvae in the intestine and are passed out in the feces. These larvae can either develop into infective parasitic larvae or into free-living worms of either sex.

The parasitic larvae enter a new host by penetrating the skin. They then migrate to the lungs, travel up the trachea and are swallowed. The free-living larvae mate, but do not produce more free-living larvae, only infective larvae that must enter a host to survive.

Ingested larvae go directly to the small intestine where they burrow in the intestinal wall andmature there, causing ulceration of the lining of the intestines and subsequent bloody diarrhea is often seen. Bowel movements may be dark and tarry, or black in color indicating ulceration and bleeding in the intestines.

Larvae that penetrate the skin migrate to the lungs are coughed up and swallowed, and take up final residence in the small intestine. Autoinfection can also occur if the first-stage larvae develop to infective third-stage larvae before passing out of the host. If this occurs, the larvae penetrate the mucosa of the rectum or perianal skin and migrate through the body as before.

#### **Herxheimer Reaction To Parasitic Die-Off:**

- While taking parasite drugs, a Herxheimer reaction might occur in severe infections with a heavy parasite burden. This is an immune system response to toxic chemicals (endotoxins) released in the blood & tissue by parasites & pathogens killed off by medication.
- While the body is detoxifying, the released toxins might (but not always) either worsen the symptoms being treated or create their own.
- As a result, you might feel worse before feeling better.
   Although you may not feel very well, the Herx effect is actually a sign that healing is taking place.
- $\circ$  The important thing is that worsening symptoms do not indicate failure of the treatment in question. In fact, it's usually just the opposite.
- So brace yourself if dying parasites & toxins make a mass exodus from every pore & orifice of your body.

### **Dosing Tips:**

- Recommend taking these antiparasitic vitamins & supplements, especially Zinc & Sulfur (MSM), during treatment: http://curezone.com/forums/fm.asp?i=1575262#i.
- Eat high-fiber foods & drink 8 glasses of fluids daily to flush out parasitic die-off & toxins. During drug treatment, stop using herbal remedies, flushes, cleanses, DE, laxatives, colonics, MMS, nutrasilver, RIFE, zappers, & other OTC parasite products.
- None of these methods cure parasite infections. They can be toxic, destroy beneficial flora in the gut, & impair organs of the digestive system.
- They cause parasites to scatter everywhere, carrying enteric pathogens from the GI tract with them. And they counteract & remove parasite drugs from your system.
- O Disseminated parasites & toxins throughout the body create hyperinfectin & severely impact the immune system. Then it's an uphill battle to eradicate them & repair damage done to the body.
- For constipation or if worms or larva migrate to lower colon out of reach of meds in upper intestines:

### **Peripheral Nervous System Disorders**

- The peripheral nervous system refers to parts of the nervous system outside the brain and spinal cord. It includes the cranial nerves and spinal nerves from their origin to their end. The anterior horn cells, although technically part of the CNS, are sometimes discussed with the peripheral nervous system because they are part of the motor unit.
- Motor neuron dysfunction results in muscle weakness or paralysis. Sensory neuron dysfunction results in abnormal or lost sensation. Some disorders are progressive and fatal.
- o 17.1.1 Anatomy
- A motor unit consists of an anterior horn cell, its motor axon, the muscle fibers it innervates, and the connection between them (neuromuscular junction). The anterior horn cells are located in the gray matter of the spinal cord and thus are technically part of the CNS. In contrast to the motor system, the cell bodies of the afferent sensory fibers lie outside the spinal cord, in dorsal root ganglia. Nerve fibers outside the spinal cord join to form anterior (ventral) motor roots and posterior (dorsal) sensory root nerve roots. The ventral and dorsal roots combine to form a spinal nerve. Thirty of the 31 pairs of spinal nerves have dorsal and ventral roots; C1 has no sensory root (Fig. 1: Spinal nerve. ). The spinal nerves exit the vertebral column via an intervertebral foramen. Because the spinal cord is shorter than the

vertebral column, the more caudal the spinal nerve, the further the foramen is from the corresponding cord segment. Thus, in the lumbosacral region, nerve roots from lower cord segments descend within the spinal column in a near-vertical sheaf, forming the cauda equina. Just beyond the intervertebral foramen, spinal nerves branch into several parts.

O Branches of the cervical and lumbosacral spinal nerves anastomose peripherally into plexuses, then branch into nerve trunks that terminate up to 1 m away in peripheral structures. The intercostal nerves are segmental. The term peripheral nerve refers to the part of a spinal nerve distal to the root and plexus. Peripheral nerves are bundles of nerve fibers. They range in diameter from 0.3 to 22 μm. Schwann cells form a thin cytoplasmic tube around each fiber and further wrap larger fibers in a multilayered insulating membrane (myelin sheath).

### **Etiology**

Disorders can result from damage to or dysfunction of the cell body, myelin sheath, axons, or neuromuscular junction. Disorders can be genetic or acquired (due to toxic, metabolic, traumatic, infectious, or inflammatory conditions—see Table 1: Some Causes of Peripheral Nervous System Disorders ). Peripheral neuropathies may affect one nerve (mononeuropathy), several discrete nerves (multiple mononeuropathy, or mononeuritis multiplex), or multiple nerves diffusely (polyneuropathy). Some conditions involve a plexus (plexopathy) or nerve root (radiculopathy). More than one site can be affected; eg, in the most common variant of Guillain-Barré syndrome, multiple segments of cranial nerves, usually the 2 facial nerves, may be affected.

#### Motor neuron\*

Acquired, chronic Amyotrophic lateral sclerosis, paraneoplasticsyndrome, postpolio syndrome, progressive bulbar palsy

- Nerve root Hereditary Neurofibroma
- o Acquired Herniated disk, infections, metastatic cancer, spinalforaminal stenosis, trauma
- o Plexus Acquired Acute brachial neuritis, diabetes mellitus, hematoma, local tumors (eg, schwannoma), metastatic cancer, neurofibromatosis (rare),
- o traction during birth, severe trauma Peripheral nerve
- o Infectious Hepatitis C, herpes zoster, HIV infection, Lyme disease, syphilis
- o In developing nations: Diphtheria, leprosy, parasite Infections

#### **Recovery:**

- o Damage to the myelin sheath (eg, by injury or Guillain-Barré syndrome) can often be repaired by surviving Schwann cells in about 6 to 12 wk.
- o After axonal damage, the fiber regrows within the Schwann cell tube at about 1 mm/day once the pathologic process ends. However, regrowth may be misdirected, causing aberrant innervation (eg, of fibers in the wrong muscle, of a touch receptor at the wrong site, or of a temperature instead of a touch receptor).

### **Finding Cause to Consider**

Symmetric, diffuse deficits Diffuse disorders (eg, toxic metabolic, hereditary, infectious, or inflammatory disorders; most immune-mediated disorders)

Unilateral deficits Focal disorders (eg, mononeuropathies, plexopathies)

Deficits localized to one or more peripheral nervous system structures (eg, nerve root, spinalnerve, nerve plexus, single peripheral nerve,  $\geq 2$  discrete nerves in separate areas [multiplemononeuropathy])

### Lesion in a peripheral nervous system structure

- o Stocking-glove distribution of deficits Diffuse peripheral polyneuropathies, possibly axonal Disproportionate weakness of proximal muscles (eg, difficulty climbing stairs or combing hair) with no sensory deficits
- o Diffuse muscle dysfunction, as occurs in diffuse myopathies
- o Possibly disorders of the neuromuscular junction if the eyes are affected
- o Chronic, progressive weakness affecting mostly distal muscles with no sensory deficits Motor neuron disease

#### Buzzing and tingling with motor weakness and decreased reflexes

#### Demyelination

Profound **proximal** and distal (away from the center line ) motor weakness with minimal atrophy

### Acquired demyelinating polyneuropathy

Deficient pain and temperature sensation; painful, often burning sensations

Weakness proportional to atrophy; disproportionately mild reflex abnormalities,

Vascular disorders (eg, vasculitis,ischemia, hypercoagulable states)

## **Threadworm (Strongyloides)**

- o Threadworm infection: Cream-colored parasites as thin as a thread.
- $_{\circ}$  When Provoked (treated) They often come out in the stool by the hundreds.
  - Threadworm infection, also called

# strongyloidiasis are red.

- Strongyloides ate also called <u>red worms</u> or <u>blood worms</u>.
- Strongyles are the really bad kids on the block. They top the most-unwanted list on pretty much any list of parasites.
- They pose a serious threat to health.
- Acute infection is generally characterized by gastrointestinal
   (GI) and pulmonary symptoms, whereas chronic infection is characterized by skin involvement.
- Severe strongyloidiasis (hyperinfection, disseminated disease) may be insidious; occasionally, symptoms may have an abrupt onset. Fever is almost always present in disseminated disease. [34] Invasion of larvae into tissue is potentially massive. As a result, patients present with an exaggeration of the symptoms of established infection found in patients who are immunocompetent. In addition, as larvae penetrate the intestinal wall, they may allow enteric flora to escape, causing bacteremia, sepsis, meningitis, and endocarditis. Thus, a diagnosis of severe strongyloidiasis should be suspected with unusual GI or pulmonary symptoms or an unexplained Gram-negative bacilli sepsis.

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Strongyloides is a parasitic infection of the intestines that is

caused by a type of roundworm called Strongyloides stercoralis. Unlike the more common roundworm infection **Ascariasis**, a threadworm infection may also spread to the skin. • The infection is transmitted to humans when a person comes into contact with soil

that is contaminated with **S. stercoralis**. This usually occurs when an individual walks barefoot on soil. The larvae enter the human through the skin. Once inside the body, they migrate to the lymph nodes where they are carried into the lungs. Once in the lungs, the larvae migrate to the patient's throat. When the patient coughs, the larvae are swallowed and they enter the digestive tract. Once in the intestine, the larvae mature into egg producing adults. The eggs are then released in the patient's feces. Adult threadworm may grow to be one to two inches long. • Threadworm infections are not fatal. However, if left untreated, threadworm infections can last for as long as 45 years. • Although this infection can occur in most areas of the world, it is most prevalent in tropical and subtropical climates.

Threadworms Contracted through the soil in south Asia and south-eastern USA, larvae penetrate the skin and invade the intestine wall or lungs. Can reproduce entirely in a human host or grow as a free-living worm. Infections can last for 30 years.

### **Symptoms:**

The signs and symptoms of threadworm infection vary, depending on the stage of the disease.

After the larvae enter the body through the skin, the area may be swollen and itchy, similar to a bug bite. Patients with long-term threadworm infections may develop an itchy skin rash near the buttocks, abdomen, and/or thighs.

Some patients may only have mild diarrhea and cramping, while others may have nausea, vomiting, fever, fatigue, and blood or mucus in the stools.

When the larvae move to the lungs and airways, the patient may develop a dry cough, fever, or difficulty breathing and may cough up blood or pus.

#### **Gastrointestinal manifestations**

- o Gastrointestinal symptoms are vague, including epigastric abdominal cramping, indigestion, anorexia, nausea, vomiting, chronic diarrhea, constipation, pruritus ani, bloating and, rarely, small bowel obstruction. The common signs are bloating, greasy stools, diarrhoea and pulmonary disorders.
- Strongyloides May bring on intense itching in the area around the rectum and cause periodic bouts of diarrhoea alternating with constipation, cough and fever.

 Prolonged malabsorption of both fat and protein can lead to a celiac-like syndrome, characterized by steatorrhea, hypoalbuminemia, and peripheral edema.

#### In disseminated disease,

Abdominal symptoms are similar to those of chronic infection, but they are more severe. Gut flora invade host tissues either through penetration of infective larvae from bowel lumen or through damaged intestinal epithelium. *Escherichia coli* and *Klebsiella* species are the most common organisms involved. Bloody stools and/or blood diarrhea may occur along with severe abdominal pains. Massive GI tract bleeding has also been reported.[35]-

#### **Complications**

o If the larvae spread to other organs in the body, the condition is called hyperinfection syndrome. Symptoms of this condition may include inflammation of the heart tissue, stomach ulcers, perforations of the intestines, blood poisoning, meningitis (which often causes fever, headache, vomiting, nausea, confusion, and fatigue), sudden and life-threatening drop in blood flow throughout the body (called shock), and possible death.

http://www.naturecures.co.uk/parasites.htm

#### **TREATMENT**

#### **Pharmacudical Treatment for Threadworms**

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#### **Natural TREATMENT FOR THREADWORMS**

o **Butternut** this herb relieves constipation and expels intestinal worms. **NOTE** This herb should not be taken for a long period of time. **Neem Leaves** Grind neem leaves to a fine paste. For one week, take a marble sized ball of this paste every morning on an empty stomach. Do not take this for the second week. For the third week, take the neem leaves again as before on an empty stomach. The whole family should take this treatment together for curing **threadworms**. **Carrot** Can eliminate **threadworms** from children. A small cup of grated carrot taken every morning for three days, with no other food added to this meal, can clear these worms quickly. **Chinese Rhubarb Root** Mix 1 teaspoon of **rhubarb** powder to 1 cup of water. Then, bring to boil and simmer at a reduced heat for 10 minutes. Add a little honey to sweeten.

#### #Indigo [Indq]

Is a remedy for **ascarides or thread-worms** in melancholy children, with intense pain in the umbilical region, also convulsions from worms.

### **Trichinosis:**

- Trichinosis is a type of parasitic infection that is caused by the **roundworm** Trichinella.
- Trichinella (trichinella spiralis) diarrhea, headache, muscle pains, chills, eye swelling, Contracted by eating undercooked or raw pork. Cysts are present in infected pork and once eaten the immature roundworms are released and can travel to human muscles. They burrow into the larynx, chest, abdomen, jaw and upper arm and cause tender calcified cysts in muscles and fever.

#### **Vector**

 Humans become infected when they eat undercooked meat (usually beef or pork) that is contaminated with Trichinella larvae.

### **Development**

o Once inside the human, the larvae mature in the intestine into adult worms over the course of several weeks. The adults then produce larvae that migrate to various body tissues, including muscle.

### **Symptoms**

- Some patients may have mild, if any, symptoms.
- Symptoms of trichinosis range from mild to severe, depending on the number of parasites in the body.
- o Patients with a very mild form may experience no symptoms at all.
- When the parasite is in the intestine, common symptoms include diarrhea, abdominal pain, and general feeling of discomfort.
- About one week after the parasite enters the body, the females produce larvae that penetrate body tissues, including muscles. Symptoms at this stage may include <a href="high-fever">high-fever</a>, muscle pain and tenderness, weakness, <a href="swelling-of-the-eyelids-or-face">swelling-of-the-eyelids-or-face</a>, <a href="sensitivity-to-light">sensitivity-to-light</a>, headache, and <a href="pinkeye">pinkeye</a> (called conjunctivitis).
- Upon infection 2-4 weeks of acute gastrointestinal symptoms, thereafter severe muscle pain plus a wide range symptoms which can masquerade as a variety of more familiar diseases. In the advanced stages the <a href="Trichinella encysts within muscles">Trichinella encysts within muscles</a>, causes extreme dehydration, swelling of the lips and eyelids, difficulty breathing, enlarged lymph glands, meningitis and pneumonia.
- However, if the patient is infected with hundreds of worms, it may lead to permanent tissue damage. This is because the larvae burrow into the patient's muscle and other tissues in the body.

#### **Prevention**

The United States Centers for Disease Control (CDC)
 recommends cooking meat products until the juices run clear or to an internal temperature of 170 degrees Fahrenheit in order to kill
 Trichinellalarvae.

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#### **Treatment**

Albendazole 400 mg po bid for 8 to 14 days

### **Pinworms**

#### o **Pinworms**

http://www.nytimes.com/health/guides/disease/pinworms/overview.html?module=Search&mabReward=relbias%3Aw

http://www.healthline.com/health/pinworms#Overview1

o Pinworms are small worms that infect the intestines.

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#### **Alternative Names**

Enterobiasis; Oxyuriasis; Threadworm; Seatworm;
 Enterobius vermicularis; E vermicularis; "Helminthic"sl. infection

Symptoms are itching and irritation of the anus or vagina, digestive disorders, insomnia, irritability or nervousness.

#### **Causes**

- o Pinworm eggs are spread directly from **person to person**.
- They can also be spread by touching bedding, food, or other items contaminated with the eggs.

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#### **Vector**

- o Typically, children
- They are most common in school-age children.
- Children are infected more than any other segment of the population, but any age group can have pinworms
- o Infected persons unknowingly touching pinworm eggs and putting their fingers in their mouths. The eggs are swallowed, and eventually hatch in the small intestine. The worms migrate and mature in the colon.
- Female worms then move to the anal area, especially at night, and deposit more eggs. This may cause intense itching. The area may even become infected. When the person scratches the anal area, eggs can get under fingernails. These eggs can be transferred to others or items in the house.

### **Prevalance / Numbers**

- Worldwide, about 500 million are infected with pinworms.
- Pinworms are the most common worm infection in the United States.
- Enterobiasis or oxyuriasis, is the most common type of worm infection in humans in the United States.
- Pinworms About 30 to 40 million people in the USA are believed to be infected with pinworms. Most prevalent in children, eighty percent of children between 2-10 years of age will contract pinworms.

#### **Transmission**

o Contracted through contaminated water, soil, house dust and also by human-to-human contact. The adult female pinworm inhabits the intestine and moves outside the anus to lay eggs at night. The eggs can then be transferred to the mouth by the fingers after itching the anal area and infections can also be spread to other family members via bathtubs, toilet seats, and bedding.

- Female worms crawl out of the anus to lay their eggs around the anal region at night. One female can deposit over 15,000 eggs that become infective immediately or within hours. Adult pinworms are about 0.5 in. (12.7 mm) long and look like little white threads. Pinworm eggs are so tiny; you'd need a microscope to see them.
- o Pinworms are very infectious and can cause a lot of itchiness in the anal area. "The worms deposit their eggs mostly at night, contaminating pajamas and bed linen," writes Dr. Brooks. "The eggs are readily transported through the air, and it is not uncommon to find them in every room of the house....Complications are much more common in women than in men" . Pinworms can also be found in the vulva, uterus and fallopian tubes because the female worm loses her way while trying to return to the anus after depositing her eggs.
- The eggs are normally caught on tape, placed near the anus at night. When the worms lay eggs, it can cause itching. If the child scratches, the eggs can cling to the child's fingers and get stuck under the fingernails.
- The eggs then stick to things the child touches, such as clothing, dishes, toys, and furniture. The eggs can live 2 to 3 weeks outside the body.
- When you touch something the child has touched, the eggs get on your hands. Then if you touch food or your mouth, you can swallow the eggs. This starts the cycle over again.
- Pinworms spread easily in homes, day care centers, schools, and other places where groups of people spend time together. So if one person in your family has pinworms, others probably do too.
- o It's possible to get pinworms by inhaling airborne eggs, but this is rare. It's also rare to get pinworms from a swimming pool.
- o **Pinworms** are spread from person to person. Pets don't get pinworms and can't spread them to humans.
- The crawling of the female worm on the skin around the anal area often causes intense itching causing a person to scratch, getting eggs on their hands. If unwashed hands touch the mouth or food, the eggs are swallowed and hatch in the lower colon where the worms mate, and the cycle continues.

#### Risc Factors

While anyone can get a pinworm infection, the following groups are more susceptible:

Children who attend daycare, preschool, or elementary school
 Family members or caregivers of infected children and adults
 Individuals who live in institutions or other crowded accommodations
 Children who don't practice regular and careful hand washing prior to eating

### **Symptoms:**

o Irritation and intense itching in the area around the rectum especially at night. Pinworm infection can produce an enormous range of diverse mental and behavioural symptoms including epilepsy, hyperactivity and vision problems.

Children who have a habit of sucking their thumbs

- Difficulty sleeping due to the itching that occurs during the night
- Intense itching around the anus
- Irritability due to itching and interrupted sleep
- Irritated or infected skin around the anus, from constant scratching
- o Irritation or discomfort of the vagina in young girls (if an adult worm enters the vagina rather than the anus)
- Loss of appetite and weight (uncommon, but can occur in severe infections)

#### **Exams and Tests**

- Pinworms can be spotted in the anal area, especially at night when the worms lay their eggs there.
- O Your doctor or nurse may have you do a tape test. A piece of cellophane tape is pressed against the skin around the anus, and removed. This should be done in the morning before bathing or using the toilet, because bathing and wiping may remove eggs. The doctor will stick the tape to a slide and look for eggs using a microscope.
- o (enterobius vermicularis) **Pinworms** are a common human parasite, causing enterobiasis. Adult females range from 8 to 13

millimetres in length and have a long, pin-shaped posterior, for which the worm is named. Pinworms mate by traumatic insemination - the male stabs the female with his penis - after which the male dies. They make their home in the host's intestines, but unlike many parasites they do not pass into the blood and cannot survive in other parts of the body for any length of time.

#### **Treatment**

- o Anthelmintic (anti-worm) medicines are used to kill the pinworms (not their eggs). Your doctor or nurse will likely recommend one dose of mebendazole or albendazole. These are available over-the-counter and by prescription.
- More than one household member is likely to be infected, so the entire household is often treated. Another dose is usually repeated after 2 weeks. This treats worms that hatched since the first treatment.
- o To control the eggs:
- Clean toilet seats daily
- Keep fingernails short and clean
- Wash all bed linens twice a week
- Wash hands before meals and after using the toilet
- Avoid scratching the infected area around the anus. This can contaminate your fingers and everything else that you touch.
- Keep your hands and fingers away from your nose and mouth unless they are freshly washed. Be extra careful while family members are being treated for pinworms.

#### **Outlook (Prognosis)**

o Pinworm infection is fully treatable.

### When to Contact a Medical Professional

- Call for an appointment with your health care provider if:
- You or your child has symptoms of pinworm infection
- You have seen pinworms on your child

#### **Prevention**

 Wash hands after using the bathroom and before preparing food. Wash bedding and underclothing frequently, especially those of any affected family members.

#### **Supplements FOR PINWORMS**

- Garlic Chop finely, or crush, 4 cloves of garlic and mix with 1 glass of liquid (water, juice or milk) and drinkdaily for three weeks. Chinese Rhubarb Root Mix 1 teaspoon of rhubarb powder to 1 cup of water. Then, bring to boil and simmer at a reduced heat for 10 minutes. Add a little honey to sweeten. Mugwort An infusion of the dried leaves and flowers helps expel pinworms. Infusion: 1 ounce dried herb or fresh leaves to 1 pint boiling water. Steep for 5-10 minutes then strain and sip slowly.
- o #Teucrium. [Teucr]

The remedy for ascarides or pin-worms; there is much irritation caused by them in the rectum. Hughes prefers the tincture or lower dilutions, saying that it rarely fails in this condition. Another remedy for pin-worms is Sinapis nigra.

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- The information in this monograph is intended for informational purposes only, and is meant to help users better understand health concerns. Information is based on review of scientific research data, historical practice patterns, and clinical experience. This information should not be interpreted as specific medical advice. Users should consult with a qualified healthcare provider for specific questions regarding therapies, diagnosis and/or health conditions, prior to making therapeutic decisions.

#### References

Dent AE, Kazura JW. Enterobiasis (Enterobius Vermicularis). In: Kliegman RM, Behrman RE, Jenson HB, Stanton BF, eds. Nelson Textbook of Pediatrics . 19th ed. Philadelphia, Pa: Saunders Elsevier; 2011:chap 286.

## **Whipworm**

http://www.nytimes.com/health/guides/disease/whipworm-infection/overview.html?module=Search&mabReward=relbias%3Aw

- $\circ$  Whipworm infection: Whipworm infection is an infection of the large intestine with a type of roundworm.
- Symptoms of whipworms are bloody stools
- O Whipworm infection: Symptoms range from mild to severe. Common symptoms include abdominal pain and diarrhea. A severe infection may cause bloody diarrhea, iron-deficiency anemia and,

sometimes, rectal prolapse, which occurs when the rectum slips down outside the anus.

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#### **Alternative Names:**

#### Trichuriasis

 Whipworm infection occurs when a parasitic worm, called Trichocephalus trichiura, infects the large intestine. Once inside the body, the eggs hatch and attach themselves to the wall of the large intestine.

#### Causes

- Whipworm infection is caused by the roundworm, Trichuris trichiura. It is a common infection that mainly affects children.
- Children may become infected if they swallow soil contaminated with whipworm eggs. When the eggs hatch inside the body, the whipworm sticks inside the wall of the large intestine.

#### **Prevalence**

- Whipworms are found around the world, especially in countries with warm and humid weather.
- Whipworm infections are estimated at several hundred million worldwide.
- Whipworm is found throughout the world, especially in countries with warm, humid climates. Some outbreaks have been traced to contaminated vegetables (believed to be due to soil contamination.
- Humans become infected after they consume foods that are contaminated with soil that contains whipworm eggs.
- This infection primarily affects children. This infection often infects and is spread to the entire family.
- Whipworms are named for their whip-like appearance, and are transmitted through the ingestion of larvae and eggs on infected foods like fruits and vegetables.

### Whipworms

These insidious creatures actually inject a digestive fluid that converts the colon tissue into liquid that the worms sucks up. Dr. Norman Stoll, a former worm expert at the Rockefeller Institute for Medical Research, estimated that the roundworm infects about 644 million people in the world. This was in the 1940s and there are no

doubt a lot more people infected with roundworm now! Dr. Brooks believes nutritional deficiencies are seen in heavy roundworm infections.

### **Symptoms**

0	Symptoms range from mild to severe. Sometimes, there are
no sy	mptoms. A severe infection may cause:

- Bloody diarrhea
- Iron-deficiency anemia
- Fecal incontinence (during sleep)
- Rectal prolapse
- Hemorrhage can occur when worms penetrate the intestinal wall and bacterial infections usually follow.
- o Can grow to 1 to 2 inches length.
- o After infection, mucus will be passed from the anus due to the increased **mucoidal** secretions produced by the injured intestine.
- An infestation will also most likely cause diarrhea.
- Whipworms are known to cause **anemia** and diarrhea.
- o In addition, children with large infestations have been noted to suffer mental retardation.

#### **Exams and Tests**

 A stool ova and parasites exam reveals the presence of whipworm eggs

#### **Treatment**

 Mebendazole taken by mouth for 3 days is commonly prescribed when the infection causes symptoms. Albendazole or

Ivermectin may sometimes be used.

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### (Prognosis)

Full recovery is expected with treatment.

#### When to Contact a Medical Professional

 Seek medical attention if you or your child develop bloody diarrhea. In addition to whipworm, there are many other infections and illnesses that can cause similar symptoms.

#### **Prevention**

- o Improved facilities for feces disposal have decreased the incidence of whipworm.
- Always wash your hands before handling food. Thoroughly washing food may also help prevent this condition

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   Mandell GL, Bennett JE, Dolin R, eds. Principles and Practice of Infectious Diseases . 7th ed. Philadelphia, Pa: Elsevier Churchill Livingstone; 2009:chap 287.
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### **Fluke**

#### **Blood fluke**

#### **Flatworm**

http://www.nytimes.com/health/guides/disease/schistosomiasis/overview.html

- o **Flatworm blood fluke** (schistosoma mansoni, S. haematobium, S. japonicum) Flukes are flatworms from a few millimetres to 2.5 cm in length (1 inch) that tend to live in tissues other than the lumen of the digestive tract. The most common human parasitic flukes are liver, blood and lung flukes.
- o Blood flukes live in the final host over a span of several years. Eggs produce minute granulomata and scars in organs where they lodge or are deposited. The symptoms experienced by humans depend on the location of the parasite in the human host.

### Clinical stages.

o Generally, three clinical stages of infection can be delineated. In the first phase, there is an itchy erythematous or petechial rash at the sites of penetrates of the cercariae, the free-swimming larvae; this lasts from two to five days. In the second clinical phase, four to five weeks later, the symptoms are primarily allergic and of varied

severity. There may be fever, urticaria, malaise, respiratory symptoms, and the liver and the spleen may be temporarily enlarged. In two to eight weeks, the person becomes asymptomatic. The final clinical phase, which can occur six months to several years after infection, is characterized by diarrhea, dysentery, intestinal tumors, portal hypertension, and hepatic insufficiency.

It should be noted that these parasites could live for many years. Some people with light infections are asymptomatic and never have signs or symptoms of parasitic disease. (2) Course and severity. Remember that the course and severity of the disease caused by the blood fluke depends on three elements:(a) The number of adult worms present.(b) The number of eggs produced.(c)The sites of the lesions the worms cause.(3) Intestinal blood flukes. Early symptoms are diarrhea, dysentery, and abdominal pain. Symptoms of later stages include anorexia, weight loss, polypoid intestinal tumors. Signs of portal hypertension and hepatic insufficiency are also in evidence.(4) Urinary blood flukes. In the urinary form of the disease, the patient may have the signs/symptoms of ureteral and renal damage ending in fatal uremia. The infected human might die of bladder carcinoma many years after being infected. (5) Oriental blood flukes. Symptoms are similar to those of intestinal blood flukes, but the disease is more serious. Complications that come from chronic infection can be fatal.

#### **Exams and Tests**

- The doctor or nurse may/will examine you. Tests that may be done include:
- Antibody test to check for signs of infection
- Biopsy of tissue
- Complete blood count (CBC) to check for signs of anemia
- Eosinophil count to measure the number of certain white blood cells
- Kidney function tests
- Liver function tests
- Stool examination to look for parasite eggs
- Urinalysis to look for parasite eggs

#### Treatment.

- The drug of choice for all three species of blood fluke--intestinal blood fluke, urinary blood fluke, and Oriental blood fluke--is praziquantel.
- Alternative drugs are as follows:(1)Intestinal blood fluke disease--oxamniquine
- Around 200 million people are infected with **blood flukes** of which some 20 million are severely ill and many more just show symptoms. The impact of flukes can play havoc with economic development, especially in tropical developing countries.
- Schistosomiasis is at present the world's second most prevalent infectious disease - second only to malaria. Schistosoma, is responsible for the second most common parasitic infection of humans: bilharzia. Liver fluke infections have been reported in Europe and the United States, as well as the Middle East, China, Japan and Africa. Lung fluke infections are common in the Far East, South-east Asia, Africa, Central and South America, Indonesia and the Pacific Islands.
- You get a schistosoma infection through contact with contaminated water. The parasite in its infective stages is called a cercaria. It swims freely in open bodies of water.
- On contact with humans, the parasite burrows into the skin, matures into another stage (schistosomula), then migrates to the lungs and liver, where it matures into the adult form.
- The adult worm then migrates to its preferred body part, depending on its species. These areas include the bladder, rectum, intestines, liver, portal venous system (the veins that carry blood from the intestines to liver), spleen, and lungs.

### **Symptoms**

- $\circ$  Symptoms vary with the species of worm and the phase of infection.
- Heavy infestation (many parasites) may cause fever, chills,
   lymph node enlargement, and liver and spleen enlargement.
- Initial invasion of the skin may cause itching and a rash (swimmer's itch). In this condition, the schistosome is destroyed within the skin.
- Intestinal symptoms include abdominal pain and diarrhea (which may be bloody).
- o Urinary symptoms may include frequent urination, painful urination (dysuria), and blood in the urine (hematuria).

### **Types**

- There are several types of fluke blood flukes, liver flukes, oriental lung flukes, sheep liver flukes and intestinal flukes, tissue flukes, zoonotic flukes, lancet flukes and a host of others. The differences vary in where and how a person has been infected and where and how they will damage the system internally.
- Liver, oriental lung, sheep liver, and intestinal flukes are transmitted via food; blood flukes are transmitted in swimming or bathing water. If living in the UK, Europe, North America or Canada it is likely that any fluke contamination will have been picked up on holiday in somewhere tropical. Areas such as Africa (especially Egypt), South America, the Caribbean and China are worst affected. They can also be brought right to the table in food imported from abroad. They have complex life cycles whereby eggs are passed through urine or faeces into fresh water, where the larva must pass though an intermediate snail host before a larval stage emerges that can infect a mammalian host by directly penetrating the skin. The parasite causes inflammation (swelling) and damage to organs, particularly the liver. The adult worms can persist in their human host for decades, and may not cause any symptoms for years. They leave the host in faeces and spend part of their lifecycle in a snail host.
- Passing <u>rolled-up tomato skins</u>, pointed <u>red leaf</u> shapes, or brownish small <u>cinnamon sticks</u> are symptoms of <u>flukes</u> (see <u>Fasciolopsis</u> <u>Buski</u>).

The worms produce eggs (up to 25,000 eggs per worm per

### Fasciolopsis Buski

Fasciolopsis buski, also called giant intestinal fluke, is a duodenal digenetic		
trematode, of the Fasciolidae family. It was described for the first time by Busk in the duodenum of a sailor in 1843 in London and its life cycle in humans was first described by Barlow in 1925. Mainly found in Southeast Asia and is found in water chesnuts, bamboo, water caltrops etc.		
o Fasciolopsis		

buski lives in the small intestine

day) that are passed in the host's feces.

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The trematode flatworm parasite *Fasciolopsis buski* is the largest intestinal fluke of humans. Fasciolopsis buski is found in Asia and the Indian subcontinent, especially in areas where humans raise pigs and consume freshwater plants.

### **Pathology**

- More worms, more disease- usually asymptomatic 0
- Large number of worms attaching to mucosa bleeding, inflammation and ulceration
- Diarrhea (foul smelling greenish-yellow stools), abdominal pain, intestinal obstruction, edema
- Toxic products from worms may be absorbed and cause toxemia
- Death is rare

discharged into the intestine and stool. Eggs become embryonated in water and release miracidia, which invade a suitable snail intermediate host. In the snail, the parasites undergo several developmental stages: sporocyst, redia, and cercaria. The cercariae are released from the snail and encyst as metacercariae on aquatic plants. The mammalian hosts become infected by ingestingmetacercariae o

Immature eggs are

n the aquatic plants. After ingestion, the metacercariae excyst in the duodenum and attach to the intestinal wall. There they develop into adult flukes (20 to 75 mm by 8 to 20 mm) in approximately 3 months, attached to the intestinal wall of the mammalian hosts (humans and pigs). The adults have a life span of about one year. Mas-Coma et al. (2005) reviewed the biology, diagnosis, treatment, and epidemiology of fasciolopsiasis (Mas-Coma et al. 2005 and references therein).

> Fasciolopsis buski is one of the largest digenean trematode flatworms infecting humans. Pigs are the only important reservoir.

#### **Presentation**

- Individuals with moderate infection present with occasional loose stools, some weight loss, malaise and generalised abdominal pain.
   Severe infection:
   Initially causes diarrhoea alternating with constipation and hunger.
- As the infection progresses and the worm burden increases, oedema of the face, abdominal wall, and lower limbs occurs, as well as ascites and generalised abdominal pain.
- Anorexia, nausea, and vomiting are also common.
- The diarrhoea persists, becoming greenish-yellow and very smelly.
- Patients may develop weakness, with grey and harsh skin and oedema of the face and lower extremities.
- In people infected with H. heterophyes, embolisation of the eggs can lead to myocarditis, chronic heart failure, and/or cerebral emboli.

### Management

- Symptoms and infection may resolve without therapy, although treatment can be provided with <u>praziquantel</u> or <u>triclabendazole.[1]</u>
- Treatment may also include antispasmodics to relieve abdominal pain, and iron supplements to treat anaemia (which may require transfusions in severe cases).

#### **Prognosis**

- Light infections may resolve spontaneously within one year, even without treatment. However the prognosis may be grave in patients with heavy infection.
- o Immunocompromised hosts may be at an increased risk of complications. For example, Gymnophalloides seoi worms were found to penetrate into colonic lymphoid tissue in a patient with colon cancer.

- Death from infection is rare and usually occurs only in persons with a heavy worm burden who present with severe cachexia and prostration. Other intercurrent infection may also cause death.
- In cases of infection with H. heterophyes or M. yokogawai, death may occur after embolisation of the eggs to the heart or brain.
   Embolisation to the brain and spinal cord can also cause focal neurological disease.

Furst T, Sayasone S, Odermatt P, et al; Manifestation, diagnosis, and management of foodborne trematodiasis. BMJ. 2012 Jun 26;344:e4093. doi: 10.1136/bmj.e4093.

Fasciolopsiasis; DPDx, Centers for Disease Control and Prevention

Heterophyiasis; DPDx, Centers for Disease Control and Prevention

Metagonimiasis; DPDx, Centers for Disease Control and Prevention

Echinostomiasis; DPDx, Centers for Disease Control and Prevention

#### Lancet Flukes

- Lancet Flukes (dicrocoelium dendriticum) Start life as eggs found in the dung of cattle. This dung is fed upon by snails which allow the eggs to enter the snail's intestine. Once inside, the eggs hatch and burrow into the digestive gland of the snail. The flukes reproduce in this gland and are expelled from the snail in slime trails. Ants happen upon the slime and consume it as a source of moisture thus taking the new flukes into their system. Once inside, the parasite shows an interesting tactic. By controlling nerve centres of the ant they are able to control its behaviour. When the sun sets and temperatures drop the ant is compelled to attach itself to a tall blade of grass by its mandibles. Here it waits to be ingested by some grazing animal. If the ant survives the night the sun prompts it to return to the colony and live its life normally, until the next night. Flukes living within ants are eaten by cattle while grazing. The flukes will enter the digestive system and force their way into the cow's liver, where they will grow to adults capable of producing eggs. These eggs are then expelled in the dung of cattle to begin their life cycle. To evidence the presence of flukes a blood test may reveal an elevated white blood cell (eosiniphil) count, although this only occurs early in the infection process.
- <u>Eosiniphil count</u> is a general indicator that the host may have more than one type of parasites such as roundworms, hookworms,

Toxocara, pinworms, and/or Strongyloides. Even certain drugs will raise the white blood cell count. Many doctors dismiss the elevated eosiniphil count as being caused by allergies, pneumonia, or an infection, not realizing that the primary allergen is a parasite itself.

- Urine testing may also evidence blood fluke eggs in the urine sediment, but few doctors test for this.
- Since the female adult fluke lays her eggs around the anal area, the application of clear tape to the anal area first thing in the morning will recover the eggs of the blood fluke as well as those from beef and pork **tapeworm**. Symptoms: fever, aching, cough, diarrhea, swollen glands, lethargy, urinary problems, liver problems, hepatitis, abdominal pain, liver abscesses, fibrosis, diabetes, diarrhea, and vomiting and jaundice in the case of the liver flukes.

### **Outlook (Prognosis)**

 Treatment before significant damage or severe complications occur usually produces good results.

### **Possible Complications**

- Bladder cancer
- Chronic kidney failure
- Chronic liver damage and an enlarged spleen
- Colon (large intestine) inflammation with bloody diarrhea
- Kidney and bladder obstruction
- Pulmonary hypertension
- Repeated blood infections can occur, because bacteria can enter the bloodstream through an irritated colon
- Right-sided heart failure
- Seizures

#### When to Contact a Medical Professional

- o Call your health care provider if you develop symptoms of schistosomiasis, especially if you have traveled to a tropical or subtropical area where the disease is known to exist or if you have been exposed to contaminated or suspect bodies of water.
- TREATMENT FOR FLATWORM BLOOD FLUKE includes:

- Black seed, cloves, gentian root, fennel seeds, green black walnut husks, hyssop leaves, oregano, peppermint leaves, and pumpkin seeds, thyme leaves.
- Parasite Cleanse, Remove solvent
   source and remove them from the body with vitamin C (3 grams) and vitamin B2 (300mg) Dr. Clark
- Artemisia Absinthum, Eugenia carypphyllata, juglans nigra

#### **Prevention**

- Avoid swimming or bathing in contaminated or potentially contaminated water
- Avoid bodies of water of unknown safety
- Snails are an intermediate host for the parasite. Getting rid of snails in bodies of water used by humans would help prevent infection.

#### References

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- Maguire JH. Trematodes (schistosomes and other flukes).
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### Ringworm

Ringworm or Tinea is a typically mild disease of the skin, scalp or nails caused by a fungus. Personal hygiene, supported by availability of adequate quantities of water are important preventive measures. Ringworm rash on small boy

#### Skin:

On the scalp, ringworm begins in the form of a pimple or sore, which then spreads into a ring shape. Hair becomes brittle, breaking easily and falling out, leaving bald spots on the scalp. On the body, ringworm may first appear as red or pink, flat or slightly raised, patches on the skin. The circular sores may be dry or scaly crusted or moist. As the sores become larger, the central area clears, leaving a ring of infected tissue around the clear area. Infection in the nails usually begins at the site of an injured nail and may spread to the other nails. Infected nails become thick, pitted, grooved and

abnormal in shape and colour. A bioresonance practioner with a parasite database can identify if tinea or other parasites are present under the skin.

### **Symptoms**

Ringworm:
 Athlete's foot (tinea pedis)
 may cause burning or itching
 anywhere on the feet.
 Symptoms are usually most
 noticeable in between the

toes. Patients may also develop itchy blisters, cracked or peeling skin, dry skin, or toenails that are thick, crumbly, discolored, or pulling away from the nail bed.

- Jock itch (tinea cruris) typically causes the skin near the genitals, buttocks, and inner thighs to become red and itchy. The skin may also start to peel or crack.
- o Patients with ringworm of the scalp (tinea capitis) typically develop a circle-shaped rash on the skin that is swollen. The skin may be scaly and itchy. There may be small black dots on the scalp. Patients may lose small patches of hair. However, the hair will grow back once treatment is started.
- Ringworm of the skin (tinea corporis) causes a circular red rash to form on the skin. This rash typically develops in patches and may be raised. The skin may be also be scaly and flakey.

### **Others**

#### White worms

They come in all sizes from tiny pinworms to those that look like spaghetti or angel hair pasta.

#### Red worms

These look just like earthworms. They exude from the colon wrapped in balls. They reach up to 6 inches in length.

#### Inch worms

These are thick (pencil size), black and bumpy and about 2 inches long.

#### Black worms

These are 1-12 inches in length and leave the colon wrapped in "yellow acid water" mixed together. They nest deep, impacted in the colon wall.

#### "Little Fish"

About 1/2'' long, these are fish-type parasites with heads and tails. They swim out of the colon in "schools".

#### "Fuzz Balls"

Round parasites with fur-like growth on them. About 1/4 - 3/4" in diameter, yellow in color.

### "Spiders"

Look like a spider and are colored brown, often 1" long.

### Stickpin worms"

One inch long and a head like a pea, perfectly round. Small ones are white, adults are black.

### **Histoplasmosis:**

The most common form of histoplasmosis causes no symptoms. However, the parasite is present in the body for the rest of his/her life.

Patients who are symptomatic usually develop symptoms three to 17 days after exposure. Common symptoms include fever, headache, dry cough, chills, chest pain, weight loss, and sweats.

When a fungal infection enters the bloodstream and affects multiple body tissues and organs, the condition is often life threatening.

Histoplasmosis may spread to virtually any part of the body, including the liver, bone marrow, eyes, skin, adrenal glands, and/or intestinal tract. When this happens, the condition is called disseminated histoplasmosis. Symptoms vary depending on which organs are infected.

### **Complications**

1. Disseminated histoplasmosis may lead to severe and fatal complications, including pneumonia, pericarditis, meningitis, and/or adrenal insufficiency.

### **Culicoides (Ceratopogonidae)**

M. Ozzardi & Perstans -> Filariasis