The Hypoglycemic Health Association

NEWSLETTER

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The NEWSLETTER of the Hypoglycemic Health Association is distributed to members of the Association and to Health Professionals with an interest in nutritional medicine and clinical ecology.

The recent upheaval about the closure of hospital beds around the state brings home the precarious nature of medical services in our community. Unless we ourselves take some personal responsibility in maintaining the best possible health through lifestyle changes, nutrition, exercise, avoidance of pollution - which means actively participating in environmental politics - we will become entirely dependent on a faltering medical service for health matters. A recent Swedish study showed that about one third of licensed physicians displayed a positive attitude toward "alternative medical modalities" and that "there was general dissatisfaction with the scientific approach of modern academic medicine", (**Scan J Soc Med 20(1),** 55-60 (1992)). The trend towards complementary medicine is growing. One in four patients already consult an alternative medical practitioner apart from their doctor. Unfortunately only the wealthy can afford this luxury. The cream of the medical dollar flows to the upper end of medical practice, the high tech specialists. In the mean time, family doctors are forced to rely more on "prescription writing" to squeeze out a living. Thus they are less likely to embrace modern nutritional medicine. It is one of our aims to inform readers about medical choices. You can support our Association by forwarding your $15 subscriptions ($10 for pensioners) to the Association. Your expiry date is shown on the address label. APPLICATION FORM has been printed on the last page. If you want to preserve the Newsletter, please take a photocopy of the form and send it in to the Association.

Our Next Public Meeting will be at 2 PM
on Saturday, the 7 September, 1996
at the YWCA,
2 Wentworth Ave, Sydney and
our guest speaker is

Dr Joachim Fluhrer

who will be speaking
on the subject of

"OxygenTherapy"

Dr Joachim Fluhrer is a registered medical practitioner and is the principal and owner of his practice at Manly, which is called **"Sydney Natural Medical Centre"**. Dr Fluhrer's main interest lies in the area of chronic illness. The practice is shared with other professionals in General, Nutritional and Environmental Medicine, Allergies, Acupuncture, Osteopathy, Homeopathy, Support for cancer patients, Chelation Therapy, Immune Therapies and, General and Biological Dentistry. The Institute caters for chronic degenerative diseases, chronic fatigue syndrome, chronic toxicity, mercury-amalgam toxicity, immune disorders, attention deficit disorders and, of course OXYGEN THERAPY which will be Dr Fluhrer's topic at the next meeting.

Tribute to

DON PEMBERTON

23 May, 1928 - 15 July, 1996

By Jur Plesman

It is with great personal sadness that I find myself writing this obituary about my friend **DON PEMBERTON** who suddenly died from a heart attack on Monday night, the 15 June 1996. He is to be remembered for his great contributions to the knowledge of clinical nutrition and toxicology. Don is, of course, a friend of many people and each one of us who came in contact with him has a special story to tell.

My acquaintance with Don can be traced to the early days when our paths crossed in the "Sydney Push", a miscellaneous group of university students, artists, non-conformists, intellectuals, and "Bohemians" centred around the "Lincoln" coffee shop in Rowe Street, Sydney in the early 1950's. I mention this, because Don never lost his "less than total respect for authority", which was a characteristic feature of members of the Push. In other words, he was primarily a lateral thinker, and would not accept a proposition just because it came from an authority.

I met Don again about twenty years ago, when he joined my counselling group for drug-addicts and alcoholics and became a co-facilitator for many years. Don was a "recovered alcoholic" and therefore a valuable member of the group. He was a lecturer of nutritional biochemistry and toxicology. At that time, I myself was disillusioned with the "psychology only" approach to the treatment of drug-addiction. My wife had bought me a book by Roger Williams "Nutrition Against Disease" and I was impressed by Alexander Schauss' "Diet, Crime and Delinquency". I was ready and looking for a change but horrified of biochemistry, which turned me off. Biochemist friends were unable to explain to me, what happens when a person is addicted to heroin. But Don could. He was not only a professional lecturer of biochemistry, he was an excellent teacher with huge amounts of patience. He began to educate me and he was able to pass on his love of biochemistry to me. Soon we would have long sessions after group sessions with Don writing biochemical reactions on the white board with incredible speed demonstrating how enzymes transform molecules into other molecules, how energy was produced in the citric acid cycle, how adrenaline causes the liver to release glucose from glycogen and so on. Our interest was in hypoglycemia as a factor in drug-addiction and other behavioural problems. At this stage, a third partner joined us and this was Dr George Samra, another lateral thinker, who happened to have a surgery in the same clinic, where our groups were being conducted. He was able to medically prove the link between hypoglycemia and drug-addiction, but additionally, to fix the parameters for diagnosing hypoglycemia.

There has always been a special chemistry between Don and myself. Whenever we met we would leave with a new vista on the world. Don, who was a devout Christian, and I, as a humanist, shared a great love of "nature". Thus he was a excellent advocate in defence of the environment. He, voluntarily, took up the cudgels on behalf of various environmental groups against many influential companies who brought in "overseas" chemical experts to argue their case. ("The Organo Chlorine Debate" Hypo Newsletter, Dec 90, 5 is an example)

Don was an excellent public speaker. He had a knack of assessing at what level his audience was operating and then he would lift them up to a level where he could make his point clear. I have seen testimonies of his students, who wrote to him when he retired as a lecturer, and they all expressed genuine love and respect for Don. He was a man without pretence and without malice.

Don Pemberton is well known for his dry sense of humour. I still remember his story of how he "stuttered" his way out of court on a traffic offence, when it would take him about 15 minutes to say: "Y y y y y y your your your M M M Ma Ma Ma Majest majest Majesty, M m m y my K K K car....." when the Magistrate interrupted him and said: "Get this man out of my court!" He had us in tears of laughter. Or when he tried to stop a police officer from smelling alcohol on his breath, by leading him around and around his car and asking him whether his tyres were alright. Or how, as an industrial chemist working for a company, he made chicken soup by reducing the chicken meat to just a few molecules with a cocktail of other chemicals to make it taste like chicken-soup. I remember sessions of belly-laughters in the group. This would relieve a lot of tension and teach us to take life with a grain of salt. Yet, Don never belittled another person. He was able to make fun of situations and himself.

He was totally committed to improving the lot of his fellow humans, especially the underdog. He was a great believer in education for natural health.

Don was a good listener. Clients loved to talk to him as he had a rich experience of life, unlimited compassion, but he also believed that somewhere along the line we have to take responsibility for our happiness. In the group we taught how another person could become your 'other persona'. This is a psycho-therapeutic means by which you imagine to have a conversation with your 'other persona' to deal with a problem. Don will remain my 'other persona' and his spirit will live on in my mind giving me a direction when I am lost in the woods. Don has left a trail of 'other personas' in many people's hearts and it is going to be a long time, before we will give him up.

Thank you Don for meeting us.

Any opinion expressed in this Newsletter does not necessarily reflect the views of the Association.

Previous Copies of the Hypoglycemic Newsletter

Back issues of the Hypoglycemic Newsletters are available at the NSW State Library, Macquarie Street, Sydney. They are filed under NQ616.466006/1 in the General Reference Library

ADVERTISING MATERIALS appearing in or with this Newsletter does not necessarily imply any recommendation by the Hypoglycemic Health Association.

Books for sale at the meeting

Jur Plesman: **GETTING OFF THE HOOK**

This book is also available in most public libraries

Sue Litchfield: **SUE'S COOKBOOK**

Dr George Samra's book

The Hypoglycemic Connection

(now out of print) is also available in public libraries.

Contributions of articles by members and practitioners are very welcome. The Editor is interested in meeting any person aspiring to research natural medicine and contribute articles as a sub-editor to this Newsletter.

The Newcastle branch of the Association are still meeting with the assistance of Bev Cook. They meet on the last Saturday of each month beginning 1.30 pm to 3.30 pm at the Hillsborough Primary School. Enter the school from the Waratah Avenue. For further information ring Mrs. Bev Cook at 049-59-4369.

Organise local meetings

If any member would like to organise meetings in their local area or meet other members, we can help by advertising your name and phone number in this Newsletter.

Entrance fee at meetings

Because of increase in costs the Committee has decided to charge an entrance fee of $2 per person or $3 per family at our public meetings.

Donations for raffle

One way of increasing our income is by way of raffles. If any member has anything to donate towards the raffle, please contact Dr George Samra's surgery at 19 Princes Highway, Kogarah, Phone 553-0084.

Lynette Wright won the Lucky Door Prize and **Patricka Sheiles** won the Raffle Prize at our last public meeting on 1 June, 1996.

Committee members

The Association is in need of your support and ask members to help out with sending the Newsletter to our members. We also need committee members and if you are interested please contact Dr George Samra's surgery at **553-0084.**

Research into illnesses

Members who are interested to have an informative article written on a particular illness or disease, should contact the Editor, c/- PO Box 8, Sylvania Southgate NSW. The editor is willing to research literature on the illness and report in the newsletter with the known traditional and complementary treatment . Or he may refer any medical question to an expert in the field. However, it must be understood clearly that treatment remains the responsibility of your doctor or health practitioner and that such articles are only designed to provide some enlightenment to the patient or to complement his/her discussion of the illness with the professional practitioner. The Association does not take any responsibility for any self-diagnosis or self-treatment undertaken by the reader on the basis of anything published in this Newsletter.

Down's Syndrome:

Inevitable mental disorder or treatable metabolic disorder?

A ray of hope!

By Dr Robyn Cosford

Down's syndrome is the commonest cause of mental retardation and it affects many families. I am sure that most people will know someone with Down's syndrome. For many years it has been regarded as a syndrome in which mental retardation and poor health is inevitable. However, this need not necessarily be so I would like to present two cases to illustrate.

Danny

My first case concerns a young man called Danny. He is a 28 year old man who presented moderately retarded, with limited speech and single word- or two-word sentences. He did not use any prepositions. He lived in a sheltered home facilities and worked in a sheltered workshop. We deal with a lot of behavioural and learning difficulties in our clinic, and it was on this basis that he was brought in to see me.

He was very agitated and was inclined to be violent; he would kick and bite. He was also very aggressive and confrontational and he had crying fits and would sob. He had been on traditional medications such as Melleril, a phenothiazine used in psychotic disorders and a very sedative drug, and on Valium another tranquilliser. He did not appear to have much benefit from these drugs.

He also suffered from sleep disturbances. He had to perform night-time rituals and they had to be done in the right order. Unless these were done, he would not go to bed. Consequently, he did not get to bed until at least 11:30 pm and sometimes 3:30 am. Thus he had variable waking times in the morning. He had also self-comforting rituals, often observed among disturbed children, such as rocking behaviours or swinging from side to side. He simply could not sit still.

Jo

The other case was a young man called Jo. He was a 25 year old and was born looking apparently normal. However, he had gradually developed widely spaced teeth, flat feet, a small head and he vomited a lot as a baby. No doubt this was due to food sensitivities. He became developmentally delayed and later was profoundly mentally-retarded with a low IQ. He had uncontrollable tempers, he was hyperkinetic, restless, over-active and he too rocked a lot as self-comforting behaviour. He had occasional seizures. He was found to have an abnormal EEG (electroencephalogram) and other neurological abnormalities.

Danny's treatment

I treated Danny as I tend to treat many behaviourally disordered patients, given that we were dealing with a 28 year old man, who went out and was uncontrollable in terms of diet and nutrition when out. As far as possible we eliminated sugars, preservatives and colours from his foods. We steered him onto whole foods, again as far as possible.

There are certain nutrients that are indicated in children with behaviour disorders. It would be difficult to mention them all as these apply to individuals with what Roger Williams would call "biochemical individuality". Among these are vitamin B6 (pyridoxine), magnesium, zinc, and certain amino acids, particularly phenylalanine, glutamine and adenosine. I also use tissue salts and homoeopathic remedies (Bach flowers and strammonium & veratrum), aroma therapy oils, such as lavender oil, and herbs passion flower, hops and valerian to sleep. We also introduced, wherever possible, behavioural interventions in the home situation. Over a 12 months period he gradually improved, although I did not then see him for a space of 8 months.

He re-appeared, this time with his mother and carer, in my surgery. She told me that he had now no agitation, he was now calm and controlled. There was no shouting, fighting, or violence. He had ceased to be aggressive or confrontational. There were no crying fits and other symptoms of what we call being 'emotionally labile'. His sleeping pattern improved. The next bit of information surprised me. Danny was seeing his older brother, whom he had not seen for some 18 months, and after a while his brother said to his mother: "Mum, what have you done? He is different." In addition, his vocabulary had increased and he had begun using prepositions

So here we have a man who had been operating at an intellectual level far below his potential for many years, who now had learned to speak. Yet, he had not received remedial teaching. He was using prepositions that he had never used before. He was communicating better and his function in his workplace improved and he was moved to a new position. I had not expected this to happen. I was rather excited to see the effects of a change in nutrition. Unfortunately, I had not done a earlier EEG to compare the results, but I was convinced that his learning and brain functions had improved.

Danny was a typical Down's Syndrome case.

Jo was a slightly different case. Jo's treatment was a very simple nutritional intervention, removing a single amino acid from his diet; *phenylalanine.* Jo was what is called a phenylketonuric. A Guthrie test[1] at birth now detects phenylketonuria (PKU). This is an inborn metabolic disorder caused by the absence or a deficiency of the enzyme, *phenylalanine hydroxylase,* responsible for the conversion of phenylalanine (an amino acid) into tyrosine[2]. It is an autosomal genetic disorder. People can be carriers and only partially be affected. The incidence is 1 in 10-20,000 and fortunately is rarely seen in adults now. Accumulation of phenylalanine is toxic to the brain tissue. If undetected at birth the patient may among other symptoms become mentally retarded. Treatment consists of a diet low in phenylalanine. It is important to realise that PKU inhibits pathways of an important neurotransmitter, *serotonin,* and this affects behaviour and moods. It also interferes with the Gamma-aminobutyric acid (GABA) pathway. This amino acid has a neurotransmitter-like activity, is found in the brain (and other organs) and keeps a person calm. It is an inhibitory chemical substance which, if it is disturbed, will result in problems with aggression. It is important that phenylketonuria be detected at or near birth, for if left untreated loss of 5 IQ points for every 10 weeks delay in diagnosis will result in mental retardation. Little benefits from treatment may be expected after 3 years of age.

Phenylketonuria and Down's syndrome therefore have similarity in that nutritional intervention can influence brain function.

Down's syndrome

Down's syndrome is the commonest single cause of mental retardation, and is caused by complete or partial excess of chromosome 21 (trisomy 21). In 89-90 per cent of cases this is caused by nondysjunction, where chromosomes don't separate properly during cell division. One cell line has 47 chromosomes (instead of 46), and this line replicates; the other, 45 chromosomes, and this cell line dies. Rarely, Down's syndrome may be inherited (6%) or resulting from nondysjunction later in cell division (Mosaicism - about 2%).

There are quite a few predisposing factors for this to occur.

The incidence of Down's syndrome increases with maternal age; that is the older the woman giving birth the greater the incidence. One in 600 births give rise to Down's syndrome in women below the age of 30. The incidence increases to one in 40 births by women above 45 years of age.

Recent research suggests that this is possibly due to oxidant damage to the ovaries, causing nondysjunction. Studies show that mothers who are 35 years old or younger at the time when they give birth to a Down's syndrome child have five times the risk of developing dementia themselves. This indicates increased oxidative damage in the body, as the link between oxidative damage and dementia is becoming increasingly apparent.

It is interesting to note that antioxidant treatment has a well-established role in the prevention of dementia.

Other risk factors include;

- infection prior to conception
- abdominal X-rays prior to conception
- OC (contraceptive pill) use within three months of conception
- coeliac disease in either parent
- diabetes; thyroid disease; Chronic fatigue syndrome; SLE in either parent

Many of these risk factors could also be operating via oxidative damage to the ovaries.

Genetic defects

A chromosome is a package of a multiple genes.

Chromosome 21 provides a genetic blueprint for various proteins, enzymes and other metabolic substances. Therefore, a duplication of this chromosome as in Down's syndrome will result in the disturbance of numerous metabolic pathways, and an excess of some gene products.

In phenylketonuria, in comparison, there is a lack of a single enzyme which results in the disturbance in one pathway in particular, and so a single corrective factor is adequate.

Superoxide dismutase enzyme (SOD)

Chromosome 21 encodes for several enzymes as now known. Perhaps the most important of these is the copper-zinc dependent enzyme*Superoxide Dismutase enzyme* (SOD).

This enzyme functions in the body to protect cells from harmful singlet oxygen and hydroxyl radicals[3], which are produced during normal cellular metabolism. Under the influence of the enzyme, the hydroxyl radicals and singlet oxygen[4] are converted to hydrogen peroxide, a very reactive chemical. Under normal circumstances, the selenium-dependent enzyme, *glutathione peroxidase,* neutralises the hydrogen peroxide. However, in Down's syndrome, there is an excess of SOD production and thus an excess production of hydrogen peroxide and a deficiency of zinc. The body tries to compensate for this by increasing the production of glutathione peroxidase with resultant deficiencies of glutathione and selenium. This imbalance in hydrogen peroxide, glutathione, selenium and zinc, may well be the mechanism behind the oxidative damage and early aging and increased presenile dementia seen in Down's syndrome.

Purine metabolism

Also encoded for on chromosome 21 are enzymes necessary for correct function of the *purine metabolism* pathway.

Purines[5] are the nitrogenous end products in the digestion of certain proteins. An inability to metabolize and excrete purines may develop into gout.

There is evidence that among Down's syndrome patients there is an increased incidence of gout, which can then be treated by avoiding foods containing high levels of purines and encouraging a purine-free diet, and increasing levels of folic acid to inhibit excess synthesis of uric acid.

Interferon

Chromosome 21 also encodes for the alpha/beta interferon receptor.

Interferon is a natural cellular protein formed when cells are exposed to a virus. It induces the production of 'translation inhibitory protein' (TIP) in noninfected cells. TIP blocks translation of viral RNA, thus giving other cells protection against both the original

and other viruses. Interferon is species specific meaning that interferon produced in one species of animals cannot be used by others. It has been trialled in the treatment of such diseases as hepatitis and also in cancer. Interferon is an important component of the immune system. The genetic blueprint in Chromosome 21 for the receptor is found to be disturbed in cases of Down's syndrome. Receptor sites for interferon on leucocytes may interfere with maturation and lead to failure in immunological development. Down's syndrome is recognised as having chronic and recurrent illness, and having 10 times the occurrence of leukaemia compared to the general population.

Amino acid metabolism

Another important enzyme which it has been discovered is encoded for in chromosome 21 is an enzyme involved in amino acid metabolism. Amino acids are the building blocks of proteins and hence affect the manufacture of various enzymes. The particular enzyme is *Cystathionine beta synthetase (CBS)*, responsible for the production of cysteine[6] as follows:[7]

(Homocysteine + Serine) —> Cystathionine
—>[CBS]—> **Cysteine** + alpha-ketobutyrate

Cysteine is a nonessential amino acid, but nutritionally important since it spares the essential amino acid *methionine*[8]. Cysteine is also important in that it is the precursor of *taurine*[9] in the formation of coenzyme A from the vitamin pantothenic acid (B5).

The result of this disturbance in CBS is that there is an increase in certain amino acids and a decrease in others. This has been confirmed in studies with Down's syndrome, in which it has been found, that there are deficiencies in serine[10], methionine and taurine.

Physical characteristics of Down's Syndrome

Characteristics are not always shown at birth as maternal metabolism tends to compensate.

Typically they have slow growth, they are short with a small head and have dry skin.

They show an abnormal face, with a flat back of the head (flat occiput) and usually they have slit eyes (epicanthic folds), pinpoint, white or light yellow spots on the iris of the eye (Brushfields spots). They may show middle ear abnormalities and abnormal skin formation around the fingers often indicative of chromosomal anomalies (abnormal dermatoglyphics). They may also display abnormal dental growth.

Patterns of dysfunction

Down's syndrome patients are typically retarded with an average IQ of about 55 untreated. Fortunately, this can be improved upon through nutrition and intensive learning and socialisation programmes.

Thyroid dysfunction is quite common among Down's syndrome patients. There is a marked dysfunction of the immune system, and so they suffer recurrent illnesses. As mentioned, studies show that individuals with Down's syndrome have ten times the incidence of leukaemia.

Significantly, they have severe gut dysfunction, marked by frequent constipation. They often lack the necessary digestive enzymes to absorb their food. They invariable have a lactose intolerance.

They are typically emotionally labile, meaning they suffer from mood swings from extreme joy to friendliness, stubbornness and aggression.

They have an increased incidence of diabetes.

Down's syndrome patients usually show signs of presenile dementia or Alzheimer's disease, typically in their 40's. Other signs of a metabolic disorders are increased incidence of gout and *hyperuricaemia* or presence of high levels of uric acid in blood.

Other biochemical disturbances currently known

As pointed out chromosome 21 encodes for various enzymes and amino acids, many of which are defective in Down's syndrome.

- The same chromosome 21 controls the behaviour of various amino acids, the building blocks of protein. Often we find a disturbance in the *glutamine/glutamate* pathways. Although these amino acids are normally produced in the body, in Down's syndrome patients they may be dysfunctional. Glutamate and glutamine are intimately involved in brain processes by converting toxic ammonia into harmless substances via the urea cycle to be excreted in the urine. By supplementing Down's syndrome patients with glutamine or glutamic acid we may be able to supply the brain with energy and improve intellectual capacity.
- Similarly, a disturbance in the *tryptophan* metabolism may cause a decrease in available *serotonin*, an important neurotransmitter affecting a person's emotions.
- It is known that Down's syndrome patients are low in *insulin growth factor no 1* (IGF1). It is responsible for making growth hormones. During baby-hood the equivalent is *insulin growth factor 2* (IGF2) which should become IGF1 in adulthood. The enzymes involved are zinc dependent and zinc supplementation has been shown in studies to normalise IGF1.
- Disturbance in the carbohydrate metabolism contributes to an increased incidence of *diabetes* among persons with Down's syndrome.
- Closely related to this is a reduced levels of *pyridoxine* (B6) which affect protein, carbohydrate metabolism and production of *serotonin* from tryptophan.
- *Vitamin B12* is found to be generally deficient, which may be due to problems of absorption involving absence of the *intrinsic factor* from the stomach, *pH levels* in the gut, or interference by other medical drugs. Vitamin B12 and *folic acid* are essential in heme production, which form part of red blood cells. B12 is essential in the correct production of DNA, without which cells cannot mature.
- *Digestive enzyme deficiencies* are common among Down's syndrome patients and this results in general malabsorption of nutrients. Of particular importance is *lactose intolerance* so milk products should be avoided. Antibodies have been found to bile ducts (75%), parietal cells (62%), and smooth muscles.
- It is common to find *thyroid dysfunction* among Down syndrome patients. They usually have a small thyroid gland. Again auto antibodies are found to the thyroid. The consequence is that they under-produce thyroxine (T4), an important hormone involved with the rate of metabolism, that will affect many bodily parameters such as temperature, metabolic rate, growth hormones, skeletal maturation, regulation of fat, protein and carbohydrate metabolism, synthesis of enzymes, muscle tone. This explains why the skin tends to be dry, why they have a lot of constipation and problems with weight. It is interesting to note that among others thyroxine also stimulates the conversion of beta-carotene to vitamin A in the liver.[11] Zinc has been shown to normalise *thyroid stimulating hormone (*TSH) and T3 levels of thyroid function.
- Vitamin B1 *thiamine* has been found to be particularly low. This vitamin is necessary for correct carbohydrate metabolism and for nerve function.

Interhemispheric communication in the brain

Studies show that Down's syndrome patients have a disturbance in their brain function, particularly in communication between one side of the brain and the other. This is something that is treatable. Patients seem to have a particular sensitivity to a drug called *atropine* (used for relaxing smooth muscles in the intestines and other organs). This drug has the effect of blocking receptor sites for *acetylcholine*, a neurotransmitter mediating synaptic activity of the nervous system and muscles. This disturbance of acetylcholine in the brain is of great interest.

As indicated earlier Down's syndrome patients are prone to develop dementia. It has been found that they have difficulties binding aluminium, which has been implicated in the development of dementia. A study has shown a defective gallium-transferrin binding in the brains of Down's syndrome patients with Alzheimer's disease. In Alzheimer's patients this mechanism is thought to be involved in the accumulation of aluminium.

Management

It is important to realize that treating a Down's syndrome child is treating a whole person, and not simply "pill taking". We have to look at the social side of the illness, family support and behavioural management. There

is a need for boundaries of acceptable behaviour and discipline.

Specific features which need to be attended to are:

- **Eyes/vision** problems should be looked at by a developmental optometrist. 50 percent of Down's syndrome children have eye and vision problems in the form of refractive errors, cataracts, keratoconus (noninflammatory protrusion of the central part of the cornea), amblyopia (defective vision, approaching blindness, usually as a result of strabismus or the inability of both eyes to focus on the same object). They may need spectacles or to do eye exercises.
- **Heart** condition, which are usually picked up at birth. 30-60 percent of Down's syndrome children have some form of cardiac condition.
- Together with structural abnormalities in the face, **ears and hearing** impairment is common. 65 percent of Down's syndrome children have conductive hearing loss. It is important to correct hearing losses if we are dealing with learning disabilities and/or speech impediments.
- Orthopaedic problems are very common because of lax ligaments and poor muscle tone. 10-20 percent of Down's syndrome children have what is called an *atlanto-axial instability*, involving the atlas and axis of the vertebral column, and affecting the whole spinal part of the body, and related to this
- a lot can be done by regular *exercise regimes* promoting coordination, muscle tone and stimulation.

Nutrition

It goes without saying that we have to provide nutritional support to various parts of the body, to the thyroid gland, the digestive system, carbohydrate metabolism and so on. The earlier this regime is started the more beneficial it will be to the child. If the condition is diagnosed early enough, the child does not need to develop Down's syndrome, or does not have to develop mental retardation and an abnormal face. Treatment should include:

- **Exclusion diets:** diets should exclude food sensitivities which could be detected by a cytotoxic food testing, diets should exclude *gluten* in the case of coeliac disease, or milk products to avoid *lactose intolerance, and sugar, artificial colourings, flavours and preservatives.*
- avoid **aluminium** cooking utensils
- **Add** green leafy vegetables, olive cooking oil, flaxseed oil on salad and vegetables, ginger, onion and garlic.
- **Vitamin/mineral support** is essential to maintain a proper carbohydrate metabolism. The B-complex vitamins and the minerals magnesium and chromium are an integral part of treatment.
  Specific vitamins and minerals include:
  1) Pyridoxine (B6) involved in protein metabolism and tryptophan conversion to serotonin, 2) folic acid for its methylation action together with vitamin B12, necessary for DNA synthesis, 3) zinc, a coenzyme in many enzymatic processes in the body, important to the immune system, and to correct insulin factor-1 (IGF-1) abnormality.
- **Thyroid support** includes the checking of function of the thyroid gland at 6 monthly intervals. The diet should be supplemented with kelp, for its iodine source, and tyrosine and where necessary thyroxine. Adequate zinc levels are necessary.
- **Amino acid correction** can be implemented after a urinary amino acid profile. The important amino acids supplements should include serine, methionine, glutamine/glutamate, tryptophan, taurine, tyrosine and others as indicated. Studies have shown that a serotonin enhancing diet can decrease aggression by 95 percent.
  Recent studies have shown that supplementation with L-carnitine produced significant improvement in mental status in Down's syndrome patients, but not other categories of mental patients.[12]
- **Phosphatidyl- choline plus vitamin B5** will help the interspheric imbalance in the brain and promote neural transmission. Lecithin is rich in choline and so is choline bitartrate obtainable in health food stores. Additionally, choline supplementation will improve sleeping abnormalities.
- Supplementation of **antioxidants** such as vitamins A, C, E, Beta-carotene and including minerals zinc, copper, manganese, selenium, glutathione (essential in the superoxide dismutase). This is to protect against the oxidant damage and imbalance caused by the excess SOD enzyme and consequent deficiencies of zinc, selenium and glutathione.

Conclusion

A Californian paediatrician, Dr Jack Warner, has treated over 700 Down's syndrome children in "the Warner House" following the principles outlined in this article. They employed a team of experts in their fields such as a physical therapist, developmental optometrist and behavioural therapist and with their combined approach were able to produce Down's syndrome children, able to attend normal schools. This, of course, depended on the age at which children commenced treatment. Early treatment resulted in Down's syndrome children having growth rates equivalent to normal, with an IQ in the normal range, general reduction in chronic illnesses and significantly, normalisation of facial features. There is a record of one child, age 5, obtaining an IQ score of 140!

Down's syndrome stems from chromosomal defects resulting in abnormal cellular biochemistry. Some of these effects, at least, are reversible, others are preventable and others can be moderated.

Therefore, Down's syndrome does not mean inevitable mental retardation: it is a metabolic disorder that can be treated to an extent that patients can function normally in society.

Clinical nutrition provides, indeed, a ray of hope!

Footnotes

1) In a Guthrie test a small amount of blood is obtained and placed in a medium with a strain of *Bacillus subtilis*, a bacterium that cannot grow without phenylalanine. It is a screening test for phenylketonuria used to detect the abnormal presence of phenylalanine metabolites in the blood.
2) Tyrosine is a forerunner of thyroxine, a hormone excreted by the thyroid gland which among other controls the metabolic rate.
3) Hydroxyl radicals - a particular reactive, damaging type of free radical, formed when superoxide radicals react with hydrogen peroxide. They are thought to be the damaging agent to joint membranes in arthritis.
4) Singlet oxygen is an activated energetic reactive form of oxygen which can damage important macromolecules such as DNA
5) Purines are also present in many medications and other substances such as caffeine, theophylline (an alkaloid found in tea and related to caffeine, also used in some broncho-dilators), diuretics and muscle relaxants. Foods high in purines are anchovies and sardines, sweetbread, liver, kidneys and other organ meats, legumes and poulrtry. Foods lowest in purine includes eggs, fruits, cheese, nuts, sugar, gelatin and vegetable other than legumes.
6) Formed from methionine, cysteine is easily transformed into usable cystine, together with methionine are major sulphur containing amino acids. It is a component of the glucose tolerance factor (with glycine, glutamic acid, niacin and chromium). Cystine or cysteine are essential for utilization of B6. Production of cysteine from methionine is prevented in chronic disease. Protects against effects of alcohol and smoking. Involved in hair strength. Involved in insulin production. Flexibility of skin through attack on free radicals.
7) Stryer,L(1988),**Biochemistry,**WH Freeman & Co NY,page 584
8) Methionine: An essential amino acid which among others control the overload of fat in your liver. Methionine's sulphur content helps the liver to manufacture lecithin, a valuable fat-fighting substance. Methionine acts as an antioxidant, deactivating free radicals. It also 'chelate' or grab hold of metallic substances and heavy metals (lead, mercury & cadmium) that might otherwise cause toxicity. It is a methyl donator in B12 metabolism. Gives rise to taurine and cysteine and cystine. Detoxifies excess histamine (important in histadelic schizophrenia). Essential for selenium bioavailability, an essential co-enzyme in glutathione peroxidase, which attacks free radicals.
9) Synthesised from methionine (or cysteine in liver), taurine helps to nourish brain cells. It works with choline to maintain neurotransmitters that promote thinking. It is often used as an addition in the treatment of epilepsy. Inhibited by estriadol. Helps overcome obstruction in the flow of bile (cholestasis). Taurine in serum rises with low zinc serum, and results in low taurine levels in the brain, thereby increasing the chances of 'fits'.
10) Serine - A nonessential amino acid (meaning the body can synthesise it) found in many proteins in the body. It is also the precursor of glycine, which in turn is involved with porphyrins and heme proteins.
11) Nutrition Search Inc (1979)**, Nutrition almanac,** 14
12) Carnitine is synthesised in the liver from lysine and methionine, vitamin C being essential for its conversion. Has a major role in transferring fatty acids into cells where they are used as energy sources.

Further reading next page---->

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HYPOGLYCEMIA AND ESSENTIAL FATTY ACIDS

By Jur Plesman

There is very little in the scientific literature dealing with the relationship between essential fatty acids and hypoglycemia. One reason seems to be that the term *"hypoglycemic syndrome"* is not recognized by orthodox medicine, and so does not merit any special attention. Had we used the term *"prediabetic syndrome"*, the story might have been different, as the latter term may have been more digestible to traditional medicine. The other reason seems to be that 'alternative medicine' does not attract the investment dollar to fund research, although such research would save the government many Medicare dollars. For the sake of this article I will use the *hypoglycemic syndrome* and *prediabetic syndrome* interchangeably.

The hypoglycemic syndrome

In summary, this syndrome may have the following features:

- People with a hypoglycemic syndrome (also described as 'Hyperinsulinism') may present any one or more of the following symptoms: addicted to sugar, depression, suffer migraine headache, irritability, bad tempers, uncontrollable violence, mood swings, difficulty coping with stress, impulsive behaviour, lack of attention, loss of memory, confusion, fatigue, depression and insomnia, nervousness, excess perspiration, bedwetting among children, apathy and lack of motivation.
- Patients usually show abnormal concentrations of blood glucose levels, fluctuating between extreme highs and lows. Dr George Samra[1] claims that in a four hour Glucose Tolerance Test (GTT) a drop in glucose levels exceeding 2.8 mmol/L (50mg per 100ml) in any one hour or over 1.9 mmol/L (35mg per 100ml) in any half an hour would be diagnostic of the hypoglycemic syndrome. There may also be, what is called a "flat-curve" hypoglycemia, which is believed to be due to an underactive thyroid gland, needing further medical attention.
- Insulin resistance in diabetes has been associated with a specific genetic marker, *the insulin gene*, situated on the short arm of chromosome 11. One form of the gene, called the UU allele, and present in some people, shows a delayed response to oral glucose and reduced glucose tolerance, which may render them susceptible to type II diabetes *(Non insulin dependent diabetes mellitus)* and by implication hypoglycemia.[2] Genetic influences should

be considered in the right context and will be further discussed below under the "Pottenger's cats".

- It is known that excess alcohol consumption can bring about a hypoglycemic reaction[3] and that this is often accompanied by a bout of violence. In fact, this connection was established by the University of Helsinki showing that violent behaviour among criminals were related to a sudden drop in blood sugar levels.[4] The brain accounts for some 60 per cent of the utilization of glucose by the whole body in the resting state.[5] Alcohol impairs the brain's ability to use glucose as fuel.[6] It seems that when the brain is deprived of glucose - its normal source of fuel - the central nervous system activates the adrenal medulla to produce adrenaline which in turn acts upon the adrenaline receptors of liver cells to release glycogen in the form of glucose and thereby supply the brain with energy.[7] The blood concentration of adrenaline almost increases a thousandfold in seconds or minutes in the fight or flee reaction.[8] It would seem that the varied symptoms in the hypoglycemic syndrome are in fact a manifestation of excess adrenaline.
- The primary cause of the hypoglycemic syndrome is said to be excess consumption of *sucrose*, a disaccharide of glucose + fructose. The glucose fraction is absorbed immediately into the blood stream. Insulin from the pancreas controls the blood glucose levels by converting it to glycogen, which is then stored in liver, and muscle tissues. The brain does not store glycogen. It is high consumption of sucrose that causes the pancreas to overproduce insulin, which ultimately may lead to diabetes. In that case, either the body fails to produce enough insulin or insulin receptor sites do not respond. The fructose fraction, on the other hand, is transported to the liver where it is converted to *glyceraldehyde 3-phosphate* + *dihydroxyacetone phosphate* which can then be called upon to convert to *pyruvate* to enter the citric acid cycle. This is another source for energy.[9] It is a cruel quirk of history that chemists were able to produce commercial quantities of sucrose and NOT fructose, that led to a conservative estimate of one per cent of the western population becoming diabetic[10]. The number of *prediabetic* persons must necessarily be much higher, especially among those living in third world living conditions. It must be understood that the "hypoglycemic syndrome" share symptoms with others such as heavy metal intoxication.
- Thus the first rule in the treatment of the hypoglycemic syndrome is avoidance of *sucrose* as is the case with diabetes. As blood glucose levels fluctuate every three hours, it is important to have regular three hourly high protein snacks. However, people should be cautioned against excess high protein diet from animal sources as this may place a premium on the urea cycle or transformation of nitrogenous substances into urine. In diabetics, and presumably hypoglycemics, the transformation of glucogenic amino acids (proteins) into glucose is a very active process and proceeds at a much higher rate than in normal people.[11] Too much protein can facilitate diabetic nephropathy.[12]
  The hypoglycemic diet should be accompanied with supplements of various vitamins and minerals involved in the breakdown of glucose into energy, especially zinc, vitamin C, B-complex vitamins, B1 (thiamine). I have argued elsewhere, that fructose used as a sweetener is preferable to others on the market. However, immoderate amounts of fructose can lead to higher levels of triglycerides. Excessive consumption of aspartame (NutraSweet) with its content of phenylalanine may be deleterious, especially among hyperactive children.[13]
- Monitoring of glycosylated haemoglobin (GHb/Hb A1c) concentration by your doctor allows you to measure the average blood glucose level over the previous several weeks, indicating that levels have been properly controlled. Your doctor could also prescribe you Keto-Diastix kit to measure glucose and ketone levels in the urine.
- Another feature of the hypoglycemic syndrome, which is shared with Type 2 diabetes, is a tendency to be overweight. We need to look at this further to find the link between hypoglycemia and essential fatty acids.

Obesity

It was reported in a British newspaper that nearly half the male population of Britain is overweight, and *two-thirds* have a high risk of heart disease.[14]

How often have we heard doctors tell us, that we are overweight and that all we need to do is exercise and eat less. A simple prescription for obesity! The problem is it does not work! The patient feels guilty when he finds himself unable to comply with the doctor's instruction, not realizing that the doctor is often not aware of the metabolic aspects of obesity. At least, the health practitioner has an excuse to blame his patient for his own ignorance. This is not unlike the tactic used when treating drug addiction or alcoholism. The fact is that almost 95 percent of dieters regain all of their lost weight within 12 to 36 months with a vengeance. They gain even more weight! Very low calories diets (VLCR) only seem to work if some sort of 'life-style' changes in the form of psychological programme is adhered to[15],[16]. The obese population as a whole does not show an elevated incidence of psychopathology. There appears to be no global personality traits or profiles that are associated with obesity.[17] Some of the dangers of strict dieting are overlooked. With weight loss, the muscle mass of the heart and left ventricle decrease. These changes may affect abnormalities and during dieting any copper, potassium and magnesium deficiencies may play an important part in promoting an electrically unstable heart. Stress could provoke arrhythmias (deviation in heart-beats) in a subset of the obese population on very low calories diets.[18] During strict dieting the risk of gallstone formation is markedly increased.[19] Some authors even suggest not to diet at all![20] This may be the other extreme. Restriction of calorie intake coupled with exercise may be beneficial for a host of other health reasons, apart from aiming at weight reduction. But strict dieting for the express purpose of weight reduction should be under close supervision by a doctor. When obesity is due to a metabolic disorder "dieting" may not only be useless, but harmful both for physical and psychological reasons.

Thyroxine and protein synthesis

Many obese individuals may have hypothyroidism because of dysfunction of the thyroid gland. Thyroxine stimulates increased consumption of glucose, fatty acids and other molecules. It stimulates RNA and protein synthesis.[21] As may be expected, people with subclinical hypothyroidism have an abnormally basal metabolic rate and experience weight gain and lethargy. If one's temperature is consistently below 36.5 C or 97.8 F before getting out of bed in the morning, one should suspect some form of hypothyroidism, which should be discussed with your doctor. Recent studies have shown that one enzyme in thyroid hormone metabolism is dependent on selenium.[22] Hypothyroidism was a major theme of Barnes.[23] Thyroxine is considered to be a anabolic hormone like growth hormone. We can help the thyroid gland produce thyroxine by supplementation with phenylalanine, tyrosine, kelp (source of iodine), zinc, selenium and vitamin B5, but only under your doctor's supervision, because of the complex nature of thyroid synthesis. Hypothyroid patients do not convert beta-carotene to vitamin A efficiently in the liver and should take preformed vitamin A.[24]

Growth hormone and obesity

In obese subjects growth hormone secretion from the anterior pituitary gland has been found to be clearly impaired. The reason for this is not clear. Impaired secretion causes obesity. The release of growth hormone (GH) is regulated by two hypothalamic hormones: *growth hormone releasing hormone* (GHRH) which stimulates the release of growth hormone and *somatostatin* (SS), which inhibits that release. These hormones are in turn controlled by feed-back mechanisms (for instance by a hormone *Insulin-like Growth Factor-1* or IGF-1). The exact role of IGF-1 in obesity has been question[25]. The secretion of GH follows a circadian ("about a day") pattern, increasing during sleep and decreasing during periods of wakefulness. Hence, could exercise before bedtime be more beneficial than in the morning? Growth hormone secretion is stimulated by an increase of plasma concentrations of amino acids (thus by a high protein meal) and by a decrease in plasma glucose concentrations (hypoglycemic dip). Thus during periods of high blood sugar levels secretion of GH is inhibited and could perhaps stimulate the

inhibitory somatostatin hormone, leading to obesity.

It is interesting that a drug called *pyridostigmine* - a drug used in myasthenia gravis - has markedly increased GH response to GHRH.[26] This is a nerve-stimulating cholinergic drug that interferes with the enzyme cholinesterase that breaks down *acetylcholine.* Acetylcholine is a neurotransmitter widely distributed throughout the body, which facilitates communication between the nervous system and skeletal muscles. Thus an increase in acetylcholine seems to be related to an increase in growth hormone. Perhaps supplementation with choline/inositol, or phosphatidylcholine found in lecithin could help increase the growth hormone and thereby improve obesity? It is noted that Ross Trattler mentions lecithin as one agent that may reduce weight.[27]

Diabetics produce too much insulin

Another reason why diabetics and prediabetics tend to put on weight is that excessive concentrations of insulin dramatically increases the rate of the conversion of carbohydrates into *triacylglycerols* (via pyruvate —> citric acid cycle), the very substance that make up our fat layers (adipose).[28]

It has also been suggested that in Type II diabetes, the pancreas often makes too much insulin because insulin resistance reduces its effectiveness. Apparently, the body lowers the number of insulin receptors in the face of too much insulin, making insulin less effective.[29] Thus, here we have a circular causal link between diabetes and obesity. Which came first the chicken or the egg? There appears to be no simple solution to the problem of obesity. What other factors play a role?

Essential fatty acids have been overlooked

A clue comes from some studies that show babies' appetites increase enormously when they are fed formulas deficient in essential fatty acids and return to normal on essential fatty acid-rich breast milk.[30] Human breast milk is a rich source of *gamma-linolenic acid* (GLA) which is the immediate forerunner of prostaglandin series no 1. Significantly, these prostaglandins *prevent obesity and enhance the effects* of insulin among many other benefits.

I refer to table **Figure 1** which shows the biochemical pathways of *essential fatty acids* (EFAs), its food sources, and vitamins and minerals requirements at different enzymatic steps. The productions of the three kind of prostaglandins shown are: *1-prostaglandins* (PGE1), *2-prostaglandins* (PGE2) and *3-prostaglandins* (PGE3) which are of central interest.

Prostaglandins are described as short-lived *local* hormones, because in contrast to the more uniform actions of global hormones, such as adrenalin, insulin and glucagon, their actions vary from one type of cell to another in the local area of the body.[31]

Table 1 shows the effects of prostaglandins and attention is drawn to beneficial effects of PGE1 and PGE3, as compared with PGE2.

A diet high in dairy products, animal meat, eggs and cow's milk will tend to over-produce *arachidonic acid* (AA) which are then converted to PGE2. Table 1 shows the results of an excess intake of arachidonic acid (AA) and production of PGE2, such as depressed immunity, increased clotting, constrict arteries leading to hypertension, and promote inflammation. It is obvious from the literature that the standard diet of eggs, meat and milk produces an imbalance in PGE synthesis and that we should help the body produce prostaglandins series 1 & 3, by increasing vegetable, fruit, and fish sources of our food.

Prostaglandins control a multitude of essential functions in the body in relation to such seemingly unrelated disorders as heart attack, high blood pressure, mood elevation, arthritis, menstrual cramps, allergies, eczema, asthma, migraine headaches, weight reduction, atherosclerosis, fertility, glaucoma and multiple sclerosis. Prostaglandins cannot be stored and tissues must make them as the need arises. It would be impossible here to cover all the functions of prostaglandins. The link between hypoglycemia and allergies, dermatitis, atherosclerosis, blood pressure, headaches, migraines, asthma and so on becomes obvious to those who have been diagnosed to have a hypoglycemic syndrome.

Balance between linoleic (N-6) and linolenic (N-3) fatty acids

The source of prostaglandins are *essential fatty acids*, such as linoleic (N-6), alpha-linolenic (N-3) and arachidonic acid. Intake of essential fatty acids, both N-6 and N-3, need to be in balance. The body can produce important derivatives from essential fatty acids: docosahexaenoic acid (DHA) from N-3 EFAs[32], (fish oils), which is prominent in the brain, the retina and spermatozoa; arachidonic acid from a predominant N-6 fatty acid in most other tissues. If there is a high ratio of linoleic acid (N-6) to alpha-linolenic acid (N-3) in the diet, DHA is depleted.

In a study with rhesus monkeys it was found that N-3 deficiency is characterised by impaired vision, abnormal electro-retinograms and polydipsia (excessive thirst). Provision of N-3 fatty acids is essential during pregnancy, lactation and infancy and both linolenic (N-3 FAs) and DHA are found in human milk. "These studies support the conclusion that there should be adequate amounts of both N-3 and N-6 fatty acids in the diet throughout life and that their ratio is of great importance".[33]

Christie claims that the ideal ratio between N-6 and N-3 families of essential fatty acids is about 6:1. In fact, N-3 supplementation alone when N-6 derivatives are low actually *brings on* deficiency symptoms, because N-3s inhibit processing of whatever N-6 is available.[34] It would appear that this ratio differs among different communities, depending on whether their ancestors were primarily inland meat-eaters or came from fish-eating coastal areas. Thus "one man's meat is another man's poison" if you use the wrong ratio.

A defective Delta-6-desaturase may be the cause of the diabetes

Linoleic acid is the main source of dietary EFAs, but before participating in prostaglandin synthesis, linoleic acid must first be converted to *gamma-linolenic acid* (GLA) by the enzyme, *delta-6-desaturase (*D6D). This metabolic pathway may be blocked in atopic eczema. The blockage may be by-passed by taking *evening primrose oil*, about ten per cent of which is gamma-linolenic acid. Other nutritional sources of GLA are black currant seed oil, borage oil and fungal oil, all of which have been used successfully in experiments to reduce blood pressure in specially bred rats.[35] Another experiment with rats showed that evening primrose oil "is the most appropriate for treatment of hypercholesterolaemia (high cholesterol). [36]

A double blind cross-over study of the effect of various doses of evening-primrose oil in 99 patients with atopic eczema showed that the preparation produced a significant clinical improvement when taken in high doses. No side-effects were noted.[37]

The *delta-6-desaturase enzyme (D6D)* is of crucial importance, as this enzyme could be defective or missing altogether in diabetic and prediabetic patients. This was proposed in some studies.[38] Here we have another reason why prediabetic people tend to be overweight as a defective or missing delta-6-desaturase cannot synthesize gamma-linolenic acid, which is the precursor to prostaglandins series 1. The latter prostaglandins are responsible for appropriate handling of lipids, including cholesterol and body fat. There is no doubt that the enzyme is a delicate one, easily knocked out of operation by many factors. Referring to **Figure 2** we find that there are many barriers along the linoleic-to-prostaglandins E1 biochemical pathway.

Agents that inhibit conversion of LA (Linoleic acids) to GLA[39],[40]

- Foods rich in saturated fats such as whole milk and certain milk proteins (peptides)
- Foods rich in cholesterol like red meat
- Trans-fatty acids[41] used in margarine and other oils
- Stress hormones such as adrenaline and cortisol
- Low levels of zinc, magnesium, vitamins B6 or B3, and vitamin C (transport mechanism may be faulty in diabetes) because of food refining
- Alcohol in excess of two glasses of wine per day or the equivalent
- Allergies and other atopic conditions like eczema
- High blood sugar in diabetes & hypoglycemics
- Lack of insulin
- Ageing
- Viral infections
- Cancer, Tuberculosis[42]
- Drugs: such as lithium[43], phenytoin, aspirin, NSAIDS and steroids and many oth-

ers
- Excess N-3 fatty acids (fish oil)

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) & PGE1

Inhibition of PG synthesis by drugs such as steroids and non-steroidal anti-inflammatory drugs is well-correlated with relief of symptoms. On the other hand, there is much evidence that steroids and NSAID do not favourably influence the long term course of most chronic inflammatory conditions in humans. Colchicine, a gout suppressant drug and very toxic, are able to selectively enhance the formation of PGE1. Horrobin states that treatment with NSAIDs will control overproduction of the 2 series prostaglandins but will have little effect on the low PGE1 production or even exaggerate the PGE1 deficiency.[44]

One sometimes wonders why doctors use a small aspirin to reduce platelet aggregation, thereby deactivating delta-6-desaturase, which helps the body produce the very prostaglandins that would prevent platelet stickiness. Perhaps by supplementing *evening primrose oil* we may be able to overcome this impasse and have the best of both worlds. This should be discussed with your doctor.

Obesity and supplements

Further investigation is required to clarify the relation between diabetes/hypoglycemia and obesity. However, victims of this syndrome can not afford to wait for scientific research to catch up and give us some more definitive answers. In the meantime, they can increase the necessary nutrients to improve their health, especially under the guidance of a health practitioner qualified in clinical nutrition.

There is some evidence that supplementation of evening primrose oil has the effects of reducing weight. When normal and obese people were given evening primrose oil in a study, almost all the active group lost weight, although one subject gained 6 kg.[45] Other studies agreed.[46]

It takes about a year to build up fatty-tissue LA levels on a high-LA diet so one needs a lot of patience to reduce weight.[47] When Dr Peter Oster and his colleagues studied a group of 650 German men they found an inverse relationship between obesity and high blood pressure, and low linoleic acid levels and vice-versa.[48] Amounts of 4 grams of EPO per day were used in these studies.

During a trial on evening primrose oil for schizophrenia at Bootham Park Hospital in York it was discovered that several patients who were more than 10% above their ideal weight lost weight while taking evening primrose oil. It had no effect on people whose weight was within 10% of their ideal body weight.[49]

Coenzyme Q10 (CoQ10) could be useful in a weight reduction programme. Half of 27 obese people have been found to have reduced CoQ10 levels and those subjects lost weight when CoQ10 was supplemented.[50]

When volunteers were given drinks containing either glucose, fructose or aspartame before a meal, those given the fructose drink consumed fewer calories and fat.[51] This is another reason for choosing fructose as a sweetener.[52]

Supplementing one's diet with the amino acids *arginine* and *ornithine* may not only help to build up the immune system but also stimulate muscle growth and the burning of excess fat and wastes.[53]

The herb, *Brindle Berry* (Garcinia cambogia) has been found to be an appetite suppressant and to inhibit the production of fats from carbohydrates. It is useful in slimming and weight control. This is due to a natural fruit acid called *hydroxycitric acid* contained in Brindle Berry, which appear to inhibit an enzyme in the production of fats. This substance can now be obtained in pure supplemental form and some of its features are: no "yo-yo" weight gain after discontinuance, no development of tolerance, safe for extended

Figure 1

Vitamins & Minerals needed for The Essential Fatty-Acid-to-Prostaglandin Pathway

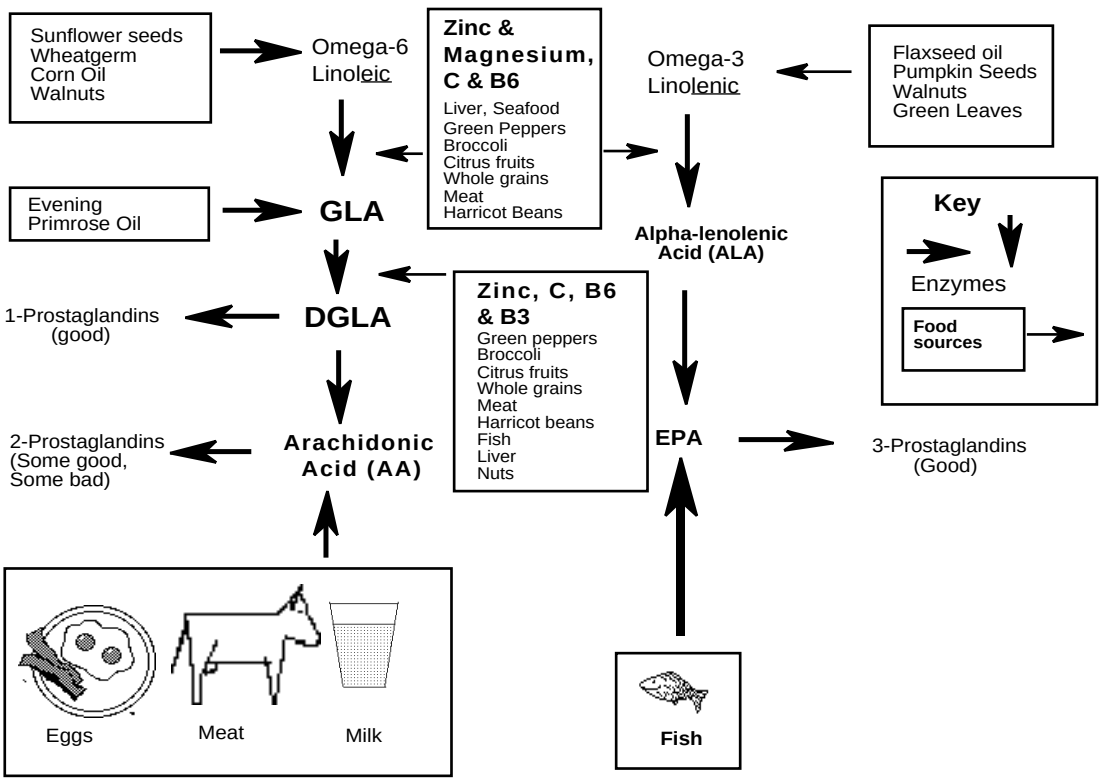
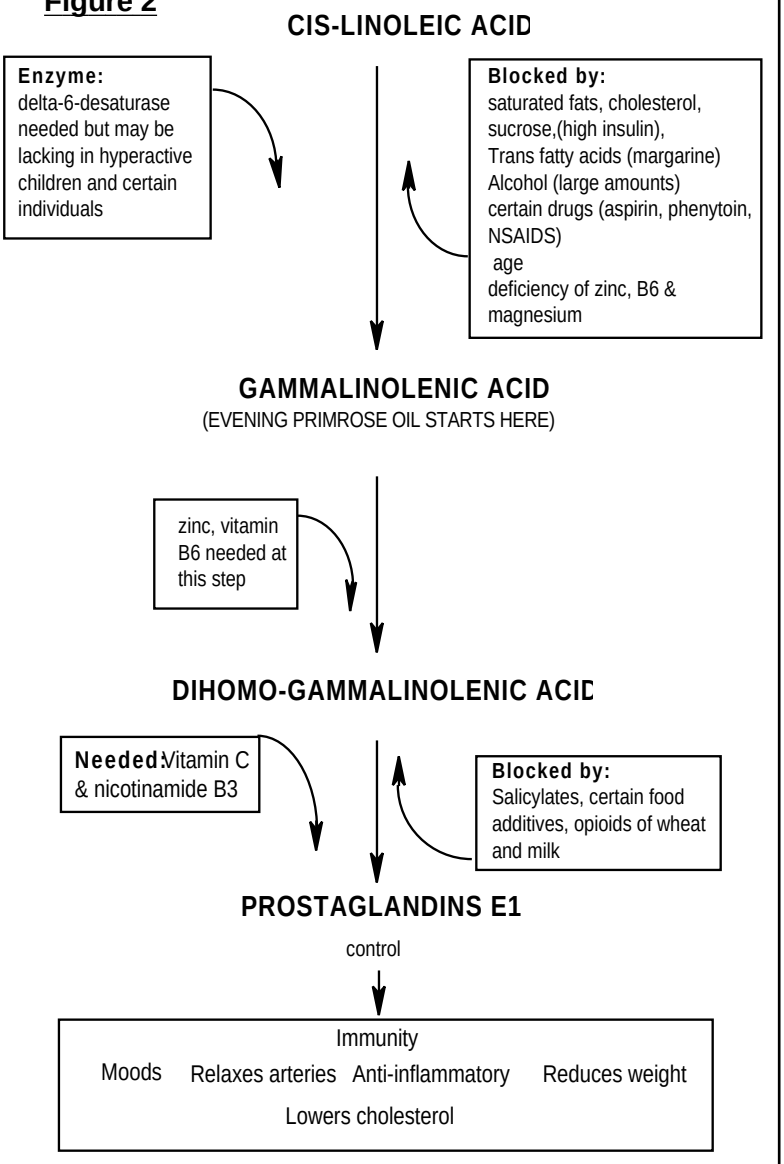


Table 1

The Effects of prostaglandins

1-Prostagladins (from GLA - Evening primrose oil)	2-Prostaglandins from AA - (meat, eggs, milk)	3-Prostaglandins from EPA (Fish)
	Depress immunity **(bad)**	Lubricate arteries
Enhance immunity	Lubricate arteries (good)	Reduce clotting
Elevate moods	Increase clotting **(bad)**	Lower Triglyceride
Reduce clotting	Constrict arteries **(bad)**	**(All good!!!)**
Relax arteries	Inflammatory **(bad)**	
Anti-inflammatory		
Lower cholesterol		
Move brown fat		
(All good!!!)		
Inhibit 2-prostagladins by slowing AA release	Inhibit 1-prostaglandin production from DGLA	Inhibit 2-prostaglandins by slowing AA production

Figure 2



consumption, does not stimulate central nervous system, decreases synthesis of fats including triglycerides and cholesterol, increased clearance of LDL cholesterol, increased production of glycogen, increased 'thermogenesis', does not cross the blood-brain barrier, safe for diabetics, long-term consumption contra-indicated only for pregnant/lactating women and young children.[54]

Other plant sources that may have a role as complementary agents in the treatment of diabetes and therefore hypoglycemia are *Fenugreek* (Trigonella foenum-graecum) and *Juniper berries* (Juniper communis). Controlled studies showed reduced blood glucose levels and weight loss.[55]

Based on studies with mice, *Glutamine* may prove useful as a supplement for patients with non-insulin dependent diabetes (NIDDM).[56]

Konjac glucomannan, a soluble fibre supplement (3.9g daily) was found to be an effective cholesterol-lowering dietary adjunct.[57]

Diabetic neuropathy

One of the many complications of diabetes is diabetic neuropathy due to nerve damage. It is usually limited to peripheral nerves. Orthodox medicine apparently has no cure. It is suspected that years of hyperglycaemia (high blood sugar levels) in non-diabetic patients, as in prediabetes, may be one causative factor.[58] Reviews of the literature show that the intake of evening primrose oil is helpful in diabetic neuropathy. It is claimed it can even reverse the nerve damage.[59]

133 patients in a double-blind, placebo-controlled trial in the UK and Finland who had diabetic nerve damage, receiving GLA from *Efamol* improved while those receiving the placebo deteriorated.[60]

Again 11 patients with diabetic neuropathy taking *Efamol* (4x500 mg morning *and* evening) showed improvements in eight aspects of nerve functions as compared with 11 receiving a placebo.[61]

GLA supplementation may improve symptoms

A defective delta-6-desaturase (D6D) goes a long way in explaining the hypoglycemic syndrome and development of diabetes. Whether we are dealing with obesity, hypertension, allergies, ineffective responsiveness to insulin, high cholesterol levels leading to atherosclerosis; they all point the finger to a deficiency of gamma-linolenic acid as the immediate forerunner of prostaglandins series 1. Therefore, we should reduce the intake of red meat, milk, eggs[62] and replace this with a fish meal, whenever possible. It would seem that a near-vegetarian diet would have much in its favour. Any change in diet should always be discussed with your medical or health practitioner.

If deficiency of essential fatty acids in general is implicated then we should increase consumption of oils of sunflower seed, wheat

germ or corn and walnuts, together with vitamin C, B6, zinc, magnesium as a source of N-6 fatty acids. Flaxseed, pumpkin seed, walnuts and their oils would supply us with *both* N-6 and N-3 fatty acids. This would be a cheap alternative to evening primrose oil.

However, if we have a defective D6D enzyme we can bypass the blockage by taking GLA directly in the form of *evening primrose oil* (about 3-4 g per day). This should always be taken with vitamin E to prevent oxidation of the oil. Vaughan Bullivant claims that *Starflower oil* (also known as Borage - Borago officinalis) is very similar to evening primrose oil, however, it is approximately two and a half times richer in GLA.[63]

Side effects of evening primrose oil

Any nutritional supplements taken in large amounts will produce side effects. However, evening primrose oil produce very few serious side effects. Christie[64] mentions the following;

- Hard cysts under the skin in those prone to cysts, due to EPO's sebum-thinning effect. Start by eating fish before starting EPO treatment
- Nausea, try small doses at first or perhaps take also hydrochloric acid
- Early morning-insomnia (avoid EPO before bedtime if you want to sleep in), probably due to raised energy levels
- Temporary euphoria
- Loose stools (diarrhoea) an advantage in constipation
- Condition in temporal lobe epilepsy (not schizophrenia) may worsen
- People prone to migraines and/or for whom alcohol triggers migraines

Pottenger's Cats

In discussing degenerate diseases and especially diabetes and the hypoglycemic syndrome, it is often claimed that the course of this disease is determined by one's genetic make-up. Thus, your age and genetic dice are loaded against you.

This gloomy and fatalistic picture does not contribute any practical solution to treatment. The emergence of genetic abnormalities is not primarily a function of the ageing population, but rather the legacy of our parents forced to live in the twentieth century, a period of near-total destruction of the natural environment of humans.

Modern technology, with crazy agricultural practices, that have produced among others the 'mad cows disease' have so altered our natural habitat that *"We have actually created an environment in which we are struggling to survive"* to quote Professor Ron Laura.[65]

Christie in his book *Food for vitality*[66] describes how Dr Frank Pottenger[67] studied cats over a ten year period. One group of cats were fed healthy natural food appropriate to their species and another group were fed scraps from Pottenger's Sanitarium. The first group grew into healthy adult cats with little signs of disease, whereas the second group develop skin disorders, thinner bones, narrow skulls and smaller jaws and crowded teeth. They would 'develop all kinds of allergies'. They would sneeze, wheeze, scratch and would be irritable, nervous and did not purr. Healthy first generation cats would produce second generation kittens with allergies, and by the third generation, the incidence is almost 100 per cent. Parasites, infections, along with heart problems, near- and far-sightedness, arthritis and hypothyroidism are common. *It would take four generations for the depleted cats to become healthy once more when they were returned to their natural diet.*

Looking at humans, we are like Pottenger's cats who after four generations of environmental destruction in the twentieth century, wonder why we loose our teeth, develop dermatitis, arthritis, grow to be cranky and aggressive, and why so many of us develop the hypoglycemic syndrome, the forerunner of diabetes.

But Pottenger's experiment also shows that genetic trends can be reversed, perhaps not in our own generation, but certainly over the next few generation by showing our children the way back to nature, in an environment where we were meant to live.

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Arachidonic acid (C20:4 n-6) Dairy products, egg yolk, milk, liver, kidney, Atlantic salmon, turkey, some in chicken, pork, beef, lamb and peanuts.

Arginine (Non-essential AA, but essential to children) avoid in herpes found in: *Almonds, bacon, brazil nuts,* buckwheat, *carob, chicken-breasts, chocolate,* cashews, coconuts, eggs, gelatine, *hazel nut,* lentils, linseed, millet, oats, cooked oatmeal, oysters, *peanuts, peanut butter,* green peas, chick peas, *pecans,* popcorn, raw cereals, raisins, rice (brown), sesame, *skim milk, Beef,* soy-beans, sunflower, *turkey, walnuts,* wheatgerm, white flour, wholewheat bread. Also found in garlic and ginseng. <u>*Supplements*</u>: take on an empty stomach)(Excess could promote herpes, kidney and liver failure). **Ornithine** derived form arginine and vice versa shares many of arginine's properties and stimulates thymus gland to produce Lymphocytes. <u>*Supplement*</u> of arginine and ornithine to be taken on empty stomach with juice or water, no proteins.

Beta carotene: Apricots, asparagus, avocados, *beetroot greens, broccoli,* Brussels sprout, rockmelon (cantaloupe), carrots, chard, chilli peppers, yellow corn, cress, pink grapefruit, greens (mustard, turnip, beet, collard etc), kale, lettuce, butterhead or Romaine lettuce, mandarin oranges, mangoes, papayas, parsley, peaches, bell peppers, plums, pumpkins, tangerines, *spinach,* squash, sweet potatoes, tomatoes and water melon. Conversion of beta carotene to vitamin A stimulated by thyroxine from the thyroid gland. (Almanac, 14)

Bioflavonoids (rutin, hesperidin): Found in skins of fruit and vegetables, The pith of Citrus fruits, orange, lemon, lime, tangerine peels, fruits, black currants, buckwheat. Green growing shoots of all plants.

Calcium: Milk and milk products, green leafy vegetables, potatoes, almonds, mushrooms, shellfish, molasses, Broccoli, Bone meal*, Dolomite*, Calcium citrate*.

Carnitine: synthesised in liver from lysine and methionine dependent on vitamin C. Not found in vegetable form. Major sources are muscle and organ meats & dairy products and richest source among plant foods avocado. (for oxidation of fatty acids). Supply of carnitine enhanced by lysine ingestion.

Choline: Egg yolk, organ meats, green leafy vegetables, Brewer's yeast, wheat germ, soybeans, fish, legumes, lecithin*.

Coenzyme Q10 (CoQ10 or Ubiquinone): Functions as oxygen transfer coenzyme. Important in heart muscle. Synthesised in body, depends on vitamin E. Available in supplemental form and used in treatment of angina pectoris and other heart problems. Rich source is yeast.

L-Cysteine and glutathione: (NEAA) (contains sulphur and inactivates free radicals) is a precursor of glutathione (a tripeptide of of glutamate, cysteine & glycine), a major antioxidant. Source: Egg, meat, dairy products and some cereals. Take with Vit C as a precaution against kidney-stone and bladder-stone formation. Caution: dangerous to diabetics.

Cystine: (NEAA)The more stable form of cysteine and formed from *methionine*.

Folic acid: dark-green leafy vegetables (spinach, chard, kale), broccoli, bean sprouts, Brussels sprouts, carrots, melon, apricot, pumpkins, avocado, dark rye flour, organ meat (liver), tortula yeast, Brewer's Yeast, root vegetables, whole grains, wheat bran, wheat germ, oysters, salmon, milk, egg yolk.

Glutamine, Glutamic Acid: Synthesized in body. Glutamine* available as a supplement synthesized from glutamate.

Inositol: Whole grains, whole wheat bread, citrus fruits, Brewer's yeast, molasses, milk, nuts, vegetables, dried lima beans, organ meat, raisins, grapefruit, lecithin*, lime, green beans (unshelled), Rockmelon (Cantaloupe), (diets high in unsprouted seeds and grains are rich in phytates, which may prevent proper absorption of many trace elements. By leavening the grains or germination prior to use phytates are eliminated and inositol is liberated into food product). Important cofactors of inositol are folic acid, B12, B6, choline, betaine, methionine.

Iodine: sealife-plant and animal-seaweed (kelp), fresh salt water fish, sea salt (many do not contain iodine), mushrooms, Irish moss (depending on iodine content of soil), seafood, dairy products, iodied salt, Morton Light Salt substitute*, Nutritional yeast*.

Methionine: (EAA) (contains sulphur and inactivates free radicals) Pork, fried liver, Brazils, Parmesan Cheese, skim Milk, flounder baked, tuna canned in oil drained, Edam Cheese, lamb, trout (Raw), Sesame Seeds, salmon canned pink, soya flour, turkey, Fish Cod (canned), pumpkin seeds, sirloin steak, chicken breasts, roast beef, roast pork, cooked prawns, cooked liver, calf liver, Cottage cheese, chicken liver, boiled eggs, roast veal, pistachios, cashews, walnuts, peanuts, chickpeas, almonds, Lima beans, yoghurt, buttermilk, brown rice.

Omega-3 or fish oil: salmon, mackerel, herring, sardines, sablefish, lake trout, fresh tuna, whitefish and anchovies. Others halibut, blue fish, rockfish, rainbow and sea trout, ocean perch, bass, hake, pollock, smelt and mullet. Among shellfish, oysters supply fair amounts of oil. *Plant sources:* soybean, legumes, rapeseed oils, walnuts chestnuts flax seed (linseed), green leafy vegetables.

Omega-6 Essential Fatty Acids: (Precursor of GLA—>DGLA—>Prostaglandins Series 1 (Good), Saffower seeds oils, Sunflower seed oils, Wheatgerm, Corn oil, Walnuts, Evening Primrose Oil (bypasses defective enzyme and contains GLA)

Phenylalanine: (EAA) Soybeans, soy products, dry skim milk, cottage cheese, fish, meat, poultry, almonds, peanuts, Brazil nuts, pecans, pumpkin seeds, sesame seeds, lima beans, chickpeas, lentils. (As supplemt* may suppress hunger if taken one hour before meals with juice or water, mood elevator)

Purines: (To be avoided in gout) Foods high in purines are anchovies and sardines, sweetbread, liver, kidneys and other organ meats, legumes and poulrtry. Foods lowest in purine includes eggs, fruits, cheese, nuts, sugar, gelatin and vegetable other than legumes.

Selenium: Important antioxidant. Tuna, herrings, Brewer's yeast, wheat germ, garlic, onions, Brazil nuts, bran, broccoli, whole grains.

Vitamin A (retinol): Fish, Liver, Eggs, Yellow fruits, vegetables, dark green fruits and vegetables, whole milk, milk products, fish-liver oil or Cod liver oil*.

Vitamin A: Made in the body from —> beta-carotene. Liver, eggs, egg yolk, yellow fruits and vegetables, dark-green fruits and vegetables, whole milk products, fish-liver oil*, in fishoils such as cod, salmon and halibut.

Vitamin B1 (Thiamine): Most vegetables, Brewer's Yeast, dried yeast, whole grains, blackstrap molasses, bran, brown rice, organ meats, meats (pork or liver), fish poultry, egg yolk, legumes, milk, peanuts, rice polish, sunflower seeds, potatoes and nuts, wheat germ, whole wheat. Enemies: Cooking, caffeine, alcohol, food-processing methods, air, water, oestrogen sulphur drugs.

Vitamin B6 (pyridoxine) Meats, whole grains, organ meats, Brewer's yeast, blackstrap molasses, milk, eggs, beef, rockmelon, cabbage, sunflower seeds, rice (whole), soya beans, canned tomatoes, Baker's yeast, wheat germ, legumes, green leafy vegetables, liver (beef), salmon, Malt extract, Flour (refined desiccated liver*, don't take more than 100 mg of B6.(see NHS205.21)

Vitamin E (Tocopherol): Almonds, Brazil nuts, cold-pressed oils, eggs, wheat germ oil, sunflower seeds (& oil), safflower oil, sesame oil, peanut oil, corn oil, hazelnuts, olive oil, organ meats, molasses, nuts, soybean oil, sweet potatoes, leafy vegetables, wholegrains, desiccated liver* (See NHS205.21)

Vitamin B12 (Cobalamin): organ meats, fish, pork, pig liver, pig kidney, fatty fish, beef, lamb, white fish, eggs, chicken, cheeses, milk and milk products. (Spirulina, nori and other sea vegetables, tofu and other soy products, fermented or otherwise, pasteuroized or not pasteurized, grains, yeast, cereals are **NOT** good sources according to Hendler (1990, 65).

Zinc: (Depending on soil) Unprocessed bran, beef steak, Brazil nuts, brown rice, cashews, almonds (nuts), cheddar cheese, chic peas, oysters, ginger root, hazelnuts, lamb chops, lentil, soya sprouts, sunflower seeds, split peas, beef liver, nonfat dry milk, egg yolk, whole wheat, rye, oats, seafood, organ meats, mushrooms, Brewer's yeast, soybeans, walnuts, wholemeal flour.

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