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.

Please note that the new venue for our next meeting is at: GLEBE NEIGHBOURHOOD CENTRE, 160 St Johns Road, GLEBE (Corner Mt Vernon St). Access by public transport is by any Parramatta Rd bus. Five minutes walk from "Foot bridge" at the Sydney University. Go to Steamer St, then past Catherine St into Mt Vernon St, until you reach St Johns Rd.

Our Next Public Meeting will be at 2 PM on Saturday, the 5 September, 1998 at ***Glebe Neighbourhood Centre*** 160 St Johns Rd (Corner Mt Vernon St) and our guest speaker is

Dr Paul Ameisen, M.B.B.S.

Dip Ac, ND, FACNEM

who will be speaking

on the subject of

"The Art of Breathing in Asthma and other diseases"

Dr Paul Ameisen has been a medical practitioner for twenty-three years with considerable experience in Obstetrics, Paediatrics, Anaesthetics, working in major hospitals in Sydney, in country areas, Boston (USA), and Baragwanath South Africa. He now has a practice at Edgecliff. As well as medical degrees, he has a *Diploma of Naturopathy*, a *Diploma Medicina Alternativa* and is a *Fellow of The Australian College of Nutritional and Environmental Medicine*. He is also the author of a book, "Every Breath You Take", describing the "Buteyko" breathing method developed by a Russian physician, Professor Buteyko. This is a technique for helping patients with asthma and other respiratory disorders managing their disease without the use of drugs. He will also discuss the relationship between hypoglycemia and asthma, which should be of great interest to our members.

Books for sale at the meeting

Jurriaan Plesman: **GETTING OFF THE HOOK**

This book is also available in most public libraries (state and university)

Sue Litchfield: **SUE'S COOKBOOK**

The Newcastle branch of the Association are still meeting with the assistance of Bev Cook. They now meet at ALL PURPOSE CENTRE, Thorn Street, TORONTO. Turn right before lights at Police Station, the Centre is on the right next to Ambulkance Station. For meeting dates and information ring Mrs. Bev Cook at 02-4950-5876.

Entrance fee at meetings

Due to diminishing income from our quarterly meetings we regrettably have to increase our fees. Entry fees for non-members will be $5.00, members $3.00 & families $5.00

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.

Other libraries holding copies are: Stanton Library, North Sydney; Leichhardt Municipal Library; The Tasmanian State Library; The Sydney University; The University of NSW, Newcastle University. The Association will provide free copies to any library upon request.

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 9553-0084.

At the last meeting on the 6th of June 1998 **Maria Keith** won the lucky door prize and **Joy Sharp** won the raffle.

Fund raising activities

We need money, ideas, donations, bequests (remember us in your will).

Local meetings at Wahroonga

Marina Bridle would like to meet other members of the Association for mutual support and discussions. Her phone number is **9487 2910**

With many thanks to **Sue Litchfield** for her generous donation of 5 cookbooks plus a beautiful hand-spun, hand-dyed and hand-knitted woollen jumper to be raffled at either the next meeting or when 200, 50 cent tickets have been sold.

Jane Graves is a supporter of the Hypoglycemic Health Association and we thank her for her donation.

Hyperinsulinism, Hypoglycaemia, Weight Problems & Diabetes

from a lecture given to the Association on 6 June 1998

By Dr Robyn Cosford

DR GEORGE SAMRA has estimated that about 3-4% of the population has hypoglycaemia. About 25 percent of Western population are affected by obesity. Recent statistics from the Australian Bureau of Statistics show that 40% of Australian males are overweight. Figures for females were a little less than that. Diabetes affects 16 million people in the USA. Insulin resistance and hyperinsulinism provides the common link between these and many other western society diseases.

Insulin and its physiology

Insulin receptor resistance is central to hyperinsulinism. *Insulin* is an anabolic hormone; that is a hormone involved with building up (conversion of simple substances to more complex compounds) in contrast to catabolic hormones, which break down complex substances. It is released from the Islets *of Langerhans* in the pancreas in response to a rise in blood glucose after ingestion of a meal. It functions to get that glucose into the cells for further metabolism as a major source of energy, and to put glucose, blood fats and amino acids into cells for storage as fats and protein. More insulin is released when we eat food with a high *glycaemic index* about which we will talk a little later. We produce less insulin when we have a regular exercise pattern. Insulin stimulates growth and stimulates insulin growth factor 1.

Insulin inhibits certain catabolic processes such as the breakdown of liver and muscle glycogen into glucose, or the breakdown of fatty acids into glucose. Thus it inhibits the break down of your body fat stores. Insulin also inhibits the production of glucose in gluconeogenesis from lactates and amino acids. It signals to the body that glucose is abundant and that glucose is the preferred form of fuel to be used.

Studies reveal that insulin resistance occurs in about 25% of the normal (non-diabetic

and non-obese) US population. When we include the diabetic and obese these percentages are much higher. Insulin resistance exists when there is an abnormal response of the cell receptors to insulin. The insulin cannot bind or the receptor response is weak. The effects of insulin resistance are 1) a reduced glucose and fatty acid entry, 2) a temporary raise in blood sugar level (hyperglycaemia), 3) an increased release of insulin by the pancreas, and 4) a raised blood levels of insulin (hyperinsulinism) over time.

Hyperinsulinism and associated diseases

Over the last few years research has established a positive link between hyperinsulinism and various diseases.

Syndrome X refers to a combination of three clinical factors: 1) *hypertension* or high blood pressure, 2) *hyperinsulinism* or raised blood levels of insulin and 3) *dyslipidaemia* or abnormal blood lipids, such as high triglycerides.

Obesity is linked with hyperinsulinism. However, not all obese people have hyperinsulinism and not all people with hyperinsulinism are obese, but there is a strong correlation between the two.

Ischaemic heart disease has been linked with hyperinsulinism, manifested by heart attacks, build-up of aortic plaques and other related symptoms. Again there is a high correlation, which means that some people with hyperinsulinism do not suffer from ischaemic heart disease and vice versa.

Non-insulin dependent diabetes mellitus (NIDDM) is the adult onset diabetes, which initially can be controlled by diet and exercise. Initially insulin injections are not required. All cases of NIDDM have insulin resistance. It means that there is an insufficient insulin response to put glucose into the cells. The level of insulin is not enough to stimulate entry of glucose into the cell, because the cell receptors are resistant. The result is high blood sugar levels.

Hyperandrogenism or excess steroid hormone levels that increase male characteristics have been found to be linked with hyperinsulinism. It alters the ratio of the androgens and oestrogens in the female as a result of increased androgens in the blood. These get flushed out in the kidneys and are not converted to oestrogen as normally happens. Thus women have free androgens in the blood which binds to the various receptors in the body, resulting in *hirsutism* or excessive hair-growth, and menstrual irregularities. In males increased free testosterone in the blood may lead to benign enlargement of the prostate or *prostatic hyperplasia*.

Polycystic ovary syndrome has been linked to hyperinsulinism. In this condition there is a two-fold problem. There is a block in the enzymatic conversion of testosterone to oestrogen. But in addition there is also a block in the production of androgens themselves. In normal circumstances the body produces DHEA (*dehydroepiandrosterone*) from a precursor called pregnenolone. DHEA can be converted to corticosteroids or it shunted to testosterone, which then turns into oestrogen. In hyperinsulinism there is a blockage in the production of DHEA, and there is reduction of what is called a sex-binding globulin (SBG) which normally binds testosterone. This allows only a small amount to be free in the blood. A disturbance in these hormones throws the system out of kilter. In males increased free testosterone causes prostate enlargement.

As a consequence ***infertility*** in females have also been linked to the problems of oestrogen caused by hyperinsulinism.

Inflammatory disease are linked because hyperinsulinism has the ability to increase the production of cytokines, which are inflammatory mediators from white blood cells. Also excess insulin will disturb the essential fatty acid metabolism and these are also involved in inflammatory reaction.

The metabolism of ***essential fatty acids*** (EFAs) are related to inflammatory diseases. There are two classes of essential fatty acids (EFA) which are produced from polyunsaturates in the food: The omega-6 essential fatty acids (6-series PUFA) and the omega-3 essential fatty acid (3-series PUFA). "Essential" refers to the fact that the body needs to obtain them from the diet. (See **Figure 1** page 4)

The dominant type of EFA in the western diet is 6-series PUFA which are mostly found in seeds and in vegetable oils. The flavour of the month is evening primrose oil. These two types should be in balance. The Israeli diet has a ratio of 6-series to 3-series PUFA of 25:1, which is far too high. A standard western diet has a ratio of about 12:1. It would appear from studies that the ratio should be in the vicinity of 3:1.

Omega-6 PUFA is broken down in the body by several enzymes called desaturases. It is known that high insulin levels interferes with the delta-6-desaturase, which converts linoleic acid (LA) to gamma-linolenic acid (GLA). The latter gives rise to prostaglandins series 1 (PG1) which are anti-inflammatory chemical substances. Hence the popularity of Evening Primrose Oil which contains about 10% of GLA and thus by-passes the dysfunctional delta-6-desaturase.

A diet high in meat, eggs, and saturated fat, typical of western diet, is more likely to be broken down into ***arachidonic acid*** via the same biochemical pathway. Arachidonic acid gives rise to chemicals called *eicosanoids*. These are secondary messages in the body producing prostaglandins series 2 (PG2) and series 4 leukotrienes (LT) and which are pro-inflammatory.

In short an excess 6-series PUFA, typical of the western diet which is high in fat and meats can produce arachidonic acid which is responsible for pro-inflammatory chemicals or eicosanoids or if you are lucky, they can produce prostaglandins series-1 which are anti-inflammatory. All saturated fats are broken down into arachidonic acid and therefore are pro-inflammatory.

On other hand the omega-3 essential fatty acids (3-series PUFA), which are found in abundance in flaxseeds and fishoils are broken down into beneficial chemicals. For example, starting from alpha-linolenic acid it can be broken down into docosahexaenoic acid, which is known to be important in brain function and important in learning difficulties in children. The 3-series PUFA is also converted via eicosapentaenoic acid (EPA) to series 3 prostaglandins, which are anti-inflammatory. (See **Figure 1**, page 4)

Thus it is clear that hyperinsulinism, that interferes with the enzymes (desaturases) in the metabolism of essential fatty acids, is highly correlated with inflammatory diseases.

Kidney stone formation has also been associated with hyperinsulinism, which is thought to be due to a disturbance in the ratio of calcium and magnesium.

Causes of insulin resistance

There are several factors involved in the causation of insulin resistance and one of them is ***genetics.***

It is well known that when the Aboriginal society are placed on the western diet they develop a ten times higher rate of heart disease and diabetes. They have a genetic susceptibility to insulin resistance. But when they eat their traditional diet - that is a low glycaemic index diet - it is not an issue. When placed on a western diet with a high glycaemic index, and an increase in omega-6 essential fatty acids and saturated fats it becomes a major problem. The same sort of picture is emerging in other pacific island races as well.

Obesity itself causes insulin resistance. It reduces the effectiveness of insulin in lowering blood sugar.

We have already mentioned how ***imbalance of essential fatty acids*** is related to insulin resistance. In this area we often come across cycles of causation. The imbalance of essential fatty acids not only causes insulin resistance, but insulin resistance or hyperinsulinism causes imbalance in fatty acids by the way it affects the delta-6-desaturase. This in turn causes inflammation. And again insulin resistance promotes obesity as insulin channels all glucose across to the fat storage cells. Therefore the body gets involved with quite a few metabolic cycles. As mentioned before, it is important to obtain the right ratio of omega-6 and omega 3 essential fatty acid.

Consumption of ***trans-fatty acids*** in margarine and processed fats have an adverse effect on essential fatty acids and is similar to the effects of excess arachidonic acid. ***Monounsaturated fats*** such as olive and canola oils have a protective effect. The key to the way the essential fatty acids, such as the 0mega-3 FAs, influence insulin resistance seems to be that they modulate the cell membrane fluid, rendering the receptors supple, enabling them to move. Fatty acids therefore have a major influence on the receptors and membrane function.

Other causes of insulin resistance are ***a high refined carbohydrate/high glycaemic diet.*** Foods that cause a higher glucose response initially causes a temporary insulin resistance. The blood glucose level goes up. The pancreas sensing the high blood glucose level secretes insulin, but the cell receptor is

temporarily insulin resistant. So the initial response is not very good. The result is that the pancreas puts out some more insulin which leads to hyperinsulinism.

Salt is another factor because excess salt releases stored calcium in cells which leads to a calcium/magnesium imbalance inside the cell. When we don't have enough magnesium within cells, we become twitchy, leading to improper responses. Some researchers believe that the altered balance between calcium and magnesium within cells is a common mechanism which causes insulin resistance. It is essential that we have sufficient magnesium in our body, which we mainly derive from our diet.

Lack of exercise is also seen as a cause of insulin resistance, because it means that our cells need more insulin to get the glucose in. When we exercise we increase the cell's receptor responsiveness to insulin. This is because in exercise we increase the availability of a glucose transporter molecule, called ***Glut-4***, that helps to transport glucose into the cell. As Glut-4 facilitates the entry of glucose, less insulin is required. Hence exercise promotes Glut-4 production, which in turn helps the entry of glucose into the cell and less insulin is required. When we lack exercise we produce less Glut-4 and insulin becomes less effective. Hence insulin resistance. Strength training or resistance exercise (weight lifting etc.) increases muscle mass and thus increases the amount of glucose used.

When we use ***stimulants*** such as nicotine and coffee, we increase the production of interleukin-2 by T lymphocytes - one of a group of proteins that acts as messengers in the immune system - and these are shown to promote insulin resistance. They also stimulate the sympathetic nervous system. Studies seem to indicate that the combination of interleukin-2 and activated sympathetic nervous system may be responsible for hyperinsulinism.

Stress is nearly always one of the factors involved in the causes of diseases. Stress affects our hormone systems. Chronic stress elevates cortisol and adrenaline and both of these have been shown in studies to promote insulin resistance.

Immune activation refers to the cycle of inflammatory reactions causing insulin resistance, which in turn causes inflammatory diseases.

Thus we are building up a big picture. It is not simply that insulin resistance disturbs blood glucose, it does a lot more than that.

Diagnosing Hyperinsulinism

How do we find out that we have hyperinsulinism. Having in mind that an estimated 25 per cent normal non-obese, non-diabetic population in the US have it, the same percentage may apply in Australia. In my clinical experience spanning over a number of years and based on laboratory tests of many patients, not every obese person has hyperinsulinism, nor has every person with hypertension. A family history of diabetes, hypertension and heart disease is suggestive. A history of blood lipid abnormalities [although inherited hypercholesterolaemia (genetic high blood cholesterol) does not reveal the same lipid abnormality we find in hyperinsulinism], kidney stones, infertility, high blood pressure, high triglycerides and obesity adds to the probability of underlying hyperinsulinism.

Clinical features

The kind of obesity may give an indication. The ***'pear shaped'*** form of obesity is not a common clinical feature. However, the ***'apple shaped'*** obesity is more typical for hyperinsulinism. The reason is that the apple shaped obese person is storing fat internally in adipose tissues usually around the waist. Thus a waist greater than 100cm or a weight/height ratio of more than .98 (1.00 for women) may be indicative.

Hypertension or high blood pressure, ***hirsutism*** or more hair on women's faces and other signs of ***abnormal lipid balance*** are important signs. Signs of raised body fats may well be related to an ***earlobe crease*** and ***xanthomas.*** The latter are fatty deposits under skin and are often found around the eyes.

Laboratory assessments

Here we can get a fixed diagnosis.

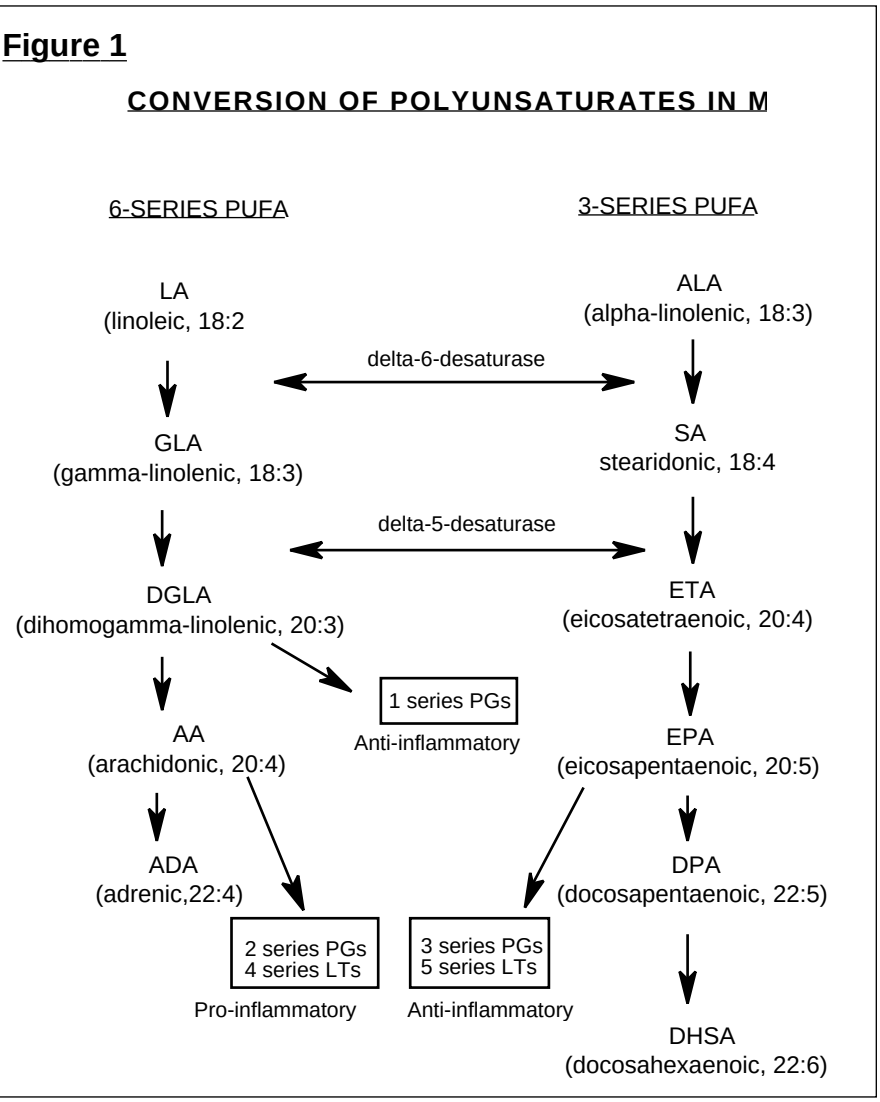
- Raised ***fasting serum insulin*** (>15mmol/ml) would be a clear indication. But it should be noted that some diabetic patients have a normal fasting serum insulin levels. The same applies to some patients with hyperinsulinism.
- In my opinion the only way to prove hyperinsulinism is by way of a full ***glucose tolerance test*** and also looking at insulin levels throughout that test. In my clinical experience several diabetic patients would not have been detected for diabetes on normal medical grounds for another five years or so.
- An ***insulin level*** of >30 mmol/ml would be too high. This is based on my personal experience, as I have been unable to obtain world-wide measures of 'normal' insulin levels.

Other laboratory findings are further supportive in the diagnosis, but before we list the various blood concentration let me explain the relationship of ***cholesterol*** with hyperinsulinism.

There are four different ways of carrying cholesterol around the body.

1) ***Chylomicron*** carry fat directly across the intestinal wall into the blood. It has not been processed.
2) Other substances are ***VLDL*** or the *very low-density lipoproteins*.
3) ***LDL*** or *low-density lipoproteins* and
4) ***HDL*** *or high-density lipoproteins*.

Figure 1



All of these contain cholesterol. The difference lies in the protein carrier and the ratio of cholesterol to the protein carrier; and the amounts of triglycerides in it. The VLDL has predominantly triglycerides plus a small amount of cholesterol. These have been found to be 'baddies'. Studies have found that high triglyceride concentrations are an independent risk factor in heart disease, with other measures being 'normal'. Again in my personal experience I would associate hypertriglyceridaemia with hyperinsulinism. The HDL cholesterol is much safer than the LDL cholesterol. The cholesterol portion in HDL does not get oxidised as easily as in LDL cholesterol. Oxidised cholesterol attracts the attention of tissue macrophages which cause atherosclerotic plaques and heart disease. Thus the absolute cholesterol content is not the issue, but rather the form in which it is, and whether it can be easily oxidised.

In hyperinsulinism we find increased VLDL, often also LDL but not always necessarily, and reduced HDL cholesterol.

Lab tests should look at:

- ***Hypertriglyceridaemia***
- a reduction in the protective levels of cholesterol - thus a ***reduced HDL cholesterol***
- ***raised LDL cholesterol***
- ***Elevated serum uric acid.*** Research has not turned up to explain why an elevated serum uric acid level is associated with hyperinsulinism. But it does give us a link with the inflammatory diseases, and particularly gout.
- ***Elevated plasminogen activator inhibitor 1*** is another factor that has been linked with heart disease. The ability of the body to break down clots seems to be lower, thus increases the risks of blood clots and consequently strokes.
- ***Hair mineral analysis*** may show low levels of magnesium, chromium and vanadium. Vanadium (found in dill seeds, radishes, black pepper, soybean-,corn-,olive-oil gelatine) is an important co-enzyme in many enzymes and high levels have been found to lower cholesterol and triglycerides levels.
- ***Raised serum ferritin*** or high blood iron may also be an indication of hyperinsulinism.

Management of hyperinsulinism

The treatment is basically similar to that of hypoglycaemia and diabetes.

Dietary considerations are:

Small frequent meals. It has been found that a short term fast will improve the ability receptors to bind with insulin. Long-term fasting for example for 3-14 days will increase the number of insulin receptors. Increased meal frequencies has been found in tests to correlate with a reduction of serum lipids and insulin levels.

Increase protein/carbohydrate ratio is a concept very familiar among hypoglycaemics. Tests have found that increased proteins in the diet increase glucagon secretion. Glucagon is a hormone produced by the pancreas which increases blood sugar levels. It is basically the opposite to insulin. Whereas insulin drives glucose into the cells where it may be stored as fat in the form of glycogen, glucagon actually stimulates the liver to break down glycogen, encourages the break-down of fat, thereby raises the blood sugar level. This reduces the pancreatic insulin response and thereby reduces the insulin resistance. Studies indicate that the protein/CHO should be in the order of 3:4.

Higher fat content. We have been told by the Heart Foundation and by the Diabetic Association to reduce fat intake. Amazingly enough, research reveals several studies showing that a higher fat diet - *but of the right kind* - with lower carbohydrates reduces the fasting glucose, insulin and triglycerides levels. This is quite different of what we have been told in the past. The question is what fat? It is the saturated fats that we should avoid. These raise the *arachidonic acid.*

If we increase the omega-6 fatty acids we find that the delta-6-desaturase and/or the delta-5-desaturase may be adversely affected by hyperinsulinism and thus promote *arachidonic acid*. This again stimulates the pro-inflammatory prostaglandins and interleukins and will increase the risk of heart disease. Thus in a situation where we have high levels of insulin the biochemical pathway down from the omega-6 essential fatty acids may not help us in producing the friendly (anti-inflammatory) prostaglandins series 1 (PGE1).

We need to increase instead the omega-3 essential fatty acids, because these fatty acids can not be broken down into arachidonic acid! They can *only* produce the anti-inflammatory series 3 prostaglandins (PGE3) and 5 series leukotrienes. Insulin also affects the delta-6 and delta-5 desaturases in the omega-3 biochemical pathway. But as western diet is abnormally high in the omega-6 essential fatty acids, there is a competition between the two biochemical pathways. By redressing the ratio between omega-6 and omega-3 - in other words, by increasing the sources of omega-3 EFAs - we can enhance the production of the friendly prostaglandins and leukotrienes stemming from *eicosapentaenoic acid* (EPA).(See **Figure 1**)

This can be achieved by consuming ***flaxseed oil*** or ***walnut oil*** which are rich sources of ***alpha-linolenic acid***, and which is the precursor of *eicosapentaenoic acid*. A daily table spoon of flaxseed oil or ground flaxseed and/or two to three serves of fish per week would be an adequate source of EPA. We have to make sure that if we choose to take fishoil that it is not adulterated with pollutants from the oceans. There is a question now with mercury contamination in fishoil supplements. People with an established diagnosis of hyperinsulinism would have the enzyme blockages and it is recommended that they take fishoil to bypass these obstructions. However, increased omega-3 fatty acids also require higher intake of antioxidants (i.e., *vitamin E*), as these fatty acids are easily oxidised.

As mentioned before hyperinsulinism channels all glucose into fat storage cells and one way of correcting this is by increasing essential fatty acids to correct the membranes of cells and correct the insulin receptors. This will improve the efficacy of insulin. No amount of exercise will reduce obesity unless you correct the hyperinsulinism and restore insulin sensitivity of membrane receptors.

High fibre diet is well-known to improve the insulin response, blood glucose control and blood lipid profile. It does this by the slowing down the release of glucose across the gut membrane into the blood. It also seems to inhibit the starch being degraded. The best sources of soluble fibre is ***oats, psyllium and flaxseed.***

Starch type relates to the glycaemic index of food. It has now been established that *rice-cakes* has one of the highest glycaemic indices in foods. Now-a-days they take white bread as the standards which has the 100% mark and glycaemic responses to all other foods are compared with this new standard (see HNL March 98, 12). Compared to white bread at 100%, puffed rice comes in at 132%, and the only food higher that puffed rice is glucose. Compared to white bread, sucrose is 83%. Sucrose added to food does not make a great deal of difference to the glycaemic index. This may be because sucrose is not pure glucose. Unexpected results have been obtained with the glycaemic index and we find that this is so, because the starch type is important.

The starch ***amylose*** compared to amylopectin improves the impact on insulin. High concentrations of amylose is to be found in oats, rye and legumes. Wheat and rice is much higher in the other form of starch, amylopectin. For some reason the amylopectin is much more quickly metabolised into glucose, than the amylose. Coming back to sugars, ***fructose*** is fruit sugar and has a beautiful glycaemic index.

All foods that have a high glycaemic index have a high insulin response. In this respect comparisons can be made. For example among the sugars, glucose has a higher insulin response than sucrose, which in turn is higher than fructose.

Among the starches amylopectin is worse than amylose. Rice is worse than wheat, which is worse than rye, which is worse than oats and which is worse than legumes. This does not mean that we should select only low glycaemic foods but rather that we should balance and diversify our food sources.

If you have coeliac disease you would be intolerant to the gluten in the white bread. Perhaps you might switch to foods that are gluten free, such as rice, corn, potato flour, rice flour, sago, soya bean flour, corn flour. In a totally gluten free diet you might want to choose legume flour bread or rice bread. However, you need to be careful that you consume a properly balanced protein diet.

In hyperinsulinism, the food ratio in the diet may have a powerful effect on insulin, glucagon and eicosanoids. The ratio of ***protein*** to carbohydrates to fat should be 30: 40: 30. Good protein sources are: fish, lean chicken, legumes, nuts, seeds and eggs. In the past we were told that eggs contain high cholesterol

and should be avoided. It is oxidised cholesterol that should be avoided and hence when eggs are boiled and not fried they are a good source of bioavailable proteins. They also contain biotin, deficiency of which may contribute to glucose intolerance.

Fats sources are flaxseed oil, and fish oils. Olive oil being a monounsaturated oil is protective of oxidation of fats.

Carbohydrate sources are: unrefined rye, oats and soy flour.

Micronutrients

The major micronutrient ***magnesium*** is probably the most important in hyperinsulinism. Studies of diets have shown that most people are deficient in magnesium, probably because it is easily lost in food processing. Magnesium is consistently low in the diabetic population. It has been confirmed that low intracellular magnesium impairs insulin action and worsens insulin resistance in NIDDM and hypertension population. Supplementation has been shown to improve insulin-mediated glucose uptake by the cells. In my practice magnesium is probably the most prescribed supplement and this is not only because of hyperinsulinism, but for other conditions as well. The magnesium in dolomite is in a inorganic form and therefore difficult to absorb. You need good stomach acids to digest it. Furthermore, dolomite might be contaminated with pollutants. The best form of magnesium is the one that has been chelated with amino-acids, such as magnesium orotate or magnesium aspartate. Bonemeal also contains magnesium as well as calcium, but basically it is delivering it in the form that bones prefer. I would like magnesium to be combined with potassium. Salt upsets the intracellular magnesium/calcium balance. Magnesium is found in most foods, especially fish, meat, seafood, green vegetables, apples, apricots, avocados, bananas, nuts, kelp, legumes.

Vitamin E improves fasting insulin levels in non-diabetic. It increases insulin sensitivity in NIDDM and decreases triglycerides and LDL cholesterol. It protects the cell membranes and is also protective of the inflammatory condition as a result hyperinsulinism.

Chromium is a well-recognised mineral playing an important role in the glucose tolerance factor. It is thought that it may influence the insulin receptors and promote insulin responsiveness. Chromium deficiency has been related to elevated blood glucose, cholesterol and aortic plaques. It has been shown to improve blood serum lipids, blood sugar, insulin levels, glycated haemoglobin in NIDDM. Good sources are Brewer's yeast, black tea and Brazil nuts, followed by cheeses, meat whole grains and brown rice.

Vanadium - vanadyl sulphate has been shown to have an insulin-like effect on glucose metabolism. At present little is known about this mineral and what its exact function is. But studies support the hypothesis that it improves insulin sensitivity in NIDDM patients. Vanadium is found in dill, fish, olives, meat, radishes, snap beans, vegetable oils and whole grains. Tobacco use decreases the uptake of vanadium.

Biotin deficiency has been shown to impair glucose tolerance and increased intake resulted in improved glucose utilisation from animal studies. Cooked eggs yolks are a good source of biotin. Raw egg white contains the anti-biotin avidin, which prevents absorption.

Alpha-lipoic acid increases insulin-stimulated glucose disposal.

Essential fatty acids helps to overcome the blockage (by hyperinsulinism) of the delta-6-desaturase and thus promotes the friendly anti-inflammatory prostaglandins.

Carnitine is an amino-acid involved with the transport of fatty acids into fat cells (adipocytes) and is especially important in hyperinsulinism and diabetes in that it reduces triglycerides and helps to convert fats into energy, thus reduce obesity. It is normally synthesised in the liver from methionine and lysine, dependent on iron, vitamin C, thiamine (B1), pyridoxine (B6). It is not found in vegetable proteins, but abundant in muscle and organ meats and dairy products.

The ***treatment*** of hyperinsulinism is basically through a change in diet. The Japanese health authorities have emphasised the importance of diversity. They recommend that a healthy diet should consist of more than 15 food types each day. If we eat the same thing day after day we are likely to develop some sort of intolerance or food sensitivity.

Hypoglycaemia and hyperinsulinism - the connection?

We can now tie all these factors together and see the stages of development from hyperinsulinism and hypoglycaemia and then to diabetes.

Stage 1: When we consume high glycaemic index foods it will result in a transient hyperglycaemia or high blood sugar level. This hyperglycaemia itself causes a transient cellular insulin resistance. It also causes a increase in pancreatic secretion of insulin (hyperinsulinism). When the cell sensitivity to insulin restores there is a sudden drop in blood sugar and reactive hypoglycaemia ensues. This is a temporary insulin resistance.

Over time when we consume more high glycaemic foods, instead of a transient insulin resistance we have a fixed insulin resistance. Then we get not only a reduction in insulin sensitivity at receptors on the cell's membrane, but also a loss of the number of insulin receptors over time. We now have hyperinsulinism upon a glucose challenge.

Stage 2: Here we see a fasting hyperinsulinism. In this stage people are still normo-glycaemic, that is they are able to maintain normal blood sugar levels, although somewhat unstable. I find this stage to be similar to *reactive hypoglycaemia.*

Stage 3: We may have fasting hyperinsulinism and higher insulin levels on glucose challenge, but the insulin is no longer enough to be effective at the membranes' receptors. Patients are now observed to be hyperglycaemic on glucose challenge in a glucose tolerance test. These are the people who may intermittently show sugar in the urine. They are now officially diagnosed as *non-insulin dependent diabetic mellitus* (NIDDM). At this stage the condition can still be altered by keeping to a *diabetic diet.*

Stage 4: There is now a loss of insulin secretion by the pancreatic cells. They are burned out. With this loss we have uncontrolled fasting glucose, but patients no longer have hyperinsulinism. These people now become insulin dependent thus IDDM.

Insulin resistance and hyperinsulinism thus underpins many of the common diseases in modern society.

Melatonin: an effective sleeping pill?

By Jurriaan Plesman

The pea-sized pineal gland, located deep in the brain below the posterior border of the Corpus callosum, had until recently been considered a vestigial or rudimentary "third eye" sensitive to the cycles of day and night. When the retina of the eyes signals the pineal gland that it is night, the pineal secretes an antioxidant hormone, *melatonin*, into the blood stream. It is claimed this not only hastens sleep, but may also strengthen the immune system, and reduce free radicals in the body.

We have often heard how tryptophan, an amino acid found in many foods such as milk and bananas, may help a person to fall asleep.

No wonder. Tryptophan in the presence of vitamin B6 and B3 (tryptophan is also the forerunner of niacin) produces serotonin, which is then converted to melatonin under conditions of darkness. This is the very substance that the pineal gland uses for conversion to melatonin as follows:

Tryptophan —> 5-Hydroxytryptophan —> 5-Hydroxytryptamine (Serotonin) —> N-Acetylserotonin —> Melatonin.

The enzyme converting N-Acetylserotonin to Melatonin is hydroxyindole-o-methyl transferase (HIOMT) and is to found *only* in the pineal gland.

Melatonin may have many other uses and has been reported to make people feel better, strengthen the immune system, and reduce free radicals in the body. Current research is underway to determine melatonin's effect as an anti-oxidant, immuno-modulator in cancer, delayed sleep-phase disorders, and jet lag. As a fat-soluble antioxidant it permeates all parts of the body and within the cell it provide protection for the nucleus which contains the DNA. Melatonin also stimulates the enzyme

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Complementary, Alternative, Unconventional, and Integrative Medicine: Call for Papers for the Annual Coordinated Theme Issues of the AMA

[Editorial]

By Dr Phil B. Fontanarosa is a Senior Editor and
Dr George D. Lundberg is Editor of JAMA.

Archives of Family Medicine, American Medical Association, 515 N State St, Chicago, IL 60610. Volume 7(1) January 1998 pp 18-19

FROM ACUPUNCTURE to aromatherapy, from homeopathy to hypnosis, and from relaxation therapy to reflexology, numerous practices that are termed complementary, alternative, unconventional, or integrative medicine have become increasingly prevalent and popular. Even though many of these therapies encompass diverse modalities and philosophies that usually are considered outside the realm of mainstream allopathic medicine, the use of complementary medicine interventions, visits to alternative medicine practitioners, and expenditures for these therapies are substantial. In the United States, the estimated 425 million visits to unconventional medicine practitioners in 1990 exceeded the number of visits to primary care physicians and the use of unconventional therapy generated expenditures estimated at $14 billion. (1) Complementary therapies are used by 20% to 50% of the population in many European countries (2) and by 48% of the population in Australia. (3)

Despite increasing public interest and worldwide use of complementary and alternative therapies, high-quality scientific evidence that clearly establishes the effectiveness (or lack thereof) of these interventions is lacking. (4,5) Consequently, many physicians traditionally have viewed alternative medicine in general, and most practices contained therein, with skepticism and mistrust.

However, recent developments indicate changing attitudes toward these unconventional therapies, and demonstrate increasing recognition of the need to critically investigate the safety and efficacy of complementary and alternative medicine practices and to determine how some of these therapies could be integrated into clinical practice to improve patient care. For instance, the US National Institutes of Health (NIH) spends approximately $40 million per year on research related to complementary and alternative medicine (largely involving dietary manipulation and behavioral medicine), (6) and the NIH Office of Alternative Medicine, which was established in 1992, is now under consideration to have its status upgraded to a full-fledged national center (for complementary and alternative medicine research). (7) In their review of published surveys, Ernst and colleagues (8) found that, on average, physicians perceive complementary medical therapies (such as acupuncture or manipulation) as moderately effective. Berman et al (9) reported that more than half of family physicians they surveyed considered alternative medicine interventions (including diet and exercise, biofeedback, hypnotherapy, and massage therapy) to represent "legitimate medical practices." At least 34 US medical schools have been reported to have started or are developing courses on alternative medical practices in their medical education programs. (10) New biomedical journals devoted to the scientific evaluation of unconventional health claims also have been launched. (11)

Given the burgeoning interest in alternative medicine among the general public, patients, physicians, academic medical centers, and health care payers, the JAMA Editorial Board and senior staff and the editors of the American Medical Association (AMA) Archives Journals, using our annual modified Delphi process, ranked alternative medicine among the top 3 subjects (of 86) for our journals to address in the coming year. (Last year, the editorial board ranked alternative medicine 68th of 73 subjects.) Moreover, in a recent survey, (12) JAMA physician readers identified alternative medicine as the seventh (of 73) most important topic for publication in THE JOURNAL. Considering that complementary and alternative medical therapies have the potential to involve patients of physicians in virtually all specialties, the editors of the AMA scientific journals have selected complementary and alternative medicine as the subject for coordinated theme issues to be published late in 1998.

The format for the concurrent theme issues on complementary, alternative, unconventional, and integrative medicine will be similar to theme issues on "Quality of Care" (November 1997) and "Managed Care" (October 1996), in which the AMA scientific journals devoted all or many of their pages, as merited after editorial evaluation and peer review, to a common topic. The 1998 coordinated theme issues will provide a unique, multidisciplinary forum for the publication of original research studies and scholarly articles that present new scientific information and innovative ideas on complementary and alternative medicine to the medical and scientific community. By stimulating research and giving emphasis to this topic, we hope to promote widespread attention in the medical literature and the lay media, foster education among health care professionals, and increase knowledge among patients and the public.

We invite authors from the United States and from other nations, especially authors from countries with an extensive history of non-Western, nonallopathic practice (eg, studies of acupuncture from China), to submit original manuscripts on topics pertaining to complementary and alternative medicine for consideration for publication in JAMA or in 1 of the AMA Archives Journals. The manuscript may be a report of original research, a review article, an opinion piece, or in the format of any of the other regular features of 1 of the AMA scientific journals. High-quality research studies (especially randomized clinical trials) that evaluate the efficacy, safety, outcomes, and cost-effectiveness of complementary and alternative medicine interventions are of particular interest. Manuscripts that assess the integration of complementary medical therapies into conventional clinical practice and papers that examine alternative medicine from the perspective of patients, health care organizations, or academic medical centers also are welcome.

The editors of the AMA scientific journals look forward to receiving manuscripts for consideration for publication in the coordinated theme issue on complementary, alternative, and integrative medicine. Submitted manuscripts are subject to our usual rigorous editorial evaluation and peer review, and advance acceptance for any paper cannot be guaranteed. Articles accepted for publication by JAMA or by 1 of the AMA Archives Journals but not included in the theme issues will be published in other issues of these journals. Authors should consult the Instructions for Authors for JAMA (13) or the appropriate Archives Journal for guidelines on manuscript preparation and submission. Manuscripts received by April 1, 1998, will have the best chance of acceptance for the coordinated theme issues.

References

1. Eisenberg DM, Kessler RC, Foster C, et al. Unconventional medicine in the United States: prevalence, costs, and patterns of use. N Engl J Med. 1993;328:246-252. [Back to Paragraph 1]
2. Fisher P, Ward A. Complementary medicine in Europe. BMJ.1994;309:107-111. [Back to Paragraph 1]
3. MacLennan AH, Wilson DH, Taylor AW. Prevalence and cost of alternative medicine in Australia. Lancet. 1996;347:569-573. [Back

to Paragraph 1]
4. Practice and Policy Guidelines Panel, National Institutes of Health Office of Alternative Medicine. Clinical practice guidelines in complementary and alternative medicine. Arch Fam Med. 1997;6:149-154. [Back to Paragraph 2]
5. Linde K, Clausius N, Ramirez G, et al. Are the clinical effects of homeopathy placebo effects? a meta-analysis of placebo-controlled trials. Lancet. 1997;350:834-843. [Back to Paragraph 2]
6. Jonas WB. Researching alternative medicine. Nat Med. 1997;3:824-827. [Back to Paragraph 3]
7. Wadman M. Row over alternative medicine's status at NIH. Nature. 1997;389:652. [Back to Paragraph 3]
8. Ernst E, Resch KL, White AR. Complementary medicine: what physicians think of it: a meta-analysis. Arch Intern Med. 1995;155:2405-2408. [Back to Paragraph 3]
9. Berman BM, Singh BK, Lao L, et al. Physicians' attitudes toward complementary or alternative medicine, a regional survey. J Am Board Fam Pract. 1995;8:361-366. [Back to Paragraph 3]
10. Jacobs JJ. Building bridges between two worlds: the NIH's Office of Alternative Medicine. Acad Med. 1995;70:40-41. [Back to Paragraph 3]
11. Stapleton S. New journal examines alternative medicine claims. American Medical News. November 3, 1997:14. [Back to Paragraph 3]
12. Lundberg GD, Paul M, Fritz H. A comparison of the opinions of recognized experts and ordinary readers as to what topics a general medical journal should address. Presented at The International Congress on Biomedical Peer Review and Global Communications; September 20, 1997; Prague, Czech Republic. [Back to Paragraph 4]
[illegible] JAMA. 1997;27:30-76. [Back to Paragraph 7]

Vanadium as an aid in diabetes

By Jurriaan Plesman

Vanadium is very much a mysterious trace element. Nutritionists are not sure of its metabolic and nutritional importance. Christie claims that vanadium not only potentiates insulin's action but has an insulin-like action. It normalizes blood glucose and preserved heart action according to a *Science* paper(22 March 1985) and cited in Christie (1991).[1] Also see article by Dr Robyn Cosford in this issue.

Vanadium is needed for cellular metabolism and for the formation of bones and teeth. There may be an interaction between vanadium and chromium and if these are taken in supplemental form, they should be taken separately. Tobacco use decreases uptake of vanadium.[2] Vanadium deficiencies in humans have not been recorded, in fact some authors believe that an intake above 10mcg per day could well lead to toxicity.[3]

Pharmacologic amounts of vanadium (ie, 10 to 100 times normal intake) affect cholesterol and triglyceride metabolism, influence the shape of erythrocytes, and stimulate glucose oxidation and glycogen synthesis in the liver. Vanadium's primary mode of action is as a cofactor that enhances or inhibits enzymes. Recent evidence suggests that vanadium may be essential for higher animals. After their mothers had been fed carefully formulated vanadium-deficient diets, second-generation goat kids suffered skeletal damage and died within 3 days of parturition. Although ubiquitous in air, soil, water, and the food supply, vanadium is generally found in nanogram or microgram quantities, which makes it difficult to measure. Estimates for the American intake of vanadium (based on a food intake of 500 g dry weight) are 10 to 60 micrograms/day. Vanadium levels in diets from five regions of the United States range from 30.9 +/- 1.5 in the Southeast to 50.5 +/- 1.5 micrograms/kg dry weight in the West. Although vanadium is thought to be essential for goats, new data may soon support its essentiality in human beings.[4]

It is thought to be involved in the regulation of the sodium/potassium pump, which plays an important role in the in the maintenance of the balance between Na^+/K^+ in the Loop of Henle within the kidney. Consequently, vanadium may have an effect on blood pressure.[5]

A growing body of experimental and clinical research indicates that the trace element, vanadium, exerts potent insulin-mimetic effects in vitro and in vivo when used in pharmacological doses. A major advance in the use of vanadium as an insulin-mimetic has been the development of organic vanadium complexes which are 2 to 3 times as potent as inorganic vanadium and have been extensively studied. There is an emerging role for the use of vanadium in human diabetes and the recently conducted clinical trials support this contention.[6]

Nevertheless, excess vanadium has been associated with some human diseases, such as non-filarial elephantiasis of the feet and legs, recently renamed *podoconiosis*. This condition is characterized by tremendous swellings of the legs and feet. Soil collected in an area of the Ethiopian Rift Valley, the borough of Ocholo, known for its high prevalence of podoconiosis (5.06%), has been submitted to mineral analysis. High values of various minerals were found in basaltic bed-rocks of the area. One of these was a high content in vanadium, above 250 ppm, in half of the soil samples collected in this region.

There are several studies to show that elevated vanadium has been reported in the plasma of patients with mania and depression and in the hair of patients with mania.[7] Increased plasma vanadium is negatively associated with Na^+-K^+-ATPase activity in manic-depressives, but not in normals.[8] This could explain why medications such as *phenothiazines* and *monoamine oxidase inhibitors* (MAOs) are used as they antagonize the effects of vanadium. *Lithium* is known to cause folic acid deficiency, but increase Na+-K^+-ATPase activity (Naylor, 1984). Some therapies for bipolar disorders (manic-depression) aim at reducing vanadium concentrations (e.g. ascorbic acid, EDTA, methylene blue and low vanadium diet).[9]

Nutritional source of vanadium are given in **Table 1** below.

Table 1
Vanadium in Mcgs per Food 100g

(Figure immediately following food items indicate the approximate content of vanadium in Mcgs per 100 g)

Buckwheat 100, Parsley 80, Radishes 79, Soybeans, Vegetable 70, Egg Yolks 68, Safflower Oil 64, Eggs 42, Sunflower Seed Oil, refined 41, Egg White 37, Egg Yolk, dried 37, Oats, without husk, whole grain 35, Olive Oil 30, Cod, 19, Beans, French beans, String Beans 15, Corn 15, Sunflower Seeds 15, Dill Pickles 14, Dill Weed 14, Green Beans 14, Oysters 11, Peanut Oil 11, Cabbage 10, Carrots 10, Garlic, cloves 10, other possible sources seafood, mushrooms, gelatin, liver.

Footnotes

1) Christie, JS (1991), **Food for vitality,** Bantam Books Sydney,142
2) Balch JF & Balch PA (1997), **PRESCRIPTION FOR NUTRITIONAL HEALING:** A practical A to Z reference to drug-free remedies using vitamins, minerals, herbs & food supplements, Avery Publishing Group, NY, 29
3) Somer,E (1995), **ESSENTIAL GUIDE TO VITAMINS AND MINERALS**, HarperPerennial, NY, 146
4) Harland BF. Harden-Williams BA (1994), Is vanadium of human nutritional importance yet? **Journal of the American Dietetic Association. 94(8):** 891-4, [Review] [28 refs]
5) Dafnis E. Sabatini S (1994), Biochemistry and pathophysiology of vanadium [editorial], **Nephron. 67(2):** 133-43
6) Verma S. Cam MC. McNeill JH (1998), Nutritional factors that can favorably influence the glucose/insulin system: vanadium, **Journal of the American College of Nutrition. 17(1):** 11-8 [Review] [65 refs] .
7) Naylor GJ (1984), Vanadium and manic depressive psychosis, **Nutr & Health 3(1-2):** 79-85
8) Dick DA et al (1981), Plamsa vanadium concentrations in manic-depressive illness, **J Physiol 310:** 27
9) Werbach,M.R.(1987), **NUTRITIONAL INFLUENCES ON ILLNESS**, Third Line Pres,Inc.,Tarzana,Cal. 88

Continued from page 6

glutathione peroxidase, another antioxidant.[1]

Seven days treatment with melatonin at a level of 2 mg daily can improve sleep patterns in elderly patients with insomnia.[2] According to many, one of the more effective means of taking a supplement such as melatonin is the sublingual (under the tongue) method. A high quality sublingual melatonin supplement will usually contain 2.5 mg. of melatonin (often in a base of sorbitol and a natural flavor such as peppermint). One tablet can be dissolved under the tongue before retiring. Wurtman and Zhdanova have observed that a relatively low dose of 0.3mg can be just as effective, which is taken 30 minutes before retiring. Subjects reported a better quality sleep and were free of morning sleepiness or hangovers. [3]

Production and secretion of melatonin is stimulated by the activity in a region of the thalamus[4], called the *suprachiasmatic nucleus (SCN)* via activation of neurons to the pineal. During the day neural pathways from the retina of the eyes depress the activity of SCN thus decreasing melatonin secretion. The diurnal secretions of melatonin provide the "internal clock". Derangements in such cycles may result in such phenomena as *jet lag* and *winter depression* also called *seasonal affective disorder* (SAD). Patients may sleep and/or eat excessively, be withdrawn and have lowered libido. This can be treated with light therapy. In one study patients who took morning walks in sunlight for a minimum of one hour showed positive results.[5] SAD patients were found to have high levels of melatonin, an important point to remember when patients happen to be night-shift workers.

It is also known that a decrease of melatonin is associated with maturation of the gonads in many lower vertebrates and that excessive melatonin secretion may delay the onset of puberty. Melatonin production is highest among children between the ages of one and five and decreases thereafter, reaching its lowest level at the end of puberty (when concentrations are about 75% lower than during childhood).[6]

Melatonin is being considered as a component of the antioxidant defence system because *in vitro* experiments have shown that it is able to neutralise toxic radicals.[7] Mice treated with melatonin showed a 50 percent reduction in tumour weight when compared to control mice. [8] It has also been reported that melatonin can inhibit the synthesis of the LDL cholesterol (the bad cholesterol).[9]

Some concern has been expressed about the variable quality of supplemental preparations in some countries. Much of the melatonin is produced from bananas and tomatoes. Melatonin rapidly degrades, raising questions about the potency of products available.[10] Melatonin could also exert an inhibitory effect on the thyroid gland's uptake of iodine. [11]

Another concern would be that treating insomnia with supplementation of melatonin alone may overlook many other factors involved. A hypoglycemic dip in the middle of the night can also cause a patient to wake up. Melatonin by itself will not correct an underlying hypoglycemic condition; nor would it mend a manic-depressive illness. Excess caffeine, alcohol, smoking, deficiencies of vitamin B3 or B6 and anxiety are some other factors to be taken into account.

Nevertheless, administration of melatonin would offer a great advantage over the plethora of narcoleptic drugs now prescribed by mainstream medicine.

Perhaps the best solution would still be to use tryptophan[12] - rich in skim milk, cottage cheese, peanuts and bananas - together with vitamin B6, zinc and niacinamide as the major precursors of melatonin. Other natural sources are black mungo beans, turkey, brewer's yeast, fried pork, soya flour, pumpkin seeds and cheeses, although tryptophan may have to compete with other amino acids. Absorption of tryptophan can be increased by intake of a high carbohydrate diet. This provokes the pancreas to secrete insulin. Insulin increases the relative concentration of tryptophan by causing the tissues to soak up competing amino acids from the blood. This leaves tryptophan with less competition transferring from the blood to the brain.[13]

The administration of melatonin is best left in the hands of skilled health professionals who may want to use it to treat specific clinical conditions, such as cancer or persistent insomnia. Studies have shown that as a sleeping pill, melatonin has short term beneficial effects, but people soon build up a tolerance that require more and more melatonin to have the initial effects. Furthermore, it needs to be taken at the right time of the day. In the hands of the inexperienced it can easily be abused like all *'sleeping pills'* and upset the finely balanced metabolism of the body. It is not yet available over the counter in Australia as it is in the USA, but it is great pity that tryptophan - its precursor - in supplemental form has been banned.

Melatonin has also been found in some herbs such as Feverfew *(Tanecetum parthenium)*, St John's wort *(Hypericum perforatum)* and Baical skullcap *(Scutellaria baicalensis)* which may explain why they are used for migraines.[14]

From a practical point of view, knowledge about melatonin may help us to improve health if we employ its precursors - regular sunlight and darkness, tryptophan, B6 and zinc - and then let nature take its course.

FOOTNOTES:

1) Balch JF & Balch PA (1997), **PRESCRIPTION FOR NUTRITIONAL HEALING: A practical A to Z reference to drug-free remedies using vitamins, minerals, herbs & food supplements,** Avery Publishing Group, 45 NY
2) Dr. Haimov, British Medical Journal, 1994
3) Wurtman RJ, Zhdanova I (1995), Improvement of sleep quality by melatonin, **Lancet 346,** 1491
4) The thalamus act primarily as a relay centre through which all sensory information (except smell) passes on the way to the cerebrum, the largest portion of the brain (accounting for about 80% of its mass) and responsible for the higher mental functions. Neurons also extend to the pineal gland.
5) Gutfeld G (1993), The new science of rays and rhythms - Cutting edge light therapies that can brighten your health, **Prevention 45 No2 (Feb 93):** 67-71
6) Fox SI (1993), **Human physiology,** Wm C Brown Pubs, Melbourne, 278
7) Reiter RJ (1996) Functional aspects of the pineal hormone melatonin in combating cell and tissue damage induced by free radicals, **Eur J Endocrinol 134,** 412-420
8) Dr. Berkowitz, **Journal of Urology**, 1988
9) Dr. Muller, **Biochemical and Biophysical Research**, 1994
10) Huether G (1996), Melatonin as an antiaging drug: between facts and fantasy, **Gerontology 42,** 87-96
11) **Harrison's principles of internal medicine,** 1971, 575
12) Tryptophan 500 mg obtainable on doctor's prescription, crushed and added to lukewarm milk is a standard treatmment for insomnia. The herb *valerian* should also be considered. We are looking forward to the day when tryptophan tablets - a nutrient - will again be available in health food stores as in the good old days.
13) Werbach MR (1993), **Healing through nu-**

LETTERS TO THE EDITOR

Aspartame

Cindy Thummel of the Lismore Women's Health Centre Inc. referring to article "*Diet for Drug Addiction and Alcoholism"* in HNL June 98 p 8, writes:

"I was very concerned to see your suggestion to replace sugar with the additive Aspartame. This chemical is known to increase frequency of seizures in epileptic patients and can even begin seizures in young people who had previously been seizure free. (See Dr Sandra Cabot's recent book). I can also verify this as my own son, aged 16, who had never experienced a seizure has had two within the last 18 months. Both times he was chewing sugar-free gum. He rarely has any sort of gum. Aspartame will increase sensitivity to flashing lights precipitating his seizures. The growing number of children who fit while watching TV and video games is a concern. I wonder how many of these have also linked with Aspartame ingestion prompted by parents who want to limit sugar intake so substitute 'Sugar Free'?"

Editor: How right Cindy Thummel is. A warning should have been included in the article referred to. We did publish an article on aspartame in HNL March 1993, 6. Aspartame is a chemical compound consisting of 50% phenylalanine (PHE), 40% aspartic acid and 10% methyl alcohol (or methanol). Methanol is readily converted to the immune suppressing toxic substance formaldehyde.

Some people consuming aspartame were found to have symptoms similar to hyperthyroidism (Graves Disease) including palpitations, tachycardia, "anxiety attacks", headaches, weight loss, thinning of hair, enlarged eyes and hypertension. People most likely to suffer these symptoms were those who were simultaneously dieting and exercising vigorously. These symptoms would disappear after two days of removing aspartame from the diet. According to HJ Roberts aspartame causes metabolic changes involving satiety and alterations in neurotransmitters and hormones (insulin, growth hormone, glucagon and cholecystokinin) . [Cholecystokinin is a hormone produced by the mucosa of the upper intestine, that stimulates contraction of the gallbladder and the secretion of pancreatic enzymes.]

A decade ago the FDA listed over 7,300 individuals with adverse reactions to aspartame.

Roberts HJ (1997), Aspartame and hyperthyroidism - a presidential affliction reconsidered, **Townsend Letter for Doctors & Patients 166:** 86-88

Food and Cancer

By Karen Bridgman[1]

Hippocrates has been credited with saying, 'Let your food be your medicine and your medicine be your food'. 60-90% of cancers are diet and lifestyle related. We get the types of cancer according to the foods we eat and this is a cultural process.[2]
For example, in Japan there is a greater incidence of stomach cancer because their diet is higher in pickles and they drink very hot tea. However they are largely protected against hormonally based cancers such as breast and prostate cancer.[3]
To improve our health and to protect against the common cancers we need to be aware of the foods that can potentially cause problems and those that we need to eat more of for protection.
In our society we have nutrient-poor, high fat, high meat diets and this predisposes us to bowel, breast, prostate and some skin cancers. Lung cancer is associated with smoking.
There are many foods that are believed to help prevent degenerative diseases such as cancer that can also help regain health if you have symptoms of this, or other degenerative disease.
I have chosen only a few foods that have been well-researched and have been shown to be so powerful they come into the category of super foods. Increasing these foods in your diet will improve your health.

Soya

Soy is a major food that has had remarkable effects on preventing the hormonally-based cancers such as breast cancer and prostate cancer. Soya contains compounds called phytooestrogens that counteract cancer-promoting oestrogen in much the same way as the drug, Tamoxifen - without the side-effects. Soya also contains other substances that block cancer cells by mechanisms not related to oestrogen. 'Soya beans have protected Asian women against cancer - women in Singapore[4] who eat twice the soy protein as other women, had half as much breast cancer.[5] Typically they ate about 85 grams of soya beans daily. This can be taken as soya milk, tofu, miso, tempeh, soya flour, soya beans etc. It appears that the soya protein is protective, but not soya sauce or soya oil.

Soya has also been shown to protect against prostate cancer in men who eat it daily. Prostate cancer is lower in men who eat soy protein regularly because it decreases dihydrotestosterone (DHT), the excess of which causes the problem.

Garlic and onions

Garlic and onions have similar effects. Garlic has been shown to help destroy cancerous cells in much the same way as chemotherapeutic drugs - without side-effects. *Ajoene* has been shown to be three times as toxic to malignant cells as normal cells. Garlic act as a *biological response modifier* that boosts immune function by boosting the anti-cancer activity of macrophages and T-cells. Garlic and onions have more that 30 different anti-cancer substances in them that have been shown to reduce stomach (possibly its antibiotic effect against Helico-bacter pylori), breast, skin, lung and liver cancer.

Research: eating 85 grams of garlic, onions, chives, spring onions etc, reduces stomach cancer by 40%.[6]

Green tea

Green tea is a major anti-cancer drink.[7] The major therapeutic effect is created by substances in green tea called *catechins*, particularly *epigallocatechin gallate* (EGCG) - a side effect is to regulate blood cholesterol. Black tea has only 10% as much EGCG as green tea and so is not as useful. These substances are powerful antioxidants and reduce the damage caused by free radicals - unstable molecules in our bodies that cause many of the problems we have with our health.

Research: drinking green teas in concentrations normally consumed by humans (approximately six to eight cups daily), blocked up to 78% of skin cancer, 58% of stomach cancer and 56% of lung cancer in mice.[8]

Beetroot

Beetroot and other red coloured vegetables and fruits help detoxify the liver and provide oxygen to cells. When cells become cancerous, a major enzyme that uses oxygen to make energy for the cell is destroyed, so the cell shifts to using a different form of energy production that does not require oxygen (often glucose or glutamine).

Raw beetroot has been shown to be a major food that switches the cell back to using oxygen again - thus reversing the effect of cancer. Beetroot also detoxifies the liver. You need to eat more than 250 gram daily or drink as juice with carrot and celery. Beetroot must be fresh, not canned.

Research: 21 out of 22 patients eating one kilogram of beetroot daily showed a reduction in tumour size, weight gain, improvement in blood profiles and improvement in their overall health and feeling of well-being. These patients have cancer of the lung, stomach, colon, breast, uterus, prostate and skin.[9]

Fibre

Food fibres have a major protective role in the battle against cancer and degenerative diseases. By speeding up the processing of food residue through the bowel and by changing the bacteria to those that are beneficial to our health, they reduce faecal mutagen/carcinogen concentrations, reduce the length of exposure to mucosa to mutagens (by increasing bowel transit time), inhibit faecal mutagen synthesis by lowering the acidity (pH) and encourage correct bowel flora. Fibre, particularly the soluble fibres, also increase butyrate, the energy source of the bowel and a major bowel inhibiting metabolite. Fibre ferments to butyrate with the correct bacteria.

Best fibres are dietary fibre-resistant starch such as whole cereals, fruit, vegetables, green bananas or the fibre supplements - slippery elm, psyllium, oat, rice, barley bran. Along with fibre in your diet, adding foods that provide the correct bowel bacteria also helps. These are foods such as sauerkraut, miso, acidophilus, yoghurts and other traditional fermented foods.[10]

Fruit and vegetables

There are many bioactive plant substances available in fruits and vegetables such as carotenoids, vitamin A, C and E, and folate. These also include powerful anti-cancer substances such as glucosinolates and flavonoids found in cassava, garlic, onions, purple, red and black fruit - blueberries, eggplant, red

cabbage, beetroot - dark green vegetables and dark yellow-orange vegetables. The cabbage family - broccoli, Brussels sprouts, cauliflower etc. - has many anti-cancer substances available in them such as isothiocyanates, indoles and sulphorane. Eating four to six serves of fruit and vegetables per day is an excellent and tasty way to maintain your health.

Overall, a diet that is protective against all cancers is one that is high in fruit and vegetables, high fibre - 30 to 35 grams of fibre daily and low animal (saturated) fat. Fat intake should be less than 30% of calories eaten.[11]

1) Reprinted with permission from Newsletter (Summer/Autumn Edition 1997) for ICAN, *International Cancer Association Network*, Suite 401, 4th floor, BMA House, 135 Macquarie Street, Sydney, NSW 2000, Telephone 02-9251-4140
2) Carper J (1988), **Food Pharmacy,** Simon and Schuster.
Carper J (1994), **Food, Your Miracle Medicine,** Simon and Schuster
Werbach M (1987), **Nutritional Influences on Illness,** Third Line Press Inc, Tarzana Cal.
Werbach M (1991), **Nutritional Influences on Mental Illness,** Third Line Press Tarzana, Cal.
3) Caragay A (1992), Cancer preventive foods and ingredients, **Food Technology 46:** 65-68
Thomas J (1987), Diet and cancer, **International Clinical Nutrition Review** Jan 1987 (7): 1
Truswell S (1996), Lecture notes from AIMS Conference 1996.
Clifford C, Kramer B (1993), Diet as risk and therapy for cancer, **Clinical Nutrition July 1993; 77(4):** 725-744
4) Lee H (1991), Dietary effects on breast cancer risk in Singapore, **Lancet 337:** 1197-1200
5) Barnes S (1990) Soybeans inhibit mammary tumours in models of breast cancer, **Progress in Clinical and Biological Research 347:** 239-253
Holm L (1992), Treatment failure and dietary habits in women with breast cancer. **Journal of the National Cancer Institute 84:** 561-9.
Kushi L(1992), Diesary fat and postmenopausal breast cancer, **Journal of the National Cancer Institute 84:** 1092-1099.
Messina J et al. (1994), Soy intake and cancer risk: A review of the in vitro and in vivo data, **Nutrition and Cancer 21(2):** 113-131.
Messina M (1991), The role of soy products in reducing risk of cancer, **Journal of the National cancer Institute 83(8):** 541-46.
6) Lau B (1991), Garlic compounds modulate macrophage and T-lymphocyte functions, **Molecular Biology 3:** 103-7.
7) Steinman D (1994), Why you should drink green tea, **Natural Health 24:** 56-58
8) Editorial (19914), The potential of green tea against cancer, **Environmental Nutrition,** 3
Imai K (1995), Study of the effects of drinking green tea on cardiovascular disease and liver disorders, **British Medical Journal, March 1995:** 693-697
9) Buist R (1986), Beetroot as cancer therapy, **International Clinical Nutrition Review July 1986, (6):** 3
10) Alberts D (1990), Effects of dietary wheat bran fibre on rectal epithelial cell proliferation in patients with resection for colorectal cancers, **Journal of the National Cancer Institute 82:** 1280-1285
Howe G (1992), Dietary intake of fibre and decreased risk of cancer of the colon and rectum, **Journal of the National Cancer Institute 84:** 1887-1896
Willet W (1990), Relation of meat, fat and fibre intake to the risk of colon cancer in a prospective study among women, **New England Journal of Medicine 322:** 1664-1672
11) Block G (1992), Fruit, vegetables and cancer prevention: A review of the epidemiological evidence, **Nutrition and Cancer 18:** 1-29

Herbs to be avoided in pregnancy

Botanical name	Common name
Alchemilla vulgaris	Ladies' Mantle
Angelica sinensis	Dong quai
Astragalus membranaceus	Astralagus
Berberis vulgaris	Barberry
Bryonia alba	English mandrake
Caulophyllum thalictroides	Blue cohosh
Centella asiatica	Gotu kola
Chelidonium majus	Greater celandine
Cinchona spp	Peruvian Bark; Cinchona
Coix larchryma-jobi	Native barley
Datura stramonium	Jimson weed, Thorn apple
Dysosma auranticocaulis	Six-cornered lotus
Gentiana lutea	Gentian root
Hedeoma pulegiodes	Pennyroyal
Hydrastis canadensis	Goldenseal
Hypericum sampsonii	Cow's soapwort
Juniperus communis	Juniper
Lobelia inflata	Indian tobacco
Mentha pulegium	European pennyroyal
Morinda citrifolia	Noni or Nono
Paullinia cupana	Guarana
Prunus serotina	Wild cherry bark
Rhamnus frangula	Alder buckthorn bark
Rheum palmatum	Rhubarb root
Sanguinaria canadensis	Blood root
Sarothamnus scoparius	Irish broom
Senecio aureus	Life root, Squaw weed
Tanacetum parthenium	Feverfew
Thuja occidentalis	Arbor vitae, Thuja
Veronica peregrina	Bone-Knitting Grass, Purslane

RESEARCH SNIPPETS

Antioxidants reduce susceptibility to LDL oxidation

It is important that patients with high risk for cardiovascular events, such as people with hypercholesterolaemia (high cholesterol) and with established coronary heart disease, take antioxidant supplementation. A group of 45 patients were randomly assigned to groups receiving low-dose 400 IU vitamin E, 500mg vitamin C and 12 mg beta-carotene, high-dose (double the low-dose) or placebo. Conclusion: "A high-dose combination of antioxidant nutrients reduces the susceptibility of LDL to oxidation in patients with cardiovascular disease and may be useful in secondary prevention.

Mosca,L Rubenfire M, Mandel C, et al (1997), Antioxidant nutrient supplementation reduces the susceptibility of low-density lipoprotein to oxidation in patients with coronary artery disease, **J Am Coll Cardiol 30:** 392-399

Antioxidants provide protection for diabetes

Diabetes is associated with vascular complications thought to be due to increased free radical activities. This study compared 28 patients with insulin-dependent diabetes (IDD) and 24 patients with non-insulin dependent diabetes (NIDD). Both groups of patients had diabetes uncomplicated by vascular disease. Serum antioxidant status were compared and found to be significantly lower in IDD patients. Total antioxidant activity did not differ significantly between NIDD and controls, however serum vitamin C levels were significantly decreased in NIDD patients. As free radical oxidation damage is implicated in the development of diabetic complications antioxidant supplementation may play a beneficial role in diabetes.

Maxwell SR, Thomason H, Sandler D et al (1997), Antioxidant status in patients with uncomplicated insulin-dependent and non-insulin-dependent diabetes mellitus, **Eur J Clin Invest 27:** 484-490

Editor's note: This supports the reasoning in a previously published article ***Hypoglycemia and atherosclerosis*** in HPNL Dec 97, Page 7 suggesting that supplementation with vitamin C is especially beneficial for patients with a glucose intolerance problem.

Maitake mushroom may have a role in diabetes treatment

Diabetic mice were used to study the effects upon glucose levels of Maitake mushroom in powder form. Results after 15 minutes and 30 minutes indicated glucose levels were 0.64 and 0.76 times those of controls respectively. Analysis revealed that Maitake mushroom influences the metabolism of absorbed glucose rather than directly inhibiting glucose absorption.

Kubo K, Nanba H (1997), Anti-diabetic mechanism of Maitake, **Townsend Letter for Doctors & Patients 168:** 86-88

Risks and benefits of vegetarianism

A review article looks at the benefits and risks of vegetarian/vegan diets based on large scale studies such as the 27,500 Seventh Day Adventists over a period of 21 years. Vegetarian diets reduce the risk of obesity, cardiovascular diseases, hypertension, some types of cancer, constipation, gallstones NIDD diabetes and may result in higher longevity. However, people on vegan diets (without animals products whatsoever) may be at increased risks of iron, zinc, calcium, vitamin B12, vitamin D3 and omega-3

Continued on follwing page -->

fatty acid deficiencies. Breast-fed infants of vegan mothers have lower rates of growth in first five years of life. Although soy products contain vitamin B12-like substances, they have little activity in humans. The same is true of vitamin B12 analogues in spirulina. Noria and chlorella seaweed do appear to provide some biologically active vitamin B12, but vegans consuming these foods were still found to be deficient.

Walter P (1997), Effects of vegetarian diets on aging and longevity [Review], **Nutrition Reviews 55:** (II)S61-S68 (refs: 49)

INTERNATIONAL CLINICAL NUTRITION REVIEW

By Editor

Dr Robert Buist, Editor in Chief of the ICNR, has indexed the **International Clinical Nutrition Review** which will be updated in the last issue of each year.

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Serious students might also consider the one year correspondence course in Nutritional Medicine. The course is registered with ATMS and the $850 fee includes all texts, tapes and study guides as well as subscription to ICNR.

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Do frequent meals affect glucose levels?

Hypoglycemics and diabetics are usually advised to have frequent three hourly snack to reduce the insulin load and level out glucose levels. Thomson and co-workers claim that results of groups of NIDD patients did not show any differences in glucose or insulin responses, when they were placed on a 3 meals per day or 8 meals a day routine. There was a slight decrease of HDL with 8-meal diet. The trial lasted two weeks, and this may be a criticism (Editor). However Jenkins still maintains that the more frequent meals a day benefit both diabetic and non-diabetic subjects, but agrees that longer-term studies are required to clarify the matter.

Thomson C, Christiansen C, et als (1997), Comparison of the effects of two weeks' intervention with different meals frequencies on glucose metabolism, insulin sensitivity and lipids levels in non-insulin-dependent etc., **Ann Nutr Metab 41:** 173-180

Jenkins DJ (1997) Carbohydrate tolerance and food frequency, **Br J Nutr Suppl 1:** S71-81

Women athletes may experience more knee injuries

Researchers from the UCLA School of Medicine have found oestrogen receptors in the fibroblasts affecting the anterior cruciate ligament - an important ligament in the knee - which could explain why women athletes may suffer more knee injuries. Oestrogen levels at the site were observed to fluctuate. Increased levels of oestradiol, such as occur during the menstrual cycle or when using oral contraceptives are associated with decreased collagen synthesis. This could dispose women athletes to injuries of this nature.

Liu SH, Al-Shaikh RA (1997), Oestrogen affects cellular metabolism of the anterior cruciate ligament, **Am J Sports Med 25(5),** 704-709

THE HYPOGLYCEMIC HEALTH ASSOCIATION
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Phone: _____ **Age:** _____

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Life Membership $150

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NEW MEMBER ☐

Occupation _____

Do you have hypoglycemia? YES/NO Does a family member have hypoglycemia? YES/NO

1998 *MEETING DATES ON SATURDAYS*

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