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.

TAKE NOTE that on the 4 March 2000 we will have an **Annual General Meeting** one half hour earlier before the lecture given by Dr George Samra. A copy of the Statement of Income and Expenditure for the year ending 31 December 1999 and Auditor's report will be made available at the meeting. We thank Hugh Macfarlane, Chartered Accountant, for his free time given to the Association in the preparation of these documents.

This Association has been in existence for the last fifteen years and much has been achieved in having *hypoglycemia* recognized as a disease entity by the medical profession. It is clear that hypoglycemia - more appropriately called *dysglycemia* - is the forerunner of diabetes and closely related to many other metabolic disorders. Many forward-looking health practitioners such as our own Dr George Samra, Dr Chris Reading, Dr Ian Brighthope, Dr Joachim Fluhrer, Dr Robyn Cosford, Dr Paul Ameisen, Daniel Baden, Dr Robert Buist, Dr Mark Donohoe, Roger French, Dr John Hart, Dr Katrina Watson, not forgetting Don Pemberton and Bill Vayda and many many others who have given talks at our public meetings are forging a new direction in orthodox medicine, known as *complementary,* or *integrative medicine.* Special courses are being designed to prepare health practitioners for this new millennium, albeit mostly outside traditional universities. It should not be forgotten though that this momentum stems from an educated and informed public - the

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Our Next Public Meeting will be at 2.00 PM
on Saturday, the 4 March, 2000
at ***YWCA***
2 Wentworth Ave, SYDNEY
and our guest speaker is

Dr George Samra

MB, BS (Sydney), FACNEM

who will be speaking
on the subject of

"How Hypoglycemics can prevent Diabetes"

DR GEORGE SAMRA is of course well-known to our members. He is the patron of our Association as well as a pioneer in Nutritional Medicine. It is mainly through the personal effort by Dr George Samra that the concept of hypoglycemia is recognised as a major cause of ill-health and an important factor in human behaviour. Naturally, since the foundation of the Association the concept has broadened to include the whole range of clinical nutrition and ecology, as well as traditional medicine. Dr George Samra is now well-known among probation officers, the judiciary and legal profession in assisting them to determine to what extent a program of rehabilitation can prevent criminal behaviour. He also specialises in allergies. Dr Samra's surgery is located at the Total Therapies Medical Centre in Kogarah, practising with like-minded practitioners.

Dr Samra's chosen topic should prove to be very interesting.

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 and Newcastle University. The Association will provide free copies in PDF format to any library upon request to jurplesman@hotmail.com

Donations by professionals

Many professionals have donated $50 to the Association and we have acknowledged this by **printing their business card** in the Newsletter. We hope to receive more of these requests, which would help to financially sustain the Association and be of benefit to the doctors and practitioners.

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**

Dr George Samra's book

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

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

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 Ambulance 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

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 4 December 1999, lucky Doug Reay won both the raffle and the lucky door price.

Fund raising activities

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

Ms Bousfield has requested us to place an ad in this Newsletter calling for interested members to start a discussion group in the Gymea area. Please call Ms Bousfield at **9525-9178**

Lyn Grady of Bowral has donated a hand-knit cardigan worth $200 to be raffled after the sale of 50 cent tickets avaiable at Dr George Samra surgery, 19 Princes Highway Kogarah, and also at the public meeting on the 9 September 1999.

The Association wishes to thank **Dr George Samra** for hiring the room at YWCA on behalf of the Association.

Please note that the Editor Jurriaan Plesman can now be reached on the internet. His e-mail address is: jurplesman@hotmail.com and fax No: 02 9130 6247 The Editor would like to hear from any member with internet facilities to help him out with typing manuscripts.

My Success Story

by Bev Cook

My qualifications to relate my 'story' is the fact that I was diagnosed some 14 years ago by Dr George Samra of Kogarah Total Therapies Medical Centre, as being a hypoglycemia sufferer.

A four hour Glucose Tolerance Test established I had type III, ie., combination of type I and II, because there is a history of diabetes in my father's family and I reacted to sugar as well. Over ensuing weeks a further pathology test (cytotoxic) revealed I also reacted to quite a number of daily consumed foods - in other words, lots of allergies, viz. wheat, sugar, milk, caffeine, all night shades, eggs, yellow food colouring and food preservatives, yeast in the main. Later after the avoidance of the foregoing, a trial testing of each 'offender' established the correctness of the pathology cytotoxic test and which foods produced the worst reactions - hence I was all too happy to avoid these until my immune system was built up to a point I can tolerate small indulgences of the allergy foods.

How did I get to Dr Samra?

From teenage years I suffered worsening migraines and sinus problems. In my early 40's other symptoms were increasing. I noticeably was pushing myself through each day - until at age 49 I had repeated visits to my local GP due to lack of energy, depression, increasing pain throughout the body. Each pathology test and examination revealed nothing. Finally, at 50 years of age my doctor said the recent test showed I was like a 17 year old athlete, but visibly there was something radically wrong. My doctor was baffled to know what it was and so referred me to a rheumatologist at Royal Newscastle Hospital. Seventeen days of every conceivable test with approximately twelve or so doctors and specialists, it was established I had sarcoidosis (inflamed and enlarged lymph nodes) for which there is no treatment. I also had low blood sugar level but *-not to worry about that!!*

At that stage my vision was as if looking through jelly - I couldn't read - I couldn't watch TV, memory gone, could not hold a conversation, words would elude me, written words would be back to front.

After discharge from hospital I was developing a phobia - I just could not leave home on my own. Travelling cars that I normally loved became intolerable. For 3 months after hospitalisation, Wally, my very supportive husband, took me to the hospital clinic each week with 24 hour urine collections, blood tests galore, pulmonary function tests (which I loathed being a claustrophobic) and x-rays. The specialist at the end of 3 months pronounced: "You haven't got any better, you haven't got any worse - but you are going to be like this for the rest of your life". I couldn't comment, but I recall thinking: "No, there has to be something better than this, he has written me off as an invalid!" Paine killers was his prescription.

Well-meaning friend recommended I consult a naturopath and biochemist, which I did to no avail. My husband, Wally, was working night shifts and had to do all the cooking, housework, washing, shopping, as my total energy of each day was used in having a shower and dressing, after which I could only rest. Life was not very joyous for us all those months. I felt my back was to the wall as I had tried everything but could not find the cause of all my problems and I was not coping at all. Having a strong belief and faith in God I prayed for his help - not a miracle - help to find out what was causing the problem so I could cope better. Shortly after my calls for help I met a visiting minister and his wife who were very familiar with my problems, as the wife, Lynette, had suffered similar things all her life until 2 years previously when a friend gave them Dr Samra's book *"The Hypoglycemic Connection"*. Lynette identified her problems after reading the book. She consulted Dr Samra, was diagnosed and was set on a course of treatment which was a major turning point for her. Lynette gave me Dr Samra's address and two weeks later I consulted him and tests revealed that that I had

•Type III hypoglycemia

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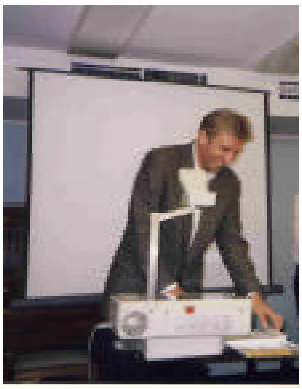
Migraines and Headaches

by
Dr Joachim Fluhrer
edited by Jurriaan Plesman,
BA, Post Grad Dip Clin Nutr



Dr Joachim Fluhrer
giving a talk on Migraine & Headaches

Headaches can be of different kinds. A **tension headache** is usually described as a dull, steady pain that feels like a band tightening around the head.

A throbbing pain around one red watery eye, with nasal congestion on that side of the face is called a **cluster headache.**

A steady pain in the area behind the face that gets worse if you bend forward and is accompanied by congestion is named **sinus headache.** Other categories name **toxic headaches** and **musculo-sketal headaches.** Among the latter is vertebral misalignment as a cause of headache. Another example is misalignment of of the jaw known as TMJ or temperomandibular joint.

These headaches should be distinguished from symptoms of *aneurysm* - a balloon-like swelling in the wall of an artery - or high blood pressure headache, tumor of the brain,when immediate medical help is required.

Migraine headaches are recurring vascular headaches which are usually preceded by an *aura,* in which case it is called a classical migraine. Without the aura it is referred to as a common migraine, but its difference is of little therapeutic value. The common migraine develops slowly and may last from two to seventy hours. Another kind of migraine is called *migrainous cranial neuralgia* characterized by closely spaced episodes of excruciating migraines occurring in clusters within a few days or weeks which may then be followed by relatively long remission periods.

The earliest phase of a migraine headache - the prodromal aura - may be characterized by visual disturbances, flickering lights, waving lines, strange taste or odor, numbness, tingling, vertigo, tinnitus, or a feeling that part of the body is distorted in size or shape. The acute phase may be accompanied by vomiting, chills, excessive urination, sweating, facial edema, irritability and extreme fatigue. This is followed by a severe pulsating pain on one side of the head, sensitivity to light, loud noises and nausea. The pain may be so severe that the victim is forced to lie down in a quiet, darkened room until he recovers. Aspirin seldom provides relief during attack, although ergotamine - a drug that causes dilated vessels to narrow - may prevent the headache from developing when used in combination of other drugs (caffeine, phenobarbital, belladonna). But ergotamine can be addicting as well as being risky in patients with vascular disease. Any other drug seems to treat the symptoms rather than its cause. The word migraine comes from the Greek *hemikrania*, meaning appropriately "half a skull".

Migraine headaches occur more frequently among women (25-30%) in which the hormone estrogen is thought to play a role. Around the time of menstruation when estrogen levels are low, women typically get migraines. Male migraine sufferers account for (15-20%). It often begins in childhood characterized by colic, abdominal pain, vertigo and motion sickness. Migraines peak in the age group of 20-25 years and tend to decline with age.

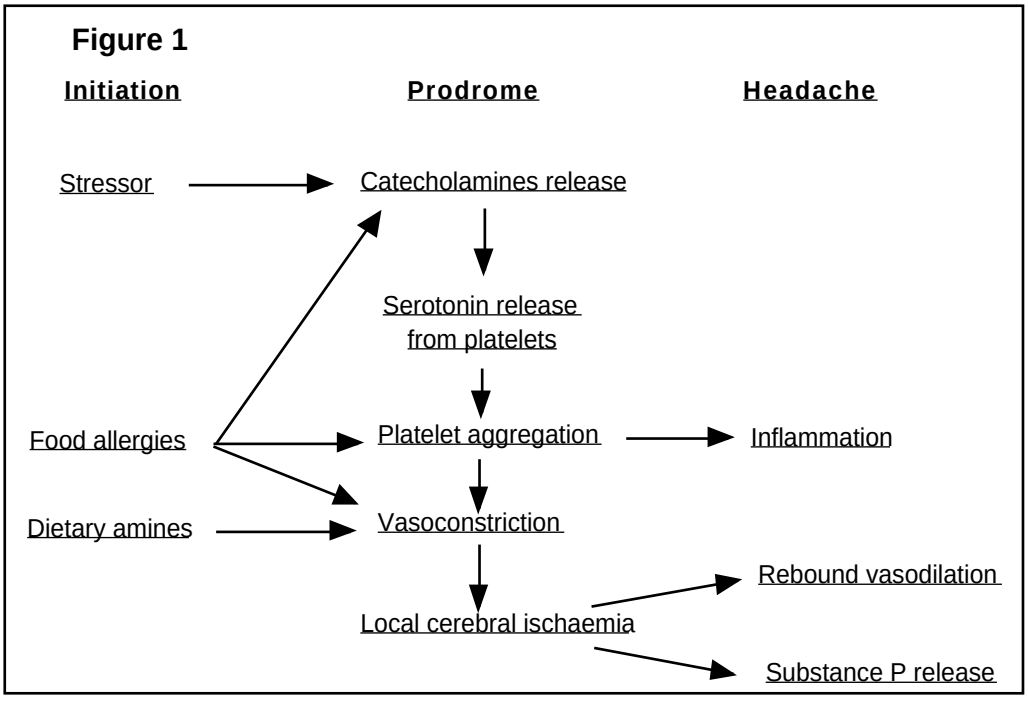
Studies have shown that among migraine sufferers 16% definite and possibly 15% of these patients are found to have a protrusion of one or both cusps of the mitral valve in their heart, known as **mitral valve prolapse (MVP)**, compared to 7-8% in a control group. MVP is closely associated with damaged platelets and is prone to aggregation of platelets. 85 per cent of mitral valve prolapse patients have latent tetany due to chronic magnesium deficiency, which is a common finding among migraine suffering population in general.

The exact mechanism responsible for the disorder is gradually being uncovered, although many questions remain unanswered. The head pain is related to the dilation of cranial vessels. A greatly increased amount of vasodilating polypeptide (a long chain of amino acids chained together by peptide bonds) related to *bradykinin* has been found in tissues of migraine sufferers. Bradykinin refers to naturally occurring polypeptide of non-protein origin consisting of nine amino acids; it is a powerful vasodilator and causes contraction of smooth muscles.

A generally accepted model of all the factors that may be involved in the development of migraine may be found in illustration **Figure 1** which shows that there are three main categories that may initiate migraine headaches.

Stressors refer to any physical or emotional stress that is capable of causing the release of catecholamines or stress hormones in the body. Thus a domestic argument or the strain in a work situation, or emotional changes especially "let-downs" just like any physical illness may provoke headaches via the release of catecholamines. Early reports for the treatment of migraine include psychotherapy as one component. Things that can trigger migraine in susceptible individuals include allergies, too much or too little sleep, constipation, emotional upsets, liver malfunction, hormo-

Figure 1



nal changes, sun glare, lack of exercise changes in weather and barometric pressures, dental problems; even smoking may affect blood vessels.

Catecholamines (or stress hormones) belong to a group of physiological important substances or neurotransmitters, including adrenaline, noradrenaline and dopamine which influence the functioning of the sympathetic and central nervous system. We know for example that an unusual fall in the blood sugar level can trigger the release of adrenaline which in turn functions to stimulate the conversion of glycogen into glucose (mainly in liver cell). This increases blood sugar concentrations. Thus hypoglycemia or unstable blood sugar levels may be one of the causes of migraines. These stress hormones release serotonin from platelets - the smallest cells in the blood - and normally involved in coagulation of blood.

Serotonin (a derivative of tryptophan, an essential amino acid) is a hormone produced by blood platelets that constricts blood vessels. Thus the first phase in the development of migraine is the constriction of blood vessels in the brain.

The release of serotonin is the body's first line defence against damage to blood vessel walls. It causes platelet aggregation or the clumping together of white cells and in turn releases inflammatory substances, biologically active compounds that occur in leukocytes, such as leukotrienes, prostaglandins etc.. It is interesting to note that a patient with migraine seeks the comfort of a dark and quiet room. One could speculate that perhaps he seeks to rid the body of excess serotonin by converting it into melatonin - a hormone secreted by the pineal gland during darkness that induces sleep and is derived from serotonin. Melatonin is also released from cells in the GI tract, but does not seem to be related to the circadian cycle.

The resulting vasoconstriction is responsible for **local cerebral ischemia** in the brain which follows a decreased supply of oxygen to that part of the brain. Like all ischemias it is accompanied by excruciating pain and organ dysfunction, like the eschemic pain of angina.

What happens next is the opposite to the effects of excess serotonin: **rebound vasodilation**. This may be due to the release of **substance P** or a polypetide neurotransmitter substance that is synthesized by the body and acts to stimulate vasodilation of blood vessels feeding the various parts of the body. There seems to be an imbalance between two antagonist hormones: serotonin and substance P trying to reestablish harmony among opposing forces. The very low and critical levels of blood flow to the brain during the prodromal stage is now followed by increased blood flow for up to 48 hours.

We have been following the flow emanating from stressors in **Figure 1.** But there are other factors at play in the development of migraines. One of these is the influence of food allergies.

Food sensitivities is implicated in 60% of attacks of migraine, which has often led investigators to assume that migraine is a food allergy. A list of foods that are found by studies to be involved with food allergies and intolerance is found in **Figure 2.**

Certain foods are more likely than others to provoke migraines and among these are **vasoactive amines.**

Amines are not allergens since they act above a certain threshold of tolerance, unlike allergens. The level of tolerance to amines differ among individuals which explains partially the unpredictability of migraine attacks. Amines are chemical compounds that contain nitrogen. They are formed by the decarboxylation of amino acids, the basic units from which proteins are made. They are important in foods. Phenylethylamine (formed from phenylalanine), tyramine (formed from tyrosine), and tryptamine (from tryptophan) are some of the amines responsible for migraine attacks. *Tyramine* are found in ripened cheeses, chicken liver, pickled herring, fermented sausages, sour cream, red wine. *Phenylethylamine* is found in chocolate, yeast, wines and fermented foods. They stimulate the sympathetic nervous system, and can cause high blood pressure, and they may initiate migraines. Amines are deactivated by the enzyme monoamine oxidase and diamine oxidase, and some antidepressants such as monoamine oxidase inhibitors, inhibit this enzyme. Studies show the activity of this enzyme is considerably reduced in platelets of migraine sufferers, except during pregnancy. Its activity is dependent on the coenzyme magnesium and vitamin B6 and this may explain why supplementation with vitamin B6 may reduce migraine after consumption of monosodium glutamate (The Chinese Restaurant syndrome). Consumption of alcohol blocks the activity of diamine oxidase.

Figure 2
Migraine Headaches
FOOD ALLERGY/INTOLERANCE

Common Foods

Cow's Milk	57-67%
Wheat	43-57%
Chocolate	26-55%
Egg	22-60%
Orange	13-52%
Benzoic acid	35%
Cheese	32%
Tomato	14-32%

Other possible sources of allergies:
Tartrazine, Rice, Rye, Fish, Grapes, Onions, Soy, Pork, Peanuts, Alcohol, MSG, Walnut, Beef, Tea, Coffee, Nuts, Goat's Milk, Corn, Oats Cane Sugar, Yeast

Bowel toxaemia can contribute to migraines. Colonic bacteria - streptococcus faecalis and E. coli - convert tyrosine to tyramine one of the vasoactive amines responsible for migraines in susceptible people. The forerunner of tyrosine is phenylalanine found in almonds, avocados, bananas, dairy products, lima beans, pumpkin seeds, sesame seeds and many others. One of the remedies against this conversion are herbs that contain the alkaloid, berberine, that blocks this conversion. Berberine (a herbal antibiotic) is an ingredient of Barberry (Berberis vulgaris), Chen's Barberry (Berberis chengi), Columba (Jateorhiza palmata, Cocculus palmata), Chinese thoroughwax (Bupleurum falcatum), Goldenseal (Hydrastis canadensis), Oregon graperoot (Berberis aquifolia).

Nitrites are substances added to meats as preservatives and turn them red. They may provoke migraine attacks in susceptible people. Nitrate is reduced to nitrite in saliva, which then reacts with dietary amines to form nitrosamines. This reaction is catalyzed by dietary factors such as thiocyanate, iodide, and tobacco smoke. Best protection is vitamin C + E or antioxidants. Nitrites are found in bologna, hot dogs, salami, sausage.

Among the food additives must be counted **aspartame** and other artificial sweeteners that can provoke migraines.

Caffeine contained in coffee, soft drinks and tea (herbal teas may be caffeine-free) can cause migraine attacks among heavy coffee drinkers (4 to 5 cups of coffee). When they abstain from coffee, they may activate caffeine-withdrawal headache often worsened by exercise.

Lactose (milk sugar) has been re-

Figure 3 **Migraine Headaches**
Food Allergy/ Intolerance

Study	% Responding	Method
Mansfield	30	Elimination
Carter et al	93	Oligoantigenic diet
Hughes et al	80	Fasting, Rotation Elimination
Egger et al	93	Elimination
Monro et al	70	RAST, Elimination, Sodium cromoglycate
Grant	85	Elimination

ported as a major cause of migraine headaches among several researchers. Of all the allergies, milk seems to top the lists of food to be avoided by sensitive people. see **Figure 2.**

Histamine is compound found in all cells, produced by the breakdown of histidine. It is released in allergic, inflammatory reaction and causes dilation of capillaries, decreased blood pressure, increased secretion of gastric juice, and constriction of smooth muscles of the bronchi and uterus. Histamine is released in large amounts after skin damage producing flushing and weals. Excessive histamine (>0.2 ng/ml) can provoke migraine attacks in sensitive people. It is increased by alcohol consumption, histamine in food and by spontaneous histamine release. Food allergies can produce excess histamine. Its activity is blocked by H1-blockers.

If histamine cause headaches a diet low in these compounds (cheese, red wine, tomatoes, some fish) must be followed. Substances which inhibit the main histamine metabolizing enzyme diamine oxidase need to be avoided. Diamine oxidase is located in the jejunum - that part of the small intestine between the duodenum and the ileum - and is the major histamine-metabolising enzyme in the GIT. It is also present in the liver, blood and kidneys. Alcohol, other amines and more than 90 pharmaceutical drugs inhibit this enzyme, resulting in increased plasma histamine levels. Among these are clavulanic acid, dihydralazine, isoniazide, metoclopramide, promethazine, verapamil.

Findings suggest that histamine intolerance is associated with vitamin B6 deficiency, since it is an essential cofactor to the enzyme.[1]

Dehydration has been noted as another factor in the genesis of migraines. It disturbs the balance of essential electrolytes: sodium, potassium and chloride. This may follow a bout of diarrhoea, prolonged fever, vomiting, acidosis and other conditions that deplete body fluids. Signs of dehydration are abnormal skin colour, flushed dry skin, coated tongue, diminished urination, irritability, and confusion.

Magnesium deficiency could easily be regarded as one of the major factors in the etiology of migraine attacks. Magnesium concentrations has an effect on serotonin receptors and neurotransmitter. It has been estimated that 50 per cent of patients during an acute attack of migraine have lowered levels of ionized magnesium.[2] [3]

Treatment

With food allergies and intolerance being a major factor it is no wonder that programs that include avoidance of allergies and food sensitivities has been widely reported as being most successful as reported by several studies. See **Figure 3.**

It is important to investigate and identify the food and/or chemical sensitivities of the patient. The practitioner may consider a course in stress management if psychological stress is seen as a major trigger in migraines.

The diet should be closely watched, especially in regard to water intake, caffeine and alcohol intake. The patient should be encouraged to adopt a "clean diet". His or her medication should be reviewed and replaced with those that do not interfere with the metabolism of diamine oxidase.

A program of bowel and liver detoxification should be undertaken. Thus a therapeutic approach involves;

- Identification and avoidance of preciptating factors
- Avoidance of initiators described above
- Attention to diet
  - ° Elimination of food allergies
  - ° Rotation Diet
  - ° Avoidance of vasoactive amines (initially)
  - ° Low animal fat
  - ° Foods high in ingredients that inhibit platelet aggregation such as Essential Fatty Acids (EFAs), garlic, onion, fish

Nutritional Support

- B-complex - stress support
- B6 - if histamine intolerant and cru cial for diamine oxidase activity
- Magnesium and calcium - relaxation vascular and skeletal muscle, antiinflammatory
- Vitamin C and bioflavonoids
- Fish oil - anti-inflammatory, inhibit PGE2 and platelet serotonin release.

Herbs

- St Mary's thistle, Dandelion root (liver tonics), Goldenseal
- Passionflower, Skullcap, Chamomile, Valerian (nervines)
- Feverfew, Ginger (anti-inflammatory

Others

- **Manipulation,**Osteopath, Chiropractor, Physio, Massage
- TMJ Dysfunction
- Relaxation training/biofeedback
- TENS
- Acupuncture

References:

1)Jarish R, Wantke F,**Int Arch Allergy Immunol, 1996; 110:** 7-12 Refs 45

2) Mauskop A, Altura BM; Role of magnesium in the pathogenesis and treatment of migraines, **Clin Neurosci 1998: 5(1):** 24-7

3) Mishima K et als; Platelet ionized magnesium, cyclic AMP, and cyclic GMP levels in migraine and tension-type headache; **Headache 1997, Oct; 37(9):** 561-4

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medical consumers - who demand a change of medical thinking without which the push towards *complementary medicine* would fail. The demand for modern medicine, especially in the area of degenerative diseases, will grow stronger as time goes on and as medical knowledge falls into the public arena. Nutritional supplements are often beyond the reach of those who need them most. Only those who can afford it seem to have the benefits of complementary doctors, but the vast majority of the patient population are forced to rely on government funded medicine, reliant almost exclusively on expensive hi tech procedures and pharmaceuticals. Thus the struggle for access to complementary medicine by all is just beginning. As a member of this Association you can contribute to this movement by attending our public lectures given by complementary doctors and health practitioners. This will enable you to understand the nature of health, improve your well-being, support and choose the right health practitioner if needed.

Bev Cook Continued from page 2

- •Several food allergies or sensitivities
- •A large problem with candida overgrowth
- •Chronic fatigue - (M.E.) Fibro myalgia
- •Salicylate intolerance
- •Gluten intolerance
- •Very low levels of essential body miner als
- •Very high toxic levels of aluminium,

Then of course there was sarcoidosis, the only condition the hospital system was able to diagnose.

So with all the foregoing physical problems, no wonder my life was such a misery! I was so happy to have an answer to my prayer asking for help to find the cause of all my woes and give direction. I started to cope better each day - each week - seeing a light at the end of a long painful tunnel.

No doubt many readers can identify with at least some if not all the problems which concerned me. I have had many people phone me asking me the address of a local doctor who would be able to help with the symptoms I experienced.

I recommend a short cut - and happily gave them Dr Samra's phone number.

Researchers cite possible link between autism, schizophrenia and diet

By Melanie Fridl Ross
<http://www.health.ufl.edu/post/post0399/post03_19/1.html>

Findings from two novel animal studies indicate autism and schizophrenia may belinked to an individual's inability to properly break down a protein found in milk, UF researchers report in this month's issue of the journal Autism.

The digestive problem might actually lead to the disorders' symptoms, whose basis has long been debated, said UF physiologist J. Robert Cade, M.D.,cautioning that further research must take place before scientists have a definitive answer. When not broken down, the milk protein produces exorphins, morphine-like compounds that are then taken up by areas of the brain known to be involved in autism and schizophrenia, where they cause cells to dysfunction.

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Osteoporosis

by Nicola Reavley

Bookman Press
Level 10, 227 Collins St, Melbourne 3000
with kind permission to publish

Osteoporosis, which literally means "porous bones", is the result of a long-term decline in bone mass which, in severe cases, causes the bones to break under the weight of the body. Particularly badly affected bones include the spinal vertebrae, the thigh bone and the radius (shorter arm bone). Over 25 million Americans may be affected by osteoporosis and 80 per cent of those are women. Although the problem also occurs in men, postmenopausal women are particularly susceptible, with around 35 per cent of women suffering from osteoporosis after menopause.

Symptoms of osteoporosis

The symptoms of osteoporosis are often absent until fractures occur, although in some cases there may be a loss of height, a hunched back or back pain. Osteoporotic fractures affect 50 per cent of women and 30 per cent of men over 50. These fractures are particularly serious as demineralized bones shatter when they break and usually take longer to heal. Radiological examination can be used to measure bone mineral density and assess the risk of fracture.

Causes of osteoporosis

Around 35 per cent of women suffer from osteoporosis after menopause and, although it is less common, the problem occurs in a similar way in men. Osteoporosis is more common in Caucasians and Asians because they are often smaller boned.

Most of the bone loss seen in osteoporosis in women occurs in the first five to six years after menopause due to a decline in circulating female hormones and an age-related reduction in vitamin D production. Genetic factors seem to play a part in osteoporosis but behavioral and hormonal factors may be more important. Sufficient body fat and muscle are necessary to keep hormone levels high enough to maintain bone mineral content. Athletes and premenopausal women whose menstrual periods have stopped may also be at increased risk of osteoporosis due to alterations in their hormone levels.

Adequate intakes of calcium, vitamin D, magnesium and boron are also necessary. Diets high in dairy products, protein, sugar, alcohol, salt, caffeine-containing drinks and very high in fiber also seem to increase the risk of the disorder, most likely due to effects on mineral absorption and metabolism. People on weight-reducing diets are also at risk as they avoid foods high in bone-building nutrients.

Inactivity leads to an increased risk of osteoporosis, as does gastric surgery and certain types of medications such as corticosteroids.

Treatment of osteoporosis

The conventional treatment for osteoporosis is estrogen therapy but this is not suitable for some women due to the increased risk of breast cancer. Some women are treated with calcitonin, a hormone that inhibits removal and promotes formation of bone. It is available in injection forms and as a nasal spray. Intake of calcium and vitamin D must also be adequate. Newer osteoporosis drugs include alendronate, which inhibits bone breakdown; and raloxifene, a selective estrogen receptor modifier.

Osteoporosis prevention

Exercise

Regular exercise plays a vital part in preventing loss of bone mass. Weight-bearing exercises such as walking, jogging and yoga contribute to increases in bone density and prevention of bone loss. Exercise also helps build muscle mass which can help protect bones from injury. It also improves strength and flexibility, decreasing susceptibility to falls.

Diet

A healthy diet can reduce the incidence of osteoporosis by ensuring the development of a favorable peak bone mass during the first 30 to 40 years of life. Adequate nutrient intake early in life is vital for bones to reach their maximum density so that they are strong enough to support the body even when they lose mass later in life. However, it is never too late to slow the bone loss seen in osteoporosis, and early postmenopausal years are an important time to ensure optimal intake of nutrients including calcium, magnesium, boron and vitamin D.

Recent research suggests that including soybeans in the diets of postmenopausal women may decrease the risk of osteoporosis. Soybeans contain compounds called phytoestrogens which act in a similar way to estrogen and have beneficial effects on bone mineral density.

Caffeine-containing drinks can increase the loss of calcium in the urine. Diet soda drinks which contain phosphoric acid can alter the calcium phosphorus balance and contribute to calcium loss from the bones. Consuming large amounts of these drinks can increase the risk of osteoporosis. Nicotine and alcohol also adversely affect bone mineral density. High salt intakes seem to increase calcium excretion, lowering bone mineral density and increasing the risk of osteoporosis. In a study published in 1995, Australian researchers investigated the influence of urinary sodium excretion on bone density in a 2 year study of 124 postmenopausal women. The results showed that increased sodium excretion was linked to decreases in bone density.[1]

While dairy products are good sources of calcium, there is concern that their protein content can actually increase the loss of calcium from bone. Researchers involved in the Nurses Health Study analyzed the diets of over 77 000 participants in the study and looked at the rates of bone fractures. Results showed that women who drank two or more glasses of milk per day had around a 45 per cent increased risk of hip fracture and a 5 per cent increased risk of forearm fracture compared to women who drank one glass or less per week. There was also no drop in risk with intake of calcium from other dairy foods.[2] A varied diet which includes nondairy sources of calcium is likely to be more beneficial in protecting against osteoporosis.

Vitamins, minerals and osteoporosis

B vitamins

B vitamin deficiencies may contribute to osteoporosis, particularly those of folate, vitamin B12 and vitamin B6. This may be partly due to the effects of increased homocysteine levels on bone metabolism.

Vitamin D

Vitamin D regulates the absorption and use of calcium and phosphorus, which are vital for normal growth and development of bones. Vitamin D is necessary for calcium absorption and increases the deposition of calcium into bones. In cases of vitamin D deficiency, the body increases production of parathyroid hormone which removes calcium from the bones and leads to bone thinning.

Research suggests that there may be a genetic link between vitamin D receptor types and osteoporosis. It is also possible that patients with osteoporosis have impaired conversion of vitamin D to its most active form. The ability to produce vitamin D in the skin

may decline with age and bone loss may increase in the winter months when people have less exposure to sunshine. People with a certain type of vitamin D receptor may be more susceptible to osteoporosis, and research suggests that women with different types of vitamin D receptors respond differently to vitamin D supplements.[3]

A study done in 1997 at Tufts University in Boston showed reduced rates of bone loss and fractures in men and women over 65 who took calcium and vitamin D supplements. Researchers assessed the effects of calcium (500 mg per day) and vitamin D (700 IU per day) on 176 men and 213 women aged 65 years or older. After a three-year period, those taking the supplements had higher bone density at all body sites measured. The fracture rate was also reduced by 50 per cent in those taking the supplements.[4]

Vitamin D supplements may also be useful in preventing bone loss in patients taking corticosteroid drugs. In a study published in 1996, researchers at the University of Virginia found that calcium and vitamin D supplements helped prevent the loss of bone mineral density in those taking the drugs for arthritis, asthma and other chronic diseases.[5] Vitamin D supplements may also be useful in reducing the risk of osteoporosis due to long-term use of anticonvulsant drugs.

However, other studies have not shown any reduction in fracture rates in those taking vitamin D supplements. A 1996 study which was carried out in Amsterdam looked at the effects of either vitamin D or a placebo on 2500 healthy men and women over the age of 70 who were living independently. The participants received a placebo or a daily dose of 400 IU of vitamin D for a three-and-a-half year period. Dietary calcium intake was the same in both groups. Forty-eight fractures were observed in the placebo group and 58 in the vitamin D group.[6]

Vitamin K

Low levels of vitamin K have been seen in sufferers of osteoporosis. In a Japanese study published in 1997, researchers investigated the relationship between bone mineral density, vitamin K levels and other biological parameters of bone metabolism in 71 postmenopausal women and 24 women with menopausal symptoms receiving hormone replacement therapy. The results showed that women with reduced bone mineral density had lower levels of vitamin K1 and K2 than those with normal bone mineral density.[7] Low levels have also been seen in osteoporotic men.[8]

Boron

Boron acts with calcium, magnesium and phosphorus in the metabolism of bone. Deficiency seems to affect calcium and magnesium metabolism and affects the composition, structure and strength of bone, leading to changes similar to those seen in osteoporosis.[9] Combined boron and magnesium deficiency seems to worsen osteoporosis, suppress bone building and cause decreased magnesium concentrations in bone.[10] Supplements of around 3 mg per day have been shown to enhance the effects of estrogen in postmenopausal women. This is likely to contribute to its beneficial effects on bone health.[11] Studies done in 1994 in athletic college women suggest that boron supplements decrease blood phosphorus concentration and increase magnesium concentration. Both of these changes are beneficial to bone building.[12]

Calcium

Osteoporosis is not merely a loss of calcium from bone, although calcium deficiency does contribute to osteoporosis. The National Osteoporosis Foundation estimates that the average adult in the US gets only 500 to 700 mg per day. The US government has recently raised its recommendation for daily calcium intake. For men and women aged from 19 to 50, the RDA is now 1000 mg, and for those over 50 it is 1200 mg.

The new RDA for adolescents is 1300 mg and adequate calcium intake during this time of life plays a vital part in allowing bones to reach their maximum density so that they are strong enough to support the body even when they lose density later in life. Studies suggest that calcium intake in adolescence is often below the recommended levels. Researchers involved in a 1994 USDA study measured calcium intake in 51 girls aged 5 to16 years old. They found calcium intake to be below the recommended dietary allowance for 21 out of 25 girls aged 11 or over. These studies suggest that the current calcium intake of American girls during puberty is not enough to enable bones to develop maximum strength, and that increased intakes may be necessary.[13] A 1993 study published in the *Journal of the American Medical Association* suggests that calcium supplements may be beneficial in adolescent girls. Researchers gave daily calcium doses of 500 mg or placebo to 94 girls and then measured bone mineral density and bone mineral content at the lumbar spine. The results showed that increasing calcium intake led to significant gains in bone mass.[14]

However, it is never too late to slow the bone loss seen in osteoporosis, and early postmenopausal years are also an important time to ensure optimal intake. A 1997 study done at King's College Hospital in London suggests that high calcium intakes are linked to bone mineral density in elderly women. Researchers assessed calcium intake in 124 women aged from 52 to 62 and also measured bone mineral density at the spine, hip and the os calcis bone in the foot. Results showed that women with high calcium intakes had higher bone mineral density.[15] Results from the Rotterdam Study, which involved 1856 men and 2452 women aged 55 years and over, show that high calcium intakes also protect against bone loss in men.[16]

Taking calcium supplements later in life can slow the bone loss associated with osteoporosis, and treatment which combines calcium and estrogen is likely to be better at building bone than treatment with estrogen alone. In a 1998 study, researchers analyzed the results of 31 studies and found that the postmenopausal women who took estrogen alone had an average increase in spinal bone mass of 1.3 per cent per year, while those who took estrogen and calcium supplements had an average increase of 3.3 per cent. Increases in bone mass in the forearm and upper thigh were also greater in women taking supplements. The added benefit from the calcium was seen when the women increased their intake from an average of 563 mg per day to 1200 mg per day.[17]

It is recommended that postmenopausal women who are not on estrogen therapy consume 1500 mg calcium per day. Multivitamin supplements often do not provide enough calcium and separate supplements may be necessary. Supplements should be taken in divided doses throughout the day, with a maximum of 500 mg being taken at any one time.

Fluoride

Bones seem to be more stable and resistant to degeneration when the diet is adequate in fluoride. Sodium fluoride supplements have been used to treat osteoporosis.[3] Researchers involved in a 1998 study published in the *Annals of Internal Medicine* compared the vertebral fracture rates in 200 women over a four-year period. One group was given 20 mg of fluoride and 1000 mg of calcium daily, and the other group received only calcium. The rate of new fractures in the fluoride group was 2.4 per cent compared to 10 per cent in the calcium only group.[18] Sustained release of fluoride in doses of 23 mg per day appears to be more beneficial than forms which are quickly absorbed from the gut.[19] However, a 1996 study done in Argentina suggests that the increases in bone mineral density are not maintained after sodium fluoride therapy is stopped.[20]

The treatment of osteoporosis with fluoride supplements is controversial as there is the possibility that fluoride bone is not always stronger than normal bone. There may be an increase in the number of hairline fractures in the hips, knees, feet and ankles. In 1983/1984, a study of bone mass and fractures was begun in 827 women aged 20-80 years in three rural Iowa communities selected for the fluoride and calcium content of their community water supplies. Residence in the higher-fluoride community was associated with a significantly lower radial bone mass in premenopausal and postmenopausal women, an increased rate of radial bone mass loss in premenopausal women, and significantly more fractures among postmenopausal women.[21] Fluoride therapy may increase the requirement for calcium as more is needed for bone formation.

Magnesium

Magnesium and calcium interact in many body functions including that of bone formation. Women with osteoporosis may have lower magnesium levels than women without the disorder. In a 1995 study, results showed that women whose dietary intakes were less than 187 mg per day had a lower bone mineral density than women whose average intakes were more than 187 mg.[22]

Magnesium is essential for the normal function of the parathyroid glands, metabolism of vitamin D, and adequate sensitivity of bone to parathyroid hormone and vitamin D. Magnesium deficiency may impair vitamin D metabolism which adversely affects bone building.[23] Magnesium deficiency is also known to cause resistance to parathyroid hormone action which affects calcium balance and may cause abnormal bone formation.[24] However, magnesium excess inhibits parathyroid hormone secretion which means that bone metabolism is impaired under positive as well as under negative magnesium balance.[25] Maintaining normal calcium-to-magnesium balance is very important in the prevention of osteoporosis.

Supplements may help to increase bone mineral density in postmenopausal women, thus reducing the risk of osteoporosis. In a 1990 study, US researchers investigated the effect of a dietary program emphasizing magnesium instead of calcium for the management of postmenopausal osteoporosis. Nineteen women on hormone replacement therapy (HRT) received 500 mg magnesium and 600 mg calcium, and seven other women on HRT did not receive supplements. The results showed that in one year, those women given the supplements had greater increases in bone mineral density than those who were not. Fifteen of the 19 women had had bone mineral density below the spine fracture threshold before treatment; within one year, only seven of them still had values below that threshold.[26]

In a 1993 study, Israeli researchers assessed the effects of supplemental magnesium in 31 postmenopausal women who received six 125 mg tablets daily for six months and two tablets for another 18 months in a two-year trial. Twenty-three symptom-free postmenopausal women were assessed as controls. The results showed that 22 patients responded with a 1 to 8 per cent rise of bone density. The mean bone density of all treated patients increased significantly after one year and remained unchanged after two years. In control patients, the mean bone density decreased significantly.[27]

Zinc

Zinc accompanies calcium in the mineralization of bone, and is lost when calcium is lost from bone. Recent research in monkeys suggests that diets low in zinc during adolescence may increase the risk of osteoporosis later in life, as bones may not develop properly.

In a 1996 study, researchers studied zinc deficiency in two groups of ten monkeys. Both groups were given nutritionally balanced diets but one group received 50 mg of zinc per gram of food while the other group only received 2 mg of zinc per gram of food. Eight of the monkeys were then studied throughout their lives to ages equivalent to that of ages 10 to 16 in human girls. The researchers found that the monkeys on low zinc diets had slower skeletal growth, maturation and less bone mass than the other monkeys, with substantial differences noticed in the lumbar spine. The differences were only apparent during rapid growth phases in the monkeys, especially during pregnancy.[28]

Other minerals

Chromium may help to boost the bone-building effects of insulin and may have a role in the maintenance of bone density and prevention of osteoporosis.[29] Copper is necessary for bone formation, and inadequate intake can cause the loss of calcium from bones, reduced bone formation and deformities. Manganese deficiency may also increase loss of calcium from the bone. Silicon may have a role in the prevention and treatment of osteoporosis, and supplements are used to increase bone mineral density.

Herbal medicine and osteoporosis

Herbs used to treat osteoporosis include horsetail (*Equisetum arvense*), oat straw *(Avena sativa*), alfalfa (*Medicago sativa*) and hawthorn (*Crataegus oxyacantha*). Herbs commonly used to alleviate the side effects of menopause include black cohosh (*Cimicifuga racemosa*) and dong quai (*Angelica sinensis*).

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Fenugreek stabilizes Blood Sugar Levels in Hypoglycemics and Diabetics

By Jurriaan Plesman, BA(Psych), Post Grad Dip Clin Nutr

There are an estimated 400,000 people diagnosed with diabetes in Australia, and a further 400,000 that have not as yet been diagnosed according Diabetes Australia. There are at present 120 million people with diabetes in the world and this number is expected to increase by 100 million by the year 2010. Thus according to Diabetes Australia there are 800,000 diabetic Australians, half of whom don't know that they are diabetic. The Australian figure will increase to 1 million by year 2010. Some experts have claimed that in fact we are dealing with an epidemic. As the forerunner of diabetes, the number of people suffering from hypoglycemia is suspected to be far greater than the 3-4% previously estimated.

When we are talking about diabetes we are talking about all the diseases that accompany diabetes such as obesity, atherosclerosis, heart disease, strokes, diabetic neuropathy (loss of sensation in the feet), and diabetic retinopathy leading to blindness, possible amputations of the lower limbs and so on. The medical costs are staggering! Diabetes is an end-disease and this all starts with the first signs and completely ignored by orthodox medicine of what we know to be "hypoglycemia". Many experts now agree that hypoglycemia is precedes diabetes.[1], [2] *The tragedy is that hypoglycemia is so easily diagnosed by a simple 4 hour glucose tolerance test, perfected by our patron Dr George Samra and described in his book,* **The Hypoglycemic Connection.**[3]

What is hypoglycemia?

For those doctors that are unfamiliar we will give the shortest possible description of the disease. Although most doctors are familiar with diabetes, only a few recognise hypoglycemia as a non-diabetic disease entity. The term hypoglycdemia is an unfortunate one. It arose at a time when the connection between hypoglycemia and diabetes was not clear. A more appropriate term would have been ***dysglycemia***, or hyperinsulinism. The confusion arises because in hypoglycemia (dysglycemia) blood sugar levels fluctuate widely from high to low, depending on the individual case This explains some of the psychological symptoms; the mood swings, uncontrollable anger bout, head aches, depression and nervousness and so on. The brain representing about 2 per cent of the body by weight accounts for some 60 per cent of the utilization of glucose by the whole body in the resting state.[4] The body does not seem to respond to insulin produced by the beta-cells of the pancreas. Often excess insulin produced in response to blood glucose - derived from refined carbohydrates in food - drives glucose levels well below the normal, hence "hypoglycemia".

Dr George Samra [4,41] has determined diagnostic criteria for hypoglycemia based on thousands glucose tolerance tests of his patients.

Relative Hypoglycemia - *fall in blood glucose of over 2.8mmol/L (50mg per 100 mls) in any one hour, or over 1.9mmol/L (35mg per 100mls) in any 1/2 hour.*

Absolute Hypoglycemia - *Any blood glucose recorded below 3.4mmoll /L(62mg per 100ml ls)*

Combined relative and Absolute Hypoglycemia - *also know as "relative hypoglycemia".*

Flat Curve response - *where no blood glucose value is more than 1.3mmol/L (24mg per 100mls)*

Fasting Hypoglycemia - *The fasting glucose is below 3.4 mmol/L (62mg per 100mls)*

Cellular Hypoglycemia - *with a normal GTT but usually an abnormal zinc/copper ratio*

Thus we have a very simple test for prediabetes, that could prevent millions of people from developing the dreaded disease at a later stage.

No doubt so long "sugar" is regarded as a non-poisonous substance in our society - and enriching the sugar industry - statistics for hypoglycemics and diabetics will steadily rise. The struggle to have cigarettes recognised as a serious health hazards by the tobacco companies pales into insignificance when it comes to altering the culture of the powerful sugar industry and their lobbyists. We desperately need an alternative to sugar as a major ingredient in our foods. It is surprising that all those people afflicted with either hypoglycemia or diabetes have not come together to bring political pressure to bear on the creation of an alternative to the sugar industry; not a chemical alternative such as aspartame, but rather an organic one such as fructose or better still "Stevia".[5] Diabetes Australia appear to be tame in this respect.

The **treatment** of both hypoglycemia and diabetes are similar: the avoidance of excess refined carbohydrates such as sugar and sugary foods, of saturated fats and the supplementation of vitamins and minerals (especially zinc and chromium). Hypoglycemics and diabetics are very sensitive to oxidative stress which is the major cause of diabetic complications. Thus the addition of antioxidants to the diet is mandatory, and of these perhaps vitamin E (500-800IUs per day) is the most important. Vitamin E has been reported to reverse the development of diabetic neuropathy [6,7,8].

Nowadays most doctors are catching up with the significance of nutritional supplementations in most degenerative diseases, especially hypoglycemia and diabetes. However there remain still a few doctors who tend to deride the virtues of vitamins and minerals more from ignorance than scepticism.

Research on Fenugreek

There are many herbs that have the ability to stabilize blood sugar levels. Juniper berries[9], Coriander, Maitake mushroom, Goat's Rue, Gymnema, Milkthistle, and perhaps another sixty or so herbs have been repoirted as having hypoglycemic actions. Among these Fenugreek, a herb of the legumes family and therefore adaptogenic, has been well-researched in the scientific literature. The herb not only has hypoglycemic activity, but also lipid lowering actions. Fenugreek's constituents are steroidal saponin (diosgenin), alkaloid (trigonelline), bitter principle, mucilage, volatile oil.

The use of Fenugreek *(Trigonalle foenum graecum)* dates back to the ancient Egyptians, Greek and Romans as a herbal remedy for a variety of illnesses involving poor digestion, debility, gastric inflammation, to promote lactation, sore throats, muscle aches, impotence and locally for skin inflammation. Our interest in the herb is in its ability to stabilize blood sugar levels in both hypoglycemics and diabetes.

The seeds of the annual herb growing up to 60cm high with slender smooth stem and slim ovate trefoil leaves have a strong odour lovage and a bitter taste.[10] In fish and some other cold-blooded animals saponins have been known to be toxic, but not in the case of mammals.[11]

In 1986 a study found in a group of diabetic dogs that a subfraction called "a" was responsible for the antidiabetic properties of fenugreek seeds which contained the testa and endosperm and is rich in fibres.[12]

In 1990, Sharma and his colleagues[13] gave 50g with each meal of defatted fenugreek to a group of Indian diabetic patients (Type 1) and found that fasting glucose, urinary glucose excretions, total and LDL serum cholesterol (HDL levels remained unchanged) and serum triglycerides were lowered and the glucose tolerance tests were improved. This would be significant in a country like India where many diabetics cannot buy insulin and other medication. A similar study with 10 type II diabetic subjects produced similar results with a report that erythrocyte insulin receptors were increased.[14]

An ethanol extract from fenugreek seeds was used to study the effects of lipid lowering

in rats by Stark and Madar[15] at a dose of 30-50g/kg for 4 weeks. The reduction in plasma cholesterol levels was 26%. The authors suggested that this effect was due to interference with bile acid absorption.

In 1996, researchers gave sixty poorly-controlled diabetic (NIDDM) subjects 25g of fenugreek seed powder daily in conjunction with a low-fat diet for a period of 24 weeks. They returned to the clinic for 2 hour oral glucose tolerance tests (OGTT) at weeks 4,8,12 and 24. Some subjects complained of minor gastrointestinal disturbances, but these symptoms disappeared after 3-4 days. By the end of the study, the area under the OGTT plasma glucose and insulin curves had decreased by 40%. After only 8 weeks of supplementation, urinary sugar levels and glycosylated haemoglobin[16] levels had fallen by 12% ($p<0.001$). Glycemic control improved in approximately 80% of patients.[17]

Rao and his colleagues investigated the toxic effects of fenugreek in rats at doses of 5, 10 and 20% of diet over a 90-day period. Fenugreek had no effect on weight gain, liver function or a range of blood parameters. The only effect noted was a significant decrease in serum cholesterol in rats consuming 10 to 20% fenugreek diets.[18]

Al-Habori and Raman[19] provide an overview of the lipid, cholesterol and glucose lowering effects of fenugreek. Antidiabetic and hypocholesterolaemic activity is thought to be due to fenugreek's high fibre and saponin content. The hypoglycaemic activity is believed to result from the inhibition of carbohydrate enzymes. Insulin secretion has been shown to be stimulated by*4-hydroxyisoleucine* in vitro and fenugreek may enhance plasma insulin levels in vivo. The lowering of cholesterol has been linked to the galactomannan fibre, saponins and sapogenins. Toxic effects were not reported.

In October 1999, scientists investigated the effect of 4-hydroxyisoleucine (4-OH-Ile) - an amino acid extracted from fenugreek seeds - on normal and diabetic rats (type II). The lactonic form of (4-OH-Ile) had no effect on normal rats. In non-insulin-dependent diabetic (NIDD) rats, a single intravenous administration of 4-OH-Ile (50 mg/kg) partially restored glucose-induced insulin response without affecting glucose tolerance. So, the antidiabetic effects of 4-OH-Ile on NIDD rats result, at least in part, from a direct pancreatic B cell stimulation.[20]

Sauvaire Y, Petit P et als. from the University of Montpellier II in France[21] claim that the stimulating effect of 4-hydroxyisoleucine was found to be strictly glucose dependent; indeed, ineffective at low (3 mmol/L) or basal (5 mmol/L) glucose concentrations, the amino acid potentiated the insulin secretion induced by supranormal (6.6-16.7 mmol/L) concentrations of glucose. The stimulation of insulin was a direct effect on isolated islets of Langerhans from both rats and humans. They showed

1) that the pattern of insulin secretion induced by 4-hydroxyisoleucine was biphasic,

2) that this effect occurred in the absence of any change in pancreatic alpha- and delta-cell activity, and

3) that the more glucose concentration was increased, the more insulin response was amplified.

Moreover, 4-hydroxyisoleucine did not interact with other agonists of insulin secretion (leucine, arginine, tolbutamide, glyceraldehyde). Therefore, they conclude that 4-hydroxyisoleucine insulin stimulating activity might, at least in part, account for fenugreek seeds' antidiabetic properties. This herb may be considered as a novel drug with potential interest for the treatment of NIDDM.

The last report underlines the adaptogenic nature of fenugreek: that is, it has glucose lowering effects when levels are high, but no such effects when glucose levels are low. There is strong possibility that fenugreek provides relief from the debilitating "psychological" symptoms of hypoglycemia - due to extreme fluctuations in blood sugar levels. Daily supplements of the herb by hypoglycemics are expected to alleviate much of their suffering.

Furthermore fenugreek has lipid lowering effects which is of great significance, once hypoglycemia has developed into full-blown diabetes.

Fenugreek powder can be obtained from most Indian spice suppliers and Asian shops for a reasonable price.

Patients should discuss these findings with their health practitioners to see whether they could benefit from fenugreek in the treatment of their condition, whether hypoglycemic or diabetic.

The author would appreciate hearing from members about their experience when they supplement their diet with fenugreek seed powder.

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GLYCONUTRIENTS:
Another Look at Sugars

by Dr Robyn Cosford

We are all acquainted with sugar in Western society. We know it is something to make things sweet and provide energy, and think of glucose as being the final common pathway for the metabolism of all sugars. Recent research however is revealing that there is more to sugar than sweetness, and more than one sugar relevant to our health.

Sugar breaks down to the mono(one)-saccharides glucose and fructose (the fruit sugar), and the fructose is further metabolised to glucose. Milk sugar (lactose) breaks down to glucose and galactose. These (glucose and galactose) are only two of some 200 monosaccharides found in nature. Eight of these naturally occurring monosaccharides are found in large amounts in the human body, but not as part of the energy producing pathways. They occur as structural and functional glycoproteins and glycolipids.

Glycoproteins are sugar-containing protein molecules. The sugars contained may include glucose and galactose, but more often contain multiple mannose units, linked to other monosaccharides, such as fucose, xylose, n-acetylglucosamine, n-acetyl-galactosamine, or saliac acid, with some glucose and galactose units. These monosaccharides have the amazing capacity to bind in a multiplicity of branching units - four monosaccharide units can be joined in over 100,000 different ways whereas four amino acids can only be joined in 24 different ways. This ability to form such an array of different molecules makes them ideal candidates to be information-rich molecules in many biological processes, such as for cell to cell messages, for example. Indeed, this is one of the functions of glycoproteins. Our cells float in our extra-cellular fluid, which comprises some 60 per cent of our total body, and this fluid is full of glycoproteins. These make contact with our cells via specific receptors for the sugars of the glycoproteins (usually glycolipids) activating specific cellular activities. Glycoproteins are also structural, occurring as part of the cell membrane, but always facing outwards. In this capacity, they function in cell to cell recognition.

Glycoproteins are particularly relevant for the immune system. For example, a carbohydrate called **Lewis X** *(sialic acid-galctose-fucose-N-acetylglucosamine)* has been found to be a glycoprotein in the cell walls of the white blood cells, which binds to receptors on cells lining the blood vessels, and thereby starts an immune reaction. Antibodies are actually glycoproteins, and function to enable white blood cells to recognise 'foreign' substances and bind to them to destroy them. Perhaps the documented, ability of sugar to temporarily depress the immune system is via disruption of the cellular communication as a result of being flooded with excess glucose.

The nervous system is also very dependent on these sugars for intercellular communication, with cell-surface carbohydrates believed to be the mechanism by which developing neurones communicate to establish brain pathways. Gangliosides - sugar containing fats, or glycolipids - make up 6% of the total brain fat and have an important function in brain development and function.

Glycoproteins are made in the body from monosaccharides glucose, galactose, mannose and xylose. The body easily makes fucose, sialic acid and N-acetylglucosamine from mannose. Should mannose or the other monosaccahrides be deficient, mannose, and all the other sugars can be manufactured from glucose and galactose, but require multiple enzyme steps, and much cellular energy, and mistakes can occur.

Out typical Western society diet provides ample sugar, giving glucose and fructose, ample milk giving more glucose, with galactose, and some fruit, providing some fructose, but little else by way of these monosaccharides. Even our babies are deficient, as infants are fed less and less breast milk. We think of breast milk as having only milk-sugar, or lactose, but studies are revealing that over 100 different oligosaccharides, rich in these monosaccharides, are found in milk. These may have special significance in blocking the ability of gut bacteria to bind to the gut wall and cause infection. The protective effect of breast feeding on reducing the incidence of gastrointestinal infections is well-known, as is the effect on protecting against illness in general, and reducing the incidence of allergies. Perhaps some of these effects are mediated via providing these monosaccharides, to help immune function.

Supplementation of glyconutrients (specific formulations rich in these monosaccharides, especially mannose) has been found to improve function (in lab studies) by up to 50% in normal individuals, and 400% in people with Chronic Fatigue Syndrome. Clinical studies are indicating their benefit in learning and behavioural difficulties, cancer, auto-immune disease, and many other conditions where immune dysfunction is a feature.

As always, whole food, natural sources are preferable where possible. Food sources of glyconutrients include brown rice, Japanese mushroom, and aloe vera juice (carefully processed to guarantee maximal acemannan content). The herb Astragalus is also well known for its immune modulating effects, via its content of these glyconutrients. And of course, breast milk for babies. As it would appear that glyconutrients are essential carbohydrates, if the immune system is not functioning well, or the diet is inadequate, consider supplementation with a specific glyconutrient formulation.

Sugar is sweet and can be good for you after all - just choose your sugars!

M R Ross, **Continued from page 5**

The animal findings suggest an intestinal flaw is to blame, said Cade, whose team also is putting the theory to the test in humans. Preliminary findings from that study - which showed 95 percent of 81autistic and schizophrenic children studied had 100 times the normal levels of the milk protein in their blood and urine- have been presented at two international meetings in the past year but have not yet been published.

When these children were put on a milk-free diet, at least eight out of 10 no longer had symptoms of autism or schizophrenia, said Cade, a professor of medicine and physiology at UF's College of Medicine and inventor of the Gatorade sports drink. His research team includes scientist Zhongjie Sun, M.D., and research associate R. Malcolm Privette, P.A.C.

"We now have proof positive that these proteins are getting into the blood and proof positive they're getting into areas of the brain involved with the symptoms of autism and schizophrenia," Cade said.

More than 500,000 Americans have some form of autism, according to the Autism Society of America. The developmental disability typically appears during the first three years of life and is characterized by problems interacting and communicating with others. Many individuals exhibit repeated body movements such as hand-flapping and may resist changes in routine.

Schizophrenia is noted for disturbances in thinking, emotional reaction and behavior and is the most common form of psychotic illness. More than 2 million Americans suffer from it, according to the National Institute of Mental Health. People with schizophrenia often hear voices not heard by others, or believe others are reading their minds, controlling their thoughts or plotting to harm them. In addition, their speech and behavior can be so disorganized that they may be incomprehensible to others.

In the UF studies, researchers injected rats with the protein beta-casomorphin-7, one of the key constituents of milk and the part that coagulates to make cheese. They then observed their behavior and later examined brain tissue to see whether the substances accumulated there.

Beta-casomorphin-7 was taken up by 32 different areas of the brain, Cade said, including sections responsible for vision, hearing and communication.

"This could explain several of the things one sees in autism and schizophrenia, such as hallucinations," he said. "If part of the brain puts out a false signal because of casomorphin, it could result in the person seeing something that's not really there.

"There are a whole number of behaviors that the rat has after beta-casomorphin-7 that are basically the same as one sees in the human with autism orschizophrenia," Cade added.

Researchers suspect the process begins in the intestine, where the body absorbs the protein when a person eats foods containing it.

Recipe Corner

by Sue Litchfield

Here we are at the start of a brand new century and please do start putting on those thinking caps by helping me out with all those recipes and helpful hints that are hidden away in those cupboards. Remember some of those old hints that belonged to Gran can be of great help to all of us.

HINT: Use yoghurt flavoured with a few chopped fresh herbs instead of sour cream. Yoghurt can become watery if used in cooking. To prevent this from happening thicken with a little corn flour or arrowroot. Try thickening casseroles with mashed potatoes instead of normal flour.

RECIPES

Apple Crumble

100 grams butter or margarine, 3/4 cup brown rice flour, 1/4 cup "Lowans" soya flour, 1/2 teaspoon Stevia, 100 50 grams coconut, 50 grams flaked almonds, 1 teaspoon cinnamon, 1 tin unsweetened pie apple (Goulburn Valley)

Grease oven proof dish and spread apple in. Rub butter with the rest of ingredients and place on top of apple. Bake in mod. oven approx 25 mins.

Variation

Add either Blackberry or Blue berry unsweetened jam for a bit of variety.

Meringues

3 egg whites, 3 tabs Rice syrup, 1 teaspoon vanilla

Beat egg white until very stiff, very slowly add rice syrup, beating all the time, lastly add vanilla.

P;lace in small heaps on baking tray lined with baking paper in slow oven approx 1 1/2 to 2 hours or till dry.

Bev Cook send me the following recipe:

Pizza base

3/4 cup rice flour, 1/2 Polenta, 1/2 cup potato flour, 1/2 cup olive oil, 2/3 cup water

Sift drying, add oil and water combined, mix to smooth dough, turn onto board dusted with rice flour, knead till smooth. Lightly oil a 30cm pizza tray and press dough to cover tray.

Top with preferred allowed topping - bake in hot oven approx 20 mins till cooked through.

Rice Custard Tart

2 cups brown rice- cooked, 1 egg lightly beaten, 1/2 cup currants, 1/2 teaspoon ground nutmeg

Lightly grease 23cm pie plate with dairy free margarine. Combine rice & egg in bowl, mix & press mixture evenly over base and side of pie plate. Put plate on oven tray, bake mod. oven approx 15 mins or until just set, cool.

Lightly beat 3 eggs with 1 1/2 teas Vanilla essence, 1 1/2 cups soy milk and 2-3 drops Stevia sweetener. Pour filling into base, sprinkle with the currants. Bake in mod oven 30 mins, sprinkle with the nutmeg, bake further 30 mins or until custard is set. Serve hot or cold after cooling & refrigerating.

Mr Ivan Fewarnside wrote in to say that one source for STEVIA is:

THE FRAGRANT GARDEN
25 Portsmouth Road
ERINA 2250
Phone: (02) 4367-7322
Fax: (02) 4365-1979
Mobile Phone: 0408-444-212

The Editor rang this firm and they advised that they are growing Stevia but that it is not ready for marketing. We ask members to pursue this matters and report any other sources of Stevia in Australia.

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By Editor

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