The Hypoglycemic Health Association

NEWSLETTER

Correspondence: THE HYPOGLYCEMIC HEALTH ASSOCIATION, P.O. BOX 830, KOGARAH, N.S.W. 1485

Phone: (02) 9553-0084, **Fax:** (02) 9588-5290

PATRON: **Steve McNaughton, BE (NSW)**

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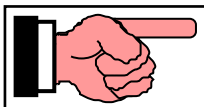
The NEWSLETTER of the Hypoglycemic Health Association is distributed to members of the Association and to Health Professionals with an interest in nutritional medicine and clinical ecology.



Please take note that on the 3 March 2001 we will have a **Annual General Meeting** one half hour earlier before the lecture given by Dr George Samra. A copy of Income and Expenditure for the year ending 31 December 2000 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 theses documents. We will be asking **members to join the Steering Committee** as some past serving members have left for the country. We desperately need new committee members to assist in the running of the Association. The position of Secretary is still vacant. The work involved is not onerous and consists of just attending some Committee Meetings once every three months at Dr Samra's surgery. Members with access to computers are especially welcome, but this is not essential.

Members are reminded that annual memberships expire on the 31 December of each year. The expiry date is shown at the top-right hand corner of address labels. Payments can be made by filling in the application form on page 12 of the Newsletter, and sending it together with the fee of $20 p.a. (Pensioners $15 p.a.) to the Association at PO BOX 830, KOGARAH NSW 1485. The cost of running the Association, organising public meetings at which expert give lectures and the preparation of Newsletters is entirely borne by members. We hope to be able to continue sending free Newsletters to doctors and other health professionals with the aim of promoting **complementary medicine** for the benefit of not only members but for the larger community. The ever increasing cost of traditional orthodox medicine force people to take greater personal responsibility for their health by applying knowledge gained of natural medicine.

Our Next Public Meeting will be at 2.00 PM
on Saturday, the 3 March, 2001
at ***YWCA***
5-11Wentworth Ave, SYDNEY
and our guest speaker is
Dr George Samra

who will be speaking
on the subject of

"Healthy Hearts and Hypoglycemics"

DR GEORGE SAMRA is of course well-known to our members. He is the President 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. 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

The Association also has a web site at: <www.companyontheweb.com/hypoglycemia_australia> where there are some Newsletters in PDF format, as well as articles on clinical nutrition and self-help psychotherapy.

Books for sale at the meeting

Sue Litchfield: **SUE'S COOKBOOK**

Dr George Samra's book

The Hypoglycemic Connection

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

Jurriaan Plesman: **GETTING OFF THE HOOK**

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

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

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 donations at meetings

Entry donation is tax deductible and for non-members will be $5, for members $3 and family $5. People requiring a receipt for taxation purposes will be issued when asked for it.

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 or Sue Litchfield at 9971-5657 or (litch.grip@bigpond.com).

At the last meeting on the 2 Dec 2000, Gersina Den Dulk won the lucky door prize.

Ms Marcia Walker won the raffle, consisting of a fan.

Fund raising activities

We need money, ideas, donations, bequests (remember us in your will), donations over $2 are tax deductible.

Raffles

Raffle tickets are available for towels at $1 each or 3 tickets for $2, which will be drawn at the next meeting of 3 March 2001. After that date an Alarm Radio Clock will be raffled. Tickets will be $2.00 each or $3 for five tickets.

All tickets can be bought at Dr George Samra's surgery, 19 Princes Highway Kogarah or at the next meeting.

Attention is drawn to our new Web Site at:

www.companyontheweb.com/hypoglycemia_australia

where you'll find articles on clinical nutrition and self-help psychotherapy. Copies of Newsletters are also available.

The following Committee Members can be contacted by email at:

Sue Litchfield, Treasurer
litch.grip@bigpond.com
Jurriaan Plesman, Editor
jurplesman@hotmail.com
Lynette & Reg Grady,
lgrady@fastrac.net.au

NEW SUPPORT GROUP - With the help of Bill and Margaret Witton and Lorraine Smith I have been able to form a support group for persons with Hypoglycemia and their family and friends. Dr. Samra has kindly donated a room at his surgery as the venue for the meetings. We have already held two meetings and intend to meet every 3 months beginning with this year on the 10th February. The dates and times for this year's meetings are as follows -

Time - 1.45pm. to 4.pm.

Dates - 10th February, 12th May, 11th August and 10th November.

Venue - 1st floor, Dr. Samra's surgery (19 Princes Highway, Kogarah between Stanley and Regent Streets). Parking is available in both streets.

The purpose of the meetings is to have a mutual support and discussion group for Hypoglycemics. Guest speakers will also be included at the meetings whenever possible.

At the first two meeting members found the information shared concerning different types of foods we can eat, and where to purchase them most helpful. We also exchanged recipes and breakfast menus.

Dr. Samra will be speaking at our meeting on 12th May.

If you would like to come along please do, we would love to see new faces. The cost is $1 per meeting. Afternoon tea is provided, however if you wish to share a special cake, biscuits or snack with the group please bring along a sample.

Letter from Sue Litchfield

Happy New Year to you all!

We ended last year on a happy note with a good attendance at the Christmas party although it would be great to see a few more faces. The raffle raised $86, which I was very excited about.

At the next meeting we are raffling towels, a great present for Mothers Day!! The tickets are on sale now at the Surgery.

Due to my husbands work and an illness in the family I am sorry I have not done anything about recipes for this magazine. However, if anyone has a few good recipes please do not hesitate to send them in, besides I would love to try a few new ones for a change.

I am planning on going away till the end of June but do not despair as this trusty old computer and all that goes with it will be packed up and going north. All the banking and bookwork will carry on as usual and the email will be read on a regular basis. In fact, I love hearing from people when away from home as it can get a little lonely at times (Email: litch.grip@bigpond.com).

As I will be away for 2 meetings and therefore unable to do anything about the afternoon tea is there anyone out there who could help out even just a few sandwiches or a cake would be of great help to us?

All the best and shall see you all in September.

Sue

A Sucrophilia Sufferer

By Jeanette Bousfield

My first memory of Sucrophilia (sugar cravings) dates back to when I was 4 years old. My mother worked at the Echo Point Kiosk and whenever we visited there on her day off, the owner would let me have a bag of confectionery of my choice. Probably because while my mother and her were talking I would stand in front of the glass counter and stare at the fabulous range of confectionery available.

We moved back to Sydney when I was 5 and being a child with overwhelming cravings for sweet food and drinks I became a regular raider of the pantry. Did I raid the biscuit tin? No - we only had Sao biscuits. Instead I would consume jelly crystals, jam and condensed milk by the spoonfuls.

Ever since I can remember I had unexplained illnesses and near fainting turns. Dad was a chronic alcoholic and that meant a lot of chaos and emotional suffering in our lives. He brought home a bag of mixed lollies for my 2 brothers and myself every payday; of course I looked forward to payday. I always ate my share that night. Dad most likely suffered from Sucrophilia himself, as alcohol contains large amounts of sugar. The alcohol shortened his life; he died from Cirrhosis of the liver at age 54.

When aged between 10 and 12 I walked home from school each Thursday for lunch

Continued at page 10

Hepatitis C - A Naturopathic Approach

by
Daniel Baden

The subject of today is about the naturopahic approach in the treat ment of Hepatitis C (HCV) and its relationship to detoxification and natural immune enhancers. This is becoming a prevalent disease, especially in Sydney, where this is a big issue. Last year Australia and News Zealand spent $86 million on hepatitis C.

Hepatitis Overview

Hepatitis C is usually of viral origin, but can also originate from metabolic or immunological disorders. Hepatitis can result from other conditions such as drug taking. Thus Hepatitis C may be a primary problem or secondary problem. The two major types are: A) infectious, faecal, or via oral route, B) via serum and blood. Hepatitis is a serious problem accounting for 300 million world wide; of these 30 per cent have chronic hepatitis .

The Hepatitis C virus was originally cloned from the serum of chimpanzees and was found to be generally responsible for Non-A and Non-B types. Thus for many years doctors could not identify the agent responsible for Hepatitis C. There are other forms of hepatitis , eg HDV, HEV, HGV, but these are not as destructive as HCV.

Drugs that cause hepatitis

Isoniazid, a drug used for tuberculosis, affects vitamin B6, B9 (folic acid), B3. ***Methyldopa*** a drug used in hypertension, affects B9 and B12. ***Nitrofurantoin*** in the treatment of urinary tract infections interfere with B9. ***Phenytoin*** used in seizures affect B9, Calcium, Magnesium, vitamin K, and B12. Then there are the antibiotics that may disrupt metabolic pathways affecting the liver.

Hepatitis C is a threat to public health and currently affects 230,000 Australians and New Zealanders or about 1 per cent of the population. It is estimated that there are between 8000 an 12000 new infections each year. These are usually under-reported due to the method of infection like needle sharing.

Facts about Hepatitis

Hepatitis C was first heard of about 30 years ago and it is known that an RNA virus, of the Flavivridae family, is responsible. The RNA of cells is involved in the replication of the cell. It has 6 major genotypes and mutates quite rapidly, and this causes problems designing a test for it or planning treatment. Twenty per cent of people with Hepatitis C have acute hepatitis.

Another problem is that it is a slowly progressive disease, so you really don't see symptoms over 2-5 decades. It can take 12 years before the initial symptoms start to occur. Eighty-five per cent of people fail to clear the virus and thus the disease becomes chronic. Thus only 15 per cent of patients recover from Hepatitis. It causes fibrosing liver disease and in 25 per cent of cases it causes cirrhosis. It is a major cause of liver transplants as well as of primary liver cancer.

Being a chronic illness it requires ongoing support. In 1/3 of patients there is significant morbidity. It affects 60 percent of males with a peak infection occurring in the age group of 20-29 years. Symptoms start to become most obvious at age 40-49 years.

The geographical outcomes vary, for some unknown reason, but sufferers from Egypt and Italy deteriorate much faster. On the other hand some patients experience no change at all, even after 30 years. As a naturopath I would like to think that the way people look after their immune system may have an influence on the prognosis of the disease.

Risk Factors

In 80 per cent of cases infection occur as a result of **injecting** drug use. Another risk factor is a **blood transfusion** before 1990, when the clearing test for Hepatitis C were nowhere as good as they are now. People with **haemophilia** - a hereditary bleeding disorder - and patients on **dialysis** have a higher risk. People working in hospitals as doctors or as nurses with a threat of **needle stick injury** are in a high risk category. **Tattooing and Body piercing** increases the risk. Interestingly, children from HCV mothers only acquire the disease in 10 per cent of cases even when breast feeding. Only 50 per cent of HCV mothers show the virus in milk, but this does not appear to affect the risk in babies.

The sources of the virus transmission is unknown in 40 per cent of cases, according to one medical record.

Clinical Presentation

People with Hepatitis C often present no symptoms or have low grade non-specific symptoms, such as fever, general feelings of being unwell. Occasionally we have acute infections of the liver rarely accompanied with jaundice. Fibrotic progression occurs in stage 1 on average between 5 - 9 years after infection. Signs of liver disease are **lethargy, enlargement of liver (hepatomegaly), little spider veins (spider naevi)** often around the stomach, and raised enzymes from the liver (**ALT)** (alanine aminotransferase) which usually indicates acute liver damage.

If you are interested in **iridology** from a naturopathic point of view, we can detect what is called liver lesions in the iris, and also yellow-brown wash. In Chinese medicine they would notice a thick exudate on the tongue with a yellow brown tinge to it.

Emotionally patients would often present with anger, frustration and irritability. Sometime they experience a mental fog and feel disoriented. They may have difficulty concentrating, menstrual problems are common and they are often depressed. When they find out about the condition they may even become more depressed because treatment is rather difficult.

Other signs are headaches, mild fever, mild spontaneous sweating, diarrhoea and nausea.

Causes

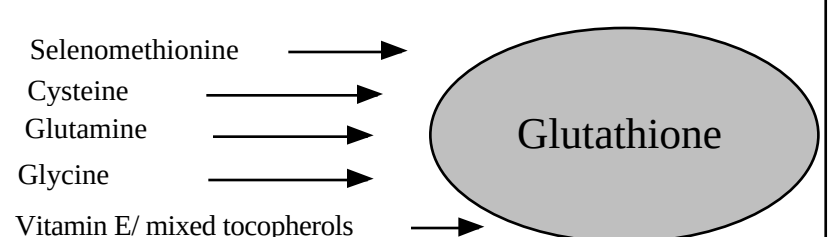
The pathogenesis is not fully understood. But there is some evidence of direct viral effects and immune mediated injury. Meaning that the virus itself can cause some problem, but the virus can also trigger other parts of the immune system to cause problems.. Thus essentially there is a two-pronged attack upon the liver. We often also find auto-antibodies attacking the body itself. Lymphoid follicles may contain activated B and T lymphocytes found within portal tracts to the liver.

Diagnosis

Every five to nine years we can almost

FIGURE 1

Glutathione is a tripeptide containing L-glutamic acid, L-cysteine, and glycine, which functions as the reductant of toxic peroxides by the action of selenium dependent glutathione peroxidase. It protect cells against the destructive effects of hydrogen peroxide.



diagnose the stage of hepatitis by measuring the **ALT enzyme** which in 80 per cent of patients show ten fold increases. In the second stage - that is from ten to twenty years - the portal tracts, the large arteries in the liver, start to expand. This is because all the tissues around the arteries become fibrous and contract, and so we start to see fibrotic changes. In biopsy of the liver we can actually see stripes of white tissues and some evidence that the liver is trying to regenerate as well. Cirrhosis of the liver develops in 25 per cent of patients.

We need to look at people's **life style** and isolate high risk factors such as using injectable drugs, having regular tattoos. Of all the tests the liver biopsy is the most reliable for HCV, but there are side effects including bleeding. The Chinese discovered that a lot of sufferers had **bleeding gums** as a symptom of hepatitis B. This should be checked out as there are other causes of bleeding gums such as vitamin C deficiency, which should be ruled out.

Other signs health practitioners look for are dilated veins on abdominal wall, peripheral oedema or swelling of the hands and feet, Sjögren's Syndrome or the dry eye syndrome affecting the mucous membranes, thyroid dysfunction, Liver Palms characterized by redness surrounding the outside of the palm, Hirsutism mainly affecting women who show excess hair growth.

One can also have a ultrasound to look at the size of the liver.

Differential diagnosis

It is important to distinguish between different types of liver diseases. From a naturopathic point of view we look at increased toxic load, either environmentally or internally produced toxins. Ross River virus, having similar symptoms, should be excluded, as well as Epstein-Barr virus (EBV), Rubella, cytomegalovirus (CMV) and Chronic Fatigue Syndrome (CFS).

Treatment

Counselling is perhaps one of the most important aspect in the treatment of Hep C. Sufferers should be fully informed of all, aspects of Hepatitis C, as there is a lot of misinformation. People should be aware of treatment options.

First thing to do is to boost the immune system and reduce inflammation.

You need to make an assessment in changes of lifestyle and addictions, such as stress, diet and smoking. Small regular meals seems to help, whereas alcohol is out of the question in Hepatitis C, because it reduces carnitine to which we will return later. Many patients feel better on a low fat diet. In a high fat diet several enzymes are suppressed. These are a particular group of enzymes in the liver which are responsible for breaking down a lot of substances such as estrogens, harmful substances from cigarette smoking and other toxins. With a high quality protein diet these enzymes become activated.

Some drugs that doctors use are: **Ribavarin** (antiviral guanosine analogue) with common side effects of nausea and loss of appetite. You get best result with **Interferon**, especially with patients under the age of 45. The 50 percent of success rate will be adversely affected if people use illegal substances. It also depends on how ill people are, whether they have additional immune stress such as HIV, Chronic Fatigue Syndrome, toxic overload. A medical journal reported that Interferon is more effective if patients had high levels of the amino acid, cysteine a derivative of the essential amino acid methionine. Cysteine works primarily in the liver and they seem to work together in that organ. However, Interferon reduces the body's antioxidants, so doctors should supplement their patients with antioxidants which will also improve the performance of Interferon.

There are certain foods that have a synergistic effect with Interferon, enhancing its capacity such as Reishi and Shiitake mushrooms. These are mushroom that grow on trees contain ***beta glucan*** known to enhance the action of Interferon. *Beta glucan* is also available as a separate supplement.

Interferon suppress viral duplication and normalizes ALT. There are a number of herbs that provide antioxidants and are precursors of glutathione which plays an important role in detoxification.

Liver Cell Regeneration

It is important to assist the liver in cell regeneration. **Excess glucose** inhibits liver cell division and repair, thus a high sugar diet would affect the livers ability to repair itself. Also liver regeneration is clearly dependent on essential fatty acids such as flaxseed oil and fishoil, and the amino acid carnitine. Alcohol consumption pushes carnitine out of the body, therefore hepatitis patients should avoid alcohol at all cost.

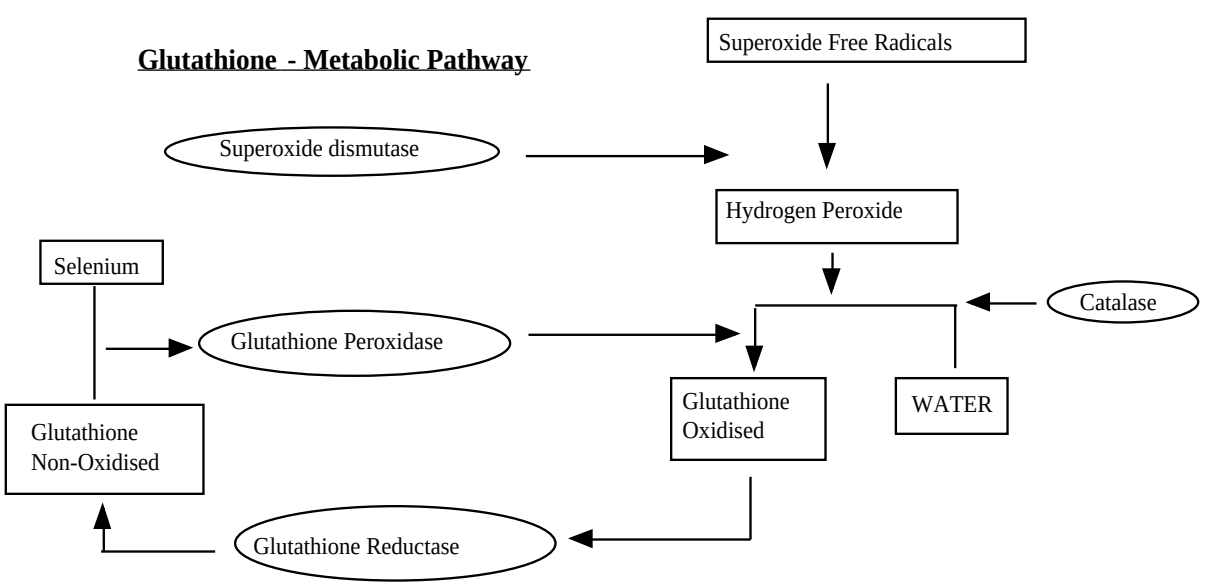
Herbal Remedies

There are some wonderful herbal treatment for hepatitis. One of the area we look for in herbs are the adaptogens. This is a class of herbs that help people to adapt to the environment during stressful periods. The most well-known among these are **Withania** (Withania somnifera) and **Siberian Ginseng** (Eleutherococcus senticosus)**.** Most of the work done on adaptogens come from the Russians where Siberian ginseng is used extensively. They found in different studies, where people were put under artificial stress those taking adaptogens like Withania and Siberian Ginseng coped much better, both emotionally and physically.

There are herbal anti-inflammatories such a **Bupleurum falcatum** also known as Chinese thoroughwax, which has anti-mutagenic action.

Curcuma longa, also known as Curcumin;

FIGURE 2



Turmeric, Bengal turmeric, Indian saffron, has powerful antiinflammatory activities. I would strongly suggest to people with chronic inflammation and arthritis to use turmeric in their cooking - a wonderful herb with antioxidant properties as well.

Silymarin marianum, or St Mary's Thistle or Milk Thistle, is regarded as the king of the liver tonics. Studies have shown that Milk Thistle can increase glutathione concentration by up to 35 per cent involved in detoxification. It is a good antioxidant as well.

Antiviral Herbs

Among the herbs specifically fighting viruses are **Astragalus membranaceus,** a well-know Chinese herb. It has antiviral and antibiotic properties which can be safely used with conventional medicine. We will discuss astragalus in more detail as this is one of the most important herbs in the treatment of hepatitis.

Similarly, **Echinacea augustifolia,** has antiseptic, antiviral, antifungal and antibiotic properties. The herb boosts the cells' production of a natural virus-fighting interferon, and because the effects are relatively short-lived, the herb is best taken at frequent intervals. This herb may however cause an allergic reaction in people with a sensitivity to flowers in the daisy family.

Phyllanthus amarus is a liver herb used in acute liver infection, but has negative results in hepatitis B.

Schizandra chinensis and **St Marys Thistle** are among the herbs helping the liver detoxify the body.

There are special herbs that can protect the liver called **Hepatoprotectives.**

These herbs are:

Chelidonium majus (Celandine, Greater celandine, garden celandine)

Curcuma longa (Turmeric)

Schizandra chinesis (Shizandra)

Silybum marianum (St Mary's Thistle)

Taraxacum off. (Dandelion)

Even if you do nothing but drink dandelion coffee this can be very helpful according to several studies done.

Some of these herbs can even restore the liver, especially Bupleurum facatum, Silybum marianum (St Mary's Thistle) and Panax ginseng (Korean Ginseng).

Astragalus membranaceaus is used in parts of America and, in Europe used as echinacea. It has immunostimulant properties, similar to echinacea. Astragalus is a tonic to the heart and vascular system, it acts as a diuretic and reduces blood pressure. It can be supplemented with most hypotensive drugs as its hypotensive action is mild. It is mainly indicated in chronic viral infections, hepatitis and debility. It is available as tablets, as liquid as a root that can be chopped up and taken as a tea. In tablet form take 200mg of Astragalus once or twice a day. Choose a products with 0.5% glucosides and 70% polysaccharides. The herb appears to have no side effects of any kind. Its main action is a liver tonic and an anti-viral agent. It stimulates interferon production. A number of studies have shown a significant response of 86 per cent in chronic persistent hepatitis. Other studies show that it lowers elevated liver enzymes within two months in 70 per cent of cases.

Astragalus increases the metabolic activity of the liver, it enhances NK Natural Killer cells, and increases **Super Oxide Dismutase** (SOD). This is is the body's main detoxifying enzyme.

It is contraindicated in acute infections.

Bupleurum falcatum contains triterpene saponins, sapogenins and saikogenins. The latter has a potent effect on the recovery of enzyme activity in liver protoplasmic membranes, lysosomes and mitochondria. The saponin compounds have been shown to enhance liver function in the long term. Thus Bupleurum is antiinflammatory, protects the liver (hepatoprotectivve), adaptogenic and above all **suppresses liver fibrosis** and inhibits, liver cell damage induced by macrophages. Macrophages is part of the immune system and the fall-out of macrophages can cause some of the problems themselves.

Schizandra has been used by Chinese herbalist for many illnesses such as cough, wheezing, spontaneous sweating, insomnia and forgetfulness. Studies show that it elevates liver function. This herb has strong liver protective effects (hepatoprotective), probably due to the inhibition of lipid peroxides. When the liver is under stress it produces a lot of free radicals. Lipid peroxides are a long term insipious free radicals. It lodges in the fatty tissues of the liver cells and they are responsible for long term damage. Schizandra has been shown to have the ability to raise cytochrome P-450 enzymes, a protein involved with extramitochondrial electron transport in the liver and in drug detoxification. Its lignans stimulate liver glycogen and protein synthesis.

Animal,al studies have shown that Schizandra has a stimulatory effect on the Central Nervous System (CNS) and has antidepressant activity.

Russians use Schizandra as an adaptogen, and have found that it can increase human endurance, which is one of the definitions of an adaptogen in herbalism. Several studies suggest that Schizandra significantly reduce SGPT (Serum glutamic pyruvic transaminase), also known as ALT (Alanine amino transferase). The measurement of these enzymes is used in the diagnosis of acute liver disease. However, these levels may rebound when treatment is ceased. Other studies by *Chang and Butt (1987)* demonstrated that Schizandra improved mental efficiency, sight and sound sensitivity and improved adaptation to light changes. Fine motor movements were also improved.

St Marys Thistle (Silybum marianum)

The principle active ingredients in Milk Thistle are a group of Flavonolignans that are collectively known as Silymarin. Silymarin comprises a total of 2.5 per cent of Milk Thistle. These flavonolignans are: dehydrosilybin, isosilybin, silybin, silychristin, silydianin. They are known to increase the activity of Superoxide Dismutase (SOD), which is the main enzymatic mechanism to clear superoxide radicals from the body. Milk Thistle protects the liver against the effects of the extremely toxic Amanita Phalloides Mushroom. Silymarin stabilizes the liver's cell membrane and protects against the toxicity of solvent carbon tetrachloride and alcohol. Milk Thistle is an effective treatment for Fatty Liver. It improves the liver function of hepatitis patients after 3 months treatment with silymarin found in Milk Thistle, according to studies using a dosage of 240 mg per day. The effects of Milk Thistle's silymarin are to restore the liver's ability to synthesize proteins within the body, especially within the liver.

Phyllanthus amarus

Phyllanthus is a liver herb which contains lignins Phyllathin/hypophyllanthin, flavonoids and alkaloids, which inhibit the Hepatitis B virus (HBV) DNA polymerase. It reduces the Hepatitis virus surface antigen. It reduces the liver's ability to pick up the Hepatitis virus. Studies demonstrated that Phyllanthus removed surface antigen markers in 59 per cent of HBV carriers in 15 to 20 days. There was no recurrence in 9 months.

Turmeric (Curcuma longa)

This herb has strong antioxidant properties and inhibits the oxidation of LDL cholesterol. Having a high cholesterol does not mean that you are going to have a heart attack. It is not so much the cholesterol per se, but rather the level of oxidized cholesterol that should be considered. When cholesterol is oxidized it will cause cardiovascular problems. Humans receiving 20 mg of Curcumin per day for 60 days experienced a significant reduction in oxidation of their LDL cholesterol. Living in our present day environment with an overload of pollutant I personally feel that we need daily antioxidant supplements for preventing all these sorts of ÔmodernÓ diseases.

Turmeric suppresses damage to liver cells (hepatocytes) caused by hepatitis C. It can also reverse damage caused by aflatoxin, a type of fungus that grows on peanuts. Curcumin also stimulates the production of glutathione by the liver. Glutathione is a tripeptide containing L-glutamic acid, L-cysteine, and glycine, which functions to protect against toxic peroxides by the action of selenium dependent enzyme called glutathione peroxidase. It protect cells against the destructive effects of hydrogen peroxide. (**See Figures 1**)

Oxidative Stress

This is a huge issue for liver function. It has a role in the pathogenesis of many gastrointestinal diseases, including pancreatitis, gastric and duodenal; ulcers, Irritable Bowel Disease (IBD), and Gastro-Intestinal Tract (GIT) cancers, hepatic-alcohol injuries, metal storage disorders, hepatitis, ischaemia/reperfusion injury. The latter occurs when a patient undergoes surgery and vessels are clamped. When the clamps are taken off the blood rushes through the arteries and cause a sudden rise in free radicals. When a person has major surgery, he should supplement with antioxidants especially vitamin E and C.

People with hepatitis C have a low level of vitamin C in their blood, and therefore they should increase the vitamin C levels. This vitamin is water-soluble and is excreted in the urine. We can increase the time it remains in the body by taking lipoic acid, a coenzyme together with vitamin B1 in the oxidative

decarboxylation of pyruvate to acetyl-CoA in glucose metabolism. Thus lipoic acid increases the effectiveness of vitamin C.

When you have a lot of oxidation it affects the glutathione in the liver. Glutathione is the body's main detoxifier in every cell and tissue of the liver. Every type of cancer, Chronic Fatigue, HIV, Chronic viral infection have a common thread in that people have low levels of glutathione. In the case of cancer people have only 25 per cent of glutathione, hence supplementing them with glutathione they would feel much better. One of the components of glutathione - cysteine - is a sulphydryl donor and improves the response to interferon. It has also been observed that patients with hepatitis C have higher levels of iron their blood, and iron inhibits interferon activity. Reduced iron also reduces ALT. One of the reason of the effects of high levels of iron is, that it increases the red blood cells' capacity to carry oxygen, which in turn exposes the body to greater amounts of free radicals. Thus when the body is under stress the iron levels should be monitored very carefully. The herbal antioxidants such as:

- Curcuma longa
- Ginkgo biloba
- Silybum marianum
- Camellia Chinensis (Tea: for their catechins)
- Bilberry and Rapeseed (Rich in Procyanidolic Oligomers, OPC's)

All of these above increase glutathione!

Other supplements

When taking **Beta Carotene** make sure you take the natural source of beta carotene. In recent years a natural substance called *Dunaliella saline* living in the ocean has been found to be a incredibly rich source of natural beta-carotene together with other carotenoids. It has a better therapeutic effect and it does not oxidize easily.

Glutathione exists in two states: oxidized state and reduced state. The herb **Ginkgo biloba** stimulates the cycle from the oxidized state to the reduced state. (**See Figure 2**).

Natural Immune Enhancers

Beta (1,3/1,6) Glucan was discovered in 1960 by Dr Diluzio-Tulane University. There are two types of glucan. In the Shiitake mushrooms contain Beta 1,3 Glucan, and the one extracted from then yeast is the Beta 1,6 Glucan. The macrophages in the immune system have receptor sites for the Beta (1,3/1,6) Glucan. The macrophages become stimulated by these Glucans and produce more interferon indirectly and activate NK cells. The ***beta Glucan is 200 times more effective than Echinacea.***

Mushrooms (Reishi/Shiitake) provide a rich source of Beta 1,3 Glucan. They stimulate IgA and increases the potency of vitamin C.

In Japanese hospitals they use the Reishi/ Shiitake mushroom for breast cancer and they have written many papers on this. These mushrooms contain the Beta D-Glucan (polysaccharide) and triterpenes. One can use any of the mushroom available in the Chinese grocers. They have antitumour properties, stimulate the immunity, they provide broad-spectrum cardiovascular support, and they are effective in the treatment of hepatitis C. Collective research indicates that 71-98 per cent of patients recover. These mushrooms also protect against chemically induced hepatitis and stimulate macrophages and helper T-cells.

Colostrum.

Colostrum is the fluid secreted by the breast during the first three days postpartum lactation begins. It contains immunological active substances (maternal antibodies). All mammals produce colostrum and it has immune enhancing properties, immunoglobulins. In naturopathy colostrum is used therapeutically, which is extracted from cows. A cow may produce colostrum for weeks, and it has a feedback mechanism. The more the calf takes the more the cow produces. It is usually extracted every second day with the calf stimulating further production every other day.

It has highly active antibacterial materials for the neutralization of bacterial toxins. Colostrum is a rich source of copper (Cu), manganese (Mn), molybdenum (Mo) and zinc (Zn). A study published in the *Journal of infectious Diseases (1995; 172,* demonstrated that colostrum could completely block HCMV (Human Cyto-Megalo virus) infection and inhibit HIV induced diseases of cells (cytopathic) effect. It is used in the treatment of any damage to the gut.

A study with rats in *J Am Physiological Soc 1994* confirmed increased growth rate of intestinal crypt cells (in the gastro-intestinal tract) and increased intestinal weight from prolonged use of IGF (Insulin Growth Factor) found in dairy products.

Dietary Whey Protein

New interest has been shown in the beneficial effects of a diet rich in whey protein, derived from a by-product of cheese making, that otherwise would be fed to animals. Whey protein is now prescribed for the treatment of various cancers such as breast cancer and especially colorectal cancer. It has been shown to improve thymus development, important in the T cell production (thymus dependent cells) as part of the immune system, and to reduce the involution - that is the gradual decrease in size - of the organ. It enhances lactoferrin, an iron complex in human milk (only a trace in cows milk), which plays a role inhibiting the growth of *E.coli.*

It provides greater protection against laboratory induced cancers than meat and soy proteins. It helps AIDS patients who are usually superinfected with HCMV (Human Cyto-Megalo virus). These patients are well known to have low serum glutathione, an essential substance necessary in detoxification.

Whey Protein Concentrate (WPC) has been proven to elevate antibody production in response to a T cell respondent antigen.

WPC helps maintain glutathione (GSH) levels. A six months Canadian study on children with a HIV/wasting syndrome show significant in blood lymphocyte glutathione.

Lactoperoxidase

Lactoperoxidase is found most abundantly in colostrum at the rate of 30mg /l.

Glycoprotein (8-10% CHO, Heme Group) It is heat stable (modern pasteurization = 30% loss). Lactoperoxidase is and antimicrobial agent killing *polio virus, candida albicans, inhibits streptococci, S.cremoris, S mutans.*

Lactoferrin

Lactoferrin is an iron-binding glycoprotein found in all mammalian fluid secretions in iron-free form. It was first discovered in 1939 and isolated in 1960. It has two primary roles: 1) Host defence, 2) Iron metabolism. Receptors for lactoferrin are found on intestinal tissues, monocytes, macrophages, neutrophils, lymphocytes, platelets and on some bacteria.

It acts by damaging the outer layer membranes of gram-negative bacteria, such as *E.coli, Salmonella.* It has a broad spectrum antimicrobial action by directly binding to the surface of microorganisms. It inhibits the macrophage uptake of cholesterol and decreases oxidation of LDL cholesterol.

Neutrophils contain lactoferrin which is released when these cells are activated by the inflammatory response. Lactoferrin is also present in the membranes of various mucous membranes.

Lactoferrin favours a gut flora rich in *bifidobacteria,* increases natural killer cells activity (NK cells), and therefore is beneficial to HIV patients. It also inhibits the Herpes virus (HSV1), which is responsible for what is called oral herpes affecting the facial areas.

Lactoferrin has been shown to rapidly clear the liver and its serum antiviral activity is predominantly active early in the infective cycle. However, its activity may be reduced by high levels of ions of calcium, magnesium and iron.

Fructo-oligosaccharides (FOS)

This is a large molecule of a sugar that cannot be absorbed by the gut, but it stimulates *bifidobacteria* growth. It is commercially made from cane-sugar and has about 70 per cent of its sweetness, when used as a alternative sweetener.

- FOS decreases pathogens in the gut by reducing the pH
- Regulates faecal frequency and consistency
- Maintains favourable colon flora during antibiotic treatment
- decreases serum cholesterol and triglycerides
- aids in the absorption of B vitamins
- aids in calcium absorption
- alters the metabolism of bile
- Reduces carbohydrate and lipid absorption, thereby normalizing blood glucose and serum lipids.
- Ameliorates derangements of carbohydrate and lipid metabolism in diabetics.

In short, the wholistic management of Hepatitis C (and other forms) with appropriate diet, herbal medicines and supplements can make an important contribution to the prognosis and pathological outcome. Treatment should commence immediately upon diagnosis and be considered long term with regular counselling.

A Unified Theory of Human Cardiovascular Disease Leading the Way to the Abolition of This Disease as a Cause for Human Mortality

Matthias Rath M.D. and Linus Pauling Ph.D

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"An important scientific innovation rarely, makes its way by gradually winning over and converting its opponents. What does happen is that its opponents gradually die out and that the growing generation is familiar with the idea from the beginning."
-Max Planck

This paper is dedicated to the young physicians and the medical students of this world

Abstract

Until now therapeutic concepts for human cardiovascular disease (CVD) were targeting individual pathomechanisms or specific risk factor,. On the basis of genetic, metabolic, evolutionary, and clinical evidence we present here a unified pathogenetic and therapeutic approach. Ascorbate deficiency is the precondition and common denominator of human CVD. Ascorbate deficiency is the result of the inability of man to synthesize ascorbate endogenously in combination with insufficient dietary intake. The invariable morphological consequences of chronic ascorbate deficiency in the vascular wall are the loosening of the connective tissue and the loss of the endothelial barrier function. Thus human CVD is a form of pre-scurvy. The multitude of pathomechanisms that lead to the clinical manifestation of CVD are primarily defense mechanisms aiming at the stabilization of the vascular wall. After the loss of endogenous ascorbate production during the evolution of man these defense mechanisms became life-saving. They counteracted the fatal consequences of scurvy and particularly of blood loss through the scorbutic vascular wall. These countermeasures constitute a genetic and a metabolic level. The genetic level is characterized by the evolutionary advantage of inherited features that lead to a thickening of the vascular wall, including a multitude of inherited diseases.

The metabolic level is characterized by the close connection of ascorbate with metabolic regulatory systems that determine the risk profile for CVD in clinical cardiology today. The most frequent mechanism is the deposition of lipoproteins, particularly lipoprotein (a) [Lp(a)], in the vascular wall. With sustained ascorbate deficiency, the result of insufficient ascorbate uptake, these defense mechanisms overshoot and lead to the development of CVD. Premature CVD is essentially unknown in all animal species that produce high amounts of ascorbate endogenously. In humans, unable to produce endogenous ascorbate, CVD became one of the most frequent diseases. The genetic mutation that rendered all human beings today dependent on dietary ascorbate is the universal underlying cause of CVD- Optimum dietary ascorbate intake will correct this common genetic defect and prevent its deleterious consequences. Clinical confirmation of this theory should largely abolish CVD as a cause for mortality in this generation and future generations of mankind.

Key words

Ascorbate, vitamin C, cardiovascular disease, lipoprotein(a),hypercholesterolemia. hypertriglyceridemia, hypoalphalipoproteinemia, diabetes, homocystinuria.

Introduction

We have recently presented ascorbate deficiency as the primary cause of human CVD. We proposed that the most frequent pathomechanism leading to the development of atherosclerotic plaques is the deposition of Lp(a) and fibrinogen/fibrin in the ascorbate-deficient vascular wall. In the course of this work we discovered that virtually every pathomechanism for human CVD known today can be induced by ascorbate deficiency. Beside the deposition of Lp(a) this includes such seemingly unrelated processes as foam cell formation and decreased reverse-cholesterol transfer, and also peripheral angiopathies in diabetic or homocystinuric patients. We did not accept this observation as a coincidence. Consequently we proposed that ascorbate deficiency is the precondition as well as a common denominator of human CVD. This farreaching conclusion deserves an explanation; it is presented in this paper. We suggest that the direct connection of ascorbate deficiency with the development of CVD is the result of extraordinary pressure during the evolution of man. After the loss of the endogenous ascorbate production in our ancestors, severe bloodloss through the scorbutic vascular wall became a life-threatening condition. The resulting evolutionary pressure favored genetic and metabolic mechanisms predisposing to CVD.

The Loss of Endogenous Ascorbate Production in the Ancestor of Man

With few exceptions all animals synthesize their own ascorbate by conversion from glucose. In this way they manufacture a daily amount of ascorbate that varies between about 1 gram and 20 grams, when compared to the human body weight. About 40 million years ago the ancestor of man lost the ability for endogenous ascorbate production. This was the result of a mutation of the gene encoding for the enzyme L-gulono-g-lactone oxidase (GLO), a key enzyme in the conversion of glucose to ascorbate. As a result of this mutation all descendants became dependent on dietary ascorbate intake.

The precondition for the mutation of the GLO gene was a sufficient supply of dietary ascorbate. Our ancestors at that time lived in tropical regions. Their diet consisted primarily of fruits and other forms of plant nutrition that provided a daily dietary ascorbate supply in the range of several hundred milligrams to several grams per day. When our ancestors left this habitat to settle in other regions of the world the availability of dietary ascorbate dropped considerably and they became prone to scurvy.

Fatal Blood Loss Through the Scorbutic Vascular Wall - An Extraordinary Challenge to the Evolutionary Survival of Man

Scurvy is a fatal disease. It is characterized by structural and metabolic impairment of the human body, particularly by the destabilization of the connective tissue. Ascorbate is essential for an optimum production and hydroxylation of collagen and elastin, key constituents of the extracellular matrix. Ascorbate depletion thus leads to a

destabilization of the connective tissue throughout the body. One of the first clinical signs of scurvy is perivascular bleeding. The explanation is obvious: Nowhere in the body does there exist a higher pressure difference than in the circulatory system, particularly across the vascular wall. The vascular system is the first site where the underlying destabilization of the connective tissue induced by ascorbate deficiency is unmasked, leading to the penetration of blood through the permeable vascular wall. The most vulnerable sites are the proximal arteries, where the systolic blood pressure is particularly high. The increasing permeability of the vascular wall in scurvy leads to petechiae and ultimately hemorrhagic blood loss.

Scurvy and scorbutic blood loss decimated the ship crews in earlier centuries within months. It is thus conceivable that during the evolution of man periods of prolonged ascorbate deficiency led to a great death toll. The mortality from scurvy must have been particularly high during the thousands of years the ice ages lasted and in other extreme conditions, when the dietary ascorbate supply approximated zero. We therefore propose that after the loss of endogenous ascorbate production in our ancestors, scurvy became one of the greatest threats to the evolutionary survival of man. By hemorrhagic blood loss through the scorbutic vascular wall our ancestors in many regions may have virtually been brought close to extinction.

The morphologic changes in the vascular wall induced by ascorbate deficiency are well characterized: the loosening of the connective tissue and the loss of the endothelial barrier function. The extraordinary pressure by fatal blood loss through the scorbutic vascular wall favored genetic and metabolic countermeasures attenuating increased vascular permeability.

Ascorbate Deficiency and Genetic Countermeasures

The genetic countermeasures are characterized by an evolutionary advantage of genetic features and include inherited disorders that are associated with atherosclerosis and CVD. With sufficient ascorbate supply these disorders stay latent. In ascorbate deficiency, however, they become unmasked, leading to an increased deposition of plasma constituents in the vascular wall and other mechanisms that thicken the vascular wall. This thickening of the vascular wall is a defense measure compensating for the impaired vascular wall that had become destabilized by ascorbate deficiency. With prolonged insufficient ascorbate intake in the diet these defense mechanisms overshoot and CVD develops.

The most frequent mechanism to counteract the increased permeability of the ascorbate-deficient vascular wall became the deposition of lipoproteins and lipids in the vessel wall. Another group of proteins that generally accumulate at sites of tissue transformation and repair are adhesive proteins such as fibronectin, fibrinogen, and particularly apo(a). It is therefore no surprise that Lp(a), a combination of the adhesive protein apo(a) with a low density lipoprotein (LDL) particle, became the most frequent genetic feature counteracting ascorbate deficiency.' Beside lipoproteins, certain metabolic disorders, such as diabetes and homocystinuria, are also associated with the development of CVD. Despite differences in the underlying pathomechanism, all these mechanisms share a common feature: they lead to a thickening of the vascular wall and thereby can counteract the increased permeability in ascorbate deficiency. In addition to these genetic disorders, the evolutionary pressure from scurvy also favored certain metabolic countermeasures.

Ascorbate Deficiency and Metabolic Countermeasures

The metabolic countermeasures are characterized by the regulatory role of ascorbate for metabolic systems determining the clinical risk profile for CVD. The common aim of these metabolic regulations is to decrease the vascular permeability in ascorbate deficiency. Low ascorbate concentrations therefore induce vasoconstriction and hemostasis and affect vascular wall metabolism in favor of atherosclerogenesis. Towards this end ascorbate interacts with lipoproteins. coagulation factors, prostaglandins, nitric oxide, and second messenger systems such as cyclic monophosphates. It should be noted that ascorbate can affect these regulatory levels in a multiple way- In lipoprotein metabolism low density lipoproteins (LDL), Lp(a), and very low density lipoproteins (VLDL) are inversely correlated with ascorbate concentrations, whereas ascorbate and HDL levels are positively correlated. Similarly, in prostaglandin metabolism ascorbate increases prostacyclin and prostaglandin E levels and decreases the thromboxane level. In general, ascorbate deficiency induces vascular constriction and hemostatis, as well as cellular and extracellular defense measures in the vascular wall.

In the following sections we shall discuss the role of ascorbate for frequent and well established pathomechanisms of human CVD. In general, the inherited disorders described below are polygenic. Their separate description, however, will allow the characterization of the role of ascorbate on the different genetic and metabolic levels.

Apo(a) and Lp(a), the Most Effective and Most Frequent Countermeasure

After the loss of endogenous ascorbate production, apo(a) and Lp(a) were greatly favored by evolution. The frequency of occurrence of elevated Lp(a) plasma levels in species that had lost the ability to synthesize ascorbate is so great that we formulated the theory that apo(a) functions as a surrogate for ascorbate.' There are several genetically determined isoforms of apo(a). They differ in the number of kringle repeats and in their molecular size. An inverse relation between the molecular size of apo(a) and the synthesis rate of Lp(a) particles has been established. Individuals with the high molecular weight apo(a) isoform produce fewer Lp(a) particles than those with the low apo(a) isoform. In most population studies the genetic pattern of high apo(a) isoform/low Lp(a) plasma level was found to be the most advantageous and therefore most frequent pattern. In ascorbate deficiency Lp(a) is selectively retained in the vascular wall. Apo(a) counteracts increased permeability by compensating for collagen, by its binding to fibrin, as a proteinthiol antioxidant, and as an inhibitor of plasmin-induced proteolysis. Moreover, as an adhesive protein apo(a) is effective in tissue-repair processes (8). Chronic ascorbate deficiency leads to a sustained accumulation of Lp(a) in the vascular wall. This leads to the development of atherosclerotic plaques and premature CVD, particularly in individuals with genetically determined high plasma Lp(a) levels. Because of its association with apo(a), Lp(a) is the most specific repair particle among all lipoproteins. Lp(a) is predominantly deposited at predisposition sites and it is therefore found to be significantly correlated with coronary, cervical, and cerebral atherosclerosis but not with peripheral vascular disease.

The mechanism by which ascorbate resupplementation prevents CVD in any condition is by maintaining the integrity and stability of the vascular wall. In addition, ascorbate exerts in the individual a multitude of metabolic effects that prevent the exacerbation of a possible genetic predisposition and the development of CVD. If the predisposition is a genetic elevation of Lp(a) plasma levels the specific regulatory role of ascorbate is the decrease of apo(a) synthesis in the liver and thereby the decrease of Lp(a) plasma levels. Moreover, ascorbate decreases the retention of Lp(a) in the vascular wall by lowering fibrinogen synthesis and by increasing the hydroxylation of lysine residues in vascular wall constituents, thereby reducing the affinity for Lp(a) binding.

In about half of the CVD patients the mechanism of Lp(a) deposition contributes significantly to the development of atherosclerotic plaques. Other lipoprotein disorders are also frequently part of the polygenic pattern predisposing the individual patient to CVD in the individual.

Other Lipoprotein Disorders Associated with CVD

In a large population study Goldstein et al. discussed three frequent lipid disorders, familial hypercholesterolemia, familial hypertriglyceridemia, and familial combined hyperlipidemia. Ascorbate deficiency unmasks these underlying genetic defects and leads to an increased plasma concentration of lipids (e.g. cholesterol, triglycerides) and lipoproteins (e.g. LDL, VLDL) as well as to their deposition in the impaired vascular wall. As with Lp(a), this deposition is a defense measure counteracting the increased permeability. It should, however, be noted that the deposition of lipoproteins other than Lp(a) is a less specific defense mechanism and frequently follows Lp(a) deposition. Again, these mechanisms function as a defense only for a limited time. With sustained ascorbate deficiency the continued deposition of lipids and

lipoproteins leads to atherosclerotic plaque development and CVD. Some mechanisms will now be described in more detail.

Hypercholesterolemia, LDL-receptor defect

A multitude of genetic defects lead to an increased synthesis and/or a decreased catabolism of cholesterol or LDL. A well characterized although rare defect is the LDL receptor defect. Ascorbate deficiency unmasks these inherited metabolic defects and leads to an increased plasma concentration of cholesterol-rich lipoproteins, e.g. LDL, and their deposition in the vascular wall. Hypercholesterolemia increases the risk for premature CVD primarily when combined with elevated plasma levels of Lp(a) or triglycerides.

The mechanisms by which ascorbate supplementation prevents the exacerbation of hypercholesterolemia and related CVD include an increased catabolism of cholesterol. In particular, ascorbate is known to stimulate 7-a-hydroxylase, a key enzyme in the conversion of cholesterol to bile acids and to increase the expression of LDL receptors on the cell surface. Moreover, ascorbate is known to inhibit endogenous cholesterol synthesis as well as oxidative modification of LDL.

Hypertriglyceridemia, Type III hyperlipidemia

A variety of genetic disorders lead to the accumulation of triglycerides in the form of chylomicron remnants, VLDL, and intermediate density lipoproteins (IDL) in plasma. Ascorbate deficiency unmasks these underlying genetic defects and the continued deposition of triglyceride-rich lipoproteins in the vascular wall leads to CVD development. These triglyceride-rich lipoproteins are particularly subject to oxidative modification, cellular lipoprotein uptake, and foam cell formation. In hypertriglyceridemia nonspecific foam-cell formation has been observed in a variety of organs." Ascorbate-deficient foam cell formation, although a less specific repair mechanism than the extracellular deposition of Lp(a), may have also conferred stability .

Ascorbate supplementation prevents the exacerbation of CVD associated with hypertriglyceridemia, Type III hyperlipidemia, and related disorders by stimulating lipoprotein lipases and thereby enabling a normal catabolism of triglyceride-rich lipoproteins. Ascorbate prevents the oxidative modification of these lipoproteins, their uptake by scavenger cells and foam cell formation. Moreover, we propose here that, analogous to the LDL receptor, ascorbate also increases the expression of the receptors involved in the metabolic clearance of triglyceride-rich lipoproteins, such as the chylomicron remnant receptor.

The degree of build-up of atherosclerotic plaques in patients with lipoprotein disorders is determined by the rate of deposition of lipoproteins and by the rate of the removal of deposited lipids from the vascular wall. It is therefore not surprising that ascorbate is also closely connected with this reverse pathway.

Hypoalphalipoproteinemia

Hypoalphalipoproteinemia is a frequent lipoprotein disorder characterized by a decreased synthesis of HDL particles. HDL is part of the 'reverse-cholesterol-transport' pathway and is critical for the transport of cholesterol and also other lipids from the body periphery to the liver. In ascorbate deficiency this genetic defect is unmasked, resulting in decreased HDL levels and a decreased reverse transport of lipids from the vascular wall to the liver. This mechanism is highly effective and the genetic disorder hypoalphalipoproteinemia was greatly favored during evolution. With ascorbate supplementation HDL production increases, leading to an increased uptake of lipids deposited in the vascular wall and to a decrease of the atherosclerotic lesion. A look back in evolution underlines the importance of this mechanism. During the winter seasons, with low ascorbate intake, our ancestors became dependent on protecting their vascular wall by the deposition of lipoproteins and other constituents. During spring and summer seasons the ascorbate content in the diet increased significantly and mechanisms were favored that decreased the vascular deposits under the protection of increased ascorbate concentration in the vascular tissue. It is not unreasonable for us to propose that ascorbate can reduce fatty deposits in the vascular wall within a relatively short time. In an earlier clinical study it was shown that 500 mg of dietary ascorbate per day can lead to a reduction of atherosclerotic deposits within 2 to 6 months."

This concept, of course, also explains why heart attack and stroke occur today with a much higher frequency in winter than during spring and summer, the seasons with increased ascorbate intake.

Other Inherited Metabolic Disorders Associated with CVD

Beside lipoprotein disorders many other inherited metabolic diseases are associated with CVD. Generally these disorders lead to an increased concentration of plasma constituents that directly or indirectly damage the integrity of the vascular wall. Consequently these diseases lead to peripheral angiopathies as observed in diabetes, homocystinuria, sickle-cell anemia (the first molecular disease described," and many other genetic disorders. Similar to lipoproteins the deposition of various plasma constituents as well as proliferative thickening provided a certain stability for the ascorbatedeficient vascular wall. We illustrate this principle for diabetic and homocystinuric angiopathy.

Diabetic Angiopathy

The pathomechanism in this case involves the structural similarity between glucose and ascorbate and the competition of these two molecules for specific cell surface receptors." Elevated glucose levels prevent many cellular systems in the human body, including endothelial cells, from optimum ascorbate uptake- Ascorbate deficiency unmasks the underlying genetic disease, aggravates the imbalance between glucose and ascorbate, decreases vascular ascorbate concentration, and thereby triggers diabetic angiopathy.

Ascorbate supplementation prevents diabetic angiopathy by optimizing the ascorbate concentration in the vascular wall and also by lowering insulin requirement-"

Homocystinuric angiopathy

Homocystinuria is characterized by the accumulation of homocyst(e)ine and a variety of its metabolic derivatives in the plasma, the tissues and the urine as the result of decreased homocysteine catabolism." Elevated plasma concentrations of homocyst(e)ine and its derivatives damage the endothelial cells throughout the arterial and venous system. Thus homocystinuria is characterized by peripheral vascular disease and thromboembolism. These clinical manifestations have been estimated to occur in 30 per cent of the patients before the age of 20 and in 60 per cent of the patients before the age of 40.

Ascorbate supplementation prevents homocystinuric angiopathy and other clinical complications of this disease by increasing the rate of homocysteine catabolism.

Thus, ascorbate deficiency unmasks a variety of individual genetic predispositions that lead to CVD in different ways. These genetic disorders were conserved during evolution largely because of their association with mechanisms that lead to the thickening of the vascular wall. Moreover, since ascorbate deficiency is the underlying cause of these diseases, ascorbate supplementation is the universal therapy.

The Determining Principles of This Theory

The determining principles of this comprehensive theory are schematically summarized in Figures I to 3 (pages 13 to 15).

1. CVD is the direct consequence of the inability for endogenous ascorbate production in man in combination with low dietary ascorbate intake.
2. Ascorbate deficiency leads to increased permeability of the vascular wall by the loss of the endothelial barrier function and the loosening of the vascular connective tissue.
3. After the loss of endogenous ascorbate production scurvy and fatal blood loss through the scorbutic vascular wall rendered our ancestors in danger of extinction. Under this evolutionary pressure over millions of years genetic and metabolic countermeasures were favored that counteract the increased permeability of the vascular wall.
4. The genetic level is characterized by the fact that inherited disorders associated with CVD became the most frequent among all genetic predispositions. Among those predispositions lipid and lipoprotein disorders occur particularly often.
5. The metabolic level is characterized by the direct relation between ascorbate and virtually all risk factors of clinical cardiology today. Ascorbate deficiency

leads to vasoconstriction and hemostasis and affects the vascular wall metabolism in favor of atherosclerogenesis.

6. The genetic level can be further characterized. The more effective and specific a certain genetic feature counteracted the increasing vascular permeability in scurvy, the more advantageous it became during evolution and, generally, the more frequently this genetic feature occurs today
7. The deposition of Lp(a) is the most effective, most specific, and therefore most frequent of these mechanisms. Lp(a) is preferentially deposited at predisposition sites. In chronic ascorbate deficiency the accumulation of Lp(a) leads to the localized development of atherosclerotic plaques and to myocardial infarction and stroke.
8. Another frequent inherited lipoprotein disorder is hypoalphalipoproteinemia. The frequency of this disorder again reflects its usefulness during evolution. The metabolic upregulation of HDL synthesis by ascorbate became an important mechanism to reverse and decrease existing lipid deposits in the vascular wall.
9. The vascular defense mechanisms associated with most genetic disorders are nonspecific. These mechanisms can aggravate the development of atherosclerotic plaques at predisposition sites. Other nonspecific mechanisms lead to peripheral forms of atherosclerosis by causing a thickening of the vascular wall throughout the arterial system. This peripheral form of vascular disease is characteristic for angiopathics associated with Type III hyperlipidemia, diabetes, and many other inherited metabolic diseases.
10. Of particular advantage during evolution and therefore particularly frequent today are those genetic features that protect the ascorbate-deficient vascular wall until the end of the reproduction age. By favoring these disorders nature decided for the lesser of two evils: the death from CVD after the reproduction age rather than death from scurvy at a much earlier age. This also explains the rapid increase of the CVD mortality today from the 4th decade onwards.
11. After the loss of endogenous ascorbate production the genetic mutation rate in our ancestors increased significantly- This was an additional precondition favoring the advantage not only of apo(a) and Lp(a) but also of many other genetic countermeasures associated with CVD.
12. Genetic predispositions are characterized by the rate of ascorbate depiction in a multitude of metabolic reactions specific for the genetic disorder." The overall rate of ascorbate depletion in an individual is largely determined by the polygenic pattern of disorders. The earlier the ascorbate reserves in the body are depleted without being resupplemented, the earlier CVD develops.
13. The genetic predispositions with the highest probability for early clinical manifestation require the highest amount of ascorbate supplementation in the diet to prevent CVD development. The amount of ascorbate for patients at high risk should be comparable to the amount of ascorbate our ancestors synthesized in their body before they lost this ability: between 10,000 and 20,000 milligrams per day.
14. Optimum ascorbate supplementation prevents the development of CVD independently of the individual predisposition or pathomechanism. Ascorbate reduces existing atherosclerotic deposits and thereby decreases the risk for myocardial infarction and stroke. Moreover, ascorbate can prevent blindness and organ failure in diabetic patients, thromboembolism in homocystinuric patients, and many other manifestations of CVD.

Conclusion

In this paper we present a unified theory of human CVD. This disease is the direct consequence of the inability of man to synthesize ascorbate in combination with insufficient intake of ascorbate in the modem diet. Since ascorbate deficiency is the common cause of human CVD, ascorbate supplementation is the universal treatment for this disease. The available epidemiological and clinical evidence is reasonably convincing. Further clinical confirmation of this theory should lead to the abolition of CVD as a cause of human mortality for the present generation and future generations of mankind.

Bousfield Story continued from page 2

because near the end of the week we had run out of fillings for lunches. I looked upon this as a treat because I could make myself sugar sandwiches.

After I finished schooling and then Business College I began work and nearly every morning bought a chocolate bar to eat on the train. If I didn't have time to buy a chocolate bar before catching the train, I'd buy myself a chocolate or strawberry malted milkshake when I reached the city. On reflection, I didn't even think it unusual that I was the only person in my group of friends eating chocolates at 8 a.m.

My list of illnesses kept growing and by age 30 I had quite a lot of the symptoms of Hypoglycemia as described in Jur Plesman's blue leaflet available at Dr. Samra's surgery. I certainly had 3 of the 4 symptoms that Dr. Samra refers to in his book "The Hypoglycemic Connection" i.e. Depression or moodiness – tiredness – memory impairment or poor concentration and history of sugar addiction.

Between the age of 30 and 40 I went under the knife of the surgeon 4 times (Appendix & Gall Bladder removed, Hysterectomy and then finally a kidney operation). Each Post surgery visit to the 4 different specialists I was told "You will be a new woman now". I recovered well from each operation but I still wasn't well. In fact I had reached the stage of commenting to my G.P. that I must be a Hypochondriac. He assured me I wasn't and explained how Hypochondriacs' imagine their symptoms but mine were real.

Then in my 40's I began suffering with migraine headaches. My G.P. recommended I abstain from eating chocolate, cheese, and citrus fruits as well as avoiding alcohol. I was able to stop eating cheese and citrus fruit as well as not drinking alcohol but giving up chocolate proved to be extremely difficult. However after enduring a few migraines lasting 4 days each I reluctantly gave up chocolate and increased eating sweets.

A turning point occurred in my life when one day I spoke to my Doctor about how fed up I was with always feeling unwell and often experiencing the weird sensation of internal tremors. He sent me off to have a Glucose Tolerance Test, which I had the next day. A few days later I went back to the Doctor for the results. I was stunned when he explained that I was a Hypoglycemic, with reactive Hypoglycemia, I had never heard of the illness.

The Doctors advice was to follow a Diabetic diet and he also referred me to a dietitian for guidance with the diet. The dietitian was helpful and after a while my health did improve.

Fortunately for me, not long after this diagnosis, I was listening to Dr. Sandra Cabot on the Radio and this particular evening she had a guest speaker who was Dr. Samra. The Hypoglycemia Association was mentioned during the program and I decided to join. After joining the Association I went to a meeting and thought that Dr. Samra may be able to help me even further with my diet. That was 12 years ago and actually I saw a Dr. Chen who was working with Dr. Samra at the time.

Dr. Chen ordered allergy tests and that suggested I had several food allergies, he then scrutinized and adjusted my diet while also recommending vitamin supplements. My health improved even further, and from then on as long as I keep strictly to my diet and take the vitamins daily, I can remain reasonably healthy. It is not easy and takes a lot of willpower, however I know it is worth the effort. I have an Aunty who is a diabetic and her eyesight is quite diminished, her heart is affected and she has been under the threat of losing one leg for some time, therefore I realise the consequences if I were to ignore my diet. I would be a likely candidate for diabetes. A couple of my cousins are diabetics too.

On Dr. Samra's advice I use glycerine daily in my diet that helps to give me energy when required.

Continued Page 12

Research Snippets
Extracts from

International Clinical Nutrition Review

A safer vitamin B3, inositol hexaniacinate?

As niacin may cause a range of side effects ranging from flushing and pruritis to hepatotoxicity (liver damage) and impaired glucose intolerance, a need has naturally arisen to investigate niacin esters such as inositol hexaniacinate (also called hexanicotinate or inositol nicotinate). Research has shown it to be effective in the treatment of hyperlipidaemia (high blood lipid levels), Raynaud's disease and intermittent claudication. Niacin deficiency, seen particularly in alcoholics and the elderly, responds well to therapeutic doses of vitamin B3. Favourable responses in hypertension, diabetes, dysmenorrhoea and alcoholism in trials to date suggest the need for more research.

Niacin is vital to cellular metabolism particularly due to its role in nicotine adenine dinucleotide (NAD) and nicotine adenine dinucleotide phosphate (NADP). Inositol hexanicotinate is slowly metabolised, not reaching maximum serum levels for approximately ten hours after administration. When it is administered orally, there is a sustained increase in the level of free nicotinic acid (B3) in blood and plasma. A group of 135 subjects, suffering from a range of conditions from Raynaud's disease to psoriasis, when given doses of inositol hexanicotinate from 600 to 1800mg daily, reported no side effects. This form of vitamin B3 appears to be safe of doses up to 4g daily. Approximately one third of patients with the same conditions receiving nicotinic acid suffered one or more symptoms such as flushing, nausea, vomiting, giddiness and weakness.

Head KA (2000), Inositol hexanicotinate: a safer alternative to niacin, **Townsend Letter 201,** 88-92

The underlying biochemistry in autism: a Review

In recent years some of the biochemical anomalies that may underlie autism have become clear. In a recent study it was found that 92% of the children investigated had greatly reduced plasma sulphate levels compared with age-matched controls and furthermore, the researchers were able to group the children into three distinct sub-groups.

In this study, early morning specimens of urine from 232 autistic children and 68 controls were collected and inorganic sulphate, sulphite, thiocyanate and thiosulphate were estimated in the urine by standard colorimetric methods. The results showed that autistic children had not only high urinary sulphate, sulphite and thiosulphate, but also increased levels of protein. Thiocyanate levels were reduced compared to controls. As sulphite oxidase is a molybdenum-containing enzyme, it was thought that the raised sulphite excretion might decrease on supplementation with molybdenum, but this only occurred in 36% of the children treated. The reduction in urinary sulphite, however, was accompanied by improvements in clinical symptoms.

It is known from previous research that total deficiency of sulphite oxidase leads to neurological dysfunction. The gene for slow sulphur oxidation appears to be inherited on a autosomal recessive pattern and the phenotype has been associated with a number of disease states such as rheumatoid arthritis and allergy, which are common in the family background of autistic children. The authors suggest that some of the symptoms of autism, such as stereotyped behaviour, mood swings and hyperactivity may be due to neurotransmitter amine imbalance in the central nervous system. Catecholamine levels in autistic children are often higher than normal due to lack of sulphated conjugates which inactivate them. Thus the increased concentrations of neurotransmitter amines act in a more prolonged fashion on the central nervous system. Similar effects can occur if enzymes such as sulphotransferase are low. Some autistic children, particularly those with a parent who suffers from migraines, have been found to have low levels of this enzyme. Hyperactivity in autistic children, may improve when chocolates, bananas, orange juice, vanillin and food colourants such as tartrazine are removed from the diet for the same biochemical reasons as these are not recommended for migraine sufferers. In addition, reduced sulphation has been associated with inflammation and gut dysfunction associated in particular with those autistic children who have casein and gluten allergy. The peptides excreted by such children can show "opiod" activity which may be responsible for the social withdrawal, insensitivity to pain and altered responses to sensory stimuli commonly described in autism.

Cluster analysis of the behavioural questionnaire completed by the children's parents revealed three main groups as follows: 1) those with problems such as sweating, thirst, cravings and urination probably caused by hypothalamic dysfunction, 2) those with problems such as hand-flapping, nightmares and epilepsy possibly caused by frontal lobe damage and 3) those with a family background of allergy, attention deficit disorder, hyperactivity and auto-immune dysfunction. This third group responds well to casein and gluten-free diets.

The authors remark that "potentially, subsetting autistic children by biochemical and behavioural parameters may be helpful in the future in recommending the appropriate treatments". They recommend a gluten-free diet, casein-free diet with the withdrawal also of chocolate, bananas and citrus fruits. Supplements suggested are magnesium sulphate, molybdenum, zinc and vitamin B6. In those children with low serotonin levels, serotonin precursors such as tryptophan may lead to reduced hand-flapping and an improvement in mood. In conclusion the researchers state that "as autism appears to be a heterogenous disorder there may well be no universal therapeutic recommendation although detailed knowledge of the individual biochemistry may eventually allow a more precisely targeted approach"

Waring RH, Klovrza LV (2000), Sulphur metabolism in autism, **J Nutr Env Med 10,** 25-32

Taurine improves insulin sensitivity in type 2 diabetes

Taurine is a potent antioxidant which prevents tissue injury. It also increases the excretion of cholesterol via conversion to bile acid and would be expected, therefore, to improve insulin resistance. Furthermore, it has been found in previous research that taurine improves streptozotocin-induced diabetes mellitus in which the development of the diabetes results from attack by oxygen free radicals on pancreatic beta cells.

In this study, the effects of taurine on insulin sensitivity were examined in rats with spontaneous type 2 diabetes. Taurine was added to the food of both the diabetic rats and a healthy control group for 10 weeks. The results showed that there were no significant differences in body weight between those with and without taurine supplementation. Total fat in the abdominal cavity, however, tended to be less in the taurine-supplemented group. Serum concentrations of HDL cholesterol were not found to be significantly different between the groups, but triacylglycerol and total cholesterol were lower in the taurine-supplemented rats. There were also lower concentrations of lipids in the livers of taurine-supplemented group. After glucose ingestion, blood glucose concentrations tended to be lower in the taurine-supplemented group.

Nakaya and co-workers conclude that "taurine supplementation improved insulin sensitivity significantly ....mainly due to the lipid lowering effect of taurine". The researchers also postulate that taurine's effects in lowering blood urea nitrogen concentrations and albuminuria might have been due to improved glucose and lipid metabolism.

Nakaya Y, Minami A et al. (2000), Taurine improves insulin sensitivity in the Otsuka Long-Evans Tokushima fatty rat, a model of spontaneous type 2 diabetes, **Am J Clin Nutr 71,** 54-8

Editor's Note: Taurine is synthesized from methionine in the presence of B6, B12, folic acid + zinc. Rich sources are: bean, eggs, pork, fried liver, Brazils, Parmesan Cheese, skim Milk, flounder baked, tuna canned in oil drained, Edam Cheese, lamb, trout (Raw), sesame seeds, salmon canned pink, soya flour, turkey, Fish Cod (canned), pumpkin seeds, sirloin steak, chicken breasts, roast beef, onions, garlic, lentils, soybeans, yoghurt, cooked prawns, cooked liver, calf liver, cottage cheese, chicken liver, boiled eggs, roast veal, pistachios, cashews, walnuts, peanuts, chickpeas, almonds, Lima beans, yoghurt, buttermilk, brown rice. Light deprivation may cause taurine deficiency via low levels in pineal & pituitary glands.

Adverse effects of mini-dose aspirin on renal function

Although very low dose aspirin is prescribed as a cardiovascular disease preventive

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I am most grateful that I heard Dr.Samra on the radio when I did. Now with the help of two other Hypoglycemics (Lorraine Smith and Bill Witton) we have formed a Hypoglycemic Support Group that meets every 3 months. I'm sure having contact with other Hypoglycemics, being able to discuss diets, where to buy particular foods we can eat, and sharing our problems all helps us to cope.

See notice in the newsletter concerning the group meetings.

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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and platelet aggregation inhibitor, no studies until the current one have been published on whether aspirin's renal effects occur at dosages of less than .05gm/day. It is known that high doses of more than 3 gm/day are uricosuric (promoting uric acid excretion), while low doses of 1-2 gm/day cause uric acid (UA) retention. This study evaluated the effects of daily doses of 75mg, 150mg and 325mg aspirin in 49 patients aged 61 to 94 years of age. Baseline and weekly samples of blood and urine were evaluated for uric acid clearance and plasma levels of aspirin. The results showed that patients on doses of aspirin as low as 75mg per day experienced significant changes in renal function and uric acid handling within one week of taking aspirin and this was especially true for those with pre-existing hypoalbuminaemia, so the authors concluded that "given the widespread (and often unmonitored) use of mini-dose aspirin, especially among the elderly, these findings call for clinician alertness as well as for further studies to clarify the mechanisms underlying these phenomena".

Caspi D, Lubart E et al. (2000), The effect of mini-dose aspirin on renal function and uric acid handling in elderly patients, **Arthritis Rheum 43(1),** 103-8

Editor' note: A good natural alternative is Essential Fatty Acids (Vitamin F), which not only reduce the risk of blood clotting, but also decrease blood pressure, aid in the prevention arthritis and helps lowering cholesterol and triglyceride.

Chromium may help treat depression

Dysthymic (disturbance of mood) disorder is a relatively common depressive illness, which does not always respond to anti-depressant medication. Researchers initiated a series of single-blind, open-label trials of chromium picolinate and chromium polynicotonate. Patients experienced a remission of dysthymic symptoms within a period of days to 3 weeks on receiving 200mcg chromium once or twice a daily. The symptoms returned after single-blind substitution of other dietary supplements, but they disappeared on resumption of chromium. The results showed that some patients responded to chromium alone, while others needed both sertraline (Zoloft) and chromium to control symptoms.

Mcleod MN, Gaynes BN, Golden RN (1999), Chromium potentiation of antidepressant parmacotherapy for dysthymic disorder in 5 patients, **J Clin Psychiatry 60(4),** 237-40

THE HYPOGLYCEMIC HEALTH ASSOCIATION
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