The Hypoglycemic Health Association

NEWSLETTER

Correspondence: THE HYPOGLYCEMIC HEALTH ASSOCIATION, P.O. BOX 8, SYLVANIA SOUTHGATE, N.S.W. 2224

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

PATRON: Dr George Samra

***Volume** 14 Number 4* *December, 1998*

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.

NEXT MEETING IS OUR CHRISTMAS MEETING and starts half an hour earlier at 1.30 pm. If possible bring a wrapped present worth about $5.00 for exchange and a plate of sugar-free food. For details see page 2 under "CHRISTMAS PARTY".
The meeting on the 5th December 1998 is in room 2 at **GLEBE NEIGHBOURHOOD CENTRE,** 160 St Johns Road , GLEBE (Corner Mt Vernon St). Remember the entrance to the building is in Mt Vernon St and not St John's Rd. The last meeting was held in a large echoing hall, but now we are booked in a smaller room with a carpet. A public bus **470** from Circular Quay - Town Hall - Central Railway stops and departs right in front of Glebe Neighbourhood Centre **every 15 minutes** on Saturdays. Thus there is easy access to our meeting and we hope to see you there.
We have come to the end of another year and membership fees fall due on the 1 January 1999. Please note from page 2 that membership fees have been increased by $5.00 - i.e., $20 p.a. and concession $15.00 p.a. - due to rising costs and expenditures. Your expiry date is shown on the address label and you can apply for renewal by photocopying the application form on the last page and sending in your remission to the Association c/- PO Box 8, Sylvania Southgate. Professionals have the opportunity to have their business cards printed in the Newsletter for a donation of $50.
The aim of the Association is to promote a community of *complementary medicine* among patients and professionals.

Our Next Public Meeting will be at 1.30 PM
on Saturday, the 5 December, 1998
at ***Glebe Neighbourhood Centre***
160 St Johns Rd (Corner Mt Vernon St)
and our guest speaker is

Dr John Hart,

MSptMed, MB,BS, BPE(Hons)BAppSc, BA

who will be speaking
on the subject of

"Preventative health and longevity"

Dr John D Hart graduated in medicine at The Flinders University of South Australia in 1994 and continued his studies, obtaining a Master of Sports Medicine at the University of NSW in 1998. He has degrees in Arts, Applied Science and Education. His special interests are in health and fitness - optimising health and disease prevention, Nutritional and Environmental Medicine, Sports medicine - Soft Tissue Injury Rehabilitation, Sports Science - Exercise Physiology, Environmental physiology, Nutrition and Elite Performance. With his background in education he loves teaching. Although primarily trained in Adelaide, he moved to Sydney in 1996, working for a year at Royal North Shore Hospital and then in the country. He practises at Sydney Natural Medical Centre at Manly.

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 to any library upon request.

Let us look at some bad news and good news!

New fees for Membership

The Committee regrets that it has been forced to increase membership fees by $5. **As from the 1 January 1999, membership fees will be $20 per annum and $15 for pensioners and students.** This is the first time that we increased the fees since 1985. Costs for printing, envelopes, post stamps, papers and stationery all have gone up. Fortunately, committee members are all volonteers. We want to thank Dr George Samra for acting as our secretary and making his surgery available for committee meetings.

New venue for meetings

One reason for changing our venue to **Glebe Neighbourhood Centre** at Cnr St Johns Rd and Mt Vernon St, Glebe, was the rising cost for hiring a room at our previous venue. The new venue is convenient and can be reached by Bus 470 from the city.

Professional mailing list

Professional health practitioners, doctors and various organisations with similar interests had been receiving free copies of our Newsletters in line with our aim of promoting *complementary medicine* and to foster a bond between complementary doctors and our members (their patients). The so called "Professional List" had grown to a large number over the years and added to our costs of running the Association. The President sent a circular to all professionals asking them to indicate whether they were still interested in receiving the Newsletters. The response was overwhelming. Thus we have been able to reduce the number of "freebies" and at the same have retained about 90 enthusiastic professionals still being in touch with us and being able to provide support to our members. We take the opportunity also to thank the many professionals who have given up their free time and delivered lectures at our public meetings.

Of course, if any member or professional would like to have their doctor or colleague included in the "professional list" please let us know and make sure they are genuinely interested in complementary medicine.

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

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.

Dr Katrina Watson leaves for Melbourne

Dr Katrina Watson, our Treasurer, is moving to Melbourne for family reasons. We are all very sorry and sad to see her go. She is such a friendly and cheerful person, and undertook the treasurer's job with gusto, despite her running a busy medical practice.

Ms Babs Lamont - New Treasurer

Our new treasurer will be *Babs Lamont* from Bowral and we are all glad and relieved that she has come to our rescue for this important and central job in our organisation. Without a treasurer we simply would fold up. Welcome aboard Babs.

Donations received

We thank **Elaine Campbell** for donating her hand-made patch-work cushion that will be raffled at our next X-mas party at Glebe Neighbourhood Centre on the 5 December 1998

We also want to thank **Lydia Beslik** for her generous donation to the Association. Other members who have donated are **Anna Jaccoud,** and **Catherine Sara.**

Let us not forget the donation by **Sue Litchfield** for her cook-books , but even more importantly for her hand-knit jumper. This was raffled and won by **Robert Bates** at our last meeting and contributed $171 to the Association's coffers.

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

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.

CHRISTMAS PARTY

Our next meeting at Glebe Neighbourhood Centtre on Saturday the 5th December 1998 will start one half hour earlier than usual **(1.30 pm)** to celebrate our Super Christmas Party. Members and friends are invited. Please bring along a plate of sugar-free foods.

Presents: The Committee asks every one to participate in the present Lucky Dip. Bring a wrapped present worth $5 with you and mark it "male" or "female"; but even if you don't, you won't be disappointed. There will be presents for kids, and they are welcome.

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 5 September 1998 **Sunny Miller** won the lucky door prize and **Barbara Martin** won the raffle. **Robert Bates** won the highly prized jumper hand-knit by our Sue Litchfield. Thanks a lot Sue. The Association collected about $171 for your efforts.

Fund raising activities

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

INDEX Page

The Art of Breathing in Asthma and other Diseases

By Paul J. Ameisen ,MBBS, ND, Dip Ac, FACNEM

Extracts from the author's book: *Evey Breath You Take,* Lansdowne Australia Pty Ltd, Sydney

I have been a medical practitioner for twenty-one years, with both city and country practices, and in that time I have treated thousands of asthma patients. Like every conscientious medical doctor I have kept up-to-date with the latest research, and with advances in techniques and medication, in order to help my patients to the best of my ability. This has been especially important to me, as

I take a keen interest in respiratory diseases. In addition, my work has been in Australia, where a major respiratory disease has a strong hold: Australia and New Zealand have more asthma sufferers per capita than any other country in the world. More than one million people have asthma in Australia (some estimate nearly two million) — that is, 25% of children, 15% of teenagers and 10% of adults have asthma.

Asthma is on the increase in the industrialised countries of the world. In the USA, 16 million people suffer from it, and three million in the United Kingdom. Boys have asthma more commonly than girls in childhood and about one child in four has asthma at some stage of development. About half of the children with mild asthma will improve and 'grow out of' the condition through their teenage years. The others have to continue with a disease that can interfere with their pleasure in life, their education, their sporting interests, their well-being and even their relationships with family and friends. Adult or 'late onset' asthma also occurs, more frequently in women than in men. These unlucky people not only suffer acute discomfort, disruption of every aspect of their lives and often sheer misery from their condition, but they may also be facing a threat to their life. Not only asthma itself, but deaths from asthma attacks are on the increase. In Australia in 1996, for instance, it is a frightening fact that asthma attacks caused more than 800 deaths.

Medicine in the twentieth century has not coped well with asthma. The number and availability of drugs to treat the disease have been sharply increasing since the beginning of the century — but so has the incidence of asthma. The Asthma Foundation of Australia reported that the incidence of asthma in children in Australia actually doubled between 1982 and 1992. As a doctor I could not help wishing that there was another way of helping a child control his or her asthma, instead of having to fall back on an increase in the drugs I prescribed.

Then, more than six years ago, I first became aware of the work and methods of a certain Professor Konstantin Pavlovich Buteyko, a diagnostic physician whose techniques were considered a breakthrough in Russia, after a lifetime of research and treatment of asthma patients. It was two of my patients who told me about it — a mother and daughter who had attended a clinic in Sydney and had both derived extraordinary benefit from the simple breathing technique that they were taught by the Buteyko practitioner.

I became interested, and I observed the technique over a long period. Doctors are always cautious about any new research or treatments they observe, and I was no exception. But there is nothing more convincing to a scientific mind than genuine, sustained and verifiable results and I eventually became convinced, from the objective evidence, that I was looking at a dramatically effective treatment for asthma. I began referring patients to the clinic and became supervising medical officer, which enabled me to monitor and help my patients and others even more effectively. Consequently I have also been able to make a study of the 8,000 patients treated so far in Australia, and when invited I have spoken on radio and television about the far-reaching beneficial effects of this natural, benign method. My book is the result of six years of research into the method and the results it has achieved for asthma sufferers.

Astma caused by hidden over-breathing

The results are astonishing and suggest a direct link between our breathing patterns and our level of health. The Buteyko theory is that the basic cause of asthma is habitual, hidden over-breathing (literally taking in too much air when we breathe). The treatment is based on bringing the breathing to normal levels and thus eradicating over-breathing (hyperventilation), and reversing the need for the body's defence mechanisms. These defence mechanisms, according to the theory, include: spasm of the airways; mucus production (chest, nose, throat and ears); inflammation (swelling) of the bronchial walls. These defence mechanisms are fully explained in the chapter 'How Does the Buteyko Method Work?'.

The message of the Buteyko method is that when asthma sufferers learn to alter the volume of air they habitually inhale, their asthma attacks can be significantly reduced and the use of asthma drugs and apparatus can be reduced or entirely eliminated.

It is possible that the economies of the industrialised countries worldwide could save billions of dollars spent annually on asthma drug subsidies and hospitalisation, if their health administrators took notice of the advances in asthma treatment pioneered by Professor Buteyko. It is on record as having benefited 100,000 patients in Russia, and is officially recognised by the Russian government. Professor Buteyko's experimentation and clinical trials on documented patients in Russia indicate that the great majority of asthma sufferers over four years old can be significantly relieved by the method, and any individual on asthma drug treatment can reduce that drug intake by 90% or more in a majority of cases.

Outside Russia, the first Buteyko clinical trials on asthma sufferers were completed in 1995 in Australia by Associate Professor Charles Mitchell of the Queensland University Medical School, Dr Simon Bowler of the Mater Hospital and Ms Tess Graham of the Buteyko Group.

The results of the first half of the trial, which were presented to a conference of the Thoracic Society in Hobart on March 30, 1995, supported the findings of Professor Buteyko, and a press release at the time made the general findings public. The Buteyko method is available in all capital cities in Australia[1] and has spread to workshops in country areas. The statistics of more than 8,000 cases so far (1997) in Australia show that the success rate continues to be very high. Asthma sufferers attending the clinics have found that after learning and practising the method they can reduce the use of their relievers and preventers to varying significant degrees. As I write this Introduction, the positive results of the second half of the clinical analysis of the method are due for publication.

It is impossible to overestimate the importance of the Buteyko method for asthma sufferers and their families.

I believe it is the great medical breakthrough of the twentieth century, and I am proud to be able to offer the first ever book on this subject outside Russia. This book is the result of my own investigation of the theory and practice of the method, and relies on my close experience of the clinics and the patients who have benefited from them, over the last six years. I have the sanction of Professor Buteyko and of the Buteyko clinics to reveal the method, its scientific bases and its results.

This book is not a self-teach manual, since a practitioner and a doctor are necessary to monitor each person's progress and give advice as to whether their medication can be safely reduced or stopped. One chapter of this book is devoted to a step-by-step description of the breathing technique itself: the chapter is not an instruction manual, as the method must be taught by a trained teacher.

Thus, through my book, asthma sufferers

and the millions of others who may suffer the hidden effects of over-breathing have an opportunity to assess all the aspects of the method and all the relevant medical background, and to decide whether they would like to eventually learn the technique for their own benefit, or share the discovery with others.

I have great pleasure and confidence in recommending this technique to all asthma sufferers and all practitioners — medical, paramedical, orthodox and complementary.

I am delighted to offer my book to the public, because it is the first time all the facts about the method have been available to everyone, as I believe they should be.

Effective treatment for asthma

The first chapter takes a look into the lungs to give a simple explanation of Professor Buteyko's theory concerning the underlying cause of asthma, what is believed to trigger it, and how it can be treated most effectively.

The fourth chapter in my book, 'How Does the Buteyko Method Work?', gives the full medical theory behind the method, with diagrams and graphs, and the scientifically minded may well wish to turn there first.

Here I offer a broad view, which will help asthma sufferers to understand the research and the theory, both of which concern the way we breathe. Basically, the idea of the Buteyko exercises is to restore healthy breathing, because this is the key to controlling asthma.

I also want to give hope and inspiration to the families of asthma sufferers, especially the parents of asthmatic children, who have been searching for a natural answer to the problems and anxiety they face.

We need carbon dioxide

You may have thought that in a discussion about the lungs we would talk about oxygen first and foremost. But the first thing I want to bring up here is how important carbon dioxide is in the body. In fact we know that each human cell needs a specific concentration of carbon dioxide — about 7% — to sustain normal life.

When human life first began on the planet, the composition of the atmosphere was different from what it is today — there was more than 20% of carbon dioxide in the air that living beings breathed. But by now the percentage has fallen greatly — our air contains only 0.03% of carbon dioxide. Our bodies have had to gradually compensate for this, and they have done so by creating an internal air environment in the small air sacs inside the lungs. With the action of normal, healthy breathing, these air sacs contain around 6.5% of carbon dioxide.

So, as we breathe in and out normally, that 6.5% of carbon dioxide exists inside the lungs, in balance with the oxygen that we also need to stay alive.

Carbon dioxide required to release oxygen from haemoglobin

An important factor that seriously affects that level of necessary carbon dioxide in the lungs is over-breathing, also known as hyperventilation. If we breathe in too great a volume of air for our body's needs, we breathe off carbon dioxide too rapidly, and the lungs are unable to maintain the right level in the air sacs. *When carbon dioxide is low due to over-breathing, this causes a chemical reaction which makes it hard for oxygen to be released from the blood stream into the tissues of the body. The tissues of the body then become starved of oxygen.*

Tissues starved of oxygen cannot be healthy: they become irritable, and smooth muscles react by going into spasm. Smooth muscle is found around our air tubes and around blood vessels, arteries and veins, and forms part of the wall of the intestines.

Oxygen starvation of vital organs (such as the brain) excites the breathing centre in the brain, thereby creating a state of breathing stimulation. This increases the breathing even further, creating a 'shortness of breath' sensation in the already deep-breathing person, which further deepens the breath and creates a vicious circle, because we breathe off even more carbon dioxide.

What are the results of over-breathing?

This century, a Russian respiratory physician, Professor Buteyko, came up with the theory that a majority of the human population actually over-breathes — some more severely than others. Because people are unaware of this factor, he called it hidden hyperventilation — long-term over-breathing not clearly visible to the individual.

He noticed that the result of obvious over-breathing has the equivalent effect of an acute and serious anxiety attack — shaking hands, anxiety, chest pain, air hunger, finger tingles and spasm (tetany), cramps, racing pulse. He went on to believe that the effect of less serious over-breathing, which is not noticed immediately, has equally dire consequences for a person's health, over time.

The amount of air we breathe is measured in litres. With normal breathing we use 3-5 litres of air per minute which results in a healthy level of 6.5% cabon dioxide in the air sacs. In hidden over-breathing we use 5-10 litres per minute resulting in very gradual sickness not easily noticed which develops over many years. When we are over-breathing - or hyperventilating - we use 10-20 litres of air per minute which results in what is known as 'attack', where the adult asthma sufferer, or person with a related condition, hyperventilates rapidly.

In general the person's system becomes ill through over-breathing, and is then more prone to viral illness and allergies. The shift in the rate of body activity disturbs the normal flow of chemical reactions in the body and further illness results.

If over-breathing disturbs our basic total metabolism, as the Professor believes, we can start to understand how it might cause a diverse set of symptoms: bronchospasm (spasming of the air tubes), heart blood vessel spasm, and increased blood pressure. These symptoms are recognised and help us define certain diseases: asthma, angina, hypertension. Professor Buteyko concluded that these in turn, if breathing is not corrected, lead to further deterioration of asthma, sclerosis (hardening) of blood vessels and lungs, myocardial infarction (heart attack) and strokes. The Buteyko theory states that these diseases are the body's defence mechanism against the excessive loss of carbon dioxide through over-breathing.

The chapter 'How Does the Buteyko Method Work?' goes into the medical facts about the body's activity in general, and the other diseases. Here we will concentrate on breathing and asthma. For this, it is important to remember that the human organism tries at all times to keep carbon dioxide at the normal, beneficial level in the lungs. When we over-breathe, therefore, Buteyko explains that the body adopts a defence mechanism to retain carbon dioxide. These are signs of this at work:

1. **Spasm of the airways and air sacs:** they close up to make openings narrower, in an effort to keep the carbon dioxide in the lungs.
2. **Mucus and phlegm develop:** this is another way for the body to narrow the airways in an attempt to trap the carbon dioxide.
3. **Swelling of the mucus lining and the bronchial tubes:** a further way for the body to narrow the airways. Asthma sufferers will instantly recognise the above symptoms. There is another that is not obvious to those who over-breathe:
4. **Increased production of cholesterol in the liver**, which causes a thickening of the cell walls of the blood vessels, which in turn prevents loss of carbon dioxide from the blood vessels back to the small air sacs in the lungs.

Professor Buteyko concluded that to avoid making the body ill through over-breathing, and also to avoid the uncomfortable and unpleasant effects of the defence mechanism at work, the solution was to educate the over-breathers so that they could learn to breathe in a shallower way — so that the lungs could return to normality, that is, keep the carbon dioxide level at around 6.5%.

To achieve this re-education, it was important for people to see what factors were making them over-breathe in the first place.

Triggers that are considered to cause over-breathing

1. **The belief that deep breathing is helpful and improves health.** This is received wisdom in the Western world, though not in Eastern cultures, where shallow breathing is practised for bodily and mental health. We breathe in more air when we exercise, it is true — but it does not follow that regular deep breathing is beneficial. In fact, try to make the barbecue fire catch by breathing in deeply and blowing out hard, and you will rapidly become faint. Observe top athletes and swimmers — these super fit people have the slowest pulse and shallowest breathing in the population. A fit, healthy body breathes slowly and more shallowly. Swimming is the best sport

for asthma sufferers, because swimmers hold their breath while exercising — they practise the Buteyko method without realising it.

2. Stress: from both positive and negative emotions. Both excitement and depression cause stress, and research shows that people under stress over-breathe.

3. Over-eating. When we eat too much the system has to work harder to process the food, and this can cause over-breathing. To avoid this one should not over-eat. It is also a fact that animal protein makes the body work harder. Many asthma sufferers will have noticed that red meat and cheese, for example (animal protein) sharply increase hyperventilation. To avoid over-breathing caused by the food we eat, it is better to eat more plant products than animal products. You should also eat raw food more than cooked food (raw food causes less over-breathing).

4. Lack of regular exercise. Physical activity on the other hand encourages the release of carbon dioxide from the body cells, increasing its level in the lungs. In vigorous exercise (except for swimming) of course we breathe deeply, and this results in a short-term drop in carbon dioxide; but the long-term result of fitness is a higher level of carbon dioxide in the lungs and better nourishment of all the cells in the body.

5. Prolonged, excessive sleep. Professor Buteyko's research demonstrates that lying down for a long time, especially on the back, while asleep or while bed-ridden, causes severe over-breathing. Techniques to avoid over-breathing in horizontal positions are described in another chapter. Patients should sleep only 6–7 hours if possible, on the left side, and breathe through the nose, with the mouth firmly shut.

6. Hot and stuffy environments. We over-breathe when our body detects that the air we are breathing does not contain what we need. On the other hand, mild or cold temperatures all assist shallow breathing — a conclusion reached over 40 years of research and measurement. We soon realise this when we sit in a sauna. Sweating may detoxify the body, but it also creates extra work, causing hyperventilation. When we move from a cooler climate to a hot one, a similar reaction can occur.

7. Bronchodilators. These are standard medication for asthmatics. Bronchodilators give quick relief at first, but Buteyko argues that they in fact cause further over-breathing — because they are designed to open the air passages and maximally keep them open for four to twelve hours, allowing the sufferer to continue what he or she thinks of as 'normal' breathing. Based on Professor Buteyko's research, a person who suffers from asthma is an over-breather — so after two to twelve hours the low carbon dioxide means that the airways will go into spasm again, and the bronchodilator will be needed once more — a vicious circle.

8. Excessive sexual activity. The hyperventilation in sexual activity is obvious — and normal. It is only when this activity becomes excessive because of a sex addiction that hyperventilation becomes a problem, because it lowers the level of carbon dioxide in the lungs.

9. Smoking and pollution. When we walk into a smoke-filled room we may cough — this is because we are entering a situation that is allergic and toxic. We also get the signal 'not enough air', so we over-breathe.

Some people — asthmatics included — react more sensitively to such situations than others, and have the same reaction to pollution, which causes over-breathing.

10. Alcohol and recreational drugs. These put a stress on the body due to their toxicity and overstimulation, and Professor Buteyko's studies give evidence that they lead to over-breathing.

The aim of the buteyko method

The aim is to use a series of regulated breathing exercises to teach the person who over-breathes to breathe a normal volume of air for the rest of his or her life. An adult who suffers from asthma usually breathes 5 to 10 litres of air a minute when he or she is 'well'. During an attack, the rate increases to 10 to 20 litres per minute. The simple, and achievable, aim of the method is to get the volume of air down to normal — 3 to 4 litres per minute. It can be done — thousands of relieved patients have proved it — and the technique is so simple a child can follow the method, and even have fun while learning.

As you will see from the chapter entitled 'A New Lease of Life', the joy of former asthma sufferers, once they have mastered the technique and returned their breathing and their lives to normal, is overwhelming.

1) Buteyko Breathing Australia, free call 1800 658 818

Buteko Coollege for Breathing Disorders PO Box 933, Bondi Junction,02 9962 9033

Criteria for writing artcicles in the Hypoglycemic Health Newsletter

When writing for the Hypoglycemic Health Newsletter, you should have in mind that our readers consist of lay persons with an above-average interest in natural medicine and a professional group of health care workers, doctors, pharmacists, nurses, counsellors and naturopaths. We aim to inform our readers and doctors of the latest in clinical nutrition and complementary medicine. To counter the argument that complementary or natural medicine is based on unproven, unsubstantiated, unscientific and controversial premises, we need to make sure that whatever we claim is supported by publicly accessible evidence from reputable scientific journals.

To improve the readability for this mixed audience the following criteria may be of assistance:

- **Technical terms** should be explained in the article itself or by means of a footnote.
- The article should be divided into **blocks of information** of several paragraphs with a highlighted caption immediately at the beginning.
- Although type-written articles will be accepted, we would prefer articles to be **prepared on a word-processor** and saved on a floppy disk in *Rich Text Format* **(RTF)** either formatted for an IBM or Macintosh computer, accompanied by a **hard-copy** of the articles. The editor uses a Macintosh computer and Word Processor MS Works v 3.0b and Pagemaker 4.

Graphics: These PC formats can be translated to Mac PICT

•AutoCad.DXF; GIF; HarvardGraphics. CGM; JPEG; Lotus.PIC; LotusFreelance. CGM; PCPaintbrush.PCX; TIFF; VenturaPublisher.GEM; VenturaPublisher.IMG; Windows Bitmap.BMP; WindowsMetafile. WMF; WordPerfect. WPG

Database Formats from IBM-compatible computers to Macintosh Computer, ClarisWorks (Windows) v. 1 & 3; dBASE (DBF) II, III, IV; FoxBASE; MS Works (DOS & Windows) v. 2 &3; WordPerfect Works (Windows) v. 2

If in doubt contact the Editor on 9130-6202!

- **References** can be in two forms:
  a) Authors are placed in alphabetical order at bottom of manuscript and then referenced in article by author year, page as for example: (Werbach 1993, 23) or,
  b) References are in numerical order in which they are first mentioned in the text.
- **Reference style:** The titles of journals should be abbreviated according to the style used in the Index Medicus. Reference should include the following, Author Initials, (Year), Title of article (lower case), **Name of Journal (Volume number) (Number):** page numbers. Example:

Prentice, A (1997), Is nutrition important in osteoporosis? **Proc Nutr Soc 56:** 357-367

Book reference:

Author Initials, (year), **Title of book** (Lower case), Publisher, page numbers. Example:º

Werbach MR (1993), **Healing through nutrition,** Thorson. London

Remember many of our readers are taking articles from this Newsletter to their doctors and therefore it is important to maintain high standards of scientific validation.

INTRACELLULAR STARVATION: A FORM OF CELLULAR DEPRESSION

The Common Pathways to Hypertension, Diabetes and Coronary Artery Disease

By Henry Osiecki
BSc (Hons) Grad Dip Nutr & Diet

CELLS require both oxygen and fuel in order to function adequately. If the cell lacks oxygen and shows dysfunction we call this ischemia. However, a lack of fuel, eg intracellular glucose can give rise to such serious clinical manifestations as diabetes, hypertension, hyperlipidemia and coronary artery disease.

Most practitioners are aware that **insulin resistance** is associated with **hyperglycaemia** or too much sugar in the blood, but few would recognise that this may be associated with an intracellular deficiency of glucose or **intracellular fuel starvation** - "starvation in the sea of plenty." As glucose is the main fuel of many cells, it is not surprising that the production of ATP (adenosine triphosphate - the energy currency of the cell) would decrease.

In the normal metabolism of glucose producing ATP, there is sufficient energy to allow the cell to maintain its electrical gradient of sodium, potassium calcium and magnesium ions as well as energy for various enzymatic reactions eg the activity of lipoprotein lipase (See Diagram 1, Normal Cellular Energetics). However, if the cell lacks the energy to do the above, we find the body becomes sluggish. The kidney does not excrete sodium, and this leads to volume dependant increase in blood pressure. This sodium excess also bloats cells, particularly those lining the blood vessels, causing a further increase in blood pressure. The dysregulation of calcium and magnesium metabolism associated with this intracellular fuel starvation causes the vascular smooth muscle to contract establishing this increase in blood pressure.

The body, in response to intracellular fuel starvation, produces more insulin, to try to force more glucose inside the cell. Over time this increase in insulin secretion (or levels) will be insufficient to regulate blood sugar or maintain intracellular glucose levels and diabetes will ensue. Furthermore, this higher level of insulin is associated with cellular receptor insulin resistance, causing physiological changes such as weight gain, elevation in serum lipoproteins and cholesterol levels. These changes occur due to inactivity of lipoprotein lipase, potassium wasting and overactivity of the autonomic nervous system particularly the sympathetic nervous system giving rise to symptoms of increased anxiety, increase heart beat and bloatedness (see Diagram 2, Intracellular Starvation).

Hyperglycaemia also affects the immune system, decreasing the phagocytic function of the polymorphonuclear cells (white blood cells) with an increased susceptibility to infection. All resulting from poor or inefficient energy production due either to an intracellular deficiency of glucose or to the nutrient cofactors involved in its metabolism.

It is obvious, therefore, that increasing the cellular energy production could obviate the above sequale. However we must first look at possible causes of this reduction in energy production.

1. Insulin receptor resistance

This can occur through genetics or more specifically to problems of insulin binding with its cellular receptor. Glucose tolerance factor, may be deficient due to a chromium and B3 deficiency and supplementing with these cofactors may improve the binding of insulin to its cellular receptor. Increasing receptor concentration in the cellular plasma membrane may also contribute to improved insulin sensitivity. Phosphatidyl serine and phosphatidyl choline may be of some benefit here.

2. Inadequate Insulin Secretion

Pancreatic insufficiency of beta cells due to viral infection chemical toxin or defects in glucose metabolism may be instrumental in decreased insulin production and secretion in response to raised serum glucose. Treating for viral infections and increasing intake of vitamin B3 and Nicotinamide Adenine Dinucleotide or NAD (the activated form of B3) may be of some benefit in reducing damage and improving insulin production. Furthermore bypassing the glucose metabolic block by supplementing with succinic acid or maleic acid may be an efficient means of stimulating pro-insulin biosynthesis and insulin release.

3. Lack of Particular Nutrient Cofactors

Nutrient cofactors such as vitamin B3, lipoic acid, carnitine, magnesium, vitamins B5 and B6, manganese, thiamine and Co Enzyme Q10 can all contribute to poor energy metabolism if these nutrients are not present or are activated appropriately. Supplementation with these cofactors is essential in maintaining the machinery of the mitochondrial Krebs cycle at optimum levels. In many disease states these same nutrients are lacking or are deficient, resulting in poor energy production and utilization.

4. Oxidation or Free Radical Damage of Mitochondrial and Plasma Membranes

During any inflammatory process, there is an increase in the production of free radicals. If these free radicals are not quenched or their production reduced, cellular membranes and enzymes can be deactivated. The result is poor energy production or an accelerated use of energy supplies. In either case, energy reserves will become low with the usual pathological sequale.

For instance excessive production of nitric oxide (NO) during inflammation may result in increased production of the "peroxynitrite ion", which is a very powerful oxidising agent. This ion can cause significant damage to the Krebs cycle enzymes as well as uncoupling oxidative phosphorylation. NO at high levels is known to be cytotoxic and is involved in a range of inflammatory, neurodegenerative, cardiovascular and ischemic pathologies. NO inhibits mitochondrial respiration by competing with oxygen binding at cytochrome oxidase in the electron transport chain of the mitochondria and it is this inhibition of energy metabolism which contributes to NO's cytotoxicity and cytostasis observable in some pathologies. Thus, regulation of NO synthesis is important. Reducing NO and quenching this peroxynitrite ion will require supplementation with lipoic acid, glutathione, Co enzyme Q10, vitamins B12 and B2, folic acid and vitamin E (gamma tocopherol). In addition, the lipid membranes may be oxidised by these free radicals so the requirement for omega 3 and 6 polyunsaturated fatty acids may increase.

5. Specific Toxins That Inhibit Mitochondrial Function

Many drugs such as Beta Blockers, HMG - Co A reductase inhibitors, antibiotics, chemotherapeutics, tricyclic antidepressants and radiation exposure can contribute to poor mitochondrial function. Further, toxins such as mercury, pesticides, PCB's and a myriad of other chemicals including metabolic toxins produced in the gut or through poor liver detoxification contribute to this mitochondrial poisoning. Nutrients such as lipoic acid,

Diagram 1 Normal Cellular Eneregetics

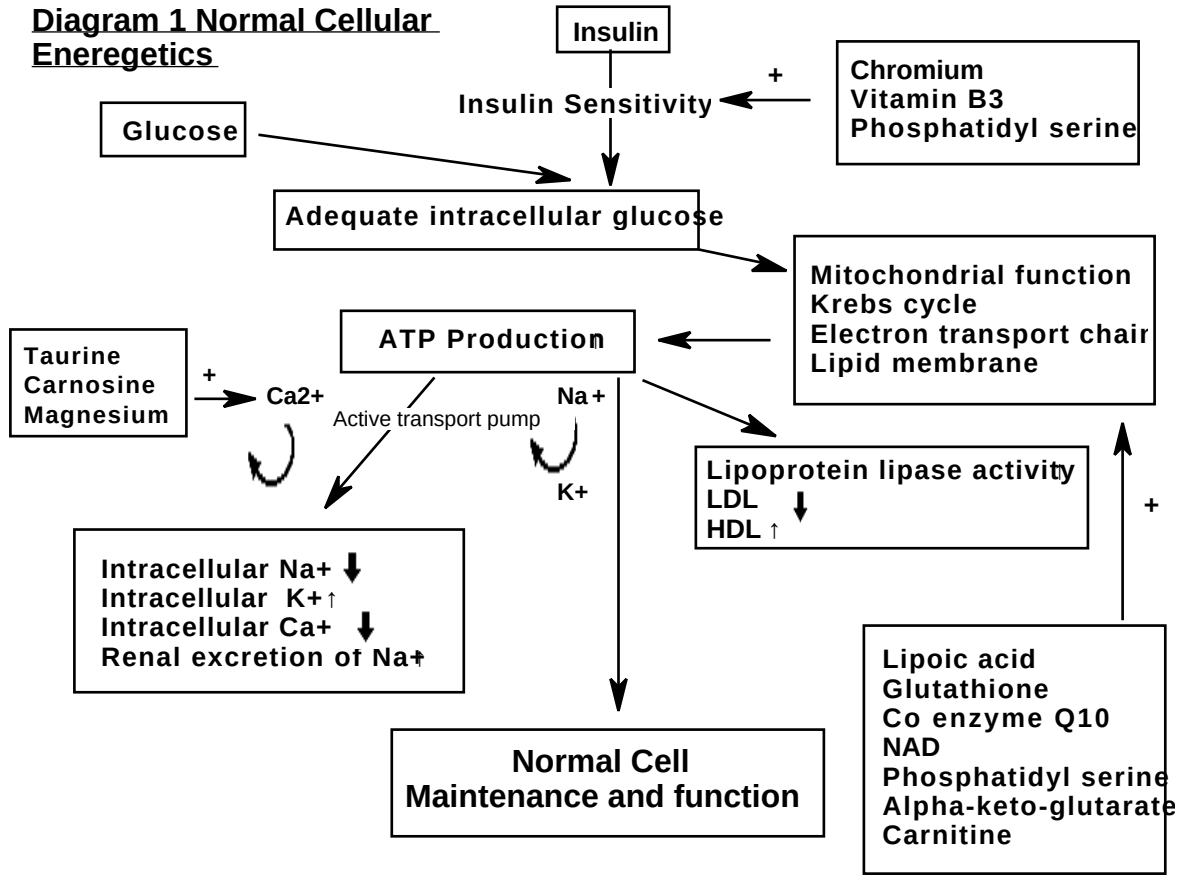
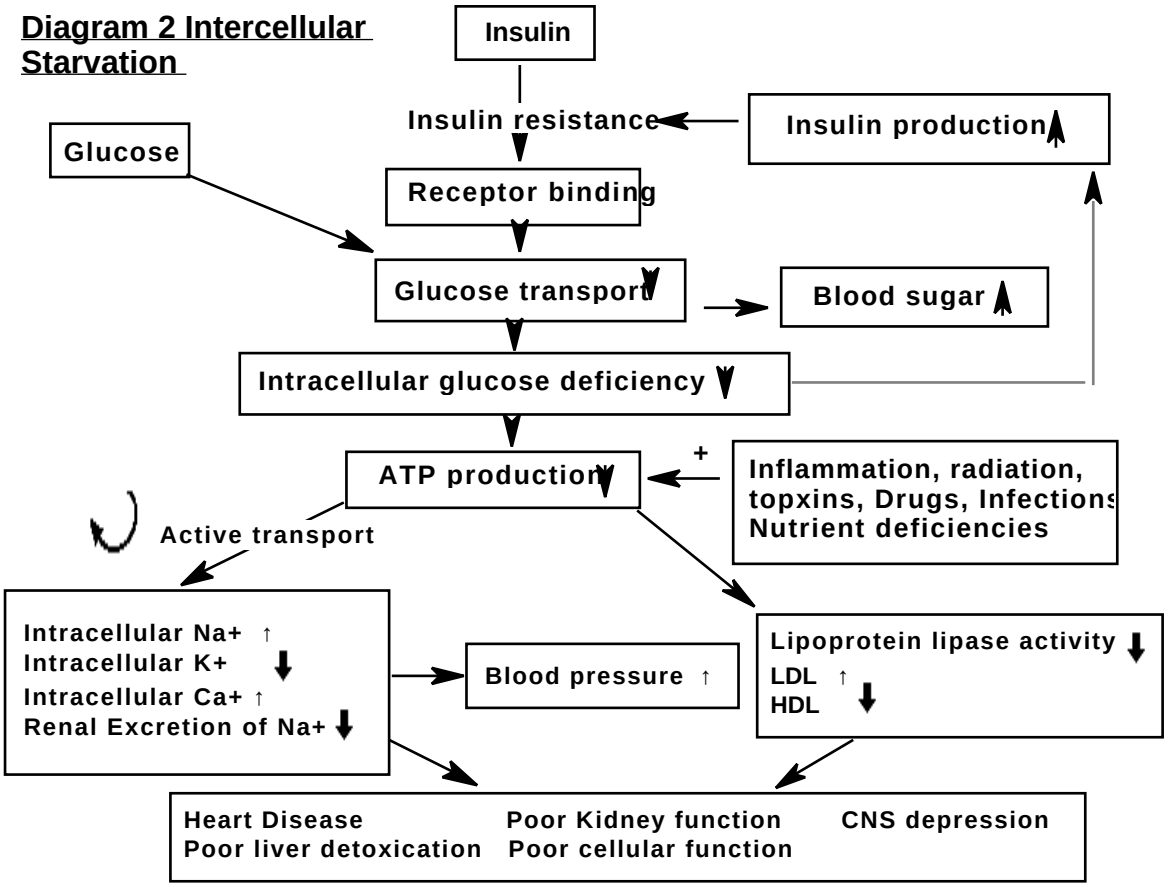


Diagram 2 Intercellular Starvation



glutathione, N-acetyl-cysteine, Co Enzyme Q10, carnitine and vitamins B3 and B5 may reduce the severity of these toxin enzymatic inhibitions and thus improve cellular energetics.

6. Viral or Bacterial Infections

Although associated with inflammation and its particular sequale, infections tend to switch cellular energy usage away from cell maintenance to immune defense. More cellular energy is required as it is used at a faster rate. If the cellular machinery is not sufficient to provide for cellular needs over a prolonged period of time, energy defects and symptoms of chronic fatigue will occur. Treating chronic infections is of primary importance to improving energy status.

In summary, recognising that intracellular starvation can be a contributing factor in many diseases with varying pathologies is a powerful metaphor for understanding and implementing new strategies in treatment. And supplementation with various contingent nutrient factors, including Co Enzyme Q10, lipoic acid, NAD and chromium can be integrated into many conventional treatment protocols for chronic disease such as high blood pressure, diabetes, chronic fatigue, hypoglycaemia and hyperlipidemia. (Diagrams 1 and 2 give a summary of the above discussions).

Further Reading

1. Davidson M B (1995 , Clinical Implications of Insulin Resistance Syndromes. **Am J Med; 99(4):** 420 - 425
2. Laws A, Reaven G M (!992), Evidence of an Independent Relationship Between Insulin Resistance and Fasting Plasma HDL-cholesterol, Triglyceride and Insulin Concentrations. **J Intern Med; 231(1):** 25 - 30
3. Flack J M, Sowers J R (!991), Epidemiologic and Clinical Aspects of Insulin Resistance and Hyperinsulinemia. **Am J Med; 91(IA):** 11S - 21S
4. Pinkney J H, Mohamed-Ali V, Denver A et al (1994), Insulin Resistance, Insulin, Proinsulin, and Ambulatory Blood Pressure in Type II Diabetes. **Hypertension; 24(3):** 362 - 367
5. Reaven G M, Lithell M, Landesburg L (1996), Hypertension and Associated Metabolic Abnormalities - The Role of Insulin Resistance and the Sympathodrenal System. **N Eng J Med; 334(6):** 374 - 381
6. Morris A D, Connell J M (1994), Insulin resistance and essential Hypertension mechanisms and Clinical Implications. **Am J Med Sci; 307(Suppl. 1)**: S47 - S52
7. Olefsky J M, Nolan J J (1995), Insulin Resistance and Non Insulin Dependent Diabetes Mellitus: Cellular and Molecular Mechanisms. **Am J Clin Nutr; 61(Suppl. 4):** 980S - 986S
8. DeFronzo R A (1981), The Effect of Insulin on Renal Sodium Metabolism. **Diabetologia; 21(3):** 165 - 171
9. Reynisdottir S, Ellerfeldt K, Wahrenberg H et al (1994), Multiple Lipolysis Defects in the Insulin Resistance (Metabolic) Syndrome. **J Clin Invest; 93(6):** 2590 - 2599
10. Smith U (1995), Smoking Elicits the Insulin Resistance Syndrome: New Aspects of the Harmful Effect of Smoking (Editorial: comment). **J Intern Med; 237(5):** 435 - 437
11. Kahn A M, Song T (1995), Insulin Inhibits Dog Vascular Smooth Muscle contraction and Lowers Ca^{2+} Influx. **J Nutr; 125(Suppl 6):** 1732S - 1737S
12. Alexiewicz J M, Kurner D, Smorgorzewski M et al (1995), Polymorphonuclear Leukocytes in Non-Insulin-Dependant Diabetes Mellitus: Abnormalities in Metabolism and Function. **Ann Intern Med; 123:** 919 - 924
13. Fournier A H (1998), Intracellular Starvation in Insulin Resistance Syndrome and Type II Diabetes Mellitus. **Med Hypoth; 51:**95
14. Malaisse W J (1994), Current Therapeutic Approach to Diabetes Using Esters of Dicarboxylic Mitochondrial Metabolites **Bull. Mem. Acad. R Med. Belg; 149(8 - 11):** 348 - 352
15. Lass A et al (1998), Electron Transport - Linked ubiquinone-dependant Recycling of Alpha Tocopherol Inhibits Auto Oxidation of Mitochondrial Membranes. **Arch. Biochem. Biophys; 352(2):** 229 - 236
16. Nishikawa M et al (1997), Role of Glutathione and Nitric Oxide in the Energy Metabolism of rat Liver Mitochondria. **Febs. Lett 415(3):** 341 - 345
17. Brown G C (1995), Nitric Oxide Regulates Mitochondrial Respiration and Cell Functions by Inhibiting Cytochrome Oxidase. **Febs. Lett; 369(2 - 3):** 136 - 39
18. Saito M (1990), Polychlorinated bipheryls - Induced Lipid peroxidation as Measured by Thiobarbituric Acid - Reactive Substances in Lower Subcellular Fraction of Rats. **Biochim Biophys Acta; 1046(3):** 301 - 308
19. McCarty F (1981), An Expanded Concept of "Insurance" Supplementation - Broad Spectrum Protection from Cardiovascular Disease. **Med. Hypothesis: 7(10):**1287 - 1302
20. McCarty F (1981), Optimised Mitochondrial Function as a Nutritional Strategy in Cancer Immunotherapy. **Med Hypothesis; 7(1):** 55 -60
21. Luft R et al (1995), Mitochondrial Medicine **J. Intern. Med; 238(5):** 405 - 421
22. Yamamoto N et al (1988), Methotrexate: Studies on Cellular Metabolism. I. Effect on Mitochondrial Oxygen Uptake and Oxidative Phosphorylation. **Cell Biochem. Funct; 6(1):** 61 - 66
23. Zeviani M et al (1997), Mitochondrial Diseases. **Neurol Clin; 7 (1):** 123 - 156
24. Suomedainen A (1997) - Mitochondrial DNA and Disease. **Ann Med; 29(3):** 235 - 246
25. Kendler B S (1986), Carnitine: an Overview of its Role in Preventive Medicine. **Prev. Med; 15(4):** 373 - 390
26. Ernster L et al (1995), Biochemical, Physiological and Medical Aspects of ubiquinone Function. **Biochim Biophys Acta 1995; 1271(1):** 195 - 204
27. Mertz W (1993), Chromium in Human Nutrition; A Review. **J. Nutr; 123(4):** 626 - 633
28. Kimura K (1996), Role of Essential Trace Elements in the Disturbance of Carbohydrate Metabolism. **Nippon Rinsho; 54(1):** 79
29. Demaison L et al (1994), Influence of the Phospholipid n-6 / n-3 Polyunsaturated Fatty and Ratio on the Mitochondrial Oxidative metabolism Before and After Myocardial Ischemia. **Biochem Biophys Acta; 1227(1 - 2):** 53 - 59

RECIPES

HAMBURGERS

Calories per serving: 370; Yield: 4 servings

2 cups ground round
1/4 cup (1 medium) egg
1 onion, chopped fine
1 tablespoon horseradish
1 teaspoon salt
1/2 teaspoon pepper
1/2 teaspoon celery seed
1 tablespoon Worcestershire sauce
1/2 bun per person, 1 slice of bread

Mix all ingredients (except bun) and shape into patties. Broil about 6-7 minutes per side. Serve on bun (or 1 slice bread)

MEAT LOAF

Calories per serving: 250 Yield: 6 servings

1/4 cup (1 medium) egg
2 cups ground round
3 slices bread, cubed fine
1/4 cup catsup
1/3 cup onion, chopped fine
2 tablespoon green pepper, chopped fine
1 teaspoon salt
1/2 teaspoon dry mustard
1 tablespoon prepared horseradish, if desired

Preheat oven to 400 degrees. Mix all ingredients well. Form into a loaf. Place in foil-lined baking pan and bake until done (15-20 minutes)

DEEP DISH APPLE PIE

Calories per serving: 165 Yield: 8 servings

4 apples, pared and sliced fine
2 teaspoons lemon juice
1/2 teaspoon lemon rind, grated
1 1/8 teaspoon salt
1/4 teaspoon nutmeg
1/2 teaspoon cinnamon
Non-nutritive sweetener equivalent to 1 cup sugar
1/2 tablespoon arrowroot powder
1 cup flour, sifted
1/3 cup diet margarine
1/3 cup water

Preheat oven to 425 degrees. Combine apples, lemon juice, lemon rind, nutmeg, cinnamon, sweetener, arrowroot and 1/3 teaspoon salt. Place in well-buttered 9" deep-dish pie plate. Combine flour and 1 teaspoon salt; cut in margarine until consistency of cornmeal. Blend in water; roll out as for a pie crust; place this on top of filling. Bake until crust is brown (about 35 minutes).

FRUIT COBBLER

Calories per serving: 150 Yield: 4 servings

2 cups water-packed fruit (cherries etc.) unsweetened
1/4 teaspoon lemon juice
1/8 teaspoon almond extract
1/2 teaspoon arrowroot powder
2/3 cup juice from fruit
1/2 cup flour, sifted
1/8 teaspoon salt
3/4 teaspoon baking powder
1 tablespoon diet margarine
1/4 cup (1 medium) egg
2 tablespoons skim milk
Non-nutritive sweetener equivalent to 1/4 cup sugar

Preheat oven to 425 degrees. Using shallow cake pan, make a layer of drained fruit. Combine lemon juice, almond extract, arrowroot powder, and drained fruit juice. Pour over fruit. Mix flour, salt, and baking powder, cutting in margarine until mixture is like coarse sugar. Mix egg, skim milk and sweetener. Stir into dry ingredients and spoon onto fruit. Bake until browned (25-30 minutes). Serve warm.

Osteogenesis Imperfecta and Juvenile Osteoporosis

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

Some of the information given is also relevant to adult osteoporosis or other collagen diseases.

Osteogenesis Imperfecta (OI) is one of a number of diseases affecting pre-pubescent children classed as *Juvenile osteoporosis.* OI is primarily a rare genetic disorder and may be classed among the collagen diseases. See list of some other diseases that involve disorders affecting collagen. **(Table 1)** Osteoporosis means 'porous bones' and is characterized by fragile, brittle bones that break easily and heal poorly.

Secondary osteoporosis affect both adults and children and result from an underlying (primary) disorder. Sometimes this may be due to medication to treat a primary disease. Drugs such as *glucocorticoids* (prednisone) used in the treatment of severe inflammations may affect bone density. Immunosuppressive agents used in the treatment of cancer, of hyperthyroidism or hyperparathyroidism or Cushing's Syndrome[1] may affect bone mass. Deficiency of phosphorus - 85% in bones - may occur through excessive intake of aluminium hydroxide antacids.[2] Other causes may be related to behaviour, such as when a child during the peak of bone formation may avoid physical activities for some reason or other, necessary for bone formation.

When no identifiable cause of juvenile osteoporosis can be found it is called *Idiopathic Juvenile Osteoporosis* (IJO). Idiopathic juvenile osteoporosis is usually a self-limiting disorder, and its progress is arrested after puberty with cessation of new fractures.[3]

Symptoms

Being a collagen disorder nearly any organ may be affected, although bone fragility is the most common. There are 4 major types of OI, ranging from mild to severe. The diagnosis is made from clinical, genetic and radiographic features. Scanning electron micrographs of bone biopsies may show marked thinning of bone. The complications of OI and the use of bone mineral density measurements, collagen analysis and prenatal ultrasound imaging are used.[4] OI is classified according to the severity of the disease, called the Sillence classification. Type I is the common mild form, type II is the perinatal (at time of birth) lethal form which is usually fatal, type III is a severe form, and type IV is a moderately severe form.[5]

The cardinal symptoms are bone-pain and pathologic fracture, which is often recognized before birth, is frequent during infancy and childhood, then decreases at puberty. Bone mineral density is markedly decreased in OI, especially of the lumbar spine. Bone deformities are frequently observed in the long bones of the extremities, and spinal deformities and compression fractures are also common. Growth retardation is extremely severe, especially in type III.[6] Other signs are a family history of frequent bone fractures, small stature, blue sclera ("white" of the eyes), possible hearing loss and dental problems. Patients with OI often have irregular bone patterns in the skull called 'wormian bones'.

Table 1

Some diseases that involve disorders of collagen

Ankylosing spondylitis (Marie-Strümpell disease, Strumpell disease), ankylosis, arthrogryposis multiplex congenita, arthritis, Bruck syndrome, cutaneous lupus erythematosis (Chronic discoid LE), dermatomyositis, Ehlers-Danlos syndrome, Marfan's syndrome, hyperparathyroidism, hyperplastic callus, glaucoma (some research), mucopolysaccharidosis (Hurler's Syndrome, Hunter's Syndrome, Sanfillipo syndrome, Morquio syndrome, Scheie's syndrome), necrotizing vasculitis, osteochondrodysplasias (Hajdu-Cheney syndrome), osteoarthritis, osteogensis imperfecta, osteoporosis, Paget's disease, polyarteritis (polyarteritis nodosa, periarteritis nodosa, necrotisng angiitis), polychondritis, polymyositis, rheumatic fever, rheumatoid arthritis, scleredema, scleroderma (progessive systemic sclerosis), solyomyositis (dermatomyositis), spondyloepiphyseal dysplasia, systemic lupus erythematosis (SLE)(Dessiminated LE), Van der Hoeve-de Kleyn syndrome.

Bone formation

Bones are part of an active, living tissue continually remodelling itself. Bone tissue consists of both cells and intercellular matrix or mould. Embedded within the bone layer tissues are cells that lay down new bones, called *osteoblasts* and those that break down bones, called *osteoclasts.* The constant activity of these cells allows bone to be remodelled throughout life. For example, orthodontic appliances cause bone resorption on the pressure bearing side and bone formation on the opposite side of teeth in children. Bone formation are regulated by *parathyroid hormone, calcitonin* and *1,25-dihydroxyvitamin D3.*

Parathyroid hormone (PTH), secreted by the thyroid glands, stimulates the ***osteoclasts*** to dissolve calcium phosphate crystals from bone tissues, when blood calcium ion levels (Ca++) are low. This raises blood calcium levels and is called ***resorption.*** Calcium ion levels are critically important in many physiological processes, such as contraction of muscles. PTH also stimulates renal excretion of phosphates. In addition, PTH stimulate the reabsorption of Ca++ in kidneys with the effects of raising Ca++ levels without promoting the deposition of calcium in bones. Finally, PTH promotes the formation of the active form of vitamin D (1,25-dihydroxyvitamin D3) in the kidneys. This vitamin (hormone) functions to increase the absorption of calcium and phosphates from the intestines.[7]

Calcitonin is secreted by the C cells of the thyroid glands and has the opposite effects to parathyroid hormone; namely it inhibits the action of *osteoclasts* and therefore reduces bone loss.(Lehninger, 746) In this respect it acts similarly to oestrogen. As we age, production of calcitonin diminishes. Women have usually lower levels than men and this may explain a higher incidence of osteoporosis among women.

In **osteogenesis imperfecta** there is a genetically heterogeneous defect of collagen synthesis, associated with low bone mass, which is thought to be due to reduced bone matrix formation. The latter stores the calcium and phosphates and other micro-minerals[8] in the form of ***hydroxyapatite.*** The inability of abnormal collagen to participate in mineralisation may be caused by its failure to interact with other bone proteins.

As a collagen disease, it should be remembered that collagen makes up almost one third of the protein mass of vertebrates. Collagen and elastin are the major fibrous proteins of connective tissues. These include the tissues enveloping the blood vessels, tendons, ligaments, structural layers under the skin and forms the extracellular ground matters between cells. Without collagen we would literally fall apart. Collagen fibrils do not stretch, whereas elastin fibrils are elastic. A cow weighing 1000 lbs is largely supported and held together by strong tough collagen fibres in its hide, tendons, cartilage, and bones (Lehninger, 157).

The cornea of the eye has sheets of collagen fibres, hence OI is often accompanied by 'blue sclera'. (Lehninger, 157) Collagen is mainly indigestible but boiling in water transform collagen into *gelatin* which is the basis of gelatine desserts.

The strength of collagen lies in the fact that it consists of three strands of chains having a specific sequence of amino acids curled around each other to form a triple-stranded collagen helix like a rope.[9]

The composition of these chains depends on the type of collagen. There are five types (type I, II, III, IV, V) depending upon the tissue involved. The one we are interested in is type I collagen that supports the skin, tendon, bone and cornea. The precursor to collagen is procollagen[10], secreted by specialised cells (fibroblasts) in connective tissues.[11] The genetically defective procollagen is believed to be responsible for the formation of abnormal collagen.

In the most prevalent type I collagen, two chains are similar and the third has a different composition.[12] Every third residue on each strand of collagen is glycine. **Glycine** is an important amino acid precisely because it occupies very little space and thereby allows different polypeptide strands to come together. The mutation of a single glycine residue - for example its replacement by cysteine or serine - in collagen causes a disruption in the triple helix, leading to the formation of abnormal collagen, characteristic of *osteogenesis imperfecta* (Stryer, 266). The abnormal collagen in the bone matrix causes the bone deformity for it cannot store the inorganic minerals - hydroxyapatite - that form bones. The medical literature suggests that other kinds of mutations also occur. Thus the genetic defect is located in procollagen type I

Medical Treatment

Being a genetic disorder traditional medicine is somewhat limited. **Calcitonin** - a potent inhibitor of osteoclasts - has been the most common therapy for OI and osteoporosis in general.[13] Its cost and side-effects (nausea, flushing, GI disturbances, urinary frequency) makes this drug of limited use.

Bisphosphonates - synthetic compounds that suppress bone resorption and reduce bone turnover - are effective in the treatment of patients with postmenopausal osteoporosis and have been shown to increase bone mass and reduce significantly the frequency of new vertebral fractures in controlled studies. These drugs include alendronate, clodronate, itedronate, olpadronate and pamidronate, all with their own side-effects.

In 1985 scientists reported for the first time the short-term effects of treatment of a patient with idiopathic juvenile osteoporosis with the nitrogen-containing bisphosphonate ***pamidronate*** (3-amino-1-hydroxypropylidene-1,1-bisphosphonate).[14] They found that this drug was less toxic than the first developed bisphosphonate, etidronate, which has a narrower therapeutic margin. *Pamidronate* inhibits the osteoclast more than the osteoblasts, thus tipping the balance in favour of bone formation. Compared with oestrogens, bisphosphonates are bone-tissue specific, have equal or greater antiresorptive effect and have fewer side effects and no known risk for carcinogenesis. Some studies suggest that bisphosphonates (and therefore pamidronate) may therefore be beneficial in postmenopausal osteoporosis. They also hold promise in treating male osteoporosis and steroid-induced bone loss.[15]

Many studies now support the use of pamidronate for the treatment of osteogenesis imperfecta and other osteoporotic disorders in young people, because it is well tolerated with fewer side-effects. There have been reports of dramatic improvements in symptoms, such as bone pain, in a matter of weeks.[16] However as bisphosphonates tend to accumulate in the skeleton there has been some concern with the long-term effect on future suppression of bone metabolism in later life.[17] These concerns require further investigations, but thus far no adverse effects have been reported.

It is understood that side effect of *pamidronate* includes bone pain and nausea. Bone pain will be dealt with later on, but it is suggested that there are several herbals to lessen the effects of nausea. These are: Ginger powder (Zingiber officinale) as a first choice, Peppermint (Mentha piperita), Clove tea (Caryophyllus aromatica), Pineapple (for its digestive juices), anise seeds (chew the whole seeds), catnip, chamomile, fennel, fenugreek, golden seal, papaya and cayenne pepper to mention just a few. A herbalist should be of assistance here.

A clinical trial for the use of pamidronate in osteogenesis imperfecta is under way in Sydney under the supervision of Professor D Sillence, Department of Clinical Genetics, Royal Alexandra Hospital for Children, Phone: 02 9845 3233, Fax: 02 9845 3204. Any queries about the drug should be addressed to him.

Nutritional treatment

Thus far there seems to be little evidence that nutritional treatment <u>alone</u> will improve osteogenesis imperfecta, because its underlying cause is a defective gene in the procollagen responsible for abnormal collagen formation. Further research in clinical nutrition specifically dealing with osteogenesis imperfecta is urgently required. However, nutritional supplements used as complementary treatment for OI and other collagen diseases - including adult osteoporosis - is expected to greatly improve the condition.

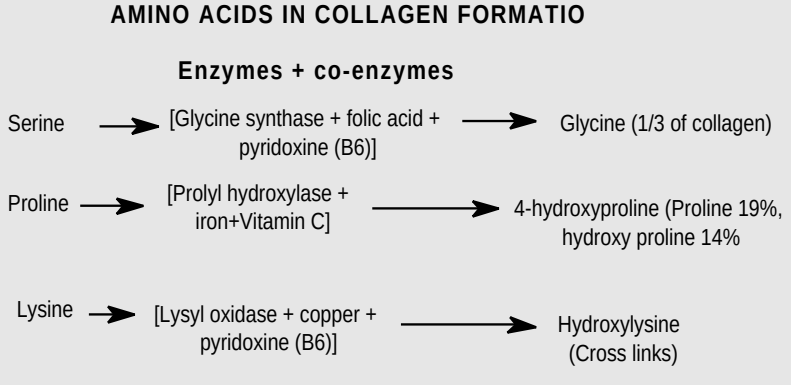
In juvenile osteoporosis one needs to keep in mind that children's taste are finicky and they often are recalcitrant dieters. Nutritional supplements are in two forms: synthetic supplements and natural sources of food items.

The disadvantage of synthetic supplements is that they are usually supplied in isolation from other nutrients for proper utilization in the body (such as calcium supplements without considering the effects on iron absorption or beta-carotene supplementation without other carotenoids). Furthermore, they may have adverse side-effects or disturb the finely-tuned metabolic balance and often are costly to the patient. On the other hand, synthetic supplements in megadoses - such as vitamin C - can have greater therapeutic effects. The B complex vitamins in tablet forms are now affordable to most patients and these supplements should form the starting point in any nutritional treatment.

Natural sources of nutrients have the advantage that they are usually combined with other necessary nutrients and simply come with the food we eat. It allows patients to select their foods according to taste. One problem may arise from the emergence of genetic engineering whereby food scientists are altering the nature of food for commercial purposes with unforeseen consequences.

*In **Table 2,** I have given a list of food items*

Figure 1



- showing approximate quantity per 100g of food - that may be of benefit for those suffering from osteoporosis. They are listed in descending order - from highest to lower concentrations. Most of the information is culled from various tables of food compositions which may not reflect Australian sources. Also many tables give different figures. But the great advantage is that they are virtually non-toxic. However, a drawback is that quantities in natural sources are not controlled and synthetic supplements may be more convenient and more accurate when targeting specific organs or disorders.

Collagen formation

Collagen contains about 35 per cent glycine and about 11 per cent of alanine residues. A high percentage of proline and hydroxyproline amounting to 21 per cent are rarely found in proteins other than collagen and elastin. Bone formation needs collagen as a matrix to deposit the inorganic calcium phosphates. *Glycine* (one third of collagen) is the smallest amino acid and therefore allows different polypeptide strands to come together. Glycine is a nonessential amino acid (NEAA), that is, the body should be able to synthesize it from its precursor *serine*, another nonessential amino acid. Mutation of a single glycine in collagen is the underlying cause of osteogenesis imperfecta. It is significant that in osteogenesis imperfecta the glycine residue is often replaced by serine or cysteine in procollagen, and one wonders whether the defect lies in the enzyme *glycine synthase,* which converts serine to glycine. This enzyme is dependent on tetrahydrofolate, which is the active form of the vitamin **folic acid** and **Pyridoxine (B6)**. (Stryer 580, Lehninger 621). I have not been able to find evidence that glycine supplementation plus necessary co-enzymes - to overcome defective glycine synthesis - will be helpful for those suffering osteogenesis imperfecta. Unfortunately, pharmaceutical companies are unlikely to spend research funds to investigate this, because nutrients are non-patentable and therefore non-profitable. Such research will have to come from universities or from the practice of complementary doctors or nutritionists, who have always been in the fore-front of clinical nutrition.

Another important component of collagen is ***proline.*** This is a precursor of *4-hydroxyproline* - a collagen compound - via the enzyme *prolyl hydroxylase* which has a ferrous ion at its active site. (Stryer, 262) A noteworthy feature of this hydroxylation reaction is the need for ascorbic acid (vitamin C). Thus the production of hydroxyproline for collagen depends on **iron** and **vitamin C.**[18] Proline supplements are given in 500mg to 1,000mg daily with vitamin C. (Chaitow, 103)

Collagen fibres are strengthened by cross-links. These are formed by *lysine* and *hydroxylysine* via the enzyme *lysyl oxidase*, which contains a **copper**[19] ion and is dependent on**pyridoxine** (B6) (Stryer 271). Lysine is an essential amino acid (EAA). We are unable to synthesize it and need it from our diet. See Table 2.

Thus collagen formation depends on: glycine, lysine, folic acid, proline, vitamin C, iron, copper and vitamin B6. See **Figure 1**.

It is interesting looking at Table 2 that ***gelatine*** has a high content of both lysine and glycine. (Gelatine contains among others: alanine 9.27%, arginine 7.45%, aspartic acid 5.63%, glutamic acid 9.58%, glycine 23%, lysine 4%, proline 13%, hydroxyproline 11%, serine 3.5%, threonine 1.82%, (with sodium 32mg, potassium 22mg, magnesium 11mg, calcium 11mg, iodide 6 mcg, vitamin B6 5.8 mcg per 100g each).[20]

Gelatine or *jelly as a desert* made from gelatine would make an ideal and tasty supplement for children with osteogenesis imperfecta. However, it should be remembered that it is devoid of any B complex vitamins (except a trace of B6) and its amino acid content is very skewed and unbalanced. Adelle Davis warns that excess gelatine should be avoided by people suffering from gout as excess glycine is excreted as uric acid in the urine. (Davis, 168) Also in kidney stone formers, 40 per cent of urinary oxalate is believed to be derived from glycine.[21] Patients with hyperoxaluria (high oxalate levels in urine) should supplement with **pyridoxine** (B6) (150 mg daily), as vitamin B6 is required to break down oxalic acid in the body. (Werbach 1993, 177). The more glycine is given the greater the urinary excretion of oxalic acid, and this excretion is decreased when vitamin B6 is given with glycine (Davis, Adelle 183). For those stone-formers that excrete calcium oxalate, increased magnesium supplementation (300 mg daily), plus pyridoxine (B6) is recommended. Magnesium oxalate - which does not form stones - reduces the amount of oxalate to combine with calcium.[22]

Bone formation

In osteogenesis imperfecta the major problem is collagen formation. Once the defective gene has been by-passed by drugs or other treatment, the problems associated with adult osteoporosis virtually do not exist or at least they are a different kettle of fish. Nevertheless, nutritional treatment of the latter may offer some assistance in a speedy recovery of juvenile osteoporosis.

Bone consists of concentric layers, called *lamellae*, around blood vessels. The bone-forming cells, *osteoblasts (or osteocytes)* are located between the lamellae or *lacuna*. They are kept alive by the supply of blood through *canaliculi* or little canals radiating around the osteocytes (Volkmann's canals). These units of bone structures are called *haversian system.* Bone consists of an organic phase, which is nearly entirely collagen, and an inorganic phase, which is calcium phosphate called ***hydroxyapatite***. These crystals are composed of calcium phosphate, calcium carbonate, with small amounts of magnesium, fluoride, sulphate and other trace elements. Specifically, the mineral structure of *hydroxyapatite* has the composition Ca10(PO4)6(OH)2. Collagen is required for the deposition of calcium phosphate crystals to form bone.

Calcium essential in bone formation

Many people believe that osteoporosis is due solely to calcium deficiency, but nutrients such as vitamin C, D, E, K, magnesium, phosphorus, silicon, boron, strontium, zinc, manganese[23],[24] and copper also play an important role. (Balch, 414) However too much magnesium and/or phosphorus found in carbonated drinks and many processed foods can inhibit calcium absorption, because these minerals compete with calcium for absorption in the blood and bone marrow. The highest rates of osteoporosis occur in countries with the highest intake of calcium, suggesting that low calcium intake is not the main factor in osteoporosis.[25] For example, excessive sugar consumption which provokes an excessive insulin response has been associated with increased calcium excretion in stone formers[26], suggesting that **hypoglycemia** or hyperinsulinism promotes urinary calcium excretion. (Davis, Adelle 233)

Most experts advise, that calcium in supplemental form should be in vicinity of 1,500 - 2,000 mg/day[27]. In tablet form the important information to look for is *elemental calcium* because calcium is usually combined with other substances to make them stable tablets. Calcium carbonate, calcium lactate, calcium citrate and calcium gluconate tend to dissolve well in the stomach. According to Werbach women over 35 who ingest less than 1 g/day of calcium before menopause, or less than 1.5 g/day after menopause are in negative calcium balance. (Werbach 1995, 219) Reasonable daily requirement would be 800 mg for non-osteoporotic patients.[28] Calcium is better absorbed when taken with a light meal than when taken alone.[29] Thiazide-type diuretics increase blood calcium levels, and complications may result if these drugs are taken with calcium and vitamin D. (Balch, 416).

But there are reports of adverse effects of high calcium intake above 2 grams per day. Symptoms may include irritability, headache, soft tissue calcification, renal failure, kidney stones (low calcium intake also associated with kidney stones). High calcium intake has been shown to interfere with absorption of iron, zinc, magnesium and phosphorous. [30]

Calcium supplements in the form of **bone meal, oyster shell and dolomite** are not recommended, because they are difficult to absorb and may contain contaminants like lead.

Calcium citrate may be more bioavailable than other calcium salts [31], [32]. 40 per cent of menopausal women have been found to have no basal gastric acid secretion[33] and deficiency impairs calcium absorption and increases urinary calcium excretion. [34]

Calcium absorption from food is not impaired by the lack of hydrochloric acid in the stomach (achlorhydria).[35]. Calcium from milk is 5 times better absorbed than from spinach, since the calcium in spinach is bound to oxalate.[36] Other high oxalate containing foods are: asparagus, broccoli, beet green, chard, chocolate, rhubarb, turnip green, collard greens, mustard greens, although they do not interfere with absorption of calcium from other foods simultaneously ingested.[37]

Animal proteins, salt and caffeine increase calcium excretion. Meat contains twenty times more phosphorus than calcium, leading to loss of calcium in bone to maintain the calcium/phosphorus balance. Fat, phytates and oxalates decrease calcium absorption, while sugar and calcium increase calcium absorption[38], but they also can cause an increase in calcium loss in the urine[39]. B vitamins decrease calcium excretion. Prentice claims that caffeine increases calcium excretion by only a small amount of 3 mg for a cup of brewed coffee which can be offset by adding milk to the coffee.[40] In a group of Greek men and women, who have 50% less fractures than in the USA, coffee consumption was found to have no discernible effect, but researchers suggested that regular consumption of olive oil may beneficially affect bone mineral density. The high vitamin E content of olive oil was thought to influence the mechanism by which prostaglandins affect bone resorption.[41] Nevertheless, most studies link coffee consumption with calcium excretion in the urine and loss of bone mineral density. Therefore coffee consumption should be restricted.

Fatty acids (fats) may decrease calcium absorption, because they form calcium soaps in the gastrointestinal tract. [42]

Milk sources of calcium

The richest source of calcium is found in dairy products, such as milk and cheeses (see Table 2). However, there is considerable controversy about the consumption of milk. It has been argued that the countries that consume the highest amounts of dairy products also have the highest rate of osteoporosis.[43] Sherrill Sellman (author of *Hormone Heresy: What women MUST know about their hormones*) quotes a one year-long study of 22 postmenopausal women which showed little improvements in calcium levels when their diets were supplemented daily with three 300mL glasses of skim milk (equivalent to 1,500 mg of calcium).[44] A twelve year study of 78,000 women showed that milk consumption does not protect against hip and forearm fractures. Female milk-drinkers actually had a significant increased risk of fracture, and teen-age milk-drinking was not protective against osteoporosis.[45] However, another study recommends that adult osteoporosis and fractures in later life can be prevented by encouraging children and young adults to drink milk in order to improve peak bone mass.[46]

The problem with milk may well be its digestion in adult life. There is a great difference between cow's milk and human milk among nine of its components. About 10-20% of children are allergic to the protein in milk, in particular beta-lactoglobulin. Human milk has alpha-lactoglobulin. Cow's milk has higher levels of total proteins, casein:whey, saturated fats, calcium, phosphorus, less lactose and vitamin C. Most non-Caucasians are lactose intolerant, because they lack an enzyme, *lactase*, that split lactose into glucose and galactose. Most people lose their intestinal lactase by the third year. Northern Europeans who since time immemorial raised cattle have retained their ability to synthesize lactase and can consume milk throughout their lives without ill effects. They seem to have larger amounts of lactase to digest milk products. This could well be compensation for the lack of sun in Northern Europe, that otherwise would faciliate calcium absorption (via vitamin D) (Trattler, 470). Lactose intolerance is inherited from both parents. People with lactose intolerance often can tolerate yoghurt or butter-milk (fermented milk). Yoghurt stimulates the synthesis of lactase. Besides its "friendly bacteria" (lactobacillus acidophilus, L. bulgaricus), yoghurt encourages the establishment of a balanced gut "flora". Thus it seems that although milk provides a rich source of calcium, other component (lactose, beta-lactoglobulin) or lack of components could well interfere with the proper utilisation of its calcium content.

Two different kinds of bones

There are two kinds of bone: *cortical* or compact bone which constitutes 80% of the skeleton found in the limbs, and *trabecular* bone or spongy bone primarily found in the spinal vertebrae. Trabecular (spinal) bones are more prone to osteoporosis because 40% is dissolved and reformed each year compared to only 10% of cortical (limb) bone. Calcium supplementation appears to be more effective in preventing cortical bone loss in the limbs and preventing hip fractures. (Werbach, 1993, 219).

Acid or alkaline foods

When food is burned the ash that remains is either acid of alkaline. *Acid ash-producing foods promotes the loss of calcium* from the body and therefore increases bone-loss.

Acid ash-forming foods are: Alcohol, asparagus, beans, Brussels sprouts, buckwheat, catsup, chick-peas, cocoa, coffee, cornstarch , cranberries, eggs, fish, meats, flour based products, legumes, lentils, meat, cheese, mustard noodles, nuts and seeds, oatmeal, olives, organ meats, pasta, pepper, plums, poultry, prunes, sauerkraut, shellfish, soft drinks, refined sugars, tea, vinegar.and these should be restricted.

Alkaline ash diet that promotes calcium absorption are: Avocados, corn, dates, fresh cocnuts, fresh fruits (except cranberry and plums), fresh vegetables (except for lentils and corn), honey, maple syrup, molasses, raisins, soy products, dairy products (except cheese).(Balch, 109)

Adherence to either an acid or alkaline ash-producing diet should be used as a guidance only as a varied diet will provide a balance.

Lacto-ovo-vegetarian females were found to have less bone mineral loss than omnivorous females, probably due to lower calcium intake, or the higher phosphorous intake among omnivores. This was again confirmed in a most recent study of 76 vegetarian and 109 omnivorous Chinese women between the ages of 70 and 89. This showed that vegetarians had a significantly higher calcium intake than the omnivores. The lacto-vegetarians had a significant higher intake of calcium than vegans.[47]

It has already been mentioned that **Vitamin D** (1,25 dihydroxycholecalciferol) promotes the intestinal absorption of calcium and phosphorus. Alternative sources of vitamin D are cod-liver oil and vitamin D fortified foods. Recommended daily intake of vitamin D is 400 IU irrespective of age. Deficiency of vitamin D may lead to *osteomalacia*, a common occurrence among Bedouin women who are clothed so that only their eyes are exposed to sunlight. (Stryer, 570)

Avoid **aluminium containing antacids** as even small amounts bind with phosphorus causing its depletion. This affects the phosphorus/calcium ratio and will increase in fecal calcium excretion, resulting in a negative calcium balance.[48]

Ross Trattler claims that **cigarette smoking** increases the acidity of the blood and thereby inhibits the conversion of vitamin D into its active form. (Trattler, 470) Another factor would be that smoking places a greater demand on vitamin C in defence against free radical oxidation[49] and that less would then be available for hydroxyproline production in collagen formation.

Magnesium deficiency has been associated with abnormally low concentration of calcium in the blood (hypocalcaemia)[50] and is believed to cause decreased osteoblastic and osteoclastic activity. Trabecular bone (spongy bone) from osteoporotic women was found to contain less magnesium than in controls and was associated with cessation of bone growth, bone fragility and larger bone crystal formation.[51] Werbach mentions that magnesium is required for the activation of alkaline phosphatase, an enzyme involved in forming calcium crystals in bone and in converting vitamin D into its active form. It also influences bone density by maintaining proper levels of parathyroid hormone[52], which stimulates the resorption of calcified bone (Werbach 1995, 220).

Fluoride, a compound of fluorine, is said to increase bone formation and the number of osteoblast. It is an essential element required in the diet to form bones and teeth. It is incorporated into hydroxyapatite to form *fluoroapatite* which causes hydroxyapatite to become larger and harder (Lehninger 783). This is important for growing children.

But there have been several studies suggesting that supplementation can easily lead to toxicity resulting in skeletal and dental fluorosis. This may especially be the case in temperate countries where the average person tends to drink more fluoridated water.[53], [54]. Fecal excretion of copper increases with increased fluoride intake which could affect the *Lysine oxidase* which forms the cross links in collagen. In case of suspected fluoride poisoning an experiment with lambs has shown that supplemental boron had a beneficial effect in counteracting adverse effects of fluoride.[55] The fluoridation of water (1ppm) is a controversial issue. It can be avoided by selecting fluoride from natural sources. Synthetic supplementation is perhaps best avoided. Fluoride occurs naturally in fruits, vegetables, grains, fish and tea-leaves and is usually quite

small (0.3-0.8mg/day) (Diesendorf, 306). See sources of fluorine in Table 2.

Vitamin K is required for the production of a non-collagen protein by osteoblasts called *osteocalcin* which chelates the calcium and holds it in place within the bone. Osteocalcin levels is a good marker for bone growth. Hence vitamin K deficiency may not only result in osteoporosis, but also in poor fat absorption and gastrointestinal disorders. Vitamin K is found in green leafy vegetables. Fat-soluble chlorophyl capsules are a good source. Osteocalcin levels were found to rise in a group of elderly women who received essential fatty acids (evening primrose oil plus fishoil). Evening primrose oil *alone* appeared to have no effect.[56]

Strontium should not be confused with radioactive strontium-90 which like strontium accumulates in bones and teeth and is extremely toxic. Non-radioactive strontium occurs in relatively large concentrations in bones and teeth where it contributes to bone strength. This element is closely related to calcium and it can replace a small proportion of the calcium in *hydroxyappatite* crystals in bones and teeth, where it provides additional strength. It occurs naturally in food, especially drinking water. Its availability depends on the soil, where the food is grown. Because of agricultural practices which replace only three or four basic mineral instead of the fifteen or so trace minerals in their fertilizers, soil content has become poorer over the years. Much of strontium is removed when grains are refined. As more and more soil erodes and is washed into the sea, the sea plants will take up these minerals as they grow. Hence seaweeds is a rich source of these lost minerals. **Kelp** contains 0.10 per cent of strontium as well as many other trace minerals that are not available in land plants. (Adams & Murray, 53) In one stud y, quoted by Murray and Pizzorno 85 per cent of patients experienced a marked reduction in bone pain and 78 per cent displayed increased bone density with strontium supplementation.(Murray & Pizzorno, 461)

Boron may prove to be an interesting mineral playing a role in the treatment of osteoporosis. It occurs in nature primarily as borax. According to Gaby (Gaby, 59), boron is known to form complexes with organic compounds that contain hydroxyl groups (combination of oxygen and hydrogen atoms - chemical symbol -OH). The synthesis of hormones, such as oestrogen and testosterone, involve one or more hydroxylation steps and hence boron supplementation has been shown to increase the levels of both these hormones [57], [58]. So boron could be expected to play a role in the prevention of adult osteoporosis (menopausal) and benign prostatic enlargement. However, one study has cast some doubts when seven women, taking additional boron supplements following a 3-week low-boron diet, did not show any effects in steroids. The authors suggest that this may have been due to faulty experimental design.[59]

Boron is known to enhance the conversion of vitamin D to its active form 1,25-dihydroxyvitamin D [60]. Boron is also known to counteract adverse effects of fluoride toxicity (Vashistha).

There appears to be a comfortable safety margin for boron. No adverse reactions in humans have been reported from 117 mg/d of boron in diet. Fatal toxicity is estimated at 3-4 g/day (Naghii et al 1993). Nutritional requirement for boron is estimated at 1-3mg/day (Gaby 62). Boron supplementation would appear to be an integral part in the treatment of osteoporosis. It should be considered in conjunction with transdermal progesterone therapy which studies have shown to not only increase bone mineral density, but also reduce fractures and bone pain.[61]

Silicon is necessary for the formation of collagen and connective tissue; for healthy nails, skin and hair and aids in the absorption of calcium. It maintains flexible arteries and therefore plays a role in preventing cardiovascular disease (Balch, 28). It is interesting that silicon appears to reduce the bioavailability of aluminium in drinking water and thus could be protective against the development of Alzheimer's disease.[62] Silicon in beer binds aluminium and destroys its toxicity. (Florence et al, 174) Silicon is said to be an essential component in proper growth of chicks and other animals.[63] Salads are said to contain 8% silica. See Table 2 for sources.

Bone pain is an unavoidable by-product of diseases of the bone. **Phenylalanine** (PHE) is an essential amino acid which is readily converted to tyrosine and two key neurotransmitters, dopamine and norepinephrine. Because of its close relationship with the central nervous system, it can elevate moods in depression and decrease pain. PHE is available in three forms, L-, D-, and DL-. The L- form is incorporated by the body's proteins. The D- & DL-form has been used in controlling pain, especially in arthritis and osteoporosis (DL-PHE 750 mg , 15-30 minutes before meals three times a day).[64], [65]. However, results have not always been consistent and supplementation can cause anxiety, headache and hypertension among some individuals. Phenylketonurics, hypertensive patients and pregnant and lactating women should avoid PHE.

Sometimes a **copper** deficiency may lower pain threshold levels. When men with a copper deficiency were replenished with Cu supplementation, enkephalin levels in the pituitary and central nervous system rose and reduced the sensation of pain. [66]

L-tryptophan 2-4 grams daily taken with sugar to facilitate absorption 90 minutes before or after meals has been found to reduce pain in some instances (Werbach 1987, 342-345).

There is a Norwegian report based on an investigation of 10,576 people, 4,490 of whom reported joint or muscle pain that there was a positive relationship between the intake of **cod liver oil** (a teaspoon of cod liver oil a day) and lower musculoskeletal pain intensity in rheumatoid arthritis and muscle pain in post-viral fatigue patients.[67]

As mentioned before 85 per cent of subject experienced relief from bone pain by **strontium** supplementation.[68] **Kelp** contains 0.10 % of strontium.

Another product that may help relieve pain is **glucosamine sulphate**. Glucosamine is an amino sugar made in the body from the simple carbohydrate glucose and the amino acid glutamine. It is not used as a source of energy, but rather as a substance to form nails, tendons, skin, eyes, bones and ligaments; in other words - it involves collagen. It is used in the treatment of osteoarthritis, bursitis, rheumatoid arthritis, food allergies and osteoporosis [69]. It has been shown to be as effective a treatment for pain as NSAIDs such as ibuprofen [70], [71]. It seems to be easily absorbed from the gastrointestinal gut and diffused to the various organs, including articular cartilage.[72]

And finally one should not overlook the reduction of pain experienced by postmenopausal women on supplementation of transdermal **progesterone** (Lee).

BOOK REFERENCES

Adams R & Murray F(1977), **MINERALS: KILL OR CURE?,** Larchmont Books,NY

Balch JF & Balch PA (1997), **PRESCRIPTION FOR NUTRITIONAL HEALING:** A practical A to Z reference to drug-free remedies using vitamins, minerals, herbs & food supplements, Avery Publishing Group, NY

Chaitow,L(1985), **AMINO ACIDS IN THERAPY,** Thorsons Pub. Inc N.Y.

Davis, Adelle(1979), **LET'S GET WELL,** Unwin Paperbacks Sydney

Florence TM & Setright RT (1994), THE **HANDBOOK OF PREVENTIVE MEDICINE,** Kingsclear Books

Fox, S.I.(1993), **HUMAN PHYSIOLOGY,** Wm.C.Brown, Dubuque IA

Gaby, AR (1994), **PREVENTING AND REVERSING OSTEOPOROSIS,** Prima Pub, Rocklin, CA

Lehninger AL(1982), **PRINCIPLES OF BIOCHEMISTRY,** Worth Publishers Inc.N.Y.

Murray, MT & Pizzorno KJ.(1990), **ENCYCLOPAEDIA OF NATURAL MEDICINE,** Optima Macdonald & Co (pub) Ltd, 461

Stryer,Lubert (1988), **BIOCHEMISTRY,** WH Freeman and Co, NY

Trattler, Ross (1985), **BETTER HEALTH THROUGH NATURAL HEALING,** Thorson Harper Collins, London

Werbach,M.R.(1987), **NUTRITIONAL INFLUENCES ON ILLNESS,** Third Line Pres,Inc.,Tarzana,Cal.

Werbach,M.R.(1995), **HEALING THROUGH NUTRITION:** A Natural approach to treating illness with diets and nutrients, Harper Collins, London

REFERENCES

1) Cushing's Syndrome, also called Addison's Disease is a metabolic disorder resulting from the chronic and excessive productiuon of cortisol by the adrenal cortex or by administration of glucocorticoids. May be due to the body's inability to regulate the secretion of adrenocorticotropic hormone (ACTH), often resulting from a pituitary tumour.
2) Davidson S, Macleod J, (1972), **Davidsons' Principles & Practice of Medicine**, Churchill Livington, NY, 62
3) Brumsen C, Hamdy, NAT, PapapoulosSE (1997), Long-Term Effects of Bisphosphonates on the Growing Skeleton: Studies of Young Patients with Severe Osteoporosis, **Reviews in Molecular Medicine 76(4)**.July 1997.pp 266-283.
4) Ablin DS(1998), Osteogenesis imperfecta: a review. [38 refs], **Canadian Association of Radiologists Journal. 49(2):**110-23, 1998 Apr. [38 refs]
5) Cole, W G, Dalgleish R, (1995), Perinatal

lethal osteogenesis imperfecta, **Journal of Medical Genetics, 32(4):** 284-289
6) Moriwake T. Seino Y (1997), Recent progress in diagnosis and treatment of osteogenesis imperfecta, **Acta Paediatrica Japonica. 39(4):**521-7, 1997 Aug. (46 Refs)
7) The production of 1,25-dihyroxyvitamin D3 from cholesterol begins in the skin under the influence of sunlight. Vitamin D3 is carried in the blood to the liver where it is converted to 25-hydroxyvitamin D3. From there it is transported to the kidneys where it is converted to 1,25-dihydroxyvitamin D3 by several enzymes in the liver and kidneys respectively. The significance of this vitamin is that it stimulates absorption of calcium and phosphates in the intestines and the reabsorption of calcium and phosphates in the kidneys. Thus vitamin D both stimulates bone resorption and bone deposition. This contradiction can be explained by the fact that the primary function of 1,25-dihydroxyvitamin D3 is the intestinal absorption of calcium and phosphates. When blood concentrations of these minerals are adequate bone deposition is promoted. Fox, S.I.(1993), **HUMAN PHYSIOLOGY**, Wm.C.Brown, Dubuque IA, p 543
8) Hydroxyappatites are composed of calcium phosphate, calcium carbonate, with small amounts of magnesium, fluoride, sulphate and other trace elements.
9) The strength of collagen is remarkable: a load of at least 10kg is needed to break a fibre 1 mm in diameter (Stryer, 264)
10) The formation of collagen is complex and undergoes about 7 steps from fibroblasts to mature collagen fibres. Procollagen is hydrolysed to Tropocollagen -> Collagen fibre -> (cross-links) -> Mature collagen fibre. Details are not important in this article except that *mutation of a single glycine in collagen can lead to osteogenesis imperfecta.*
11) **Collagen** is formed from *procollagen* by *procollagen peptidases.* Collagen fibre formation takes place in the extracellular fluid near the cell surface rather than inside the fibroblasts because the procollagen peptidases are outside the cell. (Stryer, 269)
12) The three strands of procollagen Type I are made up of two strands having the same composition [called alpha1(I)] and the third of another kind [called alpha2(I)]. This collagen Type is involved in bone formation.
13) Moriwake T. Seino Y (1997), Recent progress in diagnosis and treatment of osteogenesis imperfecta. [Review] **Acta Paediatrica Japonica. 39(4):**521-7, 1997 Aug, [46 refs].
14) Hoekman K, Papapoulos SE, Peters ACB, Bijvoet OLM. Characteristics of bisphosphonate treatment of a patient with juvenile osteoporosis. **J Clin Endocrinol Metab 61:** 952-56, 1985.
15) Hodsman A. Adachi J. Olszynski W (1996), Prevention and management of osteoporosis: consensus statements from the Scientific Advisory Board of the Osteoporosis Society of Canada. 6. Use of bisphosphonates in the treatment of osteoporosis, **CMAJ. 155(7):** 945-8, 1996 Oct 1.
16) Devogelaer J-P. Nagant de Deuxchaisnes C (1997), Use of pamidronate in chronic and acute bone loss conditions, **Medicina 57(SUPPL. 1)**, pp 101-108
17) Brumsen C, Hamdy, NAT, PapapoulosSE (1997), Long-Term Effects of Bisphosphonates on the Growing Skeleton: Studies of Young Patients with Severe Osteoporosis, **Reviews in Molecular Medicine 76(4)**.July 1997.pp 266-283.
18) This may explain why vitamin C deficiency may be responsible for **scurvy**. In 1753 James Lind, a Scottish physician, advised sailors to include lemon juice and "green and fresh vegetables with ripe fruits, are the best remedy for it"
19) There is an interaction between copper and molybdenum and a deficiency in either result in skeletal abnormalities. Loveridge N, Noble BS (1994), Control of longitudinal growth: the role of nutrition, **Eur J Clin Nutr 48:** 75-84
20) Souci SW, Fachman W, Kraut H (1994), **FOOD COMPOSITIOIN AND NUTRITIOIN TABLES,** Medpharm Scientific Pubs, Stuttgart, Germany, 303
21) Crawhall JC et al (1959), Conversion of glycine to oxalate in normal subjects, **Lancet 2:** 810
22) Johansson G et al (1982), Effects of magnesium hydroxide in renal stone disease, **J Am Coll Nutr 1(2):** 179-85 quoted from Werbach 1993, 178
23) Manganese (Mn) stimulates the production of a group of proteinlike molecules in bones called *polysaccharides.* These compounds provide structures upon which calcification take place. Mn deficiency causes impairment of bone formation, remodeling and repair. Leach RM et als (1969), **Arch Biochem Biophys 133:** 22-8 quoted in Gaby AR, 31.
24) A range of manganese (Mn) deficiency symptoms have been observed in experimental animals including growth failure and skeletal abnormalities. An antihypertensive drug, hydralazine (Alphapress, Apresoline) binds with Mn and causes its deficiency, resulting in lupus erythematosis like symptoms in 10% of patients. Mn supplementation has been helpful in these patients. Editorial, Manganese deficiency in humans: fact or fiction? **Nutr Rev 46(10):** 348-353
25) Prentice A (1997), Is nutrition inportant in osteoporosis? **Proc Nutr Soc 56:** 357-367
26) DeFronzo RA et al (1975), The effect of insulin on renal handling of sodium, potassium, calcium and phosphate in man, **J Clin Inves 55:** 845-55 & Rao PN et al (1982) Are stone formers maladapted to refined carbohydrates? **Brit J Orol 54(6):** 575-77
27) Gaby AR (1994), page 105 usually recommends 600 to 1200 mg/day in combination with other nutrients such as vitamin K, folic acid, vitamin B6, magnesium, manganese, zinc, copper, boron and silicon. He believes that doctors often do not take into account calcium derived from the diet (about 500 mg/day).
28) The mean calcium requirement of adult humans is the mean value at which calcium input and output are equal and is found to be about 550mg/day, based on hundreds of balance studies. Nordic BEC, Need AG, Horowitz M et al (1991), Osteoporosis and calcium, **Aust NZ J Med 21:** 275-278
29) Heaney RP et (1989), **Am J Clin Nutr 49:** 372-76
30) Whiting SJ, Wood RJ (1997), Adverse effects of high-calcium diets in humans, **Nutr Rev 55(1):** 1-9
31) Nicar MJ, Pak CY (1985), **J Clin Endocrinol Metab 61(2):** 391-93 & Schutte SA, Knowles JB, **Am J Clin Nutr 47(5):** 844-88
32) In achlorhydria calcium citrate is ten times as well absorbed as is calcium carbonate, Recker RR (1985), **N Engl J Med 313(2):** 70-73
33) Grossman M et al (1963), **Gastroenterol 45:** 15-26
34) Recker RR (1985), **New Engl J Med 313(2):** 70-73
35) KnoxTA et al (1989), **J Am Coll Nutr 8(5):** 450
36) Heaney RP et al (1988), **Am J Clin Nutr 47:** 707-709
37) Allen LH (1986), **Nutr. Today 21(3):** 6-10
38) Wood RJ et al (1987) **Am J Clin Nutr 46(4):** 699-701
39) Thom JA et al (1978), The influence of refined carbohydrate on urinary calcium excretion, **Br J Urology 50:** 459-64 & Holl M, Allen J (1987), **J Nutr 117:** 1229-33
40) Prentice, A (1997), Is nutrition important in osteoporosis? **Proc Nutr Soc 56:** 357-367
41) Trichopoulou A, Georgiou E, Bassiakos Y et al (1997), Energy intake and monounsaturated fat in relation to bone mineral density among women and men in Greece, **Prev Med 26:** 395-400
42) Weisser NM, in NW Solomons, IH Rosenberg, Eds (1984), **Absorption and malabsorption of mineral nutrients,** Alan R Liss, NY, 15
43) Sellman, Sherril (1998), Osteoporosis: The bones of contention, **Nexus, October - November 1998):** 21-26
44) Cottrell M, Mead N (1994), Osteoporosis and the calcium craze, **Australian Wellbeing 57:** 70-75
45) Feskanich D, Willett WC, Stamfer M, Colditz G (1997), Milk, dietary calcium and bone fractures in women: a 12 year prospective study, **Am J Public Health 87:** 992-997 quioted from Sherril Sellman, 25
46) Murphy S, Khaw K, May H, Compston JE (1994), Milk consumption and bone mineral density in middle aged and elderly women, **BMJ 308:** 939-941
47) Lau EMC, Kwok T, Woo J, Ho SC (1998), Bone mineral density in Chinese elderly female vegetarian, vegans, lacto-vegetarians and omnivores, **Eur J Clin Nutr 52:** 60-64
48) Spencer H et al (1982), Effect of small doses of aluminium-contaning anatcids on calcium and phosphorus metabolism, **AM J Clin Nutr 36:** 32-40
49) Bui MH, Sauty A, Collet F, Leuenberg P (1992), Dietary vitamin C intake and concentrations in the body fluids and cells of male smokers and nonsmokers, **J Nutr 122:** 312-316
50) Mahaffee D et al (1982), **Endocrinol 110:** 4487-95
51) Sojka JE, Weaver CM (1995), Magnesium supplementation and osteoporosis, **Nutr Rev 53(3):** 71-80
52) **Parathyroid hormone(PTH)** - A hormone secreted from the parathyroid glands that maintains a constant concentration of calcium in the extracellular fluid. PTH regulates absorption of calcium from the GI tract and mobilizes calcium from bones.
53) Dambacher MA et al (1986), Long-term fluoride therapy of postmenopausal osteoporosis, **Bone 7(3):** 199-205
54) Diesendorf Mark (1990), 2 articles: "Have the benefits of water fluoridation been oversestimeted? & The health hazards of fluoridation: a re-examination, **International Clin Nutrition Review (10)2:** 292-332
55) Vashistha SN, Kapoor V, Yadav PS et al (1997), Effect of supplemental boron on nutrient utilisation, mineral status and blood biochemical constituents in lambs fed high fluoride diet, **Fluoride 30(3):** 165-72
56) Van Papendorp DH, Coetz H, Kruger MC (1995), Biochemical profile of osteoporotic patients on eseential fatty acid supplementation, **Nutr Res 15(3):** 325-334
57) Naghii MR, Samman S (1993), The role of boron in nutrition and metabolism, **Prog Food Nutr Sci 17:** 331-349
58) Naghii MR, Samman S (1997), The effect of boron on plasma testosterone and plasma lipids in rats, **Nutr Res 17(3):** 523-532
59) Beattie JH. Peace HS (1993), The influence of a low-boron diet and boron supplementation on bone, major mineral and sex steroid metabolism in postmenopausal women, **Br J Nutr 69(3):** 871-884
60) King N. Odom TW. Sampson HW. Yersin AG (1991), The effect of in ovo boron supplementation on bone mineralization of the vitamin D-deficient chicken embryo, **Biological Trace Element Research. 31(3):**223-33, 1991 Dec
61) Lee JR (1990), Osteoporosis reversal: The role of progesterone, **International Clinical Nutrition Review 10(3):** 384-391
62) Jacqmin-Gadda H, Commenges D, Letenneur L, Dartigues (1996), Silica and aluminium in drinking water and cognitive impairment in the elderly, **Epidemiol 7(3):** 218-285
63) Carlisle EM (1972), Silicon: An essential element for the chick, **Science 178:** 619-621 & Leninger (1982), 272
64) Balagot R et al (1983), **Adv Pain Res Ther 5:** 289-292
65) Budd K (1983), **Adv Pain Res Ther 5:** 305-308

References continued on page 18

Table 2 Foods that may be benficial in the treatment of osteoporosis.

When nutrients are combined the first figure represents the first nutrients and the second figure represents the second and so on. Foods with a relatively high content has been choisen.

Boron in Mcg, in 100 g of food

Helps metabolism of calcium, magnesium, phosphorus and prevents their excretion in urine, brain function, alertness, post-menopausal osteoporosis and arthritis, build muscles.

Mushrooms, canned 4150, Cucumber 3630, Mushroom, cultivated 1820, Peas, seed, dry 1800, Peanuts, roasted 1700, Wheat Germ 1650, Black tea 1590, Almonds, roasted (nut) 1400, Raisins, Dried Grape, Sultanas 1200, Peanuts 1200, Avocados 955, Rose hips 880, Cod, 824, Walnuts 760, Pecans nut 760, Figs, dried 710, Buckwheat 680, White cabbage 600, Appricots, canned 580, Cherries, canned 570, Sweet cherries, canned 570, Oats, without husk, whole grain 568, Parsley leaf 540, Millet 520, Apricots 475, Wheat whole grain 463, Barley, without husk 458, Parsley Root 450, Garlic 440, Garlic, cloves 440, Milk, dried skimmed 435, White Beans 430, Honey 350, Plums 340, Soya flour, full fat 300, Leeks 280, Rice, unpolished 275, Brussel sprouts 270, Red cabbage 250, White wine 250, Apple 245,

Calcium in Mgs in Food100g,

Milk, dried skimmed 1290, Parmesan Cheese 1180, Kelp 1098.59, Swiss Cheese 925, Gouda cheese 45% fat 820, Edam Cheese 30% fat 800, Cheddar Cheese 752, Mozzarella Cheese 632, Camembert cheese 600, Blue Cheese 526, Sardines, canned 443, Brie Cheese 400, Carob Flour 352, Sardines in oil 330, Black tea 302, Dulse 296, Rose hips 257, Almonds, roasted (nut) 252, Collard leaves 250, Turnip Greens 246, Barbados Molasses 245, Parsley leaf 245, Hazelnuts, Cobnut 226, Cress 214, Chocolate, Milk chocolate 214, Kale 212, Parsley 203, Soya beans, 201, Corn Tortillas (lime added) 200, French beans, dried 197, Soya flour, full fat 195, Figs, dried 193, Dandelion greens 187, Salmon, canned 185, Water cress 180, Coffee powder 168, Dandelion leaves 158, Watercress 151, Coffee, roasted 146, Egg Yolk 140, Pistachio Nuts 136, Brazil Nuts 132, Chives 129, Tofu 128, Goat's milk 127, Spinach, fresh 126, Chick-peas, dry 124, Mungo beans, Black gram 123, Milk, skimmed 123, Sunflower Seeds 120, Milk, Cow's milk 120, Yoghurt, Milk 120, Beet Greens 119, Yoghurt, reduced fat 114, White Beans 113, Sesame Seeds, hulled 110, Buttermilk 109, Ripe olives 106, Tofu 105, Broccoli 105, Watermelon 105, Horseradish 105, Walnuts, English 99, Olives, green marinated 96, Cowpeas, dry 95.75, Cottage Cheese, 95, Spinach 93, Lima Beans, dry 90.43, Walnuts 87, Leeks 87, Sardines 85, Spinach, canned 85, Anchovies 82, Oysters 82, Apricots, dried 82, Raisins, Dried Grape, Sultanas 80, Celery 80,

Calcium & Magnesium in Mgs per 100g of food: The first figure represents calcium, the second the magnesium component

Milk, dried skimmed, 1290, 110, Parmesan Cheese, 1180, 41.2, Kelp, 1050, 740, Gouda cheese 45% fat, 820, 28, Edam Cheese 30% fat, 800, 59, Cheddar Cheese, 752, 29.86, Mozzarella Cheese, 632, 24, Camembert cheese, 600, 19, Blue Cheese, 526, 39, Dulse, 296, 220, Rose hips, 257, 104, Almonds, roasted (nut), 252, 170, Collard leaves, 250, 57, Parsley leaf, 245, 41.1, Hazelnuts, Cobnut, 226, 156, Chocolate, Milk chocolate, 214, 86, Kale, 212, 31, Parsley , 203, 41, Soya beans, 201, 220, Soya flour, full fat, Vegetable, 195, 247, Figs,dried, 193, 70, Dandelion greens, 187, 36, Salmon, canned, 185, 29.55, Water cress, 180, 34, Dandelion leaves, 158, 36, Egg Yolk, 140, 16, Pistachio Nuts, 136, 158, Brazil Nuts, 132, 160

Copper in mgs per Food 100g,

Aids in formation of bone, collagen, skin, connective tissues, hemoglobin, red blood cells, works with vitamin C and zinc to form elastin. Copper deficiency may lead to osteoporosis, anaemia, baldness, diarrhoea, impaired respiratory function, skin sores. Toxicity: consider copper piping and cooking utensils, depression, irritability, nausea, vomiting, joint and muscle pain. High copper levels lead to low zinc levels and vice versa.

Liver 9.9, Pickeld cucumber 8.4, Sheep's liver 7.640, Calf's liver 5.5, Crabs 4.8, Cocoa (dry powder) 4.0, Cashews (nuts) 3.7, Brewer's Yeast (GTF) 3.320, Beef, liver 3.150, Liver, beef, meat 2.8, Oysters 2.5, Black tea 2.5, Soy Lecithin 2.1, Chocolate, milk free 1.9, Rose hips 1.8, Coffee, roasted 1.730, Wheat Bran 1.550, Pork liver 1.330, Brazil Nuts 1.3, Chocolate, Milk chocolate 1.3, Pecans nut 1.3, Hazelnuts, Cobnut 1.280, Soya beans, 1.2, Split Peas 1.2, Cowpeas, dry 1.070, Wheat Germ 0.950, Walnuts 0.880,

Fluoride in Mcg per Food100g,

Black tea 9500, Walnuts 680, Pork liver 290, Lobster, Crawfish, 210, Calf's kidney 200, Beef, liver 200, Kidneys, beef, meat 200, Eel, smoked 180, Cashews (nuts) 140, Peanuts, roasted 140, Beef, liver 130, Butter 130, Peanuts 130, Barley, without husk 120, Egg, Whole egg 110, Eggs 110, Parsley leaf 110, Soya flour, full fat 110, Spinach, fresh 110, Wheat whole grain 90, Almonds, roasted (nut) 90, Coffee, roasted 90, Pike, river 80, Herring, vinegar cured 70, Radishes 70, Maize, whole grain 62, Raisins, Dried Grape, Sultanas 62, Grapes, dried 62, Rose hips 60, Blue Cheese 50, Rice, unpolished 50, Pork chops 50, Rice, polished 50, Chocolate, Milk chocolate 50, Chocolate, milk free 50, Millet 50, Apricots, dried 50, Asparagus, boiled drained 48, Sauerkraut 45, Rhubarb 40, Peas, seed, dry 40, Oats, rolled 37, Haddock 35, Chicken for roasting 33, Lettuce 32, Mushroom, cultivated 31, Salmon, flesh 30, Trout, 30, Egg Yolk 30, Mackerel 30, Flounder, fish 30, Eel, 30, White wine 30, Blackcurrants 29, Camembert cheese 28, Tuna, flesh 28, Cod, 28, Green Peas (fresh) 27, Peas in pod 27, Cress 24, Tomato 24, Strawberries 24,

Glycine in Mgs per Food 100g,

Glycine is a major component of collagen (contains 35 per cent). Retards muscle degeneration by supplying additional creatine used in construction of DNA and RNA, bile acids, promotes healing of skin, necessary in healthy nervous system function, healthy prostate, may help in preventing epilepsy, helps in mania and manic depression and hyper activity. Excess may cause fatigue. Glycine converted to serine.

Gelatine 22960, Wheat Germ 2160, Horse mackerel 1940, Hake 1740, Soya flour, full fat 1680, Peanuts, roasted 1640, Salmon, flesh 1630, Beef, sirloin steak 1590, Chicken Breasts 1560, Chicken liver 1560, Beef rump 1500, Beef, liver 1490, Beef, liver 1490, Trout, 1470, Pork liver 1460, Mutton 1430, Soya beans, 1420, Pork muscles only 1420, Calf's liver 1420, Mackerel, 1410, Pork chops 1400, Chicken for roasting 1400, Kidneys,

beef, meat 1390, Mullet 1380, Wheat Bran 1320, Eel, 1290, Lima Beans, dry 1280, Pork, Hind leg 1230, Ham 1190, Tuna, flesh 1170, Halibut 1150, Sole, fish 1140, Herring 1130, Lobster, Crawfish, 1120, Cowpeas, dry 1080, Sheep's liver 1050, Lemon sole 1040, Catfish 1040, Walnuts, 1030, Perch, river 1010, Lobster, Crawfish, 970, Oatmeal, Oat meal 960, Mungo beans, Black gram 950, White Beans 950, Cod, 940, Flounder, fish 930, Oats, rolled 850, Caviar 830, Buckwheat flour 830, Oysters 800, Buckwheat 790, Oats, without husk, whole grain 780, Wheat whole grain 720, Parmesan Cheese 700, Pike, river 620, Egg yolk 620, Peas, seed, dry 590, Bread rolls, wheat 580, Edam Cheese 30% fat 560, Barley, without husk 540, Egg, Whole egg 530, Eggs 530, Blue Cheese 510, Gouda cheese 45% fat 500, Egg White 500, Egg Yolk, dried 500, Camembert cheese 480, Cheddar cheese 470

Iron, Copper, Manganese in mgs per 100g of foods,

Kelp, 370, 0.8, 1.5, Garlic, cloves, 104, 0.149, 0.46, Brewer's Yeast (GTF), 17.6, 3.32, 0.53, Black tea, 17.2, 2.5, 73.4, Pork liver, 15.79, 1.33, 0.36, Sheep's liver, 12.4, 7.64, 0.33, Calf's kidney, 11.5, 0.37, 0.05, Kidneys, beef, meat, 9.5, 0.434, 0.1, Millet, 9, 0.85, 1.9, Wheat Germ 7.95, 0.95, 11.42, Calf's liver, 7.9, 5.5, 0.28, Liver,calf , 7.9, 5.5, 0.28, Sheep's kidney, 7.5, 0.352, 0.11, Chicken liver, 7.4, 0.406, 0.291, Egg Yolk, 7.2, 0.35, 0.125, Beef, liver, 7.01, 3.15, 0.25, Chick-peas, dry, 6.96, 0.81, 2.14, Millet, 6.8, 0.2, 0.2, Cowpeas, dry, 6.69, 1.07, 3.74, Soya beans, 6.64, 1.2, 2.71, Lima Beans, dry, 6.27, 0.804, 1.79, White Beans, Vegetable, 6.17, 0.635, 1.62, Oats,without husk, whole grain, 5.8, 0.47, 3.7, Parsley leaf, 5.5, 0.142, 2.7, Tofu, 5.36, 0.2, 0.6, Peas, seed, dry, 5.02, 0.741, 1.25, Oats,rolled, 4.61, 0.53, 4.54, Apricots, dried, 4.4, 0.8, 1.5, Almonds, roasted (nut), 4.13, 0.85, 1.9, Spinach, fresh, Vegetable, 4.1, 0.09657, 0.474, Hazelnuts, Cobnut, 3.8, 1.28, 5.7, Calf's heart, 3.7, 0.32, 0.03, Wheat Bran, 3.58, 1.55, 3.7, Brazil Nuts, 3.4, 1.3, 0.6, Wheat whole grain, 3.31, 0.459, 3.67, Figs,dried, 3.3, 0.38, 0.35, Buckwheat, 3.2, 0.8, 1.3, Dandelion leaves, 3.1, 0.17, 0.34, Barley,without husk, 2.8, 0.373, 1.65, Cashews (nuts), 2.8, 3.7, 0.84, Rice,unpolished, 2.6, 0.24, 1.1, Blueberries, canned, 2.6, 0.39, 1.9, Walnuts, 2.5, 0.88, 1.97, Pecans nut, 2.4, 1.3, 3.5, Peanuts, roasted, 2.32, 0.61, 1.24, Raisins, Dried Grape, Sultanas, 2.27, 0.37, 0.464, Beef rump, meat, 2.21, 0.07316, 0.018, Eggs, 2.1, 0.2, 0.03, Carrots, 2.1, 0.05161, 0.21, Veal, muscle only, 2.1, 0.16, 0.03, Corn flakes, 2, 0.2, 0.05, Kale, 1.9, 0.0556, 0.55, Dates, dried, 1.9, 0.33, 0.15, Dates,dried, 1.9, 0.33, 0.15, Lamb, 1.9, 0.7, 0.03, Peas in pod, 1.84, 0.326, 0.66, Green Peas (fresh), 1.84, 0.326, 0.66, Peanuts, 1.82, 0.764, 1.6, Olives, green marinated, 1.8, 0.27, 0.058,

Lysine is an essential amino acid, building block of all proteins, needed for proper growth and bone development in children, helps calcium absorption, maintains nitrogen balance, production of antibodies, hormones, enzymes, helps collagen formation, tissue repair, muscle growth, recovering after surgery, lowers triglycerides. High levels together with low levels of arginine (see) help fight herpes virus together with vitamin C. Deficiency: anaemia, blood shot eyes, irritability, lack of energy, poor appetite, weight loss, reproductive disorders

Lysine in Mgs per 100g of food:

Gelatine 3800, Brewer's Yeast (GTF) 3540, Pork, fried liver, 3046, Parmesan Cheese 2980, Gouda cheese 45% fat 2790, Milk, dried skimmed 2720, Flounder (baked), 2623, Parmesan cheese, 2607, Soya flour, full fat, 2560, Tuna, canned in oil drained, 2540, Soya flour, 2531, Edam Cheese 30% fat 2390, Blue Cheese 2380, Sardines 2280, Chicken Breasts 2270, Sardines in oil 2240, Tuna, flesh 2210, Pork muscles only 2200, Pistachios, 2190, Perch, river 2190, poultry, turkey, 2173, Beef, sirloin steak 2170, Pork chops 2160, Peas, seed, dry 2130, Edam cheese, 2111, Mungo beans, Black gram 2080, Caviar 2070, Cheddar cheese 2070, Beef rump 2050, Cod, 2050, Mullet 2040, Chicken for roasting 2040, Turkey, young with skin, meat 2030, Salmon, flesh 2020, Trout, 2020, Mutton 2000, Lobster, Crawfish, 1990, Tuna in oil 1980, Chicken liver 1960, Brie Cheese 1960, Haddock, raw 1930, Wheat Germ 1900, Soya beans, 1900, Pork, Hind leg 1900, Camembert cheese 1900, Sirloin steak, 1899, Trout (raw), 1892, White Beans, 1870, Ham 1870, Sole, fish 1860, Chicken breasts, 1852, Beef (roast), 1847, Lemon sole 1840, Pork liver 1830, Flounder, fish 1820, Rabbit meat 1810, Horse mackerel 1800, Sheep's liver 1800, pickled herring, 1775, Salmon, canned pink, 1771, Herring 1750, Beef, liver 1750, Liver,calf 1740, Turkey, adult animal with skin 1740, Mackerel, 1730, Pork (roasted) 1723, Cheddar cheese, 1702, Salami, Sausage 1690, Lobster, Crawfish, 1690, Fish, cod (canned), 1690, Halibut, raw, 1631, Chicken (breasts), 1630, Round medium fat veal, 1629, Veal roast, Roast veal 1622, Prawns (cooked), Shrimps 1595, Cowpeas, dry 1570, Halibut 1560, Hake 1560, Catfish 1560, Herring, vinegar cured 1510, Liver (cooked), 1495, Lima beans, 1488, Chicken liver, 1480, Lima Beans, dry 1470, Lamb (leg), 1457, Fresh raw cod, 1447, Cottage cheese, 1428, Chick-peas dry-raw, 1415, Kidneys, beef, meat 1410, Chick-peas, dry 1370, Eel, 1360, Pork, (loin), 1346, Eel, smoked 1340, Pumpkin seeds, 1334, Liverwurst, 1301, Egg Yolk 1300, Sheep's kidney 1300, Pork, ham, 1248, Liverwurst, Liver sausage 1240, Sausage, liver 1240, Calf's kidney 1210, Lamb (rib), 1206, Salmon, canned 1200, Peanuts 1100, Peanuts (roasted), 1080, Peanut butter, 1066,

Lysine & Glycine in Mgs per 100g Foods

Gelatine, 3800, 22960, Parmesan Cheese, 2980, 700, Gouda cheese 45% fat, 2790, 500, Soya flour, full fat, 2560, 1680, Edam Cheese 30% fat, 2390, 560, Blue Cheese, 2380, 510, Chicken Breasts, 2270, 1560, Tuna, flesh, 2210, 1170, Pork muscles only, 2200, 1420, Perch, river, 2190, 1010, Beef, sirloin steak, 2170, 1590, Pork chops, 2160, 1400, Peas, seed, dry, 2130, 590, Mungo beans, Black gram, 2080, 950, Caviar, 2070, 830, Cheddar cheese, 2070, 470, Beef rump, 2050, 1500, Cod, 2050, 940, Mullet, 2040, 1380, Chicken for roasting, 2040, 1400, Salmon, flesh, 2020, 1630, Trout, 2020, 1470, Mutton, 2000, 1430, Lobster, Crawfish, 1990, 1120, Chicken liver, 1960, 1560, Wheat Germ1900, 2160, Soya beans, 1900, 1420, Pork, Hind leg, 1900, 1230, Camembert cheese, 1900, 480, White Beans, 1870, 950, Ham, 1870, 1190, Sole, fish, 1860, 1140, Lemon sole, 1840, 1040, Pork liver, 1830, 1460, Flounder, fish, 1820, 930, Horse mackerel, 1800, 1940, Sheep's liver, 1800, 1050, Herring, 1750, 1130, Beef, liver, 1750, 1490, Liver,calf , 1740, 1420, Mackerel, 1730, 1410, Lobster, Crawfish, 1690, 970, Cowpeas, dry, 1570, 1080, Halibut, 1560, 1150, Hake, 1560, 1740, Catfish, 1560, 1040, Lima Beans, dry, 1470, 1280, Kidneys, beef, meat, 1410, 1390, Eel, 1360, 1290, Egg Yolk, 1300, 620, Peanuts, 1100, 1640, Egg, Whole egg, 890, 530,

Manganese In Mgs in Food100g,

Black tea 73.4, Wheat Germ11.42, Hazelnuts, Cobnut 5.7, Oats, rolled 4.54, Soya flour, full fat 4, Cowpeas, dry 3.74, Oats, without husk, whole grain 3.7, Wheat whole grain 3.67, Pecans nut 3.5, Soya beans, 2.71, Parsley leaf 2.7, Chick-peas, dry 2.14, Rice, polished 2, Walnuts 1.97, Blueberries, canned 1.9, Almonds, roasted (nut) 1.9, Millet 1.9, Lima Beans, dry 1.79, Barley, without husk 1.65, White Beans 1.62, Peanuts 1.6, Apricots, dried 1.5, Coconut 1.31, Rye 1.3, Split Peas 1.3, Buckwheat 1.3, Peas, seed, dry 1.25, Peanuts, roasted 1.24, Rose hips 1.2, Rice, unpolished 1.1, Whole Wheat 1.1, Blackberries 0.894, Blackberries, commercial 0.894, Bread rolls, wheat 0.89, Cashews (nuts) 0.84, Blueberries, Bilberries, Huckleberries 0.84, Tea, clear, drink 0.69, Green Peas (fresh) 0.66, Peas in pod 0.66, Cassava tuber 0.62, Brazil Nuts 0.6, Oysters 0.6, Kale 0.55, Brewer's Yeast (GTF) 0.53, Turnip Greens 0.5, Maize, whole grain 0.48, Spinach, fresh 0.474

Silicon in Mgs per Food 100g,

Oats, without husk, whole grain 425, Egg, Whole egg 300, Barley, without husk 188, Parsley leaf 12, Turnip 12, French

Index of articles in the Hypoglycemic Health Newsletters from 1 December 1997 to September 1998. For all articles prior to 1 December 1997 see Hypoglycemic Health Newsletter December 1997 page 13

Table 2 continued

beans, String Beans 10, Hazelnuts, Cobnut 10, Bananas 8, Wheat whole grain 8, Leeks 6, Blueberries, Bilberries, Huckleberries 5, Peas, seed, dry 3, Cucumber 3, Blackcurrants 3, Mandarins 3, Tomatoes, ripe 2.7, Tomato 2.7, Lettuce 2, Strawberries 2, Currants, red 2, Mutton 1, Red wine 1, Chocolate, Milk chocolate 1, Rose hips 1, Orange Juice, fresh original 1, Apple juice 1, Salmon, flesh 0.5, Parsnips 0.5, Apple 0.5, Apples 0.5, Horseradish 0.5, Rhubarb 0.5, Cauliflower 0.5, Green Peas (fresh) 0.4, Plums 0.4, Peaches 0.4, Beetroot 0.4, Eggs 0.3, Gooseberries 0.3, Grapes, dried 0.3, Grapes, natural 0.3, Pears 0.2, White cabbage 0.15, Peas in pod 0.

Others: Rice bran, rice polishings. **Herbs:** Horsetail *Equisetum arvense* contains 10% of silica.Other silica containing herbs: Eyebright, Inulin in Dandelion Tea increases calcium absorption , English plantain, Bell peppers, Lettuce contains 8%, alfalfa, beets, soy beans.

Vitamin K in mcg per 100g of food,

Kale 817, Chives 380, Spinach, canned 290, Grapeseed oil 280, Brussel Sprouts, Brussels sprouts 275, Chick-peas, dry 264, Black tea 262, Water cress 250, Spinach, fresh, Vegetable 240, Spinach, New Zealand, Vegetable 240, Spinach, Vegetable 240, Lettuce 200, Soya flour, full fat, Vegetable 200, Soya beans, 190, Cauliflower 150, Egg Yolk 147, Wheat Germ131, Mungo beans, Black gram 130, Beef, liver 100, Beef, liver 100, Broccoli 100, Cabbage 100, Liver,beef, meat 100, Rose hips 92, Calf's liver 88.5, Peas, seed, dry 81, Wheat Bran 81, Chicken liver 80, White cabbage 79.5, Oats,rolled 63, Sauerkraut 61.67, Butter 60, Corn Oil, Maize oil 60, Maize oil, 60, Pistachio Nuts 59.5, Pork liver 56, Oats,without husk, whole grain 50, Olive Oil 49.6, Egg, Whole egg 47.5,

Zinc in Mgs per Food 100g

Helps prostate functioning, immune system, protein synthesis, collagen formation, wound healing, sense of taste, bone formation, constituent of insulin, many enzymes, superoxide dismutase (SOD), antioxidant, maintains vitamin E levels, increases absorption of vitamin A, skin healing, do not take more than 100 mgs daily.

Oysters 45, Wheat Bran 16, Wheat Germ 12, Calf's liver 8.4, Yeast, dried Baker's 8, Brewer's Yeast (GTF) 8, Cocoa (dry powder) 7, Ginger Root 6.8, Pork liver 6.350, King Crab 6, Lamb 5.3, Crabs 5, Meats 5, Soya flour, full fat 4.9, Beef, liver 4.830, Oats, without husk, whole grain 4.5, Pecans nut 4.5, Split Peas 4.2, Soya beans, 4.180, Milk, dried skimmed 4.1, Blue Cheese 4.1, Beef, sirloin, meat 4.070, Oats, rolled 4.060, Brazil Nuts 4, Edam Cheese 30% fat 4, Gouda cheese 45% fat 3.9, Cheddar Cheese 3.9, Liver, beef, meat 3.9, Beef rump, meat 3.860, Egg Yolk 3.8, Chick-peas, dry 3.540, Peas, seed, dry 3.450, Camembert cheese 3.4, Peanuts, roasted 3.380, Chicken liver 3.210, Rye 3.2, Whole Wheat 3.2, Black tea 3.190, Lima Beans, fresh 3.1, Soy Lecithin 3.1, Parmesan Cheese 3, Peanut Butter 3, Peanuts. salted 3, Walnuts, English 3, Lima Beans, dry 2.970, Sardines 2.9, Peanuts 2.830, Walnuts 2.7, Wheat whole grain 2.690, White Beans 2.640, Barley, without husk 2.530, Cowpeas, dry 2.5, Buckwheat 2.5, Maize, whole grain 2.5, Almonds, roasted (nut) 2.170, Turkey, young with skin, meat 2.1, Cashews (nuts) 2.090, Beef, liver 2.080, Kidneys, beef, meat 2.080, Peanuts with skin 2, Chocolate, milk free 2, Chocolate, Milk chocolate 2, Pork, lean, meat 2, Turkey, adult animal with skin 2, Clams 1.9, Hazelnuts, Cobnut 1.870, Millet 1.8, Kidneys, Calf's 1.8, Eel, 1.750, Tuna, canned 1.7, Lobster, Crawfish, 1.6, Rice, unpolished 1.520, Shrimps, Prawns 1.5, Anchovies 1.4, Horseradish 1.4, Egg, Whole egg 1.350, Eggs 1.350, Dandelion leaves 1.2, Turnips 1.2, Bread rolls, wheat 1.1

L-Arginine beneficial for hypoglycemia and cancer

Having regard to hypoglycemia being a pre-diabetic condition with extreme fluctuation between hyper- and hypoglycemia the study by Giugliano and co-workers shows that hyperglycemia is thought to increase vascular tone. These findings naturally applies to diabetes. It appears to be related to reduced availability of nitric oxide (nitrogen monoxide), an important relaxant factor. Hyperglycemia was induced in 12 healthy subjects with an artificial pancreas and within 30 minutes heart rate, platelet aggregation, blood viscosity and blood pressure went up, while leg blood flow decreased significantly. An infusion of L-arginine (1g/min) in the last 30 minutes completely reversed the blood changes (haemodynamic) brought about by hyperglycemia. The authors conclude that hyperglycemia may reduce nitric oxide availability in humans.

Giugliano D, Marfella R, Coppala L et al (1997), Vascular effects of acute hyperglycemia in humans are reversed by L-arginine. Evidence for reduced availability of nitric oxide during hyperglycaemia, **Circulation 95(7):** 1783-90

L-arginine may also benefit cancer patients

L-arginine is known to enhance natural cytotoxicity in peripheral blood lymphocytes in patients with colorectal cancer. In a hospital trial 18 patients were treated for 3 days prior to surgery. The control group received a standard hospital diet, whereas the experimental group received and additional supplementation of 30 g per day of L-arginine. Tumours from the experimental group contained increased numbers of specific cell subsets within the tumour which expressed CD16 and CD56 surface markers. There was no difference in the total number of T and B cells, or T helper and T supersessor cells.

Heys SD, Segar A, Payne S et al (1997), Dietary supplementation with L-arginine: modulation of tumour-infiltrating lymphocytes in patients with colorectal cancer, **Br J Surg 84(2):** 238-41

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.

This makes the series of International Clinical Nutrition Review a valuable commodity in one's private library for anyone who is interested in the scientific basis of clinical nutrition.

Researchers from all corners of the world review medical and other scientific literature and cull out the latest news on the clinical application of nutrients in the treatment of disease. The serial also publishes in-depth-editorials written by experts on various topics of concern to clinical nutritionists.

No longer can it be argued that this new branch of medicine is without scientific foundation.

A "must" for naturopaths and doctors with an interest in nutrition, practitioners and interested members of the public can subscribe to this quarterly publication by sending $54.00 (New Zealand Subscribers $64) to:

Integrated Therapies
PO Box 370
MANLY NSW 2095
Australia

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

Please write to the above address for brochure.

References continued from page 14

66) Blathena S et al (1986), Decreased plasma enkephalins in copper deficiency in man, **Am J Clin Nutr 43:** 42-46
67) Eriksen W, Sandvik L, Bruusgaard D (1996), Does dietary supplementation of cod liver oil mitigate musculoskeletal pain? **Eur J Clin Nutr 50:** 689-693
68) McCaslin FE, Janes JM (1959), The effect of strontium lacate in the treatment of osteoporosis, **Proc Staff Meetings Mayo Clin 34:**329, from Murray & Pizzorno (1990), 461
69) Drovanti A, Bignamini AA, Rovati AL (1980), Therapeutic actvity of oral glucosamine sulfate in osteoarthrosis: a placebo-controlled double-blind investigation, **Clinical Therapeutics/Vol. 3.** No. 4,
70) Muller-Fasbender H, Bach GL, Haase W, Rovati LC, Setnikar I (1994), Glucosamine sulfate compared to iuprofen in osteoarthritis, **Osteoarthritis and Cartilage (1994) 2:** 61-69
71) Vaz, AL (1982), Double-blind clinical evaluation of the relative efficacy of ibuprofen and glucosamine sulphate in the management of osteoarthrosis of the knee in outpatients, **Current Med. Res. and Opinion, 8:** 145
72) Setnikar I, Palumbo R, Canali S, Zanalo G (1993), Pharmacokinetics of glucosamine in man, **Arzneim. Forsch./Drug Res. 43 (II):** Nr. 10

"Vegetarianism is harmless enough, though it is apt to fill a man with wind and righteousnous"
Sir Robert Hutchinson

THE HYPOGLYCEMIC HEALTH ASSOCIATION
P.O.Box 8, SYLVANIA SOUTHGATE NSW 222

MEMBERSHIP APPLICATION

PLEASE PRINT

Surname: _____

First Name: _____

Address: _____

Town/City: _____ **Postcode:** _____

Phone: _____ **Age:** _____

Membership $20.00 pa
Pensioners $15 pa
Life Membership $150

Please tick √

RENEWAL ☐ **Occupation** _____

NEW MEMBER ☐ _____

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

1999 *MEETING DATES ON SATURDAYS*

7th MARCH - 6th JUNE - 5th SEPTEMBER - 5th DECEMBER