The Hypoglycemic Health Association

NEWSLETTER

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

This year we are very lucky that the Association have been blessed with donations from enthusiastic supporters. This has made our accounts look pretty good. However, it would be a great mistake to rest on our laurels. In fact, membership has declined to a point that without the generous donors we would be strapped for cash. We can not rely on donations alone and we need the support of paying members to pursue our aims to educate the public and professionals of hypoglycemia and related disorders. Presently, we are sending out Newsletters to a large number of unfinancial members who may have overlooked to forward their membership fees to the Association. Please note the Expiry date on the right-hand top corner of your address label. At this time of the year it should show 31 December 2001. If you have forgotten to send in your fees, please fill in the Application Form on the last page of this Newsletter and forward it to the Association. Fees have been kept low despite rising costs and they are: $20 p.a. and $15 for pensioners and students. Members are invited to encourage their friends to become memers of the Association.

Since the founding of this Association in 1985, we have made considerable progress in making the community aware of hypoglycemia and its allied disorders. In spite of accelerating social expenditure on medicine degenerative disease are on the rise; diabetes which is the follow-up disease of hypoglycemia has now been described in terms of an epidemic. There are approximately half a million Australians walking about without knowing that they are diabetic. This Association, dealing with the forerunner of diabetes, is in a unique position to turn this around. The telltale of this disease are in fact the hypoglycemic symptoms, and by treating these conditions we are as a matter of fact preventing the further development of not only diabetes, but other degenerative diseases. Therefore, your support will contribute to this turnaround.

Our Next Public Meeting will be at 2.00 PM on Saturday, the 2 June, 2001 at YWCA
5-11 Wentworth Ave, SYDNEY
and our guest speaker is
Dr Ramesh Manocha
who will be speaking
on the subject of

“Why Meditation?”

Dr Ramesh Manocha, from the natural therapy Unit of the Royal Hospital for Women at Randwick, obtained his medical degree in 1994. He has worked in several Sydney hospitals in various capacities. Following receipt of several grants he is conducting research into Stress Management for Asthma, Daily Migraine and relaxation, Meditation for Menopausal Hot Flushes, Brain imaging elite meditators, Emotional Intelligence, Meditation for Occupational Stress and the effects of Brahmi on Brain Function.

Dr Manocha is a well-known speaker on natural health covering areas of Stress Management for GPs and nurses, midwives and presenting a holistic approach for many degenerative disease, such osteoarthritis and cancer.

His main interest is the influence of meditation on health and we are pleased to have Dr Ramesh Manocha give a talk on this fascinating topic.
Any opinion expressed in this Newsletter does not necessarily reflect the views of the Association.

The Hypoglycemic Health Association.

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

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

Donations for raffle
One way of increasing our income is by way of raffles. If any member has anything to donate towards the raffle, please contact Dr George Samara’s surgery at 19 Princes Highway, Kogarah, Phone 9535-0848 or Sue Litchfield at 9971-5657 or (litch.gr@bigpond.com).

At the last meeting on the 3 March 2001, Jadzg Bzowska won the lucky door prize. This consisted of a frame with an beautifully embroidered picture made and donated by Marie Grady of Canberra. The Association thanks her very much for her loving donation.

Marie Grady is the mother of Reg Grady.

Ms Joy Sharp won the raffle, consisting of an Alarm Radio Clock.

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

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

The Hypoglycemia support group/meets every 3 months and the next meeting will be held on 12th August 2001 at 19 Princes Highway Kogarah (1st Floor Dr. Samara's surgery) at 1.45 p.m. The cost is $1. This is a mutual support and discussion group with guest speakers whenever possible. Afternoon tea provided - family and friends welcome. For further information please telephone - Lorraine on - 95209887 or Jeanette on - 95259178

My Journey Along The Hypoglycemic Trail
by Mrs Pamela Dean

During the last six to eight months I was undergoing what I thought was an extreme case of stress at my workplace – normally being a fairly organized person I found myself not being able to discipline myself to work through a project to completion and felt like I was jumping from pillar to post each time I began a new task. Combined with symptoms of not relaxing when eating a meal, becoming quite teary on frequent occasions (thank goodness for a wonderful supportive husband), the feelings of my body/heart racing, not sleeping well, poor memory retention and lack of concentration, I had no idea at all of what was actually happening to me both physically and mentally. Quite scary I can tell you! My husband was not having the same symptoms but he was quite tired most of the time as well.

Through a contact at my husband’s work, it was suggested that we seek the advice of a nutritionist and we were highly recommended Dr George Samara, who is an expert in the field of hypoglycemia. After our initial visit in early April, Dr Samara got us to have blood and allergy tests which resulted in my husband being diagnosed a “borderline hypoglycemic” and myself being diagnosed as a type 2 absolute hypoglycemia and the possibility of type 3 combined relative and absolute hypoglycemia. Just my luck to be the worst case scenario!! I cannot put a time frame on when I started with hypoglycemia but I feel that it has probably been around for many years and now at 44 years of age, feel very fortunate to have stumbled onto the fact that I now have a “label” and know the reasons for the many symptoms I have been encountering. If I had not done anything about the symptoms at this point in time, I would probably have "rolled

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Healthy Hearts and Hypoglycemics
by
Dr George Samra, MB, BS (Sydney), FACNEM

...t will be seen from the diagrams how in atherosclerosis the plaque is building up, blocking the artery and with time it can completely obstruct the artery.

The heart is a pump. In one person’s lifetime, say eighty years it will be:

80 years x 365 (days in the years)
x 24 (hours in the day)
x 60 (minutes in the hour)
x 72 beats (per minute)

And that will be approximately 3.0725 Billion Beats in your lifetime!

The exercise is trying to get that extra billion. The Queen Mother is almost there, but a billion beats takes about twenty seven years roughly so she really has to live to 107 to have the extra billion. Some people miss out they die at 50 but they’ve almost had two billion beats.

Several things can go wrong with the pump.

1. The blood supply to the pump. The coronary arteries atherosclerosis.

We have all seen pumps like pressure pumps and swimming pool pumps around. The blood supply to the pump is through the coronary arteries  atherosclerosis. We are all familiar with the symptoms of strokes from hardening of the arteries. In fact cardiomyopathy is the most common condition that brings people to needing heart transplants. The viral damage has weakened the heart.

2. Electrical pathways within the pump. Cardiac Arrhythmias (that is irregular heart beats).

3. Muscle strength and integrity of the pump. The topic of Cardiomyopathy. Small heart size is better for muscle strength. Viruses are the most common cause for Cardiomyopathy, but it’s not always the case. In fact cardiomyopathy is the most common condition that brings people to needing heart transplants. The viral damage has weakened the heart.

4. Valve integrity of the pump - subjects like Rheumatic Fever, Endocarditis, Sclerosis (which is hardening and particularly hardening of the valves), and Regurgitation (or leakage of the valves).

5. Damage to the pipes CVA, claudication, Vascular insufficiency also atherosclerosis. The arteries and the veins are the pipes. Damage to the arteries occurs with strokes and claudication which is muscle pain usually in the back of the calf muscle.

Atherosclerosis is a disease of the arterial wall. The diagram shows thickening of the wall. (FIGURE 1) The representation of fatty matter follows the insidious accumulation of cholesterol complex to proteins within the inner lining or the endothelial lining of the artery. (FIGURE 2)

Coronary artery disease together with other cardiovascular diseases such as strokes account for about 45% of all deaths in Australia. There are a lot of routine investigations done for heart problems. The most basic is an ECG where one has electrical wires on their chest. This produces some indirect information about the electricity on the surface of the skin and the chest wall, but most of the electricity in that area is really coming from the heart. There are twelve leads and twelve print outs and we get an idea of what is going on. If there are irregularities of the heart like rapid rates or slow rates or irregular beats (atrial fibrillation) or fast beats we can find these using ECGs.

We can get clues about whether a person is having a heart attack at the time if they’ve got heart pain. The ECG is limiting, often a person with a bad heart can have a normal resting ECG because they are resting, since the pump isn’t being challenged with work or exercise.

An exercise ECG is more interesting. It’s performed with electrical leads on you whilst on an exercise bike or a treadmill and your heart is pumping away. This test is more likely to show if the heart is under strain under a workload.

The doctor can tell the location of a heart attack, whether its in the front or the back of the heart or the side, and they can recognise the patterns.

The Holter ambulatory monitor is a twenty-four hour monitor. For somebody who says to you “I get this chest pain or racy heart or my heart stops for five seconds at a time, but it happens twice a day”, using a twenty four hour monitor we might pick it up and be able to explain just exactly what is going on in your body.

The echo cardiogram, is a wonderful non invasive test and you can see the heart chambers pumping away. There are other non invasive tests like arterial ultrasounds looking for flow in different arteries particularly going to the brain or other major organs.

The most useful invasive test is the angiogram, but we won’t discuss this right now. Hopefully you can avoid an angiogram with non invasive tests because there is a morbidity and a slight mortality associated with even just doing an angiogram.
RISK FACTORS
Risk factors in the development of atherosclerosis include heredity, smoking, obesity, Diabetes, Reactive Hypoglycemia, advanced age and high blood cholesterol concentrations. This is a degenerative process and with nutritional medicine there is now an argument that it need not occur if one takes the right supplements, but since it is a degenerative process, the older you are the more blocking to arteries you’re likely to have. The more cholesterol you have in your arteries the more chance you’re going to have of blockages.

Other risk factors associated with Atherosclerosis include deficiency in Vitamin C, magnesium, selenium, a high sugar intake and high iron levels.

THE ROLE OF CHOLESTEROL INATHEROSCLEROSIS
Studies have shown that high blood cholesterol levels are not necessarily associated with the development of atherosclerosis. Although the avoidance of saturated fats in the diet prevalent in foods of animal origin and tropical oils such as coconut and palm oil is universally recommended, 80% of plasma cholesterol comes from synthesis of the liver and only 20% comes from the diet. That would be true of modern people but certainly in the olden days high cholesterol levels were common to people who were eating bacon and eggs for breakfast everyday and frying their food in lard. Changing the diets of these people resulted in massive reductions in their cholesterol levels. It seems like we’re not as good at reducing cholesterol anymore because modern people, especially as they get older are already choosing their food fairly intelligently. I notice that when the patients I see have a choice between a low fat product and a product with a higher fat content they will instinctively choose the product that contains less fat. This sort of rationale makes it much harder, a change of diet can make minor changes to a persons cholesterol reading, and we don’t really get the sort of dramatic results like we used to.

Most people with very high cholesterol levels possess a liver which is usually the culprit because it is capable of manufacturing excessive cholesterol. If we were able to measure the cholesterol levels of people who died young historically, we would probably find that their liver was manufacturing too much cholesterol and it actually blocked their coronary artery prematurely. In the past we didn’t know about cholesterol so there was nobody who could warn them.

High blood cholesterol can also occur as a result of the inherited condition known as familial hypercholesterolemia. This condition is inherited as a single dominant gene; individuals who inherit both of these genes have a lot more trouble with high cholesterol concentrations regardless of diet. This group would certainly be vulnerable to atherosclerotic heart disease.

CHOLESTEROL TYPES AND NORMAL VALUES
On the handout we have given you, you can see the normal ranges for cholesterol.

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Cut-off Range</th>
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<tbody>
<tr>
<td>LDL</td>
<td>&lt; 4 mm/L</td>
</tr>
<tr>
<td>HDL</td>
<td>&gt; 1.2 mm/L</td>
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So there is a type of cholesterol that is protective. The HDL, we’re actually measuring in terms of the more the better but I don’t think I like it to be much above 2 mm/L but certainly it should be above 1.2 mm/L to give you some protection.

The LDL is the heavy or sticky type of cholesterol and it is responsible for this atherosclerotic in the illustrations. Just to give you a bit of history on cholesterol levels, 9.3 was once the cut off for high cholesterol, now it is 5.4. What has happened have people changed?

(Audience member volunteers) Knowledge has changed.

Yes, knowledge has changed. Previously it was based on the bell curve (a population distribution curve based on height or weight). So with height, everybody above 7 foot is too tall and everybody below 5 foot is too short. On the bell curve 9.2 was a reasonable cut off, but everybody who had a blood cholesterol of 8.5 was dropping dead with heart attacks, so doctors now base the cut off levels on heart risk, which is quite intelligent when you think about it. Now 5.4 is an upper limit. Interestingly we haven’t altered the lower limit, but I would generally say the lower the better. A wonderful cholesterol level is probably 2, but below that you might have trouble making your sex hormones because they have the cholesterol molecule as a base.

The mechanism for a heart attack is a crack...
in the plaque, then a leakage of plaque material which blocks the artery completely further on. That is why we try to dissolve it.

In order to prevent Atherosclerosis progression it is reasonable to take measures to lower serum lipids and also measures to reduce platelet adhesiveness and/or aggregation, as well as lowering blood pressure if elevated.

To lower serum lipid levels:
- A low fat, low cholesterol, high fiber diet, with a high ratio of polyunsaturates to saturated fat has been well validated. It may well be of value to avoid sugar and there are some studies that show that sugar is implicated in atherosclerosis production and there are statistical correlations now you can feed everything into computers that suggest coffee consumption is associated with higher cholesterol levels and hypercholesterolaemia. But that is not necessarily the cause and effect.
- Nutritional supplementation with Niacin e.g. Vitamin B3, and Omega 3 fatty acids may be useful to promote increases in the protective cholesterol, the HDL.

To reduce platelet adhesiveness and/or aggregation:
- Sugar has been shown to increase both adhesiveness and aggregation (clumping of platelets) and should be restricted. A number of nutrients have been shown to be of benefit, including pyridoxine (50 - 150 mg), vitamin E (400 - 1200 IU), magnesium, selenium, as well as bromelain, omega-3 and omega-6 fatty acids, pantethine, garlic, ginger and onions.

I'd like to refer to a few interesting abstracts relevant to this topic.

**FIRSTLY, IS MARGARINE GOOD FOR YOU AS OPPOSED TO BUTTER?**

To the many consumers who replace butter with margarines the following should be of interest. "The actual Receptor Site. The role of insulin is to raise the actual Receptor Site. The role of insulin is to raise the higher your cholesterol level and the higher the concentration of the bad type of cholesterol (LDL). Men who drank 5 or more cups of coffee had plasma cholesterol concentrations about 0.5, micro moles per Litre higher than non drinkers after matching this up with people of similar age, ethnicity, body mass, and so on. In women, adjusted mean cholesterol levels were 0.34 mmol/L higher in coffee drinkers. The incidence of heart attacks was also shown to correlate with coffee consumption but not with tea consumption.

The epidemiological link between coffee consumption, hyperlipidaemia and atherosclerosis may be at least partly due to an association between coffee consumption and saturated fats, rather than to the effects of coffee. So what they've actually done in this observational study (American Journal of Epidemiology, 1985) is asking people how much coffee they drink and measured their cholesterol levels, but also asked about how much red meat they consumed. It was found that heavy coffee drinkers ate approximately 24% more saturated fats than non coffee drinkers, so it might not be due to the fact that people are drinking more coffee. Instead this study might actually demonstrate that people who drink lots of coffee don't look after their diet and are more likely to eat a greasy meat pies and a lot of red meat.

Strangely enough, when thirty three people with high cholesterol were asked to abstain from coffee for 5 weeks, the total serum cholesterol levels fell 10% after ten weeks, and this is from a study published in British Medical Journal, 1985. So there must be some association with drinking coffee and high cholesterol levels.

**WHAT ABOUT COFFEE?**

An observation study (from the British Medical Journal in 1985) of 1007 men and 589 women, demonstrated that there was a significant linear association between coffee consumption in men and plasma cholesterol and LDL cholesterol. So the more coffee you drink the higher your cholesterol level and the higher the concentration of the bad type of cholesterol (LDL). Men who drank 5 or more cups of coffee had plasma cholesterol concentrations about 0.5, micro moles per Litre higher than non drinkers after matching this up with people of similar age, ethnicity, body mass, and so on. In women, adjusted mean cholesterol levels were 0.34 mmol/L higher in coffee drinkers. The incidence of heart attacks was also shown to correlate with coffee consumption but not with tea consumption.

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**SO WHERE DOES SUGAR FIT IN?**

Reactive hyperglycaemia is a major cause of atherosclerosis. It is clear that people with a glucose intolerance, that is reactive hyperglycaemias and diabetics or those with hyperinsulinaemia, are especially at risk of developing atherosclerosis. The idea that fats and cholesterol in the diet may not be the main villains but rather excess sugar, is not new. Ross Hall, quoting many epidemiological studies and flawed evidence in contemporary research said categorically: The villain is sugar. And Yudkin agrees following a study of a group of people who were fed sucrose (cane sugar) and who then showed an increase of all the factors necessary in the development of atherosclerosis.

People with type 2 diabetes, Hyperglycaemia and Syndrome X all have Hyperinsulinism (too much insulin in their systems). Hyperglycaemias who eat sugar have a delayed and excessive secretion of insulin. Those Hyperglycaemias who continue eating sugar are the most likely to become the Syndrome X and then the Maturity Onset Diabetics. Their high insulin levels remain high practically all the time. This excess insulin causes resistance at the actual Receptor Site. The role of insulin is too push sugar out of the blood supply into the body cells so it can be used to make fuel. Insulin is a powerful anabolic hormone that pushes sugar out of the blood supply into the body cells to produce glycogen for storage later or can be utilized for energy. Then insulin also pushes small fatty acids from the blood stream into the body cells (particularly the fat cells) to make triglycerides (more complex fats) and also pushes amino acids (basic pro-

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**Insulin Facilitates Transport Across Membranes**

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<tr>
<th>Glucose</th>
<th>Fatty Acids</th>
<th>Amino Acids</th>
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</thead>
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<tr>
<td>A</td>
<td>B</td>
<td>C</td>
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Insulin binds to a receptor on the cell membrane allowing the entry of glucose into the cells to form glycogen, fatty acids to form triglycerides and amino acids to form protein. Insulin is an ANABOLIC hormone. At the same time it inhibits CATABOLIC processes.
tein units) across the membrane to form proteins. So insulin is a building block (anabolic) hormone.

Doctors generally think of insulin on a glucose level only, that your blood sugar and insulin are all tied up, and they are. But if one has insulin resistance, one has very high insulin levels and its moving sugar out of the blood stream into the body cells, but also moving the fats from the bloodstream into the endo, cell lining of the blood vessels. Once things go wrong and hyperinsulinemia is established, there is a predisposition to causing atherosclerosis. Sugar doesn’t actually push cholesterol into your arteries, but on the axis of free fatty acids and triglycerides this action of insulin is ultimately blocking arteries with cholesterol. (FIGURE 3)

The first recommendation would be to adopt the hypoglycemic diet, this helps the pancreas stop pumping out excessive insulin. The most important mineral supplements are chromium and zinc, because they are active at the insulin receptor site. Healing the receptor site by eating a hypoglycemic diet helps to switch off the mechanism of hyperinsulinemia and therefore stop insulin ultimately pushing cholesterol into artery walls.

IS THERE ANY VALUE TO AN OATBRAN DIET?

A study published in the American Journal of Clinical Nutrition in 1983 examined the blood pressure of twenty healthy normotensive men after they had ingested various sugar solutions following an overnight fast, similar to our modern day GTT (Glucose Tolerance Test). Different solutions were given to the men to see if their blood pressure would change, but it was not altered by ingestion of water, lactose or galactose. With glucose and sucrose blood pressure after one hour rose significantly (10 mg of Mercury) and it stayed high for two hours. After ingestion of sucrose blood pressure levels rose significantly (9 mm Mercury) and that lasted for one hour. So sugar actually pushed up the blood pressure. Obviously we see high blood pressure in heart attacks and heart disease, so sugar is tied up with these as well.

WHAT ABOUT BLOOD PRESSURE CONTROL?

A study on increased consumption of raw foods from Southern Medical Journal, 1985. For a mean duration of 6.7 months, thirty two people volunteered to eat an average of 62% of their daily diet. For a mean duration of 6.7 months, thirty two people volunteered to eat an average of 62% of their daily diet. The average blood pressure dropped significantly by 17.6 mm Hg. In addition there was significant weight loss of 3.8 kg and 80% of those who smoked or drank alcohol spontaneously ceased.

An article by Linus Pauling talks about atherosclerosis in a totally different light. It’s to do with evolution that human species are complicated, we are different to other creatures except the higher primates like chimpanzees and gorillas who also have lost their ability to make vitamin C in the adrenal glands of their bodies. We are unique in animal kingdom in that we cant make vitamin C, and Linus Pauling has written this thesis called “A Unified Theory of Human Cardiovascular Disease Leading the Way to the Abolition of This Disease as a Cause for Human Mortality”. He is suggesting that a higher dose of vitamin C (say 8000 mg daily) would stop humans developing atherosclerosis.

There are studies that show a 70kg bull makes about 10,000 mg of vitamin C every day, so the vitamin C we are getting is nowhere near as much as a bulldog. To prevent scurvy you need 25 mg per day, so we are preventing scurvy if we have an apple or orange every day, but were not getting the 10,000 mg that the bulldog is.

OTHER HEART CONSIDERATIONS

Problems with electrical pathways of the heart pump Arrhythmias.

Drugs used include Digoxin from the Foxglove Herb (Digitalis), Quinine, Ameodorene, and Beta Blockers.

To reduce cardiac arrhythmias, several studies have suggested magnesium supplementation may be beneficial, perhaps because it corrects a magnesium deficiency in the myocardium. Potassium supplementation may also be beneficial, and there is preliminary evidence that tartar might be of value.

20% of forty five consecutive patients with symptomatic atrial fibrillation had deficient serum magnesium levels (American Journal of Cardiology, 1986).

Two elderly patients with ventricular tachycardia received magnesium infusions which resulted in magnesium retention and cessation of the attacks (Acta Medica Scandinavia, 1982).

Problems with muscle strength and integrity of the pump - Cardiomyopathy.

Thirty four patients with severe congestive cardiomyopathy received 100 mg CoQ10 daily. 82% improved as shown by increased cardiac ejection fraction significant, reduced shortness of breath and increased muscle strength. These improvements lasted as long as 3 years in patients treated continuously, but cardiac function deteriorated when CoQ10 was discontinued. Of the eighty patients treated, 89% improved with supplementation (Proc. Natl. Acad. Sci., 1985).

Selenium supplementation may help. Damage to the Pipes. (Claudication, CVA, Vascular Insufficiency and Atherosclerosis).

Vitamin C, E, and Selenium help keep the pipes clean. Intermittent claudication is merely another sign associated with general atherosclerosis or the degenerative disease of blood vessels feeding the calf muscles. It typically presents with cramps of the calf after walking for a certain distance. The reason for the pain is that as with angina pectoris, muscles are starved of oxygen by a poor blood supply and also the muscle is unable to get rid of the waste products during exercise.

In a controlled experimental study, thirty two out of forty seven men with severe symptoms of claudication after walking a kilometer. After about eighteen months, twenty nine of the thirty two men on vitamin E demonstrated increased calf blood flow while ten out of fourteen men in the control group demonstrated a decrease (American Journal Clinical Nutrition, 1974).

Blood pressure and alcohol consumption

In an observational study alcohol consumption was studied in relation to blood pressure for 883 men and 959 women aged between twenty and seventy four who were not on anti-hypertensive drugs. After the data was controlled for smoking, exercise and weight, the amount of alcohol consumption was directly correlated with increases in both systolic and diastolic blood pressure (American Journal of Epidemiology, 1983).

Blood pressure and a vegetarian diet

In another observational study, ninety eight vegetarians were compared to a matched group of non vegetarians. The average blood pressure was 126/77 for vegetarians and 147/88 for the control group, a significant decrease. Only 2% of the vegetarians had hypertension (BP above 160/95) compared to 26% of the non vegetarians. Both groups had a similar sodium intake and excreted the same amounts of sodium. (American Journal of Clinical Nutrition, 1983).

Dos and don’ts.

FOOD

Vegetarian diet fish is good. Avoid allergy foods. Lower your cholesterol. Avoid excessive amounts of red meat. Avoid high cholesterol foods. Reduce coffee intake.

ALCOHOL: 2 drink limit. Avoid excess alcohol.

TOBACCO: Avoid passive smoking. don’t smoke.

BLOOD PRESSURE: Keep blood pressure under control.

EXERCISE: 45 minutes x 3 times per week walking will do. Don’t vegetate, be active!


VITAMINS: Supplement diet with Vitamin C, E, and CoQ10.

MINERALS: Supplement diet with Chromium, Magnesium, Selenium and Zinc.

HERBS: Supplement diet with garlic, Hawthorne berry, Fenugreek, Bromelain (from pineapple).

OTHER: Supplement diet with essential fatty acids, EPA/DHA.

MEDICATIONS: Aspirin and conventional blood pressure and heart drugs may be necessary.
Schizophrenia is probably the most serious of all the psychotic disorders that can afflict not only the person, but the whole of his family and his immediate social environment.

Treatment of schizophrenia should always be under the supervision of a medical practitioner, despite the fact that patients and members of the family may experience frustration with the palliative nature of drug-oriented psychiatry as it exists today. It is strongly suggested here, that treatment be sought from complementary doctors - meaning specialists who have combined their traditional medical knowledge with alternative approaches, in particular nutritional medicine.

Unfortunately, psychiatrists with post graduate diplomas in clinical nutrition are few and far between in Australia as perhaps elsewhere.

Post graduate diplomas can be obtained from:

The Australia College of Nutritional and Environmental Medicine, 13 Hilton Street, Beaumaris, Vic, 03-9589-6088 (see: http://www.acnem.org)

The International Academy of Clinical Nutrition, PO Box 370, Manly NSW, 2095 (See: http://www.intacad.com.au)

I also recommend that patients and their family members join mutual support groups for orthomolecular psychiatry, who may often be able to put you in contact with nutritionally oriented psychiatrists. (Also ACNEM has a referral service). It is only through pressure from patients and their family that will force psychiatric practitioners to respond to "market forces" and loosen the ties to the pharmaceutical companies that dominate the treatment modality of mental illness.

There is a web site, The Australian Integrative Medicine Association, (AIMA) providing names and addresses of complementary doctors in Australia at: <http://www.vanzyl.com.au/aima/>

It is imperative that the complementary medical fraternity make their presence felt and publish access to their services more openly in our community.

A good mutual support organisation is:

SOMA, which aims to see medical horizons expanded to include complementary methods in psychiatric practice, has monthly meetings, every third Monday, 10 am at St Francis Xavier Church Hall, 19 Mackenzie St Lavender Bay (behind North Sydney Station), Postal Address; PO Box 7180 Bondi Beach, 2026, Phone: 02-9789-4805, Fax: 02-9922-5747

Its patron is Dr Chris Reading, well-known orthomolecular psychiatrist in Australia and pioneer in clinical nutrition.

Symptoms of schizophrenia

It is a disease that affects the patients ability to recognize reality, characterized by hallucinations, which does not result from external stimuli but from distortions in any of the senses. Accordingly, they can be classified as auditory (affecting hearing), gustatory (affecting taste buds), olfactory (affecting sense of smell), tactile (affecting touch) and visual (affecting vision). Victims of the disease may show disorganized speech and behaviour. There usually is a lack of insight, especially during the initial stages of the disease, but patients who gain some insight into their illness, stand a better chance of prognosis.

In delusions the person tends to misinterpret incoming stimuli.

Symptoms in schizophrenia may also be defined as ‘positive’ or ‘negative’. Positive symptoms refer to the hallucinations, delusions and disorganized behaviour, whereas negative symptoms reflects a tendency towards a restricted emotional range, inexpressive speech and inability to initiate productive activity or pursue goal directed behaviour.

There appears to be a gradual deterioration in logical thinking, social skills and erratic behaviour. It usually is a life-long illness, although if treated early in its development it could remain in remission for long periods of time. Orthomolecular psychiatrists and doctors are somewhat more optimistic in believing that psychotic disorders are treatable by not only pharmaceutical as well as nutritional or other means in the not too distant future.

Schizophrenia has been subdivided into five subtypes:

Paranoid schizophrenia is often associated with schizophrenia in popular thinking, although in reality they account for a minority in the disease. They display the ‘positive’ symptoms of hallucinations and delusions. These delusions are not always paranoid - marked by suspicions - but may concern religion or ideas of grandeur. Only a small percentage of this category may pose a danger to themselves or others.

Disorganized schizophrenia refers to disorganized behaviour or emotional responses that are either blunted, "flat affect" or unusual.

Catatonic schizophrenia characterized by severe psychomotor disturbances, resulting in immobility or increased physical activity, becoming mute and displaying unusual or inappropriate behaviour.

Undifferentiated schizophrenia although ‘positive’ symptoms are present, they fail to fit in with any category.

Residual schizophrenia when negative symptoms predominate, but without major psychotic symptoms.

Causes

Underlying causes of schizophrenia are unknown. Some researchers believe schizophrenia is hereditary, and there is some evidence that some cases of the disorder are the result of an inherited defect in body chemistry in which brain chemicals called neurotransmitters function abnormally. Others theorize that it results from external factors, such as complications during birth, head injury, reaction to a virus, or environmental poisons that damage the brain. Horrobin speculates that during the evolution of the human species brain connectivity may have been improved through abnormalities in phospholipid metabolism. This schizophrenia may be seen as an intermediate step in the development of the human brain. He proposed that the changes which resulted in the schizophrenic genome “caused the emergence of humanity, and that, especially in their milder forms, they are strongly positively associated with human achievement”. (Horrobin)

Diagnosis

Just as fever is a sign of various underlying illnesses, so in schizophrenia, the clinician needs to consider a variety of other possibilities, such as mood disorders, and any fundamental medical condition that could account for the symptoms. Foremost among these are hypoglycemia, allergies and/or food sensitivities to which we will return later.

Conventional treatment of schizophrenia is by means of various antipsychotic drugs, psychotherapy and social support.

Some well-known pharmacological agents are: chlorpromazine, clozapine, fluphenazine, haloperidol, paroxetine, phenothiazines, thioridazine, trifluoperazine, and many others. Most of these are effective for positive symptoms, with the exception of clozapine, which also acts in the case of schizophrenia with negative symptoms. However, clozapine is said to have very serious side effects in one per cent of cases: it seems to destroy white blood.
cells needed to fight infection. It also may alter the heart rate, increase blood pressure, cause excessive salivation and constipation. But on the plus side it does not cause tardive dyskinesia (muscle rigidity), as with most of the other drugs. Lithium is sometimes prescribed in schizophrenia to stabilize moods. The kidney handles lithium and sodium the same way and when there is too much sodium in the body, lithium levels drop. If the body is depleted of sodium, lithium can build up and toxic reactions are possible. (Graedon, 291) Thus avoid low salt diets. Supplementation with low dose natural lithium from vegetable concentrates may sometimes be preferable (Lithomin, Nutridyn 50 g with meals) and should be tried. (Fierro AA 1988)

Many if not most major tranquilizers deplete riboflavin, vitamin B12, folic acid and vitamin C. The latter may reduce the effectiveness of the drugs (Graedon 291). Side effects of psychotropic drugs seem to be caused by interference with nutrients. Knowledge of the drug/nutrient interactions may provide steps to reduce adverse reactions. Nevertheless, any objective standards, chemotherapy alone for psychotic disorders has remained disappointing.

Since other mental disorders may mimic schizophrenia, other medications are tried, such as anti-anxiety drugs, or those treating agitation.

**Term schizophrenia under attack**

It should be noted that there are a number of psychiatrists who have serious doubts about the stigmatization of patients with the label “schizophrenia”, and even doubts the usefulness of diagnosis. They argue that there is little scientific evidence in support of the existence of the schizophrenia, and that it would be more beneficial to treat the distressing symptoms of patients directly. (Boyle)

**Tardive dyskinesia (TK)**

Long term use of antipsychotic drugs, such as chlorpromazine, thioridazine, trifluoperazine or phenothiazine and many others may cause tardive dyskinesia. “Tardive” means late (later on in treatment) and “dyskinesia” means impairment of voluntary movement. Patients may display repetitive movements, lip smacking, swinging of legs to and fro. These side effects appear to be irreversible, and may cause patients to abandon treatment. It is therefore essential to prevent the disease. It appears that the dyskinesia is associated with a errors in dopamine metabolism. (Pearson et al. 134)

Studies with a large numbers of patients have shown that megavitamin supplementation can prevent the development of these side effects.(Tkacz, Toll)

Administration of these supplements should be under strict supervision by a medical practitioner, because they ought to be considered part in the overall treatment program, and some may interact with drug treatment. Vitamins administered were:

- **Vitamin C** up to 4 grams a day
- **Vitamin B3** (Niacin or nicotinamide) up to 4 grams a day. Nowadays inositol hexaniacinate may be the preferred form, because of unpleasant but harmless side effects of niacin (Head KA)
- **Vitamin B6** up to 800 mg per day, these high doses may have side effects and need monitoring by a doctor. Some scientists do not recommend such high doses.
- **Vitamin E** 1,200 IU per day (Dahiri, Lohr). Vitamin E is thought to play a role in the antioxidation of neuroleptic drugs in certain parts of the brain.
- **Vitamin B-complex**
  - **Manganese** 15mg per day, (Vayda, 158, Kumin RA, Norris)
  - **Lecithin** providing choline has been successfully used. (Pearson, 134)

Only few studies have reported benefits with evening primrose oil in the treatment of TK.

Some researchers believed that tardive dyskinesia was associated with the body’s inability to synthesize certain amino acids involving phenylalanine and branched chain amino acids (BCAA), such as valine, isoleucine, and leucine. The liver was believed to compete with excesses of phenylalanine levels. Some improvement was noted among a few patients with supplementation BCAAs. (Richardson et al.)

**Non-pharmacological Treatment**

Studies have shown that exercising can reduce the symptoms of both depression and anxiety. (Taylor et al., Adams). It has even been demonstrated to reduce aggressive halucinatory behaviour. (Belcher, Conroy et als.). It is well known that schizophrenic patients generally have a poor diet. They tend to be overweight, but this may well be due to their medication. In addition, they seem to be heavy smokers. This could be explained by the fact that nicotine binds to nicotinic receptors in the brain, augmenting the release of numerous neurotransmitters, including dopamine, serotonin, norepinephrine, acetylcholine, gamma-amino butyric acid, and glutamate. This provides them with psychological reward and cause addiction (Quattricki et als.).

There are reports of psychotic episodes being precipitated by excessive caffeine use. Caffeine is a methylxanthine that has an behavioural effects one hour after consumption. It increases norepinephrine and serotonin and has a biphasic effect on dopamine in the CNS. (Mikelsen). Caffeine increases the anxiogenic effects among many patients. (Bruce M)

**Cognitive therapy**

Any method that aims at reducing psychological anxiety and stress, which could trigger a major depression in sensitive people, should help the patient cope with frustration. Some will therefore benefit from psychotherapy. A study compared the results of patients in groups with different treatments. 1) receiving chemotherapy alone, 2) patients receiving chemotherapy and supportive counselling and 3) chemotherapy plus intensive cognitive therapy (psychotherapy).

It was found that patients receiving cognitive behaviour therapy showed the greatest improvement in positive symptoms. Patients receiving routine care alone showed minimal change, and those who received supportive counselling showed some improvement but less so than those receiving cognitive behaviour therapy. This difference was significant for cognitive behaviour therapy compared with routine care alone. (Tarrrier 1998)

**Nutritional factors affecting the course of schizophrenia**

For many years it has been speculated that dietary factors contribute to the symptoms of schizophrenia. Apart from poor diets, some individuals may require more than the RDAs of vitamins and minerals even if diets are ‘adequate’.

In fact, schizophrenia or any other psychotic disorders could be affected by a number of dysfunctions along the route of nutritional digestion. There are about 22 digestive enzymes capable of digesting carbohydrates, proteins, sugars and fats. Magnesium is involved in about 400 enzymatic reactions, 1 percent of which is in the extracellular fluid, thus normal magnesium levels can exist in situations of intracellular depletion. (Lopez MJ, 1997) Zinc is a constituent of about 200 enzymes (Florence et al. 58).

Patients should ensure that they have adequate intake of vitamins by taking a B-complex vitamin supplements as a starting point. But let us be aware that normal dosages may be ineffective in some individuals. For example a deficiency of **vitamin B1** (Thiamine) alone may precede symptoms of chronic fatigue, irritability, memory loss, personality changes (such as aggression), insomnia, anxiety, restlessness, night terrors, appetite loss, sensitivity to noise, numbness and tingling in hands and feet, and circulation problems for those requiring higher doses.  

Another example is, the finding that many schizophrenic patients lack **hydrochloric acid** in the stomach, which is a protective barrier against diseases further down the digestive tract. Consequently, proteins are broken down into polypeptides setting up auto-immune reactions which result in “schizophrenic” symptoms. Thus it may pay for patients to take digestive enzymes; such as betaine hydrochloride, Papania, probiotics (acidophilus), dandelion root, gentian, Goldenseal, Milk thistle, Yarrow, chamomile. A Heidelberg analysis can tell if the stomach acid level is within normal range.

In my experience with people suffering from a mental and/or psychiatric disorders the adoption of the **hypoglycemic diet** markedly improved symptoms, but no claims can be made that this diet alone would remedy the condition. Common sense would tell us that fluctuating blood glucose levels supplying energy to brain cells would contribute to and exacerbate psychotic symptoms. However, the hypoglycemic diet, just as the diabetic diet - though close to a natural diet - would necessarily include some foods to which the schizophrenic patient may well be allergic.

A high intake of total fat predominantly of

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saturated fat derived from land animals was significantly associated with unfavourable ratings in the outcome of schizophrenia from survey in 8 national centers. (Christensen et al., McCready R et al).

Hydrolysis of gliadin fraction of gluten proteins from wheat in the presence of pepsin and known to produce schizophrenic symptons in gluten/glindian sensitive patients. (Washburn CF et al, Ross-Smith P et al.). But not all schizaphrenics demonstrated evidence of involuntary response to gluten. (Storms LH et al., Potkin et al).

The same applies to sensitivity to casein, the major protein found in cow’s milk (Dohan FC et al).

Results suggest that some, but not all, people with schizophrenia may benefit from a gluten-free/dairy-free diet and the best way is to have this tested by your doctor or by trial and error. (See “Finding your Allergies” at the Hypoglycemic Health Newsletter of Australia” web site).

There has been several studies showing that schizophrenics have elevated copper levels, as shown among a group of violent male per cent of cases (22/38pts) in one experiment (Werbach, 1978). But the elevated copper factor in schizophrenia is marked by fatigue, irritability, memory losses, and ear noises such as ringing. Tests for manganese levels should be conducted.

Deficiency of vitamin B12 alone can mimic symptoms of schizophrenia and patients are advised to have periodic injections of vitamin B12 and folic acid by their doctor. Absorption of B12 from food depends on the presence of the intrinsic factor, a glycoprotein synthesized in the stomach wall which binds with B12. It is subsequently bound by a special receptor in the ileum, where it is released by a releasing factor into the blood system. An abnormality along this pathway can cause severe B12 deficiency, with physical and emotional consequences. Sublingual vitamin B12 are also available and may be preferable in some circumstances.

Zinc and copper are in balance, meaning that high levels of one causes lower levels of the other and vice versa. There are several studies that patients with low zinc levels - may be due to high copper levels - responded positively with zinc sulphate and pyridoxine. (See studies see Werbach 236). Zinc deficiency is associated with apathy, lethargy, amnesia and mental retardation often with considerable irritability, depression and paranoia. On the other hand high zinc levels may lower manganese levels. (Pfeiffer CC, LaMola S, 1983). Zinc deficiency can also block omega-6 essential fatty acid metabolism into gamma-linolenic acid (GLA) and dihomogamma-linolenic acid (DGLA). (Cunnane SC et al.). As in diabetes, patients may have dysfunctional desaturas (enzymes) causing a blockage in the conversion of essential fatty acids into GLA or EPA. A natural forerunner of essential fatty acids, both omega-6 and omega-3 is flaxseed oil or walnut oil. In any case, supplementation with essential fatty acids in the form of GLA (found in Evening Primrose Oil) and fishoil (Max EPA) have been shown to improve symptoms, including side effects of anti-psychotic medication. (Laugherne JD)

Some believe that schizophrenics have a higher need for niacin. Thus some schizophrenics may be vulnerable topellagra. This is a disease resulting from a deficiency of niacin or tryptophan, or a metabolic deficit that interferes with the conversion of the precursor tryptophan to niacin. Symptoms are likely to be skin and digestive, cutaneous neuropathies, tinnitus and fatigue. (Rudin).

Several studies have shown that many schizophrenics (about 36% in one study) have low red-cell folate levels and that histamines in particular were deficient. It was suggested that folic acid therapy along with vitamin B12, niacin, vitamin C and zinc was effective in the treatment of “histapenic” schizaphrenics after 5-6 months with a rise in blood histamine levels (Werbach and other studies). (Pfeiffer CC et al., 1979). However there is a warning that supplementation with folic acid can also cause an exacerbation of psychotic behaviour if blood levels become elevated. (Werbach 221). Thus folic acid should only be used in proven deficiency.

Early reports claimed that treatment with high doses of niacin (vitamin B3) would improve psychotic symptoms in schizophrenia. But treatment with nicotinic appears to be beneficial only in cases of deficiency. Niacin treatment of schizophrenia has remained controversial following an extensive review of a series of clinical trials, which were generally negative. (Ban TA).

Adverse side effects of niacin therapy were reported, including nausea, GI irritation, increased blood sugar and uric acid. The alternative in the form of niacinamide may however produce depression and could cause hepatic toxicity. (Werbach 223a).

Studies conducted with vitamin B6 (pyridoxine) are equally contradictory. There is one case report of 2 catatonic schizophrenics, unable to tolerate neuroleptics, who responded positively to the administration of 500 mg daily of vitamin B6. (Brooks SC) But Werbach warns that doses more than 50 mg daily may cause neuropathy (Werbach 227). Excess B6 can affect the binding properties of serotonin receptors in the brain according to one study (Baumblett et al.). Other studies suggested that Pfeiffer’s observations may have been on patients whose schizaphrenics were generally negative. (Ban TA). However there is a warning that supplementation with pyridoxine can cause side effects from medication, as well as reduction in schizophrenic symptoms (Sanady R, 1990). In another study only one out twenty patients benefited from B6.

Nevertheless, the role of vitamin B6 in psychotic disorder can not be underestimated. It is required for the conversion of glutamine to GABA. Pfeiffer claims that when the blood circulation shows elevated amounts of “kryptopyroles”, it binds with both B6 (pyridoxine) and zinc, which are then excreted in the urine thereby causing their deficiencies. (Pfeiffer CC et al., 1988). Other studies suggested that Pfeiffer’s observations may have been on patients whose schizaphrenic symptoms were secondary to a metabolic disease similar to poryphria, also associated with B6 deficiency. (Cruz WT al 1978).

The administration of an amino acid, Glycine, which only plays a role in maintaining the health of the prostate gland, but also appears to influence the function of neurotransmitter in the brain, involved in memory and cognition. (File SC et al.). Glycine is used in liver detoxification in the form as glutathione, cysteine and glutamic acid) and perhaps de toxification may be responsible for the reduc.
Vitamin C is also involved with detoxification and its supplementation has been reported to benefit patients. (Sandry R 1993, Kanofsky 1992) In an uncontrolled trial, some schizophrenic patients continued a course of vitamins (4 to 10 grams of niacin or niacinamide, 4 grams of vitamin C, and 50 mg or more of vitamin B6) after being discharged from the hospital, while a control group of patients discontinued the vitamins upon discharge. Both groups continued to take their psychiatric medications. Those who continued to take the vitamins had a 50% lower readmission rate compared with those who did not. (Hawkins et al 1970) Yet some subsequent studies have not been able to confirm these results.

Tryptophan is an essential amino acid and the precursor of the important neurotransmitter Serotonin in the presence of vitamin B6. Tryptophan levels appear to be lower among schizoaffective patients compared to non-schizophrenics. Tryptophan is also needed to synthesize niacin. Sixty milligrams of tryptophan yields 1 mg of niacin (Kirshmann, 36), which could explain a abnormality in serotonin metabolism among schizophrenics.

In an uncontrolled trial, patients with schizophrenia were given 2–8 grams of L-tryptophan and 100 mg of vitamin B6 daily. This resulted in decreased agitation and less fear among patients, but these and other effects were not as great as those achieved with psychiatric medications. (Bowen 1970) It appears that schizophrenics use L-tryptophan in a different way. In a survey in populations from several English-speaking countries it was found that when tryptophan intake was low, suicide rates were high. (Kitahara) Thus tryptophan supplementation should be considered as a possibility in the treatment of schizophrenia.

What is reported that studies of serum levels of magnesium in schizophrenics is contradictory. It appears that with patients on neuroleptic medications magnesium levels may be lowered, compared to drug-free schizophrenics who have normal levels. (Werbach 224) Calcium and magnesium levels are in a ratio of 2:1. Biochemical tests should indicate whether there are any abnormalities, as these minerals are associated with many symptoms, such as anxiety, insomnia, fatigue and high blood pressure.

Some nutritional sources of brain chemistry
It is essential that any experimentation with nutrients to treat schizophrenia or any other psychotic disorder should be under the supervision of a medical practitioner.

Some scientists theorize that manic-depressive illness results from a derangement of a balance between two important nerve signal transmitters: norepinephrine and acetylcholine (ACh). Choline levels are too high in depression and norepinephrine are too high in mania.

Acetylcholine is a neurotransmitter which is important in the parts of the brain affecting primitive emotions like sex and a degree of responsiveness to outside stimuli, as in alertness versus sleep. It plays a role in memory, learning and long-term planning. When ACh levels are low, the person is easily distracted by irrelevant stimuli in the environment. Acetylcholine is used in the brain and nerve cells and required for memory, appetite, and sexual behaviour. Many hyperactive children respond positively to choline in lecithin, and this may also be useful in withdrawal from alcohol during treatment of alcoholism. (Pearson 274)

The body manufactures acetylcholine from phosphatidyl choline which is found in lecithin. Other sources are: soy, egg, brewer’s yeast, grains, legumes, fish, wheat germ. Egg lecithin may be more effective for those suffering AIDS, herpes, chronic fatigue, immune disorders. For our purpose soy lecithin granules or capsules would be the more convenient.

Vitamin B5 (pantothenic acid or Pantothenate) is required for the conversion of phosphatidyl choline in lecithin to acetylcholine. (Pearson 274). Pure lecithin contains 15-20% of phosphatidyl choline and this source of choline is more effective than from choline alone according to Wade. (Wade, C 1980).

Thus in the depressive phase of manic-depression (when acetylcholine levels are high) lecithin should be avoided, but in the manic phase it should counterbalance the high norepinephrine levels, which is thought to be responsible for the mania.

Glutamine is another important nutrient having effects on neurotransmitters in the brain. In the catabolism of amino acids nitrogen is produced, which can form free amino acids that is especially toxic to the brain. The liver can convert nitrogen to urea, which is then excreted as urine. Nitrogen can also combine with glutamic acid to form glutamine. (Chaitow, 79) Glutamine is stored in muscle tissues and needed for synthesis of skeletal muscle protein.

More importantly, glutamine is the precursor of GABA (or Gamma-aminobutyric Acid) which is inhibitory transmitter controlling the synthesis of dopamine. GABA keeps the brain and body from going into “overdrive”. GABA seems to be quite effective for anxiety disorders such as insomnia (especially the type of insomnia where racing thoughts keep the individual from falling asleep). At dosages above the 250 mg it may sometimes cause slight side effects marked by tingling. It is also a precursor of glutathione among others. Vitamin B6 is required to convert glutamine to GABA, which then plays an important role in regulating the synthesis of dopamine from dopamine cells. The importance of this is that schizophrenia is thought to be due to a disordered dopamine metabolism. GABA is available in supplemental form and should be taken with water (do not take milk), together with vitamin B6 and vitamin C.

Valium & Librium are tranquilizers that act on benzodiazepine receptors in the brain. Scientists claim that niacinamide, inositol, gamma aminobutyric acid (GABA) occupy the same benzodiazepine receptors. (Use B3, 1g; inositol 3g; 500mg GABA sublingually), and hence these may form an alternative to chemical tranquilizers. (Pearson 282).

Taurine, GABA and zinc are known to be a calming agent in the brain. (Kiffer,C.C.1978, 77), Taurine has been shown in human trials to have an anticonvulsive effect. Its apparent role is to normalize the balance of other amino acids, which in epilepsy are disordered. Serum zinc has also been found to be low in epileptics. Full spectrum light exposure results in increased levels of taurine being concentrated in the pituitary and pineal glands. Continued exposure to artificial light might cause the concentration to be reversed.

Phenylalanine is an essential amino acid and is a forerunner of a group of substances known as catecholamines, which play important roles in brain chemistry. The metabolic pathway can be represented as: Phenylalanine > tyrosine > dopa > dopamine > norepinephrine (noradrenaline) > adrenaline (epinephrine). (Lehninger, 728)

D-Phenylalanine can be decarboxylated to phenylethylamine (PEA) which has amphetamine-like stimulatory properties (found in high quantities in chocolate) and requires vitamin B6 for its conversion (Werbach, 142).

Methionine is another essential amino acid that can be converted through the action of methionine-adenosyl-transferase (MAT) to S-Adenosyl-L-Methionine or SAM. Both SAM and folic acid affect the turnover of monoamines, including serotonin and dopamine. In a study of 90 psychiatric in-patients normalization of enzyme activity was associated with clinical improvement. There was however a warning that SAM could induce mania in patients with bipolar illness. (For studies see Werbach, 143)

Ginkgo biloba is a vasodilatory effect and supplementation after 3-6 months has been shown to have positive outcomes in cognition among people suffering from Alzheimer’s Disease without hardly any adverse effects. (Pken BS). It could therefore play a role in schizophrenia.

St John’s Wort (Hypericum) seems to be worth exploring in the same way as norepinephrine. Serotonin Reuptake inhibitors) and may be an alternative antidepressant medication in moderate depression.

Pycnogenol or grape seeds extracts is a potent antioxidant and said to benefit patients with dementia and brain syndrome. (Balch et al 1997, 466)

Coenzyme Q10 improves cerebral circulation.

Conclusion
On reason why many psychiatrists are reluctant to embrace nutritional therapy may be due to the contradictory results of clinical studies in nutrition of groups of schizophrenics. This assumes that there is such a category as ‘schizophrenia’. These studies completely overlook Roger Williams concept of Biochemical Individuality (Williams, Roger 1971) which emphasizes the uniqueness of patients from a biochemical point of view. If the quantity of all the natural chemical substances in our body could be represented as spokes emanating from a central point, then individuals would present three dimensional constellations uniquely different from one another. It can
also be assumed that the deviation from the norm may be greater among schizophrenics. If this is the case, then the statistical comparisons of subgroups of ‘schizophrenics’ into control and experimental groups is inappropriate as a scientific tool. There is no control group, because all are different.

The popularity of the present scientific methodology stems from studies concerning the effects of xenobiotic chemicals on the human body, where other confounding biological factors (such as age, gender, height, economic income etc.) are controlled. Psychiatrists, using pharmaceuticals as their main tool, would be well versed in this methodology. This scientific model does fit the ‘one factor’ experimental design. But if we look at humans through the eyes of Roger Williams then it is obvious that studies investigating the influence of nutrients on mental health cannot describe people assigned into groups and subgroups (experimental and control) for statistical purposes.

It is apparent that psychiatry will have to go into a different direction if they are really to treat psychotic disorders. Future psychiatrists will have to be thoroughly trained in clinical nutrition (even herbalism), patients need to be intensively analyzed. They need to be tested for biochemical abnormalities, for vitamins and mineral deficiencies or excesses (for instance by means of hair analyses, blood and biochemical assays, allergies and food sensitivities and so on). Psychiatrists may even have to reassess their scientific methodology. Thus in the future, the curriculum for studies in psychiatry would have to place greater emphasis on nutrition and biochemical diagnostics, followed by remedies other than pharmaceuticals - away from xenobiotic to natural symptomatic remedies - which are patient-friendly.

The patient needs to be understood in terms of his biochemical individuality. There appears to be no treatment for schizophrenia as such, but there is for individual suffering from ‘schizophrenic’ symptoms. We may consider nutritional treatment a unique complementary make-up and treat him accordingly.

In the meantime, it is up to the patients and their families to seek out ‘psychiatrists’ who are familiar with not only pharmaceutical interventions, but also remain viable in the future - but more importantly with the principles of clinical nutrition and other possible modalities that can alleviate the suffering of mental disorders.

Footnotes


Pfeiffer CC, Bravermann E, and vitamin B12 therapy for the low-histamine, high copper biotype of schizophrenia, in M Betar, Ehi Reynolds Eds Folic Acid in Neurology, Psychiatry and Internal Medicine, NY, Raven Press 1987:487


Pikkarainen SP, Lehtonen TJ, discs of gliadin in the serum and the binding properties of semiconscious schizophrenic patients to cortex of rats. J Nutr 128: 1829-1835


Schaeffer MG, Gretz D, Gietzen DW, Rogers OR (1995), Dietary excess copper affects the concentrations of amino acids in that cause inorganic and serum and the binding properties of serotonin to the cortex of rats, J Nutr 128: 1321-28


Tarr R (1999), Randomised controlled trial of intensive cognitive behaviour therapy for patients with chronic schizophrenia. British Medical Journal, 1999


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2001 MEETING DATES ON FIRST SATURDAYS

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The Editor is looking for a member who is willing to help out in transcribing lectures from tape a recording. The volunteer should have access to a computer and should have facilities to email reports to the Editor. We would appreciate it very much if you could help us out.

Attention is drawn to our new Web Site at: w w w . c o m p a n y o n t h e w e b . c o m / hypoglycemia_australia where you’ll find articles on clinical nutrition and self help psychotherapy. We also have an article dealing with the treatment of drug addiction. Copies of Newsletters are also available.

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over” into a diabetic, sooner than later.

We have both been put on new nutritional diets – sugar free and yeast free – and for me also soybean free – for me personally, both as a cook and consumer, this has been extremely frustrating. Sometimes its very hard to change, even though you know its for the better in the long term, and you certainly miss the “sweet” taste of foods. When shopping you quickly discover that well over 90% of our foods have some content of sugar, yeast and preservatives contained in them. It has been extremely frustrating for me to find recipes to prepare which fit the diet schedule, as I have been so used to preparing meals with sauces and ingredients which have been sugar/yeast based, but am continually trying new ideas. Our new diet consists of chicken, fish, lamb, fresh fruit (for me only to be eaten with a meal) and vegetables, and lots of home cooking which is sugar/yeast free! Out go all my cookery books!! Both my husband and I have noticed the changes in ourselves – he has lost 4-5lbs – I don’t wish to as I only weigh 8 stone! We both eat six small meals a day and I myself need to eat every two and a half-hours! I know that if I miss this time frame then I become extremely hungry and light-headed, start to feel anxious and even feel “weepy”. At time I find this very hard to come to terms with as I have never undergone this great a change in my body before.

But thanks to the wonderful support of my husband, and my doctor who has put us both on the right track back to good health, I feel now that there is a light at the end of the tunnel and life is not as gloomy nor stressful as it was once before.

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