

# Family Tree Connection: How Your Past Can Shape Your Future Health

## A Lesson in Orthomolecular Medicine

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Genetics has become an essential part of the medical and psychiatric work up. Thus with renewed world wide interest in "Orthomolecular Medicine/Psychiatry", "Molecular Medicine", "Nutritional Medicine", "Clinical Nutrition", "Applied Nutrition" and "Clinical Ecology" then as a natural sequence, "Orthomolecular Genetics" and "Medical Genealogy" will become increasingly important. I define Orthomolecular Genetics/Medical Genealogy as the identification and recording for future generations of the genetic, metabolic, hormonal, allergic/immunological and toxic disturbances that are running in families, contributing to, perpetuating, exacerbating, and causing medical/psychiatric symptoms/signs allowing for their correction. Orthomolecular Genetics, I believe, will come to help each and every one of us. We are not able to choose our parents, but we do have the choice to do the best we can with what we have inherited, to reach our full potential.

A stumbling block, initially, to the acceptance of Orthomolecular psychiatry and medicine as applied to genetics has arisen from a misunderstanding of the definition. The correct definition is found in the Medical Journal of Australia, July 14, 1979, p. 40, where I stated "I would like to clarify what I believe Orthomolecular psychiatry is all about. Megavitamin therapy is not synonymous with it. It is only a subspecialty. Orthomolecular Psychiatry is the study of the genetic, metabolic, endocrine, immunological and toxic disturbances that are contributing to, perpetuating, exacerbating or even causing the psychiatric symptomatology.

The other half of the definition which is

often left out when people criticize Orthomolecular psychiatry, and to distinguish it from usual biological psychiatry where often vitamins, minerals, food allergies are not measured is as follows: "It is the investigation of vitamin (coenzyme) levels, mineral (cofactor) levels (or toxic levels of lead, copper and so on), hormone levels (we can't measure endorphin levels, exorphin levels, or prostaglandin levels at the moment)" [this was 1979], "immunoglobulin levels (especially IgA and IgM), electrolyte levels (especially bicarbonate, calcium (and) blood sugar, and so on). What can be corrected is corrected and the patient is followed up regularly." It clearly states that any organic factors that could be causing the psychiatric symptoms become the concern of the Orthomolecular psychiatrist, including vitamin and mineral deficiencies and food allergies. The medical literature is full of organic conditions presenting as psychiatric symptoms, and I refer to "Medical Screening of Psychiatric Patients" by Earl Gardner and Richard Hall, Journal of Orthomolecular Psychiatry, Volume 9, No. 3, pp 207-215, where they look at the incidence of medical disease in psychiatric patients and state the following conclusions seem justified from the study:

1. Approximately 80% of state psychiatric hospital inpatients have some medical illness requiring treatment.
2. It is difficult to distinguish physical disorders from functional psychiatric disorders on the basis of psychiatric symptoms alone.
3. A large percentage of patients admitted to a state psychiatric hospital have previously undiagnosed medical illnesses which cause or exacerbate their psychiatric symptoms.
4. The endocrine and central nervous sys-

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terns are the physiological systems of the body most often associated with medical illnesses which cause or exacerbate psychiatric symptoms.

5. Patients with medically determined major psychiatric symptoms are most often diagnosed as suffering from schizophrenia or depressive disorders.
6. The vast majority of medical illnesses which cause or exacerbate psychiatric symptoms, respond rapidly to treatment with medication, etc.

Also in the Archives of Psychiatry, Vol. 37, September 1980, pp 989-995, Hall et al state: "One hundred patients of lower socio-economic class were intensively evaluated medically in a research ward for the presence of unrecognized medical illnesses that might have affected their hospitalization. Forty-six percent were thought to have medical illnesses that directly caused or greatly exacerbated their symptoms and were consequently responsible for their admission, while an additional 34% of patients were found to be suffering from a medical illness requiring treatment. A diagnostic battery of physical, psychiatric, and neurologic examinations, coupled with a 34 panel automated blood analysis, complete blood cell count, urinalysis, ECG, and sleep deprived EEG, established the presence and nature of more than 90% of the illnesses detected, and is therefore recommended as an initial evaluation battery, particularly for patients facing involuntary commitment to a mental hospital."

The cases listed include 56 schizophrenics where such things as folic acid deficiency, malnutrition, hypoglycaemia, iron deficiency anaemia, thyroid, liver and adrenal disorder, infections, toxic metal poisoning and renal disorder caused or exacerbated symptoms. The same applied to other disorders such as depression, manic depression, personality disorders and organic brain syndromes, and such autoimmune diseases as Hashimoto's thyroiditis, parathyroid disorder, and adrenal disease. Thus, not surprisingly, when I looked at chronic, long term patients in Ward 5, North Ryde Psychiatric Centre, 1973, now Macquarie Hospital, North Ryde, N.S.W. Australia, using some of the tests, I increased by ten fold the discharge rate from 12

per year to 120 per year, at a time when 80 patients in hospital a year cost the state a million dollars — thus saving 1.5 million dollars in 1973.

The vitamin profiles provided free at the time by Roche Laboratories, showed that 18 schizophrenics looked at (1) 17 out of 18 or 94.5% were low in Vitamin C; (2) 15 out of 18 or 83.2% were low in Vitamin B<sub>1</sub>; (3) 13 out of 18 or 72.2% were low in Vitamin B<sub>6</sub>; (4) 13 out of 18 or 72.2% were low in Vitamin A; (5) 11 out of 18 or 62% were low in folic acid and 6 out of 18 or 33.3% were low in Vitamin B<sub>12</sub>. Of these, none were anaemic, hence my article on Latent Pernicious Anaemia, original article, Australian Medical Journal, Jan. 25th, 1975. Note 33% had five vitamin deficiencies; 50% had four; 16% had three and only one patient had one vitamin deficiency.

Nowadays, every 1,000 patients saved from dementia or chronic psychosis saves 400 million dollars over ten years.

My book "Family Tree Connection" (formerly "Relatively Speaking") with coauthor Ross Meillon, is based on over 5000 case studies to illustrate the importance of drawing up a medical, or Orthomolecular family tree, and stresses the importance of genetics and what our family tree is clearly telling us about our future, so allowing us to take practical steps to prevent serious illness from developing, or treat illnesses once present or starting to appear. It is important to study the various ways certain genetic disorders are inherited, so we understand how autosomal dominant conditions are inherited, as well as X-linked and recessive conditions, so once the genetic transmission of an illness is established, it is usually possible to know exactly who is at risk for the illness, and who should definitely escape it. So often I hear and know of patients who have worried needlessly for years about the risk of certain illnesses, and often have not married for that reason, and if married have feared to have children, when a detailed medical family tree would have allayed their fears once and for all.

I see this often in X-linked conditions when a son is very worried about a serious illness in his father, but cannot inherit that condition from his father as he can't get his X-chromosome from his father. He

gets it from his mother and the Y-chromosome from his father, making him male. Similarly, a man with a serious X-linked illness, such as manic depressive illness, certain neurological disorders, some psychoses, even familial cancer, etc., cannot pass on that X-chromosome bearing the genetic disorder to any of his sons, but all the daughters are at risk depending on whether the condition is X-linked dominant, where the daughter usually shows the condition later, or X-linked recessive, where she is a carrier. However, comforting as it is to know what one is *not* at risk for, what if one *is* at risk for, or in the firing line for a serious condition because the genetic transmission is known in your family, or if one has the illness already. This is one of the main purposes of my research and book and of relevance, I believe, to us all.

If you, or those you are concerned about, are in that situation, whatever the illness, be the cause unknown or the treatment unknown, and unlikely to be known at this stage, Orthomolecular genetics/medical genealogy behoves a detailed medical family tree to help solve the illness. The following things ideally should be included: — Ages where known, age at time of death and cause where known, medical conditions especially cancer (and type where known), diabetes, hypertension/ high blood pressure, strokes, thromboses, heart attacks, arthritis (osteo-arthritis, rheumatoid arthritis, Systemic Lupus Erythematosus, gout, etc.), pernicious anaemia, thyroid disorder and other medical conditions. Phenotypes or description of the person are important to record, including hair and eye colouring, complexion (fair or olive), whether tall or thin or short or fat. It is important to record if colour blind and those affected, and what sort — blue/green or red/green, etc. Certain conditions can be linked with colour blindness in certain families, such as severe depressive illness, psychoses, familial cancer and pernicious anaemia. For instance, in one family with five generations of manic depressive illness with mood swings, I was able to show the X-linked dominant manic depressive gene was associated with an X-linked B<sub>12</sub> deficiency or pernicious anaemia (later found to be due to allergy

to gluten containing grains) and situated on the X chromosome between the gene site for red/green colour blindness and Xg negative blood group. The depressive illness, etc. responded well to correction of the pernicious anaemia and a gluten free diet. In 1987 both the *Lancet* and *Nature* published articles confirming X-linked manic depressive illness but failed to mention my 1979 publication based on work done in 1973 where I not only showed manic depressive illness can be clearly X-linked but showed the first *correctable* X-linked factor associated with the illness.

Where the medical condition is not known, it is important to describe the patient or relative, and list the main symptoms and signs. Once this has been done, the alert family doctor or specialist will know at a glance what the medical condition is called, especially those trained in Orthomolecular medicine, psychiatry and clinical nutrition.

For example, if you had an aunt who was depressed, became very forgetful, was overweight, sluggish, had coarse facial features, was going deaf, losing her hair, developing wrist pains, had a sallow complexion, then the medical condition would most likely be an underactive thyroid, which responds well to treatment. Or suppose grandmother much of her life had migraine, arthritis, mouth ulcers, cold sores, hair loss, recurrent infection, was allergic to penicillin/sulphonamides, burnt easily in the sun, i.e. was photosensitive and suffered from severe depression, lassitude, appeared psychotic or schizophrenic, and later had dementia or a stroke or a heart attack, or even cancer, then it is highly likely she had undetected systemic lupus erythematosus (S.L.E.) for many years. Another example would be if the mother with a broad forehead, blue eyes, fair complexion, fair hair and premature greying, white spots on the forearm (vitiligo) and sallow complexion, later appeared depressed, psychotic or confused, then pernicious anaemia (low B<sub>12</sub>) would immediately spring to mind, and if she had a deep midline fissured tongue, then pellagra/low B<sub>3</sub> as well. It must be stressed that seriously low levels of B<sub>12</sub> or B<sub>3</sub> can occur in the blood or brain without anaemia or big red cells or macrocytes in the

blood. If a relative has the picture mentioned serum B<sub>3</sub> and B<sub>12</sub> should be measured regardless of the blood film. It must be remembered also that low iron or B<sub>6</sub> normally make red cells smaller than usual or results in microcytosis. Thus the *Macrocytosis* or bigger than usual red cells of low B<sub>12</sub> B<sub>1</sub>, B<sub>3</sub>, folic acid, Vitamin C, manganese and thyroxine can be masked by low iron or B<sub>6</sub> and the blood film looks normal with no macrocytes present to make the doctor suspicious of low B<sub>12</sub>/per-nicious anaemia, etc. If in doubt, vitamin and mineral assays must be done, especially with multiple sclerosis where a patient can be going blind or paralyzed before the blood film shows anaemia or macrocytosis. The same applies with psychoses, depressive illness and dementias.

Another medical condition extremely common and unnamed in the family tree so often is coeliac disease, and should the bowel biopsy be normal as it commonly is, then it is called wheat/grain (gluten, alpha-gliadin) sensitivity or intolerance or latent coeliac disease. Gluten and alpha-gliadin are very toxic fractions of most grains except rice. Here the relative has auburn or golden hair, pale freckled face, or black hair that goes steel grey early, or a middle aged person who has white hair early, especially if osteoarthritis is associated, bowel upsets/wind and many physical problems with risk for psychosis and/or depression, and later fissured tongue and picture of Alzheimer's disease or presenile dementia.

A deep midline fissured tongue like an erosion gully indicates the patient is at risk for low B<sub>3</sub>/niacin or pellagra, I believe, due mainly to low amino acid L-tryptophan especially associated with a genetic tendency to allergy/intolerance to gluten containing grains as in coeliac disease. This is especially so of the scrotal tongue or very fissured tongue of Down's Syndrome patients and very fissured tongue of any one over 40 who is starting to dement. The fissures in the tongue can be healed with adequate B<sub>3</sub>, L-tryptophan and amino acid complex supplements remembering L-tryptophan needs coenzymes/vitamins B<sub>1</sub>, B<sub>2</sub>, B<sub>6</sub>, C and cofactors/minerals magnesium, zinc, and manganese to convert it to B<sub>3</sub> or niacin.

However, early greying is also seen in

pernicious anaemia, systemic lupus and many other conditions as well as coeliac disease. Vitamin C and folic acid, as well as B-group vitamins and zinc help prevent it. Early greying is usually associated with vitamin, mineral (especially zinc) and certain amino acid deficiencies. For instance, over 12 years ago while travelling overseas, I went off vitamins and minerals for three weeks and was rapidly going grey above the ears. This rapidly reversed when back on supplements.

The next things to include on the family tree to help prevent, predict, diagnose and treat serious illnesses are the following — congenital and chromosomal abnormalities and certain major psychiatric illnesses such as schizophrenia/psychoses, manic depressive illness, chronic lassitude, weakness, dementia, severe anxiety states including obsessional/phobic conditions and learning/behavioural/sleep and other disorders in children.

Not generally known is the relationship between Alzheimer's disease (presenile dementia where people as early as in their 40's and 50's become confused, suffering severe memory impairment and usually needing constant supervision for the rest of their lives), Down's syndrome, lymphoma or bowel cancers and leukaemia, and all of these can occur in one family; often with schizophrenia an additional disorder not mentioned in the literature. Down's syndrome patients also have a high risk of leukaemia and virtually all go on to a picture similar to Alzheimer's disease. From my research, all of these conditions stem from coeliac disease or wheat/grain allergies with severe malabsorption for vitamins, minerals, amino acids, etc., and are also associated with a build up of toxic metals such as aluminum in the brain. Thus, in my experience, Alzheimer's disease patients tend to have missed low Vitamins B<sub>1</sub>, B<sub>3</sub>, B<sub>12</sub>, folic acid and zinc (and each can cause dementia per se), have toxic levels of aluminum, cadmium, lead, etc., are gluten/alpha-gliadin sensitive, have missed systemic lupus and tend to have low amino acid tryptophan, and other amino acids, and most could do with more thyroxine (thyroid hormone). They also are allergic to milk fractions as

well as grain fractions, and have other food allergies. At times they have a simian palmar crease or Sydney line.

I now look at the palmar creases in each new patient (and relatives, if present), especially the middle palmar crease to see if it continues right across the hand (Sydney line), or one crease across the hand replaces two creases — simian palmar crease, and whether there are a lot of extra lines in the thenar area more normally clear at the base of the thumb and extra lines in the hypothenar area along the inside of the hand which more normally is clear suggesting coeliac disease or allergy/intolerance to glycoprotein fractions of gluten containing grains other than gluten of alpha-gliadin. Usually extra creases or abnormal palmar creases suggest a more stormy time in utero as the palmar creases are developing in the first trimester of pregnancy.

I hope to carry out a study eventually where the abnormal creases of Down's Syndrome are compared with those of the parents, looking at where the extra chromosome comes from, where one parent has Sydney lines or simian palmar creases or extra creases like the Down's Syndrome child and one parent does not.

If the extra chromosome comes usually from that parent with the abnormal palmar creases in a high percentage of cases then nutritional/allergic/metabolic disturbances in that parent could be looked at and corrected before another child is conceived, thus preventing Down's Syndrome in future children. Also, knowing what metabolic/allergic problems are present in the parent, where the extra chromosome comes from will help unravel the causes of Down's Syndrome.

I suspect missed/untreated coeliac disease and/or systemic lupus in either the mother or the father just prior to conception as being a very high risk factor for Down's Syndrome conception or miscarriage later. I am hoping some research worker will look for coeliac disease and associated abnormal immunology in Down's Syndrome children and their parents and show that when the DS child has the immunology of coeliac disease and one parent has similar immunology which the other does not have, then the extra

chromosome usually comes from the parent with the immunology usually seen with coeliac disease. This would suggest coeliac disease and its vitamin/mineral/ amino acid deficiencies, etc. and tendency to low Cortisol just prior to conception is the major cause of Down's Syndrome rather than having an extra gene to form amyloid, etc. I now also encourage the recording of palmar creases on the family tree to help future generations understand what is associated with abnormal palmar creases so the problems can be corrected in the future. Thus the clinician trained in applied nutrition, Orthomolecular psychiatry (defined in *Journal of Orthomolecular Psychiatry*, Vol. 10, No. 1, 1981, p. 29) will look at the ill patient and the family tree, and be almost certain what the patient has inherited, where it has come from, and who is at risk for it, and what to do about it in terms of special tests and investigations. However, to further help in the diagnosis, treatment, prediction and prevention of illnesses in the family, I encourage an additional genetic approach. Here, again, the family tree can give valuable answers or clues to which investigations must be done.

The next step, then, is to record food allergies/intolerances, hypersensitivities and allergies to drugs or chemicals. Hereditary food allergies cause most of the major illnesses known to mankind. I will repeat that — hereditary food allergies cause most of the major illnesses known to mankind, because once allergic to wheat or grains, cow's milk, soya beans or legumes, etc., they severely damage the digestive system and result in malabsorption of essential vitamins, minerals and amino acids, resulting in serious conditions such as pernicious anaemia due to low B<sub>12</sub> and pellagra due to low B<sub>3</sub>. This is a very serious condition indeed, and endemic in the Australian population and not being detected because B<sub>3</sub> is not measured, except by one or two laboratories. Other very common vitamin deficiencies are B<sub>6</sub>, B<sub>1</sub>, E, A, folic acid and less often B<sub>2</sub>, B<sub>5</sub> and biotin.

In the last 5000 patients I have assayed, vitamin and mineral deficiencies are extremely common, despite what dieticians call a well balanced or healthy diet and

despite supplements; and often supplements many times the normal recommended daily allowances are not enough to maintain normal blood levels.

As far as B<sub>3</sub> is concerned, I have seen patients maintained on 850 mg per day, another on 1000 mg per day and the record in my practice is for two patients on 1800 mg per day of B<sub>3</sub> and yet still had low B<sub>3</sub> levels in the blood. One schizophrenic on over three grams per day was borderline low when assayed.

With B<sub>6</sub>, one patient was on 1600 mg per day and still low in B<sub>6</sub>. This is rare and very few patients need more than between 50-750 mg of B<sub>6</sub> to have a normal blood level. One patient on 1000 mg per day of Vitamin E was still low when measured.

I have seen a cancer patient on 40 grams per day of Vitamin C with borderline low Vitamin C level when tested the next day. With B<sub>1</sub>, some patients are low despite 400-500 mg per day. Even fat soluble Vitamin A can be low in some patients maintained on over 20,000 IU per day. One patient with breast cancer was borderline low in Vitamin A while on 100,000 IU per day. All of these patients had severe food allergies, like virtually most patients with serious illnesses.

We definitely are not what we eat but what we are able to absorb and utilize. Nowadays the old adage dating to Roman times "one man's meat is another man's poison" takes on a new significance when we consider that it means food allergies or malabsorption or essential vitamins, minerals and amino acids, or damage to vulnerable tissues or organs and vessels, or suppressed immune system/autoimmune disease/increased infections/cancer risk, etc.

The recommended daily allowance (RDA) is highly misleading, outdated and a gross underestimate to the detriment of patients suffering from Down's Syndrome, systemic lupus, schizophrenia, multiple sclerosis, depressive illnesses, anxiety states, dementia, osteoarthritis and other serious connective tissue disorders, chronic degenerative states and cancer. The RDA's for pathological states have never been recorded and the RDA is for a normal or average person, whatever that means. Thus the final thing to record on the family tree,

after food allergies and their effects, is evidence of hereditary vitamin or mineral deficiencies usually associated with the hereditary food allergies just mentioned. Here it is important to record premature greying or date when greying became noticeable, where known. Also such signs as cracked lips, especially at the corner of the mouth, suggesting not enough B<sub>2</sub> plus or minus Vitamin A, plus or minus zinc and such things as a shiny red tongue, cf. iron and B<sub>12</sub> deficiency. Such small signs as white dots in nails are often associated with not enough B<sub>6</sub>, zinc and tryptophan and with Kryptopyrrole in the urine locking away B<sub>6</sub> and zinc, a condition called pyrolleuria which is well described by Dr. Carl Pfeiffer and Donald McCabe. Schizoaffectives are very much at risk for it.

A schizoaffective is a schizophrenic with marked tendency to depression, or an atypical manic depressive where the patient appears schizophrenic at times. It is usually treated with both tranquilizers and antidepressants until the real causes such as pyrolleuria, plus or minus systemic lupus, plus or minus pellagra, plus or minus coeliac disease, plus or minus pernicious anaemia, low folic acid, B<sub>1</sub>, B<sub>6</sub>, zinc, etc. and food allergies, hypoglycaemia, etc., are diagnosed and corrected. When this happens, medication may not be required at all, or very much less — reducing risk of tardive dyskinesia, etc.

One patient presenting with tardive dyskinesia was low in B<sub>1</sub>, B<sub>6</sub>, B<sub>12</sub> and borderline low in Vitamin C, E, A, folic acid, copper, magnesium, calcium and phosphate.

The condition soon remitted with correction of these deficiencies. I did not see him for several years until he again presented with severe tardive dyskinesia which did not cease off Modecate.

This time he was low in B<sub>6</sub>, B<sub>12</sub>, copper and borderline low in B<sub>2</sub>, B<sub>3</sub>, Vitamin C, A, folic acid, magnesium, iron and phosphate.

Again he rapidly went into remission after correction of deficiencies. Vitamin and mineral deficiencies should be looked at and corrected where tardive dyskinesia has occurred and the earlier this is done, the better is the prognosis.

With the above patient, low B<sub>6</sub> and B<sub>12</sub>

appear to be the most significant deficiencies and should be looked for in tardive dyskinesia remembering if both are low, neither microcytosis of low B<sub>6</sub> will show up on blood film nor macrocytosis of low B<sub>12</sub> since both mask each other; low B<sub>6</sub> making red cells smaller than usual and low B<sub>12</sub> making red cells larger than normal usually so the red cells remain normal size thus often masking these deficiencies.

Finally I would like to stress, Orthomolecular genetics is definitely to help with the prevention of such serious illnesses as cancer, dementia, Down's Syndrome, and hopefully, chromosomal changes in dividing cells that would make them cancerous, for preventing heart attacks, thrombosis, strokes, schizophrenia/psychoses, depressive illness, chronic lassitude/weakness arthritic conditions, childhood disorders and a host of other conditions so commonly running in most families. Recording details of nutritional or allergic disturbances in family trees will open new avenues of research, and hopefully, cures eventually, for hitherto idiopathic illness, that is, illnesses where they neither know the cause of the illness, or worse, how to treat it.

Can I encourage a separate family tree of pathology results, especially where various illnesses present such as multiple sclerosis, cancer, ALS or motor neurone disease or Alzheimer's disease until eventually it is known exactly what metabolic/ hormonal/allergic/toxic fractions are causing these illnesses or associated with their presentation. Eventually common aetiological factors will emerge, especially with familial conditions. Keeping this data will eventually show up significant recurrent abnormal pathology responsible for the development of these illnesses. Each ill patient can help their family and future generations by recording their pathology results with dates and age at time of illness as well as symptoms and signs at that time.

At this stage I would like to report on some new findings of my research. The autosomal dominant condition of neurofibromatosis or Von Recklinghausen's disease can be very severe, as in the case of the Elephant Man. It presents with

neurofibroma tumours on the nerve endings and surrounding tissues. These are associated with anti-nerve antibodies and anti-collagen antibodies. I have been able to get the anti-nerve antibodies and anti-collagen antibodies to normalize with food allergy free diets and relevant vitamins and minerals. This suggests that the progressive condition may respond to nutritional intervention. Also the bone thickening as with Paget's disease appears to be associated with synovial membrane antibodies, and the titres fall when citrus and/or solanaceae are removed from the diet.

The condition rheumatoid arthritis, also with synovial membrane antibodies is helped greatly off citrus and solanaceae, plus or minus apples, plums and nectarines/stone fruit, plus or minus lamb and pork, plus or minus legumes/beans, tea and coffee, plus or minus all fruit rarely. R.S.I, also can be helped with a similar regime.

In osteoarthritis with cartilage antibodies, the patient is allergic to cow's milk/grains, usually associated with gluten and alpha-gliadin antibodies of wheat and certain grains and alpha-casein, al-pha-lactalbunin, Beta-lactoglobulin antibodies of cow's milk. However, the exact fractions of milk or grains have not been identified yet, since some patients have cartilage antibodies positive of osteoarthritis, milk/grain allergies, but all the antibodies to milk/grain fractions I have mentioned were negative. Thus there must be some other fractions of wheat, such as Beta gamma or omega gliadin that correlates every time with cartilage antibodies, and some other fraction of cow's milk such as beta or kappa casein which causes cartilage antibodies in 100% of osteoarthritic patients.

In pernicious anaemia with low B<sub>12</sub>, parietal, or stomach cell antibodies and intrinsic factor antibodies making intrinsic factor useless to latch on to B<sub>12</sub> to form a complex for absorption in the terminal ileum — the damage is usually caused by milk fractions, not grain fractions such as gluten or alpha-gliadin. The highest titres/ values for intrinsic factor antibodies are associated with allergy/hypersensitivity/ intolerance to toxic fractions of cow's milk such as alpha-casein, alpha-lactalbunin or beta-lactoglobulin.

Thus pernicious anaemia, apart from meaning low B<sub>12</sub>, is mainly due to a genetic tendency (often X-linked) to lack of certain pancreatic enzymes, most likely exopeptidases to break down toxic or immunogenic fractions of cow's milk to safe or tolerogenic fractions that don't damage the stomach cells or intrinsic factor, and so allow the normally toxic milk peptides to be broken down into safe amino acids.

The treatment for pernicious anaemia, in future, will not be intra-muscular B<sub>12</sub> for the rest of life, but milk/grain free diet, plus relevant digestive enzymes, etc. to clear up the gastritis, etc.

In familial depression with poor memory or concentration, especially where the patient has white dots in the nails, midline fissured tongue, rarely dreams, can't recall what the dreams are about or dreams are in black and white and usually unpleasant — the following is highly likely: — The patient is low in serotonin in the brain because he is low in B<sub>6</sub>, plus or minus magnesium, zinc, manganese needed to convert the amino acid tryptophan to serotonin in the brain.

Or, if low in B<sub>6</sub>, can't form another eight brain amines/neurotransmitters as well as serotonin to not be depressed — hence nine brain amines can be low with low B<sub>6</sub> and the patient remains depressed. Serotonin is usually low in the brain for dreaming pleasant, coloured dreams due to low serum tryptophan.

Tryptophan is low in gluten/alpha-gliadin or grain sensitive patients as they tend to lack trypsin, chymotrypsin and certain pancreatic exopeptidases to release tryptophan free from ingested protein. Hence low B<sub>3</sub> as well, and fissured tongue, and low picolinic acid synthesis, and can't absorb zinc efficiently, and white dots appear in the nails together with low B<sub>6</sub> and zinc.

Thus the melancholic in Greek history with 'black bile', black moods, black skin, etc. and I suspect increased pigmentation of pellagra, and photosensitivity, and black dreams or no dreams, was shunned in the past because he couldn't discuss his pretty coloured dreams with his friends. However, nowadays L-tryptophan, B<sub>1</sub>, B<sub>2</sub>, B<sub>6</sub>, Vitamin C, manganese, zinc, magnesium,

will help him to form B<sub>3</sub> and serotonin so he can have beautiful coloured, even sexy dreams, and wake up with a smile on his face ready to start the day with plenty of the relevant neurotransmitters in his limbic system and pleasure centres of his brain, good dream recall and plenty of dreams to discuss with his friends.

Finally, in my experience, especially over the last five years, I would like to suggest the following conditions and variations or combinations of them, are causing most familial conditions including cancer, arthritis, psychiatric illnesses, especially those affecting the brain and blood vessels. They are as follows:

1. Collagen/connective tissue disorders such as system lupus from early forms requiring special tests and skin biopsy (with immunofluorescence technique on unexposed skin) to diagnose, through to severe systemic lupus presenting as frank dementia, psychosis, paralysis, stroke or severe depressive illness with an M.E. like picture.
2. Severe food allergies to wheat/grains and such fractions as gluten and alpha-gliadin.
3. Severe food allergies to cow's milk, including such fractions as alpha-casein, alpha-lactalbumin, beta-lacto-globulin, and associated with allergies to albumin and globulin fractions of eggs and beef.
4. Pyroluria or Kryptopyrrole in the urine locking away B<sub>6</sub> and zinc, and a precursor of coproporphyrin raised in the urine in acute intermittent porphyria. This can cause severe psychosis and neurological symptoms as in King George III and his relatives. Pyroluria, you will recall, is associated with white dots in nails and also a tendency to china doll complexion, headaches, poor dream recall, severe inner tension, depression, and schizophrenic symptoms, if severe. It is also associated with allergies to milk, plus or minus eggs, plus or minus beef, plus or minus yeast, and is commonly associated with collagen/connective tissue disorder and low IgA immunoglobulins.
5. Undiagnosed pellagra — low B<sub>3</sub>, with fissured tongue, etc. I have detected



- over 275 cases now.
6. Other food allergies, together with allergies to milk and grains, and especially soybeans/legumes, resulting in severe malabsorption for essential vitamins and minerals, and raised levels of cadmium aluminum, copper, mercury, lead, in vulnerable tissues such as the brain. Also food allergies are associated with lack of certain digestive enzymes and low amino acids, especially in coeliac disease, etc. Associated with allergies to foods are allergies to preservatives, additives, dyes and petrochemicals, etc.
  7. Gastritis (or inflammation of the stomach cells) plus or minus thyroiditis (inflammation of the thyroid gland) plus or minus pernicious anaemia with parietal cell antibodies and intrinsic factor antibodies.
  8. Hypoglycaemia or tendency to low blood sugar, tendency to low Cortisol and to have raised levels of certain antibody groups, or types of gamma globulin associated with food allergies, especially IgM or low IgA.
  9. Anaemia due to low B<sub>6</sub> or B<sub>12</sub>, B<sub>3</sub>, B<sub>12</sub>, folic acid, E or copper, rather than iron in many cases.
  10. Low complements or raised complements, which are like antibodies, depending upon what conditions are in the family, and are usually low in milk or grain allergies, systemic lupus and associated with immune complexes.
  11. Antibodies to various tissues and organs, evidence of allergy to fractions of milk and grains.
  12. In arthritic conditions, cartilage antibodies appear in osteoarthritis, particularly due to milk/grain fraction damage, and allergies to cane sugar; and synovial membrane antibodies appear in rheumatoid arthritis, Paget's disease, along with synovitis/RSI, scleroderma, etc., mainly due, I believe, to allergy or intolerances to fractions of the deadly nightshade family or solanaceae, such as tomato, egg plant, tobacco, capsicum/peppers and even potato. Citrus can also be a culprit. In multiple sclerosis I have noted anti-nerve antibodies and antimyelin antibodies and anti-meninges

antibodies in a patient with recurrent meningioma. The meninges antibodies have become negative with food allergy free diet and antioxidants. It is too early to know whether the meningioma will now stop growing. In the condition motor neurone disease, or Amyotrophic lateral sclerosis (ALS), where the patient can lose the ability to talk and swallow (like David Niven, the actor), the last four patients I have seen have all had low B<sub>3</sub>/pellagra, which can cause inability to talk and swallow, and paralysis also. Thus any patient with low B<sub>3</sub> for long enough, could be a candidate for ALS, it seems, as well as dementia later — 2 out of 4 children at risk for ALS already had low B<sub>3</sub> in one family.

In every cancer patient I have looked at, 100% have had food allergies, 100% have had autoimmune disease seen with cow's milk/gluten containing grain allergies and 100% have shown vitamin and mineral deficiencies that could affect PGE1 series synthesis, cAMP or Cortisol levels to maintain normal homeostasis or prevent chromosomal changes or aneuploides or cancer. I suspect there are critical levels for PGE1 series below which people present with cancer.

(i) below a certain critical low level of PGE1 series those at risk for cancer due to transplacental induction or being born with damaged tissues (precancerous) present with cancer in this abnormal tissue.

(ii) another much lower critical level for PGE1 series below which even normal tissue will divide abnormally or undergo chromosomal or malignant changes.

It is well known low vitamins can increase risk of chromosomal changes and chromosomal changes increase risk of cancer. In the aetiology of cancer it has been hard to explain why being just low in Vitamin B<sub>3</sub> or Vitamin C or B<sub>6</sub> has been associated with the presentation of cancer in the susceptible. However, when one considers each can cause low PGE1 series synthesis from gamma-linolenic acid and so can low zinc, magnesium and manganese also necessary for synthesis of PGE1 series — could the common missed factor be very low PGE1 series in each case affecting those at risk with abnormal

tissue due to similar disturbances during differentiation in utero.

Thus I hope in cancer patients in future, levels of PGE2, PGE1 series, cAMP, Cortisol will be studied to ascertain the critical levels below which or beyond which chromosomal changes or cancerous changes take place. Also, which vitamin or mineral deficiencies are associated and why and here I suspect food allergies/malabsorption state are directly or indirectly a major cause of cancer.

To identify the conditions 1-12 I mentioned earlier, I know of no laboratory that can do all the tests. I am very fortunate in Sydney, to have two laboratories that between them do all of these special tests if required.

Ideally, the most useful tests to help most patients with serious medical/psychiatric illness, arthritis, blood vessel disorders, etc., are as follows: —

1. Food allergies (Immediate on RAST and delayed on BCFT or cytotoxic) and inhalant allergies to dust, moulds, etc. where indicated.
2. Antibodies to milk/grain fractions.
3. Vitamin and mineral levels, including toxic metals — in serum and hair analysis. I especially request B<sub>3</sub>, looking for pellagra — also selenium and lately germanium.
4. Antibodies to various tissues, organs and ducts, and in arthritis, also cartilage and synovial membrane antibodies as well as Antinuclear factor.
5. Immunoglobulins or antibody groups.
6. Full blood count/ESR noting size and colour of red cells. Pale and small red cells usually correlate with low B<sub>6</sub> and iron. Big red cells suggest not enough B<sub>1</sub>, B<sub>3</sub>, B<sub>12</sub>, folic acid, Vitamin C, Manganese and underactive thyroid at times.
7. Fasting blood sugar plus or minus Cortisol.
8. Kryptopyrrole in the urine (looking for pyrolleuria).

I rarely measure amino acids, unfortunately, but hope to do so regularly in certain neurological conditions from now on. Correcting these abnormal results, which are present to varying degrees in most seriously ill patients, allows correct treatment.

Relevant vitamins and minerals along with food allergy free diets, are usually sufficient to reverse all the abnormal findings in the tests. That goes for all the autoimmune diseases also. However, far more research needs to be done into the relevance of specific amino acid deficiencies, as well as the tests mentioned in more complex conditions as certain psychiatric illnesses, certainly in cancer and degenerative neurological disorders.

I look forward to the day when all sorts of fractions of foods such as lectins, are included in the delayed food allergy tests/ BCFT/cytotoxic test, and not just whole foods.

I also look forward to the day when antibodies to far more fractions or lectins of wheat and grains become routine testing, looking at antibodies to beta, gamma and omega gliadin, to glutelins, albumin, globulins, exorphins and other prolamine fractions, and lectin fractions and their digests.

The same applies to cow's milk. We should be looking at antibodies to not only alpha-lactalbumin, beta-lactoglobulin and alpha-casein, but also to beta-casein, kappa-casein, other globulins, etc., to find out exactly which fractions are doing the damage to vessels and organs and causing psychiatric symptoms.

In conclusion, very serious illnesses such as cancer, multiple sclerosis, ALS, psychoses, arthritis, will be classified into different types in terms of toxic fractions of foods responsible, by such specialists as Orthomolecular psychiatrists trained in psychoimmunology, clinical ecology and psycholectinology. Hopefully, before long, in cancer, the clinician will know exactly what:

1. toxic fractions of foods are suppressing the immune system and malabsorbing vitamins and minerals, or resulting in low amino acids, essential for the immune system.
2. food fractions or toxic peptides/lectins or exorphins are acting like hormones and growth factors, helping cells proliferate and allowing new blood vessels develop (called angiogenic factors like copper and iron), and here I suspect
3. food fractions/peptides the cancer cell has receptors for, like breast cancer cells

have receptors for oestrogen and progesterone, or both, and are dependent on them, and now lectins also as I mentioned in my book, supported by the reference — Predictive value of lectin binding on breast-cancer recurrence and survival. A. J. Leatham and S. A. Brooks, *The Lancet*. May 9th, 1987, pp 1054-1056.

4. what is the malignant environment in terms of low vitamins and minerals, prostaglandins, cAMP, hormones such as Cortisol and DHEA and amino acids, and what toxic food peptides, and chemicals and hormonal imbalance allows a malignant change in dividing cells to take place.
5. what concentration and type of hormones do we need to infuse into the blood supply of cancer/embryonic tissue or into parenteral infusions — that is — placental hormones/inducers/organizers, commensurate with the size of the cancer so that:
  - (a) cancer cells (the abnormal embryonic tissue) may no longer fear "abortion", and stop proliferating to release specific placental hormones/foetal proteins to maintain it and secondaries,
  - (b) the correct ratios of placental inducer/organizer hormones may hold the cancer cells in check and stop proliferation
  - (c) correct imbalance of placental hormones, inducers/organizers may be given to 'abort' or 'reject' the cancer.
  - (d) remove from the diet the lectins, the cancer cells have receptors for as well as giving the usual sex hormone analogs such as TAMOXIFEN, etc. in breast cancer.
  - (e) give the cancer synthetic isolectins that the cancer cells avidly trap but are useless to it because one or two amino acid sequences are different from the lectin the cancer has receptors for.
  - (f) attach cancer drugs to lectins the cancer cells have receptors for to carry the cytotoxic drugs to just the cancer and secondaries, and not normal tissue without the lectin receptors.
  - (g) restore homeostasis with relevant anti-oxidants i.e. certain vitamins/minerals/amino acids and reduce PGE2 series by avoiding food/chemical allergies, etc.

(h) choose synthetic lectins (tagged with carbon,nitrogen or sulphur isotopes) so that very small amounts will be taken up by the cancerous tissue in the body which has receptors for the lectins showing up the extent of the cancerous tissue on full body scintillograph.

Thus I hope my research and yours, will with time, ensure most serious illnesses recorded on your family tree eventually will become curable, predictable and certainly preventable. Also I would like to stress, the *aim* is not just longevity or rejuvenation with anti-oxidants, etc., but to attain *quality* of life, never forgetting the relevance of adequate exercise, sleep and relaxation, in addition to good nutrition.

### References

1. Reisman RE: American Academy of Allergy: Position Statement — controversial techniques. *J. Allergy, Clinical Immunology* 1981; 67:5, 333-338.
2. Reading CM: Report for N.S.W. Cancer Council for year ended 30th June, 1974; 89:37.
3. Reading CM: Latent Pernicious Anaemia: A Preliminary Study. *Med. J. Aust.* 1975; 1:91.
4. Reading CM: Latent Pernicious Anaemia. *Med. J. Aust.* 1975; 1:430.
5. Reading CM: Latent Pernicious Anaemia. *Med. J. Aust.* 1975; 2:111.
6. Reading CM: Psychosurgery, Orthomolecular Psychiatry and the Press. *Med. J. Aust.* 1977; 1:642.
7. Reading CM: Down's Syndrome, leukemia and maternal thiamine deficiency. *Med. J. Aust.* 1976; 12:505.
8. Reading CM, McLeay AC, Nobile S: Down's Syndrome and Thiamine Deficiency. *J. Orthomolecular Psychiatry* 1979; 8:1, 4-12.
9. Reading CM: X-linked dominant manic depressive illness: Linkage with Xg blood group, red-green colour blindness and Vitamin B12 deficiency. *J. Orthomolecular Psychiatry* 1979; 8:2, 68-77.
10. Reading CM: Orthomolecular Psychiatry. *Med. J. Aust.* 1979; 2:40.
11. Reading CM: Multiple Sclerosis: Is it transplacentally induced. *Med. Hypothesis* 1979; 5:11, 1251-1255.
12. Reading CM: Klinefelter's Syndrome and biotin deficiency. *Med. Hypothesis* 1981; 7:1105-1108.

13. Reading CM: Address on Orthomolecular Medicine/Psychiatry to Medical Benefits Schedule Revision Committee, Dec. 3rd, 1980. /. *Orthomolecular Psychiatry* 1981; 10:1,29-34.
14. Reading CM: Trace element metabolism on man and animal. Discussions (Howell J, Gawthorne J, White CL, eds. - Canberra Aust. Academy of Science) 1981; 407, 538, 603.
15. Reading CM: Relevance of cytotoxic test to detect food allergies/intolerance/hypersensitivity in psychiatric patients. *Schizophrenia Assoc. of Great Britain Conf.* 22.4.1982. Recent Trends in Biological Psychiatry.
16. Reading CM: Letter to Editor, /. *Orthomolecular Psychiatry* 1982; 11:2, 111-115.
17. Reading CM: Letter to Editor, /. *Orthomolecular Psychiatry* 1982; 11:4, 276.
18. Reading CM: Systemic Lupus Erythematosus: Nutritional Intervention. Address to 13th Annual Conference, McCarrison Society 23.8.1983, Nutrition and Mental Health; Links between food and behaviour. 1983.
19. Reading CM: Down's Syndrome; Nutritional Intervention. Address to 13th Annual Conference, McCarrison Society 23.8.1983. Nutritional and Mental Health; Links between food and behaviour, 1983. /. *Nutrition and Health* 1984;3:90-111.
20. Reading CM: Orthomolecular Psychiatry/Medicine. *Med. J. Aust.* 1984; 1:746.
21. Reading CM: Down's Syndrome; Is gluten/ alpha-gliadin sensitivity/coeliac disease the cause. /. *Biosocial Research* 1984; 6:62-65.
22. Meillon RS, Reading CM: Relatively Speaking; Family Tree Way to Better Health. Fontana Australia, Sydney, NSW, Australia 1984. (Now Family Tree Connection. Keats Publishing.)
23. Reading CM: Dietary Intervention in Systemic Lupus Erythematosus: 4 Cases of Clinical Remission and Reversal of Abnormal Pathology. *International Clinical Nutrition Review* 1985; 5:4, 166-176.
24. Reading CM: Address to Annual General Meeting of Down's Syndrome Association of Queensland, 26.3.1986. Dietary Intervention in Down's Syndrome: and a unifying theory as to its causation, prevention and treatment.
25. Reading CM: Address to Australasian College of Biomedical Scientists, Leura, N.S.W., 20.9.86. Orthomolecular Genetics/Medical Genealogy.