

THE MINERAL DEPLETION OF FOODS AVAILABLE TO US AS A NATION (1940–2002) – A Review of the 6th Edition of McCance and Widdowson*

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“A knowledge of the chemical composition of foods is the first essential in the dietary treatment of disease or in any quantitative study of human nutrition”

ABSTRACT

Over the past 60 years there have been fundamental changes in the quality and quantity of food available to us as a nation. The character, growing method, preparation, source and ultimate presentation of basic staples have changed significantly to the extent that trace elements and micronutrient contents have been severely depleted. This trend, established in a review of the 5th Edition of McCance & Widdowson's *The Composition of Foods*, is still apparent in this review of the 6th edition of the same work. Concurrently there has been a precipitous change towards convenience and pre-prepared foods containing saturated fats, highly processed meats and refined carbohydrates, often devoid of vital micronutrients yet packed with a cocktail of chemical additives including colourings, flavourings and preservatives. It is proposed that these changes are significant contributors to rising levels of diet-induced ill health. Ongoing research clearly demonstrates a significant relationship between deficiencies in micronutrients and physical and mental ill health.

Key Words: essential minerals, micronutrients, trace minerals, health, mental

*The data used as the basis for this study was published in six editions, initially under the auspices of the Medical Research Council and later the Ministry of Agriculture Fisheries and Foods, the Foods Standards Agency and the Royal Society of Chemistry: Authors R.A. McCance and E.M. Widdowson.

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An essential nutrient is one that must be obtained from the diet because the human body cannot make it in sufficient quantity, or at all, to meet its needs for normal human metabolism and reproduction: most commonly some amino acids, fats, vitamins and minerals.

Micro-nutrients and trace elements are required in ‘minute’ amounts – such as some vitamins, and minerals eg selenium (up to 200mcg/d) rather than in larger quantities, like the essential mineral calcium (up to 1.5g/d), whereas selenium in larger quantities becomes toxic. Some 90 or so minerals in the soil are essential.

INTRODUCTION

In a paper ‘A Study of the Mineral Depletion of the Foods available to us as a Nation over the period 1940 to 1991’¹ published in *Nutrition and Health* in 2003, I compared and contrasted the statistics of the essential mineral and trace element contents of foods presented in the 1st to the 5th editions of McCance & Widdowsons’ ‘The Composition of Foods’,^{2,3,4,5,6}. In the 6th edition⁷, published 2002, new analytical data have been incorporated in the ‘Milk and Milk Products’ and ‘Meat and Meat Products’ sections. This paper provides an updated look at these data with specific reference to micronutrients.

In the current paper I also provide a response to certain critical reviews of my earlier work.

It is important to note that McCance & Widdowson provide the most detailed and sophisticated historical records of the nutrient values of foods available to any nation worldwide. Currently there is a pan-european⁸ research project that will provide nutritional information on the chemical composition of foods within the EU. There is a danger that the data from this research will be seen as the benchmark against which future studies will be measured. McCance & Widdowson show that, in the UK at least, there has been a considerable depletion in minerals and trace elements in foodstuffs during the period 1940–2002.

Since the publication of my initial paper, concern and debate regarding the relationship between the quality of the food available to us and the health of the nation has gained momentum and a large number of research papers have been published detailing the close relationship between good nutrition and mental well-being. In January 2006 the McCarrison Society organized a conference entitled ‘Generating Healthy Brains’ the aim of which was to integrate the results of different fields of research relating nutrition to mental health. The current paper was presented to that conference. At the same time Sustain published their book – on a similar theme – ‘Changing Diets, Changing Minds: how food affects mental well being and behaviour’.⁹

Refer to Appendix 1 for the specific changes 14 Meat cuts and products, Appendix 2 for the changes in 9 Cheeses and to Appendix 3 for the changes in 4 Dairy products.

Review of selected data from the 6th Edition

TABLE 1

Beef – topside roast: trace elements in mg per 100gm 1940 to 2002

	1940	1978	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	76	48		62	-37	29	-18
Potassium	370	350		410	-5	17	11
Phosphorous	286	200		230	-30	15	-20
Magnesium	28	23		27	-18	17	-4
Calcium	6.2	6		8	-3	33	29
Iron	4.7	2.6		2.9	-45	12	-38
Copper	0.25	0.13	0.14	0.04	-48	-69	-84
Zinc		4.9	5.5	6.5		33	
Water (gm/100gm)	56.2			56.9			1

Table 1. This table illustrates the significant decline in essential minerals between 1940 and 2002. Closer inspection reveals very significant decreases in Na, K, P, Mg, Fe and Cu in roast topside from 1940 to 1978. This trend reverses sometime between 1978 and 2002 with the exception of copper which has virtually disappeared by 2002.

TABLE 2

Beef – rump steak: trace elements in mg per 100gm 1940 to 2002

	1940	1978	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	80		54	71	-33	31	-11
Potassium	371		360	360	-3	0	-3
Phosphorous	257		220	220	-14	0	-14
Magnesium	25		24	23	-4	-4	-8
Calcium	5.2		7	5	35	-29	-4
Iron	6		3.2	3.7	-47	8	-38
Copper			0.15	0.02		-87	
Zinc			5.3	4.7		-13	
Water (gm/100gm)	56.9			57.2			1

Table 2. This table illustrates the significant decline in essential minerals between 1940 and 2002. Closer inspection reveals a similar picture to Table 1. With the exception of Mg and Ca, trace element contents in rump steak decline significantly until at least 1991. Thereafter, Na, K, Mg and Fe stabilise or even increase. On the other hand Ca and Zn continue to decline and Cu all but disappears completely.

TABLE 3

Roast Chicken: trace elements in mg per 100gm 1940 to 2002

	1940	1978	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	80		81	100	1	23	25
Potassium	355		310	300	-13	-3	-15
Phosphorous	271		210	200	-23	-5	-26
Magnesium	23		24	23	4	-4	0
Calcium	14.5		9	17	-38	89	17
Iron	2.6		0.8	0.8	-69	0	-69
Copper			0.12	0.08	-48	-33	0
Zinc			1.5	2.7		80	
Water (gm/100gm)	61.1			65.3			7

Table 3. This table illustrates the significant decline in essential minerals between 1940 and 2002. Closer inspection reveals K, P, Ca and Fe all decline significantly between 1940 and 1991. There is little change in Na and Ca contents. Between 1991 and 2002 trace elements either increase or are stable. No analyses were made for Cu and Zn in 1940 so these are not compared.

TABLE 4

Roast Turkey: trace elements in mg per 100gm 1940 to 2002

	1940	1978	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	130		57	90	-56	58	-31
Potassium	367		310	350	-16	13	-5
Phosphorous	320		220	260	-31	18	-19
Magnesium	28.2		27	27	-4	0	-4
Calcium	38.3		9	11	-76	22	-71
Iron	3.8		0.9	0.8	-76	-11	-79
Copper			0.15	0.09		-40	
Zinc			2.4	2.5		4	
Water (gm/100gm)	59			64.6			1

Table 4. This table illustrates the significant decline in essential minerals between 1940 and 2002. Closer inspection reveals there is a marked decline in all elements (Cu and Zn excluded) from 1940 to 1991. Interestingly, as in the beef analyses above, this trend is reversed thereafter with the exception of Cu and Fe which both decline significantly up to 2002

TABLE 5

Back Bacon – fried: trace elements in mg per 100gm 1940 to 2002

	1940	1978	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium							
Potassium	517	300		360	-42	20	-30
Phosphorous	229	170		180	-26	6	-21
Magnesium	25.7	20		21	-22	5	-18
Calcium	11.5	13		6	13	-54	-48
Iron	2.8	1.3		0.6	-54	-54	-79
Copper		0.12	0.12	0.06	0	-50	
Zinc		2.6		1.9		-27	
Water (gm/100gm)	12.7			49.7			291

Table 5. This table illustrates the significant decline in essential minerals between 1940 and 2002. Closer inspection reveals similar changes as the previous tables. With the exception of Ca, essential mineral contents in back bacon decline significantly until 1978. Thereafter, there is a significant increase in K whilst and P and Mg stabilise. Ca, Fe, Cu and Zn continue a marked decline. Another significant historical change is the increase in water content by nearly 300% between 1940 and 2002.

TABLE 6

Cheddar Cheese: trace elements in mg per 100gm 1940 to 2002

	1940	1978	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	612	610	670	723	9	8	18
Potassium	116	120	77	75	-34	-3	-35
Phosphorous	545	520	490	505	-10	3	-18
Magnesium	46.9	25	25	29	-47	16	-38
Calcium	810	800	720	739	-11	3	-10
Iron	0.57	0.4	0.3	0.3	-47	0	-47
Copper	0.04	0.03	0.03	0.03	-33	0	-33
Zinc		4	2.3	4.1		78	
Water (gm/100gm)	37		36	36.6	3	2	1

Table 6. This table illustrates the significant decline in all essential minerals except Na between 1940 and 2002. Closer inspection reveals very significant decreases in K, Mg, Fe and Cu from 1940 to 1991. This trend reverses sometime between 1991 and 2002 with the content of the majority of essential minerals stabilising or increasing (Zn).

TABLE 7

Parmesan cheese: trace elements in mg per 100gm 1940 to 2002

	1940	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	755	1090	756	44	-31	0
Potassium	153	110	51	-28	-54	-68
Phosphorous	772	810	267	5	-67	-65
Magnesium	49.6	45	15	-9	-67	-70
Calcium	1220	1200	362	-2	-70	-70
Iron	0.37	0.11	0	-70	all gone	all gone
Copper	0.38	0.33	0	-13	all gone	all gone
Zinc		5.3	2.7		-49	
Water (gm/100gm)	28	18.4	27.6	-34	33	0

Table 7. This table illustrates the significant decline in all essential minerals except Na between 1940 and 2002. Closer inspection reveals very significant decreases in Fe between 1940 and 1991. Unlike Cheddar this trend continues between 1991 and 2002 with the content of Na, K, P, Mg, Ca significantly decreasing and all the Fe and Cu disappearing.

TABLE 8

Whole Milk: trace elements in mg per 100gm 1940 to 2002

	1940	1991	2002	1940 to 1991 % change	1991 to 2002 % change	1940 to 2002 % change
Sodium	50	55	43	10	-15	-14
Potassium	160	140	155	-12	11	-3
Phosphorous	95	92	93	3	1	-2
Magnesium	14	11	11	-21	0	-21
Calcium	120	115	118	-4	3	-2
Iron	0.08	0.05	0.03	-38	-40	-63
Copper	0.02	0	0	all gone	0	all gone
Zinc		0.4	0.4		0	

Table 8. This table illustrates a decline in all essential minerals between 1940 and 2002. Closer inspection reveals very significant decreases in Fe between 1940 and 1991. A trend which continues to 2002.

TABLE 9 Summary of results
 Historical essential mineral depletion – changes in 5 categories of food products

	1940 to 1991 Vegetables (n = 28)	1940 to 1991 Fruit (n = 17)	1940 to 2002 Meat (n = 14)	1940 to 2002 Cheeses (n = 9)	1940 to 2002 Dairy (n = 4)	Weighted Average (n = 72)
Sodium	-49%	-29%	-24%	-9%	-47%	-34%
Potassium	-16%	-19%	-9%	-19%	-7%	-15%
Phosphorous	9%	2%	-21%	-8%	34%	1%
Magnesium	-24%	-16%	-15%	-26%	-1%	-19%
Calcium	-46%	-16%	-29%	-15%	4%	-29%
Iron	-27%	-24%	-50%	-53%	-83%	-37%
Copper	-76%	-20%	-55%	-91%	-97%	-62%

Table 9 illustrates the weighted average changes that have taken place between 1940 and 1991 for fruit and vegetables and between 1940/1960 and 2002 for meat and dairy products. These represent the average changes in 72 food products. The results are stark. They speak for themselves. There has been a 19% loss in Magnesium, a 29% loss in Calcium, a 37% loss in Iron and a really alarming 62% loss in Copper.

There were no changes in the analytical data for fruit and vegetables presented in the 6th edition (2002) from the 5th Edition (1991) but for completeness sake, and to further illustrate the decline in the essential mineral content of all our foodstuffs over time, the data are included in an overall summary presented in Table 9. In ‘appreciating’ the declines that have taken place it is worthwhile being reminded of the reason why the ‘only’ trace elements that were analysed for in 1940 were iron and copper. This was because at that time they were the only ones known to be essential (iron in 17th Century and copper in 1928). Since then, of course, the physiological and psychological significance of many others – including Zn, Cr, Co, Se, Mo, Mn – has been proven and others such as B, V, Si, etc have been recognised.

DISCUSSION

1. Issues from the previous paper

Before discussing the significance of the data reviewed above I would like to respond to a number of criticisms leveled at my previous paper.

It has been suggested that my conclusions were invalid as analytical methods have altered over the period reviewed. Advances in analytical methods have undoubtedly occurred but I can do no better than directly quote McCance & Widdowson in their foreword to the 5th and 6th editions: **“Those methods (of 62 years ago) were no less accurate than the modern automated ones, but they took a much longer time”**.

A second criticism is that over the period reviewed, crop and animal varieties have changed to such a degree that ready comparisons are meaningless. There is no doubt that very real changes have occurred and will continue to occur particularly if GM crop varieties are introduced. Furthermore, reduced times for fruit and vegetable ‘on the plant ripening’ and transit are likely to have affected nutrient contents. Similarly in the meat sector two significant changes have taken place. Crawford¹⁰ demonstrates that not only has the lean to fat ratio decreased but perhaps more significantly the saturated fat to unsaturated fat ratio has also decreased. McCance and Widdowson in their introduction to the 6th Edition to the meat and meat products section state ‘The major source of variation in meat composition is the proportion of lean to fat, as a result of husbandry techniques and trimming practices, both at a retail level and in the home. This affects levels of most other nutrients, which are distributed differently in the two fractions’.

Which brings me to the central purpose of my analysis: I did not set out to establish the nutrient content of vegetables or meat in 1940 in comparison to their content now as a simple quantitative analysis but rather as a measure of the changing nutritive value of the British diet. Thereby helping to determine

whether, by getting less micronutrients today from essentially the same food types that were eaten 70 yrs ago this circumstance could be undermining the health of our nation in a significant way.

Consequently it is my view that the criticism mentioned previously is irrelevant as it is the intrinsic micronutrient content of food products which have the same 'name' that need to be compared; the consumer in 1940 ate an apple or an orange or a rump steak and so does the consumer in 2006. To misquote Gertrude Stein – a carrot is a carrot is a carrot and a portion of broccoli is a portion of broccoli is a portion of broccoli.

In a recent paper Davies¹¹ refers to the need to compare the dry ash contents of foods rather than fresh content. The consumer does not eat dry ash. What we are concerned with here is the continued trend towards quantity and bulk (usually more water content) over quality and taste, and the micronutrient content of fresh produce presented to the consumer.

2. The impact of nutrition on health

The impact of poor nutrition on health is common knowledge. As long ago as the early 17th century it was known that scurvy was caused by a lack of fresh fruit and vegetables. For more than a hundred years we have known about vitamin deficiency diseases and the effects of poor nutrition on populations historically in Europe and the US and to this day in the developing world. In addition to micronutrient and mineral deficiency we now suffer from indiscriminate chemical exposure from our environment. The combination of these factors is leading to outbreaks of various diseases of epidemic proportions which are now beginning to be widely recognised as the primary cause of so called degenerative diseases.

In March 2006, the UN acknowledged a new kind of malnutrition. Catherine Bertini¹² Chairperson of the UN Standing Committee on Nutrition said: "The overweight are just as malnourished as the starving, and nutritional programs in poor countries need to target rising obesity alongside hunger". She also suggested that we need a new definition of malnutrition because food availability is not really the issue. It is the quality of the food that is the problem. This new type of malnutrition, which can be categorised as multiple micronutrient depletion, has been termed 'Type B malnutrition'.

What is implicit in this diagram taken from Davies' paper¹⁴ is that nutrition is fundamental to good health. To be fit and to maintain that quality we call health we need four basic components – good quality sunlight, air, water and food. The significance of these four components cannot be underestimated – too many independent researchers have demonstrated that foods low in micronutrients undermine our physical, mental and emotional well being. This is amply demonstrated in the Appendices 1 and 2 to my first paper detailing research on micronutrients and physical and mental ill health^{15,16}.

TABLE 10.

Summary of peer-reviewed research papers which correlate various mental illnesses with mineral and trace elements deficiencies or imbalances.

	Cr	Cu	Fe	I	K	Mg	Mo	P	Se	V	Zn
ADHD		X				X		X			X
Anxiety					X	X		X	X		
Aggression			X		X						X
Bipolar disorder			X	X	X	X	X			X	
Depression	X	X	X	X	X	X			X	X	X
PMS		X	X			X					X
Schizophrenia		X	X	X		X			X		

A large number of peer-reviewed research papers written between 1941 and 2003 correlate various mental illnesses with mineral and trace element deficiencies or imbalances. Table 10 summarises these relationships, the data being extracted from 225 papers published in various respected scientific and medical journals such as the American Journal of Psychiatry, The Lancet, Canadian Journal of Psychiatry, Journal of Affective Disorders, Journal of the American College of Nutrition, British Journal of Psychiatry, Journal of Learning Disabilities etc. (a full list of references is supplied in Appendix 4)

3. Overall nutritional trends in the UK and the US

Over the past 70 years many esteemed men and women working in the fields of nutrition and health have warned us about the impact of poor nutrition on health. Among them are Sir Robert McCarrison^{17,18} Surgeon Captain TL Cleave RN¹⁹, Weston Price²⁰, Linus Pauling²¹, and Trowell and Burkitt²². It appears that their warnings have been ignored. The most damning evidence is illustrated by our children being offered with deep fried rendered meat, chips and cola and no fruit or vegetables at school meal times.

To have reached this position after the billions of taxpayers pounds spent by various government bodies (MAFF, DEFRA, FSA, various government quangos and many research establishments) on agriculture and nutritional science seems extraordinary. It has taken an obesity crisis and the indignation and passion for good food of a television celebrity chef, Jamie Oliver, to cause government policy makers to act. Oliver seems to have intuitively reached the blindingly obvious conclusion that good food is good for you. In all probability this is too late given that what used to be called adult onset diabetes is now being found in paediatric clinics. There is also a rising prevalence of childhood leukemia, childhood obesity, cardiovascular disorders, infertility, osteo and rheumatoid arthritis, mental illnesses,

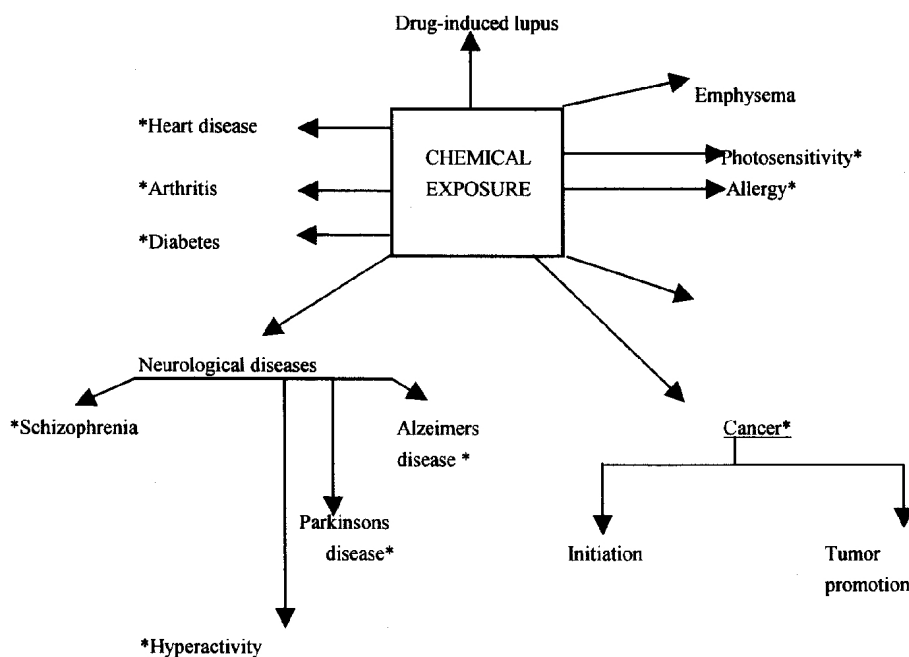


Figure 1. Diseases associated with exposure to environmental chemicals (after Forrester & Wolf¹³). The dashed lines indicate that there is circumstantial evidence to suggest that chemical exposure may play a role in the aetiology of these diseases, but this has not been proven directly. *Conditions where diet or micro-nutritional status are known to play a role.

hyperactivity, etc. All these have been shown to have some direct relationship to diet and specifically micronutrient deficiency.

However the agricultural, medical and nutritional ‘establishment’ appears to continue to ignore the obvious relationship between food and health while other sectors of the government are beginning to recognise it. For example, in a Reply by Broadley, Mead and White²³ based on their own paper ‘Historical variation in the mineral composition of edible horticultural products’ to a commentary by Davies on their paper, they recognise the decline in essential minerals over time – but contest the validity of the statistical methodology used – then dismiss these losses with the comment “Since horticultural products in general and fruit and nuts in particular are small contributors of minerals to the average diet ... changes in mineral composition are unlikely to be significant in overall dietary terms.” This is a comment made by ‘experts’ in their field who are educating our future ‘experts’ in the field of horticulture. Their comment directly contradicts the governments’ guidelines of the ‘5 a day’ campaign.

Government, academic institutions and the medical establishment continue to claim there is the need for further ‘scientific’ research when common sense tells that bad diets cause bad health and that the fast food industry

continues to be permitted to peddle junk food to children. Recently, 30 years after the confectionary and fast food establishments started it, Hastings *et al.*²⁴ proved the blindingly obvious fact that advertising to children works. Even so, according to Caraher and Lang²⁵ government agencies have all but ignored these findings. More recently Brant *et al.*²⁶ found that rats fed on organic food were slimmer, slept better and had stronger immune systems than rats fed on conventionally grown produce. To me, given the enormous knowledge base on nutrition and health available, these findings are common sense. Is it really necessary to continue to prove the obvious?

An example of a far more valid contribution to the ongoing research – which is necessary in this field – is that currently being promoted by GREEN – Gardens for Research Experimental Education and Nutrition²⁷. In this project GREEN are attempting to track the flow of nutrients from soil to plant and learn how this may be affected depending on the method of cultivation employed, to grow the same vegetables. Central to their working hypothesis is the importance of understanding and working with the soils natural micro-ecology. When plants are encouraged to work in symbiosis with soil microbes it appears to increase the transfer of essential nutrients from soil to plant. To date their research has produced potatoes and leeks with substantial increases in Ca, Mg, Fe and excellent ratios and content of Zn and Cu that far exceed any of the 1940 data. For example the calcium content of potatoes is 50 times more than that present in 1940 and for leeks the calcium content is 4 times more than that present in 1940.

In addition to measuring chemical nutrients they are also considering vitality (or freshness), visual appearance and taste of their vegetable crops. This research project should be applauded – and publicly funded.

One could ask how we ever got ourselves to this sorry position. I list below some of the reasons why the mineral content of food has diminished over time:

- i. favoring varieties of crops and animal breeds for their presentation rather than nutritional quality
- ii. increased use of trace element free NPK fertilizers
- iii. the inevitable soil depletion of essential minerals through continuous crop growing and contributed to by the overuse of NPK fertiliser with its consequent damage to endomycorrhizal fungi that help liberate essential minerals from the soil²⁸
- iv. inherent soil deficiencies of essential minerals due to parent bedrock material, the amount of organic matter present, the ionic potential of differing trace elements, the degree of soil oxidization and soil pH²⁹
- v. increased transport distances, storage times and storage methods for ‘fresh’ produce

and some of the other factors contributing to the population’s growing ill health:

- i. increased lifestyle stresses – mental, physical and emotional
- ii. increased use of stimulants – coffee, tea, tobacco, alcohol etc.
- iii. increased use of medication
- iv. dietary trends towards cheaper, more refined, quicker convenience food and drinks – high in proteins, saturated fats and refined carbohydrates but very low in micronutrients
- v. polluted air and water supplies
- vi. sunlight deprivation and quality (due to ozone depletion)

The problem is that the quality of the food and drink we consume does make a difference to our health as has been indicated above. The impact of the decline in micronutrients in modern foods compared to those foods available seventy years ago is exacerbated by two further problems – a substantial proportion of the population consume little or no fresh vegetables at all and ‘modern’ foods contain various other components that come as ‘part of the package’, namely residual herbicides, pesticides, fungicides and the ubiquitous additives of processed, convenience foods e.g. colourings, flavourings, preservatives etc. And whilst some of these have been individually tested for short term safety, no one knows what their interactions might be or their cumulative effects on the body over a lifetime.

The question arises of what is to be done about this potentially catastrophic state of affairs. During the Second World War the UK government required that, due to rationing, diets might be inadequate and all children were to receive cod liver oil and orange juice. Sixty years on we find that we are in a situation where the nation, as a whole, is overfed but malnourished and the government is again considering mandatory dietary supplementation. This would undoubtedly be a constructive short to medium term solution but only if at the same time we actively encourage, from an early age, the growing trend towards healthier eating and drinking, paying specific attention to advertising and the availability and presentation of junk foods in schools, hospitals, penal institutions and public places. There is a desperate need for the food and drinks industries to become more aware and realise they have an ever growing responsibility to their customers to provide good quality, nourishing, non-toxic products. More ‘holistic’ and local projects such as that being developed by GREEN should be encouraged. And let’s educate our children from an early age about the value and significance of our soil; how its health is directly relevant to our own, and to the health of the environment.

Assuming that the two basic premises presented here are correct – namely that the micronutrient content of our food has declined and that nutrition impacts health – there is a need for pan-European policy making bodies to encourage the growing, rearing, presentation and eating of good quality food. If we are really going to improve things, a concerted effort is needed from a variety of sources requiring the direct and integrated involvement of concerned government departments and agencies and the agriculture and food and drink industries.

CONCLUSIONS

What a dilemma we have found ourselves in. Research from all over the world has demonstrated the reality of the loss of micronutrients from our foods and provides evidence that micronutrient deficiencies significantly undermine our health, contributing towards chronic physiological and psychological illnesses in people of all ages.

Yet we continue to see the relentless pursuit of farming and marketing practices that emphasize cheapness and durability over quality to the point where the past few generations have become conditioned to accept this as the norm – with palates that have been conditioned to prefer foods containing excessive amounts of saturated fats, refined proteins, salt, sugar and other refined carbohydrates.

So what is the way forward into the 21st Century? One component to the resolution of this dilemma would be for all parties concerned to recognize the current gravity of the problem (without apportioning blame) and resolve to rectify the situation by implementing appropriate policy changes in both government and industry.

Another interesting, relatively recent, positive development is the significant and growing trend wherein individuals are beginning to make their own health choices. This trend towards nutritional awareness, with the buying of locally-grown and organic produce and a greater interest in food preparation, world cuisine and animal husbandry is a powerful force and one that is likely to bring about significant changes within the food industry.

Already this consumer-driven change has resulted in food labelling alterations as well as greater availability of organically-grown foods. We have also recently experienced within the foods and drinks industry a resistant but gradual decline in the addition of additives to food, especially artificial colours and preservatives, sugar, salt and trans-fats.

There is clearly still a long way to go and the next step will be to insist on the proper nourishment of the soils on which our food is grown and reared, preferably through organic, bio-dynamic and other sustainable farming methods, so that it will necessarily provide the minerals and trace elements that are essential for our future health and wellbeing.

REFERENCES

1. Thomas, D.E. (2003). A study of the mineral depletion of foods available to us as a nation over the period 1940 to 1991, *Nutrition and Health*, **17**, 85–115.
2. McCance and Widdowson (1940). The Chemical Composition of Foods, 1st Edition, Special Report Series No: 235. Published by Medical Research Council.
3. McCance and Widdowson (1946). The Chemical Composition of Foods, 2nd Edition, Special Report Series No: 235. Published by Medical Research Council.
4. McCance and Widdowson (1960). The Chemical Composition of Foods, 3rd Edition, Special Report Series No: 297. Published by Medical Research Council.
5. McCance and Widdowson (1978). The Composition of Foods, 4th Edition. Published by Royal Society of Chemistry/Ministry of Agriculture Fisheries and Food.

6. McCance and Widdowson (1991). *The Composition of Foods*, 5th Edition. Published by Royal Society of Chemistry/Ministry of Agriculture Fisheries and Food.
7. McCance and Widdowson (2002). *The Composition of Foods*, 6th Edition. Published by Royal Society of Chemistry/Food Standards Agency.
8. Church, S. (2005). The history of European food composition databases. On behalf of the European food information resource consortium. <http://www.eurofir.net/public.asp?id=1931>
9. Van de Weyer, C. (2006). *Changing Diets: Changing Minds: How food affects mental health and behaviour*. London: Sustain.
10. Crawford, M.A. (1991). Fat animals – Fat humans, In World Health, WHO, Geneva July/August, pp. 23–25.
11. Davis, D.R., Epp, M.D. and Riordan, H.D. (2004). Changes in USDA Food Composition Data for 43 Garden crops, 1950 to 1999. *J Am. Col. Nut.*, **23**(6), 669–682.
12. Bertini, C. (2006). UN Standing Committee on Nutrition Chair – Thirty Third Session of the Standing Committee on Nutrition Tackling the Double Burden of Malnutrition: A Global Agenda, Geneva International Conference Centre, Geneva, Switzerland www.unsystem.org/SCN/Publications/AnnualMeeting/SCN33/FINAL%20REPORT%2033rd%20SESSION.pdf
13. Forrester, L.M. and Wolf, C.R. (1990). Genetic susceptibility to environmental insults. In: *The Metabolic and Molecular Basis of Acquired Disease*, Eds. Cohen, R.D., Lewis, B., Alberti, K.G.M.M. and Denman, A.M., pp. 3–18. London: Bailliere-Tindall.
14. Davies, S. (1991). Scientific and Ethical Foundations of Nutritional Medicine. Part 1 – Evolution, Adaptation & Health, *Journal of Nutritional Medicine*, **2**, 227–247.
15. Werbach, M.R. (1998). *Nutritional Influences on Illness – A source book of clinical research*. California: Third Line Press.
16. Werbach, M.R. (1999). *Nutritional Influences on Mental Illness – A source book of clinical research*. California: Third Line Press.
17. McCarrison, R. (1936). Nutrition in health and disease, *Brit. M.J.*, **2**, 611–615.
18. McCarrison, R. (1937). Nutritional needs in pregnancy, *Brit. M.J.*, **2**, 256–257.
19. Cleve, T.L. (1977). Over-consumption. Now the most dangerous cause of disease in Westernised countries, *Public Health: The Journal of the Society of Community Medicine*, **91**(3), 127–3.
20. Price, W. (1945). *Nutrition and Physical Degeneration*. Connecticut: Keats.
21. Pauling, L. (1970), Evolution and the Need for Ascorbic Acid – *Proceedings of the National Academy of Sciences*.
22. Trowell, N., Burkitt, D. and Heaton, K. (1986). *Dietary Fibre, Fibre-depleted Foods and Disease*. London: Academic Press.
23. Broadley, M.R., Mead, A. and White, P.J. (2006). Reply to Commentary regarding “Historical Variation in the mineral composition of edible horticultural products.” *Journal of Horticultural Science and Biotechnology*, **81**, 553–555.
24. Hastings, G., Stead, M., McDermott, L., Forsyth, A., MacKintosh, A., Rayner, M., Godfrey, C., Caraher, M. and Angus, K. (2003). *Review of the Research on the Effects of Food Promotion to Children*. Glasgow: Centre for Social Marketing.
25. Caraher, M. and Lang, T. (2006). The relationship between evidence and policy. A case study of the regulation of advertising aimed at children. Centre for Food Policy, Dept of Health Management and Food Policy.
26. Lauridsen, C. *et al.* (of the Danish Institute of Agricultural Sciences) and Brandt, K., School of Agriculture, University of Newcastle (2006). Organic diet enhanced the health of rats – <http://www.darcof.dk/research/health.html>
27. GREEN published data found in The Good Gardeners Association Newsletter, Spring 2006, Issue 159. FFi www.goodgardeners.org.uk.
28. Ward, N., Stead, K. and Reeves, J. (2001). Impact of endomycorrhizal fungi on plant trace element uptake and nutrition. *The Nutrition Practitioner*, **3**(2), 30–31.
29. Thorvaldsson, G. (2005). Chair of Seminar on Essential Trace Elements for Plants, Animals and Humans – convened by the Ag. Uni. Iceland – NJF Seminar no. 370 – [www.hvanneyri.is/landbunadur/wglbhi.nsf/Attachment/LBHI-rit-3/\\$file/LBHI-rit-3.pdf](http://www.hvanneyri.is/landbunadur/wglbhi.nsf/Attachment/LBHI-rit-3/$file/LBHI-rit-3.pdf)

1991		3.2	2.9		0.8	2.7	4.6		1.3		0.9	7.8	24.2
2002		2.9	2.4	0.6	0.7	2.7	3.3	9	2.2	1.1	1.1	0.8	5.1
1940	Copper (Cu)	0.25	-	-	-	-	-	-	-	-	0.2	-	less 50%
1972		0.13	0.12	0.12	0.12	0.31	0.15		0.29				0.45
1991		0.14	0.24	0.12	0.12	0.12	0.15	0.63	0.1	0.06	0.15	0.36	1.33
2002		0.04	0.18	0.06	0.07	0.08	0.31	0.15	0.63	0.1	0.09	0.36	0.6
1972	Zinc (Zn)	4.9	2.6	2.4									less 55%
1991		5.3	5.6	5.5	1.9	2.6	2.6	2.6	3	1.3	3.2	1.7	3.9
2002		6.5	4.7	5.5	1.9	2.1	2.7	2.6	3	1.3	3.2	1.7	2.5
													less 1%

Copper – comparisons between the two figures given in 1940 with those in 2002 show a 78% decrease in Copper content

Copper – comparisons between those 1991 figures which are different from those given in 2002 – for the same cuts of meat – show a 55% decrease in Copper content

Zinc – comparisons between those 1991 figures which are different from those given in 2002 – for the same cuts of meat – show a 1% increase in Zinc content

Each individual figure represents mg per 100 gm

These statistics have been calculated by comparing and contrasting data first published in 1940 by McCance and Widdowson in 'The Chemical Composition of Foods', which was commissioned by the Medical Research Council - with that data published by the same authors in 1991 entitled 'The Composition of Foods'

This later - 5th edition - was commissioned by the Royal Society of Chemistry and the Ministry of Agriculture Fisheries and Food. The latest - 6th edition - in this series was published in 2002 by the Royal Society of Chemistry and the Foods Standards Agency.

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APPENDIX 2 – Summary of changes in the mineral content of 9 Cheeses between 1940 and 2002

Year of analysis	Mineral	camembert	chedder	cream	danish	edam	gouda	parmesan	processed	stilton	Totals	Change
1940 and 1960	Water	47.5	37	10	40.5	43.7	42.4	28	43	28.2	320.3	Plus 19%
1978												
1991		50.7	36	45.5	45.3	43.8	40.1	18.4	45.7	38.6		
2002		54.4	36.6	45.5	46.3	43.8	40.4	27.6	47.4	38	380	
1940 and 1960	Sodium (Na)	1408	612	110	1417	983	1054	755	918	1150	8407	Na Less 9%
1978		1410	610	300	*	*		*	1360	*	3680	
1991		650	670	300	1260	1020	910	1090	1320	930	8150	
2002		605	723	300	1220	996	925	756	1351	788	7664	
1940 and 1960	Potassium (K)	111	116	47	186	159	124	153	86	161	1143	K less 19%
1978		110	120	160					82			
1991		100	77	160	89	97	91	110	130	130		
2002		104	75	160	88	89	82	51	178	96	923	
1940 and 1960	Phosphorous (P)	285	545	44	425	523	469	772	480	304	3847	P less 8%
1978		290	520	100					490			
1991		310	490	100	370	530	490	810	800	310		
2002		241	505	100	344	508	498	267	768	314	3545	
1940 and 1960	Magnesium (Mg)	17.4	46.9	5.2	20.4	27.7	24.5	49.6	47.6	27.2	266.5	Mg less 26%
1978		17	25	10					24			
1991		21	25	10	27	39	38	45	22	20		
2002		14	29	10	20	34	32	15	27	15	196	
1940 and 1960	Calcium (Ca)	152	810	30	578	739	622	1220	724	362	5237	Ca less 15%
1978		380	800	98					700			
1991		350	720	98	500	770	740	1200	600	320		
2002		235	739	98	488	795	773	362	610	326	4426	

To enable an historical comparison to be made I have amalgamated the 1940 and 1960 figures together to provide a total

1940 and 1960	Iron (Fe)	0.76	0.57	0.14	0.17	0.21	0.34	0.37	0.57	0.46	3.59	Fe less 53%
1978			0.76	0.4	0.12					0.5		1.38
1991		0.2	0.3	0.1	0.2	0.4	0.1	1.1	0.5	0.3	3.2	
2002		0	0.3	0.1	0	0.3	0.3	0	0.5	0.2	1.7	
1940 and 1960	Copper (Cu)	0.08	0.03	0.04	0.09	0.03	0.06	0.38	0.03	0.03	0.77	Cu less 91%
1978		0.08	0.03						0.5		0.61	
1991			0.03				0	0.33	0.17	0.18	0.68	
2002		0	0.03	0	0	0	0	0	0	0.04	0.07	
1978 (1st	Zinc (Zn)	3	4	0.48		4		4	3.2	4	22.68	Zn less 18%
analysed)												
1991			2.3				1.8	5.3	3.2	2.5	12.8	
2002		2.1	4.1	0.5	3	3.8	3.9	2.7	2.6	2.9	18.7	

Each individual figure represents mg per 100gm.

These statistics have been calculated by comparing and contrasting data first published in 1940 by McCance and Widdowson in 'The Chemical Composition of Foods', which was commissioned by the Medical Research Council – with that data published by the same authors in 1991 entitled 'The Composition of Foods'.

This later – 5th edition – was commissioned by the Royal Society of Chemistry and the Ministry of Agriculture Fisheries and Food. The latest – 6th edition – in this series was published in 2002 by the Royal Society of Chemistry and the Foods Standards Agency.

APPENDIX 3 – Summary of changes in the mineral content of 4 Dairy products between 1940 and 2002

Year of analysis	Mineral	Whole Milk	Butter	Cream – Single	Cream – Double	Totals	Percentage Change
To enable an historical comparison to be made I have amalgamated the 1940 and 1960 figures together to provide a total							
1940 and 1960	Sodium (Na)	50		42.2	46.2	138.4	Na – Less 47%
1991		55		49	37	141	
2002		43		29	22	94	
1940 and 1960	Potassium (K)	160	15	124	79	378	K – Less 7%
1991		140	15	120	65	340	
2002		155	27	104	65	351	
1940 and 1960	Phosphorous (P)	95	24	44	21	184	P – Plus 34%
1991		92	24	76	50	242	
2002		93	23	79	52	247	
1940 and 1960	Magnesium (Mg)	14	2.4	6	3.8	26.2	Mg – Less 1%
1991		11	2	9	6	28	
2002		11	2	8	5	26	
1940 and 1960	Calcium (Ca)	120	14.8	79	50	263.8	Ca – Plus 4%
1991		115	15	91	50	271	
2002		118	18	89	49	274	
1940 and 1960	Iron (Fe)	0.08	0.16	0.31	0.2	0.75	Fe – Less 83%
1991		0.05	0.2	0.1	0.2	0.55	
2002		0.03	0	0	0.1	0.13	
1940 and 1960	Copper (Cu)	0.02	0.03	0.2	0.13	0.38	Cu – Less 97%
1991		0	0.03	0	0	0.03	
2002		0	0.01	0	0	0.01	

	Zinc (Zn)	Zn - Less 17%
1978 (1st analysed)	0.35	
1991	0.4	0.2
2002	0.4	0.2
		1.2
		1

Each individual figure represents mg per 100gm.
 These statistics have been calculated by comparing and contrasting data first published in 1940 by McCance and Widdowson in 'The Chemical Composition of Foods', which was commissioned by the Medical Research Council - with that data published by the same authors in 1991 entitled 'The Composition of Foods'.
 This later - 5th edition - was commissioned by the Royal Society of Chemistry and the Ministry of Agriculture Fisheries and Food series
 The latest - 6th edition - in this was published in 2002 by the Royal Society of Chemistry and the Foods Standards Agency

APPENDIX 4

Some references relating to the intrinsic role of mineral and trace element deficiency or imbalance in psychiatric disorders.

Boron

Boron deficiency or imbalance may play a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between boron and brain function 1,2,3,4,5.

REFERENCES

1. Penland J G, (1998). The importance of boron nutrition for brain and psychological function. *Biol Trace Elem Res*, 66, (1–3) 299–317.
2. Penland, J.G. (1996). Trace elements, Brain function and behavior: effects of Zinc and Boron. *TEKTRAN, United States Department of Agriculture. Agricultural Research Service.*
3. Penland, J.G. (1994). Dietary boron, brain function, and cognitive performance. *Environ Health Perspect*, **102** (supplement 7) 65–72.
4. Penland, J.G. (1995). Quantitative analysis of EEG effects following experimental marginal magnesium and boron deprivation. *Magnesium Research*, **8**, 341–58.
5. Forrest, N. (1999). The emergence of Boron as nutritionally important throughout the life cycle. *TEKTRAN, United States Department of Agriculture. Agricultural Research Service.*

Chromium

Chromium deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between chromium and depression 1,2,3,4.

REFERENCES

1. McLeod, M.N. and Golden, R.N. (2000). Chromium treatment of depression. *Int J Neuropsychopharmacol*, **3**, 311–314.
2. McCarty, M.F. (1994). Enhancing central and peripheral insulin activity as a strategy for the treatment of endogenous depression – an adjuvant role for chromium picolinate? *Med Hypotheses*, **43**(4), 247–52.
3. Davidson, J.R., Abraham, K., Connor, K.M. and McLeod, M.N. (2003). Effectiveness of chromium in atypical depression: a placebo-controlled trial. *Biol Psychiatry*, **153**(3), 261–4.
4. McLeod, M.N., Gaynes, B.N. and Golden, R.N. (1999). Chromium potentiation of antidepressant pharmacotherapy for dysthymic disorder in 5 patients. *J Clin Psychiatry*, **60**(4), 237–40.

Copper

Copper deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between copper and ADHD 1,2,3, depression 4,5,6, premenstrual syndrome 7, and schizophrenia 8,9,10,11,12,13,14, 15,16,17,18,19.

REFERENCES

1. Koziolec, T., Starobrat-Hermelin, B. and Kotkowiak, L. (1979) Deficiency of certain trace elements in children with hyperactivity. *Psychiatr Pol*, **28**(3), 345–53.
2. Brenner, A. (1979). Trace mineral levels in hyperactive children responding to the Feingold diet. *J Pediatr*, **94**(60), 944–5.
3. Pfeiffer, C.C. and Mailloux, R. (1987). Excess copper as a factor in human diseases. *J Orthomol Med.*, **2**(3), 171–82.
4. Hansen, Cr. Jr. *et al.* (1983). Copper and zinc deficiencies in association with depression and neurological findings. *Biol Psychiatry*, **18**(3), 395–401.
5. Narang, R.L., Gupta, K.R., Narang, A.P. and Singh, R. (1991). Levels of copper and zinc in depression. *Indian J Physiol Pharmacol*, **35**(4), 272–4.
6. Ali, S.A. *et al.* (1985), Blood levels of vanadium, caesium, and other elements in depressive patients. *J Affect Disord.*, **9**, 187–91.
7. Choung, C. and Dawson, E. (1994). Zinc and copper levels in premenstrual syndrome. *Fert Steril.*, **62**, 313–20.
8. Kornhuber, J., Lange, K.W., Kruzik, P. *et al.* (1994). Iron, copper, zinc, magnesium, and calcium in postmortem brain tissue from schizophrenic patients. *Biol Psychiatry*, **36**(1), 31–4.
9. Shore, D. *et al.* (1983) CSF copper concentrations in chronic schizophrenia. *Am J Psychiatry*, **140**(6), 754–7.
10. Tyrer, S.P. *et al.* (1979). CSF copper in schizophrenia. *Am J Psychiatry*, **136**(7), 937–9.
11. Gillin, J.C. *et al.* (1982), Zinc and copper in patients with schizophrenia. *Encephale*, **8**(3), 435–44.
12. Olatunbosun, D.A. *et al.* (1975), Serum copper in schizophrenia in Nigerians. *Br J Psychiatry*, **127**, 119–21.
13. Pfeiffer, C.C. and Illiev, V. (1972), A study of zinc deficiency and copper excess in the schizophrenias. *Int Rev Biol Suppl*, **1**.
14. Pfeiffer, C.C. and LaMola, S. (1983), Zinc and manganese in the schizophrenias. *J Orthomol Psychiatry*, **12**, 215–34.
15. Bowman, M.B. and Lewis, M.S. (1982). The copper hypothesis of schizophrenia: a review. *Neurosci Biobehav Rev.*, **6**, 321–8.
16. Gillin, J.C. *et al.* (1982). Zinc and copper in patients with schizophrenia *Encephale*, **8**(3) 435-44.
17. Matke J D, Adler M, (1971), Mode of action of D-penicillamine in chronic schizophrenia. *Dis Nerv Sys.*, **32**, 388.
18. Affleck, J.W. *et al.* (1969). Penicillamine and schizophrenia – A clinical trial. *Br J Psychiatry*, **115**, 173.
19. Nicholson, G.A. *et al.* (1966), Effect of D-penicillamine on schizophrenic patients. *Lancet*, **i**, 344.

Iodine

Iodine deficiency or imbalance plays a role in the symptoms of mood disorders 1,2. Observational and experimental studies have shown an association between iodine and aggression 3, anxiety 4, bipolar disorder 5,6,7, depression 8,9, and schizophrenia 10.

REFERENCES

1. Benvenga, S., Lapa, D. and Trimarchi, F. (2003). Don't forget the thyroid in the etiology of psychoses. *Am J Med.*, **115**(2), 159–60.
2. Yang, S.J., Wang, S.Y. and Chen, C.C. (2003). Acute psychotic state due to hyperthyroidism following excision of a mandible bone tumor: a case report. *Kaohsiung J Med Sci.*, **19**(1), 29–32. PMID – 12693723
3. Stalenheim, E.G. (2004). Long-term validity of biological markers of psychopathy and criminal recidivism: follow-up 6-8 years after forensic psychiatric investigation. *Psychiatry Res.*, **121**(3), 281–91.
4. Lee, I.T., Sheu, W.H., Liao, Y.J., Lin, S.Y., Lee, W.J. and Lin, C.C. (2003). Relationship of stressful life events, anxiety and depression to hyperthyroidism in an asian population. *Horm Res.*, **60**(5), 247–51.
5. Bauer, M., London, E.D., Silverman, D.H., Rasgon, N., Kirchheiner, J. and Whybrow, P.C. (2003). Thyroid, brain and mood modulation in affective disorder: insights from molecular research and functional brain imaging. *Pharmacopsychiatry*, **36**, Suppl 3, 215–21.
6. Arnold, L.M. (2003). Gender differences in bipolar disorder. *Psychiatr Clin North Am.*, **26**(3), 595–620.
7. Davis, J.D., Stern, R.A. and Flashman, L.A. (2003). Cognitive and neuropsychiatric aspects of subclinical hypothyroidism: significance in the elderly. *Curr Psychiatry Rep.*, **5**(5), 384–90.
8. M M Van de Vyvere, J., Vandoolaeghe, E., Bril, T., Demedts, P., Wauters, A. and Neels, H. (1996). Alterations in iron metabolism and the erythron in major depression: further evidence for a chronic inflammatory process. *J Affect Disord.*, **940**(1–2), 23-33.
9. Patterson, A.J., Brown, W.J. and Roberts, D.C. (2001). Dietary and supplement treatment of iron deficiency results in improvements in general health and fatigue in Australian women of childbearing age. *J Am Coll Nutr.*, **20**(4), 337–42.
10. Kuloglu, M., Atmaca, M., Ustundag, B., Canatan, H., Gecici, O. and Tezcan, E. (2003). Serum iron levels in schizophrenic patients with or without akathisia. *Eur Neuropsychopharmacol*, **13**(2), 67–71.

Iron

Iron deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between iron and aggression 1,2,3,4, ADHD 5,6,7,8,9, bipolar disorder 10, depression 11,12,13,14, premenstrual syndrome 15, and schizophrenia 16,17,18.

REFERENCES

1. Rosen, G.M., Deinard, A.S., Shwartz, S. *et al.* (1985). Iron deficiency among incarcerated juvenile delinquents *J Adolesc Health Care*, **6**(6), 419–23.
2. Webb, T.E. and Oski, F.A. (1974). Behavioral status of young adolescents with iron deficiency anemia. *J Spec Educat.*, **8**(2), 153–6.
3. William J. Walsh (1983). Analytical chemist, Argonne National Laboratory – reported in *Science News*, **124**, 122–5.
4. Tu, J.B., Shafey, H. and Van Dewetering, C. (1994). Iron deficiency in two adolescents with conduct, dysthymic and movement disorders. *Can J Psychiatry.*, **39**(6), 371–5.
5. Lozoff, B. and Brittenham, G.H. (1986). Behavioral aspects of iron deficiency. *Prog Hematol.*, **14**, 23–53.
6. Pollitt, E. *et al.* (1982). Behavioral effects of iron deficiency anemia in children, in *Iron Deficiency, Brain Biochemistry and Behavior*, Eds E. Pollitt and R.L. Leibel, New York, Raven Press, 195–204.
7. Ward, N.I. (1997). Assessment of chemical factors in relation to child hyperactivity. *J Nutr Environ Med.*, **7**, 333–42.
8. Koziolec, T., Starobrat-Hermelin, B. and Kotkowiak, L. (1994). Deficiency of certain trace elements in children with hyperactivity. *Psychiatr Pol.*, **28**(3), 345–53.
9. Sever, Y., Ashkenazi, A., Tyano, S. and Weizman, A. (1997). Iron treatment in children with attention deficit hyperactivity disorder. *Neuropsychobiology*, **35**, 178–80.
10. Feifel, D. *et al.* (1997). Iron overload among a psychiatric outpatient population. *J Clin Psych.*, **58**(2), 74–8.
11. Parker, S.D. (1984). Depression and nutrition: Anemia and glucose imbalances. *Anabolism*, Jan.–Feb.
12. Heinze, G., Hippus, H., Langer, G. and Matussek, N. (1997). Serum iron levels in depressed patients. *Pharmakopsychiatr Neuropsychopharmakol*, **10**(4), 239–42.
13. Fordy, J. and Benton, D. (1994). Does low iron status influence psychological functioning? *J Hum Nutr Diet.*, **7**, 127–33.
14. Ballin, A., Berar, M., Rubinstein, U. *et al.* (1992). Iron state in female adolescents. *Am J Dis Child*, **146**(7), 803–5.
15. Penland, J. and Hunt, J. (1993). Nutritional status and menstrual-related symptomatology. Abstract. *FASEB J*, **7**, A379.
16. White, T. and Brown, K. (1991). Low serum iron levels and neuroleptic malignant syndrome. Letter. *Am J Psychiatry*, **148**(1), 148.
17. Rosebush, P. and Stewart, T. (1989). A prospective analysis of 24 episodes of neuroleptic malignant syndrome. *Am J Psychiatry*, **146**, 717–25.
18. Brown, K.W. *et al.* (1987). Low serum iron status and akathisia. *Lancet*, **i**,1234–6.

Magnesium

Magnesium deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between magnesium and aggression 1,2,3,4,5,6,7,8,9,10, anxiety 11,12,13,14,15, ADHD 16,17,18, bipolar disorder 19,20,21, depression 22,23,24,25,26,27,28,29,30,31,32,33, 34, premenstrual syndrome 35,36,37,38,39,40,41,42,43,44,45,46,47, 48,49,50,51, and schizophrenia 52,53,54,55,56,57,58,59,60,61,62,63, 64.

REFERENCES

1. Izenwasser, S.E. *et al.* (1986). Stimulant-like effects of magnesium on aggression in mice. *Pharmacol Biochem Behav.*, **25**(6), 1195–9.
2. Henrotte, J.G. (1986). Type A behavior and magnesium metabolism. *Magnesium*, **5**, 201–10.
3. Bennett, C.P.W., McEwen, L.M., McEwen, H.C. and Rose, E.L. (1998). The Shipley Project: treating food allergy to prevent criminal behaviour in community settings. *J Nutr Environ Med.*, **8**, 77–83.
4. Kirow, G.K., Birch, N.J., Steadman, P. and Ramsey, R.G. (1994). Plasma magnesium levels in a population of psychiatric patients: correlation with symptoms. *Neuropsychobiology*, **30**(2–3), 73–8.
5. Kantak, K.M. (1988). Magnesium deficiency alters aggressive behavior and catecholamine function. *Behav Neurosci.*, **102**(2), 304–11.
6. Izenwasser, S.E., Garcia-Valdez, K. and Kantak, K.M. (1986). Stimulant-like effects of magnesium on aggression in mice. *Pharmacol Biochem Behav.*, **25**(6), 1195–9.
7. Struempfer, R.E. *et al.* (1985). Hair mineral analysis and disruptive behavior in clinically normal young men. *J Learn Disabil.*, **18**(10), 609–12.
8. Banki, C.M., Vojnik, M., Papp, Z. *et al.* (1985). Cerebrospinal fluid magnesium and calcium related to amine metabolites, diagnosis, and suicide attempts. *Biol Psychiatry*, **20**(2), 163–71.
9. Schmidt, K., Wier, W.R. and Asch, M. (1981). Clinical ecology treatment approach for juvenile offenders. *J Behav Ecology: Biosocial*, **2**(1).
10. Von Hilsheimer, G., Philpott, W., Buckley, W. and Klotz, S.C. (1977). Correcting the incorrigible. A report on 229 “incorrigible” adolescents. *Am Lab.*, **107**, 22–49.
11. Buist, R.A. (1985). Anxiety neurosis: The lactate connection. *Int Clin Nutr Rev*, **5**, 1–4.
12. Seelig, M.S., Berger, A.R. and Spieholz, N. (1975). Latent tetany and anxiety, marginal Mg deficit, and normocalcemia. *Dis Nerv Syst*, **36**, 461–5.
13. Durlach, J., Durlach, V., Bac, P. *et al.* (1994). Magnesium and therapeutics. *Magnes Res.*, **7**(3/4), 313–28.
14. Kirov, G.K. and Tsachev, K.N. (1990). Magnesium, schizophrenia and manic-depressive disease. *Neuropsychobiology*, **23**(2), 79–81.
15. Weston, P.G. *et al.* (1923). Magnesium sulphate as a sedative. *Am J Med Sci.*, **165**, 431–3.
16. Durlach, J. (1980). Clinical aspects of chronic magnesium deficiency, *Magnesium in Health and Disease*, Ed M.S. Seelig, New York, Spectrum Publications.
17. Koziolec, T. and Starobrat-Hermelin, B. (1997). Assessment of magnesium levels in children with attention deficit hyperactivity disorder (ADHD). *Magnes Res.*, **10**(2), 143–8.

18. Starobrat-Hermelin, B. and Koziolec, T. (1997). The effects of magnesium physiological supplementation on hyperactivity in children with attention deficit hyperactivity disorder (ADHD). Positive response to magnesium oral loading test. *Magnes Res.*, **10**(2), 149–56.
19. George, M.S., Rosenstein, D., Rubinow, D.R. *et al.* (1994). CSF magnesium in affective disorder: lack of correlation with clinical course of treatment. *Psychiatry Res.*, **51**(2), 139–46.
20. Kirov, G.K., Birch, N.J., Steadman, P. and Ramsey, R.G. (1994). Plasma magnesium levels in a population of psychiatric patients: correlations with symptoms. *Neuropsychobiology*, **30**(2–3), 73–8.
21. Chouinard, G., Beauclair, L., Geiser, R. and Etienne, P. (1990). A pilot study of magnesium aspartate hydrochloride (Magnesiocard) as a mood stabilizer for rapid cycling bipolar affective disorder patients. *Prog Neuropsychopharmacol Biol Psychiatry.*, **14**(2), 171–180.
22. Kirov, G.K., Birch, N.J., Steadman, P. and Ramsey, R.G. (1994). Plasma magnesium levels in a population of psychiatric patients: correlation with symptoms. *Neuropsychobiology*, **30**(2–3), 73–8, 1994.
23. Linder, J. *et al.* (1989). Calcium and magnesium concentrations in affective disorder: Difference between plasma and serum in relation to symptoms. *Acta Psychiatr Scand.*, **80**, 527–37.
24. Frazer, A. *et al.* (1983). Plasma and erythrocyte electrolytes in affective disorders. *J Affect Disord.*, **5**(2), 103–13.
25. Bjorun, N. (1972). Electrolytes in blood in endogenous depression. *Acta Psychiatr Scand.*, **48**, 59–68.
26. Cade, J.F.J.A. (1964). A significant elevation of plasma magnesium levels in schizophrenia and depressive states. *Med J Aust.*, **1**, 195–6.
27. Kirov, G.K. and Tsachev, K.N. (1990). Magnesium, schizophrenia and manic-depressive disease. *Neuropsychobiology*, **23**(2), 79–81.
28. Hall, R.C.W. and Joffe, J.R. (1973) Hypomagnesemia: Physical and psychiatric symptoms. *JAMA*, **224**, 1749–51.
29. Frizel, D., Copen, A. and Marks, V. (1969), Plasma magnesium and calcium in depression. *Br J Psychiatry*, **115**, 1375–7.
30. Hasey, G.M., D’Alessandro, E., Cooke, R.G. and Warsh, J.J. (1993). The interface between thyroid activity, magnesium, and depression: A pilot study. *Biol Psychiatry*, **33**, 133–5.
31. Linder, J. *et al.* (1989). Calcium and magnesium concentrations in affective disorder: Difference between plasma and serum in relation to symptoms. *Acta Psychiatr Scand*, **80**, 527–37.
32. Frazer, A. *et al.* (1983). Plasma and erythrocyte electrolytes in affective disorders. *J Affect Disord.*, **5**(2), 103–13.
33. Banki, C.M. *et al.* (1986). Aminergic studies and cerebrospinal fluid cations in suicide. *Ann N Y Acad Sci.*, **487**, 221–30.
34. Banki, C.M. *et al.* (1985). Cerebrospinal fluid magnesium and calcium related to amine metabolites, diagnosis, and suicide attempts. *Biol Psychiatry*, **20**, 163–71.
35. Posaci, C., Erten, O., Uren, A. and Acar, B. (1994). Plasma copper, zinc and magnesium levels in patients with premenstrual tension syndrome. *Acta Obstet Gynecol Scand.*, **73**(6), 452–5.
36. Rosenstein, D.L. *et al.* (1994). Magnesium measures across the menstrual cycle in premenstrual syndrome. *Biol Psychiatry*, **35**, 557–61.
37. Chuong, C.J. and Dawson, E.B. (1994). Magnesium levels in premenstrual syndrome. *Nutr Res.*, **14**(11), 1623–34.
38. Mira, M., Stewart, P.M. and Abraham, S.F. (1998). Vitamin and trace element status in premenstrual syndrome. *Am J Clin Nutr.*, **47**, 636–41.

39. Sherwood, R.A., Rocks, B.F., Stewart, A. and Saxton, R.S. (1986). Magnesium and the premenstrual syndrome. *Ann Clin Biochem*, **23**(6), 667-70.
40. Stebbing, J.B. *et al.* (1982). Reactive hypoglycaemia and magnesium. *Magnesium Bull.*, **4**(2), 131-4.
41. Rosenstein, D.L. *et al.* (1994). Magnesium measures across the menstrual cycle in premenstrual syndrome. *Biol Psychiatry*, **35**, 557-61.
42. Stewart, A. (1987). Clinical and biochemical effects of nutritional supplementation on the premenstrual syndrome. *J Reprod Med.*, **32**, 435-41.
43. Stebbing, J.B. *et al.* (1982). Reactive hypoglycaemia and magnesium. *Magnesium Bull.*, **4**(2), 131-4.
44. Abraham, G.E. (1982). Magnesium deficiency in premenstrual tension. *Magnesium Bull.*, **1**, 68-73.
45. Abraham, G.E. and Lubran, M.M. (1981). Serum and red cell magnesium levels in patients with premenstrual tension. *Am J Clin Nutr.*, **34**(11), 2364-6.
46. Stebbing, J.B. *et al.* (1982). Reactive hypoglycaemia and magnesium. *Magnesium Bull.*, **4**(2), 131-4.
47. Brown, R.C. and Bidlack, W.R. (1985). Regulation of glucuronyl transferase by intracellular magnesium, in *Proceedings of the International Symposium on Magnesium and its Relationship to Cardiovascular, Renal and Metabolic Disorders*. Los Angeles, 24.
48. Curry, D.L. *et al.* (1977). Magnesium modulation of glucose-induced insulin secretion by the perfused rat pancreas. *Endocrinology*, **101**, 203.
49. Abraham, G.E. (1986). Management of the premenstrual tension syndromes: Rationale for a nutritional approach, in *A Year in Nutritional Medicine*. Ed. J. Bland: New Canaan, CT, Keats Publishing, 125-66.
50. Facchinetti, F., Bolzella, P., Sances, G. *et al.* (1991). Oral magnesium successfully relieves premenstrual mood changes. *Obstet Gynecol.*, **78**(2), 177-81.
51. Facchinetti, F. *et al.* (1991). Magnesium prophylaxis of menstrual migraine: effects of intracellular magnesium. *Headache*, **31**, 298-304.
52. Levine, J., Rapoport, A., Mashiah, M. and Dolev, E. (1996). Serum and cerebrospinal levels of calcium and magnesium in acute versus remitted schizophrenic patients. *Neuropsychobiology*, **33**(4), 169-72.
53. Kirrow, G.K., Birch, N.J., Steadman, P. and Ramsey, R.G. (1994). Plasma magnesium levels in a population of psychiatric patients: correlation with symptoms. *Neuropsychobiology*, **30**(2-3), 73-8.
54. Kanofsky, J.D. *et al.* (1991). Is iatrogenic hypomagnesemia common in schizophrenia? Abstract. *J Am Coll Nutr.*, **10**(5), 537.
55. Kirrow, G.K. and Tsachev, K.N. (1990). Magnesium, schizophrenia and manic-depressive disease. *Neuropsychobiology*, **23**(2), 79-81.
56. Chhatre, S.M. *et al.* (1985). Serum magnesium levels in schizophrenia. *Ind J Med Sci.*, **39**(11), 259-61.
57. Paul, E.A. *et al.* (1978). Serum calcium and magnesium in schizophrenia. Relationship to clinical phenomena and neuroleptic treatment. *Br J Psych.*, **133**, 143-9.
58. Daly, R.M. and Gold, G. (1976). Serum magnesium levels in nonacute schizophrenics. *N Y State J Med.*, **76**, 188-9.
59. Hakim, A.H. *et al.* (1973). A comparative study of serum calcium and magnesium in cases of endogenous depression, reactive depression, schizophrenia and conversion reaction. *J Assn Phys Ind.*, **23**, 513-17.
60. Pandey, S.K. *et al.* (1973). An estimation of magnesium and calcium in serum and CSF in schizophrenia. *J Assn Phys Ind.*, **21**, 203-5.
61. Chugh, T.D. *et al.* (????) Magnesium in schizophrenia. *Ind J Med Res.*, **61**, 998-1001.
62. Cade, J.F.J. (1964). A significant elevation of plasma magnesium level in schizophrenia and depressive states. *Med J Aust.*, **1**, 195-6.
63. Kornhuber, J., Lange, K.W., Kruzik, P. *et al.* (1994). Iron, copper, zinc, magnesium, and

- calcium in postmortem brain tissue from schizophrenic patients. *Biol Psychiatry*, **36**(1), 31–4.
64. Levine, J., Rapoport, A., Mashiah, M. and Dolev, E. (1996). Serum and cerebrospinal levels of calcium and magnesium in acute versus remitted schizophrenic patients. *Neuropsychobiology*, **33**(4), 169–72.

Manganese

Manganese deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between manganese and aggression 1,2,3,4,5, ADHD 6,7,8,9, and schizophrenia 10,11,12,1,14.

REFERENCES

1. Marlowe, M., Bliss, L. and Schneider H.G. (1994). Hair trace element content of violence prone male children. *J Advancement Med.*, **7**(1), 5–18.
2. Gottschalk, L.A. *et al.* (1991). Abnormalities in hair trace-elements as indicators of aberrant behavior. *Compr Psychiatry*, **32**, 229–37.
3. Schmidt, K., Wier, W.R. and Asch, M. (1981). Clinical ecology treatment approach for juvenile offenders. *J Behav Ecology: Biosocial*, **2**(1).
4. Von Hilsheimer, G., Philpott, W., Buckley, W. and Klotz, S.C. (1977). Correcting the incorrigible. A report on 229 “incorrigible” adolescents. *Am Lab.*, **107**, 22–49.
5. Schauss, A.G. Comparative hair mineral analysis results of 21 elements in a random behaviorally “normal” 19–59 year old population and violent adult criminal offenders. *Int J Biosoc Res.*, **1**, 21–41.
6. Collipp, P.J., Chen, S.Y. and Maitinsky, S. (1983). Manganese in infant formulas and learning disability. *Ann Nutr Metab.*, **27**, 488–94.
7. Keen, C.L., Bell, J.G. and Lonnerdal, B. (1986). The effect of age on manganese uptake and retention from milk and infant formulas in rats. *J Nutr.*, **116**, 395–402.
8. Barlow, P.J. (1983). A pilot study on the metal levels in the hair of hyperactive children. *Med Hypotheses*, **11**(3), 309–18.
9. Barlow, P. (1979). Hair metal analysis and its significance to certain diseases. *Presentation at the 2nd Annual Trace Minerals in Health Seminar*. Boston, MA, September 8–9.
10. Pfeiffer, C.C. (1974). Observations on trace and toxic elements in hair and serum. *J Orthomol Psychiatry*, **3**(4), 259–64.
11. Pfeiffer, C.C. and LaMola, S. (1983). Zinc and manganese in the schizophrenics. *J Orthomol Psychiatry*, **12**, 215–34.
12. Pfeiffer, C.C. and Iliev, V. (1972). A study of zinc deficiency and copper excess in the schizophrenias. *Int Rev Neurobiol.*, p. 141.
13. Hoskins R G. (1934), The manganese treatment of schizophrenic disorders. *J Nerv Ment Dis.*, **79**, 59–62.
14. English, W.M. (1929), Report of the treatment with manganese chloride in dementia praecox. *Can Med Assoc J.*, **21**, 96–149.

Molybdenum

Molybdenum deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between molybdenum and bipolar disorder 1. There is also strong evidence for the neuro-protective role of molybdenum 2,3,4.

REFERENCES

1. Naylor, G.J, Smith, A.H., Bryce-Smith, D. and Ward, N.I. (1985). Trace elements in manic depressive psychosis. *J Affect Disord.*, **8**(2), 131–6.
2. Woo W H, Yang H, Wong K P, Halliwell B, (2003), Sulphite oxidase gene expression in human brain and in other human and rat tissues. *Biochem Biophys Res Commun.*, **305**(3), 619–23.
3. Johnson, J.L. and Rajagopalan, K.V. (1979). The oxidation of sulphite in animals systems. *Ciba Found Symp.*, **72**, 119–33.
4. Yamamoto, T., Moriwaki, Y., Takahashi, S., Tsutsumi, Z., Tuneyoshi, K., Matsui, K., Cheng, J. and Hada, T. (2003). Identification of a new point mutation in the human molybdenum cofactor sulferase gene that is responsible for xanthinuria type II. *Metabolism*, **52**(11), 1501–4.

Nickel

Nickel deficiency or imbalance may play a role in the symptoms of mood disorders. Observational and experimental studies have shown nickel to be associated with critical brain function 1,2,3,4,5,6,7,8,9,10.

REFERENCES

1. King, M.M. and Huang, C.Y. (1963). Activation of calcineurin by nickel ions. *Biochem Biophys Res Commun.*, **114**(3), 955–61.
2. Raos, N. and Kasprzak, K.S. (1989). Allosteric binding of nickel(II) to calmodulin. *Fundam Appl Toxicol.*, **13**(4), 816–22.
3. Lian, Q., Ladner, C.J. *et al.* (2001). Selective changes of calcineurin (protein phosphatase 2B) activity in Alzheimer’s disease cerebral cortex. *Exp Neurol.*, **167**(1), 158–65.
4. Mukai H, Ito A, Kishima K, Kuno T, Tanaka C, (1991), Calmodulin antagonists differentiate between Ni(2+)- and Mn(2+)-stimulated phosphatase activity of calcineurin. *J Biochem (Tokyo)*, **110**(3):402-6.
5. Tokoyoda, K., Takemoto, Y., Nakayama, T., Arai, T. and Kubo, M. (2000). Synergism between the calmodulin-binding and autoinhibitory domains on calcineurin is essential for the induction of their phosphatase activity. *J Biol Chem.*, **275**(16), 11728–34.
6. Pallen, C.J. and Wang, J.H. (1986). Stoichiometry and dynamic interaction of metal ion activators with calcineurin phosphatase. *J Biol Chem.*, **261**(34), 16115–20.
7. Matsui, H., Pallen, C.J., Adachi, A.M., Wang, J.H. and Lam, P.H. (1985). Demonstration

of different metal ion-induced calcineurin conformations using a monoclonal antibody. *J Biol Chem.*, **260**(7), 4174–9.

8. Chemin, J., Nargeot, J. and Lory, P. (2002). Neuronal T-type alpha 1H calcium channels induce neuritogenesis and expression of high-voltage-activated calcium channels in the NG108-15 cell line. *J Neurosci.*, **22**(16), 6856–62.
9. Mancinella, A. (1991). Nickel, an essential trace element. Metabolic, clinical and therapeutic considerations. *Clin Ter.*, **138**(3–4), 159–65.
10. Pallen, C.J, and Wang, J.H. (1984). Regulation of calcineurin by metal ions. Mechanism of activation by Ni²⁺ and an enhanced response to Ca²⁺/calmodulin. *J Biol Chem.*, **259**(10), 6134–41.

Phosphorus

Phosphorus deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between phosphorus and anxiety 1,2,3 and ADHD 4.

REFERENCES

1. Webb, W.L. and Gehi, M. (1981). Electrolyte and fluid imbalance: Neuropsychiatric manifestations. *Psychosomatics*, **22**(3), 199–203.
2. Balon, R. *et al.* (1988). Relative hypophosphatemia in patients with panic disorder. Letter. *Arch Gen Psychiatry*, **45**, 294–5.
3. Gorman, J.M. *et al.* (1986), Blood gas changes and hypophosphatemia in lactate-induced panic. *Arch Gen Psychiatry.*, **43**, 1067–71.
4. Stolley, H. *et al.* (1979), [Remarks concerning the treatment of hyperkinetic children with the so-called “diet poor in phosphate.”] *Monatsschr Kinderheilkd*, **127**(7), 450–3.

Potassium

Potassium deficiency or imbalance plays a role in the symptoms of mood disorders 1. Observational and experimental studies have shown an association between potassium and aggression 2,3,4, anxiety 5, bipolar disorder 6,7,8,9, and depression 10,11.

REFERENCES

1. Webb, W.L. and Gehi, M. (1981). Electrolyte and fluid imbalance: Neuropsychiatric manifestations. *Psychosomatics*, **22**(3), 199–203.
2. William J. Walsh (1983). analytical chemist, Argonne National Laboratory – reported in *Sci News*, **124**, 122–5.
3. Schmidt, K., Wier, W.R. and Asch, M. (1981). Clinical ecology treatment approach for juvenile offenders. *J Behav Ecology: Biosocial*, **2**(1).
4. Von Hilsheimer, G., Philpott, W., Buckley, W. and Klotz S.C. (1977). Correcting the incorrigible. A report on 229 “incorrigible” adolescents. *Am Lab*, **107**, 22–49.

5. McCleane, G.J. and Watters, C.H. (1990). Pre-operative anxiety and serum potassium. *Anaesthesia*, **45**(7), 583–5.
6. Klemfuss, H. (1995). Dietary potassium effects on lithium concentration and toxicity in humans. *Biol Psychiatry*, **37**, 42–7.
7. Jefferson, J.W. (1992). Potassium supplementation in lithium patients: a timely intervention or premature speculation? *J Clin Psychiatry*, **53**, 10.
8. Bkaskara Rao Tripuraneni, fellow in child psychiatry, Harbo-UCLA Medical Center, Torrance, California – reported in *Clin Psychiatry News* **18**(10), 3, October, 1990 and presented to the 143rd Annual Mtg of the Am Psychiatric Assoc, May 12–17, 1990, Abstracts NR 100 and NR 210.
9. Cater, R.E. (1986). The use of sodium and potassium to reduce toxicity and toxic side effects from lithium. *Med Hypotheses*, **20**(4), 359–83.
10. Webb, W.L. and Gehl, M. (1981). Electrolyte and fluid imbalance: Neuropsychiatric manifestations. *Psychosomatics*, **22**(3), 199–203.
11. Cox, J.R. *et al.* (1971). Changes in sodium, potassium and fluid spaces in depression and dementia. *Gerontology Clin.*, **13**, 232–45.

Selenium

Selenium deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between selenium and anxiety 1, depression 2,3, and schizophrenia 4,5,6,7,8,9,10.

REFERENCES

1. Benton, D. and Cook, R. (1991). The impact of selenium supplementation on mood. *Biol Psychiatry*, **29**(11), 1092–8.
2. Hawkes, W.C. and Hornbostel, L. (1996). Effects of dietary selenium on mood in healthy men living in a metabolic research unit. *Biol Psychiatry*, **39**, 121–8.
3. Benton, D. and Cook, R. (1991). The impact of selenium supplementation on mood. *Biol Psychiatry*, **29**(11), 1092–8.
4. Brown, J.S. Jr. (1994). The role of selenium and other trace elements in the geography of schizophrenia. *Schizophr Bull*, **20**(2), 387–98.
5. Foster, H.D. (1990). Schizophrenia and esophageal cancer: comments on similarities in their spatial distributions. *J Orthomol Med*, **5**(3), 129–34.
6. Foster, H.D. (1988) The geography of schizophrenia: possible links with selenium and calcium deficiencies, inadequate exposure to sunlight and industrialization. *J Orthomol Med.*, **3**(3), 135–40.
7. Alertsen, A.R., Aukrust, A. and Skaug, O.E. (1986). Selenium concentrations in blood and serum from patients with mental diseases. *Acta Psychiatr Scand.*, **74**(2), 217–19.
8. Buckman, T.D., Kling, A.S., Eiduscon, S. *et al.* (1987). Glutathione peroxidase and CT scan abnormalities in schizophrenia. *Biol Psychiatry*, **22**(11), 1349–56.
9. Abdalla, D.S., Monteiro, H.P., Oliveira, J.A, and Bechara, E.J. (1986). Activities of superoxide dismutase and glutathione peroxidase in schizophrenic and manic-depressive patients. *Clin Chem.*, **32**(5), 805–7.
10. Berry, T. (1994), A selenium transport protein model of a sub-type of schizophrenia. *Med Hypotheses*, **43**(6), 409–14.

Vanadium

Vanadium deficiency or imbalance may play a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between vanadium and bipolar disorder 1,2,3,4,5,6,7,8,9,10, 11,12,13,14,15,and depression 16,17.

REFERENCES

1. Dick, D.A., Naylor, G.J. and Dick, E.G. (1982). Plasma vanadium concentration in manic-depressive illness. *Psychol Med.*, **12**(3), 533–7.
2. Naylor, G.J., Smith, A.H., Bryce-Smith, D. and Ward, N.I. (1984). Tissue vanadium levels in manic-depressive psychosis. *Psychol Med.*, **14**(4), 767–72.
3. Naylor, G.J., Smith, A.H., Bryce-Smith, D. and Ward, N.I. (1984). Elevated vanadium content of hair and mania. *Biol Psychiatry*, **19**(5), 759–64.
4. Dick, D.A. *et al.* (1981). Plasma vanadium concentrations in manic-depressive illness. *J Physiol.*, **310**, 27.
5. Witkowska, D. and Brzezinski, J. (1979). Alteration of brain noradrenaline, dopamine and 5-hydroxy-tryptamine levels during vanadium poisoning. *Pol J Pharmacol Pharm.*, **31**, 393–8.
6. El-Mallakh, R.S. (1983). The Na, K-ATPase hypothesis for manic-depression. General considerations. *Med Hypotheses*, **12**(3), 253–68.
7. Cantley, L.C. *et al.* (1977). Vanadate is a potent (Na, K)-ATPase inhibitor found in ATP derived from muscle. *J Biol Chem.*, **252**, 7421–3.
8. Vanadium, vitamin C and depression (1981), *Nutr Rev.*, **40**(10), 293–5.
9. Naylor, G.J. and Smith, A.H.W. (1981). Defective genetic control of sodium-pump density in manic-depressive psychosis. *Psychol Med.*, **11**, 257–63.
10. Naylor, G.J., Dick, D.A., Johnston, B.B. *et al.* (1981). Possible explanation for therapeutic action of lithium, and a possible substitute (methylene-blue). Letter. *Lancet*, **ii**, 1175–6.
11. Campbell, C.A., Peet, M., Ward, N.I. (1988). Vanadium and other trace elements in patients taking lithium. *Biol Psychiatry*, **24**(7), 775–81.
12. Naylor, G.J. (1985). Reversal of vanadate-induced inhibition of Na-K, ATPase. A possible explanation of the therapeutic effect of carbamazepine in affective illness. *J Affect Disord.*, **8**(1), 91–3.
13. Naylor, G.J. and Smith, A.H.W. (1981). Defective genetic control of sodium-pump density in manic-depressive psychosis. *Psychol Med.*, **11**, 257–63.
14. Naylor, G.J. and Smith, A.H.W. (1981). Vanadium: A possible aetiological factor in manic-depressive illness. *Psychol Med.*, **11**, 249–56.
15. Witkowska, D. and Brzezinski, J. (1979). Alteration of brain noradrenaline, dopamine and 5-hydroxy-tryptamine levels during vanadium poisoning. *Pol J Pharmacol Pharm.*, **31**, 393–8.
16. Naylor, G.J., Corrigan F.M., Smith, A.H. *et al.* (1987). Further studies of vanadium in depressive psychosis. *Br J Psychiatry*, **150**, 656–61.
17. Ali, S.A. *et al.* (1985). Blood levels of vanadium, caesium, and other elements in depressive patients. *J Affect Disord.*, **9**(2), 187–91.

Zinc

Zinc deficiency or imbalance plays a role in the symptoms of mood disorders. Observational and experimental studies have shown an association between zinc and aggression 1,2,3,4,5,6, ADHD 7,8,9,10, 11,12,13, depression 14,15,16,17,18,19, and premenstrual syndrome 20,21,22,23,24.

REFERENCES

1. Walwork, J.C. *et al.* (1984). Distribution of minerals and catecholamines in rat brain: effects of zinc deficiency, in *The Neurobiology of Zinc, Part B: Deficiency, Toxicity and Pathology* (Neurology and Neurobiology V. 11B). New York, Ed. Alan R. Liss, pp. 49-64.
2. Prasad, A.S. (1983). Clinical manifestations of zinc deficiency. *Nutr Rev.*, **41**(7), 197.
3. Schauss, A.G. *et al.* (1979). A critical analysis of the diets of chronic juvenile offenders, Part II. *J Orthomol Psychiatry*, **8**(4), 222-6.
4. Walsh, W.J. (1983). Analytical Chemist, Argonne National Laboratory – reported in *Sci News*, **124**, 122-5.
5. Von Hilsheimer, G., Philpott, W., Buckley, W. and Klotz, S.C. (1977), Correcting the incorrigible. A report on 229 “incorrigible” adolescents. *Am Lab.*, **107**, 22-49.
6. Walsh, W.J. (1995). Zinc deficiency, metal metabolism, and behavior disorders. Unpublished monograph. *Health Research Institute*, 1804 Centre Point Dr., Suite 106, Naperville, IL 60503.
7. Bennett, C.P.W., McEwen, L.M., McEwen, H.C. and Rose, E.L. (1988), The Shipley Project: treating food allergy to prevent criminal behavior in community settings. *J Nutr Environ Med.*, **8**, 77-83.
8. Ward, N.I. (1997). Assessment of chemical factors in relation to child hyperactivity. *J Nutr Environ Med.*, **7**, 333-42.
9. Bekaroglu, M., Aslan, Y., Gedik, Y. *et al.* (1996). Relationships between serum free fatty acids and zinc, and attention deficit hyperactivity disorder: a research note. *J Child Psychol Psychiatry*, **37**(2), 225-7.
10. Koziolec, T., Starobrat-Hermelin, B. and Kotkowiak, L. (1994). [Deficiency of certain trace elements in children with hyperactivity.] *Psychiatr Pol.*, **28**(3), 345-53.
11. Ward, N.I. *et al.* (1990). The influence of the chemical additive tartrazine on the zinc status of hyperactive children – a double-blind placebo-controlled study. *J Nutr Med.*, **1**, 51-7.
12. Arnold, L.E., Votalato, N.A., Kleykamp, D. *et al.* (1990) Does hair zinc predict amphetamine improvement of ADD/hyperactivity? *Int J Neuosci.*, **50**(1-2), 103-7.
13. Barlow, P.J. (1983). A pilot study on the metal levels in the hair of hyperactive children. *Med Hypotheses*, **11**(3), 309-18.
14. Golub, M.R., Takeuchi, P.T., Keen, C.L. *et al.* (1996). Activity and attention in zinc-deprived adolescent monkeys. *Am J Clin Nutr.*, **64**, 908-15.
15. Tasman-Jones, C. (1980). Zinc deficiency states. *Adv Intern Med.*, **26**, 97-114.
16. Maes, M., Vandoolaeghe, E., Neels, H. *et al.* (1997) Lower serum zinc in major depression is a sensitive marker of treatment resistance and of the immune/inflammatory response in that illness. *Biol Psychiatry*, **42**(5), 349-58.
17. Maes, M., D’Haese, P.C., Scharpe, S. *et al.* (1994). Hypozincemia in depression. *J Affect Disord.*, **31**(2), 135-40.
18. Narang, R.L., Gupta, K.R., Narang, A.P. and Singh, R. (1991). Levels of copper and zinc in depression. *Indian J Physiol Pharmacol*, **35**(4), 272-4.

18. McLoughlin, I.J. and Hodge, J.S. (1990). Zinc in depressive disorder. *Acta Psychiatr Scand.*, **82**(6), 451–3.
19. Little, K.Y., Castellanos, X., Humphries, L.L. and Austin J, (1989), Altered zinc metabolism in mood disorder patients. *Biol Psychiatry*, **26**, 646–8.
20. Posaci, C., Erten, O., Uren, A. and Acar, B. (1994). Plasma copper, zinc and magnesium levels in patients with premenstrual tension syndrome. *Acta Obstet Gynecol Scand.*, **73**(6), 452–5.
21. Choung, C. and Dawson, E. (1994). Zinc and copper levels in premenstrual syndrome. *Fert Steril.*, **62**, 313–20.
22. Mira, M., Stewart, P.M. and Abraham, S.F. (1998). Vitamin and trace element status in premenstrual syndrome. *Am J Clin Nutr.*, **47**(4), 636–41.
23. Stewart, A. (1998). Clinical and biochemical effects of nutritional supplementation on the premenstrual syndrome. *J Reprod Med*, **32**, 435–41.
24. Chuong, C.J. *et al.* (1991). Baylor College of Medicine, Houston – presented at the 46th Annual Mtg. of the Am. Fertility Society, Washington, DC.

