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Editorial

Money Well Spent?

Recently Senator Michael Kirby called for a major increase in funding of psychiatric research in Canada. There have been no objections so far, especially from the psychiatrists who have always been very happy with new funds. I first noted this in the 1950s when the public began to demand that mental hospital superintendents do a much better job in treating the mentally ill in their charge. The standard reply was "Give us the money and we will do it." More money was poured into this herculean task of cleaning the stables that had been fouling for so many years. But it was very strange that even as the amount of money increased dramatically the percentage of patients who recovered did not get any better. The main effect of deinstitutionalization was the dumping of these patients from prison-like hospitals to the prisons and the streets where they still wander about today.

I conducted a comparison of over 100 schizophrenic patients I treated in a nursing home in Saskatoon, which cost 25 dollars per day, with treatment outcomes of similar patients treated in the University Hospital in Saskatoon. The cost at the University Hospital, the best in the province, a teaching hospital, was 80 dollars. The outcome was the same. I concluded that of the four important aspects of good treatment—shelter, food, kindness and medical care—that the medical care was the most important even though the other three factors had to be above what had been provided. Increasing the number of caring staff, which was the main difference, did not improve to the recovery rate. More money was not the answer. Better treatment was.

In my book *Schizophrenia, Yesterday (1950) and Today (2007), From Despair to Hope: With Orthomolecular Psychiatry* (In Press, Trafford, Victoria) I concluded that the results of treatment which depends on the use of toxic, prohibitively expensive, anti psychotics are no better than the results we saw in 1950 when the treatment

was incarceration. The evidence for this conclusion is given in the book and is based on the published conclusions of almost every paper which described outcomes. Fewer than ten percent of modern schizophrenic patients recover to the level at which they can work and pay income tax and psychiatrists do not expect them to. Here is what insider Gwen Olsen wrote in her book, *Confessions of an Rx Drug Pusher*:

"For fifteen years I served the interests of the pharmaceutical industry with dedication, loyalty and fierce competitiveness. However, a series of events over the years awakened an awareness in me that something was very wrong, indeed, very dangerous about the industry and their practices.

In particular, my experience selling psychiatric drugs as a hospital rep in which I was the caterer of the foods being consumed at hospital Grand Rounds and Mortality and Morbidity Conferences taught me that full disclosure was not being practiced with psychiatric drugs and doctors were fully aware that the therapeutics often used for mental illness could control patients but not heal them. I was trained by the industry that this phenomenon is known as the "revolving door syndrome"...There was no such thing as recovery with biochemical psychiatry, Pure and simple. These patients were customers for life and most, if not all, would suffer permanent and progressive brain damage from their treatments and medications. The drugs were also known to be highly addicting and difficult for patients to withdraw from yet patients were never informed of that fact".

Ford Motor Car company recently reported it lost billions of dollars over the past few years. Would throwing more billions into their coffers make things any better if they continued to build cars that are no longer wanted? They announced the only move able to allow them to survive. The bottom line drives industry. But it has no effect in medicine, especially in psychia-

try. Should we throw more money into psychiatry while it continues to follow the same old methods it has been following for decades, depending more and more on drugs which become more and more toxic and expensive, and which have led to the present dismal treatment results of the seriously mentally ill?

We do need much more money for research but only if that money is efficiently used to study alternative methods of treatment such as orthomolecular psychiatry. I think we should not provide more money until that new research is directed rationally and is not used to do more research for Big Pharma. Linus Pauling, in his definition of orthomolecular psychiatry, clearly showed that no xenobiotic (foreign) molecule will ever replace an orthomolecular substance that is normally found in and needed by the body. The chemical reactions in the cells are enormously complex and depend on the availability of these normal constituents such as vitamins, minerals, amino acids and essential fatty acids, in the correct concentration.

To visualize the complexity of these reactions, Roger Williams likened the cell to an orchestra. Each essential nutrient is like one member of the orchestra. A superb symphonic performance is a function of the quality of the musicians, a good conductor and all reading the same music. These are what the public demands. However, suppose during the performance the solo

violinist faints. The conductor believes the show must go on, so calls upon the lead drummer to replace the violinist. We will no longer hear a symphony; it will be a cacophony. Recently, young Julian Kuerti, assistant conductor of the Boston Symphony, learned that the scheduled pianist for the evening's performance was not able to appear. He called upon his father, renowned pianist Anton Kuerti, who was in town for the concert. The performance was superb. But there is only one Anton Kuerti. In the cells of the body each nutrient has been selected by evolution to be like an Anton Kuerti. If thiamin is removed from the cell only another thiamin can replace it, and until this is done the cell will not perform. Giving a patient a xenobiotic to replace what is missing is like replacing the violinist or pianist with the drummer. It would be like replacing Anton Kuerti with me—I would have to run for my life! The orthomolecular law is that xenobiotics cannot replace missing orthomolecular substances. The drug companies are wasting our money looking for something they will never find.

Until the profession makes a firm decision to examine very seriously alternative treatment methods I would not give them a penny. This poem, with its Part II added by Herbert Nehrlich, so well describes modern psychiatry.

—Abram Hoffer, M.D., Ph.D.
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The Ambulance Down In The Valley

by Joseph Malins (1895) – A poem about prevention

'Twas a dangerous cliff, as they freely confessed,
though to walk near its crest was so pleasant;
but over its terrible edge there had slipped
a duke and full many a peasant.
So the people said something would have to be done,
but their projects did not at all tally;
some said, "Put a fence 'round the edge of the cliff",
some, "An ambulance down in the valley."

But the cry for the ambulance carried the day,
for it spread through the neighboring city;
a fence may be useful or not, it is true,
but each heart became full of pity
for those who slipped over the dangerous cliff;
And the dwellers in highway and alley
gave pounds and gave pence, not to put up a fence,
but an ambulance down in the valley.

"For the cliff is all right, if you're careful," they said,
"and if folks even slip and are dropping,
it isn't the slipping that hurts them so much
as the shock down below when they're stopping."

So day after day, as these mishaps occurred,
quick forth would those rescuers sally
to pick up the victims who fell off the cliff,
with their ambulance down in the valley.

Then an old sage remarked: "It's a marvel to me
that people give far more attention
to repairing results than to stopping the cause,
when they'd much better aim at prevention.

"Let us stop at its source all this mischief." cried he,
"come, neighbors and friends, let us rally;
if the cliff we will fence, we might almost dispense
with the ambulance down in the valley."

Part II by Herbert Nehrlich

So the townspeople met at the top of the cliff
where the workmen put up a strong fence,
woven wire and posts that were hardy and stiff
and they lauded each other's good sense.

For a week the fence stood and no ambulance came
then one morning they woke up to see
that the fence had been cut from the cliff to the tree
in the valley they stood with their shame.

Said a voice from the sky, and they knew it was God,
"If you keep people healthy at all,
there are forces objecting as they find it quite odd
when no earls and no peasants do fall."

And instead of a fence on the edge of the cliff
they had placed at the bottom a pool,
where they'd land in the water, not ending up stiff
but each victim was seen as a fool.

And to face their disease that had caused the neglect
they would get a big bucket of pills,
though the cost of it all would not nearly reflect
that they'd taken the fence from the hills.

But the pharmacist said "It's the minds of all men,
they are missing the atoms of dope,"
and that medicine taken again and again
was the modern way's spirit of hope.

The sage who had said that the fence should be built
then spoke up, from the cliff near the edge
but the white coated doc said it must be the guilt
and he gave to the people this pledge:

"You will no longer be in the danger to fall
from the cliff, neither earl nor a peasant,
as the ordinance says that the citizens, all,
won't be wandering near any crescent."

And the sage on the edge while addressing the town
said "They're neither your neighbour nor friend."
Both the doc and his buddy then pushed the sage down
off the cliff. Thus the story does end.

American Medical Revolutions

About 170 years ago our ancestors forced the repeal of licensing laws which had created a monopoly over the practice of medicine for orthodox physicians. Ordinary people, farmers, artisans, tradesmen and others got together and forced politicians to act on their behalf. They were tired of bloodletting, and harsh medications like mercury compounds that ruined their teeth and weakened their bodies. They opted for kinder and gentler alternatives with lower casualty rates, particularly the newly introduced homeopathy. They were impressed that tiny doses of medicine were able to cure cholera much better than the massive doses used by orthodox physicians.

Homeopathy, introduced in America in 1825, was a brand new medical discipline developed by a German physician named Samuel Hahnemann (1744-1843). He was disillusioned with the results of medical practices of his day. He stopped practicing and began to study the effects

of medicine on a healthy person, himself. He tried quinine, a very popular medication, first. It caused symptoms of malaria, the disease which it was able to cure. Similarly mercury produced symptoms of syphilis on which it had therapeutic effects. This experimental evidence led to an assumption: substances which produce symptoms in healthy people can have a curative effect on sick people who experience the same symptoms. Extensive experimentation with his family and friends resulted in collection of the symptomology of 27 medications. With this information he was able to investigate the validity of his hypothesis.

Returning to the practice of medicine he found that clinical experience validated his hypothesis. By this means his hypothesis became a theory in accordance with scientific methodology. Ultimately, confirmed by other investigators, it became the law of similars.

Subsequently experimentation with varying doses disclosed that small amounts of medicines had more effect on the diseases of patients than large amounts. This experimental evidence led him to conclude that his medications were stimulating the inherent healing powers of his patients. They were getting well without the damaging side effects of excessive amounts of medicines.

Many orthodox physicians in Germany, observing Hahnemann's successes, sought training in the application of the new doctrines and began to practice homeopathy - generating a new school of medicine in the process. It became popular all across Europe. Homeopathic physicians began treating the royalty and nobility of Europe.

Homeopathic physicians didn't try to find the cause of diseases. They spent a lot of time identifying symptoms in considerable detail since each patient was considered to be unique. The symptoms defined the disease. Matching the symptoms of

the patient with the symptoms associated with medications was not an easy job. Intelligence, training and dedication were required to achieve the full benefits of homeopathic technology. Ultimately some homeopaths limited themselves to the use of low potency medications while the most effective practitioners used the high potency variety, those with the highest dilutions.

Hahnemann did not claim to have discovered the law of similars. The therapeutic systems of empiric physicians in ancient Greece and Paracelsus had included this theory. The important discovery that medicinal substances could be more active at high dilutions was his alone and he was vilified because of it. Those whose incomes depended on the sale of large quantities of drugs found it economically damaging. Orthodox physicians, whose use of excessive amounts of mercury caused their patients to lose teeth and deteriorate physically, hated it as a serious threat to their physical safety as well as their professional reputation. But many physicians trained in the orthodox tradition abandoned it and took up the practice of homeopathy with great success.

Success of homeopathic treatments with camphor, copper sulfate and *Veratrum album*, recommended by Hahnemann during the Asiatic cholera epidemic in Europe in 1832, firmly established homeopathy in France. When Hahnemann arrived in Paris in 1835 he was granted a license to practice medicine within 6 months. He subsequently cured the Marquess of Anglesea of tic de leureux which French physicians had been trying unsuccessfully to cure for 20 years. After losing prestige and patients to the homeopaths, member of the French National Academy of Medicine called them knaves, ignoramuses, charlatans and quacks. Nevertheless orthodox physicians adopted camphor, copper sulfate and *Veratrum album* as remedies for cholera.

American homeopaths were as successful treating cholera in the 1830s as the French homeopaths. They added to their reputation when in 1978 a yellow fever epidemic spread from New Orleans into the Mississippi Valley with alarming death rates: 4,600 of 27,000 cases in New Orleans, 5,000 out of 18,500 cases in Memphis with a total of 15,934 death out of 74,265 cases reported in the Mississippi Valley. Homeopathic physicians in New Orleans had treated 1,945 cases with loss of 110. In the rest of the south they had treated 1,969 cases with loss of 151–7.7%. The overall death rate for reported cases in the south was at least 16%. The French Government awarded a gold medal to a French homeopath for his work during the New Orleans epidemic. Homeopaths were popular!

Insurance companies began offering reduced rates to persons employing homeopathic physicians and homeopathic life insurance companies were being chartered. In 1870 the Homeopathic Life Office of New York reported that it had sold 7,927 policies to followers of homeopathy and 2,258 to other; 84 deaths in the first category and 66 in the second justified the lower premiums charged to the former.

As a result of these successes by 1892, homeopaths in the United States controlled about 110 hospitals, 145 dispensaries, 62 orphan asylums and old peoples homes, over 30 nursing homes and sanitarium and 16 insane asylums.

In 1889 the Westborough, Massachusetts insane asylum was run by homeopaths and the Springfield Republican reported that the cost of maintenance is much less and recoveries and general success greater than in allopathic asylums.

Meanwhile competing medical technologies and an oversupply of physicians drastically reduced the income and status of about 110,000 orthodox physicians. An average one earned \$750 per year in 1900

and about 40 per year committed suicide because of financial difficulties. But about 15,000 homeopathic physicians prospered and 26 schools of homeopathy flourished at the end of the century. Unsuspecting homeopaths, fully occupied with their lucrative practices, gave grudging support to their own organization not realizing that they were in danger.

Orthodox physicians at the American Medical Association (AMA) plotted their downfall. The first objective was reduction in the number of medical schools and medical students. This had been a cherished goal since 1846 when the founding convention of the AMA occurred.

Politically astute George Simmons, M.D. who graduated from Hahnemann Medical College of Chicago in 1882 and later attended Rush Medical School, was appointed secretary of the AMA and editor of its journal (JAMA) in 1899. Soon thereafter he was appointed secretary of a committee to consider reorganization. In 1901 a reorganized AMA changed from a loose federation of independent professionals into a political powerhouse. The reorganization substantially reduced the influence of individual physicians who had been objecting to unethical drug company advertising.

In 1904 the AMA established a Permanent Council on Medical Education. In 1905 the Council arranged a conference of state medical licensing boards to review the status of medical education and set standards for medical schools. A temporary standard required four years of high school and 4 years of medical school and examination of graduates by state boards before licensing. In 1906, the committee inspected 160 medical schools, grading 82–A, 46–B and 32–C. Fifty schools agreed to require 1 year of college sciences courses for admission.

In 1907 Arthur D. Bevan, M.D., the Council's chairman, convinced Henry Pritchard, former President of MIT, who





now headed the Carnegie Foundation, to sponsor a study of medical education. That Foundation, founded in 1905 with the objective of upgrading the status of college teachers and creating a uniform system of higher education, was a logical ally. In November of that year the trustees approved the proposed study and Pritchard hired Abraham Flexner, an educator who had graduated from Johns Hopkins University, to work on the project.

Flexner headed for his alma mater's medical school, which he used as his standard of comparison. Accompanied by Nathan Caldwell, M.D., who replaced Bevan as Chairman of AMA's Council on Medical Education, Flexner made a comprehensive survey of medical schools in 1910. His opinions of most of the schools he visited and evaluated were not flattering. Harvard University was incensed at his opinion of their medical school which had been reorganized by Charles Elliot in 1870.

Flexner was convinced, probably by Dr. Caldwell, that Hahnemann and homeopathy were frauds, since this was the official opinion of the AMA which denied that homeopathy possessed therapeutic efficacy. Flexner also bought the opinion of William Osler, M.D. that "sectarian allopathy and homeopathy" were yielding to the new scientific medicine.

Flexner's famous report, coauthored by Nathan Caldwell, caused substantial changes. It started a process that empowered the AMA, disorganized the homeopaths and forced the closure of homeopathic medical schools. Even though John D. Rockefeller favored homeopathy and repeatedly insisted that it be supported, all of his money was spent on "scientific medicine". Frederick Gates who was influential in disbursing Rockefeller's money wrote that Hahnemann was insane. John D., Jr. told his father that the homeopaths were integrating with the

allopaths. Letter requests for funds from one homeopathic school were said to have been unanswered.

Scientific medicine was designed to be capital intensive. Requirements for teaching it increased costs beyond the capability of students to support the schools with tuition and fees. As a result schools, unable to supplement their income from other sources like grants and bequests, were forced to close or consolidate. In 1910 the number of medical schools was reduced from 166 to 131. Only 63 were left in 1929. In the 1930s and 1940s, 11 homeopathic schools closed. After 1930 even the Hahnemann Medical College of Philadelphia was teaching allopathic medicine except for one or two classes of homeopathy.

New laws gave the AMA the power to control what the schools taught. Curricula were heavy in the sciences, but there was only minimal training in nutrition and pharmacology. Physicians who used to make up their own remedies began to rely on pharmaceutical company formulations and for information on drugs. Production of physicians was substantially reduced. Competing medical sects, whose members had totaled less than 10% of all physicians, were all but emasculated.

Evidently our present unsatisfactory situation came about because the frustrated monopolists of the 1820s found a way to put themselves back in the driver's seat. They convinced upper and middle class people that they were scientists who could bring the benefits of science to their patients. At least \$300 million (\$600 million according to Harris Coulter's *The Divided Legacy*) contributed by wealthy donors, supplemented by an unknown amount funneled through the JAMA by the pharmaceutical industry and other advertisers, helped them regain control. At a time when one dollar bought a 10-hour day's work, this was an irresistible flood that carried the orthodox physicians back

into power and supported the monopoly for almost a hundred years.

Once in control, efforts to reduce competition and increase income have been unceasing. Physicians who practice alternative medicine, in competition with regular physicians, are subject to harassment. In the state of Washington about 30% of them are being harassed at this time. Those who make substantial advancements in medical science often find the Federal Government moving against them. The FDA and FTC have used taxpayer money to suppress new technology in a number of cases. Even State legislators have cooperated, in cases where other means failed,

The purpose of the new licensing laws was to protect the public but, in fact, monopolized medical care, according to reports, has been killing over 200,000 of us every year and promises to bankrupt the country. These laws are used to prevent free public access to less lethal, more effective and less expensive therapies. As Daniel Haley so eloquently wrote, in *Politics in Healing*, “we don’t need government protection from things that can’t hurt us”.

Medical science should be a search for the truth and many medical scientists have spent their lives in this search. Unfortunately scientific medicine, as practiced by the medical monopoly during the last century, has rejected the discoveries of a number of medical scientists. Too many promising technologies have been consigned to the dust bin of history. As a result, medical services are much more expensive than they should be and lower in quality than they could be. Less suppression and more competition can make people healthier at lower cost.

One hundred years of suppression of advancements in medical science is enough. Even physicians have been victimized. Their expensive schools don’t teach them about the suppressed science

and give them inadequate training in nutrition and therapeutics. We can do without the high prices and poor care. Let’s recover and apply the suppressed technology and reward, rather than discourage, innovations that promise lower costs and better quality care. Replace the medical monopoly with laws guaranteeing freedom of choice in medical care.

Again in 2008, as in the 1830s, orthodox medicine is killing lots of people and creating lots of invalids. The exorbitant price of \$2 trillion a year is too much. We owe it to ourselves and our descendants to reintroduce competition into the medical marketplace. Forcing the repeal of the Medical Practices Act will be a good start. The Access to Medical Treatment Act proposed in the 2000 session of Congress might also be resurrected.

– Jack Phillips
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When the Nutritional Supplements Stop: Evidence from a Double-blinded, HIV Clinical Trial at Mengo Hospital, Kampala, Uganda

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Introduction

Last year, in this journal,¹ the authors reported on the results obtained in a prospective randomized, double-blinded clinical trial, involving 310 HIV-infected outpatients of Mengo Hospital, Kampala, Uganda. In this registered trial (International Standardized Randomized Controlled Trial Number 42274642), patients with baseline CD4 cell counts above 200, who were not receiving anti-retroviral drugs, were placed randomly into Groups A and B. The medications given to patients in Group B consisted of 30 nutrients with a filler of organic sugar. This combination of nutrients had been found, in small open trials, to stimulate appetites in HIV-positive patients.² The logic behind this approach was to establish whether greater appetite was sufficient to encourage HIV-positive individuals to increase their consumption of local foods to a point at which enough selenium and amino acids were digested to normalize glutathione peroxidase levels. Serious loss of appetite is a common symptom of HIV/AIDS.³

The capsules taken by Group A patients contained the same thirty appetite stimulating supplements, but also included an additional seven nutrients.

The latter nutrients were designed to directly promote the body's production of glutathione peroxidase and, in addition to amino-acid rich desiccated beef liver, included L-selenomethionine, N-acetyl cysteine, L-glutamine, hydroxytryptophan, alpha lipoic acid and ascorbic acid. Patients receiving this mixture of supplements did not have to rely on their own diet to provide the selenium, cysteine, tryptophan and glutamine thought necessary to boost body glutathione peroxidase levels.⁴

Results of Initial Trial

The year long study¹ examined the effects of these two combinations of nutrients on biochemical and immunologic parameters, that is, serum glutathione peroxidase levels and CD4 cell counts as the study's primary endpoint. Secondary endpoints were weight changes and patient assessed quality of life.

The mean/median serum glutathione peroxidase levels in Group A (37 nutrients) increased from 3825/3628 IU/L (International Units) at baseline to 8894/8575 IU/L at the trial's end ($p < 0.000$). Similarly, patients in Group B (30 nutrients) had an increase in mean/median serum glutathione peroxidase levels from 3862/3602 to 9839/9203 IU/L over the length of the trial ($p < 0.000$). The mean/median CD4 cell counts rose from 400/347 mm^3 to 446/388 in Group A and from 400/335 to 446/394 mm^3 in Group B ($p < 0.000$). Mean weight increases over 52 weeks were 1.0 kg in Group A and 1.4 kg in Group B, while Karnofsky scores in Group A rose from 81 to 85 and in Group B from 82 to

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86. Wilcoxon Signed Ranks Test and the Sign Test both indicated that all these measured increases within both groups were statistically significant ($p < 0.01$). Indeed, for both glutathione peroxidase levels and CD4 cell counts $p < 0.000$ in all cases. There were, however, no statistically significant differences between the measured parameters, (serum glutathione peroxidase, CD4-cell counts, weight and Karnofsky scores) in Group A compared with those of Group B ($p > 0.05$), or between males and females ($p > 0.05$).

Post-Trial Results

The year-long double-blinded clinical trial was funded by the Canadian charity, The Friends of Mengo Hospital. This allowed information on serum glutathione peroxidase levels, body weights and Karnofsky scores to be assessed six months after the trial had formally ended. CD4 cell counts were also measured at the same time, as part of normal hospital procedures.

This additional data allowed an assessment of the health implications of discontinuing nutritional supplementation in HIV-positive patients. To illustrate, the initial double-blinded closed year long trial began with 310 patients, 263 of whom completed it (84.8%). At the end of the six month open, post-trial study, CD4 cell counts were available for 213 of these patients and serum glutathione peroxidase levels from 122. Karnofsky quality of life scores also had been collected for 191 of the former participants in the closed trial and body weight for 166.

Six months after the formal closed trial had ended, the mean/median serum glutathione peroxidase levels of the 124 assessed patients had fallen by 7117/5184 IU. ($p < 0.001$). During this time period, of course, former members of Group A and Group B had not been provided with any nutritional supplements. This decline in serum glutathione peroxidase levels in

this group was almost universal.

Similarly, in the 213 patients for whom CD4 cell counts were available at the end of the six month post-trial period, mean/median levels had fallen by 155/151 mm^3 respectively. ($p < 0.001$). Similarly, in the 191 patients that had their mean/median Karnofsky quality of life scores assessed six months after the closed trial had ended, these had fallen by 5.5/5.0 respectively ($p < 0.001$).

It is clear that dramatic falls in serum glutathione peroxidase levels, CD4 cell counts and Karnofsky scores had occurred during the six months in which nutrient mixtures A and B were no longer available to former closed-trial patients. Unfortunately, these losses during the post-trial period had more than negated all the gains in quality of health indicators that most patients had achieved during the nutritional trial itself. Additional analyses indicated that such declines had occurred in the post-trial period, regardless of the nutrient group to which the patient had formerly belonged. Similarly, gender difference made no statistically significant difference ($p > 0.05$).

The only exception to this generalization involved body weight. During the six month period nutrient supplements were not provided, former trial participants had lost relatively little weight. In the 166 patients for whom this measure was available, mean/median weight had only fallen by 0.61/0.00 kilograms ($p > 0.05$).

Conclusions

The initial prospective randomized, double-blinded clinical trial demonstrated that two nutrient mixtures taken for 52 weeks by HIV-positive patients who were receiving no anti-retroviral drugs, significantly slowed their decline into AIDS. The improvement was associated with increases in serum glutathione peroxidase levels, CD4 cell counts, body weight and improvements in quality of

life scores. In contrast, the post-trial data demonstrated that if such supplementation stops, HIV-positive patients suffer a rapid health decline. This involves highly statistically significant drops in serum glutathione peroxidase levels, CD4 cell counts and Karnofsky quality of life scores ($p < 0.001$). These results are very consistent with those of smaller open trials using nutritional supplement which have been conducted elsewhere in Sub-Saharan Africa.⁵ It seems clear that inadequate nutrition plays an extremely important role in the progression of HIV-infected patients into AIDS. These results also are consistent with Foster's model⁶ of the development of AIDS which suggests that deficiencies of glutathione peroxidase play a key role in the process.

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References

1. Namulema E, Sparling J, Foster HD: Nutritional supplements can delay the progression of AIDS in HIV-infected patients: results from a double-blinded, clinical trial at Mengo Hospital, Kampala, Uganda. *J Orthomol Med*, 2007; 22(3): 129-136.
2. Bradfield M, Foster, HD: The successful orthomolecular treatment of AIDS: accumulating evidence from Africa. *J Orthomol Med*, 2006; 21(4): 193-196.
3. Shabert JK, Winslow C, Lacey JM, et al: Glutamine-antioxidant supplementation increases body cell mass in AIDS patients with weight loss: a randomized double-blind control trial. *Nutrition*, 1999; 15(11-12): 860-864.
4. Moriorino M, Aumann KD, Brigelius-Flohe R et al: Probing the presumed catalytic trial of a selenium-containing peroxidase by mutational analysis. *Z Ernährungswiss*, 1998; 37(Supp.1): 118-121.
5. Foster HD: HIV/AIDS – A nutrient deficiency disease. *J Orthomol Med*, 2005; 20(2):67-69.
6. Foster HD: How HIV-1 causes AIDS: Implications for prevention and treatment. *Med Hypotheses*, 2004; 62(4): 549-553.

The Real Story of Vitamin C and Cancer

Steve Hickey Ph.D.;¹ Hilary Roberts, Ph.D.

Introduction

In the last couple of weeks, vitamin C and cancer has become a hot news topic. For people who have followed this matter, the media's sudden interest comes as something of a surprise: the evidence that vitamin C is a selective anticancer agent has been known for decades. This story is important, as it illustrates how the head-in-the-sand conventional view (that nutritional supplements are useless) can lead to restrictive legislation, reduced health, and limited approaches to the treatment of disease.

The recent news story arose from a study by researchers at the US National Institutes of Health (NIH).¹ The NIH experiment showed that, when injected into mice, vitamin C could slow the growth of tumours. The NIH paper presents its findings as new, ignoring the long history of research into vitamin C and cancer. Far from being novel, many of the findings reported in this paper have been recognized for decades. What is strange, however, is that the media suddenly decided to report a story they had ignored for so long.

A History

One strand of this story begins with the work of an old friend, Dr. Reginald Holman. In 1957, Holman published a paper in *Nature* about how hydrogen peroxide (the chemical Marilyn Monroe reportedly used on her hair) destroyed or slowed the growth of tumours in mice.² Reg Holman met with some hostility from the medical profession, which slowed his research and clinical work over the following half century. Nevertheless, scientists have known that hydrogen peroxide kills cancer cells for over fifty years.

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In 1969, when man first walked on the moon, researchers found that vitamin C would selectively kill cancer cells without harming normal cells.³ That finding meant that vitamin C was like an antibiotic for cancer: potentially a near perfect anticancer drug. Before 1970, it was known that vitamin C was an example of a new class of anticancer substances. However, the medical research establishment largely ignored these scientific results.

In the 1970s, some members of the public and pioneering doctors experimented with high doses of vitamin C to treat cancer. By 1976, double Nobel Prize winner Linus Pauling and Scottish surgeon Ewan Cameron reported clinical trials, showing an unparalleled increase in survival times in terminal cancer patients treated with vitamin C.⁴ However, by this time Pauling was considered a quack, having claimed that vitamin C could prevent or cure the common cold, so these apparently amazing findings made little impact.

Cameron and Pauling published a second report in 1978.⁵ The Mayo Clinic responded with a study that suggested vitamin C had no effect, which the medical profession readily accepted, perhaps because it confirmed existing prejudices. However, despite the Mayo Clinic study being "considered definitive,"¹ it was highly criticized from the start. In particular, it used relatively low oral doses for short periods, rather than the lifetime combination of high oral and intravenous (IV) doses in the Pauling and Cameron study. The Mayo Clinic refused to provide Pauling with their data so he could check it. When we emailed the Mayo Clinic with a similar request, we received no reply.

If Cameron and Pauling's work, back in the 1970s, had been just a single study,

it would have been interesting and suggestive. Such a large increase in survival time demands a proper scientific follow-up and, indeed, other studies soon backed up the findings. Japanese researchers found similar survival times,⁶ apparently confirming Pauling's early results. Subsequently, Dr. Abram Hoffer, working in Canada, provided more evidence that vitamin C could enable cancer patients to live much longer. We have analyzed these results and found them to be statistically valid. They are not explicable by placebo effect or by a simple biased selection of long-lived patients. Moreover, over the last three decades, a large number of clinical and anecdotal patient reports support the claims.

A long time before the NIH's mouse experiment, Pauling also studied the effects of vitamin C on cancer in mice. He worked with Dr. Art Robinson but, unfortunately, the two researchers fell out over their interpretations of the results. Robinson left the Linus Pauling Institute (which he had helped establish) and completed the experiment alone. It was eventually published in 1994.⁷ The results were outstanding: mice with cancer that were given high dose vitamin C in the diet, or fed a diet of raw vegetables, lived up to 20 times longer than controls. Translated into human terms, this might mean that a person with one year to live might get an extra 20. Importantly, Robinson and Pauling had been inspired to do this experiment by claims from cancer sufferers in the popular literature.

Doctors Hugh Riordan, Ron Hunninghake, Jim Jackson, Jorge Miranda-Massari, Michael Gonzalez and others in the Center for the Improvement of Human Functioning, Inc., did the core research on vitamin C and cancer. They repeated and extended the early work, which had showed vitamin C would selectively kill cancer cells. They have years of experience of treating cancer patients with high dose

vitamin C. Their work is consistent with results from independent researchers and doctors worldwide.⁸

The authors of this article recently reviewed the literature on vitamin C and cancer, in our book *Cancer: Nutrition and Survival*.⁸ We found solid evidence that vitamin C, in high enough doses, acts as a selective anticancer drug. In healthy tissues, vitamin C is an antioxidant, while in cancer it acts as an oxidant generating free radicals and killing the abnormal cells. Furthermore, an understanding of its action provides insight into the cancer development process. Oxidants, such as hydrogen peroxide, are able to make cells grow and divide erroneously. So, as the cells divide, they form a population of varying cells that compete with each other for survival. It was immediately clear that oxidation could explain how cancer starts; following which Darwin's theory of evolution takes over. Given enough time, cells divide and the "fittest" are selected. In this context, the fittest to survive are those cells that grow rapidly to form an invasive cancer. Cancer is not a mysterious disease but is a result of straightforward biological processes.

This microevolutionary model for cancer makes highly specific predictions. One is that high dose vitamin C should prevent cancer and even higher doses should kill cancer cells. The model also predicts that there could be thousands of selective anticancer drugs. Animals, and especially plants, will contain these substances, because they evolved in the presence of cancer and had to develop ways to control it. If such predictions are correct, we should find a multitude of safe anticancer agents in food. Checking against medical databases, we immediately found numerous examples, such as curcumin from turmeric, alpha-lipoic acid, and vitamin D₃. Everywhere we looked, we found substances with the predicted properties. Unfortunately, many

are the very supplements the Alliance for Natural Health (ANH) is trying to protect from being banned!

To conclude our history, the NIH paper was essentially a repeat of previous animal experiments. Despite this, the NIH authors appear not to have referenced many of the scientists who did the original work on vitamin C and hydrogen peroxide in cancer. Instead, they present their work as standing alone, in an informational vacuum: with the exception of the Cameron and Pauling clinical trial, the original scientists' work is not mentioned in the NIH text. Wrongly, a reader might gain the impression that the NIH's work was fundamentally original, rather than repeating the work of others. This might mislead the media into ascribing credit for the work on vitamin C and cancer to the NIH, which would be unfair to the real pioneers of this subject.

Intravenous or Oral?

Dr. Mark Levine of the NIH claims that "When you eat foods containing more than 200 milligrams of vitamin C a day—for example, 2 oranges and a serving of broccoli—your body prevents blood levels of ascorbate from exceeding a narrow range."⁹ This statement is demonstrably false (the NIH's own data refutes it) and is an artefact of the way the NIH group interpret their experiments.

In their mouse paper, the NIH used intravenous vitamin C, rather than oral. To be more accurate, the NIH used intravenous ascorbate. Sodium ascorbate is normally used for injection, as vitamin C (ascorbic acid) can cause local inflammation at the injection site. The results they obtained are suggestive of a response, but do not show the same large effects reported by Robinson. Robinson fed his mice dietary vitamin C, in very high doses. Thus, the NIH's suggestion that only intravenous vitamin C is useful as an anticancer agent does not appear to fit

the animal data. Likewise, the idea that only intravenous vitamin C is effective against cancer does not fit the clinical data. Abram Hoffer, for example, used oral doses and obtained essentially the same results as Cameron and Pauling.

The NIH's insistence that the body has "tight controls," which prevent oral vitamin C from functioning as an anticancer agent, is wrong. In our book *Ascorbate: The Science of Vitamin C*, we have shown that the NIH claims for blood "saturation" at a low level (70 μ M/L) are incorrect.¹⁰ The NIH authors never admitted this error, despite a long email correspondence between Hickey and Levine. However, they have changed the wording they use, from "saturated" to "tight controls," and increased the level by about three times (to 200 μ M/L). It would appear that they are holding onto an outdated idea about how vitamin C acts in the body. As an alternative, we have proposed a dynamic flow model, in which, at high doses, vitamin C flows through the body, providing antioxidant support, potentially preventing cancer growth and killing cancer cells.¹¹

Dynamic Flow

Dr. Mark Levine claims:

"Clinical and pharmacokinetic studies conducted in the past 12 years showed that oral ascorbate levels in plasma and tissue are tightly controlled. In the case series, ascorbate was given orally and intravenously, but in the trials ascorbate was just given orally. It was not realized at the time that only injected ascorbate might deliver the concentrations needed to see an anti-tumor effect."⁹

As we have explained, there is no evidence for such tight control. The suggestion that the legendary scientist, Dr. Linus Pauling, or consultant surgeon, Ewan Cameron, did not know the difference between oral and intravenous administration¹² is bizarre and, again,

demonstrably incorrect.⁸ The difference between oral and intravenous vitamin C is, however, more complex than suggested by the NIH. Contrary to their conclusions, it is not clear that intravenous vitamin C necessarily provides an advantage over oral supplements in the treatment of cancer. There is a fair case for suggesting that high dose oral administration could be more effective.

At low intakes, the body prevents vitamin C from being lost through the urine; if this were not the case, we would all be at risk of acute scurvy. The body tries to retain a minimum of about 70 $\mu\text{M/L}$ of vitamin C in blood plasma. This level can be maintained with an intake as low as 200 mg a day. At higher doses, the body can afford to let some vitamin C escape in urine. This saves energy, which the kidneys would otherwise use to keep pumping the vitamin C molecules back into the blood. If dietary vitamin C is in plentiful supply, there is no need for our bodies to retain it all. So, at high doses, vitamin C flows through the body, being taken in from the gut and excreted in the urine. With such high intakes, the body has a reserve that it can call upon in times of need.

A single 5 gram dose of vitamin C can generate blood levels of about 250 $\mu\text{M/L}$; this is above the NIH paper's claimed maximum of 200 $\mu\text{M/L}$. Moreover, repeated large doses can sustain these levels. We have achieved vitamin C plasma levels above 400 $\mu\text{M/L}$, following a single dose of oral liposomal vitamin C.¹³ It seems that the claimed "tight control" concept will need revising again soon.

People vary in their responses to vitamin C. In some people, a single 2 gram oral dose of vitamin C may have a laxative effect. Our collaborator, Dr. Robert Cathcart, described this as the bowel tolerance level. Strangely, bowel tolerance has been observed to increase dramatically when a person is ill, say with the flu. A person

with a laxative effect at, say, 2 grams, may be able to tolerate 100 times more if they become ill. This increased bowel tolerance also occurs in cancer sufferers. It suggests that at times of stress or illness, the body absorbs extra vitamin C. When promoting intravenous vitamin C, the NIH authors have not considered the possibility of such increased bowel tolerance to oral doses.

To achieve the maximum blood plasma levels possible with oral vitamin C, a typical healthy person may need a total intake of about 20 grams, spread throughout the day (say 3 or 4 grams every four hours). However, cancer patients may require far more. Such massive intakes result in consistently high blood levels, which tumour tissues absorb, and which then generate the hydrogen peroxide that kills the cancer cells.

Other possible mechanisms for how vitamin C kills cancer cells¹⁴ are not covered by the NIH study. The NIH base their work on laboratory studies of mice, in which vitamin C kills cancer cells over the course of, perhaps, a couple of hours. Lower levels of vitamin C may simply take longer to kill the cells, which is a standard dose response relationship. Sustained oral doses can increase plasma vitamin C consistently, over periods measured in months or years: this may, in the end, be more effective than the short, sharp shock of intravenous therapy. Sustained levels also reduce the likelihood of tumours developing resistance to the therapy (analogous to bacterial resistance to antibiotics.)

Redox synergy

When combined with alpha-lipoic acid, selenium, vitamin K3, or a range of other supplements, vitamin C is a far more powerful anticancer agent than when used alone. Experimental data from Riordan and others shows that the cancer destroying effect of such combinations is much higher. We have described some of

these combinations in a recent book “*The Cancer Breakthrough*”.¹⁵ Strong scientific reasons suggest that such combinations, given orally, could provide cancer sufferers with a large increase in lifespan and increased quality of life.

Just as your doctor advises you to take a whole course of antibiotics continuously, until all infection is gone, vitamin C based redox therapy needs to be continuous. Like bacterial infections, cancers can rapidly become resistant to intermittent treatments. Typically, intravenous ascorbate is given at intervals, whereas oral ascorbate can maintain blood levels continuously and indefinitely. This is a valid medical reason to prefer an oral regime. Also, patients prefer the oral route, as they have greater control, lower cost, and are more involved in their treatment.

People often ask us what we would do, if we developed the disease. In the event that one of us developed a malignancy, we would opt for a vitamin C based redox therapy as our primary approach to treatment. This would be based on oral intakes: we would consider intravenous ascorbate only as an adjunct. We might use liposomal vitamin C to sustain blood levels at 400-500 μ M/L, together with alpha-lipoic acid, selenium, and other synergistic nutrients.¹⁵ While we realize malignant cancer would place us at high risk of death, we would expect to live a greatly extended life. While the assessment of increased longevity could be inaccurate (the data is not definitive), the risks are small and the potential benefits substantial.

Conclusions

Mark Levine claims that the “NIH’s unique translational environment, where researchers can pursue intellectual high-risk, out-of-the-box thinking with high potential payoff, enabled us to pursue this work.”¹⁹

However, the recent NIH study, while interesting, adds little to the studies it

replicates. More interesting is the lack of historical perspective, which may detract from the people, such as Hugh Riordan, Abram Hoffer, or Linus Pauling, who deserve the credit for carrying out original research, despite conventional medicine actively suppressing their work. The ground breaking work of doctors such as those in the British Society for Ecological Medicine, who have risked their careers to provide vitamin C based treatments for cancer and other conditions should be recognized. These pioneering doctors are often well aware of the scientific evidence and should not be described as “complementary” or “alternative”. Perhaps, one day, the media will realize the true story of vitamin C and cancer, and patients will have the opportunity to benefit.

The Alliance for Natural Health is defending our right to supplements. Over the last century, we have benefited from a large increase in life expectancy and freedom from many diseases. Much of that benefit has arisen directly from nutrition.¹⁶ We need access to supplements, which provide the possibility of disease prevention without significant risk. If this basic right is removed by Codex Alimentarius, or similar legislation—for example, the draconian regulatory measures the natural health sector is facing in Europe—even pioneering doctors will find it difficult to progress the nutritional treatment of disease. The health of most of us will suffer. We will get more illnesses, more often, and options for medical treatment of major killers, such as cancer, heart disease, and stroke, will decline.

References

1. Chen Q, Espey MG, Sun AY, et al: Pharmacologic doses of ascorbate act as a prooxidant and decrease growth of aggressive tumor xenografts in mice, *PNAS*, 2008; 105(32): 11105-11109.
2. Holman RA: A method of destroying a malignant rat tumour in vivo, *Nature*, 1957; 179(4568):1033.

3. Benade L, Howard T, Burk D: Synergistic killing of Ehrlich ascites carcinoma cells by ascorbate and 3-amino-1, 2, 4, -triazole, *Oncology*, 1969; 23: 33-43.
4. Cameron E, Pauling L: Supplemental ascorbate in the supportive treatment of cancer: Prolongation of survival times in terminal human cancer. *Proc Natl Acad Sci USA*, 1976; 73: 3685-3689.
5. Cameron E, Pauling L: Supplemental ascorbate in the supportive treatment of cancer: Reevaluation of prolongation of survival times in terminal human cancer, *Proc Natl Acad Sci USA*, 1978; 75: 4538-4542.
6. Murata A, Morishige F, Yamaguchi H: Prolongation of survival times of terminal cancer patients by administration of large doses of ascorbate, *Int J Vit Nutr Res, Suppl*, 1982; 23: 101-113.
7. Robinson AR, Hunsberger A, Westall FC: Suppression of squamous cell carcinoma in hairless mice by dietary nutrient variation, *Mech Aging Devel*, 1994; 76: 201-214.
8. Hickey S, Roberts H: *Cancer: Nutrition and Survival*, 2005; Lulu Press.
9. NIH News: *Vitamin C Injections Slow Tumor Growth in Mice*, Embargoed for Release, Monday, August 4, 5:00 p.m. EDT, 2008.
10. Hickey S, Roberts H: *Ascorbate: the Science of Vitamin C*, 2004, Lulu Press.
11. Hickey S, Roberts H, Cathcart RF: Dynamic flow, *J Orthomol Med*, 2005; 20(4), 237-244.
12. Padayatty SJ, Levine M: Reevaluation of ascorbate in cancer treatment: Emerging evidence, open minds and serendipity, *J Am Coll Nutr*, 2000; 19(4): 423-425.
13. Hickey S, Roberts H, Miller NJ: (2008) Pharmacokinetics of oral ascorbate liposomes, *JNEM*, 2008, (in press).
14. Toohey JI: Dehydroascorbic acid as an anti-cancer agent, *Canc Lett*, 2008; 263: 164-169.
15. Hickey S, Roberts HJ: *The Cancer Breakthrough*, 2007, Lulu press.
16. Wootton D: *Bad Medicine*, 2007, Oxford University Press.

Child Psychiatry: Does Modern Psychiatry Treat or Abuse?

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"It may actually be the case that children were safer before child psychiatry"

—M. McKay (2007)

Introduction

Jay, six years old, was forced into the modern psychiatric system from which he was rescued 3 years later by Dr Marty McKay and from Child Welfare by a court order. By then he had seen 60 physicians, diagnosed with dozens of diagnoses including mental retardation, ADHD, Tourette Syndrome, Oppositional Defiant Disorder, Obsessive-Compulsive Disorder, Conduct Disorder and the current favorite Childhood Onset Bipolar Mood Disorder. He was treated with toxic drugs and combinations with no evidence they were therapeutic. Toward the end of his treatment program he was on Ritalln, Divalproic Acid and Seroquel. He was saved by the relentless effort of Dr M. McKay who insisted the Hospital for Sick Children take him under care away from his psychiatrist. It took ten months to get him off the drugs. He had stopped growing. Since then he has regained his health. The long term effect of this massive long term toxic drugging is not known.

Rebecca Riley was not so lucky. She died from an overdose of two of the drugs prescribed for Jay. Under the heading "What Killed Rebecca Riley" this tragic event was featured on CBS, *Sixty Minutes*, on September 30, 2007. Rebecca was the youngest child in a dysfunctional family. Her two older siblings were already on massive drug medication. At age 2.5 years she was diagnosed ADHD and bipolar. She was prescribed Seroquel, a favorite antipsychotic for adult schizophrenic

patients, Depakote, an anticonvulsant for adults, and Clonidin, a drug for lowering blood pressure. December 13, 2006, she was found dead lying on the floor near her mother's bed, from an overdose of drugs. Her parents are charged with murder and are in jail waiting trial. December 12 she appeared to have a cold. Her mother gave her some Tylenol and some more Clonidine because she did not go to sleep. Then she laid her down beside her on the floor and fell asleep. When her mother woke up, Rebecca was dead,

The publicity given to Rebecca's death spurred Massachusetts into the beginning of regulatory action. Allen¹ reported "Although cases like the overdose of Rebecca Riley are rare, the prescription of psychiatric drugs to young children is not. Doctors last year prescribed Clonidine—a drug sometimes used to treat hyperactivity that was found in lethal quantities in the Hull girl's bloodstream—to 955 children under age 7 in MassHealth. Doctors also prescribed antipsychotic drugs, which raise the risk of diabetes and obesity, to 536 children under age 7. The largest provider of mental health services for MassHealth—Massachusetts Behavioral Health—identified 35 preschoolers in the first three months of the system who were taking three psychiatric medications or one antipsychotic drug."

Diagnosis

The North American psychiatric profession is enamored with the DSM system of diagnosing. This system is not as popular in other areas of the world, especially Australia, but doubts are developing even in North America. *The Canadian Journal of Psychiatry* featured a debate about the utility of the DSM classification of

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depression. Parker² concluded that DSM lacks explanatory power and compromises research and clinical practice. He is convinced that it does not indicate what might be causes and has no value in determining what treatment should be used. He could also have added that it is also not reliable in that several independent psychiatrists examining the same individual will come to several different diagnostic conclusions. It is not reliable it is not valid. Why are we still stuck with it? Goldney,³ defending the DSM gave a rather weak defense and hoped that it might one day be useful but did not answer the Parker charges.

In 1952 I visited Dr. Nathan Kline, Director Research of one of the New York Mental Hospitals. He was very taken with the new computers being developed. During this visit he described in detail how nosology would solve our problems. He hoped that his hospital would have the major computer which would be the diagnostic computer for the United States. I did not think much of his ideas but they must have had a major impact. I believe that the DSM is faithfully following them. The main result is nothing except for the very hefty DSM-IV. The treatment is not scientific, and has made no serious attempt to become scientific.

Pre-scientific medicine faced similar problems. It had to be descriptive since there were no laboratory or other accurate diagnostic tests available. Pain in the chest, worse on breathing and fever suggests something wrong in the chest maybe the lungs. Pneumonia was a high risk of death disease and was called the friend of the senile aged because it was a common cause of death. Before antibiotics became available one of the standard treatment was mustard plaster. Today we know there are many causes of pneumonia, that this condition is a syndrome and the real cause must be treated. Tests will determine if it is cancer, or a bacteria or

asbestos or a fungus and then appropriate treatment is given.

Modern psychiatry is in the mustard plaster stage of scientific diagnosis. This is understandable if the tests are not available but is unforgivable when available tests are not used. It would be like using mustard plasters because one did not believe in the physical and laboratory tests that are available for pneumonia. Psychiatrists over 100 years ago did use tests when they became available. Around 1900 a text book of psychiatry discussed differential diagnosis of psychosis included pellagra (vitamin B₃ deficiency) scurvy (vitamin C deficiency) syphilis of the brain and dementia praecox which later was renamed schizophrenia. The two vitamin deficiency syndromes were removed from psychiatry and came under proper care by public health and other doctors. The addition of small amounts of niacinamide to flour almost eradicated pellagra. This was one of the greatest public mental health measures ever and did more to decrease the incidence and prevalence of psychosis than all of psychiatry has done. General paresis of the insane (syphilis), was diagnosed by a blood test and disappeared from psychiatry. Dementia praecox also disappeared by being renamed schizophrenia.

There was little further progress until orthomolecular psychiatry developed.

In 1960 my research group in Saskatchewan discovered the mauve factor and used this as a way of characterizing a condition we called Malvaria and later Carl Pfeiffer renamed it pyroluria.⁴ These patients came from several diagnostic groups including schizophrenic who excreted too much of this factor into their urine. Pfeiffer eventually described a large number of different syndromes of schizophrenia. Each requires a rather different program. For if the disease is present due to a deficiency or a need for a lot of niacin it will not respond to any other vitamin

or treatment. Drugs are not specific and swamp the whole biochemical machinery of the body and affect everything. They can not be expected to be curative in the way discovering the cause and treating it is. I am sure that when the real cause of the clinical depressions usually seen by psychiatrists is discovered the profession will be totally surprised by the number of patients they had diagnosed as depression who have a nutritional cause and who respond to appropriate orthomolecular treatment. For example very few psychiatrists know that chronic food allergies can cause depression and that when these foods are removed the depression is gone. This is specific treatment and will one day be accepted by physicians who come from a different field of practice and who will know how to diagnose and treat. In the same way psychiatrists will lose their practices to doctors more willing to be scientific.

Of the forty or more different attention deficit disorders in children the main treatment is Ritalin no matter which of the forty labels is attached to the child. A child may be seen by ten different psychiatrists and given ten different diagnoses and numbers and leave the office with the same prescription. This means in fact that psychiatrists are no longer necessary since the diagnostician, and the diagnosis and the number are irrelevant. Any parent any time they are unhappy about their child's behavior would simply buy some Ritalin product over the counter.

Colleen Clements,⁵ associate professor of psychiatry, University of Rochester is a medical ethicist who writes a column for the *Medical Post*. She is very concerned about the pervasive use of Ritalin and other stimulants for the treatment of children, usually diagnosed one of the ADHD disorders. She points out (1) ADHD is a classification with dubious scientific basis; (2) There are no well established norms against which to judge the behavior of

these children; (3) Long term treatment with these drugs interferes with normal development and that society appears to benefit more than the child does from his treatment and; (4) The condition that a serious deviation must be present before treatment is started is ignored. Children are put in an illness category, which is degrading of their normality and worth. In a following issue she makes her points more dramatically. Drug prescriptions to children and adolescents increased from 275 per 100,000 in 1993-1995 to 1,438 per 100,000 in 2002. This is a five fold increase; forty percent were on another drug as well.

Between 1950, when I first became interested in psychiatry, and 1965 the older diagnoses were used. Various degrees of retardation were diagnosed based primarily on the IQ test. A very few schizophrenic children were recognized using adult criteria but these were remarkably accurate in predicting future psychoses. Dr. Loretta Bender was one of the best predictors. In one paper she reported the outcome of a number of childhood schizophrenic patients she had examined before the age of ten and who were re examined about seven years later. Half of them were adult patients in mental hospitals and the other half were psychopathic teens on the streets of New York.

Infantile Autism was so rare most doctors never saw any cases. It had been only recently described. Downs syndrome was diagnosed on physical appearance until the genetic test became available. A few hyperactive children were recognized. They were called minimally brain damaged. But this was very unpopular with their parents who heard only the word brain damage and not the word minimally. After 1960 psychiatry has been sensitive to public opinion re diagnosing. Minimally brain damage was dropped and replaced by hyperactivity.

From 1965 to today the diagnoses

have been completely changed. The APA Diagnostic Manual introduced the ADD system listing about forty different categories with their own diagnostic number. There was an explosion of diagnostic categories. All were descriptive. None had any real meaning.

The official diagnostic Bible of the American Psychiatric Association is DSM now in its fourth edition, 1994. It contains several hundred different diagnostic categories each with its own official number. I started in psychiatry in 1950. We had to consider only several dozen different diagnoses. It was simple. The explosion of diagnostic categories now listed in Number IV is fantastic. I cannot think of any other branch of medicine where the number of diseases (diagnoses) has increased by geometric progression. Does that mean that we have all developed all these new diseases, as the DSM IV would have us believe? And if this is true what will be the final count in edition V, expected to appear in 2011?

The problem is that psychiatric diagnosis contrary to all diagnosis in medicine is not scientific. It is descriptive, legal and moral. There are many variations in the way people behave and think and there is no limit to the number of descriptive diagnostic categories. I fully expect that one day the DSM will be thicker than the telephone books of large cities. There must be a reason and one is conflict of interest. A flagrant example of conflict of interest is reported by Cosgrove et al.⁶ She and her colleagues examined the financial relationship between DSM-IV panel members and Big Pharma. Out of 170 panel members 56% had one or more financial associations with Big Pharma. All the members of the panel on Mood Disorder and Schizophrenia had these ties. They recommended full disclosure. If a company has a drug released for treatment of schizophrenia it will pay them handsomely if the criteria for this

condition are so relaxed, so altered that many patients not previously diagnosed schizophrenia will become so under the new guidelines.

If diagnosis were scientific this would play little role but since the diagnosis is more psychological and political it does play a major role. At one time being gay was listed as one of the diseases in the APA manual. I keep using the word diagnosis when the APA uses the word disorder. Most people see no difference. This was quickly changed when so many of the members of the profession were gay. Two new diseases are (1) social phobia and (2) premenstrual dysphonic disorder. Psychiatric diagnosis is not causal nor does it indicate what treatment should be used.

Gerstel's report⁷ should be read. It is very critical and biting in its attack on APA diagnoses. She lists the following new diagnostic categories using the term problem. I will use the psychiatric term "disorder."

Self delusion disorder—you think you are normal and healthy.

Sibling relationship rivalry disorder—if you have problems with your siblings.

Partner relationship disorder—Guess what this means.

Phase of life disorder—concern about getting married or divorced.

Non compliance disorder—refuses to deal with your problems.

Intermittent explosive disorder—road rage or getting mad at your spouse.

I do not know the numbers for these new mental illnesses. I suspect that pretty soon any person who believes that orthomolecular psychiatry can be helpful will be labeled as suffering from a vitamin delusional disorder. I must be pretty sick but again I do not know what my number is. Several decades ago a psychiatrist in Los Angeles testified in court that his patient was delusional because she believed that

vitamin B₃ might help her.

It is clear from Gerstel's account that psychiatry knows that this is a profitable semantic game. It is not stupid. Thus Dr. M. First, director of the project to review DSM-IV, expects fewer new categories will be added because "they're hard to get rid of. It's disruptive to eliminate a disorder people have been using." This statement gives the game away. Because real diseases can not be gotten rid of so easily simply by deleting them from the diagnoses manuals. If they can be added and later deleted simply by a popular vote or by popular pressure, are they really disorders or are they sophisticated ways of describing behavior which might better be used in novels and public discourse and not tied to diseases where they do harm to victims of these diagnoses? But there is a glimmer of hope. First and Zimmerman⁸ indicate that in the new DSM-V some laboratory tests may be included in the diagnosis.

Children and Bipolar

The latest mass trend is to diagnose children as bipolar. Today in the United States there are one million children on toxic adult drugs for their bi polar disorder. They are diagnosed as early as age 3. Duffy⁹ in a very recent review concludes that as currently diagnosed, bipolar disorder does not manifest as such typically until at least adolescence. The title of her paper is a question "Does Bipolar Disorder Exist in Children? A Selected Review". After reviewing 41 published reports she concludes that it does not. She writes "Chronic fluctuating abnormalities of mood, over activity and cognition and conduct disturbances have been described in very young children. Whether this syndrome represents an early variant of BD or some other psychiatric disturbance is at this time unknown and requires further research".

In a recent report Madsen et al¹⁰ found a significant association between the amount of tranquilizers taken over years

in grams and cerebral cortex atrophy. The estimated risk of atrophy increases by 6.4% for each additional 10 grams of tranquilizer drug (in chlorpromazine equivalents). Gur et al¹¹ reported that tranquilizers increased sub cortical volumes in schizophrenic patients. These changes were not present in patients not on this medication. They suggested these changes were in response to receptor blockade and could decrease the effect of treatment. In other words these drugs damage the brain and decrease the odds these patients can ever recover. Are we preparing the ground for the next major pandemic of illness with millions of chronic schizophrenic patients becoming more and more brain damaged as they are forced to remain on their tranquilizers? And when it is fully upon us what are we going to do about it?

At age two children's brains start to develop rapidly and reach adult weight by age five. Between age two and five the brain triples in weight and this is the period when children are more impulse than control. They have to learn ways of dealing with others and with aggression so that they will become good members of society, How can the developing brain deal with these if inhibited with toxic drugs. It is well known that children are much more sensitive to drugs, even to the additives that are present in our food.

Forty years ago Dr. Ben Feingold, a well-known and respected allergist, reported that these additives made some children develop these problems. His work was totally rejected except by parents of the children who found their children became better when these additives were removed. A panel of the United States National Institutes of Health determined in 1982 that there was no scientific evidence to support these claims. The majority of clinical studies done at that time including some that were controlled, all showed that Feingold was wrong. The paradigm at that time opposed his conclusions. The paradigm is

now changing and the recent studies, also controlled, show that Feingold was right.¹² As the paradigm changes it becomes easier to insinuate these out-of-the box studies and to get them accepted. Most people do not realize that to the medical professions scientific means it has been accepted by the paradigm. If it is outside the paradigm it is not scientific.

Allowing these children to be diagnosed bipolar on vague behavioral changes that are simply a learning process is like giving a licence to kill, if not the child, then its mental growth and development

ADD and Ritalin

As the diagnostic term hyperactivity became more popular the use of stimulant drugs also increased beginning with the amphetamines (speed), and later ritalin which has evolved into different names and different formulations for the same drug. Even caffeine has been used. These stimulants had what was called a paradoxical effect on these active children. It relaxed them. Given to adults they were stimulants and were used to treat conditions with excessive sleepiness and to control excess sedation of the anticonvulsants. They were very effective and needed no double blinds to show that they did something. Children who were out of control would quickly settle down. This was great for schools who could not deal with too many hyperactive children in the classes. The drugs would be given in the morning which would keep them more or less down until they came home when the effect of the drug was gone and their hyperactivity once more exerted itself. Teachers appreciated these drugs more than did their parents. Adults were given barbiturates to help them sleep and amphetamines in the morning to waken them up. They were widely abused. One of my patients became addicted to amphetamines given to him when young to keep his weight down and later became

schizophrenic. A few children not liking the side effects of these drugs would not swallow them, and sell them to their older school chums. It is called kiddy coke. But over the past two decades they have been replaced by ritalin. Diagnosing them with one or more of the forty APA's ADDs gave the doctor permission to give them any combination of ritalin and other drugs.

Health Canada warns, (*Times Colonist*, Victoria, May 27, 2006), that ADHD drugs can be deadly, even for youngsters. It should have said especially for youngsters whose lives may be destroyed by these drugs: Adderall XR, Concerta, Dexedrine, Ritalin and Ritalin SR, Strattera, Attenade and Biphenin. The potential market is immense and explains why so many different names are used for almost the same drugs for these children. Health Canada warns that they may cause heart disease and even death but does not mention many other very serious side effects such as loss of appetite, suppression of growth and the consequences on personality by long term drug use and later addictions but some doctors are not convinced as they see more benefit than risk. This is a logical point of view if one does not know that there are much better alternatives to these drugs which are effective and do not cause any of the side effects listed.

The Ritalin advocates have new ammunition in their major attempt to retain this drug for the treatment of children, NIMH, which sponsored what it calls "The first long-term, large-scale study designed to determine the safety and effectiveness of treating preschoolers who have attention Deficit/hyperactivity disorder (ADHD) with methylphenidate (Ritalin)". Not surprisingly they found it safe and effective when used in low doses for pre schoolers, ages 3 to 5. The study found that children in this age range are more sensitive than older children to the medication's side effects and therefore should be closely monitored

Lets tease out the relevant data from this carefully worded document designed to support their conclusions

1. The study ran for 70 weeks. This may be a long time in contrast to the usual few months drug studies but is very short term in respect to these children growing into their mid teens. Malnutrition may not show its worst toxic side effects for up to 20 years. To call this a long term study is surely a major stretch. They also called it a large scale study but only 303 children were included. The term large scale is unwarranted even if that sample size was probably adequate. The description "long term" and "large scale" are used to soothe the public

2. Safety. Adverse effects are worse than with older children.

3. The medication slowed their growth rates. Over the 70 weeks of the study they grew one half inch less than the expected rates. However, a five-year follow-up study is underway to track the children's physical, cognitive, and behavioral development. Suppose we estimate what would happen if these children remained on trial for ten years, into their teens which is not that uncommon. This is difficult as growth is not linear with respect to age but we can estimate that on the average they would be 5 inches shorter and weigh 30 pounds less. How many teenagers would appreciate having their height and weight cut down that much? Height has economic and competitive advantages for both men and women. I know of no teenagers who would be happy if they knew that was going to occur. I am sure they would be even more reluctant to take the drugs and more eager to sell it to their friends as kiddie coke.

4. Eleven percent had to drop out of the study as a result of intolerable side effects. For example, while some children lost weight, weight loss of 10% or more of the child's baseline weight was considered a severe enough side effect for the inves-

tigators to discontinue the medication. Other side effects included insomnia, loss of appetite, mood disturbances such as feeling nervous or worried, and skin-picking behaviors. Can a treatment which makes one out of ten worse really be considered safe and effective?

Antipsychotics

I consider psychiatric drugs essential evils with major emphasis on the evil. They are essential for many patients but evil when used in large doses and for ever. They are less evil when used in much smaller doses and for shorter periods and if combined with orthomolecular psychiatric methods. They should be used like crutches and thrown away when they are no longer needed. Much more attention must given to the toxic side effects of the drugs. One of the major toxic long term side effects is that it is almost impossible to ever fully get well when on the medication. The natural recovery rate when patients are given proper shelter, good food and treated with civility and respect it is around forty percent. When treated by modern psychiatry it drops down to about ten percent. After over fifty years of research, mostly drug research, and billions spent on this research psychiatry has decreased the recovery rate over that achieved by the moral treatment of the insane sponsored by the Quakers from 40% to 10%. In the field of cancer has there been little improvement but it has not gotten worse than it was 150 years ago.

In Sweden government legislation enforces the "substitution principle"¹² This means that if a safer alternative is available for any toxic chemical added to the environment, food, etc., there is a legal obligation to use the safer compound. This is a very enlightened policy, not used in the North America. It should be enforced in all forms of chemotherapy including antipsychotic medication to replace drugs that are dangerous and for which there are

safer alternatives. I consider treating with psychiatric drugs palliative chemotherapy for psychiatric conditions and about as effective as is chemotherapy for cancer. And in the same way that chemotherapy for cancer leaves patients very sick so treatment with antipsychotics causes the tranquilizer psychosis which is often confused with the original psychosis.

Side effects, usually involuntary movements, can be permanent and are hence evidence of brain damage. A report in 1985 in the Mental and Physical Disability Law Reporter indicates courts in the United States have finally begun to consider involuntary administration of the so-called major tranquilizer/antipsychotic/neuroleptic drugs to involve First Amendment rights because antipsychotic drugs have the capacity to severely and even permanently affect an individual's ability to think and communicate. In *Molecules of the Mind: The Brave New Science of Molecular Psychology*, Professor Jon Franklin¹⁴ observed: "This era coincided with an increasing awareness that the neuroleptics not only did not cure schizophrenia, they actually caused damage to the brain. In severe cases, brain damage from neuroleptic drugs is evidenced by abnormal body movements called tardive dyskinesia. However, tardive dyskinesia is only the tip of the iceberg of neuroleptic caused brain damage. Higher mental functions are more vulnerable and are impaired before the elementary functions of the brain such as motor control."

Orthomolecular Treatment

By 1960 I had been using large doses of vitamin B₃ for seven years for treating schizophrenia, hypercholesterolemia, for decreasing the ravages of senility and for other conditions and I am still learning about this remarkable vitamin called a wonder drug by my friend Dr. Lars Carlson, Karolinska Institute in Sweden. What I have learned is described in the book Harold Foster and I wrote about

niacin¹⁵.

But in 1960 I had very little experience with its beneficial effects in helping children with learning and behavioural disorders. These are usually correlated as it is rare that a child will suffer from one set of these symptoms and not the other. My conclusions have been recorded in dozens of publications in *Journal of Orthomolecular Medicine* and in several books and there has been massive corroboration by physicians who used the treatment I had described.¹⁶ If this treatment is as good as I have seen and described why is not every child getting the benefit? Why is psychiatry loading these children with heavy doses of ritalin and atypical anti psychotic drugs? Why did Jay and his family have to suffer so much. Why did it take the intense dedication of Dr Marty McKay to save Jay's life and allow him to become a functioning human being. Why did the Ontario College of Physicians and Surgeons find that the psychiatrist who treated J was not to be censured?

In 1999¹⁷ I described 110 brief case histories of children under the age of fourteen I had treated with orthomolecular methods. It is obvious that many of them would today, if seen by a child psychiatrist, would be diagnosed with one or more of the ADDs and bipolar. They would have been treated with anti psychotic drugs and none would have recovered. The first three children I treated in 1960 recovered. No double blinds were needed.

In 1960 a physician called me from the United States. He was crying as he told me about his son, age 12, who was in hospital. He had just been advised that there was no treatment, no hope and that he should lock him up in a California state mental hospital and forget about him. That was very common advice. I advised his father that he should obtain some niacin and take it to the hospital to discuss with his son's psychiatrist. I did not think that any knowledgeable doctor would be afraid of

a vitamin. This was a failure as the psychiatrist became very angry, denounced the use of niacin saying that they had tested it and that it would fry his brains. Both statements were equally not true. I have been on niacin for over fifty years and so far my brain appears not to have been fried. Father then began to visit his son daily and while there, he fed him jam sandwiches made up of a slice of bread, a layer of jam, niacin powder, another layer of jam and a slice of bread. Three months later he wanted to go home. He completed grade 12 in the top 5% in the USA. Later, he studied medicine and became a research psychiatrist. He spent one summer working in Linus Pauling's laboratory.

In the same year, a female age 7 was equally disturbed and was labeled retarded, a term no longer favored. Her mother was schizophrenic. She was being prepared in New York City for schooling for the retarded. She was started on niacinamide 1 gram three times daily. She did not improve for two years but in her third year began to improve. She became normal, graduated on the Dean's list at University, became a teacher, married and recently retired. A few pennies worth of a single vitamin allowed this bright young girl to live a normal and productive life. This case illustrates the futility of psychiatric diagnosis.

Starting in 1960 I have treated well over 2,000 patients under the age of fourteen.¹⁷ There were very few failures and when they did occur it was often because the parents were not able to supervise their children's program effectively. Often one or both parents are also ill and should be treated. Schizophrenic children respond very well. In 1960 a young couple with 4 children were expelled from the city in which they were living because both parents were so psychotic the city could not deal with them and threatening to commit them to the closest mental hospital. They

fled to Saskatoon. Came under my care, and were given orthomolecular treatment. The father was well in a few months and has been well since and working full time. The mother went to university, received an MA and is now a senior administrator. Of their four children three developed behavioral problems. Dairy products were eliminated, they were given vitamins and today the entire family is normal.

The treatment theory and practice is based on the modern paradigm about the use of vitamins as treatment and not only to prevent a few deficiency diseases such as pellagra, scurvy, rickets. The treatment is more complex than just handing out a few vitamin pills. That is how it started but it became clear that the whole field of nutrition is involved. That is why in my books on children I gave so much space to nutrition. This I will not repeat.

The first element is to correct the diet of the patients. Too many consume huge amounts of food artifacts such as the sugars, free fats and products made from refined flour. This food is tasty, cheap and heavily advertised. This has been ignored for decades by government but at last the evidence has become so persuasive that attempts are being made to cut down eating of these artifacts. Just as important is to eliminate foods to which the patient is allergic. This has been totally ignored by medicine except by a few clinical ecologists who are also ignored. If the patient is sick because they are eating large amounts of milk to which they are allergic they will not recover until that has been corrected. The child can eat all good foods.

After the patients and their parents are instructed with respect to what to eat and when (i.e. to have three meals each day), they are started on the appropriate vitamins. For children with behavioral and/or learning disorders the two B vitamins B₃ and B₆ are the most important. When I first began to treat with vitamins I used only B₃ but later it became clear that

B₆ also played a role especially for autistic children. Vitamin C is needed as no one ever gets enough from food. Vitamin D is needed especially in northern countries where ultraviolet light is rare most of the year. And since it is rare for any person to have only one deficiency it is good to add a multi B- complex preparation. The most important minerals are zinc when there is evidence of a deficiency which is common when dairy products are consumed by allergic children and selenium in areas deficient like the west coast of North America.

Perhaps a more detailed description may be more persuasive.. Ben was my first child to receive orthomolecular treatment. Being the first he, his family and his response remain fresh in my mind. I will repeat what I wrote in *Healing Children's Attention and Behavior Disorders*. If a picture is worth a thousand words perhaps one good anecdote is worth dozens of brief case histories.

The Case of Ben

One evening, early in 1962, my friend George called to say he was very worried about his youngest son, Ben. Nine years old, Ben had become a behavioral problem with a learning disability. Today he would be diagnosed as suffering from ADD (Attention Deficit Disorder) or one of its many variants. Progress at school was so slow his teachers began to prepare his parents to have him go to a school for slow learners, perhaps even to a school for the mentally retarded. But before anyone was aware that Ben had such a problem, he had tested 120 on an IQ (intelligence quotient) test. To his father, a public administrator, and his mother, a teacher, this was not only perplexing but very disturbing. I asked George to bring Ben to my office on the fifth floor of the University Hospital, now Royal University Hospital, in Saskatoon. At the time, I was Director of Psychiatric

Research, Psychiatric Services Branch, Department of Public Health, Saskatchewan, and Associate Professor of Psychiatry at the Medical School.

I was not very keen on seeing Ben since I had little experience in treating children. The few children I had seen in the previous ten years were all considered either slow learners or had various degrees of severe retardation and no treatment was available for them. The 1960 view of these children was that they were primarily failures of the educational system and required special pedagogic skills and programs in order to deal with the problem. None of these special educational efforts was very effective. This was why the hospitals for the retarded were not called hospitals but rather training schools. We had one in Moose Jaw and a second one was created later on in Prince Albert after the building was closed as a special hospital for treating patients with tuberculosis. These hospitals (training schools) had more teachers and psychologists than physicians on their staff compared to mental hospitals housing schizophrenics and "real" mentally sick patients. To perpetuate this idea some American hospitals for these children called themselves "campuses:"

The modern type of hyperactive learning disordered child was extremely rare in 1960. This was also the view of celebrated pediatrician and children's health advocate Dr. Benjamin Spock. I met Dr. Spock just before we were both to appear on a TV program in Toronto in the mid 1960s and I asked him whether he had seen many "hyperactive" children when he was still practicing. He asked me to describe what I meant by hyperactive and later said that he could not recall having seen any children with this problem. But George was so disturbed I set aside my worry about making a proper assessment of Ben.

Ben came into my office with his

father. He was a good looking boy, appeared healthy, with none of the physical stigmata of the seriously retarded children seen in old psychiatric textbooks. He did not know why he had been brought to see me, and he denied having any problems or symptoms. His father gave me his developmental history. He was walking by 14 months and speaking by 20 months. Both parents considered him an ideal child until he entered Grade one when he was 7 years old. By the end of 1960, his mother noticed a change in behavior. He became more anxious, could not fall asleep at night, and if he did sleep, woke up frequently during the night. School became harder for Ben. When the family moved to a different part of the city and he was moved to a different school, he had even more problems. His teachers were worried about his erratic performance at school and told his parents he was in a "shell:" Reading and spelling were very poor. He finished Grade 3 with a D average in spite of extensive tutoring and drilling at home by his mother.

In July 1961 he was examined by a mental health clinic specializing in treating children. Ben's mother told them he had a very poor memory, reversed letters, and had no knowledge of phonics. His eyes skipped back and forth so much she tried to keep him focused by using a ruler under the lines. His teachers reported he was not working up to his best ability, spent a lot of time day-dreaming, wasting time, and therefore falling behind. His marks were very low. He did not complete his assignments and did not bother to write his exams, nor could he be motivated. At home Ben was negative to his father, missed a lot of school, and often would come home after school hours not having gone to school that day. The clinic blamed the move to a new school and sibling rivalry with his brother, a year and a half older. They recommended remedial reading, which proved to be ineffective.

After my examination, I was puzzled. Nothing appeared which could explain the deterioration of this child to his present state. I arranged to analyze his urine for the "mauve factor:" This was a substance which my research group had discovered in the urine of a majority of schizophrenic patients we treated, but it was also found in a smaller number of patients with other diagnoses. Over the previous few years, I had found that any patient with this substance in their urine more closely resembled schizophrenia than they did other diagnostic groups and that they responded very well to large doses of vitamin B₃ (niacin or niacinamide). We called it the mauve factor and later identified it as kryptopyrrole (KP).

The next day we found large quantities of KP in Ben's urine. I started Ben on niacinamide, 1 gram three times each day after meals. His parents continued this regimen for several months. George called me again that fall and told me that Ben was normal. He had been given remedial reading for two months by the clinic, who then pronounced him well, but he had shown no progress whatever before starting on the vitamin. He had spent the summer happily getting caught up with his reading.

One of his teachers prepared a report on Ben which she sent to me in 1973. George had advised her that Ben had done so badly in previous classes that he was called "stupid" in school and had responded by not answering any questions during class. But to her surprise she found him active in group discussions and volunteering answers. Here is what she wrote: "The first thing that his parents noticed in Ben's improvement after he showed an improvement in his health was his desire to go to school. Ben started to do his assignments, but at first he found the excuse of hunting for his books and pencils in his desk to delay him in starting his assignments promptly." The teacher

started keeping his books on her desk for some time, but midway through the term Ben took the initiative to get his books out promptly and began his assignments. Previous to vitamin therapy, Ben had no desire to take down all the notes given in the allotted time. When anything was dictated, Ben would have hard time keeping up. Then he would become very tense and, so to speak, “fold up” This would happen in some exams, especially in Spelling and Arithmetic, which he was slow doing, and then he would run out of time.

These problems soon began to disappear. Many other improvements were noted physically, socially, emotionally and educationally. Ben at the beginning of the term would pride himself with the fact his mother was also a teacher. Later on in the term, Ben also started to mention his father and brother. “Ben is no longer shy,” his teacher reported. “He is a sparkling personality; not afraid to speak up. He has started to take an interest in sports, in which he excels and which should be encouraged. He now gets along well with the children at school and at camp. He will assume leadership and organization duties. Ben now can read with eye-reversal not noticeable in reading and seldom in writing. Ben would go up on the stage to sing, say a speech, and read the morning scripture to the whole student body and the staff. All of the these things he did well with little nervousness and tension noticeable. Ben also reads books without being told and enjoys reading them.”

In 1966 Ben had completed Grade 7 with a low A average. In Grade 9 he went to a track meet, participated in extra curricular activities, and worked as stage manager for a school play. He was so busy he finished his scholastic year with a C average. Nevertheless, his parents were delighted with his state of normality.

In 1970 his mother wanted me to see him again. Ben had not taken niacinamide for two years, and she was worried that he

might relapse. Ben had forgotten he had ever seen me and did not understand why he should take vitamin pills. I explained the situation to him, and he agreed he would start again and keep taking vitamin niacinamide until age 18. Later Ben married. He is raising a family and has a responsible permanent job. He meets my criteria for recovery: he is free of symptoms and signs of illness, he gets on well with his family and with the community, he is employed and pays taxes.

Although Ben was one of the first children I tested for mauve factor (KP) and advised to take large doses of niacinamide, he is an excellent example of what can be done for these children with so-called learning disabilities and behavioral disorders if they are examined, diagnosed, and treated with the correct, orthomolecular approach. Ben’s treatment and response to a vitamin in large doses is a prototype of what can be achieved through diet and nutrient supplements, not only for “ill” children like Ben, but also for “healthy” children.

Discussion.

I still marvel at the fact that a disease which was very seldom diagnosed in children a few years ago is found in millions of children down to the age of two to such a degree that they are given antipsychotic drugs. This may be due to the Cascade phenomenon. Tierney¹⁸ writes “Cascades are especially common in medicine.” This phenomenon leads to widespread errors, mistaken consensus agreements. Tierney continues, “Doctors take their cues from others, leading them to over diagnose some faddish ailment (called bandwagon diseases) and over-prescribe certain treatment (like the tonsillectomies once popular for children). Unable to keep up with the volume of research, doctors look for guidance from an expert—or at least some who sounds confident.”

In his book *Good Calories, Bad*

*Calories*¹⁹ Gary Taubes presents massive evidence that the current idea about the relation between heart disease and fats in food is a severe case of mistaken consensus. The cascade effect led the medical world into a false hypothesis which has driven food guides for decades and which has not decreased heart disease as it was confidently predicted it would.

The idea that bipolar is so common originated at Harvard University with Dr. J. Biederman, Head of Child Psychopharmacology at Massachusetts General Hospital. In a CBS program he defined it more broadly so that more children could be diagnosed. He was very confident. Here we have the needed elements for a Cascade to start—the opinion by a respected scientist attached to Harvard. How many psychiatrists would stand up against a scientist from a distinguished university? The idea was also very attractive and helpful to Big Pharma which found an enormous new market for these drugs. A controlled study is now underway at Massachusetts General Hospital to test the effect of these drugs on children between ages 4 to 8 with bipolar psychosis, double blind of course. I find it bizarre that a diagnostic system which has never been validated and which is as useless and harmful as the DSM can have been accepted so quickly by the profession and that a treatment, for which there is no evidence that it works for children and has not been released for this purpose, can have become so popular in a very few years whereas orthomolecular treatment which has been developing over decades and which has been corroborated every time it has been used is hardly known. Of course the massive sweep of bad ideas is not unique to psychiatry. This phenomenon is described by Devra Davis²⁰ in the war against cancer as well as by Taube in his description of the war against the real cause of the pandemic of the metabolic disease.

Conclusion

Psychiatric diagnosis as described in DSM IV is not scientific, nor useful, either for treatment or prognosis and should be abandoned. It should be replaced by etiologic diagnosis, such as allergies, vitamin and mineral deficiency and dependency. Present diagnosis is harmful to children. It is a licence to kill. Palliative toxic psychiatric chemotherapy should follow the “substitution principle” mandated in Sweden for toxic environmental chemicals.

The adoption of these two policies would eliminate a large number of harmful conditions including brain damage, suicide, diabetes, abnormal blood lipid levels and associated cardiovascular disease. On a social level it would eliminate much pain, hardship, family disruption and chronic invalidism.

References

1. Allen S: Mass.Tracks Children on Psychiatric Drugs: Prescriptions Eyed After Overdose. *The Boston Globe*, October 7, 2007.
2. Parker G: Through a Glass Darkly: The Disutility of the DSM Nosology of Depressive Disorders. *Can J Psychiatry*, 2006;51:879-886.
3. Goldney RD: The Utility of the DSM Nosology of Mood Disorders, *Can J Psychiatry* 2006;51:874-878
4. Hoffer A, Osmond H: Malvaria: a new psychiatric disease. *Acta Psychiat Scand* 1963: 39: 335-366.
5. Clements C. ADHD: America: the drugged. Medical Post, Toronto, May 2, 2005, 12-13.
6. Cosgrove L, Krinsky S, Vijayaraqhan M & Schneider L. Financial Ties Between DSM-IV Panel Members and the Pharmaceutical Industry Psychotherapy and Psychosomatics 1996;75:154-160.
7. Gerstel J: Road Rager, Mad Spouse: Ill or Nasty. Psychiatric Labels Cast Wide Net on Human Foibles. *Toronto Star*. June 16, 2006.
8. First MB, Zimmerman M: Including Laboratory Tests in DSMIV Diagnostic Criteria *Am J Psychiat*, 2006; 163: 2041 - 2042.
9. Duffy A: Does Bipolar Disorder Exist in Children? A Selected Review. *Can J Psychiat*, 2007; 52: 400-415.
10. Madsen A, Keiding N, Karle A, Esbjerg S, Hemmingsen R: Neuroleptics in Progressive

- Structural Abnormalities in Psychiatric Illness. *Lancet*, 1998; 352: 784.
11. Gur RE, Manny V, Mozley PD, Swanson C, Bilker W, Gur RC: Subcortical MRI Volumes in Neuroleptic-Naive and Treated Patients With Schizophrenia. *Am J Psychiat*, 1998; 155: 1711-1717.
 12. Schab DW, Trinh, NH: Do Artificial Food Colors Promote Hyperactivity in Children With Hyperactive Syndromes? A Meta-Analysis of Double Blind Placebo Controlled Trials. *J Dev Behav Pediatr*. 2004;25(6) 423-434.
 13. Luymes G: Chemicals Are Quiet Killers: Report. *Times Colonist*, Victoria. October 7th, 2007.
 14. Franklin J: *Molecules of the Mind: The Brave New Science of Molecular Psychology*, 1987, Atheneum.
 15. Hoffer A, Foster HD: *Feel Better, Live Longer With Vitamin B₃*. CCNM Press, Toronto ON, 2007.
 16. Hoffer A: *Hoffer's ABC of Natural Nutrition for Children*. Quarry Press, Kingston, ON, 1999.
 17. Hoffer A: *Healing Children's Attention and Behavior Disorders*. CCNM Press, Toronto ON, 2005.
 18. Tierney J: Diet and Fat: A Severe Case of Mistaken Consensus. *New York Times*, October 9, 2007.
 19. Taube G: *Good Calories, Bad Calories*, Knopf New York, 2007.
 20. Davis D: *The Secret History of the War on Cancer*. Basic Books, www.basicbooks.com, 2007.

Are They Really Sick? A Report on Persons Who Are Electrosensitive and/or Injured by Dental Material in Sweden

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Abstract

People who attribute their symptoms to electromagnetic fields and/or dental materials typically present many symptoms simultaneously. These symptoms reportedly decrease or disappear among the Electro-sensitive when they relocate to low radiation environments, and 44% experience improved health after removal of harmful dental materials (e.g. amalgam, gold). Among Dental Material Injured, health improves for 59% when their fillings are replaced and one in five also reports significant improvement from electrohypersensitivity (EHS).

However, reduction of electromagnetic fields/radiation and removal of harmful dental materials are not enough. Nutritional supplements could also be of importance; 60% used supplements daily, mainly selenium, essential fatty acids, vitamins B and C.

This survey addresses members of FEB¹ (The Swedish Association for the Electro-sensitive) and Tf² (The Swedish Association of Dental Mercury Patients) and is financed by The Swedish Inheritance Fund.

Key Words

Dental materials, Mercury, Amalgam, Electrohypersensitivity, Electromagnetic Fields, Nutritional Therapy, Supplements

Introduction

The significant number of Swedes, who relate their symptoms to harmful electromagnetic fields (EMF) and/or dental materials, experience that they often receive either indifference or rebuttal by their health care providers during medical examinations. They frequently

report that their health care providers deem them as psychiatric cases and feel treated as though their symptoms are imagined. This may be because neither an official description of symptomatology exists nor the types of treatments known to improve health.

FEB (The Swedish Association for the Electrosensitive) and Tf (The Swedish Association of Dental Mercury Patients) are registered as disability organizations under the federal umbrella of HSO³ (The Swedish Disability Federation). In 2005 FEB and Tf received funding from the Swedish Inheritance Fund for an in-depth report of their members' experienced symptomatology and the level of care they had received from various health professionals.

The HET-Project (acronym for "health problems experienced by the Electrosensitive and the Dental Materials Injured") publicized its final report in March 2008 (www.hetprojektet.info). The outcome reveals notable severity of chronic illness from harmful sources of electromagnetic fields (EMF) and specific dental materials, but also suggest initiatives of lifestyle improvements and suitable treatment options.

Methods

A detailed study of the two membership groups were completed in the following sequences:

a) Members were invited to eight seminars in various regions of Sweden. The participants completed symptom questionnaires and took part in group discussions, at which time they clarified what type of treatments and health care

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they preferred. More than 300 members participated in these seminars.

b) Ten members contributed to in-depth interviews. Each of them had been chronically ill over a number of years. However, after the removal of injurious dental materials all of them improved, and some even fully recovered from their symptoms.

c) FEB members, who could not take part in the seminars due to their functional impairment, were encouraged to contribute by regular mail. As a result over 15 extensive personal recounts were received with detailed description of symptoms, causes or triggers and the types of treatments they viewed as improvements to their health status.

d) In addition, it was considered essential to get the professional point of view. Eight physicians and two dentists, known to effectively treat this specific group of patients, were interviewed in detail.

e) Questionnaires were distributed to members of the two organizations for quantitative analysis. The questions were designed based on the information received in previous HET surveys. Subsequently the questionnaires were mailed to one thousand members of each organization, a total of 2,000 surveys. The Electrosensitive responded with 83% and the Dental Materials Injured with 68%.

f) The result of all surveys in the entire HET-Project are included in the final report; "Are they Really Sick? A Report on the Electrosensitive and the Dental Materials Injured in Sweden."

All surveys have been publicized in separate reports on the HET-Project webpage: www.hetprojektet.info. The summary of the final report is translated here into English; the rest of the reports are written in Swedish only.

No control group was used for this study, and the survey results only disclose the experiences and insights of the Tf and

FEB members. An analysis in relation to the general population in Sweden will require further research.

Results

This research reveals that the members of both FEB (The Association for the Electrosensitive) and Tf (The Association of the Dental Mercury Patients) suffer from many symptoms simultaneously, i.e. incessant fatigue, memory and concentration difficulties, stomach and intestinal problems, chronic pain, skin irritations, anxiety and depression. Common diagnoses are fibromyalgia, psychiatric illnesses, chronic fatigue syndrome and burnout.

Symptomatology

Even though the symptoms in the two groups are similar, the accumulated data reveals notable differences between the symptoms of the Tf and the FEB members.

The Electrosensitive often experience skin and eye irritations as well as heat sensations in the brain tissue when exposed to harmful EMF and/or radiation. The Dental Materials Injured, however, characteristically experience anxiety, depression and a metallic taste in their mouths. Some are sensitized by both EMF and dental materials, and therefore the symptoms of these patient group's experiences occasionally overlap. Among the members of Tf (The Association of Dental Mercury Patients) 56% are or have been sensitive to electromagnetic fields, however this is the reality for practically all (95%) the members of FEB (The Swedish Association for the Electrosensitive). See **Table 1**, p.155)

Reduction of Harmful Sources Vital to Improved Health

Symptoms decreased in 77% of the FEB members when exposure to EMF abated, and 44% reported improved

Table 1. Most common symptoms (have or have had) in the order of frequency by FEB and Tf members.

FEB -The Swedish Association for the ElectroSensitive	Tf The Swedish Association of Dental Mercury Patients
1. Concentration difficulties 71 %	1. Excessive fatigue, exhaustion 82 %
2. Excessive fatigue, exhaustion 69 %	2. Concentration difficulties 76 %
3. Burning sensation in the skin 62 %	3. Aches & pain in shoulders, neck & thoracic region 72 %
4. Memory problems 62 %	4. Sleeping difficulties 68 %
5. Aches & pain in shoulders, neck & thoracic region 61 %	5. Muscle weakness 67 %
6. Light sensitivity 57 %	6 Depression 66 %
7. Sleeping difficulties 57 %	7. Memory problems 65 %
8. Disjointed thinking patterns 56 %	8 Recurrent irritation in the stomach & intestines 65 %
9. Muscle weakness 54 %	9. Pain in lumbar region, hips and ischias 64 %
10. Recurrent irritation in the stomach & intestines 53 %	10 Metallic taste 64 %
11. Dizziness 53 %	11. Mental stress 63 %
12. Tinnitus 53 %	12. Anxiety 62 %
13. Redness in skin 52 %	13. Pain in hands, elbows, legs and knees 61 %
14. Pain in lumbar region, hips and ischias 52 %	14. Dizziness 61 %)
15. Mental stress 52 %	15. General malaise 59 %
16. Prickling and numbing sensations in the skin 52 %	16. Pain in jaws and sinuses 55 %
17. Heart palpitations 50 %	17. Heart palpitations 55 %
18. Pain in hands, elbows, legs and knees 50 %	18. Tinnitus 54 %
19. Heat sensations in the brain tissue 48 %	19. Disjointed thinking patterns 54 %
20. Dry skin 47 %	20. Regular headaches/migraines 52 %
21. Tense muscles 46 %	21. Pain in the heart region and chest 51 %
22. Dry and 'sandy' eyes 46 %	22. Tense muscles 51 %
23. Irritation/prickling sensation in the eyes 44 %	23. Prickling and numbing sensations in the skin 51 %
24. General malaise 44 %	24. Recurrent infections 50 %
25. Pain in jaws and sinuses 43 %	25. Dry and 'sandy' eyes 50 %
26. Pain in the heart region and chest 43 %	26. Runny or congested nose 48 %
27. Regular headaches/migraines 42 %	27. Dry skin 48 %
28. A sense of physical unease 42 %	28. Allergies 48 %
29. Depression 41 %	29. High or low blood-pressure 47 %
30. Runny or congested nose 41 %	30. Light flashes in the eyes 46 %

health after having their amalgam removed. The Tf members reported 59% improvement after having injurious dental materials replaced, and one in five reported a decreased sensitivity to electromagnetic fields after the same procedure. There are therefore clear connections between these two groups. (See **Figure 1**, p.157)

Practically every member of the two associations have or have had mercury amalgam fillings and 72% respectively 82% of members in FEB and Tf have removed their amalgam fillings. This dental procedure is exceedingly common among Tf and FEB members in comparison to the general population with a small percent have replaced their amalgam fillings.⁴

Nutritional Therapy is Vital

The health status of the survey participants improved further by using nutritional therapy; 60% took nutritional supplements daily. Several reported that their health deteriorated when they discontinued this treatment regimen, but that their health improved when they reintroduced supplements to their diet again.

Among the FEB members, the most commonly used supplements are omega 3 and 6 fatty acids, vitamin C and magnesium. Among the Tf members selenium, omega 3 and 6 fatty acids and vitamin B₁₂ dominated. Moreover, 71% of the Tf members concluded that vitamins and minerals have contributed considerably to their recovery, while only 38% of the FEB members were of the same opinion. (See **Figure 2**, p.157)

An Undesirable Delay in Treatment

This survey also shows that a disproportionate number of participants had to wait many years before understanding the causes or triggers of their problems. They conceded that it was quite difficult finding a physician who could understand their symptoms. The FEB members had been sick for about two years before they were able to make connections between harmful sources and symptoms. Conversely, the Tf members suffered on an average of 12 years, while as many as 25% endured more than 20 years before suitable treatment options were presented to them. (See **Figure 3**, p.158)

About 10% of the FEB members reported that their physician suspected electromagnetic fields as the cause or trigger to their symptoms. Most discovered these sources by themselves, while some were helped by caring friends and family or found information in media exposés. The Tf members voiced similar experiences; and 8% replied that their physician had been cognizant of the causes to their symptoms.

Delays in Recuperation Time

For the Dental Materials Injured (Tf) there were considerable delays between removal of harmful dental materials and lasting improvements. Only 6% of Tf members reported immediate health recuperation after the removal procedure; and almost 40% endured more than four years before they achieved lasting improvements. (See **Figure 4**, p.158)

Abating Symptoms after Removal of Injurious Dental Materials

Among Tf members certain symptoms disappeared after the removal of injurious dental materials. The same symptoms are typical of chronic mercury poisoning, i.e. metallic taste, decreased appetite, suicide ruminations, the shivers or fever, nosebleeds, sensitive and bleeding gums, burning sensations on the tongue, panic attacks, tunnel vision, blurred vision and involuntary muscle tics.

Triggering Factors of Electrohypersensitivity

The FEB members reported symptoms mainly from computer screens (92%), fluorescent tubes and compact fluorescent lights (90%), cell phones (84%), cordless phones (82%), cellphone antennas and base stations (67%) and high voltage power lines (66%).

The most common triggers were believed to be computer and video screens (68%), amalgam (49%) and fluorescent tubes and the new compact fluorescent lights (46%).

Mitigation of EMF/radiation at home and at the workplace improved health for many, resulting in better odds of remaining in the workforce. Restful sleep was deemed vital by the participants, and therefore 8% of the FEB members had resorted to using a canopy around their beds with special EMF repellent material to prevent harmful exposure to electromagnetic fields. However, many had been forced to relocate to

Figure 1. Preferred treatments.

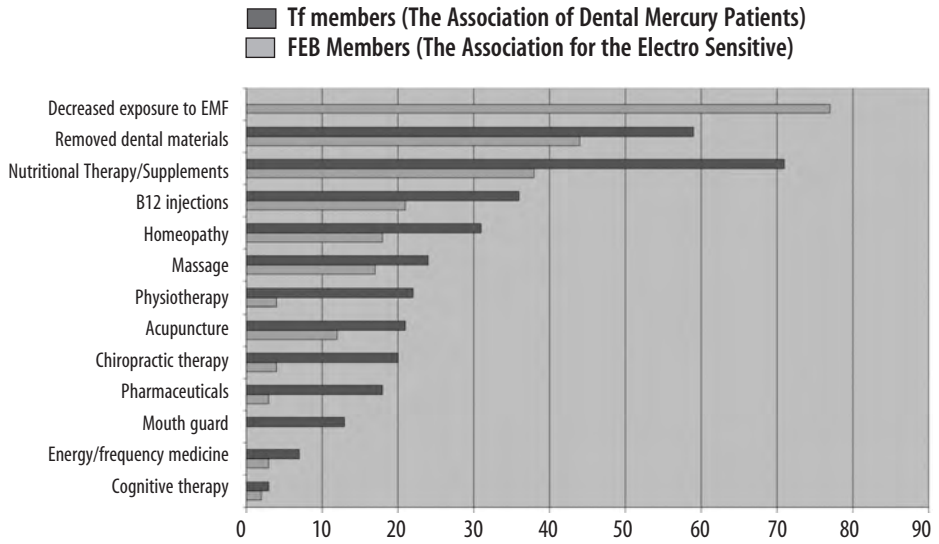


Figure 2. FEB and Tf members' use of nutritional supplements.

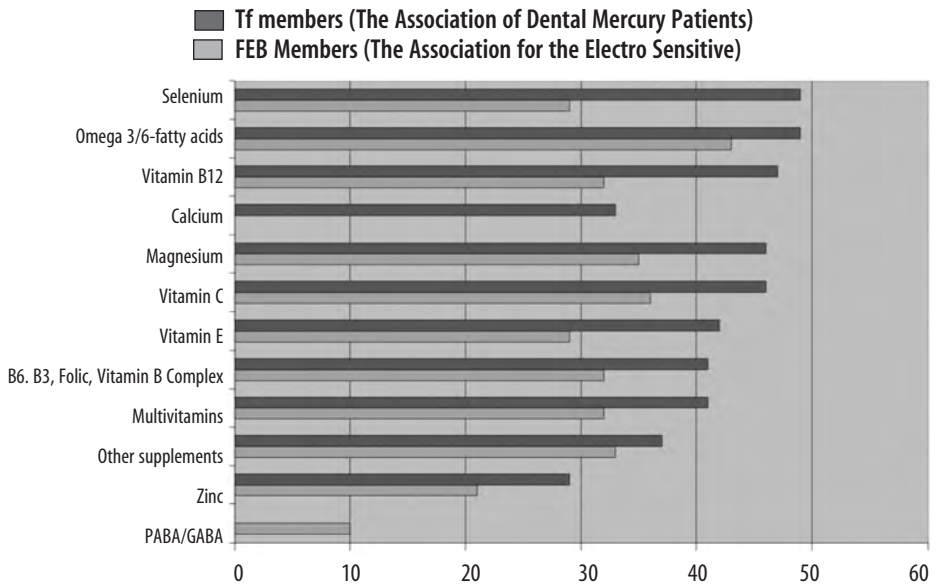


Figure 3. Number of years until the members understood the causes/triggers of their symptoms.

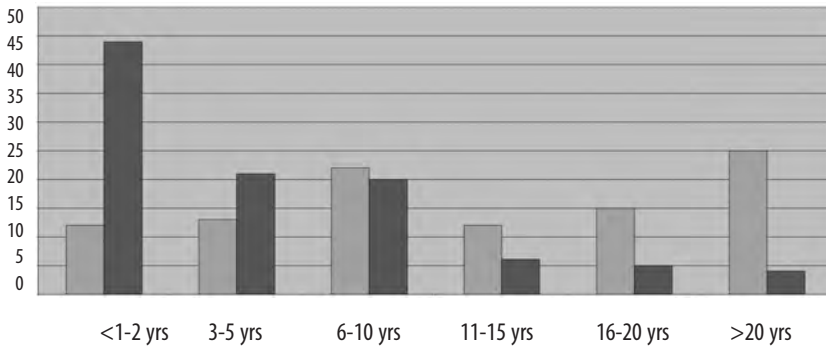
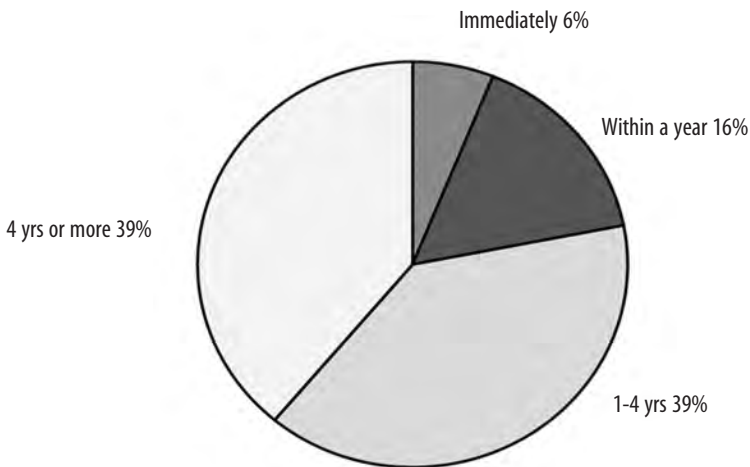


Figure 4. Time span before experienced lasting improvements after amalgam removal.



a low radiation area in the countryside to reduce their symptoms.

Discussion

Both the Electrosensitive and the Dental Materials Injured experienced many symptoms simultaneously. Certain

differences were notable in the symptomatology of the two groups, although overlaps where also apparent. The Electrosensitive complained of skin and eye symptoms, a distinct heat sensation in the brain tissue, while the Dental Materials Injured mostly reported psychiatric symp-

toms, i.e. anxiety and depression as well as a metallic taste in their mouths.

Remarkable health improvements were reported by the participants after thorough EMF mitigation, the removal of harmful dental materials and by taking vitamin and mineral supplement. However, clinically developed research projects are needed to build an evidence-based model for future health care practices.

Removal of Amalgam

Most of the members in both organizations experienced improved health after the removal of injurious dental materials, especially amalgam fillings. However, the improvement potential was not as high among the Tf members (59%) as among the participants in scientifically controlled studies; where a reported 70-80% of the patients improved health after amalgam removal.^{5,6,7}

The reason for this disparity may be due to inferior amalgam removal procedures used in Swedish practices. As many as 30% of the Tf members reported that they had to change their dental fillings two or more times due to sensitivity to the new material, amalgam remaining under the new fillings, or other ways in which the replacement was unsatisfactorily performed.

Nutritional Balance

Many questionnaire responses and individual comments revealed that a change in diet combined with vitamin and mineral supplements improved the participants' health considerably. Some avoided gluten and sugar, while others felt better after increasing their intake of B₁₂, selenium and vitamin C. This indicates that the particular health problems of the Electrosensitive and the Dental Materials Injured are complex and in need of additional treatments. Merely decreasing electromagnetic fields or removing harmful dental materials may not be enough.

Continued Research Needed

A research project with the focus on genetic differences in Electrosensitive participants and/or hypersensitivity to dental materials may be worth further investigation considering the staggering costs of chronic disease. It is now recognized that the uptake of B₁₂ and B₆ can be genetically determined just like the ability to detoxify.⁸ The genetic variant of apolipoprotein E (apo-e4) is present to a considerable degree in people with dental amalgam toxicity.⁹ Moreover, the HET-Project questionnaire responses confirm that B₁₂ injections in particular improved the participants' health status. This disclosure may indicate a genetically determined impediment in absorbing and processing vitamin B₁₂.

Detoxification

The delayed time between removal of fillings and improved health may indicate a problem with the body's ability to detoxify. This hypothesis is strengthened by some members' account of successful chelation therapies, although this form of therapy is not legal in Sweden and only a few members have actually tried it. A recent study details improved health from dental materials removal if patients also subject themselves simultaneously to chelation therapy.¹⁰

Conclusion

The Electrosensitive and the Dental Material Injured are a significant group of the chronically ill in Sweden who add to the cost in health care, insurance payments and in lost productivity. It is estimated that 3-9% of the Swedish inhabitants are Electrosensitive^{11,12} and 1-5% suffer from Amalgam Injury, which means that between 300,000 and 800,000 Swedes have such health problems. A suitable comparison would be with diabetics, estimated at 300,000 in the country. However with the availability of relevant

treatments and health care, the majority of this neglected group can recover, and sometimes completely.

The participants of this study report that most improvements occur with a decrease in harmful EMF exposures, the removal of bio-incompatible dental materials and the correction of nutritional imbalances boosting the vital organ functions with vitamin and mineral supplements. Also, there appears to be a link to genetic factors as a cause of hypersensitivity to electromagnetic fields and/or injurious dental materials.

It is therefore a socioeconomic and a financially sound solution to support further research and thereby continue the development of evidence-based EMF mitigation practices, advance dental materials removal protocols and develop beneficial nutritional therapy regimens.

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References

1. www.feb.se
2. www.tf.nu
3. www.hso.se
4. SOU 53: Dental Materials and Health. Summary in English and an overview of scientific literature (58 pages). (In Swedish: Dentala Material

och Hälsa (591 pages)) *The Dental Materials Commission, Swedish Government Official Reports. Fritzes, Stockholm 2003.*

5. Hansson, M: Health effects of amalgam removal. Suppl to *Bull Swedish Dental Mercury Assoc*, 2003.
6. Lindh U, Hudecek R, Danersund A, et al: Removal of dental amalgam and other metal alloys supported by antioxidant therapy alleviates symptoms and improves quality of life in patients with amalgam associated ill health. *Neuroendocrinol Lett*, 2002; 5/6(23): 459-480.
8. Custadio, H.M, Harari, R, Gerhardsson, L, Skerfving S, Broberg, K: Genetic Influences on the Retention of Inorganic Mercury. *Arch Environ Occup Health*, 2005; 60(1): 17-23.
9. Godfrey ME, Wojcik DP, Krone CA: (2003). Apolipoprotein E genotyping as a potential biomarker for mercury neurotoxicity. *J Alzh Dis*, 2003; 5: 189-195.
10. Wojcik D, Godfrey M, Christie D, Haley, B: Mercury toxicity presenting as chronic fatigue, memory impairment and depressions: Diagnosis, treatment, susceptibility, and outcomes in a New Zealand general practice setting (1994-2006). *Neuroendocrinol Lett*, 2006; 27/4: 415-423.
11. *The National Board of Health and Welfare (2001). Health in Sweden – Sweden's Public Health Report*, 2001.
12. Carlsson F: Känslighet för dofter och elektricitet (Sensitivities to fragrances and electricity). In *Swedish Medikament*, 2004; 9/10: 40-43.

Prevalence and Risk Factors For Lower Selenium Status Among Adult White Males in the USA

Andrew Pinfold¹

Abstract

Objectives: To establish the prevalence of lower serum selenium status (<106 ng/mL) among the adult white American male population, to determine whether certain social, economic, geographic, physical, and dietary characteristics are risk factors for lower selenium status, and to identify and evaluate potential selenium fortification vehicles that target men with lower selenium status.

Design: A cross-sectional study using nationally representative data from the National Health and Nutrition Examination Survey III, 1988-1994 (NHANES III).

Methods: 2989 white men, aged 20 or greater in the NHANES III dataset had recorded serum selenium values. These men were divided in two groups based on selenium status, those with values of less than 106 ng/mL (n=288) and those with a status greater than or equal to 106 ng/mL (n=2701). Various demographic, physical, and dietary variables were then compared between the two selenium status groups in a bivariate analysis. Multiple logistic regression was then performed to assess possible risk factors for lower selenium status.

Results: This study estimated 7.7% of White Americans adult men aged 20 years and older, a total of 4,751,618 individuals, had a selenium status of below 106 ng/mL. Several of the more than forty, social, economic, geographic, physical, and dietary characteristics examined were shown to be significantly associated a lower selenium status. Risk factors for lower selenium status (106 ng/mL) were; smoking, living in the South, an age of 60 years or older, exercising less than your peers, and having a lower income.

Conclusion: It would appear certain physical, geographic, dietary and demographic characteristics present a significant risk for lower selenium status. While, this work was unable to identify a suitable selenium fortification vehicle to reduce the prevalence of lower selenium status, it did identify risk factors that may contribute to this condition. The findings of this work could be helpful in designing a selenium augmentation/fortification program that target men with lower levels of the mineral.

Introduction

Recently, a large scale clinical trial demonstrated that selenium supplementation significantly reduced the risk of prostate cancer. The Nutritional Prevention of Cancer (NPC) trial in the United States began in 1983 in order to test whether supplementing individuals with selenium could play a role in preventing the development of cancer. Individuals were given either 200 µg of selenium per day in the form of selenized yeast or a placebo.¹

After 13 years, the NPC trial demonstrated that, although selenium supplementation did not seem to have any statistically significant effect on primary endpoint of non-melanoma skin cancer, it did provide protection against other forms of cancer. Selenium supplementation, for example, was found to significantly reduce total cancer mortality (41%) and total cancer incidence (25%).¹ The strongest inverse association between selenium supplementation was with prostate cancer. The supplemented group was 52% less likely to develop prostate cancer than the placebo group.¹ Subsequent analysis of the NPC data by Duffield-Lillico and

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colleagues showed that this inverse association between selenium supplementation and prostate cancer incidence was confined mainly to those men with blood plasma selenium levels in the lowest tertile (≤ 106.4 ng/mL).²

Prostate cancer has both large human and financial consequences in the United States. For example, in 2002, 34,446 men died as a result of prostate cancer and it was estimated that health care costs to treat the disease exceed \$1.5 billion per year.³ While not all men who develop prostate cancer have low selenium status and conversely, not all men with low selenium intake develop prostate cancer, certainly there must be some positive relationship. Given that it has been demonstrated that men with low selenium status, who are supplemented with Se, significantly reduce their risk of developing the disease, it seems very likely that some of these prostate cancer deaths could be prevented by increasing dietary intake of this trace element.

The three major objectives of this study were to estimate the prevalence of low selenium status among adults white American men, to examine the associations, if any, between a range of biological, environmental and lifestyle factors and depressed male selenium status, and lastly, to evaluate a potential dietary intervention that might be used to reduce the incidence of prostate cancer.

Methods and Materials

Data from the third National Health and Nutrition Examination Survey, 1988-1994 (NHANES III) are employed to develop comparative profiles of lower selenium status vs. higher selenium status among white adult men in the United States and to conduct multivariate analyses examining risk factors for lower selenium status. The NHANES III survey uses complex stratified multistage probability design to examine a nationally representative

sample of the United States civilian non-institutionalized population. NHANES III collected data on body measurements, demographics, physical function, dietary intake, health condition, lifestyle behaviors, and biochemical measurements of blood and urine from 39,695 individuals which were considered representative of the US population as a whole.⁴ This cross-sectional survey includes data from 2989 white men aged 20 or greater, which represented a weighted total of 61,776,414. In this study an unweighted total of 288 men had a selenium status less than 106 ng/mL, while 2701 had selenium status values greater than or equal to 106 ng/mL. (Definitions of variables used in the analysis are available from the author upon request).

Chi-square tests and confidence intervals were created for all categorical dependent variables, while T-tests were used to evaluate the differences between the low and high selenium status groups for continuous normally distributed independent variables for the bivariate comparisons. Multiple logistic regression was performed to evaluate the relationship between lower selenium status and the significant independent variables from the bivariate analysis.

Statistical analysis undertaken during this study were performed using a Statistical Analysis System software (SAS) callable version of SUDAAN, which is able to account for the complex survey design and sampling weights of NHANES III.^{5,6}

Results

The overall prevalence of selenium status below 106 ng/mL among white American adults 20 years of age and older is 7.7% (95% CI 6.5-8.8) with a weighted estimate of 4,751,618 individuals. Compared with other western countries, the prevalence with men with lower selenium is quite small since several nations have mean selenium status values below 106

ng/mL. It has been estimated, for example, that greater than 50% of the adult population of Austria, Germany, Spain, and Poland have serum selenium levels below 70 ng/mL.⁷

Bivariate Analysis

Tables 1 through 5 reveal significant differences exist between adult men with lower and higher selenium status on a range of variables including; geographic location, age, level of education, health, diet, and physical measures. Low selenium status differs significantly along geographic lines. For example, the highest prevalence of white men with lower selenium status occurs in the South, with 12.6% of the total white adult male population exhibiting this characteristic. This rate is nearly twice that of the Northeast and Midwest at 6.3% and 6.6% respectively, and almost 4 times greater than the prevalence in West region at 3.2% (see Table 1, p.164). In the United States as a whole, the majority of men with a selenium status below 106 ng/L, live in the South (54.3%), 20.0% are in the Midwest, 18.4% live in the Northeast, while 7.3% are in West. With regard to age it would appear as though low selenium status is more prevalent among older men, i.e., in the low selenium status group 31.7% (95% C.I. 25.8-37.6) are above the age of 60, whereas in the higher selenium group on 21.31% (95% C.I. 20.7-23.6) are in the oldest age category (see Table 1, p.164).

There were significant differences between the two selenium status groups in the case of income. The prevalence of poverty was significantly greater in the low selenium status group than in the higher one (28.6%, 95% C.I. 21.6-34.6 vs. 19.4%, 95% C.I. 17.6-21.2). A significant difference also occurred in the high income group, with men with lower selenium status being less likely to have a high income than those with a higher selenium status (29.06%, 95% C.I. 21.6-

36.6 vs. 45.6%, 95% C.I. 43.1-48.0). In the middle income category, prevalence rates were not significantly different between the two selenium status groups.

In terms of education, there were significant differences between the two selenium status groups. The lower selenium status group contained a significantly higher prevalence of individuals that did not graduate from high school compared to those in the higher selenium status group (30.3% vs. 20.1%).

Of the 12 health status variables examined only four; smoking status, self-reported health status, self-reported physical activity level, and a cataract diagnosis differed significantly between the two selenium status groups. Table 2 (p.165) presents the results from the analysis performed with the health status variables. While statistically not significant, the prevalence rates of many chronic diseases, including congestive heart failure, arthritis, cancer, skin cancer, and diabetes were more common within the lower selenium status group.

The prevalence of men who state that their health was fair or poor was significantly higher, 17.2% vs. 11.4% ($p \leq 0.01$) in the lower selenium status group than in the higher selenium status group. Of the variables studied, smoking status presented the most striking difference between the two selenium status groups. Using serum cotinine levels as a surrogate measure for smoking status, it was found that the majority (59.9%, 95% CI 51-68) of those in the lower selenium group were smokers, while less than 40% were smokers in the higher selenium status group. This study found that men with lower selenium are generally less active than those with higher selenium. This is not a finding that has been documented in previous studies. It is likely that exercise is a covariate of other factors which have been demonstrated to affect selenium status such as smoking, income, and educa-

Table 1. Demographic bivariate results.

Characteristic	<106 ng/mL		≥106 ng/mL	
	Number	%(95% C.I.)	Number	%(95% C.I.)
Overall Prevalence	4,751,618	7.7 (7.1-9.3)	57,024,796	92.3 (91.2-93.41)
Age**				
20-39	1,974,553	41.55 (33.68-49.42)	25,381,585	44.5 (41.9-46.6)
40-59	1,269,727	26.72 (20.06-33.37)	19,449,637	34.1 (31.3-35.7)
60+	1,507,733	31.72 (25.83-37.61)	12,193,573	21.3 (20.7-23.6)
Income**				
Low (poverty index ratio ≤ 1.85)	1,265,099	28.06 (21.6-34.6)	10,542,907	19.4 (17.6-21.2)
Medium (poverty index ratio 1.851-3.5)	1,931,989	42.9 (35.1-50.56)	19,039,406	35.0 (32.7-37.7)
High (poverty index ratio => 3.501)	1,310,131	29.06 (21.6-36.6)	24,763,722	45.6 (43.1-48.0)
Education (years attended) **				
≥ 12	1,512,733	69.9 (63.3-75.9)	45,484,599	79.9 (78.1-81.6)
< 12	3,475,348	30.3 (24.0-36.6)	11,445,280	20.1 (18.3-21.8)
Live in a Metropolitan Area				
Yes	1,848,906	38.9 (31.7-46.1)	25,508,144	44.7 (43.3-46.2)
No	2,902,712	61.1 (53.9-68.3)	31,516,651	55.3 (53.8-56.7)
Geography (% living in)**				
North East	875,556	18.4 (12.5-24.3)		
Mid West	952,311	20.0 (14.4-25.6)		
South	2,579,221	54.3 (47.0-61.5)		
West	344,530	7.3 (2.68-11.82)~		
Geography (within region prevalence) **				
North East	875,556	6.3 (4.0-8.6)		
Mid West	952,311	6.6 (3.9-7.3)		
South	2,579,221	12.6 (10.3-15.0)		
West	344,530	3.2 (1.0-5.3)~		

Source: The National Health and Nutrition Examination Survey NHANES III (1988-1994)

Notes: All reported values represent weighted estimates using Final Exam Weight: WTPFEX6.
As per NHANES III analytical guidelines all coefficients of variation above 30% are flagged with~

* stastically significant chi-square test results (95%)

** statistically significant chi-square test result (99%)

Table 2. Health variables bivariate results.

Selenium Status (ng/mL) (White US Males age >=20years)				
Characteristic	<106 ng/mL		≥106 ng/mL	
	Number	%(95% C.I.)	Number	%(95% C.I.)
Self Report Health Status**				
Fair or poor	865,542	17.2 (13.1-21.4)	6,500,634	11.4 (10.0-12.7)
Excellent, very good or good	4,144,031	82.7 (78.5-86.8)	50,504,170	88.6 (87.25-89.9)
Exercise Activity Level*				
less than peers	1,150,331	24.6 (18.15-31.0)	9,947,108	17.7 (15.8-19.6)
more or same as peers	3,520,129	75.4 (68.8-81.8)	46,141,661	82.3 (80.3-84.10)
Smoker (Serum Corinne >= 14 ng/mL = smoker)**				
yes	1,673,114	59.91 (51.24-68.58)	10,407,624	38.5 (35.5-41.6)
no	1,119,379	40.08 (31.41-48.75)	16,572,751	61.5 (58.3-64.4)
Doctor ever told: cataracts**				
Yes	532,128	11.1 (7.8-14.5)	3,222,438	5.6 (4.8-6.4)
No	4,477,445	89.3 (86.3-92.3)	53,802,357	94.4 (93.5-95.1)
Doctor ever told: congestive heart failure				
Yes	170,927	3.6 (1.2-5.9)	1,071,007	1.9 (1.4-2.4)
No	4,580,690	96.4 (94.1-98.740)	55,953,789	98.1 (97.6-98.6)
Doctor ever told: stroke				
Yes	170,927	3.6 (1.2-5.9)	1,071,007	1.9 (1.4-2.4)
No	4,580,690	96.4 (94.1-98.8)	55,953,789	98.1 (97.6-98.6)
Doctor ever told: arthritis				
Yes	925,984	19.5 (14.8-24.2)	8,813,552	15.5 (13.8-17.1)
No	3,825,634	80.5 (75.8-85.2)	48,204,415	84.5 (82.9-86.2)
Doctor ever told: asthma				
Yes	309,333	6.5 (3.2-9.7)	4,524,339	7.9 (6.6-9.3)
No	4,442,285	93.5 (90.3-96.8)	52,500,457	92.1 (90.7-93.4)
Doctor ever told: emphysema				
Yes	234,714	4.9 (2.5-7.4)	1,579,760	2.8 (2.2-3.4)
No	4,516,904	95.1 (92.6-97.5)	55,423,214	97.2 (96.4-97.8)
Doctor ever told: gout				
Yes	264,187	5.6 (3.0-8.0)	2,412,023	4.2 (3.3-5.1)
No	4,487,431	94.4 (91.9-96.9)	54,612,773	95.8 (94.9-96.7)
Doctor ever told: skin cancer				
Yes	285,855	6.0 (3.6-8.42)	3,189,124	5.6 (4.8-6.4)
No	4,465,763	94.0 (91.6-96.4)	53,835,671	94.4 (93.6-95.2)
Doctor ever told: other type of cancer				
Yes	162,970	3.4 (1.6-5.3)	1,656,493	2.9 (2.3-3.6)
No	4,588,647	96.6 (94.7-98.4)	55,368,303	97.1 (96.4-97.7)
Doctor ever told: sugar diabetes				
Yes	278,784	5.9 (3.4-8.3)	2,754,481	4.3 (3.9-5.8)
No	4,472,834	94.1 (91.7-96.6)	54,248,907	95.7 (94.2-96.1)

Notes: All reported values represent weighted estimates using Final Exam Weight: WTPFEX6.
 As per NHANES III analytical guidelines all coefficients of variation above 30% are flagged with ~.

* stastically significant chi-square test results (95%)

** statistically significant chi-square test result (99%)

tion. Of the health conditions examined, the prevalence of only one, cataracts, differed statically significantly amongst the two selenium status groups (5.6% in the higher SS group vs. 11.1 in lower SS group, $p= 0.02$).

Of the 13 individual foods and food groups examined, only two differed significantly among the selenium status groups (see Table 3, p.167). More men in the higher selenium status group take a vitamin and eat dark bread.

The only significant difference between the two serum selenium groups with regard to specific food, or food group involved the consumption of dark bread. In the lower selenium group, 29.6% (95% C.I. 22.6-36.1) of respondents said they ate ten or more serving of dark bread per month vs. 38.3%(95% C.I. 36.7-40.6) in the higher selenium status group.

The two selenium status groups also differed in their stated use of vitamin supplements. Perhaps not surprisingly, the prevalence rate of those that took a supplement was greater in the higher selenium status group than the lower selenium status group (38.2% vs. 29.5%). Unfortunately, the NHANES survey does not specify what types of vitamins or minerals an individual takes, and therefore there is no way of knowing whether an individual's supplement contained selenium, and if it did, how much.

On a number of different physical measures men, with lower selenium status differ significantly from men with higher levels of the mineral (Table 4, p.168 and Table 5, p.169). When looking at measures of cholesterol and at body mass index, the prevalence rates of men who are considered obese or overweight, or who have high cholesterol, do not differ significantly between the two selenium status groups see Table 4.

Of the five micronutrients examined, the mean levels of three; lycopene, calcium, and beta-carotene were significantly lower

in the low selenium group (see Table 5). The levels of the other two micronutrients, vitamins C and E, were also lower in this group, though not significantly. Mean lead levels were significantly higher in the low selenium group (4.81 ug/dL vs. 4.18 ug/dL, $p\le 0.05$).

Multivariate Analysis

A multiple logistic regression was performed to evaluate the relationship between the dependent variable (i.e., selenium status < 106 ng/mL) and the significant independent variables from the bivariate analysis. Micronutrients and toxins were not added to the model as they have not been shown in the literature to have a causal relationship to selenium status. The results of the multivariate analysis are presented in Table 6, p.169.

Multiple logistic regression analysis indicated that all but one of the explanatory variables in the model were significantly associated with a selenium status below <106 mg/mL.

The results of this analysis showed that those in the age cohorts below the age of 60 were less likely to have lower selenium status (i.e., age 20-39; OR= 0.63 CI 0.54-0.73 and age 40-59; OR= 0.49 CI 0.43-0.56). In addition, living in the South appears to be significant risk factor for lower selenium status when compared with the other three regions (i.e. Midwest vs. South; OR= 0.45; CI= 0.41-0.50, Northeast vs. South; OR= 0.32 CI= 0.29-0.35, West vs. South; 0.38; CI= 0.29-0.50). As well, higher/medium income reduces the likelihood of having lower selenium status (i.e. higher/medium income vs. lower income; OR=0.82; CI= 0.74-0.91) and as does being a non- smoker (i.e. non-smoker vs. smoker OR=0.45; CI= 0.37-0.54). With regard to the lone dietary variable, those that consume dark breads are less likely to have lower selenium status than those who do not (i.e. high consumption vs. no consumption; OR= 0.45; CI= 0.53-0.77,

Table 3. Dietary variable summary.

Selenium Status (ng/mL) (White US Males age ≥ 20 years)				
Characteristic	<106 ng/mL		≥ 106 ng/mL	
	Number	%(95% C.I.)	Number	%(95% C.I.)
Have you taken vitamins/minerals in past month*				
Yes	1,276,112	29.5 (22.6-36.4)	23,136,412	38.2 (35.9-40.5)
No	3,048,381	70.5 (63.6-77.4)	37,440,535	61.8 (59.5-64.1)
Dairy Servings Per Month				
<30				
30-59	1,124,706	26.0 (18.8-33.2)	12,423,909	20.5 (18.7-22.4)
>60	1,688,109	39.0 (31.3-46.8)	24,788,272	40.9 (38.6-43.3)
	1,511,679	35.0 (27.5-42.4)	23,334,133	38.5 (36.3-40.8)
Meat Servings Per Month				
<30				
30-59	840,923	19.4 (13.4-25.5)	13,826,558	22.8 (20.9-24.8)
≥ 60	2,330,544	53.9 (46.1-61.7)	30,937,235	51.1 (48.8-53.4)
	1,153,026	26.7 (20.1-33.2)	15,777,575	26.1 (24.0-28.1)
Fruit and Vegetable Servings Per Month				
<60				
60-119	1,386,639	32.1 (24.3-39.8)	16,891,090	27.9 (25.8-30.0)
≥ 120	1,819,827	42.1 (34.2-50.0)	25,389,361	41.9 (39.6-44.3)
	1,118,026	25.8 (19.6-32.1)	18,265,864	30.2 (28.1-32.2)
Cereals Servings Per Month				
<10				
10-29	2,316,019	53.6 (45.7-61.44)	32,499,575	53.6 (51.3-56.0)
≥ 30	1,211,444	28.0 (21.0-35.0)	17,180,364	28.4 (26.2-30.5)
	797,030	18.4 (13.2-23.7)	10,907,633	18.0 (16.3-19.7)
Serving of Dark Bread Per Month*				
0				
1-9	1,751,833	40.5 (32.8-48.2)	18,343,404	30.3 (28.2-32.4)
≥ 10	1,291,724	29.9(22.4-37.4)	18,984,227	31.4 (29.1-33.6)
	1,280,936	29.6 (22.6-36.1)	23,196,566	38.3 (36.7-40.6)
Servings of White Bread Per Month				
>9				
10-29	1,200,664	27.8 (30.4-35.2)	19,498,801	32.2 (30.0-34.4)
≥ 30	963,941	22.3 (15.5-29.1)	14,164,832	23.4 (21.3-25.6)

medium consumption vs. no consumption; OR= 0.78; CI= 0.69-0.91). Also, men that say they exercise the same or more than their peers are less likely to have a selenium status below 106 mg/ml (OR= 0.79; CI= 0.69-0.91). Finally, after controlling for the other explanatory variables, education level (i.e. graduated

high school vs. did not graduate from high school) was not shown to be a significant predictor of a selenium status below 106 mg/mL (OR 1.02; CI=0.92-1.12).

Discussion

This is the first study that has identified various factors which may contribute

Table 4. Prevalence of selected health characteristics of American men age 20 and over with higher and lower selenium status.

Selenium Status (ng/mL) (White US Males age >=20years)				
Characteristic	<106.4 ng/mL		>106.4 ng/mL	
	Number	%(95% C.I.)	Number	%(95% C.I.)
LDL Cholesterol				
Normal	1,582,032	83.9 (74.3-93.4)	19,114,719	80.0 (77.2-82.8)
High or Very High	304,453	16.1(6.6-25.7)	4,769,294	20.0 (17.2-22.7)
Serum Triglycerides				
Normal	2,693,802	77.5 (70.2-84.8)	35,012,979	78.6 (76.4-80.9)
High or Very High	782,187	22.5 (15.2-29.8)	9,509,224	21.4 (19.1-23.6)
Total Cholesterol				
Normal	4,049,366	85.2 (79.4-91.0)	46,695,648	81.9 (80.1-83.7)
High or Very High	702,252	14.8 (9.0-20.6)	10,324,385	18.1 (16.3-19.9)
Body Mass Index				
Normal Weight	1,514,447	32.7 (25.9-39.5)	21,860,526	38.7 (36.3-41.1)
Obese or Overweight	3,112,878	67.3 (60.7-74.1)	34,600,849	61.3 (58.9-63.6)

Source: The National Health and Nutrition Examination Survey NHANES III (1988-1994)

Notes: All reported values represent weighted estimates using Final Exam Weight: WTPFEX6.

As per NHANES III analytical guidelines all coefficients of variation above 30% are flagged with ~.

* stastically significant chi-square test results (95%)

** statistically significant chi-square test result (99%)

to a selenium status below 106 ng/mL in a nationally representative sample of White American men. The importance of this group comes from previous research that has demonstrated that men with a selenium status less than 106 ng/mL, supplemented with 200 µg of selenium, significantly reduce their risk of developing prostate cancer.²

Using the NHANES III survey this study estimated that between the years 1988-1994, 7.7% of White American adult men aged 20 years and older, a total of 4,751,618 individuals, had a selenium status of below 106 ng/mL.

Several of the more than forty,

social, economic, geographic, physical, and dietary characteristics examined by this study were shown to be significantly associated a lower selenium status. Risk factors for lower selenium status (106 ng/mL) identified by this study were; smoking, living in the South, an age of 60 years or older, exercising less than your peers, and having a lower income.

Based on the findings of this study it is difficult to identify an intervention strategy to increase the selenium status among men with lower levels of the mineral for a number of different reasons. In the past, micronutrient deficiencies and their associated illnesses were suc-

Table 5. Selected mean blood micronutrient and toxin levels of American men age 20 and over with higher and lower selenium status.

Selenium Status (ng/mL) (White US Males age \geq20years)				
Characteristic	<106.4 ng/mL		>106.4 ng/mL	
	Number	Mean (95% C.I.)	Number	Mean (95% C.I.)
Serum Vitamine E (ug/dL)	4,728,599	1076.3 (995.8-1156.8)	56,783,214	1171.0 (1147.2-1196.5)
Serum Beta Carotene (ud/dL)*	4,728,599	14.55 (13.14-16.0)	56,783,214	17.5 (16.6-18.4)
Serum Vitamin C (ug/dL)	4,717,095	0.61 (0.54-0.68)	56,353,064	0.69 (0.67-0.71)
Serum Lycopene (ud/dL)*	4,728,599	22.4 (20.2-24.6)	56,783,214	25.5 (25.0-26.1)
Serum Calcium (mmol/L)*	4,678,005	2.26 (2.25-2.28)	56,248,603	2.31 (2.30-2.32)
Serum Lead (ug/dL)*	4,751,617	4.81 (4.37-5.24)	56,992,361	4.18 (4.05-4.32)

* statistically significant t-test results (95%)

Table 6. Multiple Logistic Regression Results for Selenium Status <106 ng/mL.

Dependent Variable	P value	Odds Ratio	95%	C.I.
Selenium Status <106 ng/ml				
Significant Explanatory Variables				
Age (20-39 vs >60)	<.0001	0.63	0.54	0.73
Age (40-59 vs >60)	<.0001	0.49	0.43	0.56
Region (Midwest vs South)	<.0001	0.45	0.41	0.50
Region (Northeast vs South)	<.0001	0.32	0.29	0.35
Region (West vs South)	<.0001	0.38	0.29	0.50
Income (higher/medium income vs lower income)	0.0003	0.82	0.74	0.91
Smoking Status (non smoker vs smoker)	<.0001	0.45	0.37	0.54
Dark Bread Consumption (high consumption vs no consumption)	<.0001	0.64	0.53	0.77
Dark Bread Consumption (medium consumption vs no consumption)	0.0169	0.78	0.64	0.96
Exercise (more or the same active vs less active)	0.0014	0.79	0.69	0.91
Insignificant Explanatory Variable				
Education (graduated vs. not graduated)	0.7239	1.02	0.92	1.12

cessfully overcome with large scale food fortification, such was the case with iodized salt to prevent goiter, vitamin D fortified milk to prevent rickets, and folic acid supplemented grains to reduce the incidence of spina-bifida. With regard to making a case for supplementing a specific food in order to augment the selenium intake of those men with lower selenium status this study provides little guidance. The ideal food candidate for supplementation would be one that was consumed significantly more by men in the lower selenium status than in the higher group. With such a food, you would have the best chance of augmenting the selenium of status of those with lower serum levels of this mineral and minimize the odds (however small) of providing those with already higher levels of the with too much. However, of the 13 individual foods and food groups examined by this study, none were significantly consumed more by the lower selenium status group than by the higher status group.

Furthermore, the need to identify and perhaps implement a specific intervention aimed at augmenting selenium status is perhaps redundant. The modifiable factors for lower selenium status identified by this study were smoking, exercising less, and poverty, all of which are risk factors for many other diseases and are currently being addressed by several public health agencies in the United States.

There are two points are important to consider when interpreting the results from this study. First, the NHANES III survey examined the non-institutionalized civilian population of the United States, thus it does not include persons in institutions such as hospitals, nursing homes, or prisons. Second, it should be noted that the conclusions of this study are based upon a survey that was conducted from 1988-1994, as a result applying the results to today's (2007) population should be done with caution.

Conclusion

This study concludes that lower selenium status is significantly associated with various socio-economic, demographic, physical, and dietary factors. The factors reported in this study are consistent with risk factors for many diseases. Determinants of lower selenium status include modifiable lifestyle factors such as cigarette smoking and exercise which, if changed, could not only have a positive effect upon selenium status but decrease the risk of developing other diseases.

Cigarette smoking was one of the most striking determinants of low selenium status. However, it is unclear whether fortifying tobacco with selenium would be a good strategy to increase selenium status among men with depressed levels of the mineral. Laboratory experiments which have added selenium to tobacco show the mineral reduces the mutagenicity and toxicity of cigarette smoke.^{7,8} Yates and colleagues suggest that the mechanism by which selenium generates relief in cigarette smoke induced toxicity is to react with the constituents in the smoke itself and not by stimulating a protective mechanism in the cell. Thus, the action of adding selenium to tobacco appears to reduce the toxicity of the smoke as opposed to having a supplemental effect. In any case, the effects fortified tobacco with regard to selenium status in humans has not been studied. While this may be a good strategy to reduce the harmful effects of tobacco smoke, further research is needed to determine whether adding selenium to tobacco would have a positive effect upon selenium status.

This study was unable to identify a suitable food fortification vehicle in order to augment the selenium status of men low levels of the mineral using the monthly recall survey in NHANES III. A problem with the NHANES survey it that it simply asks for the number of servings of a certain food or food group that a

person consumes per month. This type of survey is problematic for a number of reasons. First, NHANES does not define a standard serving size for each food or food group, so one person may consider a serving of dark bread to be one slice, whereas another would consider it to be two slices. Second, NHANES does not account for different nutrient levels in the same type of food. For example, various types of dark bread may contain different concentrations of nutrients, including selenium. Other problems include memory lapses and the desire to appear more concerned about health than the interviewee really is. Future research could use a different dietary survey, such as a 24 hour dietary recall to perhaps gain a better perspective as to the eating habits of men with lower selenium status

Despite not identifying a clear fortification vehicle, increasing the selenium content of common food stuff, in order to augment selenium status on a population level, is a practice that should be considered and merits further investigation. As mentioned earlier, selenium supplementation has been shown to decrease the incidence rates of various types of illness from cancer to viral infections. The only dietary variable shown reduce the probability of depressed selenium status was the consumption of dark bread. While, dark bread consumption maybe a surrogate measure for a healthy diet in general, it has been shown to contain more than twice the amount of selenium per 100g (36.6 μg vs. 17.3 μg).¹⁰ Therefore, augmenting the selenium of white bread to the same level of dark bread could have a positive effect with regard to reducing the prevalence of lower selenium status. This type of fortification has being attempted before. In 2005, Waitrose launched selenium-fortified bread in Great Britain which contained approximately 40 μg of selenium per 100g.¹¹ Sales of this bread were slow and the product was later pulled

from the selves. The company blamed a lack of public awareness of the benefits of selenium for the sluggish sales of the product.¹²

Large scale fortification of foods with selenium, by means of widespread fertilization, has been safely conducted in Finland since 1984. Further research in this area could include estimating the impact of various levels of selenium in fertilizers and subsequent impacts on national and global (because of food exports) selenium status.

The findings of this study may have a further significance should the ongoing SELECT trial demonstrate that selenium supplementation has a protective effect against prostate cancer among men with lower selenium status. Should this be the case, the results of this study could be used to identify a target population that would benefit most from selenium supplementation. In the event that chemoprevention of prostate cancer with selenium is demonstrated to be effective among men with low levels of the mineral, this work could also be used as a screening tool to by physicians. Patients presenting with risk factors for low selenium status could be blood tested to determine if this was the case and if so, prescribed a daily selenium supplement. Though conservative, this approach would eliminate the possibility, however small of toxicity, and deliver the mineral in a controlled dose.

References

1. Clark LC, Combs GF Jr, BW Turnbull, EH et al: Effects of selenium supplementation for cancer prevention in patients with carcinoma of the skin. A randomized controlled trial. Nutritional Prevention of Cancer Study Group. *JAMA*, 1996; 276(24): 1957-63.
2. Duffield-Lillico AJ, BL Dalkin, ME Reid, et al: Selenium supplementation, baseline plasma selenium status and incidence of prostate cancer: an analysis of the complete treatment period of the Nutritional Prevention of Cancer Trial. *BJU International*. 2003; 91 (7): 608-12.
3. U.S. Department of Health and Human Ser-

- vices. 2005. United States Cancer Statistics: 1999–2002 Incidence and Mortality Web-based Report. Atlanta, GA: Centers for Disease Control and Prevention and National Cancer Institute; 2005. www.cdc.gov/cancer/npcr/uscs
4. National Center for Health Statistics (NCHS). 1996. Plan and Operation of the Third National Health and Nutrition Examination Survey, 1988–94. NHANES III reference manuals and repost (CD-Rom). *Centers for Disease Control and Prevention, Hyattsville, MD.*
 5. SAS Institute, Inc. SAS Language: Version 9.1. SAS Institute, Cary, NC. 2006.
 6. Shah, BV, Barnwell BG, Hunt PN, Lavange LM. *SUDAAN User's Manual, Release 9.0.* Research Triangle Institute, Research Triangle Park, NC. 2006.
 7. Combs GF: Selenium in global food systems. *Br J Nutr*, 2001; 85: 517-547.
 8. Yates IE, OT Chortyk, and JL Lanier: Selenium mediated reduction of the toxicity expression of cigarette smoke condensate in *Photobacterium phosphoreum*. *Bull Environ Contamin Toxicol*, 1986; 36 (2): 278-85.
 9. Chortyk OT, JL Baker, and WJ Chamberlain. Selenium-mediated reduction in the mutagenicity of cigarette smoke. *Environ Mol Mutagen*, 1988; 11(3): 369-78.
 10. U.S. Department of Agriculture, Agricultural Research Service (USDA). 2003. USDA National Nutrient Database for Standard Reference, Release 16. Nutrient Data Laboratory Home Page, url: <http://www.nal.usda.gov/fnic/food-comp>. Accessed July, 2007.
 11. Nutrilaw. 2005. Selenium Enriched Food. url: http://www.nutrilaw.co.uk/selenium_enriched_food.htm. Accessed Jul, 2007.
 12. Daniells S. 2006. Selenium supplements needed for elderly women, say researchers. url: <http://www.nutraingredients.com/news/ng.asp?n=64658-selenium>. Accessed July, 2007.

The Swedish Society for Orthomolecular Medicine Founded April 26, 2008



The photograph shows the dozen people present at this occasion. Back row, left to right: Tommy Lewander, MD, PhD, associate professor of neuroscience, special interest: polyunsaturated fats; Klas Cederwall, PhD, professor emeritus, Royal Institute of Technology; Bjorn Regland, MD, PhD, associate professor of neuropsychiatry; published in 1992 the hypothesis on a relationship between homocysteine and cognitive decline; Bo H. Jonsson, MD, PhD, Chairman; Bo Zackrisson, investigative medical journalist; Mats Humble, MD, special interest: vitamin D. Front row, left to right: Inger Hallqvist Lindvall, MD, Green Party politician; Birgitta Brunes, MD, special interest: MS; Ann Gardner, MD, PhD, special interest: mitochondrial medicine; Karin Munsterhjelm, MD, Vice-chairman, experienced in orthomolecular work in schizophrenia and thyroid disorder; Ulla Sandklef, former psychologist/psychotherapist, now organization analyst; Elisabet Carlsson, Master of Political Science, journalist

Origin of the 'Vitamin D Toxicity' Myth

Currently, around the world, renowned scientists urge us to increase the daily intake of vitamin D, especially in countries with moderate climates. Leading vitamin D experts and also more general nutritional scientists, like Walter Willett of Harvard School of Public Health, undersigned the Call to Action Statement of Grassroots Health, a public promotion organization for vitamin D in the US. These scientists

state: "Any risks of vitamin D inadequacy considerably exceed any risks of taking 2000 IU/day of vitamin D3, which the NAS-IOM regards as having no adverse health effect." <http://www.grassrootshealth.org>

However, adequate measures are not put in place by the responsible authorities. In my country, the Netherlands (with a moderate climate), it is still forbidden to recommend a food supplement exceeding 200 IU per day.

Possibly the main obstacle of the supposed toxicity of vitamin D, which already exists for decades, has been described by Reinhold Vieth, in 1999, in an excellent paper on the safety of vitamin D.¹ Vieth is professor at the University of Toronto and specializes in vitamin D. In 2003 he gave a presentation on this subject at the Orthomolecular Medicine Today Conference in Toronto. Vieth is also one of the signatories of the mentioned Action Statement. Vieth reveals in his paper of 1999 the origin of the 'vitamin D toxicity' myth:

“Throughout my preparation of this review, I was amazed at the lack of evidence supporting statements about the toxicity of moderate doses of vitamin D. Consistently, literature citations to support them have been either inappropriate or without substance. The statement in the 1989 US nutrition guidelines that 5 times the RDA for vitamin D may be harmful² relates back to a 1963 expert committee report,³ which then refers back to the primary reference, a 1938 report in which linear bone growth in infants was suppressed in those given 45–157.7 mg (1800–6300 IU) vitamin D/d⁴ The citation is not related to adult nutrition and it does not form a scientific basis for a safe upper limit in adults. The same applies to the statement in the 1987 Council Report for the American Medical Association that “dosages of 10,000 IU/d for several months have resulted in marked disturbances in calcium metabolism...and, in some cases, death.” Two references were cited to substantiate this. One was a review article about vitamins in general, which gave no evidence for and cited no other reference to its claim of toxicity at vitamin D doses as low as 250 mg (10,000 IU)/d.⁵ The other paper cited in the report that dealt with 10 patients with vitamin D toxicity reported in 1948, for whom the vitamin D dose was actually 3750–15 000 mg (150,000–600,000 IU)/d, and all patients recovered.⁶ If there is published evidence of toxicity in adults

from an intake of 250 mg (10 000 IU)/d, and that is verified by the 25(OH)D concentration, I have yet to find it.”

The discussion about the efficacy and safety of vitamin D is poisoned by this myth, which arose already in 1938, and was scientifically ‘white washed’ in 1989 by the National Academy of Sciences.

–Gert Schuitemaker, Ph.D.

References

1. Vieth R. Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety. *Am J Clin Nutr* 1999; 69(5):842-56
2. National Academy of Sciences. Recommended dietary allowances. 10th ed. Washington, DC: National Academy Press, 1989.
3. Blumberg RW, Forbes GB, Fraser D, et al. The prophylactic requirement and the toxicity of vitamin D. *Pediatrics* 1963;31:512–25.
4. Jeans PC, Stearns G. The effect of vitamin D on linear growth in infancy: II. The effect of intakes above 1,800 U.S.P. units daily. *J Pediatr* 1938;13:730–4.
5. Woolliscroft JO. Megavitamins: fact and fancy. *Dis Mon* 1983;29:1–56.
6. Eager JE, Meyran JC. Intoxication with vitamin D. *J Clin Endocrinol* 1948;8:895–910.

Action Statement on the internet:

http://www.grassrootshealth.org/_download/scientists%27%20letter%20050508.pdf

Correspondence

Some Comments on Folate

A first reading of the recent paper by Ware¹ provokes both an adverse reaction and a surprise; surprise because a paper critical of a vitamin has found entry into an orthomolecular journal, probably for the first time. On second reading, however, one notices the balancing act of the author. It gradually dawns upon the reader that the paper is not outrightly critical of the vitamin; it is scholarly, informative and balanced. That said, I am left wondering why the issue of mechanisms underneath the purportedly harmful effect of folic acid is not sufficiently discussed. Only the natural killer cell mechanism is discussed. Missing in the paper is the role of methylenetetrahydrofolate reductase (MTHFR) genetic polymorphism. Is it that the harmful unmetabolized folic acid (UMFA) is found only in those cases which have the genetic defect involving MTHFR? In such cases a higher, not lower, dose of folic acid is given. Now, in view of the paper by Ware, perhaps more of folate rather than folic acid should be given to the cases of genetic polymorphism. But then folate is a natural substance, to be obtained from vegetables. Taking high dose of folate through this natural route is costly and not a viable option.

Another mechanism that could be covered in the paper is the histamine factor. High intake of folic acid raises histamine level. Therefore, it is possible that high intake of folic acid would be harmful only in the cases which already have high level of histamine, the histadelics.

In conclusion, perhaps high doses of folic acid will be found harmful only for certain sub-classes of cases.

–Ratan Singh, Ph.D.
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Author's Reply

I am pleased that Dr. Singh realizes, as was pointed out in the article, that the paper in question is not in general critical of folic acid or anti-vitamin. At issue are high levels of consumption of the synthetic form.

As to why mechanisms, aside from the natural killer cell hypothesis, that might be responsible for the suspected adverse effects of unmetabolized folic acid were not discussed, the reason was the lack of studies that specifically focus on unmetabolized folic acid in this context. It is only recently that the connection with decreased natural killer cell activity is even mentioned in papers on folic acid, and most researchers treat synthetic folic acid and natural folate as identical and appear unaware of the potential for elevated circulating levels of the unmetabolized chemical. When studies are carried out that vary the folic acid/natural folate intake and thus the level of unmetabolized folic acid, its potential independent action is rarely investigated. In fact, one of the objectives of the paper was to encourage researchers to do just this. Such research would help answer the questions posed by Dr. Singh.

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London, Ontario
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Reference:

1. Ware WR: Raising concerns about unmetabolized folic acid. *J Orthomol Med*, 2008; 23: 43-51.

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For books cited:

2. Williams RJ: *Biochemical Individuality*. New York. John Wiley & Sons. 1973; 32-36.

For papers cited from books:

3. Cameron, E: Vitamin C, Carnitine and cancer. In. eds. Bland J. *A Year in Nutritional Medicine*. New Canaan, CT, Keats Publ. 1986; 115-123.

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