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Editorial

Patent Medicine and Orthomolecular Medicine

“All drug doctors are quacks.”
(attributed to Benjamin Franklin)

My (AWS) father spent most of his professional life with patents. He began as a patent draftsman, producing many technical illustrations for Eastman Kodak Co. in Rochester, NY. And, although patent illustrators are not allowed to sign their work, he did so anyway. He used Morse Code, and concealed his name in each drawing's broken shading lines. Later, he became a paralegal in the company's patent department. It was at this time that he took me, as a teenager, with him to work one day. Actually getting to his office was strikingly reminiscent of the opening of the television spy spoof *Get Smart*. We went through door after locked door, most with uniformed guards. Once he went to Washington, DC, with an attaché case handcuffed to his wrist. Cool!

Not everyone knows that there is a patent and copyright clause in the US constitution. Article 1, famous for its protection of free speech, also states that patents are intended “to promote the progress of science and useful arts, by securing for limited times to authors and inventors the exclusive right to their respective writings and discoveries”.¹ A patent grants an exclusive right to stop others from selling, making or even using the invention for a long period, typically 20 years. The patent is a negative right that prevents others from profiting from the invention. Infringing a patent monopoly has legal implications, and typically the patent holder will demand to be compensated financially. In some countries, patent infringement is a criminal activity.

Patents are supposed to drive innovation. The profits that can arise from a patent are said to promote investment in R&D, design and technical improvement. Since the patent is a published document,

others can keep up with the advance of the technology. Licensing the technology allows inventors to get their innovations manufactured and marketed. However, a company may acquire a patent simply to prevent it being exploited by its competitors. This may actually prevent innovation. In medicine and health care, patents all too often fail to promote the progress of science, and may actually hinder it.

Technology or Science

Patents are for technology and engineering; they are not part of the scientific method. Science distributes its knowledge openly without direct financial exploitation. The double helix model of DNA of Franklin, Crick and Watson was published in a short paper, although Franklin was not included as an author. This was a scientific breakthrough and not subject to patent. Eventually, the DNA model would help drive whole new areas of biotechnology, packed with patents and monopolies. The underlying science however is free and openly available with no restriction on its exploitation. Similarly, Alan Turing's description of a universal computer was unpatented basic science. Later, digital computers and software would become a highly profitable technology driving innovation in the latter half of the 20th century.

The idea that people need to be given monopolies for new ideas is contradicted in that the typical patent is a minor technical advance. These patented, small technical advances directly depend on the increase in fundamental scientific knowledge.

Pharmaceuticals

Medical patents exploit the sick for profit. They provide exclusivity rights to drugs and treatments and prevent competition.² The problem has been highlighted by developing countries which

are often unable to afford the inflated drug prices.³ Since these countries are not able to provide massive profits, the drugs that they need for malaria and other diseases may not be properly investigated or developed.

Recently drug companies have contributed a token portion of their profits to healthcare in developing countries. However, this can be interpreted as a minor aspect of public relations by companies that are characterised by marketing rather than R&D. Claims that patents and intellectual property laws contribute to a framework that allows for humanitarian and fair distribution of drug R&D are meaningless unless they are substantial.

The claim that drug companies need exclusive rights, a monopoly in the market, and inflated prices to reward the need for R&D is overstated and overplayed.⁴ The funds described as research and development may be exaggerated and can be lower than the marketing costs.⁵

Recent Nobel Prize winner Sir John Sulston described proprietary restriction on medicines as morally corrupt.⁶ The inequality in the availability of drugs has generated increasing anti-patent opinion. The lack of available HIV and AIDS drugs in several parts of the world has been a popular concern. Developing countries are challenging international patent law in medicine. Their argument is simple and could not be more clear: human lives are more important than profits for drug companies. Modern drug treatment for HIV/AIDS, tuberculosis and cancer are largely unavailable in many parts of the world. In 2002, Thailand switched to using generic antivirals manufactured in India, and the price dropped from over \$500 to about \$30. More recently, Brazil declared that the availability of the antiviral medication efavirenz was in the public interest and demanded appropriate prices. As the problems continue, other

developing countries may introduce local generic drugs, rejecting patents in favour of the public interest. Developing countries can override patent law in times of need but attempts to do so are likely to produce legal and political challenges.

Owning Life Itself

While the double helix was considered a scientific discovery belonging to humankind, it is now possible to patent genes. Patenting the genetic code is controversial and subject to challenge. A bill with the US Congress may invalidate patenting of human gene sequences.⁷ Patents on cells⁸ and whole living creatures⁹ have been applied for and granted. The scene was set for an entire higher animal to be patented, and sure enough, a patent on a mouse was granted to Harvard University in 1988.¹⁰ This Harvard mouse and its offspring are owned by DuPont with the registered trademark Oncomouse™. Perhaps the next step will be patented cheese to feed it. The patent for this particular genetic modification is extended to non-human mammals such as elephants or cats. However, objections to the patent in Europe, because plant and animal varieties were not patentable, were dismissed since the patent was not for a specific “animal variety”.¹¹ Similarly, humans are currently excluded—for now. Perhaps in future years the precise legal wording might not be taken to exclude humans containing a specific gene modification. By 2005, 20% of human genes were already subject to patent.¹² How far this takes us to the possibility of a new form of social Darwinism, or worse, where people are patented, trademarked, and owned, is an open question.

Medicine

Patents may be unsuitable for use in medicine and health care. Medicine is properly the application of science

to health. This application involves technology and the potential for patents. However, there is an ethical dilemma. Should a monopoly be allowed on a life saving treatment? When should it be justified for a sick person to suffer and die, because the monopoly holder will not make a sufficiently large profit from the treatment?

Sick patients are vulnerable and their vulnerability increases with the severity of the disease. A terminal patient may be willing to sell their car, house, and the future of their family for a cure. Medicine has fought hard to acquire legislation to prevent the unscrupulous peddling of quack cures. Indeed, the very term “patent medicines” emerged in the 19th century as a phrase associated with charlatans and the exploitation of the sick. Today, the vast profits that can be made from monopolies and exorbitant drug pricing in medicine has led to an inversion. Patent medicines are now seen as the evidence-based answer to disease. They are not. Not one cell in the human body is made from a drug, patented or not. Nutrients, quite unpatentable unless modified, are not even close to being as profitable as drugs are. The fact that nutrients are often more clinically effective, and that nutrients are invariably safer, does not enter the patent-pensive world of pharmaceutical finance. Nutrients are generic, and that’s a dead end. Ascorbic acid at \$35 a kilo does not excite stockholders and does not excite accountants. Wonder drugs do.

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Are Antipsychotic Drugs Safe?

Antipsychotic drugs can kill. Orthomolecular physicians have known for many decades that the use of antipsychotic drugs for patients with schizophrenia and bipolar disorder only rarely helps the patient, and indeed can actually prolong the patient’s illness. While in the short term they can help to bring some control to the condition, over the long term they interfere with the natural history of the illness converting what might have been a self-limiting state into one which is chronic and unrelenting.

For example, Bleuler, in his studies of the natural history of schizophrenia, long before the advent of the earliest antipsychotic drugs in the 1950s, showed

that at the first presentation of schizophrenia, one-third would become well again without recurrence, one-third would pursue a relapsing course (acute episodes alternating with remissions) until they became chronic, and one-third would become chronic.

In the hands of conventional psychiatrists who use antipsychotic drugs, the published studies seldom describe complete, drug-free remission.

Orthomolecular physicians frequently report complete,² drug-free remission in their patients using the full range of the orthomolecular armamentarium, i.e diet, vitamins, minerals, attention to pollutants and food sensitivities.

Because patients taking antipsychotic drugs alone do not feel well, cannot function normally in society, and cannot use whatever skills they may have, a small proportion do commit suicide, the first way that such drugs can kill.

Antipsychotic drugs are conventionally divided into two classes, the "typical" and "atypical". The typical drugs include Chlorpromazine, Thioridazine, Triifluoperazine, and Haloperidol. The atypical drugs include Clozapine, Olanzapine, Quetiapine, and Risperidone.

There is increasing epidemiological evidence linking the typical antipsychotic drugs with sudden cardiac death¹⁻⁴. The mechanism appears to be QT abnormalities, resulting in fatal *torsades de pointe*. Moreover the risk is dose dependent: the higher the dose the greater the risk, with older patients more at risk.

When the atypical antipsychotic drugs were introduced, they were promoted as being less prone to side effects and hence safer. However, no long term studies were carried out to demonstrate their safety compared with the typical drugs.

For all their claimed superiority over typical drugs, the long term patient compliance with all except the smallest doses does not seem to be superior over

the typicals. Moreover they do carry the increased risk of patient death by two mechanisms, unrelated to each other.

Clozapine is a special case. Its propensity to cause bone marrow suppression especially of the white blood cell progenitors is very well known with the risk of fatal agranulocytosis. A failure to organize regular complete blood counts with patients taking Clozapine is regarded as malpractice.

In general, patients taking atypicals are prone to marked weight gain. There may be two explanations for this. It may be a direct pharmacological action. Alternatively, or as well, such patients tend to have poor incomes ("mandated patient poverty") and, hence, be unable to afford anything other than cheap foods rich in refined carbohydrates.

The result is a rising incidence of Metabolic Syndrome (the combination of hypertension and non-insulin dependent diabetes mellitus) among such patients. This carries a serious risk of cardiovascular disease, often ultimately, and unacceptably, fatal.

What of sudden cardiac death? Ray and his colleagues from Tennessee have found that the incidence of sudden cardiac death from atypical antipsychotic drugs is similar to that of users of typical⁵. It was a remarkably well-performed epidemiological study. They used information from the state Medicaid system of tens of thousands of both typical and atypical antipsychotic drug users comparing with a matched, control group of nearly two hundred thousand non-users. The incidence of sudden death was higher² in both drug using groups. One interesting finding was that the incidence of sudden cardiac death among former drug users dropped to that of the control group.

In the corresponding editorial⁶, various measures were proposed to reduce the risk of sudden death, such as performing an ECG (EKG) on every patient before ini-

tiating such drug therapy, restricting their use in off-label situations (in children and the demented elderly), more strict attention to other cardiac risk factors, and markedly reducing the doses which are prescribed.

However, they made no mention of the role of orthomolecular techniques in mitigating the problem, a serious omission.

In my opinion initiating orthomolecular therapy simultaneously with the initiation of antipsychotic drugs is the only ethically acceptable policy. It has two important, relevant advantages: allowing an earlier reduction in the doses of the drugs (and even cessation entirely); and a direct cardio-protective effect from high doses of niacin and ascorbate,

This is not to say that antipsychotic drugs should not be used, since they do have their value in the appropriate circumstances. But they ought to be used only after a far more thorough medical, not just psychiatric, assessment of the patient has been performed, including such factors as homocysteine, folate, vitamin B₁₂, and thyroid status⁷. Then they ought to be used for as short a time as possible.

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What if this was your treatment for heart disease?

A growing community of people are choosing a natural way to help treat heart disease. It's called the orthomolecular approach to health, which emphasizes the right nutrients for your body. Using naturally occurring vitamins and minerals, you can help treat and prevent many conditions and illnesses — and make a difference in your health.



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Orthomolecular Treatment of Schizophrenia

Abram Hoffer, M.D., Ph.D; Frances Fuller, RNCP (Cand)¹

Introduction: An Apology and Explanation

For many years we have advised physicians and psychiatrists about the therapeutic advantages of giving their schizophrenic patients vitamin B₃ in the right doses as a part of the treatment they were already receiving. There were two general reactions: many psychiatrists became interested and spent one or more days with AH. With one exception they all became orthomolecular practitioners and many became the pioneers and leaders of this new field. However, very few doctors who did not visit AH ever tried to follow the treatment, even though it has been described over and over in many papers and books. Why the difference?

One factor was that the psychiatric profession became corrupted by the observation that the powerful psychiatric drugs quickly controlled abnormal behavior. It was concluded this was the cure, and was all one needed to do—similar to giving an antibiotic to a patient with pneumonia or putting a cast on a broken bone, it would heal rapidly, and the psychosocial aspects of the doctor-patient relationship did not matter very much. Psychiatrists concluded the very rapid changes induced by the drugs were the same as a cure. One day you would be dealing with a very agitated, hospitalized patient, and the next day he or she would be tranquilized and apathetic, apparently much better. One enterprising psychiatrist installed a noise meter in one of the chronic wards of his mental hospital and recorded the level of noise before and after tranquilizing his patients on that ward. He provided objective evidence that the noise levels went way down. He assumed this meant

patients were improved. All it showed is that they were less noisy.

What was not realized was that tranquilizing disturbed patients was not the same as curing them of the disease that had made them behave so badly in the first place. In the same way one can teach an autistic child new habits and ways of doing things, but it does not mean that their basic biochemical pathology is corrected. Depending upon these drugs as the treatment meant one could ignore all the other elements of a good treatment program—shelter, good food, civility and good care—which are part of any healthy doctor-patient relationship.

To give a bit of background, in the 1940s and 1950s psychiatric experience was usually gained in mental hospitals on patients for whom there was no treatment, and anything that would settle them was preferable to no treatment. A few doctors were more enterprising and were willing to use very harsh treatment such as insulin coma and ECT in order to help their patients; the results were not good, took a long time, and were unpredictable. Drugs appeared to settle all these issues. Discharging these patients—no better but tranquilized—became one of the new objectives of the mental hospitals. The clinical evidence that while drugs are helpful, they are not and never will be curative was, and is, ignored. The word “cure,” like the “N-word,” is forbidden in modern psychiatry.

The doctors who did not visit me (AH) did not see the results that I and the doctors who visited me were seeing. They were therefore not impressed by anything I wrote. Over the last years of my practise in psychiatry, forty medical students in their third or fourth year visited me and spent one or two days

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observing and interacting with me and my patients. They were completely surprised when they saw my recovered or recovering patients. During their training they had never seen even one schizophrenic patient who was as well.

We apologize because we did not understand that advising psychiatrists to add vitamins meant adding one new drug, so they could still ignore the other three essential elements of any good therapeutic program. Had we understood this, it might have been more effective to preach to doctors who were already practising good therapy including those three elements. Most of the early pioneers around 1960 were trained as psychoanalysts: Allan Cott, David Hawkins, Jack Ward, Harvey Ross, and Moke Williams. They were accustomed to spending a lot of time with their patients.

The total dependence on drugs eliminates the basic three elements of good treatment for any disease: shelter, food, and treatment with civility and respect. These are the basic elements of the Moral Treatment of the Insane practised by the Quakers 150 years ago, which allowed nearly half of their psychotic guests to recover without drugs. No doctors or nurses were allowed into these treatment homes. We will not discuss shelter and food, as this should be so obvious: living on the streets or rundown slum areas, 'dumpster diving' or eating modern hospital food is not good care. In this report we will concentrate on civility and care, and the doctor-patient relationship.

A second factor is that the journals usually read by psychiatrists refused to publish articles reporting positive orthomolecular findings. Many years ago an assistant editor of the American Journal of Psychiatry told me that he would never allow any of my papers to appear in his journal, no matter how good they were. He kept his word. But even worse is that MedLine, which is supposed to abstract

and review scientific articles in the world scientific press, undertook a censoring function to keep orthomolecular reports out of medical awareness. It resolutely refused to abstract our journal, considering that Readers' Digest is more scientific. There are also no ads in the standard medical journals extolling the virtues of vitamins, whereas up to fifty percent of the pages of some medical journals carry very impressive drug ads. We think that many journals are in fact advertising sheets with a little content so they can call themselves medical journals. This stranglehold on the public dissemination of information has come to an end with Google and other Internet search devices. This journal can now be downloaded from Google. Perhaps it is time to say goodbye to MedLine.

The Objective of the Doctor-Patient Relationship and Its Enhancement

We will describe a first interview with a schizophrenic patient and his mother in order to demonstrate our objective-recovery and how to achieve it. We believe patients must be taught something about their illness and must have hope that it can be treated successfully. We do not follow the usual mantra of modern psychiatry which is; (1) You will never get well; (2) You will never be off drugs; and (3) You will never complete your education. We have seen too many examples of patients who have been given this advice and have recovered.

On November 13, 2007, John came with his mother from hundreds of miles away. Age 20, he was tall, good-looking, quiet, and his face was frozen in anxiety. His mother looked weary and fearful. John had been diagnosed schizophrenic, or schizo-affective, and was on parenteral drugs. On his own he had stopped taking Zyprexa a month earlier with few withdrawal symptoms. When AH asked how could we help him, he was very vague and spoke very softly. Fortunately, prior to his

appointment, his mother had sent us a very good history of his illness.

I (AH) then opened up the topic of schizophrenia, telling him that I wished I had his genes but not that I wished to be sick. I emphasized that schizophrenia genes are good genes if you feed them properly, which meant giving his genes the vitamins he needed, especially niacin. Immediately he woke up and became much more interested. I assume he thought I would once more make him tell me his history.

I then outlined why in our opinion schizophrenic genes are such good genes. On the physical side their possessors tend to be good looking (he was), they aged gracefully, hardly ever got arthritis and rarely got cancer. We told him that out of 5,000 schizophrenic patients AH had seen, only ten had gotten cancer and they had all recovered with treatment that included vitamins. By this time he was wide awake. We then told him that, psychologically, possessors of these genes tend to be very intelligent, creative, and talented, and we described some of creative successful patients who had been treated. He told us he had received top marks in Grade 12 but after that he deteriorated, was struggling in his second year of university, and could not even hold minor jobs. He previously loved to play classical guitar and had been an excellent athlete.

Why did we use this approach? We did it because the information we gave him is true, as anyone reading AH's books will realize, and secondly, no one will ever recover without hope and a reason to live. The usual negative mantra of modern psychiatrists to their schizophrenic patients is correct, as they only use drugs.

The transformation in this young man in just a few minutes of discussion was amazing. He was now fully alert and taking part in the discussion. His mother now and then cried softly from relief, and kept saying she should have brought him

to see me a few years earlier. We also had to neutralize the word schizophrenia, so stigmatized and, of course, dead wrong. The term itself is meaningless, does not tell us anything about what is really wrong, and does not indicate the correct treatment. We told him that the correct term is pellagra and explained in detail what we meant.

What were we hoping to achieve? First, we had to start the process of giving him back his self-respect. He had been a very intelligent, creative young man and this had been taken away from him. He could again hold his head high, knowing that he had a biochemical disorder for which he was not to blame, and that this disorder, if treated properly, gave the possessor a whole set of highly desirable properties that most of us would like to have. Most people think that schizophrenic genes are bad genes. They are asked about them and a family history is taken, as if the whole family has been tainted. In our opinion there are no bad genes except for those that do not permit survival. If any individual has been well for even a short period of time, then the genes are not bad; they have been badly treated by not providing them with the essential nutrients in their environment, and by overwhelming them with the toxins with which our planet is now so overly loaded. If a brilliant scientist develops Alzheimer's disease at age 75, one cannot say that his or her genes were bad because they did so well for so many years. They have not been well treated (well fed) for about 20 years. With proper orthomolecular treatment they would continue to serve as good genes.

Our second objective was to restore hope that the condition was treatable. Until now all he could look forward to was a life of chronic pain, medication, failure, and indifference from the psychiatric profession. The best way to restore hope was to tell him stories about other patients who were equally sick who had

recovered; like the teenager with schizophrenia who was seen in 1973, now a professor at a famous university, or the teenaged girl practically on the streets, who recovered, married, raised her family, and then learned a new profession which she is pursuing successfully. These stories inspire hope and are very therapeutic.

The Treatment Approach

Then we asked about his diet and whether he had allergies. He did not think he had allergies but he did show some evidence of these including dark rings under his eyes, called allergy shiners, as well as a few white spots in his fingernails characteristic of dairy allergy. His mother told us that he drank a lot of milk when he was three years old, and although he did not have many colds or earaches, he did suffer many episodes of strep throat. We talked with him about the need to rule out whether he had an allergy or not. We advised him to totally eliminate all dairy products for one month, and gave him an instruction sheet to guide him. After the end of the month he would do a challenge test by eating a dairy product. To illustrate what I meant, I told him about a few patients I had seen and how they had responded. One particularly striking example was a young man, age 21, who complained he had been depressed all his life. After two weeks eliminating all dairy products he was normal, completely free of depression. He then ate some ice cream. Within two hours his depression had come back, and after another hour he was psychotic. He was very agitated all night, fell asleep in the morning, awakened after three hours and has been well since, off all dairy products.

Food allergies are trigger factors and have to be eliminated, as the constant inflammation of the gastrointestinal tract creates the 'leaky gut' syndrome and prevents the adsorption of nutrients, vitamins and minerals from the small

intestine. Milk intake is also associated with iron deficiency anemia and with zinc deficiency; being aware of this makes it easier for patients to accept that they will have to take nutrients in order to make up what they have been missing for many years.

Then we listed each of the nutrients John needed including the following recommendations:

tid three times daily

bid two times daily

od once daily

Niacin 500 mg tid after meals for two weeks, and then 1000 mg tid. This is a starting dose and one may have to go much higher depending upon the response. The most common minor and non-harmful side effect is the vasodilatation or flush. Niacin itself is the best anti-niacin-flush product and after a few days schizophrenic patients will have stopped flushing. The flush was discussed with him in detail so that he would not be surprised or frightened.

Vitamin C 1000 mg tid. This is a major antioxidant, anti-stress nutrient, and decreases the incidence of colds and the flu—a time when patients have an increased tendency to relapse.

B complex 100 mg od to replace some of the other B vitamins which have not been absorbed well for several years.

Vitamin D 6000 IU od in the winter and 4000 IU in the summer for Canadians. No Canadian gets enough from September to April unless they supplement, or holiday in Florida or California. Even in southern areas there is now so much unreasonable fear of the sun that many southern residents also need to take vitamin D.

Omega 3 essential fatty acids – salmon oil 1 gram tid

Zinc citrate 50 mg od. Dairy allergy often causes zinc deficiency and he had signs of deficiency.

He was advised to take all the pills

together at the end of his meals. Finally, he was advised that if he had any reaction to any of the pills that worried him, he should immediately call us by phone or contact us by email. All questions are usually answered within 24 hours,

The first part of the interview took about 30 minutes. The rest of the hour was open to questions from John and his mother. Every question was answered.

Discussion

At the time AH started in psychiatry, when no effective treatment was available, it was the fashion to prepare very long histories, almost brief biographies. The less that was known about causes and treatment, the more information was piled into the charts for the unfortunate secretaries to transcribe. This was based on Adolf Meyer's view that everything was important. But with growing orthomolecular experience over the past fifty years it has become clear that most of the history is not essential, unless it is needed for legal reasons or to impress one's superiors while a student or resident. A brief history such as is taken by doctors not practising psychiatry is adequate and should not take more than a few minutes. The only essential facts are when it started, what were the stresses (trigger factors), what was the treatment and response, and the present situation. Almost every schizophrenic patient AH saw was referred by their general physicians, as he did not accept any non-referred patients. Almost all had failed to respond to previous multi drug treatments or they would not have been referred. Usually the diagnosis was made by other doctors and psychiatrists and in most cases I agreed with it. Therefore, taking a history need not cut too much into the time needed for the real objective of the visit: to establish adequate treatment that will increase the patient's chance of becoming normal.

Orthomolecular treatment is so-

phisticated, effective and safe and not time-consuming as many more patients can be seen. Patients need not be seen as frequently because they recover, in contrast to those given only drugs. The saving in time and money is enormous; there is nothing more economical than recovery. Unfortunately, because the medical profession has not endorsed orthomolecular treatment and learned how to use it, patients are denied their chance for recovery and to take their place in a normal society. Sadly, it is a treatment for the people who can afford to travel long distances to get this treatment. It remains beyond the reach of the poor who have to remain dependent upon the drugs-only therapy offered to them and enforced by government. A few patients recovered by following the regimen outlined in AH's books. Some of these cases are described in *Mental Health Regained*, published by International Schizophrenia Foundation, Toronto, 2007.

Postscript December 1, 2007: John just emailed to tell us he was doing much better. According to him he has more energy, his sleeping patterns have returned to normal, his thoughts are much more organized and studying has been easier. He also said he is enjoying exercise and continues to hold athletic aspirations. John, who had been so vague about what was wrong with him, and somewhat withdrawn, especially during the early part of the consultation, made this email contact himself and was able to express clearly what was going right.



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Antioxidant Vitamins Reduce the Risk for Cancer: Part One

Michael J. Glade, Ph.D., FACN, CNS¹

Introduction

The published scientific evidence that increased consumption of antioxidant vitamins, vitamins C and E, reduces the risk of cancer has been growing over the last several decades. This review assesses the evidence that was reviewed previously by the Food and Drug Administration along with the evidence that has appeared since that review was completed. The conclusions that are drawn are based on the totality of publicly available scientific evidence, with emphasis on well-designed studies that were conducted in a manner which is consistent with generally recognized scientific procedures and principles and which provide credible scientific evidence. These conclusions are drawn with the recognition that an apparent finding of “no effect” is not equivalent to a finding of a “negative effect” and that studies that demonstrate neither beneficial nor harmful effects do not “oppose” studies that do observe a beneficial effect.

This scientific evidence reveals that vitamin C and vitamin E reduce the risk for cancer in general. Individually, they each reduce the risk of several site-specific cancers, including colon cancer, squamous cell carcinoma of the esophagus, gastric carcinoma, laryngeal cancer, lung cancer, cancer of the oral cavity, pancreatic cancer, pharyngeal cancer, renal cell cancer, cancer of the salivary glands, bladder cancer, brain cancer, cervical cancer, and rectal cancer.

Vitamin C Reduces the Risk for Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for cancer. In the 24-year

prospective Western Electric Company Study conducted in Chicago, IL, the risk of death from cancer was reduced significantly by greater intakes of vitamin C (RR, daily vitamin C intake 113 to 393 mg vs 21 to 82: 0.61; $p < 0.05$; adjusted for age, systolic blood pressure, BMI, serum total cholesterol concentration, smoking status, family history of cardiovascular disease, alcohol consumption and dietary intakes of energy, cholesterol, iron, saturated fatty acids and polyunsaturated fatty acids).¹ This protective effect of vitamin C was more pronounced among smokers. In another, 17-year prospective study of 2,974 men in Basel, Switzerland, mean serum vitamin C concentrations were significantly lower in men who died from cancer than they were in men who remained cancer-free.^{2,3}

Consistent with these reports, when men and women who had participated in the National Health and Nutritional Examination Survey II between 1976 and 1980 were contacted again, 12 to 16 years later, the adjusted risk of dying from any cancer was found to be increased significantly in men with serum ascorbate concentrations $< 28.4 \mu\text{M}$, compared to the risk in men with serum ascorbate concentrations $> 73.8 \mu\text{M}$, in 1976-1980 (RR: 1.62; 95% C.I.: 1.01, 2.59; adjusted for age, race, education, cigarette smoking, alcohol consumption, history of diabetes, serum total cholesterol concentration, systolic blood pressure and BMI).⁴ Women were not similarly affected. However, the results of observing a cohort of 11,580 initially cancer-free residents of a retirement community for 8 years indicated that the risk of developing cancer in women (but not in men) was inversely correlated with the daily consumption of vitamin

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C.⁵ In addition, in a case-control study, persons with cancer affecting different sites (breast, head and neck, genitourinary, lung, gastrointestinal and others) exhibited significantly lower mean serum vitamin C concentrations.⁶

On the other hand, the results of a 13.8-year prospective observational study of 2,112 Welsh men indicated that differences in vitamin C intakes did not affect mortality from cancers of the respiratory tract or from cancers of the digestive tract (adjusted for age, smoking status, social class, BMI, daily intakes of total energy and fat and alcohol consumption).⁷ In the 8-year prospective Nurses' Health Study of 89,494 women in the US, the risk of developing cancer was not affected by differences in vitamin C intakes.⁸ Consistent with these reports, in a prospective observational study of 605 men and women with coronary heart disease, there were no differences in the average vitamin C intakes between those subjects who developed cancer during the study and those who did not.⁹ Similarly, in a 28-year prospective observational study in Washington County, MD, differences in vitamin C intake had no effect on hazard ratios for all-cause mortality or death from cancer but 50% of subjects consumed less than the RDA for vitamin C.¹⁰ These data suggest that among vitamin C deficient adults, the degree of deficiency has no effect on all-cause mortality or death from cancer and increased risk for premature death is a feature of chronic vitamin C deficiency.

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for cancer. The evidence documented by a prospective observational study⁵ and a retrospective observational study⁶ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for cancer. In addition, the scientific evidence indicates that increased consumption

of vitamin C reduces the risk for death from cancer. The evidence documented by 4 prospective observational studies¹⁻⁴ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for death from cancer.

Vitamin C Reduces the Risk for Bladder Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for bladder cancer. The results of several retrospective observational studies are consistent with this conclusion. In a case-control study conducted in Los Angeles, CA, compared to the consumption of less than 62 mg/day of vitamin C, the consumption of more than 168 mg/day of vitamin C reduced significantly the multivariate-adjusted odds of developing bladder cancer (OR: 0.52; 95% C.I.: 0.56, 0.95; adjusted for education, number of cigarettes smoked per day, number of years smoking, current smoking status, lifetime use of nonsteroidal anti-inflammatory drugs and number of years employed as a hairdresser or barber).¹¹ In a similar case-control study of middle-aged men and women conducted in Washington State, individuals consuming the most dietary vitamin C experienced significantly less risk for bladder cancer (OR, dietary vitamin C intake > 156 mg/day vs < 78 mg/day: 0.50; 95% C.I.: 0.28, 0.88; adjusted for age, sex, county, smoking and daily energy intake).¹² Similarly, individuals who consumed the most vitamin C from dietary supplements experienced significantly less risk for bladder cancer (OR, supplemental vitamin C intake > 502 mg/day vs none: 0.40; 95% C.I.: 0.21, 0.76; adjusted for age, sex, county, smoking and daily energy intake) and individuals who consumed the most total vitamin C from foods and dietary supplements experienced significantly less risk for bladder cancer

(OR, total vitamin C intake from foods and dietary supplements > 335 mg/day vs < 95 mg/day: 0.45; 95% C.I.: 0.26, 0.79; adjusted for age, sex, county, smoking and daily energy intake).¹² Consistent with the results of these studies conducted within the US, investigators reported that men and women in Turkey with grade 1, 2 or 3 transitional cell carcinoma of the bladder had significantly lower serum concentrations of vitamin C than cancer-free men and women.¹³

In contrast, an epidemiologic analysis of the data obtained during the prospective, double-blind, randomized, placebo-controlled Alpha-Tocopherol, Beta-Carotene Cancer Prevention study of 29,133 middle-aged male cigarette smokers in Finland who supplemented their diets with 50 mg of vitamin E, 20 mg of beta-carotene or placebo for 5 to 8 years, indicated that the risk of developing bladder cancer was not affected by differences in the dietary vitamin C intakes of smokers.¹⁴ However, the results of this epidemiologic analysis are relevant only to populations that match the parent experiment's subjects – middle-aged male life-long cigarette smokers, and despite the design of the parent experiment, carry no more “weight” than any other epidemiologic findings.

Several other prospective observational studies have failed to document a chemopreventive effect of vitamin C against bladder cancer. The results of the 12-year prospective observational Health Professionals Follow-Up Study of 51,529 initially cancer-free men aged 40 to 75 years indicated that the risk for bladder cancer was not affected by differences in vitamin C intakes (adjusted for cigarette smoking, region of the US, total daily fluid intake and total daily consumption of cruciferous vegetables).¹⁵ Similarly, in the 20-year prospective Nurses' Health Study of 88,796 women in the US, differences in daily vitamin C intakes from

foods or supplements did not affect the multivariate-adjusted risk of developing bladder cancer (adjusted for age, pack-years of cigarette smoking, current smoking status and total daily energy intake).¹⁶ In the largest of such studies, the 16-year prospective observational American Cancer Society Cancer Prevention Study II of 991,522 men and women in the US, the regular consumption of any amount of supplemental vitamin C for any length of time had no effect on the risk of dying from bladder cancer.¹⁷ A lack of effect of vitamin C consumption on the prevention of bladder cancer also has been observed outside of the US; for example, the results of a 6.3-year Dutch prospective observational study of 58,279 men and 62,573 women aged 55 to 69 years (the Netherlands Cohort Study) indicated that the age- and sex-adjusted risk of developing bladder cancer was not affected by differences in vitamin C intakes.¹⁸

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for bladder cancer. The evidence documented by three retrospective observational studies¹¹⁻¹³ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for bladder cancer.

Vitamin C Reduces the Risk for Breast Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for breast cancer. In an 8-year prospective observational study of 59,036 women aged 40 to 76 years in Sweden (the Swedish Mammography Cohort), among women with BMI > 25, consuming more than the RDA for vitamin C reduced significantly the risk of developing breast cancer (HR: 0.61; 95% C.I.: 0.45, 0.82; adjusted for age, family history of breast cancer, BMI, education, parity, age at first birth, total daily energy intake, alcohol consumption and daily intakes of dietary

fiber, monounsaturated fatty acids and polyunsaturated fatty acids).¹⁹

The results of several retrospective observational studies also support the conclusion that increased consumption of vitamin C reduces the risk for breast cancer.²⁰⁻³² In a case-control study conducted in western New York state, the multivariate-adjusted odds of developing breast cancer were reduced significantly among premenopausal women by daily vitamin C intakes greater than 223 mg (OR, daily vitamin C intakes > 223 mg vs < 132 mg: 0.53; 95% C.I.: 0.33, 0.86; adjusted for age, education, age at first birth, age at menarche, history of first-degree relatives with breast cancer, personal history of benign breast disease, BMI and total daily energy intake).²⁰ This significant reduction in risk was independent of the intakes of other dietary antioxidants and did not require but was not attenuated by dietary supplementation with vitamin C, although the protection afforded by supplemental vitamin C became slightly less important with increasing consumption of vegetables. In this study, the multivariate-adjusted odds of developing breast cancer were reduced significantly in both premenopausal and postmenopausal women without a family history of breast cancer and who consumed the most vitamin C (OR, premenopausal women with daily vitamin C intake > 232 mg vs < 132 mg: 0.7; 95% C.I.: 0.5, 0.9; OR, postmenopausal women with daily vitamin C intake > 232 mg vs < 132 mg: 0.6; 95% C.I.: 0.4, 0.9; both adjusted for age, education, age at menarche, age at first pregnancy and BMI).²¹ These protective effects were not enjoyed by similar premenopausal women who had a positive family history of breast cancer, suggesting that these adequate but relatively modest intakes of vitamin C were insufficient to override other predisposing factors.²¹

In a case-control study conducted in Germany, the odds of developing breast

cancer were halved by vitamin C intakes greater than the RDA (OR, vitamin C intake > 134.4 mg/day vs < 58.5: 0.49; 95% C.I.: 0.28, 0.88; adjusted for age, total daily energy intake, age at menarche, age at first birth, age at menopause, family history of breast cancer, current smoking status, personal history of benign breast disease, BMI, daily alcohol consumption and current or recent use of hormone replacement therapy).²² In a case-control study conducted in Seoul, Korea, the odds of developing breast cancer were reduced significantly by daily vitamin C intakes greater than 210 mg (compared to daily vitamin C intakes less than 100 mg, OR: 0.37; 95% C.I.: 0.19, 0.84; adjusted for age at menarche, total number of menstrual periods, parity, total number of full-term live births, total months of breastfeeding, family history of breast cancer and BMI).²³

In a case-control study conducted in Moscow, USSR, the odds of developing breast cancer in postmenopausal women were reduced significantly by vitamin C intake (OR, greatest vitamin C intake vs the lowest: 0.20; 95% C.I.: 0.06, 0.70).²⁴ In another case-control study conducted in Navarra, Spain, the odds of developing breast cancer were reduced significantly by the consumption of vitamin C (OR, greatest vitamin C intake vs the lowest: 0.40, 95% C.I.: 0.2, 0.9).²⁵ In another case-control study of women conducted in western India, the odds of developing breast cancer were significantly lower among women who consumed the most vitamin C, compared to the odds among women who consumed the least (OR: 0.42; 95% C.I.: 0.22, 0.80).²⁶ In another case-control study conducted in Uruguay, the odds of developing breast cancer were reduced significantly by moderately increased daily vitamin C intakes (OR, 3rd quartile of vitamin C intake vs 1st quartile: 0.61; 95% C.I.: 0.40, 0.93; adjusted for age, residence, urban or rural status, family history of

breast cancer in a first-degree relative, BMI, age at menarche, parity, menopausal status and total energy intake).²⁷

In another more recent case-control study conducted in Uruguay, the likelihood of breast cancer in premenopausal women was inversely correlated with vitamin C intake.²⁸ The data collected from a cross-sectional ecological survey in 65 Chinese rural counties indicated that breast cancer mortality was inversely correlated with serum ascorbate concentrations.²⁹ In other case-control studies conducted in Shanghai, China,³⁰ Tianjin, China,³¹ Italy,³¹ and Switzerland,³² the odds of developing breast cancer were significantly inversely correlated with daily vitamin C intake. In addition, in a case-control study of women conducted in western India, the odds of developing breast cancer were significantly lower among women with the highest plasma ascorbate concentrations, compared to the odds among women with the lowest (OR: 0.23; 95% C.I.: 0.10, 0.53).²⁶ (Circulating concentrations of vitamin C can be used as biomarkers of exposure to dietary vitamin C; even small changes in vitamin C intake are reflected in changes in plasma ascorbate concentration.³³)

The results of a meta-analysis of retrospective case-control studies indicated that there was a statistically significant inverse association between vitamin C intake and risk for breast cancer.³⁴ In addition, other investigators performing a meta-analysis of published data on the relationship between breast cancer and the intake of vitamin C also concluded that the risk of developing breast cancer was reduced significantly by vitamin C consumption (RR, "high" daily consumption of vitamin C vs "low": 0.80; 95% C.I.: 0.68, 0.95).³⁵

In contrast to this large body of evidence demonstrating that increased consumption of vitamin C reduces the risk for breast cancer, the prospective

observational data collected from women during the Nurses' Health Study and Nurses' Health Study II in the US failed to reveal a relationship between vitamin C consumption and the incidence of breast cancer.³⁶⁻³⁸ After the first 6 years of the prospective Nurses' Health Study II of 58,628 women in the US, differences in total vitamin C intakes from foods and supplements had no effects on the adjusted risks of developing nonproliferative benign breast disease, proliferative benign breast disease without atypia or benign breast disease with atypical hyperplasia (adjusted for age, time period, total daily energy intake, supplement use, family history of breast cancer, oral contraceptive use and BMI).³⁶ After 8 years, the results of the prospective observational Nurses' Health Study II of 90,655 premenopausal women aged 26 to 46 years, the multivariate-adjusted risk of developing breast cancer was not affected by differences in the daily intakes of vitamin C from foods or from foods plus supplements (adjusted for age, smoking status, height, parity, age at first full-term birth, BMI, age at menarche, family history of breast cancer, personal history of benign breast disease, oral contraceptive use, menopausal status, alcohol consumption, daily energy intake and daily intake of animal fat).³⁷ Similarly, in the 14-year prospective Nurses' Health Study of 83,234 women in the US, the multivariate-adjusted risk of developing breast cancer was not affected by differences in daily intakes of vitamin C from foods alone or from foods and dietary supplements (adjusted for age, length of follow-up, daily energy intake, parity, age at first birth, age at menarche, history of breast cancer in a mother or sister, history of benign breast disease, alcohol consumption, BMI at age 18 years, change in body weight since age 18 years, height, age at menopause and postmenopausal hormone therapy).³⁸

Three other prospective observational

studies also failed to reveal a relationship between vitamin C consumption and the incidence of breast cancer.³⁹⁻⁴¹ In a prospective observational study of 34,387 postmenopausal women in the state of Iowa in the US (the Iowa Women's Health Study), the multivariate-adjusted risk of developing breast cancer was not affected by differences in vitamin C intakes (adjusted for age, daily energy intake, age at menarche, age at menopause, age at first live birth, parity, BMI at entry into study, BMI at age 18 years, family history of breast cancer, personal history of benign breast disease, alcohol consumption and education).³⁹ In addition, data obtained from 4,697 women, initially cancer-free and aged 15 years or older, after 25 years of observation failed to reveal a significant relationship between differences in daily vitamin C intakes and the occurrence of breast cancer⁴⁰ and after the first 4.3 years of a prospective observational study of 62,573 women aged 55 to 69 years (the Netherlands Cohort Study), the risk of developing breast cancer was not affected by differences in vitamin C intakes.⁴¹

The results of several retrospective observational studies⁴²⁻⁵² also failed to demonstrate the protective effect of increased vitamin C consumption against breast cancer. In a case-control study of women conducted in North Carolina, the multivariate-adjusted odds of developing breast cancer were not affected by dietary supplementation with any amount of vitamin C (adjusted for age, age at menarche, age at first full-term pregnancy, menopausal status, lactation history, family history, BMI, waist-to-hip circumference ratio, education, alcohol consumption, smoking history and daily intakes of fruits and vegetables).⁴² Similarly, the odds of developing breast cancer were not affected by differences in vitamin C intakes in upstate New York.⁴³ Investigators performing a case-control

study nested within the Canadian National Breast Screening Study of 56,837 women, also reported that the multivariate-adjusted odds of developing breast cancer were not affected by differences in the daily intakes of vitamin C from either foods or dietary supplements (adjusted for age, daily energy intake, age at menarche, surgical menopause, age at first live birth, education, family history of breast cancer, and personal history of benign breast disease).⁴⁴

In a set of case-control studies conducted in China (the Shanghai Nutrition and Breast Disease Study⁴⁵ and the Shanghai Breast Cancer Study⁴⁶⁻⁴⁸), differences in vitamin C intakes had no effects on the odds of developing nonproliferative benign breast disease, proliferative benign breast disease without atypia or proliferative benign breast disease with atypical hypertrophy. In case-control studies conducted in Italy, the energy-adjusted odds of developing breast cancer were not affected by differences in vitamin C consumption.^{49,50} In case-control studies conducted in Greece, the odds of developing breast cancer were not affected by differences in vitamin C intakes.^{51,52}

In a case-control study nested within the Danish Diet, Cancer and Health Study of postmenopausal women, the odds of developing breast cancer were reported to increase significantly with increased intake of vitamin C, an anomalous finding that the investigators could not explain and considered artefactual.⁵³

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for breast cancer. The evidence documented by a prospective observational study,¹⁹ 13 retrospective observational studies²⁰⁻³² and 2 meta-analyses^{34,35} supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for breast cancer.

Vitamin C Reduces the Risk for Cervical Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for cervical cancer. The results of several retrospective observational studies support the conclusion that increased consumption of vitamin C reduces the risk for cervical cancer.⁵⁴⁻⁵⁶ Most importantly, in a case-control study conducted in the Seattle, WA, area, the odds of developing cervical cancer were halved ($p < 0.05$) by increased daily intakes of vitamin C.⁵⁴ In addition, the results of a case-control study conducted in four Latin American countries indicated that the odds of developing cervical cancer were inversely correlated with vitamin C intakes.⁵⁵ In a case-control study conducted in India, the odds of developing cervical cancer and the severity of cervical cancer were both inversely correlated with serum ascorbate concentrations.⁵⁶

In contrast, the results of a 2-year, double-blind, placebo-controlled, randomized, factorial study in which women with colposcopically and histologically confirmed minor squamous atypia or cervical intra-epithelial neoplasia (CIN; an established precursor lesion to cervical cancer) supplemented their diets with either placebo, 30 mg beta-carotene, 500 mg vitamin C or 30 mg beta-carotene plus 500 mg vitamin C suggested that the rate of lesion regression was not accelerated by supplementation with this amount of vitamin C.⁵⁷ The results of several retrospective observational studies are consistent with this conclusion.⁵⁸⁻⁶⁰ In a case-control study conducted in the state of Hawaii, the multivariate-adjusted odds of developing cervical dysplasia (a precursor to cervical cancer) were not affected by differences in plasma ascorbate concentrations (adjusted for age, ethnicity, smoking status, alcohol consumption and presence or absence of human papillomavirus).⁵⁸ In a case-control study conducted

in the state of Alabama in the US, the multivariate-adjusted odds of developing cervical dysplasia were not affected by differences in vitamin C intakes (adjusted for age, race, age at first intercourse, number of sexual partners, parity, smoking status, use of oral contraceptives and presence of human papillomavirus infection).⁵⁹ In a case-control study conducted in the Portland, OR area, the age-adjusted odds of developing precancerous cytological abnormalities of the cervix were not affected by differences in daily vitamin C intakes.⁶⁰

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for cervical cancer. The evidence documented by three retrospective observational studies⁵⁴⁻⁵⁶ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for cervical cancer.

Vitamin C Reduces the Risk for Colon Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for colon cancer. In a prospective study that compared patients with adenomatous colonic polyps (an accepted risk factor for colon cancer) to subjects without polyps, one month of dietary supplementation with vitamin C (750 mg/day) produced a significantly greater decrease in cell proliferation within crypts of macroscopically normal-appearing colonic mucosa in subjects with polyps than was produced by placebo consumption, while there was no change in subjects without polyps – suggesting that vitamin C does not interfere with normal cell cycling but does slow abnormally accelerated proliferation in the colon epithelium.⁶¹ Consistent with this evidence of a protective effect of supplemental vitamin C, in a prospective observational study of 35,215 women aged 50 to 69 years in Iowa (the Iowa Women's

Health Study), the age-adjusted risk of developing colon cancer was reduced 33% in women who consumed more than 60 mg of supplemental vitamin C daily, compared to the risk in women who did not consume vitamin C supplements (RR: 0.67; 95% C.I.: 0.49, 0.92).⁶²

The results of several retrospective observational studies also support the conclusion that increased consumption of vitamin C reduces the risk for colon cancer.⁶³⁻⁶⁵ In the case-control North Carolina Colon Cancer Study, a group of men and women with "high" vitamin C intakes (median: 644 mg/day) experienced half the risk for colon cancer than was experienced by another otherwise similar group of men and women with "low" vitamin C intakes (median: 59 mg/day; OR: 0.5; 95% C.I.: 0.3, 0.8).⁶³ The responses of whites and African-Americans to vitamin C intake were not different.⁶³ On average, individuals with colon cancer consumed significantly less vitamin C, although vitamin intakes appeared to have no effect on the relative incidence of microsatellite instability (a biomarker for risk for colon cancer).⁶⁴ Similarly, in a case-control study conducted in the Seattle, Washington area, the age- and sex-adjusted odds of developing colon cancer were reduced significantly in men and women who supplemented their diets with vitamin C (OR, daily supplemental vitamin C intake > 500 mg vs none: 0.61; 95% C.I.: 0.40, 0.91).⁶⁵

In a case-control study conducted in Shanghai, China, the odds of men developing colon cancer also were reduced significantly by greater daily intake of vitamin C (OR, vitamin C intake > 30 mg/day vs < 30 mg/day: 0.7; 95% C.I.: 0.5, 0.9), although the odds of women developing colon cancer were not affected by differences in vitamin C intakes.⁶⁶ However, in a 17-year prospective study of 2,974 men in Basel, Switzerland, in which dietary and lifestyle patterns were assumed to remain static,

differences in prestudy serum vitamin C concentrations had no effect on the risk of developing colon cancer, a result that may reflect changing dietary and lifestyle patterns during the last quarter of the 20th century more than inherent relationships between vitamin C and the colon epithelium.^{2,3}

In a case-control study conducted in Denmark, the odds of adenomatous polyp recurrence were inversely correlated with daily intakes of vitamin C.⁶⁷ In contrast, in other case-control studies, the odds of adenomatous polyp occurrence⁶⁸ or recurrence⁶⁹ were not affected by differences in daily vitamin C intakes.

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for colon cancer. The evidence documented by a prospective clinical trial of vitamin C supplementation,⁶¹ a prospective observational study⁶² and 5 retrospective observational studies⁶³⁻⁶⁷ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for colon cancer.

Vitamin C Reduces the Risk for Colorectal Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for colorectal cancer. The results of the 14-year prospective observational American Cancer Society Cancer Prevention Study II of 711,891 men and women who were initially cancer-free indicated that the age- and sex-adjusted risk of developing colorectal cancer was reduced significantly by 10 or more years of dietary supplementation with any amount of vitamin C (OR: 0.77; 95% C.I.: 0.6, 0.90).⁷⁰ In addition, the results of several retrospective observational studies support the conclusion that increased consumption of vitamin C reduces the risk for colorectal cancer.⁷¹⁻⁷⁶

In a case-control study conducted in

France, the multivariate-adjusted odds of developing colorectal adenoma were reduced significantly by the consumption of greater amounts of vitamin C (OR, men, daily vitamin C consumption > 114 mg vs < 61 mg: 0.6; 95% C.I.: 0.4, 0.9; OR, women, daily vitamin C consumption > 114 mg vs < 61 mg: 0.6; 95% C.I.: 0.4, 0.9; both adjusted for age, sex, BMI, tobacco use, daily energy intake and alcohol consumption).⁷¹ In a case-control study conducted in Italy, the multivariate-adjusted odds of developing colorectal cancer were reduced significantly by increased vitamin C intakes (OR, vitamin C intake > 188 mg/day vs < 189 mg/day: 0.72; 95% C.I.: 0.6, 0.9; adjusted for age, study center, sex, education, level of physical activity and daily intakes of energy and dietary fiber).⁷² In a case-control study conducted in the Canton of Vaud, Switzerland, the multivariate-adjusted odds of developing colorectal cancer were reduced significantly by “intermediate” intakes of vitamin C (median: 112 mg/day) compared to “low” intakes (median: 65 mg/day; OR: 0.51; 95% C.I.: 0.3, 0.8; adjusted for age, sex, education, smoking status, alcohol consumption, BMI, level of physical activity and daily intakes of energy and dietary fiber).⁷³ Consistent with these findings, the results of a case-control study conducted in northern Italy indicated that the odds of developing colorectal cancer were reduced significantly by vitamin C consumption (OR, 5th quintile of daily vitamin C intake vs 1st quintile: 0.40; $p < 0.05$)⁷⁴ and in a case-control study conducted in western New York state, the odds of developing colorectal cancer were inversely correlated with vitamin C intakes.⁷⁵ In addition, men in Turkey with colorectal tumors had significantly lower mean plasma vitamin C concentration than healthy men.⁷⁶

In contrast, the results of a double-blind, randomized placebo-controlled clinical trial in which men and women

supplemented their diets with either placebo, beta-carotene (25 mg/day), vitamin C (1000 mg/day) plus vitamin E (400 mg/day) or all three antioxidants for 4 years indicated that combined dietary supplementation with this amount of vitamin C did not affect the incidence of colorectal adenoma (RR: 1.08; 95% C.I.: 0.91, 1.29; adjusted for age, sex, number of prior adenomas, actual length of time between clinical evaluations and study center).⁷⁷ Consistent with this finding, in a 2-year double-blind randomized placebo-controlled human clinical trial in which patients who were thought to be free of colorectal polyps after polyp removal added either placebo or a supplement containing 400 mg of vitamin C and 400 mg of vitamin E to their diets, the multivariate-adjusted risk of developing new polyps was not affected by the combined supplement (adjusted for age and the usual frequency of consumption of meats and fish).⁷⁸

Also consistent with these findings, the results of a secondary endpoint analysis of the data obtained during the prospective, double-blind, randomized and placebo-controlled Alpha-Tocopherol, Beta-Carotene Cancer Prevention study of 29,133 middle-aged male cigarette smokers in Finland who supplemented their diets with 50 mg of vitamin E, 20 mg of beta-carotene or placebo for 5 to 8 years indicated that the risk of developing colorectal cancer was not affected by the intake of vitamin C, although more than 50% of these subjects consumed less than the RDA for vitamin C.⁷⁹ However, the placebo-controlled trials were of inadequate duration to measure accurately the incidence of new polyps or tumors; even in patients who have undergone polypectomy, the minimum time before re-examination recommended by the 2006 Consensus Update on Guidelines for Colonoscopy after Polypectomy of the US Multi-Society Task Force on Colorectal

Cancer and the American Cancer Society is 5 years.⁸⁰

The results of two retrospective observational studies failed to support the conclusion that increased consumption of vitamin C reduces the risk for colorectal cancer.^{81,82} In a case-control study conducted in Los Angeles, CA, the multivariate-adjusted odds of developing colorectal adenoma or colorectal adenomatous polyps were not affected by differences in vitamin C intakes from foods or from supplements (adjusted for daily intakes of calories, saturated fat, folate and fiber, alcohol consumption, current smoking status, BMI, race, level of daily physical activity and use of nonsteroidal anti-inflammatory drugs).⁸¹ In another case-control study conducted in North Carolina, the multivariate-adjusted odds of developing colorectal adenoma were not affected by differences in vitamin C intakes in men or women (adjusted for age, BMI, daily energy intake, smoking status, use of dietary supplements, family history of colon cancer and daily intakes of fat, dietary fiber and alcohol).⁸²

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for colorectal cancer. The evidence documented by a prospective observational study⁷⁰ and six retrospective observational studies⁷¹⁻⁷⁶ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for colorectal cancer.

Vitamin C Reduces the Risk for Endometrial Cancer

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for endometrial cancer. The results of three retrospective observational studies⁸³⁻⁸⁵ support the conclusion that the consumption of increased amounts of vitamin C reduces the risk for endometrial cancer. In a case-control study nested within the Western New York Diet Study,

the multivariate-adjusted odds of developing endometrial cancer were reduced significantly in women who consumed amounts of vitamin C greater than the median (OR, daily vitamin C intake > 172 mg vs < 129 mg; 0.6; 95% C.I.: 0.4, 0.9; adjusted for age, education, BMI, diabetes, hypertension, pack-years of cigarette smoking, age at menarche, parity, use of oral contraceptives, menopausal status, postmenopausal use of estrogen and daily energy intake).⁸³ Similarly, the results of a case-control study conducted in Shanghai, China, indicated that the multivariate-adjusted odds of developing endometrial cancer were reduced significantly among women with greater daily vitamin C intakes (OR, daily vitamin C intake > 42 mg/1000 kcal vs < 30 mg/1000 kcal: 0.6; 95% C.I.: 0.4, 0.9; adjusted for age, education, menopausal status, diagnosis of diabetes, alcohol consumption, BMI, level of physical activity and dietary intakes of animal products, fruits and vegetables and energy).⁸⁴ In another case-control study, conducted in the Swiss Canton of Vaud and in Northern Italy, the energy-adjusted odds of developing endometrial carcinoma were reduced significantly by increased intake of vitamin C (OR, 5th quintile of daily vitamin c intake vs 1st quintile: 0.6; $p < 0.05$).⁸⁵

In contrast, the data obtained from the 10-year prospective Canadian National Breast Screening Study of 56,837 women indicated that the risk for endometrial cancer was not associated with differences in daily intakes of vitamin C.⁸⁶ Similarly, in a case-control study conducted in the state of Hawaii, the multivariate-adjusted odds of developing endometrial cancer were not affected by differences in the intake of vitamin C from foods (adjusted for parity, use of oral contraceptives, use of unopposed estrogen, history of diabetes and BMI).⁸⁷

The scientific evidence indicates that increased consumption of vitamin C re-

duces the risk for endometrial cancer. The evidence documented by three retrospective observational studies⁸³⁻⁸⁵ supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for endometrial cancer.

Vitamin C Reduces the Risk for Adenocarcinoma of the Esophagus

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for adenocarcinoma of the esophagus. The results of two retrospective observational studies^{88,89} support the conclusion that the consumption of increased amounts of vitamin C reduces the risk for adenocarcinoma of the esophagus. In a case-control study conducted in the US, compared to men and women with daily vitamin C intakes less than the 25th percentile, men and women with daily vitamin C intakes greater than the 75th percentile exhibited significantly reduced odds of developing esophageal adenocarcinoma (OR: 0.45; 95% C.I.: 0.33, 0.61; adjusted for sex, state of residence, age, race, income bracket, education, BMI, cigarette smoking, alcoholic beverage consumption and total daily energy intake).⁸⁸ In a similar case-control study conducted in Germany, the multivariate-adjusted odds of developing adenocarcinoma of the esophagus were reduced significantly in men who consumed more than 100 mg of vitamin C daily (RR, daily vitamin C intake > 100 mg vs < 100 mg: 0.33; 95% C.I.: 0.11, 0.92; adjusted for unspecified "known risk factors").⁸⁹

On the other hand, in a case-control study conducted in New York state, the odds of developing adenocarcinoma of the esophagus were not affected by differences in vitamin C intakes.⁹⁰ In another case-control study conducted in northeast China, the multivariate-adjusted odds of developing any esophageal cancer were not affected by differences in daily vitamin C intakes (adjusted for alcohol consump-

tion, smoking status, income and occupation).⁹¹ In a case-control study of the impact of vitamin C deficiency on adenocarcinoma of the esophagus conducted in Sweden, the multivariate-adjusted odds of developing squamous cell carcinoma of the esophagus were not affected by differences in vitamin C intakes in a vitamin C deficient population (adjusted for age, sex, BMI and smoking status).⁹²

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for adenocarcinoma of the esophagus. The evidence documented by two retrospective observational studies^{88,89} supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for adenocarcinoma of the esophagus.

Vitamin C Reduces the Risk for Squamous Cell Carcinoma of the Esophagus

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for squamous cell carcinoma of the esophagus. The results of several retrospective observational studies^{88,89,93-96} support the conclusion that the consumption of increased amounts of vitamin C reduces the risk for squamous cell carcinoma of the esophagus.

Among men participating in a case-control study conducted in the US, white men who consumed the most vitamin C from vegetables or who consumed dietary supplements containing vitamin C cut their risk of developing squamous cell carcinoma of the esophagus in half ($p < .05$; adjusted for age, residence, smoking and alcohol consumption).⁹³ Similarly, in the same study, black men who consumed the most vitamin C from fruit also cut their risk of developing squamous cell carcinoma of the esophagus in half ($p < .05$; adjusted for age, residence, smoking and alcohol consumption).⁹³ In another case-control study conducted in the US, compared to men and women with daily vitamin

C intakes less than the 25th percentile, men and women with daily vitamin C intakes greater than the 75th percentile exhibited significantly reduced odds of developing squamous cell carcinoma of the esophagus (OR: 0.53; 95% C.I.: 0.36, 0.79; adjusted for sex, state of residence, age, race, income bracket, education, BMI, cigarette smoking, alcoholic beverage consumption and total daily energy intake).⁸⁸ In a case-control study conducted in Uruguay, the multivariate-adjusted odds of developing squamous cell carcinoma of the esophagus also were reduced significantly by increased intakes of vitamin C (OR, 2nd quartile of vitamin C intake vs 1st quartile: 0.59; 95% C.I.: 0.37, 0.92; adjusted for age, sex, residence, urban or rural status, birthplace, education, BMI, smoking status, years since quit smoking, number of cigarettes smoked per day by current smokers, alcohol consumption, mate tea consumption and total daily energy intake).⁹⁴ In a case-control study conducted in France, the multivariate-adjusted odds of developing squamous cell cancer of the esophagus were reduced significantly by intakes of vitamin C greater than the RDA (OR, daily vitamin C intake > 90 mg vs < 60: 0.44; 95% C.I.: 0.24, 0.81; adjusted for interviewer age, smoking status and daily consumption of beer aniseed aperitives, hot Cakvados, whisky, total alcohol and total energy).⁹⁵ In a case-control study conducted in Germany, the multivariate-adjusted odds of developing squamous cell carcinoma of the esophagus were reduced significantly in men who consumed more than 100 mg of vitamin C daily (RR, squamous cell carcinoma, daily vitamin C intake > 100 mg vs < 100 mg: 0.31; 95% C.I.: 0.11, 0.88; adjusted for unspecified "known risk factors").⁸⁹ In another case-control study conducted in Uruguay, the multivariate-adjusted odds of developing any esophageal cancer were reduced significantly by daily vitamin C intakes greater than the lowest

quartile of intake (OR: 0.36; 95% C.I.: 0.19, 0.67; adjusted for age, gender, residence, urban or rural status, education, BMI, smoking status, alcohol consumption, total energy intake and daily intakes of alpha-carotene, beta-carotene, lutein, lycopene, beta-cryptoxanthin, vitamin E, glutathione, quercetin, kaempferol, total flavonoids, beta-sitosterol, campesterol and stigmasterol).⁹⁶

On the other hand, in one case-control study conducted in northeast China, the multivariate-adjusted odds of developing any esophageal cancer were not affected by differences in daily vitamin C intakes (adjusted for alcohol consumption, smoking status, income and occupation).⁹¹ In another case-control study of the impact of vitamin C deficiency on squamous cell carcinoma of the esophagus conducted in Sweden, the multivariate-adjusted odds of developing squamous cell carcinoma of the esophagus were not affected by differences in vitamin C intakes in a vitamin C deficient population (adjusted for age, sex, BMI and smoking status).⁹²

The scientific evidence indicates that increased consumption of vitamin C reduces the risk for squamous cell carcinoma of the esophagus. The evidence documented by six retrospective observational studies^{88,89,93-96} supports this conclusion and there is no evidence that increased consumption of vitamin C may increase the risk for squamous cell carcinoma of the esophagus. In addition, the evidence documented by a retrospective observational study⁹² demonstrates that squamous cell carcinoma of the esophagus is not prevented by vitamin C deficiency.

Part 2 of 4 will follow in the next issue of the Journal of Orthomolecular Medicine.

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Food Allergy: A Case Study

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Introduction

Food allergies have become very common, and the trend is up.¹ Most medical practitioners find that we have to face this problem more and more on a daily basis. A recent public survey in the UK has shown that almost half the population report that they have an “allergy” to some food or foods.² However, the official figures for a “true allergy to food” are around 1% of the population in most developed countries.¹ The reason for this confusion is that majority of food reactions/allergies/intolerances do not produce a typical allergy test profile (raised IgE or IgG with positive prick test and/or positive RAST test). There have been different attempts to classify this group as type B food allergy, metabolic food intolerance or simply food intolerance, rather than a “true” allergy.³ In this group a person may react to many different foods or combinations of foods. Quite often the person is not sure what food produces the reaction, because the reaction may be immediate or delayed (a day, a few days or even a week later). As these delayed reactions overlap with each other, the patients can never be sure what exactly they are reacting to on any given day.^{1,3} Additionally, there is a masking phenomenon, when reactions to a regularly consumed food run into each other (the new reaction begins when the previous has not finished yet), so the connection with that food and symptoms, it triggers, is not apparent.⁴ Food allergy or intolerance can produce any symptom under the sun: from migraines, fatigue, PMS, painful joints, itchy skin to depression, hyperactivity, hallucinations, obsessions and other psychiatric and neurological manifestations. However, the most immediate and common symptoms in the

vast majority of patients are digestive problems: pain, diarrhoea or constipation, urgency, bloating, or indigestion.^{3,5,6}

Naturally, many people try to identify which foods they react to. As a result, many forms of testing have appeared on the market, from blood tests to electronic skin tests. Many experienced practitioners get disillusioned with most of these tests, as they produce too many false-positives and false-negatives.⁶ They lead to a simple conclusion, that if you remove the “positive” foods from the diet, it will solve the problem. In some cases, indeed, elimination of a trigger food helps. However, in the majority the help is not permanent: the patients find that as they eliminate some foods, they start reacting to other foods to which they did not seem to react before. The whole process leads to a situation where the person ends up with virtually nothing left to eat, and every new test finds reactions to new foods. The majority of experienced practitioners come to the same conclusion: the simplistic idea of “just don’t eat foods, you are allergic to” does not address the root of the problem.^{3,6} We need to look deeper at what causes these food intolerances. In order to understand it, I will present a case history of one of my patients.

Case Study

Stephanie S, 35 years old, asked for my help in “sorting out her food allergies”. A very pale, malnourished looking lady, (weight 45 kg, height 160 cm) with low energy levels, chronic cystitis, abdominal pains, bloating and chronic constipation. She was consistently diagnosed anaemic all her life.

Family background: she was born naturally from a mother with digestive problems and migraines, her sister suffered from severe eczema and her brother

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from GI problems. She did not have information on her father's health.

She was not breast fed as a baby and at the age of 3 months got her first urinary infection with the first course of antibiotics. Since then the urinary infections became a regular part of her life, usually treated by antibiotics; now she is suffering from chronic interstitial cystitis. Through childhood she was very thin, always found it difficult to gain any weight, but otherwise she considered her health to be "OK" - she completed school and played sports. At 14 years of age her menstruations stopped, having started a year before. She was put on a contraceptive pill, which seemed to regulate her menstruations. Around 16 she was put on a long course of antibiotics for acne, after which developed lactose intolerance, severe constipation and bloating. She was advised to stop dairy at 18, which helped with constipation for a while, but other symptoms remained. She developed progressively low levels of energy, abdominal cramps, dizzy spells, very low body weight and very dry skin.

Following numerous medical consultations and food allergy testing she started removing different foods from her diet, but was never sure if it made much difference: some symptoms seemed to improve, others did not and new symptoms appeared. She became sensitive to loud sounds and local pollution, her shampoo and make up and some domestic cleaning chemicals. Her cystitis became chronic and was pronounced psychosomatic by her doctor. Her diet at the time of the consultation was very limited: she seemed to tolerate (but was not entirely sure) breakfast cereals, sheep's yoghurt, soy milk, some varieties of cheese, a few vegetables and rarely fish. Following several food allergy tests she has removed all meats, eggs, nuts, all fruit, whole grains and many vegetables.

This example is very common and demonstrates clearly that just remov-

ing "offending" foods from the diet does not solve the problem. We have to look deeper and find the course of the patient's malady. In order to do that we have to examine Stephanie's health history.

Infancy

Stephanie was born from a mother with digestive problems and was not breast fed. What does that tell us? We know that unborn babies have sterile gut.⁷ At the time of birth the baby swallows mouthfuls of microbes, which live in the mother's birth canal.⁸ These microbes take about 20 days to establish themselves in the baby's virgin digestive system and become the baby's gut flora.^{7,8} Where does the vaginal flora come from? The medical science shows that the flora in the vagina largely comes from the gut. What lives in the woman's bowel will live in her vagina.^{9,10} Stephanie's mother suffered from digestive problems, which indicates that she had abnormal gut flora, which she passed to her daughter at birth.

Breast milk, particularly colostrum in the first days after birth, is vital for appropriate population of the baby's digestive system with healthy microbial flora.^{9,11} We know that bottle-fed babies develop completely different gut flora to the breast fed babies.¹¹ That flora later on predisposes bottle-fed babies to asthma, eczema, different other allergies and other health problems.¹² The most important abnormalities develop in the digestive system, as that is where these microbes make their home. Having acquired abnormal gut flora from her mother at birth, Stephanie was compromised further by bottle feeding.

Chronic Cystitis

Apart from the gut, in the first few weeks of life other mucous membranes and baby's skin get populated by their own flora, playing a crucial role in protecting those surfaces from pathogens and toxins.¹³ As baby Stephanie acquired

abnormal flora in her gut, her groin and vagina got abnormal flora too (as it normally comes from the gut).¹⁰ At the same time the urethra and the urinary bladder would get similar to vagina flora: in a normal situation it should be predominated by Lactobacteria, largely *L. Crispatus* and *L. jensenii*.¹⁴ This flora produces hydrogen peroxide, reducing the pH in the area, which does not allow pathogens to adhere.¹⁵

Unprotected urethra and bladder fall prey to any pathogenic microbes, causing urinary tract infections. The most common pathogens, which cause UTIs, are *E.coli*, *Pseudomonas aeruginosa* and *Staphylococcus saprophyticus*, coming from the bowel and the groin.¹⁵ Urine is one of the means of toxin elimination from the body.¹⁶ In gut dysbiosis large amounts of various toxins are produced by pathogens in the gut and absorb into the bloodstream through the damaged gut wall.^{16,17} Many of these toxins leave the body in urine: accumulating in the bladder, this toxic urine comes into contact with the bladder lining.

The beneficial bacteria in the bladder and urethra maintain a GAG layer of the bladder: a protective mucous barrier, largely made from sulphated glucosaminoglycans, produced by the cells of the bladder lining.¹⁷ As the GAG layer gets damaged, toxic substances in urine get through to the bladder wall causing inflammation and leading to chronic cystitis.¹⁸ That is what happened to Stephanie: at the age of 3 months she got her first urinary infection.

As her gut flora, vaginal flora and the flora of urethra and the bladder were not corrected, she suffered from urinary infections all her life and eventually developed chronic cystitis.

Further Damage to Gut Flora

Because of regular urinary tract infections Stephanie had to have regular

courses of antibiotics through her entire life, starting from infancy. Every course of antibiotics damages beneficial species of bacteria in the gut, leaving it open to invasion by pathogens, resistant to antibiotics.^{10,19} Even when the course of antibiotics is short and the dose is low, it takes different beneficial bacteria in the gut a long time to recover: physiological *E. Coli* takes 1-2 weeks, Bifidobacteria and Veillonelli take 2-3 weeks, Lactobacilli, Bacteroids, Peptostreptococci take a month.^{10,20} If in this period the gut flora is subjected to another damaging factor(s), then gut dysbiosis may well start in earnest.²¹ After many short courses of antibiotics Stephanie took a long course for acne at the age of 16. That is when she got pronounced digestive problems: constipation, bloating, abdominal pain and lactose intolerance, indicating that her gut flora got seriously compromised.

From the age of 14, Stephanie has been taking contraceptive pills for many years. Contraceptives have a serious damaging effect on the composition of gut flora, leading to allergy and other problems, related to gut dysbiosis.^{22,23}

Malnutrition-The Consequence of Abnormal Gut Flora

Stephanie suffered from malnutrition all her life despite the fact that her family always cooked fresh wholesome meals and Stephanie ate well. She was always pale, very thin and small and could not gain weight. This is not surprising taking into consideration the state of her gut right from birth. The microbial layer on the absorptive surface of the GI tract not only protects it from invaders and toxins, but maintains its integrity.^{20,21} The epithelial cells called enterocytes, which coat the villi, are the very cells which complete the digestive process and absorb the nutrients from food.²⁴ These cells only live a few days as the cell turnover in the gut wall is very active. These enterocytes are constantly

born in the depth of the crypts. Then they slowly travel to the top of the villi, doing their job of digestion and absorption and getting more and more mature on the way. As they reach the top of the villi, they are shed. This way the epithelium of intestines gets constantly renewed to insure its good ability to do its work well.²⁴

Animal experiments with sterilization of the gut found that when the beneficial bacteria living on the intestinal epithelium are removed, the process of cell renewal gets completely out of order.¹⁰ The time of cell travel from crypts to the top of the villi becomes a few times longer, which upsets the maturation process of absorptive cells and often turns them cancerous. The mitotic activity in the crypts gets significantly suppressed, which means that much fewer cells will be born there and fewer of them will be born healthy and able to do their job properly. The state of the cells themselves becomes abnormal.^{9,25} That is what happens in a laboratory animal with sterilized gut. In a human body the absence of good bacteria always comes with pathogenic bacteria getting out of control, which makes the whole situation much worse. Without the care of beneficial bacteria while under attack from pathogenic flora, the gut epithelium degenerates and becomes unable to digest and absorb food properly, leading to malabsorption, nutritional deficiencies and food intolerances.^{19,21,25}

Apart from keeping the gut wall in good shape, the healthy gut flora populating this wall has been designed to take an active part in the very process of digestion and absorption.^{19,21} So much so, that the normal digestion and absorption of food is probably impossible without well-balanced gut flora. It has an ability to digest proteins, ferment carbohydrates, break down lipids and fibre. By-products of bacterial activity in the gut are very important in transporting minerals, vitamins, water, gases and many other nutrients through

the gut wall into the bloodstream.¹⁰ If the gut flora is damaged, the best foods and supplements in the world may not have a good chance of being broken down and absorbed. A good example is dietary fibre, which is one of the natural habitats for beneficial bacteria in the gut.²⁵ They feed on it, producing a whole host of good nutrition for the gut wall and the whole body. Bacteria absorb toxins, and take part in water and electrolytes metabolism to recycle bile acids and cholesterol. It is the bacterial action on dietary fibre that allows it to fulfil all those good functions in the body.^{20,21} When these good bacteria are damaged and are not able to “work” the fibre, dietary fibre itself can become dangerous for the digestive system, providing a good habitat for the harmful pathogenic bacteria and aggravating the inflammation in the gut wall. This is when gastroenterologists have to recommend a low-fibre diet.¹⁹ Consequently, dietary fibre alone without the beneficial bacteria present in the gut can be detrimental.

Stephanie also became lactose intolerant after the long course of antibiotics prescribed for her acne. Indeed lactose is one of those substances, which most of us would not be able to digest without well functioning gut flora.²⁵ The explanation offered by science so far is that after early childhood the majority of us lack the enzyme, lactase, to digest lactose.²⁶ If we are not meant to digest lactose, then why do some people seem to manage it perfectly well? The answer is that these people have the right bacteria in their gut. One of the major Lactose digesting bacteria in the human gut is *E.coli*.¹⁰ It comes as a surprise to many people that physiological strains of *E.coli* are essential inhabitants of a healthy digestive tract. They appear in the gut of a healthy baby in the first days after birth in huge numbers: 10^7 - 10^9 CFU/g and stay in these same numbers throughout life, providing that they do not get destroyed by antibiotics and other

environmental influences.^{9,19}

Apart from digesting lactose, physiological strains of *E. Coli* produce vitamin K and vitamins B₁, B₂, B₆, B₁₂, produce antibiotic-like substances called colicins, and control other members of their own family which can cause disease. In fact having your gut populated by the physiological strains of *E. Coli* is the best way to protect yourself from pathogenic species of *E. Coli*.²¹

Unfortunately, this group of beneficial bacteria are very vulnerable to broad spectrum antibiotics, particularly aminoglycosides (Gentamycin, Kanamycin) and macrolides (Erythromycin).^{9,10} Apart from *E.coli*, other beneficial bacteria in the healthy gut flora (Bifidobacteria, Lactobacteria, beneficial yeasts) will not only ensure appropriate absorption of nutrients from food but also actively synthesise various nutrients: vitamin K, pantothenic acid, folic acid, thiamin (vitamin B₁), riboflavin (vitamin B₂), niacin (vitamin B₃), pyridoxine (vitamin B₆), cyanocobalamin (vitamin B₁₂), various amino acids and other active substances.^{9,10,25} In the process of evolution Nature made sure that when the food supply is sparse, we humans don't die from vitamin and amino acids deficiencies. Nature provided us with our own factory for making these substances—our healthy gut flora. When this gut flora is damaged despite adequate nutrition we develop vitamin deficiencies. Every tested child or adult with gut dysbiosis shows deficiencies in those very vitamins, which their gut flora is supposed to produce.²⁵ Restoring the beneficial bacteria in their gut is the best way to deal with those deficiencies, particularly vitamin B deficiencies.^{10,19,21}

On testing over the years Stephanie consistently showed deficiencies in most B vitamins, fat soluble vitamins, magnesium, zinc, selenium, manganese, sulphur, iron and some fatty acids.

Anemia—Another Consequence of Gut Dysbiosis

Stephanie suffered from anemia all her life, unsuccessfully treated by courses of iron tablets. The majority of patients with gut dysbiosis look pale and pasty and their blood tests often show changes typical for anemia.²¹ It is not surprising. They not only cannot absorb essential vitamins and minerals for blood from food, but their own production of these vitamins is damaged. People with damaged gut flora often have a particular group of pathogenic, iron-loving bacteria growing in their gut (*Actinomyces spp.*, *Mycobacterium spp.*, pathogenic strains of *E.coli*, *Corynebacterium spp.* and many others).^{13,25} They consume dietary iron, leaving the person deficient. Unfortunately, supplementing iron makes these bacteria proliferate, bringing unpleasant digestive problems and does not remedy anemia. To have healthy blood the body needs other minerals and a host of vitamins: B₁, B₂, B₃, B₆, B₁₂, C, A, D, folic acid, pantothenic acid and some amino acids.^{10,24} It has been shown in a large number of studies all over the world, that just supplementing iron does not do much for anaemia.²⁷

The Pathogens in the Gut

The most studied pathogens, which overgrow after numerous antibiotic courses, are clostridia and yeasts, which normally belong to the opportunistic group of gut microbes.²⁸ The opportunistic gut flora is a large group of various microbes, the number and combinations of which can be quite individual. There are around 400 different species of them found in the human gut.²⁵ These are the most common: Bacteroids, Peptococci, Staphylococci, Streptococci, Bacilli, Clostridia, Yeasts, Enterobacteria (Proteus, Klebsiella, Citrobacteria), Fuzobacteria, Eubacteria, Spirochaetaceae, Spirillaceae, Catenobacteria, different viruses and many others.¹³ Interestingly, many of

these opportunistic bacteria when in small numbers and under control actually fulfil some beneficial functions in the gut, like taking part in the digestion of food, breaking down lipids and bile acids. In a healthy gut their numbers are limited and tightly controlled by the beneficial flora.²⁰ But when this beneficial flora is weakened and damaged, they get out of control. Each of these microbes is capable of causing various health problems.²⁹ The best known is the fungus *Candida albicans*, which causes untold misery to millions of people.³¹ There is an abundance of literature published about *Candida* infection. However, I have to say that a lot of what is described as *Candida* Syndrome is in effect a result of gut dysbiosis, which would include activity of lots of other opportunistic and pathogenic microbes. *Candida albicans* is never alone in the human body. Its activity and ability to survive and cause disease depends on the state of trillions of its neighbours—different bacteria, viruses, protozoa, other yeasts and many other micro-creatures.^{9,19,31}

In a healthy body *Candida* and many other disease-causing microbes are very well controlled by the beneficial flora. Unfortunately, the era of antibiotics gave *Candida* a special opportunity. The usual broad-spectrum antibiotics kill a lot of different microbes in the body—the bad and the good but they have no effect on *Candida*. After every course of antibiotics, *Candida* is left without anything to control it, so it grows and thrives.^{30,31} Stephanie had many symptoms of *Candida* overgrowth in her body: low energy level, dry skin, recurrent vaginal thrush and cystitis, bloating, constipation, foggy brain and lethargy. The *Clostridia* family was given a special opportunity by the era of antibiotics too, because *Clostridia* are also resistant to them.³⁴ There are about 100 members of this family and they all can cause serious disease. Many of them are found as opportunists in a healthy

human gut flora.^{25,33} As long as they are controlled by the beneficial microbes in the gut, they normally do us no harm. Unfortunately, every course of broad-spectrum antibiotics removes the good bacteria, which leaves *Clostridia* uncontrolled and allows it to grow. Different species of *Clostridia* cause severe inflammation of the digestive system and damage its integrity, leading to many digestive problems and food intolerances.^{32,33}

Food “Allergies” and Intolerances

Normal gut flora maintains gut wall integrity through protecting it, feeding it and insuring normal cell turnover. When the beneficial bacteria in the gut are greatly reduced, the gut wall degenerates.^{9,10,21,25} At the same time various opportunists, when not controlled by damaged good bacteria, get access to the gut wall and damage its integrity, making it porous and “leaky.”^{6,28,29} For example, microbiologists have observed how common opportunistic gut bacteria from families Spirochaetaceae and Spirillaceae, due to their spiral shape, have an ability to push apart intestinal cells braking down the integrity of the intestinal wall and allowing through substances which normally should not get through.^{13,25} *Candida albicans* has this ability as well. Its cells attach themselves to the gut lining, literally putting “roots” through it and making it “leaky.”³¹ Many worms and parasites have that ability as well.^{9,10,35} Partially digested foods gets through the damaged “leaky” gut wall into the blood stream, where the immune system recognizes them as foreign and reacts to them.^{36,37,38} This is how food allergies or intolerances develop. There is nothing wrong with the food. What is happening is that foods do not get a chance to be digested properly before they are absorbed through the damaged gut wall. In order to eliminate food allergies, it is not the foods we need to concentrate on, but the gut wall. In my clinical experience, when the gut wall is healed many food intolerances disappear.

Healing the Gut Wall – the Diet

How do we heal the gut wall? We need to replace the pathogens in the gut with the beneficial bacteria, so effective probiotics are an essential part of the treatment. However, the most important intervention is the appropriate diet. There is no need to re-invent a wheel when it comes to designing a diet for digestive disorders. There is a very effective diet with an excellent 60 years record of helping people with all sorts of digestive disorders, including such devastating ones as Crohn's disease and ulcerative colitis. This diet is called Specific Carbohydrate Diet or SCD for short. SCD was developed by a renowned American pediatrician Dr. Sidney Valentine Haas in the first half of the 20th century,³⁹ when doctors used to treat their patients with diet and natural means. Carrying on with the work of his colleagues Drs. L. Emmett Holt, Christian Herter and John Howland, Dr. Haas spent many years researching the effects of diet on celiac disease and other digestive disorders. He and his colleagues found that patients with digestive disorders could tolerate dietary proteins and fats fairly well but complex carbohydrates from grains and starchy vegetables made the problem worse. Sucrose, lactose and other double sugars also had to be excluded from the diet. However, certain fruit and vegetables were not only well tolerated by his patients, but improved their physical status. Dr. Haas treated over 600 patients with excellent results: after following his dietary regimen for at least a year there was "complete recovery with no relapses, no deaths, no crisis, no pulmonary involvement and no stunting of growth."

The results of this research were published in a comprehensive medical textbook *The Management of Celiac Disease*, written by Dr. Sidney V. Haas and Merrill P. Haas in 1951. The diet, described in the book, was accepted by medical community all over the world

as a cure for celiac disease and Haas was honoured for his pioneer work in the field of pediatrics. Unfortunately, "happy end" does not occur too often in human history. In those days celiac disease was not very clearly defined. A great number of various conditions of the gut were included in the diagnosis of celiac disease and all those conditions were treatable by the SCD very effectively. In decades that followed, Celiac disease was eventually defined as a gluten intolerance or gluten enteropathy, which excluded a great number of various other gut problems from this diagnosis. As the "gluten free diet" was pronounced to be effective for celiac disease, the SCD diet was forgotten as outdated information. All those other gut diseases, which fell outside of the realm of true celiac disease, were forgotten as well.

The true celiac disease is rare, so the "forgotten" gut conditions would constitute a very large group of patients, which used to be diagnosed as celiac and which do not respond to treatment with gluten free diet. Incidentally, a lot of "true" celiac patients do not get better on the gluten free diet either. All these conditions respond very well to SCD diet, developed by Dr. Haas.³⁹

Following the whole controversy about celiac disease, the Specific Carbohydrate Diet would have been completely forgotten if it wasn't for a parent, Elaine Gottschall, desperate to help her little daughter, who suffered from severe ulcerative colitis and neurological problems. She went to see Dr. Haas in 1958. After 2 years on SCD her daughter was completely free of symptoms, an energetic and thriving little girl. Following the success of the SCD with her daughter Elaine Gottschall over the years has helped thousands of people, suffering from Crohn's disease, ulcerative colitis, celiac disease, diverticulitis and various types of chronic diarrhoea. She has reported very dramatic and fast recoveries in young children,

who apart from digestive problems had serious behavioural abnormalities, such as autism, hyperactivity and night terrors. She has devoted years of research into biochemical and biological basis of the diet and has published a book, called *Breaking the Vicious Cycle. Intestinal Health Through Diet*.³⁹ This book has become a true saviour for thousands of children and adults across the world and has been reprinted many times. Many websites and web-groups have been set up to share SCD recipes and experiences. I have been using SCD for many years in my clinic and have to say that it is the diet for food allergies. As I work largely with children with learning disabilities, such as autism, ADHD, dyslexia, dyspraxia, I have grouped these patients under the name Gut And Psychology Syndrome or GAPS.⁴⁰ I had to adopt some aspects of SCD for these patients and they have named their diet—the GAPS diet. Over the years I have developed a GAPS Introduction Diet for the more severe end of the spectrum (www.gapsdiet.com). I find that the Introduction Diet is particularly effective in food allergies, as it allows the gut wall heal more quickly. The Introduction Diet is structured in stages. Unless there is a dangerous (anaphylactic type) allergy to a particular food, I recommend my patients to ignore the results of their food intolerance testing and follow the stages one by one. The Introduction Diet in its first stages serves the gut lining in three ways:

1. It removes fibre. With damaged gut wall fibre irritates the gut lining and provides food for the pathogenic microbes in the gut. This means: no nuts, no beans, no fruit and no raw vegetables. Only well-cooked vegetables (soups and stews) are allowed with particularly fibrous parts of the vegetable removed. No starch is allowed on the GAPS diet, which means no grains and no starchy vegetables.

2. It provides nourishment for the gut

lining: amino acids, minerals, gelatin, glucosamines, collagens, fat soluble vitamins. These substances come from homemade meat and fish stocks, gelatinous parts of meats well-cooked in water, organ meats, egg yolks and plenty of natural animal fats on meats.

3. It provides probiotic bacteria in the form of fermented foods. The patients are taught to ferment their own yoghurt, kefir, vegetables and other foods at home. These foods are introduced gradually in order to avoid a “die-off reaction.”

On the first two stages of the Introduction Diet most severe digestive symptoms, such as diarrhoea and abdominal pain disappear quite quickly. At that point the patient can move through the next stages, when other foods are gradually introduced. As the gut wall starts healing, the patients find that they can gradually introduce foods, which they could not tolerate before. When the Introduction GAPS Diet is completed, the patient moves to the Full GAPS Diet. I recommend adhering to the Full Diet for 2 years on average in order to restore normal gut flora and GI function. Depending on the severity of the condition, people take different time to recover. Children usually recover quicker than adults. Stephanie had to follow the Introduction Diet for 7 months before she started gaining weight and feeling stronger. By the time she moved to the Full GAPS Diet she had normal stools, no bloating and no cystitis symptoms; her energy levels were much improved, though she still looked slightly pale. In about a year from the start of the treatment she disappeared for 18 months, then emailed me with an update: she was doing well, her energy level was good, she had no symptoms of cystitis and her GI function was good. She gained weight though she was still quite slim, but within the normal range. In the last two months she started eating some foods not allowed on the diet and found that she can tolerate

them on an occasional basis, including pasta, chocolate and some goods from the local bakery.

Probiotics

In order to heal the gut wall apart from the appropriate diet we need to replace the pathogenic microbes in the gut with the beneficial ones. The fermented foods in the diet will provide some probiotic microbes. However, an effective probiotic supplement is essential in most cases. There is a plethora of studies accumulated about benefits of probiotic supplementation for most digestive disorders, as well as many other health problems.⁴¹⁻⁴⁷ The market is full of probiotics in the form of drinks, foods, powders, capsules and tablets. Majority of them are prophylactic, which means that they are designed for the fairly healthy people, they are not designed to make a real difference in a person with a digestive disorder and a “leaky gut.” These people need a therapeutic strength probiotic with well-chosen powerful species of probiotic bacteria. A therapeutic probiotic will produce a so-called “die-off reaction”: the probiotic bacteria kill the pathogens in the gut, when these pathogens die, they release toxins. As these are the toxins which give the patient his or her unique symptoms, their release makes these symptoms worse, which is called the “die-off reaction”. This reaction can be quite serious and must be controlled. That is why I recommend to start the therapeutic probiotic from a very small dose, then build the dose very gradually up to the therapeutic level. Once on that level, the patient needs to stay on it for a few months: how long depends on the severity of the condition. Once the symptoms of the disease are largely gone, the patient can start gradually reducing the daily dose to the maintenance level or can stop altogether. Stephanie took a particular therapeutic probiotic. She took one capsule per day (2 billion live cells) for

a week, then increased to 2 capsules per day. On this dose her skin became itchy, she got loose stool and her cystitis symptoms got slightly worse. She understood it to be a “die-off”, so stayed on this dose for as long as it took for these symptoms to subside in 2.5 weeks. Then she increased her dose to 3 capsules a day. This increase produced another “die-off reaction,” so she had to stay on the 3 capsules per day for a month before she could move on. In this manner she gradually got up to 8 capsules a day—her therapeutic dose. I recommended her to stay on this dose for 6 months. In this period of time all her main symptoms subsided and some started going. After 6 months, she decided to stay on the therapeutic dose for longer, as she felt well on it. After another 4 months on 8 capsules per day, she felt strong enough to start reducing the dose. She gradually reduced it to 4 capsules a day—her maintenance dose. After about 2 years on this dose she found that she could discontinue the probiotic and only take it occasionally, when she was under particular stress.

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The Scientific Assessment of Health Claims— When Only The Best Will Do

Introduction

Food and/or food constituents play a crucial role in health promotion and reduction of risk of major diseases as established through scientific evidence. A powerful form of conveying information on the potential health benefits of foods and food constituents is through making health claims in accompanying communications. Such claims could enhance the knowledge of nutrition and health among consumers¹⁻² and improve public health. In response to increased consumer interest in foods and food constituents with potential health benefits, regulatory bodies in several countries have developed guidelines for assessing health claims on foods and food constituents.

The Nutrition and Health Claims Regulation on Foods in the EU— A Brief Overview

In 2007, a regulation on nutrition and health claims made on foods was introduced in the European Union³ (hereafter referred to as the HCR). The HCR defines a “health claim” as “any claim that states, suggests or implies that a relationship exists between a food category a food or one of its constituents and health. The HCR allows two types of health claims to be made on foods/food constituents:

Article 13 of the HCR covers Health Claims other than those referring to reduction of disease risk or children’s health. These are health claims that refer to:

- (a) the role of a nutrient in growth, development and the functions of the body
- (b) psychological and behavioral functions
- (c) slimming or weight control.

Included under Article 13(5) are claims based on newly developed scientific data or which include a request for the protection of data.

Article 14 of the HCR covers Health Claims that refer to reduction of disease risk or children’s health. The HCR defines reduction of disease risk claim as “any health claim that states, suggests or implies that the consumption of a food category, a food or one of its constituents significantly reduces a risk factor in the development of a human disease.” All claims have to comply with the general principles that they are not false, ambiguous or misleading (as laid down in article 3), and they have to be scientifically substantiated (article 6).

The Recent Assessment of Health Claims in Europe – How Scientific is it?

The HCR also describes a process for the approval of the two types of health claims described above. Article 13 health claims (except those covered by Article 13(5)) will have to be based on “generally accepted scientific evidence” and have to be submitted to the EC for approval based on a list of relevant scientific references. Health claims covered by Article 13(5) and Article 14 will require dossiers of scientific evidence for these claims to be submitted to the EC for approval. The EC has forwarded the Article 13 lists as well as the Article 13(5) and Article 14 dossiers to the European Food Safety Authority (EFSA) for its scientific opinion.

In the Terms of Reference (ToR) for evaluating Article 13 health claims, provided by the European Commission to EFSA on 24 July, 2008, the Commission explicitly requests that EFSA shall evaluate the *extent to which the claimed*

effect of the food in the identified function is beneficial. Also, in assessing scientific evidence based on generally accepted science by taking the totality of scientific data into account and weighing the evidence, EFSA is invited to *comment on the nature and quality of the totality of the (scientific) evidence provided according to consistent criteria.*

However, several shortcomings have been observed in the scientific evaluations and opinions on health claims released by EFSA to date. Importantly, EFSA has: (1) failed to provide a grading of the 'strength of evidence' when assessing the relationship between food/food constituents and health; (2) omitted providing a clear definition of what it considers "generally accepted science;" (3) omitted clearly defining the standards it will apply in assessing the evidence from individual scientific studies (eg. what standards are applied in assessing biomarkers and surrogate end-points used in individual studies); (4) provided excessive emphasis on evidence from human intervention randomized-controlled trials (RCTs) in assessing relationship between food/food constituents and health and risk of disease. In doing so, EFSA has unfortunately failed to achieve the highest possible standards of scientific review.

Scientific Assessment of Health Claims – Fine-Tuning the Process

It is critical that the large body of established and emerging scientific evidence of the role of diet and certain specific food constituents in promoting health and reducing risk of chronic diseases is accurately and effectively relayed to the consumers to enable improvement of public health.

One important aspect of evaluating the scientific evidence substantiating health claims is providing a clear rating of its strength. It is with this goal in

mind that the World Health Organization (WHO) developed a grading system to evaluate the strength of the scientific evidence for the relationship between a food/food constituent and health.⁴ Evidence is classified into four grades based on its totality, as well as on the quality and consistency of individual studies. Importance is also given to regular review and updating of the classification based on emerging science. A similar method is also applied by the World Cancer Research Fund (WCRF)⁵ and advocated in the PASSCLAIM report.⁶ Application of such established methods by EFSA in the scientific evaluation of health claims would increase transparency of the process by clearly showing what individual studies were evaluated to provide the ranking as well as the rigor of the evaluation. It would also enhance consistency since such a grading system would allow other trained scientists to come to similar conclusions using the same database, while a regular review of the grading would give room for emerging science.

In assessing the quality of individual scientific studies supporting health claims, the type of study that has been conducted (ie. whether it is an observational study or a randomized controlled trial or an animal study) is highly relevant. Both the WHO and WCRF in their scientific evidence grading system describe evidence as being "convincing" when it is based on several high-quality studies of more than one type with consistency and biological plausibility. Numerous scientific publications have criticized excessive reliance on evidence from randomized controlled trials (RCTs) alone, and have suggested a well-rounded approach using evidence from both human observational-epidemiological studies and interventional studies, as well as supportive evidence from mechanistic studies to draw con-

clusions on the association between food/food constituents and health.⁷⁻¹⁰ In its report titled Evolution of Evidence for Selected Nutrient Disease Relationship,¹¹ the Institute of Medicine observes that RCTs appear to be less successful in investigating benefits of single nutrients in reducing risk of chronic diseases since chronic diseases develop over a long period of time and may be affected by various other factors at different times during that period.

Another essential aspect in assessing scientific evidence substantiating health claims is setting and defining a clear standard the degree of scientific agreement. In the United States, the Food and Drug Administration requires that scientific evidence substantiating health claims has to be based on 'significant scientific agreement', which the FDA defines as "an authoritative statement from a scientific body of the United States Government or the National Academy of Sciences."¹² Setting such clear standards also becomes important when evaluating the quality of individual scientific studies. For example, there are several biomarkers backed by scientific studies that can be used as surrogate end-points for risk of a specific disease. Additionally, biomarkers of specific food constituent intake are often based on food recall records and food composition tables. In the absence of clearly-defined and validated standards in either of the above cases, the evaluation of the quality of a study becomes vague and questionable. Clarification of the term "generally accepted science" by EFSA, as well as specification of the standards or benchmarks against which quality of individual studies will be evaluated would lead to consistency in the quality of the studies, and hence avoid any ambiguity in their evaluation and maintain uniformity in the health claim assessment process.

The relationship between diet and health has been strongly established by science. EFSA has been given the challenging task of validating the science and thereby determining the crucial message (claim) that will be relayed to the millions of consumers in Europe with impact on their health, safety and well-being. It is therefore upto EFSA to ensure that it applies no less than the highest possible scientific standards in every step of the process.

– Geetha Achanta, M.S., Ph.D.

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What if this was how you cut your cholesterol?

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ORTHOMOLECULAR
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Correspondence

Vitamins on Trial: Bad Science— Misleading Conclusions

People are confused about vitamins: Should we take them, or are they a waste of money? Conventional dietary wisdom says we can get all the vitamins we need from a good diet. But Canadians are nonetheless enthusiastic vitamin poppers. A 2005 survey by Health Canada showed that 57 percent are regular consumers of vitamin supplements, and believe they increase everyday well being and ward off the threat of future serious illness.¹

Skeptics argue that such faith is misplaced; that research shows vitamin supplements simply don't work. Vitamin poppers are wasting their money, dupes of the supplement industry. And this is where the argument gets emotional. Defenders of vitamins parade the many published studies that do seem to support a role for vitamin supplements in chronic disease prevention, while the skeptics gleefully jump all over any new study that appears to show otherwise.

Recently, it's the skeptics who have been winning. First we learned 14,641 male U.S. doctors given 400 IU of vitamin E every other day (or placebo), or 500 mg of vitamin C daily (or placebo), were not protected from cancer or heart disease, and might even have increased their risk of hemorrhagic stroke. Then a study of antioxidants C, E and selenium for the prevention of prostate cancer was cut short after a midway analysis of the data failed to show any protection.

Breaking Basic Laws of Physiology

While there are many criticisms that could be levied at these studies, one stands out above all others – you simply cannot get reliable data by studying vitamins one at a time. The reason for this should be obvious. No vitamin works alone. Rather, each works in concert with all the other vitamins and essential nu-

trients – the trace minerals, amino acids and essential fats. Roughly 40 essential nutrients are required to maintain and repair tissues, and regulate the innumerable body processes required for health. And deficiency of any one of these will cause illness.

Therefore, studying the health effects of vitamins one at a time, or even in small combinations, breaks this fundamental law of physiology. When we investigate the impact of vitamin C and E supplements on heart disease or cancer in isolation we have no way of capturing the influence of other possible deficiencies that might be present in our study population. For example, deficiencies of vitamin D, omega 3 fats or B-vitamins are also known risk factors for heart disease and cancer, and all too common in North America.²⁻⁴

The Problem with Studying Complex Systems

Suppose the auto industry makes the observation that cars must have spark plugs in good working order to run well. Although this might seem obvious to any car owner, if we follow the current logic of vitamin research we would not blindly accept a claim like this, but rigorously test it. So we put new spark plugs in a series of malfunctioning cars hoping to make them run again. Might work in some, not in others.

But if a significant number of cars failed to respond, we wouldn't be entitled to conclude that spark plugs are useless. If we did, we might expect to draw a scathing response from the auto industry, and leave knowledgeable car owners shaking their heads in disbelief. But this is what we've been doing with vitamin research. We test vitamins in isolation, and when we can't get them to work on their own, we discard them.

Such singularly focused "spark plugs research" tells us nothing, one way or the

other, about the role of vitamin supplements in health and disease. The scientific approach that reduces everything to studying one variable at a time may work for drugs or surgical interventions. But it is generally doomed to fail when we try to study complex systems, whether it is cars or human health.

Thoughtful researchers are at last beginning to see the limitation of single nutrient clinical trials and have called for a moratorium on research until we can figure out how to study nutrition in all its complexity.⁵ In the meantime, we need to acknowledge the inherent difficulties of testing intimately interactive nutrients one at a time. And we should rightly remain skeptical about the results of clinical trials that persist in this approach.

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The Doctor Who Lived: Holistic Psychiatrist Defeats the Maryland Board

Alice W. Lee-Bloem, M.D., a holistic psychiatrist practicing in Olney, Maryland, has successfully defeated the Maryland Board of Physicians and protected her legal right to continue practicing orthomolecular psychiatry and energy medicine.

After a raging, two-year battle in the Maryland courts and at the administrative level, Dr. Lee-Bloem delivered a crushing legal defeat to the Board in three ways. First, the Administrative Law Judge (ALJ), Geraldine A. Klauber, of the Maryland Office of Administrative Hearings, dismissed most of the charges by the Maryland Board against Dr. Lee-Bloem, stating that as a matter of law, the Board of Physicians could not prosecute the practice of alternative medicine and energy medicine through the peer review process. To keep the prosecution alive, the Board grasped at straws and charged Dr. Lee-Bloem with violating the "standard of care" of one patient only. Second, after a three-day trial, the ALJ wrote a 50-page decision, stating that the Board had no legal grounds to prosecute Dr. Lee-Bloem in the first place, having failed to define what the "standard of care" was, let alone convince her of any violations of the same. And third, as of February 5, 2009, the Board issued its final decision to dismiss all charges against Dr. Lee-Bloem without any conditions or probation. This complete dismissal of a case by the Maryland Board has set a new precedent and is the first decision of its kind in the history of the State of Maryland for a holistic physician.

Mr. Jacques Simon, the lead attorney in this case, brilliantly executed the legal defense and assault against the Board on behalf of Dr. Lee-Bloem through the proceedings in the state courts and at the administrative level. With national legal expertise in protecting integrative medi-

cine and physicians who practice cutting edge medicine, he defeated the Maryland Board in its efforts to quash alternative medicine, which efforts were marred by legal and constitutional deficiencies. Mr. Alan Dumoff, an attorney practicing in Maryland, added many years of additional experience, acumen, and skill in defending alternative medicine as he supported Mr. Simon and Dr. Lee-Bloem on this case as the local counsel.

-<http://www.drbloem.com/hp/victory.htm#readmore>

Book Reviews

Micronutrients: Metabolic Tuning-Prevention-Therapy

by Uwe Grober

MedPharm Scientific Publishers
Stuttgart, Germany, 2009, 478 pages.

*What are little boys made of?
Snips and snails, and puppy-dogs' tails,
That's what little boys are made of.
What are little girls made of?
Sugar and spice, and everything nice,
That's what little girls are made of.*

Actually, we are made of oxygen, nitrogen and other minerals from the atmosphere and oceans. These essential elements are combined in a large variety of molecules which are found naturally in the living body. They include the amino acids and proteins, the fats, the sugars and a whole slew of other essential compounds that have been termed "orthomolecular" by Linus Pauling. But these compounds that nature created for the living bodies are only a fraction of all the possible compounds that can be made. Those not made by nature are called xenobiotics; the body can tolerate some of them, which have only mild to moderate poisonous effects, but cannot live with most of them because they are so toxic. So this little children's poem is correct, since everything listed is a natural ingredient. Over millions of years, life has learned to use those compounds that are essential—such chromium, iron, and zinc—and eliminated toxic substances that are harmful such as mercury and lead.

Therefore, in order to grow, mature, stave off disease and maintain health, we need to provide our bodies with those essential nutrients which are used as substrates to construct other substances. One compound, nicotinamide adenine dinucleotide (NAD) is involved in at least 200 different reactions in the body and also is used as a substrate for three dif-

ferent pathways. Life has specialized from simple one cell organisms—which can make almost everything they need, provided they are given water and oxygen and an environment in which to grow—to very complex organisms (like us) who have lost the capacity to make many amino acids and all the vitamins. In losing the need to synthesize everything, so much energy was released that movement and, eventually, modern humankind could evolve. Plants are so busy making what they need they have little energy left to move about and develop brains.

What I have written above is not exotic science and is well recognized by anyone who has studied what life is. We probably have identified all of the essential nutrients but we have not yet made sure that everyone ingests these elements that are available. All the modern nutritional studies report that modern diets do not contain enough of these essential nutrients. The only group who appears not to know this is the pharmaceutical industry. If it does know this it, the industry certainly acts as if it does not, and it promotes the view that their dangerous and toxic xenobiotic products are all one needs to prevent and cure disease. So, it is very useful and helpful to have essential information contained in *Micronutrients: Metabolic Tuning-Prevention-Therapy* by Uwe Grober. Forewords were written by Bruce N. Ames, by Gerhard Uhlenbruck, and myself.

In my opinion, this is a very good book and I endorse it as a very valuable addition to all orthomolecular libraries. It covers all vitamins, both water and fat soluble, some accessory nutrients, minerals including trace elements, fatty acids and amino acids. The second half of this book discusses both prevention and treatment for a large number of diseases. We have the information. Why do we not have companies whose main concern is the health and welfare of the public rather than the size of their annual distribution of

money to their shareholders? There would be an enormous saving of money since none would have to be spent looking for the pot of gold at the end of the rainbow and more could be spent really looking at the health of the people. It has been said that big pharma does not want to do basic research as it might interfere with their own patents. We need governments which are not fearful of the patent system. We need companies that will work with the information available in this book.

–Abram Hoffer, M.D., Ph.D.

**Practicing Medicine Without a License?
The Story of the Linus Pauling
Therapy for Heart Disease**

by Owen Fonorow with Sally Snyder Jewell
Lulu.com, 2008. Paperback, 230 pages.

In 1973, I first learned of Linus Pauling's interest in ascorbate. Mostly due to his well-publicized advocacy, I, like so many others, started taking vitamin C: a whopping 500 mg per day. Now I take 15,000 mg or more daily, and vastly more if needed. My relatives think I am nuts. I don't care. I very rarely need to see a doctor; it was 12 years since my last visit. My recent physical confirmed an especially low risk for cardiovascular disease. To me, this is good evidence that Dr. Pauling was right.

Personal experiences are sometimes more compelling when not our own. Owen Fonorow's book, *Practicing Medicine Without a License*, is full of how-I-beat-cardiovascular-disease case stories from people of all ages, in all walks of life. All of them used the "Pauling Therapy." This is generally taken to mean an absolute minimum of 6 g of vitamin C and 2 to 6 g L-lysine per day. These are usually accompanied with other supplements such as proline, omega-3 oils, magnesium, CoQ

10, and vitamins E, D and B-complex. Those so doing consistently reported dramatic improvement in cardiovascular problems. While so-called "evidence based medicine" winces at anecdotes, one cannot read this collection without being impressed. If the testimonial writers are all just making it up, they must be the best actors in the world.

Mr. Fonorow claims that in his 12 years of communicating with the public, he has "never encountered heart disease in any person who takes more than 10,000 mg of vitamin C daily." The author is in a position to make this statement because he has had considerable experience interacting with people reporting their successful use of vitamin C. His Vitamin C Foundation website has long been, and remains, an excellent internet resource. Among other benefits, it offers the public the complete text of Irwin Stone's classic vitamin C book, *The Healing Factor* for free downloading at <http://www.vitamin-cfoundation.org/stone/>.

Practicing Medicine Without a License is much more than an assemblage of anecdotes. It discusses the failings of the cholesterol-causes-CVD theory; indeed, the book actually opens with Mr. Fonorow's ready admission that he eats bacon and eggs for breakfast. The book neatly traces a good deal of the history of vitamin therapy for heart disease. It also, predictably, provides background on Pauling's own struggles to educate the medical profession, conventional nutritionists, and the media. Pauling quotes and specific dosage tips abound. Literature citations are provided, but a page and a half is not enough. Given the book's unequivocal stance, it would not be amiss to greatly expand the reference section. That may be in the offing: Fonorow writes that this is actually volume one in a planned Pauling series, volume two to be entitled *The Great Suppression*. Or it may not, as the author also writes: "Published clinical

studies run by medicine to test the Pauling Therapy: There have been none.”

A very good reading list is provided, although the “Resources” section may be leaning a bit to the proprietary side. And, there is a small error on p 91. The author cites a 1994 Pauling interview as being printed in JOM (the Journal of Orthomolecular Medicine), but it in fact was published by ION (Institute for Optimum Nutrition).

In some ways, to some readers, *Practicing Medicine Without a License* will prove to be an irritating book, especially to orthodox physicians and dieticians. The book is, above all, a personal statement by the author. It is frequently confrontational, and shamelessly assertive on every page. It has attitude. I like that, and furthermore, I recommend it.

—Reviewed by Andrew W. Saul

Vitamin C: The Real Story. The Remarkable and Controversial Story of Vitamin C
by Steve Hickey, PhD, and Andrew Saul, PhD
Basic Health Publications Inc., CA
2008, 192 pages.

A curious title. Thousands of children take Flintstone multis every day; don't they get enough vitamin C? Many adults take some C when they have a cold and, even without supplements, don't most people eat enough vitamins and minerals in their fruits and veggies? What could be remarkable or controversial about vitamin C? Authors Hickey and Saul think we need to know the truth about vitamin C. Their fascinating book presents some truly remarkable discoveries, introduces us to vitamin C's multiple health-maintaining functions and outlines its health-restoring capabilities, while warning us about vitamin C factoids.

Steve Hickey, PhD and Andrew Saul, PhD present the facts clearly and carefully.

Readers will gradually realize that the vitamin C story has two dimensions. On the bright side, for decades, scientific and medical researchers have documented vitamin research, clinical progress and success. Books and medical journals explain that vital amines, as nutritional substances, are essential for health and healing. Over the past 100 years, a succession of scientific researchers studied the biochemistry of vitamin C and learned that vital amines help to maintain normal metabolism. They determined that minimal doses of vitamin C can heal scurvy and sustain life. During decades of follow-up research, scientists discovered that optimum doses of vitamin C have remarkable health-restoring capabilities. Researchers conducted clinical trials, detailed patient recoveries, corroborated findings and wrote journal articles and reference books. However, the vitamin C story also has a disturbing, dark side. Even though decades of research found vitamins safe and effective, millions of patients suffer and deteriorate while professional skeptics devalue the care provided by orthomolecular doctors (who complement standard treatments with therapeutic doses of vitamins). Rather than telling us the facts, certain health professionals dismiss the vitamin C research, ignore the progress reports, minimize vitamin C's health-maintaining functions and disparage health-restoring claims linked to vitamin C. These skeptics use factoids to support their denials, also outlined in this book.

Skeptics cannot rewrite medical history or hide the truth about vitamins. In the early 1900s, biochemists, physicians and researchers discovered that certain nutrients are essential for life. Test rats did not grow or develop unless their diets included vital amines (as vitamins were first described). Medical scientists determined that tiny quantities of vitamins are also necessary for human health. They

linked four diseases to vitamin deficiencies: beriberi to B₁, pellagra to B₃, scurvy to C and rickets to D. The history of medicine records the involvement of Christiaan Eijkman, Gerrit Grijs, Sir Frederick Hopkins and Casimir Funk. Dr. Eijkman and Dr. Hopkins received Nobel prizes for discovering that vitamins are essential for human health. Researchers then searched for the chemical identities of the essential nutrients. Dr. Szent-Gyorgi received a Nobel prize for discovering that vitamin C was ascorbic acid.

After discovering vitamins, clinical researchers wondered if essential nutrients might have clinical applications. If so, they needed clinical trials to determine the optimum doses. Scientific and medical professionals mapped the biochemical pathways and determined which metabolic processes required vitamins as co-factors. They quickly realized that a few milligrams of essential nutrients can sustain health but it took decades to discover that therapeutic doses of vitamins can restore health. Centuries ago mankind faced an epidemic of scurvy. Most people know that thousands of British sailors died during long voyages. In 1795, Dr. James Lind did the first clinical trial and discovered how to heal scurvy. It took many decades before sea captains finally added citrus fruits to ships' stores. British sailors who stayed healthy were then called limeys. What if cancer patients run low on vitamin C today; might these patients develop scurvy-like symptoms? Can megadoses of vitamin C help cancer patients? "Of course not," scoffed the skeptics, while orthomolecular doctors researched and discovered that optimum doses of vitamin C can indeed help cancer patients feel better and live longer. Other doctors discovered that therapeutic doses of vitamin C can help patients recover from life-threatening infections such as polio, pneumonia and AIDS, reduce toxic levels of lead and mercury and neutralize

toxins injected by the bites of venomous snakes and spiders.

Like a Swiss-army knife, vitamin C has multiple capabilities. When we pick up a Swiss-army knife for the first time, we expect to find large and small blades but we may not inspect it carefully. In an emergency, we happily discover that a Swiss-army knife comes with a versatile set of built-in tools: a screwdriver, a tooth pick, a cork screw and a file. After these tiny tools save lives, the word steadily gets out until the public knows that each Swiss army knife comes with life-saving tools. Consider the metabolic capabilities of vitamins as tools for restoring health. In milligram doses, vitamin C enables essential metabolic pathways to sustain life and heal scurvy. If taken in large enough doses when a patient has cancer, an infection or an overload of toxins, vitamin C can heal and restore health. The general public still does not know that vitamin C has lifesaving capabilities but the real story keeps coming out. Meanwhile, certain experts, who should know better than to publish false information, scoff at vitamin C research, forget its biochemistry, ignore its metabolic functions and refuse to prescribe it. Why don't scientific and medical experts study the vitamin C research, review the clinical trials, interview recovered patients and learn that therapeutic doses of vitamin C have proved safe and effective enough to restore health and save lives? How can trusting patients know if our doctors understand and apply the healing capabilities of vitamin C or rely on false factoids to withhold restorative care-by-vitamins? Patients and families, caregivers and health professionals have to read the real story to learn the facts for ourselves.

Vitamin C: The Real Story reminds us that a hundred years after the discovery of vitamin C, mankind is still researching vitamin biochemistry and developing medical applications. We understand that vital

amines, trace minerals, amino and fatty acids, hormones and many other nutrients are essential for sustaining life. We are still learning that optimum doses of vitamins can restore health. Orthomolecular health professionals know that vitamin C and other nutritional supplements, if given in the right doses, can help patients recover and live well. They routinely prescribe supplements and adjust the doses to suit each patient's diagnosis and biochemical individuality. Readers of this book will learn to distinguish the facts about vitamin C from factoids. Patients can ask their doctors about vitamin research, optimal doses and patient recoveries. Readers are cautioned to take care with their health. Anyone can read this book to learn the basic facts about vitamin C and then study its clinical applications: therapeutic doses of vitamin C can restore health when taken as recommended by qualified medical professionals who understand its biochemistry and know when to prescribe vitamin C as a complementary and restorative treatment.

–Review by Robert Sealey, BSc*

A Short Vitamin C Reading List

The Cancer Breakthrough: A Nutritional Approach for Doctors and Patients by Dr. S. Hickey & Dr. H. Roberts, 2007.

Healing Cancer: Complementary Vitamin & Drug Treatments by Abram Hoffer, PhD, MD, with Linus Pauling, PhD, 2004, CCNM Press.

Orthomolecular Medicine for Everyone Megavitamin Therapeutics for Families and Physicians by Abram Hoffer, MD, PhD and Andrew Saul, PhD, 2008, Basic Health.

Vitamin C, Infectious Diseases & Toxins: Curing the Incurable, by Thomas Levy, MD, JD, 2002, Xlibris Corp.

*Author of Finding Care for Depression, Mental Episodes & Brain Disorders 90-Day Plan for Finding Quality Care
www.searpubl.ca

The Journal of Orthomolecular Medicine

Since 1970, this quarterly Journal for health professionals has published the best of nutritional research and clinical trials. New articles describing the orthomolecular approach to health management and treatment of disease are accompanied by lively editorials, book reviews, letters and reports. The Journal of Orthomolecular Medicine has led the way for a quarter century in presenting, far in advance of other medical journals, new health concerns and treatments including: Candidiasis; Mercury Amalgam Toxicity; Niacin Therapy for Schizophrenia and Coronary Disease; Chronic Fatigue Syndrome; Vitamin C and Cancer; Allergies and Behavioral Disorders; Drug and Alcohol Abuse; Tissue Mineral Analysis; and Orthomolecular Treatment for AIDS and Cardiovascular Disease.

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For journal references cited:

1. Pauling L, Itano HA, Singer SJ, et al: Sickle cell anemia: a molecular disease. *Science*, 1949; 110: 543-548.

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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