

PROSPERITY

KINDNESS

IMAGINE A DAY WHEN FOOD
IS ONCE AGAIN HEALTHY, NATURAL AND REAL...

HEALTH
URGENCY

LOVE
DIGNITY

ACTION
NATURE

COMMON GOOD

NUTRITION

WORLD



NATURAL
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NUTRITION AND HEALTH

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Nutrition is nothing less than the foundation of medicine, the cornerstone of all medical therapeutics.

— A Forum on Nutrition and Health, JON 2(4), 1993

Prevention cannot start too early. Neither can it start too late.

— Rima E. Laibow, MD, 2005

1. Introduction

Nutrition brings together the goals and objectives of public health policy planners, medical/health systems and consumers. Global health policy experts know that health, itself, is a great bargain since people who are robustly well:

- Create and fully participate in functioning communities stabilizing society;
- Work and fully contribute to local and world economies;
- Consume far fewer scarce resources for preventable diseases since they require less conventional, industrial-economy-based health resources than do hungry and ill-nourished people;
- Enjoy life and treasure the stability that supports their health.

Robust individual and family health objectives thus become goals that are shared between people and society.

The core of robust health is a level of nutritional abundance which supports and meets the global right and the unique personal nutritional requirements of each individual to optimal nutritional status and health. This is, however, a challenging goal to achieve in a world in which the degradation of the quality of the food supply is an increasingly severe problem in both the developing and the developed world.

A 2003 WHO/FAO joint expert consultation report, *Diet, Nutrition and the Prevention of Chronic Diseases*¹ states that chronic diseases are preventable and that the developing world is facing the medical consequences of a nutritionally compromised food supply.. It goes on to identify the consequences of under-nutrition on the development of what it refers to as a “non communicable epidemic,” noting that increasingly sedentary lifestyles, sharp decreases in the nutritional adequacy of food and globally produced, commercially prepared food are in stark contradistinction to local and national food supplies and trading economies supportive of access to fresh and local food and sustainable economic and agricultural practices.

The non-communicable epidemic to which the WHO/FAO refers is in fact a global pandemic, one marked by a growth in the prevalence of widespread chronic illness which is impacting heavily the developing nations of the world. The pandemic is, by contrast, already well established in the developed world and is now worsening in both economic sectors of the world.

This WHO/FAO report identifies obesity, type 2 diabetes mellitus, cardiovascular disease, hypertension and stroke and some types of cancer as increasingly significant causes of premature death and disability in both developing and developed countries. These diseases place additional burdens on already overtaxed medical systems.²

“Nutrition is coming to the fore as a major, modifiable determinant of chronic disease with scientific evidence increasingly supporting the view that alterations in diet have strong effects, both positive and negative, on health throughout life,” adds the

¹ http://www.fao.org/documents/show_cdr.asp?url_file=/DOCREP/005/AC911E/AC911E00.HTM),

² http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf, p12

WHO/FAO report. “Most importantly, dietary adjustments may not only influence present health, but may determine whether or not an individual will develop such diseases In many developing countries, food policies remain focused only on under nutrition and are not addressing the problem of chronic disease”³ and overall long-term health status, including immune capacity to resist infectious diseases, parasitic infestations and other health assaults dependant upon an optimally and healthy underlying nutritional matrix in each individual.

In fact, that nutritional matrix is crucial. To illustrate this point, it is useful to examine the importance, and dangers, of saturated fat. It has become a matter of axiomatic truth that saturated fat is a component of an unhealthy diet and that intake is correlated strongly with elevated LDL-cholesterol and the resulting increase in cardiovascular disease. However, the biochemical and nutritional picture does not support that well-held belief without significant qualification.

In the context of the modern nutrient-depleted diet which most urban and urbanizing people consume saturated fat (SFA) is the strongest dietary predictor of LDL-cholesterol levels in the presence of inadequate antioxidant levels. In the presence of adequate nutritional components (i.e., unmodified saturated fat from healthy plant and animal sources including free range, naturally fed animal protein) SF not a health threat; rather, it is of great benefit. Dietary recommendations and strategies must take into account the nutritional context of the person being nourished, supported and advised. Single findings of depletion or depression of nutrients usually have concomitant significance far beyond the specific nutrient profile or symptom associated with that finding and should be addressed as part of a total nutritional picture which involves the biochemical, dietary and physiological matrix of that individual. Attention should be paid to that person’s relationship with food and supplements as well as the production, preparation, preference and consumption of food for optimal health.,

Weston Price⁴, an American dentist, carried out decades of observation and examination of people living on native and modern diets all over the world over many decades. His conclusion was that when native foods were replaced by the "displacing foods of modern commerce"⁵—sugar, white flour, condensed milk, canned foods, chocolate, jams and pastries—results were not only serious dental abnormalities, but the development of the diseases of modern civilization’s diet, including the epidemic development of diabetes, obesity and cardiovascular disease.⁶

This global exploration of diet and health found that universally, healthy people eat saturated fat in the context of optimal nutritional intake of vitamins and minerals along with co-factors found both in meat and in fats. Optimal nutrition is supported by a complex and complete relationship between intake of fats and nutrients. It is important to

³ Ibid

⁴ Price, W, DDS, Price, Nutrition and Physical Degeneration, 1945, Price-Pottenger Nutrition Foundation, San Diego, CA, (619) 574-7763

⁵ Ibid

⁶ http://www.westonaprice.org/traditional_diets/nasty_brutish_short.html

note that dietary SF, recently vilified in Western science, play many critical roles in human health and biochemistry.

Saturated fatty acids:

- Constitute at least 50% of the cell membranes, giving them necessary stiffness and integrity
- Play a vital role in bone health,^{7, 8}
- Lower Lp(a), a marker which indicates proneness to heart disease^{9, 10}
- Protect the liver from the damage caused by alcohol ingestion;^{11, 12}
- Enhance the immune system^{13, 14}
- Required for the proper utilization of essential fatty acids;^{15, 16}
- Are the preferred food for the heart^{17, 18}
- Have important antimicrobial properties against harmful microorganisms in the digestive tract.^{19, 20}

Perhaps even more important, animal fats are carriers for vital fat-soluble vitamins A and D which are needed for a host of processes, from prevention of birth defects to health of the immune system to proper development and maintenance of bones and teeth. In fact, Price was convinced that these "fat-soluble activators" were key to the beautiful facial development, freedom from dental caries and absence of chronic degenerative diseases that characterized the people he studied.

The diets of traditional groups noted for longevity are rich in animal fats: The people of Hunza consume large quantities of fermented goat milk products. Goat's milk is higher in fat, and contains more SF, than cow's milk; the inhabitants of Vilcabamba in Ecuador

⁷ Watkins, B A, et al, "Importance of Vitamin E in Bone Formation and in Chondrocyte Function" Purdue University, Lafayette, IN, AOCS Proceedings, 1996

⁸ Watkins, B A, and M F Seifert, Food Lipids and Bone Health, "Food Lipids and Health", R E McDonald and D B Min, eds, p 101, Marcel Dekker, Inc. New York, NY

⁹ Khosla, P, and K C Hayes, J Am Coll Nutr, 1996, 15:325-339

¹⁰ Clevidence, B A, et al, Arterioscler Thromb Vasc Biol, 1997, 17:1657-1661

¹¹ Nanji, A A, et al, Gastroenterology, Aug 1995, 109(2):547-54

¹² Cha, Y S, and D S Sachan, J Am Coll Nutr, Aug 1994, 13(4):338-43

¹³ Cohen, L A, et al, J Natl Cancer Inst, 1986, 77:43

¹⁴ Kabara, J, The Pharmacological Effects of Lipids, J Kabara, ed, The American Oil Chemists Society, Champaign, IL, 1978, 1-14

¹⁵ Oliart Ros, R M, et al, Meeting Abstracts, AOCS Proceedings, May 1998, p 7, Chicago, IL

¹⁶ Garg, M L, et al, The FASEB Journal, 1988, 2:4:A852

¹⁷ http://www.westonaprice.org/traditional_diets/nasty_brutish_short.html

¹⁸ L D Lawson and F Kummerow, B-Oxidation of the Coenzyme A Esters of Vaccenic, Elaidic and Petroselaidic Acids by Rat Heart Mitochondria, Lipids, 1979, 14:501-503

¹⁹ Kabara, J, The Pharmacological Effects of Lipids, J Kabara, ed, The American Oil Chemists Society, Champaign, IL, 1978, 1-14

²⁰ Cohen, L A, et al, J Natl Cancer Inst, 1986, 77:43

consume fatty pork and whole milk products; and the long-lived inhabitants of Soviet Georgia also eat liberally of pork, whole milk yoghurt and cheeses. In fact, a Soviet study found that longevity was greatest in rural communities where people ate the most fatty meat, compared to town dwellers who ate more carbohydrates.²¹, ²²

Dr Price found that healthy, disease free people eating their native diets which, without exception, contained, compared to the processed Western diet:

- At least four times the nutritional minerals—calcium, phosphorus, magnesium, iron, etc.,
- Ten times the fat-soluble vitamins. Vitamin A from animal sources is not the same as its precursors, the carotenes, which are found in plant foods. The conversion of carotenes in the human body is often compromised, and even under optimal conditions is not efficient enough to supply the amount of true vitamin A needed by the body.

Price found in the diets of healthy isolated populations.

- The richest sources of vitamins A and D from the very foods modern man eschews: animal fats, organ meats, lard, fish eggs, shellfish, eggs and butter—but not pale, commercial store-bought butter which is virtually devoid of these nutrients.²³, ²⁴

It is vitally important to note that the so-called laboratory “norms” and population definitions of “nutritional adequacy” and “health” upon which our understandings of levels and limits of nutrients are built may not express normal findings but, rather, normative ones for an unhealthy population. Our conventional definitions of nutritional adequacy may be, in reality, standards developed from populations eating a compromised diet (ours) and prone to develop preventable diseases (and, on a statistical basis, already in the process of developing them at the time that any lab value might have been drawn or other data gathered). Thus, the concept of limitation of nutrients to a supposed standard of normality drawn from the most disease-prone population the world has ever known, a population which habitually eats foods depleted in nutrients and whose genetic potential for health is manifestly not supported by that diet is not based in either science or sense, despite its familiarity.

This fully flawed premise leading to nutrient and nutritional status “norms” based on ill people, rather than healthy ones, is neither rational nor logical. And, most important, it does not allow for decisions on either a personal, regulatory or population basis, which support optimal health and the fullest possible expression of the genetic capacity for health and productivity inherent in optimal nutrition. These cautions pertain to levels of both macro and micro nutrients. Dr. Price’s important data is supported by other medical

²¹ http://www.westonaprice.org/traditional_diets/nasty_brutish_short.html

²² Pitshelauri, G Z, *The Long Living of Soviet Georgia*, 1982, Human Sciences Press, New York, NY

²³ Sally Fallon, "Vitamin A Vagary," *PPNF Health Journal*, Price-Pottenger Nutrition Foundation, Summer 1995, 191

²⁴ Evidence, B A, et al, *Arterioscler Thromb, Vasc Biol*, 1997, 17:1657-1661:(2):1-3 (619) 574-7763

and physical anthropologists, physicians, cultural and health workers. (See for example,^{25, 26}).

Like SF, carbohydrate intake must be evaluated in the context of its quality and the food environment of the patient. Dr. Price found that while seed foods (i.e., grains, legumes and nuts) were consumed in native diets among disease free peoples consuming native diets, they were always prepared with great care in traditional societies by sprouting, roasting, soaking, fermenting and sour leavening.^{27, 28} These processes neutralize substances in whole grains and other seed foods that block mineral absorption, inhibit protein digestion and irritate the lining of the digestive tract. Such processes also increase nutrient content and render seed foods more digestible. In these societies, in fact, seed foods are not consumed unless they meet these preparation criteria. In essence, the food value of our carbohydrates and the native diet ones is as different as the saturated fat from the animals they eat and that from our animal food sources.

Thus, it is important to view dietary consumption an impact of carbohydrates and saturated fat on, for example, blood lipids in the context of a pattern of dietary intake because healthy native peoples routinely eat as much saturated fat as they can get but do so in the context of a dietary environment in which food is not degraded in nutrient density and carbohydrates are, in fact, enhanced in nutritional value.

Since the dietary matrix of modern populations is far inferior to that of the societies Dr. Price studied, it is important to correct intake to those levels to help move populations back toward healthy intakes and healthy outcomes.

Hunger and under-nutrition are devastating realities for the vast majority of the world. WHO estimates that 30% of the world's population is malnourished²⁹. If standards of nourishment are modified to rest on the nourishment of truly healthy people, such as those studied by Dr. Price in his extensive research, the result would be to include most of the world's population in the undernourished category. Current definitions of homeostatic values would no longer obtain since these are the homeostatic maladaptations of challenged and compromised individuals who either have, or are in the process of developing, preventable chronic degenerative diseases of under-nutrition.

This expansion of the under-nourished, is medically and scientifically-based although politically problematic, would, in fact, be a positive advance since until malnutrition is recognized, public health policy designed to correct it cannot be created and implemented. Thus, the important distinction between normal (i.e., well nourished with an end point of optimal nutrition) and normative (ill-nourished with an end point of diagnosable dietary deficiency diseases which may not yet clinically apparent) is a crucial one. Using the second definition, what is, and what is not, a dietary deficiency disease is determined by the state of nutritional medicine's knowledge. As diseases are added to

²⁵ <http://www.agron.iastate.edu/courses/agron342/diamondmistake.html>

²⁶ <http://www.beyondveg.com/billings-t/comp-anat/comp-anat-8b.shtml>

²⁷ Steinkraus, Keith H, ed, *Handbook of Indigenous Fermented Foods*, 1983, Marcel Dekker, Inc, New York, NY

²⁸ http://www.westonaprice.org/traditional_diets/nasty_brutish_short.html

²⁹ http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf, p. 18

the category of dietary deficit disorders, more of the world is categorized as malnourished.

With this perspective in mind it becomes clear that the current definitions of biological homeostasis are both incorrect and misleading.

On the other hand, using optimal nutrition as a public health goal leads to the development of an appreciation for, and an awareness of, the importance of supporting real physical and physiological well-being based in the achievable goal of population-wide optimal nourishment.

Although the problem is of massive proportion, unlike many other complex issues it is manageable and relatively inexpensive to solve. In 2001 preventable chronic disease accounted for more than 60% of all global deaths (nearly 34 million deaths) and approximately 46% of the global disease burden.³⁰ All estimates suggest a sharp increase in the problem since that time.

In fact, nutritional treatment and prevention has a major contribution to make in the developing world because of the unique susceptibility of poorly fed and culturally disrupted populations. These populations are increasingly open to chronic preventable diseases and highly susceptible to infectious diseases because of long-term absence of optimal nutrition which predisposes them to enhanced vulnerability.

Although HIV/AIDS, malaria, tuberculosis and other infectious diseases are predominant cause of death in Sub Saharan Africa (and will continue to be for some time), of those deaths attributable to chronic disease, 79% occur in the developing countries.³¹

WHO projections predict that chronic degenerative diseases, although misnamed “diseases of affluence” are, in fact, burdens of the developing world as well so that by 2020 preventable chronic diseases will account for nearly 75% of world deaths and that, of those, non-optimally nourished people of the developing world will account for

- 71% of all ischemic heart disease deaths
- 70% of all diabetic deaths
- 75% of all stroke-related deaths.

Diabetes will increase among the non-optimally nourished in the developing world from its 1995 rate of 84 million cases to 228 million in 2025.³²

The WHO and FAO notes that “countries that have actively intervened in the diet and nutritional behavior of their populations ... have seen decreases in their risk factors and falling rates of chronic disease.”³³ Adoption of a national policy of optimal nutrition could have a tremendous impact on the economic status of a country while enhancing the well-being of her people in an unprecedented manner.

³⁰ Ibid

³¹ http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf, p. 14

³² http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf, p. 15

³³ <http://www.scielo.org/pdf/rpsp/v13n5/a15v13n5.pdf>

The US provides a sad but illustrative example of a country spending enormous sums on everything except the powerful combination of preventive nutrition and natural medicine focused on optimal health. Her efforts are toward slamming the high tech barn door after the nutritional horse is well away. The declining health of Americans testifies to the fact that this is an alluring, but incorrect strategy toward wide spread health facilitation. Estimates of cost reduction per year looking *only at hospital costs* if preventive nutrition were instituted in the US are illustrative of the vast amount of money spent on preventable diseases which could be better spent on increasing health and well-being by sparing the distress and loss of productivity and life which disease brings. Savings which could be expected if adequate nutritional prevention impacted the US population are listed below:

Disease	Reduction in Hospitalization Cost/year
Cardiovascular Disease	\$ 22 Billion US
Cancer	\$ 1 Billion US
Low Birth Weight	\$500 Billion US
Neural Tube Birth Defects	\$ 70 Billion US
Cataract	\$ 2 Billion US ³⁴

Although the percentage of the US Gross National Product which the United States spends on health care is enormous (15%, twice that of any other nation)³⁵ and decidedly unproductive in terms of the health of her people compared to other countries with a more natural approach to health care (unnecessary medical procedures and drugs cause nearly 800,000 preventable deaths and hundreds of thousands of severe drug reactions per year³⁶ in the US)³⁷, the point here pertains to any economy. Preventing disease through economical and remarkably safe, non-toxic nutritional strategies offers substantial savings over allopathic medicine and substantial increase in health and well being. As drug side effects are eliminated and toxic loads on the body are reduced to the virtual null point obtaining and maintaining optimal health becomes easier.

In fact, the side effects and toxicity of pharmaceuticals are without parallel in the nutrition world. No class of medicaments in the pharmacological realm offers the safety of nutrients. The difference is orders of magnitude apart in favor of nutrients.

Nutritional status and immune competence are the cornerstones upon which the house of health rests. Assertions are frequently made that a good diet will supply all needed nutrients. If this unproven assertion were true, those people eating such a diet should not develop the preventable diseases of under-nutrition and should not respond to the introduction of nutrients with resolution of those diseases. In fact, clinical nutrition confirms what biochemistry and immunology explain: optimal nutrition is difficult or impossible to achieve through diet alone given

³⁴ McDonald, A, Role of Nutrition in Prevention of Disease, PowerPoint

³⁵ <http://www.burtonreport.com/InfHealthCare/ManagedHlthCare.htm>

³⁶ http://www.lef.org/magazine/mag2004/mar2004_aws_i_death_02.htm

³⁷ <http://www.huppi.com/kangaroo/L-healthcare.htm>

- Widespread demineralization of agricultural soils leading to, and proceeding from, the use of synthetic fertilizers, pesticides and herbicides.
- The degradation of food quality and nutrient density with increased time and processing between harvest and consumption
- The increased need for nutrients imposed by a toxic load far in excess of that experienced by our agrarian ancestors.³⁸

When the nutritional support available to the individual falls below the combined requirements of metabolic and environmental challenges, the result is either immediate or delayed sub-optimal health resulting in

- Increased susceptibility to infection
- Failure to reach neurologic, intellectual and physical genetic potential
- Lowered vitality including chronic fatigue and diminished productivity
- Decreased level of well-being
- Development of chronic cell injury which can lead to chronic degenerative diseases
- Craving and addictions
- Reduced fertility
- Increased rates of mental and emotional, attentional and neurological disorders
- Increased violence.

Given the difference between the quality of food available to us and the food quality that we need to flourish, supplements make sense and add health promoting options to large number of people. It makes sense then, that dietary supplements of micronutrients are beneficial in the prevention and/or treatment of disease as part of the maintenance of optimal health and in helping to end world hunger.³⁹

Dr. Arline McDonald of The Feinberg School of Medicine of Northwestern University's points out "Nutrients are the raw materials that support physiologic and metabolic functions needed for maintenance of normal cellular activity. Malfunctioning of cellular activities due to an inadequate level of support from available nutrients is initially expressed in biochemical changes that will eventually develop into clinical symptoms characteristic of the particular roles of the nutrients involved. Nutrient deficiencies may develop as a result of inadequate intake, impaired absorption, increased demand, or increased excretion. Excessive intakes of some nutrients may promote deficiencies of others through impaired absorption, increased demand, or increased excretion.

Chronic disease can be considered the result of cellular change resulting from nutrient insufficiencies since they are, in fact, an expression of cumulative cellular damage due to environmental assaults for which the threshold of exposure at which damage is incurred is defined by genetics. An imbalance in dietary patterns is among the environmental factors that contribute to the development of chronic diseases. Diet may either be

³⁸ http://www.price-pottenger.org/Articles/kidney_liver_answers.htm

³⁹ Leibovitz, B, *Nutrition at the Cross Roads*, JON 2(4), 1993

directly involved in the pathogenesis of the disease or it may exacerbate pathological changes due to other environmental factors.”⁴⁰

High Dose Supplementation (HSD) with available and inexpensive nutrients like folic acid, the B Complex, (especially vitamins B3, 6 and 12) magnesium, essential fatty acids, methionine, reduced glutathione, alpha ketoglutarate, dietary fiber, and vitamin C offer the body the opportunity to repair nutritional and toxic cellular damage and support the pathways of detoxification overwhelmed by an imbalance of the required nutritional load (low) and the toxic load (high). Nutrients have both nourishing (metabolic) and supportive/corrective functions in the body. Physiological doses of nutrients are required for metabolic support while HSD is required for correction of tissue, organ, cellular and sub-cellular pathology and for detoxification support.

Metabolic functions include supporting growth and maintenance, immune surveillance, maintaining homeostasis and maintaining adequate reserves for efficient function. Intake imbalances at the metabolic level result in the classic nutrient deficiency symptoms and diseases.

Supportive/Corrective nutrient functions are evoked when HSD offers the body a larger amount of the nutrient(s) than commonly found in food. The resultant easily available nutrients drive reaction toward repair and re-equilibration within the physiological capacity of the organism, often resulting in the cellular and sub-cellular restoration of balance and health. Nutrients have a remarkably low toxicity profile so that very large doses may be offered with great safety. Thus, HSD nutrition stimulates biochemical and tissue restoration of health or maintains those processes when such restoration is not possible.

Supplementation of metabolic levels of nutrients supports

- Production of storage and release of energy
- Maintenance of lean body mass and skeletal mass
- Tissue synthesis
 - Requires
 - Protein
 - Fat
 - Zinc
 - Vitamin A
 - Vitamin C
 - Iron
- Membrane potentials (brain, heart, etc.)
- Neuromuscular activity
- Plasma and cellular fluid volumes
- Synthesis of Bioactive Compounds (enzymes, hormones, immune substances)
 - Requires
 - Amino acids
 - Vitamin B6, fatty acids
 - Selenium
- Regulatory functions (enzyme activation, cell messengers, gene induction)

⁴⁰ McDonald, A, *Relationship of Nutrition to Prevention of Diseases*,
<http://www.feinberg.northwestern.edu/nutrition/tools-resources/sbm.html>

HSD of nutrients supports

- Detoxification (biotransformation and conjugation)
- Immune function (mitogenic, microbicidal and phagocytic activity)
 - Requires
 - Zinc
 - Vitamin C
 - Proteins
 - Vitamin A
 - Vitamin B6
 - Folate
- Inflammatory response modulation by cytokines, prostaglandins
- Antioxidant activity (free radical scavengers)
 - Includes
 - Vitamin C
 - Carotenoids
 - Vitamin E
 - Selenium
- Enzyme induction and inhibition
- Homeostatic and feedback control
- Gene expression

Offering nutrients at either the metabolic or HSD dosage levels should:

- Optimize cellular activity and tissue/organ function
 - provide sufficient supplies to meet continuous demand
 - maintain adequate reserves for intermittent increased demand
- Stimulate inherent reparative capacity by providing sufficient nutrient support to allow robust cellular health
- Reduce the metabolic burden imposed on cardiac, pulmonary, neurological, renal, hepatic, and musculoskeletal systems by environmental factors
 - minimize workload of organ systems
 - support reparative or compensatory responses required to maintain normal function
- Support cellular defenses that protect tissue integrity
 - maintain immunocompetence
 - promote detoxification of chemicals
 - prevent oxidative damage

Inadequate nutrient intake can be detected by clinical symptoms if the deficiencies are severe or prolonged. Use of clinically-defined signs of deficiency to determine whether nutrient intakes are adequate has significant limitations that underscore the need for dietary assessment as a critical component of clinical evaluation. One is that many nutrients, particularly trace elements and some vitamins do not have well-defined signs or symptoms that are targeted to a specific nutrient. Consequently, metabolic abnormalities may develop that could cause cellular injury or compromise defenses. In the early stages of iron deficiency, for example, cellular energy production is compromised even though hemoglobin levels may still be in a clinically normal range. Another example of the same process is in folic acid deficiency. Megaloblastic cellular changes due to folic acid

deficiency occur in the late stages of progression of the deficiency long after metabolic abnormalities were established.

Nutrient status is seriously compromised in the face of environmental or dietary imbalances. The resulting compensatory mechanisms stress the organs, the glands and the nutrient base creating a cycle of deepening deficiency which is ultimately expressed as signs or symptoms of chronic disease, often after serious or irreversible tissue damage has occurred. "If these responses are sustained by prolonged imbalances in intakes of these nutrients, then adaptive changes to counter these responses may contribute to cellular injury and pathology. Compensatory responses and/or adaptive changes are among the risk factors identified for chronic diseases that can be managed by modifications in dietary habits."⁴¹

The urgency of not ignoring early stages of nutrient deficiencies because of the absence of clearly defined clinical deficiency symptoms is illustrated by vitamin B₁₂. Development of a Vitamin B₁₂ deficiency that is unrelated to lack of intrinsic factor (pernicious anemia) is not uncommon among adults over age 65, but the nonspecific pattern of symptoms associated with a deficiency of this vitamin (e.g., depression, dementia, lassitude, memory loss, unsteady gait, numbness and tingling in the extremities, etc.) are difficult to separate from other possible causes in this age group. Consequently, abnormalities in vitamin B₁₂-dependent cellular activities may go uncorrected resulting in irreversible damage to nervous tissue (e.g., demyelination).⁴²

The functional capacity of a cell depends on its genetics, nourishment, exposure to environmental toxins like smoking, personal hygiene, exercise, stress levels, environmental pollutants like pesticides and UV exposure, and habitual nutrient intake.

The absorption of ingested nutrients depends upon absorptive efficiency; the rate at which they are consumed is determined by metabolic demand; excretory rate excreted depends on excretion efficiency.

Unless given by a novel route such as transdermal application or IV, nutrient absorptive efficiency depends upon a healthy GI tract and kidneys (e.g., no diarrhea, loss of electrolytes, vomiting or fat malabsorption which would limit absorption of Vitamins A, D, E, K). Bio-availability of Iron, Calcium and Zinc depend on pH, and the absence or presence of factors and anti-factors such as phytic acid.

Ratios of nutrients are crucial for absorption and utilization. For example, Calcium and Iron must be in a proper balance with each other as must Iron and Zinc. Zinc must also be in an appropriate ratio to Copper and these balances must be maintained over extended periods whether the dose of nutrients is low, intermediate or in the HSD range with the proviso that there are absolute limits on safe intake some nutrients like copper if absorption is normal although persons with impaired absorptive capacity. The concept of optimum nutrition, with its inherent recognition of biochemical individuality, allows for

⁴¹ Ibid

⁴² Ibid

personal deviation from the statistical norm since normal values is statistically, not biologically, derived.

Absorptive deficiencies (often based in absolute or relative nutrient imbalances) can prevent proper absorption of other nutrients. For example, if Vitamin B12 is low, folate deficiency will result, if Vitamin D is low, calcium will be deficient and if Magnesium is low, Calcium absorption will be impaired. Thus, not only absolute amounts of nutrients must be considered, the unique biochemical makeup of that person at that particular time needs to be factored in for efficient and effective nutritional therapy so that it either enhances long-term well-being or addresses the current situation on a responsive, as well as a preventive, level.

Health, disease, balance/imbalance in nutrients, medications and sequestrants all change the excretion pattern of nutrients. Happily, because the toxic profile is so low, there is a huge safety margin for nutrients. Because of this, it is easy to have a beneficial impact using nutrients but very hard to do harm using them, unlike drugs where the opposite is true.

During apparent health (before deep cellular changes make themselves apparent in symptoms or disease entities) metabolic requirements for nutrients go up rapidly under many circumstances.

Growth, for example, increases the need for Iron, Zinc and Folate, Pyridoxine, Vitamins A and D and Calcium while stress (including alcohol intake) not only causes mineral wasting and, over a long period, accelerates bone loss, but also causes Vitamin C to be rapidly depleted. Magnesium, Vitamin K, Zinc, Magnesium, Chromium, Pyridoxine and all antioxidants are depleted rapidly as a consequence of dietary excesses.

On the other hand, once disease (accumulated profound cellular damage) develops and symptoms and dysfunctions are apparent, other shortfalls develop rapidly. During infection, for example, Iron, Zinc, Pyridoxine and Vitamin C are in short supply. All of the functions they carry out and modulate are now either halted or challenged to keep up with the metabolic demands which are usually increased in illness. Alcoholism depletes Magnesium, Zinc, Magnesium and Thiamin while medications routinely deplete CO Q 10, Vitamin D, Folate and Pyridoxine plus many other nutrients. It is important to remember that just as the requirements for a nutrient are individual points along a continuum of requirement and that point may change with age, nutritional status, stress, toxicity, etc., so, too, the absorptive pathways and particulars of nutritional requirements vary widely within and between people.

For those people who neither absorb nor metabolize vitamins well, an activated form of, for example, B1 (Thiamin Pyrophosphate or TPP), B6 (Pyridine 5 Phosphate or P5P) or B12 (dibenzoyl) is required to provide optimal nutritional status.⁴³ The reality, then, is that the individual status of a patient determines his/her nutrient requirements and that appropriate treatment takes this into account and capitalizes on it.

Few people live in the pristine environments many of our ancestors did 250 years ago. We are, regrettably, no longer free of pesticides, human and veterinary drug residues, xenobiotics, flame retardants, petrochemicals, formaldehyde and heavy metal residues in

⁴³ Erasmus, U., *Fats that Heal Fats that Kill*, Revised, Alive Books, Burnaby BC, 1993, p. 78

our bodies and our surroundings. Fewer people still partake of an unadulterated food chain grown in fully mineralized soil, diets which provide them with perfect ratios of omega 3, 6 and 9 fats, zero hydrogenated and other dangerous fats, adequate unprocessed and well prepared food which has made its way swiftly from a nearby farm to their daily feast.

Assuming that these pristine eaters (and breathers) never come into contact with any of the toxins of an industrial food supply and the industrial world, they may find themselves in the tiny minority of people who might be able safely to avoid judicious consideration of nutritional supplement at one or more points of their lives. Vitamins and minerals are essential for both cellular processes and cellular detoxification. The greater the body burden of toxins, the greater the requirement for nutrients and, in the uniquely toxic modern world, the greater level and diversity of nutrients required to support not just a standard of minimal health (which can be described as the absence of overt deficiency diseases), but optimal and robust health. This sought-after and achievable goal requires nutritional support at a level far greater than the amount of these nutrients reasonably consumed from food.

Personal nutritional optimization though supplementation in addition to the best available diet is an effective strategy which, when properly individualized, offers a country the opportunity to increase the well being of its citizens while reducing the financial and social cost of medical care to the society substantially compared to a national strategy of allowing cellular damage/chronic degenerative diseases to develop and suppressing their symptoms with toxico-pharmacology.

Put simply, **prevention of the staggering personal, social and economic burden of nutritional diseases can be accomplished through nutritional supplementation, but cannot be accomplished without it.** The paradigmatic opposite of preventive health is reactive medicine, in which a condition presents itself with already apparent pathology and that pathology, not the underlying cause of it, is addressed, usually with pharmaceuticals that inhibit or poison enzyme pathways to produce specific desired (and undesired) changes. Reactive medicine is effective and important in acute and traumatic situations while preventive medicine is significantly more effective at reducing the personal and social burden of degenerative disease. Preventive medicine is significantly less expensive than reactive medicine since it is low tech and uses substances whose costs are low; the bulk of the nutrient materials cannot be patented since they are found in nature.

Properly designed, an economical and effective health system which makes an active and meaningful contribution to ending preventable ill health and disease combines the strengths of conventional reactive, allopathic medicine and those of natural, preventive medicine and public health. Reactive medicine's strength in acute/trauma care and diagnosis can be effectively used to guide preventive and natural medicine. Natural medicine excels at prevention and at repairing underlying cellular damage while prevention of disease represents one of conventional allopathic medicine's weakest areas.

Nutritional strategies are employed at various levels of organization from the cellular to the organismic in order to achieve several goals which conventional medicine is rarely able to achieve:

1. **Optimization of Cellular Activities.** Since cellular activities are absolutely dependent upon nutrient components being both abundantly present and available for use, nutritional strategies are uniquely critical to support of the metabolic processes upon which both health and life depend. Common examples of optimization in clinical situations through nutritional strategies include the nutritional correction of diverse mechanisms for similar problems. For example, two different anemias require different treatment they are not conditions of the blood, but hematic expressions of underlying nutritional imbalances:
 - a. **Iron-deficiency anemia** (microcrystal, hypochromic), is the most frequent nutritional deficiency disease globally and is a part of a cluster of signs and symptoms associated with insufficient oxygen carrying capacity.
 - b. **Folate deficiency** (megaloblastic anemia) is the most prevalent vitamin deficiency disease and results when replication, but not growth, of cells is inhibited.

2. **Reduction of Metabolic Burden Imposed on Organ Systems.** Poor quality or unwise intakes (e.g., excess refined carbohydrates, trans fats, excess omega-6 and -9 fatty acids, etc.) and dietary excesses impose a metabolic burden on organ systems necessitating compensatory responses to support vital functions in the absence of sufficient amounts of nutrients. If these responses are sustained by prolonged imbalances in intake/absorption of these nutrients, maladaptive changes to counter these responses will cumulatively contribute to cellular injury and pathology. While diet and physical exercise habits are vitally important in this response pattern, supplemental nutritional strategies are strikingly effective in moderating the cellular metabolic status. Examples of these compensatory responses to alleviate diet-related metabolic burdens include:
 - a. **Elevated Insulin** (Hyperinsulinemia) is a compensatory response designed to normalize fasting blood glucose levels when insulin receptors are down-regulated by excess dietary intake of refined sugars and fat. Chromium picolinate and vanadium (vandyl sulfate) are important nutritional tools for the normalization of insulin levels as are Omega 3 Fatty Acids, Biotin, Calcium, Manganese, Magnesium Co Q 10, Soluble Fiber, N-Acetyl Cysteine, Selenium, Taurine, Cysteine, Vitamins B 1, 3, 6 (especially pyridoxal 5-phosphatate), C, E and Zinc.

Elevated blood pressure (Hypertension) is a compensatory response which often (but not always) results from hypervolemia required to maintain plasma osmolality/osmolarity and volume when Sodium intake exceeds renal excretory capacity⁴⁴. Increased Potassium in conjunction with decreased Sodium, Calcium supplementation, Garlic, L-tryptophan, Vitamins A, B 3, B6, C, E, Co Q 10, Zinc, Omega

⁴⁴ It should be noted that there are people in whom hypertension results when renal excretory capacity is excessive. The compensatory mechanism in these people is an increase in aldosterone and resultant hypertension which is responsive to an **increase** in sodium.

3 Fatty Acids and Flax Seed meal, Magnesium, Taurine and Arginine all help to correct this underlying maladaptive response.

3. **Protection of Tissue Integrity and Support of Cellular Defense Systems.**
 - a. **Free radical damage** from activated oxygen species is responsible for initiation of cellular injury underlying most disease processes. Nutrient-dependent cellular defense systems include antioxidant protection against free radical damage, detoxification enzymes, and immune function. All of the critical cellular antioxidant systems either are nutrients or are derived from nutrients. These critical components include free radical scavengers made from Carotenoids, Vitamins A, C and E, enzymes which require Selenium, Manganese, Iron and Glutathione (synthesized from amino acids).
 - b. **Detoxification** Biologically active endogenous and exogenous compounds are detoxified by Phase I and Phase II detoxification enzymes induced and moderated by dietary factors that include nutrients and other bioactive compounds found in foods of plant origin such as Beta-Sitosterol. They can be easily compromised or overwhelmed and require vigorous support with Iron, Vitamin E, Vitamin C, Selenium, Alpha Carotene, Beta Carotene, Lutein/Zeaxanthine and Lycopene.⁴⁵
 - c. **Immune compromise** Long before classical disease states manifest, the cellular damage resulting from the early stages of deficiencies of a wide array of nutrients results in an increased vulnerability to infection secondary to compromised immune function. Subtle immune compromise of nutrients such as Iron, Zinc, Vitamins A, and C, Folate, Pyridoxine, protein and energy intake are often first suggested by frequent or opportunistic infections, poor healing and poor infection resolution. The immune system is dependent on an available abundance of a large number of nutrients including those which
 - i. Preserve epithelial barrier function
 - ii. Support increased rates of lymphocyte proliferation and differentiation
 - iii. Contribute to synthesis of immune substances
 - iv. Maintenance of immune cell activities and inflammatory response.

Nutritional strategies for prevention, mitigation, treatment and cure are exceptionally powerful modalities in any disease condition or degenerative trend which occurs because of:

- Depleted nutritional status secondary to
 - Poor intake (reduced nutrient density and diversity in available foods)
 - Poor lifestyle choices (prepared, processed food diminished in nutritional value, over-reliance on a small number of foods)
 - Impaired digestive capacity (e.g., insufficient hydrochloric acid, etc.)

⁴⁵ <http://www.natap.org/2003/DDW/day10.htm>

- Impaired pH balance (e.g., choice of acidifying foods, paucity of alkalinizing ones)
- Impaired ability to absorb or utilize nutrient in the available form
- Underlying disease states
- Poor underlying nutritional status when faced with acute demand (e.g., wound healing, disease recovery, pregnancy and birth, etc.)
- Inadequate nutritional supplementation specific to individual needs
- Immune compromise secondary to
 - Pharmaceuticals (e.g., steroids, etc.)
 - Environmental contamination (e.g., pesticides, industrial by-products, etc.)
 - Pathogenic organisms (e.g., viral load, parasites, pathogenic bacteria, etc.)
 - Compromised nutritional status (e.g., macro-nutrients, micro-nutrients, co-factors)
 - Radiation poisoning
- Accumulation of toxic materials such as
 - Metabolic by-products (e.g., Phase I metabolites, fecal contaminants, etc.)
 - Metabolic poisons (e.g., heavy metals, organophosphates, petrochemical contaminants, etc.)
 - Toxic contaminants of food (e.g., aflatoxin, etc.)

A comprehensive discussion of the prevention, mitigation, treatment and cure of all preventable diseases and treatment strategies for acute conditions is beyond the scope of this discussion so a few representative diseases and conditions have been chosen to demonstrate the power, depth and profundity of nutritional strategies to the nutritional treatment of serious, potentially life threatening disease. The outlines of the nutritional therapy picture will be sketched here to be filled in later. Nutritional strategies are not only promising in their prevention and treatment but in many cases, have already been proven and effective.

Several framing assumptions are important here.

A. Natural Health and Western Medicine. There is no intent to undermine or displace conventional Western Medicine its areas of strength

1. Technical diagnosis (including radiological and other visualization techniques and laboratory studies of all types)
2. Acute care for emergencies and trauma. No other type of medicine offers as much power in acute and traumatic situations as allopathic medicine.

B. Natural Health and Botanicals. This paper focused on nutritional strategies for

- Prevention
- Mitigation
- Treatment
- Eventual elimination of chronic degenerative disease

No discussion is offered of the many important herbal and non-nutritional strategies which form a vital core in the natural and non-toxic prevention and treatment of disease. Exercise, smoking cessation, safe or absent alcohol use, lack of illicit drug use, sanitation, education and other vital areas of public health have likewise not been addressed.

The current threat to consumer (and health professional) choice is to nutrients for which unwarranted and dangerous restrictions are being urged by various international organizations. It would be foolhardy to attempt to deliver wise and effective natural health care without botanicals and other non-nutrients or without considering the other aspects of a healthy – or an ill -- population.

C. Natural Health across the Life Span and Diversity of Chronic Degenerative Diseases. The field of Natural Health encompasses the immense areas of

- Health promotion
- Health maintenance
- Treatment of chronic preventable illness.

Although this field is vast, it is crucial to this dialogue that the power and depth of Natural Health options based in nutrition be clear. The representative conditions which are presented here were selected because they present important public health problems and are paradigmatic for the remainder of the universe of chronic degenerative illnesses and are suitable for treatment at a reduced monetary cost to the health care system compared to conventional Western Medicine at a significant savings in the human dimension. in efficacy and population well-being.

2. Nutrients as Foods

Nutrients are essential ingredients necessary to feed, supply, activate, regulate and indeed, create our cell structures, interstitial materials, bones, hormones and, importantly, our enzymes. “The physiology of organisms is based on the existence and speed of chemical [enzyme] reactions⁴⁶ : the optimal function of our enzymes depends on optimal nutrient availability when and where those nutrients are needed. Optimum enzyme function is intimately associated with a supra-abundance of nutrients since excesses are either excreted from the body or stored in fat for future use.

Optimal health is possible only in the sustained presence of sufficient nutrients in the form, amount and type required at that moment. Optimal health is one of the common goals of people everywhere. People know instinctively, from countless eons of human memory, that food and health are intimately linked. They may or may not know biochemistry and nutritional science, but they understand that health and food are deeply connected.

Biochemistry has illuminated many of the mechanisms by which this common human truth is expressed. At any given moment, about 35,000 enzymes in each cell are actively carrying out all of the processes of life. They are, in fact, the very stuff of life. In his presentation speech of the 1965 Nobel Prize in Medicine, Professor Sven Gard, a member of the Nobel Committee, stated, “one of the principal functions of genes must be to determine the nature and number of enzymes within the cell, the chemical apparatus which controls all the reactions by which the cellular material is formed and the energy necessary for various life processes is released. There is thus a particular gene for each specific enzyme”⁴⁷

Thus, every aspect of biological life can be seen as providing what enzymes need so that they can carry out the functions which, in total, comprise a human being. Nutrition is provided to the cells so that their enzymes have the means to carry out their functions and, in the process, life emerges. But illness emerges, too, if poor choices, limited availability, degraded foods from degraded soils and chemical contamination a variety of sources force a distortion on the sequence of events, numbers, status or health of those enzymes.

Common sense and the life sciences converge at the perception that nutrients are foods:

- Nutrients have their origin in foods stuffs although they may be sometimes be synthetically produced in the modern industrial context
- Nutrients are used by the body through their interaction with enzymes and other biological systems which have evolved over the course of human development to deal with the components of the food supply

⁴⁶ <http://www.mcli.dist.maricopa.edu/syris/modules/index.php?yr=0102&id=5>

⁴⁷ *Nobel Lectures, Physiology or Medicine 1963-1970*, Elsevier Publishing Company, Amsterdam, 1972

- Nutrients support biological processes, they do not interfere with them
- Nutrients are required for every biological process either for the energy they supply or the molecules which the enzymes of the body require for every action
- Nutrients have a negligible toxicity profile. Even fat soluble vitamins require massive, nearly impossible to attain, doses to produce any signs of toxicity. Polar bear livers in the hands of starving arctic explorers are in short supply in most of the world
- Nutrients have been extensively consumed (billions of doses) but the number of deaths or serious outcomes which can accurately be attributed to them is less than miniscule.
- Most diseases are the result of long-term, sub clinical states of nutritional inadequacy. Cellular damage is produced during the period of uncorrected, undetected, early and mid-stage nutritional deficit
- Nutrients are required for optimal health in dosages which vary widely depending on the multi-variant, complex and dynamic biological status of the individual which is impacted by Biochemical Individuality which is made up of
 - Genetic factors/family history
 - Digestive capacities
 - Absorptive capacities
 - Toxic load
 - Heavy metal or other enzyme inhibitors
 - Underlying disease
 - Adrenal status
 - Immune competence and status
 - Acid-Base balance
 - Enzyme production
 - Lymphatic efficiency
 - Mineralization of bone
 - Hormone status
 - Age
 - Gender
 - Life cycle stage
 - Dietary intake
 - Treatment with drugs, radiation
 - Emotional status
 - Vaccination status
 - Gut ecology
 - Etc.
- Nutrient status in food has declined over the course of the industrialization and resulting adulteration of the food supply resulting in a sharp rise in the incidence of the chronic diseases of degeneration and neurological conditions.

- Nutrients frequently correct the underlying cellular functional damage which results in the chronic degenerative diseases without introducing toxic or damaging compounds.
- Nutrients, like other foods, are so safe that no protective or restrictive regulation to limit their use is desirable, necessary, prudent, scientifically supported or logical.

Our current norms for biological normality and health have been derived from information gathered from a population which has the poorest health of any society in the history of mankind-- ours. We have more cardiovascular disease, cancer, diabetes, autism and other neurological disorders, macular degeneration, arthritis, osteoporosis, MS and a host of other diseases than any population known to history. Human remains of the past hundreds of thousands of years make it clear that our ancestors were better fed and therefore better nourished than we. Numerous observers of pre-technological peoples and of the archeological record conclude atherosclerosis, cardiovascular disease, cancer diabetes, osteoporosis, rickets and other common western diseases were absent when humans were eating much more nutrient dense food and became common as the nutrient density in our food declined.

Weston Price, DDS, studied both the food supply and the health status of pre-technological peoples all over the world and found them to be superior to the peoples of the industrialized world in their physique, dentition, and health. He documented that rather than being short and ill, these peoples were tall and healthy. Moreover, they were free of cardiovascular disease, cancer, osteoporosis, diabetes and other diseases of the developed and developing world. These tribes of the world ate a diet that was many fold richer in nutrients than our modern diet and which a prudent modern diet with nutritional supplementation could approach.

The populations Dr. Price studied consumed food that contained at least:

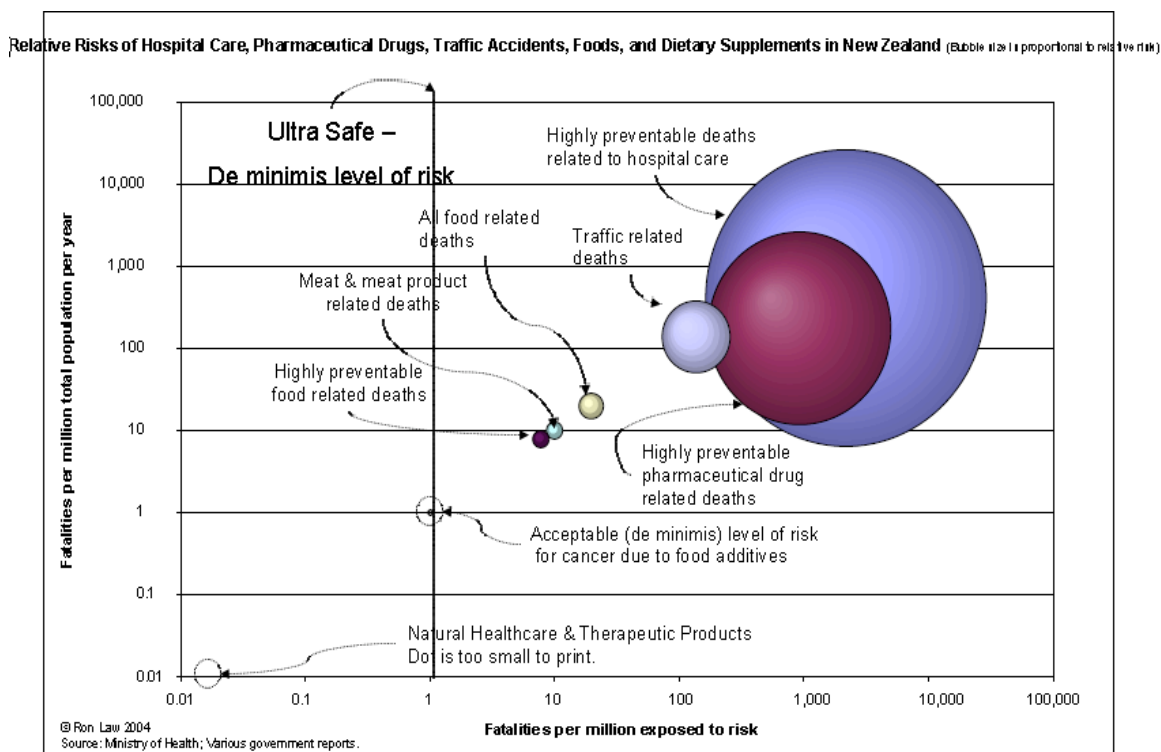
- Four times the nutritional minerals—Calcium, Phosphorus, Magnesium, Iron, etc.,
- Ten times the fat-soluble vitamins. Vitamin A from animal sources is not the same as its precursors, the carotenes found in plant foods. The conversion of carotenes in the human body is often compromised, and even under optimal conditions is not efficient enough to supply the amount of true vitamin A Price found in the diets of healthy isolated populations.

The human experience and the life sciences tell us that nutrients are food and that their toxic profile is virtually inconsequential with the possible exception of nutrients derived from genetically modified sources. But the options available to us in the conventional Western medical tradition when our nutrient status fails us, when we develop the preventable diseases of under nutrition, are not substances and procedures with a low toxicity profile. In fact, in the conventional allopathic model, when nutrition fails and disease develops, pharmaceuticals are routinely employed.

Pharmaceuticals are, however, dangerous and highly toxic. Drugs work by a common mechanism: they poison enzyme systems. If the outcome is acceptable, it is labeled a “therapeutic effect”. If not, it is a “side effect”. Side effects pile on top of one another until the body can no longer compensate or tolerate the poisoning and, if the drug is not discontinued, the iatrogenic (doctor caused) problem either results in another drug being introduced to counter the first or death. In fact, in the United States alone, *properly used prescription drugs* are the 4th leading cause of death (a minimum of 106,000 people per year)⁴⁸ while total drug-related deaths reach at least 200,000 per year.⁴⁹

It is important to remember that in the conventional Western medical model, nutrition is neglected until disease manifests and then toxic, expensive and, if the goal is restoring underlying health, ineffective treatment is instituted. In short, devastatingly toxic drugs are the conventional option for treatment if nutrient status is not adequate to prevent the development of disease. Dietary adjustments may not only influence present health, but may determine whether or not an individual will develop such diseases. Some statistical analyses will make the point clear through Ronald Law, MD’s simple and illustrative figures.

Risk of Hospital Care, Drug Treatment, Traffic Accidents, Foods and Dietary Supplements

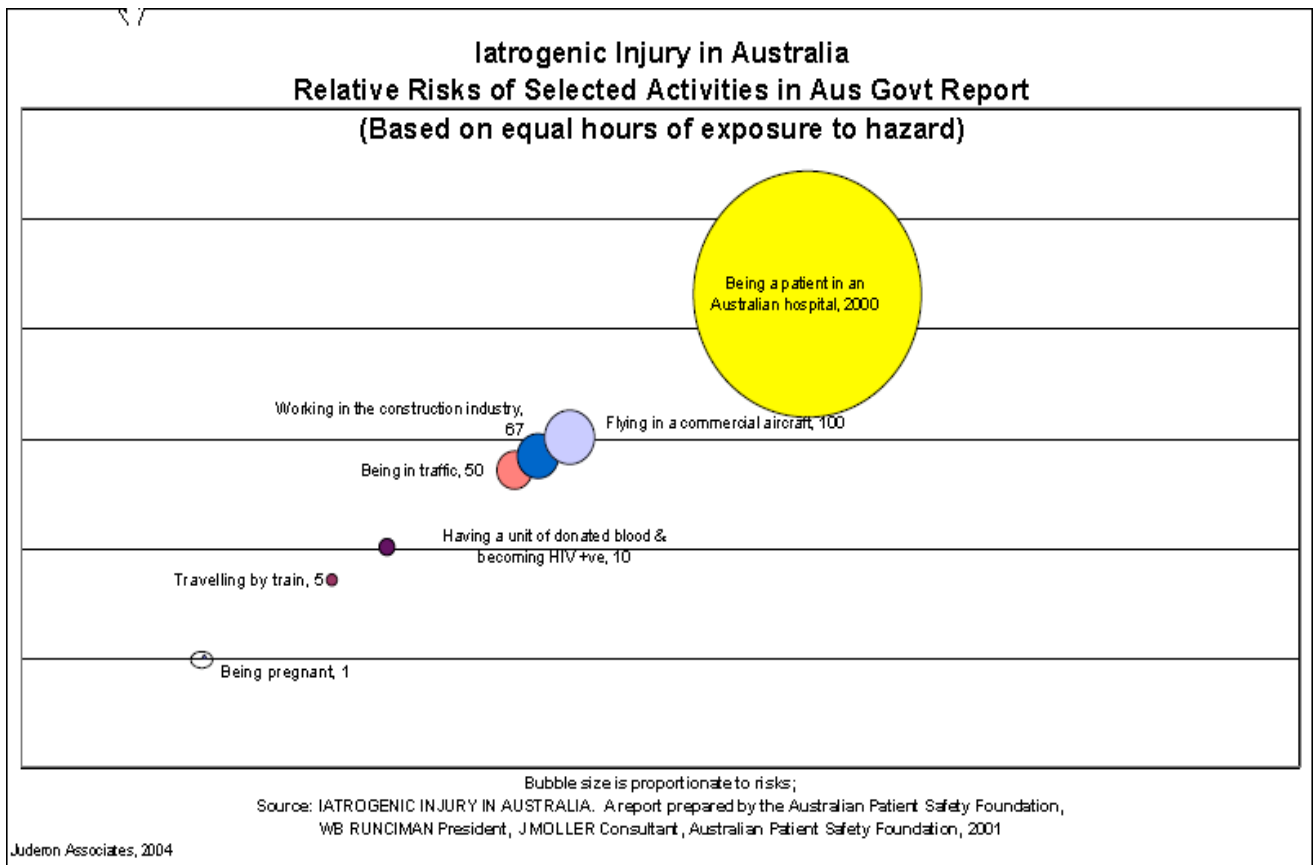


⁴⁸ http://www.lef.org/magazine/mag2004/mar2004_aws_i_death_02.htm

⁴⁹ Ibid

In the prevention, treatment and mitigation of the preventable chronic diseases of under nutrition, Natural Medicine has tremendous safety issues illustrated here in several different ways.

Risk of Iatrogenic Injury (Australia)



Much of the risk of being a patient in a hospital is due to the risk of medication reaction or death.

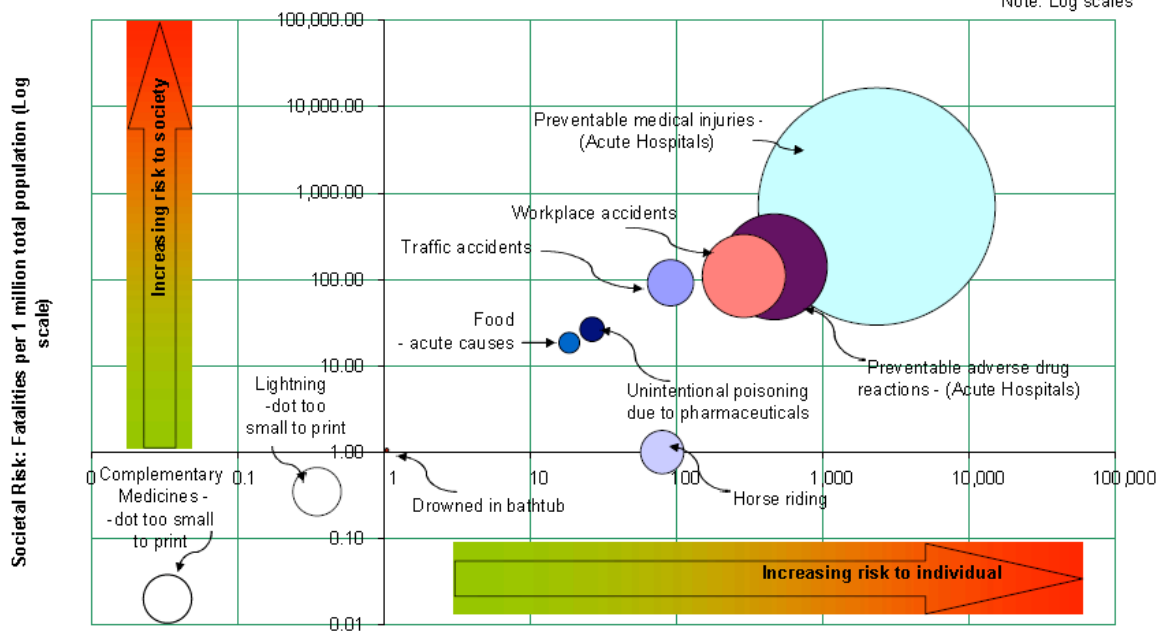
In a study of patients leaving the hospital, out-patient adverse events from drugs occurred in at least 66% of patient⁵⁰ Nutrients are orders of magnitude away from that disastrous level of risk.

⁵⁰ Forster AJ, Murff HJ, Peterson JF, Gandhi TK, Bates DW. The incidence and severity of adverse events affecting patients after discharge from the hospital. *Ann Intern Med* . 2003 Feb 4;138(3):161-7.

Relative Risk of Death from Natural vs. Western Medical Compared to the Risk of Dying in a Boeing 747 Crash

Societal vs Individual Risk in Australia

Bubble size represents risk relative to 1: million individual risk or equivalent to the risk of a single flight on a Boeing 747 anywhere in the world.
Note: Log scales

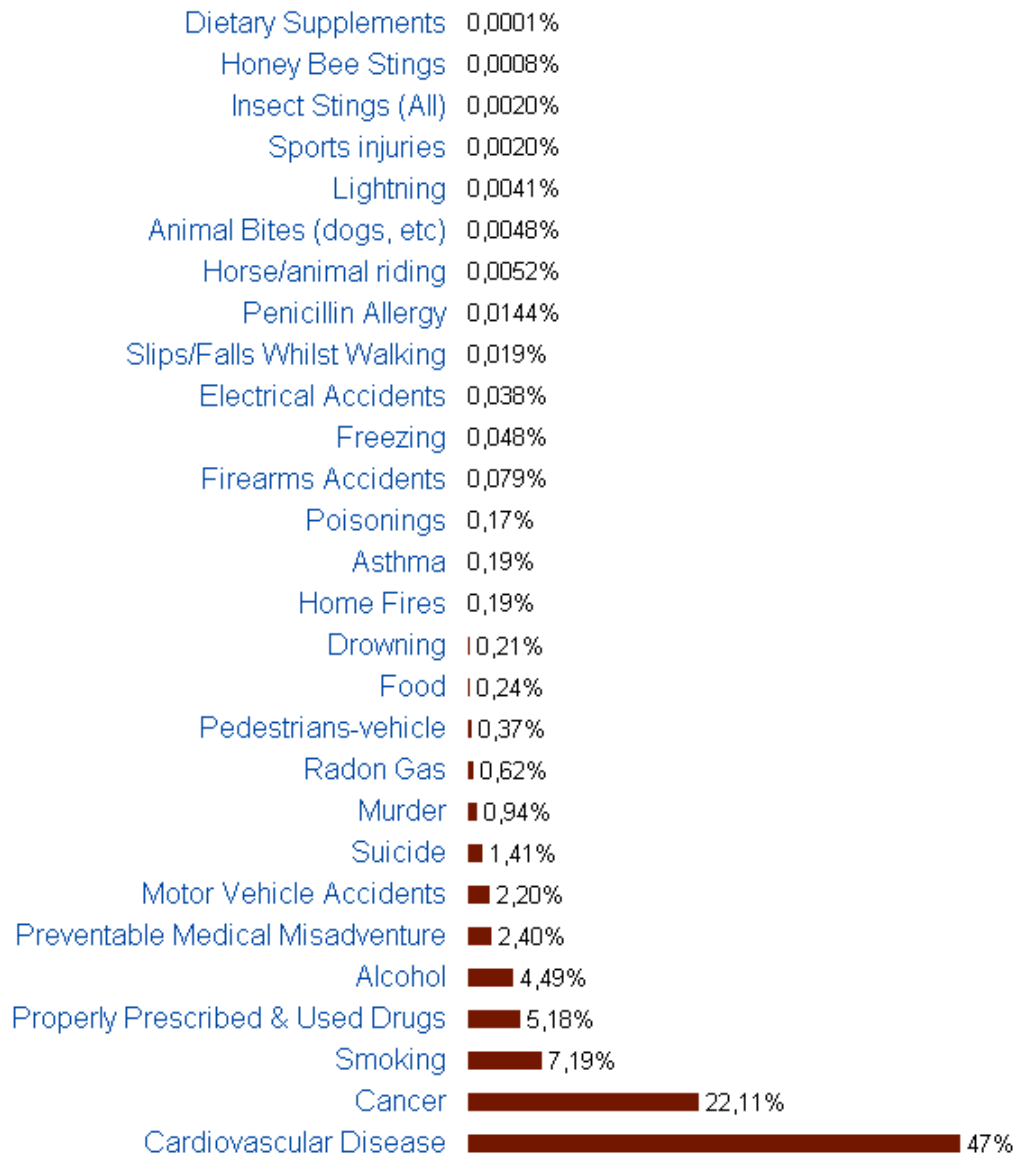


Sources: Variety of Australian Government and NGO databases and reports.

Individual Risk: Fatalities per million people at risk (Log scale)

© 2004, Juderon Associates

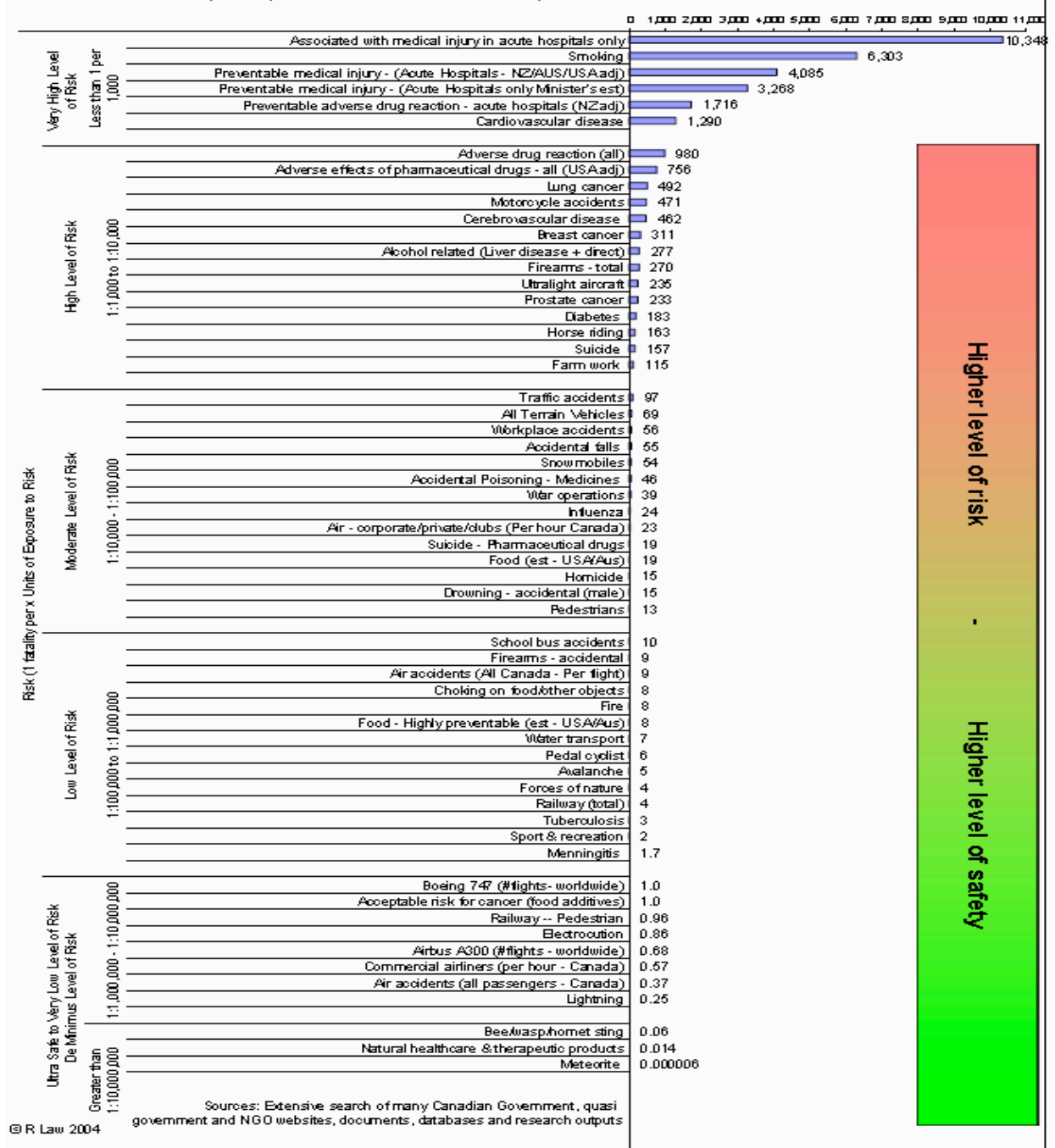
Statistical Risk of Death from Various Causes, Australia, 2004



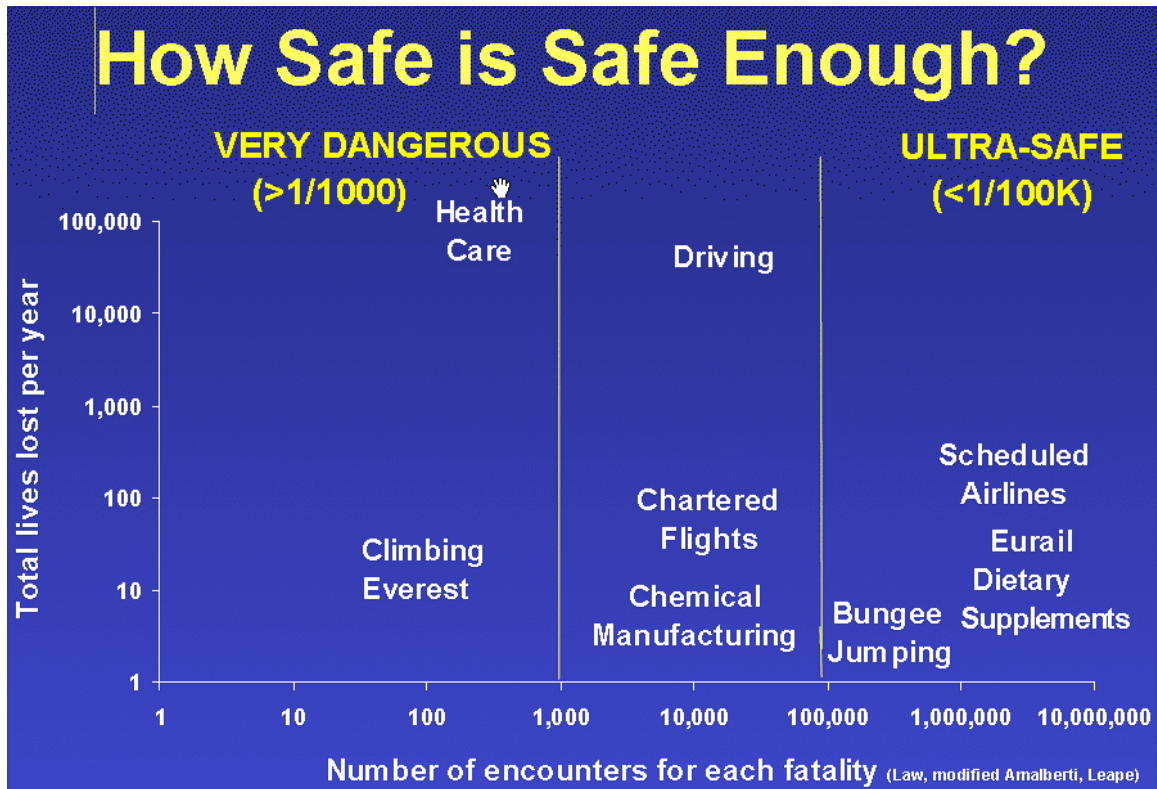
Risk of Dying in Canada Relative to Being Killed on a Boeing 747

Risk of Dying in Canada Compared to Being Killed on a Boeing 747 Flight

(See separate table for units of risk used)



Do Ultra Safe Options Need Regulation?



Accompanying his graphs, Ron Law, MD, includes this relevant information which is quoted in full:

- “□ Properly researched, regulated, prescribed and properly used drugs are the fourth most common cause of death – but they are never reported. (Source, Journal of the American Medical Association - Range 90,000 to 160,000 deaths per year.) That’s a Boeing 747 crashing every day! 46 people die every day from Aspirin alone in the USA.
- Avoidable medical misadventure is the sixth most common cause of death. (Source, CDC - range 40,000 to 90,000) In Australia 9,000 people die from avoidable medical misadventure every year. (Source, Australian Medical Journal). In Australia 50,000 people are maimed by medical misadventure every year. (AMJ)
- The figures used in this chart are at the lower end of the range (we wouldn’t want to be accused of exaggerating!)
- Food poisoning/adverse reactions cause between 5,000 to 9,000 deaths per year. (Source, CDC.)

- Dietary supplements have averaged less than 5 confirmed deaths per year over the past 25 years in the USA. Most of those relate to a single batch of genetically engineered tryptophan introduced in the late 1980's. (Source, CDC/FDA) There have been no proven deaths to dietary supplements in NZ.
- A wide range of dietary supplements are consumed by over 50% of the population in both the USA and New Zealand (Source, NIH/MOH)
- You are less likely to die from taking a supplement than dying from bee stings, sports injuries, lightening, animal bites, horse riding, radon gas, etc, etc.
- Dietary supplements are incredibly safe.
- Dietary supplements have the potential to reduce deaths from cancers and heart disease by over 50%. (Optimists would go as high as 75%)
- Greater than 26,000 times more people die from preventable medical misadventure and properly regulated properly prescribed and properly used drugs than from dietary supplements.
- You can have every confidence in assuring the safety of dietary supplements.
- There have been two deaths reported as being linked to dietary supplements in NZ – both were in people with malignant cancer who consumed the herbal mixture K4. Neither were proven to be due to K4. The coroner in one case said there was no evidence to link K4 to one of the deaths – he had terminal cancer of the liver, took K4 and died of liver failure. Officials tried to blame his death on K4. Despite the evidence to the contrary, K4 was banned.
- There was a recent media report linking Ginkgo Biloba to the death of a heart patient due to cerebral haemorrhage. The patient had been taking Ginko for some time. He was taking blood thinning drugs which are notorious for causing cerebral haemorrhage. Contrary to media reports, papers obtained by the NNFA under the official information act revealed that the MARC did not find Ginkgo to be the cause of death.”⁵¹

In ratifying the Vitamin and Mineral Guideline (VMG) on July 4, 2005, the Codex Alimentarius Commission made several serious errors which will, if enacted by Codex member countries, have disastrous impact on the health of their peoples. Codex adopted a standard which allows for the assessment of risk without the possibility of any consideration of benefits. Thus, if a nutrient could be said to be toxic at any level it can be considered a dangerous substance. Clearly, this is not a game being played on a level playing field: any substance, including oxygen and water, are toxic at some dose. Worse yet, the definition of an adverse event accepted is the scientifically untenable position that any substance which changes a bio marker so that it is no longer in homeostasis should have an Upper Limit (UL) created for it by Codex's modified risk assessment procedures.

Codex toxicology is far an appropriate methodology for determining upper limits for Vitamins and Minerals if they were needed. Toxins and dangerous industrial chemicals

⁵¹ http://www.laleva.cc/petizione/english/ronlaw_eng.html

should have safe ULs. We have seen how far from toxic or dangerous nutrients are in Dr. Law's figures.

The VMG specifies that 'upper safe levels of vitamins and mineral established by scientific risk assessment based on generally accepted scientific data' should be used to determine these values. Since there is no "scientific risk assessment based on generally accepted scientific data" which exists for the evaluation of a substance essential to life and beneficial over a wide dosage range, this is not a meaningful requirement. The inappropriate application of the tool will inevitably lead to incorrect answers to incorrect questions. In fact, Codex nutrient risk managers are urged to create ULs out of incomplete and inaccurate data which does not pertain to the populations under regulatory control by making corrections on imaginary corrections on top of corrections based on poor quality or absent data. The net result for those countries which adopt this system of thought will be mandated under-nutrition and a predictable rise in the death and suffering resulting from preventable diseases.

Risk assessment is a discipline of toxicology which is designed for, and has been peer reviewed and evaluated within the context of, the evaluation of the highest dose of a toxin which can be tolerated by a human being before there is a discernable change in that human being's state. It is appropriately used for poisons, dangerous industrial chemicals, agricultural chemicals and the like.

Codex has accepted the use of Risk Assessment procedures for nutrients without scientific justification either in the focus of the risk assessment analysis, (i.e., ultra low toxicity components of food) or the methodology used to make the determinations of dangerousness in order to set ULs on nutrients.

Codex acted unwisely in ratifying the Vitamin and Mineral Standard which focuses only on risk of nutrients with no consideration of benefits. Risk Assessment is a methodology relevant only to toxicology and both irrelevant and antithetical to Nutritional Science and Biochemistry. The Risk Assessment methodology employed by CODEX has been arbitrarily modified without scientific validation or professional consensus to restrict permissible dosages of nutrients essential to life to levels which can, by intent, have no meaningful impact on any human being, no matter how sensitive. This misapplication, distortion and misconstruction of Risk Assessment is in clear contradiction to the principles of toxicology and scientific Risk Assessment procedures which have been developed to determine the highest dosages of dangerous industrial and natural toxins to which humans can be exposed to without discernable effect. For this reason, instead of evaluating vitamin and mineral upper limits using inappropriately modified and unscientific Risk Analysis, the Natural Solutions Foundation is urging the US to change its policy on this and related issues. We urge the use of Nutritional Science rather than toxicology to support the liberal access to nutrients enjoyed under legislative protection in the US. Under the Dietary Supplements Health and Education Act, passed by unanimous Congressional consent in 1994, while a nutrient may be dealt with by the

FDA if it is shown to pose a significant risk to health and safety, barring that, nutrients are treated as foods and, as such, may have no upper limits set upon their use.

The inappropriate classification of nutrients as dangerous substances from which the public needs protection lacks a scientific basis. None the less, this is precisely what Codex articulated in its October 2004 document on Risk Assessment⁵². In that document, the preface states:

The need for an internationally relevant or “harmonized approach for nutrient risk assessment is well recognized. The increased consumption of fortified foods, formulated so-called “functional foods” and dietary/food supplements has made nutrients risk assessment highly relevant to protecting public health and to the practice of setting science-based international standards for food”⁵³

when, in fact, the premise of public danger is not supported by data or experience. Nutrients are not toxins or dangerous industrial chemicals.

The document then goes on to make it clear that the form of risk assessment used is not science-based because the application of risk assessment to substances vital for life is a newly created mis-application of the process:

“Certain nutrients and related substances, **like other ingested substances (e.g., food additives, contaminants)** can produce adverse effects **if intake exceeds a certain amount**. This potential for harm is described by the process of risk assessment, which is a science-based evaluation of available data followed by a series of decision points. **Risk assessment is well established for non-nutrient chemicals in foods.** However, **nutrients and related substances are unlike non-nutrients** in that, within a range of intake, **they provide benefit**. For this reason, **new paradigms have had to be considered that build upon the principles established for assessing the risk from non-nutrients**, but also go beyond to **incorporate additional or different principles that take into account the special characteristics of nutrients** and related substances.”⁵⁴ [Emphasis added]

The bold face phrases make clear the problematic and illogical process at work here.

- Nutrients are NOT like food additives or contaminants and should not be treated as if they were.

Some, but not all, nutrients could cause adverse effects if intake exceeds a certain amount. Billions of doses of nutrients chosen freely from a vast array has not produced a single death attributable to nutrients with the exception of children swallowing a whole bottle of pills they thought were candy or other mishaps unrelated to the substances

⁵² Joint FAO/WHO Development of a Scientific Collaboration to Create a Framework for Risk Assessment of Nutrients and Related Substances <http://www.lwho.int/ipcs/highlights/en/nrbacground.pdf>

⁵³ Ibid, preface.

⁵⁴ Ibid. p. 2

unrelated to the substances themselves. Risk assessment requires a risk. Nutrients do not meet that requirement.

- The process of risk assessment as employed by Codex and WHO/FAO is profoundly **un**-science based. It has not been established through a meaningful process of peer review and validation but instead is a jury-rigged system cobbled together to serve a political, not a scientific purpose. Scientific assessment systems are not created by committee and then employed with no peer input or clinical-world testing before the system is used on real people in a grand statistical experiment.

The October 2004 document states

- “Based on discussion papers to be developed, workshop participants will be asked to formulate an internationally-relevant scientific approach for nutrient risk assessment.
- Then, to test and demonstrate the application of the approach, workshop participants will apply the approach to a subset of nutrients, specifically several vitamins and minerals. The approach will then be refined based on this experience.”⁵⁵

It should be noted that the document goes on to state that it is the intent to apply this procedure to all nutrients in all categories since the “overarching interest is dietary substances that provide benefit but *may* cause harm at a *different level of intake* [and] “should have applicability to other nutrients and related substances.”⁵⁶

When the FOA/WHO released the results of the workshop (anticipated by the October, 2004 documents announcing it], the demonstrated bias was evident in that report⁵⁷. Despite its dire health consequences, this system has not been tested in the real world and, since it is explicitly not based on real data, can be expected to create confusion and inaccuracy.⁵⁸

In the table of contents to the 357 page FAO/WHO document, there is no mention of benefits from nutrients. The report does mention that while attention was paid to the hazards of high nutrient intake (a fictitious risk), no attention was paid to the impact of low nutrient intake (a very real risk).⁵⁹

⁵⁵ Ibid. p. 5

⁵⁶ Ibid.

⁵⁷ 130106 FAO WHO risk assessment full_report.pdf

⁵⁸ Ibid. p. 18

⁵⁹ Ibid. p. 12

This flight of imagination continues as the report goes on to state that the risk assessment process has been modified in novel ways and that the fact that risk assessment is being used to deal with non toxic substances “influences approaches used to estimate an upper level of intake and also necessitates that the homeostatic mechanisms specific to essential nutrients be taken into account”⁶⁰

This means that an entirely different standard of scientific relevance has been introduced to create a new standard of nutrient impact without any scientific justification: *“biochemical changes outside the homeostatic range can be relevant surrogates for adverse health effects associated with nutrient substances.”*⁶¹ [Emphasis added]

The dangerous illogic of this methodology is apparent: homeostatic measurements derived from an unhealthy population are now the yardstick for toxicity: any nutrient which brings about a change (or is likely to lead to an enhanced stated of health) will be declared a hazardous substance at that dose. “Hazard” is defined in this document as “the inherent property of a nutrient or related substance to cause adverse health effects depending on level of intake.”⁶² By this standard, all substances are hazardous, nutrients among them. Since adverse events are defined as biochemical responses, including desirable ones, and since there was no participation in this workshop of nutritionists, clinicians or other persons knowledgeable in the clinical uses of nutrients, the toxic slant given to all nutrients may make some sort of sense politically. But the imposition of this skewed system on trade and health makes no sense and is a very dangerous policy.

The report makes it clear that anything other than a definition of nutrients as hazardous was outside of the interest of the group, “...other aspects of evidence-based systematic review, notably the kinds of questions it seeks to address were generally not viewed as appropriately suited to nutrient risk assessment.”⁶³ This is further confirmation that clinical and population health concerns were “not appropriately suited” to the task at hand.

Of course, if every nutrient is toxic under this definition no possible significant deviation based on consumer preference or individual biochemistry will be possible. Although this system was created de novo and does has not been validated or tested, the world's health can be expected to be seriously impacted by it.

The report states that “issues related to the physiological severity of adverse health effect are considered separately rather than as a component of selecting the critical adverse health effect.”⁶⁴ The impact of this curious standard is that even the tiniest “adverse effect” is enough to ban a nutrient at a dose which is greater than the dose at which the

⁶⁰ Ibid. p. 13

⁶¹ Ibid. p. 15

⁶² Ibid. p. 14

⁶³ Ibid. p. 17

⁶⁴ Ibid. p. 17

effect was noted: for example, the flush felt with niacin at, for sensitive people, 10 mg, would make that an adverse reaction.

The fact that the physiological severity is to taken into account means that Niacin could be regarded (incorrectly) as a toxin at that level. Applying a safety margin of 100, as risk assessment procedure demands, the permitted dose would then be 100 ug, a meaningless dose designed to have no discernable impact on the human body.

For a science-based document, the workshop report is surprising. For example, the document makes clear that there are few circumstances in which data exists for the substance being controlled but that, "... adjustments for uncertainly must make use of uncertainly. Factors....In any case, the these uncertainty considerations must be checked against the level of recommended intake relative to biological essentiality or the levels of intake associated with the demonstrated impact on health. ...After uncertainties are taken into account, the resulting value is the UL for the specified sub-population. **When data are insufficient for setting a UL for one or more sub-populations (as is often the case) the risk assessor fills the gap by adjusting a UL that has been established for another sub-population. It is desirable [but not necessary – author] to make these adjustments based on understandings of physiological differences between the groups. Lacking such information, however, an alternative is the use of scaling based on body weight. This type of scaling adjusts the UL on the basis of energy requirements.**"⁶⁵ Most nutrients have little or no impact on energy requirements so setting intake criteria on this basis is more than a little surprising. Biochemistry and biochemical individuality is nowhere to be found.

The weaknesses of the system are prominent in the report. Consider, for example, "**If available** intake data obtained from individuals is the most useful type of data. The group recognized, however, that **such data are rare** in most regions of the world. Thus, the report outlines approaches that **allow the use of aggregated data**. The derivation of an intake distribution may be accomplished **even with limited aggregated data by using special statistical methods to estimate and refine a distribution curve** for the (sub) population of interest. Special considerations were given to considerations for strategies for **combining data from different sources** in order to estimate intake."⁶⁶ [Emphasis added]

The clinical, medical and scientific weakness of this method, which is central to the entire nutrient risk assessment process, is clear. Imagined data will be combined with other imagined data to create ULs on nutrients which will have no adverse effect even if that is a statistical concept, not a clinical one. This pro-illness system does not even require data for its globe-spanning determinations.

⁶⁵ Ibid. p. 18

⁶⁶ Ibid. p.18

According to the Workshop Report, the process of risk assessment is designed primarily to meet the “risk manager’s special needs”⁶⁷. Those needs are referred to many times in the document but they are never specified.

It is clear that rigorous data have only a facultative part to play in this process since “risk managers” are told that they may “make additional corrections”, the nature of which is

not specified. Managers are told, “Because a nutrient risk manager typically needs a UL even in the face of limited data, efforts should be made to establish ULs if at all possible. Of course, the nutrient risk assessor clarifies the degree of uncertainty surrounding the value of the UL which in turn enables the nutrient risk manager to take this factor into account in his or her decision making.”⁶⁸

Even more surprising is the following statement which follows immediately after the previous one: **“The absence of evidence of an adverse health effect is not equivalent to evidence of the absence of an adverse health effect. That means that it is inappropriate to make conclusions about the risk or lack of risk associated with nutrient substances based solely on studies designed for purposes other than studying risks”**⁶⁹ which says quite plainly that all nutrients are guilty until proven innocent but that there will be only kangaroo trials for nutrients for some time to come!

The Report struggles with how to deal with substances for which no known risk is known and decides that the upper limit will be the highest observed dose **despite any evidence of toxicity**. The report does not state the rationale for this odd position nor does it give a reason for stating that it does not consider people who are ill or in poor nutritional status to need separate guidelines although should data come to light, perhaps other limits could be set.⁷⁰

Nutrient Risk Managers, who are unelected bureaucrats, are given the authority to remove nutrients from the food supply through regulation or other means. Given the poor quality data which is allowed, this is a very ominous empowerment.

In short, the application of risk assessment to nutrients is unwarranted, unscientific and admittedly based on poor quality data. Since there is no health problem, “fixing” it with a restriction of nutrients is a highly irrational act.

⁶⁷ Ibid. p. 19

⁶⁸ Ibid.

⁶⁹ Ibid.

⁷⁰ Ibid. p. 20

3. Clinical Examples of Nutrition in Prevention and Treatment

A. Nutrition, Wounds and Trauma

Nutritional status is strongly predictive of disease or trauma outcome.⁷¹ Maintenance of optimal nutritional status has been shown to reduce the incidence of complications such as infection/sepsis, respiratory distress, acute renal failure, hepatic encephalopathy, congestive heart failure, and multiple organ system failure.

Recovery from disease or trauma is more likely to occur over a shorter duration and with fewer complications in well-nourished individuals. Optimal nutritional status also allows more aggressive treatment to be tried with minimal risk of adverse events. The ability to restore optimal nutritional status to a poorly nourished individual with a serious illness or injury is limited not only by the accompanying metabolic, physiological, and hormonal perturbations associated with the conditions, but also by the patient's lack of interest or ability to consume the additional amounts of nutrients required.⁷²

Malnutrition causes a decreased rate of fibroblastic proliferation and neo-vascularization and impairs both cellular and humeral immunity. A high rate of metabolic activity is present at the wound site, especially within new granulation tissue. If nutrients necessary for those activities are not provided, the health of the tissue is tenuous. Proteins and their amino acid building blocks, such as Methionine, Proline, Glycine, and Lysine, are essential for normal cell function and the repair of cutaneous wounds. Linolenic and linoleic acid must be supplied in the diet, which is why they are termed essential fatty acids.⁷³

Because they are critical constituents of the cell membrane and are the source of prostaglandins that mediate inflammation, deficiency of essential fatty acids causes impaired wound healing. Deficiency of vitamins C or K leads to scurvy and coagulopathy, respectively. Minerals, including calcium, iron, copper, zinc, and manganese, must be delivered to the wound milieu to act as cofactors for vital reactions in the synthesis of proteins needed in the healing process. If the diagnosis is impaired

⁷¹ http://www.acnem.org/journal/14-2_november_1995/wound_healing_and_nutrition.htm

⁷² McDonald, A, *Preventive Medicine Lectures*, Feinberg School of Medicine, Chicago, IL

⁷³ <http://www.emedicine.com/plastic/topic477.htm>

wound healing resulting from malnutrition, ensure that the patient receives adequate protein and energy (caloric) intake. Specific vitamin and mineral supplements [often at high doses] may be required for rapid recovery of the necessary nutrients.⁷⁴

Cellular injury occurs in trauma and disease and requires abundant and diverse nutrient availability to provide the factors and co-factors necessary to support and provide:

- Structural and functional components of the repair process. (e.g., chondroitin and glucosamine^{75, 76, 77, 78, 79, 80, 81, 82})
- Chemical mediators needed to elicit cellular responses (e.g., Vitamin C^{83, 84, 85})
- Enzyme function and pH (e.g., Manganese^{86, 87} Bromelain^{88, 89})
- Cellular replication and differentiation (e.g., ornithine alpha ketoglutarate [OKG]^{90, 91})

⁷⁴ <http://www.emedicine.com/plastic/topic477.htm>

⁷⁵ Morrison LM, Murata K. *Absorption, distribution, metabolism and excretion of acid mucopolysaccharides administered to animals and patients*. In: Morrison LM, Schjeide

⁷⁶ OA, Meyer K. *Coronary heart disease and the mucopolysaccharides (glycosaminoglycans)*. Springfield: Charles C. Thomas, 1974, 109–27

⁷⁷ Denuziere A, Ferrier D, Damour O, et al. *Chitosan-chondroitin sulfate and chitosan-hyaluronate polyelectrolyte complexes: biological properties*. *Biomaterials* 1998;19:1275–85

⁷⁸ McCarty MF. *Glucosamine for wound healing*. *Med Hypotheses* 1996;47:273–5 [review]

⁷⁹ Prudden JF, Wolarsky ER, Balassa L. *The acceleration of healing*. *Surg Gynecol Obstet* 1969;128:1321–6 [review].

⁸⁰ Glade MJ. *Polysulfated glycosaminoglycan accelerates net synthesis of collagen and glycosaminoglycans by arthritic equine cartilage tissues and chondrocytes*. *Am J Vet Res* 1990;51:779–85.

⁸¹ Suyama T, Iga Y, Shirakawa H. *The acceleration of wound healing with chondroitin sulfate A and its acidic hydrolysates*. *Jpn J Exp Med* 1966;36:449–52.

⁸² Prudden JF, Allen J. *The clinical acceleration of healing with a cartilage preparation; a controlled study*. *JAMA* 1965;192:352–6.

⁸³ http://www.vitacost.com/science/hn/Supp/Vitamin_C.htm

⁸⁴ Levine M. *New concepts in the biology and biochemistry of ascorbic acid*. *N Engl J Med* 1986;314:892–902 [review]

⁸⁵ Mazzotta MY. *Nutrition and wound healing*. *J Am Podiatr Med Assoc* 1994;84:456–62 [review]

⁸⁶ Shetlar MR, Shetlar CL. *The role of manganese in wound healing*. In: Klimis-Tavantzis DL, ed. *Manganese in health and disease*. Boca Raton: CRC Press, Inc.; 1994:145-157

⁸⁷ <http://lpi.oregonstate.edu/infocenter/minerals/manganese/>

⁸⁸ Izaka K, Yamada M, Kawano T, Suyama T. *Gastrointestinal absorption and anti-inflammatory effect of bromelain*. *Jpn J Pharmacol* 1972;22:519-34.

⁸⁹ Hayashi M. Pharmacological studies of Shikon and Tooki. (3) *Effect of topical application of the ether extracts and Shiunko on inflammatory reactions*. *Nippon Yakurigaku Zasshi* 1977;73:205-14 [in Japanese].

⁹⁰ Cynober L. *Amino acid metabolism in thermal burns*. *JPEN* 1989;13:196.

⁹¹ Cynober L. *Ornithine alpha-ketoglutarate in nutritional support*. *Nutrition* 1991;7:313–22 [review]

- Platelet activity (Calcium, Vitamin K⁹²
- Protein synthesis (e. g., arginine^{93, 94, 95, 96})
- Membrane ion transport (Chloride, Sodium ions⁹⁷)
- Osmotic activity (honey⁹⁸)
- Immune response (e. g., Vitamin A, Vitamin E^{99, 100, 101, 102, 103})

Tissue-specific requirements for nutrients depend upon the nature of the disease (renal, hepatic, pulmonary, cardiovascular) or type of injury (burns, fractures, head injury, multiple trauma, major surgery) while person-specific requirements for dosage vary greatly dependant upon underlying nutritional, genetic, toxicologic, health status and other factors unique to each individual. Careful clinical assessment of the clinical complex consisting of the patient and his/her disease or condition rather than assessment of the disease or condition isolated from the underlying host realities of nutrition, genetic and biochemical individuality. This is true whether the issue is wound healing or any other disease or condition requiring healing.

The hyper metabolic states which frequently follow trauma alter cellular metabolism and create special nutritional requirements. In addition to increased energy, there is a

⁹² <http://sln.fi.edu/biosci/blood/platelet.html>

⁹³ Kirk SJ, Hurson M, Regan MC, et al. *Arginine stimulates wound healing and immune function in elderly human beings*. Surgery 1993;114:155–60

⁹⁴ Barbul A, Lazarou SA, Efron DT, et al. *Arginine enhances wound healing and lymphocyte immune responses in humans*. Surgery 1990;108:331–7.

⁹⁵ De Bandt JP, Coudray-Lucas C, Lioret N, et al. *A randomized controlled trial of the influence of the mode of enteral ornithine alpha-ketoglutarate administration in burn patients*. J Nutr 1998;128:563–9.

⁹⁶ Cynober L. *Ornithine alpha-ketoglutarate in nutritional support*. Nutrition 1991;7:313–22 [review].

⁹⁷ <http://www.fasebj.org/cgi/content/abstract/19/3/379>

⁹⁸ <http://www.manukahoney.co.uk/article5.html>

⁹⁹ Ehrlich HP, Tarver H, Hunt TK. *Inhibitory effects of vitamin E on collagen synthesis and wound repair*. Ann Surg 1972;175:235–40.

¹⁰⁰ Hunt TK. *Vitamin A and wound healing*. J Am Acad Dermatol 1986;15:817–21 [review].

¹⁰¹ Hunt TK, Ehrlich HP, Garcia JA, et al. *Effect of vitamin A on reversing the inhibitory effect of cortisone on healing of open wounds in animals and man*. Ann Surg 1969;170:633–41.

¹⁰² Bartolomucci E. *Action of vitamin E on healing of experimental wounds on parenchymatous organs*. JAMA 1939;113:1079 [abstract].

¹⁰³ Palmieri B, Gozzi G, Palmieri G. *Vitamin E added silicone gel sheets for treatment of hypertrophic scars and keloids*. Int J Dermatol 1995;34:506–9.

corresponding increased demand for vitamins, minerals and cofactors which support energy metabolism under these conditions. The demand for increased metabolic and cellular support is superimposed upon the spot where the patient rests upon the nutritional continuum which ranges from highly deficient through marginal on towards adequate and finally reaching optima.

The nutrients required in abundance specifically for wound healing include:

- **Coenzymes** for their role in the reactions of oxidative metabolism
 - Niacin
 - Riboflavin
 - Thiamin
 - Pantothenic Acid
- **Vitamins and Minerals** involved in
 - synthesis and hydrolysis of ATP
 - Phosphorus
 - Magnesium
 - Electron Transport
 - Iron
 - Copper
 - Hemoglobin Synthesis
 - Iron
 - Copper
 - Vitamin B6

Loss of Lean Body Mass (LBM), a common sequel of trauma, reduces the size of the metabolically active compartment responsible for repairing the underlying cellular injury and fighting infection. A weight loss of 20% of total body weight is associated with impaired wound healing and host immune defense with potentially cataclysmic results. All tissues except brain lose LBM proportionately to the total weight loss. Critical tissues such as cardiac and respiratory muscle are not spared. Increased hydration and fatty infiltration of muscle and liver may actually underestimate actual weight loss of metabolically active tissue. When appropriate nutrition is provided, immune system and plasma proteins recover cell mass most rapidly, but skeletal muscle may take months or even years to fully recover.¹⁰⁴

Glucose and protein metabolism are severely altered in traumatic hyper metabolism and wound healing. In addition to macronutrients and energy, specific amino acids are required for wound healing:

- Increased Urea Synthesis
 - Ornithine
- Direct Oxidative Energy Source
 - Branched chain amino acids (BCAA)

¹⁰⁴ Ibid

- White Blood Cell, Small Intestine Mucosal Cell Energy source and Collagen Synthesis
 - Glutamine.
- Nucleic Acid Synthesis
 - Zinc
 - Iron
 - Folic Acid
- Purine, Pyrimidine Synthesis
 - Folic Acid
 - Taurine
- Epithelial, Bone Marrow Cell Differentiation
 - Vitamin A
 - Vitamin D
- Protein Sparing Fuel, Prostaglandin Substrates
 - Fatty Acids
- Protein Metabolism
 - Vitamin B6
 - Taurine
- Wound Healing
 - Vitamin A
 - Ascorbic Acid
 - Vitamin B1
 - Zinc
- Collagen Synthesis
 - Thiamin
 - Ascorbic Acid
 - Copper
 - Iron
- Hemostasis, Clotting
 - Vitamin D
 - Calcium
 - Vitamin K
- Hemoglobin Synthesis, Erythropoiesis, Fluid, Electrolyte Balance
 - Protein
 - Ascorbic Acid
 - Copper
 - Iron
 - Zinc
 - Vitamin B12
- Protect Cells from Oxidative Damage
 - Vitamin A
 - Vitamin C
 - Vitamin D
 - Vitamin E

- Glutathione
- Selenium
- Magnesium
- Iron
- Folic Acid
- Riboflavin
- Niacin
- Promote Nitrogen Balance
 - Branched chain amino acids
 - Aromatic amino acids
 - Vitamin B₆
- Preserve Bone Mass
 - Calcium
 - Vitamin D
 - Potassium
- Hepatic Support
 - Iron
 - Folate
 - Vitamin B12
 - Riboflavin
 - Niacin
 - Vitamin B6
 - Vitamin D¹⁰⁵
- Preterm special need
 - Cysteine
 - Taurine
- Wound Healing
 - Pantothenic Acid¹⁰⁶
 - Thiamine¹⁰⁷
 - Vitamin A^{108 109}
 - Thiamin¹¹⁰
 - B Vitamin Complex¹¹¹
 - Vitamin C^{112 113 114}

¹⁰⁵ Ibid

¹⁰⁶ Aprahamian M, Dentinger A, Stock-Damge C, et al. *Effects of supplemental pantothenic acid on wound healing: experimental study in rabbit.* *Am J Clin Nutr* 1985;41:578–89.

¹⁰⁷ Alvarez OM, Gilbreath RL. *Effect of dietary thiamine on intermolecular collagen cross-linking during wound repair: a mechanical and biochemical assessment.* *J Trauma* 1982;22:20–4.

¹⁰⁸ Hunt TK. *Vitamin A and wound healing.* *J Am Acad Dermatol* 1986;15:817–21 [review].

¹⁰⁹ Hunt TK, Ehrlich HP, Garcia JA, et al. *Effect of vitamin A on reversing the inhibitory effect of cortisone on healing of open wounds in animals and man.* *Ann Surg* 1969;170:633–41.

¹¹⁰ http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=6120245&dopt=Abstract

¹¹¹ Bosse MD, Axelrod AE. *Wound healing in rats with biotin, pyridoxin, or riboflavin deficiencies.* *Proc Soc Exp Biol Med* 1948;67:418–21.

- Vitamin E¹¹⁵
- Copper¹¹⁶
- Manganese¹¹⁷
- Glucosamine and Chondroitin^{118, 119}
- Glutamine¹²⁰
- Zinc^{121, 122, 123}
- Arginine^{124, 125, 126, 127, 128}
- Essential Fatty Acids
- Beta Carotene
- Glutathione¹²⁹
- Bromelain^{130, 131}

¹¹² Levine M. *New concepts in the biology and biochemistry of ascorbic acid*. N Engl J Med 1986;314:892–902 [review].

¹¹³ Mazzotta MY. Nutrition and wound healing. *J Am Podiatr Med Assoc* 1994;84:456–62 [review].

¹¹⁴ Ringsdorf WM Jr, Cheraskin E. Vitamin C and human wound healing. *Oral Surg Oral Med Oral Pathol* 1982;53:231–6 [review].

¹¹⁵ Bartolomucci E. *Action of vitamin E on healing of experimental wounds on parenchymatous organs*. JAMA 1939;113:1079 [abstract].

¹¹⁶ Rucker RB, Kosonen T, Clegg MS, et al. Copper lysyl oxidase, and extra cellular matrix protein cross-linking. Am J Clin Nutr 1998;67(5 suppl):996s–1002s.

¹¹⁷ Leach RM. *Role of manganese in mucopolysaccharide metabolism*. Fed Proc 1971;30:991.

¹¹⁸ Morrison LM, Murata K. *Absorption, distribution, metabolism and excretion of acid mucopolysaccharides administered to animals and patients*. In: Morrison LM, Schjeide OA, Meyer K. Coronary heart disease and the mucopolysaccharides (glycosaminoglycans). Springfield: Charles C. Thomas, 1974, 109–27.

¹¹⁹ Prudden JF, Allen J. *The clinical acceleration of healing with a cartilage preparation; a controlled study*. *JAMA* 1965;192:352–6.

¹²⁰ Romito RA. *Early administration of enteral nutrients in critically ill patients*. AACN Clin Issues 1995;6:242–56.

¹²¹ Sandstead HH. *Understanding zinc: Recent observations and interpretations*. J Lab Clin Med 1994;124:322–7.

¹²² Liszewski RF. *The effect of zinc on wound healing: a collective review*. J Am Osteopath Assoc 1981;81:104–6 [review].

¹²³ Pories WJ, Henzel JH, Rob CG, Strain WH. *Acceleration of healing with zinc sulfate*. Ann Surg 1967;165:432–6.

¹²⁴ Barbul A, Rettura G, Levenson SM, et al. *Wound healing and thymotropic effects of arginine: a pituitary mechanism of action*. Am J Clin Nutr 1983;37:786–94.

¹²⁵ Kirk SJ, Hurson M, Regan MC, et al. *Arginine stimulates wound healing and immune function in elderly human beings*. Surgery 1993;114:155–60.

¹²⁶ Barbul A, Lazarou SA, Efron DT, et al. *Arginine enhances wound healing and lymphocyte immune responses in humans*. Surgery 1990;108:331–7.

¹²⁷ Barbul A, Rettura G, Levenson SM, et al. *Wound healing and thymotropic effects of arginine: a pituitary mechanism of action*. Am J Clin Nutr 1983;37:786–94.

¹²⁸ Kirk SJ, Hurson M, Regan MC, et al. *Arginine stimulates wound healing and immune function in elderly human beings*. Surgery 1993;114:155–60.

¹²⁹ Werbach, M, Nutritional Influences on Illness, Op. Cit., pp 615–618

¹³⁰ Blonstein J. *Control of swelling in boxing injuries*. Practitioner 1960;203:206.

¹³¹ Tassman G, Zafran J, Zayon G. *A double-blind crossover study of a plant proteolytic enzyme in oral surgery*. J Dent Med 1965;20:51–4.

- Ornithine alpha-ketoglutarate (OKG)^{132, 133}
- Carnosine¹³⁴
- Omega 3 Fatty Acids¹³⁵

Delayed wound healing represents a massive burden on the personal and social productivity as well as the costs of care and is predictably delayed or complicated by under nutrition, specific and general shortfalls in nutritional status and failure to treat the wounded with appropriate nutritional support based not on the nature of the wound, but the reality of the spot they occupy at that moment on the nutritional continuum.

Easy access to a wide variety of high quality nutrients along with easily available information on the nutritional support of wound healing is a very good bargain for any country and for its people.

¹³² De Bandt JP, Coudray-Lucas C, Lioret N, et al. *A randomized controlled trial of the influence of the mode of enteral ornithine alpha-ketoglutarate administration in burn patients.* *J Nutr* 1998;128:563–9.

¹³³ Cynober L. *Ornithine alpha-ketoglutarate in nutritional support.* *Nutrition* 1991;7:313–22 [review].

¹³⁴ Roberts PR, Black KW, Santamauro JT, Zaloga GP. *Dietary peptides improve wound healing following surgery.* *Nutrition* 1998;14:266–9.

¹³⁵ Daly JM, Lieberman MD, Goldfine J, et al. *Enteral nutrition with supplemental arginine, RNA, and omega-3 fatty acids in patients after operation: immunologic, metabolic, and clinical outcome.* *Surgery*. 1992; 112:56-67.

B. Nutrition, Blood Lipids and Blood Pressure

Cardiovascular disease (CVD) presents a major world health problem which can be positively influenced by nutritional approaches both in prevention and treatment. Prevention is obvious, but often ignored by those who develop CVD until they can see for themselves that their lives are threatened by the impact of their behavioral choices, that is, when they realize that they now have CVD. Since the medical impact of risk factor-enhancing behavior (e.g., smoking, unbalanced diet, lack of exercise, etc.) is delayed, sometimes for decades, behavioral change is often initiated long after cellular damage has occurred. In spite of this, the cardiovascular system is remarkably responsive even to late-stage nutritional strategies which therefore thus have a tremendous role to play in the prevention, mitigation and remediation of CVD.

WHO notes in the Global Strategy on Diet, Physical Activity and Health¹³⁶:

- CVD accounted for 16.7 million, or 29.2% of total global deaths according to World Health Report 2003.
- Around 80% of CVD deaths occurred in low and middle-income countries.
- By 2010, CVD will be the leading cause of death in developing countries.
- At least 20 million people survive heart attacks and strokes every year; many require continuing costly clinical care creating a vast and costly burden of continuing care.
- Heart disease has no geographic, gender or socio-economic boundaries

While nutritional deficiencies and imbalances are believed to pose another set of significant risk factors.¹³⁷ These nutritional deficiencies and imbalances include Vitamin C and other vitally important nutrients which are either low or undetectable in many CVD patients. It should be remembered that a biological marker which is low may actually be severely depleted since the norms for biological markers and nutrient levels were developed in a population which was ill nourished and ill.¹³⁸

WHO notes further that of that 16.7 million global CVD deaths per year, an estimated 16.7 million (29.2%) result from the various forms of cardiovascular disease (CVD) which are preventable by positive action on the major primary risk factors: unhealthy diet, physical inactivity, and smoking. More than 50% of the deaths and disability from heart disease and strokes, which together kill more than 12 million people each year, can be cut by a combination of simple, cost-effective national efforts and individual actions to reduce major risk factors such as high blood pressure, high cholesterol, obesity and smoking. And, once CVD is diagnosed, nutritional strategies can often reverse or cure many types of CVD.

CVD is no longer only a disease issue of the developed world: according to the WHO, some 80% of all CVD deaths worldwide took place in developing, low and middle-

¹³⁶ <http://www.who.int/dietphysicalactivity/publications/facts/cvd/en/>

¹³⁷ <http://www.thecureforheartdisease.com/owen/HeartCure.htm>

¹³⁸ See pp. 2-5 of this document for a discussion of this important issue.

income countries, while these countries also accounted for 86% of the global CVD disease burden while WHO estimates that by 2010, CVD will be the leading cause of death in developing countries.¹³⁹ In fact, WHO identifies CVD as “the major contributor to the burden of disease among the non communicable diseases....In the next two decades, the increasing burden of CVD will be borne mostly by developing countries”¹⁴⁰

CVD is subdivided into the following clinical pictures:

- Coronary (or ischemic) heart disease (heart attack)
- Cerebrovascular disease (stroke)
- Hypertension (high blood pressure)
- Heart failure
- Rheumatic heart disease

According to WHO, of the 16.7 million deaths from CVDs yearly:

- 7.2 (43%) million are due to ischemic heart disease
- 5.5 (32%) million are due to cerebrovascular disease
- 3.9 (23%) million are due to hypertensive and other heart conditions.¹⁴¹

In addition, at least 20 million people world-wide survive heart attacks and strokes every year. A significant proportion of these CVD patients require ongoing and costly clinical care, which puts a major burden on long-term care resources. This burden is costly in man power, money, lost productivity and other resources. CVD makes itself apparent through clinical manifestations primarily in the otherwise economically and socially productive mid-life years, undermining socioeconomic development, not only of affected individuals, but families, communities and nations. Members of lower socioeconomic groups generally have a greater prevalence of risk factors, diseases and mortality in developed countries and a similar pattern is emerging as the CVD epidemic matures in developing countries.¹⁴²

Thus, preventing and controlling the maturing non-communicable CVD epidemic is especially beneficial to nations whose resources must be devoted to their emergence into healthy, stable and powerful economies with a healthy and socially productive work force and community structure.

Primary Risk Factors:

WHO notes that while CVD is influenced by a host of risk factors,¹⁴³ the five strongest influences, representing the majority of the risk¹⁴⁴, are those which can be positively modified by dietary strategies:

¹³⁹ <http://www.who.int/dietphysicalactivity/publications/facts/cvd/en/>

¹⁴⁰ http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf p. 81

¹⁴¹ Ibid

¹⁴² Ibid

¹⁴³ Grundy et al. *Assessment of Cardiovascular Risk*, J Am Coll Cardiol 1999;34:1348--59

- **Hyperlipidemia**
- **Hypertension**
- Obesity
- Diabetes mellitus¹⁴⁵,
- **Sub optimal Vitamin C levels**^{146 147 148},

Items in boldface are currently believed to have the greatest impact on CVD morbidity and mortality, especially in combination.

WHO further states “In developing countries, the effect of the nutrition transition [to nutrient poor and processed food] and the concomitant rise in the prevalence of cardiovascular disease will be to widen the mismatch between healthcare needs and resources, and already scarce resources will be stretched even more thinly. Because unbalanced diets, obesity and physical inactivity all contribute to heart disease, addressing these, along with tobacco use, can help stem the epidemic.¹⁴⁹

Although they are independent risk factors, obesity and diabetes mellitus are usually associated with elevated blood lipids and blood pressure thus compounding their dangers. While successful weight loss and blood glucose control alone reduce CVD risk, to achieve the most meaningful results, blood lipids and blood pressure and nutritional status must be controlled along with them.

Blood Lipids: Risk Factors for Morbidity and Mortality

Serum Markers for Risk Factors Predictive of CVD

↑ Total Cholesterol	↑ LDL Cholesterol	↑ Tri-Glycerides	↑ CRP
↓ HDL Cholesterol	↑ LDL/HDL Ratio	↓ Vitamin C	↓ Nutrient Status

Dietary modifications that reduce total and LDL-cholesterol must not also decrease HDL cholesterol or contribute to elevated triglyceride levels. Oxidation of LDL-cholesterol is a key step in the formation of fatty streaks, the initial lesions of atherosclerosis. LDL can be protected from oxidative stress by reduction of sources of free radicals that come into contact with it and by nutritional enhancement of free radical scavengers. Absolute reduction of LDL levels also reduces the potential amount of oxidized LDL that may be formed regardless of the level of oxidative stress.

Compensatory Markers of the Chronic Degenerative Changes Associated with CVD:

¹⁴⁴ Lowe, L, Greenland, P, Ruth, K, Dyer, A, Stamler, R, Stamler, J, *Impact of Major Cardiovascular Disease Risk Factors, Particularly in Combination, on 22-Year Mortality in Women and Men*, *Arch Intern Med.* 1998;158:2007-2014.

¹⁴⁵ <http://www.who.int/dietphysicalactivity/publications/facts/cvd/en/>

¹⁴⁶ <http://www.internetwks.com/owen/Synopsis.pdf>

¹⁴⁷ <http://www.nutraingredients.com/news/news-ng.asp?n=56749-high-dose-vitamin>

¹⁴⁸ <http://www.thecureforheartdisease.com/owen/suppress.htm>

¹⁴⁹ http://www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf, p. 81

Blood Pressure: Hypertension increases risk of both coronary heart disease and stroke. Elevations in either systolic or diastolic pressure are associated with increased CVD risk. The age-related increase in blood pressure typically observed may be minimized or delayed by dietary modifications earlier in life that normalize and sustain normal blood pressure and by nutritional interventions later in life either before or after hypertension has become clinically established.

Diabetes Mellitus: Diabetes mellitus increases risk of CVD independent of weight, blood lipids and blood pressure. Both Type I diabetics (5-10% of total prevalence) and Type II diabetics (90-95% of total prevalence) experience increased risk of cardiovascular complications. Type II diabetes is often accompanied by obesity (especially metabolic fat), hyperlipidemia and hypertension. Cellular insulin resistance is the common link between each of these risk factors. The relationship between insulin and increased CVD risk is probably related to its vasoactive and proliferative effects. Nevertheless, two prospective studies have recently confirmed that poor blood glucose control can contribute to vascular complications of diabetes with or without accompanying hyperinsulinemia.^{150 151}

Obesity: Although obesity is not as strongly associated with risk of CVD as hyperlipidemia or hypertension, it may have a greater impact on morbidity and mortality because the prevalence is higher. Obesity among children is increasing at a much greater rate than among adults. Obesity is linked to consumption of high fat foods/high energy “food” in the face of reduced energy output including abandoning free play by children in favor of passive activities like television and computer games. Inappropriate intake of sweetened, nutrient empty drinks and food substitutes like candy and pastries with their low nutrient density and high energy input is contributory to the growing epidemic of overweight and obesity.

The relationship is so common between obesity and diabetes that some health experts have begun to refer to the constellation as “diabesity”^{152 153 154 155}

Nutritional Status: Suboptimal serum Vitamin C levels, although the subject of intense controversy has a long-established history of association with CVD beginning with the

¹⁵⁰Diabetes Control and Complications Trial, 1995

<http://diabetes.niddk.nih.gov/dm/pubs/control/#what%20is>

¹⁵¹United Kingdom Prospective Diabetes Study, 1998

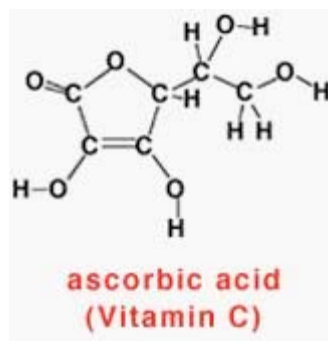
<http://www.dtu.ox.ac.uk/index.html?maindoc=ukpds/>

¹⁵²<http://www.eurodiabesity.org/>

¹⁵³<http://www.shapeup.org/diabesity.html>

¹⁵⁴<http://www.annecollins.com/obesity/diabetes-diabesity.htm>

¹⁵⁵<http://www.medicineau.net.au/clinical/diabetes/diabete2262.html>



lectures and teaching of Nobel Laureate Linus Pauling.¹⁵⁶ Dr Pauling, his associates and followers postulate that CVD cannot develop in the presence of optimal levels of Vitamin C and state that amount of Vitamin C manufactured in **stress free** primates of our size would be between 8 and 10 grams per day¹⁵⁷. Stress of all types (including nutritional insufficiency) is well known to increase the need for one of the most potent antioxidants known: Vitamin C. For example:

- People who supplement with more than 700 mg/day of vitamin C have a 62 per cent lower risk of dying from heart disease than do people with a daily intake of 60 mg/day or less.¹⁵⁸
- Supplementation with 2 g/day of vitamin C has been found to reduce adhesion of monocytes (white blood cells) to the lining of blood vessels and thereby reduce the risk of atherosclerosis.^{159, 160, 161}
- Harvard medical researchers found that vitamin C was the only one of 880 substances tested that caused heart muscle cells to regenerate from stem cells.¹⁶²
- Vitamin C supplementation (2 g/day) also effectively reverses the vasomotor dysfunction often found in patients with atherosclerosis¹⁶³
- Research carried out in Japan has shown that restenosis (re-closing of opened arteries) after angioplasty can be significantly reduced by supplementing with ascorbic acid (500 mg/day). When appropriate Vitamin C is available, cholesterol is not oxidized to form LDL-cholesterol and fatty streaks and other types of damage primate and, indeed, the experience of physicians using high dose

¹⁵⁶ <http://www.lbl.gov/Science-Articles/Archive/pauling-and-vitamin-c.html>

¹⁵⁷ <http://www.lbl.gov/Science-Articles/Archive/pauling-and-vitamin-c.html>

¹⁵⁸ Sahyoun, Nadine R., et al. *Carotenoids, vitamins C and E, and mortality in an elderly population.* *American Journal of Epidemiology*, Vol. 144, No. 5, September 1, 1996, pp. 501-11

¹⁵⁹ Weber, Christian, et al. *Increased adhesiveness of isolated monocytes to endothelium is prevented by vitamin C intake in smokers.* *Circulation*, Vol. 93, No. 8, April 15, 1996, pp. 1488-92

¹⁶⁰ Lehr, Hans-Anton, et al. *Protection from oxidized LDL-induced leukocyte adhesion to microvascular and macrovascular endothelium in vivo by vitamin C but not by vitamin E.* *Circulation*, Vol. 91, No. 5, March 1, 1995, pp. 1525-32

¹⁶¹ Heitzer, Thomas, et al. *Antioxidant vitamin C improves endothelial dysfunction in chronic smokers.* *Circulation*, Vol. 94, No. 1, July 1, 1996, pp. 6-9

¹⁶² <http://www.sciencedaily.com/releases/2003/04/030401073122.htm>

¹⁶³ Levine, Glenn N., et al. *Ascorbic acid reverses endothelial vasomotor dysfunction in patients with coronary artery disease.* *Circulation*, Vol. 93, No. 6, March 15, 1996, pp. 1107-13

- Vitamin C both orally and intravenously with CVD patients supports this hypothesis.¹⁶⁴
- Further evidence of the importance of supplementing with Vitamin C in CVD is shown by studies which show that Vitamin C lowers both blood pressure and cholesterol levels, helps thin the blood and protect it against oxidation and works in close synergism with vitamin E.^{165 166 167 168 169 170 171 172}
- Vitamin C also helps prevent atherosclerosis by strengthening the artery walls through its participation in the synthesis of collagen, and by preventing the undesirable adhesion of white blood cells to damaged arteries.^{173 174 175}
- An adequate intake of vitamin C is highly protective against stroke and heart attack.^{176 177 178}
- Regular doses of Vitamin C are necessary to support the positive impact on cardiovascular factors. In 1982 researchers at the Institute of Preventive and Clinical Medicine in the Slovak Republic, advised that "In every form of high cholesterol therapy, an adequate vitamin C supply should be ensured in doses capable of creating maximal steady-state levels of ascorbate in human tissues."¹⁷⁹
- Oxidation of cholesterol particles (LDL and lipoprotein (a)) increases the risk of arterial disease by 14 times.¹⁸⁰
- Vitamin C, in doses easily obtained through oral supplementation, however, inhibits 75% of that oxidation.¹⁸¹

¹⁶⁴ http://www.newmediaexplorer.org/sepp/2003/11/09/vitamin_c_beats_statins_in_cholesterol_heart_disease.htm

¹⁶⁵ Rath, Matthias. *Eradicating Heart Disease*. Health Now, San Francisco, CA, 1993

¹⁶⁶ Whitaker, Julian M. *Reversing Heart Disease*. Warner Books, NY, 1985

¹⁶⁷ Trout, David L. *Vitamin C and cardiovascular risk factors*. *American Journal of Clinical Nutrition*, Vol. 53, January 1991, pp. 322S-25S

¹⁶⁸ *Annals N Y Academy Science* 258:410–21, 1975; *Atherosclerosis*. 24:1–18, 1976; *Experientia* 32:687–9, 1976

¹⁶⁹ Moran, John P., et al. *Plasma ascorbic acid concentrations relate inversely to blood pressure in human subjects*. *American Journal of Clinical Nutrition*, Vol. 57, March 1993, pp. 213-17

¹⁷⁰ Johnston, Carol S., et al. *Vitamin C elevates red blood cell glutathione in healthy adults*. *American Journal of Clinical Nutrition*, Vol. 58, August 1993, pp. 103-05

¹⁷¹ Kagan, Valerian E., et al. *Recycling of vitamin E in human low density lipoproteins*. *Journal of Lipid Research*, Vol. 33, March 1992, pp. 385-97

¹⁷² Kontush, Anatol. et al. *Antioxidant and prooxidant activity of alpha-tocopherol in human plasma and low density lipoprotein*. *Journal of Lipid Research*, Vol. 37, No. 7, July 1996, pp. 1436-48

¹⁷³ Rath, Matthias. *Eradicating Heart Disease*. Health Now, San Francisco, CA, 1993

¹⁷⁴ Weber, Christian, et al. Op. Cit.

¹⁷⁵ Lehr, Hans-Anton, et al. *Protection from oxidized LDL-induced leukocyte adhesion to microvascular and macrovascular endothelium in vivo by vitamin C but not by vitamin E*. *Circulation*, Vol. 91, No. 5, March 1, 1995, pp. 1525-32

¹⁷⁶ Gale, Catherine R., et al. *Vitamin C and risk of death from stroke and coronary heart disease in cohort of elderly people*. *British Medical Journal*, Vol. 310, June 17, 1995, pp. 1563-66

¹⁷⁷ Woodhouse, P.R. and Khaw, Kay-Tee. *Seasonal variations in vitamin C status, infection, fibrinogen and cardiovascular disease - Are they linked?* *Age and Ageing*, Vol. 23, Supplement No. 2, 1994, p. P5

¹⁷⁸ Sahyoun, Nadine R., et al. Op. Cit.

¹⁷⁹ *J Lipid Research* 24:1186–95, 1983

¹⁸⁰ Tsimikas S., Brilakis E. S., Miller E. R., McConnell J. P., Lennon R. J., Kornman K. S., Witztum J. L., Berger P. B. *Oxidized Phospholipids, Lp(a) Lipoprotein, and Coronary Artery Disease*. *N Engl J Med* 2005; 353:46-57, Jul 7, 2005.

This well-documented fact alone could reduce the burden of CVD significantly. Vitamin C is a safe, non-toxic and highly useful nutrient public health at little expense if adequate doses of Vitamin C are achieved population-wide.

Unstable arterial plaque is associated with more than a half-million sudden-death heart attacks that occur annually, mostly in males with normal or low circulating cholesterol levels.¹⁸² This strongly suggests that sudden normo-cholesterol heart attack risk can be sharply reduced with this simple, and remarkably inexpensive, dietary strategy.

Arterial disease is initiated by activation of the peroxisome proliferators-activated receptors (PPARs). Sub-optimal Vitamin C "severely compromises collagen deposition and induces a type of plaque morphology that is potentially vulnerable to rupture."¹⁸³ The mechanism by which Vitamin C controls and eliminates this genetic initiation of intimal inflammation is identical to the mechanism of the statins. Vitamin C is as effective as the statins at controlling this inflammatory reaction: "[this research] provides incontrovertible evidence to support the view that both statins and vitamin C have identical effects on the expression of genes coding for PPARs" at levels "*well within the permissible dose of this vitamin*."¹⁸⁴

The question, of course, is what is an adequate or optimal supply of this particular (or any) nutrient and what would be classified as "within the permissible dose" of a substance with many-fold variation in requirements for optimal dosing and without a meaningful toxic profile? Appropriate doses must be determined on a clinical, not an administrative or epidemiological, basis.

In addition to Vitamin C, briefly considered here for the purposes of illustration are a host of vitally important nutrients which must be repleted in order to stop the progression, or even reverse CVD. Since the amounts of food which would provide high dose nutrients is impractical and unhealthy for consumers to eat in a day, the only practical alternative is to use nutrient supplementation both under the guidance of professionals and by free consumer choice.

Secondary Risk Factors

A number of less familiar risk factors for CVD may also be amenable to control by dietary means. Of these, the strongest evidence supports a role for elevated plasma homocysteine levels and alterations in levels of blood coagulation factors through nutritional strategies.

Homocysteine: Hyperhomocysteinemia (elevated blood homocysteine) is recognized as an independent risk factor for coronary heart disease, cerebrovascular disease, and peripheral vascular disease. At moderate elevations of only 12% above the upper limits of normal, homocysteine has been prospectively associated with a three-fold risk in acute

¹⁸¹ Chu YF, Liu RH, *Novel Low-Density Lipoprotein (LDL) Oxidation Model: Antioxidant Capacity for the Inhibition of LDL Oxidation*. *J Agric Food Chem* 2004, 52: 6818-6823

¹⁸² *Circulation* 105: 1485-90, 2002

¹⁸³ *European Journal Clinical Nutrition* 59: 978-81, 2005

¹⁸⁴ *Ibid*

myocardial infarction in men. Elevated homocysteine predisposes to arteriosclerosis and stroke. In fact, in 47% of patients with arterial occlusion, a moderate elevation of homocysteine was found.¹⁸⁵ Homocysteine is an intermediate on the pathway to methionine. If it is not methylated in the presence of adequate Folate, using either the B6 or the Folate and B12 dependant pathways, (and B2 in those with a genetic mutation in the MTHFR gene), it is instead oxidized to homocysteic or cysteic acids. It is believed that the dangerous impact of homocysteine is, in fact, the result of the oxidative pathway of homocysteine to homocysteic acid which occurs when insufficient Folate and B12 are available.¹⁸⁶

Homocysteine metabolism is accomplished via one of two known pathways in humans: one is dependent upon the presence of adequate B6 and the other upon adequate levels of Vitamin B12 and Folate. Insufficient effective levels of B6, B12 and Folate (which may be the result of dietary, genetic and/or pathological conditions) lead to the accumulation of homocysteine in the blood with toxic impact upon brain, bone and vascular systems. Blocks in either of these pathways due to inadequate amounts of these or other vitamins can produce hyperhomocysteinemia. Other causes of this condition include inherited disorder of metabolism and impaired renal function. At elevated levels, homocysteine stimulates proliferation of smooth muscle cells and inhibits proliferation of endothelial cells by a mechanism that is not well understood.

Blood coagulation factors: Atherosclerosis promotes endothelial injury which initiates platelet adhesion, aggregation and formation of a thrombus. Hemodynamic alterations associated with turbulent blood flow or hypertension may be involved in thrombogenesis associated with endothelial injury. Dietary factors are involved at several points in the coagulation cascade as cofactors for prothrombin and thrombin formation. High fat intakes resulting in dangerous postprandial lipemia may enhance thrombogenic activity through association with increased levels of Factor VII. Dietary fat composition may influence platelet aggregation through synthesis of prostanoids either favoring blood clot production or retarding it. Dietary fiber may have an antithrombogenic effect as levels of intake have been inversely associated with levels of plasminogen activator inhibitor-type, possibly mediated by effects of fiber on insulin.

Dietary enzymes may reduce the predisposition toward thrombogenesis and are an important preventive strategy as well as a treatment option should a clot form in, or migrate to, the brain. Enzymes like nattokinase and lumbrokinase have profound impact upon clot formation and, with sustained regular use, safely digest extra fibrin, lessening clot formation and promoting good circulation while reducing clot formation.^{187, 188, 189, 190, 191, 192}

¹⁸⁵ <http://bioline.utoronto.ca/archive/00002649/01/ni04107.pdf> p. 312

¹⁸⁶ <http://bioline.utoronto.ca/archive/00002649/01/ni04107.pdf> p. 311

¹⁸⁷ National Vital Statistics Report, Vol. 50, No. 16, September 16, 2002.

¹⁸⁸ Sumi, H., Hamada, H., Nakanishi, K., Hiratani, H. *Enhancement of the fibrinolytic activity in plasma by oral administration of nattokinase*. *Acta Haematol* 1990; 84(3):139-43.

¹⁸⁹ Sumi, H., Hamada, H., Mihara, H. *A novel strong fibrinolytic enzyme (nattokinase) in the vegetable cheese "natto"*. *International Journal of Fibrinolysis and Thrombolysis*. Abstracts of the Ninth International Congress on Fibrinolysis, Amsterdam, 1988 Vol. 2, Sup. 1:67

¹⁹⁰ Sumi, H., Hamada, H., Tsushima, H., Mihara, H., Muraki, H. *A novel fibrinolytic enzyme (nattokinase) in the vegetable cheese Natto; a typical and popular soybean food in the Japanese diet*. *Experientia* 1987, Oct 15;43(10):1110-1

Diet and the Impact of Diet on Blood Lipid Levels: Control of blood lipid levels by dietary modifications is the first step in prevention and treatment of hyperlipidemia in both adults and children. It is crucial to the immediate and long term health of a population to improve the lipid profile by natural, effective, non-toxic and inexpensive methods which will allow low-to-absent side effect levels, provide workable long term solutions for prevention and attractive options to patients with already established disease. Medications for these elevations and ratio distortions are toxic, expensive and have a serious side effect profile which nutritional interventions lack.¹⁹³

According to the National Guidelines Clearinghouse of the US government, findings of the Institute of Medicine support the use of nutritional treatment for the reduction of blood lipids and state in their Guideline on CVD and nutrition, “**Benefits of Nutrition Management to Patients**”,

- “The benefits of nutritional screening and intervention in patients at risk for or with established coronary heart disease (CHD) are considerable. The extent to which serum cholesterol declines depends on the extent to which dietary modification is instituted and maintained. Improvements in CHD mortality are also "dose-dependent". Statistically significant reductions in cardiac mortality ranging from 32 to 66% have been demonstrated in a number of trials of dietary fat restriction in CHD. Unlike trials of medications used to treat CHD, dietary trials have yielded no evidence of an excess of all-cause mortality.
- Serum cholesterol levels can be effectively lowered by dietary and lifestyle modification. Recommendations regarding increased consumption of foods rich in the B-complex vitamins and Vitamin E are reasonable and will result in a diet that is palatable and compatible with good general health and may reduce cardiovascular risk.

Benefits of Nutrition Management to Health Services Providers

- Total blood cholesterol level is conclusively linked to the development of CHD. Most of this risk is associated with low-density lipoprotein (LDL) cholesterol concentrations. A 1-mg/dl reduction in LDL cholesterol levels results in an approximate 1 to 2% reduction in relative risk of CHD.
- Virtually all lipid modification trials, including all trials involving pharmacologic agents, have utilized dietary counseling and dietary modification as cornerstones of therapy. With approximately 3 million first-time coronary events estimated to occur over a 10-year period in individuals with total cholesterol levels exceeding 200 mg/dl (5.17 mmol/L), reducing saturated fat intake by 1 to 3% would reduce the incidence of CHD by 32,000

¹⁹¹ [Ryu GH et al. Antithrombogenicity of lumbrokinase-immobilized polyurethane. J Biomed Mater Res. 1994 Sep;28\(9\):1069-77](#)

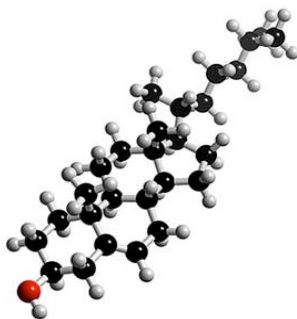
¹⁹² [Park Y, Ryu E, Kim H, Jeong J, Kim J, Shim J, Jeon S, Jo Y, Kim W, Min B. Characterization of Antithrombotic Activity of Lumbrokinase-Immobilized Polyurethane Valves in the Total Artificial Heart. Artificial Organs, Volume 23, Number 2, February 1999, pp. 210-214\(5\)](#)

¹⁹³ http://www.guideline.gov/summary/summary.aspx?ss=15&doc_id=4225&nbr=3230#s23

- to 99,700 events. This would yield combined cost savings in medical expenditures and lost earnings ranging from \$4.1 to 12.7 billion over the next 10 years.
- Failure to adequately control LDL cholesterol levels with diet alone usually results in the prescription of one or more antilipemic drugs to reduce CHD risk. Costs of treatment can easily run \$1,000 to 2,000/drug annually. Such costs can be significant to older persons living on fixed incomes and to the health care system as a whole. Failure to emphasize the importance of diet as primary or adjunctive therapy in the management of CHD frequently results in the need to use larger drug doses or drugs in combination. Either of these alternatives contributes to increased medical costs and to the increased risk of side effects and adverse drug interactions.¹⁹⁴

The same source notes that only downside to the use of nutrients as a treatment for CVD is that the frail elderly must be monitored carefully against loss of weight.¹⁹⁵

Serum Cholesterol: Saturated fat (SF) intake is generally considered to be a strong predictor of elevated LDL-cholesterol. However, as we have seen, this relationship only obtains when relevant nutrients are in short supply and carbohydrates have not been prepared in a wholesome fashion.



Cholesterol Molecule

Consideration of the hazards and necessary precautions which must be taken when SF is consumed are, it should be remembered, not applicable when the SF is from healthy and uncontaminated sources. While eating SF from healthy, free range animals untreated with chemicals, veterinary drugs or contaminated feed, is a not a risk factor, it is true that SF from sick, toxic and stressed animals (unhealthy saturated fat or “USatFat”) in the context of a poor nutritional environment raises LDL-cholesterol by decreasing expression and functional activity of hepatic LDL receptors. Polyunsaturated fat (PUFA) lowers LDL-cholesterol, but at twice the level of intake that it takes for USatFat to raise it

¹⁹⁴ Ibid

¹⁹⁵ Ibid

by the same increment. When consumed in high amounts (>12% of total energy), PUFAs will lower also lower HDL-cholesterol however if ω -6 fatty acids (linoleic and arachidonic acids) are the predominant PUFAs consumed, suppression of immune function may occur. It is very important for cardiovascular, immune and brain function that the PUFAs consumed have a beneficial ratio of ω -3, 6 and 9 moieties. Monounsaturated fat (MUFA) lowers total and LDL cholesterol as effectively as PUFAs, but in contrast to PUFAs, will not unfavorably alter HDL-cholesterol levels. Thus substitution of a proportion of the unsaturated fat component of total fat with MUFA (10% of total energy) will promote an optimal ratio of total to HDL-cholesterol.

Total fat intake may also contribute to increased LDL-cholesterol levels when large amounts are consumed (> 30% of total energy). At high levels of total fat intake, it is more difficult to keep healthy at optimal levels (< 8 - 10% of energy) or to maintain an energy intake compatible with weight control. High intake of dietary cholesterol and total fat also result in elevated chylomicrons which are produced in order to facilitate absorption. When triglycerides are released from these chylomicron particles, the cholesterol-rich remnants have atherogenic effects similar to LDL-cholesterol itself. The life span and composition, as well as the size of chylomicrons vary with the composition and nature of ingested fats with the most favorable profile being derived from coconut oil and medium chain triglyceride diets. Thus, dietary fat type and amount have a profound impact on the dynamic metabolism of lipids in the blood and the composition of those lipids.¹⁹⁶

Not only is the composition of the fats consumed significant for cardiovascular and immune health, the ratio of PUFAs to USatFat is also of great importance to heart health. LDL-cholesterol levels may be raised by dietary cholesterol but only if the ratio of PUFAs to USatFats is low (< 1). Adequate intake of PUFAs relative to USatFats can lower LDL-cholesterol while inadequate levels promote the oxidation of cholesterol to form LDL-cholesterol.

At intakes higher than 400 mg, dietary cholesterol inhibits hepatic cholesterol synthesis by negative feedback control. However when large amounts of USatFats are consumed with significant amounts of cholesterol (< 400 mg), this feedback control is overwhelmed and cannot by itself effectively reduce plasma cholesterol levels. The effect, however, is more complex: several theories have been proposed to explain the synergy between saturated fat and dietary cholesterol which elevates LDL-cholesterol levels by more than can be explained by an additive effect. These theories focus on mechanisms involving down regulation of hepatic cholesterol receptors and/or increased activity of cholesterol ester transfer protein.

In addition to dietary fat, total energy, simple carbohydrates [prepared in familiar Western ways, rather than those which promote healthy carbohydrate utilization], fructose, and alcohol intake may also affect blood lipid levels. Elevated LDL-cholesterol levels have been found in individuals consuming low fat diets when the fat component

¹⁹⁶ E. Levy, C. C. Roy, R. Goldstein, H. Bar-On and E. Ziv, *Metabolic fate of chylomicrons obtained from rats maintained on diets varying in fatty acid composition*, Journal of the American College of Nutrition, Vol 10, Issue 1 69-78

has been replaced by carbohydrates consisting mostly of simple sugars from low fiber sources. If these individuals also have elevated triglycerides in response to this dietary pattern, they are characterized as “carbohydrate-sensitive”. Carbohydrate sensitivity is seen almost exclusively with high intakes of fructose or sucrose (fructose + glucose), especially when consumed in liquid form. The mechanism for this effect is unknown but may relate to metabolism of fructose to glyceraldehyde-3-phosphate which is involved in endogenous triglyceride synthesis. However, de novo lipogenesis accounts for only a small amount of hepatic VLDL production and the increase in VLDL synthesis is not accompanied by decreased LDL in carbohydrate sensitive individuals. The greatest

sources for many people for their intake of fructose are soft drinks, candy, and desserts secondary to increased industrial usage of high fructose corn syrup as a sweetening agent.

It is interesting to note that fructose was forbidden as a sweetener in these items because of its deleterious impact until the soft drink industry forced the US FDA to change their regulation to allow it since it was permitted under the relevant Codex standard.¹⁹⁷

In contrast to total and LDL-cholesterol, HDL-cholesterol is less responsive to dietary modifications. Physical activity and alcohol have a substantial impact on raising HDL-cholesterol. Alcohol may also increase triglyceride levels which are generally inversely related to HDL. Thus consumption of large amounts of alcohol would not be an effective means for raising HDL in individuals with low HDL levels accompanied by elevated triglyceride levels. In fact, the neurotoxic, hepatotoxic, nutrient depleting and caloric impact of alcohol makes it clear that it is not a cardiovascular health promoting agent.

Consumption of garlic extracts and allium vegetables (onions, garlic, leeks, etc.) has been shown to lower LDL-cholesterol effectively while raising HDL, probably due not only to factors intrinsic in these vegetables but also to their high content of manganese, Vitamin C, Iron, Folate and Vitamin B6.¹⁹⁸

Cranberries contain polyphenols which inhibit the oxidation of LDL cholesterol. According to a recent study, 100 grams (100,000 mg) of cranberries are equivalent to 1000 mg of vitamin C or 3700 milligrams of vitamin E in countering LDL cholesterol oxidation.¹⁹⁹ Fresh cranberries typically provide 0.3% polyphenols, while cranberry extracts typically provide 7.0% polyphenols. A new type of concentrated cranberry extract (CRAN-X) yields 30 percent polyphenols, making it at least as good at inhibiting LDL cholesterol oxidation as an equal amount of vitamin C. In addition, cranberries have potent anti-adhesion factors that help prevent bacteria and cholesterol from sticking to artery walls.²⁰⁰

¹⁹⁷ Personal communication with Jim Turner, Citizens for Health, 2005

¹⁹⁸ <http://www.happyherbalist.com/garlic1.htm>

¹⁹⁹ http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15982671&dopt=Citation

²⁰⁰ Phytochemistry July 28, 2005; Critical Review Food Science Nutrition 42: 301–16, 2002

Serum Triglycerides: Alcohol and simple sugars consumed from low fiber sources can elevate serum triglycerides. Conversely, this cardiovascular risk factor is highly responsive to dietary strategies which include supplementation with

- Fish Oil²⁰¹
- Inositol^{202, 203, 204}
- Green Tea (4 cups or equivalent)²⁰⁵
- Choline^{206, 207}
- Niacin (vitamin B3)^{208, 209}
- Pantethine^{210, 211, 212}
- Chromium^{213, 214, 215, 216, 217}

²⁰¹ Prichard BN, Smith CCT, Ling KLE, Betteridge DJ. *Fish oils and cardiovascular disease*. BMJ 1995;310:819–20 [editorial/review].

²⁰² Brown WV. *Niacin for lipid disorders*. Postgrad Med 1995;98:183–93 [review]

²⁰³ Head KA. *Inositol hexaniacinate: a safer alternative to niacin*. Altern Med Rev 1996;1:176–84 [review]

²⁰⁴ Murray M. *Lipid-lowering drugs vs. inositol hexaniacinate*. Am J Natural Med 1995;2(8):9–12 [review]

²⁰⁵ Imai K, Nakachi K. *Cross sectional study of effects of drinking green tea on cardiovascular and liver diseases*. BMJ 1995;310:693–6.

²⁰⁶ Buchman, Alan L, Ament, Marvin E, Sohel, Mir, Dubin, Mark, Et al, *Choline deficiency causes reversible hepatic abnormalities in patients receiving parenteral nutrition: Proof of a human choline requirement: A placebo-controlled trial*, JPEN, Sep/Oct 2001

²⁰⁷ Watkins SM, Zhu X, Zeisel SH., *Phosphatidylethanolamine-N-methyltransferase activity and dietary choline regulate liver-plasma lipid flux and essential fatty acid metabolism in mice*. J Nutr. 2003 Nov;133(11):3386-9

²⁰⁸ Berra K. , *Clinical update on the use of niacin for the treatment of dyslipidemia*. J Am Acad Nurse Pract. 2004 Dec;16(12):526-34.

²⁰⁹ Shepherd J, Betteridge J, Van Gaal L; European Consensus Panel, *Nicotinic acid in the management of dyslipidaemia associated with diabetes and metabolic syndrome: a position paper developed by a European Consensus Panel*. Curr Med Res Opin. 2005 May;21(5):665-82.

²¹⁰ Arsenio L, Bodria P, Magnati G, et al. *Effectiveness of long-term treatment with pantethine in patients with dyslipidemia*. Clin Ther 1986;8:537–45.

²¹¹ Avogaro P, Bon GB, Fusello M. *Effect of pantethine on lipids, lipoproteins and apolipoproteins in man*. Curr Ther Res 1983;33:488–93.

²¹² Maggi GC, Donati C, Criscuoli G. *Pantethine: a physiological lipomodulating agent, in the treatment of hyperlipidemias*. Curr Ther Res 1982;32:380–6.

²¹³ Uusitupa MI, Kumpulainen JT, Voutilainen E, et al. *Effect of inorganic chromium supplementation on glucose tolerance, insulin response, and serum lipids in noninsulin-dependent diabetics*. Am J Clin Nutr 1983;38:404–10

²¹⁴ Hermann J, Chung H, Arquitt A, et al. *Effects of chromium or copper supplementation on plasma lipids, plasma glucose and serum insulin in adults over age fifty*. J Nutr Elderly 1998;18:27–45.

²¹⁵ Lee NA, Reasner CA. *Beneficial effect of chromium supplementation on serum triglyceride levels in NIDDM*. Diabetes Care 1994;17:1449–52.

- Fructo Oligosaccharides/Inulin^{218, 219, 220, 221}
- Calcium²²²
- Vitamin E^{223, 224, 225, 226}
- Vitamins B6 and B12
- Vitamin C^{227, 228}
- Vitamin E^{229, 230}
- Linoleic Acid²³¹
- Vitamin Blueberry Leaf Extract (Hydroxy Cinnamic Acid/Chlorogenic Acid)²³²

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²¹⁷ Thomas VL, Gropper SS. *Effect of chromium nicotinic acid supplementation on selected cardiovascular disease risk factors.* Biol Trace Elem Res 1996;55:297–305.

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²¹⁹ Jackson KG, Taylor GRJ, Clohessy AM, Williams CM. *The effect of the daily intake of inulin on fasting lipid, insulin and glucose concentrations in middle-aged men and women.* Br J Nutr 1999;82:23–30

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²²³ Diabetes Care 1994;17:1449–52

²²⁴ Postgrad Med 1995;98:183–93 [review].

²²⁵ Lipids 1972;7:202–6.

²²⁶ J Nutr 1991;121:165–9

²²⁷ Fidanza A, Audisio M, Mastroiacovo P. 1982. Vitamin C and Cholesterol, in Vitamin C: New Clinical Applications in Immunology, Lipid Metabolism, and Cancer, ed. A. Hanck. Hans Huber, Bern, pp.153–171.

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²³¹ Ibid

²³² Abidoff M. 1999a. *Evaluation of glucose lowering properties of blueberry leaves extract. Part 1: Double blind placebo controlled study at the Russian Center for Modern Medicine. Part 2. Effect of*

Seasonal Cholesterol Variation: Seasonal fluctuations observed in blood cholesterol levels have been attributed to changes in vitamin C intake. The controlling enzyme of bile acid synthesis (cholesterol- β -hydroxylase) is dependent on vitamin C to provide antioxidant protection for the iron moiety at its catalytic site.

Postprandial Hyperlipemia: The period following ingestion of a meal containing fat is a time of active lipid and lipoprotein metabolism. In general, plasma triglycerides peak 3 hours following a meal and return to fasting within 9-12 hours. Although postprandial lipemia typically extends over a 9-12 hour period, its duration can be modified by the total amount of fat and dietary fiber consumed. The major effect of postprandial lipemia on fasting lipids is observed in the concentration and composition of the lipoprotein fractions. The duration of postprandial lipemia is believed to be associated with eventual development of fasting hyperlipemia and insulin resistance. Hyperinsulinemia may result from sustained high levels of fatty acids in the portal circulations which decrease hepatic insulin clearance.

Effects of Diet on Regulation of Hypertension: Blood pressure is a function of cardiac output and total peripheral resistance. Control of blood pressure is achieved over the short term by sympathetic nervous system activity and over the long term by renal mechanisms involving control of urinary sodium excretion. Dietary factors can influence blood pressure through either direct effect on plasma volume and vasoactivity or by interfering with sympathetic and renal control of these parameters. Dietary contributions to the control of blood pressure include:

- Folate^{233 234 235 236}
- Calcium^{237 238}
- Potassium^{239 240}

blueberry leaves extract on plasma glucose level in Type II diabetics. February 1999 - December 1999.

<http://www.enhansulin.com/research.htm>

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²³⁴ Qureshi M; Khsandwala H; Haq IU; Prasad K. Elevated levels of plasma homocysteine in hypertensive patients with diabetes mellitus. *J Cardiovasc Pharmacol Ther* 2003 Dec;8(4):261-6.

²³⁵ John P. Forman, MD; Eric B. Rimm, ScD; Meir J. Stampfer, MD, DrPH; Gary C. Curhan, MD, ScD. Folate Intake and the Risk of Incident Hypertension Among US Women. *JAMA*. 2005;293:320-329.

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²³⁷ Y Kageyama, H Suzuki, K Arima and T Saruta, Oral calcium treatment lowers blood pressure in renovascular hypertensive rats by suppressing the renin-angiotensin system, *Calcium Hypertension*, Vol 10, 375-382

²³⁸ Henry HJ et al. Increasing calcium lowers blood pressure: The literature reviewed. *J Am Diet Assoc* 85: 182-5, 1985

²³⁹ Geleijnse JM; Kok FJ; Grobbee D. E. , Blood pressure response to changes in sodium and potassium intake: a meta-regression analysis of randomised trials. *J Hum Hypertens* 2003 Jul;17(7):471-80

- Magnesium^{241 242 243}
- Vitamin C^{244 245 246 247 248 249}
- Co Q 10^{250 251 252 253}
- Fish Oil^{254 255 256 257 258 259 260 261}

²⁴⁰ Whelton PK; He J; Cutler JA; Brancati FL; Appel LJ; Follmann D; Klag MJ, *Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials.* JAMA 1997 May 28;277(20):1624-32

²⁴¹ Altura BM, Altura BT. *Magnesium ions and contraction of vascular smooth muscles: Relationship to some vascular diseases.* Fed Proc 40(12):2672-9, 1981

²⁴² Seelig MS, Altura BT, Resnick BT, et al. *Low magnesium: a common denominator in pathologic processes in diabetes mellitus, cardiovascular disease and eclampsia.* Abstract. J Am Coll Nutr 11(5):597-637, 1992

²⁴³ Altura BM, Altura BT. *Interactions of Mg and K on blood vessels: Aspects in view of hypertension.* Magnesium 3(4-6): 175-94, 1984

²⁴⁴ <http://www.umm.edu/altmed/ConsSupplements/VitaminCAscorbicAcides.html>

²⁴⁵ <http://lpi.oregonstate.edu/s-s00/arteries.html>

²⁴⁶ <http://archives.cnn.com/1999/HEALTH/12/20/vitamin.c.hypertension/>

²⁴⁷ http://www.pdrhealth.com/drug_info/nmdrugprofiles/nutsupdrugs/vit_0264.shtml

²⁴⁸ Duffy SJ, Gokce N, Holbrook M, et al. *Treatment of hypertension with ascorbic acid.* Lancet. 1999; 354:2048-2049.

²⁴⁹ Ness AR, Chee D, Elliot P. *Vitamin C and blood pressure—an overview.* J Hum Hypertens. 1997; 11:343-350.

²⁵⁰ Langsjoen P, Langsjoen P, Willis R, et al. *Treatment of essential hypertension with coenzyme Q₁₀.* Mol Aspects Med. 1994;15(suppl):S265–S272.

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²⁵² Hodgson JM, Watts GF, Playford DA, et al. *Coenzyme Q (10) improves blood pressure and glycaemic control: a controlled trial in subjects with type 2 diabetes.* Eur J Clin Nutr. 2002;56:1137-1142.

²⁵³ Singh RB, Niaz MA, Rastogi SS, et al. *Effect of hydrosoluble coenzyme Q₁₀ on blood pressures and insulin resistance in hypertensive patients with coronary artery disease.* J Human Hypertens. 1999;13:203–208.

²⁵⁴ <http://patients.uptodate.com/abstract.asp?TR=hyperten/9501&viewAbs=1&title=1>

²⁵⁵ <http://patients.uptodate.com/abstract.asp?TR=hyperten/9501&viewAbs=3&title=3>

²⁵⁶ <http://patients.uptodate.com/abstract.asp?TR=hyperten/9501&viewAbs=5&title=5>

²⁵⁷ Howe PR. *Dietary fats and hypertension: focus on fish oil.* Ann N Y Acad Sci. 1997; 827: 339–352.

²⁵⁸ Morris MC, Sacks F, Rosner B. *Does fish oil lower blood pressure? A meta-analysis of controlled trials.* Circulation. 1993; 88: 523–533

²⁵⁹ Appel LJ, Miller ER3rd, Seidler AJ, et al. *Does supplementation of diet with ‘fish oil’ reduce blood pressure? A meta-analysis of controlled clinical trials.* Arch Intern Med. 1993; 153: 1429–1438.

²⁶⁰ Mori TA, Watts GF, Burke V, et al. *Differential effects of eicosapentaenoic acid and docosahexaenoic acid on vascular reactivity of the forearm microcirculation in hyperlipidemic, overweight men.* Circulation. 2000; 102: 1264–1269.

- Garlic^{262 263 264 265},
- Choline^{266 267},
- Taurine^{268 269 270 271 272 273},
- Vitamin B6 (or P5P)^{274 275 276 277 278}

Effects on Plasma volume: Increased plasma volume may result when renal capacity for sodium excretion is decreased. Urinary sodium excretion imposes an osmotic workload on the kidney that can exceed functional capacity if sodium intakes are repeatedly high. When this point is reached, the obligatory amount of fluid is retained with sodium in the extra cellular (interstitial and plasma) compartment. The level of sodium intake at which decreased efficiency of sodium excretion is observed will vary with age, ethnicity, and family history of hypertension. Moderate reductions in sodium intake can improve the efficiency of renal sodium excretion and thus enhance the therapeutic index of diuretics and minimize side effects associated with their use.

²⁶¹ http://circ.ahajournals.org/cgi/content/full/106/21/2747?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&searchid=1042470374029_7387&stored_search=&FIRSTINDEX=0&volume=106&firstpage=2747&search_url=http%3A%2F%2Fcirc.ahajournals.org%2Fcgi%2Fsearch&journalcode=circulationaha#R48-118058

²⁶² http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12907130&dopt=Abstract

²⁶³ <http://www.docguide.com/news/content.nsf/news/8525697700573E1885256F310062F9F6>

²⁶⁴ <http://www.healthy.net/library/books/textbook/Section5/ALIUMS.PDF>

²⁶⁵ <http://www.drlwilson.com/Articles/hypertension.htm>

²⁶⁶ Ibid

²⁶⁷ <http://www.umm.edu/altmed/ConsConditions/PrimaryPulmonaryHypertensionccc.html>

²⁶⁸ <http://www.drlwilson.com/Articles/hypertension.htm>

²⁶⁹ Kohashi N, Katori R. *Decrease of urinary taurine in essential hypertension.* Jpn Heart J 1983;24:91–102.

²⁷⁰ Abe M, Shibata K, Matsuda T, Furukawa T. *Inhibition of hypertension and salt intake by oral taurine treatment in hypertensive rats.* Hypertension 1987;10:383–9.

²⁷¹ Fujita T, Ando K, Noda H, et al. *Effects of increased adrenomedullary activity and taurine in young patients with borderline hypertension.* Circulation 1987;75:525–32.

²⁷² Militante JD, Lombardini JB, *Treatment of hypertension with oral taurine: experimental and clinical studies.* Amino Acids. 2002;23(4):381-93

²⁷³ http://pubs.nrc-cnrc.gc.ca/cgi-bin/rp/rp2_abst_e?cjpp_y99-060_77_ns_nf_cjpp

²⁷⁴ <http://www.drlwilson.com/Articles/hypertension.htm>

²⁷⁵ http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2399675&dopt=Abstract

²⁷⁶ <http://www.knowledgeofhealth.com/report.asp?story=Do%20High-Dose%20Vitamin%20B6%20Supplements%20Produce%20Health%20Benefits>

²⁷⁷ Nutrition Metabolism Cardiovascular Diseases, Volume 9, Pages 55-63, April 1999

²⁷⁸ Molecular Cell Biochemistry, Volume 188, pages 137-48, 1998

The efficiency with which sodium is excreted can also be modified by dietary factors other than the amount of sodium consumed. These factors include calcium and potassium which promote sodium excretion, and long-term consumption of high protein and high glycemic index diets which may decrease it. Modifications in intakes of these dietary factors can influence tolerance to any level of sodium intake. High potassium and calcium intakes will enhance urinary sodium excretion, thus enabling higher sodium intakes to be consumed when renal excretory capacity is reduced without an increase in plasma volume. High intakes of protein have been proposed to reduce renal functional capacity over time as a consequence of glomerular capillary damage from the repeated high perfusion pressures required to excrete excess nitrogenous waste. High glycemic index diets may contribute to the eventual development of hyperinsulinemia which may decrease the efficiency of sodium excretion through stimulation of sympathetic activity. Insulin also inhibits sodium efflux from cells through effects on membrane ion transporter activity and thus hyperinsulinemia would favor renal sodium retention.²⁷⁹

Effects on Vasoactivity: Changes in vascular tissue reactivity are observed in response to changes in efficiency of sodium excretion in order to maintain consistency of blood flow through the peripheral vasculature with fluctuations in plasma volume. When sodium is retained as a result of decreased renal excretion, plasma volume is increased and thus the peripheral vasculature must compensate by decreasing vessel diameter to maintain a constant blood flow through the capillary bed. Increased stimulation of sympathetic nervous system activity also increases vasoconstriction of the peripheral vasculature.

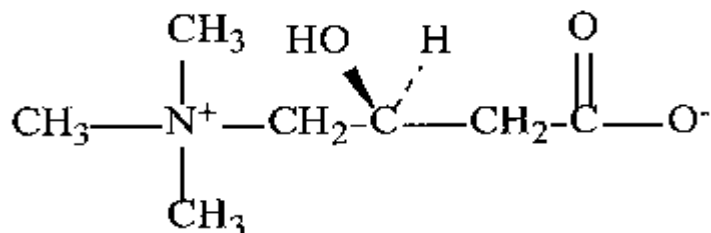
Dietary patterns that favor elevated fasting insulin levels can contribute to increased vascular resistance by insulin-mediated effects on sympathetic activity and renal sodium retention. In contrast, calcium, potassium, and polyunsaturated fatty acids enhance sodium excretion and thus would favor relaxation of the peripheral vasculature. Increased availability of magnesium directly affects capillary vessel resistance by inducing dilation through relaxation of vascular smooth muscle. As precursors for synthesis of the prostacyclin and thromboxane, the balance of intakes between ω -6 and ω -3 PUFA will determine whether vasodilation or vasoconstriction activities will dominate.²⁸⁰

Other Effects: Excess alcohol consumption adversely affects blood pressure by mechanisms that have not been clearly defined. Among the possibilities proposed are induction of sodium retention by stimulation of vasopressin and increased sympathetic nervous system activity.²⁸¹ Injury to vascular endothelial tissue can interfere with ability of the peripheral vasculature to normalize blood pressure with changes in plasma volume. Vitamin C and vitamin E protect the vascular endothelium from oxidative injury.

²⁷⁹ McDonald, A. Feinberg School of Medicine Lectures in Preventive Medicine, used with permission

²⁸⁰ Ibid

²⁸¹ Ibid

Heart Healthy Nutrients:

L-Carnitine

L-carnitine is synthesized in a reaction which is catalyzed by five enzymes which require

- Lysine^{282 283 284},
- methionine²⁸⁵
- Vitamin C,
- Vitamin B₆,
- Niacin as nicotinamide adenine dinucleotide (NAD).²⁸⁶

Because L-Carnitine is so important in energy production and management, an early sign of vitamin C deficiency is fatigue, related to decreased synthesis of L-carnitine.²⁸⁷

L-Carnitine provides myocardial support and increases the efficiency of cardiac contraction when taken on an on-going basis. Used in the immediate post-myocardial period, the same substance has both immediate impact on survival and long term effect on the regaining of cardiac function.

- L-Carnitine treatment has been found to reduce injury to heart muscle resulting from ischemia in several animal models.²⁸⁸

²⁸² Ruiz MC, Ayala V, Portero-Otin M, Requena JR, Barja G, Pamplona R. *Related Articles, Protein methionine content and MDA-lysine adducts are inversely related to maximum life span in the heart of mammals.* Mech Ageing Dev. 2005 Oct;126(10):1106-14.
PMID: 15955547

²⁸³ <http://www.advance-health.com/lysine.html>

²⁸⁴ <http://www.vrp.com/art/798.asp>

²⁸⁵ Seneviratne CK, Li T, Khaper N, Singal PK. *Effects of methionine on endogenous antioxidants in the heart.* Am J Physiol. 1999 Dec;277

²⁸⁶ Seim H, Eichler K, Kleber H. L(-)-Carnitine and its precursor, gamma-butyrobetaine. In: Kramer K, Hoppe P, Packer L, eds. Nutraceuticals in Health and Disease Prevention. New York: Marcel Dekker, Inc.; 2001:217-256.

²⁸⁷ Food and Nutrition Board, Institute of Medicine. Vitamin C. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. Washington D.C.: National Academy Press; 2000:95-185.

²⁸⁸ Lopaschuk G. *Regulation of carbohydrate metabolism in ischemia and reperfusion.* Am Heart J. 2000;139(2 Pt 3):S115-119.

- In humans, the administration of L-carnitine immediately after the diagnosis of MI improved clinical outcomes in several small clinical trials. In one trial, half of 160 men and women diagnosed with a recent MI were randomly assigned to receive 4 grams/day of L-carnitine in addition to standard pharmacological treatment. After one year of treatment, mortality was significantly lower in the L-carnitine supplemented group (1.2% vs. 12.5%), and attacks of angina were less frequent²⁸⁹.

In coronary artery disease, the accumulation of atherosclerotic plaque in the coronary arteries may prevent parts of the heart muscle from receiving adequate circulation, ultimately resulting in damage and impaired pumping ability. In MI, heart tissue may be damaged resulting in compromised pumping ability and clinical heart failure. Damage to the heart results in degraded exercise tolerance and decreased left ventricular ejection fraction (LVEF), indicative of heart failure when less than 40%.²⁹⁰

The addition of L-carnitine to standard medical therapy for heart failure has been evaluated in several clinical trials.

- In a randomized, single-blind, placebo-controlled trial in 30 heart failure patients, oral administration of 1.5 grams/day of propionyl-L-carnitine for 1 month resulted in significantly improved measures of exercise tolerance and a slight but significant decrease in left ventricular size compared to placebo.²⁹¹
- A larger randomized, double blind, placebo-controlled trial compared the addition of propionyl-L-carnitine (1.5 grams/day) to the treatment regimen of 271 heart failure patients to a placebo in 266 patients for 6 months.²⁹² Overall, exercise tolerance was not different between the two groups. However, in those with higher LVEF values (greater than 30%), exercise tolerance was significantly improved in those taking propionyl-L-carnitine compared to placebo, suggesting that propionyl-L-carnitine may help to improve exercise tolerance in higher functioning heart failure patients.

In a randomized, placebo-controlled crossover trial in 44 men with chronic stable angina, 2 grams/day of L-carnitine for 4 weeks significantly increased the **exercise workload tolerated**

²⁸⁹ Davini P, Bigalli A, Lamanna F, Boem A. *Controlled study on L-carnitine therapeutic efficacy in post-infarction*. Drugs Exp Clin Res. 1992;18(8):355-365.

²⁹⁰ Trupp RJ, Abraham WT. *Congestive heart failure*. In: Rakel RE, Bope ET, eds. Rakel: Conn's Current Therapy 2002. 54th ed. New York: W. B. Saunders Company; 2002:306-313.

²⁹¹ Anand I, Chandrashekhnan Y, De Giuli F, et al. *Acute and chronic effects of propionyl-L-carnitine on the hemodynamics, exercise capacity, and hormones in patients with congestive heart failure*. Cardiovasc Drugs Ther. 1998;12(3):291-299.

²⁹² *Study on propionyl-L-carnitine in chronic heart failure*. Eur Heart J. 1999;20(1):70-76.

prior to the onset of angina and decreased ST segment depression during exercise compared to placebo.²⁹³

In a recent randomized placebo-controlled trial in 47 men and women with chronic stable angina, the addition of 2 grams/day of L-carnitine for 3 months significantly improved exercise duration and decreased the time required for exercise-induced ST segment changes to return to baseline compared to placebo.²⁹⁴

²⁹³ Cherchi A, Lai C, Angelino F, et al. *Effects of L-carnitine on exercise tolerance in chronic stable angina: a multicenter, double-blind, randomized, placebo controlled crossover study.* Int J Clin Pharmacol Ther Toxicol. 1985;23(10):569-572.

²⁹⁴ Iyer RN, Khan AA, Gupta A, Vajifdar BU, Lokhandwala YY. *L-carnitine moderately improves the exercise tolerance in chronic stable angina.* J Assoc Physicians India. 2000;48(11):1050-1052

C. Nutrition and Glucose Control

Both absolute glucose values and fluctuations in those values are of extreme importance in the short and long term outcome of diabetics. Digestible carbohydrates make a major contribution to post-prandial glucose levels. In addition to the macro nutrient impact that carbohydrates have on blood glucose, minerals and other nutrients which are part of the diet or taken as supplemental nutrition have a significant and crucial role to play in mediating the body's ability to restore or maintain post-prandial blood glucose levels to fasting levels over time. When properly mediated, this cycle allows proper absorption and utilization of glucose from the GI tract. Minerals and other nutrients have a secondary impact on hormones and mediators which in turn impact the secretion of insulin, clearance and cellular response to it.

Numerous diseases, life style choices and conditions, as well as pharmaceuticals impair or impact glucose control. In each condition or drug reaction they are mediated not only with the often recommended physical exercise and diet, but are also strongly impacted by nutritional correction and control of blood glucose. Disease conditions impacting glucose include: metabolic syndrome

- Diabetes mellitus
- Hypertension
- Hyperlipidemia
- Obesity
- Liver disease
- Renal disease
- Cancer
- Trauma, injury
- Sepsis
- Medications (e.g., hydrochlorothiazide, prednisone, chlorpropamide, propranolol, etc.).²⁹⁵

Hyperinsulinemia, (with or without hyperglycemia), is the most commonly observed abnormality in blood glucose regulation. Large amounts of rapidly absorbed simple sugars which lead to an insulin "overshoot" (a sharp rise in insulin greater than that required to accommodate the total glucose load). This is rapidly followed by a rapid decline to below-fasting levels of glucose. This hyperglycemia is rapidly followed by hypoglycemia within 60 to 120 minutes following the carbohydrate intake. As a consequence of this recurring cycle over time, widespread consequences result including metabolic obesity and serious renal, retinal, cardiovascular, neurological, endocrine and reproductive abnormalities and diseases.

Habitual dietary abuses create pathological metabolic changes and aberrations at the cell receptor level which are difficult to mediate without adequate dietary and supplemental nutrients. In fact, long term glucose patterns have a greater impact than short-term intakes because tissue response to insulin is a receptor-mediated phenomenon with more or fewer receptors induced by characteristic dietary intake²⁹⁶

²⁹⁵ McDonald, A. Feinberg School of Medicine Preventive Medicine Lectures, Op. Cit.

²⁹⁶ Ibid

Sucrose intake should be restricted. Both laboratory animals²⁹⁷ and healthy humans²⁹⁸ caused glucose tolerance to be impaired and rendered the tissues less sensitive to glucose.

- Sucrose encourages the development of diabetic retinopathy and nephropathy.²⁹⁹
- A diet with more than 35% of calories from sucrose generates a chromium deficiency since increased sucrose metabolism leads to increased urinary chromium excretion.³⁰⁰

Since fructose is a disaccharide composed of glucose and a sucrose moiety, the glycemic response in diabetics is determined largely by the glucose fraction.³⁰¹ Fructose, however, is superior to sucrose and may be similar to starch in terms of glycemic control.³⁰² However, it is the fructose moiety of the sucrose molecule which appears to be responsible for the adverse effects of sucrose on serum lipids³⁰³ so the addition of fructose may increase insulin resistance.

Because fructose leads to increase copper excretion, inclusion in the diet of diabetics can lead to a copper deficiency which can, in turn, impair glucose tolerance.

Changing not only the amount, but also the timing of meals and snacks has a beneficial impact if the change is to small frequent meals with no increase in caloric intake over the desired range. Improvement in both serum insulin and 24 hour urinary C peptide levels decreased significantly in type I and type II diabetics. Blood glucose was reduced in diabetic subjects but not in normals. Non diabetic normals showed the same sharp improvement in their values which, in their case, took them from normal to near-optimal.³⁰⁴

Among Australian aboriginal peoples diabetes was totally unknown as long as a hunter-gatherer lifestyle was followed. In the 1970s missionaries introduce flour and sugar to their converts. By the 1980's diabetes was rampant, diagnosed in more than 20% of urban aboriginals. Prior to urbanization, they lived on what they could kill or collect and ate meat from wild animals so their diet was high in protein and dietary fiber but low in fat.

²⁹⁷ Storlien LH et. al., Effects of sucrose vs. starch diets on in vivo insulin action, thermogenesis, and obesity in rats. *Am J. Clin Nutr* 47:420-7, 1998

²⁹⁸ Reiser S et. al., *Effects of sugars on indices of glucose tolerance in humans*, *Am J. Clin Nutr* 43(1):151-9, 1986

²⁹⁹ Cohen, AM, *Experimental models in diabetes* in *Sugars in Nutrition*. Sna Francisco, Academic Press, 1974:483-511

³⁰⁰ Kozlovsky, A S et al. *Effects of diets high in simple sugars on urinary chromium losses*. *Metabolism* 35(6):515-18, 1986

³⁰¹ Hughes, TA, *Glycemic responses in insulin dependent diabetics: effect of food composition*. *Am J Clin Nutr* 49:658-66, 1989

³⁰² Thornburn, A W, et. al, *Fructose induced in vivo insulin resistance and elevated plasma triglyceride levels in rats*. *Am J Clin Nutr* 49:1155-63, 1989

³⁰³ Reiser, S et al, *Blood lipid, lipoproteins, apoproteins and uric acid in men fed diets containing fructose or high-amylose cornstarch*. *Am J. Clin Nutr* 49:832-9, 1989

³⁰⁴ Jenkins, DJ, *Metabolic advantages of spreading the nutrient load: effects of increased meal frequency in non insulin dependent diabetics*, *Am J Clin Nutr* 55:461-7, 1992

- Ten urbanized Type II diabetics agreed to return to the hunter-gatherer lifestyle for 7 weeks. Average age was 54 and all were overweight and exercised little. Before the study, most ate a diet high in fatty meat and several had hypertension while half of the study group drank heavily.

During the 7 week study, their activity level increased and food intake was reduced to 1200 calories per day. Although 64% of their diet (which they caught) was protein and only 13% was fat all subjects lost weight steadily. Blood pressure and triglyceride levels fell. Glucose tolerance improved greatly from an average fasting glucose of 200 mg/dl to 120 mg/dl indicating major improvement in post-prandial clearance.³⁰⁵

Juvenile intake of coffee, tea,³⁰⁶ nitrates³⁰⁷ and nitrosamines³⁰⁸ potentiate the development of insulin dependent diabetes. Many foods are diabetogenic including wheat, soy³⁰⁹ and cow's milk³¹⁰ especially in children and infants. Other factors may potentiate insulin resistance but, regardless of cause, the final common expressions of hyperinsulinemia and hyper/hypoglycemia are responsive to dietary strategies for regulation of glucose and prevention or amelioration of glucose related degenerative pathologies.

Non-diet-related insulin resistance is frequently precipitated by pregnancy, stress or sepsis. Post-receptor defect in signal transduction involving glucose transporter synthesis and activity is a frequent complication of injury or sepsis which is potentiated by stress-mediated elevations in epinephrine, glucagon, and cortisol. Although diet is not involved in the development of insulin resistance in conditions associated with metabolic stress, dietary adjustments and nutritional strategies can prevent the worsening of hyperinsulinemia and hyperglycemia frequently observed in pregnant, injured or septic patients.³¹¹

Protein restriction may delay or prevent the development of nephropathy. Twenty two insulin dependant diabetics randomly received either an unrestricted protein diet or a moderately protein-restricted diet for 6 months. Patients on the unrestricted diet showed progressive decline in glomerular filtration rate with no change in proteinuria. Protein-restricted patients showed a marked decrease in proteinuria and a stabilization of glomerular filtration rate independently of changes in blood pressure or glycemic control.³¹²

³⁰⁵ O'Day, K, *Marked improvement in carbohydrate and lipid metabolism in diabetic Australian aborigines after temporary reversion to traditional lifestyle.* Diabetes 33(6):596-603, 1984

³⁰⁶ Virtanen, S M, et. al, *Is children's or parent's coffee or tea consumption associated with the risk for insulin dependent diabetes mellitus in children?* Eur J Clin Nutr 48:279-85, 1994

³⁰⁷ Kostraba, JN, et al., *I* 15(11):1505-8, 1992

³⁰⁸ Dahlquist, GG, et. al, *Dietary factors and the risk of developing insulin dependent diabetes in children.* Br Med J 300:1302-6, 1990

³⁰⁹ Scott, F W, et. al, *Milk and type I diabetes, Examining the evidence and broadening the focus.* Diabetes Care 19(4):379-83, 1996

³¹⁰ Atkinson, M A, Ellis, T M, *Infants diets and insulin dependent diabetes: evaluating the "cow's milk hypothesis and a role for anti bovine serum albumin immunity.* J Am Coll Nutr 16(4): 334-40 1997

³¹¹ McDonald, Op. Cit.

³¹² Raal, FJ, et. al, *Effect of moderate protein restriction on the progression of overt diabetic neuropathy: a 6 month prospective study.* Am J Clin Nutr 60:579-85:1994

In patients whose insulin secretion is insufficient to carry out the tasks of glucose regulation, a wide variety of metabolic burdens are imposed on every cell and tissue in the body. Correction and protection of the widespread damage caused by this metabolic deviation requires vigorous nutritional supplementation. Commonly used pharmaceuticals exacerbate the nutritional requirements and their use makes the employment of nutritional strategies particularly important in order to reduce the complications of diabetes and assist in good control of the patient's glucose.³¹³

Availability of dietary glucose from food sources is the primary contributor to post-prandial hyperinsulinemia since maximal levels of circulating insulin are attained immediately following ingestion of a carbohydrate source. However, total energy intake and its distribution throughout the day, type and amount of fat, type and amount of protein, and intakes of specific micronutrients may each modify the insulin response to a particular glucose load and/or protect against or modify the enzymatic, cellular, organ, tissue and systemic impact of poor glucose control. Nutrients which show an important impact on insulin and glucose utilization and correction, amelioration or protection of hyperinsulinemia and diabetes-related damage include:

- Minerals

- Chromium^{314 315 316 317 318 319 320 321}
- Vanadium^{322 323 324 325 326 327 328 329 330}
- Calcium^{331 332 333 334 335}

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- Selenium^{336 337}
- Copper^{338 339 340}
- Manganese^{341 342}
- Potassium^{343 344 345 346 347} with caution
- Magnesium^{348 349 350 351 352 353 354}
- Zinc^{355 356}
- Amino Acids
- Arginine^{357 358 359 360 361 362}

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- Taurine^{363 364 365 366 367}
- Glutathione^{368 369 370}
- N-Acetyl Carnitine^{371 372 373 374}
- Fats and Oils
 - Omega 3, 6 fatty acids^{375 376 377}
- Vitamins and Co-Factors
 - Vitamins B1^{378 379 380 381 382}
 - Vitamin B6^{383 384 385 386}
 - Vitamin B12^{387 388}
 - Vitamin C^{389 390 391 392 393 394 395}

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○ Vitamin E^{396, 397, 398, 399, 400, 401, 402, 403, 404, 405}

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- Co Q 10^{412, 413, 414, 415, 416}
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- Folic acid^{423 424 425}
- Proanthocyanidins^{426 427 428}
- Dietary Fiber^{429 430 431 432 433434}
- Ferulic Acid^{435 436}
- Pancreatic enzymes^{437 438 439 440 441}

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The following summary is used, with permission, from the excellent Preventive Medicine lectures of Dr. Arline McDonald, who teaches this subject at the Feinberg School of Medicine of Northwest University, Chicago, IL.⁴⁴²

CONSEQUENCES OF DIET-INDUCED INSULIN RESISTANCE: The role of diet-induced hyperinsulinemia and insulin resistance in the etiology of noninsulin-dependent diabetes mellitus (NIDDM) has not been clearly defined. Hyperinsulinemia and hyperglycemia may precede development of diabetes characterized by insulin resistance by a 5 to 10-year period. Both of these abnormalities can have adverse cellular and systemic effects. Adverse effects associated with hyperglycemia are a consequence of cellular injury in noninsulin-dependent tissues that occurs with increased intracellular concentration of glucose. These include vascular, renal, ocular, and infectious complications which are frequently observed in poorly controlled diabetes. In the absence of diabetes, hyperglycemia is not usually observed in association with hyperinsulinemia because normalization of blood glucose is achieved at the expense of higher insulin levels.

Adverse effects of sustained hyperinsulinemia include increased sympathetic nervous system activity, alteration in calcium transport by smooth muscle cells, and increased proximal and distal tubular reabsorption of sodium. Each of these effects may contribute to increased peripheral vascular resistance and elevated blood pressure. Insulin also has mitogenic activity that promotes proliferation of vascular smooth muscle cells resulting in thickening of capillary and blood vessel walls and narrowing the arterial lumen. These structural changes contribute to increased peripheral resistance and formation of atherosclerotic plaques. Fibrinolytic activity may also be increased with hyperinsulinemia since levels of plasminogen activator inhibitor are increased by insulin. Insulin resistance is frequently observed in both hypertension and coronary heart disease. Insulin resistance induced by dietary factors (e.g., obesity) differs from that which characterizes noninsulin-dependent diabetes mellitus (NIDDM) in severity and associated metabolic derangements. Diet-induced insulin resistance also tends to be peripheral (e.g., skeletal muscle) rather than hepatic and appears to affect nonoxidative glucose disposal (e.g., glycogenesis) more than oxidative pathways. In NIDDM, insulin is ineffective in suppressing hepatic glucose output and insulin resistance interferes with adipocyte lipogenesis and oxidative glucose metabolism by skeletal muscle.

THE GLYCEMIC INDEX: The glycemic index is a physiological measure that is used to predict the effects of a source of digestible carbohydrate on blood glucose. The glycemic index is calculated as the AUC of the blood glucose response over a 2-hour period to the amount of carbohydrate ingested in a 100 g serving of a food expressed as a percentage of the response to a standard (white bread or glucose solution) providing an equivalent amount of carbohydrate. For example, the AUC for whole wheat bread is 811 compared to the AUC for an isocarbohydrate serving of white bread which is 866. The glycemic index of whole wheat bread would then be calculated as 0.94 (811/866) or 94%. A glycemic index of 94 means that whole wheat bread will elicit a blood glucose response that is 94% of the response that would be observed with white bread over the same time period. Glycemic response to a mixed meal can also be estimated using the glycemic indices of the individual foods. Each food is then weighted by the proportion of total

⁴⁴² McDonald, A, Nutrition and Blood Glucose, Preventive Medicine Lectures

carbohydrate it contributes to the meal to obtain an estimate for the meal. The percent difference among meal glycemic indices has been shown to accurately predict the mean incremental glycemic response areas for different mixed meals consumed by groups of subjects.

SPECIFIC EFFECTS OF DIETARY COMPONENTS ON BLOOD GLUCOSE CONTROL.

Carbohydrate: Digestible carbohydrate contributes directly to glycemic load which is the primary determinant of insulin response. Foods that provide the largest glycemic loads are sources of carbohydrate that are readily digested to soluble sugars, and then rapidly and completely absorbed. The glycemic effects of simple sugars (disaccharides and monosaccharides) can be predicted from differences in solubility in aqueous solutions. Soluble sugars consumed in liquid form, e.g., beverages, will empty from the stomach faster than the identical sugars consumed in solid form and will thus reach the surface mucosal digestive enzymes sooner. Simple sugars in solid form will solubilize more rapidly when consumed from highly refined (processed) sources, e.g., candy, beverages, pastries, processed cereals, jelly, and will be absorbed more rapidly than the same simple sugars consumed in fruits and whole grain cereals where solubility is reduced by the presence of dietary fiber. Sucrose is the most soluble simple sugar while lactose (milk sugar) is the least soluble. Fructose is relatively insoluble and contributes less to an increase in plasma glucose than the equivalent amount of carbohydrate consumed as sucrose. The presence of sodium will accelerate the absorption of simple sugars in both liquid and solid form.

The glycemic effects of starches or polysaccharides can be predicted primarily from differences in digestibility which will determine how quickly the oligosaccharide and disaccharide fragments are released from amylase activity and solubilized in intestinal fluids. Starches are consumed from cereal grains (e.g., wheat, corn, rice, barley, rye, oats), legumes (e.g., lentils, navy beans, chickpeas), and vegetables (e.g., potato). Undigested starches are less soluble than simple sugars, but if starch is consumed from a refined source, it may be more quickly digested and thus solubilized and absorbed faster than simple sugars consumed from a source rich in dietary fiber. Not all processed starches are rapidly digested and absorbed due to differences in molecular structure which can substantially influence digestibility. Methods of processing that include extrusion (pasta and cereal shapes), as well as exposure to high temperature followed by cooling, promote a rearrangement of starch crystals such that the glycosidic bonds become resistant to digestive enzymes (alpha-amylases). A high content of amylopectin (branching) relative to amylose (straight chain) will also facilitate digestion because the branch points of amylopectin provide more available sites for amylase to act upon. Starches from unprocessed or less processed sources are digested more slowly than refined starches (e.g., whole wheat bread vs. white bread), and thus more slowly absorbed, because of the higher amount of dietary fiber present.

The glycemic effects of carbohydrate sources that also contain dietary fiber are differentiated by the viscosity of the fiber. Viscous dietary fibers are classified as soluble fiber and include gums, pectins, mucilages, glucans, and hemicelluloses. Soluble fiber has the greatest effect on slowing the rate of digestive and absorptive processes. This type of dietary fiber forms a viscous gel when mixed with the aqueous solutions that comprise gastric and pancreatic fluids. Viscosity creates a barrier that reduces access of the digestive enzymes to the food matrices. Soluble fiber also increases the viscosity of

the unstirred water layer adjacent to the mucosal absorptive surface, thus slowing the rate of diffusion of disaccharides and monosaccharides to the mucosal membranes where digestive enzymes and membrane transport systems are located. Soluble fiber is found in oats, barley, citrus fruit, legumes, and psyllium.

Fat: Dietary fat can reduce the glycemic load available from ingestion of digestible carbohydrate by slowing gastric motility through stimulation of enterogastrone secretion in the duodenum. A decrease in gastric motility will delay gastric emptying, thus slowing the release of sugars available to undergo digestion and absorption. These short-term effects of fat may be offset by the long-term adverse effects of habitual high fat intakes on insulin sensitivity. When consumed in large amounts, dietary fat reduces insulin sensitivity by increasing fat stores. Body fat as measured by body mass index has been significantly correlated with dietary fat intake in both lean and obese adults. Insulin sensitivity has also been related inversely to fat intake. High fat diets apparently predispose to weight gain to a greater extent than other energy sources at equivalent energy intakes when total energy intake is moderate. This observation has been explained by differences in fuel storage regulation between fat and carbohydrate. The rate of carbohydrate oxidation increases as intake increases once glycogen stores have been maximized because the capacity to store carbohydrate as glycogen is limited. In contrast, fat oxidation does not have to increase as intake increases to regulate the total body pool because fat storage capacity is virtually unlimited.

Dietary fat can also affect tissue sensitivity to insulin without weight gain or an increase in body fat. A possible explanation is that high fat intakes proportionately reduce the amount of carbohydrate consumed, and thus may reduce the number of glucose transporters in the intracellular pool and down-regulate insulin receptors. The composition of fat consumed may also influence tissue insulin sensitivity. Because circulating fatty acids are deposited in membrane phospholipids, membrane fatty acid composition will reflect dietary fat composition. A high saturated fat content in cell membranes will decrease membrane fluidity and adversely affect recruitment of glucose transporters to the plasma membrane from the intracellular pool. The number of glucose transporters measured in rat adipocytes was higher in animals fed polyunsaturated fat than in those fed saturated fat. Intakes of both monounsaturated fatty acids and medium chain saturated fatty acids (coconut oil) can promote insulin secretion. This effect of these fatty acids may be helpful for controlling blood glucose among “carbohydrate-sensitive” individuals. A significant proportion of diabetics are unable to decrease total fat intake by proportionately increasing carbohydrate without an increase in serum triglycerides and cholesterol. For these people, increasing the proportion of monounsaturated fat ingested will allow a higher total fat to be consumed without compromising insulin sensitivity.

Protein. Insulin responsiveness to dietary protein is a function of its amino acid composition. The ratio of insulin to glucagon determines whether metabolism will favor increased (low ratio) or decreased blood glucose (high ratio). The insulin to glucagon ratio also controls accretion of lean body mass by favoring either protein synthesis (high ratio) or catabolism (low ratio). It also influences cholesterol metabolism by either stimulating (high ratio) or inhibiting (low ratio) the activity of HMG-CoA reductase, the rate-limiting step of cholesterol synthesis. Lysine has been shown to be particularly effective in raising the ratio of insulin to glucagon in both animals and humans while

arginine appears to diminish the effect of lysine. Plant protein sources tend to be lower in lysine and higher in arginine than animal protein sources such as casein (cow's milk).

Micronutrients. Insufficient intakes of vitamins and minerals that support insulin function and carbohydrate metabolism may also contribute to impaired glucose tolerance. These micronutrients include chromium, potassium, magnesium, and vitamin E. Chromium is required for normal glucose tolerance, but the mechanism of its effect has not been identified. Tissue chromium levels appear to decrease with age coincident with increased glucose intolerance and risk of NIDDM. The requirement for chromium is increased by high intakes of refined carbohydrate. Since the primary dietary source of chromium is whole grains, replacement of whole grain products with refined grain products will reduce chromium intake while increasing requirements for the mineral.

Potassium and magnesium are more involved with glucose metabolism than with direct effects on insulin function. Potassium is a cofactor for phosphofructokinase, a rate-limiting enzyme for glycolysis. Magnesium is required for oxidative metabolism of glucose. It is unclear whether supplementation with these minerals will improve glucose tolerance above the response to correction of the deficiencies. Abnormal blood levels of copper, zinc, and magnesium are frequently observed in individuals with diabetic complications, but it is unknown whether these abnormalities are a cause or an effect of the associated pathology. Low blood levels of chromium, magnesium, potassium, and pyridoxine have been reported in pregnant women with gestational diabetes. This condition is characterized by insulin activity that is insufficient to balance the glucose-elevating effects of the placental, pituitary, and adrenal hormones.

Vitamin E may improve glucose tolerance by inhibiting membrane lipid peroxidation and thus preserving membrane integrity. Optimal insulin tissue activity is observed when cell membranes are enriched with polyunsaturated fatty acids. The high degree of unsaturation in membrane lipids increases their fluidity and also their vulnerability to oxidative damage. Plasma peroxides have been measured at higher concentrations in diabetics than in controls. A recent study reported significantly lower fasting and two-hour plasma insulin, and a significant increase in nonoxidative glucose metabolism, among postmenopausal women taking vitamin E supplements at doses of 900 IU/day.

Energy Intake and Distribution. Excess energy intake can contribute to insulin resistance by increasing the concentrations of glucose and fatty acids in circulation and by contributing to increased body fat stores. Obesity has been associated with both receptor and post-receptor defects in insulin function. Abdominal obesity is more strongly related to insulin resistance than gluteal or peripheral obesity. Hyperinsulinemia is more likely to develop with abdominal obesity because hepatic insulin clearance is inhibited by high concentrations of free fatty acids in the portal circulation. The mobilization of free fatty acids from abdominal fat depots also stimulates hepatic glucose production, which initiates the metabolic cascade that begins with hyperglycemia and is followed by establishment of hyperinsulinemia and down-regulation of insulin receptors. Abdominal adipocytes also require significantly higher levels of insulin to promote glucose uptake than peripheral adipocytes.

The increase in body fat associated with aging occurs at the expense of skeletal muscle and is primarily the result of decreased physical activity. Muscle mass enhances insulin sensitivity because skeletal muscle accounts for the majority of insulin-facilitated glucose

uptake. Exercise also protects against accumulation of fat stores by increasing energy expenditure. Exercise enhances insulin sensitivity directly by lowering the Km of skeletal muscle glucose transporters both during activity and after activity to replenish muscle glycogen stores. Exercise also enhances the mobilization of free fatty acids from adipocytes for uptake by working (skeletal) muscle and favors fatty acids mobilization from abdominal adipocytes. Thus abdominal fat is more responsive to exercise resulting in a rapid improvement in insulin sensitivity as a result of a decline in portal free fatty acid levels that inhibit hepatic insulin clearance.

The pattern in which energy is distributed throughout the day may also influence insulin sensitivity. A high frequency of eating occasions (e.g., “grazing”) is often recommended to prevent the wide excursions in insulin levels between fasting and postprandial periods. Since the post-prandial state typically lasts 3-4 hours following ingestion of a meal, timing of eating occasions at 3-4 hour intervals should stabilize delivery of glucose during the day, provided that the total energy requirement is evenly distributed and not exceeded, and the composition of the meal provides a moderate to low glycemic load. Since post-prandial hyperinsulinemia must be chronic to elicit glucose intolerance by down-regulating receptors, frequent consumption of high glycemic loads could offset the benefits of dividing total energy intake into frequent small evenly divided meals.

AUTHOR'S NOTE

I have practiced Natural Health for approximately 35 years since I was graduated from the Albert Einstein College of Medicine⁴⁴⁴, in 1970. Practicing Natural and drug-free Psychiatry and drug free Primary Care Medicine during that time I have come to expect “near-miracles” on a daily basis as reliably diagnosed “irreversible”, “terminal” and “hopeless” patients made their way to my office from around the world. Using simple, but physiologically and biochemically profound, treatment options, it has been my privilege and honor to preside over the change in status from “hopeless” to “radiantly well” of thousands of patients. I have been fortunate to have the tools in my hand to offer many other patients (and, inevitably, friends and family members as well) the opportunity to defy the customary expectations for health and longevity derived from the statistics of an ailing and ill population. The results have been gratifying in the extreme as people confronting illness and death, or the sad decay which we mistakenly identify as “normal ageing” have chosen options for themselves of continuing robust and healthy life in keeping with the genetic potential given to us all but foreshortened for so many through poor nutrition and industrial toxicity.

It is possible for many, perhaps most, people to enjoy robust good health in the midst of continuing and escalating challenges to the detoxification and immune systems which an increasingly industrial society and food supply present to us all if inexpensive, simple, science-based and logical prevention and treatment strategies are available to us.

If my experience were unique, however, it would be of little concern. In fact, every doctor and health care professional who has chosen to practice their profession using natural means to treat the underlying causes of disease (as conventional Western Medicine cannot do) has a similar clinical experience to which they can testify.

It is for that reason that the Revised Vitamin and Mineral Guideline has been endorsed by several eminent associations and organizations including

- **The American Academy of Environmental Medicine**⁴⁴⁵
- **The National Association of Nutrition Professionals**⁴⁴⁶
- **The Neurotherapy and Biofeedback Certification Board**⁴⁴⁷
- **Great Smokies Medical Center of Ashville**⁴⁴⁸
- **Institute for Health Research**⁴⁴⁹
- **Freedom Club USA**⁴⁵⁰
- **BioRenew LLC.**⁴⁵¹

⁴⁴⁴ <http://www.aecom.yu.edu/home/>

⁴⁴⁵ <http://www.aaem.com/>

⁴⁴⁶ <http://www.nanp.org>

⁴⁴⁷ <http://NBCB.org>

⁴⁴⁸ <http://www.gsmcweb.com/>

⁴⁴⁹ <http://www.inhere.org/>

⁴⁵⁰ <http://www.aaem.com/>



NUTRITION AND HEALTH

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- Andrew Saul, PhD

Nutrition is nothing less than the foundation of medicine, the cornerstone of all medical therapeutics.

— A Forum on Nutrition and Health, JON 2(4), 1993

Prevention cannot start too early. Neither can it start too late.

— Rima E. Laibow, MD, 2005



ADDITIONAL MATERIALS

- › CODEX BROCHURE
- › DVD COVER

AVAILABLE ONLINE:

WWW.HEALTHFREEDOMUSA.ORG
HEALTHFREEDOM@OPTONLINE.NET

CODEX ALIMENTARIUS

[*Latin for "Food Rules"*]

IS FOOD TERRORISM:

LIKE ANY OTHER TERRORISM,
IT GAINS POWER FROM OUR SILENCE.

OUR MISSION

TO PRESERVE, PROTECT AND DEFEND
HEALTH AND HEALTH FREEDOM IN THE
UNITED STATES AND GLOBALLY.

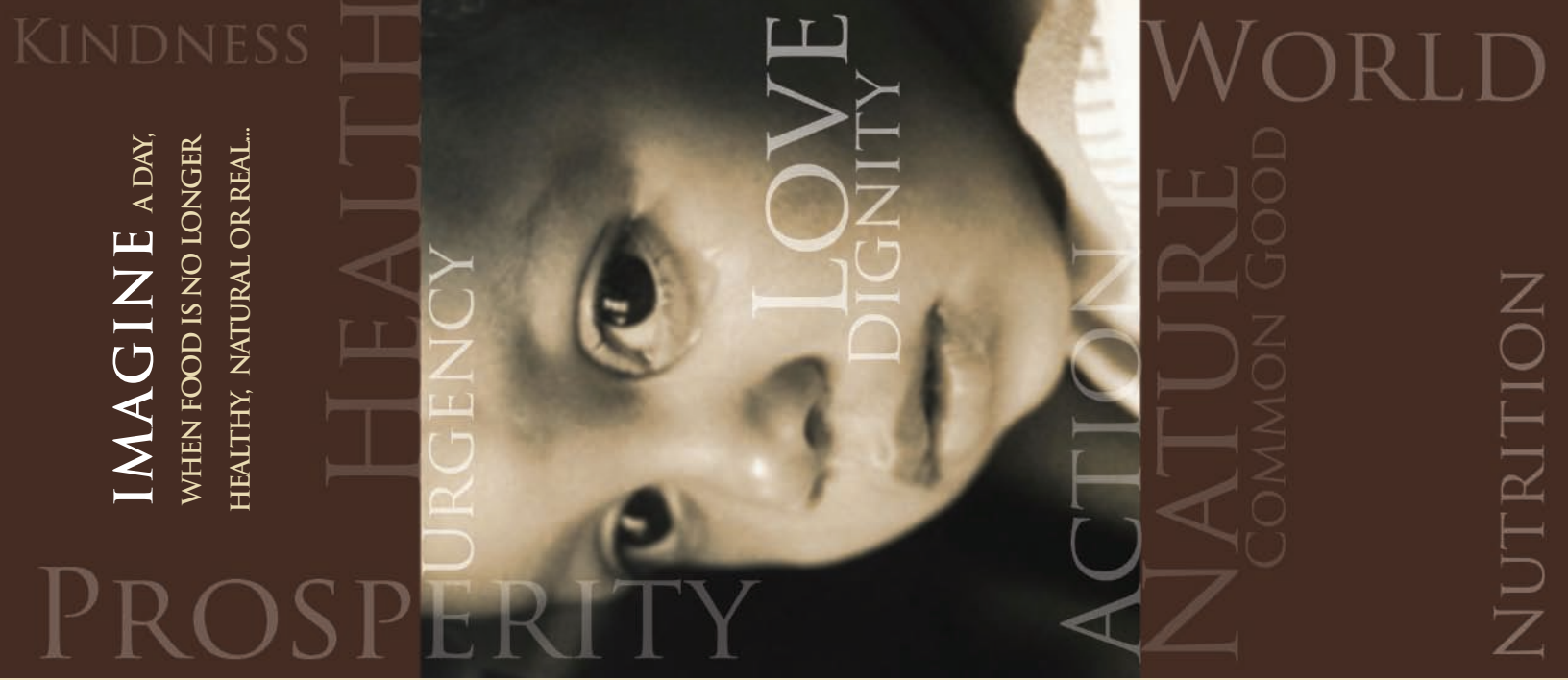
YOU HAVE THE POWER TO KEEP
AMERICA CODEX FREE!

GRASS ROOTS = GREAT RESOURCES.
CONGRESS KNOWS THAT YOU CAN VOTE
AND THAT YOU HAVE FRIENDS
WHO CAN VOTE, TOO.



YOUR HEALTH IS YOUR WEALTH.
JOIN THE FIGHT TODAY. TIME IS RUNNING OUT.

- 1 Statement at 28th Codex Alimentarius Commission Meeting by Dr. Kerstin Lettner, Assistant Director-General, Sustainable Development and Healthy Environments, World Health Organization, July 6, 2005, Rome
- 2 DSHBA, the Dietary Supplements Health and Education Act of 1994 classifies supplements as foods on which no upper limit can be set.
- 3 Codex Allows Seven Deadly Pesticides Banned by Stockholm Convention DSHBA was passed by unanimous Congressional Consent in 1994 and US Law <http://www.prweb.com/releases/2005/8/prweb276701.htm>
- 4 <http://www.nifa.org/codex/>, <http://crnusa.org/> <http://www.cfsan.fda.gov/~dms/dscodex.html> <http://www.snopes.com/politics/medical/vitamins.asp>



WHO IS BEHIND CODEX?

Codex Alimentarius, established in 1962 as a UN trade Commission, serves corporate greed with no interest in health or consumer protection. The World Health Organization says Codex Alimentarius “*Has not made a contribution in human health in its 42 years of existence.*”

CODEX ALIMENTARIUS SPONSORS:

[Connect the dots]

- **BIG PHARMA** - drug companies make HUGE profits on unnecessarily expensive drugs
- **BIG MEDICA** - pro-illness industry thrives when you and/or your family are ill
- **BIG CHEMA** - profitable toxic chemicals used on food and fields, including deadly pesticides banned in the US
- **BIG AGRIBIZ** - industrial farms use antibiotics, drugs, hormones to increase profits
- **BIG BIOTECHNA** - creates dangerous untested Genetically Modified Organisms (GMOs) planned to become legal worldwide unlabeled

WHAT IS THE CODEX THREAT?

Codex Alimentarius has no actual legal standing BUT exerts a lot of influence since it is used by the World Trade Organization [WTO] to decide trade disputes. Codex-compliant countries win automatically regardless of the merits of the case. Devastating trade sanctions result so the US is now racing to destroy protective laws to clear the way to “HARMonize” with Codex.

CODEX ALIMENTARIUS HAS A DEADLY SURPRISE IN STORE FOR YOU:

- Nutrients have been declared to be toxins and Codex is supposedly “protecting” us from them!
- Bans virtually all natural health options
- All effective, high potency nutrients will be illegal with or without a prescription
- Only a few ultra low-dose nutrients will be legal—all others banned while deadly drugs are sold unopposed

- Dangerous growth stimulants [hormones] mandatory in all meat and milk [e.g., Monsanto’s rBGH]
- Deadly banned pesticides permitted in your food³
- Untested, un-labeled Franken-food in your kitchen [GMOs - Genetically Modified Organisms]
- Free-Radical rich Irradiated food
- Widespread under-nutrition resulting in increased cancer, diabetes, heart disease, stroke, auto-immune diseases, depression, obesity, autism—the list is endless
- Weak and meaningless “Organic Farming” standards allowing dangerous drugs and chemicals in so-called “Organic” food

WHERE IS CODEX LURKING?

Codex Alimentarius is global in scope and affects all World Trade Organization member countries [including the U.S.] despite misinformation to the contrary.⁴

WHEN WILL CODEX STRIKE?

The Vitamin and Mineral Guideline was recently ratified on July 4, 2005 at the Rome, Italy Codex meeting. Codex will go into global effect on **DECEMBER 31, 2009**.

WHY IS THIS HAPPENING?

Healthy people are bad for the most powerful industries in the world: Big Pharma and Big Medica. Adulteration of food and elimination of nutrients means people cannot get healthy or stay that way—huge profits result for the adulterators in the process.

JOIN THE FIGHT!

WE NEED YOUR HELP

- 1 Join the Natural Solutions Foundation and stay up to the minute on our Health Freedom. [Your email and information will never be shared for any reason.]
- 2 Contact Congress - www.HealthFreedomUSA.org/bills makes it easy with our petition letters. No internet access? Call Congress direct: [202] 224-3121
- 3 Visit Congress in their local offices with a group of friends. Let them know you expect them to protect your health freedom and defeat all of the bills before Congress clearing the way for “HARMonization” with Codex.
- 4 Give generously. We depend on your donations of time and/or money to help us protect your Health Freedom. [Visit our website to see how your money is being used.]

Name _____

Address _____

Email _____

- ☐ Yes, I want to be a grass roots facilitator. Tell me more.
- ☐ I have an idea. I think we should: [Please print] _____

- ☐ I have a special talent to share and would like to: _____
- ☐ I would like to volunteer some of my time. _____

[List times best for you]

- ☐ Yes, I want to support the Natural Solutions Foundation!

• Here is my donation of:

☐ \$25 ☐ \$50 ☐ \$75 ☐ Other \$ _____

[For credit card or PayPal donation, visit www.HealthFreedomUSA.org/donate]

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DIETARY SUPPLEMENTS ARE FOODS



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Biochemistry, Nutritional Science, Integrative and Nutritional Medicine's clinical practice and wisdom all offer important guiding information in developing a framework in which optimal nutrition can be supported in a population reducing the joint burdens of the personal, social and economic cost of preventable disease.

Consumers and their health providers have the right, the need and the wisdom to make choices about nutrients without governmental restraint or regulation provided only that the nutrient is clean, unadulterated, and poses no unreasonable threat. Within that framework, it is the right and the responsibility of consumers and care givers to use that freedom wisely. If they do not, however, the inherent lack of toxicity in nutrients protects those who might be immoderate. No such protection rests in pharmaceuticals which are usually toxic and, with appropriate or excessive use, often cause disease and death. Nutrients are inexpensive and allow public health systems to use the same amount of money spent on non-acute, non-emergency care for the few and instead provide outstanding care for the many at the same or smaller cost.

Rima E. Laibow, MD
Medical Director
Natural Solutions Foundation

⁴⁵¹ <http://www.biorenew.com/>