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CALENDAR OF EVENTS

- March 3-4, 2007** (Houston, TX)
Spirometry and Pulmonary Disease
Instructor: *Jack Kessinger, DC DABCI*
- March 10-11, 2007** (Charlotte, NC)
Electrocardiography and Phonocardiography
Instructor: *William Kleber, DC DABCI*
- March 16-18, 2007** (St. Louis, MO)
DABCI Getaway Weekend
Location: *St. Louis Airport Marriott*
- March 24-25, 2007** (Kansas City, MO)
Immunology and Allergy, Part 1
Instructor: *William Kleber, DC DABCI*
- April 14-15, 2007** (Houston, TX)
Geriatrics
Instructor: *Jack Kessinger, DC DABCI*
- April 21-22, 2007** (Charlotte, NC)
Pharmacognosy (Herbal Therapy)
Instructor: *Dan Richardson, DC DABCI*
- April 28-29, 2007** (NUHS, Chicago, IL)
ABCI Board Testing
- April 28-29, 2007** (Kansas City, MO)
Allergy Part 2- Management of the Hypertensive Patient
Instructor: *Jack Kessinger, DC DABCI*
- May 5-6, 2007** (Houston, TX)
Urinary Disorders and Hair Biopsy Assessment
Instructor: *Frank Strehl, DC DABCI*
- May 5-6, 2007** (Charlotte, NC)
Chronic Degenerative Disease
Instructor: *Jack Kessinger, DC DABCI*
- May 19-20, 2007** (Kansas City, MO)
Common Diseases Affecting the Arterial System
Instructor: *Jack Kessinger, DC DABCI*
- June 2-3, 2007** (Houston, TX)
Immunology and Allergy, Part 1
Instructor: *William Kleber, DC DABCI*
- June 9-10, 2007** (Charlotte, NC)
Pediatrics
Instructor: *Frank Strehl, DC DABCI*
- June 16, 2007** (Chicago, IL)
Session 5 - Physical Examination Workshop
Instructors: *Cindy Howard, DC DABCI & Frank Strehl, DC DABCI*
- June 23-24, 2007** (Kansas City, MO)
Evaluating Vascular & Venous Disorders by Instrumentation
Instructor: *Jack Kessinger, DC DABCI*
- July 7-8, 2007** (Houston, TX)
Allergy Part 2 - Management of the Hypertensive Patient
Instructor: *Jack Kessinger, DC DABCI*
- July 14-15, 2007** (Charlotte, NC)
Spirometry and Pulmonary Disease
Instructor: *Jack Kessinger, DC DABCI*
- July 20-22, 2007** (Las Vegas, NV)
Symposium
- July 28-29, 2007** (Kansas City, MO)
Peripheral Vascular Disease Workshop
Instructor: *Tim McCullough, DC DABCI*
- August 4-5, 2007** (Houston, TX)
Common Diseases Affecting the Arterial System
Instructor: *Jack Kessinger, DC DABCI*
- August 11-12, 2007** (Charlotte, NC)
Geriatrics
Instructor: *Jack Kessinger, DC DABCI*
- August 25-26, 2007** (Kansas City, MO)
Facts of Neoplastic Process & Examining the Cancer Patient
Instructor: *Jack Kessinger, DC DABCI*
- September 8-9, 2007** (Houston, TX)
Evaluating Vascular & Venous Disorders by Instrumentation
Instructor: *William Kleber, DC DABCI*
- September 15-16, 2007** (Charlotte, NC)
Urinary Disorders and Hair Biopsy Assessment
Instructor: *Frank Strehl, DC DABCI*
- September 29-30, 2007** (Kansas City, MO)
Malignant Diseases, AIDS, & Their Management & Treatment
Instructor: *William Kleber, DC DABCI*
- October 6-7, 2007** (Houston, TX)
Peripheral Vascular Disease Workshop
Instructor: *Tim McCullough, DC DABCI*
- October 13-14, 2007** (Charlotte, NC)
Immunology and Allergy, Part 1
Instructor: *Jack Kessinger, DC DABCI*
- October 20-21, 2007** (Kansas City, MO)
Upper Gastrointestinal Disease
Instructor: *Jack Kessinger, DC DABCI*
- October 27-28, 2007** (Chicago, IL)
Introduction to Chiropractic Internal Disorders
Instructor: *Jack Kessinger, DC DABCI*
- November 3-4, 2007** (Houston, TX)
Facts of Neoplastic Process & Examining the Cancer Patient
Instructor: *Jack Kessinger, DC DABCI*
- November 10-11, 2007** (Charlotte, NC)
Allergy Part 2 - Management of the Hypertensive Patient
Instructor: *William Kleber, DC DABCI*
- November 17-18, 2007** (Kansas City, MO)
Lower Gastrointestinal Disease
Instructor: *Frank Strehl, DC DABCI*
- November 17-18, 2007** (Chicago, IL)
History Taking
Instructor: *Jack Kessinger, DC DABCI*
- December 1-2, 2007** (Houston, TX)
Malignant Diseases, AIDS, & Their Management & Treatment
Instructor: *William Kleber, DC DABCI*
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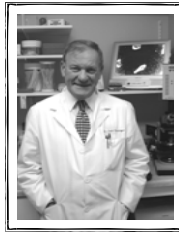
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From the Editor's Desk

by Jack Kessinger, DC, ND, DABCI



Dr. Jack Kessinger

Want to Live Longer? Avoid Cardiovascular Disease, Cancer & Doctors!

Just the other day I had a new patient who remarked that he “did not have a regular doctor.” I replied that “he would probably live longer.” On January 14, 2007, *Parade* magazine echoed the Center for Disease Control (CDC) report that the US is number one in only one category concerning health care and that is the amount of money spent. The US spends more money on health care than any other industrialized nation, yet lags far behind every other nation in overall health in all categories. The bad news is the other countries are not doing so hot either.

The *New England Journal of Medicine* (January 18, 2007; 356(3):213-15) reports that globally, the prevalence of chronic, non-communicable diseases is increasing at an alarming rate. About 58 million people die every year from cardiovascular disease, for which diabetes and hypertension are predisposing factors. Propelling the upsurge in cases of diabetes and hypertension is the growing prevalence of being overweight and obesity which *The Lancet* (2005; 366:1197-209) reports during the past decade has joined underweight, malnutrition and infectious disease as major health problems threatening the developing world. The aforementioned *New England Journal of Medicine* article also reports that in the last 20 years, the rate of obesity has tripled in developing countries that have been adopting a Western lifestyle involving decreased physical activity and over consumption of cheap, energy-dense food.

Population-based surveys of 75 communities in 32 countries show that diabetes is rare in communities in developing countries where a traditional lifestyle has been preserved. Until recently, the average people did not think that their diets could be accused of causing any health problems. It is not uncommon to read a statement from a reader to a “health” columnist that they are in

good health, but taking cholesterol lowering medications, blood pressure medications, etc.

Actually, the term “health care” is most commonly a misnomer. Think about this, a drug store advertises itself as a “Health Mart” with an almost unlimited supply of products listing an alarming number of unwanted adverse side effects. Furthermore, these so-called health products are usually found in the back of the store while in the front are items commonly recognized as unhealthy (i.e., cigarettes and other tobacco products, candy, soda pop, alcoholic beverages, etc.) that customers have to pass on their way to acquire drugs.

Doctors are the third-leading cause of death in the US, causing 225,000 deaths each year, according to the *Journal of the American Medical Association* (July 26, 2000; 284(4):483-5). Doctor Barbra Starfield of the Johns Hopkins School of Hygiene and Public Health conservatively describes how the US health system possibly caused deaths: 12,000 from unnecessary surgery, 7,000 from medication errors in hospitals, 20,000 from other errors in hospitals, 80,000 from nosocomial (hospital acquired) infections and 106,000 from the negative effects of properly prescribed drugs.

(Continued on next page)

As if these figures are not sobering enough, according to another report in the *New England Journal of Medicine* (March 26, 2005; 352(21):2211-21), “Most major drugs are effective in only 25-60% of patients and more than 2 million cases of adverse drug reactions occur annually in the United States, including 100,000 deaths.” In essence, up to 75% of drugs do not work as intended.

The leading cause of death in the US continues to be cardiovascular disease, and drugs have not helped; in fact, they often do damage. The *Journal of the American College of Cardiology* (2005; 46:1225-8) states that the introduction of statins nearly two decades ago marked a turning point in the effort to develop pharmacologic agents to reduce morbidity and mortality from coronary disease. However, despite the development of increasingly potent statins capable of markedly lowering cholesterol levels, coronary disease remains the leading cause of death in the Western societies. In addition to the obvious lack of help, several studies, like the one reported in the *Journal of the American Medical Association* (2003; 289:1651-90), relate that how statins injure is not clear, but they are associated with rhabdomyolysis and myositis and are exacerbated by hepatitis, renal, hypothy-

roid, and diabetes.

The *New England Journal of Medicine* (March 23, 2006; 354(12):1307-09) admits that, to date, no drugs specifically target arterial plaque. Even though it is well documented that healthy lifestyle habits, diet, nutritional supplements, essential fatty acids, exercise, and chelation therapy will reduce plaquing, the search continues for therapeutic interventions to act as anti-atherosclerosis agents.

Now, Bill Gates, the founder of Microsoft and a global health philanthropist, expressed his belief that there is no reason we can't cure the top 20 diseases. However, international health organizations will need to greatly expand their efforts, especially in low-income countries, to prevent and treat non-communicable diseases. It is amazing to me how someone who is smart enough to store a thousands songs on an instrument that is about the size of a matchbook cover can't understand that we already know what causes 90% of our illnesses. It certainly is not due to a deficiency of drugs or being born with too many parts. Dr. Julian Whitaker once remarked that if the doctor of today does not become the dietician of tomorrow, the dietician of today will become the doctor of tomorrow. ♦

The Legacy Continues



by A. Jay Kessinger IV, DC ND

Truth, justice and the American way. Faster than a speeding bullet. More powerful than a locomotive. Yada, yada, yada. Sounds like the prelude to Superman, with a splash of Seinfeld jargon, huh? Chiropractic kind of fits into this genre. American-born from the heart and loins of a Canadian immigrant. It may be looked at as an offshoot of the original osteopathic movement, another American innovation, however, chiropractic was, and continues to be, a distinct, stand-alone profession.

The concept of innate intelligence is the foundation of chiropractic's science and philosophy. It is each individual's portion of the universal intelligence, what is required to attain, then maintain optimal health. Interfering in natural matters impedes innate intelligence's ability to flow from producer to product and is the cause of natural disasters — sickness, disease, and death.

Unfortunately, there are a couple of movements in chiropractic pulling it away from its roots, which are firmly grounded in the expanding role of natural health care. One is to become either a limited subspecialty in the field of musculoskeletal maladies. The other is seeking to acquire prescriptive pharmaceutical rights which would most likely result in being absorbed into medicine like osteopathy.

The problem with chiropractic as a limited subspecialty is that the competition is fierce and their backing, no pun intended, is seemingly unlimited. Physical therapists are acquiring the same autonomy, state by state, that chiropractors have enjoyed since practicing chiropractic was legalized. Massage therapists are hot on the heels of physical therapists and are making inroads to the consumers' pocketbooks. Thus, chiropractors running musculoskeletal-only clinics are vulnerable to the economic pinch of the non-specific, non-diagnostic influence of professional massage therapists.

Against physical therapy's desire to legally manipulate the spine (and I assume massage therapists will also want to do this in the future), I personally see no long-lasting defense. If that were the case, in 1895, excluding osteopaths, no one would have been able to adjust the spine, and there you have almost the rest of the story. I just wonder why it is that every time a layperson manipulates the spine, and there's a problem, the headline states the injury was resulted from a "chiropractic adjustment." Why don't they attribute it to an "osteopathic manipulation?"

To continue, the problem with chiropractic, in my opinion, holding prescriptive pharmaceutical rights is that this could not help but cloud the public's understanding. Besides, aren't there enough dispensers of the Big Pharma out there already? What we, as full-scope chiropractors, do works. We never kill anybody. When patients present with a problem (disease), we diagnose it the same way any other science-based diagnostician does, then we strive to return them to normal through diet, exercise, specific nutritional supplementations, and chiropractic adjustments. With a natural approach to the return of normalcy, there is no need to just control an illness until it runs its course (i.e., death). Instead we are able to return our patients to homeostasis — the optimal health level of health for that person at that particular time and period of their life.

Maybe D.D. Palmer, the first DC, wouldn't have made a difference with the chiropractic services he offered to Mexico when it was in the midst of a rampant epidemic (he was denied by the American government), but he had the will to work toward much more than their sore backs. I submit that the chiropractic profession work toward the diagnostic principle parlayed by the DABCI council as the norm rather than the extraordinary standard. ♦

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TO SUBMIT ARTICLES

Book Review

The Natural Fat-Loss Pharmacy



by Harry Preuss, MD, MACN, CNS
and Bill Gottlieb

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Currently there are about fifty different nutrients, herbs, and food extracts on the market being sold as aids for weight loss, but there is shockingly little information for consumers concerning which of these supplements are helpful and which are downright harmful. This spring, all of that is changing with the publication of *The Natural Fat-Loss Pharmacy: Drug-Free Remedies to Help You Safely Lose Weight, Shed Fat, Firm Up, and Feel Great* (Broadway Books; January 9, 2007; Paperback Original; \$14.00). Harry Preuss, a doctor and university-based researcher, and Bill Gottlieb, former editor-in-chief at Rodale Books, have written the first and only reliable, science-based, practical guide to the nutritional supplements and herbs that can safely help you lose weight – and those that may be unsafe.

As a research fellow at the National Institutes of Health and an investigator for the American Heart Association, Harry Preuss, MD, MACN, CNS, is an authority on natural supplements. Having personally conducted extensive research on natural supplements in the laboratory and clinical trials, no one is better qualified to write this guide book. In *The Natural Fat-Loss Pharmacy*, Dr. Preuss explains the differences among the good, the useless, and the dangerous. He guides readers through the dozen or so supplements that really work, explains how to choose the ones that are best for any given individual, and gives advice on how to use them for maximum effect. He suggests which supplements work best for individuals depending on how much weight they need to lose, their food habits, age, body type, exercise and muscle profiles, as well as other pre-existing diseases and conditions.

Dr. Preuss advocates exercise and a healthy diet, but knows that people (including himself) need that extra helping hand to aid in weight loss and body maintenance. By reading *The Natural Fat-Loss Pharmacy*, you'll learn how to:

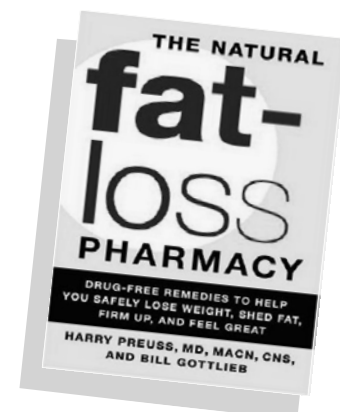
- Speed fat burning – with green tea extract
- Lose fat and build muscle without dieting or exercise – with CLA (conjugated linoleic acid)
- Stop weight regain – with MCT (medium-chain triglycerides)
- Reduce carbohydrate cravings – with 5-HTP (5-hydroxy L-tryptophan)
- Balance blood sugar for easier dieting – with chromium
- Block the absorption of excess starch and sugar – with white kidney bean extract and L-arabinose
- Reduce appetite and feel fuller faster – with chitosan
- Get off a plateau, where pounds don't seem to budge – with HCA (hydroxycitric acid)
- Turbocharge fat-burning exercise – with HMB (hydroxy methylbutyrate) or BCAA (branched-chain amino acids)

With the constant search for the "magic bullet" in weight-loss (too often at the risk of personal safety), *The Natural Fat-Loss Pharmacy* provides necessary, research-based information on the efficacy of numerous products that really work, and exposes those products that aren't likely to work or are possibly unsafe. It will be the go-to natural supplement handbook for years to come.

About the Authors

Harry Preuss, MD, MACN, CNS, is a tenured professor at Georgetown Medical Center. His current research centers on the use of dietary supplements and nutraceuticals to favorably influence or even prevent obesity, insulin resistance, and heart disease. The author of hundreds of medical papers and abstracts, he is co-author of The Prostate Cure (Crown 1998). He lives in Fairfax, Virginia.

Bill Gottlieb is the author of Alternative Cures (more than 1 million copies) and the co-author of several other health books. He lives in Middleton, California. ♦



Is Coral Calcium A Fraud?

by James A. Howenstine, MD

Several authorities in the field of natural health care have been very critical of coral calcium. The arguments raised against coral calcium include lack of efficacy, contamination by dangerous metals, and fraudulent advertising. Information about body pH changes in disease, oxygenation of body tissues, mineral reserves of the skeleton, and vitamin D status in US citizens can help resolve whether coral calcium might have value in therapy.

Importance of pH in Health and Disease States

The normal pH of the body ranges from 7.35-7.45. During rest, metabolism of the muscle tissues that support the body, heart contractions, and diaphragmatic movement continuously generate lactic acid that lowers pH. Dietary residues also usually generate acidic substances that must be eliminated to prevent chronic acidosis. Acidic waste products are removed by respiration (carbon dioxide), excretion by the kidneys, sweating, and fecal evacuation. Acidic states below the ideal slightly alkaline pH of 7.4 are rigorously corrected by the body using buffering minerals such as calcium, potassium, sodium, magnesium, strontium, cesium, and rubidium.

Persons living in developed societies spend most of their days in buildings and homes which have artificial light. Very little time is spent in sunlight, and when in the sun most persons wear sunglasses and are covered with sunscreen. This sets the stage for a serious deficiency of ultraviolet light and subsequent cholesterol-converted vitamin D, which is a critical nutrient for the body. Vitamin D is essential for proper absorption of minerals. When ultraviolet light is lacking, there is a failure to properly absorb alkaline minerals (calcium and magnesium) which the body stores in bone and keeps in reserve to prevent acidosis from becoming severe. Persons lacking ultraviolet light have a decreased ability to combat acidosis and lower vitamin D and calcium levels.

These stored alkaline minerals are a resource which enables the body to preserve a satisfactory pH when acidotic states occur. In the event of chronic acidosis, the body is able to preserve pH close to normal (7.4) by the steady release of calcium and magnesium from bone. Eventually, if the acidotic state does not resolve, the

body's bone mineral reserves become exhausted. By the time this becomes apparent, the bones have become weak and soft (osteoporosis), and the incidence of hip, spine, and other fractures skyrockets.

Nations (e.g., US, Germany, and Sweden) that eat a diet high in animal protein (meat, fish, and cheese) have 40 times more hip fractures than nations eating predominantly a vegetable and fruit diet (e.g., Thailand). This difference is explained by the fact that a high protein intake promotes acidosis, whereas a high fruit and vegetable diet results in alkalosis.

When the bone mineral reserves have been exhausted, the acidosis becomes worse, and there is a serious deficit of oxygen in body tissues. The consequences of this are the appearance of degenerative diseases including cancer, arteriosclerosis, allergic disorders, hypertension, depression, and arthritis.

Meat, poultry, fish, cheese, coffee, carbonated beverages, alcohol, cigarettes, sugars, bread, and cereals break down into acid residue. Alkaline residues are created from the intake of fruits, vegetables, eggs, nuts, seeds, legumes, milk, yogurt and herbal tea. Citrus fruits contain weak citric acid, but the alkaline minerals they contain more than balance the citric acid, resulting in an alkaline residue.

Every day we are exposed to tobacco smoke, pesticides, herbicides, fossil fuels, fluorohydrocarbons, carbon monoxide, acid rain, refined processed food, and sugar — all of which create acid residue. All pharmaceutical drugs are metabolized into acid residues.

Because alkaline fluids contain more oxygen than acidic fluids, the very slight increase in alkalinity from pH 7.3 to 7.45 raises the oxygen reserves carried by our blood by 65%. We are all wise to eat a preponderance of alkaline foods.

Genes dictate which degenerative disorders an individual is prone to develop, but the development of anoxia and chronic acidosis appears to play a more important role in the development of illness than does a person's genetic makeup. Genes are a convenient scapegoat for the failure of modern medicine to stop the rising tide of cancer, arteriosclerosis, Type 2 diabetes, and allergic diseases.

In acidotic states, potassium is unable to enter the cell, and the level of potassium in the blood level tends to be high normal or elevated. Because of the inability of potassium to enter the cells, the intake of neutral potassium

(Continued on next page)

chloride can be dangerous as it can cause a rise in blood potassium levels which may lead to heart stoppage (cardiac arrest). In alkalotic states, potassium readily enters the cell, and the blood potassium values tend to be low normal or below normal. The brilliant physician, Dr. Max Gerson, often administered three alkaline salts of potassium (gluconate, acetate, and phosphate) to successfully treat cancer patients. Many of these patients had elevated levels of potassium in their blood prior to the institution of potassium alkalizing salts. On first glance this might appear dangerous, but the alkalizing effects of the three potassium salts were immediate, and there was prompt movement of potassium into the cells preventing high levels of potassium from developing. Among the cured patients was Dr. Albert Schweitzer.

In 1924, Dr. Otto Warburg, a Nobel Prize winning physician, demonstrated that the development of cancer occurred in the absence of oxygen. When he lowered the oxygen level in tissues by 35 percent, cancer could be produced almost at will. When oxygen insufficiency is present in a cell, glucose ferments into two particles of lactic acid which greatly aggravates the already abnormally acidic interior of the cell. This intracellular environment of excess acid and lack of oxygen causes failure of normal DNA production. A carcinogen (free radicals from heavy metals, pesticides, synthetic manufactured fats, nitrites, tars from cigarettes, benzene, etc.) can enter these abnormal cells and cause a mutation of the DNA leading to a cancer cell.

Adequate stores of cellular calcium keeps the adhesion between cells intact. When calcium stores are lost, the ability of cells to bind together is impaired, and it becomes easier for cancer cells to spread locally — by blood and lymphatic channels (metastases).

The alkaline minerals (calcium, magnesium, potassium, sodium, strontium, and cesium) decrease the acidosis that leads to many diseases. Parts of the world lacking sunlight have a much higher incidence of cancer, multiple sclerosis, and allergic diseases than areas with plentiful sunlight. We need to realize that ultraviolet light is an important nutrient similar to food. Sunlight must not be blocked with sunscreens or sunglasses.

The tribes of the world known for longevity, with one exception (Okinawa), are found at high altitudes (Tibetans, Hunzas of North Pakistan, Armenians, Azerbaijanis, Georgians, and the Titicacas of Peru), and all are characterized by the drinking of eroded rocky water and the consumption of food grown with this water. This water contains larger than normal amounts of alkaline minerals (calcium, magnesium, potassium, cesium).

The Okinawa populace are growing food with and drinking water that has an alkaline pH. This water has a pH of 8.6¹ (very alkaline). Blood pH values in Okinawa are also higher than persons living in other nations. When a gram of fossilized stony coral minerals with 86 percent calcium is placed in a glass of distilled water the pH rises to 9.5-11.5.

The Hunzas and the Hopi Indians of Arizona do not get cancer unless they leave their homelands. The Hopis are protected by large amounts of alkaline rubidium in their food and water and the Hunzas by large amounts of alkaline cesium. The Hunzas and Hopis eat copious amounts of apricot pits which contain vitamin B₁₇ (laetrile). Persons eating B₁₇ never get cancer, and cancer therapy survivors who stay on laetrile (B₁₇) do not relapse.¹

Calcium has many important functions, including providing electrical energy for the heart to beat and muscles to move. The nourishing of cells depends on calcium facilitating the movement of nutrients into the cells. One of the most vital functions of calcium is involved in the replication of DNA. If calcium is lacking, poor repair of tissues and premature aging will result because of inadequate replication of DNA. Possibly the most critical use for calcium is in controlling the pH of the body. Acidic solutions have depleted oxygen levels, and alkaline solutions have an abundance of oxygen. The presence of calcium in tissues mops up acid and increases the oxygen in the tissue. Thus calcium contributes to increasing the alkalinity of body tissues and helps prevent the occurrence of cancer and other degenerative diseases.

In 1954, Dr. Carl J. Reich, an innovative Canadian physician, treated four patients with chronic diarrhea or asthma and hay fever that he felt had calcium deficiency. He treated them with intravenous calcium and vitamin D -rich halibut oil. These patients promptly recovered, as did a seven-year-old boy who had asthma from birth. Dr. Reich became convinced that mineral deficiency produced acidosis, leading to diseases. His studies suggested that many allergic conditions were actually caused by deficiencies of minerals and vitamin D. He began treating a wide variety of patients with megadosages of vitamins and minerals, especially calcium and Vitamin D, along with daily sun exposure, without sunscreens or sunglasses, for 1-2 hours. Many illnesses, including cancer, were greatly improved. Eventually this simple concept — that vitamins in large dosage could reverse or prevent diseases, including cancer — became so disturbing to the medical authorities his medical license was revoked.

Dr. Reich realized that when the body was lacking cal-

cium and minerals, bone was broken down under the influence of parathyroid hormone to create calcium. Calcium was also being removed from saliva, cerebrospinal fluid, and other body fluids to try to keep the blood pH normal at 7.4. A simple pH test of the saliva two hours after eating can disclose whether mineral deficiency may be present. Repeated test results in the very acidic pH (5.0) level suggest that alkaline mineral reserves are in danger of being depleted if the acidotic state is not corrected.

Free radicals are strongly felt to be responsible for many degenerative conditions (cancer, cataracts, arthritis, Parkinson's disease, Alzheimer's disease, arteriosclerosis, diabetes, etc.). Free radicals are electron deficient and have a positive charge. They are effectively mopped up by the presence of large amounts of negatively charged alkaline minerals in an alkaline pH and thus prevented from producing disease.

Coral polyps digest minerals and convert them into an organic substance that may be easier to absorb than conventional calcium supplements. In addition, they contain 73 other minerals often lacking in food grown from our trace mineral-depleted soil.

Serious depletion of soil minerals has occurred through-

out the world but is worst in North America. The farmers' financial struggles do not encourage the supplementation of soil with trace minerals. Lack of trace minerals impairs the function of enzymes, which do not work normally until the trace minerals are restored.

60% of US Citizens Have Vitamin D Deficiency

Lack of vitamin D has always been found among the elderly and the housebound. This deficiency is related to higher rates of cancer (breast, ovary, colon, and prostate), increased incidence of multiple sclerosis, progression of osteoarthritis, impairment of the immune response, high blood pressure, mood disorders (including serious depression), Type 1 diabetes, and tuberculosis. Lack of vitamin D appears to be a prime factor in the rising incidence of depression, along with a lack of omega 3 fatty acids in the diet. Patients with Parkinson's disease, multiple sclerosis, congestive heart failure, and Alzheimer's disease have all been found to have significant deficits of vitamin D.

In an important study from the March 19, 1998, *New England Journal of Medicine*, 290 patients admitted to Massachusetts General Hospital were studied for evidence of vitamin D deficiency. Fifty-seven percent of these patients were found to be deficient, and in 22% the deficiency was severe. In a subgroup of 77 healthy pa-

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tients with an average age of 44 years, 42% were vitamin D deficient, and in 11% the lack was severe.

A surprising and disconcerting finding in this study was that 46% of those regularly taking vitamin supplements were found to be lacking adequate vitamin D in their blood. Research by Dr. Reinhold Vieth appears to unravel this puzzle. Dr. Vieth and his colleagues at the University of Toronto have learned that any amount of vitamin D below 800² IU daily was unable to prevent vitamin D deficiency from occurring. He thinks that the proper dosage of vitamin D may be 1,200 IU or more. Coral complex is an exception, as this fossilized stony coral mineral complex contains 1,200 IU of vitamin D.

Osteoporosis is now being diagnosed in women in their 40s. These patients exhibit intense bone pain, muscle weakness, and even difficulty in walking. Some women with fibromyalgia, who have similar symptoms, have been discovered to have lower than normal bone density, which is very suspicious of osteoporosis. Twenty percent of the patients in rheumatologists' offices are suffering from fibromyalgia, which is a very common and very disabling problem for many women. All persons suspected of fibromyalgia should have vitamin D blood levels checked. Therapy is easy with vitamin D, calcium, and magnesium. However, for optimum health one should receive a complete whole food mineral complex.

Dr. G.A. Plotnikoff, of the University of Minnesota Medical School, measured 25 (OH) D [calcidiol] blood levels in 150 patients in a chronic pain clinic. He discovered that 100% of the black, Hispanic, East African, and American Indians in this clinic were vitamin D deficient³, and 93% of all patients were vitamin D deficient. Young women in their childbearing years were found to be at great risk for not being diagnosed. Many of these patients had been having pain for years without ever having vitamin D levels checked. This suggests that chronic musculoskeletal pain is often caused by undiagnosed vitamin D deficiency. Pigmented individuals appear to be at greater risk because they do not absorb ultraviolet light nearly as well as non-pigmented persons through their skin. Almost certainly the widespread use of sunglasses and sunscreens is contributing to this problem.

Persons living in intensely sunlit regions are not immune to vitamin D deficiency. A group of 360 chronic back pain patients in Saudi Arabia were treated with 5,000-10,000 units of cholecalciferol daily for three months. All reported improvement in their chronic pain⁴ and none became hypercalcemic. This was reported in the

prestigious journal, *Spine*.

A serious problem in the elderly is falls. These falls often cause hip fractures, which frequently prevent the patient from returning to independent living. Specific receptors for vitamin D have been identified in muscle tissue. Vitamin D deficiency results in muscle weakness and impaired balance, which contributes to falls in the elderly. A 49% reduction in falls⁵ was found in a geriatric facility containing 120 elderly women when 1,200 mg of vitamin D was added to the 1,200 mg of calcium given to all women. Muscle testing disclosed significant improvement in the group getting 1,200 units of vitamin D when compared to those getting only calcium. Magnesium should always be provided in the therapy of osteoporosis, as failure to do so can make the bone structure even more fragile. The correct ratio for this repletion should be about 2 mg of calcium for every 1 mg of magnesium replaced.

Blood pressure values are lower in the tropics, and blood pressure tends to rise in the winter when there is less sun exposure. Vitamin D appears to be a promising new therapy for hypertension⁶ that is safe, effective, and inexpensive. When the ultraviolet rays from the sun strike the skin, precholesterol reacts with the UVB wavelength of ultraviolet light to create cholecalciferol. The cholecalciferol is transported to the liver where it becomes calcidiol [25(OH)D]. The calcidiol is then transported to the kidneys where it is transformed into the steroid calcitriol, which enters the blood and regulates calcium⁷ in the body. Calcitriol regulates calcium metabolism in the body and has important beneficial effects in human development, diabetes, hypertension, heart disease, autoimmune illnesses, 13 different cancers, and depression.

Blood calcidiol levels⁸ should be measured to follow vitamin D status. Values of calcidiol between 35-50 ng/ml are normal. This vitamin greatly increases the absorption of minerals from the intestine.

Blood levels of calcidiol [25(OH)D] below 35 ng/ml are found in 70% of US citizens. Normal values are considered to be 35-50 ng/ml. In the northern US, no vitamin D is made during the six winter months when the sunlight is weak. Lifeguards and persons living near the equator have calcidiol values near 50 ng/ml. Toxicity from excess vitamin D during sun exposure does not occur because ultraviolet light degrades vitamin D after 20,000 units have been produced, thus leading to a steady state and no toxicity.

A young white male makes 20,000 units of vitamin D within minutes of whole body exposure to the summer

sun (before redness of skin appears). This is five times the amount of vitamin D considered capable of initiating toxic reactions⁹ by the Institute of Medicine, proving that these guidelines are set far too low. Dark-skinned individuals need 5-10 times longer in the sun to produce an equivalent amount of vitamin D depending, on the extent of their pigmentation.

Avoid sunburn, which can injure the skin. Sunscreens with a protection factor of 8 block 95% of vitamin D production. With equal sun exposure, an 80-year-old produces only 50% of the Vitamin D made by an 8-year-old.

Living in cities, working in factories, screening out ultraviolet light with glass windows, wearing more clothing than when farming, using sunscreens and dark glasses all contribute to the deficient vitamin D levels found today. One of the routes for ultraviolet light to be received by the body is through the eye. Ultraviolet deprivation may be responsible for the dramatic increase in cancer seen in certain parts of Africa. Dr. Albert Schweitzer noted that when he arrived in Africa, the natives did not wear sunglasses, and that he rarely saw cancer. Later natives could be seen pulling their canoes down the river wearing sunglasses and not much else.

The failure to receive ultraviolet light through their eyes because of sunglasses made them vulnerable to ultraviolet light deficiency, as black-skinned persons do not absorb much ultraviolet through their skin.

Dr. John Ott states that we need more ultraviolet light than we obtain from artificial light through windows. Ultraviolet light of the short wave length, germicidal ultraviolet, is mostly filtered out by the earth's atmosphere. This fear of getting too much ultraviolet light is creating a deficiency of an essential life supporting energy.¹⁰

Dr. Ott further relates that there is probably a relationship between chronic diseases and lack of sunlight: "My studies have shown that light is a nutrient, similar to all the other nutrients we take in through food, and that we need the full spectrum range of natural sunlight. If human skin is not exposed to solar radiation (direct or scattered) for long periods of time, disturbances will occur in the physiological equilibrium of the human system. The result will be disorders of the nervous system, vitamin D deficiency, weakening of the body's defenses, depression and an aggravation of chronic diseases."

He calls this state malillumination (lack of necessary
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sunlight). This develops when wavelengths are missing in various types of artificial light or are filtered from natural light passing through window glass, windshields, tinted eyeglasses, smog, and sunscreen lotions. The minerals and chemicals in the individual cells of our bodies that would normally be nourished by the missing wavelengths remain in the equivalent of darkness.

In other words, energy cannot be extracted from food materials if the proper wavelengths of light are not available to help break them down chemically.

Can Coral Calcium Help Patients?

In the testimonial section of a book by Robert Barefoot, *Death By Diet*, there are documented recoveries after taking coral calcium from a golf ball-sized neck malignancy (probably a lymphoma), lung cancer, prostate cancer (four cases), leukemia, fibromyalgia, simultaneous breast and colon cancer, brain tumor, heel spurs and a painful form of multiple sclerosis with high blood lipids.

Patients receiving coral calcium might be helped by at least three possible mechanisms. The first of these is alkalinization of the body. When a 1 g of coral calcium is placed in a glass of distilled water, the pH rises to 8.5-11.5. Raising the body pH could decrease the incidence of chronic diseases, thus benefiting health. Improved oxygenation and cellular nutrition could explain this. Better tissue oxygen levels might be expected to make it more difficult for malignant cells to survive. Persons with malignancies might wish to take three capsules of coral calcium daily at bedtime to bring their saliva pH up closer to 7.4. High doses of coral calcium (three capsules, three times daily) may have an adverse effect on renal function. A patient with pancreatic cancer went into acute tubular necrosis with uremia when taking three capsules of coral calcium three times daily along with 500 mg of CoQ 10 daily. This patient's pancreatic pain subsided in 24 hours.

A second way that health benefits could appear in persons taking coral calcium would be by correcting the mineral deficits found in chronic acidotic states. Restoring bone mineral losses would strengthen bones making fractures less likely. Correcting cellular mineral deficits should improve cellular metabolism. Coral calcium contains 71 minerals. The repletion of trace minerals lacking in the standard American diet should improve health and enzyme function.

At least 60% of the US population is lacking adequate stores of vitamin D. Every capsule of coral calcium contains 400 IU of vitamin D. Certainly any of the 60% of US citizens who lack vitamin D could be greatly helped

by the 1,200 units of vitamin D received from a three-capsule daily dosage. Many musculoskeletal pains might melt away with this dosage of vitamin D. My suspicion is that many of the persons improving while taking coral calcium therapy are actually persons with undiagnosed vitamin D deficiency.

Recently it has been learned that vitamin D is a potent antioxidant¹¹ active in fatty tissues as well as water. Certainly this antioxidant effect could also benefit the patient taking 1,200 IU of vitamin D from coral calcium.

There does not appear to be anything magical about the source of coral calcium. Coral from any part of the world should be just as effective as that found in Okinawa.

Recently I did an Internet search for "coral calcium". All the first 10 sites found were very critical of coral calcium. This criticism was primarily based on personal problems experienced by Robert Barefoot, but did not actually relate to coral calcium therapy. Any personal problems Mr. Barefoot has encountered should have nothing to do with whether coral calcium is a valuable health product or not. My impression is that the 1,200 IU dose of vitamin D in this product has helped many patients overcome a multitude of musculoskeletal problems caused by undiagnosed vitamin D deficiency

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A Simple Procedure Combining the Evaluation of Whole Body Sufficiency for Iodine With the Efficiency of the Body to Utilize Peripheral Iodide: The Triple Test

*by Guy E. Abraham, MD
and David Brownstein, MD*

Introduction

A re-evaluation of the role of the essential element iodine in medicine, called The Iodine Project, was initiated seven years ago.¹ One of the goals of The Iodine Project was to assess the optimal daily requirement of iodine for whole body sufficiency and optimal physical and mental well-being.^{2,3} In the process of testing the bioavailability of a tablet form of Lugol containing 12.5 mg elemental iodine, five normal subjects ingested 12.5 mg of the preparation, and iodide was measured in the 24-hour urine collection following ingestion of the tablet. The subjects excreted a mean of 20% of the ingested amount.⁴ This low recovery of iodide in the urine samples could be due to either low bioavailability of the product tested or high retention in the body.

According to medical textbooks, urinary iodide excretion is the best index of iodine intake. Therefore, according to medical textbooks, this low recovery of iodide was due to low bioavailability of the product tested. In order to elucidate the cause of this low iodide excretion, we continued the administration of the supplement in those subjects for one month. Then, we repeated the 24-hour urine collection and iodide was measured again in the 24-hour urine samples. The percentage of the oral dose excreted in the 24-hour urine sample increased significantly, with a mean group value of 50%.⁴ Contrary to medical textbooks, 80% of the iodine/iodide ingested was retained. After one month of supplementation, the body still retained 50% of the ingested amount. The iodine/iodide loading test evolved from these observations. However, instead of a one-month loading test, further studies were performed to shorten this test to a single ingestion of the preparation.³

For the loading test, the subjects ingest 50 mg of iodine/iodide and the percentage of the load excreted is evaluated by measuring the amount of iodide excreted in the 24-hour urine collection. Following orthoiodosupplementation at 12.5-50 mg/day, the percentage of the load excreted progressively increased over several months to

reach levels above 90% of the amount ingested. Because of the improved overall well-being reported by the subjects who achieved 90% or more iodide excretion, sufficiency was arbitrarily defined as 90%.³ Implementation of orthoiodosupplementation based on the loading test revealed that sufficiency was not achieved in some subjects even after two years of iodine supplementation at 1-2 tablets/day (12.5-25 mg iodine/day). Following a daily ingestion of 50 mg Lugol in a tablet form, most normal subjects achieved sufficiency by three months, retaining 1.5 g of iodide at sufficiency.⁵

In some patients, the pre-supplementation loading test suggested whole body iodine sufficiency because the percentage of the load excreted was 90 or greater, but these patients did not display the beneficial effects expected from iodine sufficiency. That was unexpected. These patients reported significant improvement in cognition, energy level, breast pain, and bowel movement following orthoiodosupplementation at 50 mg/day. But the repeat loading test 1-3 months post-supplementation showed a marked drop in the percentage of the load excreted. The clinical improvement did not follow the usual expected increase in the percentage of the load excreted. That was also unexpected. Follow-up with loading tests revealed increased excretion of the load in these patients to eventually reach sufficiency 6-9 months post-supplementation.

We evaluated one patient with high urinary excretion of the iodine load by collecting serial blood samples for 11 hours following the loading test.⁶ The patient, a 52-year-old woman (height 64 inches; weight 140 lbs.), had a past history of hyperthyroidism followed by hypothyroidism and had taken Synthroid 50 µg/day for five years. She developed side effects to orthoiodosupplementation and could tolerate only half a Lugol tablet/day (6.25 mg iodine/day) due to detoxification from elevated bromide levels. She was evaluated with serial serum samples before and after three months on a sustained released form of vitamin C at 3 g/day.

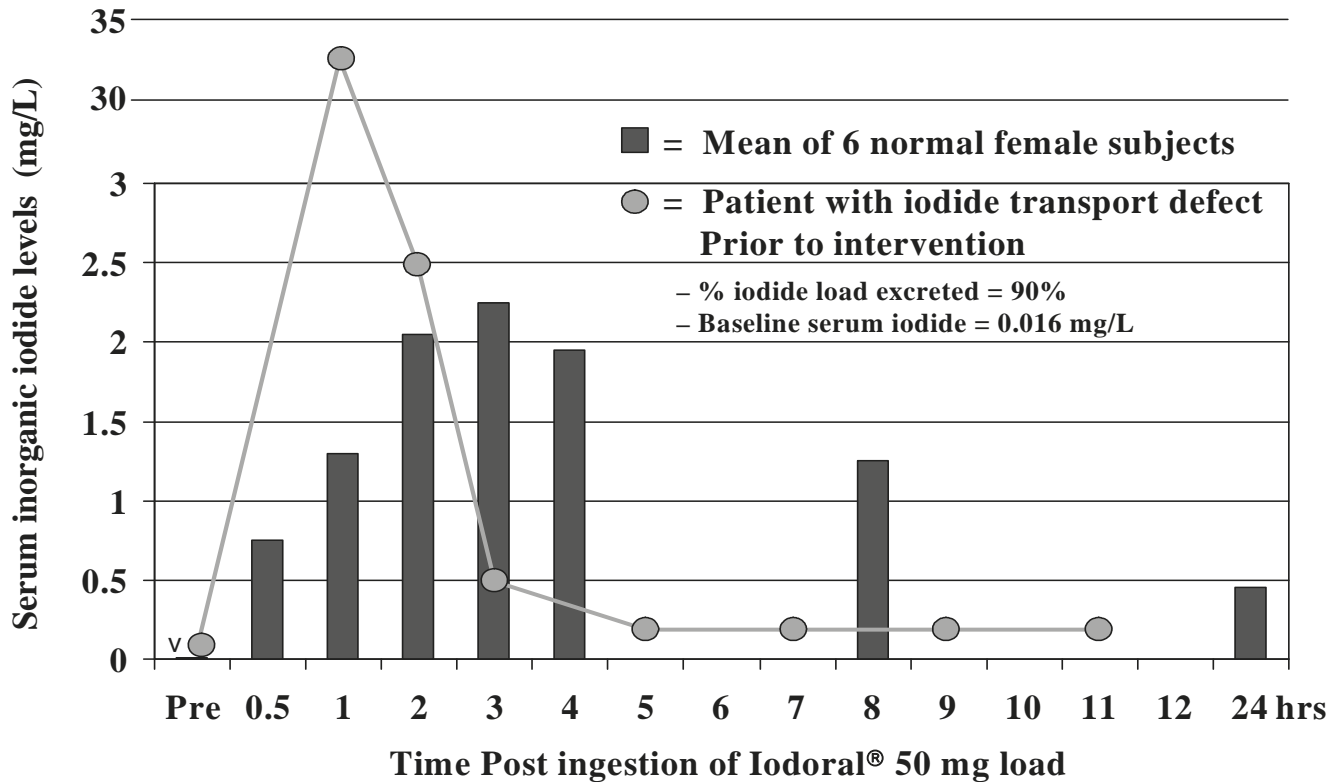
Pre-vitamin C loading test showed 90% of the load excreted in the urine, but her baseline serum iodide level was only 0.016 mg/L, compared to the expected levels of 0.85-1.34 mg/L in normal subjects who achieved whole body iodine sufficiency.⁵ The pattern observed in serum iodide levels pre- and post-vitamin C are displayed in Figure 1, superimposed on the mean value observed in six normal women.

Prior to intervention with vitamin C, the sharp peak of serum iodide at 32 mg/L at one hour post-load followed

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Figure 1

Serum Profile of Inorganic Iodide Levels following the Iodine/Iodide Load in Six Normal Female Subjects and a Patient with Iodide Transport Defect



The patient with the iodide transport defect excreted 90% of the iodine load, but her basal serum inorganic iodide level was very low — 0.016 mg/L. This pattern suggests a defect in the iodine retention mechanism. Following three months of intervention with sustained-release vitamin C at 3 g/day, she excreted 49.2% of the iodine load, and the baseline serum level was 0.42 mg/L, evidence of improved function of the iodine cellular transport mechanism.

by a rapid drop suggests that the gastrointestinal absorption of iodine was very efficient, but she was unable to transfer efficiently the serum iodide into the target cells. Following three months on vitamin C, the same test was repeated. The data revealed a normal profile of serum inorganic iodide levels. Her baseline serum inorganic iodide increased from 0.016 mg/L to 0.42 mg/L, and she retained 50% of the iodine load (49.2% recovered in 24-hour urine collection), compared to 10% of the load prior to supplementation with vitamin C.

To our knowledge, this was the first case report of a patient with evidence of a very defective retention mechanism for iodine who was studied with serial serum iodide levels prior to and following intervention. A combination of orthoiodosupplementation in amounts of iodine the patients could tolerate and administration of the antioxidant vitamin C via the oral route improved the performance of the iodine retention mechanism. Repair of a defective iodine cellular transport mechanism following orthoiodosupplementation combined with a complete nutritional program may explain our observation that in some cases a repeat loading test three months after orthoiodosupplementation resulted in a decreased percentage of the load excreted instead of the expected increase, even though the patients felt better on orthoiodosupplementation.

The milder forms of iodine retention inefficiency, either due to inefficient cellular uptake of peripheral iodide or inefficient utilization of intracellular iodide, will probably be overlooked until a more refined procedure is worked out to assess accurately the efficiency of the iodine transport and utilization mechanisms. Obviously, serial serum measurement of iodide would not be practical on a routine basis to evaluate patients with high percentage of the iodine load excreted prior to supplementation. A simple test was needed for the combined assessment of whole body sufficiency for iodine with the assessment of the efficiency of the body to utilize peripheral iodide.

Uptake and Utilization of Peripheral Iodide

The essential element iodine is present in every organ and tissue of the human body, not just the thyroid gland.⁷ Several cells beside the thyrocyte concentrate peripheral iodide against a gradient. So far the list of these iodide concentrating cells besides the thyrocyte has increased to include: white blood cells, salivary and lacrimal glands, ciliary body of the eye, renal cortex, the pancreas, the liver, gastric, small, and large intestinal mucosa, nasopharynx, choroid plexus, skin, adrenal cortex, mammary gland, placenta, uterus, and ovary.⁸ In the target cells studied, the mechanism used to concentrate peripheral

iodide involved an energy-dependant transport of one atom of iodide sandwiched between two atoms of sodium across the cell membrane.

Recently, a second mechanism for cellular transport of iodine has been reported by several investigators in the thyroid, mammary gland, and renal cortex, namely a chloride/iodide transporter identified as pendrin.⁸ The iodine transporter, pendrin, was speculated to function at the apical membrane of the cell. Rodriguez, *et al.*,⁹ identified a third human protein, homologous to NIS at the apical membrane of the human thyrocyte. This new protein does not catalyze the accumulation of iodide like NIS, but mediates its passive transfer. It was designated as human Apical Iodide Transporter (hAIT).

Among target cells for iodide uptake and utilization, the predominant research has focused on the thyrocyte. In the thyrocyte, the sodium/iodide symporter (NIS) is located in the basolateral membrane. The peripheral iodide enters the thyrocyte via the symporter in the basal membrane and crosses the thyrocyte as iodide to exit the thyrocyte via the apical membrane transporter just prior to oxidation and organification (Figure 2).

Iodide must bind to a site called the halide symporter binding site before cellular uptake. Other substances compete with iodide for these binding sites. These competing substances are called goitrogens, because they sometimes cause goiter by creating a relative iodide deficiency in the thyroid gland. These substances interfere also with iodide transport and utilization in several organs besides the thyroid gland and a better term would be iodide transport inhibitors and iodide utilization inhibitors instead of goitrogens, depending on whether the inhibition is at the cell membrane transport system or at intracellular sites of iodide oxidation and utilization (Table 1).

The Saliva/Serum Iodide Ratio

The salivary glands use a mechanism similar to the thyroid gland to concentrate peripheral iodide with subsequent oxidation and organification of iodide. Although the salivary glands can incorporate iodine in thyrosine to form mono- and di-iodothyrosine, they cannot couple iodinated thyrosine to form thyroid hormones.¹²

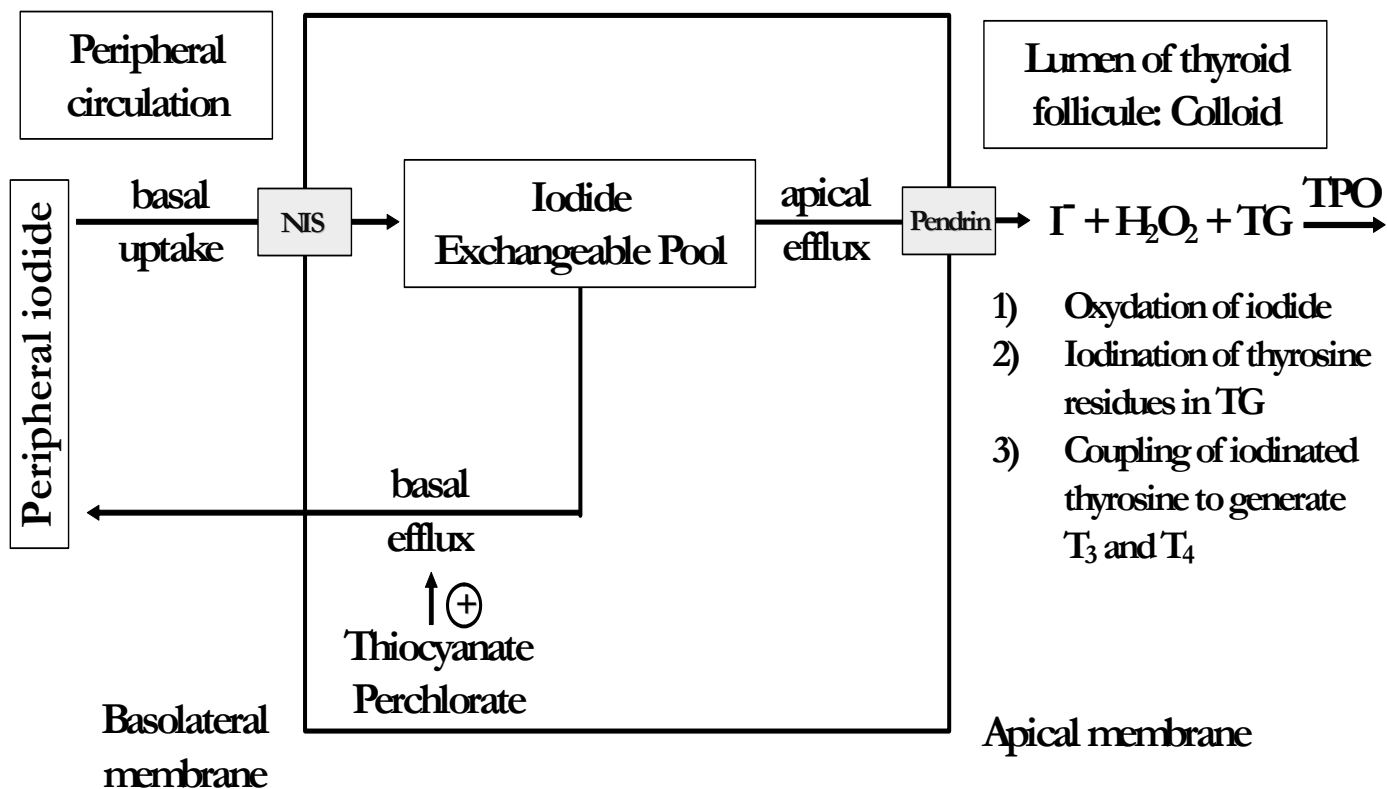
The saliva/serum iodide ratio measures the ability of the salivary glands to concentrate peripheral iodide. The assumption made is that the sal/ser ratio of iodide is an index of iodide uptake by target cells throughout the whole body.

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Figure 2

Uptake of Peripheral Iodide by the Thyrocyte followed by Oxidation and Organification for the Synthesis of the Thyroid Hormones

Thyrocyte



The saliva radioiodide/serum radioiodide ratio is used in neonates with elevated TSH and low thyroid hormones in order to confirm a congenital iodide symporter defect.¹³

The procedure involves injecting radioactive iodide in the neonate and measuring the ratio of radioactivity between saliva and serum. According to Viljder and Vulsma, a ratio >10 is considered normal; 3-10, borderline; and <3 is considered abnormal.

Stable iodide instead of radioactive iodide to assess the efficiency of the iodide transport system has not been previously reported because of technical difficulties in measuring low levels of iodine in biological fluids. Measurement of stable inorganic iodide in serum and saliva under standardized conditions seems the ideal procedure for fine tuning the assessment of iodide transport efficiency, and it is the least invasive way to assess response of the symporter function following intervention. This approach would obviate the need to expose the patient to radioactive iodide. A ratio near unity would indi-

cate a severe defect/damage/inhibition of the symporter function.

We previously reported a procedure to measure saliva and serum inorganic iodide 24 hours following ingestion of 50 mg of iodine in the form of Lugol tablets.⁸ The normal range of saliva/serum ratios was 28-74 with a mean of 44.2 ± 12.7 in 14 normal subjects. Low saliva/serum ratios were observed in breast cancer patients with high serum bromide levels. Orthoiodosupplementation at 50-100 mg/day resulted in decreased serum bromide and increased saliva/serum ratio.

In some patients with autoimmune thyroiditis and hypothyroidism, an unexpectedly high saliva/serum iodide ratio was observed (>74) concomitant with a high pre-supplementation excretion of the iodine/iodide load.¹⁴ The only explanation would be a normal transport but deficient oxidation and organification of iodide resulting in an increased exchangeable pool of iodide.

Currently, the procedure used to assess organification

defect of the thyroid gland, called the thiocyanate or perchlorate discharge test, is to give the patient radioiodide followed a few hours later by an oral dose of 1 g potassium thiocyanate or 1 g of potassium perchlorate. The percentage of radioactivity in the thyroid is measured before and after iodide is discharged by thiocyanate or perchlorate.^{10,11} Thiocyanate and perchlorate not only block the uptake of iodide by the thyroid gland but also cause a discharge of the inorganic iodide present in the thyroid. Under normal conditions, symported iodide is quickly organified and the amount of radioactive iodide discharged following thiocyanate and perchlorate is insignificant. When there is a blockage of organification of iodide, the percentage of radioiodide discharged from the thyroid gland is usually greater than 50% due to an increase in the exchangeable pool of iodine.

The Triple Test

The expected levels and ratios of serum iodide and of the inorganic and protein bound forms of saliva iodine in patients with deficient uptake and/or utilization of iodide are displayed in Table 2. In the case of inefficient symport of iodide inside the cell, but with normal oxidation and organification of the symported iodide, the expected results displayed in Table 2 under Subsection A would apply. In the case of normal symport of iodide with inefficient oxidation and organification, the results displayed in Subsection B would apply. In the case of inefficiency of both symport and utilization of iodide, the results displayed in Subsection C would apply.

The advantages of the Triple Test procedure over the currently used thiocyanate¹⁰ and perchlorate¹¹ discharge test to identify organification defect is that the Triple Test will obviate the need to inject radioiodide into the patient followed by administration of these iodine uptake inhibitors. Also, as displayed in Table 2, the radioiodide discharge test would be normal in patients with a combined inefficiency of uptake and utilization of iodide and also in patients with inefficient symport and normal organification, whereas the Triple Test could identify patients with these defects.

The Triple Test involves the collection of urine for 24 hours following ingestion of 50 mg of elemental iodine in the form of Lugol tablets. At the end of the 24-hour period, serum and saliva samples are collected. Inorganic iodide is measured in the 24-hour urine collection, in the serum, and in the saliva samples by the ion-selective electrode procedure as previously described.¹³ The saliva sample is processed further for the measurement of total iodine, both inorganic and protein bound iodine.

Collection of Urine Sample

Discard the first morning urine of Day 1. Take four tablets of Iodoral[®] 12.5 mg with a glass of water. Collect all urine samples for 24 hours following ingestion of the loading dose. Include the first morning urine on Day 2. At the end of the 24-hour collection, shake the 3-liter bottle well. Measure the volume of urine in the 3-liter bottle by looking at markings on side of bottle, and pour 1-2 ounces into a 2-ounce plastic bottle. Two plastic 2-ounce bottles should be supplied in case the 24-hour urine volume is greater than 3 L. Have the patient return the other empty 2-ounce bottle with your package, if the total volume is 3 L or less.

If the 3-liter bottle is full before the end of the collection, the same 3-liter bottle can be used to continue collection after measuring the total volume. Measure the volume of urine in the 3-liter bottle by looking at markings on the side of bottle. Pour 1-2 ounces of urine in the 2-ounce plastic bottle. Write the name, date, and volume of urine on the 2-ounce bottle. Write on the label, "Part 1 of 2 collections." Then discard the urine in 3-liter bottle. Use the same 3-liter bottle to continue collection. At the end of 24-hour collection, measure the volume of the urine again. Pour 1-2 ounces of urine into the second 2-ounce plastic bottle. Write the name, date, and volume of urine on the 2-ounce bottle. Write on the label, "Part 2 of 2 collections. When the patient's information form is completed, under *total volume*, write: "Collected in 2 parts, both sample bottles enclosed."

The concentration of iodide in the 24-hour urine collection will be the sum of both values obtained in the two specimens. Please note that in this case, two measurements will be performed instead of one to compute the percentage of the load excreted. For example, if you measure 6 mg/L in Part 1 with a volume of 3 L, and you measure 4 mg/L in Part 2 with a volume 1.5 L. The computation of the total amount of iodide excreted is:

$$\text{Part 1} = 6 \text{ mg/L} \times 3 \text{ L} = 18 \text{ mg}$$

$$\text{Part 2} = 4 \text{ mg/L} \times 1.5 \text{ L} = 6 \text{ mg}$$

$$\text{Total} = 24 \text{ mg}$$

$$\text{Percent excreted} = 48\%$$

The food grade coloring FD&C Green #3 is added at a concentration of 1 ml of a 1% solution per liter of urine. It is used for its antiseptic properties and also as a marker for spent chromatography cartridges.⁶ Sodium azide at a final concentration of 0.01% is usually the antiseptic used during collection of urine samples.²³ FD&C Green #3 and sodium azide can both be used for maximum inhibition of mold and bacteria. The com-

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Table 1
Some Examples of Interferences
with Iodine Uptake and Utilization

- A) Cellular Uptake Inhibitors¹**
- Perchlorate
 - Fluoride
 - Bromide
 - Thiocyanate
- B) Cellular Utilization Inhibitors**
- 1) TPO inhibitors¹
 - Thionamides (Antithyroid drugs)
 - Goitrin
 - Bromide
 - Thiocyanate
 - 2) Decreased H₂O₂ production
 - FAD deficiency¹⁰
 - NADPH-cyt c reductase deficiency¹¹

bined addition of FD&C Green #3 and sodium azide is used routinely for urine, serum, and saliva samples in our R&D Lab. An aqueous solution containing 15% sodium azide and 1% FD&C Green #3 is added to urine samples at one drop/ounce and to serum and saliva samples at 1 drop per 5-8 ml.

Serum Collection

Plasma contains fibrinogen which may coagulate and impair the flow of eluate through the cartridges during chromatographic separation of the halides. This causes an increased pressure in the cartridges which results in a distortion of the elution profile. For this reason, serum samples are recommended. After venipuncture and collection of 10-15 ml of whole blood in red top tubes without separator gel, let the blood clot and retract. Centrifuge the clotted blood. Decant 5-8 ml of serum in plastic tubes containing one drop of an aqueous solution containing 15% sodium azide and 1% FD&C #3.

Saliva Collection

After rinsing the mouth with water two to three times to remove any food particles, collection of saliva for a total volume of 5-8 ml can be done passively by letting it flow through a straw into a test tube or by spitting into a container with a wide opening. One drop of an aqueous solution containing 15% sodium azide and 1% FD&C Green #3 is added to the collecting vial. Xylitol and citric acid which increase saliva flow may be used in patients who cannot produce enough saliva within 5-10 minutes. Centrifuge saliva to remove coagulated mucus which interferes with the chromatographic separation of

the halides. The biological fluids are stable for up to one week at room temperature when collected in containers with sodium azide and FD&C Green #3. However, it is best to freeze the samples if they are not processed within 48 hours of collection.

Comments

Combining the percentage of the iodine load excreted in the 24-hour urine collection with the measurements of serum and saliva inorganic iodide 24 hours post-load gives an assessment of iodine sufficiency of the whole body and also efficiency of the cellular uptake and utilization of peripheral iodide.

The iodide-selective electrode used in our laboratory is specific for inorganic iodide, and therefore, will not measure protein-bound iodine. The ICP-MS procedure measures total iodine and could be used to compute organic iodine by subtracting the amount of iodine measured by ICP-MS from the level measured by the ion-selective electrode procedure. Measuring protein-bound iodine (PBI) in saliva can also be performed directly by the appropriate technique. The value obtained by ICP-MS should be equal to the sum of the value obtained by the ion-selective electrode procedure and the value obtained by the PBI measurement.

We are currently collecting samples from normal subjects and patients with various clinical conditions in order to standardize the normal range of organic/total iodine ratio and to correlate these levels with clinical response following nutritional intervention in patients with abnormal ratios.

About the Author

Guy E. Abraham, MD, is a former Professor of Obstetrics, Gynecology, and Endocrinology at the UCLA School of Medicine. Some 35 years ago, he pioneered the development of assays to measure minute quantities of steroid hormones in biological fluids. He has been honored as follows: General Diagnostic Award from the Canadian Association of Clinical Chemists, 1974; the Medaille d'Honneur from the University of Liege, Belgium, 1976; the Senior Investigator Award of Pharmacia, Sweden, 1980. The applications of Dr. Abraham's techniques to a variety of female disorders have brought a notable improvement to the understanding and management of these disorders.

Twenty-five years ago, Dr. Abraham developed nutritional programs for women with premenstrual tension syndrome and post-menopausal osteoporosis. They are now the most commonly used dietary programs by

American obstetricians and gynecologists. Dr. Abraham's current research interests include the development of assays for the measurement of iodide and the other halides in biological fluids and their applications to the implementation of orthoiodosupplementation in medical practice.

David Brownstein, MD, is a family physician who utilizes the best of conventional and alternative therapies. He is the Medical Director for the Center of Holistic Medicine in West Bloomfield, Michigan. A graduate of the University of Michigan and Wayne State University School of Medicine, Dr. Brownstein is board certified by the American Academy of Family Physicians. He is a member of the American Academy of Family Physicians and the American College for the Advancement in Medicine. Over the past few years, he has had extensive experience in the use of orthoiodosupplementation in his practice.

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Table 2 Expected Finding in Cases of Inefficient Symport, Inefficient Utilization, and a Combination of the Two

- A) **Inefficiency of iodide transport with normal oxidation and organification of iodide**
 - Decreased intracellular iodide
 - Decreased exchangeable pool of iodide
 - Decreased saliva iodide
 - Decreased sal/ser iodide ratio
 - Normal saliva organic iodine/total iodine ratio
 - Normal nitrate and perchlorate discharge test
 - Hypothyroidism and goiter in severe cases only
- B) **Inefficiency of oxidation of iodide with normal transport**
 - Increased intracellular iodide
 - Increased exchangeable pool of iodide
 - Increased saliva iodide
 - Increased sal/ser iodide ratio
 - Decreased saliva organic iodine/total iodine ratio
 - Increased discharge of iodide following nitrate and perchlorate
 - Hypothyroidism and goiter in severe cases only
- C) **Combined inefficiency in transport, oxidation, and organification of iodide**
 - Decreased intracellular iodide
 - Decreased exchangeable pool of iodide
 - Decreased saliva iodide
 - Decreased sal/ser iodide ratio
 - Decreased saliva organic iodine/total iodine ratio
 - Normal nitrate and perchlorate discharge test
 - Hypothyroidism and goiter in severe cases only

The Clinical Importance of Vitamin D (Cholecalciferol): A Paradigm Shift with Implications for All Healthcare Providers

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Introduction and Overview

While we are all familiar with the importance of vitamin D in calcium absorption and bone metabolism, many doctors and patients are not aware of the recent research on vitamin D and the widening range of therapeutic applications available for cholecalciferol, which can be classified as both a vitamin and a pro-hormone. Additionally, we also now realize that the Food and Nutrition Board's previously defined Upper Limit (UL) for safe intake at 2,000 IU/day was set far too low and that the physiologic requirement for vitamin D may be as high as 5,000 IU/day, which is less than half of the >10,000 IU that can be produced endogenously with full-body sun exposure.^{1,2} With the discovery of vitamin D receptors in tissues other than the gut and bone—especially the brain, breast, prostate, and lymphocytes—and the recent research suggesting that higher vitamin D levels provide protection from diabetes mellitus, osteoporosis, osteoarthritis, hypertension, cardiovascular disease, metabolic syndrome, depression, several autoimmune diseases, and cancers of the breast, prostate, and colon, we can now utilize vitamin D for a wider range of preventive and therapeutic applications to maintain and improve our patients' health.³ Based on the research reviewed in this article, the current authors believe that assessment of vitamin D status and treatment of vitamin D deficiency with oral vitamin D supplements should become a routine component of clinical practice and preventive medicine. Vitamin D supplementation with physiologic doses of 2,000 to 5,000 IU per day for adults is clinically safe and physiologically reasonable since such doses are less than that obtained by full-body sun exposure. Periodic assessment of serum 25-OH-vitamin D (25(OH)D) and serum calcium will help to ensure that vitamin D levels are safe and effective for health maintenance and disease prevention. Clinical research supporting the use of vitamin D in the management of type 2 diabetes, osteoporosis, osteoarthritis, hypertension, car-

diovascular disease, metabolic syndrome, multiple sclerosis, polycystic ovary syndrome, musculoskeletal pain, depression, epilepsy, and the prevention of cancer and type 1 diabetes is presented along with our proposal for the interpretation of serum 25(OH)D laboratory values.

Basic Physiology of Vitamin D

Vitamin D is obtained naturally from two sources: sunlight and dietary consumption. Vitamin D₃ (cholecalciferol) is the form of vitamin D produced in the skin and consumed in the diet. Vitamin D₂ (ergocalciferol), which is produced by irradiating fungi, is much less efficient as a precursor to the biologically active 1,25-dihydroxyvitamin D (calcitriol). Additionally, since ergocalciferol forms a number of metabolic by-products that are not naturally found in humans, it is potentially more toxic than cholecalciferol.⁴ Although ergocalciferol is occasionally used clinically and in research studies, cholecalciferol is the preferred form of supplementation and will be implied in this article when supplementation is discussed.

Vitamin D can be described as having two pathways for metabolism: one being "endocrine" and the other "autocrine," (within the cell) and perhaps "paracrine" (around the cell). This elucidation, recently reviewed by Heany,⁵ is vitally important in expanding our previously limited conception of vitamin D from only a "bone nutrient with importance only for the prevention of rickets and osteomalacia" to an extraordinary molecule with far-reaching effects in a variety of cells and tissues. Furthermore, Heany's distinction of "short-latency deficiency diseases" such as rickets from "long-latency deficiency diseases" such as cancer provides a conceptual handle that helps us grasp an understanding of the differences between the acute manifestations of severe nutritional deficiencies and the delayed manifestations of chronic subclinical nutritional deficiencies.⁵

In its endocrine metabolism, vitamin D (cholecalciferol) is formed in the skin following exposure to sunlight and then travels in the blood to the liver where it is converted to 25-hydroxyvitamin D (calcidiol, 25(OH)D) by the enzyme vitamin D-25-hydroxylase. 25(OH)D then circulates to the kidney for its final transformation to 1,25-dihydroxyvitamin D (calcitriol) by 25-hydroxyvitamin D₃-1 α -hydroxylase (1-OHase).⁶ Calcitriol is the most biologically active form of the vitamin and increases intestinal calcium absorption, increases phosphorus absorption, promotes calcium deposition in bone, and promotes a reduction in parathyroid hormone (PTH). While increased calcium absorption is obviously important for

nutritional reasons, suppression of PTH by vitamin D is also clinically important since relatively lower levels of PTH appear to promote and protect health, and higher levels of PTH correlate with increased risk for myocardial infarction, stroke, and hypertension.^{7,8} Relatedly, Fugita⁹ described the “calcium paradox” wherein elevations of PTH cause an increase in intracellular calcium and may thereby promote the cascade of cellular dysfunction that can contribute to the development of diabetes mellitus, neurodegenerative diseases, malignancy, and degenerative joint disease.

In its autocrine metabolism, circulating 25(OH)D is taken up by a wide variety of cells that contain both 1-OHase as well as nuclear vitamin D receptors (VDR). Therefore, these cells are able to make their own calcitriol rather than necessarily relying upon hematogenous supply. Cells and tissues that are known to contain 1-OHase, and which therefore make their own calcitriol, include the breast, prostate, lung, skin, lymph nodes, colon, pancreas, adrenal medulla, and brain (cerebellum and cerebral cortex).^{3,10} Cells and tissues with nuclear, cytosolic, or membrane-bound VDR include islet cells of the pancreas, monocytes, transformed B-cells, activated T-cells, neurons, prostate cells, ovarian cells, pituitary cells, and aortic endothelial cells.¹¹ Indeed, given the wide range of cells and tissues that metabolize vitamin D in an autocrine manner, we see that there is biological potential for vitamin D to influence function and pathophysiology in a wide range of processes and disease states.

Since many cells and tissues of the body have the ability to metabolize vitamin D, we should not be surprised that vitamin D plays a role in the function of these cells. Calcitriol is known to modulate transcription of several genes, notably those affecting differentiation and proliferation such as *c-myc*, *c-fos*, and *c-sis*;⁶ this may partially explain the inverse relationship between sun exposure and cancer mortality.¹² Vitamin D appears to modulate neurotransmitter/neurologic function as shown by its antidepressant¹³ and anticonvulsant¹⁴ benefits. Vitamin D is obviously immunoregulatory as manifested by its ability to reduce inflammation,¹⁵ suppress and/or prevent certain autoimmune diseases,¹⁶⁻¹⁸ reduce the risk for cancer,¹² and may even reduce the severity and frequency of infectious diseases, such as acute pneumonia in children.¹⁹

Clinical Applications and Therapeutic Benefits of Vitamin D

Support for a broad range of clinical applications for vitamin D supplementation comes from laboratory experiments, clinical trials, and epidemiologic surveys.

Despite the imperfections of current data, we can still see significant benefits from vitamin D supplementation in a variety of human diseases, as briefly reviewed below.

Cardiovascular Disease: Deaths from cardiovascular disease are more common in the winter, more common at higher latitudes and more common at lower altitudes, observations that are consistent with vitamin D insufficiency.²⁰ The risk of heart attack is twice as high for those with 25(OH)D levels less than 34 ng/ml (85 nmol/L) than for those with vitamin D status above this level.²¹ Patients with congestive heart failure were recently found to have markedly lower levels of vitamin D.

Hypertension: It has long been known that blood pressure is higher in the winter than the summer, increases at greater distances from the equator and is affected by skin pigmentation—all observations consistent with a role for vitamin D in regulating blood pressure.²² When patients with hypertension were treated with ultraviolet light three times a week for six weeks their vitamin D levels went up 162% and their blood pressure fell significantly.²³ Even small amounts of oral cholecalciferol (800 IU) for eight weeks lowered both blood pressure and heart rate.²⁴

Type 2 Diabetes: Hypovitaminosis D is associated with insulin resistance and beta-cell dysfunction in diabetics and healthy young adults.²⁵ Healthy adults with serum 25 (OH)D levels above 40 ng/ml (100 nmol/L) had significantly lower 60 minute, 90 minute and 129 minute postprandial glucose levels and significantly better insulin sensitivity. The authors concluded that the 60% improvement in insulin sensitivity afforded by vitamin D appears to be “more potent than either troglitazone or metformin,” two medications commonly prescribed to treat type 2 diabetes. In fact, supplementation with even small amounts of cholecalciferol (1,332 IU) for a short time (30 days) resulted in significant improvements in insulin secretion.²⁶

Osteoarthritis: Many practitioners know that vitamin D helps prevent and treat osteoporosis, but fewer know that the progression of osteoarthritis, the most common arthritis, is lessened by adequate blood levels of vitamin D. Framingham data showed osteoarthritis of the knee progressed more rapidly in those with 25(OH)D levels lower than 36 ng/ml (90 nmol/L).²⁷ Another study found that osteoarthritis of the hip progressed more rapidly in those with 25(OH)D levels lower than 30 ng/ml (75 nmol/L).²⁸

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Multiple Sclerosis: Since the autoimmune/inflammatory disease multiple sclerosis (MS) is notably rare in sunny equatorial regions and becomes increasingly prevalent among people who live farther from the equator and/or who lack adequate sun exposure, it is not surprising to find that vitamin D deficiency is common among patients with MS. In a clinical trial with 10 MS patients, Goldberg, Fleming, and Picard¹⁷ prescribed daily supplementation with approximately 1,000 mg calcium, 600 mg magnesium, and 5,000 IU vitamin D (from 20 g cod liver oil) for up to two years and found a reduction in the number of exacerbations and an absence of adverse effects. This is one of very few studies in humans that employed sufficient daily doses of vitamin D (5,000 IU) and had sufficient duration (2 years). More recently, Mahon *et al*²⁹ gave 800 mg calcium and 1,000 IU vitamin D per day for six months to 39 patients with MS and noted a modest anti-inflammatory effect.

Prevention of Type 1 Diabetes: Type 1 diabetes is generally caused by autoimmune/inflammatory destruction of the pancreatic beta cells. Vitamin D supplementation shows significant preventive and ameliorative benefits in animal models of type 1 diabetes. In a study with more than 10,000 participants, Hypponen *et al*¹⁶ showed that supplementation in infants and children with 2,000 IU of vitamin D per day reduced the incidence of type 1 diabetes by approximately 80%. Relatedly, several studies using cod liver oil as a rich source of vitamin D have also documented dramatic reductions in the incidence of type 1 diabetes.

Depression: Seasonal affective disorder (SAD) is a particular subtype of depression characterized by the onset or exacerbation of melancholia during winter months when bright light, sun exposure, and serum 25(OH)D levels are reduced. Recently, supplementation with 100,000 IU of vitamin D was found superior to light therapy in the treatment of SAD after one month.³⁰ Similarly, in a study involving 44 subjects, supplementation with 400 or 800 IU per day was found to significantly improve mood within five days of supplementation.¹³

Epilepsy: Seizures can be the presenting manifestation of vitamin D deficiency.³¹ Hypovitaminosis D decreases the threshold for and increases the incidence of seizures, and several “anticonvulsant” drugs interfere with the formation of calcitriol in the kidney and further reduce calcitriol levels via induction of hepatic clearance. Therefore, antiepileptic drugs may lead to iatrogenic seizures by causing iatrogenic hypovitaminosis D.³² Conversely, supplementation with 4,000 – 16,000 IU per day of vitamin D2 was shown to significantly reduce seizure

frequency in a placebo controlled pilot study by Chirstansen, *et al*.¹⁴

Migraine Headaches: Calcium clearly plays a role in the maintenance of vascular tone and coagulation, both of which are altered in patients with migraine. Thys-Jacobs³³ reported two cases showing a reduction in frequency, duration, and severity of menstrual migraine attacks following daily supplementation with 1,200 mg of calcium and 1,200 – 1,600 IU of vitamin D in women with vitamin D deficiency.

Polycystic Ovary Syndrome: Polycystic ovary syndrome (PCOS) is a disease seen only in humans and is characterized by polycystic ovaries, amenorrhea, hirsutism, and obesity. *In vivo* studies have shown that calcium is essential for oocyte activation and maturation. Vitamin D deficiency was highly prevalent among 13 women with PCOS, and supplementation with 1,500 mg of calcium per day and 50,000 IU of vitamin D2 on a weekly basis normalized menstruation and/or fertility in nine of nine women with PCOS-related menstrual irregularities within three months of treatment.³⁴

Musculoskeletal Pain: Patients with non-traumatic, persistent musculoskeletal pain show an impressively high prevalence of overt vitamin D deficiency. Plotnikoff and Quigley³⁵ recently showed that 93% of their 150 patients with persistent, nonspecific musculoskeletal pain were overtly deficient in vitamin D. Masood *et al*³⁶ found a high prevalence of vitamin D deficiency in children with limb pain, and vitamin D supplementation ameliorated pain within three months of supplementation. Faraj and Al Mutairi³⁷ found vitamin D deficiency in 83% of their 299 patients, and supplementation with 5,000 – 10,000 IU of vitamin D per day lead to pain reduction in nearly 100% of patients after three months.

Critical Illness and Autoimmune/Inflammatory Conditions: Deficiency of vitamin D is common among patients with inflammatory and autoimmune disorders. In addition to the previously mentioned epidemic of vitamin D insufficiency in patients with MS, we also see evidence of vitamin D insufficiency in most patients with Grave’s disease,³⁸ ankylosing spondylitis,³⁹ systemic lupus erythematosus,⁴⁰ and rheumatoid arthritis. Clinical trials with proper dosing and duration need to be performed in these patient groups. A recent trial of vitamin D supplementation in patients with prolonged critical illness showed a significant and dose-dependent “anti-inflammatory effect” evidenced by reductions in IL-6 and CRP.⁴¹ However, the insufficient dose of only 400 IU per day (administered intravenously) for only ten days precluded more meaningful and beneficial results,

and we present guidelines for future studies later in this paper.

Cancer Prevention and Treatment: The prevalence of many human cancers is inversely proportional to exposure to ultraviolet light and serum vitamin D levels. Vitamin D has anti-cancer effects mediated by anti-proliferative and proapoptotic mechanisms which are augmented by modulation of nuclear receptor function and enzyme action,⁴² and limited research shows that synthetic vitamin D analogs may have a role in the treatment of human cancers.⁴³ Grant¹² has shown that inadequate exposure to sunlight, and hence hypovitaminosis D, is associated with an increased risk of cancer mortality for several malignancies, namely those of the breast, colon, ovary, prostate, bladder, esophagus, kidney, lung, pancreas, rectum, stomach, uterus, and non-Hodgkin lymphoma. He proposes that adequate exposure to ultraviolet light and/or supplementation with vitamin D could save more than 23,000 American lives per year from a reduction in cancer mortality alone.

The aforementioned clinical trials using vitamin D in a wide range of health conditions have helped expand our concept of vitamin D and to appreciate its manifold benefits. Guidelines for the critique and design of clinical trials are proposed in this article to aid readers and researchers in evaluating and designing clinical studies for the evaluation of the therapeutic efficacy of vitamin D.

Diagnosing Vitamin D Inadequacy with Measurement of Serum 25-OH-Vitamin D

Periodic monitoring of serum calcium and serum 25-OH-vitamin D [25(OH)D] levels can guide dosage modifications to ensure that treatment is both safe and effective. Current laboratory reference ranges for 25(OH)D simply report average levels for the population, most of whom are deficient. They do not report ideal levels so they will mislead the practitioner unless he or she is aware of current research. The low end of the reference range is set too low due to previous misinterpretations of the research resulting in an overestimation of vitamin D toxicity and an underappreciation of the benefits and safety of higher vitamin D levels.^{44,45} Therefore, new reference ranges need to be determined based on the current research, and we present our proposals here and in Figure 1:

Vitamin D Deficiency: less than 20 ng/mL (50 nmol/L).

Serum 25(OH)D levels below 20 ng/mL (50 nmol/L) are clearly indicative of vitamin D deficiency. However, several authorities note that this level appears to be too

low; Heaney⁵ and Holick⁴⁴ both state that 25(OH)D levels should always be greater than 30 ng/mL (75 nmol/L).

Vitamin D Insufficiency: less than 40 ng/mL (100 nmol/L). According to Zittermann¹¹, hypovitaminosis D, wherein tissue levels are depleted and PTH is slightly elevated, correlates with serum levels of 30 - 40 ng/mL (75 - 100 nmol/L). Independently, Dawson-Hughes *et al*⁴⁶ showed that serum levels of PTH begin to elevate when 25(OH)D levels fall below 45 ng/mL (110 nmol/L) in elderly men and women, and these findings were supported by Kinyamu *et al*⁴⁷ who found that optimal PTH status deteriorates when 25(OH)D levels fall below 49 ng/mL (122 nmol/L) in elderly women. Therefore, in order to maintain physiologic suppression of PTH, serum levels of 25(OH)D need to be greater than 40 ng/mL (100 nmol/L).

Optimal Vitamin D Status: 40 – 65 ng/mL (100 - 160 nmol/L).

Based on our review of the literature, we propose that the optimal—"sufficient and safe"—range for 25(OH)D correlates with serum levels of 40 - 70 ng/mL (100 - 175 nmol/L). This proposed optimal range is compatible with other published recommendations: Zittermann¹¹ states that serum levels of 40 - 80 ng/mL (100 - 200 nmol/L) are "adequate", and Mahon *et al*²⁹ recently advocated an optimal range of 40 - 100 ng/mL (100 - 250 nmol/L) for patients with multiple sclerosis. The lower end of our proposed range is consistent with suggestions by Mercola^{49,50} who advocates an optimal range of 45 - 50 ng/mL (115 - 128 nmol/L) and by Holick⁴⁴ who states that levels should be 30 - 50 ng/mL (75 - 125 nmol/L). The upper end of our proposed optimal range is modified from the previously mentioned ranges offered by Zittermann¹¹ (up to 80 ng/mL [200 nmol/L]) and Mahon *et al*²⁹ (up to 100 ng/mL (250 nmol/L)). According to the authoritative monograph by Vieth¹, there is no consistent, credible evidence of vitamin D toxicity associated with levels below 80 - 88 ng/mL (200 - 220 nmol/L). Vieth¹ states, "Although not strictly within the 'normal' range for a clothed, sun-avoiding population, serum 25(OH)D concentrations \leq 220 nmol/L [88 ng/mL] are consistent with certain environments, are not unusual in the absence of vitamin D supplements, and should be regarded as being within the physiologic range for humans." Similarly, in his very thorough review of the literature, Zittermann¹¹ concludes that serum 25(OH)D concentrations up to 100 ng/mL (250 nmol/L) are sub-toxic. Additional support for the safety of this upper limit comes from documentation that sun exposure alone can raise levels of 25(OH)D to more than 80 ng/mL (200 nmol/L)¹ and that oral supplementation with 10,000 IU per day in healthy men resulted in serum levels greater

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than 80 ng/mL (200 nmol/L) with no evidence of toxicity.³ Until more data becomes available, we have chosen 65 ng/mL (160 nmol/L) rather than 80 ng/mL (200 nmol/L) as the upper end of the optimal range to provide a safety zone between the optimal level and the level which may possibly be associated with toxicity, and to allow for other factors which may promote hypercalcemia, as discussed below. Long-term prospective interventional studies with large groups and clinical trials involving patients with vitamin D-associated illnesses such as multiple sclerosis will be needed in order to accurately define the optimal range—the serum level of vitamin D that affords protection from illness but which does not cause iatrogenic complications. In reviewing much of the current literature, we found no evidence of adverse effects associated with a 25(OH)D level of 65 ng/mL (160 nmol/L), and we found that this level is considered normal by some medical laboratories⁶ and that it can be approximated and safely exceeded with frequent full-body exposure to ultraviolet light¹ or oral administration of physiologic doses of 5,000 - 10,000 IU cholecalciferol per day for twenty weeks.² Prospective studies and interventional clinical trials comparing different serum levels of 25(OH)D with clinical outcomes are necessary to elucidate the exact optimal range in various clinical conditions. There are no acute or subacute risks associated with the 25(OH)D levels suggested here. Conversely, there is clear evidence of long-term danger associated with vitamin D levels that are *insufficient*.

Vitamin D Excess: Serum Levels Greater than 80 ng/mL (200 nmol/L) with Accompanying Hypercalcemia. Serum levels of 25(OH)D can exceed 80 ng/mL (200 nmol/L) with ultraviolet light exposure in the absence of oral vitamin D supplementation^{1,6} and with oral supplementation with 10,000 IU per day as previously mentioned²— in neither scenario is toxicity consistently observed. 25(OH)D greater than 80 ng/mL (200 nmol/L) are not indicative of toxicity unless accompanied by clinical manifestations and hypercalcemia. Vieth¹ notes that hypercalcemia due to hypervitaminosis D is always associated with serum 25(OH)D concentrations greater than 88 ng/mL (220 nmol/L), and Holick⁶ stated, “Vitamin D intoxication does not occur until the circulating levels of 25(OH)D are over 125 ng/mL [312 nmol/L].” Assessment for hypervitaminosis D is performed by measurement of serum 25(OH)D and serum calcium.

Monitoring for Vitamin D Toxicity with 25(OH)D and Serum Calcium

Hypercalcemia can occur with vitamin D supplementation by either causing direct toxicity (rare) or by being associated with a vitamin D hypersensitivity syndrome (more common). If serum calcium becomes abnormally high, then vitamin D supplementation must be discontinued until the cause of the hypercalcemia is identified; however, direct vitamin D toxicity will rarely be the sole cause of the hypercalcemia.

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The most important indicator of direct vitamin D toxicity is elevated serum calcium associated with a 25(OH)D level greater than 90 ng/ml (225 nmol/L). Elevated 1,25(OH)D levels are commonly—though not always—seen with vitamin D toxicity. Severe vitamin D intoxication is rare and usually seen only with industrial accidents, such as overdosing the fortification of milk, or with long-term administration of more than 40,000 IU of vitamin D per day. Severe hypercalcemia may require urinary acidification and corticosteroids to expedite the reduction in serum calcium.⁵¹

Induction of vitamin D toxicity generally requires 1 - 4 months of 40,000 IU per day in infants.⁵¹ In adults, toxicity generally requires several months of supplementation of at least 100,000 IU per day. Hypercalcemia appears to be the mechanism of vitamin D toxicity (rather than a direct toxic effect of the vitamin), and 25-OH-vitamin D levels may be normal in patients who are vitamin D toxic and hypercalcemic, particularly with vitamin D hypersensitivity syndrome. It has therefore been suggested that serum calcium be measured on a weekly and then monthly basis in patients receiving high-dose vitamin D. Manifestations attributable to hypervitaminosis D and hypercalcemia include anorexia, nausea, and vomiting followed by weakness, nervousness, pruritus, polyuria, polydipsia, renal impairment, and soft-tissue calcifications.

As a cause of hypercalcemia, vitamin D hypersensitivity syndromes are more common than vitamin D toxicity, and they generally arise when aberrant tissue uncontrollably produces the most active form of vitamin D (calcitriol). Primary hyperparathyroidism, granulomatous disease (such as sarcoidosis, Crohn's disease, and tuberculosis) and various forms of cancer may cause the syndrome. 25(OH)D levels are normal or even low in vitamin D hypersensitivity while serum calcium and 1,25(OH)D levels are elevated. Additional causes include adrenal insufficiency, hyperthyroidism, hypothyroidism, and adverse drug effects, particularly with thiazide diuretics. Whatever the cause, patients with persistent hypercalcemia should discontinue vitamin D supplementation and receive a thorough diagnostic evaluation to determine the cause of the problem.

Past and Future Vitamin D Studies: Critique and Design

Nearly all published clinical trials have suffered from flawed design, including inadequate dosing, inadequate duration, wrong type of vitamin D being used, failure to test serum vitamin D levels, and/or failure to ensure that serum vitamin D levels entered into the optimal range. The following guidelines are provided for clinicians and

researchers using vitamin D in clinical practice and therapeutic trials.

Dosages of vitamin D must reflect natural supply and physiologic requirements and should therefore be in the range of 4,000 – 10,000 IU per day: The physiologic requirement for vitamin D appears to be approximately 4,000 IU per day in adults.² Exposure to ultraviolet light (e.g., sunshine) can produce the equivalent of 10,000 IU of vitamin D3.¹ Therefore, intervention trials with supplemental vitamin D should use between 4,000 IU per day, which is presumably sufficient to meet physiologic demands, up to 10,000 IU, which is the physiologic dose attained naturally via full-body sun exposure. Based on these physiologic criteria, we see that the majority of intervention studies in adults have used inadequate, subphysiologic doses of vitamin D. Therefore, many studies that failed to identify therapeutic benefits from vitamin D supplementation were flawed due to insufficient therapeutic intervention—the dose of vitamin D was too low.

Vitamin D supplementation must be continued for at least 6-10 months: Since serum 25(OH)D levels do not plateau until after 3 months of supplementation, and we would expect clinical and biochemical changes to become optimally apparent some time *after* the attainment of peak serum levels, any intervention study of less than 6-10 months is of insufficient duration to determine either maximal benefit or that vitamin D supplementation is ineffective for the condition being investigated.

Supplementation should be performed with D3 rather than D2: Although cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2) are metabolized similarly, D3 is the human nutrient and is 70% more efficient in raising serum 25[OH]D levels.¹¹ The type of vitamin D must always be clearly stated in published research reports.

Effectiveness of supplementation must include evaluation of serum vitamin D levels: Oral supplementation is a means by which to raise vitamin D levels; supplementation is not therapeutic in itself unless it raises serum 25(OH)D levels. To assess both efficacy and compliance, serum 25(OH)D levels must be monitored in clinical trials involving vitamin D supplementation. Assessment of serum levels is important also to assess the relative dose-effectiveness of different preparations of vitamin D; e.g., some evidence suggests that micro-emulsification facilitates absorption of fat-soluble nutrients.^{49,52,53} Measurement of 1,25-dihydroxyvitamin (calcitriol) is potentially misleading and is not recommended for the evaluation of vitamin D status.

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Serum vitamin D levels must enter the optimal range:

The majority of clinical intervention studies using vitamin D have failed to use supplementation of sufficient dosage and duration to attain optimal serum levels of vitamin D. Our proposed optimal range for 25(OH)D is 40 - 65 ng/mL (100 - 160 nmol/L) and is presented in Figure 1.

The above-mentioned criteria will aid future researchers in designing interventional studies that can accurately evaluate the relationship between vitamin D and human illness. Clinicians who are not conducting research but rather are interested in attaining clinical improvement in their patients, should follow these guidelines as well when using vitamin D supplementation in patients, while remembering to monitor for toxicity with the triad of clinical assessments, serum 25(OH)D, and serum calcium. Clinicians and researchers need to remember, however, that optimal clinical effectiveness often depends on synergism of diet, lifestyle, exercise, emotional health, and other factors. Single intervention studies are a reasonable research tool only for evaluating cause-and-effect relationships based on the presumption of a simplistic, linear model that is generally inconsistent with the complexity and multiplicity of synergistic and interconnected factors that determine health and disease. Thus, single intervention studies with vitamin D supplementation will be useful from an intellectual standpoint insofar as they will help us to further define the role of vitamin D in human physiology and pathophysiology. However, optimal clinical results with individual patients are more easily attained with the use of multicomponent treatment plans that address many facets of the patient's health.⁴⁸

Interventional Strategies to Treat Vitamin D Deficiency by Increasing Serum Vitamin D Levels

Human physiology adapted to and was shaped by a natural environment with ample exposure to sunlight.⁵ Full-body exposure to ultraviolet light on clear days in equatorial latitudes can easily provide the equivalent of 4,000 - 10,000 IU of vitamin D¹ or approximately 20,000 IU of vitamin D₂.⁸ Slightly longer durations of full-body sun exposure of approximately thirty minutes (3x the minimal erythemal dose) will produce 50,000 IU of vitamin D in lightly pigmented persons, while 5x longer durations are required for more darkly pigmented people to attain the same vitamin D production.⁵⁴ The dose of vitamin D required to obtain adequate blood levels depends on latitude, sun-exposure, skin type and dietary sources. Therefore, vitamin D supplementation above the current Food and Nutrition Board's 2,000 IU Upper Limit (UL) for adults and 1,000 IU for infants and children should always be guided by calcium and 25(OH)D

levels. 1,25(OH)D (calcitriol) has no place in routine monitoring for vitamin D supplementation, as it will mislead the practitioner.

Vitamin D Supplementation in Adults: When 28 men and women were administered 4,000 IU per day for up to five months, in the absence of UVB from the sun, serum 25(OH)D levels reached approximately 40 ng/mL (100 nmol/L), and no toxicity was observed.⁴ When 67 men were administered 5,000 and 10,000 IU of cholecalciferol per day for twenty weeks, again in the absence of UVB from the sun, serum levels of 25(OH)D increased to approximately 60 ng/mL (150 nmol/L) and 90 ng/mL (225 nmol/L), respectively, and no toxicity was observed.² Vitamin D administration above 2,000 IU/day in adults should be periodically monitored with serum calcium and 25(OH)D levels to ensure safety and the attainment of optimal 25(OH)D levels.

Vitamin D Supplementation in Pregnant Women: In 1966, two case reports and a brief review of the literature showed no adverse effects of 100,000 IU per day of vitamin D in hypoparathyroid pregnant women.⁵⁵ In 1971, a study of 15 hypoparathyroid pregnant women was reported wherein the women received more than 100,000 IU per day of vitamin D with no adverse effects to the mother or child, leading the authors to conclude that there was "no risk from vitamin D in pregnancy."⁵⁶ Doses of vitamin D for pregnant women were extensively reviewed by Hollis and Wagner⁵⁴ immediately prior to the completion of this article, and the authors concluded that doses of 100,000 IU per day were safe for pregnant women. The authors write, "Thus, there is no evidence in humans that even a 100,000 IU/d dose of vitamin D for extended periods during pregnancy results in any harmful effects." Data from several placebo-controlled clinical trials with pregnant women show that vitamin D supplementation results in superior health status for the mother and infant. The current daily reference intake (DRI) for vitamin D of 200 - 400 IU per day is therefore "grossly inadequate", and administration of less than 1,000 IU vitamin D per day to pregnant women is scientifically unjustifiable and ethically questionable. Hollis and Wagner⁴⁵ conclude that up to 4,000 IU per day is necessary for pregnant women, and this conclusion is consistent with previously cited research on physiologic requirements and endogenous vitamin D production in the range of 4,000 - 10,000 IU per day. In order to ensure safety and efficacy, vitamin D administration above 2,000 IU/day in pregnant and lactating women should be monitored with serum calcium and 25(OH)D levels.

Vitamin D Supplementation in Infants and Children:

In Finland from the mid-1950's until 1964, the recommended daily intake of vitamin D for infants was 4,000 – 5,000 IU, a dose that was considered safe and was associated with significant protection from type 1 diabetes.⁵⁴ More recently, in a study involving more than 10,000 infants and children, daily administration of 2,000 IU per day was safe and effective for reducing the incidence of type 1 diabetes by 80%.¹⁶ Thus, for infants and children, doses of 1,000 IU per day are certainly safe, and higher doses should be monitored by serum calcium and 25(OH)D levels.

Options for Raising Vitamin D Blood Levels: We have two realistic options for increasing vitamin D levels in the body: supplementation and sunlight (e.g., ultraviolet radiation). Sunlight is commonly unavailable on rainy or cloudy days, during the winter months, and in particular geographic locations. Furthermore, since many people work indoors where sunshine is inaccessible, or they are partially or fully clothed when outside, reliance on sunshine to provide optimal levels of vitamin D is generally destined to provide unsatisfactory and inconsistent biochemical and clinical results. The use of UVB tanning beds can increase vitamin D levels; but this option is more expensive and time-consuming than oral supplementation. Additionally, excess ultraviolet radiation exposure expedites skin aging and encourages the development of skin cancer. Given the impracticalities and disadvantages associated with relying on sun exposure to provide optimal levels of vitamin D year-round, oral vitamin D supplementation is the best option for ensuring that biochemical needs are consistently met.

Vitamin D is either absent or present in non-therapeutic amounts in dietary sources. One of the only major dietary sources of vitamin D is cod-liver oil, but the amount required to obtain a target dose of 4,000 IU per day would require patients to consume at least three tablespoons of cod-liver oil, or the amount contained in 18 capsules.³⁹ Clearly this would be unpalatable and prohibitively expensive for most patients, and it would result in very low compliance. Additionally, such a high dose of cod-liver oil may produce adverse effects with long-term use, particularly with regard to excess vitamin A, and perhaps with an increased tendency for bleeding and reduced biological activity of gamma-linolenic acid.^{48, 57}

Discussion and Conclusions

Vitamin D is not a drug, nor should it be restricted to prescription availability. Vitamin D is not a new or unproven "treatment." Vitamin D is an endogenous, natu-

(Continued on next page)

rally occurring, photochemically-produced presteroidal molecule with essential functions in systemic homeostasis and physiology, including modulation of calcium metabolism, cell proliferation, cardiovascular dynamics, immune/inflammatory balance, neurologic function, and genetic expression. Insufficient endogenous production due to lack of sufficient sun exposure necessitates oral supplementation to meet physiologic needs. Failure to meet physiologic needs is synonymous with insufficiency/deficiency and results in subtle yet widespread disturbances in cellular function which may promote the manifestation of subacute long-latency deficiency diseases such as osteoporosis, cardiovascular disease, hypertension, cancer, depression, epilepsy, type 1 diabetes, insulin resistance, autoimmune disease, migraine, polycystic ovary syndrome, and musculoskeletal pain. In case reports, clinical trials, animal studies, and/or epidemiologic surveys, the provision of vitamin D via sunlight or supplementation has been shown to safely help prevent or improve all of the aforementioned conditions.

Vitamin D deficiency is epidemic in the developed world, particularly among people of color, the old, the sun-deprived and medical patients^{58,59} and is notably common in patients with chronic musculoskeletal pain³⁵⁻³⁷ and disorders such as rheumatoid arthritis,¹⁸ multiple sclerosis,²⁹ Grave's disease,³⁸ heart disease, hypertension, diabetes, cancer, ankylosing spondylitis,³⁹ and systemic lupus erythematosus.⁴⁰ As a medically valid diagnosis (ICD-9 code 268.x) with a high prevalence and clinically significant morbidity, vitamin D deficiency deserves equal attention and status with other diagnoses encountered in clinical practice. Given the depth and breadth of the peer-reviewed research documenting the frequency and consequences of this problem, failure to diagnose vitamin D deficiency and failure to correct hypovitaminosis D by providing vitamin D supplementation is indefensible and is below the level of good healthcare. Failure to act prudently based on the research now available in favor of vitamin D supplementation appears likely to dwarf the previous failure to act on the research supporting the use of folic acid to prevent cardiovascular disease and neural tube defects—a blunder that appears to have resulted in hundreds of thousands of unnecessary cardiovascular deaths⁶⁰ and which has contributed to incalculable human suffering related to otherwise preventable neural tube defects, cervical dysplasia, cancer, osteoporosis, and mental depression. Currently, Grant¹² estimates that at least 23,000 and perhaps as many as 47,000 cancer deaths⁶¹ might be prevented each year in America if we employed simple interventions (i.e., sunshine or supplementation) to raise vitamin D levels. Of course,

additional lives may be saved and suffering reduced by alleviating the morbidity and mortality associated with hypertension, autoimmune disease, depression, epilepsy, migraine, diabetes, polycystic ovary syndrome, musculoskeletal pain, osteoporosis, and cardiovascular disease. Until proven otherwise, the balance of the research clearly indicates that oral supplementation in the range of 1,000 IU per day for infants, 2,000 IU per day for children and 4,000 IU per day for adults is safe and reasonable to meet physiologic requirements, to promote optimal health, and to reduce the risk of several serious diseases. Safety and effectiveness of supplementation are assured by periodic monitoring of serum 25(OH)D and serum calcium.

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Sodium to Potassium Ratio in a Hair Sample... A Remarkable Tool in Assessing Your Patient's Health

by William Risley, DC

Hardly a day passes in my work as a consultant to Analytical Research Laboratories that I do not marvel at the legacy left by Dr. Paul Eck. Information discovered by Dr. Eck is years ahead of its time and is catalogued in the laboratory. It is used in assessing physicians' patients through hair analysis worldwide.

Dr. Eck's work, along with nutritional recommendations for improvement in patient health, is sometimes met with skepticism and resistance. This is especially true of those with reasons that are less than ethical, those that are ignorant of the science, and those that simply prefer not to look objectively at the discoveries. His discoveries, however, have stood the tests of accuracy and reliability through 2 million hair assessments.

One of Eck's major postulates is that the ratio of sodium and potassium in a hair sample can be used to assess overall immune system integrity and the concomitant state of health of the adrenal cortex.¹ The concept is invaluable in clinical patient care. It is important to remember, however, that this, and other ratios, can only be used in assessing hair test results if the sample is not pre-washed at the laboratory. Leroy, *et al*, have shown that a pre-wash of the hair sample by laboratory standards, seriously compromises the test validity of certain more water soluble elements, especially sodium and potassium.²

Sodium is an extra-cellular mineral, normally balanced by potassium inside the cell, via the sodium/potassium pump mechanism. The normal sodium to potassium ratio in the hair sample is determined by simply dividing a normal sodium level of 25mg% by a normal potassium level of 10mg%. This ratio has therefore been set at 2.5:1. Increases of sodium in the normal hair ratio over and above the 2.5:1 occur when stresses upon the system mandate an increase in adrenal cortical activity. The hormonal response is secreting aldosterone, which is known to regulate both sodium and potassium.

Elevations of sodium on the hair graph occur concomitantly with certain physiological actions suggestive of a defensive posture, e.g., elevation of blood pressure, high glucose levels, and generally increased metabolic activity. An increase in metabolic activity results in a subsequent increase of acid by-products, which in turn enhance sodium's passage into the cell, mineral solubility, etc. Sodium's entrance into the cell allows glucose, proteins, minerals, and other nutritional assets to feed what is now a more active cellular response. More energy can be produced to rise to the occasion of fighting a stressor or departing the locale where the stress is occurring. Hans Selye's "fight or flight" response is an apt description.

Elevation of blood pressure, under the circumstances of increased stress is a highly desirable response on the part of the body for the moment. Unfortunately, excessive and prolonged elevations of many of these factors can become a direct or potential pathology.

Reduction of salt intake (sodium chloride) for its presumed effect on blood pressure was thus accepted decades ago as a positive health procedure. Unfortunately, today's medical dictum ignores the need for increases in salt for those patients with low blood pressure and poor adrenal function. A particular patient comes to mind with significant adrenal gland insufficiency that puts extra salt on pretzels and on bacon! He no doubt craves the potential sodium boost to his sagging adrenal gland function.

Moderate elevations of sodium over the established optimum ratio of 2.5:1 are indications of normal responses to the usual daily stressors. The balancing effect of this sodium increase is, of course, cortisol. Cortisol secretion has been found in our laboratory to be a direct corollary in the hair sample to the level of potassium. Cortisol is anti-inflammatory in nature, and it balances the inflammatory trend of aldosterone as the described defensive measure.

Mild to moderate reactions on the part of immune/adrenal axis occur consistently throughout the life of the subject, and most of these are handled with quiet efficiency, completed without alarm or notice by the patient. In the healthy patient, the balanced Na:K ratio, may elevate above the 2.5:1 expected norm without noticeable symptoms.

When a person is exposed to much more severe stressors — external or internal, emotional or physical — the ratio now approaches what Eck had found to be a

threshold at which some inflammatory process becomes apparent. Sodium to potassium at a ratio of 4.0:1 or greater has been determined to be the “break point” at which normal immune responses of an every day nature may become noticeable and alarming symptomatology.

Chemotaxis may now begin in earnest, concomitant with an obvious or occult inflammatory process somewhere in the system. Various blood cells, empowered to protect tissue or destroy invaders, now congregate in the area of the lesion. Sound theory also has been broached that cholesterol is the “plaster cast” produced to shore up inflamed blood vessels, which then may be recognized as having atherosclerotic lesions. Stress is clearly a major factor in atherosclerosis development.

As a physician, you may confidently state that a patient with a Na:K ratio in excess of 4.0:1 in a hair sample that has not been subject to a laboratory pre-wash is in fact a patient with overt or occult inflammatory lesions that threaten his or her health. The doctor should also be aware that sodium-treated water systems (water softeners) can elevate sodium readings in the hair, either by external contamination (e.g., showers, etc.) or internally (e.g., through drinking, ice cubes, cooking, etc.). It is indeed valuable to note improvement in the patient’s conditions and symptoms when the ratio improves toward the more balanced level of 2.5:1. It has proven a virtually indispensable monitor of the patient’s response to your therapy.

According to Eck, this is the probable scenario. Stress occurs, of whatever nature, and the adrenal cortex responds in a linear fashion to protect the health of the body. If the threat is severe enough, concomitant increases in aldosterone are secreted, and blood pressure elevates, along with other protective mechanisms, to enhance metabolic function ... fighting or fleeing from the real or imagined stress which is threatening harm to our patient.

Increases of sodium outside the cell cause more migration of this element intra-cellularly, carrying with it glucose, protein, vitamins, and minerals. That which is needed by each individual cell to increase energy production is subsequently provided, if the system has stored sufficient resources or they are otherwise available. The subsequent increase in energy allows the subject to escape from whatever threat is providing the stress.

Increased metabolic activity results in an additional

increase of resultant acid by-products such as carbonic acid, lactic acid, and dissolved carbon dioxide. This increased acidity enhances mineral solubility, as well as electrical receptivity of cellular processes, again enhancing energy production for the “fight or flight” response.

Eck found that when the ratio of sodium to potassium exceeds about 4:1, all of this activity results in a controlled inflammation, and we know that all healing is enhanced and/or mandated by inflammation. The aforementioned chemotaxis may occur, summoning the soldiers to do repair work on a damaged tissue or fight off bacterial or viral invaders when the skin is breached. Many other protective mechanisms will, of course, enter the fray.

This enhanced state of increased metabolic activity and the subsequent move toward a relative acidity and other Type A tendencies have been included in the description of the phrase “fast oxidizer” by both Eck and George Watson.³ Such a state is a perfectly normal reaction on the part of the human body to a major or minor threat to the health of the host. The temperature increase referred to as “fever” may also be indicated, hopefully avoiding such excesses again, which may be pathological in themselves.

If prolonged, the assets available to the body (i.e., vitamins, minerals, proteins, etc.) may be depleted to the point of physiological “burnout.” This burnout inhibits further optimum defensive responses on the part of the immune system, which may then result in the host’s lapse into chronic disease, if not actual death.

History is replete with references to gray hair occurring overnight and the incidence of rheumatoid arthritis or other severe debilitation due to this overuse of body nutrient resources. Actual cannibalism of body parts can occur in a desperate attempt to save the life of the victim. If the stomach wall is “robbed” of protein/amino acids, the integrity of the stomach lining may be compromised, and an ulcer may develop. The *Merck Manual* states that the usual finding on ulcer surgery is normal hydrochloric acid. It would seem unlikely that *H. pylori* or excess acidity are causes of the ulcer. It is more likely that compromised stomach-wall integrity impairs the protective resources of the stomach, and HCl and/or *H. pylori* become the presumed villains.

The “borrowing” of protein from joint synovial fluid carries with it the potential of a rheumatoid arthritis

(Continued on next page)

syndrome. Colitis may result when protein is lost from the wall of the colon.

A depleted immune system and adrenal cortex can still “rise to the occasion” when called upon, but it is in effect, another “nail in the coffin.” Chronic asset depletion is the result, and the system responds to further stresses at further costs to its diminishing resources and capabilities.

The elevated Na:K ratio indicating inflammation, now drops to less than 2.5:1, and may continue in further decline with worsening of the chronic disease state at each numerical drop upon the way. The patient has simply worn out in the fight against the aggressive stressor of whatever nature.

Numerical values of this ratio that decline to 0.5:1 or lower suggest definitive concerns of the patient’s ongoing good health and his/her ability to adequately cope with a significant injury or infection (viral or bacterial). In fact, the laboratory has found a percentage of these patients that may be faced with their possible demise. The concept of biochemical individuality is always a factor, and although this ratio at such a low level has been occasioned with death, other patients recover with diligent effort, and go on to lead productive lives. At the very least, the physician should be aware that such low ratios are a portent of significant concern.

Aggressive therapy and optimum patient cooperation offer significant hope for improvement, but the physician should be aware that an optimistic prognosis might be in question.

It is important to note that corticosteroid therapy commonly brings this ratio up to normal values on the hair graph. You should question the patient relative to such medicinal therapy. Relief is common with anti-inflammatories, such as hydrocortisone, and although it can certainly seem to be a blessing, a cure of the condition is usually elusive.

Assessing this Na:K ratio and finding a normal level of 2.5:1 without the support of medication is almost universally suggestive of excellent health. When the ratio exceeds 4.0:1, an inflammation or excessive stress is presumed, which tends to rapidly deplete nutrient assets when fighting that which is causing the stress response. When this ratio drops below about 2.2:1, the patient may be presumed to have a compromised immune system with the potential for severe further decline in health in a linear manner as the value continues to

worsen. Such a patient may have a chronic disease syndrome currently, or some significant assault has occurred to place him in a compromised position of health. Monitoring the changes as a result of your therapy enables you, as the physician, to successfully guide your patient’s return to health. Observing this ratio and its positive or negative changes is an essential component to the clinical practice of healing.

About the Author

Dr. William Risley is a 1961 graduate of the Palmer College-Midwest and has chaired post-graduate faculty positions in Neurovascular Diagnosis and Hospital Protocol at Palmer. He is currently on the continuing education faculty at Texas Chiropractic College and is a consultant and educator for Analytical Research Laboratories in Phoenix. He was a former instructor in plethysmography and Doppler for Diagnostics International. He is the author of 12 textbooks in general use by the chiropractic and medical professions and was a developer and owner of First Chiropractic, a 175-franchise organization of chiropractic offices. His books are available at www.chirobooks.com.

Comments may be addressed to Dr. Risley at Drrisley@arltma.com or Mediserv@mindspring.com. At no cost, information on ratios and copper can be requested at either e-mail address, requesting “ratios and copper” and supplying the sender’s name and address.

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ABSTRACTS OF INTEREST

New Details Regarding B₁₂ Deficiency in Diabetics Taking Metformin

By Steve Austin, N.D.

Design: Prospective (nested case-control) observational study.

Participants: All subjects were diabetics who had been taking metformin (Glucophage; also a component of Glucovance). Cases (n=155) were identified in which vitamin B₁₂ deficiency developed during follow-up (mean serum B₁₂ =149 pg/ml). An additional 310 controls were found who had not developed a B₁₂ deficiency during follow-up (mean serum B₁₂ =466 pg/ml).

Outcome Measures: Researchers were looking for correlations between B₁₂ deficiency and dose and duration of metformin use.

Key Findings: Each additional 1 g/day metformin dose increment nearly tripled the risk of B₁₂ deficiency (odds ratio: 2.88; 95% CI, 2.2-3.9, P<0.001). (Dosages of metformin are tailored to individual patients but typically range between 0.5 and 2.5 g/day.) In addition, those using metformin for at least three years had well over twice the risk compared with those using the drug for less than three years (odds ratio: 2.4; 95% CI, 1.5-3.9, P=0.001). After excluding subjects with borderline deficiencies, dosage remained the strongest predictor of risk.

Practice Implications: Metformin interferes with the absorption of B₁₂. The findings of the new report remind us to ask all diabetic patients about their medications. Anyone taking metformin either as Glucophage or in Glucovance should be asked about whether the prescribing doctor is monitoring serum B₁₂ levels. As a result of these new findings, we now know the problem is tied directly to both dosage and duration. In the fine print of the article, when compared with the risk in meat eaters, the adjusted risk of developing the deficiency (including borderline deficiency) in metformin-taking diabetics was over 1600% in vegetarians! This suggests that *all* vegetarian diabetics on this drug should be supplementing with B₁₂.

Ting R Z-W, Szeto CC, Chan M H-M, *et al.* "Risk factors of vitamin B₁₂ deficiency in patients receiving metformin." *Arch Intern Med* 2006; 166:1975-9.

Pycnogenol® Improves Diabetic Microangiopathy in Small Pilot Study

By Donald Brown, N.D.

Design: Prospective, placebo-controlled, parallel groups study.

Participants: 60 adult patients (aged 55-68 years) with diabetes mellitus and severe microangiopathy. All patients had been treated with insulin for at least 3 years and had stable glycemic control. Neuropathy was assessed by evaluation vibration sensory threshold and with electromyography.

Study Medication and Dosage: Pycnogenol® (Horphag Research, Ltd, United Kingdom) or placebo-50 mg t.i.d. Patients were allowed to continue taking previously prescribed drugs for diabetes.

Duration: 4 weeks.

Outcome Measures: At baseline and at the end of the 4-week treatment period, all subjects were seen and the following measures of microcirculation were completed: laser Doppler flowometry (LDF), skin flux at rest in the foot (RF), and capillary filtration (measured as the rate of ankle swelling [RAS] with strain-gauge plethysmography). The venoarteriolar response (VAR; the response reflex on standing) was measured as a percentage of RF (RF-SF/RF:100; where SF was the flux measured on standing).*

Key Findings: Microcirculation and hematologic parameters were comparable in both groups at baseline. Following 4 weeks of treatment, subjects in the Pycnogenol group had a significant decrease in capillary filtration (RAS) compared to controls (p < 0.05). The VAR was significantly enhanced in those taking Pycnogenol (median value change from 23 to 38.7; p < 0.05) while RF was decreased significantly as well (p < 0.05). No adverse events were reported in either group. As a secondary observation, 14 patients in the treatment group were evaluated for visible foot and ankle edema. The researchers report a very fast decrease in edema over 5 to 8 days in this subset of patients.

(Continued on next page)

Practice Implications: Diabetic microangiopathy is a common progressive complication of diabetes and contributes to various complications including foot and ankle edema, ulcerations, as well as retina and kidney disease. Although a small pilot study with questionable statistical analysis, this short clinical trial suggests that a relatively low dose of Pycnogenol may reduce peripheral circulatory complications associated with diabetic microangiopathy. It's interesting to note that same group of Italian researchers have also found the topical use of Pycnogenol promising for the treatment of venous ulcers of the leg¹ and the combined use of oral and topical Pycnogenol for diabetic ulcers of the leg.² Hopefully, these results will set the stage for longer studies on the effect of Pycnogenol on microcirculation in diabetics.

*Note: In normal, healthy individuals, the VAR decreases distal skin flux in seconds after standing. In most normal individuals, the decrease in flux can be within 35% and 55% of RF. This flux reduction (VAR) is greatly altered in diabetics due to the presence of different degrees of neuropathy and alterations in the axon reflex producing the venoarteriolar response.

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Bifidobacterium infantis Effectively Treats Irritable Bowel Syndrome

By Donald Brown, N.D.

Design: Randomized, double-blind, placebo-controlled, multicenter, dose-ranging study

Participants: 362 female primary care IBS patients (ages 18–65 years old) who met the Rome II criteria for the diagnosis of IBS (any bowel habit subtype were included) and in whom organic diseases, including inflammatory bowel disease, and significant systemic diseases had been excluded. Pregnancy was also an exclusion factor.

Study Medication and Dosage: Following a 2-week run-in period, participants were randomized to receive

either placebo or freeze-dried encapsulated *Bifidobacterium infantis* 35624 (Proctor and Gamble, Egham, U.K.) at a dose of either 1 x 10⁶, 1x 10⁸, or 1x 10¹⁰ cfu/mL daily.

Duration: 4 weeks.

Outcome Measures: IBS symptoms were monitored daily and scored on a 6-point Likert scale (LS) with the primary outcome measure being abdominal pain or discomfort. Symptoms evaluated included abdominal pain/discomfort, bloating/distension, urgency, incomplete evacuation, straining, and passage of gas. A composite symptom score, the subject's global assessment of IBS symptom relief, and measures of quality of life (using the IBS-QOL instrument) were also recorded. Symptom evaluations were collected weekly for the 4-week treatment period and for an additional two weeks following the end of treatment.

Key Findings: *B. infantis* 35624 at a dose of 1x 10⁸ cfu/mL was significantly superior to placebo on improvement of the primary outcome of abdominal pain/discomfort (change from baseline of -0.89 vs. -0.58, p = 0.023). The other dosages were not significantly different from placebo. At 4 weeks, variables of bloating/distension, sense of incomplete evacuation, and passage of gas were all significantly changed in the 1x 10⁸ group compared to placebo (p = 0.014). Benefits were also noted for the 1x 10⁸ dose when post hoc analysis was performed by IBS subtype. No significant adverse events were observed.

Practice Implications: This is a follow-up to an earlier 8-week trial reviewed in this column in June 2005 (*Gastroenterol* 2005;128:541–51) which found that 1x 10¹⁰ cfu/mL of *B. infantis* 35624 was more effective than a similar daily dose of *Lactobacillus salivarius* subspecies *salivarius* UCC4331 in both men and women with IBS. While the earlier trial was a bit more robust in design, it's interesting to note that the 1x 10¹⁰ cfu/mL dose was not effective compared to placebo this time around while a lower dose of 1x 10⁸ cfu/mL was effective. Probiotics should continue to be a primary consideration for the management of IBS and future trials should focus on the use of *B. infantis* for treatment durations longer than 4 or 8 weeks.

Whorwell PJ, Altringer L, Morel J, *et al.* "Efficacy of an encapsulated probiotic *Bifidobacterium infantis* 35624 in women with irritable bowel syndrome." *Am J Gastroenterol* 2006; 101:1581–90.

(Continued on page 44)

Zinc Dramatically Reduces Signs of Acne Rosacea

By Steve Austin, N.D.

Design: Randomized double blind crossover clinical trial

Participants: Of 25 rosacea patients, (aged 21–64 years), 19 completed the trial.

Study Medication and Dosage: 100 mg of zinc sulfate (ZnSO⁴) containing 40 mg Zn was administered three times per day for three months.

Main Outcome Measure: Rosacea severity scores

Key Findings: In the arm that initially received ZnSO⁴ therapy, the mean baseline severity score was 8. (Scores of 5–10 reflect moderate severity, with scores below 5 reflecting mild signs and those above 10 reflecting severe signs.) This number fell to 5.7 at one month, to 3.4 at two months, and to 1.6 at the end of the three-month intervention ($p < 0.01$ compared with baseline).

In the arm that received ZnSO⁴ during the second half of the trial, the mean baseline severity score was 7, which fell to 5.9 at one month, 3.9 at two months, and 1.9 at the end of the three-month ZnSO⁴ intervention ($p < 0.01$ compared with the end of the placebo period).

In both groups, erythema began improving after two months, though papules and pustules showed improvement after only one month. Telangiectasia scores did not improve in either group.

During placebo administration, scores began moving back toward baseline in the group that had initially been assigned to ZnSO⁴, though the mean score had only risen to 2.6 by the end of the third month. In the arm initially assigned to placebo, severity scores increased mildly during the placebo administration.

Practice Implications: In the 1970s and '80s, several trials reported that Zn supplementation aided patients with acne vulgaris. The new report extends those findings to patients with acne rosacea.

The effect was dramatic -- an average of approximately a 77% decrease in severity in three months. The mechanism behind the effect remains unclear, though the re-

searchers speculate the Zn might have an effect against demodex mites or other potential pathogens. Alternatively, they suggest that a free radical-scavenging effect of Zn might be responsible.

This trial was conducted in Iraq. Much of the Middle East contains a diet low in Zn, complicated further by the use of phytate-rich unleavened bread, which reduces bioavailability of Zn. Serum Zn levels were not measured in the new report, so it remains possible that the effects observed here were simply the function of overcoming a deficiency that may not exist in western populations. However, there is much overlap between acne vulgaris and acne rosacea, and the earlier reports of positive effects of Zn supplementation in vulgaris patients came mostly from western trials. Thus, a therapeutic trial of Zn supplementation in rosacea patients is now worth consideration. Long-term (three months or longer) Zn administration should be accompanied by supplementation with 1–3 mg per day of copper to prevent a Zn-induced copper deficiency.

Sharquie KE, Najim RA, Al-Salman HN. "Oral zinc sulfate in the treatment of rosacea: a double-blind, placebo-controlled study." *Int J Dermatol* 2006; 45:857–61. ♦

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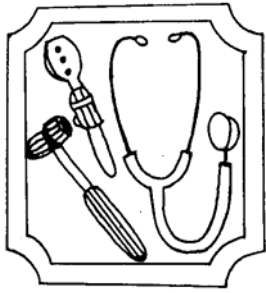
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