SUNLIGHT AND VITAMIN D PACKET CONTENTS

Important UPDATES

7/2011 Information on the importance of vitamin C is newly included. While not generally promoted, low, sufficient, and excess intake of vitamins change everything. Vitamins aren't just 'co-enzymes'. In particular vitamins A, D and C have genomic functions. This means these elements control what genes are turned on and off based on tissue levels of individual elements and the balance of these elements with each other. Excess A with normal D may cause symptoms of D deficiency, excess D with low or normal vitamin A may results in symptoms of vitamin A deficiency. Not having enough vitamin C my alter your ability to turn sunlight or supplement vitamin D into the active 25(OH)D and 1,25(OH)2D. Health is always about the milieu (environment/surroundings) inside and outside of your cells.

December 5, 2009 The Solgar Vitamin D3 Cholecalciferol 1,000 IU Soft Gels from fish liver oil (also containing 3,000 IU A per each) are no longer available. This is the product I have used successfully for the past 10 years. As I have little experience with other brands, your dosing may need to be adjusted.

The Solgar Vitamin D3 Cholecalciferol 1,000 IU Soft Gel made prior to this date effectively and safely raised vitamin D at moderate doses (typically 2,000 IU daily or less). Other brands for unknown reasons consistently have required more. In 2001 a respected researcher came to me for a consult after taking 4,000 IU of dry (dry capsule) D3 for many months and seeing NO improvement, zero change, when testing 25(OH)D.

When I questioned several manufacturing companies it was thought perhaps the technique of drying D for encapsulation/tableting using cellulose, an indigestible fiber, as the binding agent may have reduced the availability of the D in both dry capsules and tablets.

This suggests the use of cellulose as a binding agent, particularly with fat soluble nutrients and/or hormones, may be a mistake AND give the impression much larger doses are needed/safe because MUCH of the dose is NOT BEING ABSORBED. In 2009 Forest Pharmaceuticals/Armour and other thyroid manufacturers changed their desiccated thyroid binding agent from lactose to cellulose. Thyroid patients found their regular dose/s no longer worked and they had to take increasing doses of thyroid to get the same serum and physiological results they had gotten from the 'old' thyroid. (Information from Mary Shomon, about.com, and http://stopthethyroidmadness.com)

And from poor absorption to excess- some of the mycelized (emulsified) vitamin products have been found to have more or less than stated on the label in testing done by Consumer Labs. Because mycelized vitamins are pre-emulsified and extremely concentrated into drops the likelihood of absorption AND mis-dosing greatly increases leading to a greater possibility of excess/harm.

It is not about a DOSE but consistent use of a brand/product. I do not mean that one brand is BETTER than any other, all D3 supplements contain D3, but that as each is formulated somewhat differently you need to take whatever brand you choose consistently, test, and then raise your lower your dose, of the SAME brand to maintain your D between 40-60 ng/ml year round. If your brand is discontinued or you choose another source PLEASE repeat the testing process. http://www.bloodtestathome.com/vitamin_D_test.html $55 It will be worth the expense and effort.

Krispin Sullivan, Clinical Nutritionist 1-775-831-0292

REASON FOR THIS REPORT AND PACKET, PLEASE READ CAREFULLY This preliminary report was put together for the support of my clients and their families. It has been made available to the general public because others made direct requests. It may not be copied for any purpose. It may not be put on the internet in all or part. It may not be used as material, excerpted or even quoted, for other health publications or other publications without express written permission from me, the author.

Read carefully and do not think you understand vitamin D when you have finished. NUMBER ONE POINT TO REMEMBER: VITAMIN D IS NOT A VITAMIN. IT IS A PRO-HORMONE. IT IS MISUNDERSTOOD, MISNAMED AND MISLABELED. The book, Naked at Noon, Understanding The Importance of Sunlight and Vitamin D, has many chapters, each with information that often directly contradicts what you may currently believe to be true about sunlight and vitamin D. Order from http://sunlightd.org

Under no circumstances should vitamin D supplementation be advised without testing before and after supplementation. Testing every 8-12 weeks until serum 25(OH)D reaches mid-optimal range works well. Retest the second and third year at least twice.

Do not think you understand this process (I am speaking to physicians as well as patients and clients). None of us fully understand vitamin D yet. All of this information is very new. I have carefully considered all KNOWN opinions. **Some health care providers and some researchers feel that high doses of D are safe. I know from experience that doses of >2,000 IU daily can lead to toxicity in a significant percentage of persons. I have seen the test results and the side-effects. I know that some persons suggest we can get all the D we need from food, without sun exposure, again, I know from research and experience this is rarely true. Others believe that one 'dose' of D is right (safe and adequate) for everyone. This also is absolutely not true, confirmed by testing.**

Some feel that one type of D is toxic or more active or safer. D3, cholecalciferol, as found in fish oil is the sunlight produced D, cholecalciferol which we make in our skins. It is biologically the most active and potentially the most toxic. The symptoms of deficiency and toxicity are subtle. By the time you recognize that you or your patient suffer from chronic (not acute) deficiency or toxicity many of the associated conditions may be irreversible. Test, test and retest.

Read slowly and carefully. Try to understand the material and if you don't, get help. Do not proceed to treat yourself or anyone else without testing serum 25(OH)D. Do not test and treat without follow-up testing to 1) make sure the dose is correct- not too high or low and 2) make sure absorption is taking place. **Test a minimum of 3-4 times the first year.** Test a minimum of twice the second and third years. A significant number of clients may have absorption problems which would also indicate they poorly absorb A, E and K in addition to D. Others may become toxic over time using a constant daily dose.

If you need help applying the information I am available for consultation to y our physician or for y ou personally. For legal and medical reasons I am unable to advise persons who are not directly consulting with me.

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Brief Report on the Importance of Sunlight and Vitamin D

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Contents

Vitamin D Makes The News

Sunlight is mans' primary source of vitamin D. D is most known for its role in the prevention of rickets, a disease of the bones in infants and children. From this humble beginning, making our bones straight and strong, vitamin D, no longer considered a vitamin, is now surfacing as a major pro-hormone. Vitamin D in conjunction with calcium influences the growth and regulation of all body cells and systems.

To maintain optimal levels of vitamin D, sunlight UV-B exposure is absolutely critical. Current North American recommendations for sunlight exposure, 10-20 minutes three times a week before 10AM or after 2PM, and/or supplementation of vitamin D, 200-800 IU, cannot fulfill our daily needs.
In April of 2000 an abstract of a clinical observation published in the Archives of Internal Medicine caught my attention. Dr. Anu Prabhala and colleagues reported 5 patients with severe weakness and fatigue confined to wheelchairs were found to be suffering from severe vitamin D deficiency. All five patients became mobile after 6 weeks of treatment with 50,000 IU D per week. (1) This dose is equivalent to 7,000 IU a day.
People in wheelchairs 'got up and walked' when supplied with vitamin D. As a clinical nutrition researcher I found myself wondering what I had missed about this amazing vitamin.
After reading the full paper, which confirmed both diagnosis and treatment, I began a search for current information on vitamin D, it's actions, how much we really need and how we get it. The following is a small part of what I found. A more thorough examination of the information will be published in 2005 in Naked At Noon, Understanding Sunlight and Vitamin D by this author.

Reasons to make sure you get enough D

- Vitamin D is a more effective anti-oxidant than vitamin E in reducing lipid peroxidation and increasing superoxide dismutase and glutathione in hepatocytes. (2)
- Vitamin D is a membrane anti-oxidant inhibiting liposomal peroxidation. (3)
- Research suggests that low levels of D may contribute to or be a cause of Syndrome X with associated hypertension, obesity, diabetes and heart disease. (4)
- Vitamin D deficiency decreases biosynthesis and release of insulin. [5,6,7]
- Glucose intolerance has been inversely associated with the concentration of 25(OH)D. That is, low D may predispose to glucose intolerance. [4,8]
- Risk of senile cataract is reduced in persons with optimal levels of D and carotenoids. [9]
- Estimations of clinically evident serum D deficiency in the US range from 41%-57% of the general population. [10] Note that clinical deficiency does not define optimal amounts of D.
- High dietary levels of calcium, when D is insufficient, may contribute to calcification of the arteries, joints, and kidney, and perhaps even the brain. [11]
- Vitamin D regulates vitamin D binding proteins and some calcium binding proteins, which are responsible for carrying calcium to the 'right location' and protecting cells from damage from free calcium. [12]
- Calcium deposits in the arteries lead to heart attack. In the March 1995 issue of Analyst, Scottish researchers found hair calcium inversely correlated with arterial calcium; the more plaque (calcium) in the arteries, the less calcium in the hair. 90% of men experiencing myocardial infarction had low hair calcium. Vitamin D raised beard calcium and this rise continued as long as the vitamin D was consumed. Almost immediately after stopping the D, beard calcium fell to

pre-supplement levels. [13]

- Northern countries have higher levels of cardiovascular disease and more heart attacks occur in winter months. [14,15]
- Vitamin D and/or sunlight (UV-B, not UV-A or UV-C) have been shown to lower blood pressure, restore insulin sensitivity, and lower cholesterol.[16]
- Sunlight, UV-B, and vitamin D normalize food intake and normalize blood sugar. Weight normalization is associated with higher levels of D and adequate calcium.[17,18]
- Obesity is associated with vitamin D deficiency. [19,20]
- Obese persons have impaired production of UV-B stimulated D and impaired absorption of food source and supplemental D. [21]
- PCOS (Polycystic Ovarian Syndrome) has been corrected by supplementation of D and calcium.[22]
- Vitamin D plays a role in regulation of both the 'infectious' immune system and the 'inflammatory' immune system. [23]
- Low vitamin D is associated with several autoimmune diseases including Multiple Sclerosis, Sjogren's Syndrome, rheumatoid arthritis, thyroiditis and Crohn's disease. [24] [25,26]
- Osteoporosis is strongly associated with low vitamin D. Post-menopausal women with osteoporosis respond favorably (and rapidly) to higher levels of D plus calcium and magnesium.[27]
- D deficiency has been mistaken for fibromyalgia, chronic fatigue or peripheral neuropathy. [1,28]
- Infertility is associated with low vitamin D.[29]
- Vitamin D supports production of estrogen in men and women.[30]
- PMS has been completely reversed by addition of calcium, magnesium and vitamin D.[31]
- Menstrual migraine is associated with vitamin D and calcium.[32]
- Breast, prostate, skin and colon cancer have a strong association with low levels of D and lack of sunlight. [33]
- Michael Holick suggests that optimal levels of 25-hydroxyvitamin D contributes to lower levels of all epithelial cell cancers including, breast, prostate, colon and skin.[34,35,36]
- Activated vitamin D in the adrenal gland regulates tyrosine hydroxylase, the rate-limiting enzyme necessary for the production of dopamine, epinephrine and norepinephrine. Low D may contribute to chronic fatigue and depression. [37] [38]
- Seasonal Affective Disorder has been treated successfully with vitamin D. In a recent study covering 30 days of treatment comparing Vitamin D and 2 hour daily use of 'light boxes' depression completely resolved in the D group but not in the light box group.[39]
- High stress may increase the need for D or sunlight (UV-B) and calcium.[40,41,42]
- People with Parkinson's and Alzheimer's have been found to have lower levels of D.[43,44]
- Hospitalized or immobilized patients may need more D. Immobilization may require active 1,25(OH)2D as conversion of D3 seems to fail. [45,46]
- Low levels of D, and perhaps calcium, in a pregnant mother and/or later in the child may be the contributing cause of 'crooked teeth' and myopia. When these conditions are found in succeeding generations it means the genetics require higher levels of one or both nutrients to optimize health.[47] [48]
- Behavior and learning disorders respond well to D and/or calcium combined with adequate diet and trace elements.[49]
- Single, infrequent, intense, skin exposure to UV-B light suppresses the immune system but chronic, low level exposure (as per the guidelines) normalizes immune function, enhancing NK cell and T cell production, reducing abnormal inflammatory responses such as found in autoimmune disorders, and reducing occurrences of infectious disease. [24] [50,51] [52,53]

- Increasing levels of polyunsaturated omega-6 fatty acids and monounsaturated fats in the diet may decrease the binding of D to D binding proteins. Saturated fats and omega-3 fish oils do not have this effect. [54] D binding proteins are key to local and peripheral actions of vitamin D. This is an important consideration as the American diet has dramatically increased intake of polyunsaturated omega-6 fatty acids and monounsaturated fats and decreased intake of saturated fats and omega-3 fatty acids over the past 100 years.

Traditionally animal fats and fish oils contained from trace to significant amounts of D. Both reduction of natural saturated animal fats and omega-3 fish oils and increase of polyunsaturated omega-6 contribute to the current vitamin D insufficiency. Additionally excess intake of omega-6 fats down-regulate the production of vitamin D_3 upregulated protein 1, necessary for production of NK (natural killer cells) as well as other immune components making high intake of omega-6 fats promoters of cancer.[55]

So there you have it, lots of great reasons get tested for D and if you need more bump up your D supplementation or get out to the beach (even if it is only the 'beach' in your back yard).

What The Experts Say

Vieth's Review of D Sufficiency

Am J Clin Nutr 1999 May;69(5):842-56
Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety.
Vieth R
Department of Laboratory Medicine and Pathobiology, University of Toronto, Mount Sinai Hospital, Ontario, Canada.

For adults, the 5-microg (200 IU) vitamin D recommended dietary allowance may prevent osteomalacia in the absence of sunlight, but more is needed to help prevent osteoporosis and secondary hyperparathyroidism. Other benefits of vitamin D supplementation are implicated epidemiologically: prevention of some cancers, osteoarthritis progression, multiple sclerosis, and hypertension. Total-body sun exposure easily provides the equivalent of 250 microg (10,000 IU) vitamin D/d, suggesting that this is a physiologic limit. Sailors in US submarines are deprived of environmentally acquired vitamin D equivalent to 20-50 microg (800-2,000 IU)/d. The assembled data from many vitamin D supplementation studies reveal a curve for vitamin D dose versus serum 25-hydroxyvitamin D [25(OH)D] response that is surprisingly flat up to 250 microg (10,000 IU) vitamin D/d. To ensure that serum 25(OH)D concentrations exceed 100 nmol/L, a total vitamin D supply of 100 microg (4000 IU)/d is required. Except in those with conditions causing hypersensitivity, there is no evidence of adverse effects with serum 25(OH)D concentrations < 140 nmol/L, which require a total vitamin D supply of 250 microg (10,000 IU)/d to attain. Published cases of vitamin D toxicity with hypercalcemia, for which the 25(OH)D concentration and vitamin D dose are known, all involve intake of > or = 1000 microg (40,000 IU)/d. Because vitamin D is potentially toxic intake of > 25 microg/d (1,000 IU) has been avoided even though the weight of evidence shows that the currently accepted, no observed adverse effect limit of 50 microg/d (2,000 IU) is too low by at least 5-fold.

According to Vieth current recommendations for vitamin D are woefully understated. 200-400 IU will prevent rickets in children but does not come close to optimum D sufficiency. He suggests 4,000 IU as a common daily dose.

Vieth's research takes place in Canada, at higher latitude where it is difficult to get D from sunlight at any time of year. He may have found 4,000 IU to be non-toxic in this environment but his research covers months not years. His recitation of data claiming 10,000 IU of D is produced by a day in the sun and that 10,000 IU of D daily is safe is also questionable. Review of these studies suggests the correlation between sun exposure and oral intake of vitamin D overlooked key factors.

It is likely many of us need more sun or vitamin D than we currently get. While more research is needed, the minimal daily requirement of D will more likely be 800-1,000 IU instead of the 200-400 IU currently suggested. This amount is calculated from all sources, food, and supplements. It does not include sun exposure.

Vieth's estimate, 4,000 IU, is 10 times the current RDA and matches Dr. Price's observation. Weston A. Price, writing in Nutrition and Physical Degeneration, noted that the primitive diet contained 'at least ten times' the amount of 'fat-soluble vitamins' as the white man's diet. [56] Vieth's new research seems to agree that this amount is what is necessary to maintain optimal health.

Footsteps Of Our Forefathers...A Short History Of D

Weston Price concluded primitive communities ingested 10 times the current level of fat soluble vitamins, A, D and K. Equatorial communities got adequate UV-B to produce D. Their neighbors to the far north and south depended on food sources of D as significant UV-B was hard to find. I mention this because food and sunlight interact. In a global context dark skins occur in areas where much UV-B hits the ground. In South America skins are not so dark, likely due to the cloud and tree cover which reduces surface UV-B exposure.

Our ancestors did not get D in large, infrequent doses as is the current treatment by physicians for D deficiency. While vitamin A can be gotten in a large infrequent dose from consumption of animal or fish liver and we have storage capacity in our livers for this vitamin, the same is not true as regards vitamin D.

While D does store in body fat, this storage is not sufficient to maintain optimum blood levels during winter months.[57,58] A single exposure to UV light will raise levels of 25(OH)D over the next 24 hours and then return to baseline within 7 days.

This issue of storage is covered more fully in the book but a condensed version is that the true 'reserve' of vitamin D is from cholecalciferol and 25(OH)D in the blood stream. Excess vitamin D from supplements (you can't get excess from food) is carried by chylomicrons from your liver to your fat cells. It is likely this is not a 'storage' site for D reserves at all but your body's way of keeping excess out of the blood to protect cells in the same way fat soluble toxins such as pesticides are removed and stored in body fat.

When levels of 25(OH)D drop there is no evidence body fat releases vitamin D to replenish serum 25-hydroxyvitamin D. These stores are released during starvation, fasting, and dieting (as are other toxins stored in your fat cells).

Historically our natural need for D was satisfied by moderate daily exposure to sunlight and/or daily intake from food, including eggs, fatty fish, and high vitamin dairy and organ meats. Consumption of the whole animal or whole fish provided significant amounts of many nutrients not found in typical US diets. Animals kept in sunlight (wild game, pastured animals, grass-fed beef and the like, in lower latitudes where UV-B is available) contain D. Lichen [59] consumed by reindeer contains pre-vitamin D supplying D where there is little sun. Our ancestors got their D from sun with additions from eating eggs, animals including the skin and organs, and fish including organs, skin and fat.[56]

Low fat diets and consumption of processed vegetable oils and hydrogenated fats contribute to worldwide D insufficiency as is presently occurring. Highly processed fat and processed carbohydrate diets are low in vitamin D and actually increase the need for vitamin D.[60]

Studies with rats showed problems with bones and other tissues on a Western Diet, high in refined carbohydrates and processed fats. Giving extra vitamin D and calcium reversed the negative changes even while continuing the poor diet.

Clearly sunlight contributes significantly to vitamin D status even in most northern and most southern areas considering the alteration of skin pigmentation as UV-B light declines. Melanin, the pigment that gives skin its color, binds UV-B. Lighter skin allows for greater exposure to free UV-B leading to higher levels of D. [61,62,63,64,65,66]

Typical food intakes of cholecalciferol in sun-deprived areas among native persons eating traditional foods such as fatty fish including the skin, fat, and organs, and seal oil range from 1,000 IU – 3,000 IU daily.

It is probable that some vitamin D from food was also common at other latitudes as consumption of the whole fish or animal, especially glands and organs all contain D which survives cooking and drying. 4 oz. of kippered (dried) herring contains >1,000 IU D_3. Sardines and salmon as well as pickled herring remain excellent food sources of D.

Vitamin D Endocrine System and Nutrient Balance

Adequate calcium, magnesium, trace minerals, vitamins A, K and C are critical parts of D sufficiency. Vitamin D maintains serum calcium, not bone calcium. If daily calcium intake and therefore serum calcium is insufficient calcium will be withdrawn from bone. This is the reason persons using bisphosphonate drugs such as Fosamax, which keep calcium in the bone, must supplement calcium. The drug prevents their body from regulating serum calcium by preventing the movement of calcium in and out of bone.

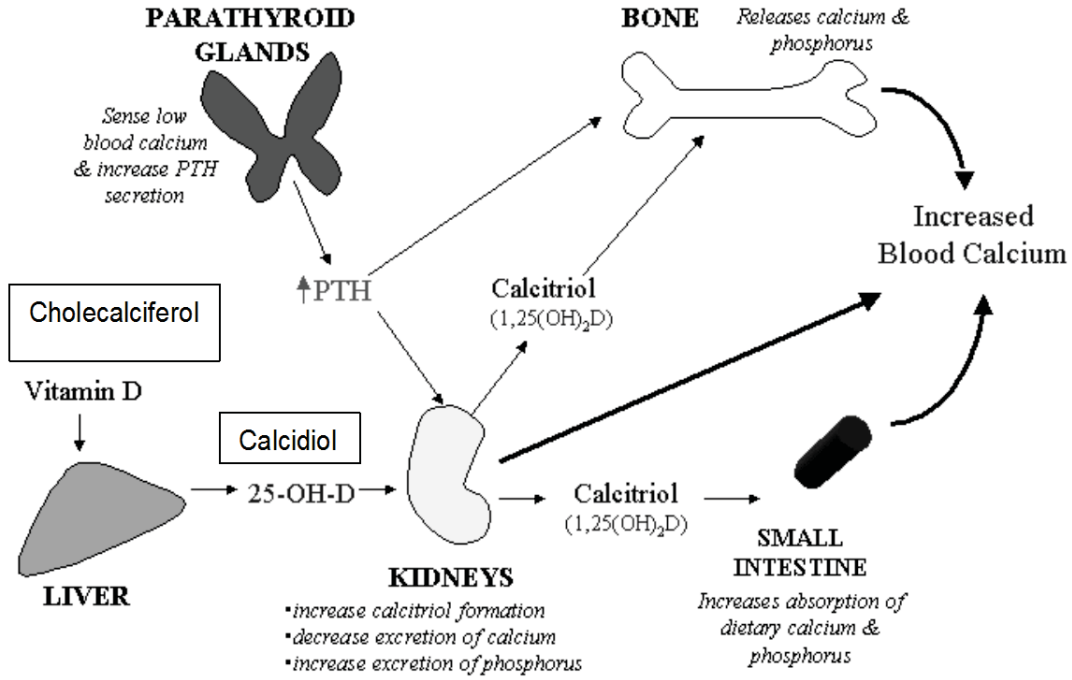
Do not supplement with D or sunlight unless calcium and magnesium are consumed regularly. Diets high in sugar and low in calcium, magnesium, and potassium contribute to significant bone loss.

Levels of 25(OH)D can test low but not actually indicate a need for more D if calcium in the diet is low. If you do not regularly consume calcium consider a calcium/magnesium supplement for a month before testing your D.

Balancing Act, Dancing on the Head of a Pin

The chart of the vitamin D endocrine system does not show many of the genomic functions of vitamin D. Vitamin D regulates the growth and death of many types of cells not just bone.[67,68,69,70,71,72] This broader view of the importance of vitamin D is quite new and as yet not well understood. Calcidiol (25(OH)D), thought to be an inactive metabolite of D is appearing to play a key role in many cell systems.[34,73,74,75,76]

Figure 0-1 The Vitamin D Endocrine System



Living systems are bounded by homeostatic mechanisms. To control the range of calcium in the blood our cells communicate with each other regulating when to excrete excess and when to absorb more. When calcium or vitamin D, 25(OH)D is below 32 ng/ml, parathyroid hormone may be elevated.[77] Studies in Europe found instances of elevation of PTH when 25(OH)D dropped below 40 ng/ml (100 nmol/l). [78,79]

PTH increases to increase conversion of 25(OH)D to 1,25(OH)2D, the form of vitamin D that increases gut absorption of calcium. (note-There are a number of vitamin D metabolites with differing roles) Elevation of PTH is important as it prevents hypocalcemia, low blood calcium. But there is a down side to higher levels of PTH.

Elevated parathyroid hormone is associated with elevated levels of interleukin-6 (Il-6). Il-6 stimulates production of inflammatory C-Reactive Protein and other inflammatory products in the liver and fat cells.[80,81] Inflammation and elevated PTH are general markers for any number of diseases including cancer, heart disease, diabetes, obesity and osteoporosis. [80,82,83]
Along with making sure you take enough A, K and C, monitoring serum PTH (parathyroid hormone) as well as 25(OH)D may be the best way to determine the health of your vitamin D endocrine system. If your serum calcium is within normal range, 25(OH)D between 40-60 ng/ml and PTH low normal it is likely all of the related cells of your body are happy, not just your bones.

About those other vitamins- Not reflected in the chart are the important roles of vitamins A, K and C, briefly covered here. Vitamin A, enough but not too much, is important to your immunity and your bone health. The relationship between A and D occurs on the gene itself where there are TWO vitamin A receptors for every ONE vitamin D receptor. In addition to playing an important role in human health, in every cell, excess or insufficient A will change the ability of vitamin D to function normally.[84,85,86,87,88,89]
Vitamin K regulates a protein controlling the movement of calcium, enough K keeping vitamin D in hard tissues, including bone, hair and nails, and out of soft tissues like arteries, joints and kidneys, helping to prevent osteoporosis and osteoarthritis.[90,91,92,93,94,95,96,97]
Vitamin C is not yet reaching its place among bone health experts. Recently researchers found mice bred to make less vitamin C (mice make their own, humans can't) rapidly developed osteoporosis when stressed. Their damaged C producing genes could not produce sufficient C when stressed (we ALL need extra C when stressed, physically, mentally or emotionally) and this insufficiency changed the ability to make and maintain bone.

HOUSTON -- (May 11, 2010) -- Vitamin C, or ascorbate, plays an important role in maintaining bone mass – promoting the balance between old bone resorption and new bone formation, said researchers from Baylor College of Medicine and Lexicon Pharmaceuticals in a report that appears online in the Journal of Biological Chemistry.

"The assumption is that everyone gets enough vitamin C in their diet," said Dr. Kenneth Gabbay, professor of pediatrics – molecular diabetes and metabolism at BCM. "However, multiple studies of large groups of people show that higher intakes of vitamin C are associated with higher bone mass and lower fracture rates. Our study shows that vitamin C or ascorbate is critical to maintaining the homeostasis necessary for healthy bone mass."

In particular, he referred to the Framingham Osteoporosis study and the Women's Health Initiative, both of which involved thousands of participants.

Gabbay and his colleagues built on the fact that mice can actually synthesize vitamin C, an ability that is lacking in humans. They identified two enzymes critical to this process by providing the building material for vitamin C – aldehyde reductase and aldose reductase. Aldehyde reductase is responsible for 85 percent of vitamin C production and aldose reductase, the remaining 15 percent. Mice bred to lack both enzymes cannot make any vitamin C and develop scurvy, a condition that affects many organ systems including bone.

However, if mice lack only aldehyde reductase, they and their skeletons develop and grow normally on the 15 percent ascorbate or vitamin X generated through aldose reductase until they face a stressor that requires more vitamin C, such as pregnancy or the loss of sex hormones that accompany menopause and aging.

"Then they fall off a cliff and develop early profound osteoporosis," said Gabbay.

…His studies (in mice) show that ascorbate or vitamin C both suppresses osteoclasts, which promote bone resorption, and stimulates the development of osteoblasts that make new bone, thus enhancing new bone formation. The constant renewal of bone is crucial to healthy bone architecture.

Many treatments for osteoporosis, including bisphosphonates such as Fosamax and Actonel, suppress the function of osteoclasts, and hence blocks bone resorption and mechanisms of bone repair. Unfortunately, these treatments do not stimulate osteoblast formation and new bone is not made. Many anti-oxidants such as resveratrol (found in red wine) and pycnogenol do the same thing. ***Only vitamin C affects both sides of the equation – osteoclast suppression and osteoblast development****, said Gabbay.*

…Most experts recommend vitamin D, calcium, exercise and bisphosphonates to keep bones healthy, said Gabbay.

"Vitamin C is never mentioned, whereas it's likely an equally important element for maintaining strong healthy bones" he said. "Our studies necessitate formal studies in patients to evaluate the usefulness of vitamin C therapy in susceptible populations."…

In addition to regulating bone dissolution and regeneration vitamin C also plays a role in hydroxylation, a process necessary to convert D3 from supplements or sunlight into the active 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D.

Supplementing Minerals

For healthy bones (and a healthy body) you need all of the essential minerals including calcium, magnesium, and potassium.[98,99,100,101,102] Potassium is important because it reduces the requirement for calcium.[101] as well as protecting against stroke and hypertension.

If the diet does not contain sufficient calcium and other associated minerals, possible only with daily consumption of whole fruits, vegetables, bone based broths and/or significant amounts of dairy as milk and cheese,[56], you may need to add supplements.

Multi-mineral tablets with calcium, magnesium and other minerals are an excellent source of needed calcium with synergistic minerals. Calcium carbonate and lactate are inexpensive and well-utilized sources of calcium. [103] Bone meal (Solgar or Kal), dolomite powder (Kal), or calcium lactate powder or tablets (Solgar, Now or Twinlab) are good sources, inexpensive and safe. All of these brands have been tested to be free of lead and other heavy metals.

If your diet is high in protein, calcium lactate or carbonate are probably better sources of calcium than bone meal.[104] Bone meal is very low in magnesium, which is acceptable if your intake of dark green leafy vegetables, nuts and legumes is daily and large. If you don't eat greens consider another calcium source that has magnesium in it.[105] Magnesium is critical to bone health as well as copper and zinc.[106,107,108,109]

Look at the label carefully to see how much elemental calcium is contained in each dose or tablet. If the label says a serving size is 3 tablets and contains 1,000 mg of calcium you must take the full serving size to get that amount. Labels are often difficult to read and understand. Most brands of calcium sold in health food stores list the elemental amount. Brands found in your local pharmacy may not.

Depending on the type of calcium, supplements may contain as little as 75 mg per tablet or capsule (Now Foods Calcium Lactate) to as much as 500 mg calcium (some calcium carbonate formulas). Calcium carbonate is 40% calcium, calcium citrate, 24%, calcium lactate contains 18.3% calcium, and dolomite contains 21% calcium and 13% magnesium.

Oyster shell and eggshell provide calcium carbonate containing 40% calcium. Bone meal contains 32% calcium, 15% phosphorous, 0.4% magnesium, and 4-12% protein and trace amounts of fluorine, iron, copper, chromium and manganese.

Some untested brands of dolomite, eggshell, oyster shell and bone meal may contain varying amounts of lead and other toxic metals. Avoid them.

Companies offering well-formulated multi-mineral supplements include Now Foods, Country Life, Solgar, Twinlab, and Nutrition Resource.

Higher amounts of calcium (with other essential minerals) are important for anyone diagnosed with bone loss or if serum parathyroid hormone is somewhat elevated. Total daily calcium from supplements may range from 800 mg to 1,500 mg depending on diet, current bone status and your body size.

Make the effort to split up your daily dose. Do not take all your calcium and magnesium once a day. A higher % of the calcium dose is absorbed if delivered in smaller, more frequent amounts.

500 mg. calcium-one dose	29% absorption
500 mg. calcium-two doses	36% absorption
500 mg. calcium-3 doses	40% absorption
2,000 mg- one dose	14% absorption

Heaney, RP et.al. J of Bone and Mineral Research, 5:11; 1990 p.1135-1137

Supplementing Accessory Nutrients

The Germans determined a minimum dose of 900 mcg vitamin K is necessary for full carboxylation of the calcium controlling protein. That is almost 1 mg. and not available in any multivitamin supplement. Life Extension http://lef.org and Complementary Prescriptions http://cpmedical.net (use my pin to order

230288) sell K supplements of sufficient dose to get what you need taking one a day with a fatty meal (K is fat soluble).
The dose of C required to keep us healthy is certainly much greater than the current DRI of 600 mg. It is likely we would all benefit from a minimum of 2,000 mg vitamin C TWICE a day. Some persons may benefit from much higher amounts, such as 4,000 mg two or three times a day, and If stress or illness or injury occur even greater amounts may be needed for a period of time.
Vitamin A is discussed in other parts of this packet. Typical safe and adequate dose of PRE-FORMED vitamin A (retinol, not beta-carotene) is likely 3,000-10,000 IU daily (about one large serving of beef liver once a week). If you use cod liver oil to get your vitamin D make sure it does NOT have excess A. No one yet knows the ratio to daily intake but it is likely 3,000 IU A to each 1,000 IU D.

Salivary pH Testing

C. Reich, MD and Weston Price, DDS found that monitoring AM pre-meal saliva (or mid-morning, two hours post eating) gave a good reading of ionized calcium and alkaline reserve. Current research confirms the value of this testing. [110] This test may be used for monitoring your alkaline reserve and your response to calcium supplementation BUT ONLY IF YOUR D IS WITHIN OPTIMAL RANGE.
Low pH does not mean you should raise your supplemental D. Use serum (blood) testing to determine D levels and retest after supplementing to check dose response.
Use pH papers to determine your overall alkaline reserve (intake of alkaline minerals including calcium, magnesium, potassium), and monitor the efficacy of mineral supplementation.
Optimal salivary pH values taken on arising before eating or drinking typically range from 6.8-7.2. This suggests an adequate alkaline reserve if vitamin D is within range.
Use pH papers with a range of 5.5-8.0 in 0.2 increments. Papers with 0.5 increments are not sensitive enough to use for pH monitoring. You may find sensitive papers at your local drugstore, online, or you may order them from Pike Agri-Lab Supplies RR 2 Box 710 Strong, ME 04983 1-207-684-5131
IF SERUM 25(OH)D IS NOT WITHIN OPTIMAL RANGE SALIVARY PH HAS NO DIAGNOSTIC VALUE AS TO MINERAL RESERVES.

Using Supplemental D Safely

400 IU of vitamin D, commonly available in multiple vitamins or as a single tablet or soft gel seem to have little value in addressing vitamin D sufficiency. Whether it is the type of D or just that the dose is so low remains to be determined. In studies with postmenopausal women 800-1,000 IU were necessary to alter 25(OH)D over a period of 6 or more months.

Cod liver oil contains both A and D and as yet our understanding of these two vitamins, their relationship to each other, is poor. What the literature does support is the common finding that elevated levels of D cause symptoms similar to vitamin A deficiency and elevated levels of vitamin A, symptoms of vitamin D deficiency. It is better to have too much A and D (at the same time) or too little A and D than have elevated levels of just one of these pre-hormone vitamins.

In Sweden women using a multivitamin containing as little as 5,000 IU of vitamin A suffered excessive bone loss and in man and animals excessive A contributes to fragile bones, hip fractures and osteoporosis.[85,86,87] Giving vitamin A when vitamin D is in excess helps protect bone.[84,89] Too little vitamin A also contributes to poor bone formation.[88,111]

What it means: We need some A and some D. Currently there is no recommendation for this relationship. In Sweden, with little sun, extra A may be too much, out-weighing vitamin D. In the tropics vitamin A intake may be too little as there is rapid turnover of vitamin when skin is exposed to UV light.[112]

Current suggestions regarding this problem of balance have a high probability of being wrong. That being said, it is likely the relationship between A and D is equivalent to a ratio of 5:1. That is 5 times more A than D. It is certain the intake of 5,000 IU A and 400 IU D, 12.5 times more A than D is not sufficient without regular UV-B sun exposure.

Testing Vitamin D

For many years the acceptable level of 25(OH)D has been >40 nmol/l (>16 ng/ml). Holick believes >20ng/ml (>50nmol/l) should be the lower acceptable limit.[113] Vieth proposes that this is far from optimal and has collected a large amount of data to suggest he is right. [114] Optimal levels are certainly >80 nmol/l (>32 ng/ml) and preferably closer to 100-150 nmol/l (40-60 ng/ml). In a personal correspondence with Dr. Holick August, 2000, Dr. Holick states optimal levels will be higher, in agreement with Vieth.

There are two readily available vitamin D tests- 1,25(OH)2D (calcitriol) and 25(OH)D (calciol). The first is active D, which is often normal even when the precursor, 25(OH)D, is insufficient. 25(OH)D is the better marker of overall D status and it is this marker that is most strongly associated with overall health. The correct test for D sufficiency and the one for which values are given in this paper is the 25(OH)D also called 25-hydroxyvitamin D.

Test results may be expressed in ng/ml or nmol/l (nanograms per milliliter or nanomoles per liter). It depends on the testing done by the lab. These numbers can be converted back and forth. 1 ng/ml is equivalent to 2.5 nmol/l so just multiply or divide by 2.5 to convert.

When you check with your physician he or she may not be familiar with this test. Be very clear about what you want. For most labs this is a send-out and results will take 1 week to receive. A Physician Protocol is available from me. Testing is available from http://lef.org (Life Extension website) costs $67 for non-members; http://privatemdlabs.com for about $60, less with coupon. For more information and testing sources visit http://sunlightd.org

If your 25(OH)D is sub-optimal supplementation is safe as long as sarcoidosis, liver or kidney disease is not present. Have your physician test your levels prior to supplementing to substantiate need for supplementation and retest to verify your supplements are working. **Don't assume you do or do not need vitamin D.**

One other condition requires caution. Hyperparathyroidism means your blood test showed elevated levels of parathyroid hormone (PTH). The condition may be primary, caused by a tumor, or secondary to kidney disease or calcium or vitamin D deficiency.
If your serum calcium is normal or low and your PTH is elevated you are a candidate for calcium and vitamin D. If your PTH is elevated and your calcium is elevated above normal you need careful monitoring by an endocrinologist experienced with your symptoms.
In some cases enlarged parathyroid may not be caused by a tumor and may be successfully be treated with vitamin D and calcium. The first step is to determine levels of both 25(OH)D and 1,25(OH)2D, serum calcium and serum PTH.

Supplements or Sunlight?

Supplements may be used during winter months and summer months when it is impossible to get outside, 'naked at noon', regularly. Some overlap of sun and supplementation at the levels suggested in this paper would not cause excess elevations of vitamin D in most cases.
As you will see, white or light skin does not require much sun to get a full dose of vitamin D when levels of UV-B are high. Limit exposure to the time needed to get your daily D, depending on skin type, latitude, altitude, and season (info further on). Combining excess vitamin D or cod liver oil and sunlight is not a good idea. Some enthusiastic supplement users combining D with sunlight have raised their 25(OH)D to potentially excessive values in excess of 80 ng/ml (200 nmol/l)

Your need for sunlight and vitamin D must be determined by you. You are also the person who must determine how to get D, by supplements or sunlight or a combination. Food cannot supply your daily need.
When first increasing vitamin D a paradoxical transient and non-complicating hypercalciuria, excess calcium in the urine, may occur. This is not a sign of toxicity and should not alarm you or your physician. It is quickly resolved when calcium and vitamin K supplementation is adequate.
There is an exception. If hypercalciuria, excess urinary calcium, occurs in combination with elevated 25(OH)D all sunning and supplements, including calcium, must be stopped immediately and avoided until 25(OH)D reaches 50 ng/ml or lower.
Two other transitional 'symptoms' have been noted by persons optimizing vitamin D. The first is transient midday sleepiness, like a child's naptime slump. It seems to last for about one week, and not always every day. Take a nap and keep going. The second is a 'reawakening' of old injuries, with associated discomfort. It is expected that calcium deposits within these injuries may resolve but we do not have data at this time.

Supplementing Vitamin D

Modern food is a very poor source of vitamin D. [56,115]
AVAILABLE SUPPLEMENTS- All of the companies have now switched to D3 cholecalciferol from irradiated sheep's wool fat. There is some evidence oil based vitamin D may absorb better, most supplements now contain dry vitamin D.

If you have symptoms of D insufficiency or you know you have not gotten adequate sun speak with your physician and arrange for testing.
Supplements of 2,000-5,000 IU may be needed on a regular basis. Higher levels should only be recommended and monitored by a knowledgeable health care practitioner.
There has been ongoing controversy concerning the use of vitamin D_3, cholecalciferol, from fish oil (as a concentrate- just using cod liver oil may put the ratio of vitamin A to D too high for treating insufficiency) or irradiated sheep's wool fat, or plant source D_2, ergocalciferol, such as found in fortified foods and some supplements. Some researchers suggest the problems of D toxicity only occur with ergocalciferol. There is no clinical data to prove this. Both D2 and D3 are implicated in hypervitaminosis D (too much D).
D_2, found in plants and made active by irradiation is less biologically active in humans. D_3, cholecalciferol, as found in eggs, organ meats, animal fat, cod liver oil and fish. This D_3 is the same D_3 formed on our skins from UV-B.
Taken orally, D_3 is potentially more toxic than D_2 at high doses because it is more biologically active.[116,117,118] but we are not concerned here with the mega-doses of D being used in research. Our concern is finding the supplement that works best for daily use.
D3 from irradiated sheep's wool fat, at moderate doses determined by testing, works best because it contains the most biologically active D_3 important to our health.
Cod liver oil, a standard in past decades, is not an optimal source due to taste, short storage life, and more importantly due to the often high level of vitamin A in relationship to D content. Vitamin A and D interact on the nuclear membrane. Too much A may bring on symptoms of D deficiency including bone loss [85,119,120,121] and too much D (both as D and in relationship to vitamin A intake) can cause symptoms similar to vitamin A deficiency.[122]
Carlson Cod Liver Oil has adjusted the A and D ratio so that one can get adequate D without an excess of vitamin A. It is the only cod liver oil I would recommend. It is possible that cod liver oil might be an adequate source for some persons in some locations. Moderate doses of cod liver oil combined with moderate sunlight may offer optimal A and D. Testing can help determine this.
Testing is necessary to determine dose. Testing must be continued over several years as I have found that the initial treatment dose frequently becomes an excessive dose sometime during the first year or second year of supplementation. Supplementation without testing may result in excess D.

What about toxicity? Isn't too much D or sun dangerous?

It is impossible to talk about D or sunlight without the words toxic or dangerous, appearing in the conversation. Yes, fat soluble vitamins can be toxic (which, by the way, includes vitamin E) and yes sunlight does contribute to skin cancer, BUT we need light and we need fat soluble vitamins and we need however much we need which it seems has not been, so far, truly understood.
With light and D there is too little, just right and too much (remember Goldilocks?). For each of us these values will be somewhat different. Genetics and location- light skinned persons nearer the equator, dark skinned persons more distant, become very problematic- we were designed for the food and light of our ancestors and, well, we moved.
Obviously some guidelines are needed. Yes, there is possible excess or toxicity but it is not caused by normal, skin safe, exposure to mid-day UV-B containing sun and it is not caused by sensible supplementation when deemed necessary and monitored by testing.
Potentially excessive levels of D occur when blood 25(OH)D reach values in excess of 200 nmol/l (80 ng/ml), for extended periods of time. Holick suggests serious, life-threatening toxicity (chronic poisoning) may begin at values of 125 ng/ml or 312.5 nmol/l. (Note as of 5/2009: Since the new NORMALS are as high as 100 ng/ml or 250 nmol/l we may be approaching a vitamin D disaster.)

Levels of 200-300 nmol/l or higher have been seen in several studies using high dose supplementation in research and quickly resolve when supplementation is stopped. In such cases no long-term problems have been reported. However, the excess has usually occurred rapidly by use of very high dose vitamin D and resolves quickly because frequent testing uncovers the excess before damage might occur.
In real life chronic use, over months or years, of high dose D (more than 1,000 IU daily in sensitive persons), with or without sunlight, may so saturate fat stores 25(OH)D may take as long as a year to return to normal.
Vieth suggests that critical toxicity may occur at doses of 20,000 IU daily over an extended period of time. It is also suggested that the UL (upper limit of safety) be set at 10,000 IU rather than the current 2,000 IU. [123] [114] My opinion, after 10 years of studying the research and seeing the results of high dose D used for supplementation is that he is wrong.
In the second and third years of vitamin D supplementation, in clients using doses as low as 2,000 IU, I frequently began to see signs of hypervitaminosis D. Just 2,000 IU taken daily for two or three years combined with summer sun has resulted in elevated 25(OH)D.
Toxic levels are not thought to occur with sun exposure [114] but may occur with certain diseases or excess use of D supplements or foods contaminated with D or combining D with sunlight.
I have seen 4 cases of D excess from sunlight exposure after regular supplementation of vitamin D, have heard of more than 30 cases with other clinicians and have worked with two cases of D excess caused by sunlight alone, thought not to be possible by researchers.
Concerns about vitamin D excess should occur when supplemental levels exceed 1,000 IU daily for extended periods of time combined with regular sun exposure or 2,000 IU daily for months or years. This can be confirmed if blood levels of 25(OH)D exceed 200 nmol/l, 80 ng/ml.[114] Problems may occur in some with values of 162 nmol/l, 65 ng/ml.
This value has been thought by some researchers to be too low as values of 162 nmol/l, 65 ng/ml are seen regularly in sun-exposed individuals in the lower latitudes with no indication of toxicity. The problem is these higher values may be causing bone loss or heart damage (arterial calcification) without the accepted hypervitaminosis D symptoms of elevated serum calcium or elevated urinary calcium.
As of 2009 most laboratories in the US now consider 32-100 ng/ml as NORMAL (80-250 nmol/l) I have yet to understand why this change has been approved.
There is no long-term clinical evidence levels of 25(OH)D above 65 ng/ml (163 nmol/l) are advantageous or SAFE. Testing prevents the possibility of exceeding optimal, safe limits. There is no research showing values of vitamin D above 60 ng/ml provide any additional health benefits as compared to values between 40-60 ng/ml.
The genetic variable for utilization and storage of D is great. Some may be able to get all the D they need from sunlight, particularly if they live in a sunny location. Others may not be able to maintain optimal D even when sunning regularly due to aging of the skin[124,125], skin color[65,126,127,128] or location[129,130], latitude and altitude.
The need for supplementation seems to vary from 'none' (with sunlight providing all the D) to as much as 4,000 IU daily, monitored twice a year. In some few older men and women with aging skin and limited sun exposure a slightly higher dose may be needed.
There is no clinical evidence that daily supplementation, in most healthy adults, need exceed 2,000 IU for optimal daily maintenance and this level would only be appropriate if testing showed an insufficiency. This dose would NOT be appropriate in summer months unless the sun is completely avoided or your skin is dark. Persons with darker skins may need higher doses summer and winter.
Sunbathing and supplements should not be combined unless you are carefully monitoring your response.
Because of genetic variations testing is the only way to determine individual need for vitamin D. While there is no clinical evidence, work by Vieth and testing done by various physicians and health care

practitioners has shown that certain persons may need as much as 3,000 IU of D daily to reach 25(OH)D values >112 nmol/l, >40 ng/ml. Over longer term I have found that rarely is this dose needed for maintenance.
High initial doses used in clinical studies range from 10,000-500,000 IU or in rare cases 5,000,000 IU. given one time or daily, weekly or monthly. The rationale for these doses is either as a prophylactic or because compliance is considered problematic. There seems to be some evidence, and logic suggests, D works better, without toxicity, when provided in lower daily doses, amount determined by testing, rather than 100,000 IU month, however a onetime dose of 100,000 IU did replete low levels of D in adolescents during winter. [131]
As I see it, there is a problem using high, infrequent dosing. Blood levels moved from low to extremely high, >300 nmol/l 24 hours after a 50,000 IU oral dose, in one recent study [131] well above normal or optimal ranges, and then slowly return to pre-treatment sub-optimal levels. Clearly this mega-dosing with vitamin D must disrupt normal feedback mechanisms in the vitamin D endocrine system and thereby calcium regulation. This unusual raising and lowering of D may have other as yet unknown consequences and D intake regulates many receptors, enzymes, binding proteins and genes..
The long-term answer to vitamin D sufficiency is testing, giving optimal daily dose of D or sunlight and verifying that the dose is appropriate for the individual. Test, treat, retest.

Skin Cancer

Skin cancer is consistently related to sun exposure. While intermittent, intense exposure is associated with melanoma [132,133] chronic moderate exposure to higher levels of UV-B providing vitamin D has been associated with a decreased risk of melanoma. [134]
Basal and squamous cell cancers and sunlight damage can be prevented by careful use of sun and use of oral or topical vitamin A, vitamin C and/or selenium, caffeine or green tea or post sun exposure application of extra virgin olive oil, just to mention a few of the substances being studied.[135,136,137] [138,139,140,140,141,141,142,143,144,145,146,147]

A contributing cause to the current epidemic of skin cancers is a shift in diet from saturated and omega-3 fatty acids to readily oxidized polyunsaturated omega-6 fatty acids, which incorporate into skin cells and are oxidized (damaged) by UV light. [148] It is unfortunate that many sunscreens contain these oils.
Unwise over-exposure to sunlight is always harmful and never necessary. Light skins need UV-B but just a little and only when UV-B is present in sufficient amounts, which is summer, mid-day, between 10AM and 2PM in most of the US.
UV-A is present all day summer and winter and is strongly associated with melanoma.[149,150] Sunning before 10AM or after 2PM exposes us to UV-A with no appreciable UV-B to produce protective vitamin D.

Med Hypotheses. 2009 Apr;72(4):434-43. Epub 2009 Jan 19.
Increased UVA exposures and decreased cutaneous Vitamin D(3) levels may be responsible for the increasing incidence of melanoma.
Godar DE, Landry RJ, Lucas AD.
US Food and Drug Administration, Center for Devices and Radiological Health, 10903 New Hampshire Avenue (HFZ-120), Silver Spring, MD 20993-0002, USA. DEG@CDRH.FDA.GOV
Cutaneous malignant melanoma (CMM) has been increasing at a steady exponential rate in fair-skinned, indoor workers since before 1940. A paradox exists between indoor and outdoor workers because indoor workers get three to nine times less solar UV (290-400 nm) exposure than outdoor workers get, yet only indoor workers have an increasing incidence of CMM. Thus, another "factor(s)" is/are involved that increases the CMM risk for indoor workers. We hypothesize that one factor involves indoor exposures to UVA (321-400 nm) passing through windows, which can cause mutations and can break down vitamin D(3) formed after outdoor UVB (290-320 nm) exposure, and the other factor involves low levels of cutaneous vitamin D(3). After vitamin D(3) forms, melanoma cells can convert it to the hormone, 1,25-dihydroxyvitamin D(3), or calcitriol, which causes growth inhibition and apoptotic cell death in vitro and in vivo. We measured the outdoor and indoor solar irradiances and found indoor solar UVA irradiances represent about 25% (or 5-10 W/m(2)) of the outdoor irradiances and are about 60 times greater than fluorescent light irradiances. We calculated the outdoor and indoor UV contributions toward different biological endpoints by weighting the emission spectra by the action spectra: erythema, squamous cell carcinoma, melanoma (fish), and previtamin D(3). Furthermore, we found production of previtamin D(3) only occurs outside where there is enough UVB. We agree that intense, intermittent outdoor UV overexposures

and sunburns initiate CMM; we now propose that increased UVA exposures and inadequately maintained cutaneous levels of vitamin D(3) promotes CMM.
PMID: 19155143 [PubMed - in process]
Adv Exp Med Biol. 2008;624:86-8.
At what time should one go out in the sun?
Moan J, Dahlback A, Porojnicu AC.
Department of Radiation Biology, Institute for Cancer Research, Montebello, Oslo, Norway. johan.moan@labmed.uio.no
To get an optimal vitamin D supplement from the sun at a minimal risk of getting cutaneous malignant melanoma (CMM), the best time of sun exposure is noon. Thus, common health recommendations given by authorities in many countries, that sun exposure should be avoided for three to five hours around noon and postponed to the afternoon, may be wrong and may even promote CMM. The reasons for this are (1) The action spectrum for CMM is likely to be centered at longer wavelengths (UVA, ultraviolet A, 320-400 nm) than that of vitamin D generation (UVB, ultraviolet B, 280-320 nm). (2) Scattering of solar radiation on clear days is caused by small scattering elements, Rayleigh dominated and increases with decreasing wavelengths. A larger fraction of UVA than of UVB comes directly and unscattered from the sun. (3) The human body can be more realistically represented by a vertical cylinder than by a horizontal, planar surface, as done in almost all calculations in the literature. With the cylinder model, high UVA fluence rates last about twice as long after noon as high UVB fluence rates do. In view of this, short, nonerythemogenic exposures around noon should be recommended rather than longer nonerythemogenic exposures in the afternoon. This would give a maximal yield of vitamin D at a minimal CMM risk.
Publication Types: Research Support, Non-U.S. Gov't
PMID: 18348449 [PubMed - indexed for MEDLINE]

What this means: Sunbathing when UV-B is present, summer mid-day for most of the US, maximizes vitamin D, stimulates the immune system, and protects from the most dangerous skin cancer, melanoma.

WHAT? Yes, I did say that. Appropriate amounts of UV-B sunlight are health protective. Excess amounts of UV of any type, including UV-A, always present from sunrise to sunset, damage human skin and the immune system.

Safe Sun and Your Skin- Soaps and Lotions, Dangerous Chemicals

Your skin's outmost layer is the stratum corneum. Thought to be a useless layer of dead skin cells it is now known to be the outermost protective layer of skin that holds in water, protects underlying cells from UV damage and pathogenic microbes (bacterial infections). [151]

Sun sensitivity, the result of damage to the stratum corneum, is a side-effect of some common skin treatments and cleansers. If you decide to sun avoid topical chemicals. These chemicals, including skin peels and hydroxy acids damage the stratum corneum leaving newly developing cells open to water loss, radiation damage and infection.[152,153,154,155,156,157,158] When using a product such as 'fruit acids' to peel the skin it takes a minimum of 7 days to restore the stratum corneum to provide protection from the outside environment, including UV light. .

If you are using the sun to get your vitamin D never use products containing Retin-A; Alcohol (Isopropyl); Fluoride; Parabens; Sodium Lauryl Sulfate (SLS); DEA; Fragrance; Polyethylene Glycol (PEG); TEA; DMDM; Hydantoin; MEA; Propylene Glycol (PG); Triclosan; FD&C Color; Mineral Oil; Sodium Laureth Sulfate (SLES); Urea; or Hydantoin. Never use products containing alpha hydroxy acid (AHA) or other strong exfoliants.[159]

Read labels, including labels on your sunscreen and your shampoo and conditioner. The use of lotions, cosmetics and soaps containing sun sensitizing chemicals and other lipid dissolving chemicals damage the skin's barrier function and make your skin more susceptible to sun damage, water loss, and infection.

For a list of chemicals and reasons to avoid them read Beauty To Die For by Judi Vance or Dying to Look Good by Christine H Farlow.

Aubrey Organics and Zia have a number of safe shampoos, conditioners and other creams and lotions. If a label lists just a few ingredients it is likely there are more ingredients and the unlisted ones may be the ones you don't want.

While some of the increase in skin cancer incidence may be caused by increased exposure to the sun, especially in light skinned individuals living in lower latitudes, it is statistically more likely use of topical

soaps and chemicals, cosmetics, shaving creams, and other abuses of the stratum corneum make an equal or larger contribution to our skins' decline.
If you factor in the increased consumption of omega-6 fats, which in large quantities increase inflammation and susceptibility to UV damage, and decreased consumption of omega-3 fats, our skins have a right to find us unforgivable. What you put on your body and what you put in your body both alter your response to your environment. [160,161,162]

Sourcing Sunlight D, How Does It Happen?

Much of the US does not have sufficient sunlight to provide adequate UV-B to produce (optimal) D and supplies of D drop during winter months. In Northern California July sun gives all the D needed for skin types 1 or 2, in 15-30 minutes, ½ of the total time on each side. By September the timed exposure increases to 40-60 minutes for the same level of D production.

Ultraviolet (UV) light is divided into 3 bands or wavelength ranges, which are referred to as UV-C, UV-B and UV-A. [163]

- UV-C is the most energetic and shortest of the UV bands, and will burn human skin rapidly in extremely small doses, however it is completely absorbed by the ozone layer.
- UV-C is present in some lights, fluorescent and halogen and other specialty lights.
- UV-C and UV-A in halogen, fluorescent, black and other specialty lights can damage skin and may contribute to skin cancer incidence.
- UV-A, known as the "tanning ray" common in tanning beds, is primarily responsible for darkening the pigment in our skin. Most tanning bulbs have a high UV-A output, with a small percentage of UV-B.
- UV-A is less energetic than UV-B, so exposure to UV-A will not result in a burn unless the skin is photosensitive or excessive dosages are used.
- UV-A penetrates more deeply into the skin than UV-B. A concern for skin cancers.
- UV-A was not blocked by sunscreens until recently and most sunscreens still block UV-A poorly.
- UV-A is considered to be a major contributor to the high incidence of non-melanoma skin cancers. [164]
- UV-A is present throughout the day, for much longer than UV-B.
- 78% of UV-A penetrates glass and is the radiation responsible for non-melanoma cancers on the left arms of men and right arms of women associated with sunlight and auto use exposure over the past 20 years.
- UV-B, sometimes called the "burning ray" is the primary cause of sunburn (erythema) caused by overexposure to sunlight. However,
- UV-B initiates beneficial responses, stimulating the production of Vitamin D necessary to help our bodies absorb and utilize calcium and much more. .
- UV-B stimulates special skin cells called melanocytes to produce melanin, which becomes additional protective pigmentation and so increases the sun protection factor (SPF) in our skin.
- UV-B stimulates the production of alpha-MSH, melanocyte stimulating hormone, an important hormone in weight loss and energy production. [165]
- UV-B does not penetrate very deeply into the skin.
- The darker the pigmentation or more tanned the skin, the less UV-B is available for D conversion. Melanin 'quenches' UV-B, like an anti-oxidant..

- UV-B sunlight produces vitamin D on the skin. The amount produced depends on exposure time, latitude and altitude of location, amount of skin surface exposed, skin pigmentation and season.

Sunlight and vitamin D are critical to health in all life forms.

Higher latitudes need D from sun and supplements to achieve optimal benefit. Our ancestors got D from fish, wild game and organ meats and by spending significant time out of doors as well as living without glass.

Glass (as in windows) allows only 5% of the UV-B light range that produces D to get into your home or auto.

Sun exposure before 10AM or after 2PM will cause burning (from UV-A) before it will supply adequate D (UV-B). This finding may surprise you, as it did the researchers. It means in the US safe sunning for vitamin D must occur between hours we have been told to avoid. Sunning near noon during summer months (or winter months in lower latitudes) for 10-90 minutes, depending on skin type (color), will form adequate D before burning occurs. [166]

Absorption of D containing oils takes about 30 minutes, conversion to active 25(OH)D about 2.5 hours. [62]

The current suggested exposure of hands and face or arms to 10-20 minutes three times a week during the summer would provide only 200-400 IU D each time (about 100-200 IU a day). 85% of body surface needs exposure to prime mid-day sun for 10-20 minutes (light skinned) to 90-120 minutes (dark skinned) to achieve optimal levels of D. [167,168]

During the months UV-B is significantly present sunbathing is necessary to reach the optimal dose. Just exposing arms, legs and face two or three times a week as is currently recommended is not sufficient. [28]

We make D in our skins. Each cellular 'factory' can only make so much D within a given period of time. Arms and legs, especially if you are standing up and walking around, just don't get enough exposure or have enough 'factories' to make sufficient D. For some persons supplementation is critical.

The surface area of exposed skin times the intensity of UV-B exposure determines your skin's production of vitamin D.

- Latitude and altitude determine the intensity of UV light. Latitudes higher than 30° (both north and south) have insufficient UV-B sunlight 2-6 months of the year (even mid-day).
- Latitudes higher than 40° have insufficient sunlight to achieve optimum levels of D 6-8 months of the year.
- Most of the US is between 30° and 45°. In much of the US 6 months or more during each year have insufficient sunlight (UV-B) to produce optimal D levels.
- In far north or south locations, latitudes 45° and higher, even summer sun is too weak to provide optimum levels of D. [169]

Quick comparisons between

UV-B	UV-A
It is the only range of ultraviolet to activate D on human skin.	
It stimulates melanin formation (tanning) and alpha-MSH. (melanocyte stimulating hormone	It 'browns' melanin produced by UV-B, your tan and moles, sun or "age" spots.
It is strongest between near noon in most of the US	It is at full strength from sunrise to sunset.

It is strongest at the equator.	It is strong from pole to pole.
It is stronger at high altitudes.	It is strong at any altitude.
It does not penetrate deeply into skin. It is thought to cause superficial basal cell skin cancers.	It penetrates deeply into the skin reaching skin cells' DNA and **it has been linked to 67% of malignant melanoma**
It reflects off shiny or white surfaces (water, sand, concrete, etc.) but also penetrates water to some degree, if the water is clear.	
It is 1,000 times more biologically active than UV-A.	It is present 1,000 times more than UV-B.
It does not penetrate glass (5% only)	It penetrates glass (78%, including car windows).

Getting D from the Sun, Safely

If sun burning always occurs at any exposure level or there is prior incidence of skin cancer you may not want to use sunlight to maintain D. Test and use a supplement.

To use sunlight in lieu of supplementation refer to the skin pigmentation guide. The guide provides an estimate of the time needed to optimal levels of D. These exposure times are sufficient but remember they are calculated on UV-B intensity. **Longer exposure, than your skin type needs, will NOT increase vitamin D production beyond optimal levels and will increase the danger of skin damage in all locations**.
This guide applies in seasons, latitudes, and altitudes of UV-B production. Please see the latitude and altitude information for UV-B levels or consider a UV-B meter. Remember, UV-B does not penetrate glass, smog, fog, some haze or clouds.

UV-B Exposure Guidelines

- For the optimal daily dose of D from sunlight 80% of skin must be exposed for the full time, that is the time just **prior** to any skin changes (pinkness).
- In most locations in the US significant UV-B is present only during mid-day and only during summer months.
- Exposure must take place when UV-B is present, between 10AM and 2PM during the summer for most of the United States, on cloud and smog free days. Near noon is best. Before 10AM or after 2PM burning may occur before optimal D is produced.

The closer to noon the less time needed to achieve D production. Do not over sun.
The time needed to produce adequate D depends on skin color.

Skin Type	Time Needed for Daily D Production
Type 1- Always burns easily, never tans, extremely sun-sensitive skin. **Examples:** Red-haired, freckles, Celtic, Irish-Scots.	5-7 minutes per side, 10 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 2- Always burns easily, tans minimally, very sun-sensitive skin. **Examples:** Fair-skinned, fair haired, blue-eyed, Caucasians	10-15 minutes per side, 20 minutes full body at UV Index 9-10, above 10 less time is needed.

Type 3- Sometimes burns, tans gradually to light brown, sun-sensitive skin. **Examples:** Darker Caucasians	20-30 minutes per side, 25 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 4- Burns minimally, always tans to moderate brown, minimally sun-sensitive. **Examples:** Mediterranean type, Caucasians.	40 minutes per side, 70-80 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 5- Rarely burns, tans well, sun-insensitive skin. **Examples:** Middle Eastern, some Hispanics, some Blacks	50 minutes per side, 90-100 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 6- Never burns, deeply pigmented, sun-insensitive skin. **Example:** Blacks	60 minutes per side, 120 minutes full UV-Index 9 or above body at UV Index 9-10, above 10 less time needed.

For type 1-3 skins sunbathing near noon will assure enough UV-B to produce D before burning occurs. For skin types 4-6 UV intensity is critical. Many parts of the US may not provide adequate UV-B to optimize D.

A note about the UV Index-

The weather service puts out daily bulletins estimating the UV intensity. Go to http://iwin.nws.noaa.gov/iwin/us/ultraviolet.html. The problem with these ratings is that they do not actually look at the local UV-B but estimate from 'sources'. I prefer to use my Sunsor™ UV-B meter. It is battery free and about the size of a small calculator. I carry it with me and can know instantly how much time I need to produce my daily D or if UV-B is too low to make sunning worth my while. A number of UV-B meters are available but expensive. Sun meters (not UV-B) won't work as they include more UV-A in their calculations.

UV Index Value and Sun Exposure Levels- National Weather Service's UV Index. Note UV-B is present during primarily mid-day in locations with values of <10. Time is for mid-day exposure. Index readings are for the mid-day value when UV is highest. Most of the day values will be lower than that indicated by the UV Index.
0 - 2 (Minimal) - An Index reading of 0 - 2 indicates minimal danger from the sun's UV radiation for the average (Type 2) person. Most people can stay in the noon sun for up to an hour without burning. Sufficient vitamin D will not be produced at this level before skin damage would occur. **(No D all skin types)**
3 - 4 (Low) - An index reading of 3 -4 indicates low risk of harm to the skin from the sun's radiation. Type 2 individuals can experience a burn in 30 -60 minutes. It would be necessary to stay in the sun for longer than 60 minutes to activate D and burning may occur before D was produced. **(No D all skin types)**
5 -6 (Moderate) - An index reading of 5 - 6 indicates some significant risk of skin damage due to the sun. Unprotected exposure can result in a burn in only 20 - 30 minutes. At this level burning or skin damage would also occur before adequate D production. **(D in light skin types only, but not much)**
7 -9 (High) - An index reading of 7 - 9 indicates high risk of harm from unprotected exposure to the sun. Time in the sun should be limited during midday (10:00 A.M. - 4:00 P.M.) as skin may burn in as little as 13 - 20 minutes. Enough D would be generated in 10-20 minutes (before burning) in type 1 and 2 skins. **(D in light and medium skins midday, very little D in dark skins)**
10+ (Very High) - An index reading of 10 - 15 indicates very high risk of harm from unprotected sun exposure. Between 10:00 A.M. and 4:00 P.M. the length of time to burn may be less than 13 minutes without protection. Enough D would be generated in 10 minutes (before burning) in type 1 and 2 skins. **(D all skin types, very little time needed for light skins, use caution.)**

While some D might be produced in light skins at the lower UV Index readings the damage from excess exposure to UV-A would outweigh the worth of sunning.

At higher latitudes, most of the US, the UV Index is for midday, not morning or afternoon. Only Hawaii, Florida, south Texas, Southern Arizona, and some higher elevations, have significant UV-B for a longer stretch of the day.

If sun exposure is or must be limited or avoided, use supplements. Testing is critical. We are all different and our responses are unique. Tropical sun must not be judged by these values. As little as a few minutes may be needed for persons with very light skin in subtropical or tropical locations.

Sunlight and supplementation should not be mixed. Cases of elevated D have occurred by persons supplementing and sunning. BUT if the sun you sun in is far to the north rarely will it have much UV-B and sunning and supplements may work together. This mix of sunlight and D should not be attempted without testing. Several persons over the last few summers found themselves having excess levels of D by combining D and sunlight. Others have found sunlight exposure altered their need for supplementing D very little.

TEST. This is particularly important in the US where during much of the year, in many locations, UV-B is not present in amounts able to produce D before burning. Depending on latitude this variable ranges from 2-3 months in San Diego and other cities of similar latitudes to 8 months in Portland, OR, upstate New York and other locations at higher latitudes.

The exact time needed, varying with skin types at various US latitudes and altitudes, is not yet adequately understood. A UV meter used with sunbathing and initial frequent testing of 25(OH)D may be useful to determine your optimal use of sun and supplements.

Genetics, both skin coloring and genes related to vitamin D response, strongly play a role in your need for D. Vitamin D receptor polymorphisms, genetic alterations regulating how we absorb and process D, are only recently being studied and the information applied to D sufficiency. [170]

Ozone and UV-B

While the popular press and some environmental groups suggest ozone depletion is responsible for the increased incidence of skin cancer there is no evidence this is true. If more UV-B were reaching the ground in the US or anywhere else on the globe we would have more vitamin D and there is no data showing such an increase.

What has changed is increased manmade ozone hovering over cities. You hear about it on the news when air pollution warnings are given for people suffering from asthma and other respiratory diseases.

This artificial layer of ozone has significantly decreased levels of UV-B in populated cities during hot summer months.[171,172] One possible explanation for increases in certain types of cancers in populated areas with increased ozone is less availability to sunlight vitamin D.[173] Los Angeles and San Diego suffer from intermittent, year-round, ozone blocking of UV-B as do many large cities throughout the US.

Sunscreen yes or no?

Sunscreens block UV-B effectively for 90-120 minutes. Newer sunscreens also block UV-A but less effectively.[174] As UV-A is implicated in melanoma and UV-A is present from sunrise to sunset, this leaves most unprotected. Blocking UV-B dramatically reduces or prevents production of vitamin D.[167,175]

Some studies suggest sunscreens blockage of D production is not a problem because tested subjects using sunscreen still had ‘adequate’ vitamin D but the values being used in these studies as adequate, less than 30 ng/ml, would now be considered inadequate.[176,177]

Perspiration or swimming, especially in pools with chlorine, may rapidly remove sunscreens or blocks and thereby any protective effect. Suggested use says to reapply frequently. As

much as one tube per person per beach day might be needed for full protection; expensive and unlikely to happen.
Sunscreens are the most common cause, 87%, of skin allergic reactions, often sensitizing the skin to other chemicals.[178,179]
The best way to protect yourself from excess sunlight, UV-A or UV-B, is seeking shade and wearing clothing.[180,181,182] In a European study researchers found clothing protective against the development of nevi (moles, a risk factor for melanoma) but NOT sunscreen.[183]
Wearing clothing, any type, not special 'sun-protective' clothing, and a broad brimmed hat provides sun protection and allows you to remove your clothing to get your daily dose of vitamin D. For strong protection wear tightly woven cotton in dark colors. White or light fabrics, especially synthetics, loosely woven, give the least protection (but still do protect).[184]

THE T-SHIRT/UV EXPERIMENT IN TIBET

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ABSTRACT
An experiment has been conducted in Lhasa, the capital of Tibet, at an altitude of 3650 m above sea level, to test the protective capability of T-shirts from solar UV radiation. The experiment was conducted on the 9th of June, 2000, with clear sky conditions using 6 different types of T-shirts, which were selected randomly according to color, fabrics and condition. The global solar UV irradiance and the corresponding transmission properties of the T-shirts were obtained by two identical instruments. The effective erythemal dose rate ratios between covered and uncovered instruments were measured to be in the range from 0.4 up to 6 %, depending mainly on fabrics and thickness of the shirts. Estimates of the corresponding daily Tibetan erythemal UV dose show that 3 of the shirts exceed the 1 MED dose level during summer. Thin cotton shirts have daily doses in the range 0.2 to 1.2 MED. The highest daily dose was found to be above 2.5 MED for the mixed fabricated shirt labeled 6% polyester and 94% cotton. Thick and loosely woven T-shirts of 100 % cotton have the best protective capability even when UV-B is at its peak.
What does it mean? Researchers tested the sun protective capacity of t-shirts in Tibet, during summer, at the point of highest UV-B. Tibet is 29° north and 3658 meters, (12,000 ft) high. At this latitude and altitude UV-B is more intense than at any location found in the US including Hawaii. The highest erythemal UV dose was between 9 and 10 kJ/m^2 during the study (this is UV dose not UV index). In the US, including Hawaii, the HIGHEST UV erythemal dose, during summer, is between 4 and 6 kJ/m^2, and UV dose approaching 6+ occurs at a very few locations.
. Clothing that would protect in Tibet will protect in the US.

UV dose [kJ/m^2]	T-shirt #1 [MED]	T-shirt #2 [MED]	T-shirt #3 [MED]	T-shirt #4 [MED]	T-shirt #5 [MED]	T-shirt #6 [MED]
2.10	0.26	0.27	0.04	0.59	0.16	0.08
7.60	0.94	0.96	0.13	2.13	0.59	0.29
9.18	1.14	1.16	0.16	2.57	0.71	0.35

Safe sunning keeps the daily dose of UV to less than 1 MED (minimal erythemal dose). As an entire day of sun in most of the US would not exceed 6 kJ/m^2 all but the cotton/polyester mix t-shirt would provide adequate sun protection.
Clothing, loose, comfortable and fun, with a sun protective hat provides adequate sunscreen without extra cost. Shade is also protective. Beach umbrellas, beach tents, and trees offer other sunscreen options without chemical overload. `
If the activity requires scant clothing and shade is not available light skinned persons may make use of sunblocks (not sunscreens) which typically block both UV-A and UV-B. Consider formulations made for babies which tend to contain the fewest potential allergens. Frequent use of these products is not advised. The data on sunscreen use and skin protection is mixed at best. In Australia the best protection was found from clothing.

Sunscreen use does not protect against melanoma and seems to increase overall sun exposure.[185] When clothing and sunscreen were compared, looking at actual skin cell damage, sunscreen was only 33% effective, clothing reduced cell damage by 66%. [186] While sunscreen may prevent burning it does not prevent cellular changes in skin and skin cell damage.

US LATITUDES

The map shows US latitudes from 50°, 40° and 30°. Latitudes above 30° have insufficient UV sunlight to produce optimal vitamin D 4-6 months of the year. Latitudes near or above 40° have insufficient sunlight 6-9 months of the year. Latitudes above 50° rarely have enough UV sunlight any month of the year, including summer months. Studies show exposure at latitudes 30°-45° will provide optimal amounts of D if exposure is mid-day, scant clothing, during summer months. [130]

Altitude compensates for degrees of latitude. Each 1,000 ft above sea level increases UV-B by about 10% and shortens the time needed to stimulate vitamin D production. Knowing your latitude and altitude can help you make wise decisions regarding vitamin D supplementation and sun exposure. Minimum vitamin D need from all sources to maintain optimal 20(OH)D is estimated to be 2,000 IU daily.
Some researchers have estimated lifeguards and farmers in latitudes below 30° make about 10,000 IU daily. I disagree with this finding. I believe that it is more likely the equivalent of 2,000-3,000 IU is produced. Lifeguards spend more than normal amounts of time in the sun. Optimal sun for your location (latitude and altitude) and skin type should provide enough D to maintain a blood level of 25(OH)D equal to or greater than 100 nmol/l or 40 ng/ml [114]
Skin pigmentation in the lower latitudes is darker, allowing for and actually requiring longer exposures to UV-B light to produce adequate vitamin D. [187] Persons with light skin should limit daily exposure to the beneficial UV-B rays to short periods, 10-20 minutes mid-day and use clothing as a barrier. When living in latitudes lower than that of their ancestors the time needed to produce D is shortened and may be as little as a few minutes. (Lower means latitude degree- 0-30° N or S)

Persons of color, i.e., type 3, 4 or 5 need longer exposure times in all latitudes. In higher latitudes the amount of UV-B in sunlight may not be sufficient even during summer months to provide optimal levels of D. You will need to be your own experiment.

Blacks in the U.S. in northern cities will find it impossible to get optimal D from sunlight at any season. [63,65,66,188] Studies show rickets in Texas, latitude near 30°, in children of African-American and Hispanic origin. [189] This is likely caused by failure to actually spend time in the sun.

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Toxicity and Other Concerns re Preliminary Report on D

For more than ten years now I have been working with physicians, their patients, and my clients to optimize D. The following is a short list of some reoccurring problems and considerations. Please call with questions or if you need help in understanding or applying the protocol. There will be a charge invoiced for telephone consults. 1-775-831-0292

- • ☐A client took 3,400 IU D daily, which initially corrected a significant D deficiency. This client
  - refused regular testing, (every 3 months the first year and every 6 months years two and
  - three) and by the end of the second year serum 25(OH)D reached 94 ng/ml. The client
  - experienced significant bone loss, loosing all bone gained during the first year of treatment
  - and more. TEST AND RETEST
- • ☐A health writer, on her own volition without testing, after reading Vieth, took 4,000 IU D
  - daily for almost one year and when tested had D levels of 25 ng/ml clearly malabsorption
  - and/or use of a type of D that didn't work. TEST
- • ☐Another health writer began taking 2,000 IU D for 10 months, also without testing. When
  - tested her D was 7 ng/ml TEST AND RETEST
- • ☐A man being treated with prescription UV-B light for psoriasis tested at 127 ng/ml- a
  - serious excess of D requiring avoidance of D in supplements AND avoidance of sun
  - exposure for many months to return serum values to within normal range..
- • ☐A client living in the northwest US with moderate sun exposure and moderate fish intake
  - tested at 60ng/ml- in optimal range with no need for supplements. Any supplements or
  - change in sun habits could potentially raise this above safe levels.
- • ☐Adams found that in some persons 25(OH)D above 67 ng/ml were associated with BONE
  - LOSS. Do not use supplements without testing. Rajasree, et all, found levels over 80 ng/ml
  - were associated with an increased incidence of heart disease.

What you are seeing in all examples is that the type of D, basic diet, genetics and other yet unknown factors profoundly influence levels of D and the effectiveness of D or sunlight supplementation. This optimization of D cannot be accomplished safely without testing.

Protein intake is important as D attaches to D-binding proteins. Low protein diets may contribute to D deficiency and insufficiency. Fats are important. Saturated fats such as coconut oil, butter and lard enhance D uptake and binding as do omega-3 fatty acids. Omega-6 fats, which include all seed oils such as soy, safflower, sunflower and even flax, may alter absorption and cell binding. Hydrogenated fats also potentially block D binding.

If your gallbladder function is impaired or you have long pursued a low fat diet make sure to take lecithin granules or lipase or bile salts or other fat digesting enzymes with your daily D. The easiest way to ensure D absorption while enhancing your protein intake is to consume one or more eggs at your D meal. One scant teaspoon of lecithin granules may alternatively be used, taken with your D.

Do not use emulsified or micellized vitamins. They are often so concentrated excessive doses may be taken by accident. Vitamin A and vitamin D are without question toxic in high doses. Use moderate doses and, if needed, lecithin to enhance absorption.

Lectins and grains (especially glutens) may, in certain persons, cause malabsorption of D or actual D depletion. For more on order the manuscript and/or call for a consultation. 1-775-831-0292 Information on lectins is available from the http://krispin.com web site.

Please test, test and retest. Do not under any circumstances undertake D supplementation without testing and unless you are in a year-round sunny southern location it is unlikely you get enough D from the sun alone.

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How Humans Store D And Why This Is Really Important

The fat-soluble vitamins A and D store in the human body. The upside of storage? Mechanisms and capacity for storage imply we have the ability to hold onto a reserve available for use when environmental or dietary sources are low. The downside? This capacity for storage can be dangerous. Hypervitaminosis is the term used to indicate the presence of excess A or D and the metabolic consequences, which in the case of too much D can be very serious indeed.

In 1972 a team from the University Department of Medicine, The Royal Infirmary, Manchester, England set out to determine if and where vitamin D is stored in the human body. Vitamin D metabolites, including the three we are focusing on, are very very very small. Vitamin D in blood and tissue is measured in nanogram or nanomole amounts. To give you an idea of just how small, a nanogram is one billionth of a gram and if you haven't got around to metrics yet it takes 28,349,520,000,000 nanograms to make one ounce. As our bodies contain 'nano' amounts of any of the D metabolites, tissue storage studies need comparatively large amounts of tissue to analyze.

The Royal Infirmary study design used radioactively labeled vitamin D_3. As the study explains 'Because of the small dose of radioactivity used in these studies, large samples were required…adequate material could be obtained only at autopsy or from an amputated limb.' The authors explain further that this is an 'opportunistic study' which I will define as meaning, 'under these conditions, dieing or having a limb amputated to be able to complete the study, you take whatever you can get'. 60 patients volunteered and received the radioactively labeled vitamin D injection. Of these 60 an unfortunate 6 supplied the samples for the study. Two other patients, who did not receive the D_3 injection, but had been treated with oral ergocalciferol (D_2) up until time of their deaths, also provided tissue samples.

With one exception all tissue donors were patients suffering from illness or injury serious enough to cause death or amputation. Four died from chronic renal (kidney) failure, one from cancer, one from post-operative complications, and one from biliary disease. In addition to the problem of serious illness possibly clouding the outcome, one of the subjects had been treated with large amounts of ergocalciferol (D_2) before receiving the injection of radioactive vitamin D_3. The remaining volunteers receiving the injection were low or deficient in vitamin D. A few of the limitations of the storage study include small sample size, time elapsed between the injection and analysis of samples, possible complications due to kidney failure as the kidney plays a major role in D metabolism and prior treatment with vitamin D. Even considering the limitations the study does offer us an approximation of D storage.

The researchers analyzed tissue samples of liver, spleen, kidney, heart, lung, thyroid, pancreas, adrenal, intestine, skin, bone, marrow; muscle, tendon, and fat to see where the radioactive vitamin D ended up and how much ended up in each part. They looked for just two of our Ds, cholecalciferol, and 25(OH)D.

And the results: Every tissue studied demonstrated vitamin D activity. This is interesting because at the time very little was known about the actions of D in tissues other than bone. Later researchers would locate receptors for vitamin D in all parts of our bodies as you have learned.(1)

After the injection the cholecalciferol cleared rapidly from the blood. Quick review: Whether from skin production, oral intake or an injection vitamin D travels first to the liver where the

cholecalciferol begins its metabolic journey by conversion to metabolite 25(OH)D and other less well known metabolites. In this study some of the injected cholecalciferol was excreted in bile, either unchanged or as one of several metabolites of D, some was converted to 25(OH)D circulating in the bloodstream, and some, both cholecalciferol and 25(OH)D, was removed from circulation by being partitioned off into fat cells or bound to tissue proteins. Our livers play an important role in handling all of the fats and fat-soluble substances we ingest or produce in our bodies.

Your body has metabolic pathways designed to break down and get rid of excesses thereby maintaining the normal balance, homeostasis, necessary for functioning. The liver breaks down fat-soluble toxins and other fat-soluble elements such as estrogen and excretes them in bile. Fat-soluble toxins, drug residues from antibiotics or chemicals such as DDT or PCBs that overload the system are unable to be metabolized and excreted in bile. Excesses can be kept out of the bloodstream by being stored in fat tissues as a protection from toxicity. In this study the path of vitamin D excess appears to be similar. When serum levels were elevated by the injection the body responded by storing excess in fat cells.

Perhaps this occurred because the dose given was much greater than a physiological dose, like a toxin needing to be trapped outside of circulation to prevent damage. Circulating in the bloodstream high levels of vitamin D can be dangerous causing damage to arteries and promoting calcification of soft tissues.(2;3) At a minimum, excessively elevated serum D may disrupt normal feedback mechanisms necessary to regulate the D endocrine system. Perhaps this occurred as a natural process to keep blood levels of vitamin D within normal ranges and store any extra for later use.

.Back to the study-The tissues containing the highest amounts of vitamin D, totals that include both cholecalciferol and 25(OH)D, were fat and bone marrow (high in fat content). Total D activity recovered from voluntary muscle nearly equaled that found in fatty tissue because while concentrations of D were lower, total body mass was greater; the body contains more muscle than fat. As discussed before vitamin D goes through many changes over time becoming any one of a number of metabolites. The time between the initial injection and the analysis of tissue ranged from 4 days to 90 days. At any time point the blood and skin samples contained primarily 25(OH)D with one exception. In the samples from patients on prior vitamin D therapy the blood and skin contained higher levels of unmetabolized D, cholecalciferol. The liver, kidney and lung tissues had a significantly higher percentage of 25(OH)D than cholecalciferol. As the interval of time increased, D in muscle shifted from cholecalciferol to the metabolite 25(OH)D. In bone marrow and fat there was a mix of cholecalciferol and 25(OH)D with the unmetabolized cholecalciferol predominating in most samples.

GP overview: An injection of D moderately raises levels of serum 25(OH)D over a number of days. Excess (my word, not the study's) is stored in tissues, in fatty tissues as the unmetabolized D, cholecalciferol, and in muscle initially as unmetabolized D but shifting to 25(OH)D over time. The skin contains vitamin D primarily as the metabolite 25(OH)D.

Vitamin D's fate is to be finally broken down and excreted in bile, as is the fate of all fat-soluble substances in our bodies. D is also removed as our bodies shed the cells of our mucous linings and skin, both of which contain all 3 metabolites of vitamin D. Traveling from initial intake, by mouth or skin, to active metabolites to degradation and elimination takes a significant period of time.

When vitamin D supplies are diminished or absent vitamin D stored in tissues and organs is rapidly released while fat stores of D release very slowly.(2) In patients who had previously been on extended D therapy large amounts of vitamin D activity were detectible in tissues even after 15-20 months.

What this study doesn't tell us: It does not offer any insight into optimal levels of vitamin D, in blood or in tissues. It doesn't tell us if storage in fat offers a useful reserve of vitamin D when serum levels decline. Holmes and Kummerow found vitamin D in fat (also from excess doses of D) loath to leave, dispersing extremely slowly.(4)

This storage study doesn't tell us what normal humans, getting their D from sunlight and diet, actually do with vitamin D. Do they have D stored in their fat? If so in what form and how much is where?

Storage studies, all using excessive D, have firmly entrenched the idea we store D in fat, as a reserve. From this premise experts suggest getting D from sunlight in summer produces excess vitamin D, which is stored and may be accessed by the body in winter to maintain vitamin D. This assumption is either not true or we are gravely 'under-sunned'.

Studies in the United States and other countries, many of them in tropical or sub-tropical countries, show winter time levels of D to be deficient or insufficient and frequently associated with bone loss.(5-9).(10-18)

These studies all confirm that while D might be sufficient in summer women, men, children, seniors, healthy, or infirm, all have lower values of D during winter months, frequently dropping to seriously deficient values. So much for storage.

The data suggests that, in the real world with real people, storage is not a large factor in year round vitamin D sufficiency, especially if summer D is insufficient. In persons living at latitudes greater than 35° only those who spent significant time (months) in the tropics during the winter maintained optimal D. All others using sunlight their major source of D saw a significant drop in serum 25(OH)D by the end of October with a slow but continuous further decline until spring.

In a study published in 2002 Barger-Lux and Heaney checked the 25(OH)D levels of men spending the summer out of doors participating in activities including landscaping, construction, farming and recreation. The average value of D reached at summer's end was 48.8 ng/ml. Approximately 5 ½ months later the average of the 26 participants dropped to 29.6 ng/ml. Three of the men had values less than 20 ng/ml and 15 had less than 30 ng/ml.(19) GP, the reality of storage looks quite different from the premise doesn't it?

Studies evaluating the system overload of an injection, with or without prior D therapy, or single or chronic high dose D as used in animal storage studies, may offer clues to D metabolism. The doses of D used in these studies may also completely imbalance the system so that the primary question answered is 'What happens to vitamin D in the human or animal body when an overload is given?' (4)

At no time, ever, from any source, could the human or animal body have gotten the massive doses of vitamin D being used today in research, in medicine and as additives in feed in animal husbandry.

This storage study helps to show some of the difficulties encountered in understanding the relationship between our bodies and vitamin D. As there is general acceptance within and outside of the medical community that vitamin D stores in the body as a natural process clinicians have developed the protocol of giving high dose vitamin D. The idea is very user friendly. An injection once every three months or a pill once a week or other prescription allows the physician to treat the condition and not have to be concerned about compliance.

Many of the studies using D for osteoporosis or other D related disorders used tens of thousands and even hundreds of thousands of International Units of vitamin D. The current prescription vitamin D supplement Calciferol, contains 50,000 IU of vitamin D_2. Protocols for various

conditions continue to suggest 50,000 IU to as much as 700,000 IU daily or intermittently (weekly, monthly or once every 3 or 6 months) to treat hyperparathyroidism, vitamin D resistant rickets, osteoporosis, osteomalacia, vitamin D myopathies or just to build or maintain levels of 25(OH)D.(20-23)

When these doses are given they must be given by prescription and monitored by the physician. At the doses used there is always a possibility of toxicity(24) and if the patient inadvertently combines the treatment with sun exposure(25) or with supplements that contain vitamin D the possibility for toxicity increases exponentially. What is unclear to me is why these doses were ever used. Physiological doses of D and sunlight are effective.

Continuing excerpts-

Too Much Vitamin D?

A review from the section on testing: Normal values of 25(OH)D range from 20 ng/ml – 57 ng/ml Optimal levels are probably 35-60 ng/ml or the range may turn out to be as broad as 30-70 ng/ml. In Vieth's review of vitamin D he was unable to find studies showing toxicity at levels of 25(OH)D below 56 ng/ml.(26) As mentioned earlier chronically sun-exposed individuals in the tropics or subtropics reach higher values. Some researchers have made comments that these naturally derived elevations of D, such as found in one farmer in Puerto Rico, 90 ng/ml, are from sun and therefore normal and without danger. Past and recent research suggests the safety of elevated levels of D from sun is less clear.

When I first began exploring vitamin D in 2000 I spoke with Barbara Boucher, M.D. from the Department of Diabetes and Metabolic Medicine, Medical and Dental School, Queen Mary, University of London, Royal London Hospital, Whitechapel, London, U.K. Dr. Boucher is one of the world's top researchers regarding vitamin D and Syndrome X, that is the complex comprised of insulin resistance, obesity, hypertension and adult onset non-insulin dependent diabetes. At the time of our conversation I had mentioned Reinhold Vieth's idea of higher doses of D being safe and perhaps even necessary. Dr. Boucher was unaware of Vieth's work but strongly disagreed with any suggestion that D in high doses for extended periods of time would be safe and suggested that I review this problem carefully. Her tone was so very intense and serious the caution stuck with me over the three years of book preparation.

Studies, however scientific, don't prove as much as the researchers, health promotion agencies, media and advertising would like us to think. Journals are filled with researchers comments arguing among themselves on why results often differ. Methods, testing procedures, number of participants, kind of participants and contrary results ruffle feathers frequently. Studies don't give us truth they give us approximations. While there may not be sufficient evidence to say levels of 25(OH)D above 70 ng/ml are absolutely harmful there is equally no sufficient evidence to support values above 70 ng/ml as natural, necessary, optimal, or safe.

Hypervitaminosis D indicates excess intake of vitamin D leading to elevated levels of 25(OH)D though D_2 or D_3, the vitamin you take or make, can also be elevated in hypervitaminosis Vitamin D toxicity, intoxication, or poisoning are terms reserved for very high levels of D accompanied by elevated serum calcium and potential or actual calcification of soft tissues. As usual the research community continues to discuss these terms among themselves and define what they might 'really mean'.

For our purposes I use the term hypervitaminosis D to expressly mean chronically elevated, >70 ng/ml, levels of 25(OH)D, caused by supplementation and/or UV-B light exposure whether from sunlight or UV-B lamps.

In August 1997 a letter was published in the Annals of Internal Medicine. The title,- *Gains in Bone Mineral Density with Resolution of Vitamin D Intoxication*, should give us pause. The authors, Dr. John S. Adams and Gene Lee found 4 patients they determined to have hypervitaminosis D. These patients were discovered during a 1992-93 admittance screening at the Cedar-Sinai Bone Center in Los Angeles, CA. The purpose of the screening was to determine if a policy of giving a standard testing profile including fasting blood levels of parathyroid hormone, TSH (thyroid stimulating hormone), calcium and 25(OH)D and fasting urine levels of calcium and creatinine would help evaluate patients for osteoporosis or low bone mineral density. (27)

The four women had high levels of calcium in fasting urine, three times greater than normal values, demonstrated bone loss, and a 25(OH)D >50 ng/ml. There were no other abnormalities in the blood with the exception of a lower level of parathyroid hormone. TSH, $1,25(OH)_2D$ and serum calcium were all within normal ranges. All four patients had experienced demonstrable bone loss.

Two of the four women had unknowingly been taking supplements containing high levels of vitamin D. All of the women had been taking a minimum of 1,000 mg of calcium prior to and at the time of their diagnosis. This is important to note because the extra calcium did not protect them from the loss of bone attributed to excess 25(OH)D. In a manner as yet undetermined the elevated level of 25-hydroxyvitamin D caused or contributed to loss of calcium in the urine and increased bone loss.

While there are some arguments among experts about whether these women actually had hypervitaminosis D and whether it was the cause of their bone loss all four patients regained bone mass when elevated 25(OH)D and urinary calcium decreased to more normal values. The drop in D and decrease in urinary calcium took several months. Bone mass increased by an average of 2% a year following the resolution of hypervitaminosis D, very good news.

In the two women with the highest levels of 25(OH)D, 89 ng/ml and 80 ng/ml, the vitamin D causing the elevation was being taken without their knowledge. The high amounts of vitamin D were found to be an unlisted ingredient in preparations purchased from health food stores. None of the patients KNOWINGLY took more than 1,200 IU of vitamin D daily. One of the supplements tested contained 3,600 IU of D, not listed on the label.

According to the vitamin D endocrine system model $1,25(OH)_2D$ (calcitriol) is the active D, the hormone. All of the women with hypervitaminosis D had normal levels of $1,25(OH)_2D$. Only 25(OH)D was elevated. Adams doesn't know why bone loss occurred but he suggests that the high levels of 25(OH)D may have displaced $1,25(OH)_2D$ in cell function, potentially stopping or slowing the formation of new bone or otherwise altering production of elements necessary for bone maintenance.

Adams, Holmes, Kummerow, and the next study from India have documented damage to bones or arteries when 25(OH)D is elevated. In some of the studies done by them or reviewed by them damage occurred when the only abnormalities were elevated 25(OH)D and excess calcium in the urine.(3;4;27-29) Some very experienced vitamin D researchers believe there isn't a known upper limit on 25(OH)D if serum calcium remains normal. They accept hypercalcemia (too much calcium in the blood) as the indicator of excess D. This may be a mistake.

Researchers from the Department of Cardiology and Achutha Menon Centre for Health Science Studies, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India considered that high levels of D are used to cause heart disease in experimental animals.(30;31) They wondered if high levels of 25(OH)D generated from exposure to tropical sun would also contribute to heart disease. In comparing $25(OH)D_3$ levels between a control group of 70 men without heart disease and 143 men with known heart disease, either recent heart attack or coronary artery blockage, levels equal to or greater than 89 ng/ml were present in 22.1% of the

controls and 59.4% of the heart disease patients. The numbers indicate a strong association yet to be understood.(32) As low and high levels of D may contribute to heart disease I asked one of the studies authors the percentage of those with heart disease in the range I believe is optimal, 40-60 ng/ml. Fifteen percent had 25(OH)D within this range, 85% had levels higher or lower.

The researchers in India knew these elevated levels were from sunlight, not supplements, because the test was specifically for 25(OH)D_3. D_3 is only produced on the skin or taken in vitamin D_3 supplements such as cod liver oil. Vitamin D_3 supplements are not generally available in many parts of the world including India. The test used commonly in the U.S. tests total 25(OH)D, which includes 25(OH)D_2 and 25(OH)D_3. Both D_2 and D_3 supplements are available here and both will raise 'total' 25(OH)D. This is important because the Indian research suggests the possibility of too much D from sun, generally thought not to be possible. (That old assumption trap.)

In December of 2001 a client being medically treated for psoriasis with a special narrowband UV-B light prescribed by his dermatologist tested 25(OH)D at 97 ng/ml. The light treatment was immediately stopped by the choice of the client, not the dermatologist. The client then left for his yearly trip to Hawaii staying three months from Jan-March of 2002. He did not intentionally avoid the sun, believing, as I did at the time, hypervitaminosis D from natural sunlight was impossible. He ate local Hawaiian foods, including eggs and fish. On his return to northern California his 25(OH)D had risen to 127 ng/ml.

This man used no supplements containing D. His calcium supplementation was between 1,000-1,200 mg daily in addition to food sources. His D dropped slowly, just 6 ng/ml each month with complete avoidance of sunlight and any foods that might contain a significant amount of D. After 8 months of sun avoidance and avoidance of fish and eggs his D dropped to 78 ng/ml.

His dermatologist refused to request the initial test for vitamin D. After being presented with the results the dermatologist stated he had no information on elevations of vitamin D from UV-B light treatment nor did he know if this was good, bad, or insignificant. After several attempts to get medical advice the client was referred to an endocrinologist who also didn't know anything about elevated D and UV-B light treatment. He was willing to order testing, one time. The clients last test reported a 25(OH)D of 57 ng/ml. after 12 months of complete sun avoidance and no fish, eggs, or supplements containing vitamin D.

His elevated 25(OH)D was caused by prescription psoriasis treatment with narrowband UV-B light made worse by exposure to tropical sun. This is iatrogenic (induced by a physician's treatment) hypervitaminosis D. When he first reported the elevation of vitamin D to his dermatologist a partner in the practice admitted they had seen excessively elevated levels of 25(OH)D before in persons treated with narrowband UV-B before. They did not know what it meant, or whether or not it might be a problem, nor was it their policy to monitor D. They demonstrated no interest in testing serum calcium, PTH, or fasting urinary calcium and refused a request to do so. Narrowband UV-B light can raise 25(OH)D to hypervitaminosis D levels.

A woman with severe osteoporosis, demonstrating a lumbar SD –4.6 and low 25(OH)D began taking D_3, cholecalciferol, 3,000 IU a day. Her serum 25(OH)D rose from below 20 ng/ml to 42 ng/ml within 4 months and a follow up bone scan after 8 months of D supplementation showed some not significant (very slight) bone gain had occurred. The client did not want the expense of testing, it was not supported by her physician or HMO, so did not test D again. She continued to take the original 3,000 IU of D about five days a week. This dose is slightly lower than the dose of 4,000 IU sometimes suggested or used by some D researchers and clinicians. She also took between 1,000-1,500 mg calcium in addition to food sources and did not avoid foods containing D. The next bone scan, about 1 year after the scan showing a slight gain and 2 years after starting vitamin D, showed normal PTH (43 ng/ml); 25(OH)D 95 ng/ml; and lumbar SD –4.8 in

addition to more loss in the femur for SD –2.2 to –2.5. Overall she experienced a 6% loss of bone density.

Her blood and urine tests confirmed her bone loss to be a case of hypervitaminosis D. She had the requisite elevated calcium in the urine with all other tests normal, serum calcitriol, PTH and serum calcium, just as Dr. Adams had found in his screenings.
When the clients second scan showed bone loss her doctor, an osteoporosis specialist, did not want to test her D. Her physician believed the problem was hormonal and insisted on tests for TSH and PTH, both normal, and other similar studies. The only reason the patient got her D tested is because she stood her ground and demanded it, forcing her doctor to order the test.

Had the vitamin D test not been done her physician would have determined 'bone loss with unknown cause'. The physician had immediately prescribed Fosamax and estrogen for treatment. These treatments would have been ineffective in treating the problem, excess supplementation of vitamin D, though they do help correct bone loss from hypervitaminosis D. What she needed to do was to stop taking the D.

The test showing 95 ng/ml was done in July. All supplements were stopped and foods with vitamin D, fish, eggs and fortified dairy were avoided. Summer sunlight was neither avoided nor sought out. In November 25(OH)D reached 110 ng/ml. Vitamin D levels dropped slowly over the next six months, about 8 ng/ml per month. At last testing her D was 61 ng/ml.

For about six months prior to the July test showing 95 ng/ml she had been experiencing 'bone ache', fatigue and depression. Within several weeks of stopping vitamin D the pain resolved and energy and mood returned to normal, interesting because her 25(OH)D continued to climb during this time. Her serum calcium remained normal at all times.

Message: Don't take vitamin D in amounts beyond the 400-800 IU range without testing and when you begin to take D (or sun) test every three or four months the first year and every six months the second and third year to make sure you have the right dose.

Retest if you move to a different latitude or increase or decrease your exposure to UV-B (summer or tropical sun). Find a healthcare provider that will support you in this process. Studies using high doses of D, in excess of 2,000 IU, to my knowledge have never lasted longer than 6 months. In many cases elevated D doesn't appear until the second or third year of supplementation.

Lessons To Be Relearned

There are several important points to consider in these real stories.

First and foremost test, test, and retest D. There is no way to know your levels without testing and there are no obvious symptoms of too much or too little D. While some persons with elevated levels did complain of a general feeling a weakness and fatigue this is hardly a specific symptom and most persons with elevated D had no symptoms. While initial supplementation with vitamin D may normalize values, continued supplementation over time at the same dose may cause hypervitaminosis D. Doses above 800-1,000 IU a day may contribute to problems in persons particularly sensitive to vitamin D. The risk increases further at doses greater than the UL of 2,000 IU.

Second, it is possible to produce hypervitaminosis D from UV-B containing light, either that in tropical climes as in the case of the heart disease patients in southern India or through treatments with narrowband UV-B light for medical conditions such as psoriasis. I cannot say at the current time if suntan beds could also contribute to excess. It's a question for researchers. When UV-B makes up 5% or less of total UV (UV-B and UV-A combined) as in most tanning bed lights in the U.S. and natural sunlight at latitudes higher than 35° north or

south, the UV-A or other bands present in the light will break down precholecalciferol in the skin before conversion to 25(OH)D. Sunning midday in summer in latitudes between 35-40° has rarely produced levels of calcidiol beyond the mid 50-60 ng/ml in light skinned persons. This does not prove more intense exposure levels of UV-B rays from sunlight in the tropics can't raise D to unhealthy levels, especially in persons with little melanin. Narrowband UV-B and tropical sun clearly can and in certain situations does raise 25(OH)D beyond known safe values. While we wait for further research to clarify this issue response is ALWAYS individual, whether to supplements or light. Test, test, and retest.

Third, combining vitamin D supplementation, at a dose that gives great values for D in winter, 40-55 ng/ml, with summer sun can rapidly elevate 25(OH)D in some persons in some locations. Having plenty of vitamin D doesn't stop your skin from producing more.(25;33) In the summer of 2002 several persons reported going from 50 ng/ml or thereabouts to levels greater than 75 ng/ml within weeks of summer sun exposure. They had continued to take an appropriate 'winter' dose of D into the sunlight of summer.

You have to make a choice. Because you have enough D, from supplements or food, you do not stop producing D in your skin. Levels just increase. You may take D and avoid the sun or figure out your winter D dose and stop in the summer or combine some D and some sun. To do any of these safely you must test, test, and retest.

Am I sounding like someone's mother? I worry. I remember Dr. Barbara Boucher's voice from the U.K. She had seen the problems with too much D given in infant formulas and fortified foods. There the problem stemmed from well-intentioned but unwise supplementation. The U.K. far north, across the ocean from Canada, with often overcast skies. Getting excess D from sunlight at any season would be extremely difficult. Here in the U.S. the situation is much more complex. We have a higher UV-B range at any season and in some areas subtropical sun. I firmly believe that we all need vitamin D and having too little contributes to both chronic and acute conditions that could be corrected if we got enough.

I also firmly believe getting too much vitamin D from supplements or light or a combination can create a serious and potentially dangerous situation. There are rarely obvious symptoms of D marginal deficiency or overabundance. Conditions associated with too much or too little vitamin D involve inappropriate mineralizaton or demineralization of bone, or joints or calcification of tissues and organs that include arteries, bone, kidneys, brain, or muscles.

I have seen an advertisement on TV for a calcium supplement that seems to suggest very high levels of vitamin D and calcium are safe and will cure any number of things. I have also visited websites produced by well-meaning persons suggesting massive doses of D, cod liver oil or a 'natural' source, is safe in any amount and providing 'references' to prove this. The references are without merit and the statements are completely and dangerously false.

You can take too much of anything but the consequences of too much D are extreme and many of these consequences are not reversible. In seeking light we must use moderation. We need a little sunlight and D, more perhaps than we get right now but not ever levels higher than reasonably available from food or natural sunlight.

The moral of all of these stories is simple. America is a BIG melting pot with latitudes tropical, subtropical, temperate, and arctic and skins from very light to very dark. We are all different. We get different light. We respond to light and supplements differently. We live in different places. We have different skin colors and varied ability to produce and store D. We can all avoid damage by testing and responding sensibly to the results.

Below I quote Dr. Adams in his reply to criticism regarding his observations as published in the Annals of Internal Medicine Letter, March 1998:

"The most crucial point raised is the importance of detecting the opposite condition, vitamin D deficiency. We agree with the recommendation to increase the current recommended daily allowance for oral vitamin D consumption by 50% to 100% (from 400 to 600 or 800 IU daily), particularly in elderly persons who have limited sunlight exposure and cutaneous vitamin D synthetic capacity. This intake level is safe and will not cause hypercalciuria. What is not always safe and reliable is the label of a food supplement not regulated by the U.S. Food and Drug Administration. In our patients, the vitamin D content of the supplement was at least one order of magnitude greater than that advertised on the label. **It is fortunate that the serum 25(OH)D level is the best screen for both hypervitaminosis D and hypovitaminosis D. Physicians should take better advantage of this versatile screening tool. "** (Underlining and bold added by me.)

A complicating addition to Dr. Adams' comments: Remember the two women with optimal D, the lucky ones who got to spend winters in the tropics? Both women were postmenopausal and candidates for a blanket prescription. of calcium and D. ANY supplementation would have elevated D to unnecessary levels. Even Dr. Adams' moderate dose of 800 IU is inappropriate if 25(OH) D is adequate. Testing really is critical.

The Causes- A Review

In our examples the elevated vitamin D came from supplements or sunlight or a combination of both. Remember the complex systems model and my warning about the devil being in skipping the details? Not getting enough vitamin D is clearly an issue in many parts of the world and so is getting too much. With all the voices, research, opinion, sales, media, the only safe path is working out your own need and your response and knowing that it works, safely, because you have tested.

The following list concerns low level hypervitaminosis D as that seen with Adam's cases of bone loss or the calcification of arteries in the southern Indians NOT clinical intoxication with elevated serum calcium.

- Vitamin D excess may be caused by too much sunlight.(32)
- Vitamin D excess may be caused by supplementation of D from unlabeled vitamin products. (34)
- Vitamin D excess may be caused by knowingly taking high doses of vitamin D over a short or long period of time.(23;35;36)
- Vitamin D excess may occur when supplements and sunlight are mixed. The body does not have an effective feedback mechanism to stop production or absorption of vitamin D either in the skin or from oral intake. A level of supplementation that might work well in winter may become too much when combined with summer sun.(25)

The Complications Of Too Much D

Our few examples showed bone loss and calcification of soft tissue, in this case the arteries. In research extremely high levels of vitamin D are given to lab animals, rats in most cases, to cause a condition similar to heart disease with calcifications in arteries. While the doses of vitamin D used to cause artery damage in rats are extreme after considering the Indian study, with a strong association between heart disease and elevated 25(OH)D, there should be a concerned response and more research in this area. It certainly appears that both low and elevated levels of vitamin D may contribute to heart disease.

Holmes and Kummerow found elevations of 25(OH)D, independent of serum calcium, caused damage to tissues. Excess 25(OH)D was found primarily in the kidney, liver, lung, aorta, and heart. places known to develop calcifications with D intoxication. As in our cadaver storage study Holmes found unconverted D primarily in fat and 25(OH)D in blood. Remember DBP prefers

25(OH)D. Research suggests DBP entry into cells may be a normal process. Usually only 1% of DBP contains vitamin D, any of its metabolites. Holmes suggests elevations of vitamin D may increase DBP binding of D to as much as 40%.

The question of what constitutes hypervitaminosis D revolves around whether deposits of calcium in soft tissues are a result of excess calcium in the blood or excess 25(OH)D. Holmes and others have found calcium deposits related most to elevations of 25(OH)D. The pathologic changes he found in tissues, calcium infiltration of cells, occurred at levels lower than those causing elevated serum calcium. His theory as to why this happens suggests D-binding protein excess 25(OH)D may alter cell membrane permeability allowing entry of calcium.(4;37) Increased calcium influx in a cell leads to calcium deposits, cell damage or death.(38;39)

Most researchers have noticed excess calcium in the urine as 25(OH)D levels increase and this has been attributed to improved absorption of calcium. Dr. Adams did not find that to be true and even if you aren't concerned about arteries or bone loss consider this- Excess calcium in the urine combined with elevated 25(OH)D has been associated with kidney stones.(40)

Vitamin D moves calcium around our bodies; into and out of bone, muscle, hair, arteries, and cells. Both low and high levels of vitamin D appear to contribute to misbehaving calcium. Goldilocks Rule: Not too much, not too little, just right.

In vitamin D poisoning calcification of the kidney and kidney failure are listed as cause of death. These unfortunate conditions have occurred in rare instances when children or adults have unknowingly been exposed to hundreds of thousands of units in a very short period of time or a somewhat lower dose of vitamin D over weeks or months. It is unlikely that any of you will find yourself suffering from poisoning in any of these ways. My concern is that in trying to be as healthy as you can be you may, in your enthusiasm, raise levels of 25(OH)D above the upper limit of safety, which to my mind is lower than some experts seem to believe.

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Please test, test and retest. Do not under any circumstances undertake D supplementation without testing and unless you are in a year-round sunny southern location it is unlikely you get enough D from the sun alone.

Normal Values most labs since 2008. These values may not reflect all lab reported normals. Levels below 20 ng/ml (50 nmol/l) are strongly associated with abnormal calcium status and elevated PTH. For optimal health, stay in the Optimal range.

Optimal 25-hydroxyvitamin D values: 100-150 nmol/l 40-60 ng/ml Values as high as 75 ng/ml, 187.5 nmol/l are found in tropical sun exposed persons. Levels above 70 ng/ml, 175 nmol/l may be safe or problematic for certain persons yet to be determined.	Normal 25-hydroxyvitamin D lab values typically are: Old 50-140 nmol/l New 80-250 nmol/l Old 20-56 ng/ml New 32-100 ng/ml Higher acceptable values have been common since 2008

☐ **Values below 30 ng/ml (75 nmol/l) may indicate bone loss.**

☐ **Values below 40 ng/ml (100 nmol/l) may indicate abnormal cell replication with implications for the development of cancers and other cellular abnormalities.**

☐ **Levels above 70 ng/ml (150 nmol/l) may indicate chronic excess in some individuals.**

☐ **Levels above 125 ng/ml (310 nmol/l) indicate chronic/acute toxicity.**

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Getting D from the Sun, Safely

If sun burning always occurs at any exposure level or there is prior incidence of skin cancer you may not want to use sunlight to maintain D. Test and use a supplement.

To use sunlight in lieu of supplementation refer to the skin pigmentation guide. The guide provides an estimate of the time needed to optimal levels of D. These exposure times are sufficient but remember they are calculated on UV-B intensity. **Longer exposure will increase vitamin D production beyond optimal levels in some locations and will increase the danger of skin damage in all locations**.
This guide applies in seasons, latitudes, and altitudes of UV-B production. Please see the latitude and altitude information for UV-B levels or consider a UV-B meter. Remember, UV-B does not penetrate glass, smog, fog, some haze or clouds.

UV-B Exposure Guidelines

For the optimal daily dose of D from sunlight 80% of skin must be exposed for the full time, that is the time just **prior** to any skin changes (pinkness). In most locations in the US significant UV-B is present only during mid-day and only during summer months.
Exposure must take place when UV-B is present, between 10AM and 2PM during the summer for most of the United States, on cloud and smog free days. Near noon is best. Before 10AM or after 2PM burning may occur before optimal D is produced. The closer to noon the less time needed to achieve D production. Do not over sun.

The time needed to produce adequate D depends on skin color.

Table 1 UV Guidelines

Skin Type	Time Needed for Daily D Production
Type 1- Always burns easily, never tans, extremely sun-sensitive skin. **Examples:** Red-haired, freckles, Celtic, Irish-Scots.	5-7 minutes per side, 10 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 2- Always burns easily, tans minimally, very sun-sensitive skin. **Examples:** Fair-skinned, fair haired, blue-eyed, Caucasians	10-15 minutes per side, 20 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 3- Sometimes burns, tans gradually to light brown, sun-sensitive skin. **Examples:** Darker Caucasians	20-30 minutes per side, 25 minutes full body at UV Index 9-10, above 10 less time is needed.

Type 4- Burns minimally, always tans to moderate brown, minimally sun-sensitive. **Examples:** Mediterranean type, Caucasians.	40 minutes per side, 70-80 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 5- Rarely burns, tans well, sun-insensitive skin. **Examples:** Middle Eastern, some Hispanics, some Blacks	50 minutes per side, 90-100 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 6- Never burns, deeply pigmented, sun-insensitive skin. **Example:** Blacks	60 minutes per side, 120 minutes full UV-Index 9 or above body at UV Index 9-10, above 10 less time needed.

For type 1-3 skins sunbathing near noon will assure enough UV-B to produce D before burning occurs. For skin types 4-6 UV intensity is critical. Many parts of the US may not provide adequate UV-B to optimize D.

A note about the UV Index-
The weather service puts out daily bulletins estimating the UV intensity. Go to http://iwin.nws.noaa.gov/iwin/us/ultraviolet.html. The problem with these ratings is that they do not actually look at the local UV-B but estimate from 'sources'. I prefer to use my Sunsor□UV-B meter. It is battery free and about the size of a small calculator. I carry it with me and can know instantly how much time I need to produce my daily D or if UV-B is too low to make sunning worth my while. UV-B meters are available but expensive.

Table 2 UV Index and Sun Exposure
National Weather Service's UV Index. Note UV-B is present during primarily mid-day in locations with values of <10. Time is for mid-day exposure. Index readings are for the mid-day value when UV is highest. Most of the day values will be lower than that indicated by the UV Index.
0 - 2 (Minimal) - An Index reading of 0 - 2 indicates minimal danger from the sun's UV radiation for the average (Type 2) person. Most people can stay in the noon sun for up to an hour without burning. Sufficient vitamin D will not be produced at this level before skin damage would occur. **(No D all skin types)**
3 - 4 (Low) - An index reading of 3 -4 indicates low risk of harm to the skin from the sun's radiation. Type 2 individuals can experience a burn in 30 -60 minutes. It would be necessary to stay in the sun for longer than 60 minutes to activate D and burning may occur before D was produced. **(No D all skin types)**
5 -6 (Moderate) - An index reading of 5 - 6 indicates some significant risk of skin damage due to the sun. Unprotected exposure can result in a burn in only 20 - 30 minutes. At this level burning or skin damage would also occur before adequate D production. **(D in light skin types only, but not much)**
7 -9 (High) - An index reading of 7 - 9 indicates high risk of harm from unprotected exposure to the sun. Time in the sun should be limited during midday (10:00 A.M. - 4:00 P.M.) as skin may burn in as little as 13 - 20 minutes. Enough D would be generated in 10-20 minutes (before burning) in type 1 and 2 skins. **(D in light and medium skins midday, very little D in dark skins)**

10+ (Very High) - An index reading of 10 - 15 indicates very high risk of harm from unprotected sun exposure. Between 10:00 A.M. and 4:00 P.M. the length of time to burn may be less than 13 minutes without protection. Enough D would be generated in 10 minutes (before burning) in type 1 and 2 skins. **(D all skin types, very little time needed for light skins, use caution.)**

While some D might be produced in light skins at the lower UV Index readings the damage from excess exposure to UV-A would out weigh the worth of sunning.

At higher latitudes, most of the US, the UV Index is for midday, not morning or afternoon. Only Hawaii, Florida, south Texas, Southern Arizona, and some higher elevations, have significant UV-B for a longer stretch of the day.

If sun exposure is or must be limited or avoided, use supplements. Testing is critical. We are all different and our responses are unique. Tropical sun must not be judged by these values. As little as a few minutes may be needed for persons with very light skin in subtropical or tropical locations.

Sunlight and supplementation should not be mixed. Cases of elevated D have occurred by persons supplementing and sunning. BUT if the sun you sun in is far to the north rarely will it have much UV-B and sunning and supplements may work together. This mix of sunlight and D should not be attempted without testing. Several persons over the last few summers found themselves having excess levels of D by combining D and sunlight. Others have found sunlight exposure altered their need for supplementing D very little.

TEST. This is particularly important in the US where during much of the year, in many locations, UV-B is not present in amounts able to produce D before burning. Depending on latitude this variable ranges from 2-3 months in San Diego and other cities of similar latitudes to 8 months in Portland, OR, upstate New York and other locations at higher latitudes.

The exact time needed, varying with skin types at various US latitudes and altitudes, is not yet adequately understood. A UV meter used with sunbathing and initial frequent testing of 25(OH)D may be useful to determine your optimal use of sun and supplements.

Genetics, both skin coloring and genes related to vitamin D response, strongly play a role in your need for D. Vitamin D receptor polymorphisms, genetic alterations of how we absorb and process D, are only recently being studied and the information applied to D sufficiency. (180)

Vitamin D Testing

There are two common vitamin D tests currently available.

1. 25(OH)D also called 25-hydroxyvitamin D; or 25-hydroxy

2. 1,25(OH)2D which is 1,25 dihydroxyvitamin D; or 1,25-dihydroxy

25(OH)D is your storage D, the second, 1,25-dihydroxy, your hormone D.

The 25(OH)D test is the right test to order for monitoring vitamin D need and repletion. If your physician orders it for you your insurance may pay. Check, because if your insurance does not pay the test may be quite expensive. It depends on the lab.

As an alternative you may order the 25(OH)D test through the Life Extension Foundation (LEF). For members the cost is approximately $47, for non-members, $63. The test results are sent directly to you by LEF. Also available from http://privatemdlabs.com for about $50, $59 minus a 15% coupon code on the first page.

To order visit http://lef.org , Blood Testing, Vitamin D test; or call 800-544-4440 or to order from Private MD Labs visit http://privatemdlabs.com or order from http://www.homehealthtesting.com/vitamin-d-test.html to test at home.

I do not recommend membership in LEF, nor do I use any of the LEF supplements. I am grateful they provide a way to test D that is easy and relatively inexpensive. You may choose to join or purchase products from them but do not use either of their vitamin D products.. I absolutely do NOT recommend their vitamin D. Both products are dry D and I have found extremely poor utilization of dry D from any source. In addition the 5,000 IU product, if absorbed, may result in excess over time.

If you have an autoimmune disease or other symptoms or conditions warranting further testing you may need the following blood tests, also ordered by your physician-

25(OH)D (standard) plus

1,25(OH)2D
Plus the following tests that are usually found in a 'panel'
PTH (parathyroid hormone)
Ionized calcium
Total calcium

Full testing including the panel is appropriate for women with bone loss, persons with sarcoidosis, and persons who seem to need D but respond poorly to D supplements and/or sunlight.

UV-B Exposure Guidelines

For the optimal daily dose of D from sunlight 85% of skin must be exposed for the full time, time depending on skin color. (About 400 iu produced per 5% surface skin exposure per times depending on skin color.) 'Full body' means front and back.
In most locations in the US, those higher than 30□ significant UV-B is present only during mid-day and primarily during summer months.
Exposure must take place when UV-B is present, between 10AM and 2PM, on cloud and smog free days. Near noon is best. Before 10AM or after 2PM burning may occur before optimal D is produced.
The closer to noon the less time needed to achieve D production.
Sunbathing near noon will assure enough UV-B to produce D before burning occurs.

Skin Type	Time Needed (Summer) for Daily D Production
Type 1- Always burns easily, never tans, extremely sun-sensitive skin. **Examples:** Red-haired, freckles, Celtic, Irish-Scots.	5-7 minutes per side, 10 minutes full body at UV Index 9-10, above 10 less time is needed.
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Type 3- Sometimes burns, tans gradually to light brown, sun-sensitive skin. **Examples:** Darker Caucasians, lighter skins of other races	20-30 minutes per side, 25 minutes full body at UV Index 9-10, above 10 less time is needed.
Type 4- Burns minimally, always tans to moderate brown, minimally sun-sensitive. **Examples:** Mediterranean type, Caucasians.	40 minutes per side, 70-80 minutes full body at UV Index 9-10, above 10 less time is needed.
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Type 6- Never burns, deeply pigmented, sun-insensitive skin. **Example:** Blacks	60 minutes per side, 120 minutes full body at UV Index 9-10, above 10 less time needed.

Source estimating the UV intensity- http://iwin.nws.noaa.gov/iwin/us/ultraviolet.html. The problem with these ratings is that they do not actually look at the local UV-B but estimate from 'sources'. I prefer to use my Sunsor□ UV-B meter. It is battery free and about the size of a small calculator. I carry it with me and can know instantly how much time I need to produce my daily D or I can supplement if UV-B is too low to make sunning worth my while.

UV Index Value and Sun Exposure Levels- National Weather Service's UV Index. Note UV-B is present during primarily mid-day in locations with values of <10. **Times are for mid-day exposure.**

- □ **0 - 2 (Minimal)** - An Index reading of 0 - 2 indicates minimal danger from the sun's UV radiation for the average (Type 2) person. Most people can stay in the noon sun for up to an hour without burning. Sufficient vitamin D will not be produced at this level.
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- □ **5 -6 (Moderate)** - An index reading of 5 - 6 indicates some significant risk of skin damage due to the sun. Unprotected exposure can result in a burn in only 20 - 30 minutes. At this level burning or skin damage would also occur before adequate D production.
- □ **7 -9 (High)** - An index reading of 7 - 9 indicates high risk of harm from unprotected exposure to the sun. Time in the sun should be limited during midday (10:00 A.M. - 4:00 P.M.) as skin may burn in as little as 13 - 20 minutes. Enough D would be generated in 10-20 minutes (before burning) in type 1 and 2 skins.
- □ **10+ (Very High)** - An index reading of 10 - 15 indicates very high risk of harm from unprotected sun exposure. Between 10:00 A.M. and 4:00 P.M. the length of time to burn may be less than 13 minutes without protection. Enough D would be generated in 10 minutes (before burning) in type 1 and 2 skins.

If sun exposure is or must be limited or avoided, use supplements. The instructions above, followed carefully, provide the equivalent of 800-2,000 IU of D daily, what would be a 'full' dose.

Physician Protocol for D Sufficiency

K. Sullivan, CN

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Contraindications

This protocol is contraindicated in persons with active liver or kidney disease or prior damaged liver or kidney, and in sarcoidosis and other diseases of extra-renal over-production of active D. If the patient has had skin cancer or is taking medications or herbs that may cause sensitivity to sunlight, D supplements and calcium may still be used. This protocol should not be undertaken by any one at any time without a commitment to frequent testing.

Beginning understanding

Before using this protocol please read the Preliminary Report on the Importance of Sunlight and Vitamin D available from this author. It is my belief and experience that it is impossible to adequately address vitamin D sufficiency without testing. Vieth and Holick both suggest that precursor D, 25-hydroxyvitamin D is the critical value. See Holick on cancer at the end of this summary.

Also read the Essential Fats Update with vitamin K information.

Indications For Treatment

Elevated parathyroid hormone is a common presenting symptom of severe D deficiency. Elevated PTH with or without alteration of serum calcium (high or low) should indicate the need for testing 25(OH)D. 25(OH)D is the best maker of D deficiency, insufficiency and toxicity. (1-3)

PTH >65 ng/ml indicates hyperparathyroidism.(4) This may have a simple cause if you find 25(OH)D to be below 40 ng/ml.(3;5-8)

25(OH)D values < 40 ng/ml or >70 ng/ml may indicate insufficiency or excess. All patients should be tested for D sufficiency yearly as a part of their standard testing.

Patient Examples

Over the past three years only two clients in the San Francisco Bay Area, 38□ north, were found to have optimal 25(OH)D, between 55-60 ng/ml. Both spent winters in tropical latitudes and intentionally sunned, without sunscreen, during summer months..

Two patients taking high dose D, 4,000 IU daily, on the advice of a radio talk show host were found to be severely deficient . Four patients taking high dose D and one patient being treated with UV-B light for psoriasis were found to have excessive levels of D, greater than 110 ng/ml (275 nmol/l)..

The client with psoriasis undergoing UV-B light treatment was found to have a value of 127 ng/ml, high normal is probably 65 ng/ml. He was not using added D supplementation in any form. Independent testing verified the seriousness of the problem and treatment for hypervitaminosis D, which includes elimination of calcium supplements, addition of extra vitamin A , as retinol not beta-carotene,(9) and avoidance of all supplement, food, and sun sources of D was begun immediately. Eight months after treatment began this patient's D remained above 75 ng/ml.

When elevated levels of 25(OH)D are found, greater than 110 ng/ml (275 nmol/l) whatever the cause, further testing should include:

1. A second confirmed 25(OH)D
2. Fasting serum and 24 hour urinary creatinine and calcium
3. Serum PTH.
4. Follow-up studies of bone density and a heart scan are indicated if values exceed 100 ng/ml for extended periods of time.

Medical UV-B light, as used in treatment of psoriasis, does not contain UV-A. When pure UV-B is used, elevated and potentially damaging levels of 25(OH)D can rapidly accumulate. It has been suggested the UV-A portion of sunlight converts excess precursor D into inactive metabolites so that toxicity is not possible from natural sunlight, however, it is possible for light skinned individuals to produce excess levels of D in tropical climates from sun exposure (10) and persons taking D and sunning can also rapidly accumulate too much D.

During chronic vitamin D excess it is important to remember that 1,25(OH)2D may remain normal, as will serum calcium. Elevated 25(OH)D and hypercalciurea may be the only symptoms you will see. 1,25(OH)2D also remains normal in most conditions of insufficiency.1,25(OH)2D is not a useful marker of D sufficiency or insufficiency.

There is an as yet not well defined genetic component in response to both sunlight and D supplementation. Testing is the only way to determine need for and response to treatment.

Vitamin D from sunlight rapidly converts to 25-hydroxyvitamin D if liver function is normal. Studies show that D produced on the skin from sunlight is well regulated and stores as 25(OH)D in muscle, fat and blood. However, in much of the US and other non-tropical locations summer levels of D within the target range drop below that range within 2 months of diminished UV-B exposure.

Ergocalciferol or cholecalciferol D from supplements, when given in high doses, have been located in fat stores as unconverted vitamin D, ergo or cholecalciferol. This may be why supplementation is less efficient at raising 25(OH)D to optimal levels as the liver may be overwhelmed with D2 or D3 and unconverted D is removed to fat.

Understanding the difference between treatment and maintenance is also more difficult as values may rise slowly, reach optimal values, and then continue to rise to excessive amounts over time.

It is likely this delayed response to oral supplementation is the reason high dose oral vitamin D may seem non-toxic for many months or even a year or longer. Excess D, as unconverted D2 or D3, will be drawn off into fat stores, until these stores are saturated. Then, with no place to go, serum D2 or D3 and/or serum 25(OH)D may rise rapidly to potentially dangerous levels. As there is significant tissue saturation by this time, levels

of D may remain elevated for many months even with avoidance of D in supplements, food and avoidance of sunlight.

Retesting after determining the patient's choice of treatment will give indication of maintenance levels of sun and/or supplements. Retest 3-4 times during the first year making note of the season, latitude, sun exposure, amount of skin exposed, and supplement use.

There is substantial evidence that summer and winter variables in 25(OH)D are significant and the consequences, such as bone loss or depression during winter, may be dire. (11-13)

In latitudes above 40□ the seasonal variation will probably not be enough to alter need for D supplementation. This must be determined on an individual basis by frequent testing and careful monitoring. One reason Vieth may have found little difficulty using 4,000 IU of D3 in his supplement studies is that he is located in Canada and his subjects are not regular sunbathers. In addition his studies did not continue for 2-3 years.

Vitamin D supplementation restores D over time (as excess is depleted over time). The initial dose needed to restore D and the maintenance dose are likely quite different.

Believing you have achieved the 'goal' and not retesting may result in chronic excess and hypervitaminosis D.

Giving just 400 IU of D and 1,450 mg of calcium or just increasing dietary calcium to 800-1,450 mg significantly raised 25(OH)D in postmenopausal women over a three year period. (14) This amount of D did not result in D sufficiency but my point is that even low doses, especially when combined with calcium, do alter 25(OH)D over time.

The best and safest results have been obtained when physicians and patients commit to testing 25(OH)D every 8-12 weeks for one full year, summer and winter and retesting every six months years two, three and four.

Test values

Lab/Physician/Researcher 'norms' for 25(OH)D have changed over time. For most of the last 15 years the cutoff for clinical deficiency was <10 ng/ml. Test and validate 25-hydroxyvitamin D levels. Use 25-hydroxyvitamin D values, that is 25(OH)D, the immediate precursor to hormonal D and the true 'storage' site of pre-D (not 1,25(OH)2D, the active hormone) as per Holick and Reinhold Vieth study. (15) In the US laboratories commonly report 25(OH)D in ng/ml, other countries or laboratories use nmol/l.

Conversion factors- nmol/l = 2.5Xng/ml; ng/ml=nmol/l□2.5

Optimal 25-hydroxyvitamin D values are: **100-150 nmol/l** **40-60 ng/ml (using supplements)** **40-70 ng/ml (using only sunlight)**	**Normal (sufficient) 25-hydroxyvitamin D values are:** **Old 50-142 nmol/l New 80-250 nmol/l** **Old 20-57 ng/ml New 32-100 ng/ml**

The low optimum value is taken from a large European study showing 40 ng/ml to be the lowest value not associated with elevated PTH.(16) The older normal values 20-60 ng/ml are per Holick.(17) and the new values 32-100 ng/ml likely proposed by Vieth. As mentioned, older norms used a cutoff of 10 ng/ml. This is still a marker for rickets and osteomalacia. The likely cutoff of osteoporosis is 32 ng/ml.

I set the high optimum, sunlight, to be equal to 'tropical values' in normal lifestyle (not lifeguards) and the supplement maximum to be that which is least likely to result in excess, even if some sunning occurs. The low optimum is taken from various research studies as being the lowest value not associated with any disease state or elevated PTH.

I chose this range because it is one that can be attained naturally and a range where no reports (one exception, mentioned in toxicity section) of toxicity or negative consequences have been reported. Levels above 70 ng/ml have been associated with heart disease, elevated cholesterol, hypercalciurea and bone loss. It is my opinion, based on a thorough review of the literature, ongoing research will not support values greater than 70 ng/ml.

Using 55-70 ng/ml as a 'goal' is inappropriate. A goal might be simply >40 ng/ml. At 60 ng/ml, using supplements, exposure to summer or tropical sun may rapidly raise D to levels >80 ng/ml. There is no mechanism in the human body to regulate D when excess D is taken or supplemental vitamin D and sunlight are combined.

In the U.S. DiaSorin, Inc produces an accurate and reasonably priced 25-hydroxyvitamin D test. Physician information is available from http://www.vitamind.com or 1-800-328-1482. This test may be ordered directly from DiaSorin if you use your own office lab to draw, process and analyze blood. The DiaSorin test uses ng/ml so the rest of this paper will discuss D using ng/ml. If you have read the Vieth paper please remember he uses nmol/l as do researchers in Europe, the UK and AU.

Quest Labs in the US had some problems with D testing which related to how the calibrated their machines. This issue has been addressed. For accuracy I find it best to always use the same lab for repeat testing as there may be some slight differences between labs.

55 ng/ml are found commonly in the lower latitudes. 65 ng/ml or higher have been seen in chronically sun-exposed individuals. While it is possible to reach these high levels there is no indication in research they are advantageous and slight excess of these levels may contribute to disease states.

Levels under 40 ng/ml are associated with autoimmune disease, osteoporosis, and several types cancer. MF Holick suggests 20 ng/ml as the cutoff for clinical deficiency (18;19) Greater than 30 ng/ml is necessary for bone health.

Vitamin D_3 is more biologically active than D_2. (20;21) Solgar is the only company currently providing a 1,000 IU D_3 from fish liver oil, Solgar Vitamin D3 Cholecalciferol Soft Gels. As it is extracted from fish liver oil it also contains an excipient amount of vitamin A, listed on the back of the label. All other companies are using cholecalciferol extracted from irradiated sheep's wool fat. It is extracted as a dry D3 and I have found

products containing this D to provide a less vigorous response, requiring higher amounts of D3 to increase 25(OH)D.

Prescription D, ergocalciferol, 50,000 IU , is not necessary, is composed of the less biologically active ergocalciferol and may be toxic. See more below.

Treatment

Values below 25 nmol/l or 10 ng/ml- these values indicate severe clinical deficiency.(22-24)

The doses given assume no supplementation at time of testing and no exposure to sunlight, ie. winter months, or dark skin in latitudes above 35°, or light skin above 50°. If skin is light and it is summer and latitude is between 30-40° reduce dose by half and use safe sun guidelines.

1. Give 6,000 IU D_3 daily for eight weeks. The best response has been achieved by taking the supplement with the meal containing the largest amount of fat. If meals do not contain significant fat take with 3-6 grams of fish oil immediately before a meal. ***Fat must be present in significant amount at time of D supplementation to stimulate bile production for D absorption.***
2. Week 9-12 reduce to 4,000 IU D_3
3. After the initial week of D_3 supplementation verify dietary intake of, or give, 800-1,000 mg elemental calcium (total daily amount).
    A. If supplements are used give in divided doses, three or more times daily. Use calcium glycinate, calcium lactate, calcium carbonate, or calcium gluconate.
    B. If tolerated use a supplement with both calcium with magnesium.
    C. Give 200-300 mg calcium or calcium with magnesium with each meal three times a day and between meals if necessary. See information on split dose below.
4. Test at the end of 12 weeks. If there has been a significant rise, but not to within optimal range continue 4,000 IU dose for another three months.
5. If 25(OH)D is now within optimal range reduce to 3,000 IU for another 3 months and retest.
6. Adjust dose up or down, in 1,000 IU increments until the goal of 40-60 ng/ml is reached and maintained for one full year.
7. Check your dose every 6 months years 2-4 and yearly thereafter.
8. Do not over treat. 40 ng/ml is fine. 55-60 ng/ml is more a summer value. Treating to achieve >55 ng/ml may result in a situation where your patient must avoid all sunlight or use a total sun block. This is tough to do and most won't like it.
9. Retest every three months the first year and every six months years two, three and four.
10. Always suggest the minimum amount of supplemental D that will keep serum 25(OH)D above 40 ng/ml

Expect numbers to be higher in summer and lower in winter but try to keep winter values above 35-40 ng/ml.

If it is necessary to take the D with fish oil it is important they be taken immediately before food. If taken at the end of the meal bloating and indigestion are common and patients will likely stop taking the supplements.

If 25-hydroxyvitamin D values are above 25 nmol/l or 10 ng/ml but below optimal ranges (100 nmol/l or 40ng/ml)

This assumes no current supplementation. If your patient is taking vitamin D or cod liver oil or using sunlight and the values above are present record this information. The amounts listed are still correct, but considered as additions to their current supplements/sun.

If high doses of D are being taken and values are still low- make sure your testing facility is providing you with accurate testing, look for lectin intolerance, look for use of an ineffective supplement, look for bile or pancreatic problems.

If your patient wishes to use the safe sun guidelines, has white/light skin and it is summer, latitudes 30-45° NO SUPPLEMENTS SHOULD BE USED. Follow the Safe Sun guidelines.

1. 1,000-3,000 IU D_3 once a day for 4 weeks. Use 1,000 IU if 30-39 ng/ml, 2,000 IU if 25-29 ng/ml. Use 3,000 IU if 25(OH)D is 10-24 ng/ml. Take with fatty meal or with 2-3 grams fish oil immediately before meals.
2. Calcium 800-1,600 mg (+ magnesium 400-800 mg)daily in food or supplements split in three or more doses.
3. Continue or reduce to 1,000-2,000 IU once a day, for another 4 weeks and retest. All patients should be taking 1,000-2,000 IU daily at this point.
4. Maintain or reduce so that your patient is taking1,000 IU +/- sunlight after initial loading dose.
5. Base dose on test results. Do not over treat. 40-50 ng/ml is a excellent value. >55 ng/ml may result in a situation where your patient must completely avoid sunlight to stay within guidelines (unless this value was achieved by using sunlight)
6. Retest every three months the first year and every six months the following 3 years. Yearly thereafter.

No supplements should be taken on the day of testing. Adjust dose based on values and retest to make certain of optimal daily need. Retest 4 times over the first year to determine optimal summer and winter supplemental/sun D dose. Retest twice during the second, third and fourth years to make sure the program is working, enough vitamin D or sun, but not continuing to rise to excessive levels.

Necessary doses to maintain optimal D may range from 'just' sunlight and food sources (the fat of cold water fish) to as much as 3,000 IU D_3 daily. I have rarely seen the highest value, 3,000 IU, needed when diet, sunlight and high quality omega-3 fats are adequate. Genetics and sun exposure are the two most important factors regulating supplemental need. The one exception may be in seniors greater than 65 years of age.(25;26)

If the dose being used does not raise D to low-mid optimum, slowly increase the dose by 1,000 IU, always staying at the new dose for a minimum of 3 months before retesting. Sunlight and D can combine to cause elevated levels of 25(OH)D. Chronic dosing can create a situation that may take many months to resolve. Be conservative in treatment. As long as values continue to move toward optimum stay with the current dose.

Make sure calcium intake is adequate. When calcium is low or missing 25(OH)D is rapidly converted to 1,25(OH)2D, with consequent drop in 25(OH)D values. In some cases just adding calcium may raise 25(OH)D into normal/optimal range over time.(27;28)

In latitudes below 40° where sun is abundant and summer is approaching, if your patient 'sunbathes' according to the Safe Sun guidelines no extra D may be needed. Wait and retest in the fall.

No persons should intentionally sunbathe (without sunscreen) and also take vitamin D. In most latitudes in the U.S. light skinned patients are advised to take D to optimize levels and after reaching the goal, >40 ng/ml, stop all supplementation during summer months. Supplements can begin again in October or November. I am repeating this because it is important to clearly understand supplements and sun can combine to rapidly raise serum D. Chronic high levels of D can be difficult to lower. Excess D may contribute to deposition of calcium in soft tissues including the arteries even when serum calcium is normal.

Make sure to inform your patient of their D status. If you find the patient is resistant to supplemental D and has difficulty maintaining optimal levels make it clear they have a genetic need for extra D or sunlight.

Do not assume the dose of supplemental D is correct without repeated testing over a four year period. Excesses may not appear until the third or fourth year.

If levels are high without supplementation make sure the patient understands that supplementation of D beyond 800 IU may contribute to chronic D excess with possible bone loss or calcification of soft tissues. Too much D, whatever the cause, may not be immediately life threatening but chronic excess may cause bone loss and calcification of soft tissues including the kidney, arteries, brain and joints.

Other considerations

I most favor sunlight as a source of D. Following the Safe Sun guidelines, sunning midday when UV-B is present, according to the instructions found in the Sunlight and D Packet allows for optimal D without excess. The book, Naked at Noon , http://sunlightd.org gives more details. Sunning provides D by a pathway that has less risk of hypervitaminosis D and is affordable for anyone in a UV-B sunny location.

It is possible the combination of summer sun and moderate supplementation during winter months may be adequate for many. Only time and testing will tell. The expense of monitoring and supplementation may be too costly for much of the US population. Readily available reasonably priced 25(OH)D testing needs to be a priority.

Unfortunately fortification of foods or a Recommended Dietary Allowance of vitamin D just doesn't make any sense. Both will undershoot or overshoot the goal for large segments of the population. As vitamin D has important genomic functions long-term costs of deficiency or excess will undoubtedly be large.

Calcium and magnesium perform many synergistic functions. We have found success in using supplements containing 1,000 mg calcium combined with 500 mg magnesium in 2-8 tablets or capsules. When necessary, not common, you may need to add additional calcium from calcium glycinate, calcium lactate or calcium carbonate.

Vitamin D has been given in doses ranging from 8,000-50,000 IU daily for as many as 6-24 weeks with physician monitoring under certain conditions including severe, prolonged deficiency. The safety of this dosing is not clinically verifiable and as lower doses readily replete D are unnnecessary.

Serum testing of 25(OH)D is the most accurate and only safe guide to supplementing vitamin D. There appears to be no reason to use more than 4,000 IU daily, however low the initial testing, as D must be converted in the liver by a P450 enzyme to 25(OH)D.

Giving higher doses will increase the storage of unconverted D in fat tissue. If this deposition in fat later contributed to maintaining D it would be an easy treatment but there is little indication that stored D leaves fat storage except under conditions of dieting/starvation.(29) Lower doses, 1,000-3,000 IU, given with high quality fats to stimulate bile production or with lecithin, and sufficient calcium elevate 25(OH)D most efficiently.

Sunlight is the least expensive and safest source of D.(30) It is safest because almost all of the sunlight produced D is converted into 25(OH)D with little excess. When sun exposure stops or the sun wanes levels of 25(OH)D drop, usually within weeks.

The patient's nutritional status plays a significant role in response and benefit of optimum D. Vitamins A, C, K and B6 as well as the minerals zinc, magnesium, and calcium work with the vitamin D endocrine system altering response to treatment. (31-34) These nutrients may act synergistically like A and K or play key roles in enzyme production to produce 25(OH)D and 1,25(OH)2D as do B6 and C.

The common medical protocol for treating D insufficiency or deficiency is to give a prescription of one 50,000 IU tablet of vitamin D_2, ergocalciferol, once or several times a week. In three cases I have personally reviewed patients suffered severe symptoms of vitamin D toxicity, nausea, bone pain, hair loss, fatigue, yet 25(OH)D barely raised to just 30 ng/ml.

Literature supports the concept that high levels of ergo or cholecalciferol or 25(OH)D may be problematic and produce metabolic derangement, possibly by displacing 1,25(OH)2D in cellular functions.

Using pharmacological levels of D2 or D3 is not necessary to normalize 25(OH)D.

There is a possibility chronic massive doses may cause a rebound response. Vitamin D converted in the skin or taken in food or supplement passes through the liver and is

transported by DBP, vitamin D-Binding protein. This is the D storage protein found in blood, skin, and muscle. In addition, active D, 1,25(OH)2D, requires D receptors, VDR, on cells and D-Binding proteins and receptors respond to serum levels of ergo or cholecalciferol, 25(OH)D and 1,25(OH)2D.

More reasonable, physiologic doses in the natural form, D3, given over time seem to help the body replenish tissue stores more readily and without side effects. These doses over time best normalize D binding proteins and D receptor sites.

By physiologic doses I mean amounts that might be generated in the skin by persons living in ancestral latitudes or the amounts found in a high fish fat diet such as that of the Eskimo, Inuit, or fisherman diets in Japan or other island or coastal communities.

Sunlight and Supplement Equivalent?

Several researchers have suggested their studies show one MED (minimum erythemal dose) of UV-B produces vitamin D equivalent to 10,000 IU of D. The way in which these studies were designed suggests these results are not equivalent. Metabolism of skin produced vitamin D and oral D are quite different.

A cadaver study showed high levels of unconverted ergo or cholecalciferol in fat tissues when large amounts of D were given orally. The metabolic pathway allows only so much D to pass through the liver for conversion to 25(OH)D. The rest of the dose will enter the lymphatic system and proceed to fat tissue storage.

As these amounts of D 50,000 IU or greater or even less given over time, are NOT physiologic, the long term harm has yet to be determined but there is no question excessive vitamin D is disease producing in mammals, reptiles, and birds.

Traditional Diets

While rats prefer D2, cholecalciferol, D3, as made in human skin is the most biologically active form of D in humans.(35;36) Wild caught herring has about 750 if D_3 per 3 ounces but that is without the skin, organs or fat, which contain significant amounts of vitamin D_3 and would be consumed in traditional diets.

On cold water fish diets in which the fat and skin of the fish is regularly consumed daily D intake may be as high as 1,000-3,000 IU. Until these sources are tested it is hard to know. It is reasonable to assume that amounts equaling several thousand units could be ingested daily but in communities where there is virtually no exposure to UV-B light , so there would be no chance for excess due to the combining of food and sunlight sources.

Genetic factors seems to provide tolerance to high vitamin D and low calcium intake in northern First Nation peoples traditional diets. These diets contain very high intakes of vitamin D from whale, fish, and seal fat, yet are very low in calcium. This combination supported health in these communities and as traditional diets have been rejected serious health consequences have rapidly appeared. It is possible within these communities combining high levels of D and high levels of calcium could potentially create a disastrous situation.

It is likely maximum intake of vitamin D over time is about 1,000-2,000 IU. Perhaps higher levels of vitamin D are safe if calcium intake is minimal. This has yet to be determined.

Given that we currently do not test foods for vitamin D and that the amount of D found in eggs or animal fat is dependent on supplemental D or exposure to UV-B it is hard to argue the point that food is currently an unknown and unreliable source. Food D plus sunlight in summer or living in lower latitudes and using vitamin D supplements or fortified foods could combine to raise levels of cholecalciferol and 25(OH)D beyond reasonable and safe limits. We need D but not too much and we need other elements to make D work.

In tropical or sub-tropical locations other genetic attributes are present. As the Inuit are 'designed' to get D from marine sourced fat, persons from the African continent relied entirely on sunlight.

Both Inuit (and likely other northern First Nation peoples) and African peoples recycle calcium in the kidney efficiently so that diets containing as little as 200 mg of calcium are able to build and sustain calcium requirements. This fact may explain why African Americans are less likely to suffer from osteoporosis but manifest other symptoms of vitamin D insufficiency including hypertension, obesity, type II diabetes, and varying cancers.

In India studies have shown rapid turnover of cholecalciferol and 25(OH)D by processing enzymes which would protect from excess production of D from sunlight. This modification of metabolic pathways may occur in other gene pools as well. Lack of sun exposure would contribute to low status as life styles alter exposure to UV-B.

Civilization has contributed to low D by:

1. Dietary changes (less D containing foods and/or addition of cereals which impair D status)
2. Life style (less sun, more clothing)
3. Public health policies (warnings to avoid sunlight when it contains UV-B, mid-day)
4. Migration and immigration (moving from a location where appropriate foods or UV-B could be found for your genotype)
5. Intermarriage (mixing genes without understanding the implications for sourcing D)

Malabsorption

I have seen a few situations in which low serum 25(OH)D, followed by supplementation with 4,000 IU daily, failed to move serum D into optimal range, and in two patients 25(OH)D did not change at all.

In several cases personally known to me the patients were taking a dry form of vitamin D3. I was told by a product formulator the cellulose used to extract and concentrate D may bind the D and prevent or partially prevent absorption..

One patient had been taking, on her own from reading alternative health literature, 4,000 IU of a dry vitamin D product. After one year her 25(OH)D tested at 25 ng/ml, an osteoporotic level for this 79-year-old female. On fish oil based D plus summer sun exposure she rapidly repleted her D status to 51 ng/ml. She maintains this value by supplementing in winter and safe sunning in summer months.

Certain drugs increase or interfere with vitamin D. Corticosteroids are a well known problem.(37-39) If medications are causing D insufficiency make sure to note this and modify dose and also consider this, the need to adjust dose if medications are stopped.

In some of the cases of D insufficiency or malabsorption there was clear indication of altered gut mucosal integrity.

Loren Cordain, Colorado State University, personal communication, suggests that wheat or lectin intolerance may damage the gut to the extent that D is not absorbed or liver conversion of D_3 is altered.(40)

Early researchers found diets high in grains, particularly whole grains including oatmeal dramatically increased the need for vitamin D.(41;42)

Consider testing for gluten intolerance(43-46) or removal of possible offending lectins (soy, wheat, rye, dairy, oats, legumes, barley, nightshades) for 4-8 weeks while continuing supplementation and retest.

I have also used topical (skin) application, which does raise serum D and may be used with infants and children. For more information on lectins visit http://krispin.com/lectin.html

Other causes of malabsorption may be pathogenic bacteria, yeast overgrowth or parasites. If 25(OH)D is still not moving consider testing for pathogens. Two reliable testing sources are-

Diagnos-Techs, Inc Expanded GI Health Panel, GI-2, 1-800-878-3787, or

Genova Diagnotics CDSA + parasitology random/purge profile, 1-800-522-4762

Online go to http://www.diagnostechs.com or http://www.gdx.net

Another problem may occur following removal of the gallbladder or if bile insufficiency is present. Both conditions will significantly reduce absorption of fat-soluble vitamins including D, A, E and K. Use the Lecithin or Bile Protocol listed in the section on intolerance if this is the situation.u

Supplement choices

D_3, as found in fish oil based D, is the most biologically active D and the preferential form for humans.(47)

> Solgar Vitamin D3 Cholecalciferol soft gels, contains some vitamin A as an excipient. In my experience the best absorbed and utilized but no always appropriate.

Jarrow or other brand D3, dry source dissolved in oil (safflower, soy, or olive)
VRP, Life Extension or other brand D3 dry source capsules

These forms come in doses ranging from 800 IU to as much as 10,000 IU. I have found websites and physicians supporting the regular use of 5,000-10,000 IU daily. (My only hope is that they are using a poorly absorbed source.)

Intolerance to supplementation

Patients using dry forms of D, so called vegetarian D3 from irradiated sheep's wool grease (lanosterol) or D2 ergosterol (irradiated yeast is the usual source), have had greater difficulty repleting and maintaining D. Irradiated lanosterol is D3 but there still seems to be difficulty in absorption, perhaps because the supplement is not consumed with adequate fat or because the extraction process uses cellulose. This may explain why higher amounts of this form of D may be needed to increase 25(OH)D.

In Australia and some parts of Europe vitamin D_3 is not available but only the plant based ergocalciferol, vitamin D_2.

When poor digestion and assimilation of fats exists gastrointestinal distress may occur. If this happens use the lecithin protocol. This protocol is also necessary if the gall bladder has been removed or if cholestasis is present.(48-52)

Other patients, rare but it may happen, seem to react to a particular supplement with symptoms ranging from heart palpitations to 'a burning pain' in the gut. If this occurs consider a different brand or source, dry or oily, fish liver oil based or lanosterol based untilf you find what works for them.

Protocol to enhance D (and other fat soluble vitamins) absorption

Fat soluble vitamins which include vitamins K, D, A and E require significant bile and pancreatic enzymes for absorption. If digestive problems are current or chronic consider the following-

With (at the same time as) each daily dose of D and fish oil take (**pick one**)

1. 1 egg yolk, cooked (equiv. 1 tbl. Lecithin, it's ok to include the white) or
2. 1 heaping teaspoon of lecithin granules or
3. digestive enzymes or pancreatin with ox bile.

Lecithin, from the egg yolk or granules, or bile salts must be taken at the time of the dose so that emulsification of the fat soluble D occurs in the digestive tract. This combination overcomes problems with bile insufficiency and malabsorption of fat-soluble vitamins. (53)

If the lecithin or other digestive remedy does not stop gastrointestinal distress all or part of the vitamin D dose may be rubbed on the skin. To my knowledge this works only with the fish liver oil based D and will stain clothing and sheets. Allow about 1 hour for absorption before removing excess. The soft gel is pierced with a pin and squeezed onto the skin and then rubbed in. This method may also be used for repleting D in infants.

The importance of calcium with D

Calcium supplementation is determined by genetic need, bone density, age, height and weight. Low bone density or larger body size, whether lean or fat, need the highest dose.

Also use the higher dose if dietary potassium is low and average pre-meal AM salivary pH is below 6.5. Salivary pH reflects blood ionized calcium and blood pH and is a marker of alkaline reserve when vitamin D is within optimal range. (54) Calcium lactate combined with a diet high in potassium containing foods is particularly useful to normalize salivary pH, restore alkaline reserve and preserve bone.

D does not maintain bone calcium. It regulates serum calcium. Giving D when calcium is insufficient will withdraw calcium from bone. D_3 supplementation or sunlight should not be prescribed without knowing calcium intake and supplementation of calcium should be initiated if dietary intake is below 800 1,000 mg daily.

Elevated levels of 25(OH)D have been clinically shown to CAUSE bone loss. 40-55 ng/ml 25(OH)D is safe and sufficient. There is no clinical support for efficacy or safety of 25(OH)D above 65 ng/ml when using supplements.

If mildly elevated PTH or hyperparathyroidism is present with low serum calcium begin D and calcium together. Please see papers at the back of this report.

In the case of elevated blood calcium with elevated PTH first check 1,25(OH)2D and if normal start D supplementation, add calcium after the first 2 weeks.

Calcium supplements need to be balanced with adequate levels of D. Serum vitamin D regulates the level of ionized or free calcium and if blood calcium is low serum vitamin D will draw calcium from the bone.

Calcium and D_3 do not need to be taken at the same time. D_3 is usually taken once a day with the meal highest in fat. Calcium is absorbed more efficiently when taken 200-300 mg of calcium per dose.

500 mg. calcium-one dose	29% absorption
500 mg. calcium-two doses	36% absorption
500 mg. calcium-3 doses	40% absorption
2,000 mg- one dose	14% absorption

Heaney, RP et.al. J of Bone and Mineral Research, 5:11; 1990 p.1135-1137

Higher amounts of calcium, as well as magnesium and trace elements, may be needed with diagnosed bone loss. Total daily calcium as a supplement may range from 800 mg to 1,000 mg depending on current dietary calcium, bone status and body size.

Heaney found that post-menopausal women often needed as much as 2,400 mg of calcium daily to return PTH to low, young adult, values.(55) However when this study was done vitamin D levels were not a consideration.

D sufficiency may reduce, even dramatically reduce, the need for calcium. Some research indicates 70% absorption with D sufficiency compared to 20% absorption with D insufficiency.

An easy way to encourage calcium consumption in more frequent small doses is to prescribe children's chewable calcium and magnesium to be taken throughout the day. Nutrition Now makes Rhino Cal with 125 mg calcium, 65 mg magnesium and 50 IU D per each raspberry flavored tablet. Nature's Plus offers Nutri-Cal Hearts, each containing 250 mg calcium and 125 mg magnesium plus 50 IU vitamin D, heart shaped and vanilla malted flavor.

For patients with elevated vitamin D, when they can once again take calcium, the Vitamin Shoppe offers Vita-Bear with 250 mg calcium and 125 mg magnesium and no vitamin D. When 25(OH)D is elevated reducing calcium will more rapidly reduce excess 25(OH)D.

Tremor or tetany

If tremor, tetany or muscle tightness, or complaints of 'charlie horse' occur at higher doses of D and calcium maintain 25(OH)D by serum testing and reduce calcium dose or increase magnesium. In some patients magnesium may need to be equal to calcium intake. Magnesium glycinate (Albion Labs, in some Solgar, Metagenics, Douglas and Carlson Lab products) is the most tolerated magnesium. The decision to increase magnesium or reduce calcium should be guided by bone density status. For more information on Albion magnesium http://www.albionlabs.com/human/archive.htm

Rebuilding bones

Bones are made of calcium, magnesium, phosphorus and many trace minerals. The bone matrix is dependent on protein. Bones cannot be rebuilt without all of the necessary elements. In seniors make sure adequate protein is consumed daily. (56;57) While protein supplements may acidify and lower bone density, whole proteins increase quality and quantity of bone. Eggs are an inexpensive and safe source of protein for most persons. They do not contribute to heart disease risk (58-60) unless they are fried and consumed with bacon and the like.

Expensive 'chelated' calcium is not necessary if vitamin D intake reflected by optimal 25(OH)D is adequate. With low D status 20% of calcium is absorbed, with optimal D 70%.(61) The one exception may be seniors with hydrochloric acid insufficiency. If this is found to be the case, digestive enzymes, an increase in protein and mineral chelates, or multi-minerals containing chelated calcium and magnesium with trace minerals, may rapidly enhance calcium absorption, and bone rebuilding and repair. Now Foods Full Spectrum Minerals, Carlson Labs Mineral Compleet or Country Life Total Mins offer high quality chelates with trace elements.

The use of calcium supplements without sufficient D may cause other as yet unrecognized problems. Vitamin D controls the production of some calcium binding proteins critical to normal calcium utilization.

It is important to think of calcium, vitamin D and vitamin K as members of a team. While studies show that increasing calcium may slow bone loss even in the presence of less

than optimal 25(OH)D there are other considerations. D-binding proteins and calcium-binding proteins are the cutting edge of biochemistry and molecular biology and we are headed into discovery of and understanding of the amazing relationship of man to light (or when UV-B light is absent or low, supplemental vitamin D).

Once optimal 25(OH)D is achieved and verified by testing, maintenance by daily intake of supplements and/or sunlight should be a lifelong practice. Patients need to be instructed in how to maintain their personal need for D.

REMEMBER: Too low or too high 25(OH)D may cause bone loss and tissue calcification.

An understanding of our relationship to and need for sunlight and D is critical to health. Over the next few years more and more information will become available to validate this information. Encourage your patients (and your peers) to take the time and spend the funds necessary to understand their need for D.

If you are a physician participating in an HMO encourage your group to include 25(OH)D testing as routine. It will save many dollars in health care costs over the years of your patients' lives.

Side effects of treatment

Transient hypercalciuria may occur. This is harmless and non-complicating since this protocol is not used with patients with kidney disease. It does not increase kidney stone production. Read the information on vitamin K in the Omega-3 Update enclosed. If hypercalciurea occurs with 25(OH)D >65 ng/ml refer to the section on toxicity.

An unusual sleepiness may occur lasting for several days to as much as several weeks. This seems to relate to alteration in 25(OH)D and serum calcium levels. It is noticeable because it may occur mid-day and napping may be impossible to resist.

As long as blood testing continues and serum 25(OH)D remains between 40-60 ng/ml problems associated with excess D are unlikely to be an issue**.**

Hypercalciurea with a 25(OH)D exceeding 65 ng/ml should be a concern for the practitioner. Immediately stop all sources of D including eggs, fish, sunlight exposure and supplements.

Toxicity- Hypervitaminosis D

Do not overdose. Do not supplement without frequent testing. Vieth and Hollick have suggested that high doses of D ranging from 2,000-4,000 IU or even 10,000 IU daily are safe and may be necessary for D sufficiency. My experience over the past eight years suggests in some cases they are right, but in many these doses will raise D above optimum after 1-2 years, or sooner.

Do not consider giving D supplementation unless your patient is willing to test every three-four months the first and second year and every six months the following **two**

years. Often elevated levels of D did not appear until the end of the second or even the third year of supplementation with disturbing symptoms from depression to bone pain.

Between November 1992 and November 1993 John S. Adams, MD tested vitamin D, PTH and TSH, urinary calcium and creatinine in all new patients referred to Cedars-Sinai Bone Center for possible osteoporosis. 69 patients were tested. The study information is available in the Annals of Internal Medicine, August 1, 1997 127:203-206. Adam's outcome showed 4 patients testing with elevated calcium in the urine and elevated 25(OH)D, between 52.8-88.8 ng/ml. Each of these patients had significant bone loss, which resolved over the next three years after removing excess D.

While it is possible, and likely, that the lowest value seen in this study was not an excessive value, even this patient regained bone when the level of D dropped . The higher 25(OH)D levels, those above 65 ng/ml, fit cases seen over the past 3 years in which the goal, 40-60 ng/ml, was exceeded and quantifiable bone loss occurred. It is likely some persons are more sensitive to excess 25(OH)D.

In 6 cases I have personally monitored where 25(OH)D exceeded optimal levels the causes were mixed, two by supplementation with vitamin D, two by supplementation plus sunlight, one, sunlight alone, and one by UV-B treatment only, prescribed for psoriasis. This last patient took no supplemental D and had the highest value of 25(OH)D at 127 ng/ml.

In all of these cases there were consequences, in particular bone loss, though serum calcium and PTH remained normal. The problems were detected by bone scan and urine testing for calcium and creatinine. Unfortunately there is evidence that elevated 25(OH)D may contribute to artery damage even when serum calcium is not elevated.(10;29;62)

When hypervitaminosis D occurs recovery may be very slow. If elevated levels are detected within a month of first appearance reversal may be rapid but if the excess caused by high dose D supplementation has been going on longer, undetected by regular testing of serum 25(OH)D, levels may drop only 5-10 ng/ml per month in those I have screened. During this time all foods with D are excluded and sunlight is avoided.

Foods to exclude: fish, shellfish, eggs, fortified dairy, margarine, fortified cereals and mushrooms. Calcium must be stopped until D values go below 75 ng/ml and all supplements must be free of vitamin D, which would include any multiple. Cod liver oil must be avoided as well as animal livers. Fish oil in capsules, a source of omega-3 fats, guaranteed to not contain vitamin D because of the molecular distillation process, may be continued.

Supplements with unknown ingredients should be avoided. In the Adam's study one patient became toxic taking an herbal remedy containing unlisted vitamin D_2.

If bone loss is severe bisphosphonates are the treatment of choice. These drugs have been used successfully to prevent the side-effects of vitamin D toxicity, which include bone loss and arterial calcification. (63)

Excess vitamin D alters vitamin A metabolism and extra pre-formed vitamin A, not beta-carotene, without any vitamin D such as retinol palmitate, can moderate excess D.(64;65)

There is significant difference between chronic low dose excess and acute toxicity from high dose treatment with vitamin D_2 or D_3 (ergo and cholecalciferol), 25(OH)D (calciferol) or rocaltrol or calcijex (calcitriol).

The prescription (high dose) vitamin D_2, precursor 25(OH)D or the active hormone, calcitriol all have the potential to cause toxicity, chronic and acute. There is no evidence that these supplements are necessary except in rare genetic disorders or when liver or kidney failure is present. They are useful and safe only in the hands of a few endocrinology specialists.

Kidney Stones?

High levels of 25(OH)D, that would give serum values of 25-hydroxyvitamin D >200 nmol/l, >80 ng/ml, or daily supplementation of >1,000 IU D_3 for an extensive length of time in persons not needing D supplementation may contribute to kidney and other soft tissue calcification.

At this time the most common nutrient abnormalities related to the development of kidney stones are dietary insufficiency of B-complex vitamins, especially B-6(66), or magnesium and/or calcium deficiency. (67-69) Vitamin K at levels of 1 mg. daily reduces calcium loss in the urine in postmenopausal women. Make sure vitamin K is a part of any protocol. (70-75)

Heart Disease?

In a recent University of California study of 9,704 women 65 and older those taking calcium with vitamin D had a significantly reduced incidence of heart disease(76). BUT in Southern India high levels of 25(OH)D had a strong correlation with ischemic heart disease.(10) Enough but not too much is the rule. Levels of 25(OH)D should not exceed 65 ng/ml. Enough and safe- 40-60 ng/ml.

Oral supplementation of vitamin D is used to create a condition similar to atherosclerosis in rats used for heart research. The vitamin D dose used, 2,000 IU daily, is equivalent to giving an adult human, weighing 150 pounds, 200,000 IU daily. This is a toxic dose and would not have an equivalent in humans except under conditions of research or when persons have been exposed to high levels of concentrated D inappropriately added to foods.(77-80) Research concerned with optimal amounts of D shows adequate D reduces or prevents arterial plaque.(81)

Also important when plaque is present - adequate vitamin K, about 1 mg. daily. German studies suggest minimal K intake is 900 mcg. ten times greater than the US RDA of 90 mcg. (82-86) Since Warfarin, used to decrease clotting, lowers vitamin K this drug actually contributes to heart disease, especially atherosclerosis.(87;88) Giving omega-3 fish oil instead of Warfarin may safely prevent abnormal clotting and does not lower levels of vitamin K.(89-93)

Vitamin D, Sunlight and Skin Cancer

Sunlight has always been the most dependable source of vitamin D with food sources supplementing the low light of far northern regions. Because of the relocation of races to areas distant from ancestral optimal latitudes, darker skin types moving far north or south, lighter skin types moving nearer the equator, over and under exposure to

ultraviolet light, both UV-A and UV-B, is widespread. This migration has brought about serious consequences. One of these is an increase in skin cancers.

For lighter skin types overexposure to sunlight increases the incidence of basal and squamous cell skin cancers and, because of exposure patterns, may not provide optimal levels of D. For darker skins production of D requires longer exposures to UV-B sunlight and at latitudes above 30□,□north or south, UV-B intensity is inadequate much of the year to provide optimal D.

The best sunscreen is clothing.(94)

For more detailed and researched information on these issues please refer to the Brief Report on Sunlight and Vitamin D available from http://sunlightd.org or the book, Naked At Noon, Understanding Sunlight and Vitamin D by this author. Both contain sun exposure guidelines, with clinical studies, and information about getting and maintaining optimal levels of D.

MF Holick and the Cancer Connection

For many years researchers have suggested a connection between D, calcium and cell hyperproliferation.(95-97) In the September, 2002 issue of Osteoporosis and Metabolic Bone Disease Michael Holick presents preliminary evidence that both benign prostatic hypertrophic cells and prostate cancer cells have enzyme activity capable of converting 25-hydroxyvitamin D into active D. This enzyme is reduced in hypertrophic and cancerous tissue, between 50-90%, but still present. When these abnormal cells were cultured with 25-hydroxyvitamin D there was an up-regulation of the critical enzyme, an increase in calcitriol and marked inhibition of proliferation. (98) Holick believes that adequate levels of 25-hydroxyvitamin D, greater than 40 ng/ml, may be protective from most of the common epithelial cell cancers including breast, colon, prostate and skin.

An abundance of studies continue to confirm these findings..

More on Cancer

In January of 2002 Holt, et al, reported an inverse relationship between 25(OH)D and colonic epithelial cell proliferation. 'Tropical' levels of 25(OH)D were protective. This study reaffirms the importance of the precursor 25-(OH)D for health.

Cancer Epidemiol Biomarkers Prev 2002 Jan;11(1):113-9

Colonic epithelial cell proliferation decreases with increasing levels of serum 25-hydroxy vitamin D.

Holt PR, Arber N, Halmos B, Forde K, Kissileff H, McGlynn KA, Moss SF, Fan K, Yang K, Lipkin M.

Gastrointestinal Division, St. Luke's-Roosevelt Hospital Center, New York, NY 10025, USA.

Epidemiological evidence suggests a potential role for vitamin D in colon cancer prevention. Vitamin D, absorbed from the intestine or derived from solar ultraviolet light, is metabolized in the liver to 25-hydroxyvitamin D (25-OH D(3)). Previous studies examining effects of vitamin D upon carcinogenesis have focused upon the active metabolite 1,25-dihydroxyvitamin D [1,25-(OH)(2) D(3)], which interacts with nuclear vitamin D receptors in several organs. Until recently, the metabolism of 25-OH D(3) to 1,25-(OH)(2) D(3) was believed to occur only in the kidney, but more recent studies have shown that 25-OH D(3) conversion to 1,25-(OH)(2) D(3) can occur in other tissues. We examined the association between fasting levels of 25-OH D(3), 1,25-(OH)(2) D(3), and BsmI polymorphism of the vitamin D receptor (VDR) gene with indices of colonic epithelial cell proliferation and differentiation in a chemoprevention study, after giving vitamin D or calcium and taking rectal biopsies that were incubated with bromodeoxyuridine. Vitamin D receptor polymorphism was determined by genotyping of the 3' BsmI polymorphism in intron eight of the VDR gene. No significant changes in cell proliferation or in differentiation were found in subjects between study start and end. However, fasting serum levels of 25-OH D(3) showed a highly significant decrease with whole crypt labeling index and the size of the proliferative compartment (phi h). There was no correlation between serum levels of 1,25-(OH)(2) D(3) and the proliferative parameters. Calcium supplementation induced a significant effect upon the relationship between serum 25-OH D(3) and rectal epithelial cell labeling index and phi h when studied by covariance analysis without a relationship with 1,25-(OH)(2) D(3) levels. VDR genotype did not influence the effects of serum 25-OH D(3) or serum 1,25-(OH)(2) D(3) levels upon proliferation. These data suggest that there might be a local effect of 25-OH D(3) on colonic epithelial cells through conversion of 25-OH D(3) to 1,25-(OH)(2) D(3). Subsequent studies have demonstrated the presence of 1alpha-hydroxylase mRNA in normal colorectal epithelium and in colorectal cancer. Thus, vitamin D may have an important role in determining the effects of calcium on colorectal epithelial proliferation and may explain some of the discrepancies found previously in studies that examine the direct role of calcium on the colorectal epithelium.

Vitamin D and Multiple Sclerosis

In April, 2001, a small study at Penn State and Helen Hayes Hospital in New York showed at a daily dose of D, 1,000 IU reversed two of the markers of active MS. 1,000 IU daily of D decreased interleuken-2 associated with cells that induce MS and increased transforming growth factor beta-1 associated with remission of the disease.

Unfortunately the study designers did not consider testing and treating as described in this paper. I am sure that had they done so their results would have been even more dramatic with more rapid response.

Several MS support groups and at least one researcher have suggested high levels of vitamin D to treat MS. This suggestion is not based on research showing D improves, prevents or cures MS but on research showing less MS in areas with more UV-B light. Taking D without testing and believing that pharmacological doses of D or elevated levels of 25(OH)D may improve MS is likely to be a dangerous proposition. There is no indication that levels of 25(OH)D above 65 ng/ml are safe, nor that higher values have any benefit in MS or any other autoimmune disease. Persons in areas with more UV-B light typically have values around 50-65 ng/ml.

I am available for consultation, for a fee, as you begin to work with the protocol in your practice. I can be reached most mornings, Pacific Coast time, Monday through Friday 1-775-831-0292.

We contribute to disease incidence and promote early death by misunderstanding the importance and use of sunlight and vitamin D.

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Patient Protocol for D Sufficiency

Patient Protocol for D Sufficiency

This protocol is contraindicated in persons with active liver or kidney disease or prior damaged liver or kidney, in sarcoidosis and other diseases of extra-renal over-production of active D. If you have had skin cancer or are taking medications or herbs that may cause sensitivity to sunlight, vitamin D supplements and/or calcium may still be used. If in doubt inform your physician.

Before using this protocol please read the Preliminary Report on the Importance of Sunlight and Vitamin D available from this author. Vitamin D is a pro-hormone NOT a vitamin and too much OR too little may have grave consequences. It is my belief that it is impossible to adequately address vitamin D sufficiency without testing. Vieth , Holick and other experts suggest that precursor D, 25-hydroxyvitamin D, also written as 25(OH)D is the critical value to judge D status.

Do not consider D supplementation NO MATTER WHAT YOUR SYMPTOMS without testing and monitoring with the help of a physician or other licensed health care practitioner **who understands both the importance and the possible dangers of vitamin D**.

By means of testing I have found there is a genetic component in response to both sunlight and D supplementation. Testing is the only way to determine need for and response to treatment. Vitamin D from sunlight or supplements acts as a pro-hormone, rapidly converting into 25-hydroxyvitamin D, the circulaing storage D found in blood and tissues which is converted to 1,25 (OH)D, the active hormone, as needed.

When first addressing the issue of D insufficiency or deficiency a supplement of vitamin D may be critical to normalizing serum 25(OH)D. After a year or so of regular supplementation of a similar dose may no longer necessary and even potentially dangerous. Higher levels of D, levels above 80 ng/ml or 200 nmol/l, do not promote health. If sun is used wisely, moderate supplementation may only be necessary during winter months, Nov-March or not at all if winter vacations are taken in tropical or semi-tropical locations.

Retesting will give indication of maintenance levels of sun or supplements. Retest when the season changes in mid latitudes, 30-50° north and south, which includes most of the US and Europe. There is clear evidence that summer and winter variables may have significant negative health consequences, such as bone loss or higher incidence of infections, occurring frequently in winter months in some locations.

In latitudes above 50□, such as Canada and the UK, some supplementation may be necessary year round. In locations below 30□, such as Florida and south Texas, it may be appropriate to use no supplements, depending on sunlight year round. A majority of persons in the US may find that supplementation is needed only in winter months.

It is important to decide how you will get your D and stick with it. Chronic high dose supplementation can become toxic over time showing 'safe' values for up to three years before jumping rapidly to excessively high values. Combining supplements and sunlight can also cause serum values to reach potentially dangerous levels rapidly. If sunning when UV-B is present, summer in the US, don't use a supplement or cod liver oil. An exception would be a person with very dark skin living in more northern parts of the US, where both sun and supplementation may be necessary.

Some over enthusiastic supplement users may reach levels of vitamin D that make it necessary for them to use a complete sunblock and avoid sun until levels decline, difficult if you are vacationing in Hawaii and want to surf or swim.

Test Values for Vitamin D Sufficiency

Test and validate 25-hydroxyvitamin D levels. Use 25-hydroxyvitamin D values, that is 25(OH)D, not 1,25(OH)D as per Reinhold Vieth study. (1)

Labs use either nanograms per milliliter (ng/ml) or nanomols per liter (nmol/l). The conversion factor allows you to see your value either way.

Conversion factors-
nmol/l = 2.5 X ng/ml
ng/ml = nmol/l□□2.5

Optimal 25-hydroxyvitamin D values are:	**Normal (sufficient) 25-hydroxyvitamin D values are:**
100-150 nmol/l or 40-55 ng/ml (using supplements) 40-70 ng/ml (using sunlight)	Old 50-142 nmol/l New 80-250 nmol/l Old 20-57 ng/ml New 32-100 ng/ml

55 ng/ml are found commonly in the lower latitudes. 65-75 ng/ml have been seen in chronically sun-exposed individuals without known complications. Levels under 40 ng/ml may be associated with secondary

hyperparathyroidism, obesity, osteoporosis, and cancer. MF Holick suggests 20 ng/ml as the cutoff for clinical deficiency (2;3) Insufficiency occurs between 21-31 ng/ml. Sufficient 25(OH)D is greater than 32 ng/ml. Though the new normals allow up to 100 ng/ml current data do not support values higher than 65 ng/ml. In sensitive persons values as low as 67 ng/ml have resulted in bone loss. (BONE LOSS)

Vitamin D_3 is more biologically active than D_2. (4;5) Solgar Vitamin D3 Cholecalciferol 1,000 IU soft gels provide 1,000 IU D_3 extracted from fish liver oil. This product also contain a small amount of vitamin A, an excipient which cannot fully be removed. This may actually be a good thing as the balance between A and D is important to vitamin D status and bone health.

Most supplement companies now have D3 in doses of 400-50,000 IU. Dry encapsulated vitamin D may be difficult to absorb and not provide as rapid normalization of serum 25(OH)D as the Solgar product but if excess A is an issue they may be used. Make sure to always take vitamin D with a HIGH fat meal (or use the lecithin granules). Doses higher than 4,000 IU daily are never suggested for the long term unless definitively proven to be safe and necessary by testing. Do not forget to regularly test.

Prescription D2, 50,000 IU or greater is not necessary and may have negative consequences.

Your test values must be balanced with your current intake of D. If you are taking a supplement of D, 400 IU and you test low, follow the instructions as given. If you test low and are taking 1,000 IU or more of vitamin D in any type of supplement or cod liver oil you need to determine if you are utilizing what you are taking and if not why not, before increasing your dose.

Values below 25 nmol/l or 10 ng/ml- these values indicate severe clinical deficiency.(6) At this level you have a severe clinical deficiency and should be monitored closely by your physician (7)

This protocol assumes no exposure to sunlight. If you live between 30-50° latitude and it is winter, proceed. If it is summer and your skin is light colored and you will be full body sunning reduce the dose to half that listed or consider just using sunlight. Sunlight rapidly raises serum 25(OH)D and there is little danger of toxicity. Darker skins need the higher dose even if exposed to sunlight.

If your latitude is higher no reduction is needed. If your latitude is lower, less than 30°, some sun and half dose OR all sun following instructions for skin type in the sunlight protocol and NO supplements. (8;9)

This protocol is superceded by advice from your physician. He or she may choose to use higher doses of vitamin D for a period of time. Under supervision with frequent testing higher doses may be appropriate. Long term use of high doses of D as 25(OH)D moves closer to optimal values is inappropriate.

The first 12 weeks depending on starting values of 25(OH)D:
Take 3,000-4,000 IU D_3 once a day with your highest fat meal, 1,000-2,000 IU if also using sunlight.

You must take your daily D with a meal containing significant fat. If you are eating a meal without much fat you may add 4-6 fish oil capsules (not cod liver oil), such as Costco Kirkland Fish Oil or Trader Joe's Trader Darwin Omega-3 or Now Foods Super EPA Double Strength Fish Oil. Fish oil supplements enhance D uptake and provide important omega-3 fats. Again, this is fish oil, not cod liver oil.

Do not use cod liver oil for treating vitamin D deficiency as it contains significant amounts of vitamin A which you may or may not need and may confuse the outcome of your program. While cod liver oil may be used to maintain levels of D it is typically not sufficient for repleting D in a serious deficiency. Taking enough cod liver oil to raise D may provide excessive or toxic levels of vitamin A.

Retest at 12 weeks, whether using supplements or sunlight.

If 25(OH)D has risen but is not yet within optimal range continue at 3,000-4,000 IU for 3 more months. Retest.

If/when vitamin D has risen to within optimal values continue on using 2,000 IU daily dose and retest again in 12 weeks. If at that time D has not stayed within optimal levels, 40-60 ng/ml increase to 3,000 IU daily and retest in 3 months. Increase or decrease your D by 1,000 IU increments until you find the dose that maintains your D within optimal range, 40-60 ng/ml. Test every 3-4 months until you determine your range for summer and winter.

If your D is elevated, above 70 ng/ml, stop supplementation and/or sun exposure immediately. Consult your physician.

Always make sure your intake of calcium is between 800-1,200 mg elemental calcium (total amount) daily. If supplements are necessary take in divided doses, two or more times daily. The best way to supplement calcium, other than food, is by using a complete multi-mineral. Good formulas are available from Now Foods, Solgar, Country Life or Nutrition

Resource. There are others. Look for significant amounts of magnesium, zinc, manganese, boron and other trace elements. Many of these minerals work with vitamin D to build and protect bone, not just calcium. Magnesium is responsible for the hardness of bone.

Supplemental calcium may contribute to constipation. This is usually not the case in multi-minerals. Excess magnesium can cause diarrhea. If your bowel movements change with the intake of any supplement look for other types or brands.

One of my clients and students favorite sources of calcium and magnesium are children's chewable calcium with magnesium. They taste great, like a treat, and are easy to remember. Nature's Plus Nutri-Cal hearts are my personal favorite. Also popular, Vitamin Shoppe's Calci-Bears and Nutrition Now Rhino Cal. Make sure to chew enough throughout the day to reach your calcium goal.

Calcium need is determined by genetics (your ancestors), bone density, age, height, and weight. If you have low bone density or larger body size, whether lean or fat, you'll need the highest dose. Also use the higher dose if average pre-meal AM salivary pH is below 6.5. Salivary pH reflects blood ionized calcium and blood pH and is a marker of alkaline reserve. (10)

Salivary pH does not reflect D status. It is a useless number, to gauge your need for calcium, if vitamin D is not in the optimal range.

If serum 25(OH)D is within optimal range and the AM saliva is acidic a multi-mineral plus plenty of potassium containing foods may be the best source of alkaline reserve for you.

If at any point something feels wrong,
stop what you are doing and call your physician.

If 25-hydroxyvitamin D values are above 25 nmol/l or 10 ng/ml but below optimal ranges (100 nmol/l or 40ng/ml) –

This protocol also assumes no sunlight. If it is summer, or lower latitude, sunlight according to the protocol for your skin type may be used with NO supplements. If you have dark skin and are in latitudes above 30°, you will need to supplement, the sun just is not strong enough, even in summer, unless perhaps you spend most of the day outside with scant, very scant, clothing.

1,000-2,000 D_3 once a day for 8 weeks. Use 1,000 IU once a day if 30-39 ng/ml, 2,000 IU a day if 25-29 ng/ml. Take with a fatty meal or 4-6 grams of fish oil.

.
Calcium, 800-1,200 mg (+ magnesium 400-600 mg)daily split in three or more doses. This is the daily total from all sources. Higher levels of vitamin D may reduce your need for calcium, or extra calcium may reduce your need for vitamin D.

At 12 weeks retest and maintain, increase or reduce (+or- 1,000 IU) based on test results.

Retest every three or four months the first and second year and every six months the following 2 years to determine optimal summer and winter sun and/or supplement D dose.

> No supplements should be taken on the day of testing.
> Always adjust dose based on serum 25(OH)D
> Taking too much D cannot solve the problem of low vitamin D.
> The potential toxicity of vitamin D requires conservative action and lots of testing.
> The same dose that optimized your D when you started may over time continue to raise your D to excess levels.

Necessary doses to maintain optimal D may range from sunlight and food sources only to daily cod liver oil to as much as 2,000-3,000 IU supplemental D daily. Genetics (your skin, liver, D receptors and binding proteins) and sun exposure response (location/latitude/timing) are the two most important factors regulating sun and/or supplemental need.

Oral intake of D and sunlight may combine to raise serum D beyond optimal values. Do not take any D if you are intentionally sunning at locations, times, or seasons with lots of UV-B. Usually no D is taken during summer months. This may not be true if your skin is dark or you avoid sun or you live in the far north or far south. Testing will let you know your response. It is the only way to replete D safely.

If you find you are D resistant, your efforts to supplement or sun are not raising your D, or you have difficulty attaining or maintaining optimal levels you may have a genetic need for extra D or sunlight or you may be malabsorbing your D supplement.

If levels are high without supplementation, supplementation of D beyond the 800 IU level may contribute to D excess with ensuing deposits of calcium in soft tissues, loss of calcium in the urine and/or bone loss.

Hypervitaminosis D

Vitamin D is without question toxic in doses in excess for your genetic type. I use the word toxic to mean that short term or long term such levels will cause serious consequences in your body.

Taking D without testing can create disease. High levels of 25(OH)D are associated with bone loss and heart disease. Do not even consider this protocol if you are not willing to test frequently the first and second year and continue testing every six months the third and fourth years. By that time you should have a clear idea of your need for and tolerance of sunlight and vitamin D. If you wait to test and your dose, whether of D or sunlight and D combined, raises levels to excessive values it may take as long as a year to return your vitamin D to within normal range. Conversely if you don't test and take D you may find because of type of D taken or malabsorption you have not reached the optimal range.

Levels of D associated with bone loss are near the 'high optimal' values so the margin for error is not large. Optimal values are 40-60 ng/ml. Excess associated with bone loss may occur in some persons at values greater than 67 ng/ml (167 nmol/l).(11) Values associated with heart disease are greater than 75 ng/ml (187 nmol/l) in some persons.(12)

In unusual cases, D poisoning, relating to doses in millions of units, has caused death by rapid calcification of the kidney and heart.(13) There is no concern of that occurring with any protocol outlined in this paper.

There are NO immediate, obvious, symptoms of chronic excess 25(OH)D. TEST.

If Sunlight Or Supplements Of D Do Not Optimize 25(OH)D

In rare cases as much as 3,000 IU may be needed on a chronic basis or perhaps during winter months, low in UV-B. At my location in Northern California UV-B is 'practically' absent (would require excessive exposure times to make much D) from Sept-April.

Increasing your daily D dose, incrementally by 1,000 IU, is indicated only if two serum tests, minimum of 2 months apart, show that the current amount of sunlight or D is insufficient. Serum D may rise very slowly. **In a Nigerian study just the addition of calcium raised 25(OH)D to normal levels over a**

period of 4 months.(14) As long as your 25(OH)D continues to rise toward optimum you may want to stick with your current dose.

Each increase in D supplementation should be continued for 2-3 months and further increase considered only if no improvement is noted. Each new level of D supplementation should again be tested after 3 months.

D can build up slowly. As long as there is an increase of serum D at each testing there is no reason to increase the dose of vitamin D, even if the ideal has not yet been reached. Patience is important.

If with each increase there is no change in 25(OH)D please see below. Persistence is important as is chronic testing. D may rise slowly but then continue to rise beyond optimal levels to toxic values. Be patient, go slowly and always retest.

Malabsorption Of Supplemental D

I have seen incidences in which low serum 25(OH)D, followed by chronic supplementation as high as 4,000 IU daily, failed to move serum D. In some of these cases there was clear indication of altered gut mucosal integrity. Loren Cordain, Colorado State University, personal communication, suggests that wheat or lectin intolerance may damage the gut to the extent that D is not absorbed. Celiacs do not absorb vitamin D and this reverses when avoiding all gluten containing grains.(15;16)

Ask your physician to consider testing for gluten intolerance or consider removal of possible offending lectins (soy, wheat, rye, dairy, oats, legumes, barley, nightshades, depending on your genetics) for 4-8 weeks while continuing supplementation and retest.

If you are gluten intolerant you will not be able to absorb fat-soluble vitamins unless you completely avoid gluten (Also true of any other lectin to which you may demonstrate intolerance).

For more information on lectin intolerance http://krispin.com/lectin.html

A second cause for malabsorption of D and other fat-soluble vitamins may be caused by damage to the gut lining from pathogenic bacteria, yeast overgrowth, or parasites. Your physician can arrange testing and provide treatment to remove any discovered pathogens.

A third cause of malabsorption may be bile or pancreatic insufficiency.

If you

Have had your gallbladder removed
Are sensitive to fatty foods (they cause gastrointestinal distress)
Have been diagnosed with pancreatic insufficiency
Have been diagnosed with IBD or IBD

you will need the following protocol.

Fat Malabsorption Protocol

Bile, excreted into the upper intestine, emulsifies fats and fat soluble vitamins. Lecithin works like bile to emulsify fats. Taken with fat-soluble vitamins it allows emulsification and absorption even without sufficient bile.

With each dose of D consume **one** of the following (must be taken at exactly the same time you take the D or other fat-soluble vitamin)

Pick ONE:

1. One egg yolk (any style) With the white is fine.
2. 1 rounded teaspoon of lecithin granules (in mouth swallowed with water or juice or mix in water and juice, don't chew as they will stick to your teeth)
3. Ox bile or ox bile containing digestive aid per physician instruction

Cannot Tolerate Vitamin D

If it has been determined that you need D but when you take a supplement of D_3 your digestive tract indicates distress (gas, bloating, diarrhea) you may rub the D on your skin. It does have a 'fishy' smell so you may want to apply it before bed. This also works for repleting D in infants and children.

If you must use a dry vitamin D3 supplement make sure you take it with a fatty meal or fish fat. All vitamin D requires fat for absorption.

There is a form of D, emulsified or micellized vitamin D, that does not require fat. It is extremely dangerous to use as excess serum D may occur rapidly. Do not use this type of D unless you are frequently monitoring response and continue testing bi-monthly as long as you take it.

When using D topically you will not be able to get all of the oil out of the soft gel. Add one extra soft gel to your daily dose. You may put the D on your legs, feet, arms, face, wherever it absorbs and makes it easy for you. Do not cover the area for about an hour. You may then wipe off any excess. (Don't use soap for a few hours)

You may apply the D3, especially the Solgar D3 Cholecalciferol soft gels, which contains a small amount of vitamin A, to sun damaged skin, to areas with warts, moles or 'skin tags' or on other skin abnormalities. Some persons have reported improvement in their particular skin conditions when doing this.

Skin, Black And White And In-Between

Since darker skins need significantly more exposure to light to achieve the same amount of D in the blood, D deficiency is a more serious problem among persons of color. Blacks suffer from 70% more incidences of degenerative diseases uncluding cancers, diabetes and hypertension as compared to persons of Northern European ancestry. Latinos have a 50% higher incidence. It is possible that insufficiency of vitamin D plays a critical role in these numbers picture.

Remember, the darker your skin the more UV-B light you need to attain the equivalent 'optimal' level of 25(OH)D. The Safe Sun guidelines will provide adequate D for most persons, however, dark skins at higher latitudes will never be able to get sufficient D from sunlight alone. It is likely both sunlight and vitamin D will be needed to normalize 25(OH)D.

Bones, Calcium, Magnesium, Vitamins K and D

Bones require many nutrients for health and regeneration. The factors mentioned here are the few known factors. There are likely to be more interrelationships discovered over the next decade.

Vitamin D increases absorption of calcium. Magnesium increases bone density and hardness.(17-20) Vitamin K is necessary for making a protein that regulates deposition of calcium in bone and other hard tissues such as hair, nails, and cartilage, and helps keep it there.(21-24)

D does not maintain bone calcium. It regulates serum (blood) calcium. Giving D when calcium is insufficient may withdraw calcium from bone. Calcium and D without adequate vitamin K also contributes to deposition of calcium in soft

tissues. like arteries, rather than where it is truly needed, in our structural tissues.(25;26)

D_3 supplementation or sunlight should not be prescribed or undertaken without knowing calcium and vitamin K intake and supplementation should be initiated if intake of dietary calcium is below 800 - 1,200 mg daily and/or vitamin K intake is less than 1,000 mcg. (1 mg).

The American diet is deficient in vitamin K and unless regularly using live cultured yogurt, natto, and dark greens with butter or other fat, K should be added. Note that vitamin K is not potassium but a fat-soluble vitamin, usually sold in microgram amounts, that is responsible for GLA protein hydroxylation controlling calcium for bone health. (GLA protein not GLA fatty acid)

Complementary Prescriptions sells Vitamin K 1.5 mg (Complementary Prescriptions 1-888-401-1105 Product CP1091 use my PIN 230388 as referral http://cpmedical.net). Take one once a day with fat or fish oil. Doses of vitamin K higher than 1.5 mg may or may not be a good idea. Supplementing with less than 1 mg of vitamin K is unlikely to supply your needs.

Calcium supplements need to be balanced with adequate levels of D. I repeat for emphasis, Vitamin D regulates the level of ionized and free calcium and if these values are low elevating serum D may draw calcium from the bone.

Trace minerals including boron are also needed for healthy bones.(27-30) A well balanced diet of whole, fresh, foods are essential to realizing the benefits of adequate vitamin D.

Calcium and D_3 do not need to be taken at the same time. D_3 is usually taken once a day in the morning with fish oil or at the meal containing the most fat.

Calcium is absorbed more efficiently when taken 200-300 mg of calcium per dose. Taking a low dose calcium-magnesium three or four times a day is the best way to increase or maintain bone health..

500 mg. calcium-one dose	29% absorption
500 mg. calcium-two doses	36% absorption
500 mg. calcium-3 doses	40% absorption
2,000 mg- one dose	14% absorption

Heaney, RP et.al. J of Bone and Mineral Research, 5:11; 1990 p.1135-1137

A fun and easy way to take calcium and magnesium in smaller doses over the day is to use a children's chewable calcium-magnesium. Good choices are Nutrition Now Rhino Cal or Nature's Plus Nutri-Cal Hearts (my personal

favorite, vanilla flavor). Put out the number of chews needed to meet your daily goal and snack on them throughout the day.

Higher amounts of calcium may be needed with diagnosed bone loss. Total daily calcium as a supplement may range from 800 mg to 1,500 mg depending on current dietary calcium, bone status, and body size.

D sufficiency seems to reduce the need for calcium. Some research indicates 70% absorption with D sufficiency as compared with 20-40% with low D. You may find high intake of calcium causing symptoms as your vitamin D levels increase.

If tremor, tetany, 'charlie horse' or muscle tightness occurs when taking higher doses of D and calcium maintain D by serum testing and reduce your calcium dose and/or increase your intake of magnesium. In some persons, though rarely, magnesium supplementation may need to be equal to calcium intake.

Bones are made of calcium, magnesium and many trace minerals. The bone matrix is dependent on protein. Bones cannot be rebuilt without all of the necessary elements. Make sure adequate protein is consumed daily. (31;32) Eggs are an inexpensive and safe source of protein for most persons. They do not contribute to heart disease risks (33-35) unless they are fried and consumed with bacon and the like.

Expensive 'chelated' calcium is not necessary if D is adequate. With low D status 20% of calcium is absorbed, with optimal 25(OH)D 70%.(36)

The one exception, requiring acidified or chelated calcium, may be seniors or others with hydrochloric acid insufficiency. If this is found to be the case digestive enzymes, an increase in protein in the diet(24;37;38), and mineral chelates, or multi-minerals containing chelated calcium and magnesium with trace minerals, may rapidly enhance bone rebuilding and repair. Solgar, Now Foods, Carlson Labs, and Country Life offer multi-minerals chelates.

Taking calcium without sufficient D and vitamin K may cause other as yet unrecognized problems. Serum vitamin D controls the production of some calcium binding proteins and receptors critical to normal calcium utilization.

It is important to think of protein, calcium, magnesium, vitamin K and vitamin D as a team. While studies show that increasing calcium may slow bone loss even in the presence of less than optimal D there are other considerations. D-binding proteins and calcium-binding proteins are the cutting edge of biochemistry and molecular biology and we are headed into discovery of and understanding of the amazing relationship of man to light (or when light is absent or low, supplemental vitamin D).

As vitamin D regulates serum (blood) calcium, not bone calcium, vitamin K is equally important to keep calcium, once absorbed, in the bone and out of soft tissue.(26;39-41) The recommended daily intake of vitamin K is not less than 1 mg. (1,000 mcg.) daily.

Once optimal D is achieved and verified by testing, maintenance by daily intake of supplements and/or sunlight should be a lifelong practice. Remember to retest if you move to a different latitude or altitude or your situation changes dramatically.

An understanding of our relationship to sunlight and D is critical to health. Over the next few years more information will become available to validate this research. Take the time and spend the funds necessary to understand and put into practice your individual need for D and sunlight.

For long term health most persons in latitudes higher than 30 degrees, north or south, will require vitamin D supplementation equal to 1,000-3,000 IU daily much of or all of each year plus a multi-mineral providing 800-1,000 mg calcium 400-800 mg magnesium plus other minerals and trace minerals.

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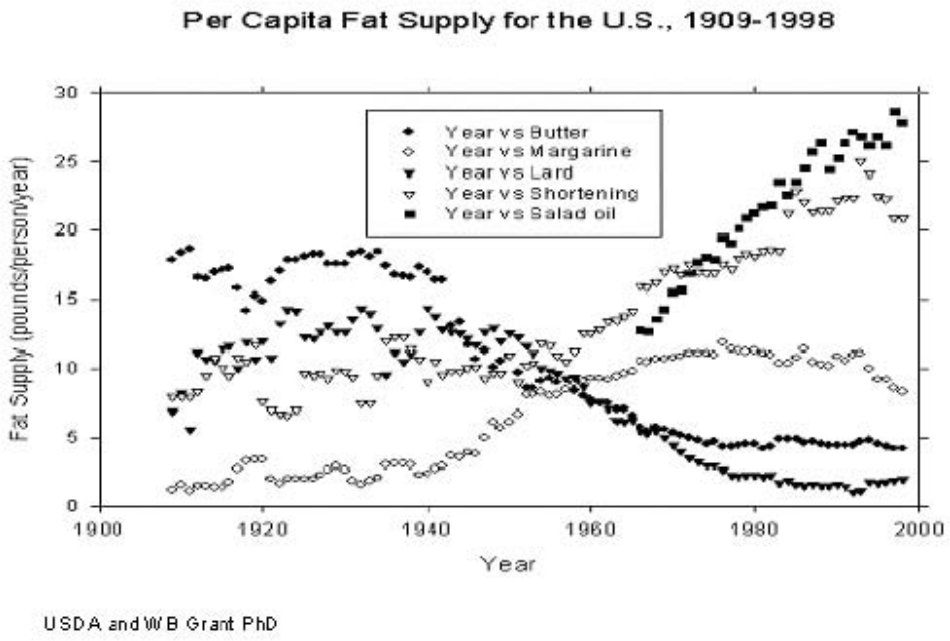
Omega-3 and the fat soluble vitamins A&D, E and K

When considering overall sunlight, vitamin D and calcium status, fatty acids must also be considered. Fatty acids change cellular responses to vitamin D and calcium. The types of fat in the diet also alter markers of inflammation including the skin's responses to sunlight. The amount of **omega-6**, which includes most nut and seed oils, in the American diet has increased dramatically over the last 100 years. During this same period of time intake of omega-3 fatty acids has significantly declined.



Decreasing intake of omega-6 fats is an important step to improvement of vitamin D status and general health. To do this you will need to **avoid all vegetable seed oils. Do not use sunflower, corn, soy, safflower, cottonseed, canola, or products that contain these oils. Avoid all hydrogenated and partially hydrogenated fats, all shortenings and all margarines (whether from the health food store or regular market).**

While omega-6 fats are essential they can easily and safely be consumed by a moderate intake of nuts or seeds. These foods are best, meaning freshest, purchased in the shell and added to foods or salads or eaten as desired. They may be eaten straight from the shell. Sprouting or light dry-roasting increases the digestibility of the proteins while retaining the stability of the fatty acids.

Acceptable oils, not omega-6, include extra virgin olive oil, fresh peanut oil (or non-hydrogenated peanut butter) and avocado oil. High oleic safflower oil, with a fatty acid profile similar to olive oil, is also acceptable. Natural (non-hydrogenated)

saturated fats are NOT a problem in D nutriture. Use butter, coconut oil, palm oil and non-hydrogenated lard as desired and as your waistline allows.

Excess omega-6 fatty acids promote inflammation. Inflammation is strongly associated with all degenerative diseases including osteoporotic bone loss, cancer, heart disease, allergies, and asthma.

Omega-3 fats combined with natural saturated fats are incorporated into your skin and protect the skin from UV damage. The long-chain omega-3 fatty acids DHA and EPA are anti-inflammatory and critical for skin, joint, brain, and nerve function. It seems that about 5-10 grams of fish fat containing 1,500-3,000 mg of EPA/DHA per day equates with positive benefits. 3 ounces of fatty fish, example- salmon, wild cooked, contains about 7 grams of fish fat of which 1.5 grams (1,500 mg) is combined EPA/DHA. This is without the skin or visible fat. Assuming our ancestors ate the skin and the fat under the skin, their consumption reached well beyond the amounts being suggested here

If you are not eating a significant portion of fatty fish, such as mackerel, sardines, or salmon a minimum of three times a week, including the skin and fat under the skin, you may need as many as 8-10 **fish oil** soft gels daily. Most brands of fish oil contain about 300-500 mg of combined DHA and EPA per 1,000 mg soft gel. Ideal intake of omega-3 is likely to be about 2,000-3,000 mg daily, 15, 000-20,000 mg a week. That is 2-3 grams daily, 15-20 grams a week of combined DHA/EPA (not total fish fat). It is likely some persons with coastal or island ancestors may need amounts exceeding this.

A good source of **omega-3 fish oil** is the Kirkland brand from Price-Costco; 300 soft gels for about $8. I consider it the best because it is affordable and very fresh due to the high product turnover rate at Costco. Costco also carries a enteric coated fish oil for those who find they burp repeatedly. It has less DHA/EPA per each so more pills will be needed and is somewhat more expensive.

Trader Joe's Trader Darwin Omega-3 or Now Foods Super EPA Double Strength are also excellent products with fewer soft gels needed per day (more EPA and DHA per each soft gel). All of these supplements are well made and due to high product turnover, very fresh. They have been tested to be free of heavy metals and contaminants.

If you 'repeat' your fish oil try keeping your soft gels in the freezer. This often solves the problem and keeps your fish oil fresher.

REVIEW: The recommended daily dose of essential long-chain omega-3 is about 2,000-3,000 mg. This number is the total of combined DHA and EPA.

What ever brand you use check the label carefully to determine how much DHA and EPA are in EACH capsule (not serving), add the numbers together to determine total EPA/DHA per each.

The Costco Kirkland brand contains 300 mg of combined DHA and EPA in each soft gel. A daily dose would be 5-10 soft gels. It sounds like a lot but translates into just

½-1 tablespoon of fish oil daily. You have the option of purchasing liquid fish oil (Carlson or Health from the Sun) and using 2-3 teaspoons per day but fish oil stays fresher in soft gels. Once your bottle is open you can keep it in the freezer to further extend its lifespan.

Fish oil may be taken with or without food. Many clients find that there is less 'repeat' when taken on an empty stomach. If taking fish oil with a meal take it first, then eat. Taken at the end of a meal you are guaranteed to burp fish oil.

As mentioned if your ancestors were coastal or island people (any coast, any island) you will likely need a higher dose. Depression, diabetes, obesity, hypertension, heart disease or any autoimmune disease would also indicate a need for higher levels of omega-3.

If you are over weight you may also need more fish oil, as much as 4,000-5,000 mg of combined DHA-EPA daily.

FISH OIL IS NOT COD LIVER OIL OR FISH LIVER OIL

It is possible to take fish liver oil, as a liquid, not in softgels, and get your daily omega-3 and vitamin A and vitamin D. The problem is relationships (see paper on Complex Systems)

If you use cod liver oil you need to make sure you get enough and not to much of each part you are looking for. If you live in a tropical location and add sun to cod liver oil the combination may result in an excess of vitamin D.

If your cod liver oil provides insufficient omega-3 you might be tempted to use more and take your vitamin A or D to extreme levels, not a good thing. In Finland, where there is little sun, cod liver oil intake seemed to increase bone loss. Depending on the brand it is possible that the product had too little vitamin D and too much vitamin A. There is a very complex relationship between these two vitamins that has yet to be studied. It is clear from the literature that when the level of A or D is normal or low and even small amounts of the other vitamin are consumed a 'relative' deficiency of the normal or low partner is created.

Normal or low vitamin D combined with 5,000 IU or more of vitamin A creates a relative D deficiency even though serum values of D appear within normal range.

Normal or low vitamin A combined with an as yet unknown level of vitamin D creates a relative A deficiency.

In animal studies animals were healthier when both A and D were low or A and D were high. In the case of D toxicity giving excess A can protect the body from damage while the levels of D are reduced.

Using fish liver oil may be extremely problematic. If you intend to use cod liver oil as a source of omega-3 proceed with caution and watch for symptoms of imbalance in either A or D. In this case testing serum D will not necessarily show you the problem, however testing will prevent you from moving into serious excess of D.

BALANCE IN ALL THINGS:

Sarcoidosis is a disease in which individual cells convert 25(OH)D into 1,25-dihydroxyvitamin D and this causes abnormal calcifications. Giving etretinate, an analog of vitamin A, has partially corrected this condition. It is likely giving vitamin A while reducing vitamin D or sun exposure might also alter the course of this disease. Tuberculosis, in some ways similar to sarcoidosis, is being successfully treated with vitamin A.(1-7)

Sunning increases the need for vitamin A but reduces the need for vitamin D. Depending on your genetics regular sunning may increase your need for vitamin A by 50% or more.

At the present time there is no accurate serum test for vitamin A. Serum A status remains normal or near normal until all body (tissue, liver) stores are depleted.

Vitamins K and E, the other fat soluble vitamins-

Fatty acids, especially omega-3, increase the need for vitamin K and vitamin E. Vitamin E needs increase to about 100 IU a day when using fish oil. Most multiples supply this or more so additional supplementation of E is not usually needed beyond the daily multiple. If you are not taking a multiple consider adding one now.

When taking fish oil the minimum dose of vitamin K should be 1 mg (1,000 mcg) daily. Source Naturals makes a 500 mcg vitamin K. Two small capsules taken once a day with your fish oil or fatty meal satisfy your requirements. Vitamin K 1.5 mg is also available from Complementary Prescriptions online at http://cpmedical.net Their product, containing 1.5 mg of mixed vitamin K_1 and K_2 is also taken once a day, just one small capsule. To order call 1-888-401-1105 and use my PIN referral 230288 and order product CP1091. The CP product is superior because it contains both forms of vitamin K and the correct daily dose in one capsule. Must be taken with some fat, fatty meal.

Vitamin K is fat-soluble so should always be taken with fish oil or a meal with significant fat.

Vitamin K is not potassium, which has the atomic symbol K. It is a vitamin found in liver , fatty fish, chard, kale and spinach. For the vitamin K in dark green leafy vegetables to be absorbed your greens must be eaten with FAT, and not an omega-6 fat to block D. So butter up those greens or use olive oil on your dark green salads. Vitamin K is also found in active culture yogurts and kefirs and in natto, a fermented soy product, and these foods may be a good source of K IF THE PRODUCTS CONTAIN FAT. Avoid fat free cultured products .

Choice of n-3, Monounsaturated and Trans Fatty Acid-Enriched Oils
for the Prevention of **Excessive Linoleic Acid Syndrome**
Harumi Okuyama, Ph.D.
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Excessive **linoleic acid (omega-6)** intake and relative n-3 deficiency syndrome
Animal experiments and epidemiological studies have revealed that excessive intake of **linoleic acid (omega-6) (LA, n-6) is a major risk factor for cancers of western type, allergic hyper-reactivity, coronary heart disease (CHD) and cerebrovascular disease (CVD) (1).** Although epidemiological studies performed in the USA failed to reveal a positive correlation between LA intake and breast **cancer** mortality, this is probably because the proximate marker for breast **cancer** is the proportion of n-6 eicosanoid precursors in phospholipids, which is saturated both in the high and low LA intake groups in the USA. Empirical equations presented by Lands indicate that both increasing the intake of n-3 fatty acids and decreasing that of n-6 fatty acids are necessary for effectively decreasing the n-6 eicosanoid precursors in phospholipids and thereby decreasing **cancer** mortality. On the other hand, high n-6/n-3 ratio but not hypercholesterolemia has been proved clinically to be a major risk factor for thrombotic diseases. Over-production of inflammatory lipid mediators of n-6 series has been shown to be a major cause for the rapid increase in allergic hyper-reactive patients in Japan.
President's Summary 1997 from the Japan Society for Lipid Nutrition
After discussion through several annual meetings of the Japan Society for Lipid Nutrition, Presidents Summary 1997 was published (in Japanese) as a review article (J. Lipid Nutr. 6:5-42, 1997), in which 20% as total fat energy was recommended for those with moderate physical activity. For healthy populations, saturated plus monounsaturated : n-6 : n-3 = 2.5 : ? 0.8 : ? 0.2 (n-6/n-3 ? 4) was recommended. For the primary and secondary prevention of those diseases described above, an n-6/n-3 ratio of 2 was recommended. The latter value was based on: 1) even the n-6/n-3 ratio of Danes was 3 in a well known epidemiology of Greenland natives; 2) the ratio of current Japanese is 4 but the incidence of **cancer**s of western type has been increasing rapidly, and the ratio of 4 or above cannot be recommended; 3) **animal experiments have shown the effectiveness of decreasing n-6/n-3 ratio to below 2 for the suppression of carcinogenesis and metastasis**; and 4) the safety of n-6/n-3 ratio of 1 has been established in animal experiments and in a retrospective study on hunters and gatherers foods.
In order to meet the recommendations described above, vegetable oils with n-6/n-3 ratios of 2 or below and those with very low n-6 fatty acid contents (e.g., high-oleic type) are useful. However, there was another criterion to be considered; the presence of minor components, which affect animal physiology seriously.

Survival time-shortening and renal injury induced by some vegetable oils and partially hydrogenated oils in SHRSP rats
Using soybean oil as a control, some oils were found to prolong the mean survival time of SHRSP rats by ca 10% (e.g., **DHA**-rich **fish oil**, perilla seed oil, flaxseed oil) while some others shortened it dose-dependently by ca 40% (double-low rapeseed oil, evening primose oil, high-oleate safflower oil, high-oleate sunflower oil, olive oil and partially hydrogenated rapeseed and soybean oil). When the rapeseed oil was lipase-treated, the resulting free fatty acid fraction was almost free of such activity, indicating that the survival-time shortening activity is due to minor components other than fatty acids in these oils. Free fatty acid fraction from partially-hydrogenated soybean oil exhibited a survival time between those of the original oil and soybean oil. It should be emphasized that lard, sesame oil and high-linoleate safflower oil were relatively safe for the SHRSP rats.

Those oils with survival-time shortening activity were found to cause renal injury; lesions in blood vessels, accelerated proteinuria, decreased platelet count and elevated gene expression for TGF?, fibronectin and renin.
Choice of n-3, monounsaturated and trans fatty acid-enriched oils
In order to decrease the n-6/n-3 ratio of our current foods to 2 or below, the intake of high-?-linolenate oils such as perilla seed oil and flaxseed oil as well as seafood and vegetables should be increased. **High-linoleate (omega-6) oils are inappropriate for human use as foods**. For deep-frying and preservation purpose, high-oleate vegetable oils are useful but all the high-oleate vegetable oils and hydrogenated vegetable oils we have examined so far exhibited the survival time-shortening activity, and I cannot recommend people to have these oils in large quantities. Instead, lard was safe for this animal model, and could be used in quantities not to induce obesity; animal fats as well as a high-LA vegetable oil intake caused insulin resistance in a NIDDM model of rats.
Reference
Okuyama, H., Kobayashi, T., and Watanabe, S. (1997) Dietary fatty acids ñ The n-6/n-3 balance and chronic, elderly diseases. Excess **linoleic acid (omega-6)** and relative n-3 deficiency syndrome seen in Japan. Prog. Lipid Res. 35: 409-457.

The points made here

Excess Omega-6 promotes inflammation, **cancer**, heart disease and allergy/**asthma**
Omega-6 must be dramatically reduced. Eliminate all obvious sources
Omega-3 is protective
Omega-3 is not necessarily available from flax, only 1 in 6 Americans can do the conversion.
Omega-3 is available from fish but daily intake is a problem due to fish toxicity; that is heavy metals, especially mercury, chemicals, pesticides, etc.
Fats are incorporated into skin- you are what you eat- a combination of **omega-3** fats and natural saturated fats including coconut oil, butter and non-hydrogenated lard (read labels) offer the **best protection** from disease, including skin **cancer**.
Omega-6 in excess promotes inflammation of tissue so that the skin is more susceptible to damage from UV light. Incorporation of omega-6 into the linings of the lungs, gut, breast, prostate, and bone increase susceptibility to carcinogens, mutagens, allergens and toxins. Fish oil, all parts of the fish oil , DHA, EPA, other omega-3 fatty acids and even the cholesterol it contains, protect these same cells.
HDL cholesterol in its own right has powerful antioxidant activity.

HDL has long been known as the “good kind of cholesterol,” protecting against heart disease and atherosclerosis. HDL has powerful antioxidant properties, similar to vitamin C, vitamin E, and coenzyme Q-10. An HDL associated enzyme, lecithin-cholesterol acyltransferase , which forms part of HDL, is a powerful antioxidant enzyme that blocks the oxidization of LDL cholesterol. Cholesterol is beneficial and without harm if it is not first oxidized.
Vohl MC, Neville TA, Kumarathasan R, Braschi S, Sparks DL: A novel lecithin-cholesterol acyltransferase antioxidant activity prevents the formation of oxidized lipids during lipoprotein oxidation. Biochemistry; 1999 May 11;38(19):5976-81

The cause of fat oxidation is simple, exposure to oxygen or light or heat. Fats stay fresh if they start out fresh and are kept away from heat, light and oxygen. Processed fats, all types, are exposed to these very damaging agents.

Saturated fats are the most stable to heat, light and oxygen. Do not reuse after heating. Do not expose to direct sunlight. Cover and refrigerate. Butter keepers, which submerge butter in water, also work to keep butter fresh and soft at the same time without refrigeration. Use natural saturated fats within 6 months.

Monounsatureated fats are somewhat stable. Most monounsaturate fats can be kept in a closed container for up to one year. If you purchase smaller containers and keep them in a dark pantry no longer than 6-12 months finishing a bottle within 1-2 months of opening little rancidity is likely. You may reserve and purchase your yearly supply from growers online.

Polyunsaturated fats are very unstable. The more polyunsaturated the fat the more rapidly oxidizable. (Throw out your vegetable oils. It is difficult to tell if they are fresh or not. Nuts and seeds both protect the fats from oxidation and easily demonstrate rancidity. You can tell when a nut is 'bad'.)

Fish oil omega-3 fats are protected by the other fats contained in fish oil. However, they are inherently unstable and should be kept in the cold as much as possible. Freezing is an excellent way to preserve fish fats until use.

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Update on Essential Fats

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Terms Used

Omega-6: Any of the fatty acids in the omega-6 family, short or long-chain. Also designated as n-6. These fats are polyunsaturates with multiple double bonds beginning on carbon 6.

Linoleic Acid: Short-chain omega-6 fatty acid also designated by LA. Can be converted into arachidonic acid.

Arachidonic acid: Long-chain essential omega-6 designated AA

Docosapentanoic Acid: Long-chain omega-6 designated DPA (displaces DHA in the brain- not a good thing)

Omega-3: Any of the fatty acids in the omega-3 family, short or long-chain. Also designated by n-3. These fats are polyunsaturates with multiple double bonds beginning on carbon 3.

Linolenic Acid: Short-chain omega-3 fatty acid designated by ALA or LNA or alpha linolenic acid. Can be converted into EPA and DHA.

Eicosapentanoic Acid: Long-chain essential omega-3 designated EPA

Docosahexanoic Acid: Long-chain essential omega-3 designated DHA. The most important fatty acid for brain function.

Monounsaturated Fatty Acids: Fats containing one double bond on carbon 9. Designated omega-9 or n-9 or oleic acid.

Saturated fats: Fats with no double bonds. They can be short or long-chained. They are ubiquitous (in all life). Several are critically important to health and may be anti-viral and anti-bacterial. Natural saturated fats are stable to heat and light and safe for higher heat cooking. They do not oxidize readily.

Trans-fats: Trans-fatty acids are created when seed and grain oils are processed such as in the making of margarine and the hydrogenated fats used in most processed foods. Trans-fats also occur in vegetable and grain oils when they are heated.

Seed and grain oils: Corn, soy, safflower, sunflower, sesame, cottonseed, canola, walnut, peanut, flax,

Fruit oils: Olive, avocado.

Essential Fats for Humans

Fatty acids play critical roles in human health and disease. Cell membranes (all cells) are composed of a double layer of fats. Your brain is about 60% fat. The fats you eat strongly influence the ability of your cell membranes and your brain to function.

Cholesterol is an alcohol, not a fat. Natural saturated fats are found in all foods to some degree. Saturation of a fat may be natural or from processing as is done to make margarine solid. Naturally occurring saturated fats are not associated with disease unless they are imbalanced by inadequate intake of the polyunsaturate essential fatty acids, especially the omega-3 fats.

Linoleic Acid is an omega-6 that is 20 (or more) times too high in the American diet and strongly implicated in degenerative diseases.

Linolenic A cid is a short chain o mega-3 foun d in perilla, canol a a nd flax con sidered by some to be important as a source of long chain omega-3 DHA and EPA but many

Americans suffer from poor conversion, an en zyme insufficien cy, or impai red gen etic ability to elongate the fatty acid to its active EPA and DHA forms.

Arachidonic Acid is fou nd in meat and fish and e ggs and dairy and made in o ur bodies from linoleic acid. It is critical for the g rowth of th e body and brain and i mmune function but needed in very small amounts.

EPA, eicosapentanoic acid and DHA, docosahexanoic acid , are found in fi sh, grass-fed beef and po ultry and wild game. Some may be mad e from Linole nic Acid, dep ending on your genetics, your liver function and other as yet unknown factors.

Omega-3 (Fish Oil) Fatty Acid Update

Omega-3 fatty acids, DHA and EPA, are essential to brain and nerve function. In cell membranes they enhance cell response (to insulin, neurotransmitters and other messengers), and facilitate repair when cells are damaged. Omega-6 fats contribute to membrane resistance, altering mood, insulin response, learning and cell repair in a negative way.

There is a chart located on the last page of this report showing the list of fat types and other charts showing fat types in foods. You may use this as a reference guide.

Some anthropologists believe the human brain would not have developed as it did without access to high levels of DHA found in fish and shellfish and to a lesser degree in wild game. Just two generations of high omega-6 and low omega-3 can lead to profound alterations in brain size and brain function in animals and probably in man. Other anthropologists believe that the human brain formed as it is today, large in proportion to body size, and that its capacity is being diminished as the diet becomes deficient in omega-3 fats. A relative omega-3 fat deficiency can be created by an overabundance of omega-6 fats, a lack of omega-3 fats, alcohol consumption or the consumption of trans-fats as has been increasingly occurring in the US for the past 50 years.

Breast milk contains DHA and EPA, equivalent to amounts present in the mother's diet (what mom eats is critical). Formula contains no omega-3. Raising children on formula or mother's milk deficient in omega-3 fats contributes to impaired visual development, poor spatial development, slower learning, decreased comprehension and early allergies and asthma.

Recent research has shown that it is critically important and the amount of **omega-6 fats** in the American diet is dramatically reduced and **omega-3 fats** increased. I have suggested the use of butter, coconut and olive oil for many years and this recommendation continues. What needs to happen now is that omega-6 fats are intentionally identified and avoided as much as is possible. Omega-3 fats need to be sought out and dramatically increased.

To do this you will need to **avoid all vegetable seed oils. Do not use sunflower, corn, soy, safflower, canola, or products that contain these oils. That is no hydrogenated or partially hydrogenated fats, no margarine, no vegetable oil, no shortening.**

Acceptable oils are extra virgin olive oil and avocado oil in moderate quantities. High oleic (omega-9) safflower or sunflower oil is acceptable in minimal quantity. Natural (non-hydrogenated) saturated fats are NOT a problem. Use butter, coconut oil, palm oil and non-hydrogenated lard as desired, also in moderate amounts.

Fats in natural foods are never composed of a single type of fatty acid. Coconut oil has polyunsaturated fats and olive oil has saturated fat. In nature fats are always mixed. Even fish oil contains saturated fat and cholesterol.

Do not worry about naturally occurring cholesterol in fish, eggs or lean meats. Do not worry about total fat intake as long as it is from actual food, whole-fat milk, real butter, the fat on fish, poultry,

meat. The change to concentrate on is reducing ADDED FAT, eliminating as much omega-6 added fat as possible. Do your best to avoid all obvious omega-6 fats. If you suffer from elevated cholesterol it is even more important that you increase your omega-3 fats and avoid omega-6. Keep your total fat grams, from food and added, to about 66 grams.

Between 1903 and 1998 ADDED FATS rose from 34 pounds per person per year, mostly butter and lard, to more than 66 pounds per person per year. All of the fat increase was in the form of omega-6 as salad dressing, margarine, shortening and hydrogenated fat added to processed foods and candies. See the chart that follows.

The ratio of omega-6 to omega-3 in the U.S. diet is somewhere between 25-50:1. The ideal ratio is somewhere between 4-1:1.

To correct this imbalance you will need to severely restrict omega-6 fats and add fatty fish daily making sure to eat the skin and fat under the skin. Remember, the fish must not be cooked in an omega-6 fat, vegetable oil or margarine, nor dipped in mayonnaise, also an omega-6.

Supplementing Omega-3

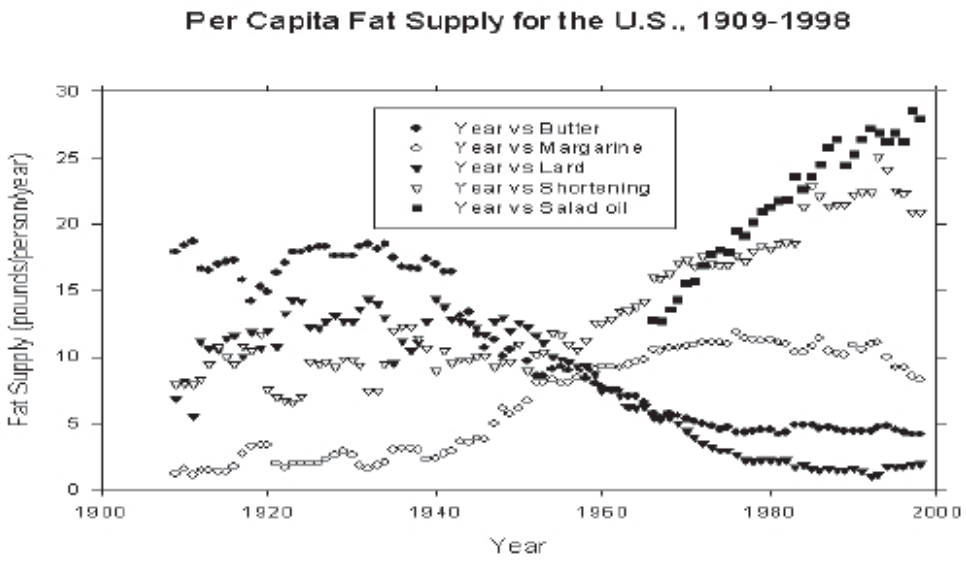
Supplemental Dose: If you are not eating fatty fish such as mackerel, sardines, salmon, daily you will need to use fish oil supplements to equal 10-30 grams of fish fat per day (equivalent to about 1-3 tablespoons of fish oil). The beneficial (active) dose is one 1,000 mg fish oil concentrate, each gel containing 180 EPA and 120 DHA, for each 10 pounds of actual body weight, for both children and adults. 180 pounds equals 18 gels, 150 pounds 15 gels. If using the Trader Joe Omega-3, slightly stronger, use 1 for each 15 pounds of body weight. Fish oil can be taken with or without food. Many clients find that there is less reflux when taken on an empty stomach. Do not take all of your fish oil at once. Split it up into two or three doses. Do not take any after 5PM, as fish oil can be very energizing.

The best source of omega-3 fish oil I have found is the Kirkland brand from Price-Costco. It is the freshest (due to high product turnover), good quality and a great price, 300 soft gels for $7.39. Trader Joe's also has a good buy. Their supplement is slightly stronger so fewer soft gels are needed.

Omega-3 and 6 fats move into cell membranes, particularly the membranes of epithelial cells-cells that compose skin, the lining of the arteries and lungs, the linings of the ducts in the breast and testes. They also are components of nerve and brain cells. The preferential fat for these cell membranes is omega-3 but n-3 be replaced by n-6, if n-3 is not available. Cells where omega-3 is displaced by omega-6 are less able to repair themselves and the DNA may be altered.

The brain and nerves so need omega-3 that they will rob it from every other cell to maintain optimum brain levels. Some of the brain and nerve conditions associated with high omega-6 and low omega-3 include, alcoholism, depression, manic depression, memory loss, impaired night vision, anxiety, insomnia, dementia, Alzheimer's, Parkinson's, ADD, ADHD, dyslexia, stress induced hostility and schizophrenia.

In the following graph of US per capita fat consumption 1909-1998 it is clear that both our overall intake of fats and intake of omega-6 fats has dramatically increased. <u>There is no indication our intake of natural saturated fats has increased over this period of time.</u>



USDA and W B Grant PhD

Added Fats and Oils U.S. Pounds Per Capita Intake 1909-1998

From the USDA	1909	1945 1970	1980 1998			
Butter In 1909 butter and cream was from grass-fed cows.	17.9	11.7	5.4 4.5 4.2			Butter from grass-fed cows is high in omega-3
Lard In 1909 natural lard, not hydrogenated, contained poly fats including omega-3	6.9	12.2	4.5 2.3 2.0			Most is now hydrogenated or from grain-fed (omega-6) pigs
Margarine 1.2		3.9	10.8	11.3	**8.3**	All Omega-6 and trans-fats
Shortening, Crisco, added hydrogenated and partially hydrogenated fats. (In 1909 shortening contained less omega-6. It had coconut or palm oil, safer)	8.0	10.0 17.3			**20.9**	All Omega-6 and trans-fats
Salad Oils Most Omega-6, often rancid, containing trans-fats.	N/A N/A		15.5	21.3	**27.9**	In 1998 a small amount was omega-9 olive oil (<0.5 lb)
Edible Tallow	N/A	N/A N/A			3.2	
Total Pounds of Added Fat Per Capita	**34**	37.8 53.5			**66.5**	Almost double by 1998

The following paper was presented in 2000 at the National Institute of Health is Washington, DC. The BOLD text is my emphasis. Keywords in understanding fats- Linoleate is omega-6, linolenate is omega-3 as is DHA and EPA and oleic is omega-9. Linoleate, DHA, EPA and linolenate are all polyunsaturated fats. Oleic omega-9 is monounsaturated fat.

Choice of n-3, Monounsaturated and Trans-fatty Acid-Enriched Oils for the Prevention of Excessive Linoleic Acid Syndrome

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Mizuhoku, Nagoya 467-8603, Japan

Excessive **linoleic acid (omega-6)** intake and relative n-3 deficiency syndrome

Animal experiments and epidemiological studies have revealed that **excessive intake of linoleic acid (omega-6) (LA, n-6) is a major risk factor for cancers of western type, allergic hyper-reactivity, coronary heart disease (CHD) and cerebrovascular disease (CVD)** (1). Although epidemiological studies performed in the USA failed to reveal a positive correlation between LA intake and breast cancer mortality, this is probably because the proximate marker for breast cancer is the proportion of n-6 eicosanoid precursors in phospholipids, which is saturated both in the high and low LA intake groups in the USA. Empirical equations presented by Lands indicate that both **increasing the intake of n-3 fatty acids and decreasing that of n-6 fatty acids are necessary for effectively decreasing the n-6 eicosanoid precursors in phospholipids and thereby decreasing cancer mortality.** On the other hand, high n-6/n-3 ratio but not hypercholesterolemia has been proved clinically to be a major risk factor for thrombotic diseases. **Over-production of inflammatory lipid mediators of n-6 series has been shown to be a major cause for the rapid increase in allergic hyper-reactive patients in Japan**.

President's Summary 1997 from the Japan Society for Lipid Nutrition

After discussion through several annual meetings of the Japan Society for Lipid Nutrition, Presidents Summary 1997 was published (in Japanese) as a review article (J. Lipid Nutr. 6:5-42, 1997), in which 20% as total fat energy was recommended for those with moderate physical activity. For healthy populations, saturated plus monounsaturated : n-6 : n-3 = 2.5 : 0.8 : 0.2 (n-6/n-3 4) was recommended.

For the primary and secondary prevention of those diseases described above, an n-6/n-3 ratio of 2 was recommended. The latter value was based on: 1) even the n-6/n-3 ratio of Danes was 3 in a well known epidemiology of Greenland natives; 2) the ratio of current Japanese is 4 but the incidence of **cancer**s of western type has been increasing rapidly, and the ratio of 4 or above cannot be recommended; 3) **animal experiments have shown the effectiveness of decreasing n-6/n-3 ratio to below 2 for the suppression of carcinogenesis and metastasis**; and 4) the safety of n-6/n-3 ratio of 1 has been established in animal experiments and in a retrospective study on hunters and gatherers foods.

In order to meet the recommendations described above, vegetable oils with n-6/n-3 ratios of 2 or below and those with very low n-6 fatty acid contents (e.g., high-oleic type) are useful. However, there was another criterion to be considered; the presence of minor components, which affect animal physiology seriously.

Survival time-shortening and renal injury induced by some vegetable oils and partially hydrogenated oils in SHRSP rats

Using soybean oil as a control, some oils were found to **prolong the mean survival time** of SHRSP rats by ca 10% (e.g., **DHA-rich fish oil, perilla seed oil, flaxseed oil**) while some others shortened it dose-dependently by ca 40% (double-low rapeseed oil, evening primrose oil, high-oleate safflower oil, high-oleate sunflower oil, olive oil and partially hydrogenated rapeseed and soybean oil). When the rapeseed oil was lipase-treated, the resulting free fatty acid fraction was almost free of such activity, indicating that the survival-time shortening activity is due to minor components other than fatty acids in these oils. Free fatty acid fraction from partially-

hydrogenated soybean oil exhibited a survival time between those of the original oil and soybean oil. It should be emphasized that **lard and sesame oil were relatively safe** for the SHRSP rats. **Those oils with survival-time shortening activity were found to cause renal injury; lesions in blood vessels, accelerated proteinuria, decreased platelet count and elevated gene expression for TGF?, fibronectin and renin.**

Choice of n-3, monounsaturated and trans-fatty acid-enriched oils

In order to decrease the n-6/n-3 ratio of our current foods to 2 or below, the intake of high-n3 linolenate oils such as perilla seed oil and flaxseed oil as well as seafood and vegetables should be increased. **High-linoleate (omega-6) oils are inappropriate for human use as foods**. For deep-frying and preservation purpose, high-oleate vegetable oils are useful but all the high-oleate vegetable oils and hydrogenated vegetable oils we have examined so far exhibited the survival time-shortening activity, and I cannot recommend people to have these oils in large quantities. **Instead, lard was safe for this animal model, and could be used in quantities not to induce obesity;** animal fats as well as a high-LA vegetable oil intake caused insulin resistance in a NIDDM model of rats.

Reference

Okuyama, H., Kobayashi, T., and Watanabe, S. (1997) Dietary fatty acids ñ The n-6/n-3 balance and chronic, elderly diseases. **Excess linoleic acid (omega-6) and relative n-3 deficiency syndrome seen in Japan.** Prog. Lipid Res. 35: 409-457.

Key Facts Regarding n-3 and n-6

Omega-6 fats in amounts more than essential promote **cancer**, **heart disease, autoimmune disorders including arthritis, diabetes and allergy/asthma.**

Omega-6 fats must be dramatically reduced. Eliminate all obvious sources.

Omega-3 fats are protective and may prevent or reverse the listed conditions.

Omega-3 is not necessarily available from flax or perilla oil.

Omega-3 is available from fish but daily intake may be a problem due to fish toxicity from chemicals, pesticides, etc.

Fats are incorporated into skin- you are what you eat- a combination of **omega-3** fats and natural saturated fats including coconut oil, butter and lard offer the **best protection** from disease, including skin **cancer.**

Omega-6 fats, in high amounts, promote inflammation of tissue so that the skin, linings of the lungs, gut, breast, prostate, and bone are more susceptible to damage from UV light, carcinogens, mutagens, allergens and toxins

Omega-3 fats including all parts of the fish oil , both DHA and EPA and even the cholesterol it contains protect these same cells.

HDL cholesterol in its own right has powerful antioxidant activity.

Omega-6 decreases and omega-3 increases HDL cholesterol.

Omega-3 DHA is the major fat in the eye and is replaced (if available) every 10 days. Increasing omega-3 with fish oil improves night vision and color vision.

Omega-3 fats are associated with the ability to smell and for seniors who have lost their sense of smell fish oil may restore it in 1-2 months.

Omega-3 fats improve memory. Just two days after increasing omega-3 the phosphatidylserine content of the brain is dramatically increased.

HDL has long been known as the good cholesterol, protecting against heart disease and atherosclerosis. It was recently discovered that HDL has powerful antioxidant properties similar to vitamin C, vitamin E, and coenzyme Q-10. An HDL associated enzyme, lecithin-cholesterol acyltransferase , which forms part of HDL, is a powerful antioxidant enzyme that blocks the oxidization of LDL cholesterol. Cholesterol is beneficial and without harm if it is not first oxidized. Vohl MC, Neville TA, Kumarathasan R, Braschi S, Sparks DL: A novel lecithin-cholesterol acyltransferase antioxidant activity prevents the formation of oxidized lipids during lipoprotein oxidation. Biochemistry; 1999 May 11;38(19):5976-81

Oleic acid is an omega-9 such as found in olive oil and avocado oil. Some sunflower and safflower oils are bred to be high in this fat, safe for cooking and mayonnaise. Look for the words High Oleic on the bottle and a high number for omega-9 on the label.

Essential Fatty Acids Explained

Many people find it difficult to believe that fat can be essential to your health, but it's true. Fatty acids are the "building blocks" of fat. Some of these fats are called "essential" because your body needs them, yet cannot make them; you must eat them.

Essential fatty acids (EFAs) are all polyunsaturated fats. The two types of essential fatty acids are omega-3 fatty acids and omega-6 fatty acids. These come in short and long chain configurations. The short-chain omega-3 is alpha-linolenic acid (LNA or ALA). Its elongated (made longer) derivatives include: eicosapentaeonic acid (EPA), docosahexaenoic acid (DHA) and a few others, less well known and less studied.

The main short-chain omega-6 is linoleic acid (LA). Like LNA, it also has elongated derivatives, the main being arachidonic acid, necessary for prostaglandin formation and brain function.

The very long-chain omega-3 EFAs are connected with brain and visual development in infants. Deficiencies in adults can lead to impaired mental processes, including learning disorders, dementias and other neuronal diseases, impaired vision, and depression. Studies suggest that prolonged deficiencies might lead to retinal and macular damage. In pregnant women low levels of the elongated omega-3 can actually reduce brain size in offspring.

How much of these essential fatty acids do you need? There is no RDA yet, but many sources agree that Americans do not get nearly enough omega-3. Even worse we get too much omega-6, which displaces omega-3 in cell membranes and neural circuits.

Researchers involved in the Workshop on the Essentiality of and Recommended Dietary Intakes for Omega-6 and Omega-3 Fatty Acids suggest "adequate intakes" of each:

- ☐ omega-3: 0.65 grams (g) of EPA and DHA combined (with neither falling below 0.22 g)
- ☐ omega-6: 4.44 g

The problem with these numbers is that a diet containing nuts and seeds or meat or even milk and eggs as a protein source has at least 12 grams of omega-6. That would mean that a minimum (not optimal) of omega-3 would be 1.95 grams which is the amount in about 6 ounces of fatty fish daily (more would be needed if lean fish is used) however, even that does not work if you are eating farmed fish as farmed fish is high in omega-3 but equally high in omega-6, canceling the much of the benefit to balance ratios.

Sources looking at the dietary ratio of omega-6 to omega-3 fatty acids suggest that in early human history the ratio was about 1:1. Currently most Americans eat a dietary ratio that falls between 20:1 and 50:1. The optimal ratio is most likely between 4:1 and 1:1. For most Americans this means greatly reducing the omega-6 fatty acids they consume and increasing the amount of omega-3 fatty acids.

Please note that this has nothing to do with rancid fats, trans-fats or hydrogenated fats. These damaged fats are not good for you but neither are the 'cold pressed' so called healthy vegetable oils like canola, soy, safflower, sunflower or corn oils. Even flax has a significant amount of omega-6 and should be avoided. If you are vegan or vegetarian use perilla oil instead and avoid so called 'essential fat' supplements that contain omega-6 and omega-9 in addition to omega-3. You can get all you need of n6 and n9 from food.

Omega-3 Fatty Acids

The best source of omega-3 fatty acids is cold-water fish, which is high in both EPA and DHA. You may need as little as three servings a week of very fatty fish (make sure to eat the skin and surrounding fat) or 8 or more ounces daily to meet your genetic need. If fish are not your thing you can use fish oil capsules. A standard fish oil concentrate soft gel weighing 1000 mg contains about 300 milligrams of omega-3. The suggested dose is one soft gel for each 10 pounds of body weight.

Important: Fish oils produce free radicals in your body. You must be sure to get enough antioxidants, particularly vitamin E and vitamin K, when you increase your fish or fish oil intake. See info at the end of this article.

You may be tempted to eat foods or use supplements that contain LNA (linolenic acid- short chain omega-3) rather than EPA and DHA from fish or fish oil (particularly if you're a vegetarian), but you should be aware that it may not be very useful to you, as many of us convert it to EPA and DHA very inefficiently. This impaired conversion is further reduced in aging. In many studies even large doses of ALA (flax or perilla) did not raise membrane DHA. Flaxseed, perilla oil and walnuts are common sources of LNA. Recent studies suggest high amounts of ALA may be associated with prostate cancer.

You may try adding perilla oil (lower in omega-6) and buying the 'algae' DHA such as Neuromins from Solgar or Nature's Way, which contain 100 mg of DHA per each soft gel. To match the fish

oil program you would need 3 or more tablespoons of perilla oil daily plus about 5-15 per day of the Neuromins providing 500-1,500 mg of DHA.

A sampling of foods high in DHA and EPA (combined):

Food	g in 100-gram serving 100 grams =3.5 ounces	g in normal-sized serving
Sardine oil	20.79	2.83 (1 tablespoon)
Cod liver oil	17.87	2.43 (1 tablespoon)
Herring oil	10.48	1.43 (1 tablespoon)
Salmon, Atlantic (farmed)***	2.15	3.89 (half fillet)
Mackerel, Pacific and jack***	1.85	3.25 (1 fillet)
Pickled herring	1.39	.42 (2 pieces)
Salmon, chinook***	1.74	2.68 (half fillet)
Salmon, pink***	1.28	1.6 (half fillet)
Mackerel, Atlantic***	1.20	1.07 (1 fillet)
Rainbow trout (farmed)***	1.15	.82 (1 fillet)
Bluefish***	.99	1.16 (1 fillet)
Sardines, canned in oil	.98	.90 (1 can, 92 g)
White tuna, canned in water	.86	.73 (3 oz, 85 g)
***cooked with dry heat		

Omega-6 Fatty Acids

Most of us get our omega-6 fatty acids from vegetable oils including foods made from vegetable oils, like margarine, Crisco and mayonnaise. The popular evening primrose and borage

supplements are also high in omega-6. As you can see from the chart below, simply changing the type of oil you use could greatly reduce your intake of LA. Also pay special attention to the difference between wild and farmed salmon as to omega-6 content. This pattern of higher omega-6 in farmed animals is repeated in wild compared to domestic game and grass-fed compared to grain-fed beef, poultry and pork. Organic dairy, meat and poultry are products from animals fed grain so omega-6 is still high.

Food	**g in 100-gram serving (3.5 ounces)**	**g in normal-sized serving**
Sunflower oil, linoleic (60% and over)	65.70	8.94 (1 tablespoon)
Corn oil	58.00	7.89 (1 tablespoon)
Sunflower oil, linoleic (less than 60%)	39.80	5.41 (1 tablespoon)
Sunflower seeds, oil roasted	37.82	25.53 (half cup)
Sunflower oil, linoleic (hydrogenated)	35.30	4.80 (1 tablespoon)
Sunflower seeds, dry roasted	32.78	20.98 (half cup)
Canola oil	20.30	2.84 (1 tablespoon)
Peanuts	15.56	11.36 (half cup)
Safflower oil	14.35	1.95 (1 tablespoon)
Almonds, unblanched	12.21	8.67 (half cup)
Flax Seed	22.636	2.693 (1 tablespoon)
Pumpkin seeds	8.76	2.80 (half cup)
Hamburger 0.590		
Chicken Breast with skin, boneless	1.480	1.382(half breast)
Egg	1.36	0.686 (per egg)

Wild Salmon	**0.562**	**0.866 (half filet)**
Farmed Salmon	**1.939**	**3.451 (half filet)**
Butter (from grain-fed cows)	1.830	0.260 (1 tablespoon)
Cheese, cheddar white	0.577	0.164 (1 slice)
Milk, full fat	0.075	0.183 (1 cup)
Olive oil	7.90	1.07 (1 tablespoon)

Vitamin E and K, Anti-oxidants Extraordinaire, Needed to Protect Omega-3

A typical breakfast in Tokyo, Japan contains natto, fish, rice and pickles. Natto is high in vitamin K. The fish contains vitamin K, vitamin D, omega-3, minerals and amino acids. The pickle improves digestion. In all traditional cultures the daily diet contains protein, dark greens and some fermented food from natto to yogurt to fermented cabbage. This wide variety assures adequate nutrition from fresh whole foods. Without this natural balance nutritional deficiencies are inevitable.

Vitamin K is a fat-soluble vitamin found in dark green leafy foods and naturally fermented foods. The symbol K is used for potassium on the periodic chart. We are not talking about potassium here, but a vitamin. Vitamin K1 is phylloquinone, found in plants. Vitamin K2, menaquinone, is found in animals and made in the human gut by 'good' bacteria. Antibiotics destroy our ability to make K in the gut. Probiotics like acidophilus restore gut K production. There is some evidence that natural gut production is not enough to support artery and bone health. Dietary sources are critical.

Vitamin K reverses postmenopausal bone loss by keeping calcium in the bone where it belongs, working better than Fosamax. Low levels of vitamin K lead to under-carboxylated calcium forming plaque in the arteries in heart disease. K is necessary for the formation of osteocalcin a bone builder. It is intimately related to the functions and actions of vitamin D. Vitamin K is an effective anti-oxidant in the cell membrane and necessary for normal blood clotting.

Large doses of the naturally occurring K1 and k2, up to 45 mg daily, have been given with few side effects. High (1-5 mg) doses of vitamin K found in dark green leafy vegetables, seaweeds and animal livers, do not 'over' coagulate the blood. Adequate vitamin K normalizes fragile membranes. It has proven useful preventing or correcting easy bruising, varicose veins and 'spider' veins. Vitamin K within physiological amounts does not make blood thicker or 'stickier'. K toxicity has occurred in infants given vitamin K3 (not a natural form of K) by injection. In research only the analog 'man-made' K3 has shown toxicity.

If you are on a blood thinning medication, such as Coumadin or aspirin or other non-steroidal anti-inflammatory, you must discuss K supplementation with your physician.

Research demonstrates a high intake of fat, whether omega-3, omega-6, omega-9 or saturated fat, increases the need for vitamins E and K. In animals given diets high in omega-3, omega-6, omega-9 or saturated fat liver content of vitamin K was reduced to 1/5 of controls (normal chow diet). The tendency of blood to coagulate more slowly or a reduction in blood platelets is often a side effect of using omega-3 fatty acids and may occur when eating a diet high in fatty fish. Normalizing levels of vitamin K with high vitamin K foods or a vitamin K supplement reverses both of these tendencies.

In the US vitamin E supplementation is common. Most health food store multiples contain 200-400 iu per daily dose. Higher levels of E have a reverse effect. In fact, high levels of E actually reduce levels of vitamin K, not a good idea for postmenopausal women needing to keep or rebuild bone or heart patients using omega-3 and vitamin K to prevent or reverse arteriosclerosis.

Finding vitamin K is much harder, whether in foods or in a supplement. The DRI is 90 mcg. but just 1 mg (1,000 mcg, more than 10 times the DRI) has reversed bone loss in post-menopausal women, reducing urinary calcium loss by 25% within a few days of starting supplementation. 10 mg (10,000 mcg) of vitamin K daily has been used by our space program to prevent bone loss in astronauts during weightlessness. A study published April 2001 in Kardiologie correlated low levels of vitamin K to under-carboxylated MGP (a protein that reflect artery health). The conclusion? Low levels of K allow calcium to leave the bone, not be delivered to the bone and promote the deposition of calcium in soft tissues.

Vitamin K works **with** vitamin D (both are equally important) to prevent bone loss and build new bone. It also influences blood sugar levels and adult onset diabetes. Low vitamin K contributes to post meal hyperinsulinemia (high insulin) and insulin resistance.

We do make some vitamin K in our guts if we have normal bowel flora (the 'good' gut bugs, they make the K for us) and normal bowel function. Constipation, diarrhea, IBS or Crohn's would all indicate a problem with vitamin K, either making it or absorbing it. Dietary fiber (soluble fiber) helps the 'good' gut bacteria thrive so soluble fibers, found in high fiber foods like berries, figs and some legumes, will increase vitamin K if the gut bugs are right.

Vitamin K is found in very dark green leafy vegetables like chard, spinach, bok choy and seaweeds such as dulse. It is also found in cod liver oil, beef and poultry livers. To be absorbed it must be consumed with fat. Fat-free veggies for dinner, no K absorbed, but with a little added butter or olive oil on your greens the K is very well absorbed. Wrapping sushi in seaweed gives a meal high in vitamin K and omega-3, both well absorbed. This rule regarding the need for fats to absorb is also true of 'green drinks'. Unless you add fat to your drink or have some with it (not later) the important fat-soluble nutrients will pass you right by.

Vitamins A, D and E also need fat for absorption. Since they are usually found in a fat containing supplement (cod liver oil) or in fats found in animal or poultry livers, butter, full-fat dairy and such, absorption is usually not a problem. Our modified 'low-fat' or 'no fat' diets are un-natural and rob us of the very important fat-soluble vitamins, A, D, E and K. Absorption of calcium, A and D from non-fat milk is poor.

If you have difficulty digesting fats it is important to address this problem ASAP. Longevity requires adequate A, D, E and K and without good fat digestion you will not get what you need, supplements or from food.

Essential and non-essential fats carry these vitamins and alter them for cellular function. Using a water based or 'dry' fat-soluble won't alter the way fat-soluble vitamins act once they are absorbed. You still need the good fats and must be able to digest them to get them 'in'.

If you have had your gallbladder removed you no longer make concentrated bile. You will need to take triple strength lecithin, about 3-4 soft gels, or a concentrated fat-digesting enzyme (lipase) or

concentrated pancreatin or ox bile at every meal, especially meals containing your fat-soluble vitamins and omega-3 oils.

If you have difficulty digesting fats but still have your gallbladder using the lecithin or lipase or pancreatin for a few weeks while getting the fat-soluble vitamins and omega-3 fats will most likely correct the problem. Use the digestive aids temporarily and see how you improve.

Now Foods has a good triple strength lecithin and a strong Pancreatin. Solgar makes a Vegetarian Digestive Aid high in lipase and Kal makes a chewable digestive 'gum' also high in lipase. Use as many as you need to emulsify the fats. You can tell because the fats won't reflux and your digestion will proceed normally.

Who Needs Extra Vitamin K?

anyone taking the suggested amounts of omega-3
anyone on a high fatty fish diet
anyone with osteoporosis or beginning bone loss (vitamin K works as well or better than Fosamax)
anyone with diabetes
anyone with IBS, celiac or Crohn's
anyone with heart disease or a family history of heart disease
post-men opausal women

A better choice Vitamin K 1.5 mg is available from Complementary Prescriptions online at http://cpmedical.net Their product, containing 1.5 mg of mixed vitamin K_1 and K_2 is taken once a day, just one small capsule. To order call 1-888-401-1105 and use my PIN referral 230288 and make sure to order product CP1091 or use my PIN online. The CP product is superior because it contains both natural forms of vitamin K and the correct daily dose in one capsule. Must be taken with some fat, fatty meal.

Other vitamin K supplements include 100 mcg. tablets from Solgar or Solaray- you need 10 per day or Now Foods Alfalfa 650 mg 10 grain, 6 per day (1,108 mcg K) both containing the less active K1..

A 10 milligram supplement of vitamin K, called Super Vitamin K, is available from:

LIFE EXTENSION VITAMINS 1229 SOLANO AVE. ALBANY CA. 94706
PHONE: 1-510-527-3005 TOLL FREE NUMBER: 1-888-771-3905 Orders can also be faxed to: 1-510-527-0902 or order online from http://www.iherb.com

The dose is too high to use as a daily supplement but you could take one Super Vitamin K twice a week.

Super Vitamin K is a potent, oil based K source. If you have varicose veins or spider veins, scars or stretch marks that have not responded to other treatments you can use this K topically as well as orally for quicker response. Vitamin K is fat-soluble and will absorb nicely through the skin.

Remember to take your fat-soluble vitamins, A, D, E and K, with the meal highest in fat or with your omega-3 dose. While the DRI for vitamin K is 90 micrograms (0.090 milligrams) there is no UL (upper limit of safety) and up to 45 mg have been given daily to treat osteoporosis. 5 mg. of vitamin K is still a physiologic amount, meaning you could get this much from food if using dark greens and naturally fermented products but has not shown better health benefits than 1 mg.

Call with questions and concerns.

From NIH Conference 2000 Essential Fats- Avoid high Short 6 and get Long 3

Use only oils in bold. Use Olive, Butter and Coconut for cooking.

Food Oils in descending rank			18-carbon		20- & 22-carbon	
by short 3/short 6 ratio	Serving size	kcals	Short 6	Short 3	Long 6	Long 3
Oil, perilla	**1 tablespoon**	**120**	**1680**	**8960**	**0**	**0**
Oil, flaxseed	1 tablespoon	120	2240	7980	0	0
Fish oil, herring	**1 tablespoon**	**123**	**156**	**417**	**39**	**1509**
Fish oil, salmon	**1 tablespoon**	**123**	**210**	**525**	**92**	**4657**
Fish oil, sardine	**1 tablespoon**	**123**	**274**	**592**	**239**	**3096**
Fish oil, cod liver	**1 tablespoon**	**123**	**127**	**254**	**127**	**2557**
Fish oil, menhaden	**1 tablespoon**	**123**	**293**	**575**	**159**	**3624**
Butter oil, anhydrous	**1 tablespoon**	**112**	**288**	**185**	**0**	**0**
Oil, canola	1 tablespoon	124	2842	1302	0	0
Oil, mustard	1 tablespoon	124	2146	826	0	0
Oil, walnut	1 tablespoon	120	7194	1414	0	0
Oil, soybean	1 tablespoon	120	6936	925	0	0
Oil, soybean lecithin	1 tablespoon	104	5465	698	0	0
Oil, wheat germ	1 tablespoon	120	7453	938	0	0
Shortening, household, lard + vegetable oil	1 tablespoon	115	1242	141	0	0
Oil, olive	**1 tablespoon**	**119**	**1067**	**81**	**0**	**0**
Oil, soybean, (hydrogenated)	1 tablespoon	120	4746	354	0	0
Oil, sheanut	**1 tablespoon**	**120**	**666**	**41**	**0**	**0**
Mayonnaise, soybean and safflower oil	1 tablespoon	99	7176	414	0	0
Shortening, industrial, lard + vegetable oil	1 tablespoon	115	2317	128	0	0
Oil, sunflower, > 70% oleic	**1 tablespoon**	**124**	**505**	**27**	**0**	**0**
Oil, rice bran	1 tablespoon	120	4542	218	0	0
Margarine-butter blend	1 tablespoon	102	2162	103	0	0
Oil, cocoa butter	**1 tablespoon**	**120**	**381**	**14**	**0**	**0**
Oil, sunflower, linoleic, (hydrogenated)	1 tablespoon	120	4801	122	0	0
Oil, palm	**1 tablespoon**	**120**	**1238**	**27**	**0**	**0**
Oil, corn	1 tablespoon	120	7888	95	0	0
Oil, sesame	1 tablespoon	120	5617	41	0	0
Oil, sunflower, linoleic < 60%	1 tablespoon	120	5413	27	0	0
Oil, cottonseed	1 tablespoon	120	7004	27	14	0
Oil, grapeseed	1 tablespoon	120	9466	14	0	0
Oil, peanut	1 tablespoon	119	4320	0	0	0
Oil, coconut	**1 tablespoon**	**117**	**245**	**0**	**0**	**0**
Salad dressing, cottonseed, oil	1 tablespoon	88	5068	0	0	0
Oil, sunflower, linoleic >60%	1 tablespoon	120	8935	0	0	0
Oil, safflower, linoleic >70%	1 tablespoon	120	10149	0	0	0
Oil, safflower, oleic > 70%	**1 tablespoon**	**120**	**1952**	**0**	**0**	**0**
Oil, palm kernel	**1 tablespoon**	**117**	**218**	**0**	**0**	**0**

Omega-3 and Fat-soluble Vitamins Protocol

General Use All

Omega-3 fish oil concentrate (remember to avoid omega-6 fats) to equal approximately 180 EPA and 120 DHA (total omega-3 300 mg.) per 10 pounds of body weight. If using Costco Fish Oil Concentrate a 150-pound woman would use about 15 soft gels, 7 or 8 twice a day. If using the Trader Joe's Trader Darwin Omega-3 it is one per each 15 pounds of body weight so 10 (5 twice a day) for 150 pounds. If using Now Foods SuperEPA it is one per each 20 pounds of body weight. **The dose is key.** Taking more than you need is not necessary. Taking less than the suggested dose won't do the job. Take on an empty stomach (but with fatty vitamins). Do not take after 5 PM

After the first 1-3 months reduce your dose to 300 mg combined DHA and EPA for each 20 pounds of body weight. This is maintenance. Increase if you feel better on the higher dose.

Vitamin K If using **Super Vitamin K** take one every other day or one three times a week such as Mon-Wed-Fri. If using **Source Natural's 500 mcg K** take 2 per day all at once with some type of fat or if using **Complementary Prescriptions Vitamin K 1.5 mg** one per day. RE osteoporosis Vitamin K works better than Fosamax without the side effects. Get into this change as soon as possible. Fosamax damages the digestive tract and that does not help with absorption of D and calcium. Take with your first daily dose of fish oil or with a very high fat meal.

Some clients have been given the mineral potassium when they asked for vitamin K. K is the atomic symbol (remember chemistry?) for potassium but there is also a fat soluble vitamin K which is what you want.

Best: Complementary Prescriptions Vitamin K 1.5 mg Product #CP1109 1-888-401-1105 Register as my client and use my PIN 230288. Dose is one a day with a fatty meal.

OK: Source Natural's Vitamin K 500 mcg. Two pills once a day with first dose of fish oil.

Too much K for daily use but will work: Life Extension Super Vitamin K 10 mg. (this is 10,000 mcg) Call 1-888-771-3905 to order (free shipping) or order online from http://www.iherb.com (pay shipping) One bottle has 90 soft gels, a six-month supply for most clients. Do not take one a day.

You may also use the Super Vitamin K supplement topically for spider veins, bruising, scars and varicose veins. Pierce the cap and use directly on your skin. Rub in and allow to absorb. Use 2-3 times a week. If using over a large area use the formula at the end of this report.

Osteoporosis (and all post-menopausal women)

Same as General Use especially note the vitamin K/Fosamax information. Plus

Solgar Vitamin D3 Cholecalciferol 1,000 IU soft gels extracted from fish liver oil (which also contains some vitamin A, see back of bottle)
Take according to response on your vitamin D blood test. Make sure to retake the test after 3-6 months to make sure your dose is right. Your dose may be as few as none and as many as 4 per day. Take with your first daily dose of fish oil.

Heart Disease

Same as for osteoporosis. Make sure to test your D and retest. Too much or too little D contributes to heart disease.

Allergy and Asthma, Includes IBS and Crohn's

Same as for osteoporosis. D is critical.

Impaired Memory, Depression, ADD, ADHD, Dementia

You may or may not need the D. Be SURE to test. Otherwise the program is the same as for osteoporosis.

Cancer

Check with me. You need this protocol but it is adjusted, amounts and times, throughout treatment. Optimal omega-3 enhances the effects of chemotherapy, radiation and helps prevent metastasis after surgery.

Problems with digestion and absorption of fats-

If you don't digest fats well or have had your gall bladder removed or eat a low fat diet you will be deficient in omega-3, and vitamins beta carotene, A, D, E and K. You will also absorb calcium poorly and use it even less well.

Fat is critical to health. The key is WHAT FAT and HOW MUCH. Natural fats as found in foods should not be avoided. Use whole milk cheese and yogurt if you tolerate dairy. Eat the skin of poultry and fish. DO NOT AVOID NATURAL FATS.

Use butter or non-hydrogenated coconut oil, from your health food store, for cooking. Also use extra virgin olive oil for cooking and salads.

If you have developed an inability to digest fats, but have your gallbladder, use a lipase enzyme or ox bile or pancreatin or triple strength lecithin for several weeks with meals and when taking the fish oil and D and K.

If you have lost your gallbladder you no longer produce concentrated bile and need to ALWAYS take one of the above with every meal and with your fish oil and K and D. If you do not do this you will rapidly become even more deficient in the fat-soluble vitamins and essential omega-3.

Conditions associated with poor fat digestion and absorption-

Celiac-Sprue (gluten intolerance)
Cro hn's Disease
Diab etes
IBS Irritable Bowel Syndrome
All of the sclerosing diseases
Gallbladder disease

Pancreatitis
Liver disease
Low fat diets
Osteo porosis

The supplements to look for follow. **Pick one** and try it till you find one that clearly relieves your digestion and that you are comfortable taking. Unless you have no gallbladder, stop after a few weeks to a month to see if your digestion has improved. If you take one of these and experience no changes in your digestion you either don't need a supplement or it is the wrong one. Digestive aids work immediately, at the meal they are taken. They don't build up. They don't store. There is no reason to buy a large bottle until you know you need them, they are one's that work for you and you know you will need them long term.

Now Foods Triple Strength Lecithin (must be triple strength or you would need to take 9-12)
Has some omega-6 fat in it. The dose is 3-4 per meal and with fatty supplements, that is the fish oil and K and D. (for fat digestion only)

Kal-N-Zyme® Gum 250 Pieces
Chew 2-3 after meal or fatty supplements (mostly for fat digestion)

Now Foods Pancreatin or Bile Salts
2-3 with meal and with fatty supplements (for fats, proteins and carbohydrates)

Now Foods Super Enzymes
As on the label but also with fatty supplements (for fats, proteins and carbohydrates)

Twinlab Super Enzymes
As on the label but also with fatty supplements (for fats, proteins and carbohydrates)

Solgar Vegetarian Digestive Aid
As on the label but also with fatty supplements

REMEMBER- you will not absorb and utilize beta-carotene, A, D, E or K if not consumed with FAT. Calcium is absorbed and utilized with FAT. Do not expect to raise calcium levels on a low fat diet.

Digestive aids do any of the following:
Lipase- digests fat
Protease- digests proteins
Amylase- digests carbohydrates
Lecithin- emulsifies fat
Ox bile- emulsifies fat
Betaine HCl- 'cooks' the food in acid. Only necessary if you know you have low acid. Not a good idea if you do not need it. Vinegar or lemon juice can be used with a meal to improve digestion if acid is low.
Pancreatin- a pancreas extract that contains lipase, protease and amylase

The amount you need depends on your insufficiency.

Vitamin K Topical Treatment-bruises, varicose veins, spider veins

Use the capsule directly for full strength, small areas. OR

1 10 mg Life Extension Super Vitamin K soft gel squeezed into
1 teaspoon (approximate) warmed coconut oil mix well
Optional:
1 10,000 iu A soft gel squeezed
1 1,000 iu D soft gel squeezed
1 400 iu d'alpha E soft gel squeezed
1 OptiZinc capsule (opened)

Pinch of ascorbic acid (you may grind the zinc and ascorbic acid finer with a mortar before adding)

Mix well and store in closed container in the refrigerator. All except the vitamin K and coconut are optional and any or all may be in or out. Do not make more than will be used in 3 days. The coconut oil will harden in the fridge. Just scrape out the amount you need and let it soften in your hand. This is not a replacement for your C-Spray. The C-Spray is anti-aging and a sun protector. The K is for spider veins, varicose veins, bruising and scars.

Every Body Needs Vitamin A, Prelim Update 3/2008

Research regarding the human need for vitamin A and vitamin D from the embryo to adult shows both A and D have genomic functions. Both function at the level of your 'genes' as 'messengers/hormones' telling cells to divide, grow, mature, and die. Throughout the life of a cell vitamin A, thyroid, vitamin D and other hormonal regulators, are necessary for health and longevity. Though they are called vitamins, both A and D are actually the raw materials from which the active hormones, retinoic acid (from vitamin A) and calcitriol (from vitamin D), are made.

Of all the hormones necessary for life and health only two require precursors that must be gotten every day from our environment , vitamin A and vitamin D.

Lack of vitamin A at ANY age results in growth failure, of few or many cells including brain and nerve cells.(1-5) This is important because as we age it had been thought vitamin A was not an important contributor to the maintenance of the human nervous system as it is in infancy. New research is proving that assumption untrue.

Recent studies show low levels of vitamin A alter brain function and thereby affect mood, memory, and sleep. Low levels of vitamin A are implicated in production of plaque in the brain. (6-11) Diets devoid of vitamin A produced plaque in the brains of rats mimicking the plaque found in Alzheimer's disease. When the rats were repleted with vitamin A the plaque regressed.

Over the past 50 years, Alzheimer's, autism, and learning disabilities, including ADD and ADHD, have exponentially increased in the US population. When increases of a particular condition occur over a broad population it is important to look for broad changes. Since the 70's we have been encouraged to reduce fat and reduce intake of animal products. Low fat milk is the norm, we eat less butter, and sales of non-fat or low fat salad dressing, yogurt, cheese and snack and cereal foods continue to climb. Fat is blamed for obesity, heart disease, and cancer, BUT the data does not hold up under scrutiny.

Natural fats found in meat, dairy, fowl and fish contain varying amounts of the fat soluble vitamins A and D as well as vitamins E and K. Organ meats, kidney, brain, heart, sweetbreads (thymus), contain more concentrated amounts of important nutrients than the other edible parts of meat, fish and fowl. Kidney and liver are excellent sources of vitamin A, iron, trace minerals and several B vitamins. Fish fat and skin, the part we often throw away, are excellent sources of natural vitamin D and some vitamin A. Prior to the 1960s liver once week was standard in most healthy homes. Prior to the promotion of refined, processed and fortified foods health advisors promoted regular intake of organ meats and fish once or twice a week.

Introduction of refined and processed foods produced a general decline in health in the US which is being repeated throughout the world. Food in the 40's and up until the mid-50's still consisted primarily of whole real foods produced locally, available in season, and always fresh. The invention of the refrigerator and freezer and the canning and packaging industries allowed the growth of packaged, processed, no longer whole or fresh, foods.

In 1937 the US had a population of 129.8 million. 2 million owned a refrigerator.

1937 more than 2 million Americans owned refrigerators.
1939 refrigerator with one section for frozen food and a second for chilled food, introduced by G. E.
1946 Mass production of modern refrigerators starts after World War II.
1947 GE two-door refrigerator-freezer combination
1955 80% of American homes now have refrigerators

2005 A domestic refrigerator is present in 99.5% of American homes

Many persons do not realize the short life of foods. Vitamin A, whether retinol or beta-carotene, declines rapidly in all foods as does folic acid, other B vitamins and vitamin C. Even refrigeration will not stop this process. Foods transported long distances also lose nutrients, even if they are 'organic'.

Whole foods, including naturally occurring animal organs, skin, and FAT, have historically provided nutrients to sustain life and health. Gravy made from fat drippings will contain vitamin D and A when made fresh using 'real' ingredients. Stews and soups prepared from a stock using marrow bones (or other whole bones) will contain vitamins A, D and K as well as other important immune and growth factors.

Until the 1950s grains and soy were not used to fatten meat for market as cost precluded such 'luxury'. All poultry were 'free range' and small farms the standard. Meat and fowl contained naturally the nutrients to sustain life or the animal would suffer and not survive. Now antibiotics can keep livestock alive in spite of poor health. Until quite recently fish were still 'natural' but with current 'fish farming' practices the omega-3 content, taurine content, and vitamin content of farmed fish are significantly lower than their wild caught relatives.

"The first self-propelled grain combines (machines that harvest grain) were introduced during World War II and they enabled U.S. farmers to produce substantially more grain than the nation's population could consume. So the farmers started feeding it to their livestock to add value to their livestock production, not as an exception, but as the rule. Feedlots were invented and that created a whole new form of livestock production. (The first commercial feedlot in Texas was started in 1950!) Consequently the supply of grain-fed cattle soared. By 1955 it had shot up to 7.4 billion pounds. It reached 22.8 billion pounds in 2004 while the non-fed beef supply fell from 5.7 billion pounds in 1955 to 3.6 billion pounds today…

In the late 1950s the poultry industry started moving the chickens out of the pastures, where they had grazed while being fed grain, into huge buildings where they were fed only grain. Now instead of taking three months or more to raise a fryer, it takes seven weeks. During the 1960s the dairy industry evolved away from the small 25-cow, pastured-based dairies to huge operations with 1,000 or more cows housed in feedlot conditions being fed grain and alfalfa. Milk production per cow soared. By the 1970s the pork industry followed suit by moving sows indoors where they were fed grain and turned into living machines that mass produced more live pigs than ever before. The piglets are weaned at 30 days and are fed more grain so they grow exceptionally fast to produce more pork than ever before imagined from a sow."
From http://www.texasgrassfedbeef.com/id91.htm
Author: Ted Shanker http://slankersgrassfedmeats.com

Today to get the same quality of food your parents, grandparents, and great grandparents consumed requires finding small local producers and the cost may be prohibitive for many Americans.

Modern animal husbandry means artificial supplements, crowded conditions, growth stimulants, pesticides, and other modern miracles of production. While these methods provide large quantities of cheap food the quality is relatively poor, contaminated, or even potentially toxic. Beef, pork and poultry may be given excessive doses of vitamin D or vitamin A or even caused to have a deficiency of certain nutrients because this manipulation of diet produces some perceived benefit to the seller (not the buyer). Beef fed a vitamin A deficient diet have more marbling of the muscle (and less vitamin A in the liver, fat and meat) when sent to market.

Vitamin A is a name given to numerous retinol metabolites as well as beta-carotene. Vitamin A deficiency is one of the oldest recorded medical conditions. From its early history vitamin A has

been associated with night blindness, the first known mention of treatment by eating animal liver some 3500 years ago in Egypt, and the condition and treatment also reported by Hippocrates.

Until some few years ago infants were routinely given cod liver oil or concentrated 'drops' of vitamins A and D during the first year of life. This was done to ensure growth as lack of vitamin D produces rickets, with profound deformation of bones and increased susceptibility to infection, and lack of A will produce failure to gain weight, increased susceptibility to infection, and at its most extreme, eye lesions and blindness.(12;13)

Some evidence suggests low vitamin A status may contribute to SIDS (Sudden Infant Death Syndrome).(14)

In 1921 J. B. Bloch wrote:

"From that moment (December 21, 1917) everyone ate butter instead of margarine and since then there has been no xerophthalmia in Denmark. It is impossible with certainty to throw any light on the extent to which change of diet in 1918 affected the two other conditions associated with xerophthalmia in young children, vis.: dystrophy and the reduced power of resistance to infection."

Currently vitamin A is considered toxic, lowered or eliminated from most supplements including some fish liver oils, and warnings to avoid during pregnancy are everywhere. The problem is between low fat diets, avoidance of poultry, dairy, and meat fats and little consumption of liver the chance of being vitamin A sufficient is declining.

Further, the blatant recent promotion of high doses of vitamin D will contribute to profound vitamin A insufficiency within the next few years.

In 2000 I finished a book, Naked at Noon, Understanding the Importance of Sunlight and Vitamin D. The journey of research and writing changed my views on public health, researchers, the supplement industry and the nature of healthcare and nutrition dramatically. I found myself struggling to put a frame of reference to the information being given to the 'general public', referred to in the book as the GP.

Nutritional science has repeatedly failed to understand the synergism between varying nutritional elements.

There is a sense we have some complete body of evidence regarding what is 'healthy'. We most certainly do not. YOUR health depends on your life style, your genetics, your inherent 'special needs', your diet, your exercise, and your exposure to pathogens and toxins.

What I learned while researching vitamin D was that we all need it and we must get it from sunlight or diet (cold water fish fat, cod liver oil, animal fat naturally containing vitamin D). We respond individually to both sunlight and diet. Some easily produce all the D they need from sunlight exposure, others in a similar location with similar exposure will make MUCH less D in their skin. Some may have the potential to make lots of D but live in a latitude where the sunlight lacks enough UV-B to produce adequate vitamin D. There are other alterations.

Some persons may take 400 IU of vitamin D and get UV-B sunlight in moderate amounts and have optimal values of storage vitamin D, serum 25(OH)D, a marker of vitamin D status that is useful in monitoring ones response to sunlight and/or vitamin D supplementation. Others may take a supplement of 4,000 IU of D daily and not change serum D at all, because the supplement they are using is not absorbed, either because of the type of supplement or malabsorption caused by taking the supplement without adequate fat to stimulate bile, or bile insufficiency, gut pathogens or celiac(gluten intolerance) or Crohn's disease. It is also possible some just don't absorb oral vitamin D well due to genetic differences in gut binding sites or gut binding proteins.

The easy part is that you can test your serum D and tell if you need more or less sun or supplement.

Vitamin D and vitamin A become, after absorption and conversion, hormones, a part of the Steroid/Retinoid/Thyroid Hormone Superfamily. Representative receptors (places where hormones bind and deliver their messages) and hormones belonging to this gene superfamily include the DNA binding and regulatory proteins and steroid hormones estradiol (E2 receptor, ER), cortisol (CORT receptor, GR), androgen (ANDR receptor, AR), progesterone (PROG receptor, PR), and aldosterone (ALDO receptor, MR), the nonsteroid hormones triiodothyronine (T3 receptor, T3R) and dihydroxyvitamin D3 (D3 receptor, VDR), and two classes of retinoid (all-trans retinoic acid and 9-cis retinoic acid) receptors (RARs and RXRs respectively).These hormones together regulate DNA transcription.

hor·mone
Function: noun
1 a : a product of living cells that circulates in body fluids (as blood) or sap and produces a specific often stimulatory effect on the activity of cells usually remote from its point of origin -- called also internal secretion –Merriam Webster Online

tran·scrip·tion
Function: noun
: the process of constructing a messenger RNA molecule using a DNA molecule as a template with resulting transfer of genetic information to the messenger RNA Merriam Webster Online

Hormones tell our cells what to do and how to do it. Instructions include divide (make new), grow (mature), and die. Variations include how many and how often. If a cell continues to reproduce and doesn't die it is a malignant cell (cancer). If a cell makes the wrong kind (different type of cell, bone instead of cartilage) it is called metaplasia. If a cell makes an impaired (abnormal, damaged) copy of itself it is a dysplastic cell. If the cell makes too many of itself it is hyperplastic (benign prostatic hyperplasia). Vitamins A and D, as hormones, play crucial roles in determining how a cell develops and when it dies.

Hormones control growth. It bears repeating, hormones control cell growth in all cells at all stages of life. Looking at the preceding list of Steroid/Retinoid/Thyroid Superfamily members there are only two hormones we must get daily from our environment.

From Forbes, D is for Debate, Robert Langreth, 2/28/08, regarding vitamin D (both emphasis is mine)-

…a vocal band of researchers are touting a far larger role for this once obscure vitamin. They cite a flurry of intriguing, if preliminary, epidemiological and lab studies hinting that vitamin D may play a role in staving off a wide range of diseases, including colon cancer, infections, multiple sclerosis and other autoimmune ailments and possibly even heart disease.

"Up until now we looked at vitamin D the way we look at an iceberg. Eighty-five percent of its function has been hidden, and ***we had no idea until two or three years ago****," says an excited Robert Heaney, an endocrinologist at Creighton University in Omaha. "The field has just exploded." Adds Medical University of South Carolina biochemist Bruce Hollis: "I often say its skeletal effects are the least interesting thing we know about it."*

Whatever is true for vitamin D is doubly true for vitamin A. We have little understanding of the genomic functions of either of these vitamin/hormones. We cannot make the genomic vitamin A and vitamin D without the precursor molecules that MUST come from outside ourselves. Every other hormone in the human body is made from elements within the body. ONLY A and D are taken directly from the environment from sunlight and food.

So, for growth of new cells (longevity, rejuvenation) we need high quality protein, high quality fats, vitamin A and vitamin D. If these elements are missing serious disease and/or death ensues. If these elements are in short supply conditions ranging from night blindness to chronic infections to acne, rickets to osteoporosis to cancer may occur. Both A and D are critical to normal cell development which is lacking in cancer, autoimmune and degenerative diseases.

Moreover, both vitamin-hormones, A and D, must be supplied in sufficient but not excessive amounts. Excess of either element also causes abnormal cell development leading to disease and degeneration.(15-26)

AND to push the limit on balance, both must be supplied in harmonious amounts which so far research has yet to determine. All ideas of balance at this moment must be guesses AND must be individual because we are not the same.

In numerous studies A without D or D without A causes harm (including abnormal, uncontrolled growth or failure of growth of varying kinds). So normal D with elevated A or normal A with elevated D, lack of both A and D and excess A and D may be damaging.

Symptoms of an imbalance of A and D often mimic the symptoms of a deficiency of the undersupplied nutrient. So taking lots of vitamin A would contribute to conditions associated with deficient vitamin D even if your serum vitamin D remained within normal range, conversely if you take lots of D or get lots of sun without A or with marginal A you will see increased skin problems, including skin cancers, suppression of the immune system, increased infections, poor night vision, and other symptoms related to too little A. (and serum retinol will not reflect tissue- skin, liver, organs, levels of vitamin A).

Beta-carotene is not A (retinol) and many people do not convert beta-carotene into active A sufficient to maintaining adequate vitamin A tissue stores.(27-29) Both beta-carotene and vitamin A (retinol) require fat to be absorbed (see article at end of chapter) and beta-carotene needs specific enzymes to split the molecule to release vitamin A. Low fat diets, salads with non-fat or low fat dressing, bile insufficiency, all lower the ability to absorb beta-carotene and genetics determines if you easily convert beta-carotene to vitamin A.

Studies have shown that humans vary greatly in their ability to convert beta-carotene to retinol.(28;29)

Pre-formed vitamin A as retinol is found in all animal, poultry and fish flesh and fat in small amounts, and in animal, fish and poultry livers in higher concentrations. As mentioned at the beginning of this chapter the earliest ‘prescribed vitamin supplement’ is known to be liver and somewhat later cod liver oil.

While there is a blood test for vitamin D, 25(OH)D, the test for serum retinol is not valid for insufficiency. Vitamin A is stored in the human liver and in fat cells. Serum vitamin A will not drop until most body stores are depleted and by that time much damage will have already occurred.(30-32) Clinicians are currently considering using a determination of ‘night blindness’ as a way of determining vitamin A sufficiency.(32) But eye symptoms rarely occur before liver stores are dangerously low.

Vitamin A is depleted with every infection be it bacterial or viral and whether the infection is respiratory, systemic, or in your gut.(33-36) Each infection lowers your store of vitamin A further unless you increase your supply.

Zinc alters both conversion of beta-carotene to vitamin A and the effectiveness of vitamin A in maintaining healthy cells.(37-44)

Vegetarians may have a high intake of beta-carotene but be deficient in vitamin A because without zinc, which is low in non-meat/fish/poultry diets, the abundant beta-carotene is inefficiently converted to vitamin A.(45)

Also a consideration in status of both vitamin A and vitamin D are the prevalent fats in the diet. The consideration of the 'why' of fats and A and D needs more research but in general omega-6 fatty acids impair functioning of A and D, whether at the level of absorption, conversion, storage or binding to DNA.(46-51)

Low protein alters the immune system and vitamin A and D status. Retinol binding protein carries vitamin A from storage to tissue and organ. If protein, zinc or vitamin A are less than optimal this system will function poorly.(45;52-54)

The tenants of my 'nutritional theories' have included (to this point, Feb. 2008):

1. The human body is composed primarily of water, protein and fat.
2. Cells are constantly being born and dying at all ages and stages of life.
3. To make new cells adequate intake of high quality proteins and high quality fats are necessary on a daily basis.
4. Minerals and vitamins are very small components of the human body, minerals between 5-7% of mass and vitamins less than 1/10 of 1%. They are present to regulate and facilitate the birth and growth of new cells through regulation of minerals, fats and proteins..
5. Whole fresh meat, dairy, fowl, and fish, should always be the primary source of proteins and fats as food sources contain the appropriate accessory nutrients for production of healthy new cells.

It's time to add

6. In addition to high quality protein and fat we all need the ONLY two hormones we must get from our environment every day- Those hormones are Vitamin A and Vitamin D

How much vitamin A and what sources will provide your need?

In my experience with clients over the past 35 years persons with ancestors from coastal or island communities need preformed vitamin A, beta-carotene won't do. There will be exceptions to this but if you consume lots of vegetables with butter or extra virgin olive oil and still show symptoms of low vitamin A status consider a supplement of vitamin A containing a concentrate from fish liver oil.

Dry supplements of any of the fat soluble vitamins may not work as well as oil based supplements. The method used to 'dry' oil soluble vitamins A, D, K and E is cellulose. Cellulose is

Supplements may be beta-carotene, retinyl palmitate, retinol (fish liver oil derived), or a combination. Food sources include butter, egg yolk, animal and fish livers, and fish liver oil. The diagram following shows the steps needed to manufacture appropriate active vitamin A metabolites. Getting vitamin A from animal/fish sources as retinol enhances vitamin A status rapidly and bypasses problems with beta-carotene conversion.

Possible symptoms of vitamin A insufficiency:

Poor night vision (13;55-57)
Overly dry or oily skin, acne at any age (58)
Premenstrual syndrome (vitamin A controls production of progesterone)

Miscarriag e (59-62)
Irregular menses (63-67)
Chronic respiratory infections (33;34;68-70)
Chronic gut infections and impaired digestion (high susceptibility to food born pathogens) (71-73;73-75)
Loss of taste or smell (76-78)
Diminished hearing ability or noise sensitivity (79;80)
Poor memory (including Alzheimer's and dementia) (1-3;6;7;81)
Disturbed sleep rhythms (1;82)
Dry eyes (83-85)
Mucous membrane changes, thick mucous without infection, postnasal drip, or dry, sensitive eyes, nose, throat (may also involve vaginal mucous membranes) (86-95)
Bumps, like goose bumps, on the backs of upper arms that do not go away
Weight gain and insulin resistance (both associated with low vitamin A and D) (96)
Thyroid problems (hypo or hyper) (37;97-99)
Frequent urination, though little urine is passed. (women) There is little research in this area and not all frequent urination is associated with low A status but the lining of the bladder is profoundly altered when vitamin A is insufficient and the tissues of the bladder become less 'elastic' so the bladder both expands and contracts poorly.

Any chronic infection including herpes or HIV would increase the need for vitamin A.

Aging alters the ability to convert beta-carotene into retinol and as low vitamin A is associated with memory and sleep deficits, amyloid plaque in animal studies, and higher rates of infections of all types, getting enough preformed vitamin A would most certainly increase longevity and the enjoyment of longevity.

Accessory Nutrients Key to Fat Soluble Vitamin Sufficiency

Nutrition is never about just one thing. Macular degeneration lipofuscin is associated with unmetabolized remnants of vitamin A. Taurine, an amino acid found in fish, shell fish, lean meat, beef heart, and wild game has been found in high amounts in the human retina and appears to protect cells. It also plays a role in fat soluble vitamin metabolism.(100-106)

As mentioned before our ancestors had access to taurine in greater amounts than many American diets today. Lack of taurine alters the function of the human eye, heart, brain and gut. When taurine is present in sufficient amounts lower levels of vitamins A and D may be required.(106-108)

Taurine is the most abundant free amino acid in the human body and is found in higher amounts in

Excerpts and chart from- Taurine, A compilation by Ch. M. Ruessheim, December 30, 2000

For a long time, taurine was considered a nonessential nutrient for humans. However, in recent years it has become clear that Taurine is a very important amino acid involved in a large number of metabolic processes and can become **essential under certain circumstances**. Taurine is important in the visual pathways, the brain and nervous system, cardiac function, and it is a conjugator of bile acids. Basically, its function is to facilitate the passage of sodium, potassium and possibly calcium and magnesium ions into and out of cells and to stabilize electrically the cell membranes. Dr. G. E. Gaull (1984) suggests that since human never develop a high level of cysteinsulfinic acid decarboxylase, an enzyme necessary for the formation of taurine from the amino acid cysteine, people are probably all somewhat dependent upon dietary

taurine. Under certain conditions of high stress or in disease states the need for taurine probably increases. Another important function of taurine is detoxification.Taurine is required for efficient fat absorption & solubilization. Studies also showed that dietary taurine supplementation ameliorates experimental renal disease including models of refractory nephrotic syndrome and diabetic nephropathy. The beneficial effects of taurine are mediated by its antioxidant action. (Trachtman H. and Sturman J.A., 1996, Amino Acids, 11:1-13). Taurine may also have an important role in renal development. One study with rats showed protective effect of taurine on TNBS-induced inflammatory bowel disease. With all these discoveries and more on the horizon taurine research is accelerating rapidly…

Due to its ability to neutralize hypochlorous acid, a potent oxidizing substance, taurine is able to attenuate DNA damage caused by aromatic amine compounds in vitro. (Kozumbo et al, Breakage and binding of DNA by reaction products of hypochlorous acid with aniline, l-…, Toxicol Appl Pharmacol, 1992, 115,107-115). **Because of taurine's unique structure, containing a sulfonic acid moiety rather than carboxylic acid, it does not form an aldehyde from hypochlorous acid, forming instead a relatively stable chloroamine compound. Hence, taurine is an antioxidant that specifically mediates the chloride ion and hypochlorous acid concentration, and protects the body from potentially toxic effects of aldehyde release. Taurine has also been reported to protect against carbon tetrachloride-induced toxicity. (ref). Another striking finding: Retinol [vitamin A] in excess amounts, i.e., unbound to retinol-binding protein, can act as a poison. When the long-term lymphoid cell lines are exposed to 10 mc M retinol, their viability decreases strikingly over a 90-minute period]. Addition of zinc improves the viability slightly. Further addition of taurine protects the cells even more. If a combination of zinc and taurine is added, there is a striking protective effect. (Gaull, 1986). This suggests that taurine and zinc - both found in animal foods - provide protection from excess vitamin A - a vitamin found in full form only in animal foods** - then this is certainly an interesting synergism, to say the least. Another study showed that taurine could reduce the toxic effects of copper.

Exposure to bacterial endotoxins has been suggested as one factor which can augment the magnitude of individual response to xenobiotics (ref). Circulating endotoxins of intestinal origin have been found to create a positive feedback on endotoxin translocation from the gut, stimulating increases in serum endo-toxin levels. In experimental animals, taurine was found to significantly inhibit intestinal translocation and to protect the animals from endotoxemic injury (ref). Therefore, it is possible that taurine might be able to modify factors underlying susceptibility to toxic chemicals…

Taurine Content of Selected Foods (mg/kg, wet weight)

taken from Nutrient Requirements of Cats, Revised Edition, 1986 which in turn has adapted from Roe and Weston, 1965, Potential significance of free taurine in the diet, Nature, 205:287.

Item	Uncooked Mean	Range	Baked Mean	Range	Boiled Mean	Range
Beef muscle	362	150-472	133	96-125	60	58-63
Beef liver	192	144-270	141	68-184	73	36-95
Beef kidney	225	180-247	138	130-144	76	68-88
Lamb muscle	473	446-510	257	220-284	126	91-184
Lamb kidney	239	128-440	154	81-290	51	47-55
Pork muscle	496	394-690	219	126-390	118	91-184
Pork liver	169	110-228	85	70-100	43	30-54
Chicken muscle	337	300-380	229	140-310	82	71-180
Cod Fish	314	233-396	294	260-328	161	125-198
Oysters	698	390-1238	264	217-308	89	59-122
Clams	2400	1450-3700	1017	587-1700	446	264-794

Source URL http://www.serve.com/BatonRouge/taurine_chmr.htm

It is difficult to know how accurate the chart is because determining taurine in foods is not a well established science. In the chart above the milligram amount is per kilo which is 2.2 pounds. It is likely consumption of any 'juices' or items prepared in soup or stew would retain taurine. The taurine content of your diet will alter production of bile and absorption of all fat soluble nutrients including A, D, E and K. Taurine is important to the retina and may help prevent both cataract and macular degeneration. Taurine alters cell membrane permeability and neurotransmission. It may be useful in protecting the brain from Alzheimer's disease and cells from vitamin A excess. (101;107-112)

Supplementation of 250-1,000 mg of taurine two or three times a day when little fish or shellfish are consumed will provide a significant contribution to overall health and to your vitamin A and D status. This may also be important if you use protein powders, none of which contain taurine.

While there is no recommended dietary allowance for taurine a safe margin is likely 30-35 mg per kilo of actual body weight (about 10-15 mg per pound). It is unlikely higher doses would be needed except following surgery, severe burns, following tissue wasting or other severe injury.

All comments regarding any benefits from vitamin A supplementation assume you are committed to consuming a balance of other nutrients, especially high quality protein, high quality fats, taurine and vitamin D.

Vitamin A Guidelines

Some writers suggest that cod liver oil is a good source of A and D and while in some locations with some persons this may be true in other locations with other persons this will not be true.(113-117) Cod liver oil may be a good supplement in winter or in locations far north or south but not appropriate in Florida or other sunny climes.

Nutrition must be personal and you must be the person in charge. Test your 25(OH)D. If it remains between 35-70 ng/ml year round (ideal 40-60 ng/ml and the higher end is in no way better than the lower end), then consider your sources of vitamin A and your symptoms or lack thereof.

If your skin, hair, and nails are healthy, your skin neither overly dry or oily, you do not have acne, you have strong healthy teeth, no cavities, nor do you have periodontal disease or gum recession, you rarely get infections or food poisoning or stomach flu, you easily navigate a dark room, your sexual organs work well (sex drive, menses, pregnancy) and sleep and memory are your friends keep doing whatever it is you are doing.

If you have more than one of the symptoms and little or no source of vitamin A in your diet or supplements consider thoughtful, monitored, supplementation over several months, keeping a record of doses and results.

If you have more than one of the symptoms and are currently taking significant amounts of vitamin A in food and/or supplements and have been doing so for many months consider that you may be consuming excessive amounts of vitamin A.

The symptoms of vitamin A excess (as true of many vitamins) are similar to the symptoms of vitamin A deficiency. Chronic consumption of excess vitamin A is harmful. While excess A taken

with excess D seems to be less 'toxic' than excess of either individually, it is certainly not as beneficial as finding out how much you need and getting what you need.

The explanation for this 'reverse effect', too much being similar to too little, may have to do with binding proteins or receptor sites on the DNA or some other as yet unidentified cause, but whatever the cause too little or too much, for you, will not benefit your long term health.

When using vitamin A or D supplements it is possible to have an insufficiency, correct the problem but continue on the 'treatment' dose, and then find yourself with a return of symptoms or new symptoms because the dose is now too high.

Deficiencies of vitamins A and D may be treated with high doses initially with good effect. However, the same dose over time will be excessive. It is most likely maintenance dosing of vitamin D for adults ranges between 800 IU and 2,000 IU depending on location, skin type and genetics. The likely maintenance dose of vitamin A is 4,000 IU- 8,000 IU daily (total combined from food and supplements) with boosters during any and all infections .

If a profound deficiency has been present for some time daily doses of 10,000-20,000 IU of vitamin A retinol may be needed for several weeks to replete body stores.

RDA and DRI, Recommended Dietary Allowances and Dietary Reference Intakes, are broad averages of what may work for most persons. They may or may not work for you. Tolerance and need are, and always will be, individual.

These values do not consider absorption variables such as gluten intolerance, IBS, gallbladder insufficiency, colitis, lectin intolerance, pancreatic insufficiency, dietary anomalies (anorexia, low fat or no fat dieting, low protein, lack of essential fats, low DHA fatty acids, bulimia, gastric bypass) or genetic variables in beta-carotene conversion.

I believe food faddists, persons, and agencies promoting sun avoidance, processed food, refined food, low fat or no fat high carbohydrate diets, and/or supplements instead of food have harmed us all. Vitamins A and D are found in the fat and organs of animals, fowl and fish, and sunlight and have provided us with our 'daily hormones' until this present day.

Summary of Recommendations re Fat Soluble Vitamin/Hormones

Test your vitamin D. Use supplements and/or sunlight to keep 25(OH)D between 35-55 ng/ml.
Make sure your diet/supplements provide a minimum of 3,000 IU of vitamin A daily, animal/fish source retinol the preference, and not more that 8,000 IU daily (unless you have determined a higher need through careful and monitored dosing).
Make sure both oral intake of D and dietary or supplemental intake of vitamin A are accompanied by significant dietary fat (article at the end).
Consume sufficient high quality proteins and fats for your genetics and body size.
If your diet is low in fish and shellfish add omega-3 fatty acids and taurine. Typical doses are 2,000-3,000 mg of combined EPA and DHA 5-7 days a week and 1,000 mg of taurine 2-3 times a day. Both supplements may be taken with or without food. Sufficient omega-3 and taurine will likely reduce your requirement for vitamins A and D by maximizing absorption.

Regarding the Marshall Protocol, avoidance of vitamin D, need for vitamin A-

Dr. Marshall treats sarcoidosis and non-specific chronic infectious diseases such as arthritis, chronic Lyme disease, fibromyalgia and chronic fatigue syndrome by eliminating vitamin D and sunlight, lowering vitamin 25(OH)D levels, and giving long term low dose anti-biotics and Benicar. He has had some success with this protocol over the past 15 years or so. Dr. Marshall believes 25(OH)D must be less than 20 ng/ml or treatment will not be effective. This value would now be considered an insufficiency of D.

I am unaware whether Dr. Marshall has considered the relationship between vitamin A and D and that 25(OH)D over 20 ng/ml could contribute to a cellular insufficiency of vitamin A if vitamin A intake is low. That would make susceptibility to and recovery from infection most difficult.

Vitamin A sufficiency has been found to reduce infections both viral and bacterial.(33;34;118;119) As Dr. Marshall uses anti-biotics to kill pathogens he need not worry about vitamin A status.

As studies world wide have shown the benefit of vitamin D in bone health, and prevention of cancers of many types I worry about long term use of the Marshall Protocol.

I would like persons with intractable infectious conditions, herpes, mycoplasmas (cell wall deficient bacteria), Lyme's Disease, and other chronic systemic infections, to first consider maximizing protein intake from real whole foods, consume fatty fish or fish oil regularly, test vitamin D and replete and balance vitamin A before considering such a treatment.

Taurine also plays an important role in the immune system and making sure you get sufficient taurine may improve your symptoms.(120-123)

When infection is present it is difficult to take too much A. The rapid turnover of vitamin A and zinc as a part of the immune system suggests we need abundant supplies during an infection whatever the pathogen.(33;73;75;124-144)

If you are unable to restore health and need to consider the Marshall Protocol for a period of time remember the difference between treatment and maintenance. Do not keep 'treating' when health is restored.

When health is restored maximize protein and fat from food, increase your taurine intake and include moderate amounts of vitamins A and D.

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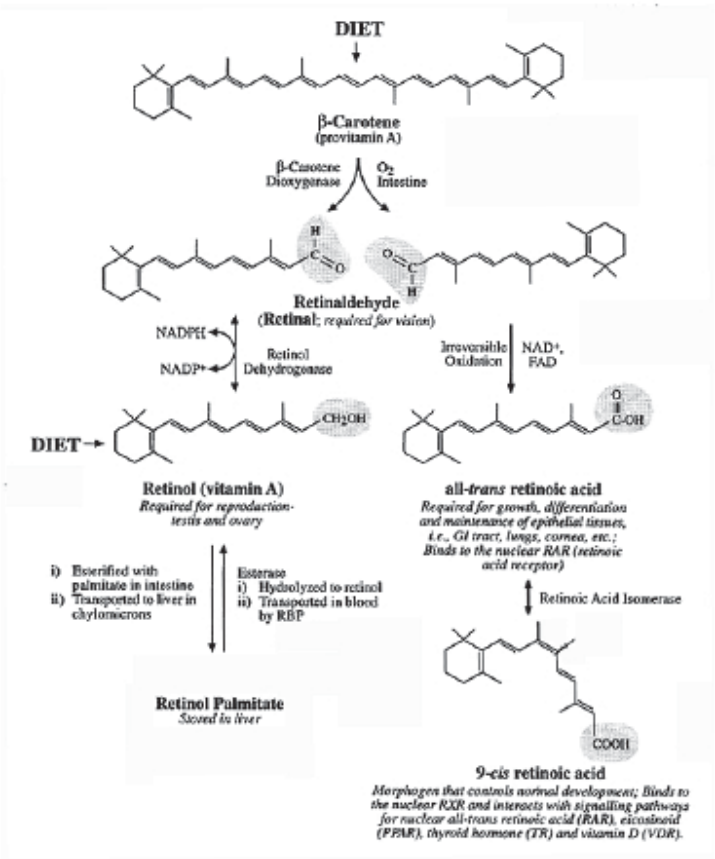

Krispin Komments on fats and food:

In the following article you will see just one of the the problems surrounding low fat eating. While the article is particularly focused on beta-carotene ALL fat soluble vitamins have the same problem, they must be eaten in a fatty food or if supplemented, taken with a fatty meal. For more

information on the problems associated with low fat diets please read Good Calories, Bad Calories by Gary Taubes Random House Sept. 2007

Wednesday, August 09, 2006

By Tara Parker-Pope, The Wall Street Journal

Are you getting the most out of your fruits and vegetables?

That's the question researchers are trying to answer as they study how our bodies absorb the healthful nutrients and compounds in foods. What they are findin is that in our quest to cut calories and fat from our diets, we may be cutting out a lot more.

It turns out that some of the best stuff in fruits and vegetables -- certain vitamins and cancer-fighting compounds -- are "fat-soluble." That means some fat needs to be present for the body to adequately absorb the nutrients. But studies are now showing that people who opt for no-fat dressing or who skip adding foods like avocado or cheese to a dish to avoid fat calories, are getting far less out of their salads and other veggies.

"What we're finding is that if you don't have some fat in the meal, all these wonderful" compounds are missed, says Steven Clinton, program leader for molecular carcinogenesis and chemoprevention and the Ohio State University Comprehensive Cancer Center in Columbus. "If the nutrients don't get into your system, then what good are they?"

Dr. Clinton's latest research looks at how adding avocado -- which is relatively high in unsaturated fat -- to salsa or a salad affects how well the body absorbs healthful compounds in the foods. In particular, the study looked at absorption of carotenoids, the red, yellow and orange pigments found in many fruits and vegetables that are believed to have cancer-fighting properties.

For the salsa study, 11 test subjects were first given a meal of fat-free salsa and some bread. Another day, the same meal was offered, but this time avocado was added to the salsa, boosting the fat content of the meal to about 37 percent of calories. In checking blood levels of the test subjects, researchers found that the men and women absorbed an average of 4.4 times as much lycopene and 2.6 times as much beta carotene when the avocado was added to the food.

Lycopene is the red carotenoid found in tomatoes and watermelon that is being studied as a potential fighter of prostate and other cancers. Beta carotene is the orange pigment in fruits and vegetables that is used in the body's manufacture of vitamin A. Studies suggest that diets high in fruits and vegetables containing beta carotene are linked to lower cancer rates.

With the salad test, the impact of adding avocado was even greater. The first salad included romaine lettuce, baby spinach, shredded carrots and a no-fat dressing, resulting in a fat content of about 2 percent. After avocado was added, the fat content jumped to 42 percent. When the salad was consumed with the avocado, the 11 test subjects absorbed seven times the lutein and nearly 18 times the beta carotene. Lutein is a carotenoid found in many green vegetables and is linked with improved eye and heart health.

Researchers noted that a small portion of the increased carotenoid levels in the blood of test subjects could be attributed to the compounds present in the avocado. However the vast majority of the increase was attributed to better overall absorption once fat was present.

Study researchers say they were not only surprised by how much more absorption occurred with the avocado was added to the meal, but they were taken aback at how little the body absorbed when no fats were present. "The fact that so little was absorbed when no fat was there was just amazing to me," says Dr. Clinton.

An earlier study done in 2004 by Ohio State University researchers showed a similar effect comparing salads consumed with no-fat, low-fat and full-fat salad dressings. When the seven test subjects consumed salads with no-fat dressing, the absorption of carotenoids was negligible. When a reduced-fat dressing was used, the added fat led to a higher absorption of alpha and beta carotene and lycopene. But there was substantially more absorption of the healthful compounds when full-fat dressing was used.

So far there isn't enough research to advise people how much fat they should consume with vegetables to get the optimal absorption of carotenoids. The basic advice is to still count calories and don't overdo the fats, choosing heart-healthy unsaturated fats like avocado or olive oil rather than foods with a high saturated-fat content.

A recent rat study by German researchers showed that the type of fat matters. They compared vitamin E absorption in rats that were fed diets with cottonseed oil or hydrogenated oils -- which contain unhealthy trans fats. The trans fats actually slowed the absorption of vitamin E compared with other type of fat.

For people watching their weight and the fat content of their diet, the balancing act might be tricky. The best nutrient absorption from the salad, for instance, occurred when diners ate dressing with 28 grams or about two tablespoons of canola oil. That translates to about 250 extra calories…

First published on August 9, 2006 at 12:00 am

Caution- Living Systems are Complex Systems

Why there cannot be an RDA (Recommended Daily Allowance) for vitamin D and why genes aren't the answer..

There is a constant pressing sense that the discovery of the secret of life is just around the corner, that there is this secret scientists are looking for and will soon find that will give us all disease and pain free eternal life. As we have yet to conquer the common cold perhaps we need a more realistic way of looking at things.

Life is dynamic, not static. The very 'essence' of life is that it is constantly changing. The earth spins around on its axis while moving around the sun as the sun moves through space and we move about the earth. Internally cells are constantly being born as old cells are dieing. The chemicals of life move in and out and through our cells moment by moment, second by second. All this MOVEMENT in response to never ending change. Life is eternal but man is not.

While certain academics attempt to sell DNA as the secret to life (though DNA is not living and cannot be made to be alive) and stem cells as key to cure of all disease and easy body repair more thoughtful scientists draw quite different conclusions based on more realistic data. Their conclusions don't sell all that well as they don't offer predictable outcomes or eternal life.

But the facts can help us deal realistically with the body we have been given, now.

Buying (or even believing in) a new drug or supplement that will purportedly lead to some utopian longevity is expensive in more ways than cash. Believing in magic may cause you ignore warnings and signals suggesting things need changing now; changes that might really make a difference in your life today and tomorrow.

Eternal life, in a body, is no where is sight, ever, for some very provable reasons. Life in all forms is comprised of interacting, constantly changing, non-linear complex systems. The nature of living complex systems is that they are not 'completed' but emerging, endlessly. How they emerge is altered by any number of other complex non-linear systems both inside out outside the living system. While calculated outcomes can be 'suggestive' they are not now nor ever will be guaranteed. Each bit of life is somewhat like every other bit and yet not alike at all.

From <u>It Ain't Necessarily So</u> edited by Richard Lewonton, Chapter 5 Not in Our Genes (with Steven P.R. Rose and Leon J. Kamin, pg 147
…it takes more than DNA to make a living organism. I once heard one of the world's leaders in molecular biology say, in the opening address of a scientific congress, that if he had a large enough computer and the complete DNA sequence of an organism he could compute the organism, by which he meant totally describe its anatomy, physiology, and behavior. But that is wrong. Even the organism does not compute itself from its DNA. A living organism, at any moment in its life, is the unique consequence of a developmental history that results from the interactions of and

determination by internal and external forces. The external forces, what we usually think of as "environment," are themselves partly a consequence of the activities of the organism itself as it produces and consumes the conditions of its own existence. Organisms do not find the world in which they develop. They make it. Reciprocally, the internal forces are not autonomous, but act in response to the external. Part of the internal chemical machinery of a cell is only manufactured when external conditions demand it…

When we interact with our environment we change it and are changed by it. Our genes may tweak the way we interact or respond but they do not create either our internal or external realities. It (life) really isn't in our genes. It's not in our proteins either. There is little evidence genetic engineering will prevent disease and it has the potential to create more unusual or devastating conditions than those it attempts to 'correct'. Stem cell research faces the same inevitable complexity with proven inability to control long term outcomes.

Why is this all so difficult? Just one small example: Your brain contains some 10,000,000,000 (10 billion) cells making some 13,000,000,000,000 (13 trillion) connections with each other, just your brain, not your entire nervous system or muscles or skin or other tissues and organs. Healthy communication between all of your cells and your cells communication with your environment and your action in and upon your body and environment, all are players in your state of health and vitality. Not one of the players stays in the same place or even in the same 'state' for any length of time. Everywhere there is movement and change. If you feel like you are spinning, good. My point is made.

Life, when expressed in its fullness is very much like 'dancing on the head of a pin'. For humans it requires keeping balanced within a moving object (your body) that is constantly changing shape, content, direction and speed on a moving object (earth) that is constantly changing shape and content. Earth's movement, direction and speed, remains somewhat constant, BUT always moving.

Food sustains life. Food that sustains life comes from life. Only life can feed life. Food provides elements that compose the human body and elements that provide fuel and flame for the energy we need to keep moving.

But life is more than food. Man is made to move. There is no cure for 'lack of exercise' except exercise. Machines have given us the illusion of freedom from work. We no longer have to move to get somewhere. We are moved through space by a machine. We no longer dig a ditch, wash a dish, scrub the floor, clean the laundry, hang out clothes to dry, push a lawn mower or scythe a field. We operate machines that do these things for us. Food is not hunted or gathered but collected by car at the local supermarket and stored in a machine to keep it fresh for days.

def. Machine: any mechanical or electrical device that transmits or modifies energy to perform or assist in the performance of human tasks

What these marvelous machines can't do for us is give us freedom from the need to move, the necessity of physical exercise.

Movement is significant character of living organisms. A hint from the universe, when movement stops it signifies death. Life IS movement and disequilibrium.

Nutrition- An Integrated Approach Third Edition Pike and Brown Chapter 9 pg 295
…All cells are units separated from their environment by a membrane. This is a barrier whose presence determines the shape and encloses the substance of the cell. Despite the variability and potential hostility of the outside environment, it is the membrane on which the constancy of the internal chemistry of the cell is dependent. …mass invasion of potentially toxic materials or rejection of essential nutrients can lead to cellular death by asphyxiation, hydration, desiccation, poisoning, starvation, or other equally effective means…

A cell in equilibrium with its environment is a dead cell. One of the fundamental attributes of a living cell therefore is the ability to prevent the establishment of an equilibrium…

What does any of this have to do with vitamin D or my health?

Essential elements of health and life include light, air, water, food, friends, environment, work, and exercise. Some necessary elements may get lost as diets and lifestyles change dramatically within a few generations. While looking to science to cure our ills we may overlook these essential elements and thereby suffer harm. Certainly sunlight and vitamin D fit this profile.

Sun avoidance, removing the fat and skin, rich sources of omega-3 and vitamin D, from fresh caught fish, overall eating fewer fish, and altering the habits of animal husbandry create significant losses in sources of vitamin D.

Vitamin D has receptors on the plasma membrane as well as the nucleus of many cells. Vitamin D is considered to be a member of the steroid hormone family.

def. hormone (from Greek horman - "to set in motion") is a chemical messenger from one cell (or group of cells) to another. All multicellular organisms produce hormones including plants.

Vitamin D, like the other steroid hormones, has genomic functions. It acts upon the receptors designed for it within living cells. Depending on the balance of all of the other hormone-messengers (dancing partners) on receptors of any given cell at any given time, instructions are transmitted to stop making something or start making something or make more or less of something.

Vitamin D does not work alone. Cells containing receptors for vitamin D also contain receptors for other steroid hormones including vitamin A derived RAR and RXR, thyroid, and estrogen. Calcium, which plays a significant role in cell-cell messaging, also interacts with vitamin D.

There is not one single nutrient, enzyme, hormone or other protein, including DNA, that can be controlled to guarantee a given cellular outcome WITHIN A LIVING SYSTEM.

In a Petri dish some control may be possible, to some small extent, but NEVER within a complex living system. It is this fact that has prevented and will continue to prevent the 'miracle' outcomes predicted by some delusional researchers.

By conversation (exchanging information) with other hormone-messengers vitamin D helps decide what happens next. The absence, presence, relative excess, or relative deficiency of any given hormone-messenger will send DIFFERENT instructions determining what happens next in that cell.

Some scientists, from the earliest beginnings of the study of our physical and natural world, believed one could 'know' a thing by tearing it apart and determining its 'elements'. The goal has been to reduce the studied organism or disease to its most elemental parts. Finding the atom has not brought us even one step closer to understanding 'life'.

When scientists use the dissection (tear into and examine the parts) approach it reduces 'variables', those differences that confound outcomes. The goal is to make the object more understandable. Unfortunate for the scientists, variables are exactly what makes life 'alive'.

def. reductionism:
noun: the analysis of complex things into simpler constituents
noun: a theory that all complex systems can be completely understood in terms of their components

Lifelines Life Beyond the Gene Steven Rose pg 79
…But living systems are not simple, they involve many interacting variables. Parameters are not fixed; properties are non-linear. And the living world is highly non-uniform. Reductionist methodology is helpful in chemistry, say, because (so far as is known) the chemical world is the same everywhere. In the living world, the exception is nearly always the rule. So if one is not careful, the simplifying constraints that the methodology offers soon cease to be helpful support to theory, and instead become straitjackets. The Zuckerman trap… awaits us if we are not careful to remember that ***what happens in the test-tube may be the same, the opposite of, or bear no relationship at all to what happens in the living cell, still less the living organism in its environment….***

Emphasis is mine.

DNA does not inevitably determine disease or outcome. Neither does vitamin D or any other hormone or nutrient or protein or chemical. OUTCOMES ARE DETERMINED BY THE PARTICIPANTS AND THE CONVERSATION. It is the elements gathered for the conversation, the time of day, the season, the latitude, the longitude, the mood, the meal, RELATIONSHIPS. There it is,

outcomes are determined by and through the relationships of living things to living things to all things.

There is not a test nor will there ever be a test to know what you will die of or what diseases you will get or avoid. What is fascinating is that some very learned academics suggest differently even though their colleagues continue to disprove the validity of any theory of genetic determinism. We continue to have choices, about what we eat and drink and how and where we live and who and what we love, in our daily lives , and these simple choices will have more effect on outcome than all scientific knowledge distilled and swallowed whole.

No one can decide for you what will make your life journey _____.
What goes in the blank? I can think of many things. Most of them are for the moment, here and gone. Lovely, joyous, thrilling, satisfying, fulfilling, peaceful, courageous, happy, content, exciting, productive.

For me the blank would be 'useful'. That is what I most want from my life, to be useful. A healthy body and mind helps me be more useful and stay useful longer.

Whatever your goal in seeking health, listen to your body and work to find those things that you need, enough but not too much, and avoid those things you do not need. Learn to make wise choices from the myriad of choices set before you every day.

When evaluating any diet, supplement, medication, lifestyle, or relationship consider equally the implications of 'dancing on the head of a pin'; the importance of disequilibrium.

def. disequilibrium- noun: loss of equilibrium attributable to an unstable situation in which some forces outweigh others

Life is, by its very nature, unstable, unequal, vibrant, moving, exciting, dangerous, insecure; like the sea, it is never still. There are no guarantees. Your dance must be constantly changing, moment to moment, to safely navigate your life's journey. Keep dancing. Maintain moderation in all things, including exercise, food, supplements, sunlight, and friends.

Gently search in your diet, environment, work, and relationships for those things that are of sensible (you are able to recognize their value) use to you..

- ☐ Is something missing
- ☐ Is this vital
- ☐ Is this toxic
- ☐ Do I have too much
- ☐ Do I have enough
- ☐ Does this feed me
- ☐ Does this poison me
- ☐ Does this make sense

Find your voice, your music, and your partners, and trust the Still Small Voice that gently reminds you of the wonder of it all; of the wonder of you; of the wonder of life.

Books I Love:

Lessons From The Living Cell- The Limits Of Reductionism . Stephen Rothman, Mcgraw Hill

It Ain't Necessarily So- The Dream Of The Human Genome And Other Illusions . Second Edition Richard Lewontin, New York Review Books

Lifelines- Life Beyond The Genes Steven Rose, Oxford

The End Of Certainty- Time Chaos And The New Laws Of Nature Ilya Prigogine, Free Press

Sync-The Emerging Science Of Spontaneous Order Steven Strogatz, Hyperion

Health, Immunity and Aging

There is significant evidence a functioning immune system is key to health and longevity. Immune function is all about walls, walls that divide 'self' from 'not-self'. It is true good walls make good neighbors.

The human body is very much like the description of a cell (also repeated later in the Workbook)-
From chapter 9, The Plasma Membrane, Nutrition an Integrated Approach , Pike and Brown, 1986
All cells are units separated from their environment by a membrane. This is a barrier whose presence determine the shape and encloses the substance of the cell. Despite the variability and potential hostility of the outside environment, it is the membrane on which the constancy of the internal chemistry of the cell is dependent. The discharge of this responsibility is made possible by the ability of the membrane to discriminate among those organic and inorganic molecules in the surrounding medium, permitting the entrance to some and rebuffing others. This is a truly vital task since either mass invasion of potentially toxic material or rejection of essential nutrients can lead to cellular death by asphyxiation, hydration, desiccation, poisoning, starvation, or other equally effective means. The cell, thus dependent on the external environment for all the raw materials from which it is made and with which it operates, by means of the membrane barrier and it fastidious selectivity, can enjoy a distinct and separate existence.
A cell in equilibrium with its environment is a dead cell.

For survival, selectivity, that is **discrimination**, is the only choice. It is necessary that the cell selects and allows those things that are needed and avoids those things that are unnecessary or toxic. This is equally true throughout your body as a whole. Your body must remain vigilant to accept that which is able to maintain life and become 'self' and to avoid or destroy that which is 'not self'. Tolerance of 'not self' brings dissolution of the cell and death.

Inflamm Res. 2000 Nov;49(11):561-70.

Unregulated inflammation shortens human functional longevity.

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Systemic inflammation, represented in large part by the production of pro-inflammatory cytokines, is the response of humans to the assault of the non-self on the organism. Three distinct types of human ailments - namely autoimmunity, presenile dementia (Alzheimer's disease), or atherosclerosis - are initiated or worsened by systemic inflammation. Autoimmunity is unregulated hyperimmunity to organ-specific proteins, inducing rapid turnover of antigen-specific T cells of the acquired immune system with ultimate exhaustion and loss of acquired immunity IL-2 and IFN-gamma production and proliferative decline, conforming to the limited capacity of clonal division (Hayflick phenonmenon). In Alzheimer's disease (AD), the primary degenerative process of amyloid-beta (AJ3) protein precedes a cascade of events that ultimately leads to a local "brain inflammatory response". Unregulated systemic immune processes are secondary but important as a driving-force role in AD pathogenesis. Atherosclerosis, an underlying cause of myocardial infarction, stroke, and other cardiovascular diseases, consists of focal plaques characterized by cholesterol deposition, fibrosis, and inflammation. The presence of activated T lymphocytes and macrophages indicate a local immunologic activation in the atherosclerotic plaque that may be secondary to unregulated pro-inflammatory cytokines too. The premature hyperimmunity of autoimmunity, the local "brain inflammatory response" to A/3 protein in AD, and the immune response to fatty changes in vessels in atherosclerosis all signal the critical importance of unregulated systemic inflammation to common neurological and cardiovascular disease that shortens the nominal longevity of humans.

Over a lifetime assaults include over-exposure to infectious pathogens including bacteria, virus, spirochetes, and mycoplasmas; damage to healthy gut microflora from pathogens or antibiotics resulting in immune failure of the gut wall; consumption of lectins, glycoproteins such as gluten or casein, that are not genetically appropriate resulting in immune dysfunction of the gut wall. These assaults can be intensified, meaning immune response does not result in full recovery, if sources of nutrients needed by the immune system, such as protein, zinc, selenium, iron, vitamin A, vitamin D, vitamin E, and others, are missing or insufficient.

The immune system exists to protect our bodies from invasion by toxins (chemicals, drugs, heavy metals, poisons), pathogens (infectious agents including virus, bacteria, parasites and fungi), allergens (injected, including vaccines, inhaled or ingested) and other environmental assaults.

Lack of exposure to 'other-not me' assaults (living in a bubble) prevents the development of critical parts of the immune system, hence the advice to allow children to have pets at an early age. BUT over stressing the immune system shortens life expectancy.

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Systemic inflammation, represented in large part by the production of pro-inflammatory cytokines, is the response of humans to the assault of the non-self on the organism. Three distinct types of human ailments - namely autoimmunity, presenile dementia (Alzheimer's disease), or atherosclerosis - are initiated or worsened by systemic inflammation. Autoimmunity is unregulated hyperimmunity to organ-specific proteins, inducing rapid turnover of antigen-specific T cells of the acquired immune system with ultimate exhaustion and loss of acquired immunity IL-2 and IFN-gamma production and proliferative decline, conforming to the limited capacity of clonal division (Hayflick phenomenon). In Alzheimer's disease (AD), the primary degenerative process of amyloid-beta (AJ3) protein precedes a cascade of events that ultimately leads to a local "brain inflammatory response". Unregulated systemic immune processes are secondary but important as a driving-force role in AD pathogenesis. Atherosclerosis, an underlying cause of myocardial infarction, stroke, and other cardiovascular diseases, consists of focal plaques characterized by cholesterol deposition, fibrosis, and inflammation. The presence of activated T lymphocytes and macrophages indicate a local immunologic activation in the atherosclerotic plaque that may be secondary to unregulated pro-inflammatory cytokines too. The premature hyperimmunity of autoimmunity, the local "brain inflammatory response" to A/3 protein in AD, and the immune response to fatty changes in vessels in atherosclerosis all signal the critical importance of unregulated systemic inflammation to common neurological and cardiovascular disease that shortens the nominal longevity of humans.

The immune system is keyed to all 'surfaces', the skin, the gut from mouth to anus, the cell walls and cell membranes. In infants the gut wall is semi-permeable at birth but rapidly develops closure in breast fed infants. Feeding of formula, even 'hypo-allergenic formula' seems to delay development of gut barrier function. (1)

Early introduction of inappropriate proteins can irreversibly alter immune function with dire results.(2-4) Evidence indicates breast feeding, even for a few months, feeding genetically appropriate foods, and early introduction of probiotics help to lower immune assaults early in life and reduce lifetime immune burden.(5)

Vaccinations prevent a number of life-threatening diseases but over-vaccination, becoming more likely with the new policies of yearly flu vaccines and increasing insistence on vaccines for other viral diseases, are likely to lead to increases in autoimmune diseases. Some researchers suggest

this may already be happening. Evidence from veterinary medicine shows a steep increase in auto-antibodies in vaccinated versus unvaccinated dogs.(6-8)

Our immune system has been most recently classified by stimulation of T cells, T helper cells, T Suppressor cells and T Killer cells. Assaults stimulate expression of T Helper cells 1 and/or T Helper cells 2 (TH1 and TH2).

Th1 function regulates the cellular or cell-mediated immune system designed to destroy, digest and expel foreign antigens out of the body via the lymph system. This is your body's 'acute inflammatory response' and is often accompanied by inflammation, fever, pain, malaise and discharge of mucus, pus, rash or diarrhea.

Th2 functions as your humoral immune system regulating the production of antibodies which recognize foreign antigens in blood.

The two types of T-helper cells are defined by the cytokines they produce. Th1 cells, involved in cellular immunity, produce IL-2, TNF-beta and IFN-gamma, while Th2 cells, with roles in humoral immunity, produce IL-4, IL-5 and IL-10. The cytokines produced by Th2 cells enhance Th2 development and inhibit Th1 development, while Th1 cytokines stimulate development of Th1 and inhibit development of Th2.

These two functions work together to protect us from 'not us'. Th2 functions act as a sense organ, 'tasting', identifying and remembering foreign invaders (not-self). Th1 functions digest and eliminate foreign invaders from the body. While cellular immunity (Th1) directs Natural Killer T-cells and macrophages to attack abnormal cells and microorganisms at sites of infection inside the cells, humoral immunity (Th2) results in the production of antibodies used to neutralize foreign invaders and substances outside of the cells.

We are daily assaulted by virus, bacteria, parasites, fungus and antigenic proteins.
In many cases, an infection is fought with both arms of the immune system. At other times predominantly one is needed to control an infection. A healthy immune system is balanced and dynamic, Th1 and Th2 activity switching back and forth as needed. This allows for a quick eradication of a threat and then a return to balance before responding to the next threat. The inability to respond adequately with a Th1 response can result in chronic infection and cancer; an overactive Th2 response can contribute to allergies, and play a role in the development of autoimmune diseases. In end stage illnesses, both arms of the immune system fail.

The food we eat, the drugs we use, the infectious agents we are intentionally or unintentionally exposed to and the health of our gut all potentially contribute to immune load. Enough but not too much is the rule. We need a certain amount of exposure to 'foreign invaders' to stimulate immune function but chronic over stimulation results in acute or chronic illness and aging of the immune system.

An example of 'stress but not too much' would be using 'exfoliation' to stimulate regeneration of sun-damaged skin. Infrequent use in some persons will produce the desired results but chronic use or even light use in susceptible persons will damage underlying structures in the skin and increase the risk of skin cancer. In all cases exfoliation renders the skin more vulnerable to chemical and sun damage for 7-10 days following treatment.

As we age there is a shift from Th1 to Th2 dominance resulting in less ability to resist and recover from infectious disease, a generalized increase in inflammation and an increased likelihood of autoimmune disease.(9-11) This change increases the risk of altered cell-cell communication resulting in higher rates of cell hyperplasia (over production of cells as found in benign prostatic hyperplasia). Chronic inflammation is recognized as a promoter in heart disease, hypertension, diabetes, osteoporosis and cancer.

Much of this aging of the immune system is modifiable. An adequate diet, normal thyroid function and adequate vitamin D support immune health even in centenarians.(12-15)

Consumption of whole, fresh foods, genetically appropriate, (Did your ancestors eat it?) including fish, shell fish, poultry, meat and organ meats supports immune health at any age. Organ meats, especially livers, provide the important fat soluble vitamins and key minerals required for immune function.(16-18) Fresh foods provide abundant anti-oxidants, protein, minerals and vitamins.

Moderate regular exercise stimulates (think 'exercises') immune functions, both Th1 and Th2 in a positive way.(19-23) Regular moderate sun exposure also enhances immunity at any age. Lack of sunlight or overexposure to sunlight suppresses immune response.(24)

Vaccination may be important in areas where exposure to the disease is likely but vaccination when exposure is unlikely may increase immune load without long-term benefit. Yearly flu vaccine will more rapidly push the immune system into Th2 response found in aged individuals. Why? Here is the hypothesis from Gary Null.

http://www.garynull.com/Documents/niin/how_vaccinations_work.htm

…A vaccination consists of introducing a disease agent or disease antigen into an individual's body without causing the disease. If the disease agent provoked the whole immune system into action it would cause all the symptoms of the disease! The symptoms of a disease are primarily the symptoms (fever, pain, malaise, loss of function) of the acute inflammatory response to the disease.
So the trick of a vaccination is to stimulate the immune system just enough so that it makes antibodies and "remembers" the disease antigen but not so much that it provokes an acute inflammatory response by the cellular immune system and makes us sick with the disease we're trying to prevent! Thus a vaccination works by stimulating very much the antibody production (Th2) and by stimulating very little or not at all the digesting and discharging function of the cellular immune system (Th1).
Vaccine antigens are designed to be "unprovocative" or "indigestible" for the cellular immune system (Th1) and highly stimulating for the antibody-mediated humoral immune system (Th2). Perhaps it is not difficult to see then why the repeated use of vaccinations would tend to shift the functional balance of the immune system toward the antibody-producing side (Th2) and away from the acute inflammatory discharging side (the cell-mediated side or Th1). This has been confirmed by observation especially in the case of Gulf War Illness: most vaccinations cause a shift in immune function from the Th1 side (acute inflammatory discharging response) to the Th2 side (chronic auto-immune or allergic response).
… There is no system of the human being, from mind to muscles to immune system, which gets stronger through avoiding challenges, but only through overcoming challenges. The wise use of vaccinations would be to use them selectively, and not on a mass scale. In order for vaccinations to be helpful and not harmful, we must know beforehand in each individual to be vaccinated whether the Th1 function or the Th2 function of the immune system predominates.
In individuals in whom the Th1 function predominates, causing many acute inflammations because the cellular immune system is over reactive, a vaccination could have a balancing effect on the immune system and be helpful for that individual.
In individuals in whom the Th2 function predominates, causing few acute inflammations but rather the tendency to chronic allergic or autoimmune inflammations, a vaccination would cause the Th2 function to predominate even more, aggravating the imbalance of the immune system and harming the health of that individual.

Sleep also plays an important role in immune health.(25-31) Studies show sleep deprivation decreases production of NK and NKT cells.

Natural Killer cells destroy cells that have become infected with a virus or cells that are replicating abnormally such as pre-cancerous cells. Natural Killer T cells secrete cytokines of both the Th1

and Th2 family and destroy infectious agents as well as protecting against autoimmune disease. Both of these sleep modifiable cell types are critical players in immune maintenance.

Our environment, those around us, family, friends and co-workers, and even the thoughts we think alter immune function. Positive thinking, a positive human support system, and faith in God improve immune health.(32-37)

To remain healthy into advanced age we need a lifetime of good friends, genetically appropriate fresh, real, whole food, faith, exposure to and full recovery from common pathogens and infectious agents, quality and quantity of good sleep, intermittent doses of probiotics or naturally fermented foods, and moderate regular exercise.

Things to avoid for long-term health: Excessive exercise, avoidance of exercise, excessive food, dead fake food, processed food, genetically inappropriate food, refined sugar, excessive alcohol, chronically depressed or angry friends or family, chronically angry or paranoid politics, a bad attitude, and sleep deprivation.

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