Vitamin D					

VITAMIN D AND SUNSHINE

I found that both vitamin D and sunshine helped me during my recovery. I particularly felt better after spending time in the sun, even though I was taking 6-8 capsules of vitamin A and D (10,000 IU of A, 400 IU of D per capsule). I shouldn't have been vitamin D deficient and if anything may have been taking too much A and D. However the added sunshine was always a plus. One member of our group who had a very persistent case of hyperT also reported that on vacations to sunny places where she was able to get into the sun every day she felt much better even though she stopped taking supplements for that week. JJ

"It was confirmed that TH (thyroid hormone) produced a peroxide of dehydrocholesterol, a precursor of vitamin D3, in the diaphysis of the femur in the increased metabolic state." cadmium inhibits lipogenesis.doc Does this mean that thyroid hormone causes an increased peroxidation of pre-vitamin D and this is the mechanism by which hyperT causes calcium metabolism problems? JJ

Wilson's disease results in excess tissue accumulation of copper and is often complicated by skeletal and mineral abnormalities. We investigated *vitamin* D metabolism in rats fed a copper-laden diet rendering hepatic copper content comparable with that found in Wilson's disease. Injection of 25-hydroxyvitamin D3 [25(OH)D3] resulted in reduced 1,25-dihydroxyvitamin D [1,25(OH)2D] levels in copper-intoxicated rats. In vitro 25(OH)D-1 alpha-hydroxylase activity was impaired in renal mitochondria from copper-intoxicated animals. Activity was also inhibited in mitochondria from controls when copper was added to incubation media. Impaired conversion of 25(OH)D to 1,25(OH)2D occurs in copper intoxication and suggests that altered *vitamin* D metabolism is a potential factor in the development of bone and mineral abnormalities in Wilson's disease. vitamin D metabolism impaired in Wilson.doc

The following information indicates that vitamin D deficiency is involved in thyroid disease. Also, there are links to obesity, diabetes, cancer, heart disease, arthritis, depression, PMS, and autoimmune disease. The article is from Dr. Mercola's site at mercola.com:

Breakthrough Updates You Need to Know on Vitamin D

The compound we call vitamin D can no longer properly be considered a vitamin. For most mammals, it is not in any sense even a nutrient. Nevertheless, vitamin D resembles true vitamins inasmuch as humans -- who are cut off from the critical solar ultraviolet wavelengths by reason of latitude, clothing, or shelter -- depend on an external source of the substance, just as they do for the true essential nutrients.

What is Vitamin D?

Vitamin D, calciferol, is a fat-soluble vitamin. It is found in food, but also can be made in your body after exposure to ultraviolet rays from the sun. Vitamin D exists in several forms, each with a different activity. Some forms are relatively inactive in the body, and have limited ability to function as a vitamin. The liver and kidney help convert vitamin D to its active hormone form.

The major biologic function of vitamin D is to maintain normal blood levels of calcium and phosphorus. Vitamin D aids in the absorption of calcium, helping to form and maintain strong bones. It promotes bone mineralization in concert with a number of other vitamins, minerals, and hormones.

Without vitamin D, bones can become thin, brittle, soft, or misshapen. Vitamin D prevents rickets in children and osteomalacia in adults, which are skeletal diseases that result in defects that weaken bones.

What are the sources of vitamin D?

Food sources

Fortified foods are the major dietary sources of vitamin D. Prior to the fortification of milk products in the 1930s, rickets (a bone disease seen in children) was a major public health problem in the United States. Milk in the United States is fortified with 10 micrograms (400 IU) of vitamin D per quart, and rickets is now uncommon in the US.

Exposure to sunlight

Exposure to sunlight is an important source of vitamin D. Ultraviolet (UV) rays from sunlight trigger vitamin D synthesis in the skin.

Season, latitude, time of day, cloud cover, smog, and suncreens affect UV ray exposure. For example, in Boston the average amount of sunlight is insufficient to produce significant vitamin D synthesis in the skin from November through February.

Sunscreens with a sun protection factor of 8 or greater will block UV rays that produce vitamin D.

Vitamin D supplements are often recommended for exclusively breast-fed infants because human milk may not contain adequate vitamin D.

Vitamin D and Bone Health

It is estimated that over 25 million adults in the United States have, or are at risk of developing osteoporosis. Osteoporosis is a disease characterized by fragile bones. It results in increased risk of bone fractures.

Rickets and osteomalacia were recognized as being caused by vitamin D deficiency 75 years ago; their prevention and cure with fish liver oil constituted one of the early triumphs of nutritional science. The requirement for vitamin D has been pegged to these disorders ever since.

Having normal storage levels of vitamin D in your body helps keep your bones strong and may help prevent osteoporosis in elderly, non-ambulatory individuals, in post-menopausal women, and in individuals on chronic steroid therapy.

Researchers know that normal bone is constantly being remodeled (broken down and rebuilt). During menopause, the balance between these two systems is upset, resulting in more bone being broken down (resorbed) than rebuilt.

Vitamin D deficiency has been associated with greater incidence of hip fractures. A greater vitamin D intake from diet and supplements has been associated with less bone loss in older women. Since bone loss increases the risk of fractures, vitamin D supplementation may help prevent fractures resulting from osteoporosis.

The use of vitamin D is well accepted, but the mere absence of clinical rickets can hardly be considered an adequate definition either of health or of vitamin D sufficiency.

The fact that it takes 30 or more years to manifest itself makes it no less a deficiency condition than a disorder that develops in 30 days. It is easy to understand how

long-period deficiency diseases could never have been recognized in the early days of nutritional science, but with modern methods and a better grasp of the relevant physiology, failing to recognize a slowly developing condition as a true deficiency state, can no longer be justified.

Vitamin D nutrition probably affects major aspects of human health, as listed below, other than its classical role in mineral metabolism. The rest of the article addresses some of the newly recognized uses of vitamin D.

Cancer

Today, it is well established that besides playing a crucial role in the establishment and maintenance of the calcium in the body, the active form of vitamin D also acts an effective regulator of cell growth and differentiation in a number of different cell types, including cancer cells.

Laboratory, animal, and epidemiologic evidence suggest that vitamin D may be protective against some cancers. Clinical studies now show vitamin D deficiency to be associated with four of the most common cancers:

- Breast (23)
- Prostate 24-27
- Colon 28-31
- Skin 32,33

Diabetes

Vitamin D deficiency has been associated with insulin deficiency and insulin resistance. (1-3) In fact, last year it was shown that vitamin D deficiency is likely to be a major factor for the development of type one diabetes in children. (4)

Heart Disease

Insulin resistance is also one of the major factors not only leading to the cancers mentioned above, but also to the number one killer in the US, heart disease. Northern countries have higher levels of heart disease and more heart attacks occur in the winter months. (5,6)

Arthritis

Progression of degenerative arthritis of the knee and hip is faster in people with lower vitamin D concentrations (33-34)

Infertility and PMS

Infertility is associated with low vitamin D(7), and PMS has been completely reversed by addition of calcium, magnesium and vitamin D.(8)

Fatigue, Depression and Seasonal Affective Disorder

Activated vitamin D in the adrenal gland regulates tyrosine hydroxylase, the rate limiting enzyme necessary for the production of dopamine, epinephrine and norepinephrine.

Low vitamin D may contribute to chronic fatigue and depression. (9-10) 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.(11)

Autoimmune Disorders

Multiple Sclerosis, (12) Sjogren's Syndrome, rheumatoid arthritis, thyroiditis and Crohn's disease have all been linked with low vitamin D levels.

Single, infrequent, intense, skin exposure to UV-B light suppresses the immune system and causes harm.

However chronic low-level exposure normalizes immune function and enhances immune cell production. This reduces abnormal inflammatory responses such as found in

autoimmune disorders, and reducing occurrences of infectious disease. (14-18)

Obesity

Vitamin D deficiency has been linked with obesity. (18, 19) Vitamin D has recently been shown to lower leptin secretion. (20) Leptin is a hormone produced by fat cells and is involved in weight regulation. It is thought that the hormone signals the brain when fat cells are "full," but exactly how the hormone controls weight is not entirely clear.

Additionally, obesity by itself probably further worsens vitamin D deficiency due to the decreased bioavailability of vitamin D(3) from skin and dietary sources, because of its being deposited in body fat. (36)

Syndrome X

Vitamin D deficiency has been clearly linked with Syndrome X. (21) Syndrome X refers specifically to a group of health problems that can include insulin resistance (the inability to properly deal with dietary carbohydrates and sugars), abnormal blood fats (such as elevated cholesterol and triglycerides), overweight, and high blood pressure.

Vitamin D and Steroids

Steroids, like prednisone, are often prescribed to reduce inflammation from a variety of medical problems. These medicines may be essential for a person's medical treatment, but they have potential side effects, including decreased calcium absorption.

There is some evidence that steroids may also impair vitamin D metabolism, further contributing to the loss of bone and development of osteoporosis associated with steroid medications. For these reasons, individuals on chronic steroid therapy should consult with their physician or registered dietitian about the need to increase vitamin D intake through diet and/or dietary supplements.

The above document was edited from:

National Institutes of Health Document on Vitamin D

DR. MERCOLA'S COMMENT:

I wish to express my sincere appreciation to nutritionist Krispin Sullivan for the years she researched this subject, which provided me with so much of the foundational background for this review. She is publishing the definitive resource for vitamin D later this year called Naked at Noon.

A preliminary copy of her vitamin D research is available on her web site.

References

- 1. Hypponen E, Laara E, Reunanen A, Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study. Lancet. 2001 Nov 3;358(9292):1500-3.
- 2. Billaudel B, Barakat L, Faure-Dussert A. Vitamin D3 deficiency and alterations of glucose metabolism in rat endocrine pancreas. Diabetes Metab 1998;24:344-50.
- 3. Bourlon PM, Billaudel B, Faure-Dussert A. Influence of vitamin D3 deficiency and 1,25 dihydroxyvitamin D3 on de novo insulin biosynthesis in the islets of the rat endocrine pancreas. J.Endocrinol. 1999;160:87-95.
- 4. Ortlepp JR, Lauscher J, Hoffmann R, The vitamin D receptor gene variant is associated with the prevalence of type 2 diabetes mellitus and coronary artery disease. Diabet Med. 2001 Oct;18(10):842-5.
- 5 Segall JJ. Latitude and ischaemic heart disease [letter]. Lancet 1989;1:1146.

- 6 Williams FL, Lloyd OL. Latitude and heart disease [letter]. Lancet 1989;1:1072-3.
- 7. Panda DK, Miao D, Tremblay ML, Targeted ablation of the 25-hydroxyvitamin D 1alpha -hydroxylase enzyme: evidence for skeletal, reproductive, and immune dysfunction. Proc Natl Acad Sci U S A. 2001 Jun 19;98(13):7498-503.
- 8. Thys-Jacobs S. Micronutrients and the premenstrual syndrome: the case for calcium. J.Am.Coll.Nutr. 2000;19:220-7.
- 9. Puchacz E, Stumpf WE, Stachowiak EK, Stachowiak MK. Vitamin D increases expression of the tyrosine hydroxylase gene in adrenal medullary cells. Brain Res. Mol. Brain Res. 1996;36:193-6.
- 10. : Gloth FM 3rd, Alam W, Hollis B. Vitamin D vs broad spectrum phototherapy in the treatment of seasonal affective disorder. J Nutr Health Aging. 1999;3(1):5-7.
- 11. Gloth FM, III, Alam W, Hollis B. Vitamin D vs broad spectrum phototherapy in the treatment of seasonal affective disorder. J.Nutr.Health Aging 1999;3:5-7.
- 12: Hayes CE. Vitamin D: a natural inhibitor of multiple sclerosis. Proc Nutr Soc. 2000 Nov;59(4):531-5.
- 13. McMichael AJ, Hall AJ. Multiple sclerosis and ultraviolet radiation: time to shed more light. Neuroepidemiology. 2001 Aug; 20(3):165-7.
- 14. Deluca HF, Cantorna MT. Vitamin D: its role and uses in immunology. FASEB J. 2001 Dec;15(14):2579-85.
- 16. Long KZ, Santos JI. Vitamins and the regulation of the immune response. Pediatr.Infect.Dis.J. 1999;18:283-90.
- 17. Ghezzi A, Zaffaroni M. Neurological manifestations of gastrointestinal disorders, with particular reference to the differential diagnosis of multiple sclerosis. Neurol Sci 2001 Nov;22 Suppl 2:S117-22
- 18 Cantorna MT. Vitamin D and autoimmunity: is vitamin D status an environmental factor affecting autoimmune disease prevalence? Proc.Soc.Exp.Biol.Med. 2000;223:230-3
- 19 Shi H, Norman AW, Okamura WH, Sen A, Zemel MB.1alpha,25-Dihydroxyvitamin D3 modulates human adipocyte metabolism via nongenomic action. FASEB J. 2001 Dec;15(14):2751-3
- 20 Speer G, Cseh K, Winkler G, Vitamin D and estrogen receptor gene polymorphisms in type 2 diabetes mellitus and in android type obesity. Eur J Endocrinol. 2001 Apr;144(4):385-9.
- 21 Henendez C, Lage M, Peino R, Retinoic acid and vitamin D(3) powerfully inhibit in vitro leptin secretion by human adipose tissue. J Endocrinol. 2001 Aug;170(2):425-31
- 22 Boucher BJ. Inadequate vitamin D status: does it contribute to the disorders comprising syndrome 'X'? [published erratum appears in Br J Nutr 1998 Dec;80(6):585]. Br.J.Nutr. 1998;79:315-27.
- 23 Grant WB An ecologic study of dietary and solar ultraviolet-B links to breast carcinoma mortality rates. Cancer 2002 Jan 1;94(1):272-81
- 24 Polek TC, Weigel NL. Vitamin D and prostate cancer. J Androl. 2002 Jan-Feb;23(1):9-17.
- 25 Luscombe CJ, French ME, Liu S, Prostate cancer risk: associations with ultraviolet radiation, tyrosinase and melanocortin-1 receptor genotypes.Br J Cancer. 2001 Nov;85(10):1504-9.
- 26 Hansen CM, Binderup L, Hamberg KJ, Vitamin D and cancer: effects of 1,25(OH)2D3 and its analogs on growth control and tumorigenesis. Front Biosci. 2001 Jul 1;6:D820-48.

- 27 Tuohimaa P, Lyakhovich A, Aksenov N, Vitamin D and prostate cancer. J Steroid Biochem Mol Biol. 2001 Jan-Mar;76(1-5):125-34
- 28 Mokady E, Schwartz B, Shany S, A protective role of dietary vitamin D3 in rat colon carcinogenesis. Nutr Cancer. 2000;38(1):65-73.
- 29 Platz EA, Hankinson SE, Hollis BW, Plasma 1,25-dihydroxy- and 25-hydroxyvitamin D and adenomatous polyps of the distal colorectum. Cancer Epidemiol Biomarkers Prev. 2000 Oct;9(10):1059-65.
- 30 Tangpricha V, Flanagan JN, Whitlatch LW, 25-hydroxyvitamin D-1alpha-hydroxylase in normal and malignant colon tissue. Lancet. 2001 May 26;357(9269):1673-4.
- 31 Lamprecht SA, Lipkin M. Cellular mechanisms of calcium and vitamin D in the inhibition of colorectal carcinogenesis. Ann N Y Acad Sci. 2001 Dec;952:73-87.
- 32 Majewski S, Kutner A, Jablonska S. Vitamin D analogs in cutaneous malignancies. Curr Pharm Des. 2000 May;6(7):829-38.
- 33 Braun MM, Tucker MA. A role for photoproducts of vitamin D in the etiology of cutaneous melanoma? Med Hypotheses. 1997 Apr;48(4):351-4.
- 34 McAlindon TE, Felson DT, et al. Relation of dietary intake and serum levels of vitamin D to progression of osteoarthritis of the knee among participants in the Framingham Study. Ann Intern Med 1996; 125: 353-359
- 35 Lane NE, Nevitt MC, Gore LR, Cummings SR. Serum levels of vitamin D and hip osteoarthritis in elderly women: a longitudinal study. Arthritis Rheum 1997; 40(suppl): S238.
- 36. Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF. Decreased bioavailability of vitamin D in obesity. Am J Clin Nutr. 2000 Sep;72(3):690-3)