Dynamic Chiropractic

VITAMINS / SUPPLEMENTS

Why Your Patients Need More Vitamin D After Age 45 (Part 2 of 2)

James P. Meschino, DC, MS

Editor's note: Part 1 of this article appeared in the June 3 issue: www.chiroweb.com/archives/22/12/17.html.

Vitamin D Prevents Cancer

Vitamin D receptors exist on intestinal cells and bone to regulate calcium absorption and bone metabolism, as discussed in part one of this article. However, these receptors are also present in a wide variety of other tissues and organs, including the brain, pancreas, skin, gonads, prostate, stomach, colon, breast, kidney, connective tissue, parathyroid gland, mononuclear cells, and activated T- and B-lymphocytes.

Recent studies indicate that tissues expressing vitamin D receptors are able to convert 25-hydroxy vitamin D, which they extract from the bloodstream, into calcitriol for their own internal use. As I have stated, calcitriol is the most potent form of vitamin D; this is not only true with respect to bone support; studies also illustrate that calcitriol exerts a number of anti-cancer effects on local tissues that convert 25-hydroxy vitamin D into calcitriol for their own purposes, such as breast cells, prostate cells and colon cells. As such, circulating levels of 25-hydroxy vitamin D serve as the raw material from which many tissues synthesize calcitriol for their own internal use. Studies reveal that higher amounts of circulating 25-hydroxy vitamin D in the blood enable local tissues, such as breast, prostate and colon cells, to synthesize greater amounts of calcitriol for their own needs.

Epidemiological (observational) studies suggest that lower blood levels of 25-hydroxy vitamin D are associated with a higher risk of developing breast, colon, ovarian, and prostate cancer. This is an important finding, as one in nine women is expected to develop breast cancer in her lifetime, one in eight men is expected to develop prostate cancer in his lifetime, and one in 16 women and one in 15 men will develop colon or rectal cancer in their lifetimes.

Low-Fat Food Sources of Vitamin D	
Foods	Approximate I.U.of vitamin D per 3.5 oz.
sardines (canned)	1150-1570
salmon (fresh)	154-550
salmon (canned)	220-440
herring (fresh)	315
herring (canned)	330
shrimp	150
halibut	44
chicken (raw)	50-67

oysters	5 I.U. per 3-4 medium-sized oysters
nonfat and 1% milk and yogurt (vitamin D-fortified)	100 I.U. per 8 ounces
low-fat cheese (less than 4% milk fat)	12-15

Interestingly, studies show that rates of breast, prostate and colon cancers increase further from the equator (north or south), and that populations residing further from the equator also have lower year-round average blood levels of vitamin D. This is related to the fact that our bodies make vitamin D in response to direct exposure to sunlight (but not through a window). When sunlight hits our skin, it triggers the conversion of 7-dehydrocholesterol into vitamin D (cholecalciferol) within our skin. This enters the bloodstream and travels to the liver, where it is converted to 25-hydroxy vitamin D, which is five times more potent than cholecalciferol in terms of its vitamin D activity.

Thus, populations living closer to the equator, who enjoy more year-round sunlight intensity and exposure, manufacture more cholecalciferol in their skin and demonstrate higher year-round average blood levels of 25-hydroxy vitamin D. As a general statement, populations in North America that live at or above the 42nd latitude have significantly higher rates of breast, colon and prostate cancer than populations living below it. This latitude essentially divides the U.S. into two equal halves: north and south (running through the middle of California, and the tops of Arizona, New Mexico, Texas, Tennessee and the Carolinas). Some exceptions to this observation exist, in that people living in large cities in the South of the U.S. also have higher rates of these cancers per capita.

C.F. Garland and F.C. Garland, who first published these data in the 1980s, explain this finding by indicating that the air pollution in large cities and tall buildings block much of the sunlight, and that city dwellers tend not to wear short-sleeve shirts and shorts as often as country folk, and tend not to be outdoors during the sunniest hours of the day. As such, individuals in large cities in the South do not have blood levels of vitamin D high enough to protect them from breast, colon and prostate cancer, as do individuals living in the more rural parts of the South, according to these researchers.

Overall, studies indicate that vitamin D blood levels of 85-120 ng/mL are associated with a high degree of protection with respect to risk of breast, prostate and colon cancer, and may significantly reduce the risk of developing multiple sclerosis via immune-modifying influences.

How does calcitriol reduce cancer risk in the breast, prostate, colon and possibly other tissues? Experimental studies reveal that calcitriol exhibits a number of anti-cancer effects. Essentially, prostate, breast, colon and other cells that contain vitamin D receptors extract 25-hydroxy vitamin D from the bloodstream, and convert it into calcitriol - once inside the cell. This, in turn, slows down the rate of replication of these cells, an effect associated with decreased cancer development.

The presence of calcitriol has also been shown to slow the rate of replication of human prostate, breast and colon cancer cells, under experimental conditions. Calcitriol also promotes newly formed cells to mature to their full adult potential, which also decreases the chance of these cells being transformed into cancer cells by some external influence. Calcitriol also exerts a favorable effect on immune function, which is thought to account for some of its anti-cancer influences. It also has been shown to transform the appearance of human cancer cells (e.g., prostate cancer cells) back to healthy, nonmalignant-looking cells, and inhibit their replication, an effect that is lost

once the calcitriol is no longer administered.

The problem with relying upon vitamin D from sunlight and food sources: From an evolutionary standpoint, exposure to direct sunlight is the principal way in which we are set up to derive our vitamin D stores. To maximize vitamin D synthesis within the skin, all that is required is 15-20 minutes of direct sunlight exposure to the face, arms and legs, three times per week. However, experts warn that even this amount of cumulative sun exposure increases risk of skin cancer over our lifetime, and that it is best to derive vitamin D from the consumption of fish, vitamin D-fortified dairy products and supplements. The point is that many people don't get 15-30 minutes of direct sunlight exposure each day, especially those of us living above the 42 degrees latitude within North America, where sunlight intensity between October and May is insufficient for our bodies to make vitamin D inside our skin.

Very few foods contain vitamin D in their native form. It is best to eat fatty fish, such as sardines, salmon and mackerel, 3-4 times per week, to help satisfy the body's requirement. Of course, there is supposed to be 100 IU of vitamin D in every eight ounces of fortified milk, but studies undertaken by M. Holick and others showed that nearly two-thirds of the whole milk samples tested in one study had less than 80 percent (and several skim milk samples had between zero percent and 50 percent) of the amount of vitamin D appearing on the label. This problem will continue, as vitamin D levels in milk are affected by season; the breed of cow; the animal's diet; its exposure to sunlight; and procedures used in fortification.

Although the 1997 recommendations by the Institute of Medicine suggest that middle-aged adults (50-70 years old) should consume 400 IU of vitamin D per day, and older subjects should consume 600 IU per day, evidence is strong to indicate that in the absence of exposure to sunlight, the adequate intake for vitamin D should be at least 800-1,000 IU per day by age 45-50. M. Holick points out that intake is completely safe up to 2,000 IU per day for ages 1 year and above, and that the risk of vitamin D toxicity is greatly exaggerated by many health policy-makers.

Ensuring Optimal Vitamin D Status

The totality of evidence suggests that many North Americans are either vitamin D deficient - or more commonly, insufficient - and would benefit from additional supplementation. Most multivitamins contain 400 IU of vitamin D, an amount that is reported to raise vitamin D blood levels (25-hydroxy vitamin D) by approximately 45 ng/mL. However, even higher amounts of total supplemental vitamin D (800-1,000 IU per day) should be implemented after the age of 45 or 50, as the body's ability to convert 25-hydroxy vitamin D to calcitriol slows down to a significant degree.

Studies prove that higher blood levels of the less potent 25-hydroxy vitamin D (in the range of 85-120 ng/mL) can compensate for the reduced synthesis and availability of calcitriol in the bloodstream after ages 45-50, and can significantly reduce your risk of osteoporosis. Furthermore, this amount is also strongly associated with a reduction in risk of breast, colon, prostate and ovarian cancers, as well as multiple sclerosis.

In a study released in 2004, K.L. Munger and fellow researchers showed that participants in the Nurses' Health Study who ingested 400 IU of vitamin D from supplements daily (most notably from a multivitamin product) showed a 40 percent reduction in risk of multiple sclerosis compared to women who did not use supplements containing vitamin D. This group of 95,253 female registered nurses, residing in the United States, has been followed by researchers since 1980.

Vitamin D has been shown to exert favorable influences on immune cells that are consistent with preventing events related to multiple sclerosis, which have been confirmed in animal and human

investigations. Over and above supplementation, note that sardines, mackerel, herring and salmon are excellent food sources of vitamin D, as well.

Resources (for parts 1 and 2)

1. Brodie MJ, et al. Rifampicin and vitamin D metabolism. *Clin Pharmacol Ther* 1980;27(6):810-4.
2. Chapuy MC, et al. Effect of calcium and chole-calciferol treatment for three years on hip fractures in elderly women. *Br Med J* 1994; 308:1081-2.
3. Chen TC, Holick MF. Vitamin D and prostate cancer prevention and treatment. *TEM* November 2003;14(9):423-30.
4. Chesney RW, et al. Decreased serum 24,25-dihydroxyvitamin D3 in children receiving glucocorticoids. *Lancet* 1978;2(8100):1123-5.
5. Crowle AJ, et al. Inhibition by 1,25 dihydroxy vitamin D3, of the multiplication of virulent tubercle bacilli in cultured human macrophages. *Infect Immun*,1987; 55:2945-50.
6. Dawson-Hughes B, et al. Effect of calcium and vitamin D supplementation on bone density in men and women 65 years of age or older. *N Engl J Med* 1997;337:670-6.
7. Dawson-Hughes B, et al. Rates of bone loss in postmenopausal women randomly assigned to one of two dosages of the vitamin D. *Am J Clin Nutr* 1995; 61: 1140-5.
8. Dawson-Hughes B. Calcium and Vitamin D requirements of elderly women. *J Nutr* 1996;126 (Suppl 4): 1165S-7S.
9. DeLuca HF. The vitamin D story: a collaborative effort of basic science and clinical medicine. *FASEB J* 1988;2:224-36.
10. Diarrhea and constipation. In: Berkow R, Fletcher AJ, Beers MH, et al, editors. *The Merck Manual of Diagnosis and Therapy*, 16th ed. Rahway, NJ: Merck Research Laboratories, 1992.
11. Feldman D, et al. Vitamin D and prostate cancer. *Adv Exp Med Biol* 1995;375:53-63.
12. Fukazawa T, et al. Association of vitamin D receptor gene polymorphism with multiple sclerosis in Japanese. *J Neurol Sci* 1999; 166(1): 47-52.
13. Gahn PH, et al. Circulating Vitamin D metabolites in relation to subsequent development of prostate cancer. *Epidemiol Biomarkers Prev* 1995;5(2):121-6.
14. Garland CF, et al. Can colon cancer incidence and death rates be reduced with calcium and vitamin D? *Am J Clin Nutr* 1991; 54(Suppl 1):193S-201S.
15. Garland CF, Garland FC, Gorham ED. Calcuim and vitamin D. Their potential roles in colon and breast cancer prevention. *Ann NY Acad Sci* 1999;889:107-19.
16. Garland FC, et al. Geographic variation in breast cancer mortality in the United States: A hypothesis involving exposure to solar radiation. *Prev Med* 1970; 19:614-22.
17. Hayes C, et al. Vitamin D and multiple sclerosis. *Proc Soc Exper Biol Med* 1997;216:21-7.
18. Healthnotes, 2000 Inc. Available from: www.healthnotes.com.
19. Holick M. Too little vitamin D in premenopausal women: why should we care? *Am J Clin Nutr* 2002;76:3-4.
20. In the news...vitamin D and colon cancer. Harvard Women's *Health Watch* February 2004;11 (6):7.
21. James WP, Avenell A, Broom J, et al. A one-year trial to assess the value of orlistat in the management of obesity. *Int J Obes Relat Metab Disord* 1997;21 (Suppl3): 24S-30S.
22. K‡llay E, Adlercreutz H, Farhan H, et al. Phytoestrogens regulate vitamin D metabolism in the mouse colon: relevance for colon tumor prevention and therapy. *J Nutr* November 2001;132(11):3490S-3493S.
23. Knodel LC, et al. Adverse effects of hypolipidaemic drugs. *Med Toxicol* 1987;2(1):10-32.
24. Lore F, et al. Vitamin D metabolites in postmenopausal osteoporosis. *Horm Metab Res* 1984; 16:161-6.
25. Martinez ME, et al. Calcium, vitamin D and the occurrence of colorectal cancer among women. *J Natl Cancer Instit* 1996;88(19):1375-82.
26. Mehta RG, et al. Prevention of preneoplastic mammary lesion development by a novel vitamin D analogue, 1-alpha-hydroxyvitamin D5. *J Natl Cancer Instit* 1997;89 (3):212-8.

27. Munger KL et al. Vitamin D intake an incidence of multiple sclerosis. *Neurology* 2004;62:60-65.
28. Odes HS, et al. Effect of cimetidine on hepatic vitamin D metabolism in humans. *Digestion* 1990; 46(2):61-4.
29. Optimal calcium intake: NIH consensus conference. *JAMA*1994; 272(24):1942-8.
30. Peehl DM. Vitamin D and prostate cancer risk. *Eur Urol* 1999;35 (5-6):392-4.
31. Rozen F, Yang XF, Huynh H, Pollak M. Antiproliferative action of Vitamin-D-related compounds and insulin-like growth factor - binding protein 5 accumulation. *J Natl Cancer Instit* 1997;89(3):652-6.
32. Schmidt J, Wittenhagen P, H¿rder M. Molecular effects of vitamin D on cell cycle and oncogenesis. *Ugeskrift for laeger*, July 20, 1998;160(30):4411-4.
33. Shabahang M, et al. Growth inhibition of HT-29 human colon cancer cells by analogues of 1,25 dihydroxy vitamin D3. *Cancer Res* 1994;54:407-64.
34. Toppet M, et al. Sequential development of vitamin D metabolites under isoniazid and rifampicin therapy. *Arch Fr Pediatr* 1998;45(2): 145-8.
35. Veith R. Vitamin D supplementation, 25-hydroxy vitamin D concentrations and safety. *Am J Clin Nutr* 1999;69(5):842-56.
36. Zerwekh JE, et al. Decreased serum 24,25-dihydroxyvitamin D concentration during long-term anticonvulsant therapy in adult epileptics. *Ann Nerol* 1982;12(2):184-6.

James Meschino, DC, MS
Toronto, Ontario
Canada
www.renaisante.com

JULY 2004