Dynamic Chiropractic

NUTRITION / DETOXIFICATION

Why Your Patients Need More Vitamin D After Age 45 (Part 1 of 2)

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Vitamin D acts in the body as both a vitamin and a hormone, exerting a powerful influence on maintaining bone density and preventing vital steps in the development of breast, prostate and colon cancers (and possibly others). There is also evidence to suggest that more optimal vitamin D levels can reduce the risk of multiple sclerosis by affecting immune system function.

Throughout younger adult life, supplementation of 400 IU of vitamin D per day from a high-potency multivitamin/mineral, combined with the normal amounts most people acquire from fortified dairy products and fish, is usually adequate to maintain blood levels of vitamin D in a range that is associated with healthy bone development; the prevention of breast, prostate and colon cancers; and multiple sclerosis, according to available studies. However, by age 45-50, the enzyme in the kidney (alpha-hydroxylase) that converts vitamin D into its most active form (1,25 dihydroxy vitamin D, also known as calcitriol) becomes less active. This results in decreased synthesis of calcitriol; thus, tissues that rely on its health-promoting influence are left to feel the effects of insufficiency.

Studies strongly suggest that the age-related decline in calcitriol synthesis in the kidneys is a major contributing factor to the development of osteoporosis in women and men older than age 50, as well as the age-related increase in risk of breast, prostate and colon cancers that is common in North America and many industrialized countries. However, studies also indicate that individuals older than age 45 can compensate for the decline in calcitriol synthesis by raising their blood levels of the less potent form: 25-hydroxy vitamin D.

Abundant evidence exists suggesting that adults who maintain blood levels of this form of vitamin D in the range of 85-120 ng/mL (nanograms per milliliter) have a significantly lower risk of developing osteoporosis, breast cancer, ovarian cancer, prostate cancer, colon cancer and multiple sclerosis. Studies also show that to ensure blood levels of 25-hydroxy vitamin D are within this protective range, it is important to increase vitamin D supplementation to 800-1,000 IU per day after the age of 45. Until then, 400 IU per day from a high-potency multivitamin/mineral combination is adequate to meet vitamin D needs in most cases. However, I strongly advise the addition of a daily supplement that contains 400-600 IU of vitamin D after age 45, in a formulation that also provides an additional 500-700 mg of calcium. (It is perfectly acceptable if the product also contains some additional magnesium, zinc and other bone support nutrients.)

Vitamin D and Bone Density

Vitamin D is required at all stages in life to optimize calcium absorption from the intestinal tract, which, in turn, helps increase bone mineral density during the developmental years and helps prevent the loss of calcium from bone after age 50. The increased deposition of calcium into our bones that occurs at an optimal rate up to age 24, and the prevention of calcium being leached from bones after

age 50, are both essential aspects related to the prevention of osteoporosis.

Unfortunately, vitamin D deficiency is widespread in North America, especially in the more northern regions (above 42 degrees latitude), where sunlight intensity is very limited between October and May. Older individuals have also been shown to be at very high risk for vitamin D deficiency, due to poor intake of vitamin D-containing foods, reduced sunlight exposure and reduced conversion of 25-hydroxy vitamin D into calcitriol (which is twice as potent as 25-hydroxy vitamin D with respect to influence on calcium metabolism, our bones and other tissues). Vitamin D deficiency correlates with a blood level of 25-hydroxy vitamin D below 20 ng/mL. Optimal levels are considered to be in the range of 85-120 ng/mL. It is this range of vitamin D status that is most strongly associated with a reduced risk of osteoporosis, many cancers and multiple sclerosis. Unfortunately, a great number of adults fall into the range above the deficiency threshold and below the optimal range of vitamin D blood levels, defined as a blood level of 21-79 ng/mL. This middle ground is often referred to as vitamin D insufficiency.

Your Bones Need More Vitamin D Support After Age 45

As mentioned, after the age of 45-50, the body is less able to convert 25-hydroxy vitamin D into calcitriol, due to a decline in the activity of the kidney enzyme known as alpha-hydroxylase. However, if blood levels of 25-hydroxy vitamin D can be raised into the range of 85-120 ng/mL, this amount has been shown to compensate for the drop-off in calcitriol levels. Studies show that supplementation with 800-1,000 IU of vitamin D per day after age 45 can elevate blood levels of 25-hydroxy vitamin D into the ideal range of 85-120 ng/mL, and has been shown to reduce risk of osteoporotic fractures by more than 40%. Let's look at several studies that clearly illustrate this point.

In a 1994 study involving 3,720 elderly women living in nursing homes, those supplemented with 1,200 mg of calcium and 800 IU of vitamin D daily showed a 43% reduction in hip fractures over a three-year period, compared to those not taking such supplements. A 1995 study showed that supplementation of postmenopausal women with 700 IU of vitamin D daily reduced the annual rate of hip fractures from 1.3% to 0.5%, nearly a 60% reduction. A three-year follow-up study showed that supplementation with 500 mg of calcium and 700 IU of vitamin D resulted in a significantly reduced hip fracture rate in men and women taking this combination, compared to the placebo group.

Ample evidence now suggests that after age 45, it is extremely prudent to increase your vitamin D supplementation to reach a total daily value of 800-1,000 IU, in order to significantly reduce your risk of osteoporosis. This advice is meant for both men and women, as osteoporosis now affects one in four women and one in eight men older than age 50. However, the benefits of increased vitamin D supplementation don't end with the prevention of osteoporosis; there are also important implications for the prevention of breast, ovarian, prostate and colon cancer, as we shall explore in part two of this article.

- 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* 2003 Nov;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(Suppl4):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: URL: 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 2004* Feb; Vol. 11(6)p7.
- 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. Kellay E, Adlercreutz H, Farhan H, Lechner D, Bajna E, Gerdenitsch W, Campbell M, Cross HS. Phytoestrogens regulate vitamin D metabolism in the mouse colon: relevance for colon tumor prevention and therapy. *J Nutr* 2001 Nov;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. IAMA 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, Harder M. Molecular effects of vitamin D on cell cycle and oncogenesis. *Ugeskrift for laeger* 1998 Jul 20;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.

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