Published in final edited form as:

South Med J. 2007 April ; 100(4): 384–385.

Vitamin D Deficiency in the Southern United States

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Vitamin D is an essential nutrient for optimal calcium homeostasis for the body. Vitamin D is a secosteroid hormone (“seco” meaning “cut”) with two forms: D₂ found in the diet, and D₃, which is either found in the diet or made in the skin from 7-dehydrocholesterol (7-DHC) upon exposure to ultraviolet B radiation (UVB) between wavelengths of 290 and 315 nm. Upon entering the circulation, vitamin D (D without subscripts refers to either D₂ or D₃) undergoes two sequential hydroxylations. The first hydroxylation occurs in the liver in the 25 position to form 25-hydroxyvitamin D (25(OH)D), the major circulating form of vitamin D with a half-life of 2–3 weeks. The second hydroxylation happens in the kidney in the 1 position to create 1,25(OH)₂D, the active form. The major role of vitamin D is to maintain adequate serum calcium and phosphorus levels for proper mineralization of bone by increased intestinal efficacy of calcium and phosphorus absorption from the small intestine.

Bone is comprised of an extracellular collagen matrix that is mineralized with hydroxyapatite crystals (Ca₁₀(PO₄)₆(OH)₂). Inadequate calcium and phosphorus levels resulting in poorly mineralized matrix caused by vitamin D deficiency is referred to as osteomalacia in adults and as rickets in children. Previously it was believed that vitamin D deficiency only occurred in older patients who were either hospitalized¹ or lived in nursing homes²; however, it has become apparent that vitamin D deficiency affects adults of all ages, including young adults between the ages of 18 to 29, who have a 36% prevalence of vitamin D deficiency at the end of the winter.³

Subjects living at higher latitudes are at increased risk for developing vitamin D deficiency due to decreased UVB radiation reaching the earth's surface, especially during the winter months when the angle of the sun is most oblique. In fact, during the winter months in Boston, (42°N) vitamin D can not be made in the skin between November and February.⁴ Other populations at risk for vitamin D deficiency include African Americans due to melanin effectively absorbing solar radiation between 290 to 700 nm (in the area of vitamin D production),⁵ obese patients due to fat sequestration of vitamin D,⁶ and patients with fat malabsorption.⁷

Several studies have revealed that there is also an alarmingly high prevalence of vitamin D deficiency in a lower latitude of the United States. One study, by Levis et al, found the prevalence of vitamin D deficiency in an ambulatory population of men and women in Miami, Florida (25° N) to be 38–40% at the end of winter.⁸ A study examining the prevalence of vitamin D deficiency in North America in women receiving osteoporosis therapy found a prevalence of vitamin D deficiency of 18% in the early winter months. There was no significant difference in the prevalence rate of vitamin D deficiency when three different latitudes were compared >42°, 35 to 42° and <35° N.

The reasons for which living in lower latitudes does not protect against vitamin D deficiency is likely due to the fact that Americans are spending less time outdoors and/or are using more sunscreen for skin cancer protection. Holick estimates that 10 to 15 minutes of sunlight exposure to 10% of the uncovered skin would result in about 1,000 IU of vitamin D being made in the skin.⁹ This amount would satisfy the body's daily requirement for vitamin D. In addition, physicians should recommend and encourage vitamin D supplementation in patients who do not have adequate sunlight exposure or already have low bone mass. The current established adequate intakes for vitamin D for most adults are between 400 to 600 IU. In the absence of sunlight or during the winter months, most adults will need 800 to 1,000 IU to maintain normal vitamin D stores.¹⁰

Those adults who are at highest risk for vitamin D deficiency or need to have optimal calcium absorption (ie, patients with osteoporosis) should have their vitamin D status determined by measuring a 25(OH)D level. There is some debate over the appropriate thresholds for determining vitamin D deficiency. Previously, Malabanan et al determined that a minimum 25(OH)D level of 20 ng/mL prevents secondary hyperparathyroidism.¹¹ Chapuy et al demonstrated that levels of PTH start to rise when 25(OH)D levels fall below 31 ng/mL.¹² Heaney et al have determined that a 25(OH)D level of at least 34 ng/mL is best to optimize calcium absorption.¹³ Most osteoporosis experts would agree that patients should have a 25(OH)D level of at least 30 to 32 ng/mL.¹⁴ Patients who are diagnosed with vitamin D deficiency should first be corrected with vitamin D 50,000 IU once a week for 8 weeks before going on a maintenance dose of vitamin D.

There is increasing evidence that vitamin D may have other nonskeletal effects. Several clinical trials have demonstrated that vitamin D may improve muscular strength and result in the reduction of falls.¹⁵ Higher 25(OH)D levels also have been determined to be protective against developing several different cancers including colon, breast and prostate.¹⁶ Finally, vitamin D may further modulate the immune system resulting in decreased type 1 diabetes mellitus and multiple sclerosis in mice models.^{17,18}

Physicians should not assume that vitamin D status is optimal in patients living in the southern United States. Patients should be questioned about dietary intake of vitamin D-containing foods, risk factors for developing vitamin D deficiency and exposure to sunlight. For those patients who are at high risk of vitamin D deficiency or have osteoporosis, it is prudent to obtain a 25(OH)D level and to correct vitamin D status before going on a maintenance dose of vitamin D.

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