Vitamin D for Chronic Pain

According to a comprehensive review of the clinical research evidence, helping certain patients overcome chronic musculoskeletal pain and fatigue syndromes may be as simple, well tolerated, and inexpensive as a daily supplement of vitamin D.

By Stewart B. Leavitt, MA, PhD

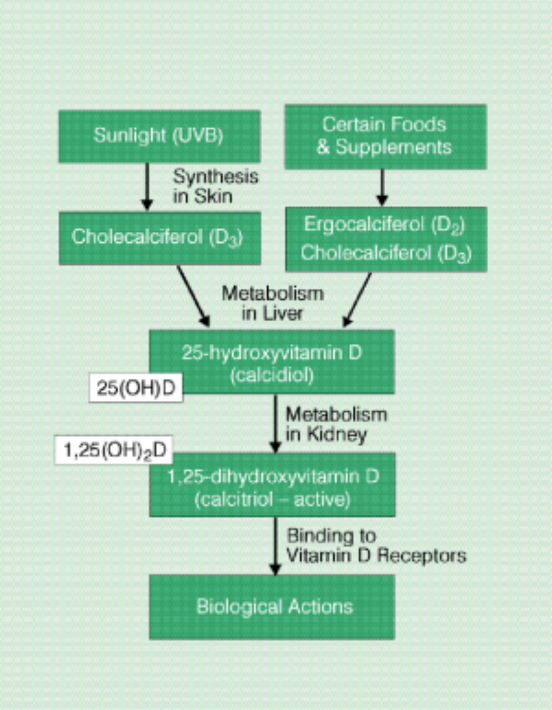
This article is adapted from the author's peer-reviewed research report, Vitamin D— A Neglected 'Analgesic' for Chronic Musculoskeletal Pain, from Pain Treatment Topics. The full report, along with a special brochure for patients, is available at www.pain-

Standing apart from the various other essential nutrients, vitamin D was spotlighted recently as having special therapeutic potential. This has important implications for the management of chronic musculoskeletal pain and fatigue syndromes.

During this past June 2008, news-media headlines heralded recent clinical research that revealed benefits of vitamin D for preventing type 1 diabetes,1 promoting survival from certain cancers,2 and decreasing the risks of coronary heart disease.3 Overlooked, however, was the traditional role of vitamin D in promoting musculoskeletal health and the considerable evidence demonstrating advantages of vitamin D therapy in helping to alleviate chronic muscle, bone and joint aches, and pains of various types.

Chronic pain—persisting more than 3 months—is a common problem leading patients to seek medical care.4-7 In many cases, the causes are nonspecific, without evidence of injury, disease, or neurological or anatomical defect.8,9 However, according to extensive clinical research examining adult patients of all ages, inadequate concentrations of vitamin D have been linked to nonspecific muscle, bone, or joint pain, muscle weakness or fatigue, fibromyalgia syndrome, rheumatic disorders, osteoarthritis, hyperesthesia, migraine headaches, and other chronic somatic complaints. It also has been implicated in the mood disturbances of chronic fatigue syndrome and seasonal affective disorder.

Although further research would be helpful, current best evidence demonstrates that supplemental vitamin D can help many patients who have been unresponsive to other therapies for pain. Vitamin D therapy is easy for patients to self-administer, well-tolerated, and very economical.



(http://www.practicalpainmanagement.com/sites/default/files/imagecache/lightbox-large/import_files/html/P0807F02/fig1.png)

Figure 1. *Vitamin D Metabolism*

It must be emphasized that vitamin D is not a pharmaceutical analgesic in the sense of fostering relatively immediate pain relief, and expectations along those lines would be unrealistic. Because Vitamin D supplementation addresses underlying processes, it may take months to facilitate pain relief, which can range from partial to complete. Furthermore, vitamin D supplementation is not proposed as a panacea or as a replacement for other pain treatment modalities that may benefit patient care.

Vitamin D and 'D-ficiency'

Pharmacology. Vitamin D comprises a group of fat-soluble micronutrients with two major forms: D2 (ergocalciferol) and D3 (cholecalciferol)10,11 (see Figure 1). Vitamin D3 is synthesized in the skin via exposure of endogenous 7-dehydrocholesterol to direct ultraviolet B (UVB) radiation in sunlight and is also obtained to a small extent in the diet (see Table 1). In many countries, some foods are fortified with vitamin D3, which is the form used in most nutritional supplements.14,20 Vitamin D2, on the other hand, is found in relatively few foods or supplements.14

Following vitamin D synthesis in the skin or other intake, some of it is stored in adipose tissue, skeletal muscle, and many organs,19 while a relatively small portion undergoes a two-stage process of metabolism (see Figure 1). First, D2 and/or D3 are metabolized via hydroxylation in the liver to form 25-hydroxyvitamin D, abbreviated as 25(OH)D (also called calcidiol).10,14,18,21,22 This has minimal biological activity and serum concentrations of 25(OH)D accumulate gradually, plateauing at steady-state levels by about 40 days19,23,24 to 90 days.25,26

The 25(OH)D metabolite is converted primarily in the kidneys via further hydroxylation to 1,25-dihydroxyvitamin D, abbreviated as 1,25(OH)2D (also called calcitriol). It is the most important and biologically-active vitamin D metabolite with a short half-life of only 4 to 6 hours27 but can remain active for 3 to 5 days.15,20 A central role of vitamin D—via its active 1,25(OH)2D metabolite—is to facilitate the absorption of calcium from the intestine and help maintain normal concentrations of this vital agent. Equally important, 1,25(OH)2D sustains a wide range of metabolic and physiologic functions throughout the body.28

Vitamin D actually is misclassified as a vitamin; it may be more appropriately considered a prohormone and its active 1,25(OH)2D metabolite—with its own receptors found in practically every human tissue—functions as a hormone. These vitamin D receptors, or VDRs, may affect the function of up to 1000 different genes18,22,29,30,31 helping to control cell growth or differentiation. The VDRs themselves can differ in their genetic makeup (poly-morphism) and activity, which may account for varying individual responses to vitamin D therapy.32,33

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