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Perspective

Extraskeletal Effects of Vitamin D: Including the Immune Function Regulation

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DESCRIPTION

When sufficient ultraviolet B radiation is present, the skin can naturally produce vitamin D by the conversion of the cholesterol precursor 7-dehydrocholesterol through photochemical and thermal processes. Only at sun angles greater than 45 degrees does cutaneous vitamin D synthesis take place effectively. In many major population centers in Europe and North America, it does not occur during six months of the year. To become physiologically active, dietary or topically applied vitamin D passes through a series of hydroxylation, primarily a hepatic 25-hydroxylation catalyzed by CYP2R1 and other enzymes, followed by a 1-hydroxylation in peripheral tissues catalyzed by CYP27B1. With a half-life of several weeks and seasonal variations in cutaneous vitamin D production, 25-hydroxyvitamin D (25D), the main circulating metabolite of vitamin D, is a steroid hormone. 1,25-dihydroxyvitamin D (1,25D), the active form of vitamin D, binds to the Vitamin D Receptor (VDR), a member of the nuclear receptor family of ligand-regulated transcription factors, which mostly but not entirely controls gene transcription to have physiological effects. Significantly, the gene that codes for CYP24A1, the enzyme that starts the breakdown of both 25D and 1,25D, is the most strongly stimulated 1,25D target gene. This gene thus functions as a physiological negative feedback loop.

The classic effects of vitamin D on musculoskeletal health are related to its most well-known roles in the metabolic and signaling processes of the body. Studies on vitamin D's non-classical, extraskeletal activities, however, have proliferated rapidly in recent years. Among these, the rising corpus of research connecting vitamin D and the immune system has stood out. Given the apparent prevalence of vitamin D deficiency in areas where Coronavirus Disease 2019 (COVID-19) infection and disease severity are equally pronounced, the potential role of vitamin D as an endogenous regulator of both innate and adaptive immunity has attracted a lot of attention in recent months.

1,25-D was first believed to only be a calcium homeostatic hormone until vitamin D was found as the treatment for nutritional rickets. However, there are connections between vitamin D and infections that date back to the ancient Greeks' use of heliotherapy to treat phthisis (Tuberculosis [TB]). When the sanatorium movement began in Europe in the middle of the 1800s to treat tuberculosis, and it was later shown that Ultraviolet (UV) light might also treat cutaneous TB, the idea was revived (lupus vulgaris). Epidemiological observations (e.g., respiratory tract infections) have recently demonstrated a protective effect for vitamin D in infectious diseases and autoimmune disorders such as multiple sclerosis and type 1 diabetes mellitus. According to a rapidly growing body of preclinical studies, vitamin D signaling is active in both the innate and adaptive arms of the immune system. The innate immune system has multiple so-called "pattern recognition receptors" (PRRs) built in that enable it to recognize a variety of diseases. Although there are many more, the two main groups of PRRs are Toll-Like Receptors (TLRs) and NOD- (Nucleotide-binding Oligomerization Domain) like receptors. Many receptor-specific pathogen-associated molecular patterns trigger the activation of PRRs (PAMPs). As a result of PRR signaling, several cytokines are produced that attract additional immune system cells to the site of infection, as well as antimicrobial peptides that have immediate antibacterial and antiviral effects.

Almost all immune system cells in both the innate and adaptive arms express vitamin D metabolic enzymes. Overall, clinical and experimental findings suggest a significant relationship between vitamin D level and vulnerability to infectious and autoimmune diseases. There is proof that a lack of vitamin D in a child's immune system increases the chance of allergies or autoimmune diseases. Support for vitamin D's potential function in preventing respiratory tract infections has come from numerous laboratory and clinical studies. The ongoing COVID-19 crisis and the treatment of other infectious diseases may both benefit clinically and financially from the evaluation of vitamin D supplementation as an adjuvant therapeutic intervention. According to the immunoregulatory abilities of vitamin D that were discussed above, increasing the level of circulating 25D may decrease the progression of disease or perhaps improve patient survival. Despite the overwhelming evidence for a mechanistic role for vitamin D signaling in immune system regulation, large-scale randomized controlled trials are still needed to determine whether maintaining adequate vitamin D levels reduces the incidence and severity of infections and/or autoimmune diseases.

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