
1: Autoimmun Rev. 2009 Jul;8(8):639-44. Epub 2009 Feb 12.

Vitamin D: the alternative hypothesis.

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Early studies on vitamin D showed promise that various forms of the "vitamin" may be protective against chronic disease, yet systematic reviews and longer-term studies have failed to confirm these findings. A number of studies have suggested that patients with autoimmune diagnoses are deficient in 25-hydroxyvitamin D (25-D) and that consuming greater quantities of vitamin D, which further elevates 25 D levels, alleviates autoimmune disease symptoms. Some years ago, molecular biology identified 25 D as a secosteroid. Secosteroids would typically be expected to depress inflammation, which is in line with the reports of symptomatic improvement. The simplistic first-order mass-action model used to guide the early vitamin studies is now giving way to a more complex description of action. When active, the Vitamin D nuclear receptor (VDR) affects transcription of at least 913 genes and impacts processes ranging from calcium metabolism to expression of key antimicrobial peptides. Additionally, recent research on the Human Microbiome shows that bacteria are far more pervasive than previously thought, increasing the possibility that autoimmune disease is bacterial in origin. Emerging molecular evidence suggests that symptomatic improvements among those administered vitamin D is the result of 25-D's ability to temper bacterial-induced inflammation by slowing VDR activity. While this results in short-term palliation, persistent pathogens that may influence disease progression, proliferate over the long-term.

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