

Vitamin D may exacerbate autoimmune disease

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Deficiency in vitamin D has been widely regarded as contributing to autoimmune disease, but a review appearing in *Autoimmunity Reviews* explains that low levels of vitamin D in patients with autoimmune disease may be a result rather than a cause of disease and that supplementing with vitamin D may actually exacerbate autoimmune disease.

Authored by a team of researchers at the California-based non-profit Autoimmunity Research Foundation, the paper goes on to point out that molecular biologists have long known that the form of [vitamin D](#) derived from food and supplements, 25-hydroxyvitamin D (25-D), is a secosteroid rather than a vitamin. Like corticosteroid medications, vitamin D may provide short-term relief by lowering [inflammation](#) but may exacerbate disease symptoms over the long-term.

The insights are based on molecular research showing that 25-D inactivates rather than activates its native receptor - the Vitamin D nuclear receptor or VDR. Once associated solely with calcium metabolism, the VDR is now known to transcribe at least 913 [genes](#) and largely control the innate immune response by expressing the bulk of the body's antimicrobial peptides, natural antimicrobials that target bacteria.

Written under the guidance of professor Trevor Marshall of Murdoch University, Western Australia, the paper contends that 25-D's actions must be considered in light of recent research on the Human Microbiome. Such research shows that bacteria are far more pervasive

than previously thought - 90% of cells in the body are estimated to be non-human - increasing the likelihood that [autoimmune diseases](#) are caused by persistent [pathogens](#), many of which have yet to be named or have their DNA characterized.

Marshall and team explain that by deactivating the VDR and subsequently the immune response, 25-D lowers the inflammation caused by many of these bacteria but allows them to spread more easily in the long-run. They outline how long-term harm caused by high levels of 25-D has been missed because the bacteria implicated in autoimmune disease grow very slowly. For example, a higher incidence in brain lesions, allergies, and atopy in response to vitamin D supplementation have been noted only after decades of supplementation with the secosteroid.

Furthermore, low levels of 25-D are frequently noted in patients with autoimmune disease, leading to a current consensus that a deficiency of the secosteroid may contribute to the autoimmune disease process. However, Marshall and team explain that these low levels of 25-D are a result, rather than a cause, of the disease process. Indeed, Marshall's research shows that in autoimmune disease, 25-D levels are naturally down-regulated in response to VDR dysregulation by chronic pathogens. Under such circumstances, supplementation with extra vitamin D is not only counterproductive but harmful, as it slows the ability of the immune system to deal with such bacteria.

"Vitamin D is currently being recommended at historically unprecedented doses," states Proal. "Yet at the same time, the rate of nearly every autoimmune disease continues to escalate."

More information: Albert PJ et al. In press. Autoimmunity Reviews. "Vitamin D: The alternative hypothesis."

Full-text preprint: autoimmunityresearch.org/trans ... s/AR-Albert-

[VitD.pdf](#)

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