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VDR Nuclear Receptor Competence is the Key to Recovery from Chronic Inflammatory and Autoimmune Disease

The VDR Nuclear Receptor is at the heart of human innate immunity, responsible for TLR2, TLR4, CAMP, TACO and IL2 expression[1]. During Th1 immune challenge, the VDR is activated by the endogenous secosteroid 1,25dihydroxyvitamin-D. We have previously described how intra-phagocytic bacterial pathogens are responsible for much chronic inflammatory disease[2,3], and our phase 2 study results have confirmed this pathogenesis. In order to induce recovery from chronic inflammatory disease, it is necessary to restore VDR functionality by removing all exogenous sources of the secosteroid we call 'Vitamin-D', and dampen down over-exuberant VDR activity, for example with the ARB Olmesartan[1]. This enables the immune system to recognize the pathogens. To date we have demonstrated recovery from Hashimoto's Thyroiditis, Rheumatoid Arthritis, Sarcoidosis, and an assortment of chronic inflammatory diagnoses. This breakthrough is the result of a collaboration between molecular scientists and a disparate group of innovative physicians, facilitated by the Internet. However, the widespread application of this pathogenic understanding will require meticulous translation of the molecular science into conventional clinical precepts.

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2. Marshall TG, Marshall FE: Sarcoidosis succumbs to antibiotics-implications for autoimmune disease. *Autoimmunity Reviews*, 2004;3(4):295-3001.
3. Marshall TG, Fenter B, Marshall FE: Antibacterial Therapy Induces Remission in Sarcoidosis. *Herald MKDTS* 2004g; Volume.III:Release.1(The Journal of the Interregional Clinical-Diagnostic Center, Kazan, in Russian translation).ISSN:1726-6149.