

JCSMR WEDNESDAY SEMINARS 2006

MOLECULAR MECHANISMS DRIVING THE CURRENT EPIDEMIC OF CHRONIC DISEASE

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[The Finkel Lecture Theatre](#)

The John Curtin School of Medical Research,
Building 54, Garran Road, ANU



It has long been thought that the diseases termed 'autoimmune' result from 'antibodies to self', and treatment regimes have therefore been based on immunosuppression. However, we identified that several idiopathic chronic diseases, including many of the 'autoimmune' diagnoses, in fact result from a defect in innate immunity, exacerbated during the 20th Century. The VDR Nuclear Receptor is at the heart of human innate immunity, responsible, inter alia, for TLR2, TLR4, CAMP, TACO and IL2 expression. During Th1 immune challenge, the VDR is activated by the endogenous secosteroid 1,25-dihydroxyvitamin-D, and de-activated by its precursor, 25-hydroxyvitamin-D. Our Phase 2 study results confirm that 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 by the use of a VDR partial agonist, for example the ARB Olmesartan. This enables the immune system to recognize the pathogens. To date we have demonstrated recovery from Hashimoto's thyroiditis, rheumatoid arthritis, sarcoidosis, CFS, 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.

[If you would like to meet with Dr Marshall, please contact Rohan.Baker@anu.edu.au](mailto:Rohan.Baker@anu.edu.au)

[Drinks and nibbles provided after the seminar](#)

The views expressed in this lecture are those of the presenter and do not necessarily represent the views of The Australian National University