Meg Mangin, J Clin Cell Immunol 2014, 5:5
http://dx.doi.org/10.4172/2155-9899.S1.017

OMICS Group Conferences
Accelerating Scientific Discovery

3rd International Conference and Exhibition on
Clinical & Cellular Immunology
September 29-October 01, 2014 DoubleTree by Hilton Baltimore-BWI Airport, USA

# Clinical evidence of immune system dysregulation caused by intracellular infection

**Meg Mangin**
Chronic Illness Recovery, USA

Intracellular bacteria may cause immune system dysregulation via actions on the vitamin D receptor (VDR), the nuclear receptor which regulates the innate immune system. Cell wall deficient (CWD) bacteria persist within cellular cytoplasm (including phagocytes), by using strategies to evade elimination. One of those strategies appears to be downregulation of the VDR. This is evidenced by elevated 1,25-dihydroxyvitamin D (calcitriol), in the absence of hypercalcemia. High levels of calcitriol suggest that bacterial ligands are able to antagonize or otherwise inhibit VDR function and prevent the receptor from expressing enzymes necessary to keep calcitriol in a normal range. Calcitriol activates the immune system by binding to the VDR to genomically induce transcription of antimicrobial peptides (AMPs) which eliminate pathogens. Normally, renal production of calcitriol is tightly self-regulated, with the end product down-regulating its own further production. Production of calcitriol in extra-renal cells is controlled by cytokines, lipopolysaccharide,nitric oxide and intracellular vitamin D binding protein; when extra-renal tissues are parasitized by CWD bacteria, excess calcitriol production is stimulated and renal control of calcitriol production is lost.Elevated calcitriol indicates the immune system recognizes the presence of parasitic bacteria and is attempting to combat them by increasing the production of calcitriol in order to transcribe AMPs. The result is chronic low-grade inflammation, persistent intracellular infection, and eventual multi-morbidity. Administration of the angiotensin blocker olmesartan medoxomil demonstrates reversal of immune system dysregulation. When used at higher than the standard antihypertensive dose, olmesartan appears to be anagonistic VDR ligand which upregulates the bacterially-inhibited VDR. This is evidenced by a significant reduction in elevated calcitriol. In addition, an apparent increase in AMP transcription, and thus elimination of intracellular bacteria, is evidenced by symptoms of Jarisch-Herxheimer reaction and eventual reduction in inflammatory symptoms.

**Conclusion:** intracellular infection compromises VDR activity and inhibits the innate immune system. Olmesartan, which enhances VDR expression and eliminates the offending bacteria, represents a clinical breakthrough in the treatment of immune system dysregulation.

## Biography

Meg Mangin, R.N. is a registered nurse licensed in the state of Wisconsin. She attended St. Mary's School of Nursing in Rochester, MN and graduated from Milwaukee County General School of Nursing. She held staff nurse positions in coronary/intensive care, and provided skilled home-nursing services in a variety of medical specialties. Meg headed the Wisconsin La Leche League for five years and served on a National Institutes of Health (NIH) State of the Science panel. She also served 6 years on an NIH Data, Safety and Monitoring Board. Meg was one of the earliest adopters of Inflammation Therapy and participated in its early research. She led a team of online nurses in counseling hundreds of patients on their road to recovery and was a presenter at Days of Molecular Medicine in Karolinska, Sweden, the Understanding Aging Conference in Los Angeles and the International Conference on Autoimmunity in Porto, Portugal. She is the author of Observations of Jarisch-Herxheimer Reaction in Sarcoidosis Patients and a co-author of a chapter in the 2006 medical textbook titled Vitamin D: New Research, published by Nova.

mmangin@chronicillnessrecovery.org