Bacteria-Induced Vitamin D Receptor Dysfunction in Autoimmune Disease: Theoretical and Practical Implications for Interpretation of Serum Vitamin D Metabolite Levels

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A new model that provides a pathogenesis for a number of autoimmune and inflammatory diseases focuses on the role of vitamin D nuclear receptor (VDR) dysfunction, which leads to an inadequate innate immune system response to infection. The VDR dysfunction is seen as the result of infection by an intraphagocytic biofilm-like metagenomic microbiota, which evades the immune system through its ability to block the VDR. A treatment approach based on this model has successfully used subinhibitory antibiotics combined with the VDR agonist, olmesartan, to induce remission/recovery in a number of autoimmune diseases, including sarcoidosis, rheumatoid arthritis, systemic lupus erythematosis, psoriasis, psoriatic arthritis, Reiter´s syndrome, Sjogren's syndrome, autoimmune thyroid disease, spondyloarthropathies and uveitis. Recovery using this approach also requires the avoidance of supplemental vitamin D. Laboratory, epidemiological and clinical vitamin D research will be revisited in the light of this model. The use of vitamin D metabolite levels for diagnosis, monitoring, assessment of disease severity and in making treatment decisions will be described. Early identification and treatment of at-risk family members will also be addressed. The degree of VDR blockage and subsequent innate immune system suppression from the intraphagocytic microbiota may vary with various factors (e.g., age, pathogen exposure sequence/timing), and this may affect observed patterns of response to bacterial, viral and parasitic pathogens and autoimmune disease incidence rates. An underlying innate immune system suppression from VDR blockage could account for the wide range of opportunistic and triggering infections implicated in various autoimmune diseases.
