AB005. Vitamin D immunomodulatory effect

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Abstract: In addition to the most familiar classical role vitamin D to maintain calcium and phosphorus homeostasis through effects on the intestine, kidney, and bone, in conjunction with parathyroid hormone, vitamin D shows a regulatory effect on a number of different cells, especially its anti-proliferative and pro-differential biological function. Through its own receptor in the immune cells, vitamin D increases the phagocytic activity of macrophages. Also, by binding to the regulatory sequences of antimicrobial peptides genes, vitamin D increases the microbicidal activity of phagocytes. Inhibition of differentiation and maturation of antigen-presenting dendritic cells, as well as direct influence on their contact with T lymphocytes, it significantly influences the type of immune response. Dendritic cells under the influence of vitamin D induce suppressor T cells, which can inhibit Th1 cell response and are critical in the regulation of immune tolerance. Vitamin D inhibits proliferation of Th1 and Th17 cells, as well their cytokine production, and suppresses the differentiation and maturation of B lymphocytes. Due to all these functions, vitamin D has shown beneficial effects in the prevention and modification of a number of autoimmune diseases. Data have shown that a broad spectrum of tissue cells, including immune cells, express vitamin D metabolizing enzymes, providing a biologically plausible mechanism for local, auto- and paracrine conversion of the native circulating forms, to the active form calcitriol. This process seems to be essential for normal immune function and therefore impaired or insufficient vitamin D levels may lead to dysregulation of immune responses. Addressing the questions as to whether vitamin D levels are related to the risk of developing autoimmunity and whether vitamin D supplementation can modify the course of autoimmune diseases? Unfortunately, clinical application of 1,25(OH)2D3 is obstructed by toxicity issues since the supraphysiological doses needed to modulate immune responses elicit concomitant calcemic side effects. The problem is the hypercalcemia in some diseases with impaired immune systems due to elevated levels of active vitamin D in the blood (sarcoidosis, tuberculosis, Crohn's disease, T cell lymphoproliferative disorders), a cytokine which is explained by stimulating the expression of CYP27B1 and CYP24A1 dysfunction (mitochondrial protein initiates the degradation of the 1,25(OH)2D3. In regulating the level of vitamin D3, this enzyme plays a role in calcium homeostasis and the vitamin D endocrine system.

Keywords: Vitamin D metabolism; immunomodulation; supplementation Vit D; vitamin D deficiency; calcium

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