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Brief Report

VITAMIN D's ROLE IN THE IMMUNE SYSTEM AS A SURVIVAL MOLECULE

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BRIEF REPORT

Vitamin D is an interesting and appealing chemical that has received a lot of attention in recent years in medicine. Its immunomodulatory and anti-inflammatory properties may match those of many naturally occurring compounds (e.g., flavonoids), but its role in biology was chosen through time to mitigate the harmful effects of the cell stress response and the immunological response. In this way, this molecule might be thought of as an ancient hormone with a primary function of pro-survival. The purpose of this review was to clarify this subject.

The anti-inflammatory and immune regulatory properties of 1,25(OH)2 vitamin D3 (calcitriol) and its immediate precursor, 25-OH vitamin D3 play a key part in vitamin D's pro-survival role in a systemic viewpoint (calcidiol). This capacity is thoroughly discussed in the current review.
Vitamin D's role as a calcium and bone physiology regulator is not limited to the well-known. Because it is present when the immunological response develops early in the newborn's life, this molecule should play a crucial role in the immune system as well. This fact should confirm the importance of this hormone-like vitamin for human health, as well as the frequently addressed nutritional information campaigns offered to ensure people have the proper intake of vitamin D and its optimal levels; however, this issue may vary depending on a subject's lifestyle and genetics.

Vitamin D and the immunological T-cell response should have a close association. For patients undergoing renal transplantation, the presence of T-helper (Th) imbalance and vitamin D deficiency is a negative predictor of survival, particularly in the presence of Th cytokine polymorphism IL13 rs20541 T allele, IL28B rs8099917 GG genotype, and IL28B rs12979860 TT genotypes; these are considered negative predictors of survival in patients undergoing renal replacement therapy, Vitamin D regulates memory and effector CD8+ T cell development in cytotoxic CD8+ T cells, which is important in the inflammatory and infectious responses.

Vitamin D may affect *in vivo* T-cell–mediated acquired immunity, according to current research. A recent randomised controlled clinical study in a single-center experiment of 38 patients found that vitamin D administration modifies immunological function mediated through T-cell response. Supplementing with 2000 IU/d of vitamin D led to more effective augmentation of vitamin D blood bioavailability and improved regulatory T-cell (T-reg) immunity in a double-blind, randomised controlled clinical study in pregnant women. Vitamin D supplementation increases the number of CD38+ B cells in the bloodstream while lowering the number of CD4+Th17 leukocytes.

Vitamin D's pro-survival effect is primarily exerted at the systemic organism level through its essential function in suppressing and regulating innate immunity. Nonetheless, recent research is examining whether vitamin D has a function in B cells, including B-regulatory cells, in autoimmune illnesses. In this way, vitamin D has an important role in regulating cytokines in both innate and acquired immunity.

Calcitriol has the capacity to rewire T cells to operate as a signal transducer and activator of transcription initiation, hence directing the production of pro-inflammatory cytokines. The activity of calcium and the calcium-sensing receptor, whose modulation is the primary function of this vitamin, are used to assess the anti-inflammatory action of calcitriol or 1, 25$(OH)_2$ D3. Increased calcium levels via the calcium-sensing receptor operate as a danger signal, triggering the formation of inflammasomes in myeloid cells and the functional elicitation of interleukin (IL)-1 by caspase-1. Vitamin D's activities should so counterbalance calcium's pro-inflammatory and apoptosis-promoting effects. The link between calcium and vitamin D in immunity is a master tuner of cell inflammation and response to oxidative stress, thus this finding is particularly intriguing.

Vitamin D's ability to modulate calcium excess is a key feature of the survival strategy, as an increase in calcium causes autophagy to be replaced by apoptosis. Vitamin D controls antigen presentation and contributes in human dendritic cell (DC) response throughout cell maturation as a regulator of proper homeodynamics between energy and immunological diseases. Vitamin D reduces the expression of membrane markers HLA-DR, CD14, CD40, CD80, CD83, and CD86, which hinders DC maturation. It also works in tandem with toll-like receptor (TLR) ligands to induce IL-8, IL-10, and IL-6 while blocking IL-12 induction mediated by lipopolysaccharide (LPS).

Vitamin D targets various components and actors of the innate immune system to maintain energy and survival equilibrium, according to this data. It should behave as a pro-survival hormone in this regard. As a result, evidence of vitamin D's capacity to produce tolerogenic DCs has been described from this perspective. Vitamin D's anti-inflammatory and pro-tolerogenic properties are important aspects of its involvement in maintaining a healthy individual's physiology.

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