

ALLERGY, VITAMINS

# WHAT THE #\*\$! DO WE KNOW!?

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It drives me crazy – the [new JACI review](#) with a detailed explanation how VDD (vitamin D deficiency) induces food allergy (FA). Unfortunately, it has never been shown that FDD is related to FA – while the opposite may be true...

Until we have the vitamin D feeding data ready for publication from the [Europrevall](#) survey, I can only look back in history when we started the vitamin hypothesis back in [1998](#). Roitt's Essential Immunology was writing at that time on page 85

## CONTROL MECHANISMS

to T-cell tolerance and greater resistance to infections. Females are also far more susceptible to autoimmune disease, an issue that will be discussed in greater depth in Chapter 17, but here let us note that oral contraceptives can induce flares of the autoimmune disorder systemic lupus erythematosus (SLE).

### **Malnutrition diminishes the effectiveness of the immune response**

The greatly increased susceptibility of undernourished individuals to infection can be attributed to many factors: poor sanitation and personal hygiene, overcrowding and inadequate health education. But in addition there are gross effects of protein-calorie malnutrition on immunocompetence. The widespread atrophy of lymphoid tissues and the 50% reduction in circulating CD4 T-cells underlies serious impairment of cell-mediated immunity. Antibody responses may be intact but they are of lower affinity; phagocytosis of bacteria is relatively normal but the subsequent intracellular destruction is defective.

Deficiencies in pyridoxine, folic acid and vitamins A, C and

E result in generally impaired immune responses. Vitamin D is an important regulator. It is produced not only by the UV-irradiated dermis but also by activated macrophages, the hypercalcemia associated with sarcoidosis being attributable to production of the vitamin by macrophages in the active granulomas. The vitamin is a potent inhibitor of T-cell proliferation and of cytokine production by Th1 cells. This generates a neat feedback loop at sites of inflammation where macrophages activated by IFN $\gamma$  produce vitamin D which suppresses the T-cells making the interferon. It also downregulates antigen presentation by macrophages and promotes multinucleated giant cell formation in granulomatous lesions. Nonetheless, in further emphasis of the potential duality of the CD4 helper subsets, it promotes Th2 activity, especially at mucosal surfaces. Zinc deficiency is rather interesting; this greatly affects the biological activity of thymus hormones and has a major effect on cell-mediated immunity.

Of course there is another side to all this in that moderate restriction of total calorie intake and/or marked reduction in fat intake, ameliorate age-related diseases such as autoimmunity.

Vitamin D is an important regulator ... The vitamin is a potent inhibitor of T cell proliferation and of cytokine production by Th1 cells.

The same view is shared in the more recent 11th edition on page 228:

By downregulating macrophage activity, Th1 effectiveness is decreased

At least I am pleased to read that

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predict that correction of VDD will lower the risk of FA but caution that supraphysiologic levels of any hormone, including vitamin D, might have untoward effects, particularly during major developmental periods, such as fetal development and infancy. Supraphysiologic levels of vitamin D are achievable only by excessive supplementation and might actually increase the risk of atopic diseases, as proposed by Wjst<sup>66</sup> and others. We believe that these dose-specific effects and the complexity of the relationship between vitamin D, immunity, and microbes might help to explain why elucidating this hormone's role in atopic disease has proved such a challenge.

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All chemical compounds of vitamin D for supplementation are supraphysiological #\*!\$