

Vitamin D Deficiency, Biology and its Functions

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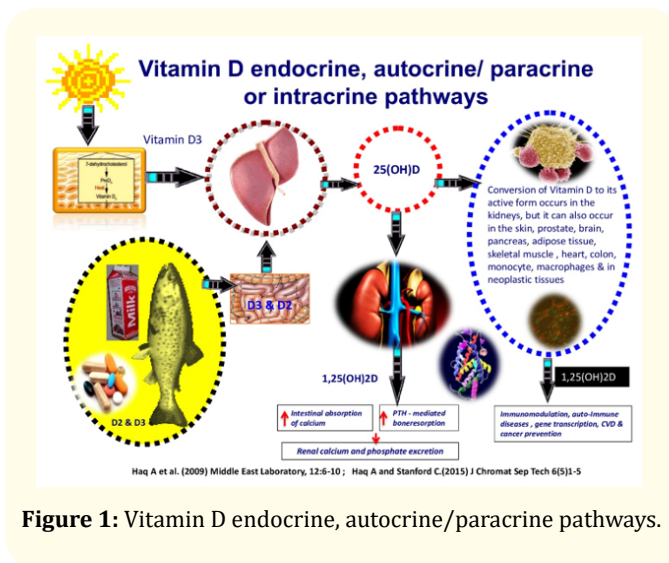
Vitamin D deficiency is a major global, public health problem and is prevalent among all ages and ethnic groups. Nevertheless, it is preventable cost-effectively, if proper public health interventions are implemented. Only a handful of expensive foods contain vitamin D and it is difficult to change people's attitude and lifestyles. Therefore, an inexpensive supplements and food-fortification programs have been recommended. More than 80% of the requirement vitamin D should generate from our skin, but sun avoidance behaviors prevent this. The combination of lifestyle and environmental issues, decreased exposures to sunlight have led to vitamin D insufficiency. This affects approximately, 50% of the global population and estimated 1.5 billion people worldwide have vitamin D deficiency [1]. Sunlight remains the primary source of vitamin D₃ that is synthesis in dermal tissues in humans and in animals. The details of vitamin D metabolism through sun or by ingested food are shown in figure 1.

Reduced blood concentration of vitamin D leads to an increased prevalence of autoimmune disorders [2], including type 1 diabetes, T helper lymphocyte; mediated inflammatory immune disorders [3], such as Crohn's disease, ulcerative colitis, multiple sclerosis [4], rheumatoid arthritis [4], type I diabetes [5], systemic lupus erythematosus [6] and psoriasis [7] etc.

While it is best to obtain vitamin D through sunlight, D₃ is the preferred supplement. Skin and dietary derived vitamins D₂ and D₃ need to be activated. The first hydroxylation step occurs in liver generating 25(OH)D and its final hormonal form, 1,25(OH)₂D is generated in kidneys. Serum 25(OH)D levels below 30 ng/mL (75 nmol/L) is considered as insufficient or hypovitaminosis D.

The key functions of vitamin D is to facilitate intestinal absorption of calcium and phosphorus, skeletal mineralization, and control cell growth [2]. Vitamin D also enhances the immune protection and fight against the invading microbes. These actions are mediated through modulation of vitamin D-dependent gene transcriptions. Beyond its use to prevent osteomalacia and rickets and prevention of falls and fractures, the evidence for other health effects of vitamin D supplementation are increasing, but still inconsistent. However, it is not surprising, as one cannot expect people to get better following a supplementation, when they are not deficient. Provision of vitamin D supplementation to D-deficient elderly and institutionalized persons, have been unequivocally shown to decrease falls, increases bone mineral density, and decrease fractures. Additional exciting biological and physiological functions of vitamin D are in the horizon.

Research during the last two decades in the field of vitamin D suggests that vitamin D is much more than a nutrient needed for bone health. Vitamin D is an essential hormone required for regu-

**Figure 1:** Vitamin D endocrine, autocrine/paracrine pathways.

lating a large number of physiologic functions and affects a large number of genes. All studies, in virtually all nations, irrespective of latitude, show that the majority of the world's population has insufficient vitamin D status. It is clear that sufficient levels of serum 25(OH)D are essential for optimum human health. However, many questions remain unanswered. For example, what are the optimal levels of serum 25(OH)D? What amount of supplementation or sunlight exposure is needed to achieve and maintain these optimum levels? There is a growing consensus among the leading researchers that the optimal range for 25(OH)D values lies above 30 to 32 ng/mL (75 - 80 nmol/L) for most populations, and it seems prudent that persons at high risk of vitamin D deficiency and/or insufficiency have their serum 25(OH)D levels assessed.

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