ACTA SCIENTIFIC NUTRITIONAL HEALTH (ISSN:2582-1423)

Volume 4 Issue 11 November 2020

Short Communication

Vitamin D Deficiency, Biology and its Functions

Afrozul Haq[1]* and Nuzhat Ahsan[2]

[1]Professor, Department of Food Technology, Jamia Hamdard, New Delhi, India

[2]DST WOS A Scientist, Institute of Life Science, Bhubaneshwar, Odisha, India

***Corresponding Author:** Afrozul Haq, Professor, Department of Food Technology, Jamia Hamdard, New Delhi, India.

Received: September 21, 2020

Published: October 28, 2020

Keywords: Vitamin D, 25(OH) D; Metabolism; Deficiency; Public Health

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 difficulty to change people's attitude and lifestyles. Therefore, an inexpensive supplements and foodfornication 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 D_3 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.

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

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, D_3 is the preferred supplement. Skin and dietary derived vitamins D_2 and D_3 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-

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.

Bibliography

1. Z Naeem. "Vitamin D Deficiency- An Ignored Epidemic". *International Journal of Health Sciences* 4.1 (2010): 5-6.

2. R Zhang and DP Naughton. "Vitamin D in health and disease: Current perspective". *Nutrition Journal* 9 (2013): 2-13.

3. E Zold., *et al*. "Vitamin D deficiency in undifferentiated connective tissue disease". *Arthritis Research and Therapy* 10 (2008): 1-8.

4. KL Munger., *et al*. "Serum 25-hydroxyvitamin D levels and risk of multiple sclerosis". *The Journal of the American Medical Association* 296 (2006): 2832-2838.

5. S Patel., *et al.* "Association between serum vitamin D metabolite levels and disease activity in patients with early inflammatory polyarthritis". *Arthritis and Rheumatology* 56 (2007): 2143-2149.

6. SB Mohr., *et al.* "The association between ultraviolet B irradiance, vitamin D status and incidence rates of type 1 diabetes in 51 regions worldwide". *Diabetologia* 51 (2008): 1391-1398.

7. PW Wu., *et al.* "25-hydroxyvitamin D and cardiovascular risk factors in women with systemic lupus erythematosus". *Arthritis and Rheumatology* 61(2009): 1387-139.