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A REVIEW ON VITAMIN D DEFICIENCY AT DIFFERENT STAGES - DURING GESTATION, LACTATION AND PAEDIATRICS

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ABSTRACT

Now a-days vitamin D deficiency plays a significant role in concern with health issues around the geographic region. Even in tropical countries, where there is abundant sunlight this deficiency is reported among all age groups. Recent guidelines suggested vitamin D at its high risk for toxicity as 25(OH)D levels <5, <15, >20 and >50ng/mL respectively. The manifestations of this deficiency vary from autoimmune diseases to cardiovascular mortality. The objective of this review is to assess the magnitude and consequences of VDD during gestation, lactation and paediatrics with its associated risk factors, prevention methods, and the possibilities of epigenetic mechanism in early foetal life. Routine supplementation starting from newborn period is being increasingly endorsed by various international organizations. Preventing from being exposed to UV rays, food fortification and routine supplementation are the current available options for handling this nutritional deficiency.

KEYWORDS: Vitamin D, gestation, lactation, foetus, autoimmune disease, deficiency.

INTRODUCTION

Vitamin D is fat-soluble which performs a key role in calcium homeostasis and bone metabolism. In particular, vitamin D deficiency induced disorder includes rickets or osteomalacia which is able to arrest normal growth and the development of infancy and childhood. It is also found that Vitamin D influences the extraskeletomuscular system as well as the immune system. There has been a growing interest in vitamin D deficiency and its supplementation where vitamin D can be obtained in three ways: through skin, diet, and from

other supplements. Vitamin D is naturally obtained when exposed to sunlight. However too much exposure can lead to skin aging and skin cancer. (Balasubramanian *et al*., 2013).

Table I: Recommendations on daily intake of Vitamin D.

Life stage	Vitamin D (IU/day)	Vitamin D (mcg/day)
Children and Teen	600	15
Gestation and lactating women	600	15

The amount of vitamin D on food and supplement labels can be given in International Units (IU) or micrograms (mcg). Since skin synthesis of vitamin D varies too much, the latest dietary recommendations assume minimal sun exposure. (Thorne-Lymana *et al*., 2012).

Decades back, understanding of vitamin D synthesis and its function has changed remarkably. The traditional concepts and current recommendations for vitamin D supplementation, duration of sun light exposure and revised management strategies for vitamin D deficiency can be re-examined.

CAUSE OF VITAMIN D DEFICIENCY

Occurrence of vitamin D deficiency is found to be about 50-90 % in Indian subcontinent and is attributed to low dietary calcium along with skin colour and changing lifestyle. Vitamin D deficiency is very common in infants due to the following factors – decreased dietary intake, decreased cutaneous synthesis because of cultural and religious practices, seasonal variation, fear of cancer, and practice of not taking the child out, increase in pigmentation, increasing rate of exclusive breast feeding, and low maternal vitamin D (Balasubramanian *et al*., 2013).

Table II. Etymology of vitamin D deficiency

Decreased vitamin D synthesis	Skin pigmentation, physical agents blocking UVR exposure, clothing, latitude, season, air pollution, cloud cover, altitude
Decreased nutritional intake of vitamin	Strict vegan diet
Age and physiology	Elderly, obese and institutionalised
Mal-absorption	Celiac disease, pancreatic insufficiency (cystic fibrosis), biliary obstruction (biliary atresia)
Decreased synthesis	Chronic liver disease
Increased degradation of 25 (OH) D	Drugs such as rifampicin, isoniazid, anticonvulsants, glucocorticoids.

PHYSIOLOGY AND VITAMIN D METABOLISM

There are two sources of Vitamin D for human. An exogenous one is provided by the diet in the form of vitamins D2 and D3. In the endogenous production, cholecalciferol (D3) which is the main source of Vitamin D, is synthesized in the skin by the action of ultraviolet B (UVB) radiation through the photolysis of 7-dehydrocholesterol and transformed into vitamin D3. (Urrutia-Pereiraa and Dirceu Soléb, 2014) The vitamin D binding protein transports the vitamin D3 to the liver where it undergoes hydroxylation to 25(OH)D (the inactive form of vitamin D) and then to the kidneys where it is hydroxylated by the enzyme 1 α hydroxylase to 1,25(OH)D, its active form. This enzyme is also present in a variety of extrarenal sites, including osteoclasts, skin, colon, brain, and macrophages, which aids in the cause of its broad-ranging effects. (kulie *et al*., 2009).

The process takes place in two phases, the first one occurs in the deep layer of the dermis and consists in the photo conversion of 7-dehydrocholesterol into pre-vitamin D or pre-calciferol. In second phase, there is a chemical isomerisation, pre-vitamin D slowly and progressively turns into vitamin D3 depending on body temperature, which has high affinity for the Vitamin D carrier protein (DBP). The pre-vitamin D remains in the skin because of its lower binding affinity. Upon reaching the skin capillary network, Vitamin D is transported to the liver and binds with DBP, where it starts its metabolic transformation.

RISK FACTORS OF VITAMIN D DEFICIENCY

The main source of Vitamin D in children and adult is exposure to sunlight, so the main cause of vitamin D deficiency is the decrease of its endogenous production. The factor that affects the transmission of UVB radiation or interferes with its skin penetration will determine the reduction of 25(OH)D. Among these, risk factors are the following.

• Use of sunscreen with a protection factor reduces the synthesis of vitamin D in the skin above 95%

• Individuals with darker skin have natural sun protection, as melanin absorbs UVB radiation, and thus they need 3-5 times longer sun exposure to synthesize the same amount of vitamin D than individuals with light skin

• Skin aging as well as age decrease the capacity of the skin to produce vitamin D due to lower availability of 7-dehydrocholesterol.

• Skin damages such as burn decreases vitamin D production.

• Atmospheric contamination and overcast may act as a sunscreen.

• The season of the year and the time of the day influence dramatically on the skin production of vitamin D.

The second cause is the reduced intake of vitamin D, as few food items are rich in vitamin D (blue fish, egg yolks). The intake of the vitamin can be increased with fortified products such as dairy products, although the amount of Vitamin D they provide may be insufficient for an adequate state of Vitamin D. (Thorne-Lymana and Fawzi 2012).

MEASUREMENT OF VITAMIN D LEVEL

The major circulating form of vitamin D is 25(OH)D, with a half-life of 2-3 weeks and its levels are the best available indicators of vitamin D status. Though, 25 (OH)2D (calcitriol) is the active form, it has a half-life of only 4 hours and it is not found to be a good indicator of vitamin D. The assays that are used to measure 25(OH)D levels should be capable of measuring both D2 (ergocalciferol) and D3 (cholecalciferol) derivatives. The total 25(OH) D [25(OH)D2 and 25(OH)D3] levels can be measured by high performance liquid chromatography (HPLC) or tandem mass spectrometry. (Urrutia-Pereiraa and Dirceu Soléb, 2014) Other methods of measurement include radio-immune assays using monoclonal antibodies to 25(OH)D and chemiluminescent protein binding assay (Balasubramanian *et al*., 2013).

TREATING VITAMIN D DEFICIENCY

If the main source of vitamin D comes from sunlight exposure, it is difficult to establish generalized requirements for the intake, especially when many variables associated with its deficiency. While excluding the exposure of habitual sunlight, it is known that what level of exposure is safe and sufficient to maintain adequate levels of vitamin D. In addition, vitamin D fortified foods are available which will refill the deficiency of vitamin D.

CONCLUSION

Deficiency of vitamin D in pregnant women, lactating and infants are at major risk. Prevention strategies should be made sure for vitamin D sufficiency in women during pregnancy and lactation. Evidence based interventions will help us to improve maternal and foetal nutrition. The ambiguities between the definitions of vitamin D status, combined with a lack of consistency in recommendations related to incorporation of routine testing of 25(OH)D levels in the prenatal period, especially women with risk factors for vitamin D deficiency. The amount of dosage levels and gestational age for the start of vitamin D

supplementation with lack of universal standards for vitamin D, lack of education about the benefits of vitamin D and the need for adequate sunlight exposure represents all important barriers to the advancement implementation of vitamin D supplemental guide.

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