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Review Article

Vitamin D and Oral Health

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Abstract

Vitamin D is a fat-soluble Vitamin that helps the body to absorb and retain calcium and phosphorus for tooth and bone mineralization. The two major forms of Vitamin D are Vitamin D2 and D3. While Vitamin D2 is manufactured through ultraviolet irradiation of ergosterol from yeast, Vitamin D3 results from ultraviolet irradiation of 7-dehydrocholesterol from lanolin exhibiting the biological activity of cholecalciferol (vitamin D3), and it is synthesized in the human skin. It is not commonly found in natural diet and are mainly taken as supplements. Vitamin D has a significant role in calcium homeostasis and metabolism. Vitamin D is also very effective along with minerals in the protection of oral health. Vitamin D helps maintain the calcium-phosphate balance and contributes to the shaping of the bone. It also has important functions by showing anti-inflammatory effects. Deficiency of Vitamin D has been linked to major changes in the tooth tissue as such developmental anomaly, teeth decay, gingivitis and periodontal disease. This article discusses the details effect of Vitamin D on tooth and its surrounding tissues.

Keywords: Vitamins; Vitamin D; Dental Caries; Periodontal Disease

Introduction

Vitamin D is a fat-soluble steroid hormone obtained mainly from exposure to sunlight, that help the body absorb and retain calcium and phosphorus, which are critical for building bone and forms an important constituent of tooth. Natural foods containing Vitamin D are not common. Taking a supplement is the prime way to get adequate vitamin D but it can be found in oily fish and oils from fish [1].

Vitamin D is available in two forms: vitamin D2 ("ergocalciferol" or pre-vitamin D) and vitamin D3 ("cholecalciferol"). Both are also naturally occurring forms that are produced in the presence of the sun's ultraviolet-B (UVB) rays, hence its nickname, "the sunshine vitamin," but D2 is produced in plants and fungi and D3 in animals, including humans. Vitamin D production in the skin is the primary natural source of vitamin D, but many people have insufficient levels because they have limited sun exposure. People with darker skin tend to have lower blood levels of vitamin D because the pigment (melanin) acts like a shade, reducing production of vitamin D (and also reducing damaging effects of sunlight on skin, including skin cancer) [2].

Measurement of serum 25-hydroxyvitamin D (25[OH]D) is a widely accepted biomarker analysis for vitamin D status [3].

Magnesium, calcium, and phosphorus are important minerals that form the basic structural components of the tooth and should be taken in sufficient levels with the diet. These minerals play a role in strengthening the tooth structure by interacting with vitamins, especially, vitamin D [4]. Various theories have been proposed to explain the role of vit D in decreasing the risk of caries. One of these mechanisms is the regulation of serum calcium, phosphate and parathyroid hormone, which are necessary for the formation, calcification, mineralization and protection of teeth. Hypocalcemia and Hyphosphatemia have been linked to Enamel and dentin defects [5].

Other role of the vitamin D as suggested by laboratory studies are reduction of cancer cell growth, help control infections and reduce inflammation. Scientists are actively investigating other possible functions of Vitamin D which may be linked to its receptors in many of the body's organs and tissues advocating its roles beyond bone health [6].

Discussion

Vitamin D deficiency has become a pandemic issue due to decreased vitamin D intake from food and lessened sunlight exposure. Attention is drawn to vitamin D and its role learned in disorders such as diabetes, cardiovascular disease and cancers including oral cancers. Vitamin D is also very effective along with minerals in the protection of oral health. Vitamin D helps maintain the calcium-phosphate balance and contributes to the shaping of the bone. It is reported that with sufficient vitamin D level, the onset and progression of caries in the tooth structure can be stopped, the formation of caries can be reduced and enamel loss can be prevented. Vitamin

D also affects the disease and health conditions of the periodontium. Anti-inflammatory and immunomodulatory functions have a role in the pathogenesis of periodontal disorders. It can reduce bone resorption and suppress the inflammatory outcome related to periodontal diseases by increasing mineral density [7]. Vitamin D has been linked with tooth decay, gingivitis, and tooth loss. Vitamin D, in particular, as a promising oral health-protective agent, is said to lessen the incidence of caries and periodontitis.

Vitamin D and dental tissue

Vitamin D has a significant role in the formation of oral hard tissue [8], comprising tooth enamel and dentin, and affects primary teeth development [9]. Vitamin D has a significant role in odontogenesis [10]. The mechanism by which vitamin D excites the mineralization of tooth enamel involves binding to Vitamin D Receptor expressed in both tooth and bone cells. Vitamin D receptors direct the transcription of several target genes, most expressed by ameloblasts and odontoblasts. VDR stimulates the formation of structural gene products in dentin, together with calcium-binding proteins and diverse extracellular matrix proteins. The gene encoding VDR is positioned on chromosome 12q13.11 and comprising several polymorphisms [11]. The VDR gene adjusts the biological role of major vit D metabolites, thus having a key role in the configuration of teeth, particularly in the mineralization of dentin and enamel. Consequently, enamel developmental deficiencies e.g., enamel hypoplasia, can take place in consequence of VDD [12]. Moreover, vit D adjusts and adapts both the innate and adaptive immune system.

During the development of the tooth, the hard tissues of the tooth are strongly affected by nutritional status and thus vitamin deficiency [13]. It is stated that there is a positive correlation between malnutrition and enamel hypoplasia and caries in the primary dentition period in children [14].

In addition, deficiencies of these minerals cause delayed tooth eruption, bleeding gums, destruction patterns in alveolar bone, periodontal disease, enamel or dentin hypoplasia. It is reported that with sufficient vit D level, the onset and progression of caries in the tooth structure can be stopped, the formation of caries can be reduced and enamel loss can be prevented. In the formation of tooth decay, the acid that is produced by bacterial fermentation of the residues on the tooth surface that are not brushed after eating sugary foods lowers the pH below 7 and plays a role in the destruction of the tooth hard tissues. However, it has been recently revealed that dental caries can be reduced with UV- B rays and vit D supplements. Considering the helpful effects of vit D on dental caries, it is thought to be effective in reducing the overall prevalence, especially in children at risk of early caries.

Early childhood caries (ECC) is one of the most common chronic diseases and can have adverse effect on individual's overall health. Early childhood caries affect the nutritional status and general health of the child. It is stated that children with ECC may have malnutrition, iron deficiency anemia and VDD [15] When the relationship between vit D intake and caries is evaluated, it is determined that the incidence of tooth decay is higher in children with low vit D or children of mothers with low vit D during pregnancy. There is an association between vit D levels in early childhood (up to 8 years old) and DMFT scores. When serum vit D concentrations are more than 50 nmol in early adolescents (10-11 years) considerably less caries is detected in permanent first molars has been found. Similarly, in children aged 6 to 17 years, they found a 0.66 decrease in DMFT for every 10 ng/ml of serum vit D level increase [15]. In general, malnutrition and shortage in vitamin intake due to malnutrition augments the incidence of enamel hypoplasia in children. Vitamin D use may have a role in the protection of caries early in life. It is thought to be a promising caries prevention agent, given that vit D supplementation is connected with a 47% reduction in caries in children according to meta-analysis studies [16]. Vitamin D deficiency during pregnancy (a vital period for tooth growth) is related with developmental defects; especially enamel hypoplasia and caries susceptibility. Also, vit D intake during pregnancy diminishes the risk of enamel defects and hypoplasia in babies and is associated with better eruption of deciduous teeth. Improving vit D levels in children from an early stage of life appears to be an important task. This requires awareness from pregnancy. Pregnant women should have their vit D levels tested routinely during the first trimester of pregnancy and the risk of VDD, VDD and vit D ingestion should be evaluated. Prenatal vit D levels appear to influence the development of primary dentition and ECC.

Vitamin D is an essential hormone for the absorption of calcium, magnesium and phosphorus from the intestine, which is necessary for the appropriate mineralization of bones and teeth. In addition, covering the surfaces of the implants with vit D during implant application, which is one of the dental procedures, increases osteointegration. Moreover, applying vitD3 intraperitoneally speeds up orthodontic tooth movement, and even patients receiving vit D and bisphosphonate therapy can obtain orthodontic treatment [16].

Currently the evidence highlights the association of low levels of vitamin D and the high prevalence of caries in both children and adults, although the mechanism remains unclear [17]. Additionally, vitamin D exerts several roles in the control of the human immune system, and an optimal vitamin D concentration (≥75 nmol/L) is associated with lower odds for dental caries in children. However, the studies' results are contradictory. A recent systematic review of controlled clinical trials, with data from 2827 children, investigated the impact of vitamin D supplementation on dental caries prevention. The results of this study show that vitamin D supplementation reduced the risk of caries in about 47%, but with low certainty [18]. Another research supports that caries-free children were twice as likely to have optimal vitamin D concentrations (≥75 nmol/L) and those with severe early childhood caries were at

nearly three times the odds of having deficient levels (<35 nmol/L) [19]. On the one hand, it is important to clarify that serum vitamin D does not change the major structure of teeth since this structure remains constant until some extrinsic factor causes its wear.

Vitamin D deficiency in tooth mineralization

Teeth are mineralized organs, surrounded by alveolar bone, and formed by three distinctive hard tissues: enamel, dentin, and cementum. The tooth mineralization process occurs parallel to skeletal mineralization, yet if mineral metabolism is disturbed then failures will occur similarly to those that occur in bone tissue. Vitamin D plays a key role in bone and tooth mineralization, and when levels are unregulated it can lead to the "rachitic tooth", which is a defective and hypomineralized organ highly susceptible to fracture and decay [20].

The mechanisms by which VDD affects tooth mineralization are well debated elsewhere. The main biological basis relies on the fact that severe VDD (<10 ng/mL) causes hypocalcemia and hypophosphatemia with secondary hyperparathyroidism (driven by hypocalcemia) [21]. This hyperparathyroidism promotes intestinal absorption of calcium (Ca2+), and renal production of 1 α,25-dihydroxyvitamin D (1,25[OH]2D), increasing bone turnover leading to elevated serum levels of Calcium and low serum levels of inorganic phosphate. The initial hypophosphatemia is then severely worsened. Ultimately, the loss of vitamin D signaling pathways in tooth cells with low concentrations of Calcium and phosphate ions inhibit proper mineralization of teeth and mineralization defects occur. Apart from its mineralization homeostasis role, circulating vitamin D can initiate a signaling pathway through vitamin D receptors (VDR). VDR is a ligand-activated transcription factor that controls gene expression through vitamin D elements (VDRE) [22]. For instance, some of these responsive genes affect bone, mineral metabolism, immune response, cell life cycle and migration, skeletal muscle, detoxification, and energy metabolism. Vitamin D up regulates VDR which, in turn, can induce structural gene products, including calcium-binding proteins and various extracellular matrix proteins (e.g., enamels, amelogenins, dentin sialoglycoproteins, and dentin phosphoproteins), resulting in the formation of dentin and enamel. Beyond the typical VDD causes, nutritional deficiency or reduction of sunlight exposure, there are genetic deficiencies originating from mutations encoding elements of the vitamin D metabolic machinery. The main causes of VDD, second to genetic mutations, are abnormal enzyme secretion (i.e., vitamin D-dependent rickets type 1, VDDR-I) and anomalous VDR function or signaling (vitamin D-dependent rickets type 2, VDDR-IIa; hereditary defects in the vitamin D receptor-effector system, HDVDR) [23]. These genetic conditions cause defective mineralized tissues, despite normal vitamin D consumption or sunlight exposure and, ultimately, will increase the risk of mineralized tooth tissue hypoplasia (i.e., amelogenesis imperfecta, dentinogenesis imperfecta, enamel hypoplasia) or higher risk of caries. Remarkably, deciduous dentition can be influenced by maternal 25 (OH) D levels, despite the influence of inherited defects of the fetus. Fetal serum-circulating levels of vitamin D follow the maternal concentration and can be used as a standard surrogate marker to the fetus. Therefore, if maternal 25(OH)D levels turn unbalanced, this may have direct repercussions on the baby's health [24] and, in particular, on tooth development. The pattern of mineralization defect depends on the specific week of gestation when maternal VDD occurred. It is known that maternal VDD at 12-16, 20-32 and 36-40 weeks results in defects at the incisal third, middle third and cervical third, respectively [25]. In a randomized clinical trial (RCT), vitamin D supplementation during pregnancy revealed that pregnant women with <15 ng/mL of vitamin D had a 14% higher risk of deciduous dentition [26]. In contrast, high-dose vitamin D supplementation during pregnancy was associated with an approximately 50% reduced odds of enamel defects. In another RCT, high-dose Vitamin D supplementation during pregnancy was linked to 50% lower risk of enamel defects in the newborn, underlying once more the likely preventive role of Vitamin D for enamel defects [27].

Vitamin D deficiency and periodontitis

Periodontitis is a complex polymicrobial disease and one of the two most prevalent diseases worldwide. Periodontitis is associated with strong socioeconomic and systemic repercussions with great impact on quality of life. The systemic link [28] between periodontitis and other diseases and conditions has escalated, such as diabetes, ischemic stroke, cardiovascular disease (CVD), rheumatoid arthritis, inflammatory bowel disease, stress, solid-organ transplanted individuals, or preterm birth. Furthermore, the impact of nutrition on periodontal health, and in particular Vitamin D Deficiency, has been intensively investigated and a recent European consensus stated that an inadequate vitamin D status impacts periodontal health and oral functions. Several cross-sectional studies have compared the levels of Vitamin D between individuals with periodontitis and without periodontitis; however, the results remain diverse [29]. While most reports show that periodontitis was associated with lower levels of Vitamin D compared to non-periodontitis, another has reported no differences. Further, vitamin D concentrations were associated with higher periodontal destruction, severe periodontitis stages and higher tooth loss. In otherwise healthy patients (CVD and diabetes mellitus), lower levels of Vitamin D were also associated when periodontitis was diagnosed. Data from the NHANES III study [30], performed in the USA, showed that individuals with the highest levels of vitamin D experienced 20% less bleeding on probing than those with the lowest levels. Other investigations also demonstrated that lower levels of gingival inflammation are associated with people without periodontitis. Comprehensively, the inflammatory and immune actions against periodontal pathogens are triggered by the host immune system. As previously mentioned, salivary low levels of vitamin D were associated with higher levels of inflammation biomarkers in periodontitis patients when compared to periodontally healthy pa-

tients [31] (namely IL-35, IL-17A and transforming growth factor), supporting the presence of an inflammatory microenvironment.

The immunological role of vit D is stimulation of the arrangement of some antimicrobial peptides, e.g., defensins and cathelicidin (LL-37), which defend against many pathogens, counting oral bacteria. Cathelicidin (LL-37 or hCAP-18) is controlled by vit D, which has both anti-endotoxin and antimicrobial properties.

Remarkably, vitamin D supplementation was linked to a decrease of salivary cytokines before nonsurgical periodontal treatment. In addition, a cross-sectional study showed through gingival samples that periodontitis patients exhibited lower levels of VDR and fewer fibroblast cells with higher inflammatory cell infiltration compared with healthy periodontal individuals [32]. Although not fully understood, Vitamin D has apparent fine-tuning, anti-inflammatory and mineralization effects on the periodontium according to the latest *in vitro* evidence. A study showed that vitamin D may decrease the number of live porphyromonas gingivalis through active autophagy [33] and might alleviate the inflammatory burden of periodontitis in rodent models: decreasing inflammatory levels (RANKL, TNF-α, IL-1, MMP-9) [113-116]; inhibiting IL-6 overexpression; and suppressing alveolar damage via inhibition of bone loss, apparently through systemic T-helper cells. In cultured human periodontal cells, Vitamin D induced a comparable mineralization effect to vitamin C [34].

Thus both preclinical and clinical studies support the idea that vitamin D, through its metabolic pathway, might be involved in the pathogenesis of periodontitis, by impacting tooth mineral density and being reversely correlated with disease severity of periodontitis.

From a genetic perspective, the role of VDR variants in periodontitis has been the subject of great consideration. In two recent evidence-based studies, a number of VDR polymorphisms were correlated with higher risk of developing periodontitis [35]. Notwithstanding, the VDR variants' impact on periodontitis still remains to be consolidated since it depends on the number of studies and is expected to increase considerably in the future.

On the other hand, the influence of vitamin D supplementation was studied in both nonsurgical and surgical periodontal treatments. Vitamin D and calcium supplementation showed moderate positive effects on periodontal health after nonsurgical periodontal treatment. Further, baseline VDD negatively influenced periodontal surgery outcomes, even when supplementation was used to compensate for low levels. Despite that these studies show vitamin D as a hypothetical hallmark for the success of patients' treatment, more studies are warranted to infer scientific fallouts and permit definite conclusions.

Conclusion

Vitamin D is a group of fat-soluble secosteroids responsible for increasing intestinal absorption of calcium, magnesium, phosphate, and many other biological effects. It has been linked with tooth decay, gingivitis, and tooth loss. Vitamin D reduces the incidence of dental decay and periodontitis. Adequate level of vitamin D is desirable in all individual for overall wellbeing of the body and simultaneously preventing the dental diseases. Vitamin D level should be regularly monitored and subsequently supplemented as and when required.

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