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Review Article

Vitamin D role in accelerating tooth movement – A review

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ABSTRACT

Purpose: The study focuses on the role of Vitamin D in accelerating orthodontic tooth movement. Vitamin D is a potent stimulator of osteoclastic activity by promoting the recruitment of osteoclast precursors in bone remodeling. In addition, the prevalence of Vitamin D deficiency is high, so it is important to investigate the clinical application of these findings, including the potential use of Vitamin D metabolites to enhance the rate of tooth movement during orthodontic treatment.
Materials and Methods: 3 databases were searched: Scopus, MEDLINE, and Web of Science. The search resulted in 42 publications from databases. 7 studies were included in the qualitative analysis.
Conclusions: The majority of studies showed the locally injected calcitriol to be clinically efficient and cost effective reducing the overall treatment time also it was found initially there was an increase in osteoclastic activity followed by osteoblastic activity, this was also evidenced by several laboratory-based investigations and questionnaire-based assessments pertaining to Vitamin D.

1. Introduction

It's well-known that calcium homeostasis is controlled by Vitamin D. It also regulates intestinal and renal calcium absorption and bone remodeling. Globally, Vitamin D inadequacy is a problem, especially among elderly patients and osteoporosis patients. Factors that contribute to low Vitamin D are lack of exposure to sufficient sunlight and inadequate dietary intake and supplementation; other factors contributing are obesity, age, use of medication, sunscreen, exposure to sunlight, and skin color. Fortunately, we find Vitamin D supplements to be widely available and relatively inexpensive.[1]

The discovery of Vitamin D was done nearly a century ago it was the nutrient which prevented rickets, a devastating skeletal disease characterized by under-mineralized bones.[2]

Vitamin D plays a crucial role in mediating calcium absorption and regulating musculoskeletal health.[3] It is a steroid hormone that has specific receptors in many target organs and tissues. The action is by activating DNA and RNA within the target cell, producing proteins and enzymes which can be used for the bone resorption process.[4] It is also involved in the formation of osteoclasts from precursor monocytes and these effects are produced at much lower doses than other hormones such as prostaglandins.[5,6] The mechanism is believed to be indirect through which $1,25(OH)_2D_3$ induces bone and cartilage calcification, by increasing in the concentration of calcium and phosphate in the serum.[7–9]

Vitamin D synthesis occurs endogenously in the skin, induced via ultraviolet radiation and exogenously through

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dietary sources like oily salt fish (mackerel, salmon, sardines and tuna), cod liver oil and egg yolk. Many countries, like the United States of America, have started fortifying dairy products with Vitamin D due to its scantness in natural foods. Vitamin D, which is obtained through supplements, is converted to 25-hydroxyVitamin D and 1,25-dihydroxyVitamin D. The current recommended daily intake for Vitamin D is 400-600 IU, and for calcium, it is 1,000 to 1,200 mg for people over 50 years of age.[10] Vitamin D deficiency was estimated to be in 1 billion people worldwide.[11,12]

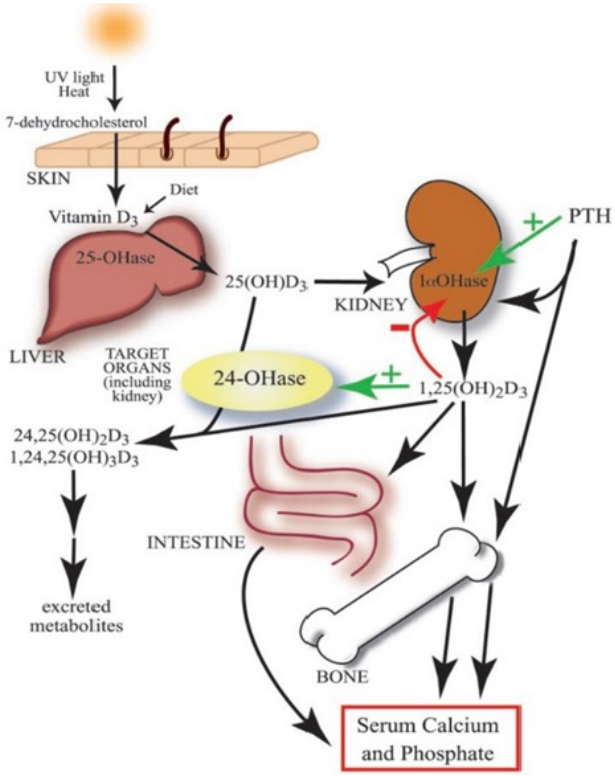
Orthodontic treatment aligns the teeth to achieve good aesthetic and occlusion function. The teeth moves in the alveolar bone by the help of orthodontic force which are applied through the brackets attached to the teeth with the help of composite adhesives; there is cellular and biochemical activity, accompanied by increased remodeling of the periodontal ligaments and alveolar bone, allowing tooth movement. Cytokines, especially interleukins, also play an important role in the Receptor Activator of Nuclear Factor-Kb/ Ligand (RANK/RANKL) system which controls bone remodeling.[13] RANKL is a protein expressed by osteoblasts regulating osteoclastogenesis binding to receptors on the pre-osteoclast surface (RANK) stimulating differentiation and activation into mature osteoclasts which results in bone resorption.[14]

Administration of Vitamin D during orthodontic tooth movement induces osteoclast formation leading to bone resorption, thus the movement of teeth occurs faster with Vitamin D as reported by a previous study.[15] Upon relieving the application of force after active tooth movement, periodontal tissue reorganization occurs consistently for stabilization of the tooth position. The resorbed areas are remodeled for which osteoblasts are needed on the compression side and new bone is formed there, as well as on the tension side. Thus osteoblast-mediated bone formation in periodontal tissue for tooth stabilization after orthodontic tooth movement is enhanced by the potential of $1,25(OH)_2D_3$.[16]

The review was done for the purpose of evaluating the importance of the role of Vitamin D in orthodontic tooth movement and stability.

2. Vitamin D Synthesis, Function, and Metabolism

Vitamin D form was first isolated and identified in 1971.[17] Important role is played by $1,25(OH)D_3$ in the regulation of cellular processes which are associated with carcinogenesis, inclusive of differentiation, proliferation, and apoptosis.[18] The natural form of Vitamin D produced in skin is Vitamin D_3, and another derived from irradiation of ergosterol is Vitamin D2, which occurs to some degree in plankton under natural conditions and Vitamin D_2 is produced from the mold ergot (which contains as much as 2% ergosterol).[19] The bioactive or hormonal form of Vitamin D is $1,25\text{-}(OH)_2D_3$ also known as calcitriol. Sequential hydroxylation's of Vitamin D_3 generates it from a secosteroid precursor obtained from the diet or produced in the skin upon exposure to UV light. The first hydroxylation of Vitamin D_3 occurs at the C-25 position which is catalyzed by Vitamin D-25-hydroxylase in the liver to produce 25-hydroxyVitamin D_3 [$25(OH)D_3$], it is also the major circulating form of Vitamin D in mammals. The substrate for a second hydroxylase is $25(OH)D_3$, the renal $25(OH)D_3$-1α-hydroxylase (1αOHase), resulting in the production of the most bioactive metabolite, $1,25\text{-}(OH)_2D_3$. A classic endocrine feedback system operates to tightly control serum levels of $1,25\text{-}(OH)_2D_3$.[20,21] For example, low serum calcium and phosphorus levels stimulates renal 1αOHase activity and PTH. The expression of 1αOHase is negatively regulated by high levels of $1,25\text{-}(OH)_2D_3$.Inactivation, or catabolism, of Vitamin D metabolites is initiated by the ubiquitous enzyme 25-hydroxyVitamin D3-24-hydroxylase (24OHase) to generate either $24,25(OH)_2D_3$ or $1,24,25(OH)_3D_3$. The 24-hydroxylated metabolites are further degraded and eventually excreted as either calcitroic acid or 23-carboxyl derivatives.$1,25\text{-}(OH)_2D_3$ regulates this catabolic process and stimulates 24OHase expression to prevent excessive synthesis of the hormone. $1,25\text{-}(OH)_2D_3$ operation is by negative feedback loop by inducing expression of the catabolic enzyme 24-OHase and by inhibiting expression of the anabolic enzyme 1αOHase. In response to low serum calcium, PTH hormone is produced which stimulates 1αOHase expression in the kidney and promoting calcium mobilization from the bone and reabsorption from the kidney (figure), $1,25\text{-}(OH)_2D_3$, thus brings about calcium absorption in the intestine and release of calcium from the skeleton.[22]



3. Bone Remodeling

Bone remodeling, also known as bone turnover, predominantly occurring on the endosteal surface and much less on the periosteal surface. The size or shape of the bone is not changed by Bone remodeling but it is responsible for maintaining integrity by the removal and repair of the damaged bone. The three major sequential phases of cellular activity at the remodeling site are activation, resorption and formation.

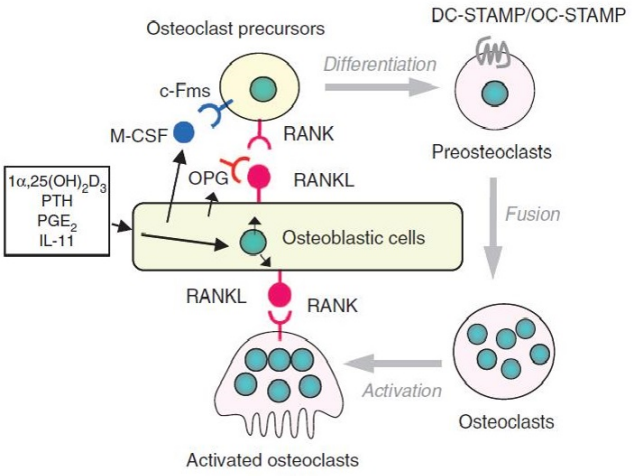
In the activation phase there is detection of an initiating remodeling signal which activates osteoclastogenesis, like the direct mechanical strain of the bone and endocrine signal such as PTH.

In the resorptive phase the osteoclastic bone resorption takes place as well as osteoblastic and osteocystic activity, signaling, recruitment and promotion of osteoclastic proliferation and differentiation.

In the formation phase, the paracrine signaling mechanism happens, allowing the transition from bone resorption to bone formation.[23]

For maintaining bone health, Vitamin D and Calcium are essential. Bone is dynamically remodeled throughout the entire lifespan replacing the damaged bone and adapting to the mechanical load, by the balanced and coupled actions of bone-forming osteoblasts and bone resorbing osteoclasts. Balanced bone remodeling is essential for the maintenance of bone mass and skeletal integrity. Bone remodeling is a complex process that is regulated by a variety of endogenous and exogenous factors. Primarily, it is regulated through the RANK/RANKL/OPG system acting directly on osteoblast/stroma cells and osteoclast precursors. RANKL, expressed by osteoblasts/stroma cells, binds to the RANK receptor on osteoclast precursors inducing osteoclastogenesis. Osteoblast-produced OPG functions as a decoy receptor, blocking the effects of RANKL. Many local and systemic factors regulating bone remodeling – including transforming growth factor-ß, bone morphogenic proteins,cytokines-likeIL-1ß, IL-6 and tumor necrosis factor-a, hormones such as PTH, 1,25-VitD_3 and oestrogen – mainly signal by influencing the RANK/RANKL/OPG system on osteoblasts/stroma cells, thus keeping the system in balance.[24]

Bone resorption-stimulating factors act on osteoblastic cells to induce the expression of RANKL as a membrane-associated factor. Osteoblastic cells constitutively produce M-CSF (Macrophage colony-stimulating factor). Osteoclast precursors express receptors RANK and c-Fms and differentiate into osteoclasts in the presence of RANKL and M-CSF. Osteoblastic cells secrete OPG, which inhibits the RANKL–RANK interaction between osteoblastic cells and osteoclast precursors. Multinucleated osteoclasts also express RANK, and RANKL induces the bone-resorbing activity of osteoclasts via the interaction with RANK. Multinucleated osteoclasts are formed by cell–cell fusion of mononuclear preosteoclasts. The dendritic cell-specific transmembrane protein (DC-STAMP), a seven-transmembrane protein, was first identified as a protein responsible for the fusion of preosteoclasts. No multinucleated osteoclasts were observed, but many preosteoclasts were detected in DC-STAM VDR null mice. The bone-resorbing activity of DCSTAMP null preosteoclasts was lower than that in multinucleated osteoclasts. DC-STAM mice develop mild osteopetrosis. osteoclast-stimulatory transmembrane protein (OC-STAMP), another seven-transmembrane protein, was also involved in the fusion of preosteoclasts. OC-STAMP null mice exhibited a complete lack of cell–cell fusion of preosteoclasts, although the expression of DC-STAMP was normal in these cells. These results suggest that the fusion of osteoclasts is regulated by both OC-STAMP and DC-STAMP.[25]



The function of Vitamin D is to increase serum calcium concentrations by 3 separate ways. First, it is the only hormone inducing the proteins involved in active intestinal calcium absorption. also, the active intestinal absorption of phosphate is stimulated by this hormone. Secondly, calcium concentrations in blood remain in the normal range even when an animal is placed on a no-calcium diet by this hormone. Thus, an animal should possess ability to mobilize calcium in the absence of calcium coming from the environment, ie, through enterocytes. Also, Vitamin D hormone stimulates osteoblasts to produce receptor activator nuclear factor-κB ligand (RANKL). RANKL thus stimulating osteoclastogenesis and activating resting osteoclasts for bone resorption.[26]

Thus, Vitamin D hormone role in allowing individuals to mobilize calcium from bone is very important especially when calcium is absent from the diet, however, in vivo both Vitamin D and parathyroid hormone are required for this mobilization even.[27,28]

4. Vitamin D Status

Concentrations of Vitamin D are the best measure of Vitamin D status. Generally considered deficient are Levels <20 nmol/L, insufficient levels are levels 20-40 nmol/L, sufficient levels 40-60 nmol/L, and likely harmless levels are levels>80 nmol/L. Several factors influence Vitamin D levels, like age, gender, diet, sunlight exposure, climate, and altitude. More than 40% of the UK population experiences Vitamin D insufficiency. During winter, this figure is generally much higher and the risk of insufficiency increases with age, adolescents being the most affected group among the young population. High levels of Vitamin D are generally found in the populations of Norway and Sweden, due to the high intake of fish and cod liver oil. The populations of Spain, Italy, and Greece have been attributed to sun avoidance and air pollution resulting in relatively lower levels of Vitamin D. Whereas in the Middle East and Asia, Vitamin D deficiency in children and adults is high, which may be probably related to skin pigmentation and sun avoidance.[29–31]

It is estimated by many researchers that the oral dose of Vitamin D_3 to attain and maintain 25(OH)D levels >80 nmol/L is 2200 IU/d if baseline levels are 20 to 40 nmol/L, 1800 IU/d if levels are 40 to 60 nmol/L, and 1160 IU/d if levels are between 60 and 80 nmol/L.[32]

5. Tooth Movement Accelerating Methods

Tooth movement is divided into three phases: the initial phase, rapid movement after the application of force; then the lag period, with little or no movement, followed by last phase, where there is gradual or sudden increase of movement. In the acute phase of tooth movement, there are acute inflammatory responses, which are characterized by leucocytes migrating out of blood capillaries, producing cytokines, which stimulate the excretion of prostaglandins and growth factors. And the chronic phase involves the proliferation of fibroblast, endothelial cells, osteoblasts, and alveolar bone marrow cells remodeling process. Experiments have been done using these molecules exogenously to enhance tooth movement both in animal experiments and humans. Examples of these are Vitamin D, prostaglandin E (PGE), cytokines that including lymphocytes and monocytes-derived factors, receptor activator of nuclear factor kappa B ligand (RANKL), and macrophage colony-stimulating factor (MCSF).[33]

6. Effect of Vitamin D on Tooth Movement

One of the earlier attempts made by Boyce and Weisbrode 1985 on female Sprague Dawley rats they evaluated the outcome of calcium rich diets and Vitamin D metabolite injection on bone formation. They found a substantial increase in the number of osteoblasts in treated rats compared to controls. As anticipated, the calcium and phosphorus levels increased and they concluded in their study that the experimental group experienced a net increase in bone formation.

Collins and Sinclair in 1988 demonstrated that intra-ligamentary injections of Vitamin D metabolites causes an increase in the number of osteoclasts, resulting in the rate of bone resorption, thus there was increase in the rate of tooth movement during canine retraction in cats. Total tooth movement on experimental group was 3.25 (1.94) mm and 2.04(1.27) mm in control group. There was significant difference in the tooth movement($p \leq 0.05$).

Later in 2004 Kale et. al. compared the effect of the administration of prostaglandin and 1,25-dihydroxy cholecalciferol (1,25 DHCC) on tooth movement. In both it was found that the amount of tooth movement was significant when compared to controls. In the experimental

Study	Sample	Number		Method		Tooth movement
		Experimental	Control	Experimental	Control	
Boyce & Weisbrode (1985)	Female Sprague Dawley rats	46	26	135 ng 1,25(OH)D, daily for 1,2,3,4.6,8, or 10 days	IP dose of ethanol	Increase tooth movement
Collins & Sinclair (1988)	Cats	10	10	With 1,25D injections	DMSO injections	Increase tooth movement
Kale et al (2004)	Male Sprague rats	37	5	Group 3: DMSO Group 4: 1,25-DHCC Group 5: Prostaglandin	Group 1: With no orthodontic force Group 2: With orthodontic force	Increase tooth movement
Kawakami & Takano-Yamamoto (2004)	Male Wistar rats	16	16	Group 1: Elastic band & Vichelon injection on right side Group 2: Elastic band & injection of 1,25-DHCC on the left side	Group 3: No elastic band & injection of vichelon on right side Group 4: No elastic band & injection of 1,25-DHCC on the left side	Improves stability after orthodontic treatment
Al-Hasani N R et al (2011)	Iraqi patients	15	15	Left side 15pg, 25pg, 40pg/0.2ml calcitriol in 10% DMSO	Right side 0.2 ml DMSO injection	25pg produced 51% faster OTM
Al-Sayag et al (2014)	Male albino rabbits	15	15	Group 1: 20μl injection of 1,25 DHCC in DMSO right side	Group 2: 20μl injection of DMSO in left side 3 times/week for 3 weeks	Increase tooth movement
Ida-Bagus Narmada (2019)	Female Wistar rats	12	12	Group K1: vit D on 7th day Group K2: vit D on 14th day	Group K3: without vit D Group K4: without vit D on 14th day	

group their was increase in the number of Howship lacunae and capillaries on the pressure side. also, the number of osteoblasts on the external surface of the alveolar bone was increased following the administration of 1,25 DHCC in comparison to prostaglandin administration. Thus, the authors concluded the role of 1,25 DHCC in facilitating tooth movement through the regulation of bone deposition and the resorption processes

Some investigators like Kawakami and Takano-Yamamoto in 2004 hypothesized that calcitriol may improve bone formation and periodontal tissue remodeling by increasing osteoblastic activity, which in turn would improve the stability of the teeth position after orthodontic tooth movement. It was found by some researches that there was an increase in the mineral appositional rate on alveolar bone after application of an orthodontic force and injection of calcitriol in the submucosal palatal area of the rats, who were subjected to tooth movement. In doing so, they found that calcitriol has a potent effect on bone formation and resulted that the use of calcitriol may promote the reestablishment of tissue supporting the teeth after orthodontic treatment. In humans, Al Hasani NR et. al. 2011 evaluated the clinical efficacy of locally injected Vitamin D_3 in accelerating orthodontic teeth movement (OTM) and reducing treatment time and cost. Statistically non-significant differences were reported for OTM between control and experimental sides, and among the three groups. However, on clinical efficacy basis, the

dose of 25 pg. calcitriol produced about 51% faster rate of experimental canine movement compared to control, while each of the 15 pg and 40 pg doses resulted in about 10% accelerated OTM. Furthermore, the periapical radiographs showed no damaging effect of calcitriol to the surrounding tissues. In conclusion, for the first time they reported that locally injected calcitriol, in dose dependent pattern, is clinical and cost effective in humans. Al-Sayag et. al. 2014 measured the amount of tooth movement and bone density from digital radiograph using planmeca Dimaxis Pro X-ray machine with Dimaxis classic imaging software at ten time points and they concluded that the 1,25 DHCC side had increased OTM when compared to its contra-lateral control side at all time intervals. Although the 1,25 DHCC side had higher bone density than the control side in all sites and at all time intervals but the difference was insignificant. The bone density increased with time. Moreover, the bone density increased progressively from cervical to apical area in both sides, in addition the medial bone density was higher than lateral bone density. Recently, in 2019, Ida Bagus Narmada et. al. indicated that during pregnancy without Vitamin D administration, the number of osteoclasts and RANKL expression was lower than groups with Vitamin D. There was a significant difference in osteoclast number and RANKL expression between groups. The orthodontic tooth movement was done by 8.0 mm-long Nickle-titanium coil spring, which was placed between the maxillary central incisors to move the molar towards the mesial and was fixed using 0.07 stainless steel ligature wire around the maxillary incisor with 10 g/mm^2 force measured using a tension gauge daily.

7. Conclusions

Several laboratory-based investigations and questionnaire-based assessments of Vitamin D and administration of exogenous biological molecules undergoing orthodontic treatment have been extensively tested in animal experiments, but clinical trials on humans are limited. Furthermore, studies are needed in the future, as many of these mechanisms in humans are not fully understandable. The dose-dependent mechanism of Vitamin D should be investigated further to reduce the treatment time and cost by enhancing the orthodontic tooth movement.

8. Source of Funding

None.

9. Conflict of Interest

None.

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