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The Significance of Vitamin D in the Development of the Periodontium

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ABSTRACT:

It has been known for a long time that one of the most important functions of this secosteroid is to regulate calcium levels, which allows vitamin D to play an important role in the preservation of skeletal health. There is a wealth of research that demonstrates a correlation between oral bone mineral density and some measure of systemic osteoporosis. This research also suggests that osteoporosis and low bone mass may be factors that contribute to the development of periodontal disease. In recent years, the nonskeletal effects of vitamin D have received increased attention for a number of different reasons. It has been established that a great number of cells that are not related with calcium homeostasis exhibit membrane receptors for vitamin D. [Citation needed] These include activated T lymphocytes and B lymphocytes, as well as cancer cells from the skin, placenta, pancreatic, prostate, and colon. In addition, vitamin D "insufficiency" is a worldwide epidemic, and epidemiologic data has connected this illness to a multitude of chronic health problems, including cardiovascular and autoimmune diseases, hypertension, and a number of malignancies. This condition is a worldwide epidemic. It is interesting to note that there is accumulating evidence supporting the concept of "continual vitamin D adequacy" in the context of the maintenance of periodontal health. This link between lower serum levels of vitamin D and higher gingival inflammation has been uncovered. It is possible that a significant part of this process is determined by the capacity of vitamin D to influence both the innate and the adaptive components of the host response. In this review, we will investigate the skeletal and nonskeletal activities of vitamin D, as well as its possible role in preventing damage to the periodontium and controlling the healing of periodontal wounds.

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Keywords: immune response; periodontal disease; vitamin D; wound healing.

INTRODUCTION:

Vitamin D is a fat-soluble vitamin that can either be produced endogenously in the skin in response to enough exposure to sunshine or consumed in food in order to receive it. Vitamin D can be gained in either way (1). Because it helps maintain normal calcium levels in the body, its significance to skeletal health has been acknowledged for a very long time (2). New data supports protective correlations between appropriate serum levels of vitamin D metabolites and cardiovascular disease, diabetes, hypertension, and cancer, probably through the regulation of inflammatory pathways. These diseases include cardiovascular disease, diabetes, and cancer (1,3).

It has also been demonstrated that vitamin D, which possesses anti-inflammatory qualities and the ability to induce the generation of antimicrobial peptides such as defensins and cathelicidin, may have a beneficial effect on dental health (4). A higher vitamin D consumption may help protect against the progression of periodontal disease in older men, according to research that was published not too long ago (5).

Endocrine mechanisms and autocrine/intracrine processes are both utilised in the functioning of vitamin D within the body. Endocrine mechanisms are the more common of the two. The endocrine route includes a mechanism that is referred to as "classic," and it is linked to increasing the amount of calcium that is absorbed by the intestinal tract as well as the activity of osteoclasts. Gene signalling and expression, protein synthesis, hormone production, the regulation of immunological and inflammatory responses, and cell turnover are all related with "nonclassic" systems that utilise autocrine and intracrine pathways (6).

There is a debate going on right now over the correct amount of vitamin D that one should take in every day. In 2010, the Institute of Medicine issued revised guidelines for the amount of vitamin D that should be consumed through diet. These recommendations were as follows: 400 IU/d (0–12 months of age); 600 IU/d (1–70 years of age); and 800 IU/d for persons over the age of 70. (7). According to the opinions of a number of industry professionals, these vitamin D levels recommended by the Institute of Medicine may prevent clinical vitamin D insufficiency (typically reported as 30 ng/mL) and deficiency (typically reported as 20 ng/mL), but they are significantly lower than the dose required to achieve optimal vitamin D status, which is regulated through autocrine mechanisms (8,9). According to estimates provided by the Third National Health and Nutrition Examination Survey (NHANES III), 77% of American adults lacked "sufficient" levels of vitamin D. (6). In this review, we will analyse the vitamin D pathways that are connected with controlling musculoskeletal health, as well as the influence that osteoporosis has on periodontal disease. The impact of vitamin D on periodontal inflammation and health, as well as the possible role of vitamin D in regulating wound healing after periodontal therapy, will also be investigated as part of this study.

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Vitamin D's impact on bone metabolism and its relationship to osteoporosis

Vitamin D, as an essential component of the interactions that take place between the kidney, bone, parathyroid gland, and gut, contributes to the regulation of skeletal and mineral homeostasis (10). Vitamin D is necessary for the maintenance of healthy skeletal tissue because of its function in regulating the amount of calcium that is found outside of cells. The active form of vitamin D (calcitriol, also known as 1,25-(OH)2D3) (11) is produced in the skin when exposed to ultraviolet light, and it is then modified in the liver and kidney before being released into the circulation. It does this by binding to carrier proteins in the plasma, which then carry it to the appropriate organs and tissues (12). The binding of vitamin D to the vitamin D receptor (VDR), a member of the steroid nuclear receptor superfamily, is the mechanism by which vitamin D exerts its effects on the body. The binding of vitamin D activates the vitamin D receptor, which has multiple roles including acting as a transcription factor for target genes (13–15).

The capacity of the small intestine to absorb calcium and phosphate from the diet is improved by the use of vitamin D. Vitamin D stimulates calcium uptake and transport in intestinal epithelial cells by binding to and activating the vitamin D receptor (VDR). This is accomplished through an increase in the expression of epithelial calcium channels, a cytosolic calcium-binding protein, and plasma membrane proteins that are responsible for the delivery of calcium to the bloodstream (16). Vitamin D helps to produce optimal circumstances for bone mineralization by boosting intestinal uptake of both calcium and phosphate. Vitamin D is essential for the development and maintenance of a mineralized skeleton since it is required for both of these processes (10, 17).

Osteoprotegerin, or OPG, is a RANKL antagonist that is produced by osteoblasts and other cells, such as activated CD4+ T lymphocytes, and is one of the most significant regulators of bone remodelling. Other molecules, such as RANKL, are also essential regulators of bone remodelling (18–21). In addition, osteoblasts, CD4+ T lymphocytes, and other cells (such as gingival fibroblasts and epithelial cells) all release interleukin (IL)-6, which has the ability to increase the creation of RANKL and drive the formation of osteoclasts (20). The differentiation of osteoclast progenitor cells into adult osteoclasts is triggered when RANKL binds to RANK, which is expressed on the surface of osteoclast progenitor cells. The OPG protein functions as a soluble receptor for the RANKL protein, blocking the interaction between RANK and RANKL as well as the maturation of osteoclast progenitor cells. It is possible, then, for the production of mature osteoclasts to be determined by the relative ratio of RANKL to OPG in the microenvironment of osteoclast precursors. The promoter of the RANKL gene contains vitamin D and glucocorticoid response elements, and studies have shown that the vitamin D–VDR complex stimulates RANKL expression in cells such as osteoblasts and bone-marrow-derived stromal cells. [Citation needed] [Citation needed] [Citation needed] [Citation needed] [Citation needed] [Citation needed] [Citation needed] [C (22,23). This combination of increased RANKL expression and decreased expression of OPG

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mediated by vitamin D would favour the differentiation and activation of osteoclasts, as well as accelerated bone resorption. Vitamin D can downregulate OPG (22–25).

On the other hand, low levels of vitamin D may increase circulation levels of parathyroid hormone, which can indirectly induce bone resorption and increase blood calcium levels. This can happen if there is not enough vitamin D in the body (26). The hormone produced by the parathyroid gland can attach to osteoblasts and cause them to produce more RANKL while at the same time suppressing OPG, which ultimately results in an increase in osteoclastic activity. However, Hofbauer et al. (27) have shown that vitamin D has a stimulating impact on OPG, and Kondo et al. (24) have observed that although vitamin D initially represses OPG, long-term exposure to vitamin D leads to a recovery of OPG expression. Both of these findings contradict each other. This points to the possibility that the catabolic effects of vitamin D are only temporary. Indeed, it has been demonstrated that vitamin D has anabolic effects on osteoblasts in vitro and in a rat model of osteoporosis. These effects include the activation of osteopontin and alkaline phosphatase (28, 29). Therefore, vitamin D seems to stimulate bone resorption, but after longer periods of exposure, it may facilitate osteoblast proliferation and differentiation by regulating the transcription of several genes associated with the osteoblast phenotype. This occurs because vitamin D appears to regulate the expression of several genes associated with the osteoblast phenotype.

Through its influence on inflammation, vitamin D can also have an effect on the metabolism of bone. It has been shown by a number of studies that vitamin D possesses anti-inflammatory properties, which, given that chronic inflammation can lead to bone loss, could have a beneficial influence on bone levels. For instance, vitamin D inhibited MAPK signalling, which led to a decrease in the production of the pro-inflammatory cytokine tumour necrosis factor-alpha (TNF-a), as well as the pro-inflammatory cytokine IL-6, in human peripheral blood mononuclear cells that had been stimulated with lipopolysaccharide. In human monocytes, vitamin D decreased lipopolysaccharide's induction of IL-6 levels as well as the generation of RANTES (CCL5, which is a chemoattractant for T cells) and a B lymphocyte chemoattractant. These effects were observed in response to the addition of lipopolysaccharide. Additionally, the synthesis of IL-17 from peripheral blood mononuclear cells was decreased by vitamin D, whereas the production of IL-4 from these cells was enhanced.

It is possible that IL-17 is implicated in a wide variety of inflammatory illnesses, including periodontitis, as it is known to induce the production of IL-6 and the neutrophil attractant IL-8 . IL-4 is responsible for significant bone-promoting and anti-inflammatory actions. In addition to this, vitamin D lowers the levels of prostaglandins in tumour cell lines by blocking the creation of cyclooxygenase-2, and it prevents the activation of nuclear factor kappaB, which is a crucial regulator in the development of proinflammatory molecules. Because of its ability to reduce inflammation, vitamin D is being investigated as a potential

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supplementary therapy for the treatment of a number of persistent disorders, including asthma and arthritis. On the other hand, vitamin D may potentially have properties that promote inflammation. For instance, it can activate the expression of trigger receptor expressed on myeloid cells (TREM) in gingival epithelial cells, which may result in the secretion of proinflammatory cytokines. Furthermore, when combined with a cross-linking anti-TREM antibody, vitamin D was able to upregulate the expression of IL-8 in these cells. Several studies have demonstrated that there is a correlation between low levels of serum vitamin D and increased expression of osteoclastogenesis-stimulating proinflammatory cytokines such as IL-6 and TNF-a. These cytokines are produced in response to low levels of serum vitamin D. A lack of vitamin D and calcium in one's diet on a consistent basis can result in a negative calcium balance, poor bone mineralization, and bone loss. Rickets, which is characterised by limited growth and malformation of the long bones, can be caused by a lack of vitamin D in children. A lack of vitamin D in adults can cause osteopenia and osteoporosis, as well as increasing the risk of fracture. In people who suffer from Parkinson's disease, a lack of vitamin D and the compensatory hyperparathyroidism that often accompanies it has been linked to an increased risk of vertebral fractures.

Patients over the age of 50 who presented to an emergency department with a fracture had a vitamin D insufficiency or deficiency in 90% of the cases, according to Bours et al. and other researchers, and many of these patients had one or more secondary causes of osteoporosis, including a vitamin D deficiency or insufficiency, according to Bours et al. and other researchers. Another study, while failing to support vitamin D supplementation to improve bone density in healthy children with normal vitamin D levels, did suggest that supplementation might significantly improve bone health in children with low serum vitamin D, although these results were not clear. While this study failed to support vitamin D supplementation to improve bone density in healthy children with normal vitamin D levels, it did suggest that supplementation might significantly improve bone health in children with (24). In premenopausal women who had systemic lupus erythematosus, increased levels of vitamin D were shown to maximise the bone mineral density response to bone-active drugs, according to a study that was conducted not too long ago by Yeap and colleagues (22). The optimal bone mineral density response to bisphosphonate therapy in the hip and spine may be achieved, according to the findings of other research, when adequate amounts of vitamin D are present.

Periodontal disease.

According to Jabbar et al., osteoporosis is a chronic, complex disease, just like periodontal disease, and the two diseases may share some risk factors like age, genetic polymorphisms, hormonal and/or nutritional deficiencies, as well as some systemic disorders and drugs. However, it is unknown how much overall bone loss contributes to oral bone loss, how strong any relationship between osteoporosis and periodontal disease is, or what processes link the two together. Numerous research have established a correlation between systemic bone

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mineral density/low bone mass/osteoporosis and alveolar bone density and tooth loss; however, another study discovered that these correlations were either weak or Martinez-Maestre et al. carried out a comprehensive analysis of clinical trials that investigated the possibility of a connection existing between osteoporosis and periodontitis. A favourable link was established in the majority of cases based on maxillary and/or mandibular radiographs, however the results were ambiguous when based on clinical periodontal examination.

They came to the conclusion that the majority of the research that were looked at provided evidence of a connection between osteoporosis and periodontitis, but they recommended that further studies that were better controlled be conducted.

However, it can be challenging to accurately interpret and compare the findings of some studies due to factors such as small sample sizes, inadequate control of confounding factors, different types of study populations, and different methods or study sites used to evaluate osteoporosis and periodontitis (21). In addition, a number of studies have provided evidence suggesting that there is no connection between bone mineral density and the loss of alveolar bone. For instance, Elders et al. (23) discovered that there was no significant link between systemic bone loss and alveolar bone height. Based on these findings, the researchers came to the conclusion that systemic bone loss does not play a substantial role in the pathophysiology of periodontitis. According to the findings of Klemetti and colleagues (24), there is not a significant association between the amount of existing trabecular or cortical bone density and the pace of alveolar crestal bone loss. The researchers Famili et al. (22) looked at 398 postmenopausal women and found minimal evidence to indicate a link between edentulousness, periodontal disease, and longitudinal changes in bone mineral density. The investigation was conducted over the course of several years.

Researchers in this field have cited the need for additional studies that are on a larger scale and better controlled to determine the role of osteopenia and osteoporosis on the prevalence and severity of periodontitis. In addition, researchers in this field have cited the need for prospective studies to determine if osteopenia and osteoporosis are associated with the onset and progression of periodontal disease. However, Jeffcoat noted that the majority of the current evidence shows an association between oral bone mineral density and some measure of systemic osteoporosis (i.e. dual-energy X-ray absorption). This is the case even though there are studies in the published literature that contradict each other. According to the information presented, osteoporosis and poor bone mass may be factors that contribute to the development of periodontitis.

The influence that vitamin D has on the body's immunological response

The finding of the vitamin D receptor (VDR) in tissues that are not involved in the regulation of calcium homeostasis, such as the skin, placenta, pancreas, prostate and colon cancer cells, and activated T and B cells, led to the possibility of vitamin D having effects outside of the skeleton. In addition, antigen-presenting cells, such as macrophages and dendritic cells,

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produce the enzyme 1a-hydroxylase, which is responsible for activating vitamin D. CYP27B1 is another name for this enzyme. Therefore, these cells have the ability to convert the precursor form of vitamin D, 25(OH)D3, which is the most prevalent form in circulation, into the active form, 1,25-(OH)2D3. By binding to the VDRs of the cells and so enhancing transcriptional control, they are able to elicit reactions from the cellular level. This autocrine mechanism is essential to two key aspects of immune function, namely innate antibacterial activity and the presentation of antigen to cells of the adaptive immune response, such as activated T lymphocytes. Both of these aspects of immune function are vital to the protection of the body against pathogens. It was first shown by Liu et al. that enhanced synthesis and activity of 1,25-(OH)2D3 in macrophages occurred in response to cellular sensing of pathogens via toll-like receptors. In a manner that is dependent on vitamin D, this resulted in an increase in the production of the antibacterial proteins cathelicidin and beta-defensins, as well as an improvement in the killing of bacteria. Other cell types inside tissues such as the skin, lungs, and placenta, as well as "barrier sites" in general, have been observed to have antibacterial responses that are comparable to these. As a consequence of this, it is not out of the question that a lack of vitamin D could lead to reduced antibacterial responses as well as increased vulnerability to a wide variety of infectious diseases.

Vitamin D's immunomodulatory actions also govern the acquired immune response, also known as the adaptive immune response. The proliferation, maturation, and differentiation of dendritic cells were all decreased by 1,25-(OH)2D3, according to in-vitro investigations. Additionally, there was a reduction in the levels of CD40, CD80, CD86, and major histocompatibility complex class 2, which led to a reduction in the activation of T cells. After being exposed to 1,25-(OH)2D3, monocytes and macrophages exhibited decreased antigen-presenting and T-cell-stimulatory capacities, which was consistent with what was observed in dendritic cells.

In addition to this, 1,25-(OH)2D3 prevents the expression of pro-inflammatory cytokines in monocytes. These cytokines include IL-1, IL-6, TNF-a, IL-8, and IL-12. It was first thought that the ability of 1,25-(OH)2D3 to reduce proliferation and immunoglobulin synthesis in B lymphocytes was an indirect impact mediated by T-helper (Th) cells. However, more recent research has shown that this is not the case. In more recent research, it was demonstrated that 1,25-(OH)2D3 had direct effects on the homeostasis of B cells. This paper also emphasised its ability to suppress the differentiation of B lymphocytes to plasma cells and class-switched memory B cells, which suggests a potential role for vitamin D in B-lymphocyte-related illnesses such as systemic lupus erythematosis. [Citation needed]

Intriguingly, the authors discovered that patients with systemic lupus erythematosis exhibited reduced serum levels of both 25 (OH) D3 and 1,25-(OH)2D3.

It is possible that the potential of vitamin D to influence T-lymphocyte proliferation and function is the one activity of vitamin D within the adaptive immune system that has received

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the most research attention. After the discovery of VDR in activated T lymphocytes, a number of research groups found that 1,25-(OH)2D3 induced highly effective antiproliferative responses in these cells.

T-helper cells appear to be the primary target among the many subsets of T lymphocytes, with 1,25-(OH)2D3 reducing proliferation as well as modifying cytokine production by these cells. This is because of the way that 1,25-(OH)2D3 works. In this way, vitamin D can serve to "shape" the adaptive immune response by selectively boosting certain T-helper-cell subsets while inhibiting other helper T cells. This allows vitamin D to play a role in the regulation of the adaptive immune response. In a test tube, 1,25-(OH)2D3 suppresses the synthesis of cytokines by Th1 cells while encouraging the production of Th2 cytokines.

In addition, it appears that Th17 cells are extremely sensitive to the suppression caused by 1,25-(OH)2D3, and a reduction in the production of IL-17 can be attributed to a direct transcriptional mechanism. In addition, 1,25-(OH)2D3 is responsible for the induction of regulatory T cells, which have been demonstrated to play a role in suppressing "overexuberant" T-cell activity, which has been linked to autoimmune responses. Therefore, the amount of vitamin D or its active serum metabolite may operate to shape the adaptive immune response by selectively stimulating various T-helper-cell subsets and their related cytokines. This may be the case since vitamin D is a serum metabolite. Throughout the course of research, it has been demonstrated that T cells play an essential part in regulating the evolution of the periodontal lesion.

T lymphocytes were shown to be the predominant cell type in what is known as the "early lesion" in the seminal research conducted by Page and Schroeder. The production of cytokines by T lymphocytes was a significant factor in the course of this lesion through the established and advanced stages, during which B lymphocytes and plasma cells predominated. Since the middle of the 1980s, when several Th-cell subsets were first identified, there have been numerous attempts to link particular subsets with the progression of periodontal disease. However, there is still no general agreement on this topic. Multiple studies have found a correlation between an elevated level of IL-17 expression and production and a higher prevalence of periodontal disease. Th17 activity and increased production of cytokines and growth factors, such as IL-1, IL-6, IL-21, and transforming growth factorbeta, that selectively push T cells along this pathway may place a subject at risk for periodontal disease, according to recent reports. These cytokines and growth factors are known to selectively push T cells along this pathway. The ability of vitamin D to inhibit the Th17 pathway and stimulate the production of anti-inflammatory cytokines lends credence to the hypothesis that "adequate" levels may serve to inhibit the maturation of Th17 cells and foster an environment that is more amenable to the resolution of periodontal inflammation.

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Vitamin D's effects on inflammation as well as the health of the gums and teeth

It has been demonstrated that vitamin D, and more specifically its hormonally active form, 1,25-(OH)2D3, has anti-inflammatory and antimicrobial effects. These effects are achieved through the modulation of inflammatory cytokine production by immune cells and the stimulated secretion of peptides with antibacterial action by cells of the monocyte–macrophage lineage. The presence of healthy gums and teeth is dependent on a number of factors, including vitamin D.

First, the active hormone 1,25-hydroxy-2D3 is essential for the upregulation of genes by the 1a-hydroxylase enzyme. These genes encode proteins that are necessary for epithelial cells to have tight adherens and gap junctions. This not only improves and reinforces the physical barrier that the epithelial cells have built, but it also makes cell-to-cell communication more effective. Second, vitamin D has a function in the innate immune system as a powerful activator of antimicrobial peptides, which may have antibacterial and lipopolysaccharide neutralising effect against periodontal microorganisms. This activity may help prevent periodontal disease.

According to Wang et al., appropriate circulating levels of vitamin D are required for the formation of cathelicidin and some defensins in the human body. Additionally, 1,25-(OH)2D3 has the potential to promote expression of cathelicidin in keratinocytes and other cell types. Vitamin D's antimicrobial actions are mediated by the vitamin D receptor (VDR), and they are coupled with an up-regulation of the cathelicidin hCAP-18 gene. There is a possibility that the potential of vitamin D to induce defensins and cathelicidin as well as to reinforce the physical barrier in the oral cavity may contribute greatly to the improvement of oral health.

It has been demonstrated that 1,25-(OH)2D3 can stimulate the differentiation of monocytes into macrophages. Macrophages are able to express the enzyme 1a-hydroxylase and create 1,25-(OH)2D3, which in turn promotes lysosomal enzyme activity and phagocytosis. Additionally, 1,25-(OH)2D3 suppresses the synthesis of proinflammatory cytokines by inhibiting the in-vitro synthesis of macrophage mRNA for IL-1, IL-6, and TNFa. This results in a reduction in the production of these cytokines. The findings of Xu et al. demonstrated that vitamin D improved the chemotactic and phagocytic capacity of macrophages. These "perio-protective" properties of vitamin D are extremely tempting for the care of patients with periodontal disease, which is caused by chronic bacterial-induced inflammation. Periodontal disease is the leading cause of tooth loss in adults.

Numerous research have found that increasing one's consumption of vitamin D and/or calcium leads to a reduction in the amount of alveolar bone loss, gingival inflammation, and/or attachment loss. In particular, Dietrich et al. used the data from NHANES III to examine the association between serum vitamin D levels and attachment loss. They discovered an inverse relationship between the two; the lower the levels of vitamin D, the

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more attachment loss that was observed, and the association was found to be independent of factors such as smoking habits and diabetes. They believed that the anti-inflammatory properties of vitamin D were responsible for this inverse connection. A separate analysis of the NHANES III data revealed that individuals with the highest levels of 25 (OH)D experienced 20% less bleeding on probing than did those with the lowest levels. This finding suggests that vitamin D may reduce the risk of gingival inflammation by exerting antiinflammatory effects (22).

The findings that mutations of the VDR gene are related with periodontitis, alveolar bone loss, clinical attachment loss, and/or tooth loss provide further credence to the hypothesis that vitamin D could play a role in maintaining periodontal health. A recent study conducted by Boggess and colleagues lends support to the hypothesis that adequate vitamin D intake is necessary to preserve periodontal health. According to what they found, an inadequate level of vitamin D in the mother is a risk factor for moderate to severe periodontal disease that can occur during pregnancy. Comparatively, pregnant women who suffered from moderate to severe periodontal disease had lower serum levels of 25(OH)D, and they were more likely to have a level of 25(OH)D that was less than 75 nM. This was the case when compared to women who had periodontally healthy gums.

The most current investigation into the connection between total vitamin D consumption and periodontal health in older males was carried out by Alshouibi et al. (5). They discovered that a negative association existed between the total amount of vitamin D consumed and the occurrence of severe periodontitis as well as moderate to severe bone loss. In addition, it appeared that a daily consumption of less than 800 IU/d was the minimum necessary to meet the criteria for preventing moderate to severe periodontitis. Several studies (110,116) found significant associations between the intake of vitamin D and calcium and periodontal health. These studies also suggested that dietary supplementation with calcium and vitamin D may improve periodontal health, increase bone mineral density in the mandible, and inhibit alveolar bone resorption.

Patients who underwent periodontal maintenance and took calcium and vitamin D supplements had better periodontal health than those who did not. This was evidenced by shallower probing depths, fewer bleeding sites, lower gingival index values, fewer furcation involvements, less attachment loss, and less alveolar crest height loss.

The influence of vitamin D levels on the results of periodontal surgery and the healing process

Periodontitis can be identified by the loss of alveolar bone, which is caused by the immunological response of the host to the bacterial infection.

Because vitamin D is so important to the health of bones and the immune system, there is good reason to assume that a shortage in vitamin D could have a harmful impact on the

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periodontium. Patients with severe, chronic periodontitis underwent periodontal surgery and received daily injections of 20 lg of teriparatide (a commercially available form of parathyroid hormone) or a placebo, along with oral calcium (1000 mg) and vitamin D (800 IU) supplementation, for 6 weeks, and were followed for 1 year in a recent longitudinal clinical trial. Patients were then evaluated after 1 year. Teriparatide was related with improved clinical outcomes, greater clearance of alveolar bone abnormalities, and quicker osseous wound healing in the oral cavity when compared to the placebo.

In 2011, the same research group investigated the results of periodontal surgery and the administration of teriparatide in persons who had appropriate levels of vitamin D and those who did not have sufficient levels (26). According to what they found, vitamin D sufficiency in placebo patients at the time of surgery led to a better clinical attachment level gain and a greater pocket-depth reduction when compared with vitamin D deficiency in patients after 12 months. According to these findings, the state of vitamin D may be an important factor in postsurgical recovery. This finding seems to agree with findings from previous studies in which a vitamin D deficiency was associated with compromised osseous healing in the oral cavity in bisphosphonate-associated osteonecrosis of the jaw in preclinical studies. These findings support a role for vitamin D in bone healing in the oral cavity. The authors claimed that an anabolic drug, such as teriparatide, benefits from vitamin D sufficiency in stimulating oral bone growth. This is due to the fact that radiographic outcomes were better in patients on teriparatide who had sufficient levels of vitamin D. A patient who had periodontitis and an intrabony defect was treated with simply open flap debridement surgery, daily systemic dose of 20 mg of teriparatide, oral vitamin D and calcium supplements for six weeks in a case report that proved the concept. There were no periodontal-regeneration methods used in this procedure. The patient's clinical and radiological outcomes improved during the course of the subsequent 4 years of follow-up.

CONCLUSION:

In addition to its ability to maintain bone health through calcium homeostasis, it has been demonstrated that vitamin D is responsible for a multitude of other functions as well. Osteoporosis and low bone mass have both been implicated as potential risk factors for periodontal disease. The majority of the data points to a relationship between oral bone mineral density and some measure of systemic osteoporosis. The accumulation of epidemiological evidence lends credence to the hypothesis that a subject with insufficient levels of vitamin D may be at an increased risk of contracting infectious diseases that have inflammatory pathologic components, such as cardiovascular disease, diabetes, arthritis, cancer, and periodontitis. The capacity of vitamin D to promote the innate immune response by means of the creation of antimicrobial peptides, such as beta-defensins and cathelicidin, would result in the physical barriers being strengthened, so making it more difficult for pathogens to penetrate the epithelium. It's possible that vitamin D's ability to mould the adaptive immune response by selectively boosting certain Th subsets can help foster an

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environment that's more amenable to the resolution of inflammation. These provide a biological explanation to explain some data that suggest a "perio-protective" role for vitamin D, and it is this role that is supported by the findings. It is abundantly clear that well-designed, larger-scale studies are required to be carried out in order to gain a better understanding of the part that vitamin D plays in this process and the amount of the vitamin that is necessary to reach the desired effect. The discovery that vitamin D plays a part in the healing process of wounds and that having "sufficient" levels of it may affect how well periodontal surgery goes may be the most fascinating aspect of this research. It will be necessary to conduct additional robust clinical trials in order to substantiate these findings and, subsequently, to shed light on the mechanisms involved in these processes.

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