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ORAL HEALTH AND VITAMIN D IN ADULT: SYSTEMATIC REVIEW

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ABSTRACT

The role of nutrition in oral diseases has grown in popularity, and recent studies have revealed increasingly important links between nutritional deficiencies and oral pathologies. The levels of vitamin D (VD) have recently garnered an increasing amount of focus in the realm of oral health. VD deficit (VDD) is related with a wide array of oralhealth issues during growth and maturity; decreased VD synthesis may speed up the progression of some of these conditions. In children, severe VDD can produce faulty toothmineralization, which can result in problems in both the dentin and the enamel. As a direct result of this, there is a potential for an increased risk of the beginning stages of dental caries as well as their advancement. In addition, VDD has been linked to a higher prevalence of periodontitis and gingival inflammation, and a number of recent research, both preclinical and clinical, have uncovered putative routes through which vitamin D may interact with the periodontium. The correction of VDD by supplementation may be one factor that contributes to the successful treatment of periodontitis; nevertheless, alveolar bone regeneration treatments conducted in individuals with baseline VDD appearto be more likely to fail. Some forms of oral cancer and osteonecrosis of the jaw may beconnected to vitamin D, as may certain oral malignancies. This study seeks to present thorough evidence of how VD levels should be evaluated to maintain excellent oral health, as well as to describe how VDD may hinder oral development and its function inparticular oral disorders.

Keyword: *Oral Health, Periodontitis, Steroid Hormone, Vitamin D*

INTRODUCTION

Vitamin D is a steroid hormone that you get mostly from the sun, but you can alsoget it from food and supplements. Vitamin D is hard to find in foods, but it can be foundin oily fish (like salmon, mackerel, and herring) and fish oils (e.g., cod liver oil).[1] VitaminD is an umbrella term for Vitamins D2 and D3. Vitamin D2 is made by exposing ergosterol from yeast to ultraviolet light.[2] Vitamin D3 is made by exposing 7- dehydrocholesterol from lanolin to ultraviolet light. 7-dehydrocholesterol has the same biological activity as cholecalciferol (vitamin D3) and is made in the human skin. Serum 25-hydroxyvitamin D (25[OH]D) is a biomarker that is widely used to measure vitamin D status.[3]

Vitamin D is first and foremost a hormone, and its endocrine activity helps keep calcium and phosphate in the blood at the right levels by controlling how well they are absorbed in the gut. Vitamin D also controls cell differentiation, cell maturation, and theinnate immune system.[4] It does this by acting as an autocrine and paracrine agent. In moredetail, the Vitamin D receptor (VDR), which is a molecule that binds to the active form of Vitamin D, is what makes Vitamin D work inside cells.[5] So, the actions of vitamin D depend on how the VDR is controlled for its genomic effects and on a membrane- associated protein for its nongenomic effects. This is because this vitamin changes the expression of a large number of genes. It is thought that it does this for 5–10% of the entire genome.[6]

The role of nutrition in oral diseases has grown in popularity, and recent studies have revealed increasingly important links between nutritional deficiencies and oral pathologies. Caries and periodontal diseases are complex multifactorial diseases that continue to be the two most common diseases worldwide.[7,8] VDD and its pathophysiologic processes are linked to both caries and periodontal disease. The mechanisms by which vitamin D affects oral health go beyond bone metabolism. VDD, according to current research, impairs odontogenesis, resulting in a hypomineralized dentition prone to fracture and caries lesions. VDD has also been linked to poor periodontal health and may be involved in the immune response to periodontal infection.[9–11]

Furthermore, VDD in conjunction with periodontitis has recently been linked to potential systemic consequences during pregnancy, during orthodontic treatment, in postmenopausal women, or in some oral pathologies.[9–11] This article investigates association between oral health and vitamin D in adult.

METHODS

Protocol

The Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 recommendations served as the foundation for this study's approach. These elements had an impact on the laws that were passed.

Eligibility Criteria

This literature review seeks to demonstrate the relationship between oral health and vitamin D in adult by assessing or analyzing previous research on this topic. This is a major concern raised in the research currently being looked at. Researchers enter research that meets the following requirements, including: 1) To be considered for publication, articles must be written in English and highlight or focus on discussing oral health and vitamin D in adult. 2) Articles published after 2017, but before this systematicreview period were considered for this evaluation. The following types of writing will notbe considered for inclusion in publication, for example: editorials, submissions that do not have a DOI, review articles that have already been published, or entries that are verysimilar to those that have already been published in a journal.

Search Strategy

The search for studies to be included in the systematic review was carried out from January, 18th 2023 using the PubMed and SagePub databases by inputting the words:"oral health", and "vitamin D". Where *("oral health"[MeSH Terms] OR ("oral"[All Fields] AND "health"[All Fields]) OR "oral health"[All Fields]) AND ("vitamin d"[MeSH Terms] OR "vitamin d"[All Fields] OR "ergocalciferols"[MeSH Terms] OR "ergocalciferols"[All Fields])* is used as search keywords.

Data retrieval

After conducting a literature review that included a review of the titles and abstracts of previous research, the author modified the inclusion and exclusion criteria. The revised criteria are included in the research's supplementary materials. This clarifiedthe scope of the problem and highlighted the aspects that require further investigation. The author reached this conclusion after conducting research on additional studies with asimilar format. Only studies that met all of the inclusion criteria were considered during the systematic review process.

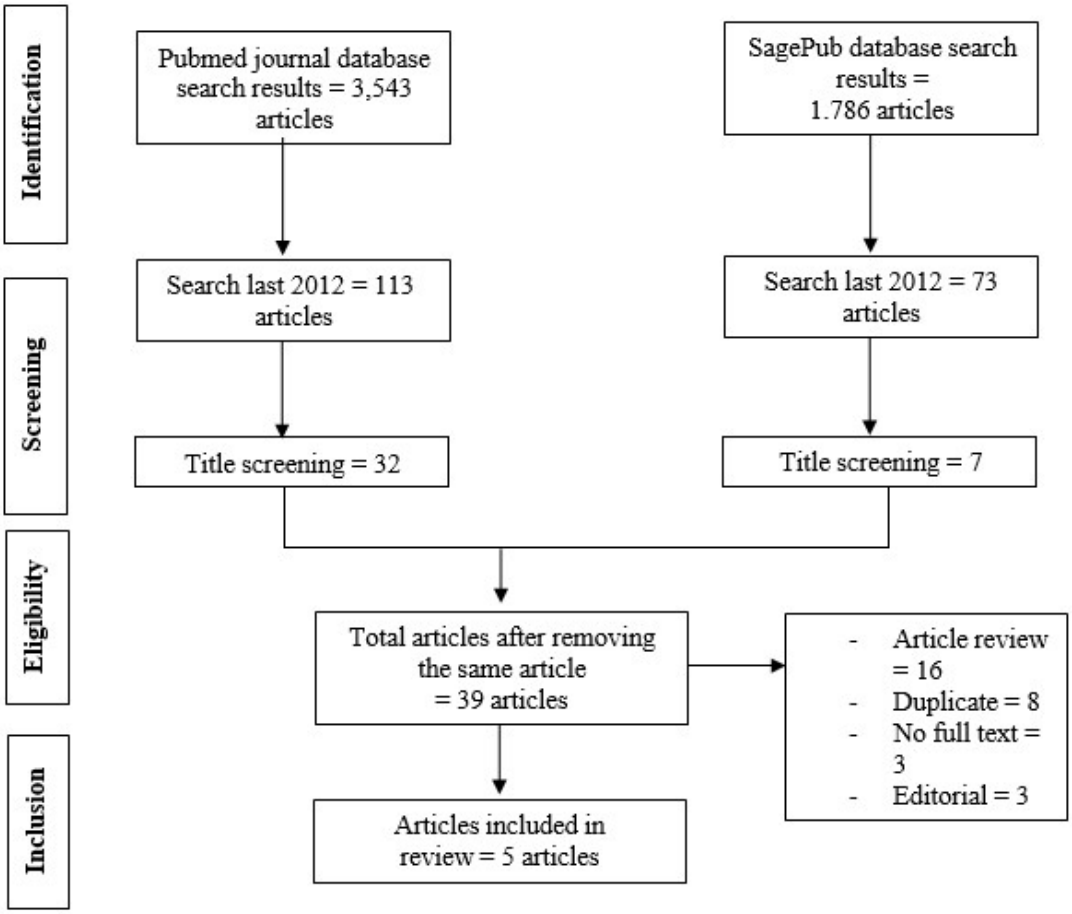

Figure 1. Article search flowchart

This ensured that only pertinent data was discovered. We did not take into accountany research proposals that did not meet all of our criteria. This ensured that a thorough examination would be carried out. This effort yielded data pertinent to the studies, such as their titles, authors, publication dates, locations, research investigation types, and parameters. These are the available item categories. These are skills that can be developed. These data can be presented in a variety of formats depending on the information source.

Quality Assessment and Data Synthesis

Each author conducted an independent investigation of a piece of researchmentioned in the titles and abstracts of the papers before deciding which articles to investigate. The full texts of publications that meet the inclusion criteria for the systematicreview will then be reviewed to determine which papers will be included in the review. This will be done in order to choose which articles will be included in the review. To make the process of selecting articles for the review easier. Which studies have high enough quality to be included in the review?

RESULT

Anbarcioghu, et al (2019)[12] showed serum 25(OH)D concentration in aggressiveperiodontitis (AgP) patients was lower (11.22 ± 4.8 ng/ml) than in controls (16.9 ± 6.4 ng/ml) and in chronic periodontitis (CP) patients (16.13 ± 8.3 ng/ml; overall p value 0.0002). After controlling for age and gender, these relationships remained significant (p = 0.002). There were no significant variations in any bone-related biomarker across the three groups, and there was no connection with periodontal disease indicators. Second study showed periodontitis was not associated with serum PTH in the case-control data or at the baseline of the intervention data. A post-therapy increase in serum PTH was found in 61% of the T1DM patients; in patients with moderate or severe periodontitis (n = 26) the average increase was 0.6 pmol/l (p = 0.016) and in patients with no or mild periodontitis (n = 27) 0.2 pmol/l (p = 0.250).[13]

Table 1. The litelature include in this study

Author	Origin	Method	Sample Size	Result
Anbarcioglu,2019[12]	Turkey	Comparative study	160 subject	The serum 25(OH)D concentration in aggressive periodontitis (AgP) patients was lower (11.22 ± 4.8 ng/ml) than in controls (16.9 ± 6.4 ng/ml) and in chronic periodontitis (CP) patients (16.13 ± 8.3 ng/ml; overall p-value 0.0002). After controlling for age and gender, these relationships remained significant (p = 0.002). There were no significant variations in any bone-related biomarkeracross the three groups, and there was no connection with periodontal diseaseindicators.
Antonoglou,2015[14]	Finland	Case control study	54 period on tit ispatients and 30period on tally healthy controls	Periodontitis was not associated with serum PTH inthe case-control data or at thebaseline of the intervention data. A post-therapy increase in serum PTH was found in 61% of the T1DM patients; in patients with moderate or severe periodontitis (n = 26) the average increase was 0.6 pmol/l (p = 0.016) and in patients with no or mild periodontitis (n = 27) 0.2 pmo l /l (p = 0.250).
Meghil, 2019[15]	USA	Double- blind pilot randomized clinical trial	23 patients with mode rate to severe period on tit is	In addition, the vitamin D supplementation (VDS)group had lower peripheral blood CD3 and CD3+CD8+ cytotoxic T lymphocyte (CTL) counts as well as lower levels of pro-inflammatory salivary cytokines than the control group. Vitamin D supplementation increasedserum 25(OH)D levels byapproximately two fold compared to baseline levels. The VDS group, on the other
				hand, showed higher amounts of autophagy-related proteins as well as other proteins that are essential for anti- microbial autophagy in whole blood PBMCs.
Patil, 201916	India	Double- blind pilot randomized clinical trial	19 chronic periodontitis patients	Vitamin D3 deficiency was discovered in all chronic periodontitis patients. The mean serum 25(OH)D3 level before supplementation was 13.96 ng/mL, but it increased dramatically to 35.12 ng/mL following 6 weeks of Vitamin D3 treatment. The mean VDR conc. in PDL tissue was -1.443 ng/mL, which increased to 2.38 ng/mL following supplementation, according to VDR analyses. After supplementation, there was a concentration dependent association between serum 25(OH)D3 levels and VDR concentration in PDL tissue.
Gao, 202017	USA	Randomize d, double-masked, placebo-controlled clinical trial	360 patients with mode rate or severe period on it is	There was a slight but significant decrease in AL and PD in both vitamin D groups compared with placebo group for moderate and deep pockets. About 2000 IU/d vitamin D3 group, 1000 IU/d vitamin D3 group, and placebo group all decreased the AL for both moderate pockets (-0.4 mm vs -0.4 mm vs -0.3 mm) and deep pockets (-1.1 mm vs -1.1 mm vs -1.0 mm) (all P <0.05). Similarly, PD was also decreased in these three groups for both moderate pockets and deep pockets (all P <0.05). In addition vitamin D supplementation was well
tolerated, and no adverseevents were reported.				

Meghil, et al (2019) showed vitamin D supplementation (VDS) group had lower peripheral blood CD3 and CD3+CD8+ cytotoxic T lymphocyte (CTL) counts as well as lower levels of pro-inflammatory salivary cytokines than the control group. Vitamin D supplementation increased serum 25(OH)D levels by approximately twofold compared to baseline levels. The VDS group, on the other hand, showed higher amounts of autophagy-related proteins as well as other proteins that are essential for anti-microbial autophagy in whole blood PBMCs.[15]

Patil, et al (2019) conducted a study in India. They showed vitamin D3 deficiency was discovered in all chronic periodontitis patients. The mean serum 25(OH)D3 level before supplementation was 13.96 ng/mL, but it increased dramatically to 35.12 ng/mL following 6 weeks of Vitamin D3 treatment. The mean VDR conc. in PDL tissue was -1.443 ng/mL, which increased to 2.38 ng/mL following supplementation, according to VDR analyses. After supplementation, there was a concentration-dependent association between serum 25(OH)D3 levels and VDR concentration in PDL tissue.[16]

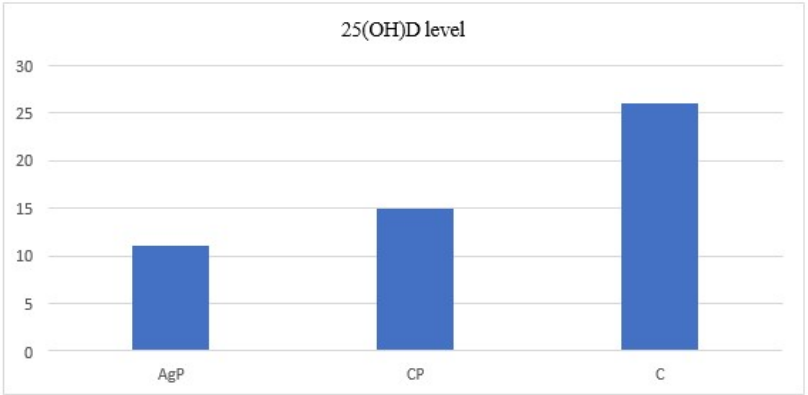

Figure 2. Comparison 25(OH)D level patients with AgP, CP, and control (C)

All chronic periodontitis patients were found to be vitamin D3 deficient in Gao, et al study (2020). Before supplementation, the average serum 25(OH)D3 level was 13.96ng/mL, but it jumped considerably to 35.12 ng/mL after 6 weeks of Vitamin D3 treatment.According to VDR studies, the mean VDR conc. in PDL tissue was -1.443 ng/mL, whichincreased to 2.38 ng/mL after supplementation. There was a concentration-dependent relationship between serum 25(OH)D3 levels and VDR concentration in PDL tissue aftersupplementation.[17]

DISCUSSION

Vitamin D is involved in a number of important processes, including bone metabolism and inflammation. There is some data that suggests vitamin D could influencethe onset and progression of periodontitis. [Citation needed] [Citation needed] An inverseconnection between periodontitis and vitamin D status, as evaluated by serum levels of 25(OH)D, has been described in the scientific literature. One case-control study found that patients with periodontitis had a considerably higher proportion of low 25(OH)D levels (i.e., <50 nmol/L) compared with healthy controls, with an adjusted odds ratio of 1.5. This was contrasted to the healthy controls who did not have periodontitis.[18]

Periodontitis is a complicated condition that is caused by several microorganismsand is characterized by persistent and ongoing inflammation. Periodontitis is one of the two most common diseases in the world, and the severe form of the condition is the sixthmost common. It has significant ramifications for both the socioeconomic system and thesystem as a whole. It has a significant bearing on one's quality of life and can be restoredwith periodontal treatment. The systemic connection that exists between periodontitis and other diseases and conditions, such as diabetes, ischemic stroke, cardiovascular disease (CVD), rheumatoid arthritis, inflammatory bowel disease, stress, individuals who have had solid-organ transplants, or preterm birth, has strengthened over the years.[19,20]

In addition, the impact of nutrition on periodontal health, and specifically VDD, has been intensively researched, and a recent European consensus stated that an inadequate vitamin D status impacts periodontal health and oral functions. VDD is the most important nutrient for maintaining periodontal health. Study showed an inverse association of serum 25(OH)D concentration and the risk of AgP. On the other hand, noassociation was observed between serum 25(OH)D and CP.[12]

There is some evidence to support the hypothesis that a lack of vitamin D contributes to the development of AgP. There is some speculation that the anti- inflammatory and antibacterial actions of vitamin D, as well as its effect on bone mineral density, may have a beneficial affect on periodontal health. Since periodontal diseases, including the more severe AgP, are infectious and are initiated in response to bacterial insult, it is reasonable to suspect that a vitamin D deficiency could negatively affect the immune response and, as a result, the periodontium. This is because periodontal diseasesare initiated in response to bacterial insult.[21]

Defensins and other naturally occurring antimicrobial peptides are activated in reaction to vitamin D, which in turn stimulates the body's innate immunological response.Specifically, A. actinomycetemcomitans, which is a periodontal infection linked with AgP, can be eradicated by beta defensin 2. As a result, having an insufficient amount of vitamin D may weaken the host's ability to fight off periodontal infections. In addition tothis, vitamin D not only boosts the body's innate immune response to a microbial threat, but it also dampens the body's overactive adaptive immunological response when it comesto fighting off viruses.[22,23]

An improper or overactive immune response to pathogenic microorganisms is hypothesized to be the cause of the illness process in autoimmune polyangiitis (AgP). Asa result, it is conceivable that a lack of vitamin D increases to the likelihood of developingAgP by lowering the host's ability to control an overactive immunological response. In recent years, a significant amount of focus has been placed on the potential significance of vitamin D in the development of chronic periodontal disease. Epidemiological researchhas shown a correlation between chronic periodontitis and serum vitamin D concentrations. In older adults (age >50 years), in postmenopausal women, and in pregnant women, an inverse correlation was observed to exist between serum vitamin Dlevels and indices of periodontal disease.[14,22]

In addition, a dose-dependent inverse connection between the serum vitamin D concentration and gingival inflammation that was evaluated by bleeding on provocation was found to exist. This association was shown to be negative. The study also took into account other factors that could have a direct influence on the status of vitamin D, such as the amounts of parathyroid hormone, calcium, or phosphate. Calcium and phosphate concentrations in the blood and interstitial fluids can be modulated thanks to a complicated feedback process that involves parathyroid hormone and vitamin D. This mechanism plays a role in the maintenance of mineral homeostasis in the body. The levelsof vitamin D in the bloodstream are inversely related to serum levels of parathyroid hormone. A lack of vitamin D can cause secondary hyperparathyroidism, which is a condition that occurs when the body's parathyroid glands produce too much parathyroid hormone.[14]

Other study showed link in a periodontitis patient cohort, between vitamin D supplementation and CD3+CD8+ cytotoxic T-lymphocytes (CTLs). CTLs have long been implicated in protective and destructive immune response s. Furthermore, preventing CTL -mediated damage is suggested in previous studies as a therapeutic approach for the treatment of some inflammatory conditions. In periodontal disease, elevation in CTL counts have been linked to the increased periodontal tissue damage. So,regulating the CTLs response during periodontal therapy via vitamin D supplementationwould be a therapeutic approach to counteract hyperresponsiveness of CTLs.[15]

CONCLUSION

According to the findings of several studies, people with low levels of vitamin Dhave a higher prevalence of periodontitis. In patients who had more chronic diseases, theassociation between vitamin D and those conditions was not significant.

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