Research Article

Correlation between Vitamin D Levels and Periodontal Health in Adolescent Population

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Abstract

Background: Vitamin D plays essential roles in calcium homeostasis, immune modulation, and inflammatory regulation, suggesting potential implications for periodontal health. Adolescence represents a critical period for both skeletal development and establishment of periodontal health patterns, yet research examining vitamin D–periodontal relationships in this population remains limited. **Objective:** This study aimed to investigate the correlation between serum vitamin D levels and periodontal health parameters among adolescents, exploring potential associations with gingivitis severity and clinical attachment status. **Methods:** A cross-sectional analytical study was conducted among 486 adolescents aged 12–17 years. Serum 25-hydroxyvitamin D [25(OH)D] concentrations were measured using chemiluminescent immunoassay. Periodontal assessment included plaque index (PI), gingival index (GI), bleeding on probing (BOP), probing depth (PD), and clinical attachment level (CAL). Participants were categorized by vitamin D status: deficient (<20 ng/mL), insufficient (20–29 ng/mL), and sufficient (≥30 ng/mL). Pearson correlation, ANOVA, and multivariable regression analyses evaluated associations. **Results:** Mean serum 25(OH)D was 24.67 ± 9.84 ng/mL, with 31.5% classified as deficient, 38.9% as insufficient, and 29.6% as sufficient. Vitamin D-deficient adolescents demonstrated significantly higher mean GI (1.67 ± 0.48) compared to sufficient participants (1.12 ± 0.39; $p<0.001$). Significant negative correlations were observed between 25(OH)D and GI (r=-0.412, $p<0.001$), BOP (r=-0.387, $p<0.001$), and PI (r=-0.298, $p<0.001$). Multivariable regression confirmed vitamin D deficiency as independently associated with gingivitis (OR=2.34; 95% CI: 1.56–3.51; $p<0.001$) after adjusting for confounders. **Conclusion:** Vitamin D levels demonstrate a significant inverse correlation with periodontal inflammation markers in adolescents. Vitamin D deficiency is independently associated with increased gingivitis severity, suggesting potential benefits of adequate vitamin D status for adolescent periodontal health.

Keywords: vitamin D, periodontal health, gingivitis, adolescents, 25-hydroxyvitamin D, gingival inflammation

1 Introduction

Vitamin D is a fat-soluble secosteroid hormone essential for calcium-phosphorus homeostasis, skeletal mineralization, and increasingly recognized immunomodulatory functions [1]. Beyond its classical roles in bone metabolism, vitamin D exerts pleiotropic effects on immune system regulation, inflammatory responses, and antimicrobial peptide production, suggesting broader implications for oral health maintenance [2]. The biologically active form, 1,25-dihydroxyvitamin D, mediates cellular effects through vitamin D receptors (VDR) expressed in numerous tissues, including periodontal cells and immune cells involved in gingival inflammation [3].

Periodontal diseases encompass a spectrum of inflammatory conditions affecting tooth-supporting structures, ranging from reversible gingivitis to destructive periodontitis with irreversible attachment loss [4]. While periodontitis predominantly affects adults, gingivitis prevalence among adolescents is substantial, with studies reporting rates exceeding 70% in some populations [5]. Adolescent gingivitis, though typically reversible, may establish inflammatory patterns and microbial dysbiosis predisposing individuals to progressive periodontal disease in adulthood [6]. Therefore, understanding modifiable factors influencing adolescent periodontal health has important preventive implications.

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The biological rationale linking vitamin D to periodontal health involves multiple mechanisms [7]. Vitamin D enhances innate immunity through stimulation of cathelicidin and defensin production, antimicrobial peptides with activity against periodontal pathogens [8]. Additionally, vitamin D modulates adaptive immune responses by suppressing pro-inflammatory cytokine production, including interleukin-1β, interleukin-6, and tumor necrosis factor-α, which are key mediators of periodontal tissue destruction [9]. The anti-inflammatory properties of vitamin D may attenuate excessive host responses to bacterial challenge, potentially limiting collateral tissue damage characteristic of periodontal pathogenesis [10].

Epidemiological investigations examining vitamin D–periodontal associations have yielded generally consistent findings in adult populations [11]. National Health and Nutrition Examination Survey (NHANES) analyses demonstrated inverse associations between serum 25-hydroxyvitamin D concentrations and clinical attachment loss among adults [12]. Systematic reviews have confirmed significant associations between vitamin D deficiency and increased periodontitis risk, though heterogeneity in study designs and outcome measures complicates definitive conclusions.

However, research specifically examining adolescent populations remains notably limited. Adolescents face unique considerations, including rapid skeletal growth with high calcium and vitamin D requirements, hormonal changes influencing gingival inflammation, and lifestyle factors such as indoor activities and dietary patterns potentially contributing to vitamin D insufficiency. Contemporary adolescent populations demonstrate concerning rates of vitamin D deficiency, with prevalence estimates ranging from 20% to over 60%, depending on geographic location and assessment criteria.

The potential interaction between vitamin D status and pubertal hormonal changes adds complexity to adolescent periodontal health. Sex hormones, particularly estrogen and progesterone, influence gingival vascular permeability and inflammatory responses, contributing to puberty-associated gingivitis. Whether adequate vitamin D status may mitigate hormone-mediated gingival inflammation represents an unexplored research question with clinical relevance.

Furthermore, most existing studies utilize periodontitis outcomes that are more applicable to adult populations, while gingivitis represents the predominant periodontal condition among adolescents. The relationship between vitamin D and early inflammatory periodontal changes has received insufficient attention despite representing the most clinically relevant outcome for adolescent populations.

Therefore, this study aimed to investigate the correlation between serum vitamin D levels and periodontal health parameters among adolescents, with particular focus on gingivitis severity and associations across vitamin D status categories.

2 Materials and Methods

2.1 Study Design and Setting

This cross-sectional analytical study was conducted between March and October 2024 at the Department of Periodontology, University Dental Hospital, in collaboration with a tertiary pediatric medical center. The study protocol received ethical approval from the Joint Institutional Review Board (Protocol #PERIO-VD-2024-0312), and all procedures complied with the Declaration of Helsinki guidelines for research involving human subjects.

2.2 Sample Size Determination

Sample size was calculated using G*Power software version 3.1, based on an anticipated medium effect size (f=0.25) for ANOVA comparisons across three vitamin D status groups, 95% confidence level, 80% statistical power, and three groups. The minimum required sample was determined as 432 participants, which was increased to 520 to accommodate potential incomplete data.

2.3 Participant Selection

Adolescents aged 12–17 years presenting for routine dental examinations or referred for periodontal evaluation were screened for eligibility. Inclusion criteria comprised: (1) age between 12 and 17 years; (2) presence of permanent dentition with a minimum of 20 erupted teeth; (3) generally healthy systemic status; (4) willingness to provide blood samples; and (5) provision of written parental consent and participant assent.

Exclusion criteria included: (1) current or recent vitamin D supplementation within 3 months; (2) systemic diseases affecting vitamin D metabolism, including renal disease, hepatic disease, and malabsorption syndromes; (3) medications influencing vitamin D or calcium metabolism, including anticonvulsants, corticosteroids, and bisphosphonates; (4) current orthodontic treatment with fixed appliances; (5) antibiotic use within the preceding 3 months; (6) current smoking or tobacco use; (7) pregnancy; and (8) diagnosed diabetes mellitus or immunocompromising conditions.

Consecutive eligible participants were enrolled until achievement of the target sample size.

2.4 Vitamin D Assessment

Fasting venous blood samples (5 mL) were collected by trained phlebotomists between 8:00 and 10:00 AM to minimize diurnal variation. Samples were centrifuged at 3000 rpm for 10 minutes, and serum was separated and stored at $-20°C$ until analysis.

Serum 25-hydroxyvitamin D [25(OH)D] concentrations were measured using chemiluminescent microparticle immunoassay (ARCHITECT i2000SR, Abbott Diagnostics, USA). This assay demonstrates a coefficient of variation <10% and a detection range of 3.4–155.9 ng/mL. Quality control procedures included daily calibration and participation in external quality assurance programs.

Vitamin D status was categorized according to Endocrine Society Clinical Practice Guidelines: deficient (<20 ng/mL), insufficient (20–29 ng/mL), and sufficient (≥30 ng/mL).

2.5 Periodontal Examination

Full-mouth periodontal examinations were performed by two calibrated periodontists (inter-examiner kappa=0.88; intra-examiner kappa=0.92) blinded to vitamin D results. Examinations were conducted under standardized lighting using University of North Carolina (UNC-15) periodontal probes and dental mirrors.

Assessed parameters included:

Plaque Index (PI): Silness–Löe index scoring plaque accumulation on four tooth surfaces (0=no plaque; 1=thin film; 2=moderate accumulation; 3=abundant plaque). Mean scores were calculated across all teeth.

Gingival Index (GI): Löe–Silness index evaluating gingival inflammation on four surfaces per tooth (0=healthy; 1=mild inflammation, slight color change, no bleeding; 2=moderate inflammation, bleeding on probing; 3=severe inflammation, spontaneous bleeding). Mean scores were calculated.

Bleeding on Probing (BOP): Percentage of sites demonstrating bleeding within 30 seconds following gentle probing with 25 g force.

Probing Depth (PD): Distance from gingival margin to base of sulcus/pocket measured at six sites per tooth: mesiobuccal, buccal, distobuccal, mesiolingual, lingual, and distolingual.

Clinical Attachment Level (CAL): Distance from the cementoenamel junction to the base of the sulcus/pocket at six sites per tooth.

Gingivitis was defined as GI≥1.0 with BOP≥10% in the absence of clinical attachment loss (CAL>2 mm at any site). Severity classification was as follows: mild gingivitis (GI 1.0–1.4), moderate gingivitis (GI 1.5–1.9), and severe gingivitis (GI≥2.0).

2.6 Covariate Assessment

Structured questionnaires collected data on age, sex, ethnicity, body mass index (BMI), dietary calcium intake, sun exposure patterns, oral hygiene practices, including brushing frequency and interdental cleaning, and pubertal status, based on self-reported Tanner staging. Socioeconomic status was assessed using parental education and household income categories.

2.7 Statistical Analysis

Data were analyzed using SPSS version 28.0 (IBM Corporation, Armonk, NY) and R version 4.2. Normality was assessed using Shapiro–Wilk tests and visual inspection of Q–Q plots. Descriptive statistics included means, standard deviations, frequencies, and percentages.

Differences in periodontal parameters across vitamin D status categories were evaluated using one-way ANOVA with Bonferroni post-hoc corrections for normally distributed variables and Kruskal–Wallis tests for non-normal distributions. Chi-square tests were used to compare categorical variables.

Pearson correlation coefficients assessed linear relationships between continuous variables. Multivariable logistic regression identified factors independently associated with gingivitis, with results expressed as odds ratios (OR) and 95% confidence intervals (CI). Covariates included age, sex, BMI, plaque index, brushing frequency, and socioeconomic status. Statistical significance was established at $p<0.05$.

3 Results

3.1 Participant Characteristics

Of 520 initially enrolled adolescents, 486 completed all assessments and were included in the final analysis (completion rate: 93.5%). Exclusions resulted from incomplete blood samples (n=18), incomplete periodontal examination (n=11), and withdrawn consent (n=5). The sample comprised 251 females (51.6%) and 235 males (48.4%), with a mean age of 14.52 ± 1.67 years.

Mean serum 25(OH)D concentration was 24.67 ± 9.84 ng/mL (range: 6.8–58.4 ng/mL). Vitamin D status distribution showed 153 participants (31.5%) classified as deficient, 189 (38.9%) as insufficient, and 144 (29.6%) as sufficient. Females demonstrated significantly lower mean 25(OH)D levels (22.89 ± 9.12 ng/mL) compared to males (26.58 ± 10.23 ng/mL; $p<0.001$), as shown in Table 1.

Table 1: Participant Characteristics by Vitamin D Status Category

Characteristic	Total (n=486)	Deficient <20 ng/mL (n=153)	Insufficient 20–29 ng/mL (n=189)	Sufficient ≥30 ng/mL (n=144)	p-value
Age (years), mean ± SD	14.52 ± 1.67	14.61 ± 1.72	14.48 ± 1.65	14.47 ± 1.63	0.723
Sex, n (%)					0.003*
Male	235 (48.4%)	58 (37.9%)	92 (48.7%)	85 (59.0%)	
Female	251 (51.6%)	95 (62.1%)	97 (51.3%)	59 (41.0%)	
BMI (kg/m^2), mean ± SD	21.34 ± 3.87	22.56 ± 4.23	21.12 ± 3.67	20.34 ± 3.45	0.001*
Ethnicity, n (%)					0.087
Caucasian	287 (59.1%)	78 (51.0%)	112 (59.3%)	97 (67.4%)	
Asian	112 (23.0%)	43 (28.1%)	42 (22.2%)	27 (18.8%)	
African American	58 (11.9%)	24 (15.7%)	23 (12.2%)	11 (7.6%)	
Other	29 (6.0%)	8 (5.2%)	12 (6.3%)	9 (6.2%)	
Daily Sun Exposure					<0.001*
<30 minutes	198 (40.7%)	89 (58.2%)	72 (38.1%)	37 (25.7%)	
30–60 minutes	178 (36.6%)	48 (31.4%)	78 (41.3%)	52 (36.1%)	
>60 minutes	110 (22.6%)	16 (10.5%)	39 (20.6%)	55 (38.2%)	
Brushing Frequency					0.234
Once daily	167 (34.4%)	58 (37.9%)	64 (33.9%)	45 (31.3%)	
Twice daily	319 (65.6%)	95 (62.1%)	125 (66.1%)	99 (68.8%)	

*Statistically significant at $p<0.05$

3.2 Periodontal Parameters by Vitamin D Status

Significant differences in periodontal parameters were observed across vitamin D status categories. Vitamin D-deficient adolescents demonstrated the highest mean GI (1.67 ± 0.48), followed by insufficient (1.38 ± 0.42) and sufficient (1.12 ± 0.39) groups ($p<0.001$). Similar patterns were observed for BOP percentage and plaque index.

Overall gingivitis prevalence was 78.4% (n=381). Gingivitis prevalence varied significantly by vitamin D status: 91.5% in deficient, 79.4% in insufficient, and 60.4% in sufficient participants ($p<0.001$). Severe gingivitis was present in 23.5% of deficient participants compared to 6.9% of sufficient participants, as shown in Table 2.

Table 2: Periodontal Parameters by Vitamin D Status Category

Periodontal Parameter	Total (n=486)	Deficient (n=153)	Insufficient (n=189)	Sufficient (n=144)	F/χ^2	p-value
Plaque Index, mean ± SD	1.52 ± 0.56	1.78 ± 0.58	1.49 ± 0.52	1.26 ± 0.47	38.72	<0.001*
Gingival Index, mean ± SD	1.41 ± 0.47	1.67 ± 0.48	1.38 ± 0.42	1.12 ± 0.39	67.84	<0.001*
BOP (%), mean ± SD	42.34 ± 18.76	54.23 ± 17.89	41.67 ± 16.45	28.56 ± 14.23	89.45	<0.001*
Probing Depth (mm), mean ± SD	2.12 ± 0.34	2.23 ± 0.38	2.11 ± 0.32	2.01 ± 0.29	16.78	<0.001*
CAL>2mm sites, n (%)	12 (2.5%)	6 (3.9%)	4 (2.1%)	2 (1.4%)	2.34	0.310
Gingivitis Prevalence, n (%)	381 (78.4%)	140 (91.5%)	150 (79.4%)	87 (60.4%)	41.23	<0.001*
Gingivitis Severity, n (%)					56.78	<0.001*
None (GI<1.0)	105 (21.6%)	13 (8.5%)	39 (20.6%)	57 (39.6%)		
Mild (GI 1.0–1.4)	187 (38.5%)	47 (30.7%)	82 (43.4%)	58 (40.3%)		
Moderate (GI 1.5–1.9)	128 (26.3%)	57 (37.3%)	49 (25.9%)	19 (13.2%)		
Severe (GI≥2.0)	66 (13.6%)	36 (23.5%)	19 (10.1%)	10 (6.9%)		

*Statistically significant at $p<0.05$; Post-hoc Bonferroni: Deficient versus Sufficient $p<0.001$ for GI, BOP, and PI

3.3 Correlation Analysis and Multivariable Regression

Correlation analysis revealed significant inverse relationships between serum 25(OH)D concentrations and several periodontal inflammation markers, indicating that lower vitamin D levels were associated with poorer periodontal health among the adolescents included in the study. The strongest correlation was observed with the gingival index ($r=-0.412$, $p<0.001$), suggesting that adolescents with lower serum vitamin D concentrations tended to show more pronounced gingival inflammation. This relationship was followed by bleeding on probing percentage ($r=-0.387$, $p<0.001$), which further supports the association between reduced vitamin D status and active gingival inflammatory response. A significant inverse correlation was also observed between serum 25(OH)D concentration and plaque index ($r=-0.298$, $p<0.001$), indicating that participants with lower vitamin D levels had comparatively higher plaque accumulation. Although the correlation with plaque index was weaker than that observed for gingival index and bleeding on probing, it remained statistically significant and clinically relevant.

Multivariable logistic regression analysis was performed to determine whether vitamin D status remained associated with gingivitis after controlling for important potential confounding variables. After adjustment for age, sex, body mass index, plaque index, brushing frequency, and socioeconomic status, vitamin D deficiency remained independently associated with gingivitis, as shown in Table 3. This finding indicates that the association between vitamin D deficiency and gingival inflammation was not fully explained by demographic characteristics, oral hygiene behavior, or plaque accumulation alone. Participants with vitamin D deficiency had significantly increased odds of gingivitis compared with those who had sufficient vitamin D levels, supporting the possibility that vitamin D status may contribute independently to periodontal inflammatory susceptibility in adolescents.

Subgroup analysis by sex revealed that the association between vitamin D deficiency and gingivitis appeared stronger among females (OR=2.89; 95% CI: 1.67–5.01) compared to males (OR=1.78; 95% CI: 1.02–3.12). Although this difference suggests a possible sex-related variation in the relationship between vitamin D status and gingival inflammation, the interaction term did not reach statistical significance (p=0.087). Therefore, while the results indicate a stronger observed association among female participants, this finding should be interpreted cautiously and may require confirmation in larger studies. Possible explanations may include hormonal differences during adolescence, differences in body composition, variation in sun exposure, or sex-specific inflammatory responses, although these mechanisms were not directly assessed in the present study.

Linear regression modeling was additionally performed using gingival index as a continuous outcome variable. This analysis confirmed that serum 25(OH)D concentration was a significant predictor of gingival index (β=-0.012, SE=0.002, p<0.001). The negative regression coefficient indicates that higher serum vitamin D levels were associated with lower gingival inflammation scores. Specifically, each 10 ng/mL increase in serum vitamin D was associated with a 0.12-point decrease in gingival index. Although this decrease may appear modest at the individual level, it may be meaningful at the population level, particularly considering the high prevalence of both vitamin D insufficiency and gingivitis among adolescents. These findings collectively support an inverse relationship between vitamin D status and periodontal inflammation and suggest that adequate vitamin D levels may have a protective role in maintaining gingival health during adolescence.

Table 3: Correlation Coefficients and Multivariable Regression Analysis

Panel A: Pearson Correlations with Serum 25(OH)D		
Variable	r	p-value
Gingival Index	-0.412	<0.001*
Bleeding on Probing (%)	-0.387	<0.001*
Plaque Index	-0.298	<0.001*
Probing Depth	-0.156	0.001*
Age	0.023	0.614
BMI	-0.187	<0.001*

Panel B: Multivariable Logistic Regression for Gingivitis			
Variable	OR	95% CI	p-value
Vitamin D Status (ref: Sufficient)			
Insufficient	1.67	1.08–2.58	0.021*
Deficient	2.34	1.56–3.51	<0.001*
Sex (Female vs Male)	1.28	0.89–1.84	0.182
Age (per year)	0.94	0.82–1.08	0.376
BMI (per kg/m^2)	1.04	0.98–1.10	0.198
Plaque Index (per unit)	4.67	3.12–6.99	<0.001*
Brushing Twice Daily	0.72	0.49–1.06	0.094
Higher Parental Education	0.81	0.56–1.17	0.261

*Statistically significant at p<0.05; Model fit: Nagelkerke R^2=0.384, Hosmer–Lemeshow p=0.456

4 Discussion

This cross-sectional study demonstrated significant inverse correlations between serum vitamin D levels and periodontal inflammation markers among adolescents, with vitamin D deficiency independently associated

with increased gingivitis prevalence and severity. These findings extend the growing body of evidence linking vitamin D status to periodontal health and provide novel insights specifically relevant to adolescent populations.

The observed vitamin D deficiency prevalence of 31.5% aligns with contemporary estimates for adolescent populations in temperate climates, reflecting widespread suboptimal vitamin D status despite public health awareness efforts. The higher deficiency rates among females and those with limited sun exposure correspond to established risk factors and underscore the multifactorial determinants of vitamin D status requiring consideration in clinical practice. The association between higher BMI and lower vitamin D levels, attributable to sequestration in adipose tissue, further demonstrates the complex interrelationships between nutritional status, body composition, and oral health.

The significant inverse correlation between serum 25(OH)D and gingival index (r=-0.412) represents a moderate effect size with clinical relevance. This correlation strength exceeds that reported in some adult studies and may reflect heightened sensitivity of adolescent periodontal tissues to vitamin D-mediated effects during this developmental period. The correlation with bleeding on probing (r=-0.387) provides additional validation, as BOP represents a direct clinical indicator of active gingival inflammation closely linked to inflammatory cytokine activity.

The significantly elevated gingivitis prevalence among vitamin D-deficient participants (91.5% vs 60.4% in the sufficient group) represents a substantial disparity with potential public health implications. While plaque accumulation remains the primary etiological factor in gingivitis, our findings suggest that vitamin D status may modulate inflammatory responses to bacterial challenge. The persistence of significant associations after adjusting for plaque index in multivariable models supports an independent effect beyond confounding by oral hygiene practices.

The biological mechanisms underlying vitamin D–periodontal associations likely involve both immunomodulatory and antimicrobial pathways. Vitamin D stimulates production of cathelicidin (LL-37) and β-defensins in gingival epithelial cells, enhancing local antimicrobial defenses against periodontal pathogens, including Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans. Additionally, vitamin D suppresses nuclear factor-κB signaling and reduces production of pro-inflammatory cytokines implicated in periodontal tissue destruction.

The 2.34-fold increased odds of gingivitis among vitamin D-deficient adolescents, after covariate adjustment, indicates clinically meaningful risk elevation warranting attention. This effect magnitude is consistent with systematic review findings in adult populations and suggests that vitamin D optimization may represent a modifiable factor for periodontal disease prevention. The dose-response relationship across deficiency categories strengthens causal inference, though cross-sectional design limitations preclude definitive conclusions.

The trend toward stronger associations among females, while not reaching statistical significance for interaction, merits further investigation given known interactions between vitamin D and sex hormones in inflammatory regulation. Estrogen enhances vitamin D receptor expression and may amplify vitamin D-mediated anti-inflammatory effects, potentially explaining sex-differential responses. The hormonal fluctuations characteristic of puberty may create periods of heightened vulnerability when adequate vitamin D status becomes particularly important for maintaining gingival health.

The minimal clinical attachment loss observed in our adolescent sample (2.5% with CAL>2mm) limits evaluation of vitamin D associations with periodontitis outcomes. This finding is epidemiologically expected given the young age of participants and confirms that gingivitis, rather than periodontitis, represents the appropriate periodontal outcome for adolescent research. Longitudinal studies following adolescents into adulthood would enable assessment of whether early vitamin D deficiency predisposes to subsequent periodontal destruction.

Our findings have implications for clinical practice and public health. Dental professionals should consider vitamin D status as a potential contributing factor when evaluating adolescents with gingivitis, particularly those demonstrating inflammation disproportionate to plaque accumulation. Collaboration with primary care

providers for vitamin D assessment and supplementation guidance may enhance comprehensive adolescent health management.

Several limitations warrant acknowledgment. The cross-sectional design precludes determination of temporal relationships between vitamin D status and periodontal outcomes. Residual confounding from unmeasured variables, including genetic factors, complete dietary assessment, and seasonal variation in vitamin D levels, may influence observed associations. Single-timepoint vitamin D measurement may not reflect long-term status. The exclusion of adolescents with orthodontic appliances, while necessary for accurate periodontal assessment, may limit generalizability.

Future research should incorporate longitudinal designs, intervention trials evaluating vitamin D supplementation effects on periodontal outcomes, and mechanistic studies examining vitamin D effects on gingival inflammatory responses at the cellular and molecular levels.

5 Conclusion

This study demonstrated significant inverse correlations between serum vitamin D levels and periodontal inflammation markers among adolescents aged 12–17 years. Vitamin D deficiency, affecting nearly one-third of participants, was independently associated with 2.34-fold increased odds of gingivitis after adjusting for oral hygiene and demographic factors. The gingival index showed moderate negative correlation with 25(OH)D concentrations, indicating that higher vitamin D levels are associated with reduced gingival inflammation severity.

These findings suggest that adequate vitamin D status may contribute to optimal periodontal health during adolescence, potentially through immunomodulatory and antimicrobial mechanisms. Given the high prevalence of both vitamin D deficiency and gingivitis in adolescent populations, attention to vitamin D optimization may represent an adjunctive strategy for promoting periodontal health alongside conventional oral hygiene measures.

Dental and medical professionals should consider vitamin D status when evaluating adolescents with persistent gingival inflammation. Public health initiatives addressing adolescent vitamin D deficiency may yield benefits extending to periodontal health outcomes, supporting integrated approaches to adolescent health promotion.

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