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Association between Vitamin D Intake and Tooth Hypomineralization in Children: A Systematic Review

Anggun Rafisa[1], Felisha Febriane Balafif[2], Nuroh Najmi[3], Faisal Kuswandani[4]

[1,2,3,4]Department of Oral Biology, Faculty of Dentistry, Universitas Padjadjaran, Indonesia

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Corresponding Author:

Anggun Rafisa

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ABSTRACT

Background: Vitamin D plays an important role in tooth mineralization; however, evidence regarding its association with enamel hypomineralization, particularly molar–incisor hypomineralization (MIH), remains inconsistent.

Objective: This systematic review aimed to evaluate the association between vitamin D exposure and enamel hypomineralization in children and to explore potential modifying factors contributing to conflicting findings.

Methods: A systematic search of PubMed and Scopus was conducted for studies published between 2015 and 2025. Eligible studies included observational or interventional research involving children, assessment of vitamin D exposure (dietary intake, supplementation, or serum 25-hydroxyvitamin D), and outcomes related to enamel hypomineralization. Study selection followed PRISMA guidelines.

Results: Six studies met the inclusion criteria. Findings were heterogeneous, with some studies reporting a protective association between higher vitamin D levels and reduced hypomineralization, while others found no significant relationship.

Conclusion: Current evidence does not consistently support a direct association between vitamin D status and enamel hypomineralization in children. Tooth hypomineralization is likely multifactorial, and future longitudinal studies with standardized assessments are needed to better define the role of vitamin D within this complex process.

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1. INTRODUCTION

Tooth hypomineralization is a developmental dental disorder characterized by defects in enamel mineralization, resulting in porous, delicate enamel with changed translucency and heightened vulnerability to post-eruptive decay.[1] Molar-incisor hypomineralization (MIH) is one of these disorders that has drawn the most attention because of its high frequency and substantial effects on oral health, such as dental hypersensitivity, a greater risk of cavities, difficulties with restorations, and a lower quality of life for children who are affected.[2-4]

The development of enamel is a highly regulated biological process that can be disrupted by systemic factors acting during prenatal, perinatal, or early postnatal life.[5] Current evidence suggests that tooth hypomineralization has a multifactorial origin, involving a complex interaction between genetic susceptibility and environmental exposures.[6] Systemic illnesses, medication use, and nutritional status during critical periods of amelogenesis have all been proposed as potential contributing factors.[6-8] However, the relative contribution of specific nutritional components remains incompletely understood.

Vitamin D is an essential nutrient involved in calcium and phosphate metabolism and plays a fundamental role in skeletal and dental mineralization.[9] Vitamin D has been shown to influence the activity of ameloblasts and the regulation of mineral deposition in developing enamel.[10] Inadequate vitamin D intake or deficiency during pregnancy and early childhood may therefore interfere with normal enamel formation, potentially predisposing children to hypomineralized enamel defects.[11-13]
In recent years, several epidemiological studies have examined the relationship between vitamin D exposure, either through dietary intake, supplementation, or serum levels, and the occurrence of tooth hypomineralization in children.[14] While some studies report an increased risk of MIH or other enamel defects associated with low vitamin D status,[15-17] others have found no associations.[18] Differences in study design, timing of vitamin D assessment, outcome definitions, and control of confounding factors may partly explain these inconsistencies.

A comprehensive evaluation of existing studies is needed to clarify the strength and consistency of this association and to identify gaps in current knowledge. Therefore, the objective of this systematic review is to critically assess and summarize the available evidence on the relationship between vitamin D intake or status, during prenatal and/or early childhood periods, and the development of tooth hypomineralization, including molar–incisor hypomineralization, in children.

II. METHOD

Study Design and Reporting Guideline

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. The review protocol was developed a priori to define the research question, eligibility criteria, search strategy, and methods for study selection and data synthesis.

Eligibility Criteria

Studies published between 2015 and 2025 were included if they were original research articles with observational (cohort, case–control, or cross-sectional) or interventional study designs, involved children as the study population, assessed vitamin D exposure including dietary intake, supplementation, or serum vitamin D levels (e.g., 25-hydroxyvitamin D), reported outcomes related to tooth hypomineralization such as MIH or other developmental enamel defects, were published in peer-reviewed journals, and were available in full-text format.

Studies were excluded if they were case reports, case series, reviews, editorials, letters, conference abstracts, or expert opinions; involved animal or in vitro experiments; or were published in languages other than English.

Information Sources and Search Strategy

A comprehensive literature search was performed in electronic databases including PubMed and Scopus from inception until the date of the final search. The search strategy combined controlled vocabulary (MeSH terms) and free-text keywords related to vitamin D and tooth hypomineralization. Boolean operators (AND, OR) were used to optimize sensitivity. Reference lists of included studies were manually screened to identify additional relevant articles.

A representative search strategy was as follows:
“vitamin D” OR “vitamin D intake” OR “vitamin D deficiency” OR “25-hydroxyvitamin D”) AND (hypomineralization OR “enamel hypomineralization” OR “molar incisor hypomineralization” OR MIH OR “developmental defects of enamel”

Study Selection

All records identified through the database searches were imported into a reference management software, and duplicates were removed. Two reviewers independently screened titles and abstracts to identify potentially eligible studies. Full-text articles were then assessed for inclusion based on the predefined eligibility criteria. Any disagreements between reviewers were resolved through discussion or consultation with a third reviewer.

Data Extraction

Data extracted from each included study comprised the author and year of publication, study design, characteristics of the study population, and main findings.

III. RESULTS

As shown in Figure 1, the database search identified a total of 119 records, comprising 51 records from PubMed and 68 records from Scopus. After the removal of 39 duplicate records, 80 records remained for title and abstract screening. During the screening process, 39 records were excluded because they did not meet the eligibility criteria. Forty-one full-text articles were assessed for eligibility and 35 articles were excluded due to reasons such as irrelevance to the research question, insufficient assessment of vitamin D exposure, or lack of appropriate outcome measures related to tooth hypomineralization. Finally, 6 studies met all inclusion criteria and were included in the final qualitative synthesis.

Based on Table 1, six studies examining the relationship between vitamin D, diet, and enamel or tooth mineralization outcomes in children were included, encompassing cross-sectional, cohort, and case–control designs with sample sizes ranging from 101 to 1,241 children aged approximately 4–18 years. The findings were heterogeneous. Two studies reported a potential protective role of vitamin D, showing that higher serum 25(OH)D levels or early-life vitamin D supplementation were associated with reduced odds of molar–incisor hypomineralization (MIH) and fewer caries-related restorations, particularly in the primary dentition. In contrast, three studies found no statistically significant associations between serum or maternal vitamin D status and the prevalence or severity of MIH or dental caries after adjustment for confounders, despite descriptive trends suggesting higher defect prevalence among children with lower vitamin D levels in some analyses. One study focusing on broader dietary factors demonstrated that enamel hypoplasia was significantly associated with high sugar consumption and vitamin D deficiency, while adequate calcium intake appeared protective.

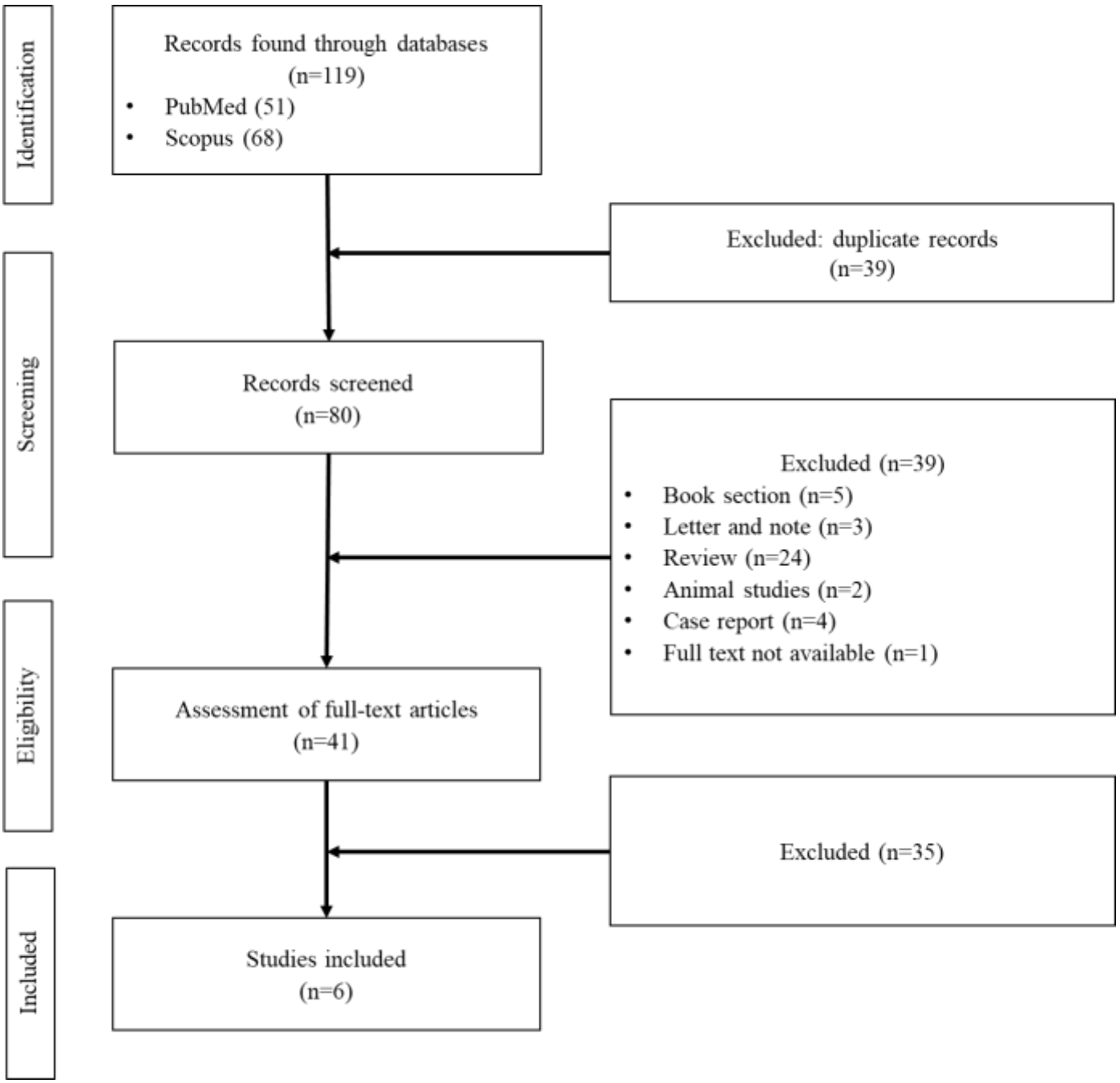

Figure 1. PRISMA flow diagram of literature search and study selection

Table 1: Overview of the Included Studies

No.	Author	Title	Study Design	Population Characteristics	Main Findings
1	Kühnisch et al. (2015)[19]	Elevated serum 25(OH)-vitamin D levels are negatively correlated with molar-incisor hypomineralization	Cross-sectional study	1,048 children aged approximately 10 years from the German GINIplus and LISAplus birth cohorts; serum 25(OH)D levels measured at age 10; MIH assessed according to EAPD criteria; caries recorded using surface-based DMF indices	Higher serum 25(OH)D concentrations were significantly associated with a lower likelihood of MIH. Each 10 nmol/L increase in 25(OH)D was associated with reduced odds of MIH after adjustment for demographic and socioeconomic confounders. Elevated vitamin D levels were also associated with fewer caries-affected permanent teeth, suggesting a protective relationship between vitamin D status and dental health.
2	Kühnisch et al. (2016)[17]	Fluoride/vitamin D tablet supplementation in infants—effects on dental health after 10 years	Cohort study	406 children examined at 10 years of age with data on fluoride and/or vitamin D supplementation during the first year of life recorded monthly through parental questionnaires	Continuous fluoride and vitamin D supplementation throughout the first year of life was associated with a significantly lower risk of caries-related restorations in the primary dentition. No significant association was found between supplementation and caries in permanent teeth or the occurrence of MIH.
3	Doğusal et al., 2021[20]	Evaluation of Serum 25(OH)D Levels in Obese and Normal-Weight Children with Carious and Hypomineralized Teeth	Case–control study	455 children aged 6–18 years attending pediatric endocrinology and pediatric dentistry clinics; included obese and age- and sex-matched normal-weight children; all had fully erupted first permanent molars and incisors	No significant association was found between serum 25(OH)D levels and dental caries or MIH in either age group. Obese children were not at increased risk for caries or MIH compared with normal-weight children. Serum vitamin D status did not significantly influence caries indices or MIH prevalence.
4	Mortensen et al., 2022[18]	Vitamin D status and tooth enamel hypomineralization are not associated in 4-y-old children: An Odense Child Cohort study	Cohort study	1,241 children aged approximately 4 years from the Odense Child Cohort with available data on maternal serum 25(OH)D in early and late pregnancy and cord blood; generally healthy mothers and children	Maternal and cord blood serum 25(OH)D concentrations were not associated with hypomineralization of second primary molars in 4-year-old children, regardless of vitamin D categorization or lesion severity. Shorter gestational age and higher maternal education were identified as independent risk factors for hypomineralization of second primary molars.
5	Børsting et al., 2024[15]	The association between serum vitamin D status and dental caries or molar incisor hypomineralisation in 7–	Cross-sectional study	101 Norwegian children aged 7–9 years with available serum 25(OH)D measurements assessed	Although children with insufficient serum vitamin D levels (<50 nmol/L) showed a higher prevalence and number of teeth affected by dental caries

		9-year-old Norwegian children: a cross-sectional study		by LC-MS/MS and standardized oral examinations	and MIH in descriptive analyses, no statistically significant associations were observed between vitamin D status and either caries or MIH prevalence or severity after adjustment for confounders.
6	Meena et al., 2024[16]	Assessing the Impact of Diet on Enamel Hypoplasia in Children	Cross-sectional study	200 children aged 6–12 years from urban and rural schools; dietary intake assessed via questionnaires; enamel hypoplasia evaluated using Modified DDE Index	Enamel hypoplasia was present in 36% of children. High sugar consumption was significantly associated with increased enamel hypoplasia (OR = 2.8; P < 0.001). Adequate calcium intake showed a protective effect, while vitamin D deficiency was significantly correlated with enamel hypoplasia (P < 0.01).

IV. DISCUSSION

Vitamin D plays a fundamental role in enamel formation through its regulation of calcium and phosphate homeostasis, which are essential for proper mineral deposition during amelogenesis.[21] The active form of vitamin D, 1,25-dihydroxyvitamin D, influences mineral metabolism by enhancing intestinal absorption of calcium and phosphate and by acting on vitamin D receptors expressed in ameloblasts and odontoblasts.[22, 23] Disruptions in vitamin D availability during critical periods of tooth development, particularly prenatal life and early childhood, may therefore interfere with enamel matrix maturation and mineralization, potentially resulting in hypomineralized enamel.[24] This biological plausibility supports the hypothesis that inadequate vitamin D status could contribute to developmental enamel defects such as MIH and other forms of enamel hypomineralization.

Despite this mechanistic rationale, findings across epidemiological studies remain inconsistent. Some investigations have reported inverse associations between serum 25-hydroxyvitamin D concentrations and MIH prevalence, suggesting a protective effect of higher vitamin D levels,[16, 17, 19] while others have failed to demonstrate significant associations.[15, 18, 20] These discrepancies may partly be explained by differences in the timing of vitamin D exposure assessment. Enamel development spans prenatal and early postnatal periods,[25] yet many studies measure vitamin D status at a single time point later in childhood, which may not accurately reflect exposure during critical windows of amelogenesis. Consequently, misclassification of relevant vitamin D exposure may attenuate observed associations.

Methodological heterogeneity across studies also contributes to conflicting results. Variations in study design, sample size, age of participants, and diagnostic criteria for MIH or enamel hypomineralization can influence outcome estimates. In addition, different laboratory methods for measuring serum 25(OH)D, including immunoassays versus liquid chromatography–tandem mass spectrometry, may yield non-comparable vitamin D concentrations.[26] Residual confounding further complicates interpretation, as factors such as gestational age, childhood illnesses, socioeconomic status, dietary calcium intake, and fluoride exposure are variably controlled across studies and may independently affect enamel mineralization.[27]

Another potential explanation for inconsistent findings is that vitamin D may not act in isolation but rather as part of a complex nutritional and environmental network influencing enamel development. Adequate calcium intake appears to be a critical modifier of vitamin D action, as vitamin D–mediated mineral absorption is ineffective in the presence of insufficient dietary calcium.[28] Furthermore, high sugar consumption, systemic inflammation, and early-life stressors may exacerbate enamel vulnerability regardless of vitamin D status.[29, 30] This interaction-based perspective may explain why some studies observe associations only in specific subgroups or populations with concurrent nutritional deficiencies or adverse environmental exposures.

Future research should prioritize well-designed longitudinal studies with repeated assessments of vitamin D status during key developmental periods, including pregnancy, infancy, and early childhood. Standardized diagnostic criteria for MIH and enamel hypomineralization, along with uniform laboratory methods for vitamin D measurement, are essential to improve comparability across studies. Additionally, future investigations should adopt a multifactorial approach by simultaneously evaluating vitamin D, calcium intake, dietary patterns, and perinatal factors. Such comprehensive models are likely to provide a clearer understanding of the role of vitamin D within the broader etiological framework of enamel hypomineralization and inform effective preventive strategies.

V. CONCLUSION

Current evidence does not consistently support a direct association between vitamin D status and enamel hypomineralization in children. Although vitamin D is biologically important for enamel mineralization, its effect appears to be influenced by timing of exposure, study methodology, and interacting factors such as calcium intake and overall diet. Enamel hypomineralization is likely multifactorial, and future longitudinal studies with standardized assessments are needed to better define the role of vitamin D within this complex process.

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VII. DISCLOSURE

The author reports no conflicts of interest in this work.

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