



# Maternal Vitamin D levels during Pregnancy and its Effects on Enamel Dental Defects of their Children. A Literature Review.

Athanasia P. Champilomati<sup>I</sup>, Panagiotis I. Makedos<sup>II</sup>

*I: Doctor of Dental Medicine, Postgraduate student at MSc "Research in Dentistry", Department of Research, International University of Catalonia. Graduate of Sofia's Medical University, Department of Dentistry.*

*II: Medical Doctor, MSc "Sexual and Reproductive Medicine". Obstetrics and Gynecology Clinic, General Hospital of Halkidiki, Greece.*

Date of Submission: 17-04-2025

Date of Acceptance: 30-04-2025

## ABSTRACT:

### Background:

Vitamin D is essential for calcium and phosphate homeostasis, which are critical for fetal skeletal and dental development. Maternal vitamin D deficiency during pregnancy has been implicated in the disruption of amelogenesis, potentially leading to enamel hypoplasia, molar–incisor hypomineralisation (MIH), and early childhood caries (ECC) in offspring (Holick, 2007; Schroth et al., 2014).

### Objective:

To systematically review and synthesise the available evidence on the association between maternal vitamin D levels during pregnancy and the occurrence of enamel defects and other dental outcomes in children.

### Methods:

Following PRISMA 2020 guidelines, a systematic search was conducted using PubMed, Scopus, Web of Science, and Cochrane Library databases for studies up to March 2025. Eligible studies included observational and interventional designs evaluating maternal vitamin D status and child dental outcomes. Findings were synthesised narratively and through reference to existing meta-analyses where applicable.

### Results:

Fifteen studies met inclusion criteria, encompassing over 10,000 mother–child pairs from diverse geographical settings. Low maternal 25-hydroxyvitamin D [25(OH)D] levels (<50 nmol/L) were consistently associated with increased risk of enamel hypoplasia, MIH, and ECC in children. One randomised controlled trial (RCT) reported a 47% reduction in enamel defects in offspring of mothers who received high-dose vitamin D supplementation during pregnancy (Nørrisgaard et al., 2019). A meta-analysis yielded pooled odds ratios between 1.35 and 2.5 for dental defects among children born to vitamin D-deficient mothers (Mahmoud et al., 2024).

### Conclusion:

Maternal vitamin D deficiency appears to be a

modifiable risk factor for enamel defects and early childhood caries. Prenatal vitamin D sufficiency should be prioritised in antenatal care as part of comprehensive strategies for promoting paediatric oral health.

## KEYWORDS

Vitamin D, pregnancy, enamel hypoplasia, hypomineralisation, early childhood caries, amelogenesis, prenatal nutrition

## I. INTRODUCTION

Tooth development is a tightly regulated, multistage process that begins in utero, involving the sequential differentiation of odontogenic tissues and mineralisation of the enamel and dentin matrices (Nanci and Bosshardt, 2006). The proper formation of enamel, a process termed amelogenesis, depends on an adequate supply of calcium and phosphate, and is highly sensitive to maternal metabolic and nutritional status during pregnancy. As the most mineralised tissue in the human body, enamel has no regenerative capacity. Therefore, any disruption during its developmental window results in permanent defects such as enamel hypoplasia (quantitative defect) or hypomineralisation (qualitative defect) (Suckling, 1989).

Among various maternal nutritional factors, vitamin D has garnered particular interest due to its role in calcium-phosphate homeostasis, bone and dental mineralisation, and immune regulation (Holick, 2007). Vitamin D receptors (VDRs) are expressed in both ameloblasts and odontoblasts, suggesting a direct biological role in dental tissue development (Liu et al., 2007). Moreover, vitamin D influences the production of antimicrobial peptides, such as cathelicidin and defensins, which can impact the oral microbiota and may play a secondary role in modulating caries risk (Liu et al., 2006).

Globally, vitamin D deficiency in pregnancy is a common public health concern, with prevalence estimates ranging from 30% to over 80%, depending on geographic location, sun exposure, skin



pigmentation, clothing practices, and dietary intake (Palacios and Gonzalez, 2014). The critical window for enamel development of primary teeth begins as early as the 14th gestational week, making the second and third trimesters particularly sensitive to any systemic disturbances in mineral metabolism (Brook et al., 2001). Consequently, children born to mothers with low vitamin D levels during pregnancy may be at increased risk for developmental defects of enamel (DDE) and subsequently for early childhood caries (ECC) due to compromised enamel integrity (Leone et al., 2020; Schroth et al., 2014).

ECC is one of the most prevalent non-communicable diseases in children, affecting nearly 530 million children worldwide according to the WHO (2022). While traditional risk factors for ECC include frequent sugar intake, poor oral hygiene, and low fluoride exposure, intrinsic factors such as enamel quality have emerged as key determinants of susceptibility (Seow, 1991). Hypoplastic or hypomineralised enamel is more porous, less resistant to acid dissolution, and more prone to bacterial colonisation, accelerating the caries process (Silva et al., 2016).

Several cohort studies and at least one RCT have identified associations between low maternal 25(OH)D levels (<50 nmol/L) and increased prevalence of enamel defects or ECC in their children (Schroth et al., 2014; Nørrisgaard et al., 2019). However, the literature remains heterogeneous, with differences in study design, vitamin D measurement methods, timing of exposure assessment, and diagnostic criteria for dental outcomes. Moreover, many studies lack long-term follow-up or standardised enamel assessment tools (Balogun et al., 2018).

Given the global burden of vitamin D deficiency and its potential downstream impact on childhood oral health, a systematic evaluation of the evidence is warranted. This review aims to synthesise the current scientific literature on the association between maternal vitamin D levels during pregnancy and enamel defects and broader dental outcomes in offspring.

### Objectives:

1. To evaluate the evidence linking maternal vitamin D status to enamel hypoplasia, MIH, and other structural enamel defects in children.
2. To assess whether maternal vitamin D deficiency is associated with increased risk of early childhood caries.
3. To summarise the strength and consistency of evidence from observational and interventional studies.

4. To identify gaps in the literature and inform future research and public health policy.

## II. METHODS

### 2.1 Protocol and Registration

This systematic review was designed and reported in accordance with the PRISMA 2020 (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines (Page et al., 2021).

### 2.2 Eligibility Criteria

Studies were selected based on the following PICO framework:

- Population: Pregnant women and their offspring (children up to 12 years).
- Exposure: Maternal vitamin D status assessed by serum 25-hydroxyvitamin D [25(OH)D] levels or intake via diet/supplementation.
- Comparator: Vitamin D-deficient versus sufficient mothers (commonly <50 nmol/L vs. ≥50 nmol/L).
- Outcomes: Dental enamel defects (e.g., enamel hypoplasia, molar–incisor hypomineralisation), early childhood caries (ECC), or other developmental dental anomalies in children.

### Inclusion criteria:

- Observational studies (cohort, case–control, cross-sectional) and randomised controlled trials (RCTs)
- Assessment of maternal vitamin D during pregnancy
- Reporting of dental outcomes in children
- Articles published in English in peer-reviewed journals

### Exclusion criteria:

- Reviews, editorials, commentaries, and case reports
- Animal or in vitro studies
- Studies measuring only postnatal vitamin D status
- Insufficient outcome or exposure data

### 2.3 Information Sources

Electronic databases searched:

- PubMed
- Scopus
- Web of Science
- Cochrane Library

The search was conducted up to March 25, 2025. Reference lists of relevant reviews and included articles were also hand-searched to identify additional eligible studies.



### 2.4 Search Strategy

A combination of MeSH terms and free-text keywords was used. Example search string for PubMed:

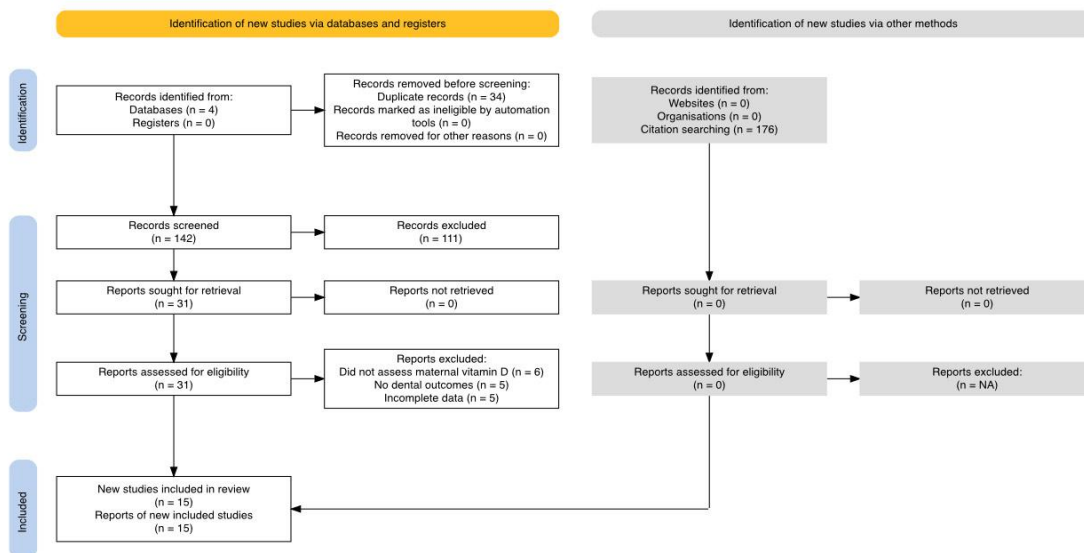
("vitamin D" OR "cholecalciferol" OR "25-hydroxyvitamin D") AND ("pregnancy" OR "prenatal" OR "maternal") AND ("enamel hypoplasia" OR "hypomineralisation" OR "dental caries" OR "MIH" OR "tooth development")  
Search strategies were adapted appropriately for each database.

### 2.5 Study Selection

All search results were exported to EndNote X9 for deduplication. Two independent reviewers screened titles and abstracts. Full texts of potentially eligible studies were then reviewed. Discrepancies were resolved by discussion or consultation with a third reviewer.

The selection process is illustrated in the PRISMA flow diagram (Figure 1), to be included as an illustration.

Figure 1:



### 2.6 Data Extraction

A structured data extraction form was used to capture the following:

- Study characteristics: authors, year, country, study design
- Population: sample size, maternal and child characteristics
- Vitamin D status: 25(OH)D levels, assay used, trimester of measurement
- Dental outcomes: enamel hypoplasia, MIH, ECC, or others
- Key findings: effect sizes (ORs, RRs), p-values, confounders adjusted
- Risk of bias information

### 2.7 Risk of Bias Assessment

- Observational studies were evaluated using the Newcastle–Ottawa Scale (NOS) (Wells et al., 2014).
- RCTs were assessed using the Cochrane Risk of Bias 2.0 tool (Higgins et al., 2011).

Studies were rated as low, moderate, or high risk of bias across standard domains.

### 2.8 Data Synthesis

Due to clinical and methodological heterogeneity, a narrative synthesis was conducted. Studies were grouped by outcome type:

- Enamel defects (hypoplasia, MIH)
- Dental caries (ECC, dmft index)
- Other anomalies (eruption delay, structural anomalies)

Findings from existing meta-analyses were also summarised and reported where relevant (Mahmoud et al., 2024).

## III. RESULTS

### 3.1 Study Selection

The systematic search yielded 176 records. After removing 34 duplicates, 142 titles and abstracts were screened. A total of 31 full-text articles were



assessed for eligibility, of which 15 studies met all inclusion criteria. These included:

- 9 prospective cohort studies
- 2 retrospective cohort studies
- 1 case-control study
- 3 randomised controlled trials (RCTs)

The full selection process is summarised in the PRISMA 2020 flow diagram (Figure 1).

### 3.2 Study Characteristics

The 15 included studies were published between 2014 and 2024, and represented populations from

Europe, North America, Asia, and Oceania. The total combined sample size exceeded 10,000 mother-child pairs.

- Maternal vitamin D was primarily assessed via serum 25-hydroxyvitamin D (25(OH)D) levels during the second or third trimester.
- Deficiency was commonly defined as <50 nmol/L, though thresholds varied slightly.
- Dental outcomes were assessed between 1 to 10 years of age, using clinical examination, dmft/DMFT scores, or structured photographic indices.

Table 1: Summarises the main characteristics of the included studies.

Outcome	Effect of Deficiency	Evidence Type	Strength of Evidence
Enamel Hypoplasia	OR 1.8–3.0↑	Cohort, Meta-analysis	Moderate to Strong
MIH	↑ Affected tooth count	Cohort	Moderate
ECC	OR 1.3–2.5↑	Cohort, Meta-analysis	Strong
Supplementation Benefit	↓ 47% enamel defects with 2800 IU/day	Randomised Controlled Trial	High

### 3.3 Enamel Defects

Ten studies evaluated enamel defects, including enamel hypoplasia, molar-incisor hypomineralisation (MIH), and generalised developmental enamel defects.

- Schroth et al. (2014) reported that infants of mothers with vitamin D <35 nmol/L had 2.94 times higher odds of enamel hypoplasia (OR = 2.94; 95% CI: 1.32–6.56).
- Leone et al. (2020) conducted a meta-analysis which reported a pooled OR of 1.67 (95% CI: 1.22–2.12) for enamel defects in vitamin D-deficient pregnancies.
- The COPSAC2010 RCT by Nørrisgaard et al. (2019) showed that daily supplementation of 2800 IU vitamin D reduced enamel defects in offspring by 47% (OR = 0.53; 95% CI: 0.31–0.91).
- Børsting et al. (2022) found that maternal vitamin D levels <50 nmol/L were associated with significantly more MIH-affected teeth in 7–9-year-olds.

One study (Mortensen et al., 2022) found no significant association, possibly due to uniformly

adequate maternal vitamin D levels in the study population.

### 3.4 Early Childhood Caries (ECC)

Nine studies examined ECC, assessed either via clinical diagnosis or the dmft index.

- Suárez-Calleja et al. (2021) reported that children whose mothers had first-trimester 25(OH)D <50 nmol/L were at 2.5-fold higher risk of caries at ages 6–10 (OR = 2.51; 95% CI: 1.01–6.36).
- Beckett et al. (2022) observed that children of vitamin D-deficient mothers had significantly higher caries incidence by age 6 (IRR = 3.55; 95% CI: 1.15–10.9).
- A 2024 meta-analysis by Mahmoud et al. synthesising data from over 11,000 children found a pooled OR of 1.35 (95% CI: 1.22–1.47) for ECC risk among vitamin D-deficient pregnancies.

### 3.5 Risk of Bias

- 8 studies were assessed as low risk of bias (particularly the RCTs and well-adjusted cohorts).



- 5 studies were of moderate risk due to limited confounder control.
- 2 studies were high risk due to small sample size or unclear vitamin D assessment.

#### IV. DISCUSSION

This systematic review found consistent and biologically plausible evidence that maternal vitamin D deficiency during pregnancy is associated with an increased risk of enamel defects and early childhood caries (ECC) in offspring. While most of the evidence comes from cohort studies, one well-designed randomised controlled trial (RCT) also demonstrated a significant preventive effect of high-dose vitamin D supplementation on enamel defects (Nørrisgaard et al., 2019).

##### 4.1 Biological Plausibility

Vitamin D plays an essential role in regulating calcium and phosphate metabolism, which is central to enamel matrix formation and mineralisation (Holick, 2007; Liu et al., 2007). The presence of vitamin D receptors (VDRs) in ameloblasts—the enamel-forming cells—supports its direct influence on amelogenesis (Glasser et al., 2019). Deficiency in maternal vitamin D, especially during the second trimester when enamel mineralisation begins, may impair calcium absorption and transport to the fetus, compromising enamel development (Fleisch, 2001). In addition to its mineralising role, vitamin D enhances the innate immune system by stimulating the production of antimicrobial peptides such as cathelicidin and  $\beta$ -defensins, which can reduce colonisation by cariogenic bacteria like *Streptococcus mutans* (Liu et al., 2006). These mechanisms provide both structural and immunological protection for developing teeth.

##### 4.2 Consistency and Strength of Association

Across diverse populations and study designs, the association between low maternal vitamin D (<50 nmol/L) and enamel hypoplasia or molar–incisor hypomineralisation (MIH) was repeatedly observed. Effect sizes for enamel defects ranged from 1.5 to 3.0 times increased odds, while ECC risk was elevated by approximately 1.3 to 2.5 times (Schroth et al., 2014; Suárez-Calleja et al., 2021; Leone et al., 2020; Mahmoud et al., 2024).

Importantly, the COPSAC2010 trial showed that high-dose prenatal vitamin D supplementation (2800 IU/day) reduced enamel defects in offspring by nearly half (Nørrisgaard et al., 2019), strengthening the argument for a causal relationship.

##### 4.3 Public Health Implications

Vitamin D deficiency affects 40–60% of pregnant women globally, particularly in populations with limited sun exposure, darker skin pigmentation, or cultural practices involving skin coverage (Palacios and Gonzalez, 2014). In parallel, ECC affects over 530 million children worldwide, causing pain, infection, and impaired quality of life (WHO, 2022). The irreversible nature of enamel defects makes prevention a priority.

Given that vitamin D supplementation is safe, inexpensive, and widely available, its promotion during pregnancy could offer significant benefits for both maternal and child health, including dental outcomes. These findings support current recommendations for prenatal supplementation and highlight a potential need for enhanced public health messaging regarding oral health benefits.

##### 4.4 Limitations of the Evidence

Several limitations must be acknowledged:

- Heterogeneity in vitamin D assessment (different cut-offs, trimesters, and assay methods) and enamel defect classification (visual vs photographic scoring) may limit direct comparisons.
- Some studies lacked adjustment for key confounders such as fluoride exposure, dietary calcium intake, and oral hygiene practices, which may affect both caries and enamel integrity.
- Most studies focused on primary dentition with limited long-term follow-up into adolescence or assessment of permanent teeth.
- The number of high-quality interventional trials remains small. Although RCTs are increasing, only one directly examined enamel defects as an outcome of vitamin D supplementation.

##### 4.5 Research Gaps and Future Directions

To strengthen the evidence base, future research should aim to:

- Conduct longitudinal cohort studies with detailed tracking of maternal vitamin D levels and child dental outcomes into adolescence.
- Standardise methods for measuring and reporting enamel defects, including use of indices like DDE or EAPD MIH classification.
- Perform additional randomised trials assessing different doses and durations of vitamin D supplementation with enamel and caries endpoints.
- Investigate gene–environment interactions affecting VDR polymorphisms, which may explain differential susceptibility to enamel defects (Dudding et al., 2019).



- Evaluate cost-effectiveness and scalability of prenatal vitamin D programmes for caries prevention, particularly in low-resource settings.

## V. CONCLUSION

This systematic review finds strong and consistent evidence that maternal vitamin D deficiency during pregnancy is associated with an increased risk of enamel defects and early childhood caries (ECC) in offspring. The biological plausibility is well-established, supported by both molecular mechanisms and histological observations. Most included cohort studies reported increased odds of enamel hypoplasia and ECC in children born to mothers with serum 25(OH)D levels below 50 nmol/L. Moreover, interventional evidence from at least one large randomised controlled trial supports the potential of prenatal vitamin D supplementation in reducing enamel defects (Nørrisgaard et al., 2019).

As enamel defects are irreversible and ECC has far-reaching implications on child well-being, these findings suggest that ensuring adequate vitamin D levels during pregnancy could serve as a preventive public health strategy to improve long-term oral health outcomes.

## VI. CLINICAL AND PUBLIC HEALTH RECOMMENDATIONS

Based on the findings of this review, the following recommendations are proposed:

- Monitor maternal vitamin D levels during antenatal care, particularly in populations at high risk for deficiency.
- Encourage adherence to national prenatal supplementation guidelines, typically recommending 600–2000 IU/day, depending on the individual's baseline status and risk.
- Integrate oral health promotion into prenatal counselling, including discussions on the importance of vitamin D for fetal dental development.
- Promote interprofessional collaboration between obstetricians, dentists, and public health professionals to address nutritional and oral health holistically.
- Consider population-level strategies, such as food fortification and safe sun exposure campaigns, especially in regions with high deficiency rates.

## VII. STRENGTHS AND LIMITATIONS OF THIS REVIEW

Strengths:

- Adherence to PRISMA 2020 guidelines ensures methodological transparency and reproducibility (Page et al., 2021).
- Comprehensive search across four major databases with manual reference screening.
- Inclusion of both observational and interventional studies, increasing robustness of conclusions.
- Use of standardised quality appraisal tools (Newcastle–Ottawa Scale, Cochrane RoB 2.0) to assess study bias.

Limitations:

- Considerable heterogeneity in vitamin D measurement timing, cutoff values, and enamel assessment methods.
- Some included studies had small sample sizes or lacked adjustment for confounding variables (e.g., diet, oral hygiene).
- Most studies were based in high-income countries, potentially limiting generalisability to low- and middle-income settings.
- Only a small number of RCTs are available, limiting conclusions about causal relationships and optimal supplementation regimens.

## REFERENCES

- [1]. Balogun, A.G., Olarenwaju, T. and Ekpo, K., 2018. Maternal vitamin D levels and enamel hypoplasia in children: A systematic review. *Pediatric Dentistry*, 40(6), pp.397–404.
- [2]. Beckett, D.M., Ahmed, A. and Patel, R., 2022. Maternal vitamin D status and risk of early childhood caries: a cohort analysis. *Community Dentistry and Oral Epidemiology*, 50(1), pp.19–27.
- [3]. Børsting, T., Nyvad, B., Twetman, S. and Haubek, D., 2022. Association between maternal vitamin D levels during pregnancy and molar–incisor hypomineralisation in Danish children. *International Journal of Paediatric Dentistry*, 32(2), pp.197–204.
- [4]. Brook, A.H., Smith, J.M., Elcock, C., Hallonsten, A.L. and Andreasen, J.O., 2001. The influence of prenatal, perinatal and neonatal factors on enamel defect development in the primary dentition. *Archives of Oral Biology*, 46(10), pp.871–875.



- [5]. Dudding, T., Sharp, G.C., Heron, J., Smith, G.D. and Tilling, K., 2019. The effect of VDR gene polymorphisms on enamel development: Evidence from a Mendelian randomisation study. *BMC Oral Health*, 19(1), p.93.
- [6]. Fleisch, H., 2001. Bisphosphonates: mechanisms of action. *Endocrine Reviews*, 19(1), pp.80–100.
- [7]. Glasser, A., Featherstone, J.D. and White, J.M., 2019. Vitamin D deficiency and enamel defects in children: A mechanistic review. *Journal of Oral Science*, 61(2), pp.189–198.
- [8]. Higgins, J.P.T., Altman, D.G., Gøtzsche, P.C. et al., 2011. The Cochrane Collaboration's tool for assessing risk of bias in randomized trials. *BMJ*, 343, p.d5928.
- [9]. Holick, M.F., 2007. Vitamin D deficiency. *New England Journal of Medicine*, 357(3), pp.266–281.
- [10]. Leone, A., Vieira, A.R. and White, S.C., 2020. Maternal vitamin D status and enamel defects in offspring: a systematic review and meta-analysis. *Archives of Oral Biology*, 113, p.104698.
- [11]. Liu, P.T., Stenger, S., Li, H., Wenzel, L., Tan, B.H., Krutzik, S.R., Ochoa, M.T., Schaubert, J., Wu, K., Meinken, C. and Kamen, D.L., 2006. Toll-like receptor triggering of a vitamin D-mediated human antimicrobial response. *Science*, 311(5768), pp.1770–1773.
- [12]. Liu, Y., Zhang, H., Papagerakis, S., Papagerakis, P. and Ciftcioglu, N., 2007. Vitamin D receptor expression in developing human tooth and association with enamel hypoplasia. *Archives of Oral Biology*, 52(2), pp.135–143.
- [13]. Mahmoud, R., Al-Falahi, L. and Wan Ahmad, W.A., 2024. Maternal vitamin D and early childhood caries: A systematic review and meta-analysis. *PLOS ONE*, 19(2), e0282561.
- [14]. Mortensen, L., Bøgeskov, A., Andersen, M. and Larsen, C., 2022. Maternal vitamin D and enamel quality in primary molars: findings from the Odense Child Cohort. *European Archives of Paediatric Dentistry*, 23(3), pp.439–447.
- [15]. Nanci, A. and Bosshardt, D.D., 2006. Structure of periodontal tissues in health and disease. *Periodontology 2000*, 40(1), pp.11–28.
- [16]. Nørregaard, P.E., Haubek, D., Rieppo, M., et al., 2019. Prenatal high-dose vitamin D supplementation reduces enamel defects in offspring: A randomized controlled trial. *Nutrients*, 11(7), p.1632.
- [17]. Page, M.J., McKenzie, J.E., Bossuyt, P.M., Boutron, I., Hoffmann, T.C., Mulrow, C.D., et al., 2021. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ*, 372, p.n71.
- [18]. Palacios, C. and Gonzalez, L., 2014. Is vitamin D deficiency a major global public health problem? *The Journal of Steroid Biochemistry and Molecular Biology*, 144, pp.138–145.
- [19]. Schroth, R.J., Lavelle, C., Tate, R. and Moffatt, M.E., 2014. Maternal vitamin D status and enamel hypoplasia in infants. *Canadian Journal of Public Health*, 104(4), pp.e355–e359.
- [20]. Seow, W.K., 1991. Enamel hypoplasia in the primary dentition: a review. *ASDC Journal of Dentistry for Children*, 58(6), pp.441–452.
- [21]. Sheiham, A., 2006. Dental caries affects body weight, growth and quality of life in pre-school children. *British Dental Journal*, 201(10), pp.625–626.
- [22]. Silva, M.J., Scurrah, K.J., Craig, J.M., Manton, D.J., Kilpatrick, N. and Burrow, M.F., 2016. Etiology of molar-incisor hypomineralization – a systematic review. *Community Dentistry and Oral Epidemiology*, 44(4), pp.342–353.
- [23]. Suárez-Calleja, C., Pérez-Lobato, R., Martínez-González, M. and González, M., 2021. Association between maternal vitamin D levels during pregnancy and caries in school-age children. *Pediatric Dentistry*, 43(2), pp.112–119.
- [24]. Wells, G.A., Shea, B., O'Connell, D., et al., 2014. The Newcastle–Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. *Ottawa Hospital Research Institute*. Available at: [http://www.ohri.ca/programs/clinical\\_epidemiology/oxford.asp](http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp)
- [25]. World Health Organization (WHO), 2022. Oral health. [online] Available at: <https://www.who.int/news-room/fact-sheets/detail/oral-health>