

Oral Health Implications of Nutritional Deficiencies: A Narrative Review from a Public Health Perspective

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Abstract

Background: Oral diseases affect billions of people worldwide and share determinants with undernutrition and poor diet quality. Nutritional deficiencies may influence tooth development, periodontal health, and oral mucosal integrity, yet the evidence has rarely been synthesised from a public health standpoint.

Objective: To narratively synthesise evidence on the oral health consequences of selected nutritional deficiencies and to discuss implications for prevention, policy, and surveillance.

Methods: A narrative review was conducted following SANRA principles. PubMed and Google Scholar were searched for English-language reviews, meta-analyses, trials, cohort, case-control, cross-sectional and case studies addressing vitamin D, vitamin C, B-complex vitamins, iron, antioxidants, and protein-energy malnutrition in relation to caries, developmental enamel defects, periodontal disease, and oral mucosal conditions.

Results: The strongest, though still heterogeneous, evidence concerns vitamin D: a meta-analysis reported a 22% higher caries risk in vitamin D-deficient children, while controlled trials suggested a protective effect of supplementation with low certainty. Findings on enamel hypomineralisation are inconsistent. Early childhood malnutrition is associated with delayed tooth eruption. Lower antioxidant and vitamin D status are associated with periodontal disease, and vitamin C depletion increases gingival bleeding. Vitamin B12, iron and folate deficiencies commonly present as glossitis and angular cheilitis, which may serve as early clinical signals in primary care.

Conclusions: Nutritional deficiencies are plausible, modifiable contributors to oral disease burden, but causal evidence is limited by observational designs and confounding by sugar intake and socioeconomic status. Public health strategies should integrate oral health with nutrition programmes, strengthen maternal and child micronutrient interventions, address free-sugar consumption, and add nutritional indicators to oral health surveillance.

Keywords: oral health; malnutrition; micronutrient deficiency; vitamin D; dental caries; periodontal disease; public health

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1. Introduction

Oral diseases remain among the most prevalent noncommunicable conditions worldwide, affecting an estimated 3.5 billion people, and the greatest burden falls on disadvantaged populations [1,2]. Despite this scale, oral health has long been neglected in global health policy and is poorly integrated with other health programmes [3].

Diet and nutrition influence oral health through both local and systemic pathways. Locally, the frequency and quantity of fermentable carbohydrate intake drives caries. Systemically, nutrient adequacy during tooth formation, and throughout life, shapes enamel and dentine quality, the host inflammatory response, and the integrity of the oral mucosa [4]. The relationship is also bidirectional: painful, missing or decayed teeth restrict food choice and may worsen nutritional status [4].

Micronutrient deficiencies, sometimes termed “hidden hunger”, remain widespread in low- and middle-income countries, including deficiencies of iron, vitamin A, zinc, folate, vitamin B12 and vitamin D [5]. Many of the same populations experience rising consumption of free sugars, producing a double burden of undernutrition and diet-related oral disease [6]. Individual studies have addressed particular nutrients, but the oral consequences of nutritional deficiencies have not been examined together with a public health lens.

This narrative review therefore aims to (i) summarise evidence on the oral health implications of selected nutritional deficiencies, (ii) identify gaps and sources of bias in the evidence, and (iii) discuss implications for public health policy, programmes, and research.

2. Methods

2.1 Study design

A narrative review design was chosen because the topic spans heterogeneous nutrients, outcomes and study designs, which precludes a single pooled estimate. The review was structured according to recommendations for narrative reviews [7] and reported with reference to the SANRA quality criteria [8].

2.2 Literature search

PubMed/MEDLINE and Google Scholar were searched without a lower date limit, with emphasis on literature from 2003 onwards. Search terms combined controlled vocabulary and free text: (“nutritional deficiency” OR malnutrition OR “micronutrient deficiency” OR “vitamin D” OR “vitamin C” OR “vitamin B12” OR folate OR iron OR antioxidant) AND (“oral health” OR “dental caries” OR “enamel hypoplasia” OR “molar incisor hypomineralization” OR periodontitis OR gingivitis OR glossitis OR “tooth eruption”). Reference lists of key articles were hand-searched. Citations were retrieved and checked against the original publication records.

2.3 Selection and synthesis

Systematic reviews, meta-analyses, controlled trials, cohort, case-control, cross-sectional studies, consensus reports, and illustrative case series published in English were eligible. Studies focusing solely on dietary sugars without a deficiency component, animal-only studies, and opinion pieces without data were excluded from the evidence tables but informed the context. Because of the narrative approach, no formal risk-of-bias scoring was applied; instead, study design and consistency of findings were weighed qualitatively. Evidence was grouped by nutrient and oral outcome, and implications were interpreted from a public health perspective.

3. Results

Table 1 summarises the principal nutrient-oral outcome associations, and Figure 1 presents the conceptual framework that links upstream determinants, nutritional deficiencies, oral outcomes, and public health responses.

3.1 Vitamin D, calcium and phosphorus: tooth development and caries

Vitamin D regulates calcium and phosphate homeostasis and acts through the vitamin D receptor in mineralised tissues. In children, severe deficiency can cause defective mineralisation, with dentine and enamel defects that may increase susceptibility to caries [9]. A systematic review and meta-analysis of cohort, cross-sectional and case-control studies found that children with vitamin D deficiency had a 22% higher risk of caries than those with normal levels (RR 1.22), with stronger associations in primary than in permanent teeth [10]. A meta-analysis of 24 controlled trials involving 2,827 children, mostly conducted between the 1920s and 1980s, reported a pooled caries rate ratio of 0.53 with vitamin D exposure, but the author rated certainty as low because of trial quality and heterogeneous vitamin D sources [11]. A case-control study reported lower 25(OH)D concentrations in children with severe early childhood caries [12].

Evidence on developmental enamel defects is less consistent. In the German GINIplus and LISAplus birth cohorts ($n = 1,048$), each 10 nmol/L increase in serum 25(OH)D at age 10 was associated with lower odds of molar-incisor hypomineralisation (OR 0.89) [13]. By contrast, a larger Dutch population-based cohort found that 25(OH)D measured in mid-gestation, in cord blood, and at 6 years was not associated with hypomineralised second primary molars or MIH [14]. Differences in timing of measurement, populations and confounder control may explain these discrepancies.

3.2 Protein-energy malnutrition

Chronic undernutrition during early childhood has been associated with delayed tooth eruption. In Haitian adolescents, a history of early childhood malnutrition was linked to altered eruption timing [15]. Delayed eruption and enamel defects may prolong or alter caries susceptibility, although evidence for malnutrition as an independent caries determinant remains limited and confounded by sugar intake and access to fluoride.

3.3 Periodontal disease: vitamin D, antioxidants and vitamin C

In a nationally representative US sample, lower serum 25(OH)D was associated with greater periodontal disease [16]. Consensus work on lifestyle and systemic determinants of caries and periodontal disease, as well as narrative reviews of nutrition and periodontal health, highlight vitamins C and D, antioxidants and dietary pattern as plausible modifiers of the host inflammatory response, while noting that much of the supporting evidence is observational [17,18]. In NHANES III, periodontitis prevalence was inversely related to serum antioxidant concentrations [19]. Vitamin C has the most direct experimental support: controlled depletion in healthy volunteers increased gingival bleeding without a corresponding rise in plaque, and bleeding improved with supplementation [20]. Frank scurvy, now uncommon, presents with swollen, haemorrhagic gingivae.

3.4 Oral mucosal manifestations: B-complex vitamins and iron

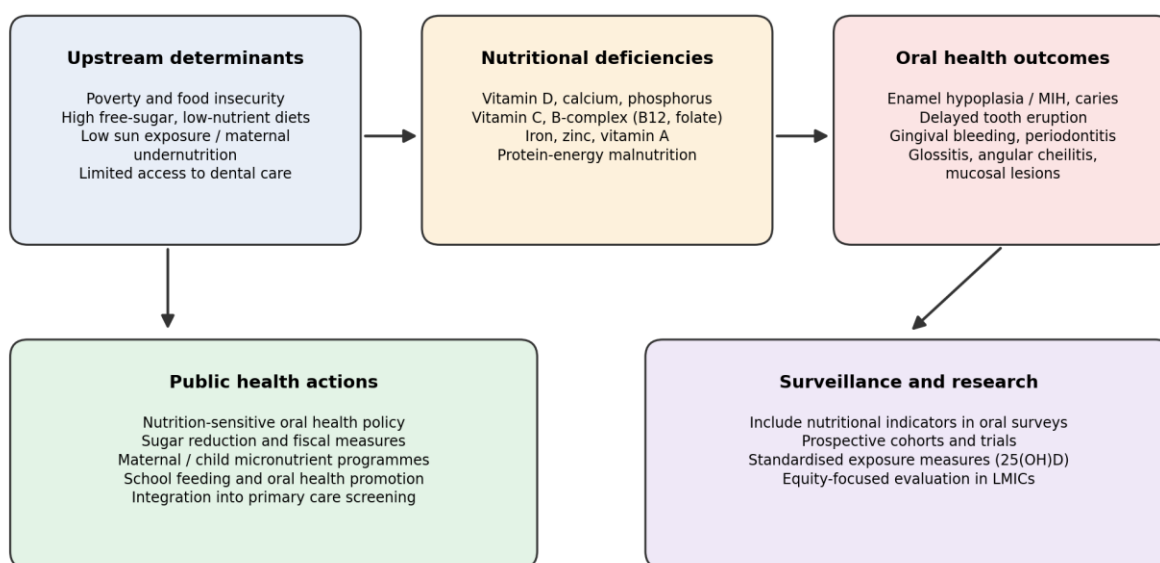
Deficiencies of vitamin B12, folate, riboflavin and iron commonly manifest as atrophic glossitis, angular cheilitis, recurrent ulceration or burning sensations. A case series described glossitis with linear lesions as an early sign of vitamin B12 deficiency, occurring in patients without anaemia or neurological symptoms [21]. Because the oral cavity is often examined before haematological abnormalities are detected, dental and primary care visits provide an opportunity for early recognition, particularly among older adults, people on restricted diets, and those with malabsorption.

Table 1. Nutritional deficiencies and associated oral health outcomes.

Nutrient / condition	Oral manifestations	Key evidence (study type)	Public health relevance
Vitamin D (with calcium, phosphorus)	Enamel hypoplasia, MIH, early childhood caries; possible periodontal inflammation	Pooled RR 1.22 for caries in deficient children [10]; supplementation trials RR 0.53, low certainty [11]; case-control: lower 25(OH)D in severe ECC [12]; cohort: higher 25(OH)D linked to less MIH [13], but not in a large birth cohort [14]; NHANES association with periodontal disease [16]	Maternal and child supplementation, food fortification, safe sun exposure; confounding and causality remain unresolved
Protein-energy malnutrition	Delayed tooth eruption, enamel defects, higher caries susceptibility in primary dentition	Observational study in Haitian adolescents linking early malnutrition to delayed eruption [15]	Child nutrition programmes may carry oral health co-benefits
Vitamin C	Gingival bleeding, swollen gums; scurvy in severe deficiency	Controlled depletion study showed increased gingival bleeding [20]; narrative reviews [18]	Low-cost dietary correction (fruit and vegetables); relevant to food-insecure groups

Antioxidant nutrients (e.g., vitamins C, E, carotenoids)	Greater periodontitis prevalence at lower serum concentrations	Cross-sectional NHANES analysis [19]; consensus and review literature [17,18]	Supports diet quality as a periodontal risk factor; evidence mostly observational
Vitamin B12, folate, iron, riboflavin	Glossitis, angular cheilitis, mucosal atrophy, burning mouth	Case series: glossitis with linear lesions as early B12 sign, even without anaemia [21]	Oral signs can prompt early detection of anaemia and malabsorption in primary care

ECC, early childhood caries; MIH, molar-incisor hypomineralisation; NHANES, National Health and Nutrition Examination Survey; RR, relative risk. Numbers in brackets refer to the reference list.



Relationship is bidirectional: oral disease also impairs chewing, intake and nutritional status

Figure 1. Conceptual framework linking upstream determinants, nutritional deficiencies, oral health outcomes, and public health responses.

4. Discussion

4.1 Principal findings

Across nutrients, the evidence suggests that deficiencies affect oral health through three broad routes: impaired tooth formation and eruption, altered host inflammatory response in the periodontium, and compromised mucosal integrity. The best-developed literature concerns vitamin D and caries, where the direction of association is generally consistent but the magnitude, causality, and relevance to enamel defects remain uncertain [10,11,13,14]. For other nutrients the evidence rests largely on cross-sectional data, small experimental studies, or case series.

4.2 Public health implications

Oral diseases and undernutrition share upstream determinants such as poverty, food insecurity, and limited access to services, which supports the common risk factor approach advocated by the World Health Organization [22]. Three implications follow. First, nutrition programmes, including maternal micronutrient supplementation, complementary feeding, school meals, and food fortification, should be assessed for oral health co-benefits, and oral health promotion

should be built into these platforms. Second, dietary sugar control remains central: restricting free sugars to below 10% of energy intake is associated with lower caries, and the World Health Organization recommends this threshold, with a conditional recommendation for below 5% [23,24]. Nutrient-deficient diets are often also sugar-rich, so interventions that address only micronutrient status will not remove caries risk [6,25]. Third, primary care and dental settings can use oral signs such as glossitis, gingival bleeding, and delayed eruption as low-cost triggers for nutritional assessment and referral [21].

4.3 Strengths and limitations of the evidence

Most included studies are observational and cross-sectional, so reverse causation is plausible; for example, caries may reduce food intake. Residual confounding by sugar consumption, fluoride exposure, oral hygiene and socioeconomic position is common. Vitamin D measurement varies in assay, season and threshold, complicating comparison. Many older trials had methodological weaknesses [11]. As a narrative review, this work is subject to selection bias, no formal risk-of-bias appraisal was undertaken, and the English-language restriction may have excluded relevant regional evidence. Nevertheless, the narrative approach permitted integration of diverse evidence types.

4.4 Recommendations for research and policy

Future work should include prospective cohorts and pragmatic trials in low- and middle-income settings, standardised exposure and outcome definitions, and explicit adjustment for dietary sugars. Oral health surveys should add nutritional indicators, and national nutrition surveys should include basic oral examinations, allowing joint surveillance. Policy-makers should consider integrating oral health into nutrition and maternal-child health strategies and evaluating equity impacts.

5. Conclusion

Nutritional deficiencies are plausible and modifiable contributors to oral disease, with the clearest evidence for vitamin D and caries, vitamin C and gingival bleeding, and B-complex vitamins and iron in oral mucosal disease. Because current evidence is predominantly observational, recommendations should be cautious and complement, not replace, established measures such as sugar reduction and fluoride use. An integrated public health approach linking nutrition, oral health, and primary care offers the most efficient route to reduce the combined burden of malnutrition and oral disease.

Declarations

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Data availability: No new data were generated; all sources are cited in the reference list.

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