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Micronutrients and Dental Caries Prevention: A Narrative Review

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ABSTRACT

Dental caries remains a common chronic disease worldwide, and its development is influenced by a complex interplay of oral, dietary, and host-related factors. This narrative review summarizes current evidence on the potential role of micronutrients in dental caries prevention, with emphasis on their biological relevance and clinical evidence. Vitamin D has been the most extensively studied micronutrient and may influence caries susceptibility through calcium and phosphate homeostasis, dental mineralization, and immune regulation. However, although observational studies frequently report an inverse association between vitamin D status and caries, findings from randomized controlled trials and Mendelian randomization studies remain inconsistent. Calcium and phosphorus are essential for the formation and maintenance of mineralized dental tissues, while zinc and magnesium may contribute to mineralization and resistance to demineralization. Vitamins A, E, and K and other trace elements may also have roles in maintaining oral health, although evidence supporting their specific effects on dental caries is more limited. Differences in study populations, nutritional assessment, caries outcomes, and study design make comparisons across studies challenging. Micronutrient status may be an important component of the biological context underlying dental caries, but current evidence does not support micronutrient supplementation as a replacement for established preventive measures. Further well-designed clinical studies are needed to clarify the effects of individual and combined micronutrients and to determine which populations may benefit most from targeted nutritional interventions.

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I. INTRODUCTION

Dental caries is one of the most prevalent chronic diseases worldwide and remains a major public health concern across the life course.[1] Despite substantial advances in preventive dentistry, untreated dental caries continues to affect billions of people and may result in pain, infection, tooth loss, and impaired quality of life.[2] The burden of dental caries is particularly important in children and other vulnerable populations, in whom oral disease may coexist with nutritional, socioeconomic, and environmental disadvantages.[3, 4]

Dental caries is traditionally understood as a biofilm-mediated, diet-modulated, multifactorial disease resulting from an imbalance between demineralization and remineralization of dental hard tissues. Fermentable carbohydrate intake, cariogenic microorganisms, salivary conditions, tooth susceptibility, and fluoride exposure are established determinants of this balance.[5] However, increasing evidence suggests that caries susceptibility cannot be explained solely by local oral factors. Systemic nutritional status may also influence the development and progression of caries by affecting tooth development and mineralization, the availability of calcium and phosphate, salivary and immune functions, and the ecological characteristics of the oral microbiota.[6-8]

Nutrition is therefore increasingly recognized as an important component of the oral-systemic interface. Vitamins and minerals are essential for the formation, maturation, and maintenance of mineralized dental tissues. Among these micronutrients, vitamin D has received particular attention because of its potential roles in calcium homeostasis, enamel mineralization, and immune modulation.[9-12] Calcium and phosphorus are fundamental components of dental hard tissues, while zinc, magnesium, and other trace elements may influence mineralization processes and the resistance of dental tissues to demineralization.[13-17] Nevertheless, the strength of evidence varies considerably among individual micronutrients, with observational associations not always being supported by randomized controlled trials or other approaches to causal inference.[18-20]

This narrative review synthesizes current evidence on the role of micronutrients in dental caries prevention. Particular emphasis is placed on their biological mechanisms and clinical evidence, including their potential effects on dental tissue mineralization and remineralization, mineral homeostasis, and host-related pathways. The review also considers the consistency and limitations of current evidence to clarify the potential role of nutritional strategies as complementary approaches to established caries prevention.

II. VITAMIN D

Vitamin D is the micronutrient most extensively investigated in relation to dental caries. Its potential role in caries prevention is biologically plausible because vitamin D contributes to calcium and phosphate homeostasis, mineralization of dental tissues, and regulation of innate immune responses.[11, 21] Adequate vitamin D status may therefore influence caries susceptibility through both systemic effects on mineral metabolism and local effects on host defence.

The relationship between vitamin D and dental caries has been examined extensively in observational studies. Lower vitamin D status, particularly lower circulating 25-hydroxyvitamin D concentrations, has frequently been associated with greater caries risk in children and adolescents.[9, 10] A cross-sectional study among US children and youth reported a protective association between vitamin D status and dental caries, while studies using the Cariogram model have similarly suggested differences in caries risk according to vitamin D deficiency.[9, 10] Prenatal vitamin D status has also attracted attention, with systematic review evidence suggesting an association between maternal vitamin D deficiency and caries in offspring.[22]

Several mechanisms may explain these observations. Vitamin D facilitates intestinal calcium absorption and thereby contributes to mineral availability during tooth development.[9, 10, 21] In addition, vitamin D has immunomodulatory effects that may be relevant to the oral environment. Vitamin D signaling can regulate antimicrobial peptides, including cathelicidin LL-37 and defensins, and may influence inflammatory responses.[11, 12, 23, 24] Evidence from children with dental caries has suggested an association between vitamin D status and salivary cathelicidin levels, supporting the possibility that vitamin D may contribute to local antimicrobial defense.[23]

Despite this biological plausibility and the consistency of some observational findings, evidence from intervention and causal studies remains less conclusive. A systematic review and meta-analysis of controlled clinical trials reported a potentially protective effect of vitamin D against dental caries, with a relative-rate estimate of approximately 0.53, but the certainty of evidence was limited by heterogeneity and potential bias.[18] Another systematic review and meta-analysis focusing on early childhood caries similarly identified an association between vitamin D and caries, although considerable variation existed among the included studies.[25]

Importantly, randomized controlled trials and Mendelian randomization studies have not consistently supported a causal preventive effect. An umbrella review of vitamin D and multiple health outcomes found discrepancies between observational and randomized evidence.[19] A Mendelian randomization analysis of circulating vitamin D concentrations also did not provide strong evidence for a causal association with dental caries.[26] Similarly, analyses of prenatal, perinatal, and early-childhood vitamin D status have produced complex findings.[27] A further Mendelian randomization study examining the causal association between 25-hydroxyvitamin D and caries also failed to establish a clear causal relationship, while a systematic review and meta-analysis of serum vitamin D levels and caries risk highlighted substantial heterogeneity across observational studies.[20, 28]

III. VITAMIN A, E, AND K

Although vitamin D has received substantially greater attention, other fat-soluble vitamins may also contribute to oral health. Epidemiological evidence suggests that higher intake of vitamins A, E, and K may be associated with lower dental caries prevalence, particularly among children and adolescents.[9, 10, 29] However, the evidence base for these vitamins is considerably smaller than that for vitamin D.

Vitamin A has important roles in epithelial integrity and tissue development and may therefore contribute indirectly to maintaining oral health. Vitamin E, through its antioxidant properties, may influence oxidative stress and salivary function, while vitamin K is involved in bone and mineral metabolism. [9, 10, 29] These biological roles provide plausible pathways through which inadequate intake might influence susceptibility to oral disease. However, direct evidence linking supplementation of vitamins A, E, or K with reduced dental caries incidence remains limited. Current evidence is primarily derived from observational associations rather than well-designed intervention studies.[9, 10, 29] Consequently, these vitamins should be considered components of adequate overall nutrition rather than established independent preventive agents for dental caries.

The broader relationship between nutrition and dental caries further supports this interpretation. Nutritional status reflects multiple interacting dietary and metabolic factors, making it difficult to attribute caries risk to a single vitamin in isolation.[30] Future research should therefore evaluate these vitamins alongside dietary patterns and other nutritional determinants rather than relying exclusively on isolated nutrient exposure.

IV. CALCIUM AND PHOSPHORUS

Calcium and phosphorus are fundamental micronutrients for the formation and maintenance of mineralized dental tissues. Enamel and dentin contain high concentrations of calcium and phosphate within hydroxyapatite crystals, making adequate availability of these minerals essential for normal tooth development and mineral homeostasis.[13, 14, 31, 32] Deficiencies or inadequate availability of calcium and phosphorus may increase susceptibility to dental caries by compromising mineralization and reducing the capacity of dental tissues to withstand acid-mediated mineral loss. Evidence from maternal nutrition research has suggested that calcium supplementation during pregnancy may influence the subsequent caries experience of offspring. A follow-up of a randomized controlled trial found an association between maternal calcium supplementation and dental caries outcomes in children at 12 years of age.[13]

The biological importance of calcium and phosphorus extends beyond tooth development. The availability of these minerals at the tooth surface is critical to the dynamic balance between demineralization and remineralization. When calcium and phosphate concentrations are sufficient, the oral environment can support mineral deposition and repair of early enamel lesions.[13, 14, 31, 32] Conversely, repeated acid exposure can drive mineral loss when mineral availability is insufficient.

An important aspect of this evidence is the distinction between systemic nutritional adequacy and topical mineral delivery. Milk-derived casein phosphopeptides (CPP) can stabilize amorphous calcium phosphate (ACP), forming CPP-ACP complexes that maintain calcium and phosphate in a bioavailable state at the tooth surface.[13, 14, 31, 32] These complexes can enhance remineralization and inhibit demineralization by maintaining supersaturation with respect to enamel mineral. Although CPP-ACP itself is a bioactive peptide-mineral complex rather than a micronutrient, its evidence provides mechanistic support for the importance of calcium and phosphate availability in caries prevention.

Research into biomimetic remineralization has further expanded this concept. Calcium- and magnesium-containing systems have been investigated for their ability to stabilize amorphous mineral precursors and promote mineral deposition in damaged dental tissues.[33] These developments suggest that future nutritional and preventive approaches may increasingly focus not only on the quantity of minerals available but also on their physicochemical form and ability to participate in controlled mineralization.

Overall, calcium and phosphorus have stronger mechanistic support than many other micronutrients in relation to dental caries. Nevertheless, the clinical effectiveness of systemic supplementation depends on baseline nutritional status, developmental stage, dietary context, and the route through which minerals are delivered. Adequate dietary intake should therefore be regarded as part of maintaining normal dental development, while topical remineralization approaches may provide additional local benefits.

V. ZINC

Zinc is a trace element that may influence dental caries through effects on mineralization, bacterial metabolism, and the physicochemical properties of dental hard tissues.[15-17, 34] Zinc is incorporated into mineralized tissues and may interact with calcium and phosphate during hydroxyapatite formation. Experimental and mechanistic evidence suggests that zinc can modify the crystallization and stability of dental mineral. Zinc incorporation into hydroxyapatite may increase resistance to demineralization, while zinc can also influence calcium and magnesium bioavailability.[13, 14, 31, 32] More recent experimental work indicates that trace zinc can affect amorphous calcium phosphate crystallization, suggesting a potential role in regulating the transition from precursor phases to more stable mineral structures.[16] Zinc may also affect bacterial activity. Its ability to interfere with bacterial metabolism provides a possible additional mechanism through which adequate zinc status could influence caries susceptibility.[15] However, the relationship between zinc exposure and caries is complex and may depend on concentration, chemical form, and the broader mineral environment.

Current evidence is predominantly mechanistic and observational rather than derived from large randomized clinical trials. Therefore, while zinc has biologically plausible anticariogenic properties, evidence remains insufficient to establish zinc supplementation as an independent strategy for caries prevention. Its importance may instead lie in maintaining adequate trace-element status as part of overall nutritional health.

VI. MAGNESIUM

Magnesium is another mineral involved in dental mineralization and may influence the formation and stability of mineral precursors.[16, 33] Its biological role is particularly relevant to biomimetic mineralization because magnesium can interact with amorphous calcium phosphate and influence mineralization kinetics. The incorporation and stabilization of magnesium within

mineral phases may help regulate crystal growth and contribute to the formation of dental tissues with appropriate structural characteristics.[16, 33] Recent research into biomimetic mineralization has highlighted magnesium-containing systems as potential approaches for promoting repair of demineralized dental tissues.[33]

Evidence concerning magnesium and caries prevalence is less extensive than that for calcium, phosphorus, or vitamin D. Available research largely focuses on mineralization mechanisms and experimental models rather than clinical supplementation trials. Consequently, although magnesium is biologically relevant to dental mineralization, its independent contribution to caries prevention remains to be established. The potential role of magnesium may nevertheless become increasingly important as research moves toward biomimetic approaches that reproduce the physicochemical processes involved in natural enamel and dentin mineralization[35].[33, 36-38]

VII. OTHER TRACE ELEMENTS

Several additional trace elements have been investigated in relation to dental caries, including strontium, boron, molybdenum, and other inorganic elements.[17, 34] These elements may influence mineralization, enamel resistance, and the local chemical environment of dental tissues. Evidence from population studies has demonstrated associations between elemental composition and caries prevalence. Studies examining multiple inorganic elements in teeth have identified differences in elemental profiles according to caries status.[34] Strontium exposure has been associated with lower caries risk in some populations, whereas exposure to potentially toxic elements such as lead has been associated with increased susceptibility.[35]

However, interpretation of these findings is complicated by environmental exposure and socioeconomic factors. Trace-element exposure may vary according to drinking water, geographic location, occupation, environmental contamination, and dietary patterns. These factors can simultaneously influence oral health and nutritional status, making causal interpretation challenging.[17, 34, 35, 39] The role of trace elements should therefore be considered within the broader context of mineral homeostasis and environmental exposure. Current evidence supports their biological relevance but does not provide sufficient evidence for routine supplementation as a caries-prevention strategy.

VII. CURRENT EVIDENCE, LIMITATIONS, AND FUTURE DIRECTIONS

Although micronutrients have biologically plausible roles in dental caries prevention, the overall evidence base remains heterogeneous. The strongest evidence concerns the fundamental roles of calcium, phosphorus, and vitamin D in mineral homeostasis and dental tissue development, but the strength of evidence for clinical caries prevention varies considerably among individual nutrients.[13, 18, 29]

One of the major challenges is the substantial variation in study design, nutritional assessment, diagnostic criteria, and caries outcomes. These methodological differences make direct comparison between studies difficult and may contribute to inconsistent findings, particularly for vitamin D.[18, 20, 28, 40] In addition, many studies rely on observational data, in which nutritional status may be closely associated with socioeconomic circumstances, dietary patterns, general health, and access to dental care.[39] Another limitation is the predominance of research in children. Although childhood represents an important period for tooth development and caries prevention, evidence among adults and other vulnerable populations remains less comprehensive.[29] Nutritional requirements and the mechanisms linking micronutrient status to caries may also differ across the life course.

Future research should prioritize well-designed longitudinal and randomized controlled trials with standardized definitions of nutritional status and dental caries outcomes.[18, 41] Dietary assessment should ideally be combined with biochemical measurements to distinguish reported intake from actual physiological status.[42, 43] Adequate sample sizes, appropriate control groups, and longer follow-up periods are also necessary to determine whether correction of micronutrient deficiencies translates into clinically meaningful reductions in caries incidence.

An additional priority is the investigation of interactions among micronutrients. Most existing studies focus on individual nutrients, whereas mineralization and immune function depend on interconnected nutritional pathways.[30] Calcium, phosphorus, vitamin D, magnesium, and trace elements may therefore exert effects that cannot be fully understood when considered independently. Genetic and molecular factors may further explain differences in individual susceptibility. Genetic variation affecting calcium and phosphorus homeostasis, enamel formation, and mineralization-regulating pathways may influence caries risk.[44-47] Integration of these factors with nutritional and metabolic assessments could contribute to more individualized prevention strategies.

Emerging technologies may facilitate this transition. Salivary biomarkers, proteomic analyses, microbiome profiling, and artificial intelligence-based diagnostic approaches may improve early identification of individuals at elevated caries risk.[48-50] Although these technologies are not micronutrients themselves, their integration with nutritional assessment could provide a more comprehensive understanding of how micronutrient status interacts with biological susceptibility.

Importantly, nutritional strategies should remain complementary to established preventive measures. Fluoride continues to have the

strongest evidence base for caries prevention through its ability to promote remineralization and inhibit demineralization.[51-53] Ensuring adequate micronutrient status should therefore be viewed as part of comprehensive oral and systemic health rather than as a substitute for fluoride-based prevention.

VIII. CONCLUSION

Micronutrients may contribute to dental caries prevention through their roles in dental tissue development, mineral homeostasis, remineralization, and host defense. Vitamin D has received the greatest attention, although evidence regarding its protective effect remains inconsistent, while calcium and phosphorus have important roles in maintaining dental mineralization. Zinc, magnesium, vitamins A, E, and K, and other trace elements may also contribute to oral health, but current evidence remains more limited. Overall, adequate micronutrient status should be considered an important component of comprehensive oral health and caries prevention, rather than a replacement for established preventive measures. Further well-designed clinical studies are needed to clarify the independent and combined effects of micronutrients and to identify populations that may benefit most from targeted nutritional interventions.

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X. DISCLOSURE

The author reports no conflicts of interest in this work.

REFERENCES

1. Bernabe E, Marcenes W, Hernandez CR, Bailey J, Abreu LG, Alipour V, et al. Global, Regional, and National Levels and Trends in Burden of Oral Conditions from 1990 to 2017: A Systematic Analysis for the Global Burden of Disease 2017 Study. J Dent Res. 2020;99(4):362-73. doi: 10.1177/0022034520908533.
2. Yadav A, Rajendra S, Seemala J, Vajrala D. Effect of Dental Caries on Oral Health-Related Quality of Life Among Young Adults: A Systematic Review. Cureus. 2026;18(3):e104744. doi: 10.7759/cureus.104744.
3. Chen X, Jia L, Wang Q, Wang JJ, Tian Y, Zhang Z, et al. Global, regional, and national burden of caries in primary teeth from 1990 to 2021: results from the global burden of disease study 2021. BMC Oral Health. 2025;25(1):1381. doi: 10.1186/s12903-025-06691-y.
4. Dipalma G, Inchingolo AM, Fiore A, Chieppa S, Carone C, Tartaglia FC, et al. Role of nutrition in prevention of dental caries in children and adolescents: a systematic review. BMC Oral Health. 2026;26(1):495. doi: 10.1186/s12903-026-07839-0.
5. Machiulskiene V, Campus G, Carvalho JC, Dige I, Ekstrand KR, Jablonski-Momeni A, et al. Terminology of Dental Caries and Dental Caries Management: Consensus Report of a Workshop Organized by ORCA and Cariology Research Group of IADR. Caries Res. 2020;54(1):7-14. doi: 10.1159/000503309.
6. Sheetal A, Hiremath VK, Patil AG, Sajjansetty S, Kumar SR. Malnutrition and its oral outcome - a review. Journal of clinical and diagnostic research : JCDR. 2013;7(1):178-80. doi: 10.7860/jcdr/2012/5104.2702.
7. Sudjatmoko AR, Santoso S, Yanuaryska RD. Effect of nutritional status on dental maturation and mandibular bone density among Indonesian children aged 6-9 Years in Yogyakarta. Journal of oral biology and craniofacial research. 2025;15(2):428-32. doi: 10.1016/j.jobcr.2025.02.010.
8. Zhang JS, Chu CH, Yu OY. Oral Microbiome and Dental Caries Development. Dent J (Basel). 2022;10(10). doi: 10.3390/dj10100184.
9. Güler Y, Derelioğlu SŞ, Yılmaz S. Caries Risk Assessment in Children with Different Rates of Vitamin D Deficiency, Using Cariogram Model. International Journal of Child Health and Nutrition. 2023;12(1):23-31. doi: 10.6000/1929-4247.2023.12.01.3.
10. Pu R, Fu M, Li N, Jiang Z. A certain protective effect of vitamin D against dental caries in US children and youth: A cross-sectional study. Journal of Public Health Dentistry. 2023;83(3):231-8. doi: 10.1111/jphd.12571.
11. Schwalfenberg GK. A review of the critical role of vitamin D in the functioning of the immune system and the clinical implications of vitamin D deficiency. Molecular Nutrition and Food Research. 2011;55(1):96-108. doi: 10.1002/mnfr.201000174.
12. Doncheva N, Mihaylova A, Zlatanova H, Ivanovska M, Delev D, Kostadinov I. Immunomodulatory properties of cholecalciferol in rats with experimentally induced inflammation. Pharmacia. 2024;71:1-9. doi: 10.3897/PHARMACIA.71.E122410.
13. Bergel E, Gibbons L, Rasines MG, Luetich A, Belizán JM. Maternal calcium supplementation during pregnancy and dental caries of children at 12 years of age: Follow-up of a randomized controlled trial. Acta Obstetricia et Gynecologica Scandinavica. 2010;89(11):1396-402. doi: 10.3109/00016349.2010.518228.

14. Reynolds EC, Black CL, Cai F, Cross KJ, Eakins D, Huq NL, et al. Advances in Enamel Remineralization: Casein Phosphopeptide - Amorphous Calcium Phosphate. Journal of Clinical Dentistry. 1999;10(2):86-8.
15. Rahman MT, Hossain A, Pin CH, Yahya NA. Zinc and Metallothionein in the Development and Progression of Dental Caries. Biological Trace Element Research. 2019;187(1):51-8. doi: 10.1007/s12011-018-1369-z.
16. Su S, Yi L, Fu Z, Zou Z. Bifunctional effect and mechanism of trace zinc on amorphous calcium phosphate crystallization. Journal of Colloid and Interface Science. 2026;721. doi: 10.1016/j.jcis.2026.140708.
17. Curzon MEJ. Combined Effect of Trace Elements and Fluoride on Caries: Changes Over Ten Years in Northwest Ohio (U.S.A.). Journal of Dental Research. 1983;62(2):96-9. doi: 10.1177/00220345830620022001.
18. Hujoel PP. Vitamin D and dental caries in controlled clinical trials: Systematic review and meta-analysis. Nutrition Reviews. 2013;71(2):88-97. doi: 10.1111/j.1753-4887.2012.00544.x.
19. Liu D, Meng X, Tian Q, Cao W, Fan X, Wu L, et al. Vitamin D and Multiple Health Outcomes: An Umbrella Review of Observational Studies, Randomized Controlled Trials, and Mendelian Randomization Studies. Advances in Nutrition. 2022;13(4):1044-62. doi: 10.1093/advances/nmab142.
20. Haworth S, Dodhia SA, West NX, Thomas SJ, Timpson NJ, Johansson I, et al. Examining the causal association between 25-hydroxyvitamin D and caries in children and adults: A two-sample Mendelian randomization approach. Wellcome Open Research. 2021;5. doi: 10.12688/wellcomeopenres.16369.2.
21. Grant WB. A review of the role of solar ultraviolet-B irradiance and vitamin D in reducing risk of dental caries. Dermato-Endocrinology. 2011;3(3):193-8. doi: 10.4161/derm.15841.
22. Bahardoust M, Salari S, Ghotbi N, Rahimpour E, Haghmoradi M, Alipour H, et al. Association between prenatal vitamin D deficiency with dental caries in infants and children: a systematic review and meta-analysis. BMC Pregnancy and Childbirth. 2024;24(1). doi: 10.1186/s12884-024-06477-0.
23. Al-Ghurabi BH, Aldhaher ZA, Kareem MWA, Abd ST, Abdullah WL, Abbas AA, et al. Effect of vitamin D on salivary cathelicidin in kindergarten children with dental caries. Journal of Emergency Medicine, Trauma and Acute Care. 2 ed2024.
24. Diah D, Prahasanti C, Rahayu RP. Bioinformatic Analysis of Vitamin D as a Pro-Inflammatory Cytokine Inhibitor in Periodontal Disease: An in-Silico Study. Journal of International Dental and Medical Research. 2024;17(2):623-7.
25. Ji S, Zhao K, Ma L, Chen X, Zheng D, Lu Y. The Association Between Vitamin D and Early Childhood Caries: A Systematic Review and Meta-Analysis. Oral Health and Preventive Dentistry. 2024;22:63-72. doi: 10.3290/j.ohpd.b4928565.
26. Hu Z, Zhou F, Xu H. Circulating vitamin C and D concentrations and risk of dental caries and periodontitis: A Mendelian randomization study. Journal of Clinical Periodontology. 2022;49(4):335-44. doi: 10.1111/jcpe.13598.
27. Navarro CLA, Grgic O, Trajanoska K, Van Der Tas JT, Rivadeneira F, Wolvius EB, et al. Associations between Prenatal, Perinatal, and Early Childhood Vitamin D Status and Risk of Dental Caries at 6 Years. Journal of Nutrition. 2021;151(7):1993-2000. doi: 10.1093/jn/nxab075.
28. Li Z, Wei X, Shao Z, Liu H, Bai S. Correlation between vitamin D levels in serum and the risk of dental caries in children: a systematic review and meta-analysis. BMC Oral Health. 2023;23(1). doi: 10.1186/s12903-023-03422-z.
29. Khan D, Hernandez-Castro I, Larvie DY, Armah SM, Cardenas A, Malin AJ. Higher fat-soluble vitamin and phosphorus intake are associated with less dental caries among children and adolescents in the United States, NHANES 2011–2018. Frontiers in Oral Health. 2025;6. doi: 10.3389/froh.2025.1617695.
30. Malin AJ, Wang Z, Khan D, McKune SL. The Potential Systemic Role of Diet in Dental Caries Development and Arrest: A Narrative Review. Nutrients. 2024;16(10). doi: 10.3390/nu16101463.
31. Aimutis WR. Bioactive Properties of Milk Proteins with Particular Focus on Anticariogenesis. Journal of Nutrition. 4 ed2004. p. 989S-95S.
32. Reynolds EC. Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: A review. Special Care in Dentistry. 1998;18(1):8-16. doi: 10.1111/j.1754-4505.1998.tb01353.x.
33. Li B, Zhong C, Yang H, Yu J. Advancing dentin biomimetic mineralization: From non-collagenous proteins to multifunctional material design. Acta Biomaterialia. 2026;217:21-43. doi: 10.1016/j.actbio.2026.05.008.
34. Riyat M, Sharma DC. Analysis of 35 inorganic elements in teeth in relation to caries formation. Biological Trace Element Research. 2009;129(1-3):126-9. doi: 10.1007/s12011-008-8305-6.
35. Bowen WH. Exposure to metal ions and susceptibility to dental caries. Journal of Dental Education. 2001;65(10):1046-53.
36. Chen Y, Yang X, Zhang S, Wang H, Xie H, Chen C. Polyacrylic acid/citrate/amorphous calcium phosphate complex for dentin remineralization and bond durability. Dental Materials. 2026;42(1):126-37. doi: 10.1016/j.dental.2025.09.016.
37. Shen D, Zhu H, Xu Y, Zhou Z, Wu Z, Fu B. In-depth Occlusion of Dentinal Tubules Induced by Polyaspartic Acid–Calcium and Magnesium Complexes. Journal of Dental Research. 2025;104(6):637-44. doi: 10.1177/00220345241309340.
38. Zhou Z, Li J, Wang Z, Zhang H, Wang Y, Shen D, et al. In-Depth Occlusion of Dentinal Tubules and Rapid

Remineralization of Demineralized Dentin Induced by Polyelectrolyte-Calcium Complexes. Advanced Healthcare Materials. 2023;12(19). doi: 10.1002/adhm.202300100.

39. Cruz Palma G, Rosa Isela Sánchez Najera C, Miguel Ángel Quiroga García C, Carlos Galindo Lartigue C, MartíNez GonzáLez GI. Dental caries and social determinants of health in Mexico. Revista Cubana de Estomatologia. 2014;51(1):55-70.
40. Kühnisch J, Janjic Rankovic M, Kapor S, Schüler I, Krause F, Michou S, et al. Identifying and avoiding risk of bias in caries diagnostic studies. Journal of Clinical Medicine. 2021;10(15). doi: 10.3390/jcm10153223.
41. Mheissen S, Khan H, Seehra J, Pandis N. Are longitudinal randomised controlled oral health trials properly analysed? A meta-epidemiological study. Journal of Dentistry. 2022;124. doi: 10.1016/j.jdent.2022.104182.
42. Chi DL, Hopkins S, O'Brien D, Mancl L, Orr E, Lenaker D. Association between added sugar intake and dental caries in Yup'ik children using a novel hair biomarker. BMC Oral Health. 2015;15(1). doi: 10.1186/s12903-015-0101-z.
43. Yang X, Zhao T, Yu P, Cai D, Liao R, Cai Y, et al. Deep quantitative proteomic analysis of gingival crevicular fluid for severe pediatric caries. Analytica Chimica Acta. 2026;1407. doi: 10.1016/j.aca.2026.345531.
44. Root AW. Genetic disorders of calcium, phosphorus, and bone homeostasis. Metabolic Diseases: Foundations of Clinical Management, Genetics, and Pathology. 2017. p. 813-46.
45. Sokolova II, Yaroshenko OH, Herman SI, Tomilina TV, Skydan KV, Skydan MI. FEATURES OF DENTAL STATUS AND METABOLISM IN CHILDREN WITH EARLY CHILDHOOD CARIES AGAINST THE BACKGROUND OF CONNECTIVE TISSUE DYSPLASIA. Wiadomosci lekarskie (Warsaw, Poland : 1960). 2021;74(10):2503-9. doi: 10.36740/wlek202110125.
46. Socorro M, Hoskere P, Roberts C, Lukashova L, Verdelis K, Beniash E, et al. Deficiency of Mineralization-Regulating Transcription Factor Trps1 Compromises Quality of Dental Tissues and Increases Susceptibility to Dental Caries. Frontiers in Dental Medicine. 2022;3. doi: 10.3389/fdmed.2022.875987.
47. Cavallari T, Arima LY, Ferrasa A, Moysés SJ, Tetu Moysés S, Hirochi Herai R, et al. Dental caries: Genetic and protein interactions. Archives of Oral Biology. 2019;108. doi: 10.1016/j.archoralbio.2019.104522.
48. Amaechi BT. Emerging technologies for diagnosis of dental caries: The road so far. Journal of Applied Physics. 2009;105(10). doi: 10.1063/1.3116632.
49. Khuwaja H, Khidri AA, Jaffari R, Javed S. AI-Powered Dental Caries Detection. Proceedings of the IEEE International Multi Topic Conference, INMIC. 2024 ed2024.
50. Salah AN, Elesawy AE, Zaki MB, Abulsoud AI, El-Sayyad GS, Elkalla WS, et al. Pharmaceutical Innovation in Salivary Biomarker Research: Transforming Oral Healthcare through Nanotechnology and Point-of-Care Systems: A Narrative Review. Journal of Pharmaceutical Innovation. 2026;21(6). doi: 10.1007/s12247-026-10857-8.
51. Vitoria Miñana I, Pericas Bosch J, Sánchez Ruiz-Cabello FJ, Soriano Faura FJ, Colomer Revuelta J, Cortés Rico O, et al. Dental health promotion. Pediatria de Atencion Primaria. 2011;13(51):435-58. doi: 10.4321/s1139-76322011000300010.
52. Yin W, Zhou Z, Huang RZ, Sun G, Zhong Y, Yang Z, et al. Arginine Dentifrices and Childhood Caries Prevention: A Randomized Clinical Trial. JDR Clinical and Translational Research. 2026;11(2):204-14. doi: 10.1177/23800844251361471.
53. Wierichs RJ, Meyer-Lueckel H. Systematic review on noninvasive treatment of root caries lesions. Journal of Dental Research. 2015;94(2):261-71. doi: 10.1177/0022034514557330.