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When the Tooth Is No Longer Enough: A Question-Driven Journey from Dental Caries to Complex Networks

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ABSTRACT

Traditionally, dental research has framed its questions around units of observation such as lesions, teeth, or specific oral diseases. However, empirical findings may reveal the limitations of these units, requiring broader explanatory lenses. This critical review reconstructs a scientific trajectory organized around eight research questions. The findings shifted the unit of inquiry from the caries lesion to the disease process and to individual, social, and behavioral contexts, oral disease burden, syndemic frameworks, and complex disease networks. Evidence that carious dentin could be retained without compromising restoration performance challenged lesion-centered intervention, while differences in fluoride effectiveness and caries polarization revealed the importance of social and behavioral conditions. Caries and periodontal indicators subsequently converged into the *Chronic Oral Disease Burden* construct across life stages. Associations with inflammatory, nutritional, metabolic, mental, and vascular conditions then extended the frontiers beyond the mouth. Throughout this trajectory, sugars remained a thread, shifting from a major causal factor for dental caries to a shared behavioral risk factor across oral and systemic conditions. Within the complex NCD network, dental caries emerged as a hub across the life course; when we incorporated its determinants, socioeconomic disadvantage, high free sugar consumption, and insulin resistance emerged as key connecting nodes. Because caries is among the earliest NCDs, these findings raise a question requiring prospective investigation: *Could early childhood caries serve as a sentinel marker of broader NCD trajectories across the life course?* The disease remains the same; what changes is the question-driven lens through which we examine it.

Keywords: Dental Caries; Periodontal Diseases; Noncommunicable Diseases; Dietary Sugars; Life Course Perspective.

INTRODUCTION

Traditionally, dental research has focused on units of observation such as lesions, tooth surfaces, teeth, or specific oral diseases. However, empirical findings may reveal the limitations of these isolated units in explaining the complexity of oral diseases and their place within the landscape of noncommunicable diseases, necessitating a broader, interdisciplinary explanatory lens.

In this manuscript, we describe a scientific trajectory organized around eight questions that collectively form a research framework. Each question emerged from limitations revealed by previous findings and progressively broadened the explanatory lens: from conservative removal of carious dentin to control of the caries process, from individual oral diseases to the proposition of the *Chronic Oral Disease Burden* construct, and from the mouth to syndemic frameworks and, ultimately, a complex network approach examining how different NCDs

interconnect. Throughout this journey, sugar remains a recurring explanatory thread, shifting from a major causal factor for dental caries to a shared behavioral risk factor across oral and systemic conditions.

METHODOLOGICAL APPROACH

This critical review provides an interpretive reconstruction of a question-driven scientific trajectory. Studies were selected based on their contribution to eight successive research questions that progressively broadened the unit of inquiry, from the caries lesion to individual, social, and behavioral contexts; oral disease burden; syndemic frameworks; and, ultimately, complex disease networks. Policy documents were included when they translated findings into clinical or public health guidance, while additional publications supported the theoretical interpretation developed in this review (Table 1). Study selection was purposive rather than exhaustive, and no systematic literature search was undertaken. The review is intended to interpret the development of this research framework rather than provide a comprehensive synthesis of all evidence in the field.

RESULTS

From the lesion to the disease process

Question 1: Can a caries lesion be arrested without the complete removal of carious tissue?

The journey began with a clinical question centered on the caries lesion: how much carious tissue must be removed? Our early study was among the first to challenge the long-standing principle that carious tissue had to be completely removed for restoration success. It showed that carious dentin could be retained without compromising clinical performance or radiographic outcomes. Beyond these outcomes, scanning electron microscopy showed the integrity of the hybrid layer after one year, even when the adhesive interface was formed over carious dentin in primary teeth (1).

A subsequent randomized clinical trial by Lula et al. showed that residual bacterial colonization after selective carious tissue removal was similar to that observed after complete carious tissue removal, suggesting that bacterial persistence alone was not a reason to reopen the cavity (2). Although softer and wetter dentin was initially associated with greater bacterial contamination during carious tissue removal, its clinical characteristics, including color, consistency, and moisture, changed substantially after sealing. Together with reduced bacterial counts, these changes indicated caries lesion arrest (3). Nearly three decades after the earliest studies, these findings continue to inform the principles of Minimal Intervention Dentistry and are cited in clinical practice guidelines issued by the Brazilian Ministry of Health (4) and the American Dental Association (5).

Lesion arrest does not necessarily imply disease control. Systematic reviews showed that caries remained a major reason for restoration failure and that patient-related factors, particularly caries risk, influenced restoration longevity (6,7). Thus, these findings exposed the limitation of the lesion as the unit of intervention: a lesion could be arrested despite retained carious dentin, but lesion control alone was insufficient to control the disease process.

From the disease process to the individual

Question 2: Does the same fluoride dose work for every child?

Once caries control was understood as extending beyond managing individual lesions, attention shifted to whether preventive interventions worked equally for every child. A randomized clinical trial comparing dentifrices containing 500 and 1,100 ppm fluoride showed that their effects differed by baseline caries experience. Children with active caries benefited more from the conventional fluoride concentration, whereas this difference was not observed in caries-free children (8). These findings exposed the limitations of low-fluoride dentifrices as a population-wide strategy: the lower concentration did not provide the same caries control for children with active disease, the group most in need of protection. Thus, the effect of fluoride concentration depended on who received it. The study was later cited in a report by the Irish Department of Health evaluating fluoride toothpaste use in children under six, underscoring its relevance to public health guidance (9).

From individual differences to social and behavioral contexts

Question 3: Why does early childhood caries affect children so unequally?

The finding that preventive interventions did not work equally for every child raised a broader question about the unequal distribution of early childhood caries. In a low-income population, caries was strongly polarized: most children had few or no lesions, whereas a small subgroup carried a substantially greater disease burden (10). Although these children shared broadly similar conditions of social vulnerability and access to health care, these common conditions alone did not fully account for the observed polarization. Frequent sucrose consumption was associated with higher caries experience, suggesting that differences in sugar exposure helped distinguish children with a greater disease burden even within the same socially vulnerable population (10).

From social and behavioral contexts to co-occurring conditions

Question 4: What else accompanies early childhood caries?

Frequent sugar consumption was associated with the polarization of early childhood caries, even among children living under disadvantaged conditions (10). A subsequent study broadened the explanatory framework by identifying undernutrition, overweight/obesity, and frequent sugar consumption as risk indicators for early childhood caries (11). These findings placed sugar exposure, dental caries, and contrasting nutritional conditions within the same empirical picture. Moreover, children with early childhood caries exhibited elevated inflammatory biomarkers in saliva and serum (12,13). Thus, a condition clinically expressed in the teeth co-occurred with contrasting nutritional states and inflammatory alterations at both local and systemic levels.

From a caries-related exposure to a shared oral risk factor

Question 5: Does the association of sugar end with dental caries?

Sugar had repeatedly appeared in relation to caries. The next step was to ask whether its association extended to the periodontium. Lula et al. provided early evidence that, in a population-based sample of American young adults, more frequent added-sugar consumption was associated with greater prevalence and extent of periodontal disease, independent of traditional risk factors (14). This association was observed when the disease affected multiple teeth, but not when it was limited to a single tooth, suggesting a relationship beyond a purely local effect. Our findings were subsequently cited in a World Health Organization policy document addressing oral diseases as part of essential noncommunicable disease interventions in Africa (15).

We later examined the question across different populations and life stages. Menezes et al. showed that higher soft-drink consumption was associated with more teeth with probing depth ≥ 4 mm with bleeding on probing among pregnant women (16). In Brazilian adolescents, Moreira et al. showed that added sugar intake above 10% of daily energy was associated with a greater extent of periodontal disease (17). Together, these findings showed that the association between sugar exposure and periodontal alterations was observed across populations and life stages.

The explanatory question then moved from sugar as an isolated exposure to dietary patterns. An unhealthy dietary pattern, including sugars, snacks, and salty/fast foods, was associated with greater periodontitis extent and severity, whereas a healthy dietary pattern was associated with better periodontal conditions. Biofilm mediated part of the association between unhealthy diet and periodontal outcomes, while the study also supported systemic pathways linking diet to the periodontium (18). Thus, the explanatory lens expanded from viewing sugars as a caries-related exposure to understanding sugars and dietary patterns as exposures associated with periodontal disease through local and systemic pathways.

From disease-specific outcomes to integrated oral health

Question 6: If dental caries and periodontal disease share common determinants, why study them separately?

We first addressed this question epidemiologically. Rather than treating dental caries and periodontal disease as separate outcomes, Carmo et al. investigated whether their shared variation could be represented by a common latent construct (19). The *Chronic Oral Disease Burden* was proposed to capture the common variance across caries and periodontal indicators, providing an integrated epidemiological representation of oral disease burden (19). The Swedish National Board of Health and Welfare later cited the study in the evidence base supporting Sweden's National Guidelines for Dental Care (20).

Using the same integrated construct, a subsequent study by Ladeira et al. found that sugar intake above international recommendations was associated with a greater *Chronic Oral Disease Burden* in adolescents (21). Notably, associations with individual caries and periodontal indicators were observed even at half the intake limits recommended by international organizations.

The construct was then tested in an American population aged 13 to 80 years. Costa et al. showed that caries and periodontitis indicators consistently converged into a common *Chronic Oral Disease Burden* across age groups, supporting the construct's consistency from adolescence to older adulthood (22).

The construct was later extended to early childhood by incorporating gingivitis as the periodontal manifestation alongside dental caries (23). Araújo et al. examined this early burden in relation to added sugar consumption and obesity within mother-child dyads (23). In the BRISA cohort, the shared variance of visible plaque, gingivitis, and dental caries was already identifiable as a common oral disease burden within the first 1,000 days. Maternal and childhood sugar consumption, together with obesity, were linked to this burden through intergenerational risk pathways (24). Together, these findings shifted the unit of inquiry from individual oral diseases to the burden they share.

From the Chronic Oral Disease Burden to a syndemic framework

Question 7: If oral diseases share determinants with systemic conditions, why should the mouth remain the boundary of explanation?

Integrating caries and periodontal disease into a common oral disease burden raised a broader question about their relationships with conditions beyond the mouth. A connection emerged between periodontal and skeletal health. By the end of the second decade of life, near peak bone mass, low bone mineral density was associated with severe periodontitis and greater periodontal disease extent (25). This finding showed that relationships between periodontal and systemic conditions were already observable in adolescence.

The shared-risk perspective was subsequently extended beyond sugars. Monteiro et al. showed that an unhealthy dietary pattern was associated with caries both directly and indirectly through dental biofilm, whereas addictive behaviors, comprising smoking and risk of alcohol dependence, were associated with caries indirectly through biofilm. Unhealthy diet and addictive behaviors were also interrelated (26). These findings positioned caries within a broader behavioral risk context shared with other NCDs.

The lens then expanded from shared risk factors to co-occurring diseases. Ladeira et al. investigated caries, periodontitis, obesity, and insulin resistance as co-occurring rather than isolated conditions, examining their relationships within a shared social and behavioral context (27). The findings showed that oral and metabolic conditions co-occurred and shared determinants, supporting their interpretation within a syndemic framework.

The explanatory framework next incorporated mental health. Barbosa et al. showed that, in adolescents, substance use behaviors and added sugar consumption were associated with both depression and suicide risk, suggesting that behavioral risks shared with NCDs also intersected with mental health (28). Building on this connection, Nogueira Filho et al. investigated dental caries and depression as correlated conditions shaped by common determinants (29). Lower socioeconomic status was associated with higher sugar consumption and depression, while sugar consumption was directly associated with both caries experience and depression. Caries and depression were also positively correlated. Thus, sugar, initially investigated as a caries-related exposure, emerged as a shared behavioral factor connecting oral and mental health within a broader context of social vulnerability.

The syndemic perspective extended beyond questions originating in oral health. Falcão et al. examined socioeconomic status, insulin resistance, asthma, and depression in adolescents without including an oral condition (30). Lower socioeconomic status was associated with higher insulin resistance and depression; higher insulin resistance was associated with asthma, which in turn was associated with depression. These findings extended the explanatory framework beyond the mouth, linking social disadvantage with metabolic, respiratory, and mental health conditions.

The temporal dimension of this framework became clearer when Barbosa et al. showed that the *Insulin Resistance Phenotype* was already associated with the *Vascular Risk Phenotype* at the end of the second decade of life. Lower socioeconomic status, harmful alcohol use, and higher body fat were also associated with both phenotypes (31). These interconnected phenotypes may represent early events along the continuums of type 2 diabetes and cardiovascular disease, long before these conditions typically become clinically manifest. Oral health re-entered this expanded framework, where periodontitis was correlated with the *Vascular Risk Phenotype* in late adolescence, and both conditions shared socioeconomic and metabolic determinants (32).

Altogether, these findings raised a broader question: if oral, mental, metabolic, and vascular processes are already interconnected by adolescence, how are multiple NCDs and their determinants organized within a complex system?

From a syndemic approach to complex disease networks

Question 8: If noncommunicable diseases are interconnected, how are they organized within a complex disease network?

While the syndemic approach allowed us to investigate disease co-occurrence within a context of shared social and behavioral risk factors, the next question required a different explanatory perspective. Complex network analysis allowed us to examine multiple NCDs simultaneously, revealing how they were interconnected and which diseases occupied the most central positions. Using this approach, Alves-Costa et al. found that dental caries emerged as a hub connecting oral and systemic NCDs across the life course. Caries formed a triad with asthma and obesity from early childhood onward, while remaining connected with other NCDs at later life stages (33).

Our earlier findings helped contextualize some of these network connections, showing that sugar-related exposures were associated not only with dental caries but also with asthma (34,35) and allergic conditions (36) early in life. Together, these findings raised another question: why was dental caries so central to the NCD network? To explore this question, we expanded the network beyond the diseases themselves to include their social, behavioral, and metabolic determinants in a population-based sample of adolescents. In this expanded network, free sugar consumption, insulin resistance, and socioeconomic disadvantage emerged as central elements connecting multiple NCDs (37).

Taken together, these findings repositioned dental caries from an isolated oral outcome to a central condition within a network of interconnected disease processes.

DISCUSSION AND FUTURE DIRECTIONS

The next step is to consider how and why these diseases emerge and become interconnected across the life course.

NCDs, including oral diseases, evolve along *continuums* of pathophysiological processes that are dynamic and non-deterministic, and may become clinically manifest across the life course. Depending on individual susceptibility, risk exposures, protective factors, disease-control interventions, and the stage of tissue damage, these processes may progress, stabilize, regress, or be interrupted. This continuum perspective has been applied to cardiovascular disease (38), type 2 diabetes (39), and dental caries (40). Periodontal disease has similarly been described through transitions from health to reversible gingivitis and, in susceptible individuals, to periodontitis with irreversible attachment loss (41). Figure 1 schematically illustrates these continuums and their potential overlap across the life course.

Our findings place oral diseases within the same inflammatory and cardiometabolic context as other NCDs. Early childhood caries was accompanied by elevated systemic inflammatory biomarkers (13), and both caries and periodontitis were associated with systemic inflammatory alterations in adolescence (42,43). During adolescence, oral diseases also co-occurred with obesity and insulin resistance (27), while periodontitis was correlated with a vascular risk phenotype (32).

Across their trajectories, some NCDs may precede others by decades. Dental caries, for instance, is among the earliest NCDs to become clinically detectable, often emerging in the first

years of life. The breakdown of mineralized dental tissues may therefore be detected one or two decades before clinically evident damage to the periodontal supporting tissues, which may already be present by the end of the second decade of life (25).

This temporal difference invites us to reconsider the role of dental caries across the life course, particularly in the current epidemiological context. After decades of stable or declining trends, the prevalence of early childhood caries has increased globally over the most recent decade (44). In Brazil, the prevalence of periodontitis has also increased since 2005, alongside increases in sugar-sweetened beverage consumption and high body mass index (45). Consistent with this pattern, sugar-sweetened beverage consumption has increased nationally and globally and is projected to rise further by 2050 (46).

Seen through this broader lens, caries represents more than an isolated oral disease. It shares behavioral determinants with other NCDs (26) and occupies a central position within complex disease networks (33). When we expanded the network to incorporate NCD determinants, its central architecture shifted: socioeconomic disadvantage, high sugar consumption, and insulin resistance emerged as key connecting nodes in adolescence (37).

CONCLUSION

The answers to the earliest questions in this scientific journey remain clinically relevant and provide support to guidelines and policies. Selective carious tissue removal is an established component of minimally invasive dentistry, helping to arrest lesion progression while reducing the risk of pulp exposure and necrosis. Likewise, twice-daily toothbrushing with fluoride toothpaste containing at least 1,000–1,100 ppm fluoride remains central to preventing caries and controlling mineral loss in individuals with active disease.

Other questions have gained attention more recently. Although caries and periodontal diseases involve distinct pathophysiological processes, they share social, behavioral, and dietary determinants, as well as dental biofilm, contributing to a chronic oral disease burden from early childhood through later life. A recent consensus on gingival and periodontal diseases in children and adolescents identified mechanical biofilm control as the foundation for preventing and controlling gingival inflammation. It also recognized that frequent consumption of simple sugars and refined carbohydrates is associated with greater biofilm accumulation, gingivitis, and dental caries, recommending reduced free sugar intake, a balanced diet, and twice-daily toothbrushing with fluoride toothpaste (47). Together, these measures provide a practical basis for the coordinated prevention of caries and gingival inflammation.

Broader explanatory lenses also generate new questions. Can interventions confined to the oral cavity sustainably control caries and gingival inflammation while children and adolescents remain exposed to the double burden of undernutrition and overweight/obesity, systemic inflammation, and co-occurring conditions? Investigating this question is particularly challenging because NCDs are connected through complex, nonlinear relationships and evolve along continuums in which early events may co-occur and interact long before clinical diagnoses are established.

The early emergence of dental caries may provide an opportunity to identify broader NCD trajectories while they are still developing and potentially modifiable. Prospective research should now determine whether early childhood caries can serve as a sentinel marker

of interconnected NCD trajectories and whether acting on their shared determinants can alter their course across the life course. The disease is the same. What has changed throughout this question-driven scientific journey is not only the lens through which we examine it, but also what its early manifestation may allow us to anticipate and prevent.

Effective health promotion may therefore require both coordinated clinical care and upstream policies that act on the contexts in which oral and systemic diseases jointly emerge. This requires confronting the social and commercial determinants that shape the production, marketing, and consumption of sugar-rich ultra-processed foods.

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REFERENCES

1. Ribeiro CCC, Baratieri LN, Perdigão J, Baratieri NMM, Ritter AV. A clinical, radiographic and scanning electron microscopic evaluation of adhesive restorations on carious dentin in primary teeth. *Quintessence Int.* 1999;30(9):591–599.
2. Lula ECO, Monteiro-Neto V, Alves CMC, Ribeiro CCC. Microbiological analysis after complete or partial removal of carious dentin in primary teeth: a randomized clinical trial. *Caries Res.* 2009;43:354-358. doi:10.1159/000231572.
3. Lula ECO, Almeida Jr LJS, Alves CMC, Monteiro-Neto V, Ribeiro CCC. Partial caries removal in primary teeth: association of clinical parameters with microbiological status. *Caries Res.* 2011;45:275-280. doi:10.1159/000325854.
4. Brasil. Ministério da Saúde. Secretaria de Atenção Primária à Saúde. Departamento de Estratégias e Políticas de Saúde Comunitária. Diretriz para a prática clínica odontológica na Atenção Primária à Saúde: manejo de lesões profundas de cárie. Brasília, DF: Ministério da Saúde; 2024. 57 p.
5. Dhar V, Pilcher L, Fontana M, González-Cabezas C, Keels MA, Mascarenhas AK, et al. Evidence-based clinical practice guideline on restorative treatments for caries lesions: a report from the American Dental Association. *J Am Dent Assoc.* 2023;154(7):551-566.e51. doi:10.1016/j.adaj.2023.04.011.
6. Maciel CM, Baroudi K, Costa LDC, Souto TCV, Vitti RP. Longevity of resin composite and amalgam posterior restorations: a systematic review. *Eur J Prosthodont Restor Dent.* 2022;30(4):267-275. doi:10.1922/EJPRD_2371Maciel09.
7. Opdam NJM, van de Sande FH, Bronkhorst E, Cenci MS, Bottenberg P, Pallesen U, Gaengler P, Lindberg A, Huysmans MCDNJM, van Dijken JW. Longevity of posterior composite

- restorations: a systematic review and meta-analysis. *J Dent Res.* 2014;93(10):943-949. doi:10.1177/0022034514544217.
8. Lima TJ, Ribeiro CC, Tenuta LM, Cury JA. Low-fluoride dentifrice and caries lesion control in children with different caries experience: a randomized clinical trial. *Caries Res.* 2008;42(1):46-50. doi:10.1159/000111749.
9. O'Mullane D, Clarkson J, Parnell C. *Report of the Working Group on the Use of Fluoride Toothpaste in Children Under Six Years of Age.* Dublin: Department of Health, Republic of Ireland; 2018. Available from: <https://www.gov.ie/en/department-of-health/policy-information/national-oral-health-policy/>
10. Nunes AMM, Silva AAM, Alves CMC, Hugo FN, Ribeiro CCC. Factors underlying the polarization of early childhood caries within a high-risk population. *BMC Public Health.* 2014;14:988. doi:10.1186/1471-2458-14-988.
11. Ribeiro CCC, Silva MCB, Nunes AMM, Thomaz EBAF, Carmo CDS, Ribeiro MRC, Silva AAM. Overweight, obese, underweight, and frequency of sugar consumption as risk indicators for early childhood caries in Brazilian preschool children. *Int J Paediatr Dent.* 2017;27(6):532-539. doi:10.1111/ipd.12292.
12. Ribeiro CCC, Pachêco CJB, Costa EL, Ladeira LLC, Costa JF, Silva RA, Carmo CDS. Proinflammatory cytokines in early childhood caries: salivary analysis in the mother/children pair. *Cytokine.* 2018;107:113-117. doi:10.1016/j.cyto.2017.12.009.
13. Lima GQT, Brondani MA, Silva AAM, Carmo CDS, Silva RA, Ribeiro CCC. Serum levels of proinflammatory cytokines are high in early childhood caries. *Cytokine.* 2018;111:490-495. doi:10.1016/j.cyto.2018.05.031.
14. Lula ECO, Ribeiro CCC, Hugo FN, Alves CMC, Silva AAM. Added sugars and periodontal disease in young adults: an analysis of NHANES III data. *Am J Clin Nutr.* 2014;100(4):1182-1187. doi:10.3945/ajcn.114.089656.
15. World Health Organization Regional Office for Africa. Promoting oral health in Africa: prevention and control of oral diseases and noma as part of essential noncommunicable disease interventions. Brazzaville: World Health Organization Regional Office for Africa; 2016.
16. Menezes CC, Ribeiro CCC, Alves CMC, Thomaz EBAF, Franco MM, Batista RFL, Silva AAM. Soft drink consumption and periodontal status in pregnant women. *J Periodontol.* 2019;90(2):159-166. doi:10.1002/JPER.16-0388.
17. Moreira ARO, Batista RFL, Ladeira LLC, Thomaz EBAF, Alves CMC, Saraiva MC, Silva AAM, Brondani MA, Ribeiro CCC. Higher sugar intake is associated with periodontal disease in adolescents. *Clin Oral Investig.* 2021;25(3):983-991. doi:10.1007/s00784-020-03387-1.
18. Costa SA, Nascimento GG, Colins PMG, Alves CMC, Thomaz EBAF, Souza SFC, Silva AAM, Ribeiro CCC. Investigating oral and systemic pathways between unhealthy and healthy dietary patterns to periodontitis in adolescents: a population-based study. *J Clin Periodontol.* 2022;49(6):580-590. doi:10.1111/jcpe.13625.

19. Carmo CDS, Ribeiro MRC, Teixeira JXP, Alves CMC, Franco MM, França AKTC, Benatti BB, Cunha-Cruz J, Ribeiro CCC. Added sugar consumption and chronic oral disease burden among adolescents in Brazil. *J Dent Res.* 2018;97(5):508-514. doi:10.1177/0022034517745326.
20. Socialstyrelsen. Bilaga: rekommendationer med tillhörande kunskapsunderlag: nationella riktlinjer för tandvård [Appendix: recommendations and supporting evidence for the national guidelines for dental care] [Internet]. Stockholm: Socialstyrelsen; 2022 [cited 2026 Sep 18]. Available from: <https://www.socialstyrelsen.se/kunskapsstod-och-regler/regler-och-riktlinjer/nationella-riktlinjer/riktlinjer-och-utvarderingar/tandvard/> Swedish.
21. Ladeira LLC, Nascimento GG, Leite FRM, Alves-Costa S, Thomaz EBAF, Alves CMC, Cury JA, Ribeiro CCC. Sugar intake above international recommendations and oral disease burden: a population-based study. *Oral Dis.* 2024;30(2):615-623. doi:10.1111/odi.14464.
22. Costa SA, Ribeiro CCC, Leite FRM, Peres MA, Souza SFC, Nascimento GG. Chronic oral diseases burden: the confluence of caries and periodontitis throughout life. *J Clin Periodontol.* 2023;50(4):452-462. doi:10.1111/jcpe.13761.
23. Araújo SMP, Silva GQTL, Costa EL, Nunes AMM, Ribeiro CCC. Pathways in the association between added sugar consumption, obesity in mother-child dyads, and chronic oral disease burden in early childhood. *Eur J Oral Sci.* 2022;130(2):e12847. doi:10.1111/eos.12847.
24. Araújo SMP, Nascimento GG, Ladeira LLC, Alves-Costa S, Saraiva MC, Alves CMC, Thomaz EBAF, Ribeiro CCC. Chronic oral disease burden at the first 1000 days: intergenerational risk factors, BRISA cohort. *Oral Dis.* 2024;30(8):5388-5396. doi:10.1111/odi.15010.
25. Costa SA, Ribeiro CCC, de Oliveira KR, Alves CMC, Thomaz EBAF, Casarin RCV, Souza SFC. Low bone mineral density is associated with severe periodontitis at the end of the second decade of life: a population-based study. *J Clin Periodontol.* 2021;48(8):1041-1050. doi:10.1111/jcpe.13525.
26. Monteiro ELO, Ladeira LLC, Costa CM, Monteiro MCC, Rebelo MAB, Alves CMC, Thomaz EBAF, Ribeiro CCC. Behavior risk factors for noncommunicable diseases and caries in adolescents: a population study. *Caries Res.* 2025;59(6):519-528. doi:10.1159/000544723.
27. Ladeira LLC, Nascimento GG, Leite FRM, Alves-Costa S, Barbosa JMA, Alves CMC, Thomaz EBAF, Batista RFL, Ribeiro CCC. Obesity, insulin resistance, caries, and periodontitis: syndemic framework. *Nutrients.* 2023;15(16):3512. doi:10.3390/nu15163512.
28. Barbosa JMA, Ribeiro CCC, Batista RFL, Brondani MA, Simões VMF, Bettiol H, Barbieri MA, Coelho SJDDAAC, Silva AAM. Behavioral risk factors for noncommunicable diseases associated with depression and suicide risk in adolescence. *Cad Saude Publica.* 2022;38(2):e00055621. doi:10.1590/0102-311X00055621.
29. Nogueira Filho R, Ladeira LLC, Oliveira ICV, Queiroz LJ, Santos CC, Alves CMC, Thomaz EBAF, Ribeiro CCC. Depression and caries in adolescents: role of social inequities and sugar consumption. *Clin Oral Investig.* 2026;30(2):66. doi:10.1007/s00784-025-06738-y.

30. Falcão AS, Ladeira LLC, Costa SA, Batista RFL, Alves CMC, Thomaz EBAF, Ribeiro CCC. Low socioeconomic status, insulin resistance, asthma, and depression: a syndemic framework in Brazilian adolescents. *Pediatr Allergy Immunol.* 2026;37(2):e70290. doi:10.1111/pai.70290.
31. Barbosa JMA, Silva AAM, Batista RFL, Salgado BJL, Nascimento JXPT, Simões VMF, Ribeiro MJS, Barbieri MA, Ferraro AA, Ribeiro CCC. Insulin resistance phenotype is associated with vascular risk phenotype at the end of the second decade of life: a population-based study. *Cardiovasc Diabetol.* 2022;21(1):284. doi:10.1186/s12933-022-01724-0.
32. Costa SA, Nascimento GG, Costa CM, Alves CMC, Thomaz EBAF, Ribeiro CCC. Periodontitis is associated with higher blood pressure and pulse wave velocity in late adolescence: a population-based study. *Oral Dis.* 2026;in press. doi:10.1111/odi.70492.
33. Alves-Costa S, Rodrigues FA, Ferraro AA, Nascimento GG, Leite FRM, Souza BF, Ribeiro CCC. Caries is the hub of a complex network of chronic diseases across the life decades. *J Dent Res.* 2025;104(8):881-889. doi:10.1177/00220345251317487.
34. Nascimento JXPT, Ribeiro CCC, Batista RFL, Alves MTSSB, Simões VMF, Padilha LL, Cardoso VC, Vianna EO, Bettiol H, Barbieri MA, Silva AAM. The first 1000 days of life factors associated with “childhood asthma symptoms”: BRISA cohort, Brazil. *Sci Rep.* 2017;7(1):16028. doi:10.1038/s41598-017-16295-4.
35. Padilha LL, Vianna EO, Vale ATM, Nascimento JXPT, Silva AAM, Ribeiro CCC. Pathways in the association between sugar sweetened beverages and child asthma traits in the 2nd year of life: findings from the BRISA cohort. *Pediatr Allergy Immunol.* 2020;31(5):480-488. doi:10.1111/pai.13243.
36. Amorim Muniz AKO, Vianna EO, Vale ATM, Nascimento JXPT, Silva AAM, Ribeiro CCC. Sugar-sweetened beverages and allergy traits at second year of life: BRISA cohort study. *Nutrients.* 2023;15(14):3218. doi:10.3390/nu15143218.
37. Alves-Costa S, Souza BF, Rodrigues FA, Ferraro AA, Nascimento GG, Leite FRM, Ladeira LLC, Batista RFL, Thomaz EBAF, Alves CMC, Ribeiro CCC. High free sugars, insulin resistance, and low socioeconomic indicators: the hubs in the complex network of noncommunicable diseases in adolescents. *Diabetol Metab Syndr.* 2024;16(1):235. doi:10.1186/s13098-024-01469-8.
38. Dzau V, Braunwald E. Resolved and unresolved issues in the prevention and treatment of coronary artery disease: a workshop consensus statement. *Am Heart J.* 1991;121(4 Pt 1):1244-1263. doi:10.1016/0002-8703(91)90694-D.
39. Ramlo-Halsted BA, Edelman SV. The natural history of type 2 diabetes: implications for clinical practice. *Prim Care.* 1999;26(4):771-789. doi:10.1016/S0095-4543(05)70130-5.
40. Featherstone JDB. The continuum of dental caries—evidence for a dynamic disease process. *J Dent Res.* 2004;83 Spec No C:C39-C42. doi:10.1177/154405910408301S08.
41. Chapple ILC, Mealey BL, Van Dyke TE, Bartold PM, Dommisch H, Eickholz P, et al. Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: consensus report of Workgroup 1 of the 2017 World Workshop on the

- Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45 Suppl 20:S68-S77. doi:10.1111/jcpe.12940.
42. Costa SA, Ribeiro CCC, Carneiro ALFC, Cintra LTA, Alves CMC, Thomaz EBAF, Souza SFC. Dental caries and systemic inflammation in adolescents: findings from a population-based study. *Caries Res*. 2026 Jan 29:1-8. doi:10.1159/000550646. Online ahead of print.
 43. Ribeiro CCC, Carmo CDS, Benatti BB, Casarin RVC, Alves CMC, Nascimento GG, Moreira ARO. Systemic circulating inflammatory burden and periodontitis in adolescents. *Clin Oral Investig*. 2021;25(10):5855-5865. doi:10.1007/s00784-021-03891-y.
 44. Costa CM, Alves-Costa S, Hugo FN, Paes AMA, Tabchoury CPM, Cury JA, Ribeiro CCC. Increasing global trends in early childhood caries prevalence over the last decade: Global Burden of Disease Study 2021. *Caries Res*. 2025 Oct 27:1–7. doi:10.1159/000549128. Online ahead of print.
 45. Alves-Costa S, Leite FRM, Ladeira LLC, Lima-Soares F, Paes AMA, Souza BF, Nascimento GG, Ribeiro CCC. Behavioral and metabolic risk factors associated with periodontitis in Brazil, 1990–2019: a multidimensional analysis for the Global Burden of Disease Study 2019. *Clin Oral Investig*. 2023;27(12):7909–7917. doi:10.1007/s00784-023-05384-6.
 46. Alves-Costa S, Nascimento GG, Souza BF, Hugo FN, Leite FRM, Ribeiro CCC. Global, regional, and national burden of high sugar-sweetened beverages consumption, 1990-2021, with projections up to 2050: a systematic analysis for the Global Burden of Disease Study 2021. *Am J Clin Nutr*. 2025;122(5):1413-1422. doi:10.1016/j.ajcnut.2025.07.033.
 47. Chapple I, Gizani S, Sanz M, Declerck D, Madianos P, Clerehugh V, et al. Gingival and periodontal diseases and conditions in children and adolescents: consensus report. *J Clin Periodontol*. 2026;53(7):1068-1099. doi:10.1111/jcpe.70033.

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CONFLICTS OF INTEREST: The author declares no conflicts of interest.

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AUTHOR CONTRIBUTIONS

Cecilia Claudia Costa Ribeiro: Conceptualization; Methodology; Investigation; Writing – original draft; Writing – review and editing.

USE OF ARTIFICIAL INTELLIGENCE

ChatGPT (OpenAI) was used solely for language editing and text organization. The author reviewed and approved the final text and takes full responsibility for the content of the manuscript.

DATA AVAILABILITY STATEMENT:

No new research data were generated or analyzed for this critical review. All information supporting the review is included in the manuscript and its cited references.

Table 1. Evidence supporting the eight research questions and translation into clinical and public health guidance

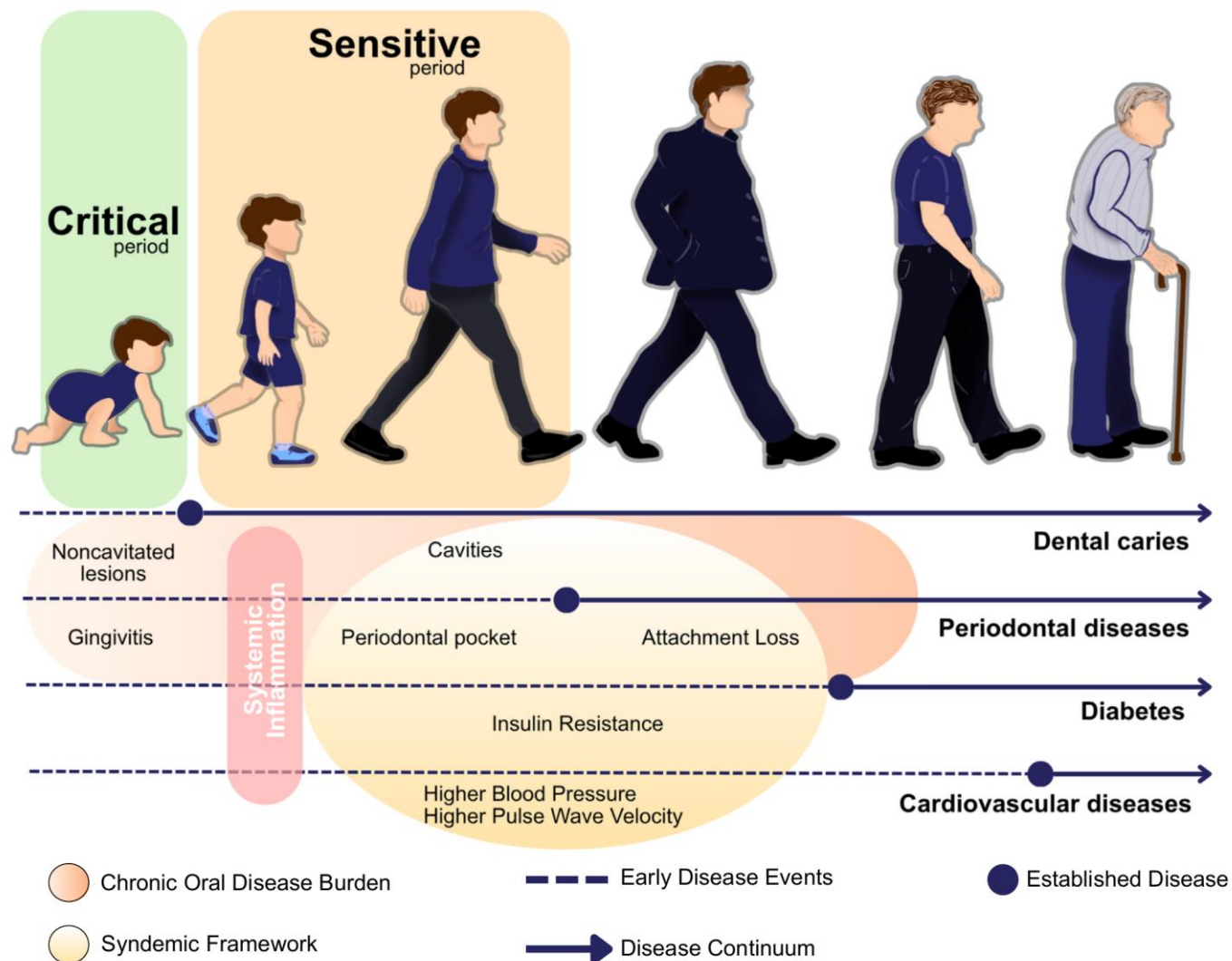
Q.	Shift in the explanatory lens	Key supporting studies	Answer developed	Guideline or policy citation
Q1	Lesion -> disease process	Ribeiro et al., 1999 (Ref. 1); Lula et al., 2009 (Ref. 2)*; Lula et al., 2011 (Ref. 3)	A caries lesion can be arrested without complete removal of carious tissue. However, lesion arrest alone is insufficient to control the disease process.	Brazilian Ministry of Health clinical guideline, 2024 (Ref. 4); American Dental Association clinical practice guideline, 2023 (Ref. 5)
Q2	Disease process -> individual	Lima et al., 2008 (Ref. 8)*	The effect of fluoride concentration depends on baseline caries experience; the same fluoride dose does not provide equivalent protection for every child.	Irish Department of Health report on fluoride toothpaste use in children under six, 2018 (Ref. 9)
Q3	Individual -> social and behavioral contexts	Nunes et al., 2014 (Ref. 10)	Shared social vulnerability does not fully explain caries polarization. Differences in sugar exposure frequency help distinguish children with a greater disease burden.	-
Q4	Caries -> co-occurring conditions	Ribeiro et al., 2017 (Ref. 11); Ribeiro et al., 2018 (Ref. 12); Lima et al., 2018 (Ref. 13)	Early childhood caries co-occurs with contrasting nutritional conditions (double burden of malnutrition) and with local and systemic inflammatory alterations.	-
Q5	Caries-related exposure -> shared oral risk factor	Lula et al., 2014 (Ref. 14); Menezes et al., 2019 (Ref. 16); Moreira et al., 2021 (Ref. 17); Costa et al., 2022 (Ref. 18)	Sugar exposure and unhealthy dietary patterns are associated with periodontal disease through potential local and systemic pathways.	WHO Regional Office for Africa policy document, 2016 (Ref. 15)
Q6	Disease-specific outcomes -> integrated oral health	Carmo et al., 2018 (Ref. 19)*; Ladeira et al., 2024 (Ref. 21); Costa et al., 2023 (Ref. 22); Araújo et al., 2022 (Ref. 23); Araújo et al., 2024 (Ref. 24)	Caries and periodontal indicators converge into a common Chronic Oral Disease Burden, identifiable from early childhood through older adulthood.	Swedish National Board of Health and Welfare, National Guidelines for Dental Care, 2022 (Ref. 20)

Q.	Shift in the explanatory lens	Key supporting studies	Answer developed	Guideline or policy citation
Q7	Oral disease burden -> syndemic framework	Costa et al., 2021 (Ref. 25); Monteiro et al., 2025 (Ref. 26); Ladeira et al., 2023 (Ref. 27); Barbosa et al., 2022 (Ref. 28); Nogueira Filho et al., 2026 (Ref. 29); Falcão et al., 2026 (Ref. 30); Barbosa et al., 2022 (Ref. 31); Costa et al., 2026 (Ref. 32)	Oral, metabolic, respiratory, vascular, and mental health conditions co-occur and are interconnected within shared social and behavioral contexts in adolescence.	-
Q8	Syndemic framework - > complex disease networks	Alves-Costa et al., 2025 (Ref. 33); Nascimento et al., 2017 (Ref. 34); Padilha et al., 2020 (Ref. 35); Muniz et al., 2023 (Ref. 36); Alves-Costa et al., 2024 (Ref. 37)	Dental caries occupies a central position in noncommunicable disease networks, while free sugar consumption, insulin resistance, and socioeconomic disadvantage structure multiple connections.	-

* Study directly cited in the corresponding guideline or policy document.

Figure 1. Oral and systemic disease continuums across the life course

Oral and Systemic Disease Continuums Across the Life Course



Conceptual illustration of oral and systemic disease continuums, early disease events, chronic oral disease burden, and their potential overlap within a syndemic framework. Timing is schematic and does not represent fixed ages, inevitable progression, or established causal relationships between conditions.

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