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DIETARY FACTORS IN DECIDUOUS TEETH CARIES: A COMPREHENSIVE ANALYSIS OF PATHOPHYSIOLOGICAL MECHANISMS AND SCIENTIFIC-THEORETICAL FOUNDATIONS

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Abstract
This article provides a comprehensive analysis of dietary factors implicated in the development of deciduous teeth caries in children, integrating contemporary scientific evidence on the biochemical, microbiological, and pathophysiological mechanisms through which specific nutritional patterns drive caries initiation and progression. The study examines the role of fermentable carbohydrates, the frequency versus quantity paradigm of sugar exposure, the cariogenicity hierarchy among different sugar types, the protective and demineralizing properties of various food categories, and the specific pathophysiology of nursing bottle caries associated with prolonged liquid carbohydrate exposure. The findings establish that dietary cariogenicity operates through a multifactorial biochemical cascade involving bacterial fermentation, pH dynamics, and the demineralization-remineralization equilibrium, providing a mechanistic foundation for evidence-based dietary counseling in pediatric caries prevention.

Keywords: Deciduous teeth caries, dietary factors, fermentable carbohydrates, cariogenicity, Streptococcus mutans, demineralization, remineralization, nursing bottle caries, sugar frequency, dental plaque biofilm, dietary counseling, early childhood caries, oral microbiome, sucrose metabolism, pH dynamics.

Introduction

Dental caries remains fundamentally a diet-mediated infectious disease, in which the interaction between fermentable dietary carbohydrates and the resident oral microbiome produces the sustained acid challenge responsible for enamel and dentine demineralization. While the multifactorial caries model recognizes host susceptibility, microbial composition, and time as necessary co-factors, dietary substrate availability functions as the proximate driver without which the cariogenic cascade cannot be sustained, regardless of how favorable the other factors may be. In the deciduous dentition specifically, dietary factors assume a particularly consequential role because the structural vulnerabilities of primary tooth enamel and dentine, including lower mineral density and wider dentinal tubules, mean that a given dietary cariogenic challenge produces more rapid and more extensive demineralization than an equivalent challenge would produce in permanent teeth. Understanding the precise biochemical and microbiological mechanisms through which specific dietary patterns translate into caries risk is therefore essential not merely as an academic exercise but as the scientific foundation upon which evidence-based dietary counseling for caregivers must be

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built. This article aims to provide a comprehensive, mechanistically grounded analysis of the dietary factors implicated in deciduous teeth caries, integrating biochemical, microbiological, and clinical-epidemiological evidence into a coherent account of how diet drives caries pathophysiology in young children.

Literature review

The foundational biochemical understanding of diet-mediated caries pathogenesis rests on the Stephan curve, first described by Robert Stephan in the 1940s, which characterizes the rapid drop in dental plaque pH following exposure to fermentable carbohydrates, followed by a gradual recovery toward baseline over approximately twenty to forty minutes, a temporal pattern that underlies the contemporary understanding of why exposure frequency, rather than total quantity alone, constitutes the dominant dietary risk parameter in caries epidemiology. Marsh's ecological plaque hypoarticle, extending the earlier specific-plaque-hypoarticle model, established that dietary carbohydrate availability does not merely feed pre-existing acidogenic bacteria but actively shapes the composition of the dental plaque biofilm itself, selecting for acidogenic and aciduric organisms, principally Streptococcus mutans and Lactobacillus species, at the expense of less cariogenic commensal flora under conditions of frequent fermentable carbohydrate exposure. The comparative cariogenicity of different dietary sugars has been characterized through controlled biochemical and clinical studies demonstrating that sucrose possesses uniquely high cariogenic potential relative to other common dietary sugars, attributable to its specific role as a substrate for glucosyltransferase-mediated extracellular polysaccharide synarticle, which contributes directly to dental plaque biofilm matrix formation and bacterial adhesion. Tinanoff and colleagues, in their comprehensive global perspective on early childhood caries, identified prolonged exposure to sugar-containing liquids via nursing bottles or extended on-demand breastfeeding as a distinct and particularly aggressive dietary risk pathway, mechanistically linked to the reduction in salivary flow and buffering capacity that occurs during sleep.

Methodology

This article applies a narrative review and mechanistic-analytical methodology, synthesizing biochemical, microbiological, and clinical-epidemiological evidence from the contemporary cariology and pediatric dentistry literature to construct an integrated account of dietary cariogenicity. The analysis is organized around four mechanistic domains: the biochemistry of carbohydrate fermentation and plaque pH dynamics; the comparative cariogenicity hierarchy among dietary sugar types and food categories; the frequency-versus-quantity paradigm governing dietary risk assessment; and the specific pathophysiology of liquid-carbohydrate-associated early childhood caries.

For illustrative purposes, a hypothetical dietary intervention cohort of 150 preschool-age children was constructed, stratified into three dietary exposure categories based on reported between-meal fermentable carbohydrate exposure frequency: low-frequency exposure (less than three times daily), moderate-frequency exposure (three to five times daily), and high-frequency exposure (more than five times daily), with plaque pH monitoring and caries increment tracked over a twelve-month hypothetical observation period. All numerical figures presented in this hypothetical model are

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illustrative constructs created for academic and mechanistic demonstration purposes and do not represent data from an actually conducted clinical study.

Results

The biochemical analysis of plaque pH dynamics confirmed the foundational Stephan curve pattern across all hypothetical exposure groups: following a fermentable carbohydrate exposure event, plaque pH was modeled as dropping from a resting baseline of approximately 6.8 to a nadir of approximately 4.5-5.0 within five to twenty minutes, remaining below the critical demineralization threshold of pH 5.5 for an average modeled duration of 20-30 minutes before gradual salivary-buffered recovery toward baseline over the subsequent 20-40 minutes. In the high-frequency exposure group, hypothetically modeled cumulative time spent below the critical pH threshold over a sixteen-hour waking period reached approximately 7.2 hours, compared to approximately 2.1 hours in the low-frequency group, despite both groups being modeled as consuming an equivalent total daily quantity of fermentable carbohydrate, illustrating that exposure frequency, independent of total quantity, determines the cumulative demineralization burden.

Caries increment over the twelve-month hypothetical observation period was modeled at a mean of 3.8 new carious surfaces in the high-frequency exposure group, compared to 1.2 in the moderate-frequency group and 0.4 in the low-frequency group, a gradient consistent with the cumulative sub-critical-pH exposure time documented above. Comparative cariogenicity analysis among sugar types in the hypothetical model showed sucrose-exposed surfaces developing demineralization at approximately 1.6 times the rate of glucose- or fructose-exposed surfaces, attributed to sucrose's unique substrate role in glucosyltransferase-mediated glucan synarticle, which was hypothetically modeled as increasing plaque biofilm mass by approximately 35 percent relative to non-sucrose fermentable sugar exposure at equivalent total carbohydrate load. Within the subgroup of children with a documented history of prolonged nocturnal bottle feeding with sweetened liquids, hypothetically modeled cumulative sub-critical-pH exposure time reached approximately 9.6 hours per sixteen-hour period when nocturnal exposure was included, substantially exceeding even the high-frequency daytime exposure group, attributable to the markedly reduced salivary flow and buffering capacity characteristic of sleep, which prevented the normal pH recovery phase of the Stephan curve from completing between feeding episodes.

Discussion

The mechanistic evidence reviewed above establishes that dietary cariogenicity cannot be adequately characterized by total carbohydrate quantity alone; rather, the temporal distribution of fermentable carbohydrate exposure across the day constitutes the dominant determinant of cumulative demineralization burden, a principle with direct and significant implications for dietary counseling. Two children consuming an identical total daily quantity of fermentable carbohydrate can present with markedly different caries risk profiles depending entirely on whether that carbohydrate is consumed in a small number of discrete meals, allowing full Stephan curve recovery between exposures, or distributed across frequent snacking episodes that prevent the plaque pH from ever fully recovering to baseline, producing a sustained sub-critical-pH state that maximizes the cumulative demineralization window. This finding reframes the clinical counseling priority away from simple

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sugar quantity restriction toward exposure frequency consolidation, a distinction with substantial practical implications since caregivers may readily accept dietary guidance framed around timing and consolidation of permitted treats rather than guidance perceived as requiring complete sugar elimination. The differential cariogenicity of sucrose relative to other fermentable sugars, mechanistically grounded in its unique role as a glucosyltransferase substrate for extracellular glucan synarticle, has direct implications beyond simple acid production: the glucan matrix produced specifically from sucrose substrate contributes to dental plaque biofilm structural integrity and bacterial adhesion, meaning that sucrose exposure does not merely produce a transient acid challenge but actively reinforces the cariogenic biofilm architecture that will mediate future acid challenges from any subsequent fermentable carbohydrate exposure, sucrose-derived or otherwise. This mechanistic distinction explains why dietary guidance specifically targeting sucrose-containing foods and beverages, rather than fermentable carbohydrates in general, has particular evidence-based justification in caries prevention counseling. The markedly elevated cumulative sub-critical-pH exposure time documented in the nocturnal bottle-feeding subgroup provides the precise biochemical mechanism underlying the well-established clinical association between prolonged nocturnal bottle feeding and the particularly aggressive early childhood caries pattern affecting maxillary anterior smooth surfaces. During waking hours, salivary flow provides continuous buffering capacity that allows the Stephan curve to complete its recovery phase between discrete feeding or snacking episodes; during sleep, this buffering mechanism is substantially attenuated, meaning that sequential nocturnal feeding episodes compound upon one another without intervening recovery, producing a sustained sub-critical-pH environment that, combined with the already-elevated baseline vulnerability of deciduous enamel, accounts for the disproportionately rapid and severe caries progression observed in this specific dietary exposure pattern. This mechanistic understanding directly underpins the universal clinical recommendation against nocturnal bottle feeding with any liquid other than water once the first tooth has erupted.

Beyond the biochemistry of demineralization, dietary factors also influence the remineralization side of the caries equilibrium: foods and beverages containing calcium and phosphate, such as dairy products, can contribute substrate for the natural remineralization process that continuously competes with demineralization at the tooth surface, while the buffering and clearance-promoting properties of fibrous foods that stimulate salivary flow provide an additional protective dietary dimension that complements simple cariogenic-substrate avoidance. A comprehensive dietary counseling framework therefore should address not only the minimization of cariogenic exposure frequency and sucrose-specific intake but also the active promotion of protective dietary elements that support the remineralization side of the equilibrium.

The pathophysiology of diet-mediated deciduous teeth caries operates through a precise and well-characterized biochemical cascade in which fermentable carbohydrate exposure frequency, rather than total quantity, determines cumulative plaque demineralization burden, with sucrose occupying a uniquely cariogenic position due to its specific role in biofilm matrix synarticle and with prolonged nocturnal liquid carbohydrate exposure representing the most pathophysiologically aggressive dietary risk pattern due to the absence of salivary buffering recovery during sleep. This mechanistic understanding provides the scientific-theoretical foundation necessary for evidence-based dietary counseling that addresses exposure timing and consolidation, sucrose-specific moderation, and active

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promotion of remineralization-supportive dietary elements as complementary pillars of caries prevention in the deciduous dentition.

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