

# PATHOLOGICAL ANATOMY AND CLINICAL PICTURE OF DECIDUOUS TEETH CARIES IN CHILDREN A COMPLEX ANALYSIS OF ETIOPATHOGENESIS, MORPHOLOGY AND CLINICAL COURSE BASED ON CONTEMPORARY SCIENTIFIC DATA

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## Abstract

This article presents a comprehensive analysis of the etiopathogenesis, morphological characteristics, and clinical course of deciduous teeth caries in children, integrating contemporary scientific evidence on the microbiological, biochemical, and structural mechanisms underlying caries initiation and progression in the primary dentition. The study examines the distinctive histological features of deciduous enamel and dentine that render the primary dentition particularly vulnerable to rapid caries progression, reviews the principal etiological factors and their interaction within the established multifactorial caries model, and characterizes the clinical staging and presentation patterns specific to early childhood caries. The findings confirm that the pathological anatomy of deciduous caries is distinguished from permanent dentition caries by a series of structural and biological features that collectively accelerate lesion progression and necessitate earlier and more aggressive clinical intervention.

**Keywords:** Deciduous teeth caries, early childhood caries, etiopathogenesis, pathological anatomy, enamel demineralization, dentine morphology, Streptococcus mutans, caries progression, clinical staging, primary dentition, caries morphology, pulp involvement, nursing bottle caries, caries risk factors, pediatric dentistry.

## Introduction

Dental caries affecting the deciduous dentition represents a complex, multifactorial infectious disease process whose pathological anatomy and clinical behavior differ in important and clinically consequential respects from caries affecting the permanent dentition. These differences arise from fundamental structural distinctions between deciduous and permanent tooth tissues, including the thinner and more permeable enamel layer of deciduous teeth, the proportionally larger pulp chambers and wider dentinal tubules characteristic of the primary dentition, and the comparatively lower degree of mineralization of deciduous enamel relative to permanent enamel at equivalent developmental stages. Taken together, these structural features render the primary dentition not merely quantitatively more susceptible to caries attack but qualitatively different in its pattern of lesion initiation,



morphological progression, and clinical manifestation, demanding a specific and well-grounded understanding of deciduous caries pathological anatomy on the part of clinicians responsible for the dental care of young children. This article aims to integrate contemporary scientific evidence on the etiopathogenesis, morphological characteristics, and clinical course of deciduous teeth caries, providing a systematic analysis applicable to both clinical practice and academic study in pediatric dentistry.

### Literature review

The foundational multifactorial model of dental caries etiology, originally articulated by Keyes and subsequently elaborated by Fejerskov and Kidd among others, identifies the interaction of a susceptible host, cariogenic microorganisms, fermentable dietary substrate, and sufficient time as the necessary and jointly sufficient conditions for caries initiation. In the context of the deciduous dentition, each of these factors operates under conditions that collectively amplify caries risk relative to the permanent dentition. The microbiological dimension of deciduous caries etiopathogenesis centers on the role of mutans streptococci, principally *Streptococcus mutans* and *Streptococcus sobrinus*, as the primary acidogenic and aciduric organisms responsible for the sustained pH reduction at the enamel surface that drives demineralization, with vertical transmission from primary caregiver to infant identified as the principal route of early colonization. The role of *Lactobacillus* species as secondary colonizers contributing to lesion progression into dentine has been additionally documented in the contemporary caries microbiology literature. The structural vulnerability of deciduous enamel has been characterized through scanning electron microscopy and microhardness studies demonstrating lower mineral density, wider intercrystalline spaces, and greater permeability to acid and ionic diffusion relative to mature permanent enamel, providing a histological basis for the clinically observed rapidity of caries progression in the primary dentition. The concept of early childhood caries as a specific and particularly severe clinical presentation of deciduous caries, characterized by the involvement of smooth surfaces and anterior teeth in a pattern inconsistent with simple dietary sugar exposure and strongly associated with prolonged nocturnal bottle feeding with sweetened liquids or prolonged on-demand breastfeeding, has been systematically defined and classified in the pediatric dental literature, with the American Academy of Pediatric Dentistry providing widely adopted diagnostic criteria.

### Methodology

This article applies a narrative review and descriptive-analytical methodology, integrating contemporary evidence from the microbiological, histopathological, and clinical pediatric dental literature to construct a comprehensive account of deciduous caries etiopathogenesis, morphology, and clinical staging. For illustrative purposes, a hypothetical clinical observation series of 180 preschool-age children presenting with active deciduous caries across varying stages of lesion severity was constructed to demonstrate the distribution of lesion morphology and clinical staging patterns across the primary dentition. Children were hypothetically stratified by caries severity according to the International Caries Detection and Assessment System staging framework, and lesion morphological characteristics were described in relation to the affected tooth tissue layer. All



numerical figures are illustrative constructs for academic purposes and do not represent data from an actually conducted study.

## Results

In the illustrative observation series of 180 children with active deciduous caries, lesion distribution across the International Caries Detection and Assessment System severity spectrum was hypothetically modeled as follows: initial enamel lesions without cavitation accounted for approximately 22 percent of all recorded lesions, established cavitated enamel lesions without dentine involvement accounted for approximately 31 percent, lesions extending into outer and middle dentine accounted for approximately 29 percent, and lesions extending into deep dentine with radiographic evidence approaching pulpal involvement accounted for approximately 18 percent of all recorded lesions. This distribution, with nearly half of all recorded lesions already demonstrating dentine involvement at the time of clinical presentation, illustrates the well-documented tendency toward delayed presentation in pediatric dental populations and the rapid progression characteristic of deciduous caries once the enamel-dentine junction has been breached. Morphological characterization of lesions in the illustrative series revealed that the deciduous enamel caries lesion is characterized histologically by a subsurface demineralization zone beneath a relatively intact surface layer, consistent with the established caries lesion body structure documented in the histopathological literature, but progressing to full cavitation more rapidly than equivalent lesions in permanent enamel due to the structural features outlined above. Once the enamel-dentine junction is reached, caries spread in the primary dentition was modeled as characteristically lateral as well as apical, reflecting the comparatively wider dentinal tubules of deciduous dentine that facilitate more rapid acid and bacterial penetration in multiple directions simultaneously relative to permanent dentine. The proportionally larger pulp chambers of deciduous teeth were represented in the illustrative series as resulting in a shorter clinical pathway between initial enamel cavitation and pulpal involvement, with pulpal signs or symptoms present in a hypothetically modeled 34 percent of children presenting with deep dentine lesions at the time of clinical examination. In the subgroup of illustrative cases classified as early childhood caries based on the involvement of maxillary anterior smooth surfaces and a reported history of prolonged nocturnal bottle feeding, the mean hypothetically modeled dmf index was 6.8, substantially higher than the overall cohort mean of 3.9, and the proportion of lesions with deep dentine involvement was hypothetically modeled at 47 percent, illustrating the particularly aggressive clinical course associated with this caries pattern relative to conventional pit and fissure or approximal caries in the primary dentition.

## Discussion

The etiopathogenesis of deciduous teeth caries operates through the same fundamental multifactorial mechanism as permanent dentition caries, namely the sustained acidogenic activity of cariogenic microorganisms metabolizing fermentable dietary carbohydrates at the tooth surface, producing local pH reduction below the critical threshold for enamel hydroxyapatite dissolution. However, the structural and biological characteristics of the primary dentition modify the expression of this fundamental mechanism in ways that produce a distinctive and clinically important pathological anatomy. The lower mineral density and greater acid permeability of deciduous enamel shorten the



time required for demineralization to progress from the enamel surface to the enamel-dentine junction, while the wider dentinal tubules of primary dentine facilitate more rapid lateral spread of the caries lesion once dentine is reached, together producing the characteristically rapid clinical progression that distinguishes active deciduous caries from caries in the permanent dentition and underscores the clinical urgency of early detection and intervention.

The proportionally larger pulp chambers of deciduous teeth constitute perhaps the most clinically consequential structural feature in terms of treatment planning implications, since the reduced distance between the enamel-dentine junction and the pulp means that even moderately advanced caries lesions in the primary dentition carry a substantially higher risk of pulpal involvement than equivalent lesions would in permanent teeth, and conservative restorative approaches that would be appropriate for managing a moderately deep lesion in a permanent tooth may be inadequate for an equivalent lesion in a deciduous tooth without pulpal risk assessment and management. This morphological reality provides the histopathological basis for the clinical guideline recommendation that deep carious lesions in the primary dentition should be evaluated for pulpal status and managed with stepwise or indirect pulp capping techniques or pulpotomy where indicated, rather than simply excavated and restored without pulpal consideration. The particularly aggressive clinical course and morphological severity documented in early childhood caries cases in the illustrative series reflects the specific pathophysiological conditions created by prolonged nocturnal bottle feeding with sweetened liquids: during sleep, salivary flow is substantially reduced, eliminating the principal physiological buffering and remineralization mechanism available to the tooth surface, while the sustained contact between fermentable carbohydrate-containing liquid and the dentition creates conditions of prolonged acidogenic bacterial activity that far exceed those produced by daytime dietary sugar exposure alone. This pathophysiological mechanism provides the scientific basis for the universal recommendation against nocturnal bottle feeding with sweetened liquids as a foundational element of early childhood caries prevention counseling.

The pathological anatomy and clinical course of deciduous teeth caries are distinguished from permanent dentition caries by a series of structural features, including lower enamel mineral density, wider dentinal tubules, and proportionally larger pulp chambers, that collectively accelerate lesion initiation, progression, and pulpal involvement in the primary dentition. Contemporary scientific evidence on the etiopathogenesis, morphology, and clinical staging of deciduous caries provides an essential evidence base for clinically informed treatment planning in pediatric dentistry and underscores the necessity of early detection, prompt intervention, and caregiver-targeted preventive education as the foundational pillars of primary dentition caries management.

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