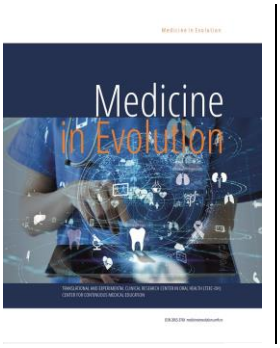
From Biofilm to Cavity: Clinical and Microbiological Determinants of Early Childhood Caries



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Abstract

Early childhood caries (ECC) is one of the most prevalent chronic diseases of childhood and a major, socially patterned public health problem. It is a multifactorial, biofilm-mediated, sugar-driven disease in which a dysbiotic shift of the oral microbiota toward acidogenic and acid-tolerant organisms — including Streptococcus mutans, lactobacilli and the yeast Candida albicans — repeatedly lowers plaque pH and shifts the balance between demineralization and remineralization toward net mineral loss. This narrative review integrates the clinical and microbiological determinants of ECC and traces the continuum leading from a dysbiotic dental biofilm to clinical cavitation. We summarize current definitions, classification and epidemiology, including data from Romania; the microbiological basis of the disease (the dental biofilm, the ecological plaque hypothesis, the roles of mutans streptococci, lactobacilli and Candida albicans, and microbiome dysbiosis); and the host, dietary and behavioral factors that modulate risk. These mechanisms are linked to clinical presentation, diagnostic criteria (dmft indices and ICDAS) and severity classification, and to implications for prevention. An integrated, ecological perspective, rather than a single-pathogen model, most coherently accounts for ECC and supports early, risk-based, family-centered and interdisciplinary prevention.

Keywords: early childhood caries; dental biofilm; Streptococcus mutans; Candida albicans; oral microbiome; dysbiosis; dental caries; preschool children; demineralization; caries risk assessment

INTRODUCTION

Dental caries is one of the most widespread chronic, non-communicable diseases affecting humans, resulting from a complex and prolonged interaction between acid-producing bacteria organized in a biofilm, fermentable dietary carbohydrates, and host factors such as the teeth and saliva, all acting over time [1]. When this process affects the primary dentition of young children it is termed early childhood caries (ECC). The American Academy of Pediatric Dentistry (AAPD) defines ECC as the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth of a child 71 months of age or younger [2]. A more aggressive form, severe early childhood caries (S-ECC), is defined by age-specific thresholds and by the involvement of smooth surfaces, and it corresponds to the pattern historically described as "nursing bottle caries" or "baby bottle tooth decay" [2,3].

ECC is not merely an early manifestation of a common disease; it has distinctive clinical and biological features. It tends to progress rapidly, often follows a characteristic pattern that spares the mandibular incisors while affecting the maxillary incisors and molars, and is closely linked to inappropriate feeding practices and the early acquisition of cariogenic microorganisms [3,4]. Untreated, ECC causes pain, infection, difficulty eating and sleeping, impaired growth and quality of life, and it is a strong predictor of caries in the permanent dentition [1,4].

The global burden of the disease is substantial. The World Health Organization estimates that oral diseases affect close to half of the world's population, and that some 514 million children are affected by caries of the primary teeth [5]. A comprehensive global review of ECC has underscored that the disease is driven by frequent sugar consumption in an environment of enamel-adherent, acid-producing bacteria within a complex biofilm, and that its distribution follows a strong social gradient [4]. In many countries, including Romania, caries prevalence in young children remains far above WHO targets, reflecting limited preventive infrastructure and unequal access to care [6,7].

Historically, ECC has been conceptualized in terms of a small number of key pathogens, most notably *Streptococcus mutans*. Contemporary evidence, however, increasingly favors an ecological, community-level understanding of the disease, in which dysbiosis of the dental biofilm — rather than the mere presence of a single species — drives net demineralization [8,9,10]. The aim of this narrative review is to integrate the clinical and microbiological determinants of ECC and to delineate the conceptual pathway that leads from biofilm to cavity. After summarizing the definitions, classification and epidemiology of ECC, we examine the microbiological basis of the disease, the host and behavioral factors that modulate it, the pathogenic sequence from biofilm dysbiosis to cavitation, and the clinical and diagnostic correlates of these processes, before considering the implications for prevention and management.

DEFINITIONS, CLASSIFICATION AND EPIDEMIOLOGY

Definitions and classification

The standardization of ECC definitions has been important for both research and clinical care. According to the AAPD, ECC denotes any caries experience (non-cavitated or cavitated lesions, missing surfaces due to caries, or filled surfaces) in any primary tooth in a child under six years of age; S-ECC is defined by more stringent, age-related criteria, including any sign of smooth-surface caries in children younger than three years [2]. These definitions are deliberately inclusive of early, non-cavitated lesions, reflecting the modern

understanding of caries as a continuum rather than a simple present/absent phenomenon [11].

At the level of the individual lesion, caries is best conceptualized as a dynamic process ranging from the earliest, subsurface, sub-clinical mineral loss, through visible non-cavitated ("white spot") lesions, to enamel and dentinal cavitation [11]. This continuum is central to the theme of the present review, because the same microbiological and dietary drivers operate throughout, and because early stages are potentially arrestable or reversible.

Global epidemiology

ECC is among the most common diseases of early childhood worldwide, with reported prevalence varying widely by region, age and social circumstances, and reaching very high levels — up to around 85% — in disadvantaged populations [3]. A global perspective on ECC has emphasized both the magnitude of the problem and the striking inequalities in its distribution, with the highest burden falling on children of lower socioeconomic status and on those with limited access to preventive dental care [4]. WHO data reinforce this picture, estimating that hundreds of millions of children are affected by caries in the primary dentition and highlighting oral disease as a major, largely preventable, global health challenge [5].

Epidemiology in Romania

Romania illustrates the challenges faced by many countries with limited public preventive programs. In a study of 6–8-year-old Romanian schoolchildren, the mean number of decayed, missing and filled teeth (DMFT) was 4.89 among the children examined, a level the authors described as very high relative to WHO recommendations for this age group [6]. Regional data are consistent with a heavy caries burden and with the influence of oral health behaviours and living conditions on disease experience [6]. More recent work in Romanian preschoolers aged 4–5 years has examined the geography of sugar intake as a risk factor, finding statistically significant differences in sugar consumption across counties but showing that household-level factors — such as parental education and socioeconomic status — appear to play a larger role than geography alone in shaping children's dietary risk [7]. Together, these findings underscore that ECC in Romania is common, socially patterned and closely tied to modifiable dietary and behavioral determinants.

THE MICROBIOLOGICAL BASIS OF EARLY CHILDHOOD CARIES

Dental plaque as a structured biofilm

Dental plaque is not a random accumulation of microorganisms but a structurally and functionally organized biofilm that develops on tooth surfaces in an ordered sequence and that, in health, maintains a diverse and relatively stable microbial composition — a state of microbial homeostasis [8]. The biofilm mode of growth confers properties that are highly relevant to caries: close physical and metabolic interactions between species, gradients of pH and oxygen, an extracellular polymeric matrix that promotes adhesion and retention, and increased tolerance to environmental stress and antimicrobials [8]. An understanding of ECC therefore requires that the microbiota be viewed as a community whose collective metabolic activity — not simply the identity of any individual member — determines whether the tooth surface remains sound or begins to demineralize.

Dietary sucrose plays a special role in this biofilm ecology. Beyond serving as a readily fermentable substrate for acid production, sucrose is the preferred substrate for the synthesis of extracellular glucans by cariogenic streptococci, and these glucans form a substantial part of the extracellular polymeric matrix that binds the biofilm together, mediates firm adherence to enamel, and creates the diffusion-limited microenvironments in which acid accumulates against the tooth surface [12,8]. The matrix is therefore not merely a scaffold but an active

contributor to virulence: it concentrates acid, protects resident organisms, and stabilizes the very community shifts that drive disease [8]. This may account for the observation that both the frequency and the chemical nature of sugar intake are of consequence, and for the fact that the identification of a single cariogenic species in isolation is less informative than the characterization of the metabolically active, matrix-embedded community as a whole [8,9].

The ecological plaque hypothesis and dysbiosis

The ecological plaque hypothesis provides the conceptual bridge between diet, microbiology and disease. In this framework, frequent intake of fermentable carbohydrates leads to repeated production of organic acids by plaque bacteria, lowering the local pH. Persistently low pH selects for acidogenic (acid-producing) and aciduric (acid-tolerant) species while suppressing the acid-sensitive organisms associated with sound enamel, driving a shift — a dysbiosis — in the composition and activity of the community toward a more cariogenic state [9]. Dental caries has thus been described as an example of an "ecological catastrophe", in which a change in local environmental conditions, principally recurrent acidification, disrupts the normal microbiota and favors demineralization [9].

Molecular studies of the oral microbiome in children support this ecological view. Analysis of the microbial communities associated with ECC has demonstrated measurable dysbiosis, with alterations in community structure that distinguish caries-active children from caries-free children, consistent with a shift toward acidogenic and aciduric taxa rather than the acquisition of a single unique pathogen [10]. This community-level perspective complements, and to some extent reframes, the traditional single-species models discussed below.

Mutans streptococci

Among individual organisms, *Streptococcus mutans* (and, in some populations, *Streptococcus sobrinus*) has historically been regarded as the principal etiological agent of caries, including ECC [12]. Mutans streptococci are strongly acidogenic and aciduric, adhere efficiently to enamel, and synthesize extracellular glucans from sucrose that promote biofilm accumulation, making them well adapted to the cariogenic niche [12]. Their role in ECC is closely tied to the timing of colonization. Classic work established that infants acquire mutans streptococci during a relatively discrete "window of infectivity", with a reported median age of initial acquisition around 26 months, though colonization can occur earlier in high-risk children [13]. Earlier and heavier colonization, frequently from the mother or primary caregiver, is associated with greater caries risk, providing a biological rationale for early preventive intervention [4,13].

Mutans streptococci must nevertheless be interpreted within this broader ecological context. Although they are strongly associated with caries and contribute substantially to acid production and biofilm formation, the disease may occur in their relative absence and does not depend on any single species; the weight of current evidence supports a polymicrobial, community-driven process in which mutans streptococci are important but not uniquely necessary contributors [9,10].

Lactobacilli

Lactobacilli are highly acidogenic and aciduric organisms that have long been associated with caries, particularly with the progression of established, cavitated dentinal lesions. Rather than being primary initiators of the disease on smooth enamel surfaces, oral lactobacilli appear to behave as opportunistic secondary colonizers that flourish once a suitable niche has developed. It has been proposed that sustained lactobacillus colonization requires a stagnant, largely anaerobic, low-pH environment with ready access to carbohydrates — precisely the conditions found within an advancing carious lesion [14]. In this model, lactobacilli represent organisms that have adapted to a niche created by the

destabilization of a healthy oral microbiome into a cariogenic one, and their abundance is therefore both a marker and a driver of lesion progression [14].

Candida albicans and interkingdom interactions

An increasingly recognized component of the ECC microbiota is the yeast *Candida albicans*. Clinical studies have repeatedly demonstrated the co-occurrence of *C. albicans* and *S. mutans* in ECC lesions, and mechanistic work indicates that the two organisms can act synergistically to enhance each other's cariogenic potential [15]. *C. albicans* can promote the adhesion and accumulation of *S. mutans*, and this cross-kingdom cooperation is mediated in part by streptococcal glucosyltransferase (Gtf) enzymes, which bind to the yeast surface and generate glucans that strengthen the biofilm matrix [15]. The resulting mixed-species biofilm is more robust, more acidogenic and potentially more virulent than either organism alone, offering a plausible explanation for the aggressive, rampant character of some cases of ECC [15]. The recognition of these interkingdom interactions has broadened the microbiological model of ECC beyond bacteria alone and has implications for both diagnosis and targeted prevention [15].

HOST, DIETARY AND BEHAVIORAL DETERMINANTS

The microbiological drivers of ECC operate within, and are strongly modulated by, host and environmental factors. Seow's foundational review of the biological mechanisms of ECC emphasized that, although the general etiology resembles that of other forms of caries — mutans streptococci fermenting dietary carbohydrates to produce acid attacks on susceptible teeth — the predisposing factors specific to very young children are distinctive and, in part, incompletely understood [12]. A subsequent unifying conceptual model organized these determinants into environmental, maternal and child-related domains that interact to produce disease [16].

Among the behavioral determinants, diet and feeding practices are of primary importance. Frequent consumption of fermentable carbohydrates, especially sugars, constitutes the central dietary driver of ECC, since each exposure provides substrate for acid production and prolongs the periods of low plaque pH [1,9]. In infants and toddlers, feeding behaviours are particularly influential: prolonged and frequent bottle use, especially with sugar-containing liquids or at night, together with on-demand nocturnal breastfeeding after the eruption of the teeth, may create sustained cariogenic conditions, particularly on the maxillary incisors [3,4]. Data from Romanian preschoolers confirm that the frequency and type of sugar intake vary across the population and are shaped by parental and socioeconomic factors, reinforcing diet as a modifiable target [7].

Saliva constitutes a major line of host defense. It contributes to the clearance of food debris and acids, to the buffering of pH, and to the supply of calcium, phosphate and antimicrobial components that support remineralization [1,11]. A reduced salivary flow or impaired salivary function shifts the balance toward demineralization [11]. In young children, the relative immaturity of erupting enamel and, in certain cases, developmental defects of enamel further increase susceptibility [4].

Transmission from the caregiver represents a further determinant. Because cariogenic organisms are frequently transmitted from the mother or primary caregiver, maternal oral health, maternal caries experience and caregiver behaviours — such as the sharing of utensils — influence the timing and magnitude of the child's colonization [4,13]. This vertical and horizontal transmission underlies the importance of a family-centered approach to prevention [16].

Finally, the socioeconomic and behavioral context exerts a pronounced influence. ECC follows a marked social gradient, with lower socioeconomic status, lower parental education, limited access to preventive dental care and suboptimal oral hygiene practices all associated with a higher disease burden [3,4]. These determinants are especially salient in the Romanian context, where caries prevalence is high and where the majority of oral health services are delivered through the private sector, generating financial and geographic barriers to care [6,7].

FROM BIOFILM TO CAVITY: THE PATHOGENIC CONTINUUM

The determinants described above converge on a single, dynamic mechanism at the tooth surface. Every time fermentable carbohydrate reaches the dental biofilm, acidogenic bacteria metabolize it and release organic acids, lowering the plaque pH. When the pH falls below the critical threshold for enamel (approximately 5.5), the surrounding fluid becomes undersaturated with respect to tooth mineral, and calcium and phosphate diffuse out of the enamel — demineralization [1,11]. When carbohydrate intake ceases and saliva restores a neutral pH, the fluid becomes supersaturated again and mineral, together with fluoride, can be redeposited — remineralization [11].

The eventual clinical outcome is therefore determined by the "caries balance": the net result of pathological factors that promote demineralization (acidogenic bacteria, frequent fermentable-carbohydrate intake, impaired salivary function) versus protective factors that promote remineralization (adequate salivary flow and components, fluoride, and favorable dietary patterns) [11]. In health, cycles of demineralization and remineralization are balanced and no net mineral loss occurs. In ECC, the combination of frequent sugar exposure and a dysbiotic, acid-adapted biofilm biases this balance toward repeated and prolonged demineralization [9,11].

Crucially, the microbiological dysbiosis and the physicochemical process reinforce one another. Recurrent low pH selects for ever more acidogenic and aciduric organisms, which in turn generate more acid — a self-perpetuating cycle that pushes the community further from health [9]. As net mineral loss continues, the earliest subsurface lesion becomes clinically visible as a non-cavitated white-spot lesion; with ongoing imbalance, the surface breaks down and the lesion cavitates, first in enamel and then extending into dentin [11]. Advancing lesions create the stagnant, anaerobic, low-pH niche in which secondary colonizers such as lactobacilli proliferate and accelerate dentinal destruction [14], while interkingdom partnerships such as that between *S. mutans* and *C. albicans* can render the biofilm more matrix-rich and virulent, helping to explain the rapid, rampant progression characteristic of severe ECC [15]. In this way, the pathway from biofilm to cavity is best understood not as the action of a single microbe but as an ecological and physicochemical continuum, driven by diet and modulated by host defenses.

A practical corollary of this continuum concerns the frequency, rather than merely the total amount, of carbohydrate exposure. Because each acid challenge is followed by a period of salivary recovery and potential remineralization, it is the repetition of demineralizing episodes throughout the day — as occurs with frequent snacking, sipping of sweetened drinks or on-demand night-time feeding — that most powerfully biases the balance toward net mineral loss [1,11]. In young children, whose salivary flow is reduced during sleep and whose feeding is often frequent and prolonged, this temporal pattern is particularly consequential and provides a clear, mechanistic rationale for the behavioral advice that underpins ECC prevention [3,4,11].

CLINICAL PRESENTATION AND DIAGNOSIS

Clinical features and pattern

The clinical expression of ECC reflects the mechanisms described above. The earliest sign is typically a chalky, opaque white-spot lesion along the gingival margin of the maxillary primary incisors — the surfaces most exposed to pooled cariogenic liquids and least protected by saliva [3,4]. As the disease progresses, these lesions cavitate and darken, and the pattern extends to the primary molars and canines, often in an age-related sequence that mirrors the eruption of the teeth and the duration of exposure to the cariogenic environment [3]. The characteristic sparing of the mandibular incisors, which are protected by the tongue and by proximity to salivary ducts, constitutes a useful diagnostic feature in distinguishing ECC from other conditions [3]. In severe cases, extensive destruction of multiple teeth, pain and abscess formation may be present [4].

Diagnostic indices and criteria

Accurate detection and quantification of ECC are essential for both clinical management and epidemiological comparison. The most widely used epidemiological measure is the dmft/dmfs index, which counts decayed, missing (due to caries) and filled primary teeth or surfaces; the mean dmft of a population provides a compact summary of caries experience and is the metric used, for example, in the Romanian studies cited above [6]. However, traditional indices at the cavitation threshold do not capture early, non-cavitated lesions and therefore underestimate the true burden of disease along the continuum [11].

To address this limitation, the International Caries Detection and Assessment System (ICDAS) was developed as an integrated, standardized visual system that classifies caries severity on an ordinal scale from the earliest visible enamel changes through to extensive cavitation into dentin [17]. ICDAS has demonstrated content and correlational validity against histological assessment and allows both early and established lesions to be recorded, making it well suited to a modern, continuum-based understanding of ECC and to caries management by risk assessment [17]. Combining a severity-sensitive detection system such as ICDAS with conventional dmft reporting provides a fuller clinical and epidemiological picture than either alone.

The role of microbiological assessment

Because ECC is a biofilm-driven ecological disease, microbiological assessment can complement clinical diagnosis and risk assessment. Traditional culture-based methods on selective media, together with quantification of colony-forming units of mutans streptococci and lactobacilli, have long been used to gauge cariogenic microbial load, while molecular techniques such as quantitative polymerase chain reaction (qPCR) and high-throughput sequencing now permit more precise and comprehensive characterization of the oral microbiome, including the detection of *Candida albicans* and of community-level dysbiosis [10,15]. Such approaches remain largely research or risk-stratification tools rather than routine diagnostics, but they reinforce the clinical value of identifying children with a highly cariogenic, dysbiotic biofilm before extensive cavitation has occurred.

IMPLICATIONS FOR PREVENTION AND MANAGEMENT

Viewing ECC as an ecological, biofilm-mediated disease has direct consequences for prevention and management. Because the disease arises from an imbalance driven by frequent sugar exposure and a cariogenic biofilm, effective prevention aims to shift the caries balance back toward health rather than simply to eliminate a single pathogen [9,11]. Key strategies supported by the mechanisms reviewed here include reducing the frequency of fermentable-carbohydrate intake and modifying feeding practices (in particular avoiding prolonged, frequent or nocturnal exposure to sugar-containing liquids) [3,4,7]; supporting

remineralization and inhibiting demineralization through the appropriate use of fluoride [11]; and disrupting the cariogenic biofilm through effective, caregiver-assisted oral hygiene from the time the first teeth erupt [2].

The timing of intervention matters. The existence of an early window during which cariogenic organisms are acquired, and the transmissibility of these organisms from caregivers, argue for early, family-centered preventive efforts that begin in infancy and address maternal oral health [4,16,13]. The AAPD emphasizes a combination of dietary counselling, reinforcement of toothbrushing with fluoridated toothpaste, professional preventive measures such as topical fluoride and sealants where appropriate, and minimally invasive restorative care when needed, all delivered within a risk-based framework [2]. Because early, non-cavitated lesions are potentially arrestable or reversible, detection systems such as ICDAS that identify disease along the continuum enable timely, minimally invasive management before cavitation occurs [11,17]. Finally, the recognition of interkingdom interactions, such as that between *S. mutans* and *C. albicans*, points toward possible future targeted approaches that address the mixed-species biofilm rather than bacteria alone [15].

Fluoride merits particular emphasis because it acts directly on the demineralization–remineralization balance that lies at the heart of the disease. By promoting the redeposition of mineral in a more acid-resistant form and by interfering with bacterial acid production, fluoride can arrest early lesions and reduce the likelihood that subsurface mineral loss progresses to cavitation [11]. Delivered as fluoridated toothpaste used in an age-appropriate amount, and supplemented where indicated by professionally applied topical fluoride, it is one of the most firmly established preventive measures and is a central component of the risk-based approach advocated for young children [2,11]. Together with dietary modification and biofilm disruption, fluoride exemplifies how an understanding of the underlying mechanism translates into concrete, minimally invasive clinical practice [2,11].

At the population level, the persistently high caries burden documented in settings such as Romania, together with the strong social gradient of the disease, indicates that individual clinical measures must be complemented by public health action — improved access to preventive dental care, community-level prevention and attention to the socioeconomic determinants that shape both diet and care-seeking [6,7].

DISCUSSIONS

This review has traced early childhood caries from its microbiological origins in the dental biofilm to its clinical endpoint in cavitation, emphasizing that the two are linked by a single, dynamic physicochemical and ecological process. Several themes emerge. First, the classic single-pathogen model centered on *Streptococcus mutans*, while capturing an important contributor, is insufficient on its own; the accumulated evidence supports an ecological, community-level understanding in which dysbiosis of the biofilm — a shift toward acidogenic and aciduric organisms under the pressure of frequent sugar exposure and low pH — is the proximate microbiological driver of the disease [9,10]. Mutans streptococci, lactobacilli and *Candida albicans* each occupy meaningful roles within this community: mutans streptococci as strongly acidogenic early contributors adapted to enamel colonization [12,13], lactobacilli as opportunistic secondary colonizers that accelerate lesion progression [14], and *C. albicans* as an interkingdom partner that can enhance biofilm virulence [15].

Second, microbiology cannot be separated from host and behavioral context. The same organisms produce disease only under permissive conditions — frequent fermentable-carbohydrate intake, impaired salivary protection, early and heavy colonization, and the socioeconomic circumstances that shape these exposures [4,12,16]. This integration is

precisely why ECC is best managed through a combination of dietary, hygienic, fluoride-based and family-centered measures rather than through any single intervention [2,11].

Third, the clinical and diagnostic tools available to the clinician map onto this continuum. The dmft index remains valuable for epidemiological comparison and captures the cavitated burden of disease, as illustrated by Romanian data [6], while severity-sensitive systems such as ICDAS extend detection to the early, potentially reversible stages that are the most promising targets for prevention [11,17]. Microbiological assessment, increasingly aided by molecular methods, offers a complementary window onto the dysbiotic biofilm that underlies clinical disease [10,15].

Several limitations of the current evidence base should be acknowledged. Much of the microbiological understanding of ECC derives from cross-sectional association studies, which cannot fully establish causal sequence or the direction of the relationship between specific taxa and disease; longitudinal and mechanistic studies are needed to clarify how community dysbiosis initiates and progresses in individual children. Definitions and diagnostic thresholds have evolved over time, complicating comparison across studies and eras, and prevalence estimates vary widely with methodology and setting. Finally, the translation of ecological and interkingdom insights into practical, targeted preventive strategies remains at an early stage. These gaps define important directions for the field, and they are also directly relevant to clinical and microbiological research on ECC in specific populations, including Romania.

CONCLUSIONS AND FUTURE DIRECTIONS

Early childhood caries is a common, preventable and socially patterned disease that is best understood as a continuum extending from a dysbiotic dental biofilm to clinical cavitation. Its microbiological determinants — a structured biofilm undergoing ecological shift toward acidogenic and aciduric organisms such as mutans streptococci, lactobacilli and *Candida albicans* — are inseparable from its dietary, host and behavioral determinants, and both sets of factors act through the same underlying balance between demineralization and remineralization. This integrated, ecological perspective explains the clinical features and progression of ECC and provides a rational basis for early, risk-based and family-centered prevention.

Future work should prioritize longitudinal and mechanistic studies that clarify the causal dynamics of biofilm dysbiosis in young children; the further validation and clinical integration of severity-sensitive diagnostic systems such as ICDAS alongside conventional indices; the exploration of targeted anti-biofilm and interkingdom strategies for prevention; and population-level research and public health measures addressing the social determinants that drive the persistently high burden of ECC in settings such as Romania. Bridging clinical observation and microbiological insight — that is, connecting the microbiological origins of the disease to its clinical endpoint — offers the most promising avenue for reducing the burden of this important childhood disease.

Author Contributions

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Conflicts of Interest

The authors declare no conflict of interest.

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