Submitted: 08 January, 2026 Accepted: 02 March, 2026 Published: 03 May, 2026 DOI:10.22514/jocpd.2026.055

Journal of Clinical Pediatric Dentistry

REVIEW

Emerging role of *Scardovia wiggsiae* as an oral pathogen in early childhood caries: a scoping review

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Abstract

Early childhood caries (ECC) and severe ECC (S-ECC) remain significant public health concerns. Emerging evidence indicates that *Scardovia wiggsiae* (*S. wiggsiae*), an acidogenic and aciduric bacterium, has been frequently detected in children with progressive carious lesions. To investigate this association, a scoping review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines. A comprehensive literature search was conducted in PubMed through December 2025 using Medical Subject Headings (MeSH) and free-text terms combined with Boolean operators. English-language human case-control studies involving children $\leq$6 years comparing ECC and S-ECC with caries-free controls were included. Eight studies met the eligibility criteria and were analysed. The observations indicated that the prevalence of *S. wiggsiae* ranged from 45% to 90.6% in ECC and 18% to 93.3% in S-ECC, compared with 2% to 90% in caries-free children. An increase in the bacterial abundance in caries-affected groups compared with caries-free controls was evident. Frequent sucrose consumption was associated with ECC and increased detection of *S. wiggsiae*. Co-detection with *Streptococcus mutans* (*S. mutans*) was commonly reported, suggesting ecological enrichment within cariogenic biofilms. To conclude, this review indicates that *S. wiggsiae* is frequently detected in association with ECC and S-ECC, notably in sugar-rich and acidic environments. The organism is detected in both caries-affected and caries-free children, although the quantitative analyses indicate that bacterial load correlates significantly with disease severity more than simple detection. However, study heterogeneity and limited longitudinal data in young children restrict definitive conclusions.

Keywords

Acidogenic bacteria; Biofilm; Dental plaque; Early childhood caries; Oral microbiome; *Scardovia wiggsiae*

1. Introduction

1.1 Overview of early childhood caries

Early childhood caries (ECC) is a multifactorial disease known by various synonyms such as nursing bottle caries, baby bottle tooth decay, and milk bottle syndrome. ECC is defined as the presence of one or more decayed, missing, or filled tooth surfaces in any primary tooth in a child at 71 months of age or younger. The striking clinical appearance of ECC is the rampant involvement of multiple surfaces of newly erupted primary teeth. ECC is typically classified into mild, moderate, and severe forms. The most advanced of the three forms, severe early childhood caries (S-ECC), is characterised by widespread carious involvement of most primary teeth, including the mandibular incisors [1]. S-ECC affects young children in an atypical and rampant pattern of dental caries, leading to agony from considerable pain, subsequently leading to difficulty in eating and talking [2–5].

ECC occurs in infants and pre-schoolers and often progresses rapidly. It disproportionately affects vulnerable populations, with consequences extending beyond individual disease to population-level health inequality. The prevalence of ECC varies widely depending on socioeconomic status, geographic location, and cultural factors. The prevalence of ECC ranges between 1% to 12% in developed countries, and as high as 70% in developing countries, and among certain disadvantaged groups of children [2–4]. This shows that the disease disproportionately affects children from low-income and underserved populations, highlighting marked disparities in oral health. National surveys report ECC prevalence of 36% in Greece, 45.8% in Brazil, 51.9% in India, 64.7% in Israel, and up to 83% in the United Arab Emirates. ECC frequently affects boys more than girls and is most prevalent in children aged 3 to 4 years [1].

In addition to localized tooth destruction, ECC significantly compromises oral health-related quality of life. The affected children frequently experience pain, infection, disturbed sleep, and difficulty in eating, which may impair adequate nutrition and growth in severe cases. ECC often leads to significant oral health complications and systemic manifestations, such as malnutrition and gastrointestinal disturbances, particularly among children in underdeveloped and developing regions, thereby posing a substantial burden on maternal and child healthcare services [6–11]. Therefore, ECC should be regarded not only as a dental condition, but as a broader paediatric health concern and a major public health problem requiring preventive and community-level interventions.

1.2 Importance of microbial factors in ECC

The primary factors determining the initiation and progression of ECC include prolonged exposure and high intake of fermentable sugars, diet and feeding practices, the cariogenic pathogens harboured, and poor oral hygiene practices [6–8]. *Streptococcus mutans* (*S. mutans*) is recognised as the primary pathogen isolated from the cultivable dental plaque biofilm of children affected by ECC [12]. The other species detected at lower relative abundance include the *Bifidobacterium*, *Actinomyces*, *Veillonela*, *Fusobacterium*, *Lactobacilli*, *Campylobacter*, *Gemella*, *Selenomonas*, *Neisseria*, *Capnocytophaga*, *Abiotrophia*, and *Granulicatella* [13–15]. In addition, the latest literature highlights the multifaceted roles of fungal species, *viz. Candida albicans* (*C. albicans*) and *S. mutans*, contribute to the poly-biofilm infections in ECC [16].

The initiation and progression of ECC are closely linked to dynamic changes in the oral microbiota. Healthy enamel surfaces are initially colonized by *non-mutans Streptococci*, *Streptococcus sanguinis* (*S. sanguinis*), *Streptococcus oralis* (*S. oralis*), *Streptococcus mitis* (*S. mitis*), *Actinomyces*, and other viridans group *Streptococci*. During plaque maturation, the microbial community shifts toward dominance by *Mutans Streptococci* and *Actinomyces* species, with pH fluctuations shaping the microbial composition [17].

Mild and infrequent acidification is countered by homeostatic remineralization processes, maintaining microbial balance. However, frequent or prolonged acidic conditions favour the selection of aciduric organisms such as *Bifidobacterium*, *Mutans Streptococci*, and *Lactobacilli*, while *non-mutans Streptococci* and *Actinomyces* decline. These acid-tolerant microbes undergo phenotypic and genotypic adaptations, including increased proton impermeability, enhanced stress-response mechanisms, and production of alkali via the arginine deiminase system, which collectively facilitate their persistence and contribute to caries progression. Progressive carious lesions show increased microbial diversity, with communities including *S. mutans*, *S. mitis*, *S. oralis*, *S. sanguinis*, multiple species of genus *Veillonella* (*Veillonella spp.*), *Granulicatella*, *Bifidobacterium*, *Scardovia wiggsiae* (*S. wiggsiae*), *Actinomyces spp.*, *Lactobacilli*, and yeasts. In cavitated enamel and dentinal lesions, *S. wiggsiae* and other aciduric bacteria become prominent, highlighting their role as secondary colonizers in the ecological shift from health to disease [17].

It is evident from the aforementioned observations that *S. mutans* serves as a primary etiologic agent in ECC, rapidly establishing plaque biofilms on tooth surfaces in the presence of dietary sucrose and driving cariogenic processes. Building on this biofilm foundation, *C. albicans* can colonize and integrate into the developing plaque, interacting synergistically with *S. mutans*. Recent evidence suggests that together they form a dual-species biofilm characterized by a shared extracellular matrix that enhances structural stability, nutrient retention, and metabolic cross-feeding. *C. albicans* has been shown to induce glucan-synthesizing genes in *S. mutans*, increasing extracellular polysaccharide production, antimicrobial resistance, and overall biofilm virulence. These cooperative interactions further amplify the pathogenic potential of the biofilm, contributing to the progression and severity of ECC [16].

1.3 Emergence of *Scardovia wiggsiae* as a cariogenic pathogen

In 2011, Tanner *et al.* [18] conducted a study on children with S-ECC with the primary objective of evaluating the microbiota and identifying other species associated with S-ECC beyond the *S. mutans*. Historically, culture-based studies identified microorganisms phenotypically, often only to the genus level. In contrast, the authors employed molecular methods, including Polymerase Chain Reaction (PCR) and 16S rRNA gene sequence analysis, enabling the detection of a broader diversity of microorganisms at the species level. Subsequently, Tanner *et al.*'s [18] study led to the discovery of a novel species associated with S-ECC, *viz.* "*Scardovia wiggsiae*". Following this, several studies were conducted to divulge the role of *S. wiggsiae* in various paediatric subjects and other pathologies of the oral cavity [12].

In view of the existing evidence, this scoping review aimed to evaluate published studies on *Scardovia wiggsiae* and its role in the development and progression of ECC in the paediatric population. The review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines [19].

2. Methodology

2.1 Study design

This review was conducted to evaluate the association between *S. wiggsiae* and ECC/S-ECC in children aged 6 years and below. The review followed the PRISMA-ScR guidelines to ensure methodological rigour, transparency, and reproducibility [19]. The review involved a structured literature search, eligibility screening, and a systematic data extraction process.

2.2 Literature search strategy

A systematic electronic literature search was conducted in the PubMed database for studies published through December 2025. The search terms included both free-text and MeSH keywords related to the population, condition, and microorganism. Terms were combined using Boolean operators (AND/OR) and applied to the title and abstract fields, with limits set to

English language and human studies. The search strategy was structured as: (child OR preschool OR infant OR paediatric) AND ("early childhood caries" OR "severe early childhood caries" OR ECC OR S-ECC) AND ("*Scardovia wiggsiae*" OR "Bifidobacterium").

2.3 Eligibility criteria

Inclusion Criteria:

- Children aged 6 years and below;
- Case-control studies comparing ECC or S-ECC with caries-free controls;
- Studies using molecular detection methods (PCR, quantitative PCR (qPCR), or 16S rRNA sequencing) for microbial identification;
- Studies reporting prevalence or quantification of *S. wiggsiae*.

Exclusion Criteria:

- Participants older than 6 years or mixed populations without separable paediatric data;
- Non-case-control study designs;
- Studies not using molecular microbial identification;
- Studies unrelated to ECC or S-ECC;
- Duplicate publications or inaccessible full-text articles.

2.4 Study selection

A total of 58 records were identified through database searching (Fig. 1). Two reviewers independently screened the titles and abstracts. Fourteen original studies were subjected to full-text review. Eight studies fulfilled the eligibility criteria and were selected in the final analysis. Disagreements were resolved through discussion.

2.5 Data extraction

Data were extracted manually and recorded in Microsoft Excel spreadsheets (**Supplementary Table 1**, Ref. [18, 20–26]; **Supplementary Table 2**, Ref. [18, 20–22, 26]). Two independent reviewers performed the extraction to ensure accuracy and consistency, following established protocols for scoping reviews (PRISMA-ScR). The variables collected from each study included: study design, sample size (cases and controls), participant age and gender distribution, sample type, microbial detection method, prevalence or quantification of *S. wiggsiae*, co-detection with *S. mutans*, caries experience measured using decayed, missing, and filled teeth/surfaces (dmft/dmfs) scores, and any reported statistically significant associations. The compiled dataset was cross-checked and verified by both reviewers to minimize errors and discrepancies, ensuring a high standard of data reliability suitable for subsequent analysis and tabulation.

3. Microbial characteristics of *Scardovia wiggsiae*

"*Scardovia wiggsiae*" was named in honour of the renowned American Microbiologist Lois Wiggs for her contributions to the field of microbiology. Scardovia is one of the seven genera within the family *Bifidobacteriaceae*. It is a relatively new bacterial genus, delineated from *Bifidobacterium* in 2002 following the discovery of genomic sequence divergence (Jian and Dong, 2002) [27]. The phylotype Scardovia C1 was described by Munson *et al.* [28] in 2004 in a study of the microbiota associated with dentinal caries based on 16S rRNA gene sequence comparisons [29]. Downes *et al.* [30] first described the *Scardovia wiggsiae sp. nov.* based on five strains isolated from the human oral cavity in 2011.

3.1 Light microscopy

S. wiggsiae is characterised as an anaerobic, non-sporing, non-motile gram-positive bacterium. The bacterial cells exhibit pleomorphism, exhibiting straight, slightly curved, or club-shaped forms, and their dimensions range from 0.6 to 0.7 μm $\times$ 1.6 to 4 μm. They appear in pairs or singly, or in short chains with some branched and diphtheroid arrangements. The bacterial colonies on the culture media appear grey, off-white or cream-coloured, 0.4 to 1.2 mm in diameter, circular, irregular, undulated, convex, and opaque [30].

3.2 Biofilm formation

The ultrastructural morphology of *S. wiggsiae* and its biofilm was first studied in depth by Bossù *et al.* [12] in 2020. Ultrastructural analysis showed that *S. wiggsiae* cells within biofilms were elongated, cigar-shaped bacilli with rounded poles and lacked surface appendages, with mean dimensions of approximately 1.18 μm in length and 0.38 μm in diameter.

The surface texture of the biofilm produced by *S. wiggsiae* appeared to be compact, spongy, and granular. The compact biofilm appeared coarse and punctuated by holes, corresponding to openings of a micro-canalicular system. The spongy areas of the biofilm resembled a trabecular structure. The granular areas of the biofilm surface seemed like a carpet of digitiform formations (similar to a coral reef) of varying thickness and irregular prismatic shape. In the loosely structured regions of the biofilm matrix, numerous packed bacilli were observed arranged in a carpet-like structure [12].

It can be inferred from the above ultrastructural observations that the compact, spongy, and granular biofilm architecture reflects a structurally organized microbial community capable of maintaining localized microenvironments. The presence of micro-canalicular openings and densely packed bacilli may facilitate nutrient diffusion, metabolic exchange, and retention of acidic by-products within the biofilm matrix. Such structural characteristics are consistent with enhanced biofilm stability and persistence under low-pH conditions, supporting the ecological enrichment of *S. wiggsiae* in advanced carious lesions, rather than implicating it as a primary initiator of disease. In accordance with these ultrastructural and ecological observations, a recent study demonstrated an association between *S. wiggsiae* and Visible Occlusal Plaque Index scores in children. It was noted that the prevalence of *S. wiggsiae* increased markedly with greater plaque accumulation, reaching levels comparable to *S. mutans* observed in the heavily plaque-covered occlusal surfaces of molars, suggesting a possible association with dense occlusal biofilms [31].

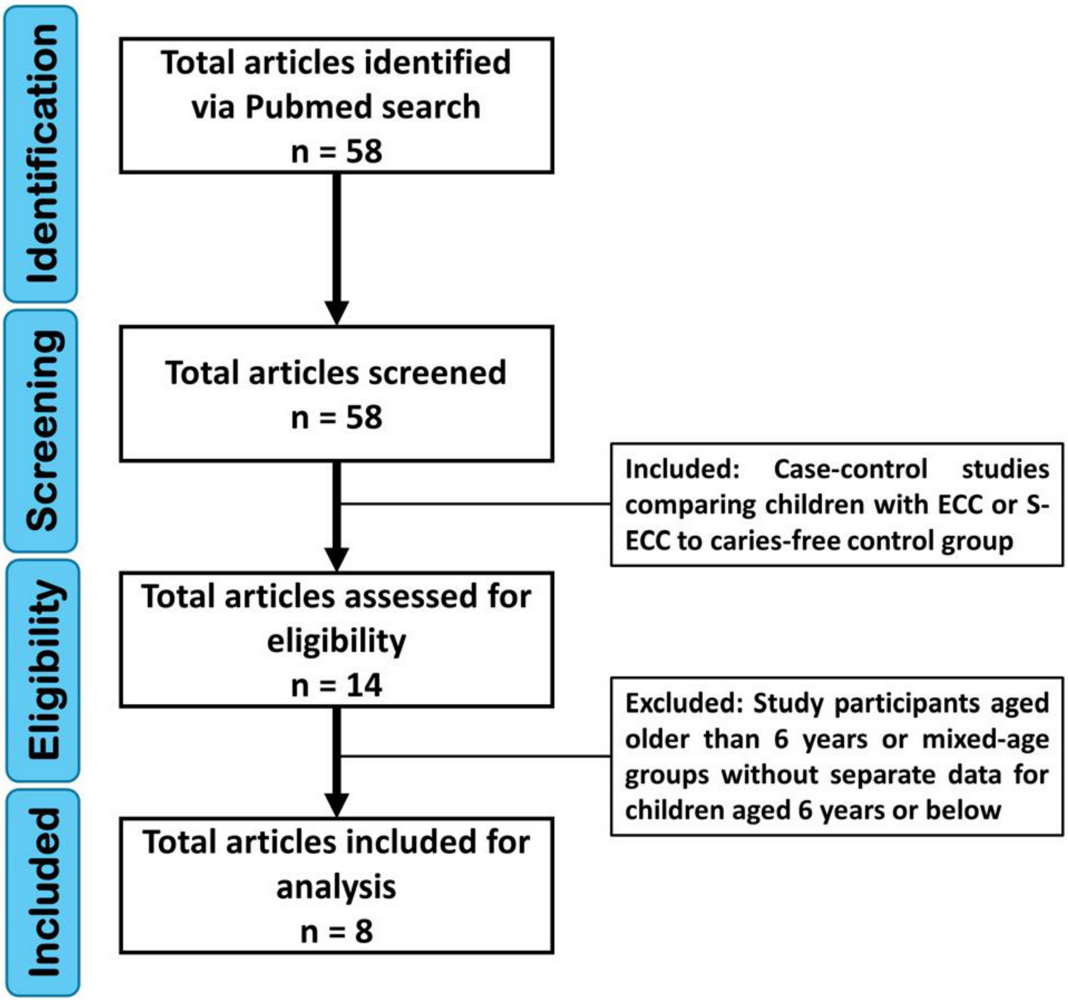

FIGURE 1. Literature review and screening process of the included studies. ECC: Early childhood caries; S-ECC: Severe early childhood caries.

3.3 Sugar metabolism

S. wiggsiae ferments glucose to chiefly produce acetic acid. The metabolic framework of *S. wiggsiae* appears to favour a modified version of the fructose-6-phosphate phosphoketolase (F6PPK) pathway (also known as the bifid shunt or phosphoketolase pathway) over the classical Embden-Meyerhof-Parnas (EMP) glycolytic route typically utilized by many streptococcal species. In this alternative pathway, fructose-6-phosphate is cleaved enzymatically by transaldolase and transketolase, directing a portion of the carbon flux toward acetate production via acetyl phosphate, rather than proceeding exclusively through pyruvate to generate lactate or formate. Genomic analyses of *S. wiggsiae* support this metabolic orientation, revealing the gene homologs encoding key enzymes of the F6PPK shunt, including transaldolase and transketolase, consistent with its proposed central role in carbohydrate metabolism [29].

In a study evaluating oral microbial profiles in relation to free sugar intake, qPCR analysis indicated higher detection of *S. wiggsiae* among individuals consuming $\geq$5% of total energy from free sugars compared with those consuming $<$5% [32]. Another study reported that flavoured milk, which typically contains approximately 5% sucrose, has been associated with increased acid production in dental plaque, potentially contributing to cariogenic conditions within the oral biofilm. The frequency and quantity of flavoured milk consumption among children with ECC may, therefore, be linked to the increased detection of acidogenic and aciduric bacteria, such as *S. mutans*, *S. wiggsiae*, and *Lactobacillus spp.* [20].

Emerging evidence suggests that alternative non-cariogenic sweeteners may effectively suppress the growth and metabolic activity of *S. wiggsiae*. Erythritol and xylitol, individually and in combination, have been shown to inhibit clinical isolates of both *mutans streptococci* and *S. wiggsiae* [33]. More recently, rubusoside, a natural sweetener, demonstrated significant bacteriostatic effects against *S. wiggsiae*, reducing its growth, acid production, and biofilm formation [34]. These findings support the potential of such sugar alternatives as preventive agents in caries management strategies targeting emerging pathogens like *S. wiggsiae*.

3.4 Influence on pH and acid tolerance

Dental plaque pH is a key determinant in the initiation and progression of dental caries. Stephan curves show that after sugar intake, plaque pH can drop below the critical level of 5.5, leading to enamel demineralization. In caries-active children, baseline pH may already be low (~5.5), dropping further to

~4.5 or lower. Notably, *S. wiggsiae* can survive and produce acid at extremely low pH levels. This aciduric trait enables it to persist and function in environments inhospitable for many bacteria. Its survival in such acidic conditions supports its role in caries progression, particularly in severe or advanced lesions [33–35].

Metabolically, *S. wiggsiae* predominantly produces acetic acid as its principal end-product, distinguishing it from many classical cariogenic bacteria, such as *S. mutans*, which primarily generate lactic acid. Sugar metabolism in *S. wiggsiae* involves the F6PPK shunt, with transaldolase and transketolase directing carbon flow toward acetate formation. Smaller amounts of formic and lactic acids are also produced, with pH-dependent metabolic shifts favouring lactic acid production under acidic conditions. Notably, acetic acid exists chiefly in a non-ionised form at a lower pH than lactic acid, which may facilitate deeper diffusion into dental hard tissues and enhance demineralization. Collectively, these metabolic characteristics provide a mechanistic explanation for the frequent detection of *S. wiggsiae* in deep dentinal lesions and support its association with caries progression, rather than lesion initiation [29].

Non-cariogenic sweeteners may influence caries risk primarily by reducing the availability of fermentable substrate and limiting sustained plaque acidification, thereby altering the ecological conditions that favour aciduric bacteria such as *S. wiggsiae* [33, 34]. In addition, reducing the frequency of free sugar intake may help prevent prolonged low-pH episodes, restricting metabolic activity and selective expansion of acidogenic species within the biofilm.

3.5 Ecological niche of *S. wiggsiae* in dental caries

S. wiggsiae is an acidogenic and aciduric bacterium that thrives in low pH environments within the oral cavity, particularly under cariogenic conditions. It is most commonly detected in deep dentinal lesions, where oxygen levels are low, and carbohydrate availability is high, favouring its anaerobic growth. Within the oral biofilm, *S. wiggsiae* shows a strong association with the dentinoenamel junction and deep layers of carious dentin, rather than on the enamel surface [36, 37]. In a recent study, *S. wiggsiae* was detected across diverse oral sites, including tooth biofilms, the tongue dorsum, and gingival crevicular fluid. Its presence in both traditional and non-traditional niches suggests a broader ecological role and possible interactions with other oral microbes, warranting further investigation into its contribution to caries progression in children and oral microbial ecology [38].

4. Association of *Scardovia wiggsiae* with ECC

The association of *S. wiggsiae* with ECC and S-ECC was assessed based on findings from the studies summarised in **Supplementary Table 1**. These studies consistently report a higher prevalence and abundance of *S. wiggsiae* in children with ECC than in caries-free controls. The detailed discussion in the following section further explores its potential role in caries development and progression.

4.1 Quantification of *S. wiggsiae* in ECC and S-ECC cases

Detection of *S. wiggsiae* varied substantially across the included studies, with reported prevalence ranging from approximately 45% to 90.6% in ECC and 18% to 93.3% in S-ECC. In caries-free control groups, detection rates also showed marked variability, spanning from approximately 2% to 90.0%, with some studies reporting negligible detection. Analysis of the selected studies (**Supplementary Table 1**) further demonstrated substantial heterogeneity in both prevalence estimates and quantitative measurements across ECC, S-ECC, and control groups, attributable to differences in study populations, sampling sites, and microbial detection methods. Importantly, studies employing quantitative approaches consistently reported higher bacterial loads of *S. wiggsiae* in ECC and S-ECC than in caries-free controls, including cases in which prevalence was comparable between groups.

4.2 Association with severity and caries progression

S. wiggsiae has been reported predominantly in advanced carious lesions, suggesting an association with later stages of childhood caries, rather than early lesion initiation. Several studies also noted increased microbial heterogeneity, with enrichment of low-abundance background taxa as the oral microbial community shifts toward dysbiosis, reflecting the complex ecological changes accompanying caries progression [35]. Consistent with the Ecological Plaque Hypothesis, findings from the included studies (**Supplementary Table 1**) indicate that *S. wiggsiae* can be detected in both caries-free and caries-affected children, with relative abundance increasing in association with disease severity. This pattern is best interpreted as reflecting ecological selection under conditions, such as frequent sugar exposure and sustained plaque acidification, which may favour the persistence of aciduric species such as *S. wiggsiae* within a dysbiotic biofilm environment.

4.3 Correlation with sugar-rich dietary habits

One study reported that flavoured milk containing approximately 5% sucrose was associated with increased plaque acid production, favouring acidogenic bacteria such as *S. mutans*, *S. wiggsiae*, and *Lactobacillus spp.* in children with ECC [20]. Studies by Tanner *et al.* [18] and Pan *et al.* [21] reported associations between ECC and the frequency of consumption of sugar-rich foods and beverages. Similar observations were described by Tantikalchan *et al*. [22], who reported S-ECC among children with frequent consumption of sugar-coated crackers. Collectively, these findings indicate that frequent consumption of sugary solid and liquid foods and beverages is associated with ECC. Notably, several studies have reported an association between *S. wiggsiae* detection and high sucrose exposure, consistent with its preferential persistence under acidic, sugar-rich biofilm conditions [32].

4.4 Co-occurrence with *Streptococcus mutans*

Co-detection of *S. mutans* and *S. wiggsiae* (**Supplementary Table 2**) was more frequently reported in children with ECC and S-ECC compared with caries-free controls. Several studies observed higher ECC prevalence among children harbouring both species compared with those harbouring only a single species. This pattern of co-detection was more frequently observed in ECC groups than in caries-free children, possibly reflecting a shared adaptation to cariogenic biofilm conditions, rather than direct microbial interaction, warranting further investigation to clarify the nature of this association.

Experimental studies have demonstrated that *S. wiggsiae* exhibits acidogenic activity comparable to *S. mutans* under both neutral and acidic conditions. Animal model studies indicate that *S. wiggsiae* alone demonstrates limited colonisation and minimal caries induction. In contrast, co-inoculation with *S. mutans* has been associated with increased colonisation and lesion development [34]. These findings are best interpreted as supporting an ecological model in which *S. mutans* is associated with early biofilm acidification; concurrently, *S. wiggsiae* may become enriched under the resulting acidic conditions, consistent with its aciduric phenotype, rather than implying a direct synergistic etiologic interaction.

4.5 Additional risk indicators for ECC

In addition to microbial findings, several host, behavioural, and environmental factors were reported to be associated with ECC and higher detection of *S. wiggsiae*. Children with parents who had active dental caries ($p = 0.027$) [18], those born prematurely ($p = 0.04$) [22], and those with a history of prolonged bottle feeding ($p = 0.001$) [22] showed higher ECC prevalence and increased detection of *S. wiggsiae*. Furthermore, infrequent dental check-ups ($p = 0.005$) [20] and a higher frequency of carbohydrate intake ($p < 0.05$) [18, 21, 22], whether in liquid form or as solid snacks, such as sugar-coated crackers, were also associated with elevated *S. wiggsiae* levels. Collectively, these findings suggest that familial, behavioural, and dietary factors may influence the oral microbial ecology in young children and are associated with conditions under which aciduric species such as *S. wiggsiae* are more frequently detected.

5. Inference and conclusions

The prevalence of *S. wiggsiae* showed wide variability across studies (**Supplementary Table 1**), ranging from approximately 45% to 90.6% in children with ECC and 18% to 93.3% in those with S-ECC. In caries-free children, reported prevalence ranged from approximately 2% to 90.0%, with some studies noting negligible detection. Notably, one study reported comparable prevalence between S-ECC (93.3%) and control groups (90.0%); however, quantitative analysis revealed substantially higher bacterial loads in the S-ECC group (1.40×10^9 cells/mL) than in the control group (1.49×10^8 cells/mL). This observation suggests that disease severity may be more closely associated with bacterial abundance than with simple detection. In addition, studies summarized in **Supplementary Table 1** demonstrated increased detection or abundance of *S. wiggsiae* in caries-affected children, supporting an association between higher bacterial burden and caries severity. The differences between ECC, S-ECC, and caries-free groups were more consistently observed in studies employing quantitative detection methods than in those relying on presence-based approaches.

The selected studies reported higher *S. wiggsiae* abundance in groups with elevated dmfs/dmft scores, suggesting a stronger association between bacterial load and caries severity than with mere detection. Although gender distribution was reported across studies, no consistent association was observed between *S. wiggsiae* abundance and gender, limiting the interpretation of gender-related susceptibility. Additionally, host-related factors, including parental caries history, premature birth, prolonged bottle feeding, and irregular dental check-ups, were reported across studies as contextual risk indicators associated with ECC and higher detection of *S. wiggsiae*, further supporting a multifactorial and ecological interpretation of disease progression.

The observed associations between *S. wiggsiae*, dietary habits, and co-detection with other cariogenic species are best interpreted within the framework of the Ecological Plaque Hypothesis proposed by Philip D. Marsh (1994) [39]. This model emphasizes that dental caries arises from environmentally driven shifts in the balance of the oral microbiota, particularly in response to frequent sugar exposure and sustained reductions in plaque pH, rather than from the action of a single pathogen. In this context, frequent consumption of sucrose-rich foods and beverages may contribute to prolonged acidic conditions that select for aciduric organisms such as *S. wiggsiae*. The frequent co-detection of *S. wiggsiae* with *S. mutans* (**Supplementary Table 2**) in ECC and S-ECC groups likely reflects shared adaptation to these cariogenic ecological conditions, rather than direct mechanistic synergy between the species. Within such an ecological framework, *S. mutans* may be associated with early biofilm acidification, while *S. wiggsiae* appears to become enriched as lesions progress and the environment becomes increasingly acidic. In addition, dental caries is widely regarded as a multifactorial and polymicrobial disease, in which shifts in overall microbial community structure, rather than dominance of a single pathogen, contribute to disease progression [40, 41]. The caries-associated dysbiosis reflects coordinated changes among multiple taxa driven by environmental pressures [42]. Therefore, the enrichment of *S. wiggsiae* in ECC and S-ECC is best interpreted as part of a broader polymicrobial response to sustained acidic conditions, rather than evidence of a direct etiologic role.

In conclusion, the evidence from the included studies indicates frequent detection of *S. wiggsiae* among children with ECC and S-ECC. Its capacity to persist under acidic conditions, frequent co-detection with *S. mutans*, and reported associations with dietary and host-related factors suggest its involvement within a dysbiotic oral environment, rather than a singular etiologic role. These observations highlight the importance of considering microbial profiles alongside behavioural and biological risk factors when developing preventive approaches for ECC.

6. Limitations and future perspectives

The findings of this scoping review should be interpreted with caution, as the included evidence primarily comprises case-control studies with small sample sizes and substantial heterogeneity in study populations, sample types, and microbial detection methods. Variability in outcome measures, including prevalence, bacterial load, and gene expression, limits direct comparability and precludes quantitative synthesis. In addition, the predominance of cross-sectional designs and limited original research in children under 6 years restricts inference on temporal dynamics. Future longitudinal studies employing standardized methodologies are needed to clarify the role of *S. wiggsiae* in ECC progression and to inform targeted preventive strategies in high-risk paediatric populations.

7. Highlights

• Quantitative abundance of *S. wiggsiae* shows a stronger association with disease severity, rather than mere detection.

• Higher levels of *S. wiggsiae* were observed in children with a habit of frequent consumption of sugar-rich foods and beverages, particularly solid carbohydrate-dense items.

• Higher co-detection with *S. mutans* was commonly reported among ECC and S-ECC participants, indicating a possible association with increased caries severity and advancement.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analysed during this study are included in this article. No additional datasets were generated.

AUTHOR CONTRIBUTIONS

BM—designed the research study. BM and SP—performed the literature search, study screening, data extraction, and tabulation. LT and DDN—analysed and interpreted the data. SP, LT and DDN—contributed to study conceptualization, provided scientific supervision, and critically revised the manuscript. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at https://oss.jocpd.com/files/article/2049718866819334144/attachment/Supplementary%20material.docx.

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How to cite this article: Barnali Majumdar, Shankargouda Patil, Luca Testarelli, Dario Di Nardo. Emerging role of *Scardovia wiggsiae* as an oral pathogen in early childhood caries: a scoping review. Journal of Clinical Pediatric Dentistry. 2026; 50(3): 1-8. doi: 10.22514/jocpd.2026.055.