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Review Article

Scardovia wiggsiae: A key cariopathogen

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Abstract

Dental caries (DC), a terminology used to describe a complex illness spawned by several factors which involves the deterioration of the hard tissues of the oral cavity i.e. the teeth due to the presence of various bacteria. Several bacteria, including Streptococcus mutans, Actinomyces, and Lactobacilli species, have been shown to have a role in the development of DC. *Scardovia wiggsiae* (*S. wiggsiae*), a novel cariopathogen, has been discovered in children suffering from early childhood caries (ECC), even in conditions where commonly found cariopathogens are not present. This new bacterium is a member of the *Bifidobacteriaceae* family and is often present in the human oral cavity. The aim of the present review is to provide a comprehensive exploration of the microbiology of *S. wiggsiae* and its connection to dental caries. The discussion will include the characteristics of this novel species, including its interactions with other microorganisms, its ability to tolerate acidic conditions, and its potential to cause tooth decay. This may have an impact on future research and development of novel diagnostic tools and treatment methods targeted at controlling this recently identified microorganism.

Keywords: Bifidobacteriaceae, Cariopathogen, Early childhood caries, Dental caries, Scardovia wiggsiae.

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1. Introduction

Dental caries (DC) is a permanent microbiological condition that damages the teeth's hard tissues. It is characterized by the loss of minerals from the tooth's inorganic component and the degradation of its organic content, which usually results in the creation of cavities.[1] Alternatively, it has also been described as a disease of the oral cavity that is caused by several factors. According to Newburn's Tetrad (1982), dental caries is influenced by the host, microbes, nutrition, and time. Microbes are particularly crucial in the formation of DC. Some of the often-linked pathogens with caries include *Streptococcus mutans* (*S. mutans*), *Lactobacilli species*, *Actinomyces species*, and others.[2]

When considering severe types of DC, such as Early Childhood Caries (ECC), *S. mutans* might make up more than 30% of the microbial culture population.[3-5] Research conducted in 2011 by Tarnow *et al.* under anaerobic culture conditions identified a previously unknown species called *Scardovia wiggsiae* (*S. wiggsiae*) as the causative agent of ECC.[6] *Scardovia* is one of the seven genera under the *Bifidobacteriaceae* family. A novel bacterial genus was delineated from the genus Bifidobacterium in 2002 based on genomic sequence discrepancies.[7] *S. wiggsiae* was identified in severe ECC as clone CX010 in 2002. Although knowledge regarding the metabolic pathways of *S. wiggsiae* is scarce, the species predominantly generates acetate from glucose. This indicates that, similar to oral *Bifidobacterium* species, *S. wiggsiae* has a distinct metabolic pathway known as the fructose-6-phosphate pathway (F6PPK shunt), which

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contrasts with the glycolytic system of the caries-associated *S. mutans*.[8-10]

The characteristics of *S. wiggsiae* indicate its acidogenic tendencies. The final products of glucose fermentation are acetic acid and lactic acid. Acid production decreases intraoral pH, resulting in microbial dysbiosis favouring acidity. Additional traits encompass an acid-producing capacity that is comparable to or exceeds that of *S. mutans*. *S. wiggsiae* are arginine deaminase negative, hence failing to generate ammonia to counteract the reduced intraoral pH.[11-14] This study focuses on the microbiology of this newly discovered species and its connection and contribution to the development of DC.

2. Materials and Methods

2.1. Information sources

Electronic and manual searches were conducted using various established databases, including The National Library of Medicine (MEDLINE PubMed), Cochrane Library, EBSCOhost-Dentistry and Oral Sciences Source, and Google Scholar. Grey literature was accessed and carefully examined, along with the bibliographies of the studies and reviews that were included, to identify any additional potential references. Articles published till July 31, 2024 were searched.

2.2. Search strategy

The key words used for the search strategy included the following:

1. *Scardovia wiggsiae*
2. Dental caries
3. Cariogenic pathogen
4. Cariogenic microorganism
5. Early childhood caries

The search terms adhered to the syntax rules specific to each database. Additional search terms were incorporated into the review using Boolean operators (OR, AND) to combine keywords. A thorough examination was conducted on all cross-reference lists of selected studies to identify any additional articles that could potentially meet the eligibility criteria of the study. Filters were employed at the conclusion of the search to restrict the results based on English language and time constraints (**Figure 1**).

2.3. Selection process

During the initial screening, two reviewers carefully examined the titles and abstracts of the publications that were found in relation to the topic at hand. A full-text report was obtained if the information that met the topic was present in the abstract, or if the title was relevant but the abstract was not accessible. A thorough examination of the articles was conducted to determine which ones satisfied the specified criteria for inclusion. Excluded from the study were reports that were only presented as abstracts. This is because previous evidence has indicated that there may be differences between the data reported in an abstract and the data provided in the final published full report. To reduce the potential for publication bias, the researchers reached out to the authors of relevant abstracts to inquire about the availability of a full text report of the study, regardless of whether it had been published or not.

3. Results of Search Strategy

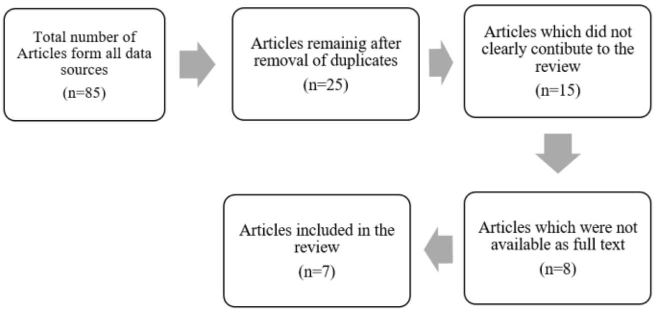

Figure 1: The results of the search strategy

4. Characteristics of *Scardovia wiggsiae*

One of the seven genera of the *Bifidobacteriaceae* family's Gram-positive, anaerobic, non-sporing, and non-motile bacilli is *S. wiggsiae*. The name Scardovia is derived from Vittorio Scardovi, an Italian microbiologist who made notable contributions to the understanding of *Bifidobacteria*. Meanwhile, the term "*wiggsiae*" is derived from Lois Wiggs, a microbiologist from America renowned for her contributions to anaerobic microbiology.[15] In 2002, a new bacterial genus was identified and classified separately from the genus *Bifidobacteriaceae*. This separation was based on a difference in the genome sequence.[16,17] According to the Human Oral Microbiome Database (HOMD), it is classified as the 195th human oral taxon.[18] Obtained from the mouth of people, this microbe may be cultivated on anaerobic blood agar plates. The colonies should be immersed in brain-heart infusion (BHI) broth containing 5 g of yeast extract per liter and cultured for 24 hours at a temperature of 37 degrees Celsius with a carbon dioxide concentration of 5%.[1] Strains have been obtained from both occlusal and root caries.[20,21] The commonly used technique is real-time polymerase chain reaction (RT-PCR). Children with ECC and S-ECC have elevated amounts of this bacterium.[22,23] A study conducted by Chandna et al[22] indicated that 86.7% of individuals with severe early childhood caries (SECC) were discovered to be infected with this bacterium, whereas only 60% of children with early childhood caries (ECC) were found to have *S. wiggsiae*. Surprisingly, a small number of infants in the caries-free group were found to have this microbe.

It is classified under the domain bacteria, phylum Actinobacteria, class *Actinobacteridae*, order *Bifidobacteriaceae*, genus *Scardovia*, and species *wiggsiae*. The width is around 0.6-0.7µm, while its length ranges from

1.6 to 4µm. It is often pleomorphic, meaning it may vary in shape. It can be slightly curved and arranged in a club-like manner, either alone, in pairs, or in short chains.[20,21,26] Varying environmental factors may alter the morphology of cells, causing them to transition from one type to another. The cell color varies from an off-white shade to cream or even gray.[16] The genomic structure of *S. wiggsiae* comprises 1.55 megabase pairs (MBP) and includes 1243 genes. The whole genetic code of *S. wiggsiae* F0424[25] was sequenced in 2012. The percentage of guanine+cystine (G+C) in it is 52.9%.[16] Being a Gram-positive bacilli, it possesses a dense peptidoglycan layer composed of A4α L-Lys-Ser-Glu in the peptide linkage, where L-ornithine substitutes L-lysine and threonine partly replaces serine.[16]

S. wiggsiae has been discovered in the oral cavity, namely in microhabitats such as the teeth, gums, tongue, and palate.[18] It is important to note that this bacterium is capable of growing in acidic environments, as shown by its ability to thrive on acid agar plates with a pH of 5. This is particularly relevant since active dental lesions are known to occur in acidic conditions.

Additionally, a variant of *S. wiggsiae* was detected in the arm injury of an individual who injects drugs intravenously. It has a preference for mesophilic conditions, with a temperature range of 25 to 45 degrees Celsius.[25]

5. Metabolism of *S. wiggsiae*

S. wiggsiae can metabolize several carbohydrates, including as glucose, sucrose, and galactose, without generating any gas. The bacterium exhibits acidogenic, aciduric, and acid-tolerant characteristics. It is an anaerobic organism that lacks the ability to produce indole, catalase, or hydrogen sulfide. *S. wiggsiae* lacks the ability to break down urea or gelatin, and thus does not reduce nitrogen levels.[16] Based on research conducted on the sugar metabolism of *S. wiggsiae*,[16] the bacterium demonstrates resistance to fluoride as well as lactic and acetic acids, and it exhibits a significant level of acid generation. *S. wiggsiae* lowered the pH of the ambient air to 3.5 by breaking down glucose. Glucose mostly yields acetic acid, with minimal quantities of lactic and formic acid. *S. wiggsiae* has the ability to adapt to acid and fluoride exposures due to its distinctive metabolic route, known as the F6PPK shunt (**Figure 2**). This process ensures the continuous metabolic flow towards the acetic acid pathway, which is resistant to fluoride. *S. wiggsiae* has a strong ecological competitiveness in acidic settings such as caries lesions due to its ability to tolerate and produce lactic acid.

6. Association of *Scardovia wiggsiae* in Dental Caries

Kameda *et al.* discovered that *S. wiggsiae* produced acid from glucose, causing the pH of the surrounding environment to decline to 3.5. This demonstrates both substantial acid production and the ability to withstand acidic conditions.[26] In addition, the acid production rate remained constant at pH 7.0 and 5.5, indicating that *S. wiggsiae's* acid production routes are capable of withstanding acidic conditions. Tanner *et al.* shown that *S. wiggsiae* is capable of flourishing and producing acid in conditions of low pH.[6] Furthermore, *S. wiggsiae* is predominantly an anaerobic bacterium, whereas *S. mutans* is facultatively anaerobic. This shows that *S. wiggsiae* may survive and create acid in mature biofilms with low amounts of oxygen. When *S. mutans* is cultivated on plates using mitis salivarius agar, a specialized medium for *mutans streptococci*, the morphology of the colony is uneven.[27]

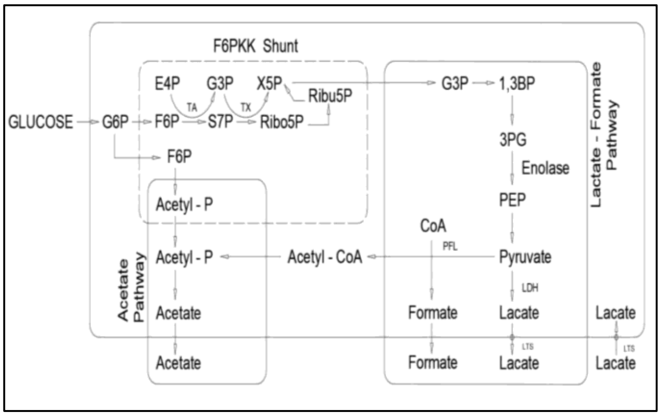

Figure 2: Metabolism of *S. wiggsiae*

The results suggest that acid produced by *S. wiggsiae* may cause the demineralization of hydroxyapatite and speed up the development of DC by reducing the pH of the oral biofilm. Research conducted by Aizawa et al revealed that Mutans *streptococci*, known for their strong cariogenic potential, are capable of reducing the ambient pH to 3.5. This suggests that *S. wiggsiae* exhibits acid production and acid tolerance similar to that of *S. mutans*.[28] This might be one of the factors contributing to the frequent presence of *S. wiggsiae* in caries lesions.[22,29,30] It has been seen in both animal and human trials of persons with ECC, even when *S. mutans* is not present.[6,31]

7. *S. wiggsiae* Acid Tolerance, its Acidogenicity and Arginine Deiminase Activity

Studies have shown that *S. wiggsiae* exhibits tolerance to acid when isolated.[6] The strains exhibit properties of aciduricity as well as acidogenicity similar to those of *S. mutans*,[32,33] as they thrived at pH levels of 7, 5.5, and 5, which were equivalent to *S. mutans*. *S. wiggsiae* has acidogenic capabilities comparable to those of *S. sobrinus*, *S. mutans*, *Actinomyces israelii* and *Actinomyces naeslundii* I and II at acidic circumstances. Therefore, *S. wiggsiae* is a very potent acid producer, comparable to or perhaps surpassing *S. mutans* in its production of acid. In addition, strains of *S. wiggsiae* do not possess arginine deaminase, suggesting that this species is unable to increase the pH via synthesizing ammonia. *S. wiggsiae* was unable to alleviate the consequences of local acid production in plaque biofilms.[31]

8. Invivo cariogenicity of *S. wiggsiae*

Due to its exceptional ability to thrive in acidic conditions and produce acid, as well as its tendency to stick together and accumulate in large quantities on teeth when exposed to sucrose, *S. mutans* is a major cause of DC.[2] Glucans, which are glucose polymers, are created from sucrose by the action of the enzyme glucosyltransferase (Gtf). It is well-known that glucans can generate adhesion that is reliant on sucrose.[34,35] *S. mutans* produces many proteins that may bind glucans. Three glucan-binding proteins (Gbps), namely Gbps A, C, and D, were believed to have a part to play in the cariogenic potential of *S. mutans*.[36] A recent study found that *S. mutans* and *S. wiggsiae* are the primary organisms associated with SECC in both blood and acid agars. However, it is worth noting that *S. mutans* was not detected in any of the children with severe ECC, as revealed by the investigation of caries-associated microbiota in ECC. The strain was injected into rats and *S. wiggsiae* proved positive.[6] The primary microbial species associated with severe early childhood caries (in instances where *S. mutans* was not present) were *Streptococcus gordonii*, *Streptococcus cristatus*, *Actinomyces gerensceriae*, *Dialister invisus*, and *Streptococcus wiggsiae*, as identified by examination of blood agar samples. When *S. mutans* is exposed *to S. wiggsiae*, the cariogenicity of *S. mutans* is heightened compared to when it is present on its own. Additionally, bacterial colonies were obtained from the dentinal tubules of mandibular molars in the experimental rats. The co-occurrence of *S. wiggsiae* together with *S. mutans* may indicate a dual infection paradigm. However, *S. wiggsiae* has a lower tendency to cause tooth decay compared to *S. mutans*, due to its restricted ability to induce lesions in experimental mice. The reduced infection and colonization rates were most likely due to the absence of appropriate attachment mechanisms in *S. wiggsiae*.[37]

Table 1: Characteristics of included studies

S. No.	Author Details	Inferences
1.	Henne et al (2015)[38]	*S. wiggsiae* was more prevalent (61.5%) in caries lesions than in controls (40%), as validated by real-time quantitative PCR and DNA sequencing. The controls had three people with the greatest absolute and relative *S. wiggsiae* counts. *S. mutans* was identical to *S. wiggsiae* in caries lesions (61.5%) but only 10% in controls.
2.	Row et al (2016)[39]	Some patients have higher percentages of *Scardovia* in their oral microbial flora. The microbial interactions that help or hinder *Scardovia* growth in the oral cavity are unknown, indicating the need for long-term studies to better understand demographic or health parameters and disease risk.
3.	Matondkar et al (2020)[40]	*S. wiggsiae* was found in most caries-free and S-ECC-affected children. Their numbers range greatly in both groups, with the S-ECC group having a stronger link to the illness process. *S. wiggsiae* is linked to severe early childhood caries in this research.
4.	Lee et al (2021)[41]	*Scardovia wiggsiae* and *Leptotrichia wadei* were found to be abundant in the caries samples, but *Neisseria oralis* was found to be abundant in the non-caries samples of children under the age of six. This was discovered using differential abundance analysis.
5.	McDaniel et al (2021)[42]	SW was found in n = 71/170 specific oral locations, with 64.7% of SW-positive participant samples harboring SW in more than one oral site, including GCF and the tongue dorsum. It may be the first to use oral site-specific analysis to determine *Scardovia* prevalence and location in clinical patient samples.
6.	Isaac et al (2022)[43]	Under PCR analysis, dentinal caries had the highest incidence of *S. wiggsiae*, followed by plaque and saliva from high-risk people. One prominent bacterium is *Scardovia wiggsiae*.
7.	Tantikalchan et al (2022)[44]	S-ECC had more *S. wiggsiae* and *S. mutans*. The dmft score and gingival index were linked to *Bifidobacterium*, *S. mutans*, and *S. wiggsiae*. Children who only found *S. mutans* had substantially lower dmft scores than those who found two bacteria, *Bifidobacterium*, and *S. mutans*.

9. Future Prospects

It is crucial to determine the precise involvement of this organism in the development and cause of DC. Future research should include further human trials in which this particular organism is isolated and its susceptibility and intensity against other cariogenic bacteria are assessed. Future research should also prioritize investigating the interaction between other therapeutic approaches, including both preventative and interceptive methods, with this specific bacterium. Developing targeted diagnostic tools for the identification of this bacterium might improve the ability to detect it early and respond effectively. In addition, the development of innovative therapeutic approaches that specifically target the unique traits of *S. wiggsiae*, such as its ability to tolerate acidic conditions, might potentially lead to improved prevention as well as management of DC. On the whole, integrating the insights obtained from *S. wiggsiae* studies into clinical practice has the potential to enhance our approach to combating dental caries and achieving superior oral health outcomes. It is important to emphasize that caries only arise when *S. wiggsiae* is present alone, hence therapy focused on *S. mutans* may not be enough for managing the condition. It is necessary to create probiotics that target this particular microbe in order to halt the course of DC in instances caused only by *S. wiggsiae*.

10. Conclusion

In conclusion, the discovery of *S. wiggsiae* as a novel cariopathogen emphasizes the complexity of dental caries, emphasizing that the cause of the disease involves more than the commensal pathogens such as *S. mutans*, *Actinomyces*, and *Lactobacilli*. Despite the lack of these common disease-causing microorganisms, this newly discovered member of the *Bifidobacteriaceae* family has been strongly associated with tooth decay in young children. *S. wiggsiae* is believed to contribute to the occurrence of DC due to its unique properties. These traits include a higher resistance to acidic conditions and the ability to possibly contribute to tooth decay via interactions with other bacteria in the mouth. DC is a complex illness of the oral cavity that involves several factors including bacterial infections. While microbes like *S. mutans* and *Lactobacilli sp.* that are often found in older individuals are known to be important in the development of DC, other species like *S. wiggsiae* also have a prominent role in the occurrence as well as progression of DC. *S. mutans* and *S. wiggsiae* are significant microbial pathogens that contribute to the occurrence of DC. The discovery of *S. wiggsiae* underscores the need for ongoing research into emerging infections and their impact on oral health, with the aim of devising more effective preventive and treatment interventions.

11. Source of Funding

None.

12. Conflict of Interest

None.

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