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Scardovia Wiggisiae - A new etiological agent for early childhood caries

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Abstract

A new micro-organism has been found responsible for causing Early Childhood Caries (ECC) -*Scardovia Wiggisiae* (S.W). It belongs to a genus of unidentified *Bifidobacterium*. The existence of S.W should thus be counted along with *S.Mutans* and *L.Bacilli*. The active lesions of the caries had more S.W count than the non-caries when assessed. Also, it was seen that among those seeking orthodontic care, the incidence of S.W was greater. As S.W is one of the causative species for caries, it may be used as a risk marker for caries. To avoid ECC, we need to find ways to reduce the S.W count. One of the studies showed that incidence of caries can be decreased by using pit and fissure sealant. However, more research is required in this matter.

Keywords: Scardovia Wiggisiae, early childhood caries, Microflora

Introduction

Dental caries in the primary dentition, early childhood caries (ECC), is epidemic worldwide with a global prevalence burden of untreated caries of 8.8% [1] with increased levels in selected populations including 32% very young (16-month-old) American Indian children with cavities [2]. A higher prevalence was observed for 3-year-olds, which ranged from 36 to 85% in the Asian countries of Taiwan, the Philippines and Korea [3].

Most of the clinical studies referring to microbiology of ECC focuses on *mutans streptococci* (MS) and *lactobacilli* (LB), which are routinely detected using selective-culture-based methods [4]. However, the microbiota of biofilms taken from ECC patients has been acknowledged to contain a broad diversity of bacteria. Some studies support the view that caries can develop in the absence of MS [5-7]. Newer molecular methods have suggested that the traditional MS and LB species bear less significance, thereby giving clues that other species may also be responsible for ECC. Thus, several bacterial species, either alone or as a group, other than MS may also play major roles in caries development [5, 8-10].

Microflora associated with ECC

In children with ECC, studies have shown that *Streptococcus mutans* (*S. mutans*) frequently exceeds 30% of the microbial flora in the cultivable biofilm plaque [11-13]. Other species, identified with a lower percentage, belong to *Veillonella*, *Granulicatella*, *Fusobacterium*, *Lactobacilli*, *Neisseria*, *Campylobacter*, *Gemella*, *Abiotrophia*, *Selenomonas* and *Capnocytophaga* [4, 14].

Also, *Actinomyces* and *Bifidobacterium* were found to be associated with initial and deep caries, respectively [8, 15]. In 2011, a study using anaerobic culture conditions allowed the identification of a newly named species, *Scardovia wiggisiae* (S.W) and *Selenomonas noxia* [16], which was significantly associated with severe ECC. S.W was detected also in children without the contemporary presence of *S. mutans*, indicating its exclusive role in this disruptive process [17]. It was classified as an unidentified *Bifidobacterium* species, present within deep dentine caries and in high proportions within infected pulp tissue in children. *Selenomonas noxia* belongs to the *Veillonella* family, which has wide distribution among various animal species [18-20]. Apart from being found in the gastrointestinal tract, it is also found in the oral cavity and may be found in higher levels among patients with poor oral hygiene [21-24]. The

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most recent literature has shown a significant relationship between the presence of *S.W* in the early stages of caries and in pediatric subjects undergoing orthodontic therapy [25-28]. *S.W* has clinical importance in S-ECCs, being a potential risk indicator for the oral health of pediatric patients [29].

Association of *S. Wiggisiae* and *S. Mutans* with Caries

Thomas *et al.* evaluated caries progression by studying demineralization of enamel chips worn in an intra-oral appliance [30]. *S. mutans* was found at higher levels in caries-active than in caries-inactive subjects. In another study, *Parascardovia denticolens* was cultured from the forefront of carious lesions with vitally exposed pulps suggesting this *Scardovia*-related species was associated with lesion progression in dentin [31]. In another study, the major taxon cultured from deciduous pulps was *S.W* (*Bifidobacterium* Ssp2 was *S. wiggisiae* by 16S rRNA sequences) [32]. In a pyrosequencing study, increased relative abundance of *S.W* was higher in dentin caries compared to caries-free sites or initial carious lesions [33]. Together these studies indicate an association of *S.W* and related taxa with dental caries and suggested that further study to examine cariogenic potential was indicated. Together these data on acid-production from *Scardovia* and *Scardovia*-related species indicate that they are strong acid producers, at a similar or greater extent than that of *S. mutans*. Further *S. wiggisiae* strains were arginine deaminase negative indicating the inability of this species to raise the pH from ammonia production.

Metabolism of *S. Wiggisiae*

Like *Bifidobacterium* species, *S.W*, via the F6PPK shunt, metabolizes sugars and produces acetic acid as an acidic end-product. In the entire sequence of its genomes F0424, the gene sequences of two main F6PPK shunt enzymes, transaldolase and transketolase, have been assigned to help F6PPK shunt involvement in *S. sugar* metabolism. The F6PPK shunt is a metabolic pathway in which F6P is produced by glucose phosphorylation.

The enzymatic activities of transaldolase and transketolase are continually degraded until glycolysis via G3P eventually reaches the latter half and produces lactic and formic acids (lactate formate pathway) or is used to transform acetyl phosphate into acetic acid (acetate pathway). Besides acetic acid, *S.W* also developed small quantities of formic and lactic acids, suggesting that both above-mentioned pathways are used. At pH 7.0, only acetic and formic acids were formed, while lactic acid was also formed at pH 5.5 implying that there was a metabolic change in the lactate-formate pathway between lactic and formic acids [34].

It has been documented that lactate dehydrogenase, which is responsible for the formation of lactic acid, acts under acidic conditions in oral streptococci, while pyruvate formate lyase is responsible for the function of formic acid production at neutral pH due to discrepancies between their optimum pH [35]. A previous study says that a higher proportion of acetic acid is non-ionized compared to lactic acid in low pH conditions, thus acetic acid is more likely to penetrate and decalcify enamel from within than lactic acid [36]. This implies that acetic acid-containing bacteria, *S.W*, cause caries to evolve and encourage the progression of caries.

S.W has high acid production and resistance to fluoride and lactic / acetic acid. It also has a distinctive metabolic pathway, F6PPK shunt, which by preserving the metabolic flow to the fluoride-tolerant acetic acid-pathway could contribute to its

fluoride and acid tolerance.

Isolation of *S. Wiggisiae* by RT-PCR

One study assessed the levels of *S.W* in caries-free, early childhood caries (ECC) and severe ECC (SECC) affected children using real-time polymerase chain reaction (RT-PCR). Forty-five children aged <71 months were randomly recruited. Fifteen children suffering from ECC, 15 with SECC, and 15 children without ECC were enrolled in the study. About 1–2 mL of unstimulated saliva was collected and subjected to microbial analysis using RT-PCR. The correlation of decayed, missing, or filled surface levels with 16s rRNA levels showed significant positive correlation with 16S rRNA in both ECC and S-ECC patients. They found that salivary levels of *S.W* were significantly associated with ECC in children [37].

Effect of sealant on *S. Wiggisiae*

One of the study was done to evaluate the effect of sealant on the *S. Wiggisiae* count. It evaluated that placement of dental sealants was sufficient to reduce the levels of detectable *Scardovia* among those patients initially testing positive (23%). However, most samples were initially *Scardovia*-negative (77%) and this study revealed a subset of these *Scardovia*-negative patients were subsequently found to harbor *Scardovia* from their corresponding post-sealant samples (28%). Despite the change from *S.W*-negative to *S.W*-positive among some of the patients evaluated, these changes in the overall percentage of patients testing *S.W* positive were not large enough to be statistically significant [38].

S. Wiggisiae count in caries risk assessment

To evaluate the duration of periodic recall, a patient's caries condition is also used. A better caries-prediction tool will help to make preventive program more suitable for the individual. It will, however, be useful for patients and parents as an educational and motivational resource. A decrease in the number of microbes may represent the success of preventive care and encourage patients to continue their good long-term oral health practice.

The quest for the NCBI BLAST revealed that the nucleotide sequences of *S. Wiggisiae*-specific PCR products amplified from three samples of dental plaque showed a 100 % match with *S.W* strain F0424 and some other *Bifidobacteria* have a partial match. In both dental plaque and dentine samples, *S.W* was found, but its proportion was lower than *S. mutans*. The incidence of both microbes was substantially higher in the ECC group than in the caries-free group in dental plaque samples.

Thus, the plausibility of applying *S. wiggisiae* as a microbiological marker in combination with *S. mutans*, for better caries risk assessment in children will give positive predictive value for caries. However, using single microorganism detection appears to compromise the sensitivity or specificity of the tests [39].

Orthodontics and *S. Wiggisiae*

The discrepancy between orthodontic and non-orthodontic patients was examined for the count of *S.W* associated with white spot lesions in one of the retrospective studies. This research selected more than one hundred saliva samples from adult orthodontic (n=49) and non-orthodontic (n=52) patients for inclusion. Subsequently, all DNA derived from these samples was analyzed using PCR, which indicated the presence of *S. P. gingivalis* (PG), *S. mutans* (SM), and *S.*

Wiggisiae (S.W), the prevalence of which varied between non-orthodontic and orthodontic patients. Nearly all the PG-positive and S.W-positive tests were also SM-positive in non-orthodontic patients. None of the S.W-positive samples were either SM- or PG-positive among orthodontic patients, however. In patients seeking orthodontic therapy, this indicates the S.W is prevalent. Thus, it will not be wrong to say that the association of S.W with white spot lesions can lead to early cavitation^[40].

Conclusion

S. wiggisiae represents a new frontier in the microbial etiology of ECC. This may lead to the development of new antimicrobial agents targeted to this organism and change in practices. Like in treatment of ECC it was found that using pit and fissure sealant would lead to reduction in its count. Also, new strategies must be formulated to prevent the growth of *S. wiggisiae* along with the other micro-organisms leading to cavitation.

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