Competence and Transformation of Oral Streptococcus sobrinus in Dental Caries

Dental caries is an infectious progression concerning the breakdown of the tooth enamel. Caries form through an intricate interface between cariogenic acid-producing microorganisms in combination with fermentable carbohydrates and other dietary factors. Furthermore, the inherent, interactive, social, and cultural factors play the critical role.

Risk factors for dental caries in young children include high levels of cariogenic microbial annexation. *Streptococcus mutans* forms a biofilm, then takes the sugars we eat and ferments them into acid, which decalcifies teeth and causes decay. Frequent exposure to dietary sugar and refined carbohydrates, inapt bottle suckling, low saliva flow rates, developmental defects of tooth enamel, low socioeconomic standing, previous caries, maternal caries, high maternal levels of cariogenic microorganisms, and poor maternal oral hygiene are also the major factors. Other risk factors include lack of access to dental care, low community water fluoride levels, inadequate toothbrushing or inadequate use of fluoride-containing toothpaste, and lack of parental knowledge regarding oral health

Although there is a second harmful microbes called *Streptococcus sobrinus* which accelerates tooth decay in some people, very little is known about this microbe. This concept may change soon since a team of Illinois Bioengineering researchers led by Assistant Professor Paul Jensen has magnificently sequenced the complete genomes of three strains of *S. sobrinus*.

According to Jensen, S. sobrinus is problematic to work within the laboratory and it is not present in all people, so researchers have instead focused their efforts over the years on understanding the more stable and prevalent S. mutans, which was sequenced in 2002. Although it is rare, S. sobrinus produces acid more quickly and is associated with the poorest clinical outcomes, especially among children, claimed Jensen, a researcher at the Carl R. Woese Institute for Genomic Biology. If S. sobrinus is present along with S. mutans, the children are at more risk for widespread tooth decay, which means there is some level of communication or synergy between the two that we do not understand yet. Now that the S. sobrinus sequencing is complete, Jensen and his scholars are building computational models to better understand how the two bacteria interact and why S. sobrinus can cause such potent tooth decay when combined with S. mutans. They have further confirmed, that S. sobrinus lacks complete pathways for quorum sensing, which is the ability of other bacteria to have sagacity and react to nearby bacteria, and ultimately proliferate.



According to Jensen, *S. mutans* microbes send out sensors in the form of a peptide to treasure out how many other *S. mutans* cells are close. Once the *S. mutans* cells reach a certain threshold, they attack and create an imbalance in a person's mouth thus resulting caries-promoting environment, resulting imbalance between good and depraved bacteria, which leads to rapid cavity formation.

"This is ground-breaking work because the field was plagued by a lack of information," claims Jensen. Jensen also stated that his team is working on a genomic-scale model, which will allow them to run virtually any experiment they want.

Armed with this knowledge, his team will then make recommendations of drug targets for further experimentation. These therapies could eventually be used to lessen the destructive power of the bacteria hence ultimately will be a weapon to fight the evil of tooth decay.

Modern advances in technology and medicine have allowed for connections to be made within our bodies that we were never aware of. Recent research as part of the Human Microbiome Project has indicated a link between the quality and wide diversity of the bacteria we carry and the range of diseases they can cause. The presence of these bacteria causes a significant impact on our overall health and susceptibility to diseases. Even cancer studies are showing that there is more value in studying the specific mutations of the tumors rather than cancer's geographical placement in the body.

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