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The Role of *Streptococcus mutans* And Oral Ecology in The Formation of Dental Caries

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Table of Contents

[Abstract](#)
[Introduction](#)
[Microbial and Host Ecology](#)
[Pathogenic Bacteria](#)
[Streptococcus mutans](#)
[Modes of Transmission](#)
[Virulence Factors](#)
[Coevolution of humans and Streptococcus mutans](#)
[Early Childhood Caries](#)
[Treatment Options](#)
[Conclusion](#)
[References](#)

Abstract

Although it is one of the most common ailments on the planet, dental caries, more commonly known as cavities, remains a poorly understood disease. Caries are caused by a complex interplay of factors, especially patient diet and the presence of the bacteria *Streptococcus mutans* on the teeth. In spite of the fact that poor dental health has been linked to multiple full-body conditions and diseases, such as Multiple Sclerosis and Heart Disease, and that eighty percent of all American adolescents will be diagnosed with caries, there are still few successful preventative treatments. Rampant caries diseases are especially common among lower-income populations, such as the devastating pediatric disease Early Childhood Caries, which has reported rates of as high as ninety percent in some subpopulations. Recent research into the intricate microbial ecology of the mouth and the other risk factors that may play a role in caries formation has provided insight into new treatment and prevention possibilities for this extremely common infectious disease.

Introduction

In the United States, basic dental hygiene techniques are common knowledge. From advertisements on television to the dentist's office, we are told to brush and floss daily in order to prevent dental caries. However, the simplified view that sugar-rich foods lead directly to cavities, though fundamentally true, belies the complex ecosystem of microflora that inhabit the human oral cavity. Over 400 microbial species can be found in the typical adolescent human mouth, and in general this ecosystem is maintained at homeostasis, with each microbe inhabiting its own ecological niche, such as the varied surfaces found within the human mouth (the teeth versus the tongue, for example) (Slavkin 1999). However, changes in the oral cavity caused by an increase in glucose consumption can shift the homeostasis of this ecosystem to particularly acidophilic bacteria known to be damaging to the teeth, resulting in dental caries. The most virulent of these species is *Streptococcus mutans*, which has been found to be the initiator of most dental caries (Tanzer, Livingston et al. 2001), and which is a transmissible bacterium that can be transmitted both horizontally and vertically (Loveren, Buijs et al. 2000; Li, Caufield et al. 2005). As a result, dental caries can be considered one of the most widespread and common infectious diseases on the

planet. Recent research has shown the human mouth to be its own ecosystem with an intricate ecology of microbial species, and the human relationship with virulent oral pathogens, in particular with *Streptococcus mutans*, to be a formerly mutual or commensal evolutionary relationship that has gone awry due to the modern diet. It is hoped that these new attitudes toward oral health and caries in particular can inform treatment options for a variety of patients. New treatments derived from an ecological or evolutionary viewpoint may be instrumental in treating Early Childhood Caries (ECC) and other persistent dental diseases that are especially prevalent in low-income populations.

Microbial and Host Ecology

Under the conditions in which the human-oral microflora relationship evolved, the bacteria that inhabit the human mouth appear to have a commensal or even mutualistic relationship with their human host and with each other. The mouth can be considered an ideal environment for the growth of microorganisms, since it is warm and moist and has a constant influx of nutrients through saliva and food intake. In fact, it has been calculated that there are as many as 4×10^{10} organisms in each gram of plaque removed from the teeth (Gibbons and Armstrong 1964). These organisms consist of, on average, more than 400 species that live together through the exploitation of very specific ecological niches. For example, Lactobacilli are known to favor the dorsum of the tongue, while *Streptococcus mutans* requires a solid, non-shedding surface for colonization, as demonstrated by the rapid appearance of *S. mutans* in the mouth of toothless infants when obturators are inserted to fix cleft palates (Tanzer, Livingston et al. 2001). This complex relationship even consists of bacteria whose presence is contingent on other, "pioneer," bacterial species. For example, the group of bacteria known as the sanguis streptococci has been found to produce nicotinamide adenine dinucleotide (NAD), which is then consumed by the species *Haemophilus parainfluenzae* (Liljemark and Bloomquist 1996). It has even been demonstrated that certain oral microorganisms can cooperate in a mutualistic manner for a common benefit, as when species collaborate by using different species-specific enzymes to break down complex host molecules that could not be metabolized by a single species (Marsh 1994).

The ecology of the mouth does not just involve interactions among microorganisms, however. In fact, the host plays a large role in maintaining a uniform ecosystem, especially through the saliva. Saliva is a complex mineral- and protein-rich solution that delivers nutrients to the many bacterial species within the mouth while also protecting host surfaces. During mastication, increased saliva flow prevents changes in oral pH, because the buffer bicarbonate is present in saliva and acts as an acid sink at a time when acidic products are being introduced into the mouth. Urea and the peptide sialin are both also present in low concentrations in saliva and produce ammonia when hydrolyzed, a basic product capable of raising pH. (Loesche 1986). This basicity and buffering counteracts the lactic acid produced by anaerobic bacteria in the mouth during the fermentation that occurs when nutrients are introduced, offsetting decay of the teeth caused by this acid. Saliva also contains glycoproteins that are known to be antibacterial, such as lysozyme and lactoperoxidase. These compounds act independently of the host's immune system, and are able to destroy invasive bacteria without harming the ecological balance of the oral cavity, since indigenous bacteria have evolved resistance (Loesche 1986).

A remarkable example of the balanced relationship between saliva and oral microbiota is the fact that saliva is actually supersaturated with calcium and phosphate ions, which precipitate to form hydroxyapatite and remineralize the teeth. This supersaturated solution should theoretically result in uncontrollable tooth growth as more and more calcium phosphate precipitates onto the teeth. However, proteins containing proline and a peptide called statherin, both of which are present in saliva, have been shown to slow the rate of precipitation of these ions to a rate which perfectly matches the rate of decay induced by bacteria during normal lactic acid formation (Loesche 1986). Under these conditions, teeth should be caries-free while the mouth remains colonized. The extensive decay and rampant levels of *S. mutans* in patients suffering from xerostomia, or reduced saliva flow, is a striking example of the role of saliva in caries prevention (Marsh 1994). In fact, patients undergoing radiation for head and neck cancers are often used as models for the progression of decay, since severe xerostomia is an almost universal side effect of such treatment (Loesche 1986).

Pathogenic Bacteria

Even without the devastation of xerostomia, caries still can be rampant. Historically, cavities were attributed to a general overgrowth of oral bacteria, termed the "non-specific plaque hypothesis." However, this was disproved by Keyes (1960), who compared the bacterial make-up of caries-active and caries-inactive hamsters and found much higher proportions of a group of bacteria termed the "mutans streptococci" in the cariogenic hamsters (Keyes 1960). He further showed that hamsters without caries did not develop them until exposed to caries-active hamsters or their feces. The mutans streptococci could be isolated from these newly caries-active hamsters, and cultures of the bacteria would also cause caries in caries-free hamsters. Keyes' hamster experiments showed that these bacteria fulfilled Koch's postulates for infectious disease and led to the adoption of the "specific plaque hypothesis," which states that only certain bacterial species are responsible for cariogenic behavior (Gibbons and Armstrong 1964).

This breakthrough led to even more questions, however. In particular, researchers were eager to discover which bacterial species were responsible for caries formation. Caries are formed when the rate of decay of the teeth caused by the lactic acid produced by anaerobic bacteria exceeds the rate of repair initiated by the phosphate and calcium ions in saliva. Lactic acid production surges when sucrose is introduced into the mouth during meals or snacks, resulting in an overall drop in oral pH. Thusly, if acidity is a prerequisite for caries formation, then only species that thrive in an acidic environment, known as acidophilic species, can play a role in producing them. In fact, when cultures simulating a community of oral bacteria were pulsed with glucose to produce a constant pH lower than 5, the acidophilic species *Lactobacillus casei*, *Veillonella dispar*, and especially *Streptococcus mutans* were able to dominate niches previously occupied by other, avirulent species, and became irreversibly over-represented in the population (Marsh 1994).

Streptococcus mutans

Streptococcus mutans has been implicated most of all as the initiator of dental caries. In an experiment in which Swedish children were given chlorhexidine to prevent *S. mutans* colonization, development of caries was delayed by an average of three years, while titers of lactobacilli and other oral bacteria hypothesized to be virulent were unaffected (Tanzer, Livingston et al. 2001). Meiers et al. (1982) collected the water spray from a high speed drill used during the filling

of both carious and non-carious lesions in the mouths of naval recruits and found that although multiple organisms were present in each fissure, *S. mutans* was the only bacterium found in significantly larger numbers in the carious lesions than on the teeth of caries-free individuals (Meiers, Wirthlin et al. 1982). Molecular analysis of bacterial samples attained from the enamel of caries-free children and from within the carious lesions of children suffering from Early Childhood Caries performed by Becker et al. (Becker, Paster et al. 2002) showed a similar cause-and-effect pattern. Only *Streptococcus mutans* and *Veillonella dispar*, a species shown to serve as a lactic acid sink and increase the activity of *S. mutans*, were found in much higher concentrations in the carious lesions (Figure 1).

What makes *Streptococcus mutans* such a potent initiator of caries? A variety of virulence factors unique to the bacterium have been isolated that play an important role in caries formation. First, *S. mutans* is an anaerobic bacterium known to produce lactic acid as part of its metabolism. Then there is the ability of *S. mutans* to bind to tooth surfaces in the presence of sucrose by the formation of water-insoluble glucans, a polysaccharide that aids in binding the bacterium to the tooth. Mutant strains developed to produce water-soluble glucans instead have extremely diminished cariogenicity, especially on the smooth surfaces of the teeth which require greater tenacity for binding to occur (Loesche 1986). Water-insoluble glucan has also been found to lower the calcium and phosphate concentration of saliva, decreasing its ability to repair the tooth decay caused by bacterial lactic acid (Napimoga, Kamiya et al. 2004). The most important virulence factor, however, is the acidophilicity of *Streptococcus mutans*. Unlike the majority of oral microorganisms, *S. mutans* thrives under acidic conditions and becomes the dominant bacterium in cultures with permanently reduced pH. Additionally, unlike many species present in plaque, whose metabolisms slow considerably at such a low pH, the metabolism of *S. mutans* actually improves, as the proton motive system used to transport nutrients through its cell wall in environments of low pH or high glucose concentration is modulated by hydrogen ion content, which increases with acidity (Hamilton and Martin 1982). In this way, *S. mutans* can actually continue to lower or maintain the oral pH at an unnaturally acidic value, leading to conditions favorable for its own metabolism and unfavorable for other species it once coexisted with. It is this lowered pH that results in demineralization and cavitation of the teeth, both of which increase with increased rates of *S. mutans*. Under acidic conditions, *S. mutans* succeeds in creating a cycle that is favorable for itself and unfavorable for others involved in the oral ecology – becoming, in effect, a pathogen.

Modes of Transmission

Like any other infectious pathogen, *Streptococcus mutans* depends on transmission routes to propagate itself among many human hosts. Although *S. mutans* favors hard, non-shedding surfaces for the establishment of permanent colonies, a fact which led many to assume that levels of *S. mutans* were undetectable in infants until the eruption of the primary teeth, recent studies have revealed that *S. mutans* can colonize the furrows of the tongue in pre-dentate infants (Berkowitz 2003). When the teeth erupt, typically between the ages of one and two, *S. mutans* can establish thriving colonies on the teeth that eventually lead to cavities, most notably Early Childhood Caries (ECC). It is the appearance of detectable levels of the bacteria on the teeth that indicate that cavity formation is possible. Detection of *S. mutans* in the furrows of the tongue reinforces the conclusion that the most common transmission route for the bacteria is vertical, from mother to child, most likely shortly after birth. Studies of the saliva samples of two to five year-old children and their mothers by Caufield et al. (1988) revealed a high fidelity in the genetic makeup of each host's *S. mutans* population (Caufield, Ratanapradikul et al. 1988). The same experimenters also concluded that plasmid DNA similarities correlate to different races, also implying primarily vertical transmission. As a result, mothers with high titers of the bacteria or who have suffered from many dental caries themselves are likely to pass the same virulence and associated problems on to their children. In fact, mothers whose salivary *S. mutans* levels exceeded 105 colony forming units were about nine times more likely to pass the bacteria on to their children (Berkowitz 2003).

Streptococcus mutans also appears capable of horizontal transmission. Children in the same nursery school class often had identical strains of the bacteria in their saliva (Berkowitz 2003), and children who had no detectable *S. mutans* titer until after the age of five often shared strains with both mother and father when the bacteria was finally acquired (Loveren, Buijs et al. 2000). Frequent close contact with others seems to be enough to transmit *S. mutans*, a fact which could impact prevention techniques in environments such as day care centers, where children harboring more virulent strains could increase the cariogenic potential of the bacteria in other children.

Virulence Factors

The time at which *Streptococcus mutans* is acquired in relation to other common oral bacteria also plays a role in its prevalence in the ecology of the mouth. The successive colonization of many different species leaves only a certain realized niche for *S. mutans*, minimizing its impact through a process that has been termed "bacterial succession." Some pioneer species, such as *Streptococcus oralis* and *Streptococcus mitis* are detectable in infants only a few days old, while *S. mutans* is virtually undetectable until around age two (Li, Caufield et al. 2005). Any alteration in this progression of colonization can lead to increased risk of dental caries. For example, Li et al (Li, Caufield et al. 2005) found that babies delivered via Caesarian section had detectable levels of *S. mutans* approximately a year earlier than those delivered vaginally, presumably because they were not colonized by pioneer bacteria found on the perineum of their mothers that babies born vaginally were exposed to.

However, the biggest virulence factor and greatest determinant of caries susceptibility is extra-bacterial: the consumption of sugar-rich carbohydrates. Each influx of sugar into the mouth results in a sharp drop in pH, conditions which favor demineralization of the teeth and heightened *S. mutans* activity. Under the conditions in which the human – oral bacteria relationship evolved, dietary sugar levels were dramatically lower and humans ate a few large meals, rather than constantly introducing sugar into the mouth by snacking. In studies tracking oral pH and eating habits over time, those who ate three regular meals a day experienced the same post-meal drop in pH as those who snacked constantly, with the critical distinction that the time difference between each drop allowed the saliva to raise pH and remineralize the teeth, undoing the damage caused by *S. mutans* metabolism at each meal time. In contrast, those who ate three meals as well as sucrose snacks experienced an overall drop in pH with no recovery ability, because the saliva did not have enough time to raise the pH before more sugar was consumed (Loesche 1986). While the increased prevalence of fluoridation of the water supply has made sugar-consumption less of a risk factor, it is still one of the biggest predictors of dental caries (Burt

and Pai 2001).

Coevolution of humans and *Streptococcus mutans*

Under prehistoric evolutionary conditions, the presence of *Streptococcus mutans* would have no adverse effects, and might even have benefited humans by preventing the colonization of harmful bacteria. Yet the taste for sugar that once ensured that our ancestors consumed foods with the maximum amount of calories now keeps us snacking in between meals and has created a multibillion dollar industry for what Loesche has called "the slow-release device for sucrose known as a candy" (Loesche 1986). Studies of societies in which access to sugar is drastically altered show the expected matching change in caries rates. For example, Inuit families following culturally traditional (low-sucrose) diets had many fewer cavities than wealthier Inuit families who could afford more Western foods (Mayhall, Dahlberg et al. 1970). Similarly, Norwegian children who grew up during World War Two, when sugar was severely rationed, experienced fewer caries than children growing up shortly after, when sugary foods were again common (Toverud 1957). Humans' relationship with both food and with *Streptococcus mutans* evolved in very different conditions than those of today. In effect, the disease initiated by *S. mutans* in our mouths is a case of evolutionary incongruence with the lifestyle enjoyed by those in developed countries.

Early Childhood Caries

One of the most striking examples of the problems caused by the conflict between the evolutionary past and the modern lifestyle is the pediatric dental disease known as Early Childhood Caries, also called Nursing-Bottle Caries. Early Childhood Caries is associated with the availability of a sugar-rich drink (often in a bottle or sippy cup) to infant or young child during periods of sleep. This allows the sugars to bathe the primary teeth at a time of decreased salivary activity. Providing a beverage containing sugars during naptime is a behavior shown to increase the risk of caries formation almost fourfold. This is a practice often associated with lower-income families, who are also at increased risk since salivary function can be lowered through iron deficiency, a frequent malnutrition problem in lower-income households, or exposure to lead products, as lead-based paint is still common in many inner-city dwellings (Campbell, Moss et al. 2000). Lower-income populations are also less likely to receive dental care due to inability to pay or a lack of availability of clinicians. In fact, only six percent of dental needs were treated in 1,198 communities termed "health professional shortage areas" (Mouradian, Wehr et al. 2000). Untreated cavity numbers fall almost linearly with family income increases, with children from families at or below the federal poverty line having, on average, more than three times the number of decayed teeth of children whose parents earn 301% or more of the federal poverty income (Mouradian, Wehr et al. 2000). It is thus not surprising that Head Start preschool programs for low income children have reported Early Childhood Caries rates of as high as ninety percent (Slavkin 1999).

Disease rates are also disproportionately distributed among ethnic minorities compared to non-Hispanic Whites. African-American and Hispanic children suffer much higher caries rates and have more untreated dental needs (Vargas, Crall et al. 1998). Native American and Alaskan Native children have some of the highest Early Childhood Caries rates in the nation (Tang, Altman et al. 1997). Preventative treatments are also less common in these groups. Only five percent of African-American 14 year-olds and seven percent of Hispanic 14 year-olds have dental sealants, a paltry rate compared to the 24 percent of all 14 year-olds who do (Service 2000).

Treatment Options

Although treatment for Early Childhood Caries is generally straightforward, young children must often be put under general anesthesia for these dental procedures due to age, behavior, or the severity of the treatment. This renders the disease extremely costly both financially and in lost work hours for parents. These costs are also unfairly distributed on the lowest-income families or on federal health insurance plans (Berkowitz 2003). Relapse rates for children suffering from the disease are also extremely high. In a study by Almeida et al. (2000) of children who required general anesthesia for the treatment of their rampant caries, 79 percent of these children were diagnosed with additional cavities within the two years after their treatment, compared to only 29 percent of an initially caries-free control group. The establishment of virulent *S. mutans* colonies at such a young age can lead to lifelong dental problems. Persistent dental disease is both costly and painful, and most importantly, it has also been suggestively linked to diabetes, high blood pressure, heart disease, and Multiple Sclerosis later in life (DeStefano, Anda et al. 1993; McGrother, Dugmore et al. 1999).

The most common treatment for Early Childhood Caries and other dental diseases has been to fill each cavity on a case-by-case basis and attempt to educate families about proper dental hygiene and dietary practices, but the high relapse rates of most patients suffering from dental caries attests to this plan's failure. In a small but suggestive study, of 38 Mexican-American parents of children with Early Childhood Caries asked about dietary practices, 25 were aware that naptime bottle feeding was associated with the disease, implying that lack of awareness is not the problem (Berkowitz 2003). However, an understanding of the complex interplay of human host and *S. mutans* can hopefully improve prevention techniques and prevent Early Childhood Caries and other rampant caries diseases from being so pervasive in lower-income populations. Even delaying initial acquisition of the bacterium by suppressing maternal *S. mutans* levels through antibiotic prescription has been shown to reduce Early Childhood Caries rates in at-risk children (Marsh 1994). Another study involving prophylaxis showed that women who rinsed their mouths daily with the bactericide chlorhexidine in their seventh month of pregnancy delayed colonization of their children's mouths by an average of four months. An alternate treatment option was put to the test in a study by Ma et al. (1987). Their study involved passively immunizing individuals against *Streptococcus mutans* by having them rinse with a solution containing genetically engineered monoclonal antibodies (MAbs). These immunized individuals showed decreased salivary rates of *S. mutans* compared to a non-immunized control group who had rinsed with saline (Ma, Smith et al. 1987).

Filling the ecological niches once dominated by *Streptococcus mutans* with less virulent species has also been proposed as a treatment method. The development of avirulent mutant strains of the bacterium shows promise as a pre-emptive colonizer, which would be introduced into the mouth before natural *S. mutans* colonization and occupy the ecological niche that would be available to virulent strains (Marsh 1994). Becker et al. (2002) found that *Streptococcus sanguinis* was present in the mouths of caries-free individuals, but was absent in the mouths of those with dental disease.

The relationship between *S. sanguinis* and oral health could be exploited by the implantation of *S. sanguinis* strains before an initial diagnosis of caries. Alternately, a mutant strain of *Streptococcus Salivarius* has been shown to displace *S. mutans* from the mouths of caries-active rats and shows promise as a treatment for humans (Marsh 1994).

Conclusion

The incredibly widespread problem of dental caries is the final manifestation of an imbalance in the multifaceted relationship between human host, *Streptococcus mutans*, and hundreds of other bacterial species that inhabit the mouth. The diets of those in developed countries are dissimilar to those of their ancestors in which the oral microbiota – human relationship evolved, which leads to selection for more virulent, acidogenic *S. mutans* populations, who can manipulate and come to dominate the ecology of the mouth. Understanding this intricacy promises novel solutions to a disease with high costs and morbidity rates. Dental caries is a seemingly simple problem with complicated causes and excessive costs, which will hopefully be resolved through simple solutions.

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About the Author

Lisa Simon is a member of the Yale College class of 2010 at Yale University. She is in Branford College. She is pursuing a B.S. degree in Ecology and Evolutionary Biology and plans to attend dental school upon graduation.

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