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# THE ROLE OF *STREPTOCOCCUS MUTANS* IN THE FORMATION OF DENTAL CARIES: AN ECOLOGICAL PERSPECTIVE

Jason Yeshaya Friedman

## ABSTRACT

The teeth are among the most distinctive and productive features of the human species. It is the longest lasting surface of the body and can be used in research studies many years after death. Yet, in the living individual, the integrity of the teeth is constantly assaulted by a microbial challenge so great that dental caries, or decay, ranks as one of the most widespread medical afflictions. According to studies, dental caries rank third in medical costs, behind only heart disease and cancer (Loesche 1996). This review will attempt to describe what is responsible for dental caries, namely a bacterium called *Streptococcus mutans*. More specifically, it will concentrate on theories regarding the precise role of *S. mutans* and what causes it to flourish at times when bacteria associated with a healthy oral cavity cannot survive. It will further explain how after performing Pure Culture and Mixed Culture studies, the results clearly provided a theory referred to as the "ecological plaque hypothesis." In this theory, it became clear that it was not the mere presence of *S. mutans* that caused dental caries, but rather it was specific environmental factors that allowed *S. mutans* to thrive while rendering the non-pathogenic bacterium insignificant. Based on these updated theories, scientists have been able to find preventative methods to inhibit *S. mutans* even in the environment that normally favors its growth.

## INTRODUCTION

Although the oral cavities of most mammalian species contain billions of microbial cells, most scientists have concluded that *Streptococcus mutans* is the bacterium that is responsible for dental caries, or decay. Over the years, scientists have been researching the precise role of *S. mutans* and what causes it to flourish at times when non-pathogenic bacteria cannot survive. It is this precise question that this review will attempt to answer. Specifically, it will concentrate on the ecology of the oral cavity and will attempt to prove, via various studies, a theory called the "ecological plaque hypothesis." This theory will appropriately explain the role of *S. mutans* and will open the door for successful prevention mechanisms.

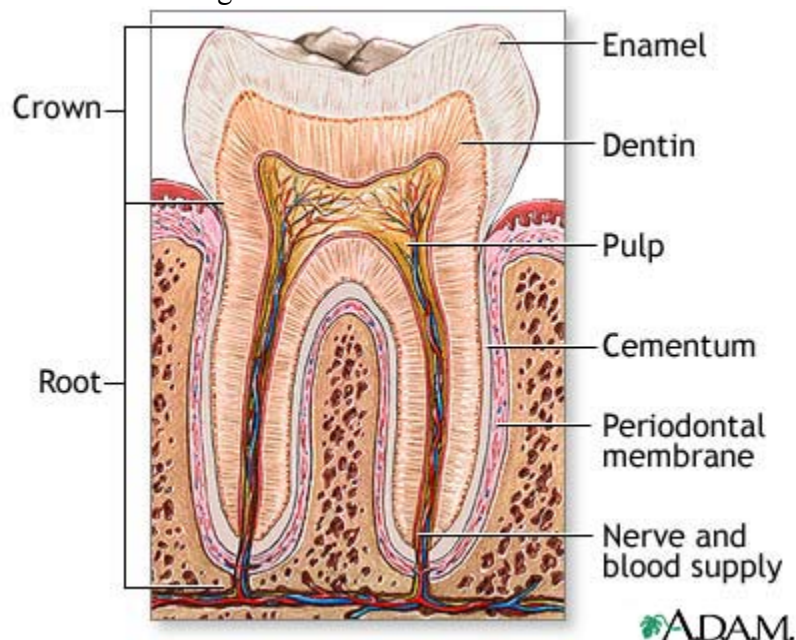
## DISCUSSION

A healthy human being has an estimated total of 100 trillion living cells (Savage 1977). However, 90% of these cells are merely micro-organisms that live in areas such as the intestinal tract, skin, and mouth. In fact, each specific habitat has a particular composition of micro-organisms which, in a healthy individual, remains stable once it's established. This is called microbial homeostasis. The stability of the microbial community in each distinct habitat is due to key ecological factors that are present. These factors include nutrients, pH levels, attachment receptors, and oxygen levels (Marsh 2001). Changes in the environment can severely alter the homeostatic balance necessary to sustain the microbial species, and this can result in disease.

From all the areas which contain these massive numbers of microbial cells, the oral cavity alone contains almost half of these microorganisms, amounting to about six billion microbial organisms (Loesche 1996). There are many properties of the oral cavity that make it an ideal habitat for microbial species. One such feature is the continuous flow of saliva. Saliva greatly enhances the ecology of the oral cavity. The pH of saliva (about 6.95) is ideal for micro-organism growth and its ionic composition provides buffering and reparation of the enamel (Scannapieco 1994). In addition, saliva keeps the mouth warm (around 35°C) and

moist. Yet, perhaps the most beneficial feature of the saliva in the mouth is its ability to promote the adhesion of microbial species by coating surfaces of the oral cavity with a selective conditioning film.

Although all surfaces in the oral cavity benefit from saliva and serve as habitats for various microbial species, there is one surface in the mouth that has certain features which render it the supreme resting place for the oral microorganisms. This surface is the tooth (Figure 1). The teeth are oral surfaces that have a unique feature which is not seen in any other surface in the body. The tooth surface rarely sheds once it is formed (Bowden 2000). This property facilitates microbial growth, because once biofilm is formed on the tooth, it will not be removed as a result of shedding of the tooth surface.



**Figure 1:** The structure of the tooth.

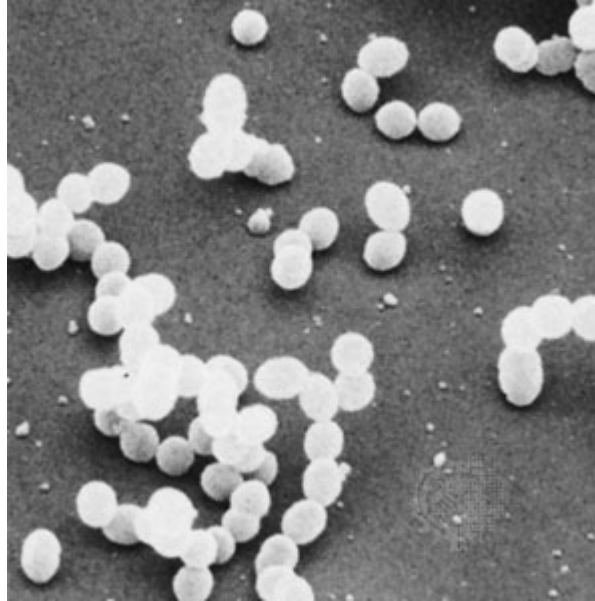
Source: David Zieve, MD, MHA, Medical Director, A.D.A.M., November 12,

The non-shedding character of the tooth makes its biofilm covering more important when studying microbial species. The extensive formation of biofilm on the teeth is called dental plaque. In healthy dental plaque formation, there is a sequence of events of microbial succession that allow its formation (Busscher and Van der Mei 2000). The first step occurs shortly after the teeth erupt. As mentioned, the host's saliva, along with some bacteria, coats the enamel surface with film. The first round of microbial species adheres to the film, using nonspecific interactions between charged molecules. These organisms grow and create a suitable environment for the colonization of more complex species. The new wave of species adheres to the already attached organisms. This process results in the dental plaque becoming a complex and multi-species biofilm.

Over the years, there have been numerous studies done on the composition of this dental plaque in order to determine which species cause it to decay and facilitate formations of dental caries. Although old hypotheses have been modified and study techniques enhanced, close to all researchers would agree the main species responsible is a bacteria called *Streptococcus mutans* (Figure 2).

The real question is not whether *S. mutans* is responsible for disease. It clearly is. Rather, researchers have been studying the exact role of *S. mutans* in dental caries. The questions scientists have been studying are about the specific triggers that allow *S. mutans* to grow and how to successfully prevent it from doing harm.

The first hypothesis in regards to the role of *S. mutans* was known as the “specific plaque hypothesis.” In this conclusion, it was believed that although there is a vastly diverse collection of microbial species present in the plaque, *S. mutans* causes the disease and caries (Emilson and Krasse 1985). It does so by rapidly metabolizing sugar into acid. This process creates an environment with a particularly low pH. Under these conditions, *S. mutans* can continue to grow and eventually wear down the enamel.



**Figure 2:** Streptococcus mutans.

Source: Encyclopedia Britannica Online. Web. 25 May, 2011.

However, the “specific plaque hypothesis” did not succeed in painting the full picture. The questions of what triggered *S. mutans* to grow and what allowed it to flourish when bacteria associated with healthy enamel could not do so were still unclear. Further study and research upon the habitats and the different factors that alter the microbial homeostasis led to the formation of an alternative hypothesis which better explains the role of *S. mutans* in dental caries and disease.

One such study is called Pure Culture Study. This study compares selected healthy and diseased stains in a controlled environment. This method provides accurate details on how environment affects the microbial species by independently varying different environmental cues and observing the microbial response. Early Pure Culture studies compared the response of *S. mutans*, the bacteria implicated in disease, and the bacteria of healthy enamel, by introducing them to different sugar and pH environments. The conclusions of these experiments showed that *S. mutans* had the ability to survive and flourish over a wider range of pH and was in fact at its optimal growth at acidic pH (about 5). In addition, *S. mutans* had a higher rate of sugar uptake and underwent glycolysis more rapidly.

As mentioned, these studies clarified the role of *S. mutans* in dental caries and disease beyond the “specific plaque hypothesis”. It confirmed the role of *S. mutans* in disease, but

expanded the theory by explaining how ecological changes are the key to allow the *S. mutans* to flourish as opposed to the bacteria associated with healthy enamel. It showed how differences seen in the composition of plaque are driven by the microbial species' response to environmental changes, resulting in the ability of previously minor components of the microflora to flourish.

To test this hypothesis, and to further explore the impact of environmental changes on the balance of the plaque microbial community, it was necessary to develop a more intricate study. This led scientists to begin a Mixed Cultures Study. In the Mixed Culture Study, researchers exploited the unique advantages of a chemostat to grow mixed cultures of oral bacteria in a range of controlled conditions (Marsh et al. 2006). Due to the fact that a chemostat allows individual parameters to be changed independently, a clear presentation is provided on how specific environmental conditions affect the physiology of the culture.

The reactions to two main environmental conditions were studied in the Mixed Culture experiment. Researchers wanted to see how oral bacteria associated with healthy enamel would react differently to *S. mutans* when presented with large doses of sugar and an environment with low pH. To best study the oral bacteria within these parameters, a chemostat was developed with a system for growing nine oral bacteria at constant temperature (37°C) and pH (7.0). The mixed culture was pulsed with glucose on ten consecutive days, either with or without pH control (Table 1) (Bradshaw et al. 1989).

**Table 1:** Effects of glucose pulses, low pH, and fluoride on the stability of a mixed culture of nine oral bacteria.

| Bacterium            | Percentage Viable Count |                           |                      |   |
|----------------------|-------------------------|---------------------------|----------------------|---|
|                      | Pre-pulsing             | With pH control<br>(pH 7) | Without pH control   | After 10 glucose pulses:<br>Without pH control,<br>with NaF |
| <i>S. gordonii</i>   | 28.3                    | 25.0                      | 0.2                  | 0.002   |
| <i>S. oralis</i>     | 15.2                    | 16.9                      | 1.3                  | 4.6   |
| <i>S. mutans</i>     | 0.3                     | 1.0                       | 18.9                 | 0.2   |
| <i>A. viscosus</i>   | 0.1                     | 13.1                      | 2.3                  | 0.4   |
| <i>L. casei</i>      | 0.1                     | 0.2                       | 36.1                 | 36.5  |
| <i>N. subflava</i>   | 0.1                     | 0.01                      | ND <sup>b</sup>      | 2 x 10 <sup>-5</sup>  |
| <i>V. dispar</i>     | 9.8                     | 28.7                      | 41.4                 | 57.8  |
| <i>P. intermedia</i> | 31.0                    | 5.6                       | 6 x 10 <sup>-4</sup> | 0.5   |
| <i>F. nucleatum</i>  | 15.2                    | 9.5                       | 2 x 10 <sup>-5</sup> | 0.2   |
| Final pH             | 7.0                     | 7.0                       | 3.83                 | 4.49  |

<sup>a</sup> The mixed culture was pulsed on 10 consecutive days to give 28 mmol/L glucose, with or without 1 mmol/L NaF. The pH was either maintained automatically throughout at pH 7.0 ± 0.1 or was allowed to fall for six h following each pulse before being returned to pH 7.0 for 18 h prior to the next pulse.

<sup>b</sup> ND = not detected.

Source: Bradshaw et al. 1989

To understand the table let us simply focus on the first three species. The first two bacteria, *S. gordonii* and *S. oralis* are bacteria associated with a healthy oral cavity. The third strain, the *S. mutans*, is associated with disease. We see in the first column, that before any glucose was pulsed, the non-pathogenic bacteria dominated over the *S. mutans*. This represents a healthy oral cavity. In the second column glucose is pulsed, yet there is pH

control. We see that the non-pathogenic bacteria still remain dominant. This proved that sugar-uptake alone is not the real source for dental decay. In the third column, glucose is again pulsed, but now there is no pH control. We see in this case that *S. mutans* greatly increase in number due to decrease in pH. It was concluded that it was the low pH and not the availability of the carbohydrates itself that was selecting for the unfavorable disease causing bacteria.

The fourth column shows the effect of NaF on oral bacteria. We see that when fluoride is added the pathogenic bacteria are destroyed even under conditions of low pH. This will be discussed later.

This experiment was repeated to find out if there was a “critical pH” in which homeostasis of the oral cavity broke down. To do this, the mixed culture was again pulsed with glucose in three repeating experiments. However, this time the pH was controlled to fall at values at around pH 5.0. Results showed that the microbial community was irreversibly disrupted only when the pH fell below 5.0. The results showed that when the pH was lower than 5.0, the *S. mutans* dominated.

Based on the Pure Culture and Mixed Culture studies, the “specific plaque hypothesis” was clearly not the most accurate assessment of the cause of dental caries. It is not the mere existence of *S. mutans* that causes dental caries and disease. Rather the most accurate hypothesis, based on the above mixed culture studies, became known as the “ecological plaque hypothesis” (Takahashi and Nyvad 2008). This hypothesis suggests that key changes in environmental conditions trigger shifts in the balance of resident plaque microflora. It is these environmental conditions that allow the sites to become predisposed to disease. It is at this time that the *S. mutans*, which has the ability to survive in these adverse conditions which altered the resident microflora (i.e. low pH), contributes to dental caries and disease.

In other words, the ecological plaque hypothesis can be explained as follows. The mouth of a healthy individual always contains some amount of *S. mutans*. However, under normal healthy environmental conditions, there are many more healthy bacteria which can survive, rendering the *S. mutans* as a small percentage, weakly competitive, and clinically insignificant.

Yet, if a person would do something to alter the environment, pathogenic bacteria can become more competitive. For example, if an individual would increase the intake frequency of fermentable sugars, the plaque environment would become much more acidic (lower pH). This low pH environment favors the growth of *S. mutans*, as shown in the above mixed culture experiment. At this time, the *S. mutans* is strongly competitive now that it can render the bacteria associated with healthy enamel as insignificant. This causes the dental caries and disease.

An important aspect of the ecological plaque hypothesis is the idea that disease can be prevented in ways that do not directly inhibit pathogenic bacteria. Prevention strategies can be formed that serve to interfere with the underlying environmental factors that enabled the growth of the pathogenic bacteria in the first place. By finding ways to maintain ecological stability in the oral cavity, we can prevent *S. mutans* from ever attaining the strength necessary to cause harm.

As mentioned, it is the highly acidic (low pH) environment which is favorable to *S. mutans*. Thus, mechanisms that can combat the *S. mutans* viability by also competing in these acidic environments can serve as a beneficial source of prevention (Marsh 2001). Thus, the most common strategy to prevent disease via the ecological plaque hypothesis is the ability to decrease the growth of *S. mutans* even in highly acidic enamel. Studies have showed that

fluoride has such capabilities. Fluoride has the capability to stabilize the environment of the enamel and plaque even during times of low pH, rendering the *S.mutans* ineffective. Thus, Fluoride Therapy became the main prevention strategy that is based on the ecological plaque hypothesis.

Based on a similar experiment to the Mixed Culture Study mentioned above, researchers have determined that fluoride can serve to be inhibitory toward pathogenic bacteria even in environments without pH control (i.e. low pH). To test this theory 1 mmol/L of sodium fluoride was pulsed along with glucose into a mixed culture of three oral bacteria (Table 2).

| <b>Table 2:</b><br>Effects of fluoride on the stability of a mixed culture of three oral bacteria with 10 glucose pulses on 10 consecutive days with 28 mmol/L glucose and without pH control (percentage viable count) |  |  |                                       |
|---|--|--|---------------------------------------|
| Bacterium   | Pre-Pulsing<br>without Sodium Fluoride | After Pulsing<br>without Sodium Fluoride | After Pulsing<br>with Sodium Fluoride |
| <i>S. gordonii</i>  | 28.3                                   | 0.2                                      | .002                                  |
| <i>S. oralis</i>  | 15.2                                   | 1.3                                      | 4.6                                   |
| <i>S. mutans</i>  | 0.3                                    | 18.9                                     | 0.2                                   |

Source: Bradshaw et al. 1989

The results clearly show the ability of fluoride to inhibit the competitive capabilities of *S. mutans*. In the first column, at pre-pulsing we see the environment of healthy enamel. The two bacteria associated with health (*S. gordonii* and *S. oralis*) are at much higher concentration than *S. mutans*. In the second column, we see the *S. mutans* become more competitive due to the glucose pulsing and lack of pH control. This is the pathogenic environment seen in the earlier Mixed Culture Experiment. The third column represents the presence of fluoride in the acidic environment. As soon as fluoride is introduced, the *S. mutans* is seen to be inhibited, thus restoring the enamel culture to health (Aioba and Fejerskov 2002). Clearly, the great strides researchers have taken in defining the specific reason for dental caries, via the ecological plaque hypothesis, have allowed scientists to form prevention strategies based on these very principles.

## CONCLUSION

In conclusion, it is evident from the research presented that ecological factors in the oral cavity play a large role in the effectiveness of *S. mutans* to cause dental caries. According to the ecological plaque hypothesis, *S. mutans* is always present. However, it is a specific environmental cue, such as low pH, which allows the *S.mutans* to become more competitive and outlive the bacteria associated with healthy enamel. Armed with this knowledge, scientists were able to realize fluoride's capability to prevent dental caries by interacting and inhibiting *S. mutans* even in adverse ecological conditions.

## REFERENCES

- Aioba T, Fejerskov O. 2002. Dental Fluorosis: Chemistry and Biology. *Critical Reviews in Oral Biology & Medicine* 13(2):155-170.
- Bowden, GHW. 2000. The Microbial Ecology of Dental Caries. *Microbial Ecology in Health & Disease* 12(3):138-148.
- Bradshaw DJ, McKee AS, Marsh PD. 1989. Oral Bacteria communities in vitro. *J Dent Res* 68: 1298-1302

- Busscher HJ, Van der Mei HC. 2000. Initial microbial adhesion events. In: Allison DG, Gilbert P, Wilson M, editors. *Community Structure and Cooperation in Biofilms*. Cambridge: Cambridge University Press. pp 25-36.
- Emilson CG, Krasse B. 1985. Support For and Implications of the Specific Plaque Hypothesis. *Scandinavian Journal of Dental Research* 93(2):96-104.
- Loesche WJ. 1996. Microbiology of Dental Decay and Periodontal Disease. 4th ed. In: Baron S, editor. *Medical Microbiology*. Galveston: University of Texas Medical Branch at Galveston. Chapter 99.
- Marsh PD. 2001. Dental Plaque as a Microbial Biofilm. *Caries Research* 38(3):204-211.
- Marsh PD, Moter A, Devine DA. 2006. Dental plaque biofilms: communities, conflict and control. *Periodontology* 55(1):16-35.
- Savage D. 1977. Microbial Ecology of the Gastrointestinal Tract. *Annual Review of Microbiology* 31:107-33.
- Scannapieco F. 1994. Oral Ecology. *Critical Review of Oral Biological Medicine* 5:203-248
- Takahashi N, Nyvad B. 2008. Caries Ecology Revisited: Microbial Dynamics and the Caries Process. *Caries Research* 42(6):409-418.