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Bacterial Specificity in the Etiology of Caries and Periodontal Disease

R. C. KESTENBAUM

With the evolution of the germ theory of disease, it became plausible that bacteria might be related in some way to oral diseases. The advent of antibiotic therapy offered a new way of demonstrating that microorganisms are implicated in the carious process. Studies by numerous investigators demonstrated the ability of antimicrobial agents to limit the carious process in experimental animals. Another relatively modern process, gnotobiotics, permitted still another approach to the implication of bacteria in caries formation. This led to the isolation, from human beings and rodents, of specific streptococci capable of inducing smooth-surface caries in gnotobiotic and conventional model rodent systems. These organisms also were shown to have the capacity to produce extracellular polysaccharides. Sucrose, the primary carbohydrate source for formation of extracellular polysaccharides, also was found to be necessary for implantation and caries production by these organisms, which suggests a possible role for these materials.

Examination of these cariogenic streptococci for the types of extracellular polysaccharides showed that some organisms produced primarily dextrans, and others produced levan from the sucrose. The inability of the oral flora to degrade dextran further suggested that its formation would be an important factor in the establishment of a cariogenic flora. Although levans can be degraded by oral microbes, it was demonstrated that implantation of a levan-forming organism into animals containing dextran-forming streptococci greatly increased caries.

A possible explanation of the role played by extracellular materials may be found in the nature of these substances. Both dextrans and levans are highly viscous, sticky materials. From sucrose medium, cariogenic organisms can produce gooey masses that adhere to glass. A similar phenomenon could occur on the hard surfaces in the oral cavity. Such an occurrence could give these acidogenic organisms an advantage over other

organisms in maintaining themselves on the tooth structure, where their metabolic products could cause destruction.

Thus, it appears that two parameters may have key roles in the carious process. The first is the ability to form plaque on smooth surfaces. The production of dextrans or levans that are not readily degraded by the oral flora may provide certain organisms with an increased ability to maintain themselves on the smooth surfaces of teeth. Adhesion of these organisms to the tooth surfaces also may provide some added protection to the other members of the oral microbiota, holding them more firmly and possibly protecting them from the cleansing action of the oral cavity. As such, these materials would serve to increase plaque.

However, increased plaque does not by itself produce caries. A model rodent system has been found that produces masses of plaque in hamsters without producing smooth-surface caries. This organism, *Odontomyces viscosus*, although it is a levan producer, induces massive soft tissue damage and bone resorption indicative of periodontal disease. However, it is only weakly acidogenic as opposed to the strongly acidogenic nature of streptococci and lactobacilli. Thus, the second key parameter in the carious process would appear to be the ability to produce sufficient amounts of acid to destroy the enamel surfaces.

In the discussion of bacterial specificity, several organisms and a number of possible mechanisms for the destruction of hard and soft tissues have been mentioned. However, perhaps the key suggestion to be made is that we now know that certain specific organisms can have an increased capacity to form plaque through the production of extracellular polysaccharides. Although the mechanisms for destruction differ in caries and periodontal disease, in both infections the most important role of specific bacteria may be based on their ability to increase the quantity of plaque produced.