**Title:** Effect of Nicotine on a Mixed Culture of *Streptococcus sanguis* and Streptomycin-Resistant *Streptococcus mutans*

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*Streptococcus sanguis* is a gram-positive, normal inhabitant of the oral cavity. Another gram-positive bacterium, *Streptococcus mutans* is a known causal agent of plaque and dental caries. *S. mutans* modifies the oral environment to make it less hospitable for other streptococcal strains by producing bacteriocins. It is well established that smokers have an increased rate of caries as a result of a higher proportion of *S. mutans* than *S. sanguis*. One of the important virulence properties of *S. mutans* is the ability to form biofilm on tooth surfaces. The aim of this study was to show that exposure of high concentrations of nicotine would provide *S. mutans* with a competitive advantage that would lead to an inverse relationship in which streptomycin-resistant (*S. mutans*R) *S. mutans* would overshadow the presence of *S. sanguis* possibly by binding to the majority of available tooth surfaces by increased bacteriocins. *S. mutans* UA159 was made resistant to streptomycin (*S. mutans*R) by step-wise growth on Tryptic Soy Agar (TSA) with 1 mg/ml streptomycin. Tryptic soy broth with 1% sucrose (TSBS) media ranging from 0 to 1 mg/ml of nicotine was added to sterile six-well plates containing three hydroxyapatite disks (representing tooth enamel) per nicotine concentration and inoculated with overnight TSB cultures of *S. mutans*R and *S. sanguis* 10556. Plates were incubated overnight at 37°C with 5% CO₂. The disks were aseptically rinsed with sterile saline to remove non-adherent bacteria and placed into a tube with sterile saline to dislodge biofilm. The biofilm was spiral plated onto 1 mg/ml streptomycin-containing TSA (to enumerate *S. mutans*) and 0 mg/ml streptomycin TSA (to enumerate both *S. mutans* and *S. sanguis*). Plates were incubated overnight at 37°C with 5% CO₂ before being counted using an automated colony counter. The data obtained suggests a dosage-dependent effect of nicotine on the growth of *S. mutans*. *S. mutans*R predominated over *S. sanguis* in the 0 mg/ml of nicotine control possibly as a result of *S. mutans* bacteriocin production. Moreover, a trend of increased *S. mutans*R was seen in 0.25 mg/ml of nicotine compared to the control. It is hypothesized that low levels of nicotine increased bacteriocin production which led to a predominance of *S. mutans*R over *S. sanguis*. Increasing concentrations of nicotine reduced the number of *S. mutans*R in a mixed biofilm culture most likely resulting from lysis. This study confirmed earlier results demonstrating that nicotine upregulated the ability of *S. mutans* to kill *S. sanguis* resulting in exclusion of *S. sanguis*. This provides further evidence of the role of nicotine in caries formation in smokers. Increased numbers of *S. mutans* would bind to available sites on tooth surfaces, creating a destructive biofilm, leading to increased demineralization of enamel. The dispersal properties of biofilm would allow it to spread to nearby surfaces and ultimately result in increased caries.