

Mechanisms of Attachment of Tobacco-Treated *Streptococcus mutans* to Human Endothelial Cells
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Smoking has been proven to cause increased dental caries, which is an infectious disease caused by *Streptococcus mutans*, a gram-positive bacteria commonly found in the oral cavity. *S. mutans* is also known for its contribution to atherosclerosis, specifically the accumulation of plaque in the coronary arteries. This is facilitated by the interaction and binding of *S. mutans* to local endothelial cells (HUVEC). This study was conducted to explore the direct effects that tobacco has on the ability of *S. mutans* to affect endothelial cells that might lead to atherosclerosis. *S. mutans* were treated with different concentrations of nicotine and cigarette smoke condensate (CSC) to test if they affect the binding capabilities of *S. mutans* to endothelial cells. Blocking reagents, enolase antibody and purified DnaK, were also used to treat the HUVEC to observe the effects these reagents have on the ability of *S. mutans* to bind to the cells. Binding was measured by performing a binding assay that incorporated these reagents and reading the absorbance using a spectrophotometer at 450 nm. To do this, sonicated HUVEC were added to a 96-well microtiter plate with 1% bovine serum albumin (BSA), followed by treated *S. mutans*, extra-avidin labeled horseradish peroxidase, and O-phenylenediamine (OPD). The experiment is still in progress; therefore, no results have been obtained thus far. However, it is expected that the nicotine/CSC treatment of *S. mutans* will increase binding to the endothelial cells thereby providing a possible mechanism of *S. mutans* contributing to atherosclerosis. The knowledge obtained from this experiment will be significant in developing treatment modalities to decrease the effects of smoking on cardiovascular disease.

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