

Serotype k *Streptococcus mutans* Binding to Collagen and Fibrinogen in Nicotine

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Background: *Streptococcus mutans* is a gram-positive coccus-shaped, facultatively anaerobic bacterium that is commonly found in the human oral cavity and is a major contributor to tooth decay. The bacterium has the potential to make its way into the blood stream and adhere to endothelial cell proteins such as collagen and fibrinogen in the arteries through specific receptors potentially leading to atherosclerosis. Endothelial cells secrete cell-associated and cell-free collagen and fibrinogen. Specifically, serotype k *S. mutans* have been associated with atherosclerosis and nicotine has been shown to increase the biofilm formation of *S. mutans* (serotype k). The focus of this research was to measure *S. mutans* ability to bind to collagen type I and fibrinogen when the cells were grown in the presence of nicotine. **Methods:** *S. mutans* serotype k strains 51, 52, and 89 were cultured in 0–2 mg/mL nicotine. Formaldehyde was added to kill the cells followed by labeling the cells with biotin. Collagen type I and fibrinogen were coated (1 µg/mL) onto 96-well microtiter plates. The plates were washed and 1% BSA was added to block the wells. Then the biotinylated nicotine-treated *S. mutans* were added, incubated to allow binding to the endothelial cell proteins, and washed. Finally, ExtrAvidin HRP and OPD were added to the plate and the optical density was measured at an absorbance of 490 nm. **Results:** The optical density was directly related to the relative number of cells bound to collagen type I and fibrinogen. **Conclusion:** The results demonstrated a significant increase in all three strains of *S. mutans* binding to the proteins when cultured in 1 and 2 mg/mL concentrations of nicotine compared to the 0 nicotine control. The increased numbers of nicotine-treated *S. mutans* binding to the endothelial cell proteins may have the ability to contribute to atherosclerosis.