Effects of Nicotine on Aerobic and Anaerobic Serotype K *Streptococcus mutans* Biofilm Formation Nicole Quint<sup>1</sup>, Grace Gomez<sup>2</sup>, Richard L. Gregory<sup>1</sup>

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Atherosclerosis is a specific form of arteriosclerosis where the walls of arteries began to thicken as a result of bacterial invasion and accumulation of inflammatory white blood cells. There could be a direct correlation of atherosclerosis and the intake of nicotine. Nicotine has been reported to increase the amount of the cariogenic oral bacteria known as Streptococcus mutans; thus possibly leading to an increase of dental caries. Serotype K S. mutans has been associated strongly with atherosclerosis. Objective: This study focused on the biofilm formation of S. mutans serotype K when incubated in various dilutions of nicotine. Methods: S. mutans UA159 (stereotype C), and stereotype K strains 89, 52, and 51 were cultured in tryptic soy broth (TSB) overnight and then added to dilutions of TSB with 1% sucrose (TSBS) containing concentrations of nicotine between 0 and 32 mg/ml. Each dilution was added to 96-well microtiter plates, inoculated with bacteria and incubated for 24 hours aerobically at 37°C in 5% CO<sub>2</sub> and anaerobically. The plates were treated with formaldehyde, crystal violet, and isopropanol and biofilm formation was measured at an absorbance of 490 nm. Results: Strains UA159, 89, 52, and 51 all demonstrated significantly higher biofilm formation (p<0.05) at a nicotine dilution of 8 mg/ml. When comparing the anaerobic results to the aerobic results, anaerobic incubation increased the overall biofilm formation across the majority of nicotine dilutions. Conclusion: It was established that when S. mutans strains UA159, 89, 52, and 51 were incubated anaerobically and aerobically biofilm formation was enhanced. Smoking can lead to a higher population of S. mutans in the oral cavity that potentially has traits of significantly enhanced biofilm formation when presented with moderately high levels of nicotine which may lead to increased binding to endothelial cells contributing to atherosclerosis.

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