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 preforming 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.

Mentors: Richard L. Gregory, L. Jack Windsor, and Fengyu Song, Department of Oral Biology, Indiana University School of Dentistry, Indianapolis, IN 46202