

Streptococcus mutans Binding to Collagen, Fibrinogen, Fibronectin, and Laminin
Sylvie N. Kristoff¹, Richard L. Gregory²

¹Dept. of Biology, Indiana University-Purdue University Indianapolis, IN

²Dept. of Oral Biology, Indiana University School of Dentistry, Indianapolis IN

Introduction: *Streptococcus mutans*, nicotine, and certain proteins may be involved in a complicated mechanism that contributes to atherosclerosis. Build up of arterial plaque causes atherosclerosis. Arterial plaque is mainly composed of fat, cholesterol, and calcium. When plaque builds up in the arteries, a clot or blockage can occur and may cause an occlusion.

Objective: *S. mutans* grows in oral biofilm and causes dental caries. These bacteria enter the blood stream from mucosal breaks in the oral cavity. There is evidence that *S. mutans* binds to endothelial cell surface proteins lining arterial surfaces. An increased incidence of *S. mutans* in arterial plaque seems to have a direct relationship with atherosclerosis. From preliminary research, there was a strong indication that increased *S. mutans* biofilm formation is caused by nicotine. The number of binding proteins on nicotine-treated *S. mutans* cell surface increases as well. In addition, results demonstrated that *S. mutans* binds to collagen type I, fibrinogen, fibronectin, and laminin, which are proteins found on endothelial cells.

Methods: To investigate protein binding, *S. mutans* UA159 was cultured in 0, 0.25, 0.50, 1.0, 2.0, and 4.0 mg/ml of nicotine and their ability to bind to human collagen type I, fibrinogen, fibronectin and laminin was assessed using an ELISA assay.

Results: *S. mutans* significantly bound to collagen type I and fibrinogen when cultured in 2 and 4 mg/ml nicotine. *S. mutans* significantly bound to laminin when the bacterium was grown in 1, 2, and 4 mg/ml. The binding of *S. mutans* to fibronectin varied when cultured in different concentrations of nicotine.

Conclusion: From the results, it can be concluded that *S. mutans* UA159 binds to collagen type I, fibrinogen, fibronectin, and laminin. This indicates that *S. mutans* and the proteins studied are very likely to be part of the mechanism that leads to atherosclerosis.