Effects of nicotine and glucose on production of inflammatory mediators in response to IL-1 and LPS in human gingival fibroblasts (HGF)

Sharlenne Sanchez, Mariah Chambers, William Laidlaw, Mansoor Madani, Ruth C. Borghaei

Department of Biochemistry/Molecular Biology, Philadelphia College of Osteopathic Medicine, Philadelphia, PA 19131

ABSTRACT

Periodontitis is the most common cause of adult tooth loss in the U.S., with an estimated 1 in 3 adults suffering from some form and 10 to 15% of adults developing severe form. In addition to its direct impact, periodontitis also contributes to the development of several other diseases, including cardiovascular disease, pre-term low birth weight, diabetes, and oral cancer. Since smoking and diabetes are frequently co-existing, modifiable risk factors for the development of periodontitis, as well as for cardiovascular disease and some types of cancer, this study is designed to determine whether and to what extent these factors interact to affect the production of proteins and products that are involved in the pathogenesis of these conditions. HGF cultures derived from patients with periodontitis were incubated with IL-1, LPS, nicotine and glucose in various combinations for 24 hours, and the levels of matrix metalloproteinase 1 and 3 in conditioned media were measured by ELISA. Results show that while nicotine alone has no effect on MMP-3 protein expression, it acts synergistically with IL-1 or LPS to increase its expression further. Glucose alone has no effect on basal or IL-1 induced MMP-3 expression, but counteracts the effects of nicotine. Similar effects were seen with MMP-1. Further experiments are planned to determine the effects of nicotine and glucose on these and other mediators of inflammation.