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“Effects of pro-inflammatory cytokines on polarized salivary epithelium”

Lay Abstract:

Sjögren’s syndrome (SS) is associated with the increased production of pro-inflammatory cytokines by infiltrating lymphocytes or by salivary epithelium. Little is known about the effects of these cytokines on salivary gland dysfunction in SS. Therefore, studies in this proposal will utilize a cell line derived from salivary gland to evaluate the role of these cytokines on ion secretion and on epithelial integrity in salivary glands. These studies may lead to a better understanding of the causes of salivary gland dysfunction that contribute to decreased saliva production in patients with SS.

Scientific Abstract and Research Proposal:

Sjögren’s syndrome (SS) is an autoimmune disease that mainly affects the exocrine glands and usually presents as persistent dryness of the mouth and eyes due to functional impairment of the salivary and lacrimal glands. The salivary dysfunction in SS is often associated with increased production of pro-inflammatory cytokines such as interleukin-1 β (IL-1 β), interleukin-6 (IL-6), tumor necrosis factor- α (TNF α) and interferon- γ (IFN γ). Pro-inflammatory cytokines associated with SS can be produced by infiltrating lymphocytes or salivary epithelium, thus implicating the salivary epithelium as a target and a source of cytokines. Although pro-inflammatory cytokines in SS may affect salivary gland functions, little is known about their effects on salivary epithelial cell tight junction (TJ) integrity and transcellular ion transport properties, information that is relevant to the mechanisms underlying infiltration of lymphocytes that target salivary gland in SS. Therefore, studies in this proposal will utilize polarized rat parotid (Par-C10) monolayers to evaluate the effects of these pro-inflammatory cytokines on transepithelial anion secretion (I_{sc}) and expression and phosphorylation of epithelial cell TJ proteins that regulate ion and epithelial integrity in salivary glands. These studies may lead to a better understanding of the pathogenesis of autoimmune-associated dysfunction of salivary gland that contributes to xerostomia in patients with SS.