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$P2Y_2$ nucleotide receptors mediate inflammatory responses in mouse salivary gland cells

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Sjögren's syndrome (SS) is a chronic inflammatory autoimmune disease characterized by destruction of salivary and lacrimal glands leading to xerostomia (dry mouth) and xerophthalmia (dry eyes). Although the mechanisms involved have not been adequately elucidated, the diminished function of exocrine glands in SS is often associated with lymphocytic infiltration of the tissue. Aberrant expression of specific adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) and intracellular cell adhesion molecule-1 (ICAM-1) is also observed in salivary gland with SS, which enable salivary epithelium to interact directly with infiltrating lymphocytes. $P2Y_2$ nucleotide receptor ($P2Y_2R$) is G protein-couple receptor that is activated by extracellular ATP and UTP. $P2Y_2R$ expression and activity is up-regulated in response to damage or stress in a variety of tissues, including submandibular glands (SMGs), where it mediates a complex set of cellular responses to injury of disease. Additionally, $P2Y_2R$ activation up-regulates VCAM-1 expression in dispersed rat SMG cell culture and human submandibular gland (HSG) cells. Our objective is to investigate weather $P2Y_2R$ up-regulation correlates with increased expression of adhesion molecules in SMGs from a mouse model for SS (C57BL/6.NOD-Aec1Aec2) as compared with normal mouse strain (C57BL/6). $P2Y_2R$ expression was measured by RT-PCR and adhesion molecules expression was determined by Western blot analysis. Salivary flow was preformed by cannulation of individual glands. We could see that $P2Y_2R$ expression and ICAM-1 expression were both up-regulated in the SMGs from a mouse model for SS as compared with normal mouse strain. And salivary flow was decreased in salivary glands from a mouse model for SS. These results suggest that $P2Y_2R$ mediate inflammatory responses related to secretory dysfunction in the mouse model for SS. Our ultimate goal would be to translate all this information to the human salivary gland in order to understand SS and to develop new therapies for salivary dysfunction in SS.