

Public Abstract

First Name:Qixing

Middle Name:

Last Name:Liang

Adviser's First Name:Gary

Adviser's Last Name:Weisman

Co-Adviser's First Name:

Co-Adviser's Last Name:

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Title:MECHANISMS OF SALIVARY GLAND CELL PROLIFERATION IN VITRO BY P2Y2R ACTIVATION

Sjogren's syndrome (SS) is an autoimmune disease in which exocrine glands including the salivary gland are targeted and destroyed by the immune system. SS and the side-effects of gamma-radiation therapies for head and neck cancers cause salivary gland dysfunction, in particular hyposalivation. Salivary gland regeneration has been considered to be a very promising approach for restoring saliva secretion. To regenerate a functional salivary gland in a clinical setting, it is first necessary to gain a better understanding of the mechanisms involved in salivary gland regeneration. This thesis describes our attempts to understand mechanisms that increase salivary gland cell proliferation which is an important part of regeneration.

P2Y2 receptor (P2Y2R) is a G protein-coupled receptor on cell membrane that can be activated by ATP or UTP. Activation of P2Y2Rs has been shown to stimulate proliferation in many cell types. Our previous studies have shown that the P2Y2R interacts with many signaling molecules to regulate multiple signaling pathways involved in cell proliferation. These studies support the hypothesis that expression and activation of P2Y2Rs in damaged salivary gland cells promotes cell proliferation. In this project, we demonstrate that activation of the P2Y2R increases the proliferation of human salivary gland (HSG) epithelial cell. Other data indicate that inhibition of Src and ERK1/2 prevents P2Y2R-mediated proliferation of HSG cells. Thus, the current results suggest that activation of P2Y2Rs by UTP promotes proliferation of HSG cells by stimulating Src-dependent signaling pathway and activating ERK1/2. Accordingly, the P2Y2R represents a promising target for salivary gland regeneration.