

# Disruption of tight junction structure in salivary glands from Sjögren's syndrome patients is linked to proinflammatory cytokine exposure

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**Objective.** Disorganization of acinar cell apical microvilli and the presence of stromal collagen in the acinar lumen suggest that the labial salivary gland (LSG) barrier function is impaired in patients with Sjögren's syndrome. Tight junctions define cell polarity and regulate the paracellular flow of ions and water, crucial functions of acinar cells. This study was undertaken to evaluate the expression and localization of tight junction proteins in LSGs from patients with SS and to determine in vitro the effects of tumor necrosis factor  $\alpha$  (TNF $\alpha$ ) and interferon- $\gamma$  (IFN $\gamma$ ) on tight junction integrity of isolated acini from control subjects. **Methods.** Twenty-two patients and 15 controls were studied. The messenger RNA and protein levels of tight junction components (claudin-1, claudin-3, claudin-4,

occludin, and ZO-1) were determined by semiquantitative reverse transcriptase-polymerase chain reaction and Western blotting. Tight junction protein localization was determined by immunohisto-chemistry