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Examining the molecular genetic changes in the etiology of Sjögren’s Syndrome
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Abstract for the Research Proposal:
Sjogren’s syndrome (SS) is an autoimmune disease characterized by lymphocytic infiltration of lacrimal and salivary glands. My preliminary studies revealed that minor salivary glands from SS-compatible patients presented with a distinct phenotype that included loss of cell polarity and defective adherens junctions (AJs) compared to tissues not compatible with SS. Our hypothesis is that subsets of SS cases arise from dysregulation of cell-cell and cell matrix adhesion and from the disruption of the extracellular matrix (ECM) that leads to the perturbation of cytoskeletal organization, loss of cell polarity and impaired secretory function. Using molecular, cell biological and biochemical approaches, we will determine expression, membrane localization, scaffold organization and cytoskeletal engagements of cell-cell and cell matrix adhesion receptors, polarity proteins and ECM composition. Properties of adhesion receptors will be aligned with their N-glycosylation status. Detected changes in expression levels of specific proteins will be further analyzed for mutations.