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Activation of the type I interferon pathway in primary Sjogren's syndrome.

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Abstract

Sjogren's syndrome (SS), a chronic autoimmune systemic disease affecting middle aged women, is characterized by lymphocytic infiltration of the salivary and lachrymal glands resulting in dry eyes and dry mouth. Recent advances have revealed a major role for activation of the type I interferon (IFN) pathway in the pathogenesis of the syndrome, as evidenced by the increased circulating type I IFN activity and an IFN "signature" in peripheral blood mononuclear cells (PBMC) and minor salivary gland (MSG) biopsies from these patients. Polymorphisms in genes involved in the IFNalpha pathway, such as IRF5 and STAT4, have been found to be associated with disease susceptibility. While the initial triggers of the innate immune response in SS remain elusive, preliminary evidence supports the role of inappropriately expressed endogenous LINE-1 (L1) retroelements as potential triggers of type I IFN activation in SS, possibly through Toll-like receptor (TLR) dependent or independent pathways. Proteins of the methylation machinery and the APOBEC family of cytidine deaminases are coordinately overexpressed, suggesting that those proteins might contribute to regulation of the inappropriately expressed L1 endogenous retroelements in SS. Given the apparent central role of IFNalpha in the pathogenesis of SS, blockade of this cytokine may be a rational therapeutic approach. In the current review we summarize the current evidence regarding the potential triggers of type I IFN activation as well as the data supporting genetic and epigenetic regulation of the type I IFN system in SS.

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