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Study provides new insight in the fight against Sjogren's syndrome

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Sjögren's syndrome is the second most common autoimmune disease affecting four million Americans—yet treatments are limited due to a lack of knowledge about its causes. A new study from the Forsyth Institute is helping to shed light on what happens in the development and the life cycle of the disease. This study is one of the first to define the immune-regulatory mechanisms operating in Sjögren's syndrome and provides a new foundation for fighting the disease.

Two specific proteins in the body, programmed death-ligand 1 (PD-L1) along with PD1 have been found to work together to suppress both protective immunity and autoimmune responses to prevent organ damage. According to research published in *Scientific Reports*, scientists at Forsyth led by Dr. Qing Yu have shown that blockade of the PD-L1-PD-1 pathway made the autoimmune response worse and accelerated the development of Sjögren's syndrome.

The hallmark symptoms of Sjögren's syndrome are dry mouth and dry eyes. However, the disease also affects many other organs and causes an array of health complications including B cell lymphoma. In this study, the research team investigated the role of PD-L1 in Sjögren's by inhibiting its function in a common disease model (non-obese diabetic mice). They found that PD-L1 hinders the development and onset of the disease.

The study, titled, "Endogenous programmed death ligand-1 restrains the development and onset of Sjögren's syndrome in non-obese diabetic

mice," is published on December 14th in *Scientific Reports*. This study investigated the previously un-explored role of the PD-L1 pathway in Sjögren's syndrome. Using an experimental mouse disease model, Dr. Yu and her colleagues characterized how the pathway serves to impede and limit the autoimmune responses and the pathologic development of this disease. This novel finding is one of the first to define the immune-regulatory mechanisms operating in Sjögren's syndrome, and provides initial foundation and justification for harnessing the immune-inhibitory pathways, such as PD-L1-PD-1, to combat this high-impact autoimmune disease.

More information: *Scientific Reports*www.nature.com/articles/srep39105

Provided by Forsyth Institute