Modulating the Dynamics of the Autoimmune Response in the Pancreatic Islets in Type 1 Diabetes

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Abstract:
The destruction of the insulin-producing pancreatic islets that causes type 1 diabetes is largely caused by T cells. However, T cells do not act independently to drive disease. We focus on understanding environmental factors and immune cell interactions that activate or inhibit the pathogenic T cells that damage the islets. Our long term goal is therapeutically enhance existing tolerance pathways and disrupt pathogenic pathways in the islets to treat type 1 diabetes.

Bio:
Rachel Friedman first got hooked on Immunology in college. She then worked on vaccine development as a technician before attending the University of California San Francisco where she completed her PhD in Biomedical Research, studying basic mechanisms of T cell activation. She then completed a postdoctoral fellowship in Immunology at the University of California San Francisco studying the immune response at the disease site in type 1 diabetes. In 2011, Dr. Friedman established her independent research group as an Assistant Professor in the Departments of Biomedical Research at National Jewish Health, and Immunology & Microbiology at the University of Colorado School of Medicine. The Friedman Lab is focused on understanding how environmental cues and immune cell interactions at the disease site drive pathogenesis or tolerance in autoimmune diabetes, with the goal of developing new therapeutics to disrupt autoimmune responses.