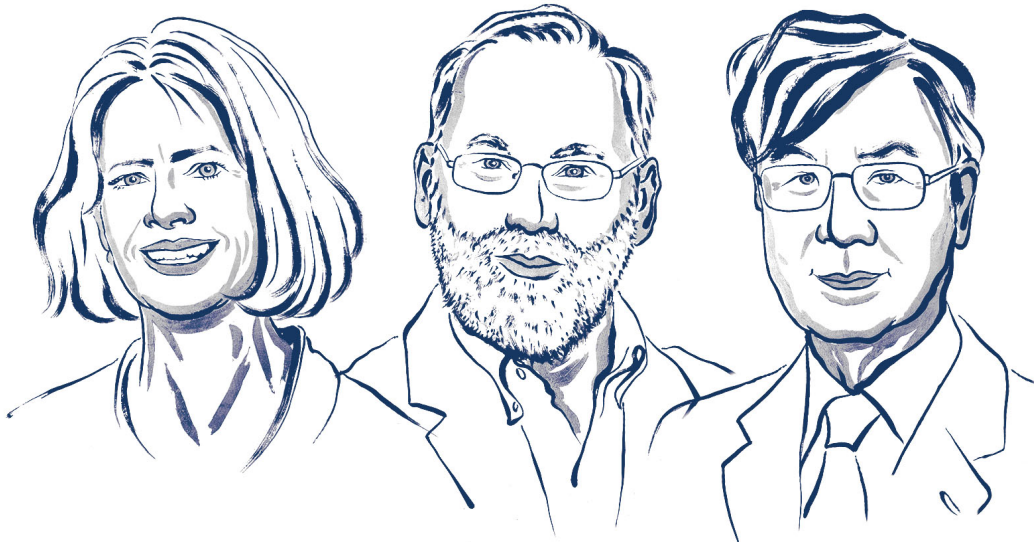

2025 Nobel Prize in Physiology

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The Nobel Assembly at Karolinska Institutet have award the 2025 Nobel Prize in Physiology or Medicine to **Mary E. Brunkow, Fred Ramsdell, and Shimon Sakaguchi** and their discoveries which have completely helped change the landscape of our understanding of how autoimmune diseases are prevented and controlled.

This wonderful news circles back to UCSF Diabetes Center and our founding director Jeff Bluestone, who after his tenure at UCSF, went on to co-found [Sonoma Biotherapeutics](#) with Fred Ramsdell, Qizhi Tang and Alexander Rudensky, who all remain scientific advisors to the clinical-stage biotechnology company developing engineered regulatory T-cell therapies to treat autoimmune and inflammatory diseases by restoring balance to the immune system.

The UCSF Diabetes Center has a rich history on the study of Treg's in the context of Type 1 Diabetes and how they are a key gatekeeper in preventing the onset of T1D. Bluestone was the first to establish that Treg's play an important role in controlling the onset of diabetes in the NOD mouse model and took this fundamental insight into both deeper mechanistic work and clinical translation. Working together with Qizhi Tang, the team went on to demonstrate that controlling the T cell receptor specificity could enhance the function and activity of Treg's for diabetes prevention and this key insight provided the framework for bringing Treg's to the clinic.

Our Diabetes Center Research group went on to publish first in human studies for clinical trials with Treg's that have now further developed into companies dedicated to developing Treg's for autoimmune diseases including Sonoma Biotherapeutics. Ramsdell's major contribution was to identify the Treg master regulatory gene *FOXP3*. Further underscoring its role in T1D was the later discovery of patients with mutations in *FOXP3* developing the IPEX syndrome that is frequently characterized by the development of multiple autoimmune diseases that frequently includes the development of T1D in the first few months of life.

