Maximize translatability of your type 1 diabetes (T1D) agent with our preclinical drug development platform. Utilize well-established and reliable models of spontaneous and accelerated T1D to assess your agent efficacy.

Select from our clinically-relevant preclinical models of T1D based on your individual research needs:

- Non-obese diabetic (NOD) spontaneous model of T1D.
- Cyclophosphamide-induced accelerated T1D.
- Consult our scientific experts for custom model builds.

**Spontaneous T1D in NOD Mice**

- Evaluate your agents in a simple, spontaneous disease model, with no confounding immune modulation due to induction reagents.
- Understand the role of T\(_{reg}\) cells in the context of your compound MoA.
- Provides an ideal model to study human autoimmune diabetes; NOD genetic predisposition leads to 60-70% T1D incidence at 21-31 weeks of age.
- Diabetes is confirmed following two consecutive (72 hours apart) non-fasting blood glucose measurements >250mg/dL.

**Cyclophosphamide-Induced Accelerated T1D in NOD Mice**

- More rapidly assess agent efficacy in a robust model of accelerated T1D.
- Benefit from higher disease incidence than spontaneous models.
- Investigate how to reverse and/or prevent the diabetes-inducing effects of low dose cyclophosphamide routinely used for anticancer regimens.
- Diabetes is accelerated through 200mg/kg cyclophosphamide administration, reducing the number of T\(_{reg}\) cells.
- T1D is confirmed following two consecutive (72 hours apart) non-fasting blood glucose measurements >250mg/dL.

**Evaluate the Efficacy of Your T1D agent through Key Endpoints**

- Histopathology assessment of pancreas, lymphocyte infiltrates, and insulin-positive islets.
- Cytokine analysis.
- Flow cytometry analysis of immune cell populations.
- Monitor blood glucose and body weight twice weekly.