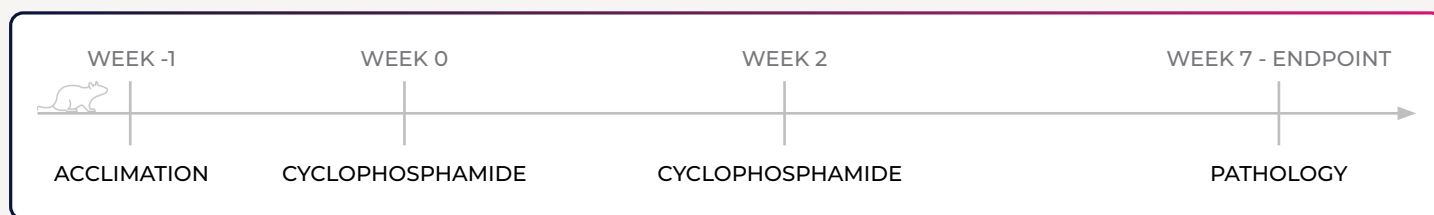


CYCLOPHOSPHAMIDE-INDUCED TYPE 1 DIABETES NOD MOUSE MODEL

Type 1 diabetes is a chronic autoimmune metabolic disorder that arises as a result of the autoimmune destruction of β cells of the endocrine pancreas.

- The pathogenesis of type 1 diabetes is multifactorial, and the following could all play a role in the genesis of autoimmune diabetes:
 - Genetic factors
 - Environmental factors
 - Prenatal and perinatal factors
 - Immunological factors
- Type 1 diabetes currently has no cure. Treatment involves the careful management of blood sugar levels using insulin. Diet and lifestyle changes are also advised to prevent complications.
- Non-obese diabetic (NOD) mice can spontaneously develop type 1 diabetes via an autoimmune pathway, but this process can take an extended period of time (around 24 weeks for 50% of mice to develop type 1 diabetes) and can be inhibited by immune tolerance.

CYCLOPHOSPHAMIDE-INDUCED TYPE 1 DIABETES PRECLINICAL *IN VIVO* MODEL



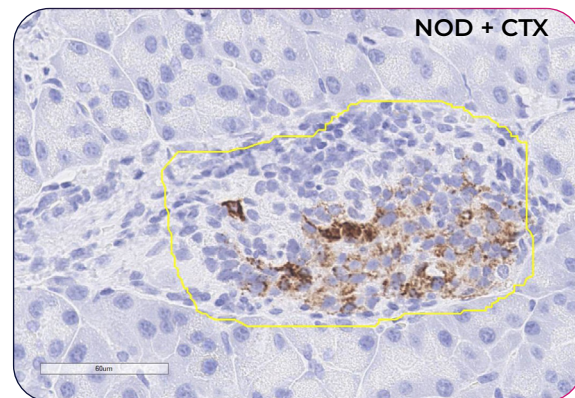
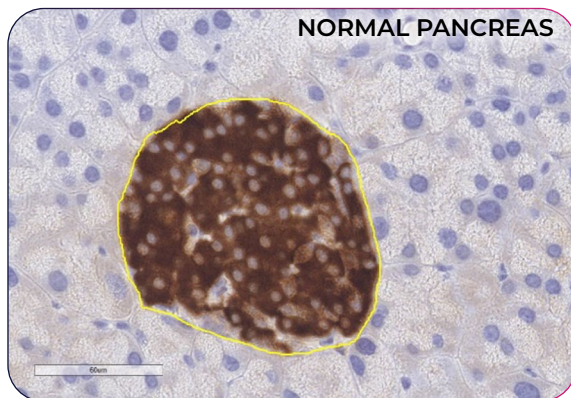
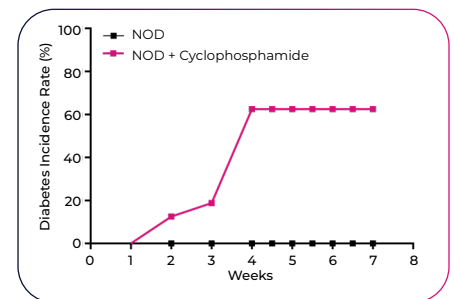
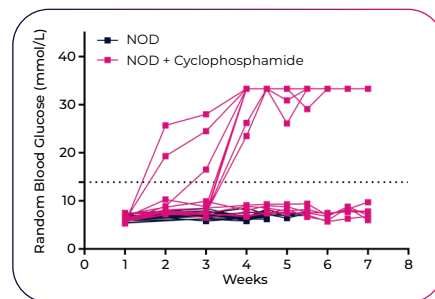
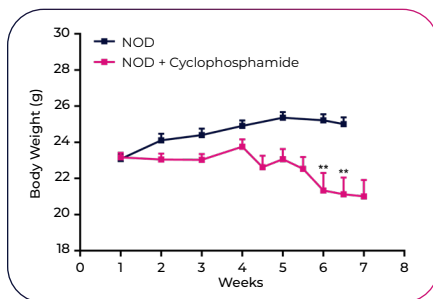
FAST
Onset of type 1 diabetes after ~4 weeks

RELIABLE
On average, 62.5% of animals develop type 1 diabetes

CLINICALLY RELEVANT
Random blood glucose levels $\geq 13.8_{\text{mmol/L}}$

CYCLOPHOSPHAMIDE-INDUCED TYPE 1 DIABETES NOD MOUSE MODEL DATA

- Mouse
 - SPF female non-obese diabetic (NOD) aged 7 weeks on arrival
- Model
 - Type 1 diabetes onset accelerated by once-a-week intraperitoneal injections of cyclophosphamide in weeks 0 and 2
- Test article administration
 - Oral
 - Intraperitoneal
 - Intravenous
 - Subcutaneous
 - *Other routes available on request*
- 62.5% of animals on study were type 1 diabetic as defined by the diabetes incidence standard: blood glucose ≥ 13.8 mmol/L measured on two separate consecutive occasions within a week
- Staining of mouse pancreas revealed a significant decrease in the number of insulin positive cells



SUMMARY

The cyclophosphamide-induced type 1 diabetic mouse model serves as a reliable and clinically relevant platform for assessing drug candidates. With an accelerated onset for disease development, lead times for assessing candidates is significantly reduced.

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