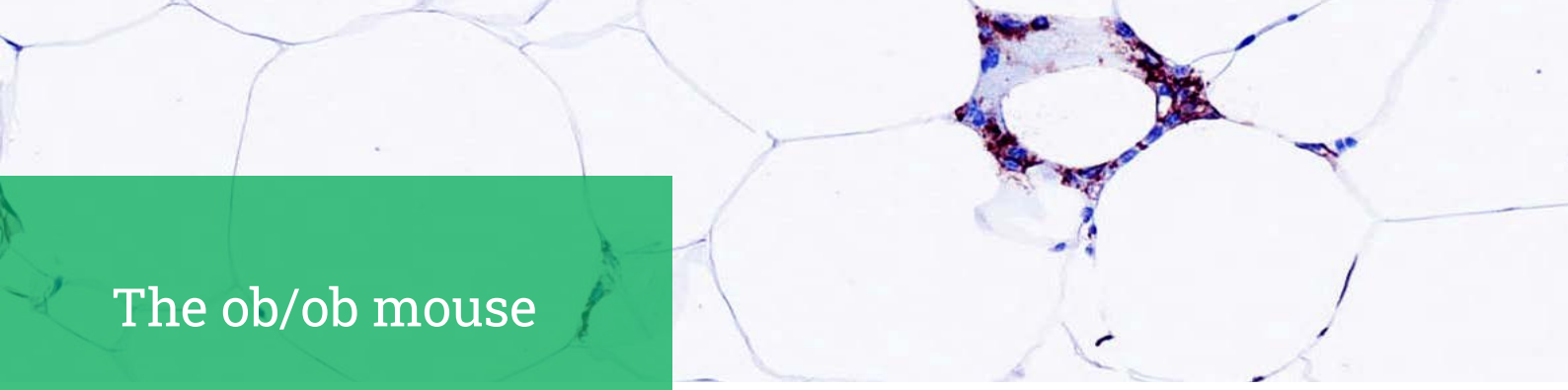


The ob/ob mouse

A hyperphagia-driven model of overt obesity for drug efficacy testing.

Standard models for profiling drug therapeutic effects in obesity

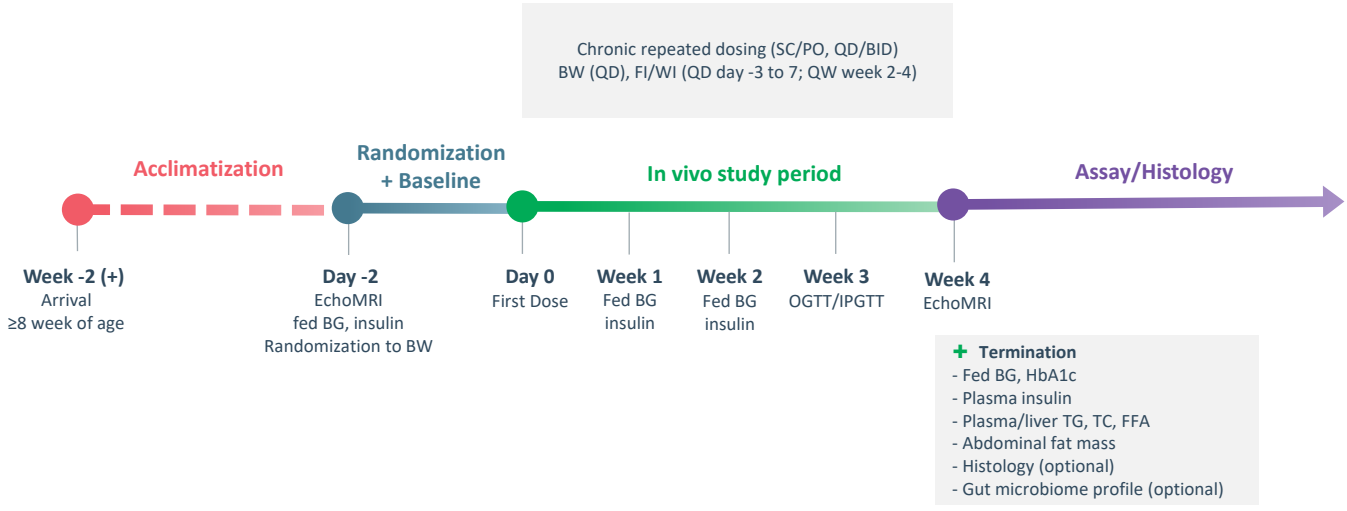
ob/ob mice carry a spontaneous 'obese' (ob) deleterious mutation in the leptin gene (Lepob), rendering the mice leptin deficient. ob/ob mice develop hyperphagia-driven severe obesity and impaired glucose homeostasis.

Key model traits

- Hyperphagia-driven overt obesity.
- Marked insulin resistance and glucose intolerance.
- Mild hyperglycemia.
- Intact leptin receptor signalling.
- Treatment efficacy across a wide range of anti-obesity and anti-diabetic drug classes.

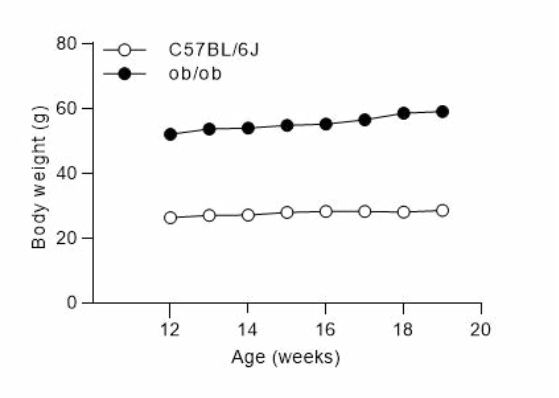
Diet	Chow (Altromin 1324)	ob/ob mice are enrolled into studies at ≥8 weeks of age. Age-matched chow-fed C57BL/6J mice may serve as lean controls.
Strain	B6.Cg-Lepob/J	

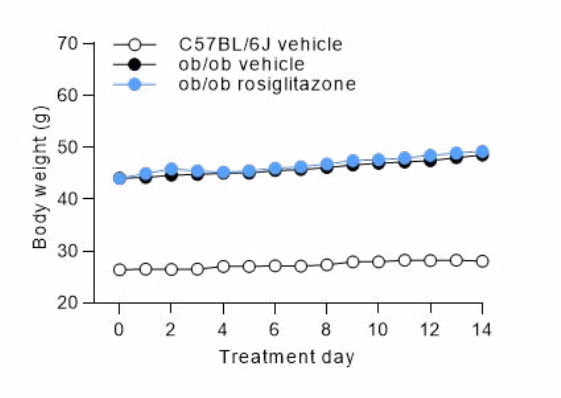
Study outline

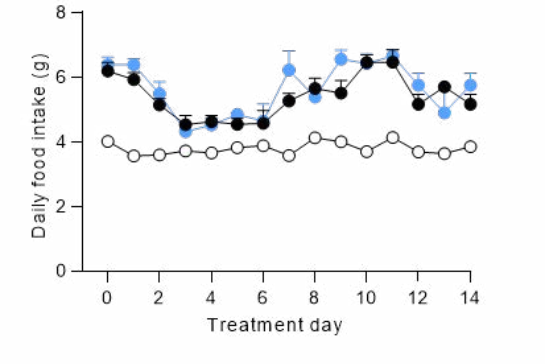


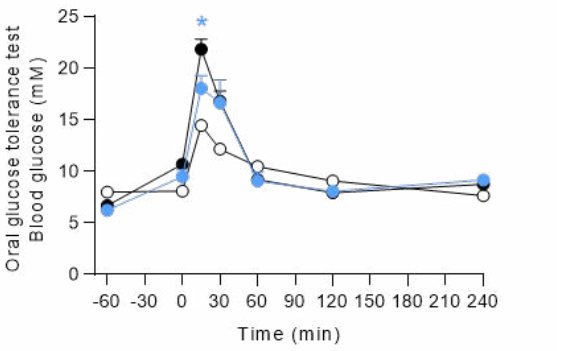
Body weight, food intake and glucose tolerance

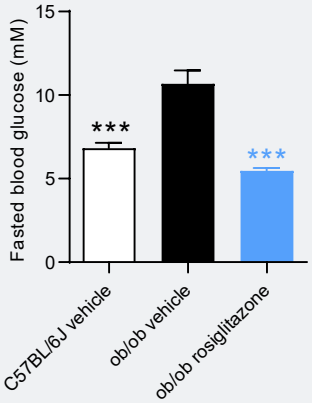
The ob/ob mouse demonstrates severe obesity and glucose intolerance. 2 weeks of rosiglitazone (PPAR-γ agonist) treatment is weight-neutral and improves glucose tolerance in ob/ob mice.

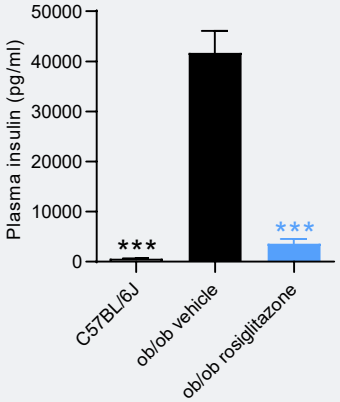












Blood biochemistry

ob/ob mice develop robust hyperglycemia, severe hyperinsulinemia and elevated plasma levels of triglycerides and cholesterol. 2 weeks of rosiglitazone treatment improves blood biochemistry in ob/ob mice.