Insulin resistant and dyslipidemic hamster

Key benefits
- Provides a noteworthy competitive advantage for your compound effects on both insulin resistance/diabetes, dyslipidemia and hepatic steatosis
- To select the best drug candidate in a very reproducible model with lipoprotein metabolism similar to humans
- To test the efficacy of novel drugs affecting both glucose and lipoprotein metabolism in a model validated with reference compounds

ANIMAL MODEL
- Background Strain: Golden Syrian Hamster
- Gender/Weight: male 90/110 g
- Diet: High fat diet + 10% fructose in drinking water
- Time on diet: 4 weeks
- Positive reference compounds: fenofibrate, rosiglitazone, sitagliptin

PHARMACOLOGICAL RELEVANCE
- A 4-week high fat diet induces hypercholesterolemia (140% increase), strong hypertriglyceridemia (300% increase), a 50% increase in CETP activity and a 20% decrease in HDL-c/total cholesterol ratio
- Fenofibrate lowers triglycerides and increases HDL-c/total cholesterol ratio (40% for both)
- Rosiglitazone lowers cholesterol (10%) and triglycerides (30%) plasma levels

Liver total cholesterol, triglycerides and fatty acids levels

- A 4-week high fat diet induces a strong hepatic steatosis with a 203, 131 and 264% increase in total cholesterol, triglycerides and fatty acids levels, respectively
- Rosiglitazone significantly decrease liver triglycerides by 20%. Fenofibrate decreases liver total cholesterol, triglycerides and fatty acids by 34, 32 and 42%, respectively

Blood glucose levels after an overnight fasting

- A 4-week high fat diet increases fasting blood glucose by 60% and plasma insulin by 180%. HOMA-IR dramatically increases by 360%.
- Both fenofibrate and rosiglitazone significantly decrease fasting blood glucose (10% and 20%), plasma insulin (69 and 52%) and HOMA-IR (72 and 61%)

REFERENCES