Physiogenex
THE METABOLIC DISEASES EXPERTS

Diet-Induced NASH (DIN™) mouse model associated with metabolic syndrome

- ✓ Unique diet-induced mouse model of non-alcoholic steatohepatitis (NASH)
- ✓ Associated with obesity and insulin resistance similar to human NASH physiopathological features

Key benefits

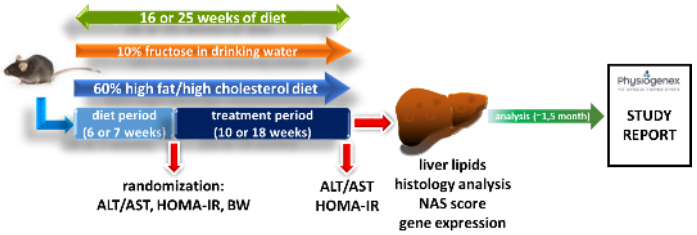
Unique proprietary diet-induced animal model that enables pharmacological studies targeting NASH and liver fibrosis: a human-like context including obesity and insulin resistance.

The diet–induced DIN™ NASH mouse model provides:

- A model pharmacologically validated with the FXR agonist Obeticholic acid to study NASH and liver fibrosis in the context of metabolic syndrome
- A model mimicking the human risk factors such as the fat-enriched diet which plays a major role in the development of NASH
- The model can be run for 16 weeks for liver steatosis/hepatocyte ballooning, or 25 weeks for advanced liver complications (inflammation/fibrosis)

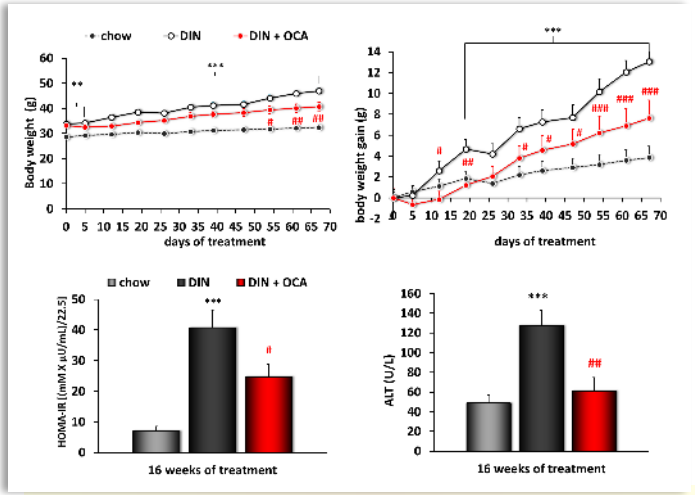
ANIMAL MODEL

- **Background strain/gender:** C57BL/6J mice, male
- **In house "Diet-Induced NASH" (DIN™):** high fat/high cholesterol + fructose in drinking water for 16 or 25 weeks
- **Reference compounds:** FXR agonist Obeticholic Acid (OCA) 25mg/kg/day in the diet (DIN+OCA)
- **Experimental design:**



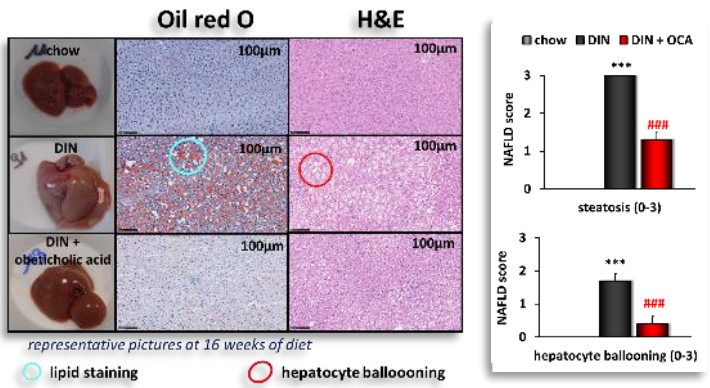
16 WEEKS OF DIET - MODEL CHARACTERISTICS

OCA REDUCES DIET-INDUCED OBESITY & INSULIN RESISTANCE



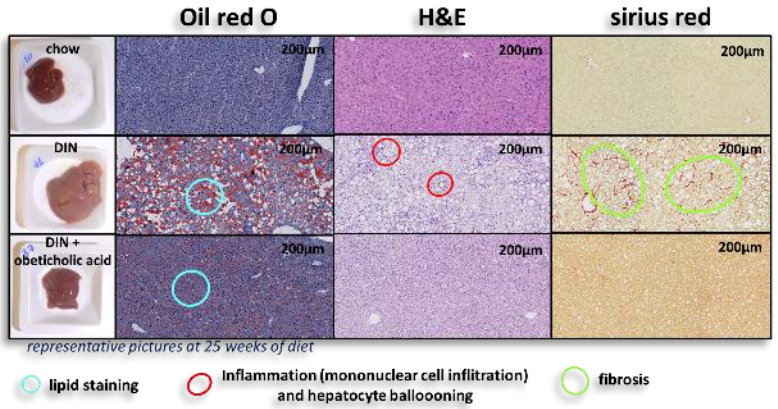
**p<0.01, ***p<0.001 vs. chow, #p<0.05 #p<0.01 ###p<0.001 vs. DIN

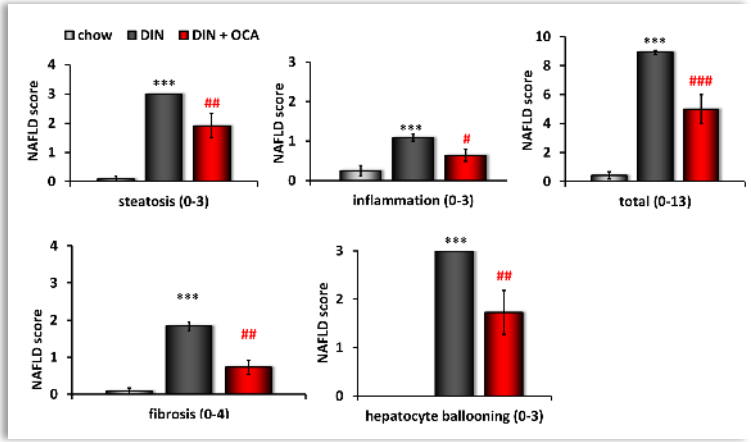
OCA REDUCES LIVER STEATOSIS AND BALLOONING SCORING



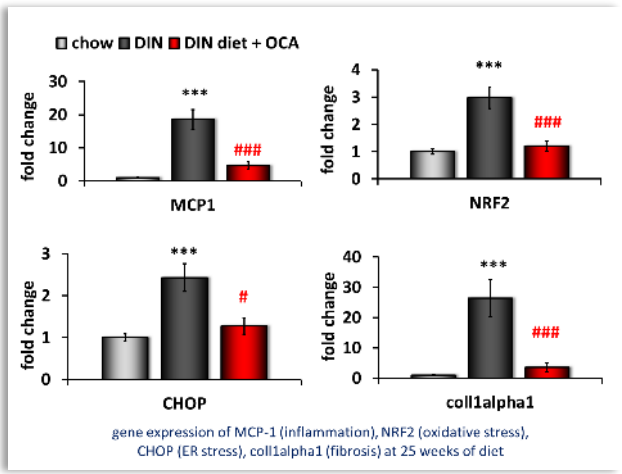
25 WEEKS OF DIET - MODEL CHARACTERISTICS

OCA SUBSTANTIALLY IMPROVES NAFLD SCORING





OCA REDUCES DIET-INDUCED HIGHER EXPRESSION OF GENE INVOLVED IN INFLAMMATION, OXIDATIVE/ER STRESS AND FIBROSIS



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