NAFLD/NASH

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MS-NASH: A more translatable polygenic model for drug development

Advance your NAFLD/NASH therapeutics with a rodent model that more closely mimics human NAFLD/NASH disease progression

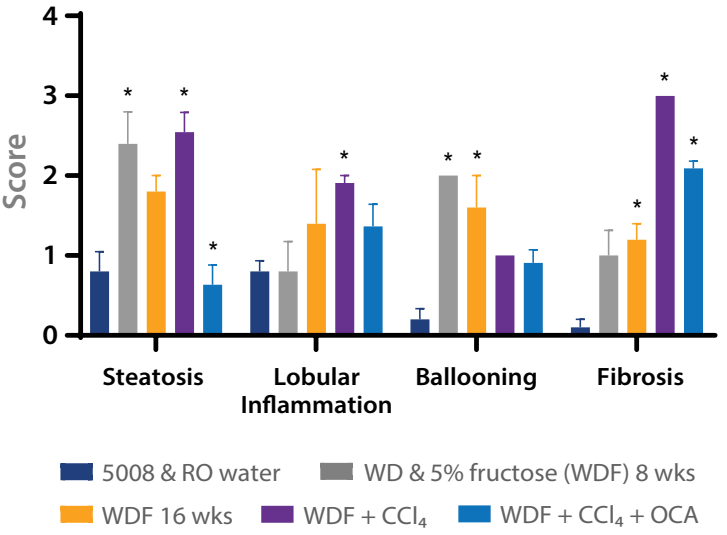
The MS-NASH mouse (formerly FATZO) features an intact leptin pathway and polygenic obesity. The model recapitulates human metabolic disease, providing an inherently dysmetabolic, obese, and diabetic model for the preclinical development of NAFLD/NASH therapies.

- Developed by crossing *C57BL6/J* with *AKR/J* mice and selectively bred for obesity, insulin resistance, and hyperglycemia.
- Develops liver steatosis leading to NASH through administration of a "Western diet" + fructose.
- Accelerated NASH progression and exacerbated pathology with CCl_4 administration.
  - Fibrosis observed in as little as 12 weeks.
- Metabolic stress on the liver indicated by elevated liver injury markers (ALT, AST) and liver triglycerides.
- Progressive worsening and histological changes from NAFLD observed over time.
- Improved NAS score and lipid profile with OCA treatment.

MS-NASH Mouse NAFLD/NASH Progression on Western Diet and Accelerated Western Diet + CCl_4

	Western Diet	Western Diet + CCl_4
Steatosis	4-8 weeks	4 weeks
Ballooning	16 weeks	4-8 weeks
Inflammation	16 weeks	8-16 weeks
NASH/Fibrosis	16-20 weeks	12-16 weeks

Liver NAS and Fibrosis Scoring in MS-NASH Mice
OCA reduces steatosis and fibrosis in MS-NASH mice



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DiscoverCrown
Trial translational rodent and NHP models for obesity, diabetes, renal disease, and NAFLD/NASH.
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