ENVIGO

Genetically engineered models (GEMS)

Ldlr knockout rat

Model	Ldlr knockout rat
Strain	HsdSage:SD-*Ldlr*^{tm1sage}
Location	U.S.
Availability	Cryopreserved

Characteristics/husbandry

+ This model possesses a 337 bp deletion and 4 bp insertion within Exon 4 on chromosome 8
+ Homozygous knockout rats display loss of LDLR protein via Western blot
+ Homozygous knockout rats have increased body weight as compared to wild type
+ Homozygous knockout rats show significantly elevated serum cholesterol levels
+ Background strain: Sprague-Dawley

Zygosity genotype

+ Homozygous

Research use

+ Atherogenesis
+ Atherosclerosis
+ Hypertension
+ Insulin resistance
+ Lipoprotein/Cholesterol transportation
+ Metabolism (Triglyceride/Cholesterol)
+ Obesity; Type II diabetes

Origin

The Ldlr KO rat model was originally created at SAGE Labs, Inc. in St. Louis, MO and distributed out of the Boyertown, PA facility. The line continues to be maintained through the original SAGE Labs animal inventory acquired by Envigo.

Description

The Low-Density Lipoprotein (LDL) receptor is directly involved in the development of atherosclerosis, due to accumulation of LDL cholesterol in the blood. Ldlr (-/-) rats show elevated serum cholesterol levels.

Ldlr mediates the endocytosis of cholesterol-rich low-density lipoproteins (LDL) and is important for the proper regulation of LDL circulation within the blood. Expressed in the liver, loss of function of Ldlr leads to accumulation of excess LDL, which directly contributes to atherosclerosis, making this a useful model for the study of cardiovascular disease. This model is currently cryopreserved.

Figure 1: Homozygous Ldlr knockout rats demonstrate significantly higher cholesterol levels at 5-weeks of age 5-week-old Ldlr knockout rats display increased serum cholesterol levels both pre- (A) and post-fasting (B) as compared to wild type animals.

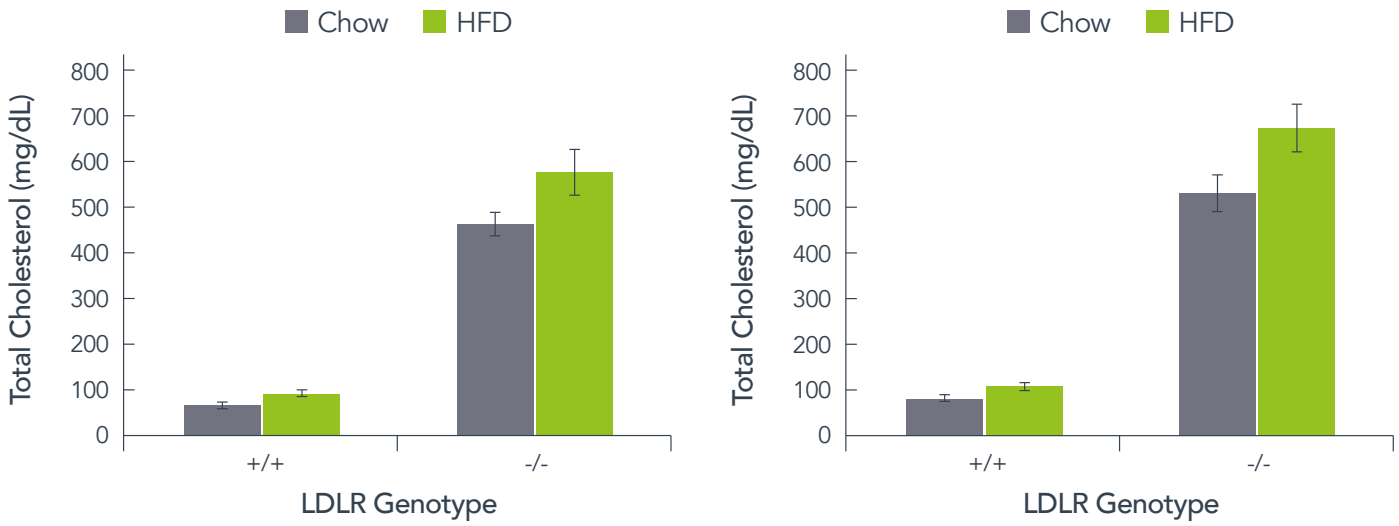
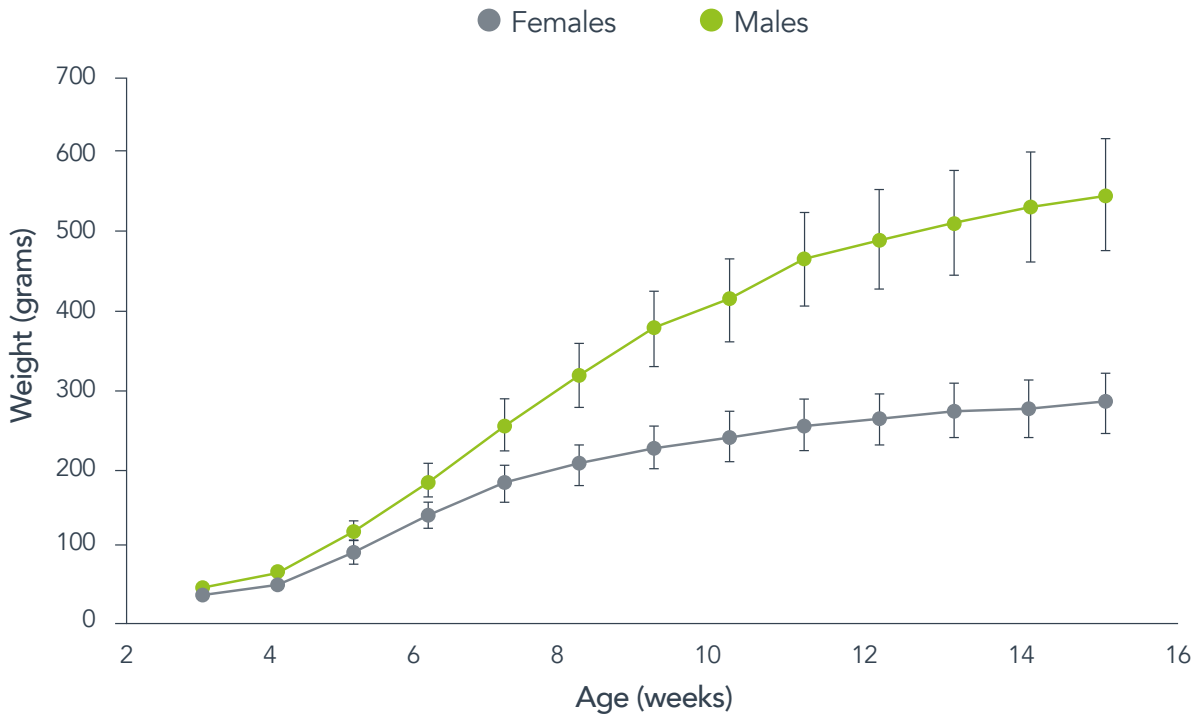


Figure 2: A graph showing the correlation between the age and weight of Ldlr knockout rats.



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