Resource Summary Report

Generated by NIF on Sep 9, 2026

Lepr^{db}/Lepr^{db}

RRID:MGI:3822315
Type: Organism

Proper Citation

RRID:MGI:3822315

Organism Information

URL:

Proper Citation: RRID:MGI:3822315

Description: Allele Detail: Spontaneous This is a legacy resource.

Species: Mus musculus

Notes: Allele Detail: Spontaneous This is a legacy resource.

Phenotype: increased circulating glucose level, obese, increased circulating insulin level, impaired adaptive thermogenesis, female infertility, insulin resistance, impaired glucose tolerance, abnormal eating behavior, increased percent body fat/body weight, insulin resistance, impaired glucose tolerance, hypoactivity, hyperglycemia, female infertility, decreased body length, abnormal female reproductive system morphology, abnormal oxygen consumption, short femur, absent estrous cycle, increased body weight, absent estrus, decreased body length, decreased lean body mass, increased circulating leptin level, polyphagia, increased percent body fat/body weight

Affected Gene: Lepr

Genomic Alteration: db

Catalog Number: 3822315

Background: involves: C57BL/6 * C57BLKS/J

Database: MGI, Mouse Genome Informatics MGI

Database Abbreviation: MGI

Availability: Availability unknown check source stock center

Source References: PMID:20068132, PMID:19015522

Organism Name: $Lepr^{db}/Lepr^{db}$

Record Creation Time: 20240120T190402+0000

Record Last Update: 20240130T201858+0000

Ratings and Alerts

No rating or validation information has been found for $Lepr^{db}/Lepr^{db}$.

No alerts have been found for $Lepr^{db}/Lepr^{db}$.

Data and Source Information

Source: Integrated Animals

Source Database: MGI, Mouse Genome Informatics MGI

Usage and Citation Metrics

We found 1 mentions in open access literature.

Listed below are recent publications. The full list is available at NIF .

King BC, et al. (2019) Complement Component C3 Is Highly Expressed in Human Pancreatic Islets and Prevents ?? Cell Death via ATG16L1 Interaction and Autophagy Regulation. Cell metabolism, 29(1), 202.