

# Resource Summary Report

Generated by [PRECISE-TBI](#) on Jul 31, 2026

[Lepr<sup>db</sup>/Lepr<sup>db</sup>](#)

RRID:MGI:3822315

Type: Organism

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## Proper Citation

RRID:MGI:3822315

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## 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<sup>db</sup>/Lepr<sup>db</sup>

**Record Creation Time:** 20240120T190402+0000

**Record Last Update:** 20240130T201858+0000

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## Ratings and Alerts

No rating or validation information has been found for Lepr<sup>db</sup>/Lepr<sup>db</sup>.

No alerts have been found for Lepr<sup>db</sup>/Lepr<sup>db</sup>.

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## Data and Source Information

**Source:** [Integrated Animals](#)

**Source Database:** MGI, Mouse Genome Informatics MGI

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## Usage and Citation Metrics

We found 1 mentions in open access literature.

**Listed below are recent publications.** The full list is available at [PRECISE-TBI](#) .

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.