Also Known As: B6 ob
Mice homozygous for the obese spontaneous mutation, \( Lep^{ob} \) (commonly referred to as ob or ob/ob), exhibit obesity, hyperphagia, transient hyperglycemia, glucose intolerance, and elevated plasma insulin. They are also hypometabolic, hypothermic, and subfertile. Wound healing is impaired and hormone production from both pituitary and adrenal glands is increased. This strain is used to model phases I and II of diabetes type II and obesity. Obesity is characterized by an increase in the number and size of adipocytes. Although hyperphagia contributes to the obesity, homozygotes gain excess weight and deposit excess fat even when restricted to a diet sufficient for normal weight maintenance in lean mice.
Mice homozygous for the obese spontaneous mutation, $(Lep^{ob})$, are first recognizable at about four weeks of age. Homozygous mutant mice gain weight rapidly and may reach three times the normal weight of wild-type controls. In addition to obesity, mutant mice exhibit hyperphagia, a diabetes-like syndrome of hyperglycemia, glucose intolerance, elevated plasma insulin, subfertility, impaired wound healing, and an increase in hormone production from both pituitary and adrenal glands. They are also hypometabolic and hypothermic. The obesity is characterized by an increase in both adipocyte number and size. Adipose tissue transplants in $Lep^{ob}$ homozygotes protect them from obesity, normalize insulin sensitivity, and restore fertility. Although hyperphagia contributes to the obesity, homozygotes gain excess weight and deposit excess fat even when restricted to a diet sufficient for normal weight maintenance in lean mice. Hyperinsulinemia does not develop until after the increase in body weight, and probably results from it. Homozygotes have an abnormally low threshold for stimulation of pancreatic islet insulin secretion even in very young pre-obese animals. Female homozygotes exhibit decreased uterine and ovarian weights, decreased ovarian hormone production and hypercytolipidemia in follicular granulosa and endometrial epithelial tissue layers (Garris et al., 2004).

As is the case with mice carrying the diabetes mutation $(Lepr^{db})$, manifestation of the diabetic syndrome is strikingly dependent on genetic background. Hyperglycemia is only transient, (subsiding around 14 to 16 weeks) on the C57BL/6J background. On the C57BLKS background, obese homozygotes (Stock No. 000696) become severely diabetic with regression of islets and early death. Injection of recombinant leptin into obese homozygotes sharply reduces body weight,
decreases food intake, increases energy expenditure, and restores fertility in male mice.

Leptin can regulate bone mass through a central, neuroendocrine signaling pathway. Similar to the effects of aging in humans, homozygotes exhibit muscle hypoplasia (quadriceps), increased marrow adipogenesis and decreased bone mass in the hindlimbs (Hamrick et al., 2004).

Genotyping Protocols
Restriction Enzyme Digest: Lep
Pyrosequencing: Lep
End Point Analysis: Lep
Genotyping resources and troubleshooting
Dietary Information
LabDiet® 5K20 formulation (10% fat)

Breeding Considerations

This strain is a good breeder.

When maintaining a live colony, these mice are bred as heterozygotes. Females homozygotes are infertile, and males exhibit a reduced ability to mate.

Wean-aged mice may show signs of barbering that include whisker picking. Affected mice typically regrow whiskers within two weeks after weaning.

Additional Breeding and Husbandry Support

Mating System
Ovarian Transplant (Homozygous ob/ob) x Heterozygote

Appearance
black, fat
Related Genotype: a/a Lep\textsuperscript{ob} /Lep\textsuperscript{ob}

black, lean
Related Genotype: a/a Lep\textsuperscript{ob} /+ or a/a ?/+

Citation
When using the B6 ob mouse strain in a publication, please cite the originating article(s) and include JAX stock #000632 in your Materials and Methods section.

Animal Health Reports

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Domestic

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