This strain is used to model phases I to III of diabetes type II and obesity. Mice homozygous for the diabetes spontaneous mutation (Lepr<sup>ob</sup>) manifest morbid obesity, chronic hyperglycemia, pancreatic beta cell atrophy and become hypoinsulinemic. Obesity starts at 3 to 4 weeks of age. Elevated plasma insulin begins at 10 to 14 days and elevated blood sugar at 4 to 8 weeks. Homozygous mice are polyphagic, polydipsic, and polyuric. The severity of disease on BKS background leads to uncontrolled rise in blood sugar, severe depletion of insulin-producing beta-cells of the pancreatic islets, peripheral neuropathy, myocardial disease and death by 10 months of age. Exogenous insulin fails to control blood glucose levels and gluconeogenic enzyme activity increases. Wound healing is delayed, and metabolic efficiency is increased.

**Genetic overview**

000662 C57BLKS/J  F131
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