BKS-DB

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One-Stop Service of Mouse Model

Stain Name: C57BLKS/JGpt - *Lepr*^{em2Cd}/Gpt
Strain Type: KO
Strain ID: T002407
Background: C57BLKS/JGpt

Description

The Obese Gene Receptor (*ob-R*) also known as LEPR gene is encoding a Type I cytokine receptor, a protein that in humans have a large relationship with obesity, hypertension, diabetes, lipid metabolism disorders, etc. [1, 2]. The *db/db* mouse is a model of obesit y, diabetes, and dyslipidemia wherein leptin receptor activity is deficient because leptin mutation are performed by CRISPR/ Cas9 technology. The disease course is greatly affected by the genetic background, *Lepr* defects in the BKS background are often accompanied by higher levels of blood sugar and body weight.

Higher levels of blood glucose and gluconeogenesis enzyme activity cannot be controlled by insulin treatment on BKS-*db* mouse, in which accompanied by phenotypes of peripheral neuropathy, myocardial disease, delayed wound healing, accelerated metabolic efficiency [3], hypothalamic lesions[4]. In addition, female *db/db* mice were observed infertility together with reduced uterine and ovarian weight and decreased estrogen secretion [5-6].

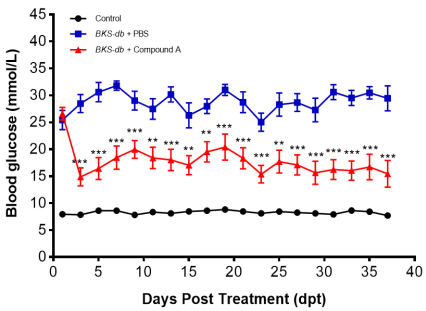
The *Lepr* gene mutant mouse (BKS-*db*) was constructed by GemPharmatech Co., Ltd using gene editing technology. By monitoring the blood glucose levels for this strain, it was found that BKS-*db* mouse have significantly higher level of blood glucose than that of the wild control. This strain is an ideal model for type II diabetes research.

Application

1. Metabolic research (diabetes and obesity). 2. Endocrine Disorder research. 3. Reproductive Biology research.

Data support

■ Blood glucose curve in *BKS-db* mice treating with drug intervention



The blood glucose levels of BKS-*db* was significantly reduced after the drug intervention.Control group (n=14), BKS-*db* + PBS group (n=12), BKS-*db* + Compound A group (n=12). The data of tumor volume and mouse body weight were expressed as mean ± standard error (Mean ± SEM). For comparison among three or more groups, one-way ANOVA with Dunnett post-hoc test method were performed. P<0.05 was considered to be statistically significant. **, P < 0.01; ***, P < 0.001.

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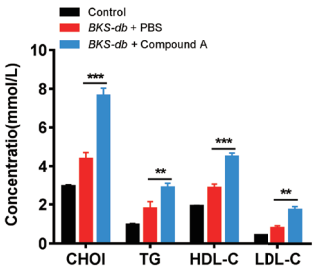
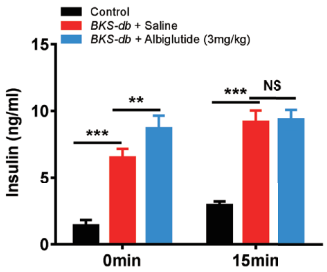
Sera blood lipids levels in BKS-db mice treating with drug intervention



The Sera blood lipids levels in BKS-*db* mice were significantly increased after the drug intervention. Control group (n=14), BKS-*db* + PBS group (n=12), BKS-db + Compound A group (n=12). The blood lipids levels were expressed as mean ± standard error (Mean ± SEM). For comparison among three or more groups, one-way ANOVA with Dunnett post-hoc test method were performed. P<0.05 was considered to be statistically significant. **, P < 0.01; ***, P < 0.001.

Sera insulin levels in BKS-*db* mice treating with drug intervention



Albiglutide intervention can significantly increase the sera insulin levels in BKS-*db* mice under background conditions on day 28. But after the oral glucose tolerance test (OGTT), there was no statistically significant difference in sera insulin levels between controls and treatment group. Control group (n=8), BKS-*db* + Saline group (n=11), BKS-*db* + Albiglutide (3mg/kg) group (n=11). The levels of insulin were expressed as mean ± standard error (Mean ± SEM). For comparison among three or more groups, one-way ANOVA with Dunnett post-hoc test method were performed. P<0.05 was considered to be statistically significant. **, P < 0.01; ***, P < 0.001.

References

1.Chen, Hong, et al. "Evidence that the diabetes gene encodes the leptin receptor: identification of a mutation in the leptin receptor gene in db/db mice." *Cell* 84.3 (1996): 491-495.

2.Yoon, Ji-Won, et al. "Genetic control of organ-reactive autoantibody production in mice by obesity (ob) diabetes (db) genes." *Diabetes* 37.9 (1988): 1287-1293.

3.Kämpfer, Heiko, et al. "Lack of interferon-gamma production despite the presence of interleukin-18 during cutaneous wound healing." *Molecular Medicine* 6.12 (2000): 1016.

4.Garris, Bryan L., et al. "Hypophyseal lipoapoptosis: diabetes (db/db) mutation-associated cytolipidemia promotes pituitary cellular disruption and dysfunction." *Pituitary* 7.1 (2004): 5-14.

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6.Garris, David R., and Bryan L. Garris. "Genomic modulation of diabetes (db/db) and obese (ob/ob) mutation-induced hypercytolipidemia: cytochemical basis of female reproductive tract involution." *Cell and tissue research* 316.2 (2004): 233-241.

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