

SWR.129X1(B6)-Apob^{tm1.1Zc}/JStock No: **007679** Congenic, Targeted Mutation

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advanced non-alcoholic fatty liver diseases (NAFLD), and atherosclerosis. In addition, all three congenic apoB38.9 strains (Stock No. 007679, Stock No. 007682, and Stock No. 007683) may be useful in conjunction with other apoB mutant mice, including Stock No. 002053 (apoB70), Stock No. 002876 (apoB48-only), and Stock No. 002877 (apoB100-only).

Donating Investigator

Gustav Schonfeld, Washington University School of Medicine

[R E A D M O R E +](#)**GENETIC OVERVIEW****Genetic Background****Generation***Apob^{tm1.1Zc}***Allele Type**

Targeted (Humanized sequence)

Gene Symbol*Apob***Gene Name**

apolipoprotein B

[V I E W G E N E T I C S](#)**RESEARCH APPLICATIONS**

Research Tools

Metabolism Research

Mouse/Human Gene Homologs

Reproductive Biology Research

Cardiovascular Research

Diabetes and Obesity Research

Internal/Organ Research

BASE PRICE

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Details

Detailed Description

Mice homozygous for this apoB38.9 allele (apoB^{38.9/38.9}) are viable with impaired fertility, bearing a premature stop codon at residue 1767 of the targeted gene. As a result, homozygous plasma shows a truncated apoB38.9 as the sole apoB protein. Plasma from heterozygous (apoB^{+38.9}) mice have reduced apoB100 and apoB48 compared to wildtype, with apoB38.9 representing 20% of total circulating apoB. This apoB38.9 truncation affects both apoB100 and apoB48 metabolism in mice, and mimics human Familial Hypobetalipoproteinemia (FHBL). Homozygous and, to a lesser extent, heterozygous mice exhibit symptoms of FHBL due to impaired lipoprotein export system/VLDL secretion, including elevated hepatic triglyceride (TG), cholesterol and free fatty acids (FFA), with decreased plasma TG and cholesterol. Because plasma and liver lipid profiles range from mild to severe in populations of heterozygous apoB38.9 mice on a mixed (C57BL/6J;129X1/SvJ) genetic background, apoB^{+38.9} congenic mice were generated on three different inbred strains with characterized differences in liver fat: SWR/J (low liver TG strain, ~40mg/dL), C57BL/6J (medium liver TG, ~140mg/dL), and BALB/cByJ (high liver TG strain, ~200 mg/dL). All three apoB^{+38.9} congenic strains exhibit significantly impaired hepatic TG secretion compared to their respective genetic backgrounds, with significant interactions observed between genetic background and apoB genotype for liver TG and FFA. BALB/cByJ-apoB38.9 mice (Stock No. [007683](#)) exhibit the greatest hepatic TG and FFA increase (probably due to elevated hepatic TG synthesis rate). Despite having the lowest degree of hepatic steatosis among the three apoB^{+38.9} congenic strains, SWR/J-apoB38.9 mice (Stock No. [007679](#)) show insulin resistance, with BALB/cByJ-apoB38.9 mice exhibiting intermediate and C57BL/6J-apoB38.9 mice (Stock No. [007682](#)) exhibiting the greatest insulin resistance. Additional comparisons between the three congenic strains reveal that C57BL/6J-apoB38.9 mice have significant increase of fatty acid synthesis rate compared to the other two congenic strains, while SWR/J-apoB38.9 mice show dynamic feedback regulation of fatty acids and TG synthesis and beta-oxidation in response to excessive hepatic TG accumulation. Gender dimorphism is observed; while BALB/cByJ-apoB38.9 males have reduced plasma cholesterol levels compared to wildtype males, females from all three apoB^{+38.9} congenic strains have reduced plasma cholesterol compared to wildtype females. Male BALB/cByJ-apoB38.9 and C57BL/6J-apoB38.9 mice have significant reduction of liver TG synthesis compared to wildtype males, while SWR/J-apoB38.9 females show the same significant reduction. These apoB38.9 mutant mice may be useful to study the genetic and molecular mechanism of apoB defects and lipid metabolism/liver fat accumulation, the relationship between hepatic steatosis and insulin resistance, the progression of advanced non-alcoholic fatty liver diseases (NAFLD), and atherosclerosis. These apoB38.9 mutant mice may also be useful in conjunction with other apoB mutant mice, including Stock No. [002053](#) (apoB70), Stock No. [002876](#) (apoB48-only), and Stock No. [002877](#) (apoB100-only).

Development

Control Suggestions

Selected References

– Genetics

+ *ApoB*^{tm1.1Zc}

– Disease/Phenotype

+ Disease Terms

+ Research Areas By Phenotype

+ Mammalian Phenotype Terms by Genotype

+ References

– Technical Support

C O N T A C T T E C H N I C A L S U P P O R T

Genotyping Protocols

[Genotyping resources and troubleshooting](#)

Breeding Considerations

When maintaining a live colony, heterozygotes are bred to wildtype siblings or to SWR/J inbred mice (see Stock No. [000689](#)). The donating investigator reports that homozygous mice are produced in reduced rates and are probably infertile.

[Additional Breeding and Husbandry Support](#)

Citation

When using the SWR.129X1(B6)-*ApoB*^{tm1.1Zc}/J mouse strain in a publication, please [cite the originating article\(s\)](#) and include JAX stock #007679 in your Materials and Methods section.

Animal Health Reports

[Facility Barrier Level Descriptions](#)

Production of mice from cryopreserved embryos or sperm occurs in a maximum barrier room, [G200](#)

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