NZO/HILtJ
Stock No: 002105 | New Zealand Obese
Inbred Strain

Also Known As: New Zealand Obese
NZO inbred mice and strains derived from them develop severe obesity, and are thus useful for studying obesity and Type 2 diabetes.

GENETIC OVERVIEW
Genetic Background | Generation
?+F73
(2021-03-31 00:00:00)
NZO mice of both sexes exhibit high birth weights and are significantly heavier at weaning age. Severe obesity (including both visceral and subcutaneous fat depots) develops even when mice are maintained on a standard diet containing 4.5% fat. Both males and females of the NZO/Hl substrain exhibit impaired glucose tolerance (IGT), but subsequent type 2 maturity onset (NIDDM) diabetes development is limited to males, with a phenotype penetrance of 50% or less. NZO/Hl mice also show anti-insulin receptor antibodies, a defect in leptin transport, and hypertension. The genetic lesion appears to be within the islets of Langerhans as transfer of pancreatic islets from normal mice returns body weights and blood glucose levels to within normal range. Ovarian granulosa cell tumors, lymphomas, duodenal, and lung tumors have also been noted to occur in NZO mice at an elevated frequency. F1 hybrids of NON/ShiLt and NZO/Hl provide a new model of obesity-induced diabetes. Male (NON/ShiLt x NZO/Hl)F1 hybrids are obese (BW = 53.5 g by 16 weeks) and almost all develop maturity onset NIDDM. F1 males on a 4% diet will develop hyperglycemia around 20 to 24 weeks of age; increasing the fat content of the diet accelerates diabetes onset to 16 to 20 weeks of age. (NZO/Hl x NON/ShiLt)F1 hybrids will develop diabetes slightly faster than their reciprocal cross due to the NZO maternal environment; however this cross is difficult to produce due to the inherently poor breeding performance of NZO/Hl females. F1 females exhibit a weight gain similar to the NZO parent, and have impaired glucose tolerance but are resistant to diabetes development. Diabetes development can be accelerated to eight to 12 weeks by fostering onto an F1 dam. Reciprocal backcrosses to the parental strains and analysis of (NON/ShiLt x NZO/Hl)F2 mice has led to the identification of a number of complex diabetes-predisposing (“diabesity”) QTLs. Dr. Leiter's research group at The Jackson Laboratory is currently developing a series of nine recombinant congenic strains (RCS) made by backcrossing the (NZO/Hl x NON/ShiLt)F1 for two generations onto the NON/ShiLt background before inbreeding (~12% NZO/Hl, 88% NON/ShiLt genomes). Preliminary analysis indicates that body weight gains of all RCS are higher than NON/ShiLt, but none are as obese as NZO/Hl; some of these RCS develop NIDDM while others are resistant. These new strains will be useful to further analyze diabesity QTLs and as new models for type 2 (NIDDM) diabetes. An additional benefit of the RCS is better breeding performance than NZO/Hl.
Genetics

- *Pctp*<sup>R120H</sup>
- *Cox7a2<sup>i</sup>*

Disease/Phenotype

- Disease Terms
- Research Areas By Phenotype
- Mammalian Phenotype Terms by Genotype
- Phenotype Information
- References

Technical Support

Genotyping Protocols
Genotyping resources and troubleshooting
Dietary Information
LabDiet® 5K54 formulation (4% fat)

Breeding Considerations

This inbred strain is a challenging breeder (can have a high rate of non-productive matings).

Additional Breeding and Husbandry Support
Mating System
Sibling x Sibling

Appearance
agouti
Related Genotype: A/A

Citation
When using the New Zealand Obese mouse strain in a publication, please include JAX stock #002105 in your Materials and Methods section.
Animal Health Reports
Facility Barrier Level Descriptions

AX12 (Maximum)

Pricing & Availability

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Domestic
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LIVE MOUSE

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RELATED PRODUCTS AND SERVICES

Mouse ES Cells
NZO/HiltJ-AC652/GrsJ cells
$995.00

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LICENSING INFORMATION

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Email: TechTran@jax.org

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All

By Allele

By Gene

By Collection