The Jackson Laboratory

B6;129S2-*Syngap1*^{tm2Geno}/RumbJ

Stock No: **029304** | SYNGAP1$^{lx\text{-}st}$ (Cre-conditional rescue)

Targeted Mutation

Typically mice are recovered in 10-14 weeks. Contact Customer Service to place an order or for more information.

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truncated/inactive SynGAP protein. These mice allow Cre recombinase-inducible restoration of full-length SynGAP expression. SYNGAP1$^{lx\text{-}st}$ mice may be useful Cre-lox studies of synapse development (specifically in dendritic spine), cognitive and behavioral maturation, intellectual disability and autism spectrum disorder.

Donating Investigator

Gavin Rumbaugh, The Scripps Research Institute

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GENETIC OVERVIEW

Genetic Background **Generation**

Syngap1^{tm2Geno}

Allele Type	Gene Symbol	Gene Name
Targeted (Inserted expressed sequence, Humanized sequence)	*Syngap1*	synaptic Ras GTPase activating protein 1 homolog (rat)

VIEW GENETICS

RESEARCH APPLICATIONS

Neurobiology Research
Developmental Biology Research
Research Tools

VIEW ALL RESEARCH APPLICATIO

BASE PRICE

Starting at:

$2,854.50 Domestic price Cryo Recovery

VIEW PRICE LIST

Details

Detailed Description

Mutations that cause intellectual disability (ID) and autism spectrum disorder (ASD) are commonly found in genes that encode for synaptic proteins. *Syngap1* encodes a synaptic RasGAP (SynGAP) that is largely localized to dendritic spines in neocortical pyramidal neurons, where it suppresses signaling pathways linked to NMDA receptor (NMDAR)-mediated synaptic plasticity and AMPA receptor (AMPAR) membrane insertion. *Syngap1* has alternative transcriptional start sites and several alternatively spliced C-terminal exons that result in many possible SynGAP isoforms. Human SYNGAP1 haploinsufficiency results from a truncation of the full-length protein. Exon 7 contains the first common methionine present in the shortest splice variant.

The SYNGAP1$^{lx\text{-}st}$ allele has a *loxP*-flanked neo-STOP cassette upstream of exon 6 of the *Syngap1* gene. Following *cre*-mediated recombination that removes the floxed region, SynGAP expression and function are fully recovered.

Prior to Cre recombinase exposure, mice heterozygous for the SYNGAP1$^{lx\text{-}st}$ allele express a SynGAP protein truncated after exon 5 (known to be an inactivate SynGAP protein), as well as diminished levels of functional SynGAP (from the wildtype allele). Therefore, the phenotype of heterozygotes (SYNGAP1$^{lx\text{-}st/+}$) is the same as mice heterozygous for the global knockout allele, and both are a model of human SYNGAP1 haploinsufficiency - exhibiting normal synaptic transmission, modest defects in synaptic plasticity, enhanced synaptic function (accelerated rate of glutamatergic synapse maturation) during early neural development and profound cognitive and behavioral abnormalities. Specifically, heterozygous SynGAP-deficiency significantly disrupts excitatory/inhibitory (E/I) balance in the neural networks that support cognition and behavior. Abnormal behavioral phenotypes can be observing as early as 21 days of age with ~100% penetrance (although physiological abnormalities can be measured even earlier through other methods such as electrophysiological recordings). Furthermore, homozygotes (SYNGAP1$^{lx\text{-}st/lx\text{-}st}$ are phenotypically the same as global knockout homozygotes - exhibiting complete postnatal lethality between 2-5 days of age.

When SYNGAP1$^{lx\text{-}st}$ are bred to mice that express Cre recombinase, the resulting offspring may be useful in generating tissue-specific restoration of full-length SynGAP expression. For example, when SYNGAP1$^{lx\text{-}st/+}$ mice are bred hemizygous for the CAGGCre-ER^{TM} transgene (Stock No. 004682), the resulting animals allow widespread, tamoxifen-inducible SynGAP restoration. In those mice, complete restoration of SynGAP protein expression in adults had no detectable benefit on behavior or cognition - demonstrating that SYNGAP1 haploinsufficiency is a disorder of brain development.

Development

Expression Data

Control Suggestions

Selected References

Genetics

Syngap1^{tm2Geno}

Disease/Phenotype

Disease Terms

Research Areas By Phenotype

Mammalian Phenotype Terms by Genotype

References

Technical Support

CONTACT TECHNICAL SUPPORT

Genotyping Protocols

Separated PCR:Syngap1-Alternate 2
Standard PCR:Generic Neo
Probe:Generic Neo
Genotyping resources and troubleshooting

Breeding Considerations

Homozygous mice exhibit complete postnatal lethality between 2-5 days of age. Heterozygous mice develop profound cognitive and behavioral abnormalities grossly demonstrable ~21 days of age. The donating investigator breeds heterozygous mice to wildtype mice from the colony, using both sexes as the heterozygote.

When maintaining a live colony, at The Jackson Laboratory Repository, heterozygous mice may be bred to wildtype mice from the colony or to C57BL/6J inbred mice (Stock No. 000664).

Additional Breeding and Husbandry Support

Mating System

Wild-type x Heterozygote
Heterozygote x Wild-type

Citation

When using the SYNGAP1$^{lx\text{-}st}$ (Cre-conditional rescue) mouse strain in a publication, please cite the originating article(s) and include JAX stock #029304 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

Pricing & Availability

Cryo Recovery

Typically mice are recovered in 10-14 weeks. Contact Customer Service to place an order or for more information.

Domestic **International**

Pricing effective for USA, Canada and Mexico shipping destinations

CRYORECOVERY - DOMESTIC PRICING

SERVICE/PRODUCT	DESCRIPTION	PRICE
Cryo Recovery	Heterozygous or wildtype for Syngap1<tm2Geno>	$2,854.50

RELATED PRODUCTS AND SERVICES

Frozen Mouse Embryo	B6;129S2-Syngap1<tm2Geno>/RumbJ Frozen Embryo	$2595.00

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