

129-Add1^{tm1Llp}/Llp

Stock No: **008672**

 **Coisogenic, Targeted Mutation**

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in compensated hemolytic anemia. On a C57BL/6J congenic background, but not a 129 background, homozygotes also have a high incidence of nonobstructive hydrocephaly, and on a 129 background, but not a C57BL/6J background, homozygotes have a high incidence of megaesophagus. An increased diameter of actin rings has been found in the neurons of homozygous null mutants and progressive axon enlargement with subsequent degeneration has been found in optic nerve, sciatic nerve, and corticospinal tract on a segregating B6;129 background.

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GENETIC OVERVIEW

Genetic Background

Generation

Add1^{tm1Llp}

Alele Type

Gene Symbol

Gene Name

Targeted (Null/Knockout)

Add1

adducin 1 (alpha)

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RESEARCH APPLICATIONS

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BASE PRICE

Starting at:

Details

Detailed Description

Adducin heterodimers form tetramers that bind actin filaments, recruit spectrin, and help cap fast-growing ends of actin filaments, thereby regulating the membrane-cytoskeletal structure. This null allele of adducin 1 results in compensated hemolytic anemia in homozygotes. The mean corpuscular hemoglobin and mean corpuscular volume are decreased, the percent circulating reticulocytes is increased, and red blood cells are osmotically fragile and spherocytic, with decreased membrane surface area. Adducin 1 is essential for adducin tetramer formation. Red blood cells and nervous system tissue from these null homozygotes show concomitant loss of adducin 2 and adducin 3, despite normal transcription of these genes in fetal liver, and platelets also lack adducin 3, which is normally the only other adducin expressed in platelets. Platelet counts are increased in males and the mean platelet volume decreased in females, but abnormalities are not found in the discoid shape of the platelets, aggregation, or bleed times. Lethal communicating hydrocephalus with "striking dilation of the lateral, third, and fourth ventricles" was first characterized in these homozygotes in the initial report by Robledo et al, 2008, who subsequently found a significant impact of genetic background. On a C57BL/6J congenic background 57 of 71 homozygotes developed hydrocephalus, and on a segregating B6;129 background 50 out of 98 homozygotes developed hydrocephalus, but hydrocephalus was only found in 1 of 29 homozygotes on a 129 background. Conversely, megaesophagus was observed in 13 of 29 homozygotes and kyphosis was found in all 29 homozygotes on a 129 background, but neither phenotype was found in homozygotes on a C57BL/6J congenic or B6;129 segregating background. Megastomach was found in one of the 29 homozygotes assessed on the 129 background. Viability is lower on the 129 background, with only 10% of pups at 10-14 days of age being homozygous. Those that survive are smaller in body size and weight, have blunter snouts, hyperkyphosis, splaying of the legs when lifted by the tail, a stiff or waddle-like gait, and decreased activity, spending increased time in the nest relative to littermate controls. Further assessment on the 129 background found vertebral fusions in dorsal portions of cervicothoracic vertebrae in some but not all homozygotes with hyperkyphosis. No muscular abnormality was found that could be causative. Instead a decrease in axon number was found in the motor branch and sensory branch of the femoral nerve.

Leite et al. subsequently reported that axon specification, neuron polarity, axon and dendrite length are all unaffected in homozygotes on a segregating B6;129 background, but actin rings have an increased diameter with age in axons of dorsal root ganglia neurons and retinal ganglion cells, although the periodicity of actin rings remains normal. They also found that progressive axon enlargement with subsequent degeneration occurs in the central and peripheral nervous systems. As homozygotes age, the number of axons with a larger than normal diameter was found to have increased in optic nerve, spinal cord, and sciatic nerve. Compared with 20 days of age there are more axons larger than 2 micrometers at 60 days of age and many more at 100 days of age, but loss of myelin was not found. Decreased density of myelinated axons of sciatic nerves was found at 20 days of age. The optic nerve appears normal at 20 days of age, but by 100 days of age there is an approximately 40% reduction in optic nerve axon density and the corticospinal tract also shows a 25% decrease in axon density at this age. At this timepoint the optic nerve and corticospinal tract shows severe nerve atrophy with an approximately 33% decreased optic nerve area.

Development

Selected References

Genetics

[+](#) *Add1^{tm1Llp}*

[-](#) 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](#)

Citation

When using the 129-*Add1^{tm1Llp}*/Lp mouse strain in a publication, please [cite the originating article\(s\)](#) and include JAX stock #008672 in your Materials and Methods section.

Animal Health Reports

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