This spontaneous point mutation causes a less severe phenotype than null mutants or other defined spontaneous mutations of this gene and is valuable for assessing the role of this gene in nervous system development and possibly congenital heart defects associated with some cases of Down syndrome.
**RESEARCH APPLICATIONS**

Developmental Biology Research  
Neurobiology Research  
Cell Biology Research  

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**BASE PRICE**
Starting at:

$2,854.50 Domestic price Cryo Recovery

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**Details**

**Detailed Description**

*Dscam*\(^{3J}\), a point mutation causing an R1018P amino acid substitution in the second fibronectin domain, causes a less severe phenotype than *Dscam*\(^{2J}\), *Dscam*\(^{del17}\), or *Dscam* targeted null mutants. The phenotype of *Dscam* null mutants varies with genetic background and includes respiratory distress, associated perinatal lethality, changes in C4 ventral root and pre-inspiratory neuron signaling, and an abnormal response to hypercapnia. *Dscam*\(^{3J}\) homozygotes display kyphosis, domed skull, muscle stiffness, and less difficulty in the righting response than is found in *Dscam*\(^{2J}\) homozygotes, which have a truncation in the extracellular domain of this protein. *Dscam*\(^{3J}\) homozygotes have enlarged central and lateral ventricles of the brain.

Characterization of the retina shows that, similar to *Dscam*\(^{2J}\) homozygotes, *Dscam*\(^{3J}\) homozygotes have expanded inner plexiform, inner nuclear and retinal ganglion cell layers, but, distinct from *Dscam*\(^{2J}\) homozygotes, the inner nuclear layer is evenly laminated. The retinal ganglion cells have defects in arborization and soma spacing, but the dopaminergic amacrine cells have reduced defects in arborization and soma spacing compared with those of the retinas of *Dscam*\(^{2J}\) homozygotes. Retinas of *Dscam*\(^{3J}\) homozygotes also have increased incidence of juxtaposed dopaminergic amacrine cells, occasional loose fasciculation of dopaminergic amacrine cell neurites, and increased numbers of bNOS amacrine cells, which also have abnormal spacing and appear hypertrophied, but the severity of these defects is less severe than the defects caused by other *Dscam* mutant alleles. The defects in neurite lamination, laminar specificity of type 2 and type 6 cone bipolar cells, and the disrupted targeting of retinal ganglion cell axons to the dorsal lateral geniculate nucleus that are found in *Dscam*\(^{2J}\) and other *Dscam* mutant homozygotes are not distinct phenotypes in *Dscam*\(^{3J}\) homozygotes. Subcellular localization of DSCAM protein from *Dscam*\(^{3J}\) homozygotes shows increased retention in the cell bodies in the retinal ganglion cell layer consistent with a model of mis-localization of this mutant protein. (See Schramm et al., 2013 for more detail.)
Genotyping Protocols
End Point Analysis: Dscam<3J>
Genotyping resources and troubleshooting

Mating System
Heterozygote x Heterozygote

Citation
When using the C3(Cg)-Dscam<3J>/GsrJ mouse strain in a publication, please cite the originating article(s) and include JAX stock #006046 in your Materials and Methods section.

Animal Health Reports
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