Homozygous Sod1<sup>tm1Leb</sup> knock-out mice are characterized by abnormal retinal and cochlear ganglions, and they have age-related defects in hearing, vasculature, and muscle. These mice may be useful in studying retinal dysfunction, autoimmunity, female reproductive development/function, glaucoma, hearing loss, Alzheimer's Disease, familial amyotrophic lateral sclerosis (ALS), and other age-related disorders.
The \textit{Sod1}^{tm1Leb} (formerly \textit{SOD1}^{m1BCM}) knock-out allele disrupts exons 1 and 2 of the Cu,Zn-superoxide dismutase-1 gene (\textit{Sod1}). This knock-out allele is the same as carried by B6;129S-\textit{Sod1}^{tm1Leb}\textit{/J} (Stock No. 002972) but is fully congenic in the present strain having been backcrossed to C57BL/6JEi for 13 generations. In the original strain no protein expression from the targeted locus is detected in brain tissues. Heterozygous mice are viable and fertile. While homozygous males reproduce normally, homozygous females are infertile. Homozygous mice have abnormalities of the retinal ganglion, cochlear ganglion, and female reproductive development/function. Homozygous mice also exhibit age-related deafness, accelerated vascular aging and age-related skeletal muscle defects. These mutant mice may be useful in studying retinal dysfunction, autoimmunity, glaucoma, hearing loss, Alzheimer’s Disease, familial amyotrophic lateral sclerosis (ALS), and other age-related disorders.
Genotyping Protocols
Standard PCR: Human HPRT
Standard PCR: Sod1
Genotyping resources and troubleshooting

Citation
When using the Sod1 KO mouse strain in a publication, please cite the originating article(s) and include JAX stock #003881 in your Materials and Methods section.

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