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In vivo Animal Models

ALS

SOD1-G93A Transgenic Mouse Model

This Amyotrophic Lateral Sclerosis (ALS) mouse model overexpresses the human SOD1 (superoxide dismutase 1) with G93A mutation under the regulatory control of the human SOD1 promoter.

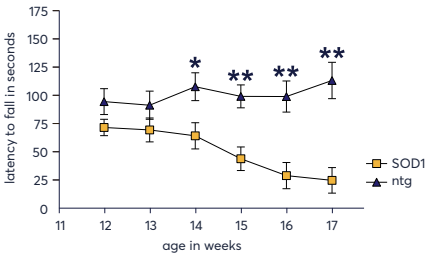
- SOD1 accumulation in spinal cord, brain stem and midbrain
- Motor neuron loss in spinal cord and brain regions such as the SN
- Neuron loss accompanied by neuroinflammation
- Strong involvement of astrocytes and microglia
- Severe motor deficits starting at 12 weeks and worsening over age

Figure 1:
RotaRod and wire hanging test of 12 - 18 weeks old SOD1-G93A mice compared to non-transgenic (ntg) mice. Time animals stay on the rod or keep hanging on the wire. Two-way ANOVA with Bonferroni's *post hoc* test. Mean ± SEM; n = 12 - 18; *p<0.05, **p<0.01, ***p<0.001.

Figure 1

RotaRod

A



Wire Hanging

B

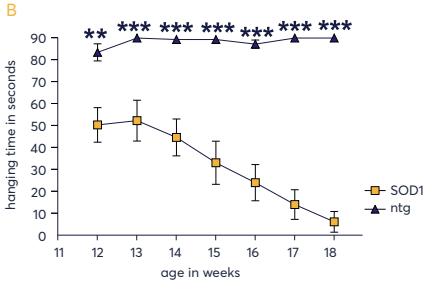
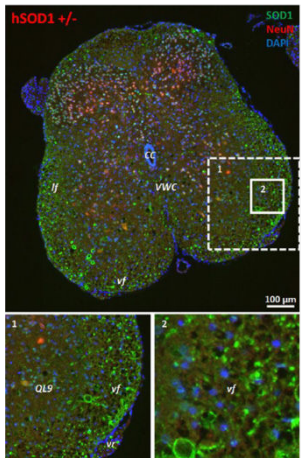


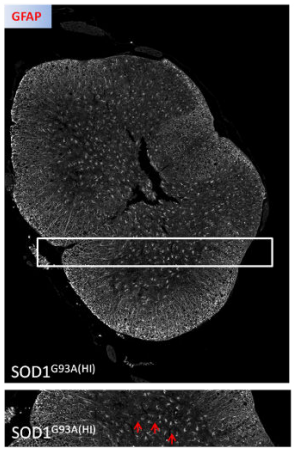
Figure 2:
A: SOD1 expression and neuronal loss in the spinal cord of SOD1G93A mice.
B: Astrocytosis in the spinal cord of SOD1-G93A mice.

Figure 2

A



B



Gurney et al. Motor neuron degeneration in mice that express a human Cu, Zn superoxide dismutase mutation. Science 1994; 264 (5166):1772-5.

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Discovery

Important note:
Representative data are shown throughout this document. However, biological variability might cause deviations from shown data.

Scantox Group, HQ
Hestehavevej 36A, Ejby
DK – 4623 Lille Skensved
clientservice@scantox.com
www.scantox.com
+45 5686 1500

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