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In vivo Animal Models

Huntington's Disease

R6/2 Transgenic Mouse Model

R6/2 mice express a N-terminal fragment of the Huntington's Disease (HD) gene under control of human gene promoter elements with >120 CAG repeats that are sufficient to produce the phenotype of HD:

- HTT aggregates
- Motor deficits
- Learning deficits
- Mean survival of appr. 100 days

Figure 1:
RotaRod test of R6/2 mice. Motor coordination expressed as time to fall off the rod in seconds of R6/2 and ntg mice. Mean ± SEM, Two-way ANOVA with Bonferroni's *post hoc* test; R6/2: n = 10, ntg: n = 19; **p<0.01; ***p<0.001.

Figure 2:
Brain atrophy in 4 month old R6/2 mice reflecting an approximately 20% grey matter loss in both investigated regions compared to age-matched ntg littermate. Mean + SEM; unpaired t-test; n = 5; ***p<0.001.

Figure 3:
Astrogliosis in R6/2 mice. Quantitative and qualitative comparison of astrocytosis in R6/2 and non- transgenic (ntg) mice.
A: Number of astrocytes per mm². Mean ± SEM; unpaired t-test; n = 5; *p<0.05; **p<0.01
B: Representative images of GFAP (red) in primary somatosens. cortex and hippocampal CA1 region. DAPI (blue).

Figure 1

RotaRod

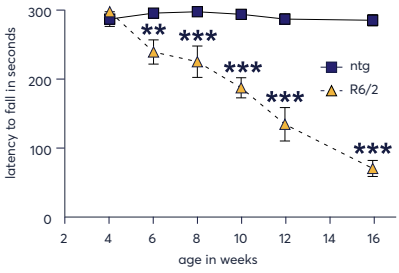
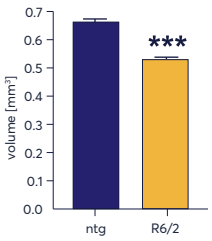


Figure 2

Brain Atrophy

Cortex



Hippocampus

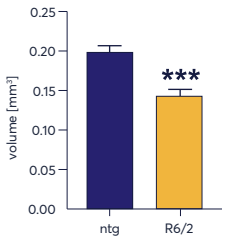
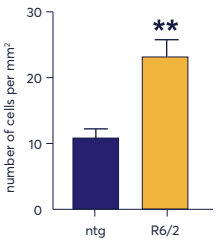


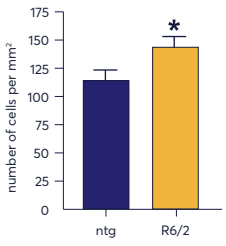
Figure 3

Cortex

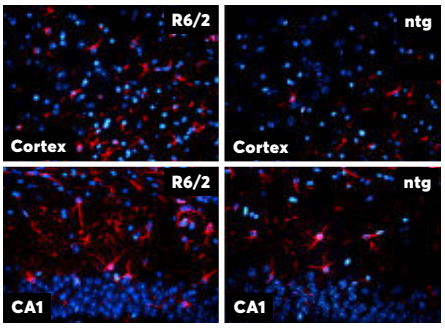
A



Hippocampus



B



Mangiarini L., Sathasivam K., Seller M., Cozens B., Harper A., Hetherington C. , Lawton M., Trottier Y. , Lehrach H., Davies S.W., Bates G.P. Exon 1 of the HD Gene with an Expanded CAG Repeat Is Sufficient to Cause a Progressive Neurological Phenotype in Transgenic Mice. Cell, 1996, Vol. 87, 493–506.

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Discovery

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

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126-35-HuntingtonsR6_2-EN (04/2024)