In vivo Animal Models

Fragile X Syndrome

Fmr1-KO Mouse Model

Fmr1-KO mice

The mouse model contains a neomycin resistance cassette substituting exon 5 of the fragile X mental retardation syndrome 1 (Fmr1) gene. The knockdown causes an increase in the number of CGG repeats that lead to hypermethylation of the Fmr1 gene and therefore inhibiting FMR protein production.

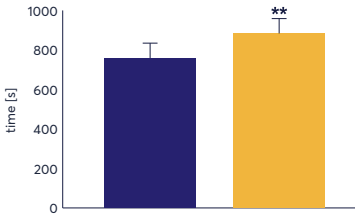
At the age of 7 weeks mice start to present core and secondary phenotypic traits of Fragile X syndrome such as:

- Hyperactivity
- Altered anxiety levels
- Repetitive behavior
- Social behavior deficits
- Vocalization deficits

Figure 1:
Activity, hyperactivity, anxiety, and repetitive behavior of male Fmr1-KO mice at the age of 7 weeks. Activity **(A)** and hyperactivity **(B)** measured in the open field test, time spent in open arms of the open field test **(C)**, and time spent grooming in the auto-grooming test **(D)** of Fmr1-KO compared to C57BL/6JRj mice. n = 15 per group. Unpaired t-test; Mean + SEM; **p<0.01.

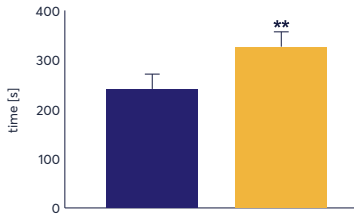
Activity

Figure 1: A



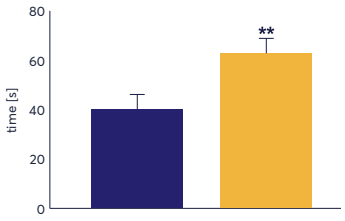
Hyperactivity

Figure 1: B



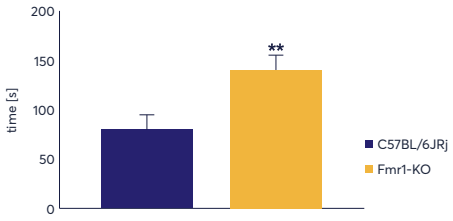
Open arms

Figure 1: C



Grooming

Figure 1: D



References:
Bakker et al. 1994. Fmr1 knockout mice: a model to study fragile x mental retardation. The Dutch-Belgium Fragile X Consortium. Cell 78(1):23-33.

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Discovery

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