

Fmr1 knockout rat

MODEL	Fmr1 knockout rat
STRAIN	HsdSage: SD- <i>Fmr1</i> ^{em1Sage}
LOCATION	U.S.
AVAILABILITY	Live colony



CHARACTERISTICS/HUSBANDRY

- This model was created in collaboration with Autism Speaks and underwent phenotypic characterization by Dr. Richard Paylor at Baylor College of Medicine
- Preliminary results suggest Fmr1 knockout rats possess perseverative chewing behavior and decreased juvenile play
- Homozygous knockout rats exhibit complete loss of target protein as demonstrated by Western blot
- An expansion of CGG trinucleotide repeat in Fmr1 has been implicated in Fragile X syndrome
- Background strain: Sprague-Dawley
- This gene is X-linked

ZYGOSITY GENOTYPE

- Homozygous (Females) / Hemizygous (Males)

RESEARCH USE

- Autism
- Fragile X syndrome

ORIGIN

The Fmr1 knockout rat model was originally created at SAGE Labs, Inc. in St. Louis, MO. The animal inventory was acquired by Envigo in 2019 and then by Inotiv in 2021. The line continues to be maintained through the original SAGE Labs animal inventory and is distributed out of the Boyertown, PA facility.

DESCRIPTION

This model contains a deletion of the Fragile X mental retardation 1 gene (Fmr1). Mutations in Fmr1 result in Fragile X syndrome, the leading monogenic cause of autism, making this rat useful for the study of both Fragile X syndrome and autism. The X-linked gene Fmr1 produces the fragile X mental retardation protein, or FMRP. FMRP is essential for normal mental development. An expansion of the trinucleotide CGG repeat in the Fmr1 gene is responsible for fragile X syndrome, a syndrome characterized by autism and mental disability.

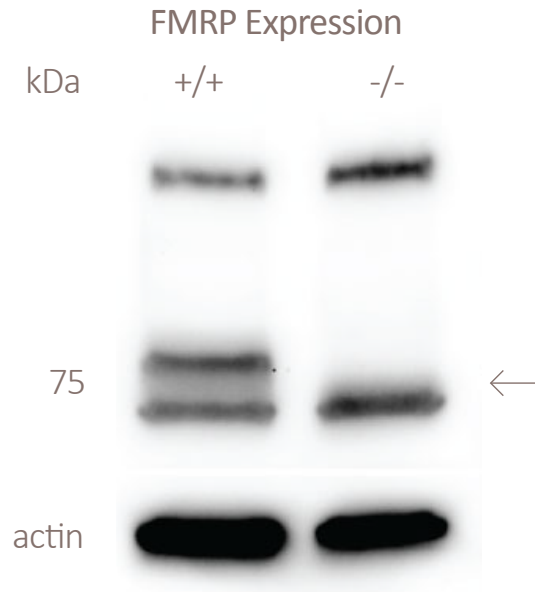


Figure 1: Loss of FMRP protein in *Fmr1* knockout rats. FMRP protein expression is disrupted in *Fmr1* knockout rats as compared to wild type controls as demonstrated by Western blot. Actin staining demonstrates equal sample loading.

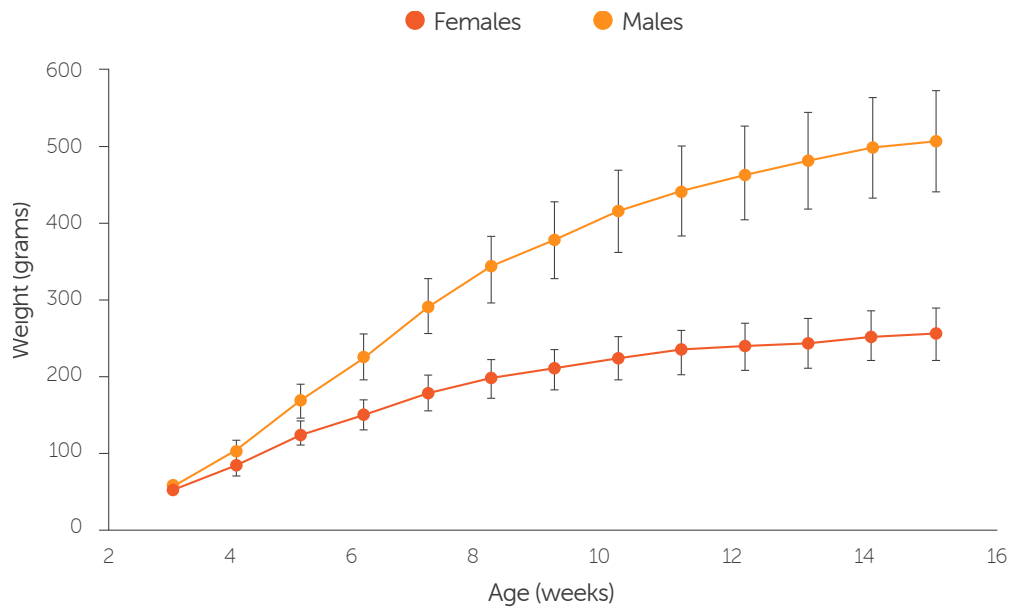


Figure 2: A graph showing the correlation between the age and weight of *Fmr1* knockout rats.