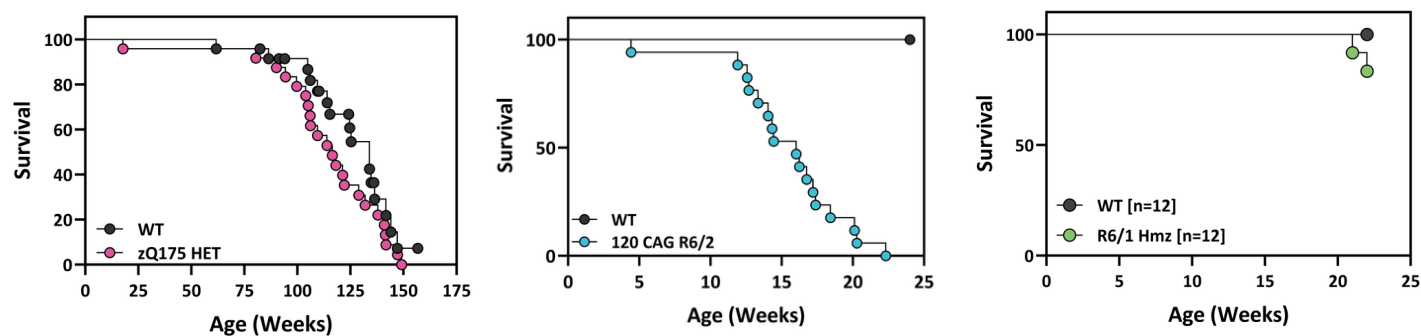


In recognition of Huntington’s Disease (HD) Awareness Month, PsychoGenics is proud to support your HD research needs.

With decades of experience in HD research, we offer three, fully characterized, mouse models of HD for assessing potential therapeutics: the zQ175 knock-in (replacement of mouse Huntingtin (HTT) exon 1 with the mutant human HTT exon 1, 190 CAG repeats), the R6/2 120 CAG (expresses the N-terminal portion of the huntingtin protein), and the R6/1 (carries the human huntingtin gene’s 5’ end, 116-120 CAG repeats).

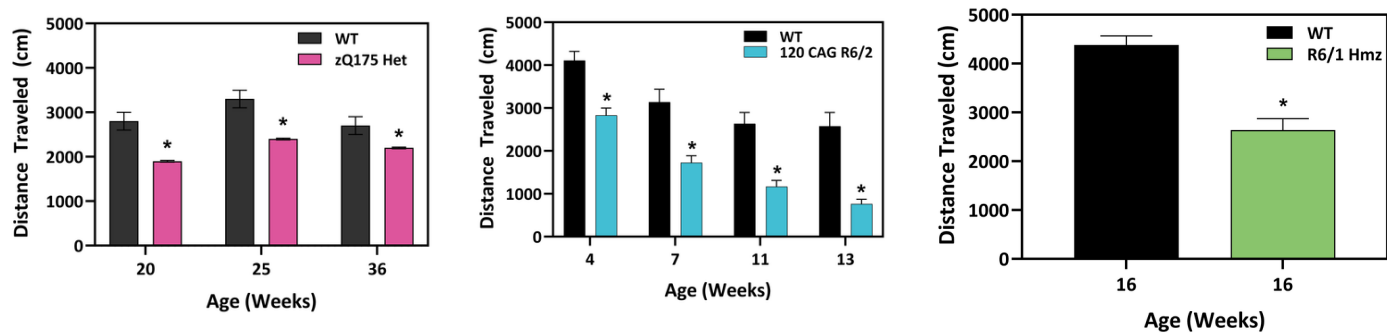
Through molecular, behavioral, histological, and electrophysiological approaches, we have characterized these models at both pre-and post-symptomatic stages. These models serve as validated and predictive tools for assessing novel HD treatments.

SURVIVAL



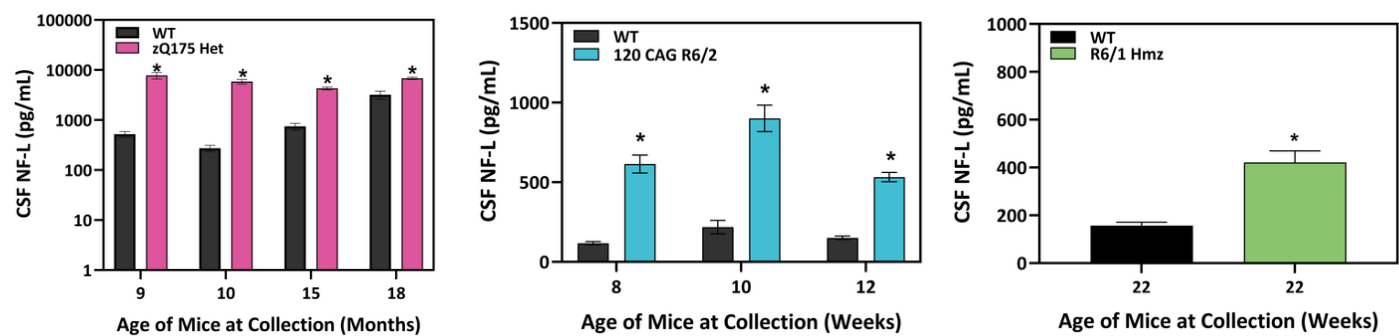
Survival data for all 3 models. zQ175 Het and R6/2 Hmz mice display reduced survival as compared to wild-type littermates, while there are no significant survival differences for R6/1 Hmz mice.

MOTOR DEFICITS



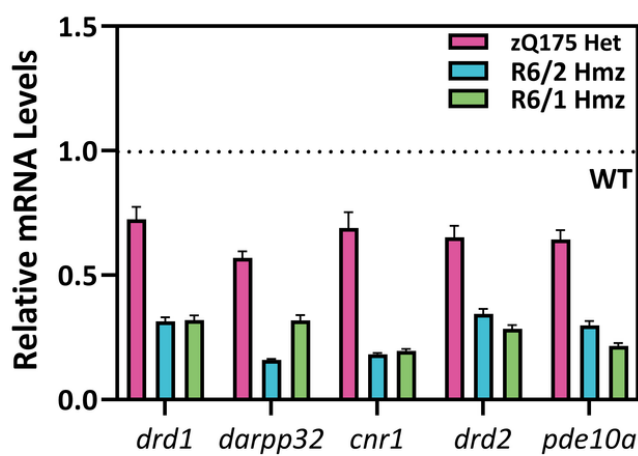
All 3 models exhibit motor deficits as assessed by open field at multiple timepoints. The total distance traveled is decreased in zQ175 Het, R6/2 Hmz, and R6/1 Hmz mice as compared to wild-type littermates.

NEUROFILAMENT LIGHT CHAIN (NFL) LEVELS



Axonal degradation is assessed by neurofilament light chain (NfL) levels in the cerebrospinal fluid. All 3 models show significant increases in NfL as compared to wild-type littermates.

MSN MARKERS



Striatal transcript expression levels of genes for medium spiny neuron (MSN) markers were assessed by qPCR. Markers were normalized to housekeeping genes (gapdh, atp5b, elf4a2), and wild-type littermates. MSN target genes were significantly decreased in zQ175 Het mice at 30-weeks of age, R6/2 Hmz mice at 12-weeks of age, and R6/1 Hmz mice at 22-weeks of age, reflecting a loss of MSNs.

Eager to know how PsychoGenics can revolutionize your research?

Let's talk. Contact us below.

[Contact Us](#)