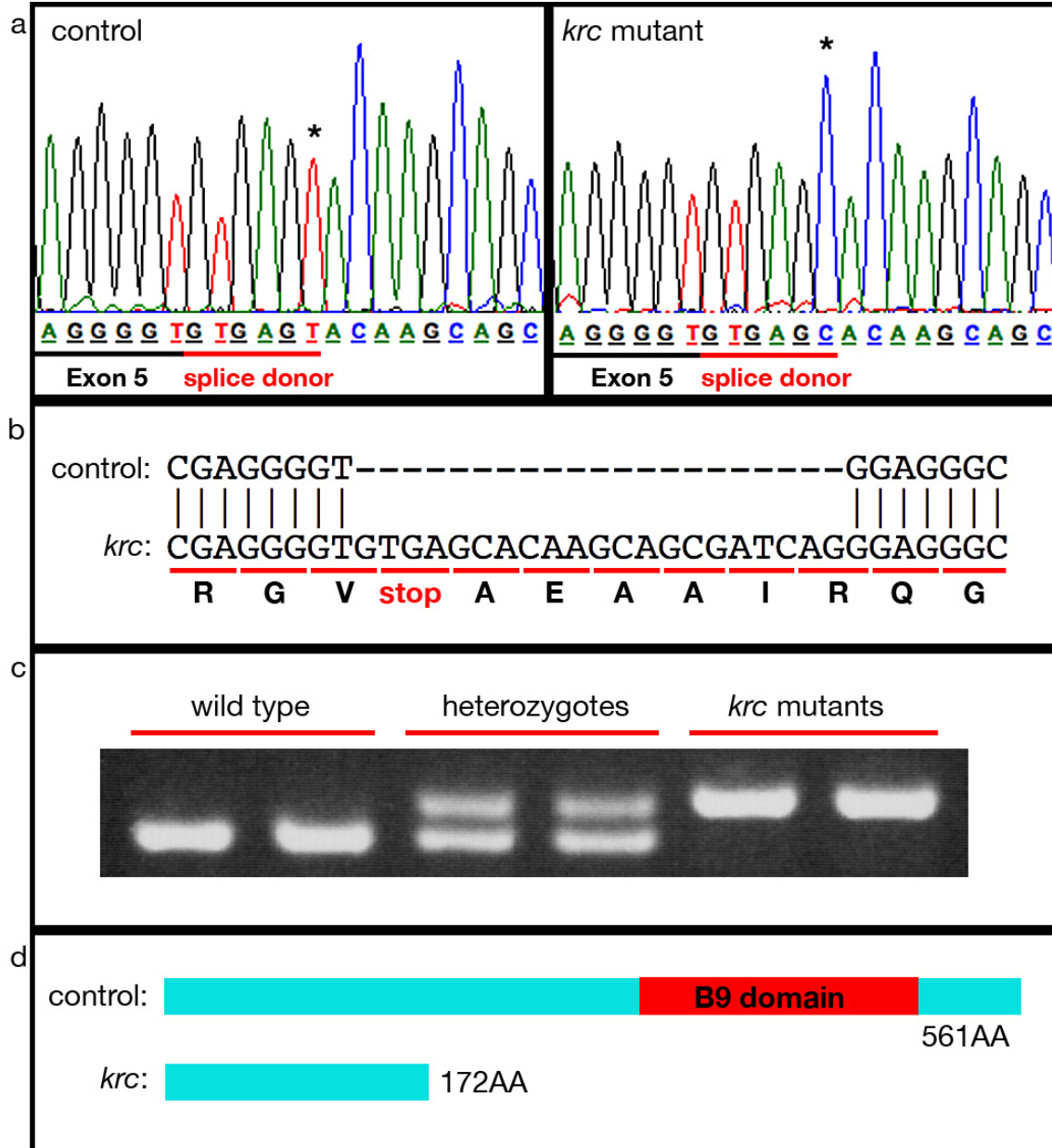


**Supplemental Figure 1**

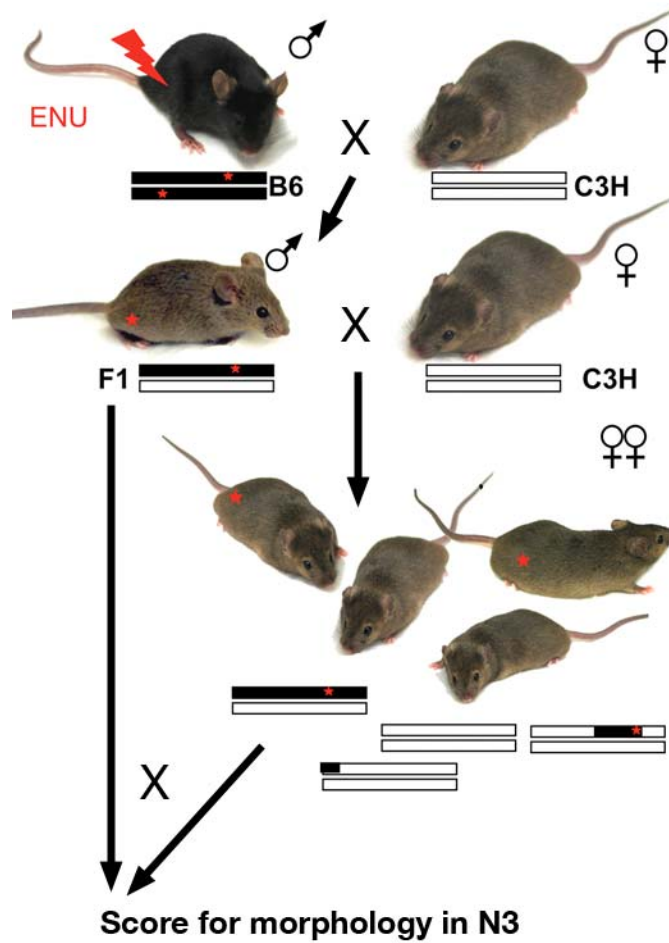


**Supplemental Figure 1. Identification of the *krc* mutation in the *Mks1* gene.**

(a) *Mks1* genomic sequencing traces showing a T>C transition (asterisks) in last position of the mutant splice donor site in intron 5. (b) Sequencing of *Mks1* transcripts identified a 21-nucleotide insertion in the *krc* mutants. The predicted translation indicates an in-frame premature stop codon. (c) RT-PCR of *Mks1* mRNA from controls and mutants show a smaller band in wild-

type animals, two bands in heterozygous animals and a single, larger band in *krc* mutants. No wild-type transcript is evident in the mutants. (d) Predicted Mks1 proteins in wild type and *krc* mutants, N-termini to the left. The only known domain in Mks1 is a B9 domain found in other basal body proteins (eg PMID: 19208769, PMID: 18337471, PMID: 18287022). The premature stop codon in *krc* mutants truncates the protein prior to the B9 domain.

**Supplemental Figure 2.**



**Supplemental Figure 2. Forward genetics screen for recessive mutations.** C57BL/6J (B6) males were mutagenized with ENU and subsequently crossed to C3HeB/FeJ (C3H) females. F1 sons of mutagenized mice were again crossed to C3H females to generate second-generation (G2) females. Several G2 females per line were mated to their F1 fathers. Embryos from this cross were examined for abnormal morphological features.