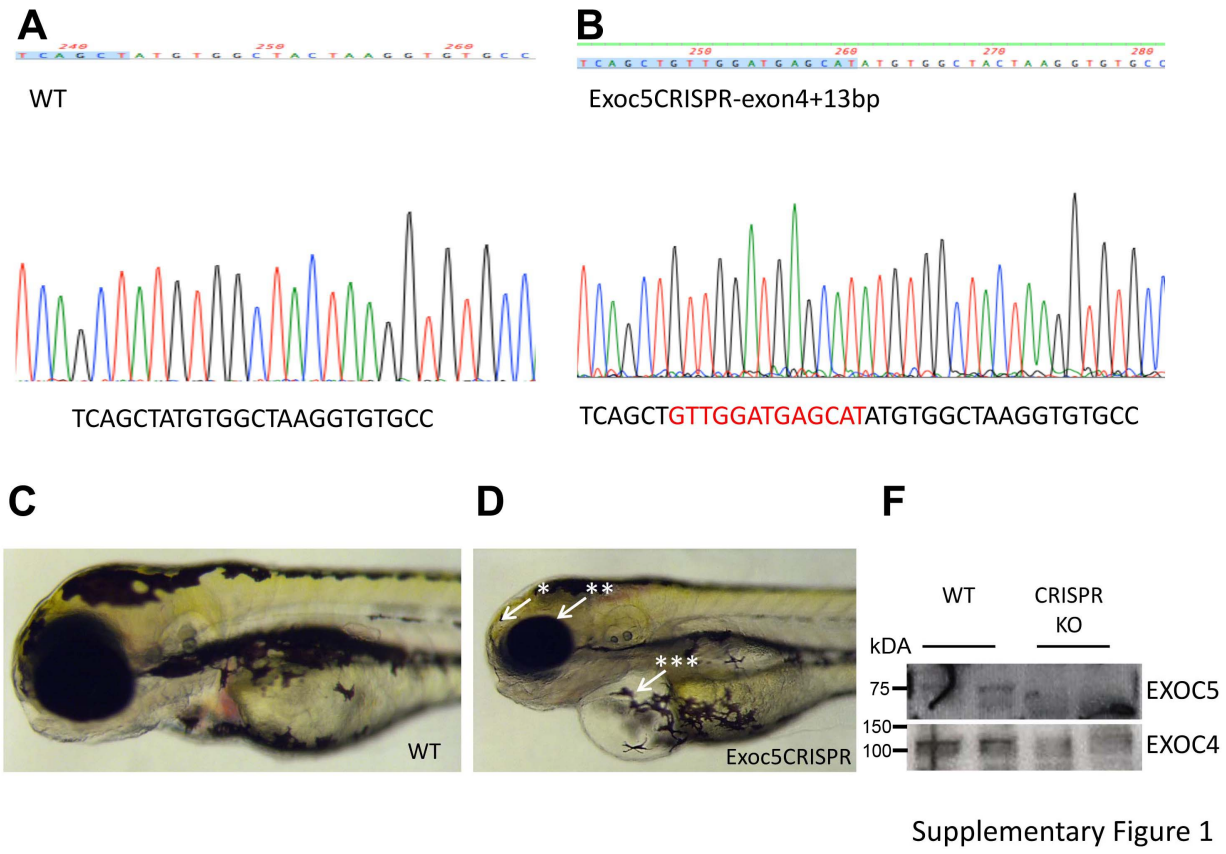
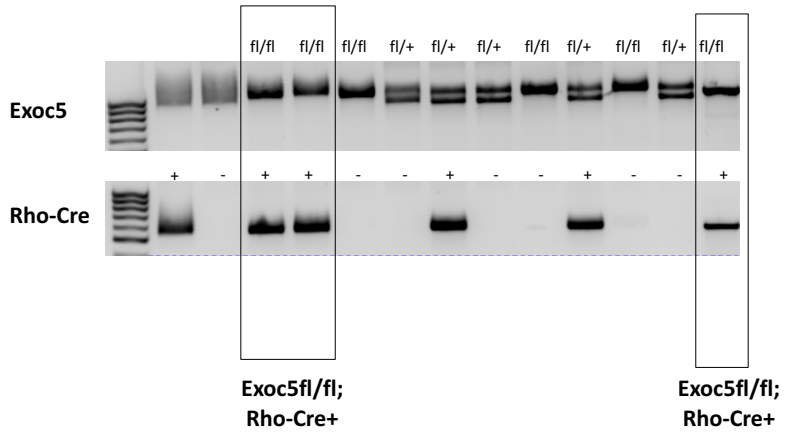


Supplementary Figure 1

Supplementary Figure 1. A second *exoc5* mutant zebrafish line, generated using CRISPR, shows ciliopathy phenotypes. (A, B) Chromatograms of Sanger sequencing reactions of wild type (WT) and homozygous *exoc5* mutant (*exoc5* Mut) zebrafish showing the 13 bp insertion from CRISPR gene editing that leads to a stop codon. (C,D) Lateral view of representative WT (C) and *exoc5* homozygous mutant (D) zebrafish at 3.5 dpf. *Exoc5* homozygous CRISPR mutants phenocopied the homozygous exoc5 mutants we received from ZIRC (Fig. 1), with cilia defects that included: *hydrocephaly; **smaller eyes, ***pericardial edema and tail curvature.



Supplementary Figure 2

Supplementary Figure 2. Genotyping for the target Exoc5 fl/fl;Rho-Cre+ mice. Using the genotyping primers described in the Methods section and PCR, we identified our target Exoc5 fl/fl;Rho-Cre+ pups resulting from our final cross of male Exoc5 fl/+;Rho-Cre+ X female Exoc5 fl/fl mice. The target Exoc5 fl/fl;Rho-Cre+ mice were positive both for Rho-Cre and for Exoc5 fl/fl (one band). Exoc5 fl/fl and Exoc5 fl/+;Rho-Cre+ littermates were used as controls for the experiments described in the manuscript.