Review of: "Myosin phosphatase target subunit 1 governs integrity of the embryonic gut epithelium to circumvent atresia development in medaka, Oryzias latipes"

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The study reports a novel model of intestinal atresia (IA). The authors have impressively generated an MYPT1 knockout medaka using the CRISPR/Cas9 system, from which they were able to isolate and maintain a mutant cell line to yield a transcriptional frame shift that alters 45 codons and a premature stop codon at the 88th codon. They report that MYPT1 mutation or loss of function causes augmented actomyosin contraction, which is responsible for the IA phenotype.

The authors indicated in the results section that the g1-4 medaka mutant developed IA but did not point to supporting evidence in this section. The evidence for this was, instead, presented in the discussion, but would have been better suited for the results. They also could not distinguish between small and large intestine, which, they say, behave differently, and indicated that the rest of the intestine appeared to be properly developed at stage 40. Given that a distinction could not be made, it is not clear what they refer to as the rest of the intestine. They also did not indicate which portion of the intestine showed atresia.

The authors report seeing dilation upstream of the atresia at the hatching stage in mutants, but have not made reference to what the normal intestine looks like in the same region.

Overall, the research is a good one and the conclusions are in sync with the findings reported.