SUPPLEMENTARY FIGURE LEGEND

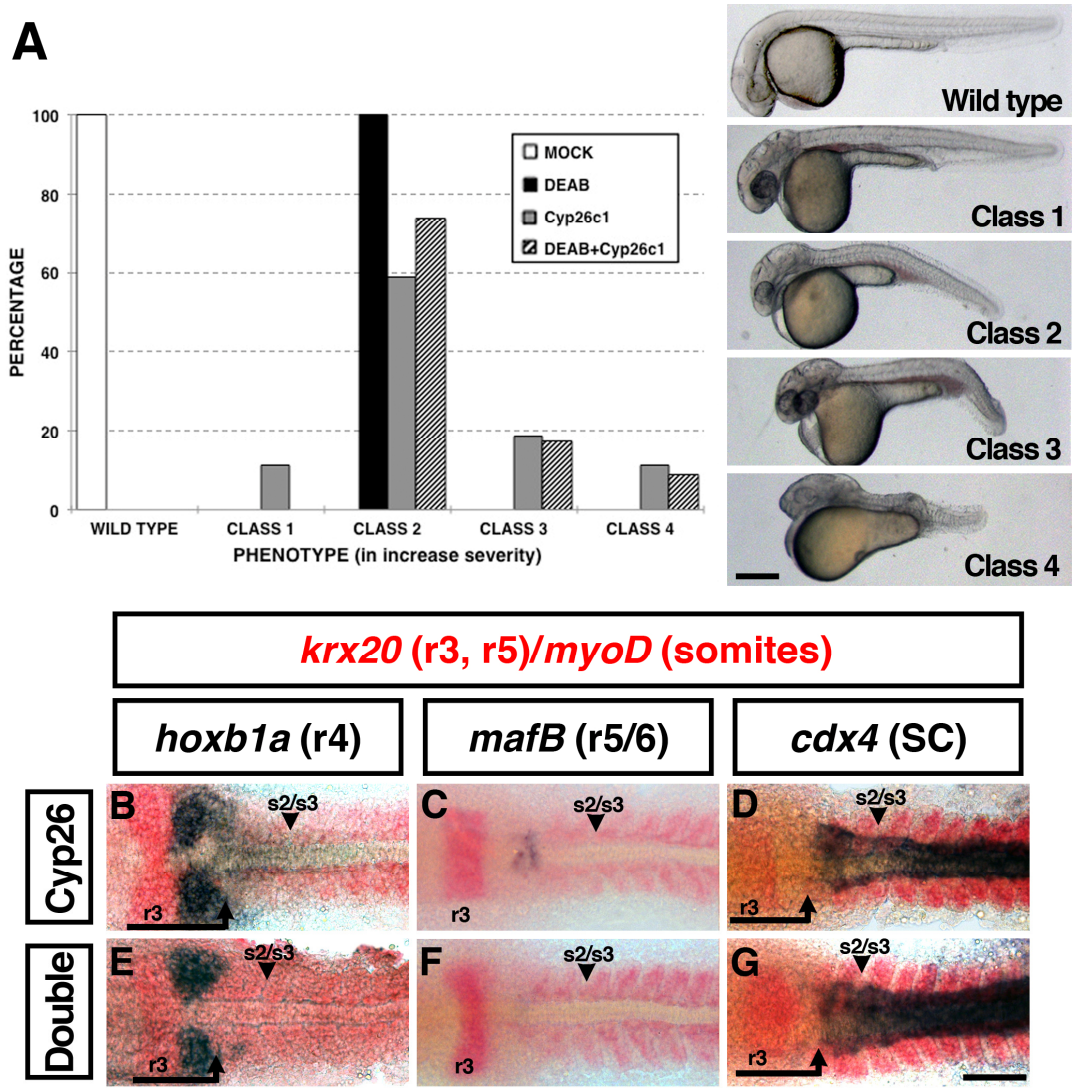

Figure S1. Overexpression of the Retinoic Acid-degrading enzyme Cyp26c1 recapitulates defects associated with loss of Retinoic Acid synthesis. (**A**) Frequency distribution of morphological defects caused by RA loss due to exposure to the RA synthesis inhibitor DEAB at 50% epiboly (black bars), overexpression of RA-degrading enzyme Cyp26c1 (gray bars) and both (hatched bars). Embryos were scored at 32 hpf using a four-class scale of increase severity. Class 1 had blood pooling. Class 2 had hindbrain size reduction, tail defects, lack of fin buds and edema. Class 3 had severe tail bending. Class 4 had a severely reduced axis and small somites. For each condition, n=80 embryos from three independent trials. Cyp26c1 treatments were statistically significant from mock control (white bar), but not DEAB treatments by pairwise chi square analysis (mock versus cyp26c1 treatment, χ^2=63, df=1, P=0; DEAB versus *cyp26c1* treatment, χ^2=1.01, df=1, $P \leq 0.314$). Embryos are mounted laterally, anterior to the left. Scale bar: 250 µm. (**B-G**) Expression analysis of *hoxb1a* (r4; B,E), *mafb* (r5/r6; C,F) and *cdx4*

(spinal cord; D,G) (purple) relative to r2/r3 (*krx20*, red) and somites 2/3 (*myoD*, red) boundaries, in Cyp26c1-overexpressing embryos that were not exposed (Cyp26c1; B-D) or exposed (Double; E-G) to the RA synthesis inhibitor DEAB. Similar changes were observed in DEAB-treated embryos (Fig. 1). Embryos at 12-somite stage are shown in dorsal view, anterior to the left. Arrows, distance from r2/r3 to the expression domain of the indicated gene; arrowheads, s2/s3 border. Scale bars: 50 μm.