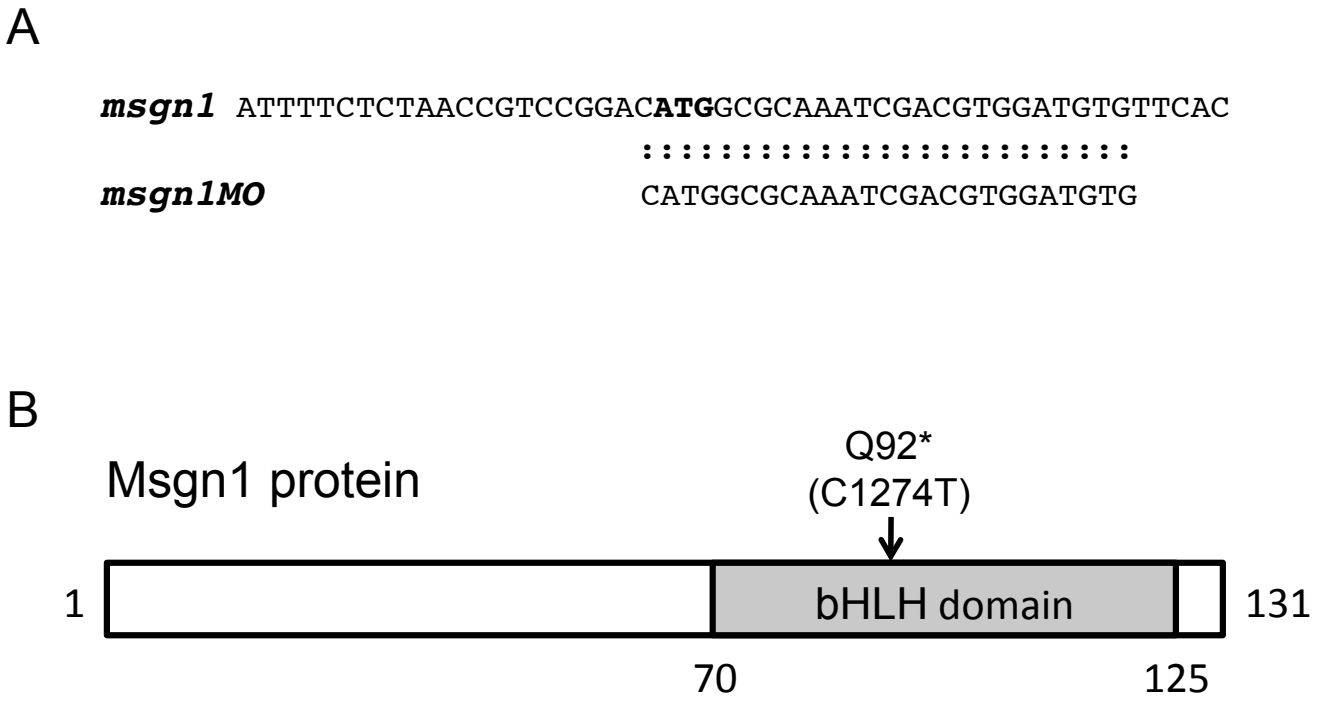

Fig. S1. Msgn1 loss-of-function strategies. (**A**) The *msgn1* morpholino oligonucleotide (*msgn1MO*) was targeted to the initiation ATG codon of the *msgn1* gene. (**B**) The *msgn^{fh273}* allele carries a point mutation that substitutes the cytosine at position 1274 with a thymine, changing the glutamine at position 92 into a stop codon. The resulting protein is truncated in the middle of the first helix of the HLH motif.

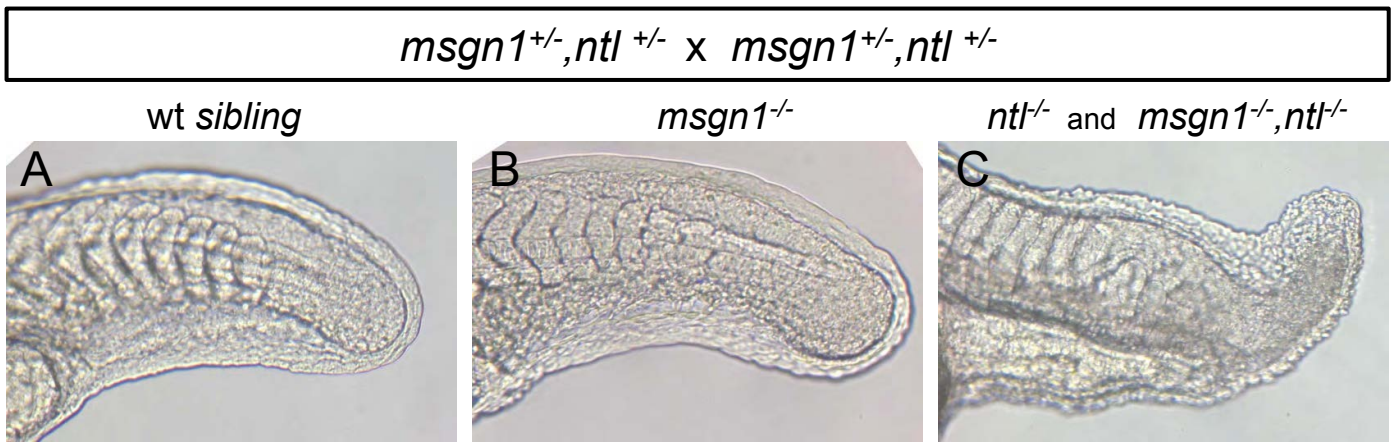

Fig. S2. Loss of *msgn1* does not enhance the tail deficiency of *ntl*$^{-/-}$ mutants. The progeny of a double heterozygous *msgn1*$^{+/-}$*;ntl*$^{+/-}$ cross were sorted into three distinguishable phenotypic classes and subsequent genotyping showed that embryos with a normal tail phenotype were wt (**A**), embryos with an enlarged tailbud were *msgn1*$^{-/-}$ (**B**) and embryos with similar severe tail truncations were either *ntl*$^{-/-}$ or *msgn1*$^{-/-}$*;ntl*$^{-/-}$ double mutants (**C**). Genotypes were present at the expected ratios.

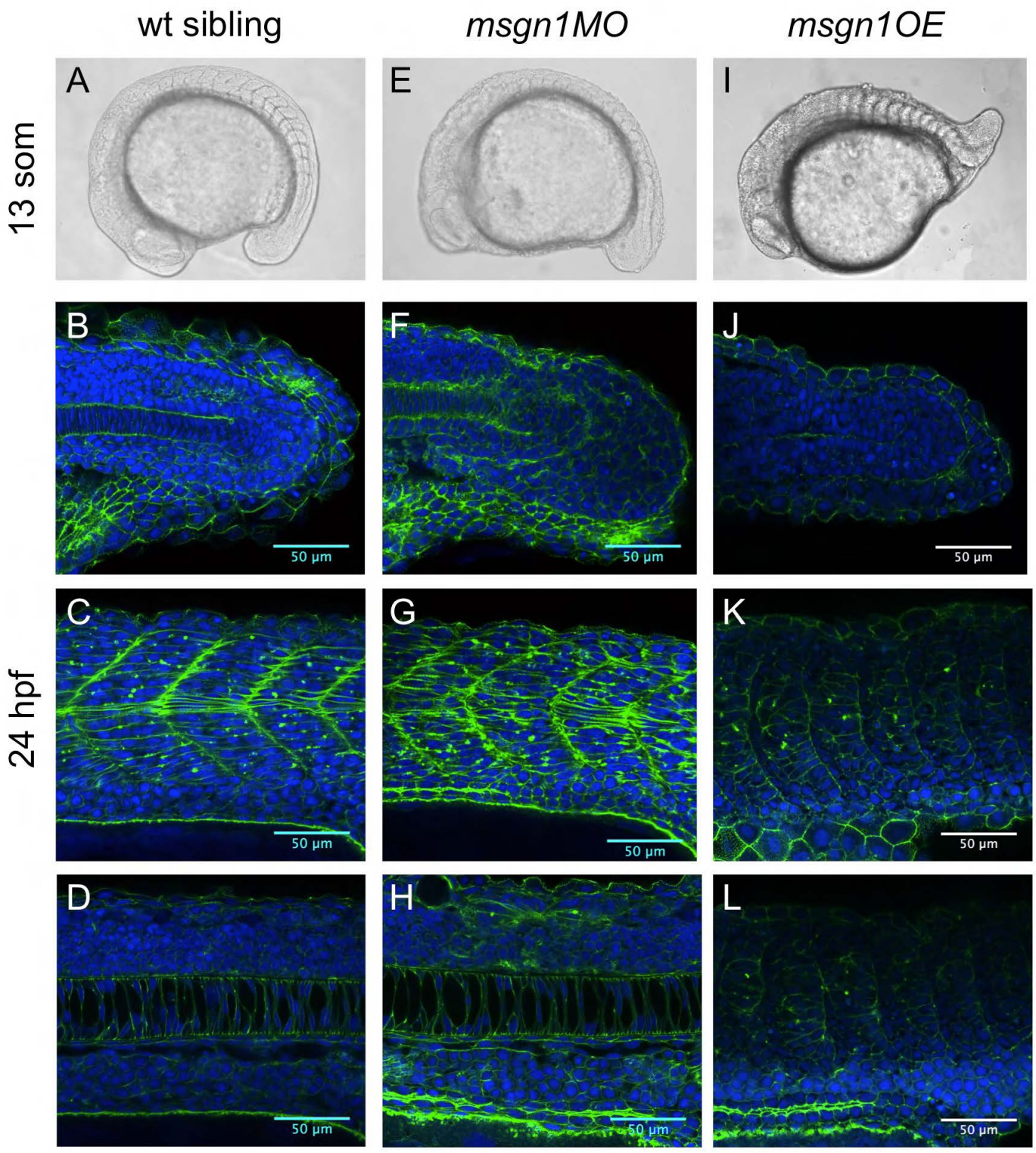

Fig. S3. Msgn1 regulates posterior development. (**A**,**E**,**I**) Live images of wt (A), *msgn1MO*-injected (E) and *msgn1*-overexpressing (I) embryos. (**B-D**,**F-H**,**J-L**) Nuclei (DAPI, blue) and F-actin (phalloidin, green) staining of wt (B-D), *msgn1MO*-injected (F-H) and *msgn1*-overexpressing (J-L) embryos. (B,F,J) Confocal sections at the level of the tailbud (B,F,J), the trunk somites (C,G,K) and the notochord (D,H,L).

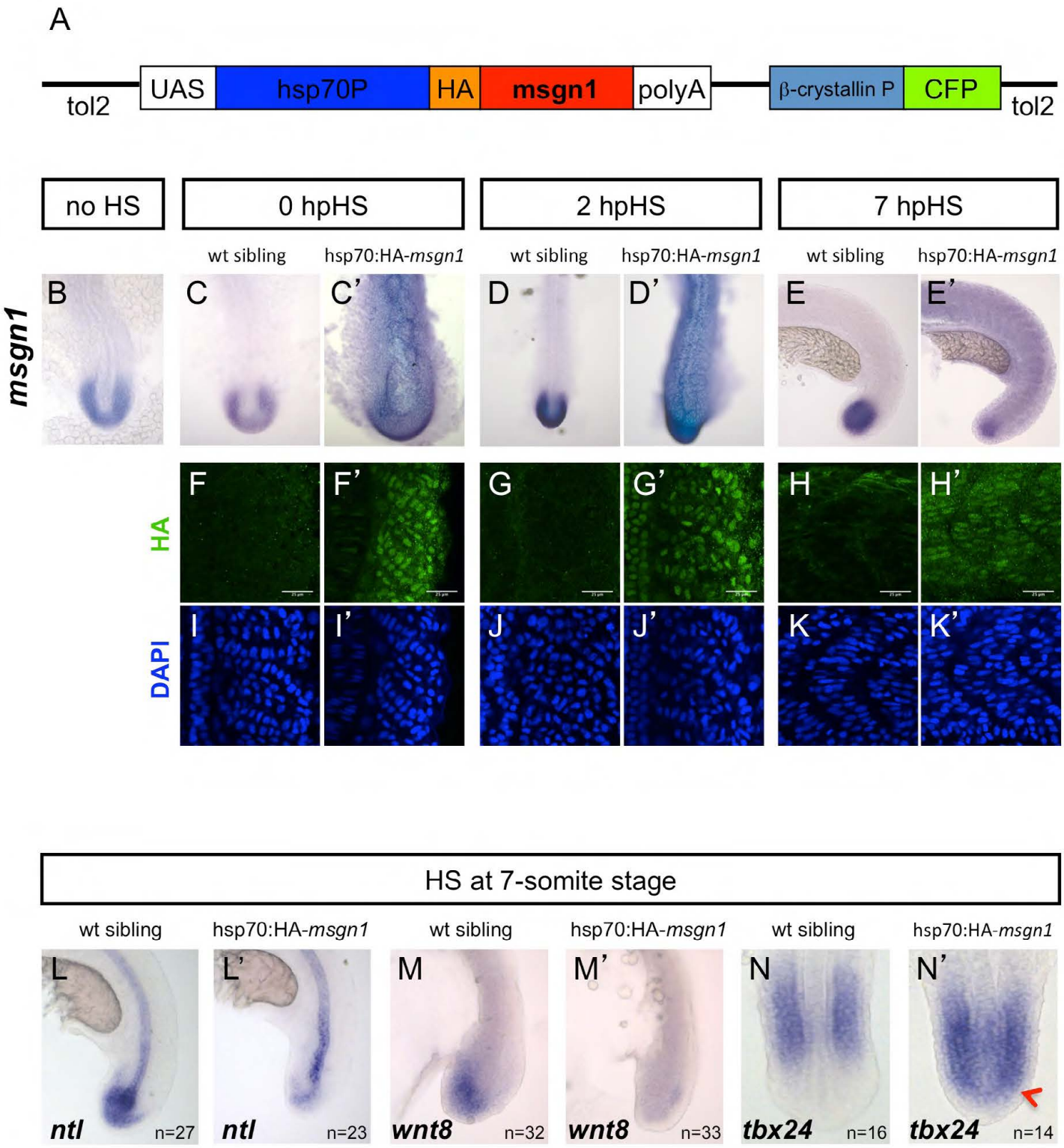

Fig. S4. Generation and validation of the hsp70:HA-*msgn1* transgenic line. (**A**) The construct used to generate the hsp70:HA-*msgn1* transgenic line. The N-terminus of the *msgn1* gene was fused with an HA tag and placed under an *hsp70* promoter. In addition, the β-*crystallin* promoter was used to drive CFP in the lens to facilitate identification of transgenic embryos. (B-N′) Embryos were obtained from a cross between hsp70:HA-*msgn1* heterozygous and wt fish, generating a batch with an expected frequency of 50% transgenics and 50% wt control siblings. (**B-E**′) In situ hybridisation showing *msgn1* mRNA levels in hsp70:HA-*msgn1* transgenic embryos and their wt sibling controls, with no heat shock (B) or heat shocked for 1 hour at the 13-somite stage and fixed immediately (C,C′), 2 hpHS (D,D′) and 7 hpHS (E,E′). (**F-K**′) Levels of HA-tagged Msgn1 protein (F-H′) at the level of the tenth somite in hsp70:HA-*msgn1* transgenic embryos fixed immediately after heat shock (0 hpHS), 2 hpHS and 7 hpHS and their corresponding control siblings. (I-K′) The same embryos as in F-H′ counterstained with DAPI to reveal the nuclei. (**L-N**′) Expression of *ntl*, *wnt8* and *tbx24* in hsp70:HA-*msgn1* transgenic embryos and their respective control siblings heat shocked for 1 hour at the 7-somite stage and fixed 7 hpHS. Red arrowhead, ectopic expression of *tbx24* in the tailbud. hpHS, hours post-heat shock.

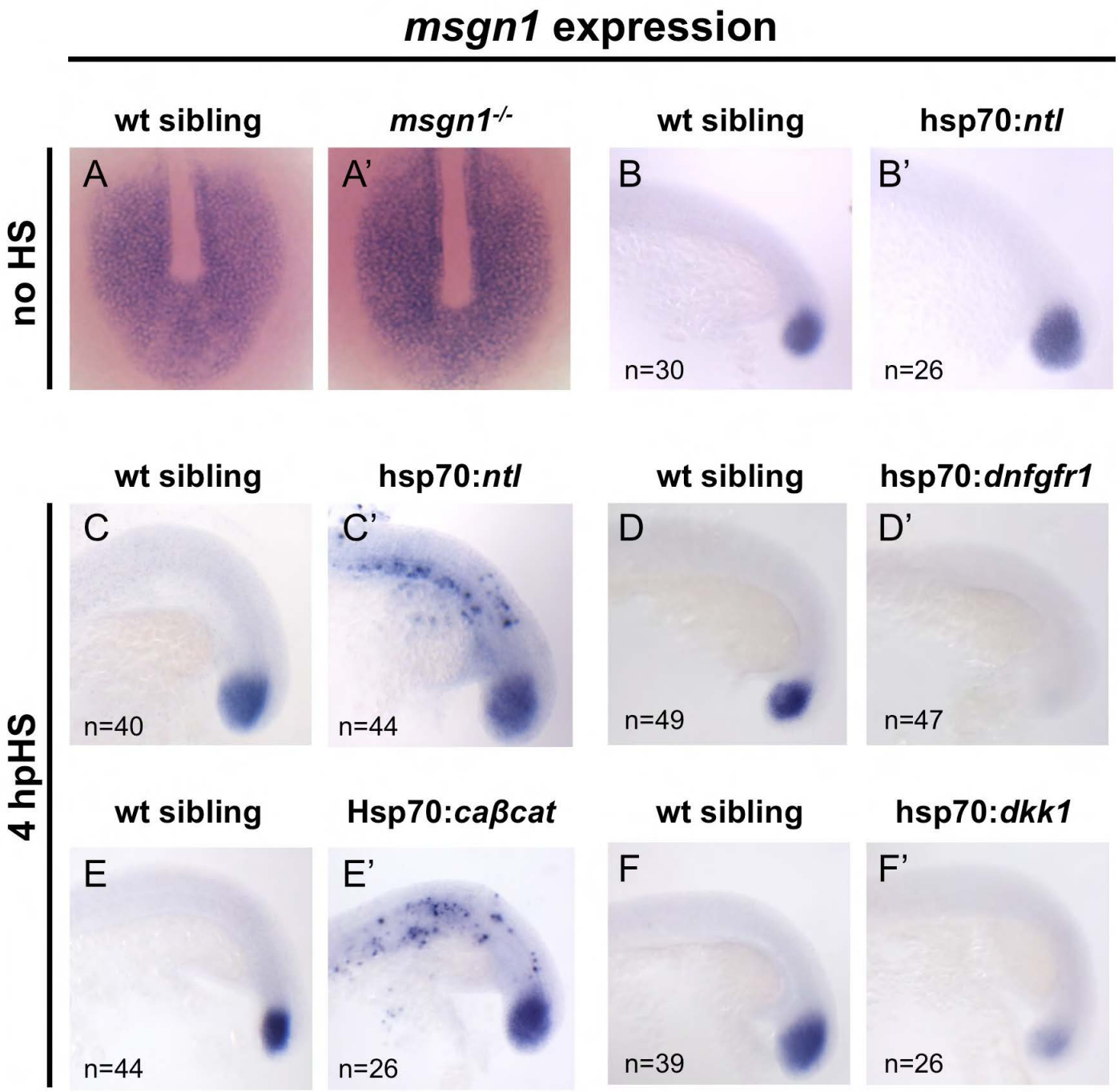

Fig. S5. Regulation of *msgn1* expression during segmentation. (**A**,**A′**) Similar expression of *msgn1* in the presomitic mesoderm of 8-somite stage embryos was detected in wt siblings (A) and *msgn1*$^{-/-}$ embryos (A′). (**B**,**B′**) With no heat shock, a normal pattern of expression of *msgn1* is observed in hsp70:*ntl* injected embryos and their uninjected siblings. (C-F′) All embryos were heat shocked for 30 minutes at the 13-somite stage. (**C**,**C′**) Ectopic expression of *msgn1* is induced in hsp70:*ntl* injected embryos when compared with their uninjected siblings. (**D**,**D′**) A complete absence of *msgn1* expression is observed in hsp70:*dnfgfr1* transgenic embryos when compared with their siblings. (**E**,**E′**) Ectopic expression of *msgn1* is induced in hsp70:*caβcat* injected embryos when compared with their uninjected siblings. (**F**,**F′**) A severe downregulation of *msgn1* expression is observed in hsp70:*dkk1* transgenic embryos when compared with their siblings. Transgenic embryos were obtained from a cross between heterozygous transgenics and wt fish, generating batches with the expected frequency of 50% transgenics and 50% wt control siblings.

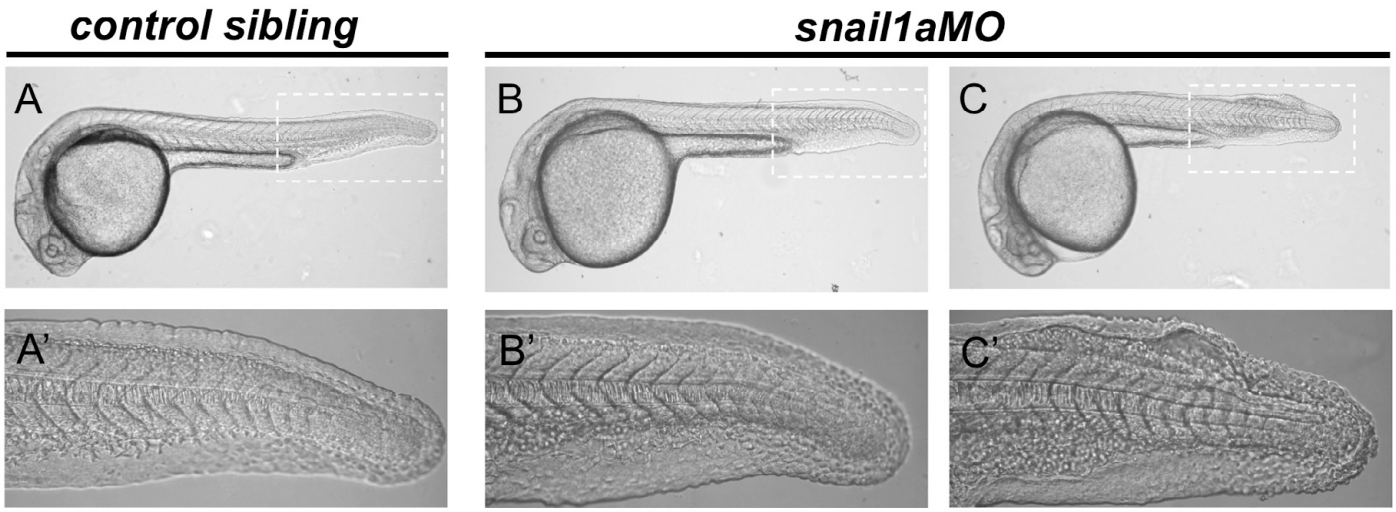

Fig. S6. The mild *snail1a* loss-of-function phenotype. Eighty percent (*n*=123, three different batches) of the *snail1aMO*-injected embryos (**B**,**B′**) show an indistinguishable phenotype from controls (**A**,**A′**) and 20% show a fin-fold phenotype (**C**,**C′**). (A′-C′) Magnification of the tail region corresponding to the embryos shown in A-C.