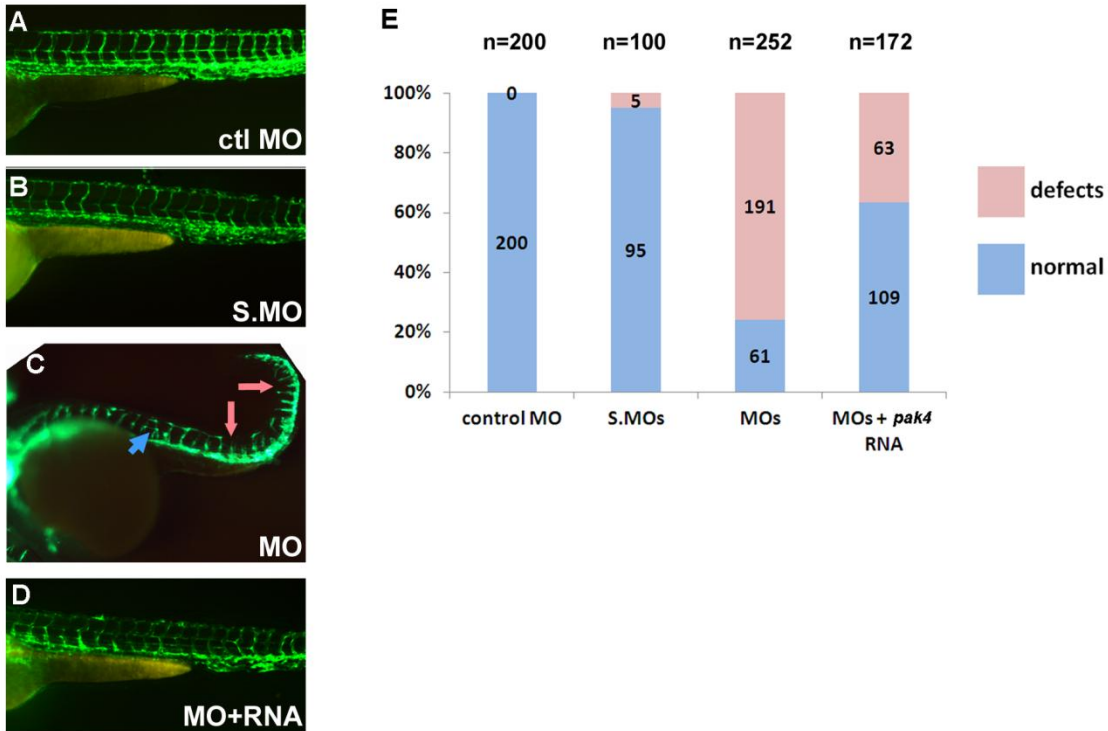**Supplementary Figure S1. Vascular defects in MZ*pak4* morphants.** *fli1a*:eGFP transgenic embryos were injected with control MO (**A**), splice-blocking MOs (**B**), or the combination of the translation- (MO-3) and splice-blocking MOs (**C**). As indicated by the fluorescing endothelial cells, splice morphants exhibited normal vasculature (**B**), while defects were observed in the MZ*pak4* knockdown embryos (**C**), including short intersegmental vessels (ISV; pink arrows), abnormal longitudinal extension of ISV at myoseptum (blue arrow) and partial loss of dorsal longitudinal anastomotic vessels (DLAV). The defects could be substantially rescued by co-injection of *pak4* mRNA (**D**). Combined results from three experiments are summarized by the histogram in (**E**). Images were recorded at 50 hpf.
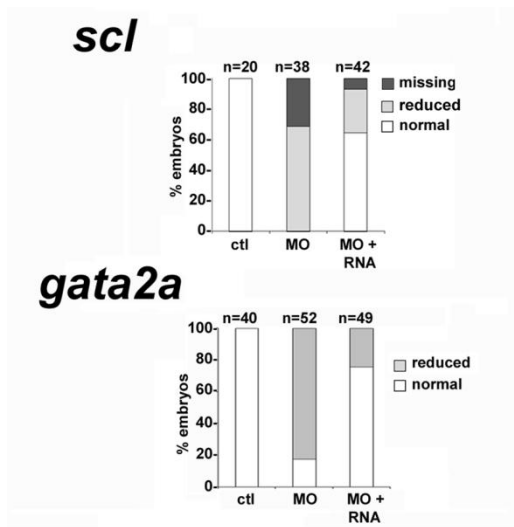
Supplementary Figure S2. MZ*pak4* knockdown-rescue data. Percentages of embryos showing complete or partial rescue for *scl* expression at 7-somite stage (Fig. 3D−I) and *gata2a* expression at 24 hpf (Fig. 3A−C). Six ng of combined MOs was injected in knockdown and rescue embryos while 800 pg *pak4* mRNA was co-injected in rescue samples.

Supplementary Figure 3
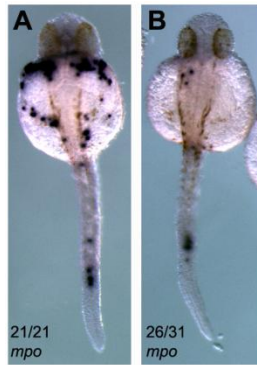
Supplementary Figure S3. Expression of *mpo* in MZ*pak4* morphants. At 30 hpf, the number of *mpo*-expressing cells was significantly reduced in the MZ*pak4* knockdown embryos (**B**) compared to the uninjected control (**A**). Numbers refer to frequency of observed phenotype.
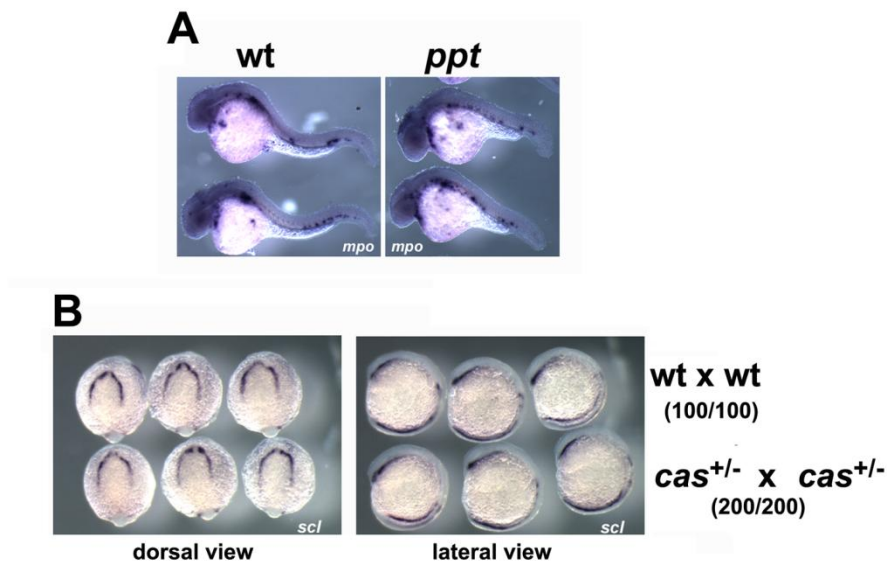
Supplementary Figure S4. Primitive hematopoiesis is not affected by convergent extension defects or disruption of signaling from endoderm. (A) In situ hybridization with *mpo* in wild type (wt) and *pipetail* mutants (*ppt, wnt5a*) at 30 hpf. Lateral views shown. **(B)** In situ hybridization with *scl* in wild type (wt) and embryos from an in-cross of heterozygous *casanova* mutants (*cas^{ta56}*; *sox32*) at 7-somite stage. Dorsal/anterior (left) and lateral (right) views are shown. Data in parentheses indicate that 100% of the *cas* in-cross gave normal *scl* expression.