



S2 Fig. Injection of human *paxillin* mRNA rescues the cardiac phenotype of MO1-Paxillin morphants. (A-C) Lateral view of MO1-*paxillin* (A) and MO1-*paxillin*+human *paxillin* mRNA (B) injected embryos at 72 hpf. (C) Bar graphs compare average of rescued embryos after co-injection of human *paxillin* mRNA compared to MO1-*paxillin* injected embryos (n=3; * $P=0.0073$). **(D)** Fractional shortening (FS) measurement of rescued embryos compared to Paxillin morphant embryos at 72 hpf (n=5). **(E, F)** Lateral view of a (E) 5bp-mismatch MO (MO2-control) and (F) MO2-*paxillin* splice MO injected embryos at 72 hpf. The heart failure phenotype of MO2-*paxillin* splice morphants was identical to that of embryos injected with translation blocking *paxillin* MO (MO1-*paxillin*). **(G)** Bar graphs compare average of affected embryos after injection of MO2-*paxillin* compared to 5bp-mismatch MO (MO2-control) injected embryos at 72 hpf (n=3; * $P=0.0001$). **(H)** FS measurements of Paxillin morphant ventricles at 48, 72 and 96 hpf (n=5 individuals per time point). **(I)** RT-PCR of control- and MO2-*paxillin*-injected embryos. Injection of MO2-*paxillin* caused partial and whole skipping of exon 2 (298 bp) leading to premature termination of Paxillin protein translation. Wild-type *paxillin* RNA was severely reduced in Paxillin morphants (527 bp product). **(J)** Western blot analysis of control- (MO1-control) and MO1-*paxillin*-injected embryos demonstrated high efficacy of MO-mediated *paxillin* gene knock-down. For each sample 50 embryos were pooled and 20 μ g of protein lysate were loaded per lane. *In vitro* translated (IVT) zebrafish Paxillin protein was used as positive control.