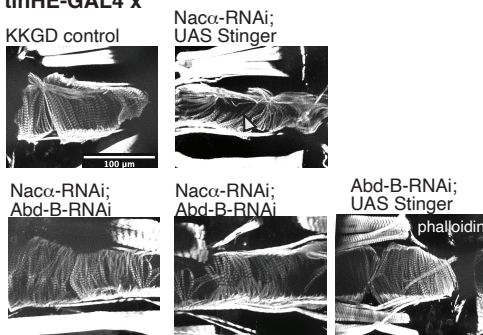
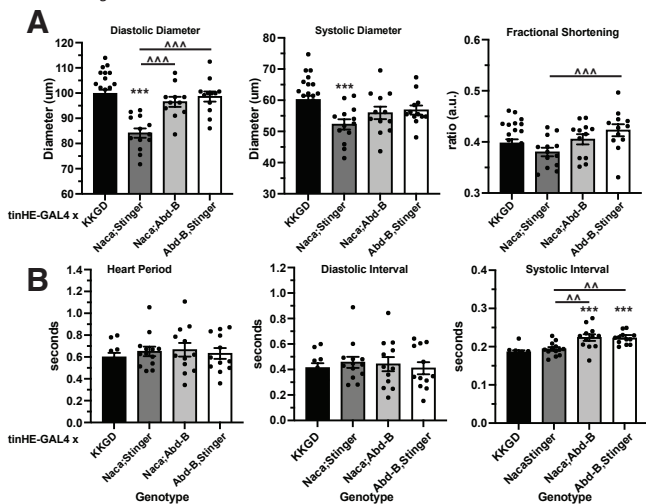


Supplemental Figure 7

Rescue using tinHEGAL4



SUPPLEMENTAL FIGURE 7: *Nacα* interactions with *Abd-B* to regulate heart function and structure.

Testing interaction of *Nacα* and Hox gene *Abd-B* using the cardiac specific tinHE-GAL4 driver by **A**, functional, **B**, temporal and **C**, structural assessment. **A**, Knockdown (KD) of *Nacα* (combined with UAS-Stinger::GFP to control for UAS binding sites) using tinHE-GAL4 caused a decrease in both diastolic and systolic diameters that produced a slight but not significant decrease in fractional shortening. KD of *Abd-B* (combined with UAS-Stinger::GFP) did not produce significant changes in fractional shortening or diameters compared to control but fractional shortening and diastolic diameters were significantly higher compared to *Nacα*;Stinger genotype. Combined knockdown of *Nacα* and *Abd-B* produced heart parameters that were not different to controls but recapitulated heart function produced by *Abd-B* KD alone, suggesting that the heart function was rescued. **B**, Temporal parameters were unchanged with *Nacα*-RNAi expression. KD of *Abd-B* lengthened systolic interval compared to controls. Combined *Nacα* and *Abd-B* KD displayed longer systolic intervals similar to *Abd-B* KD alone, suggesting a rescue. **C**, Phalloidin staining of select genotypes. Compared to controls, *Nacα* knockdown disrupted circumferential fiber organization creating gaps in the matrix (similar to Figure 2I). KD of *Abd-B* did not significantly alter circumferential fiber organization. Combined knockdown of *Nacα* and *Abd-B* (2 examples shown) improved circumferential fiber organization compared to *Nacα* knockdown alone. * vs control KKGD. ^ compared to *Nacα*;Stinger. * p<0.05, ** p<0.01, *** p<0.001.