Figure S1. Ubiquitous scox knock down phenocopies mutant phenotype.

(A) Representative examples of 3rd instar larval sizes (upper panel) and measurement of their length (lower panel) in controls (Or-R, Da::G4 and UAS-scox) and ubiquitous scox KD (Da-Gal4>scox). scox KD causes developmental arrest at early stage 3 and small sized larvae. Values are means ± S.E. n=8-12. Error bars indicate SEM (***p<0.001).

(B) Complex IV and I enzymatic activities in purified mitochondria from 3rd instar larvae normalized for Citrate Sintase activity as an indicator of mitochondrial mass. n=2. Ubiquitous scox KD causes mitochondrial Complex IV deficiency but has effect on not Complex I activity.
Cardiac-specific *scox* knockdown does increase the percentage of long diastoles

(A) Heart rate and, (B) % of long diastoles were measured for hearts from 1 and 2 week old control (*TinCΔ4::Gal4* and *UAS-scox*) and *scox* KD (*TinCΔ4-Gal4>scox* and *TinCΔ4-Gal4/+>scox/+*) flies. Cardiac-specific *scox* KD hearts exhibited a decreased Heart rate and higher % of long diastoles compared to controls. In all cases the *scox* knockdown phenotype is more severe in 2-week-old flies. Significance was determined using a 1-way ANOVA and Tukey multiple comparisons post-hoc test. Differences are relative to the *TinCΔ4::Gal4* control. Error bars indicate SEM (**p<0.001**). n= 20 to 40 flies per genotype.
Figure S3. Cardiac-specific scox knockdown does not disrupt pupal cardiac development. Confocal images of pupae hearts from control (TinCA4::Gal4) and scox KD hearts stained with Phalloidin-Alexa Fluor 594 at 10X optical magnification. scox KD does not affect pupal heart structure.
Figure S4. *dp53* over-expression in *Surf1* KD hearts does not affect heart structure.

(A) *Surf1* mRNA levels measured by qRT-PCR in 1-week-old adult hearts from *Surf1* KD (*TinCA4-Gal4>Surf1i*) and control (*TinCA4::Gal4*) flies. Relative expression of *Surf1* in adult hearts was normalized to *RpL10* expression. *TinCA4::Gal4* expression level was set as one. Compared to control, cardiac-specific *Surf1* KD hearts showed a reduction in *Surf1* mRNA levels close to 50%. Values are displayed as mean ± SEM. Statistical significance was determined by unpaired, Student’s two-tailed t test: *p < 0.05. n= 6-7 per genotype. (B) ROS production measured as DHE staining in hearts from control (*TinCA4::Gal4*) and cardiac-specific *Surf1* knockdown 2-week old adult hearts. DHE staining is enhanced in cardiac-specific *Surf1* KD compared to controls. Arrows indicate DHE staining in *Surf1* KD cardiomyocyte nuclei. (C) Confocal images of 1-week old adult hearts stained with Phalloidin-Alexa Fluor 594 to identify actin filaments at 10X optical magnification. Hearts from control (*TinCA4::Gal4*), cardiac-specific *Surf1* KD (*TinCA4-Gal4>Surf1i*) and cardiac-specific *dp53* OE alone (*TinCA4-Gal4/+/dp53/+) or in a *Surf1* KD background (*TinCA4-Gal4/+/dp53/Surf1i/dp53*) flies are shown. Cardiac-specific *dp53* OE itself causes myofibrillar disorganization which is fully rescued by *Surf1* KD (bottom panels). (D) *dp53* mRNA levels measured by qRT-PCR in 1-week-old adult hearts from *Surf1* KD (*TinCA4-Gal4>Surf1i*) and control (*TinCA4::Gal4*) flies. Relative expression of *dp53* in adult hearts was normalized to *RpL10* expression. *TinCA4::Gal4* expression level was set as one. *dp53* expression is not altered by cardiac-specific *Surf1* KD. Values are displayed as mean ± SEM. Statistical significance was determined by unpaired, Student’s two-tailed t test. n= 6-7 per genotype.
Figure S5. Reaper expression in scox KD hearts exacerbates degeneration.

(A) Confocal images of 2-week old adult hearts stained with Phalloidin-Alexa Fluor 594 to identify actin filaments at 10X optical magnification and (B) of abdominal segments 3 and 4 (A3 and A4) at 25X optical magnification (2X ZOOM). Cardiac-specific Reaper OE alone (TinCΔ4Gal4>Rpr) or together with scox interference (TinCΔ4Gal4>Scox/Rpr) are shown. Cardiac-specific Reaper overexpression exacerbates heart morphological defects caused by scox KD.
Figure S6. Heart-specific scox KD in a dp53 null background rescues heart structure phenotype.

(A) Fluorescent-phalloidin staining of 2 week old adult dp53[5A-1-4], dp53[5A-1-4], scoxi, dp53[5A-1-4], TinCΔ4::Gal4 and dp53[5A-1-4], TinCΔ4>scoxi hearts at 10X optical magnification and (B) of abdominal segments 3 and 4 (A3 and A4) at 25X optical magnification (2X ZOOM). Cardiac-specific scox KD in a dp53 null background rescues heart structure phenotype caused by scox KD alone.
Figure S7. Inhibition of apoptosis by p35 and Diap1 overexpression in scox KD hearts rescues structural phenotype.

(A) Confocal images of 2 week old adult hearts at 10X optical magnification and (B) of abdominal segments 3 and 4 (A3 and A4) at 25X optical magnification (2X ZOOM). Adult hearts are stained with Phalloidin-Alexa Fluor 594. Cardiac-specific overexpression of p35 (TinCΔ4-Gal4>p35) and Diap1 (TinCΔ4-Gal4>Diap1) alone and in a scox KD background (TinCΔ4-Gal4>scox/p35 and TinCΔ4-Gal4>scox/Diap1) are shown. Cardiac-specific overexpression of either p35 or Diap1 rescues the structural defects observed in cardiac-specific scox KD alone.