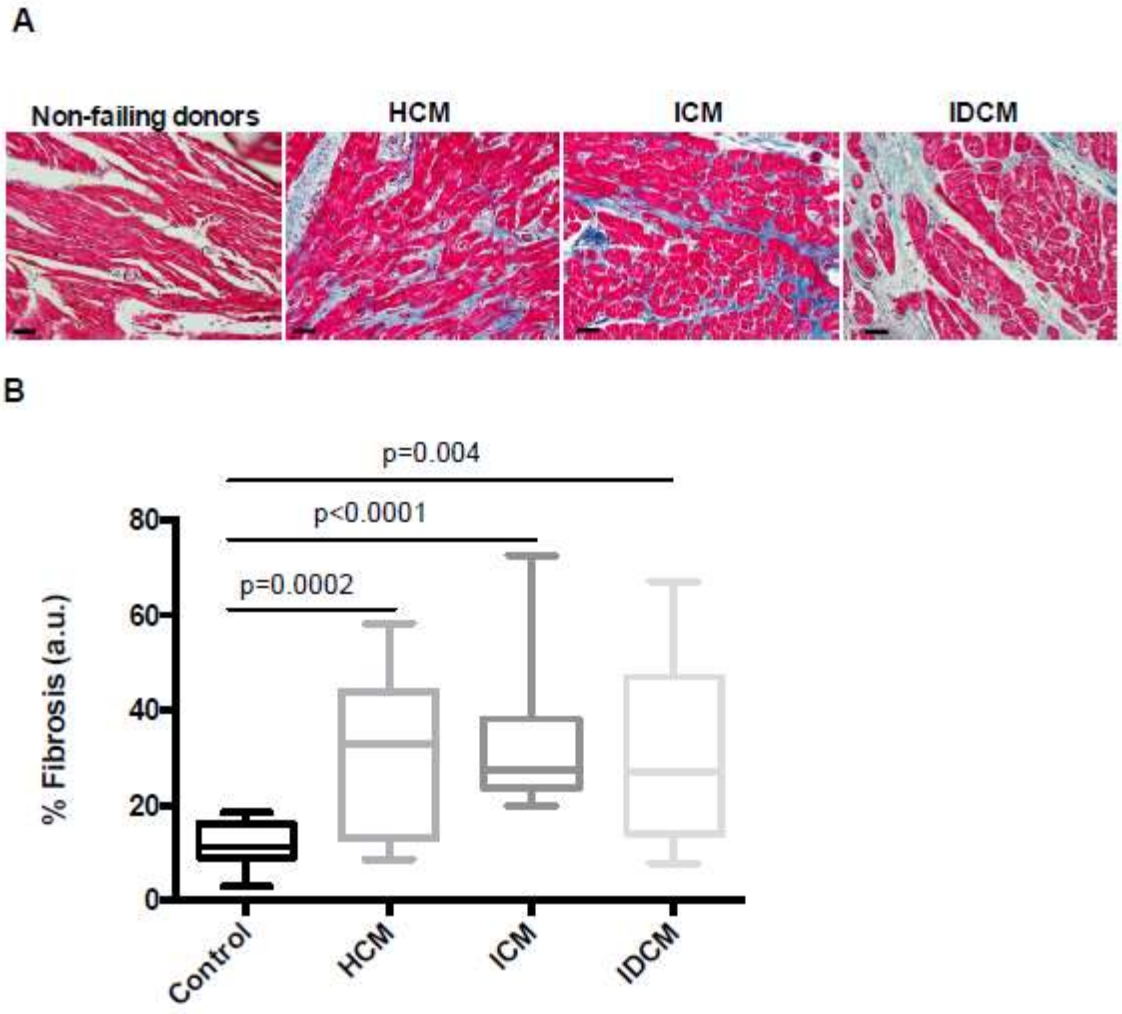
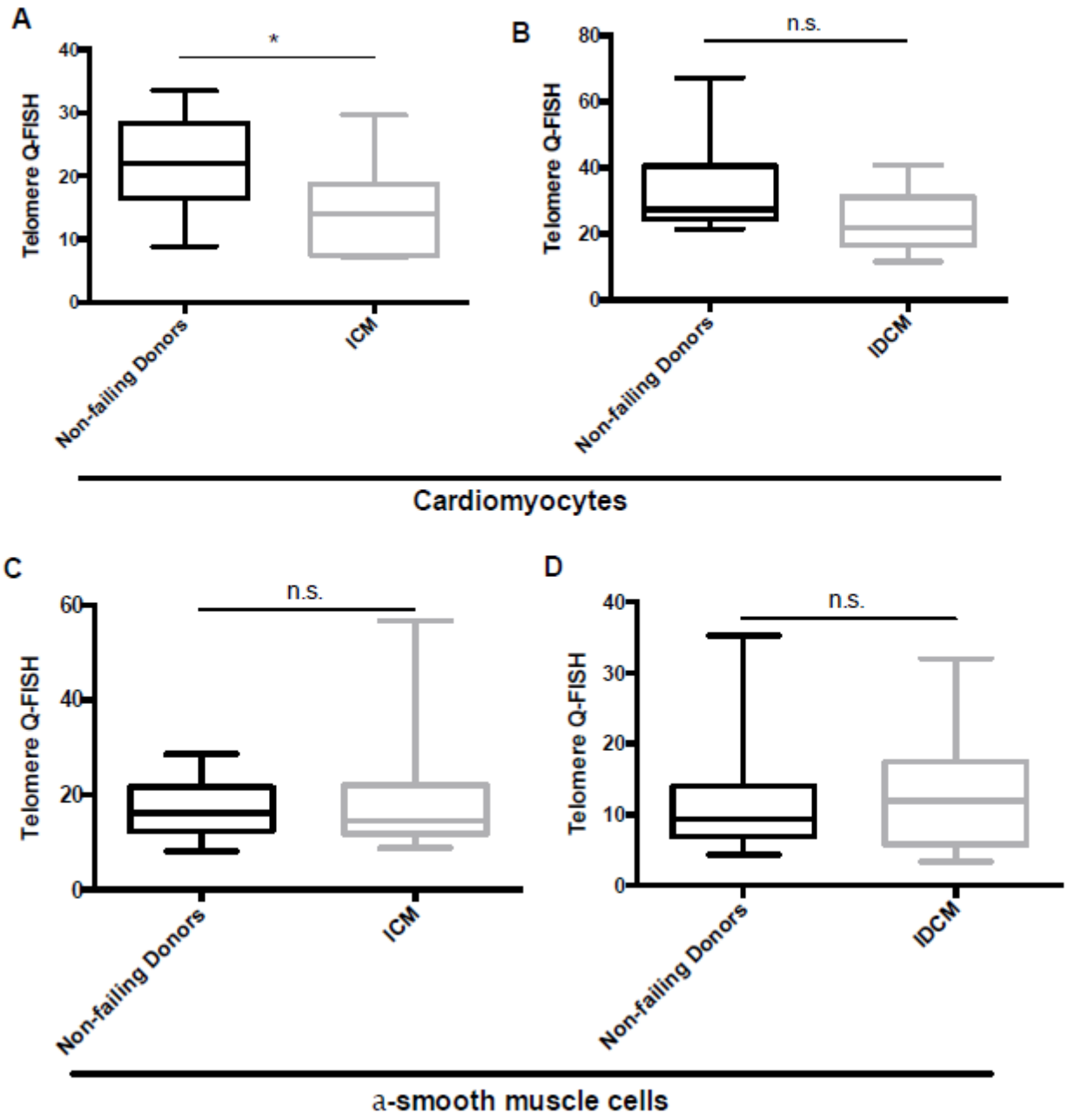
SUPPLEMENTAL MATERIAL

Figure S1. Increased fibrosis in diseased cardiac tissues.



A. Representative trichrome staining of cardiac tissues from non-failing donors, hypertrophic cardiomyopathy (HCM), ischemic cardiomyopathy (ICM) and idiopathic dilated cardiomyopathy (IDCM) patients. **B.** Calculated fibrosis boxplots graphs are presented as % of blue area (fibrotic tissue) over whole tissue area. A total of n=26 non-failing donors, n=17 HCM, n=9 ICM and n=11 IDCM patients' samples were analyzed. Statistical comparison between non-failing donors and HCM, ICM and IDCM show significance of difference (Kruskal-Wallis, $p=0.0002$, $p<0.0001$ and $p=0.004$, respectively).

Figure S2. Telomere length measurements in ischemic (ICM) and idiopathic dilated (IDCM) cardiomyopathies.



Left ventricle (LV) tissues from non-failing donors (black, n=9) and patients with ICM (grey, n=9) (**A** and **C**) and IDCM (grey, n=11) (**B** and **D**) were subjected to quantitative fluorescence *in situ* hybridization (Q-

FISH) analysis. Significant telomere shortening occurs in patient cardiomyocytes (**A**) ICM (Mann-Whitney, * p=0.041) but not in (**B**) IDCM (Mann-Whitney, p=0.101), while α-smooth muscle-positive cells from (**C**) ICM (Mann-Whitney, p=0.578) and (**D**) IDCM (Mann-Whitney, p=0.922) show comparable telomere lengths with non-failing donors. The number of nuclei (N) scored per group is shown in Table 2.