Figure S1. MEF Cultures Are not Contaminated by Cardiomyocytes or Cardiac Progenitor Cells

(A) Immunocytochemistry for vimentin, collagen1 (Col1), Nkx2.5, α-actinin, cTnT, and α-MHC-GFP, with DAPI staining in MEFs.

(B) Immunostaining for Nkx2.5, cTnT, α-actinin, and DAPI in murine neonatal cardiomyocytes provided a positive control for immunocytochemistry. High-magnification views in insets show sarcomeric organization.

(C) The percentage of vimentin+, Col1+, Nkx2.5+, α-actinin+, cTnT+, and α-MHC-GFP+ cells in MEFs (n = 3).

(D) Relative mRNA expression of Actc1, Actn2, Ryr2, Tnnt2, Cacna1c, Gja1, Postn, Snail, Fn1, Col1a1, and Ddr2 in MEFs compared to hearts (n = 3).

(E) FACS analyses for cTnT expression in MEFs and mouse hearts. MEFs did not include cTnT+ cardiomyocytes.

(F) FACS analyses for transfection efficiency of miRNA mimics (green-miR).

(G) FACS analyses for αMHC-GFP+ cells. Cells were analyzed 1 week after GMT transduction or miRNA transfection.

(H) FACS analyses for transduction efficiency of pMXs-GFP with and without miRNA mimics in MEFs. Addition of miRNA did not augment the transduction efficiency of pMXs-GFP.

(I, J) FACS analyses for αMHC-GFP+ and cTnT+ cells 1 week after GMT/miR-133 transduction with and without the JAK inhibitor 1 (JAK-I). Quantitative data are shown in (J) (n = 3).

All data are presented as means ± SEM. **, P < 0.01 vs. relevant control. Scale bars, 100 µm.