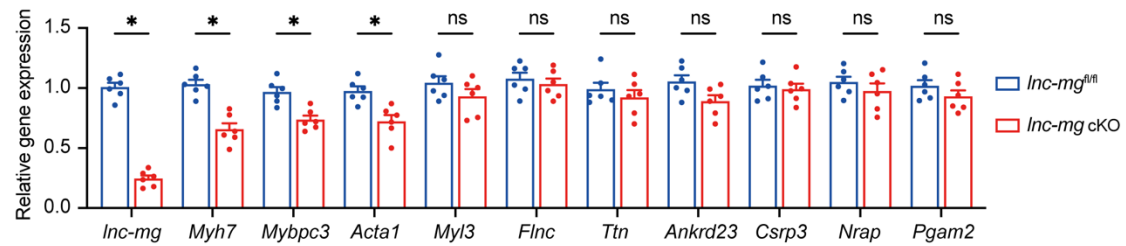
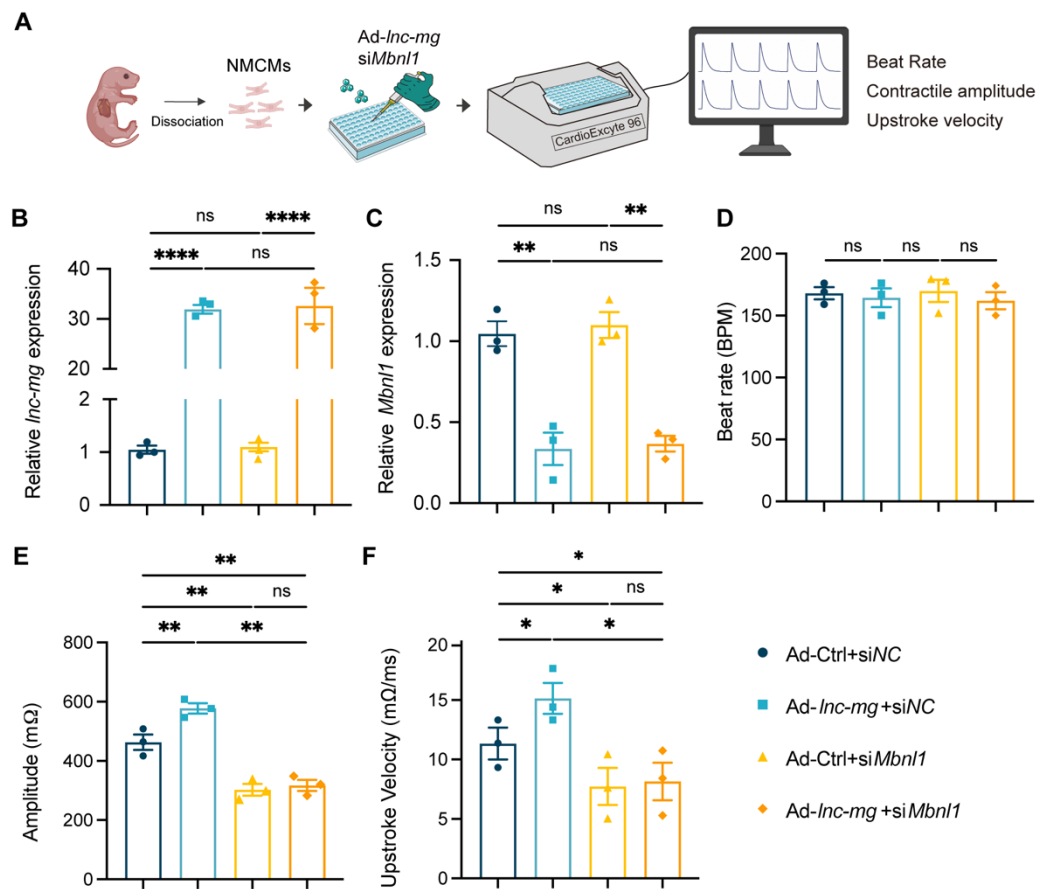


Supplementary Figures

Lnc-mg regulates cardiomyocyte contraction and promotes functional recovery after ischemic injury



Supplementary Figure S1. RT-qPCR validation of representative contractility- and myofibril-related genes identified by RNA-seq. Relative expression of the indicated genes in *lnc-mg*^{fl/fl} and *lnc-mg* cKO mouse hearts. *Myh7*, *Mybpc3*, and *Acta1* exhibited statistically significant reductions in *lnc-mg* cKO cardiomyocytes. Six independent samples were analyzed (n = 6 biological replicates per group). Each sample was measured in technical triplicate, and the technical replicates were averaged before statistical analysis. Statistical comparisons were performed using multiple unpaired t-tests. Data are presented as mean ± SEM. ns, not significant; * *P* < 0.05.



Supplementary Figure S2. MBNL1 is required for *lnc-mg*-mediated regulation of cardiomyocyte contractile function. (A) Schematic illustration of the experimental procedure. Neonatal mouse cardiomyocytes (NMCs) were transduced with Ad-*lnc-mg* or Ad-Ctrl and treated with either siMbnl1 or siNC, followed by contractile-function assessment using the CardioExcyte 96 system. (B, C) RT-qPCR validation of *lnc-mg* overexpression (B) and *Mbnl1* knockdown (C). (D-F) Quantification of spontaneous beat rate (D), impedance-derived contractile amplitude (E), and upstroke velocity (F). Three independent NCMC isolations were analyzed (n = 3 biological replicates per condition), with 24 technical replicate wells included per condition within each isolation. Each dot in panels B-F represents one independent NCMC isolation. Data are presented as mean $\pm$ SEM and were analyzed using two-way repeated-measures ANOVA followed by Šídák's multiple-comparisons test. ns, not significant; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.0001$. Panel A was created with BioRender.com [Created in BioRender. Jia, S. (2026) <https://BioRender.com/bz1ea4m>].