SUPPLEMENTAL MATERIAL

Detailed Methods

Generation of Mice: *Ttn*$^{\Delta IAjxn}$ and *Rbm20*$^{\Delta RRM}$ mice previously described[1, 2] were bred to generate homozygous *Ttn*$^{\Delta IAjxn}$ mice on that were heterozygous for WT RBM20 protein on a C57BL/6J background. In our studies we used mice 4 months old and male, unless indicated otherwise (see Figure S1 for breeding details). All experiments were approved by the University of Arizona Institutional Animal Care and Use Committee (IACUC) and followed the U.S. National Institutes of Health *Using Animals in Intramural Research guidelines for animal use*.

Skinned Cell Mechanics: Experiments were conducted in accordance with the Guide for the Care and Use of Laboratory Animals, and all protocols were approved by University of Arizona's Institutional Animal Care and Use Committee. Cells were isolated as described previously[3]. Briefly, mice were heparinized (1,000 U/kg, i.p.) and euthanized by cervical dislocation under isoflurane. The heart was removed and cannulated via the aorta with a blunted 21-gauge needle for retrograde coronary perfusion. The heart was perfused for 4 min with perfusion buffer ([in mmol/L] 90 NaCl, 34.7 KCl, 0.6 KH2PO4, 0.6 Na2HPO4, 1.2 MgSO4, 12 NaHCO3, 10 KHCO3, 10 HEPES, 10 taurine, 5.5 glucose, 5 BDM, 20 Creatine, 5 Adenosine and 5 Inosine, pH 7.4), followed by digestion buffer (perfusion buffer plus 0.05 mg/ml Liberase TM research grade; Roche Applied Science, and 13 µM CaCl2) for 20 min. When the heart was flaccid, digestion was halted and the heart was placed in myocyte stopping buffer (perfusion buffer plus bovine calf serum 0.08 [BCS]/ml and 8 µM CaCl2) with protease inhibitors ([in mmol/L] 0.4 Leupeptin, 0.1 E64, and 0.5 PMSF (Peptides International,Sigma-Aldrich)). The left ventricle was cut into small pieces, and the rest of the heart was discarded. The small pieces of left ventricle were triturated several times with a transfer pipette and then filtered through a 300µm nylon mesh filter. **Passive stress measurement in skinned cardiomyocytes.** Mouse cardiomyocytes, isolated as explained above, were skinned for 7 min in relaxing solution ([in mmol/L] 40 BES, 10 EGTA, 6.56 $MgCl_2$, 5.88 Na-ATP, 1.0 DTT, 46.35 K-propionate, 15 creatine phosphate, pH 7.0) with protease inhibitors ([in mmol/L] 0.4 leupeptin, 0.1 E64 , and 0.5 PMSF) and 0.3% Triton X-100 (Ultrapure; Thermo Fisher Scientific). Cells were washed extensively with relaxing solution pCa 9 and stored on ice. Skinned myocytes were used for mechanic studies within 48 h after time of cell isolation.

Myocyte suspension was added to a room temperature flow-through chamber mounted on the stage of an inverted microscope (Diaphot 200; Nikon). Skinned myocyte was glued at one end to a force transducer (Model 406A or 403A, Aurora Scientific). The other end was bent with a pulled glass pipette attached to micromanipulator so that the myocyte axis aligned with the microscope optical axis and cross sectional area (CSA) was measured directly. The cross sectional images of skinned cells were analyzed by ImageJ 1.41 software (National Institutes of Health) and were used to convert measured force to stress and for cell dimension study (Fig S4d). Then, the free end of the cell was glued to a servomotor (Model 308B, Aurora Scientific) that imposes controlled stretches. Sarcomere length (SL) was measured with a MyocamS and SarcLen acquisition module (IonWizard 6.2, IonOptix Co, MA) attached to a computer. To correct for ~20% lattice expansion during skinning process[4], CSA of skinned cells were divided by a correction factor of 1.44. Passive stress was measured in relaxing solution pCa 9 with protease inhibitors at room temperature. Cells were stretched from slack length at a speed of 1 base length/sec to a SL of 2.3 µm for +/+, 2.7 µm for +/- and 3.0 µm for -/-, followed by a 20 sec hold and then a release back to the original length. Recovery time of at least 7 min in between stretches was utilized to prevent memory-effects in subsequent measurements. Data were collected using a custom LabVIEW VI (National Instruments, Austin TX) at a sample rate of 1 kHz. Measured forces were converted to stress (force/unit undeformed CSA). The stress during the 1 base length/sec stretch was plotted against the SL and fitted with a monoexponential curve to derive stress-SL relationships.

Echocardiography: A Vevo 2100 High Resolution Imaging System (Visual-Sonics, Toronto, Canada) was used with the model MS550D scan head designed for murine cardiac imaging. Care was taken to avoid animal contact and excessive pressure which could induce bradycardia during conscious scanning. Imaging was performed at a depth setting of 11 mm. Images were collected and stored as a digital cine loop for off-line calculations. Mice were consciously echoed while scruffing the skin at the nape of the neck and a standard short axis (M-mode) cine loop was recorded at the level of the papillary muscles to assess chamber dimensions (LV systolic and diastolic dimensions (LVDs, LVDd)) posterior and anterior wall thickness (WTH), and cardiac function via Fractional

Shortening (%FS). Functional calculations were obtained according to American Society of Echocardiography guidelines. To investigate diastolic function we performed Doppler echo on anesthetized mice. Mice were anesthetized and ventilated with 2% isoflurane using a nose cone and body temperature was maintained at 37^{o}C. Following anesthetic induction, the mouse was placed in dorsal recumbence on a heated platform for echocardiography. Images were collected and stored as a digital cine loop for off-line calculations. The left atrial dimension was measured in the long-axis view directly below the aortic valve leaflets. Passive LV filling peak velocity, E (cm/sec), and atrial contraction flow peak velocity, A (cm/sec), were acquired from the images of mitral valve Doppler flow from 4-chamber apical views, according to American Society of Echocardiography guidelines. The heart rate of animals was maintained in the range of 450 ± 25 bpm for Doppler studies.

Pressure-Volume Analysis: In vivo pressure-volume analysis was performed in mice using a SciSense Advantage Admittance Derived Volume Measurement System and 1.2F catheters with 4.5 mm electrode spacing (SciSense, London, Ontario, Canada). Mice were anesthetized and ventilated with 1.5% isoflurane using a SAR-1000 Small Animal Ventilator (CWE). Body temperature under anesthesia was maintained at 37°C using a TC-1000 Temperature Controller (CWE). A lateral incision through the skin and muscle was made below the ribcage and the diaphragm was cut in order to expose the apex of the heart. A small puncture was made in the apex of the left ventricle using a 28G needle and the 1.2F catheter was inserted into the LV. Baseline functional parameters were assessed during a pause in ventilation in order to avoid respiratory influences. For load-independent indices, including the end-systolic and end-diastolic pressure-volume relationships, the IVC was temporarily occluded to vary the preload conditions. Data acquisition and analysis was performed using LabScribe 2 (iWorx, Dover, NH) and curve fitting was performed with MATLAB (MathWorks, Natick, MA). Diastolic PV data was analyzed using a mono-exponential fit with constant ($P = Ae^{\beta V} + C$) with the exponent (β) reported as the coefficient of stiffness.

Exercise Testing: Mice were tested for maximal running speed using a 6-lane rodent treadmill system (Exer 3/6, Columbus Instruments, Columbus, OH). After an acclimation period during which mice were running at low speed (10 m/min), exercise testing was performed by having mice run at progressively increasing speeds (speed steps of 4 m/min). Maximal speed was determined when the mouse left the treadmill and remained on a shock pad for at least 5 sec.

Quantification of Protein Expression: Flash frozen LV tissues were cut and weighed, then pulverized into a very fine powder using a mortar and pestle in liquid nitrogen. Samples were then placed into a -20°C freezer for 20minutes. 8M urea buffer ([in mol/L] 8 Urea, 2 Thiourea, 0.05 Tris-HCl, 0.075 Dithiothreitol with 3% SDS and 0.03% Bromophenol blue pH 6.8) and 50% glycerol with protease inhibitors ([in mmol/L] 0.04 E64, 0.16 Leupeptin and 0.2 PMSF) were added to the samples (in a 40:40:1 ratio) and solubilized for 10minutes at 60°C. The solubilized samples were centrifuged, aliquoted into smaller volumes, and stored at -80°C.

Titin isoform expression analysis: titin isoform expression analysis was performed via electrophoresis of the solubilized samples on a 1% agarose gel (Hoefer gel system). The gels are loaded with six different volumes of the sample and run at 15mA per gel for 3hours and 20minutes. The protein was then fixed on the gel using a fixing solution, stained with coomassie brilliant blue (Acros organics), then scanned (Epson commercial scanner) in order to capture an image for the analysis of the various Titin isoform bands. The images were analyzed using OneD scan software by determining the integrated optical density of Titin and myosin heavy chain as a function of the various volumes loaded on the gel. The isoform expression ratios of the samples are determined by using the slope of the optical density of the different loading volumes.

Western Blotting: The solubilized samples were run on SDS PAGE gels (different percentages depending on target protein). Gels were run for approximately 2 hours then samples were transferred to PDVF membranes (Immobilon, Millipore) using a semi-dry transfer unit (Trans-Blot Cell, Bio-Rad). Transfer amperage was based on area of membrane(s) multiplied by 1.3 and was run for 2.5hours. The membranes were stained with Ponceau

S (Sigma) to visually ensure protein transfer. The Ponceau S stain was later removed by .1M NaOH, and blots were placed in a blocking solution (Odyssey Block) for 1hour to prevent non-specific binding of the antibody. Blocking solution was removed and blots were then soaked in the primary antibody solution overnight. Primary antibody was removed and blots were washed with PBS-tween solution before secondary fluorescent antibody was added. The blots soaked in secondary antibody for 1hour before they were scanned using an Odyssey Infrared Imaging System (Li-COR Biosciences). Images obtained were analyzed using Li-COR Odyssey software.

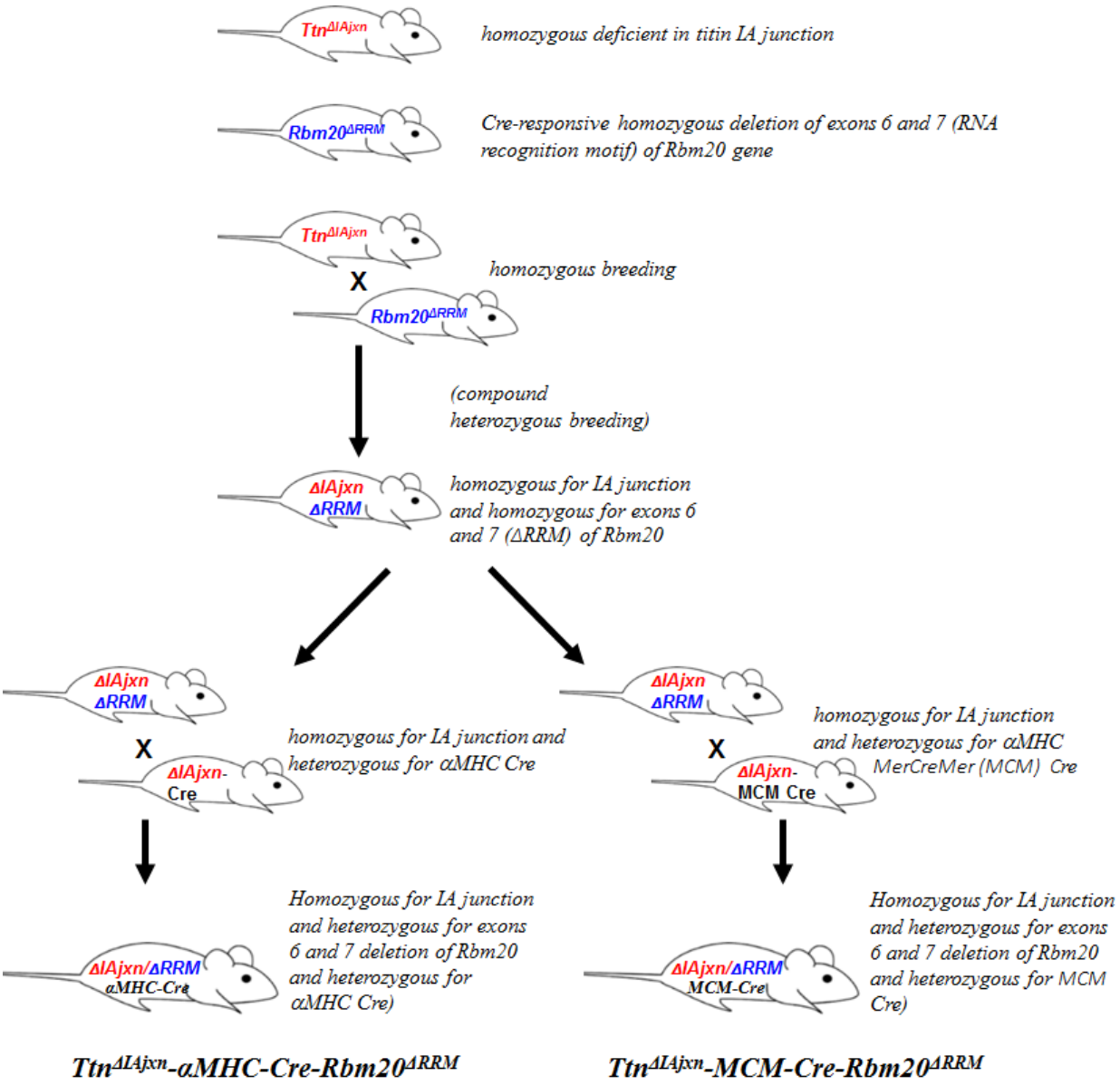
Antibody	Source	Host	Dilution
FHL2	MBL	Rabbit	1:5000
FHL1	Millipore	Rabbit	1:5000
αBCrystallin	Cell Signaling Technology	Rabbit	1:1500
GAPDH	Thermo Pierce	Mouse	1:5000

Transverse Aortic Constriction: For minimally invasive Transverse Aortic Constriction (TAC) surgeries, anesthesia was induced by intraperitoneal (i.p.) injection of ketamine hydrochloride (120 mg/kg) plus xylazine (12 mg/kg) for a depth of anesthesia ~45 min, long enough to finish the surgery. The aortic banding procedure was performed similar to that previously described in [5] with minor adaptations. Briefly, a small incision in the chest cavity was created between the first and second intercostal ribs and the transverse portion of the aorta was bluntly dissected from the surrounding tissue. A curved forceps was then placed under the transverse aorta, 7-0 silk was grasped by the forceps and moved underneath the aorta between the right innominate and left carotid arteries, and a loose double knot was made. A 27-gauge needle with OD 0.42 mm was delivered through the loose double knot and placed directly above and parallel to the aorta. The loop was then tied around the aorta and needle and secured with the second knot (this was done very quickly, to minimize ischemia). The needle was immediately removed to provide a lumen with a stenotic aorta. Following the surgery, all layers of muscle and skin were closed with 6-0 continuous absorbable and nylon sutures, respectively, and the wound was treated with Betadine. Immediately after the operation, 0.5 ml of 37°C saline was given intraperitoneally, and a dose of analgesic (SR-buprenorphine, 0.1 mg/kg) was also given subcutaneously. For the sham operation (control) the mice underwent

the identical procedure, except placing of the ligature. The surgical survival rate following TAC was >90% and mortality rate within the first month following TAC was low (<5%). Successful surgical ligation of the transverse aorta, determined by a Doppler flow velocity measurement indicated by a large pressure gradient following constriction. Animals were sacrificed, weighed and cardiac tissue was flash frozen in liquid nitrogen (-80^{o}C) for further analysis.

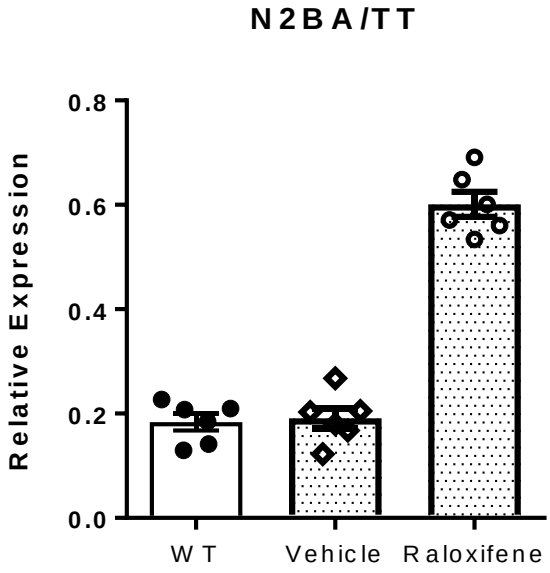
Myocyte Cross Section Area: Cross-sectional area (CSA) of cardiomyocytes was performed as described[6] with minor modifications. Hearts in diastole were removed from sham and TAC mice and the LV tissue was covered with OCT (Tissue-Tek) and frozen with liquid nitrogen–cooled isopentane and stored at −80°C. 8-µm thick sections were collected on VWR glass microscope slides and stored at −20°C overnight. The cross-sections were skinned and stained with anti-Laminin (Sigma) to demarcate cell borders and DAPI (Vector Laboratories) to stain nuclei. Images were collected on an Axio Imager M.1 microscope (Carl Zeiss) using an Axio Cam MRC (Carl Zeiss). CSAs were measured using the ImageJ program (National Institutes of Health) and ther borders of stained cells that included nuclei were traced manually. The CSA of 100 myocytes from the myocardium of the left ventricle (6 mice per group) were collected from each sample using 4 randomly selected sections.

Online Figure I



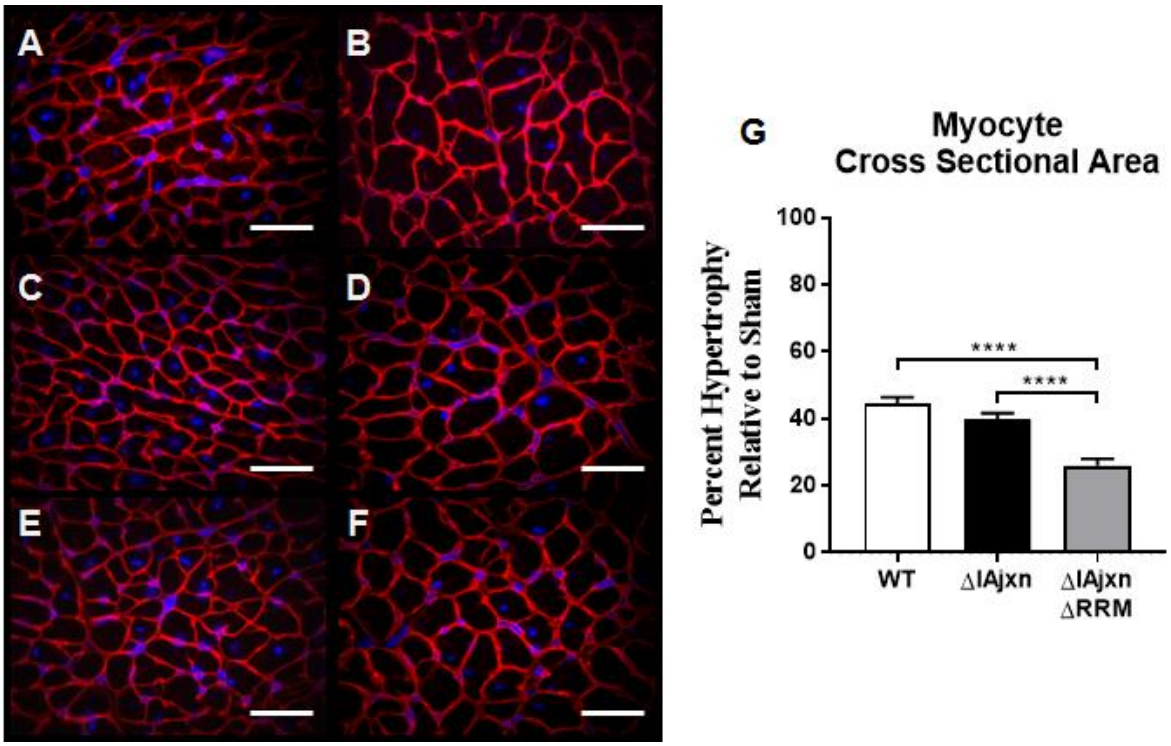
Online Figure I: Schematic explaining breeding of *Ttn*$^{\Delta IAjxn}$ to *Rbm20*$^{\Delta RRM}$ mice[1, 2]. Generation of homozygous *Ttn*$^{\Delta IAjxn}$ mice with constitutive (bottom left) or inducible (bottom right) heterozygous reduction of functional Rbm20.

Online Figure II



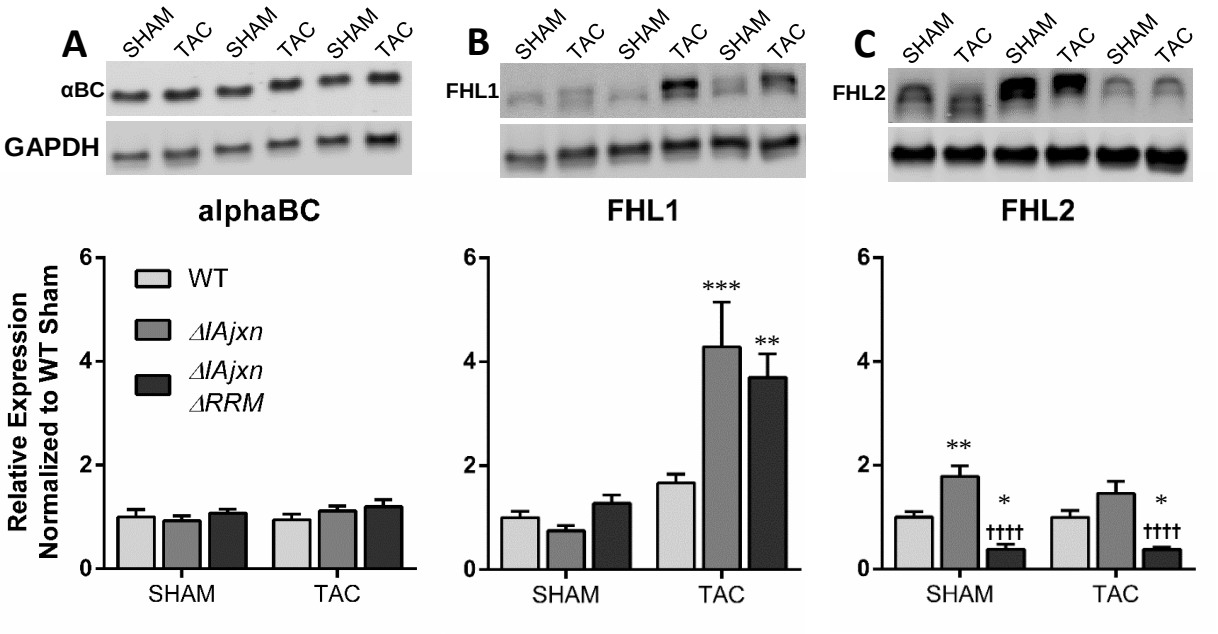
Online Figure II: Titin expression in WT and *Ttn*$^{\Delta IAjxn}$-MCM-Cre-*Rbm20*$^{\Delta RRM}$ 28 days after injection with either vehicle or raloxifene. Raloxifene triggers expression of large N2BA isoforms that comprise ~60% of total titin.

Online Figure III



Online Figure III: LV myocyte cross-sectional area analysis in LV of mice that underwent TAC. A) WT sham; B) WT TAC; C) *ΔIAjxn* sham; D) *ΔIAjxn* TAC; E) *ΔIAjxn/ΔRRM* sham; F) *ΔIAjxn/ΔRRM* TAC. G) Percent CSA increase (hypertrophy) relative to sham controls. Data shown as mean ± SEM. Statistical significance calculated by two-way ANOVA with Bonferroni correction: **** $p<0.0001$ as indicated. (*Ttn*$^{\Delta IAjxn}$- αMHC-Cre-*Rbm20*$^{\Delta RRM+/-}$ mice were used.)

Online Figure IV



Online Figure IV: Expression of N2B element localized binding proteins and their response to TAC. A) αB crystallin; B) FHL1; C) FHL2. Data shown as mean ± SEM. Statistical significance calculated by two-way ANOVA with Bonferroni correction: * p<0.05; ** p<0.01; *** p<0.001; **** p<0.0001; (versus WT ****, versus *ΔIAjxn* ††††). (*Ttn*$^{\Delta IAjxn}$- αMHC-Cre-*Rbm20*$^{\Delta RRM+/-}$ mice were used.)

Online Table I

	WT	*ΔIAjxn*	*ΔIAjxn/ΔRRM*
n	6	7	7
N2BA/N2B	0.27 ± 0.02	0.23 ± 0.02	7.8 ± 1.1 ****,††††
N2BA/TT	0.18 ± 0.02	0.16 ± 0.01	0.82 ± 0.008 ****,††††
N2B/TT	0.67 ± 0.02	0.70 ± 0.02	0.11 ± 0.01 ****,††††
TT/MHC	0.19 ± 0.02	0.27 ± 0.03 *	0.28 ± 0.03 *
N2BA/MHC	0.03 ± 0.003	0.04 ± 0.002	0.22 ± 0.02 ****,††††
N2B/MHC	0.12 ± 0.01	0.19 ± 0.01 ***	0.03 ± 0.005 ****,††††
T2/T1	0.16 ± 0.05	0.15 ± 0.03	0.09 ± 0.01
MHC (OD/mg)	123.6 ± 12.0	109.6 ± 3.8	136.1 ± 13.0
TT (OD/mg)	24.0 ± 4.9	29.4 ± 1.7	39.1 ± 7.3

Online Table I: Titin isoform Analysis. Data shown as mean ± SEM; Abbreviations: *ΔIAjxn*: (*Ttn*$^{\Delta IAjxn}$); *ΔRRM* (*Rbm20*$^{\Delta RRM+/-}$); TT: total titin; MHC: myosin heavy chain; T1: sum of titin isoforms N2BA and N2B; T2: titin degradation product. Statistical significance calculated with one-way ANOVA with Bonferroni correction: * p<0.05; ** p<0.01; *** p<0.001; **** p<0.0001; (versus WT ****, versus *Ttn*$^{\Delta IAjxn}$ ††††).

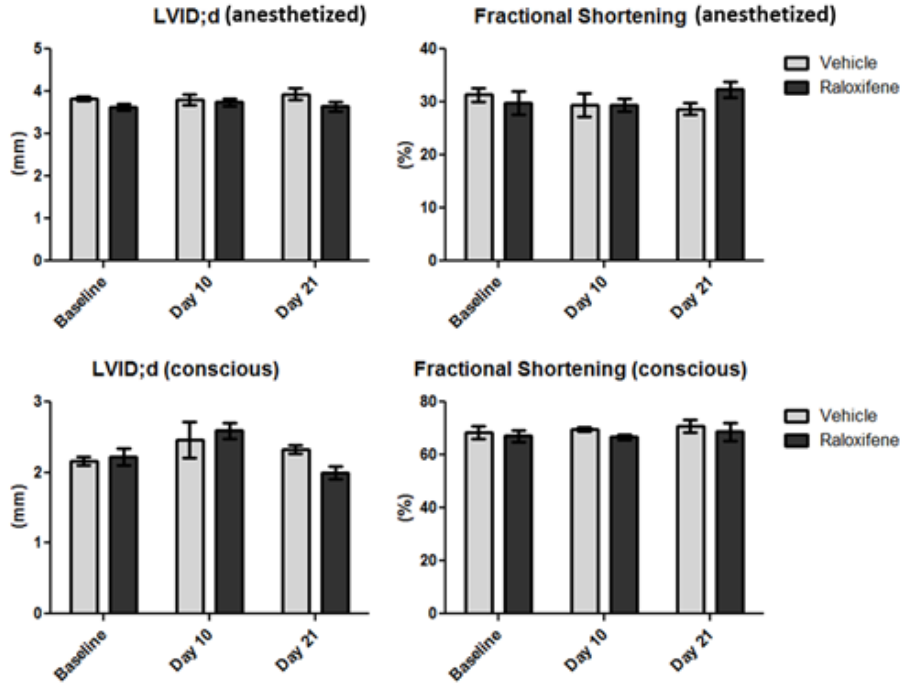
Online Table II

	WT	*ΔIAjxn*	*ΔIAjxn ΔRRM*
n	15.0	15.0	16.0
BW (g)	26.7 ± 0.6	28.3 ± 0.7	28.1 ± 0.6
HW (mg)	124.3 ± 2.2	131.2 ± 3.0	130.1 ± 2.5
LV (mg)	84.9 ± 1.3	89.5 ± 2.1	86.5 ± 1.6
RV (mg)	25.2 ± 0.7	27.8 ± 0.8	28.7 ± 0.9 **
ATR (mg)	7.1 ± 0.3	7.0 ± 0.3	7.4 ± 0.3
HW/BW (mg/g)	4.7 ± 0.1	4.6 ± 0.1	4.6 ± 0.1
LV/BW (mg/g)	3.2 ± 0.1	3.2 ± 0.1	3.1 ± 0.05
RV/BW (mg/g)	0.9 ± 0.02	1.0 ± 0.02	1.0 ± 0.03
ATR/BW (mg/g)	0.3 ± 0.01	0.3 ± 0.01	0.3 ± 0.01

Online Table II: Tissue morphometry of WT mice, homozygous *Ttn*$^{\Delta IAjxn}$ mice (shown as *ΔIAjxn*) and mice that are homozygous *Ttn*$^{\Delta IAjxn}$ and heterozygous α-MHC-Cre-*Rbm20*$^{\Delta RRM+/-}$ (shown as *ΔIAjxn /ΔRRM).* Data shown as mean ± SEM. Male mice age 3-4 months old were dissected. Abbreviations: *ΔIAjxn*: *Ttn*$^{\Delta IAjxn}$; *ΔRRM:* α-MHC Cre *Rbm20*$^{\Delta RRM+/-}$; BW: body weight; HW: heart weight; LV: left ventricle; RV: right ventricle; ATR: atrium. Statistical significance calculated with one-way ANOVA with Bonferroni correction: ** $p<0.01$ versus WT.

Online Table III

	Baseline		Day 10		Day 21		
	Vehicle	Raloxifene	Vehicle	Raloxifene	Vehicle	Raloxifene	
n	6	5	6	6	6	5	
HR (BPM)	731.9 ± 9.0	709.1 ± 15.6	735.5 ± 11.2	700.3 ± 20.9	732.8 ± 12.2	770.2 ± 17.2	Conscious
V;s (uL)	0.9 ± 0.2	1.2 ± 0.2	1.2 ± 0.3	1.6 ± 0.3	0.8 ± 0.2	0.9 ± 0.4	
V;d (uL)	17.3 ± 1.6	18.9 ± 1.7	23.8 ± 4.4	25.9 ± 3.3	18.5 ± 1.4	14.6 ± 2.3	
SV (uL)	16.4 ± 1.6	17.7 ± 1.6	22.6 ± 4.1	24.3 ± 3.1	17.7 ± 1.3	13.7 ± 2.0	
EF (%)	94.9 ± 1.0	93.8 ± 0.9	95.4 ± 0.4	94.2 ± 0.6	95.8 ± 0.8	94.8 ± 1.4	
FS (%)	68.4 ± 2.4	65.7 ± 2.1	69.7 ± 0.9	66.7 ± 1.2	70.9 ± 2.4	68.8 ± 3.4	
CO (mL/min)	15.8 ± 1.5	17.2 ± 1.4	21.4 ± 4.0	22.3 ± 2.7	17.0 ± 1.3	13.2 ± 1.7	
WT;d (mm)	1.36 ± 0.05	1.28 ± 0.04	1.13 ± 0.07	1.07 ± 0.05	1.31 ± 0.06	1.30 ± 0.03	
WT;s (mm)	1.96 ± 0.05	1.85 ± 0.06	1.88 ± 0.05	1.76 ± 0.04	1.93 ± 0.03	1.87 ± 0.03	
LVID;d (mm)	2.16 ± 0.06	2.31 ± 0.10	2.46 ± 0.25	2.59 ± 0.12	2.33 ± 0.07	2.00 ± 0.09	
LVID;s (mm)	0.72 ± 0.06	0.81 ± 0.07	0.78 ± 0.08	0.88 ± 0.06	0.69 ± 0.09	0.66 ± 0.09	
Eccentricity	1.6 ± 0.1	1.8 ± 0.1	2.3 ± 0.4	2.4 ± 0.2	1.8 ± 0.1	1.6 ± 0.1	
HR (BPM)	529.7 ± 17.2	512.6 ± 17.5	521.9 ± 13.2	459.9 ± 26.0	544.2 ± 9.4	548.3 ± 18.4	Anesthetized
V;s (uL)	25.6 ± 1.6	25.8 ± 2.7	28.0 ± 3.8	27.2 ± 1.9	31.6 ± 3.3	23.0 ± 2.8	
V;d (uL)	63.5 ± 2.4	60.3 ± 2.1	63.5 ± 5.0	63.0 ± 2.4	70.8 ± 5.0	58.8 ± 4.8	
SV (uL)	38.0 ± 1.8	34.5 ± 1.4	35.5 ± 2.4	35.8 ± 1.3	39.2 ± 2.0	35.8 ± 2.3	
EF (%)	59.8 ± 2.0	57.5 ± 3.2	56.7 ± 3.4	57.0 ± 1.8	55.8 ± 1.7	61.3 ± 2.2	
FS (%)	31.4 ± 1.4	29.9 ± 2.2	29.4 ± 2.2	29.4 ± 1.2	28.7 ± 1.1	32.4 ± 1.5	
CO (mL/min)	20.1 ± 1.1	17.7 ± 1.1	18.6 ± 1.4	16.4 ± 0.9	21.2 ± 0.9	19.5 ± 1.1	
WT;d (mm)	0.80 ± 0.03	0.82 ± 0.04	0.81 ± 0.03	0.77 ± 0.03	0.83 ± 0.05	0.78 ± 0.04	
WT;s (mm)	1.19 ± 0.03	1.20 ± 0.07	1.18 ± 0.04	1.08 ± 0.05	1.14 ± 0.06	1.19 ± 0.07	
LVID;d (mm)	3.83 ± 0.05	3.69 ± 0.05	3.81 ± 0.13	3.74 ± 0.09	3.94 ± 0.13	3.65 ± 0.11	
LVID;s (mm)	2.65 ± 0.07	2.64 ± 0.10	2.65 ± 0.16	2.65 ± 0.06	2.89 ± 0.12	2.54 ± 0.11	
Eccentricity	4.9 ± 0.2	4.5 ± 0.3	4.8 ± 0.3	4.9 ± 0.2	4.9 ± 0.4	4.7 ± 0.3	



Online Table III: Conscious and anesthetized echocardiography of raloxifene injected WT mice (age 3 months) at baseline, 10 days and 21 days after completing the injections. Vehicle: DMSO. Treatment: (40 mg/kg) raloxifene injections (x8 days). α-MHC-MCM RBM20 WT mice were used. Abbreviations: HR: heart rate; BPM: beats per minute; EF: ejection fraction; FS: fractional shortening; CO: cardiac output; WT;d: wall thickness in diastole; WT;s: wall thickness in systole; LVIDd: left ventricular internal diastolic diameter; LVIDs; left ventricular internal systolic diameter; Eccentricity: LVDD/WTH Data shown as mean ± SEM; Statistical significance calculated with one-way ANOVA with Bonferroni correction: * $p<0.05$ considered significant. No significant difference between Vehicle and Treatment groups exist.

Online Table IV

	Vehicle	Treatment
n	8	9
Age (days)	133.6 ± 9.0	136.2 ± 8.9
HR (bpm)	675.4 ± 26.3	678.4 ± 33.7
FS (%)	52.7 ± 25.7	50.6 ± 1.9
LVID;d (mm)	2.7 ± 2.7	2.6 ± 0.1
LVID;s (mm)	1.4 ± 0.1	1.3 ± 0.04
LVPW;d (mm)	1.1 ± 0.04	1.1 ± 0.04
LVPW;s (mm)	1.4 ± 0.1	1.4 ± 0.1
LVDD/WT(ratio)	2.6 ± 0.1	2.4 ± 0.1

Online Table IV: Conscious Echocardiography of inducible Rbm20 inhibition in *Ttn*$^{\Delta IAjxn}$ mice, 28 days after completing vehicle or raloxifine injection. Data shown as mean ± SEM. No differences are detected in systolic function or LV chamber dimensions. See text for details. Abbreviations: HR: heart rate; BPM: beats per minute; FS: fractional shortening; LVIDd: left ventricular internal diastolic diameter; LVIDs; left ventricular internal systolic diameter; LVPWd: left ventricular posterior wall thickness in diastole; LVPWs: left ventricular posterior wall thickness in systole. Eccentricity: LVIDd/WTd. Statistical significance calculated with Student's T-test: * $p<0.05$.

Supplemental References

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