Supplemental Information - Material and Methods

Echocardiography

Serial echocardiographic measurements were obtained at baseline, 4 and 8 weeks following MI. Echocardiographic assessments were performed in anesthetized rats (1-2% isoflurane inhalation) using a Vevo-770 echocardiogram (Visual Sonics Inc., Toronto, Ontario, Canada) equipped with 17.5 and 25 MHz transducers. Cardiac dimensions: left ventricle end-diastolic (LVEDD), end-systolic (LVESD) diameters and fractional shortening (FS) were recorded from M-mode images using averaged measurements from 3 to 5 consecutive cardiac cycles according to the American Society of Echocardiography (1). LV end-diastolic (LVEDV) and end-systolic (LVESV) volumes and ejection fraction (EF) were calculated from bi-dimensional long-axis parasternal views taken through the infarcted area. All images were analyzed using Vevo 770 3.0.0 software (Visual Sonics).

Hemodynamic measurements

Rats were anesthetized with an intramuscular injection of 0.5 ml/Kg of a mixture of 1.5 ml of ketamine (100mg/ml), 1.5 ml of xylazine (20 mg/ml) and 0.5 ml of acepromazine (10 mg/ml). A 2-F micromanometer tipped catheter (SPR-838, Millar Instruments, Houston, TX) was inserted into the right carotid artery and advanced retrograde into the left ventricle. Measurements were calibrated by injecting a hypertonic saline (15%) bolus to determine extra-ventricular conductance; relative volume units were converted to actual volume using the cuvette calibration method (2). All analyses were performed using PVAN 3.0 software (Millar Instruments, Houston, TX). Left ventricular pressure-volume loops were recorded at steady state and at varying preloads during temporary compression of theinferior vena cava.

Tissue Collection

At the end of the study, rat hearts were harvested and weighed, and a portion of the LV remote from the infarct area was flash-frozen in liquid nitrogen for total RNA

isolation and protein analysis. Remaining tissues were fixed with 10% formalin for histology.

Morphometric analysis

Rat hearts were processed using routine histological procedures. Five-micrometer sections were sliced and stained with hematoxylin/eosin (H&E) and Masson's trichrome. Myocardial infarct size was quantified by the circumferential extent of scar and percentage area fibrosis as previously described (3).

Total RNA Isolation

Total RNA from heart tissue was extracted using Trizol (Invitrogen, Carlsbad, CA). Three heart samples from each group were analyzed.The quality of RNA isolated was tested using NanoDrop1000 (Thermo Fisher Scientific Inc., Wilmington, DE). OD 260/280 ratio was in the range of 1.8 to 2.1 for all samples.

Myocyte isolation

The isolation of myocytes was performed as previously described (4). Briefly; the rats were anesthetized with pentobarbital (100mg/Kg, Sigma, St. Louis, MO) with heparin (4000 U/Kg, APP Pharmaceuticals, Schaumburg, IL). For the isolation of myocytes, the hearts were cannulated and perfused through the aorta with Ca^{2+} free bicarbonate buffer containing 120 mMNaCl, 5.4 KCl, 1.2 mM $MgSO_4$, 1.2 mM NaH_2PO_4, 20 mM $NaHCO_3$, 10 mM 2,3-butanedione monoxime, 5 mMtaurine and, 5.6 mM glucose, gassed with 95% O_2 - 5% CO_2; this was followed by enzymatic digestion with collagenase type-2 (1 mg/ml, Worthington Biochemical Co., Lakewood, NJ) and protease type-XIV (0.1 mg/ml, Sigma, St. Louis, MO).

Cardiomyocytes were obtained from digested hearts which were then mechanically disrupted, filtered, centrifuged and resuspended in a Tyrode solution containing 0.125 mM $CaCl_2$, 144 mMNaCl, 1 mM $MgCl_2$, 10 mM HEPES, 5.6 mM glucose, 1.2 mM $NaHPO_4$ and 5 mMKCl at pH7.4.

GH and IGF-I measurements

Blood was drawn 1-2 hours after the last injection at the end of the study and the serum was stored at –80ºC until the measurements were performed. All the samples were assayed together and each sample was assayed in duplicate. Rat serum GH was measured using a rat GH Enzyme-Linked Immunosorbent Assay (ELISA) Kit (DSL-10-72100, DSL Webster, TX), following the manufacturer's recommendations. This test is an enzymatically amplified “one-step” sandwich-type enzyme immunoassay, where standards, controls and unknown samples are incubated in micro-titration wells pre-coated with the anti-rat GH antibody. The standard curve of the assay was established with samples provided by the manufacturer. Rat serum IGF-I, after extraction with acid ethanol, was measured using a rat IGF-I Radioimmunoassay Kit (DSL-2900, DSL Webster, TX) following the manufacturer’s recommendations. The IGF-I assay included quality controls provided by the manufacturer. The standard curve of the assay was established with samples provided by the manufacturer.

Immunostaining

Cardiomyocytes were stained as described previously (5). Briefly, after isolation, 150 μl of cardiomyocytes in suspension were allowed to sediment and then fixed for 10 minutes in 2% paraformaldehyde. Cells were then stained with rabbit polyclonal antibody against human GHRHR at 4ºC for 24 hours followed by the secondary antibody at 37ºC for 1 hour (see table S3).

Paraffin sections were deparaffinized and rehydrated by immersion in xylene followed by a graded series of ethanol as previously described (6). Antigen retrieval was performed by a heat-induced method with citrate buffer (Dako, Carpinteria, CA). After blocking with 10% normal donkey serum, sections were incubated with a primary antibody (table S3), at 37ºC for 1 hour, followed by application of secondary antibody. Omission of the primary antibodies on parallel sections was used as negative control. Nuclei were counterstained with DAPI (Invitrogen, Carlsbad, CA). The total numbers of positively-stained cells were quantified per slide to calculate the number of cells per unit

volume (mm^3) on each sample. Morphometric analysis was performed using Adobe Photoshop CS3 (San Jose, CA).

To quantify apoptosis of cardiac cells, terminal deoxynucleotidyltransferase-mediated dUTP nick end-labeling (TUNEL) staining was performed on paraffin embedded tissue sections according to the manufacturer's protocol using a commercially available kit (*In Situ* Cell Death Detection Kit, POD, Roche Diagnostics GmbH, Germany). Slides were scanned by using a Mirax scan (Carl Zeiss,GmHB, Germany) and analyzed under 20 and 40x magnifications. Apoptotic nuclei were identified by green fluorescence staining and expressed as a percentage per cubic millimeter (mm^3). All images were obtained with fluorescent (Olympus IX81, Olympus America Inc., Center Valley, PA) or a LSM710 Zeiss confocal laser scanning module(Carl Zeiss MicroImaging). The quantification of other markers (c-kit, CD45, mast cell tryptase, myosin light chain, phospho-histone H3 [pH_3], GATA-4) was performed in a similar manner.

Quantification of immunohistochemistry staining for GHRHR

All images were obtained using a 40x objective and the settings were constant for the entire study. Ten high power fields of confocal images were taken from each sample (n=3 for each group). The quantification of the fluorescence was performed following deconvolution, using Huygen Essential software, version 3.4 (Scientific Volume Imaging, Hilversum, The Netherlands). An optical density plot of the selected area was generated using the histogram tool in the Image Pro plus version 6.3 (Media Cybernetics, MD) and the mean staining intensity (intensity/pixel) was recorded.

Capillary density

For determining the capillary density in the myocardium, three to four representative sections of each group were stained with isolectin-B4 conjugated to Alexa 488 (Invitrogen, Carlsbad, CA) for 24 hours at 4°C. Five fields on each section were randomly chosen for counting vessel profiles at the border and remote area separately. The capillary density was expressed as capillaries/high-power field (HPF).

Real Time PCR

Two micrograms of total RNA were reverse transcribed with the QuantiTect Reverse Transcription Kit (Qiagen, Valencia, CA) using Veriti 96 well thermal cycler (Applied Biosystems, Foster City, CA). Quantitative real-time RT-PCR reactions were performed using the iQ5 Real-Time PCR Detection System (Bio-Rad Laboratories, Hercules, CA).

We evaluated the mRNA expression of rat GHRHR, GHRH and β-actin. The analyses of gene expression for GHRHR, GHRH and β-actin were as described previously (5, 7). PCR conditions were 3 min at 95ºC and 30 cycles with 30 s 95ºC, 60 s 60ºC. The criteria for the design of rat specific primers were as extensively described (8). All samples were run in triplicate, and each well of PCR reactions contained 25 µL as final volume, including 2 µL of cDNA, 200 nM of gene-specific primers, and 400 nM of probes. iQSupermix (Bio-Rad) was used in the PCR. The efficiencies of all primers (Invitrogen Life Technologies, Carlsbad, CA) and probes (Integrated DNA Technologies, Coralville, IA) were tested prior to the experiments and they were all efficient in the range of 95–105%. Normal rat pituitary was used as positive control and rat beta-actin was used to normalize for differences in RNA input(5). Negative samples were run in each reaction consisting of no-RNA in reverse transcriptase reaction and no-cDNA in PCR reaction.

To examine the effect of the treatments on the molecular phenotypes of MI hearts pro-apoptotic and anti-apoptotic genes, as well as stem cell, calcium homeostasis and genes related to proliferation, were also assessed by real-time PCR. We used TaqMan probes (see Table S4) labeled with 6-carboxyfluorescein (FAM) and iQSupermix (Bio-Rad Laboratories) for real-time RT-PCR reactions, according to the manufacturer's protocol (Applied Biosystems). Relative expression ratios were calculated using Pfaffl's method (9). Calculated values are presented as mean ± SEM to indicate accuracy of measurement.

Functional assay of proliferation

In order to determine the impact of GHRH-A on proliferation of cardiac stem cells (CSCs) *in vitro,* we isolated CSCs from rat hearts after myocardial digestion with collagenase. C-kit^{pos} cells were selected by magnetic sorting using Easy Sep (Stem Cell Technology). Selection was confirmed by fluorescence activated cell sorting (FACS) and immunostaining. Proliferation was determined by measuring the incorporation of the thymidine analog EdU (5-ethynyl-2´-deoxyuridine), followed by immunodetection and quantification of EdU positive cells by flow cytometry using a commercially available kit (Click-it, Invitrogen). Cells were plated a day before the experiment at an initial density of 2-5 x 10^5cells/100 mm culture dish and pre-primed with 200 nM GHRH-A, vehicle (DMSO), MIA-602 and combination of GHRH (A+Ant).The next day, cells were treated again with drugs and Edu at a concentration of 10μM, harvested at different time points (8h-20h), fixed, stained and analyzed according to manufacturer's instructions.

Statistical Analysis

All values are shown as mean ± SEM. Significance was determined by the analysis of variance (ANOVA) followed by post-hoc tests and Student's *t* test. For a given parameter, $p<0.05$ was considered significant. All tests were carried out using GraphPad Prism software (San Diego, CA, USA) version 5.0 for Windows.

References

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2. Pacher, P. *et al.* (2008) Measurement of cardiac function using pressure-volume conductance catheter technique in mice and rats *Nat. Protoc.* 3, 1422-1434.

3. Kanashiro-Takeuchi R.M *et al.* (2009) Sex-specific impact of aldosterone receptor antagonism on ventricular remodeling and gene expression after myocardial infarction *Clinical and Translational Science* 2, 134-142.

4. Gonzalez, D. R., Beigi, F., Treuer, A. V., & Hare, J. M. (2007) Deficient ryanodine receptor S-nitrosylation increases sarcoplasmic reticulum calcium leak and arrhythmogenesis in cardiomyocytes *Proc. Natl. Acad. Sci. U. S. A* 104, 20612-20617.

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9. Pfaffl, M. W. (2001) A new mathematical model for relative quantification in real-time RT-PCR *Nucleic Acids Res.* 29, e45.

Supplemental Information - Results

Figure Legends

Figure S1. Changes over time in body weight (BW) after MI and effects of 4-week treatment on heart weight (HW) and the ratio HW/BW and HW and tibia length (HW/TL). All values represent mean ± SEM (n=7-10). Note that rrGH, GHRH-A and GHRH (A+Ant) therapy increased BW at week 8 (* $p<0.001$ † $p<0.05$ vs. placebo and MIA-602).

Figure S2. Serum concentration (ng/ml) of GH (A) and IGF-I (B) measured at the end of the study. All values represent mean ± SEM (n=7-10) Panel A: GH level was markedly increased by rrGH treatment, * $p<0.0001$ vs. all groups. Panel B: IGF-I levels were increased in both rrGH and GHRH (A+Ant) groups, * $p<0.0001$ vs. placebo, GHRH-A and MIA-602.

Figure S3. Representative confocal micrograph image of GHRH receptors (GHRHR). Bar graphs correspond to expression of GHRHR. Panels depicts a confocal microscopy images showing tropomyosin (green), GHRHR (red) and nuclei (blue). The upper right panel shows higher magnification of the GHRHR on the cardiomyocyte sarcolemmal membrane. Scale bars correspond to 10 and 50 um respectively. At the bottom, bar graphs represent the measurement of the intensity of the fluorescence of GHRHR on cardiomyocytes. All values represent mean ± SEM (n=3-4)

Figure S4. Heat map showing fold change patterns of mRNA for anti- and pro-apoptotic genes (Bcl-2 and Bax, respectively), stromal derived factor 1 (SDF-1), stem cell factor (SCF), genes related to angiogenesis (vascular endothelial growth factor A: VEGFA) and proliferation (IGF-1 and cyclin A2: CCna2), GHRH and GHRH-R. The heat map was generated in Microsoft Excel; color spectrum expands from green (lowest value) to red (highest value) through yellow.

Figure S5. Representative pressure-volume loops and corresponding end-systolic (ESPVR) and end-diastolic (EDPVR) pressure-volume relationships from placebo and

GHRH-A groups. As depicted, improved contractility was observed in the GHRH-A treated rats as compared with placebo rats.

Figure S6. Representative fluorescent microimage of endothelial cells stained for isolectin B4 (green) in placebo and GHRH-A group. Bar graphs showing capillary density per high power field (HPF) at the border zone. All values represent mean ± SEM (n=3-4).

Figure S7. Impact of treatments on expression of apoptosis ($TUNEL^{pos}$ cells) at the border zone of the infarct area. All values represent mean ± SEM (n=3-4).

Figure S8. GHRH receptor (GHRHR) expression on rat CSCs. FACS analysis of rat CSCs showing that the majority of cells from explants (SPNT) or supernatant (SPNT) are positive for GHRHR.

Figure S9. Representative fluorescent microimage of transcription factor GATA-4 (magenta), myosin light chain (MLC, green) and nuclei (blue). Bar graphs correspond to expression of $GATA\text{-}4^{pos}$ cells at the border zone of the infarct area. Data represent mean ± SEM (n=3), * $p<0.05$ vs. placebo and MIA-602.

Table Legends

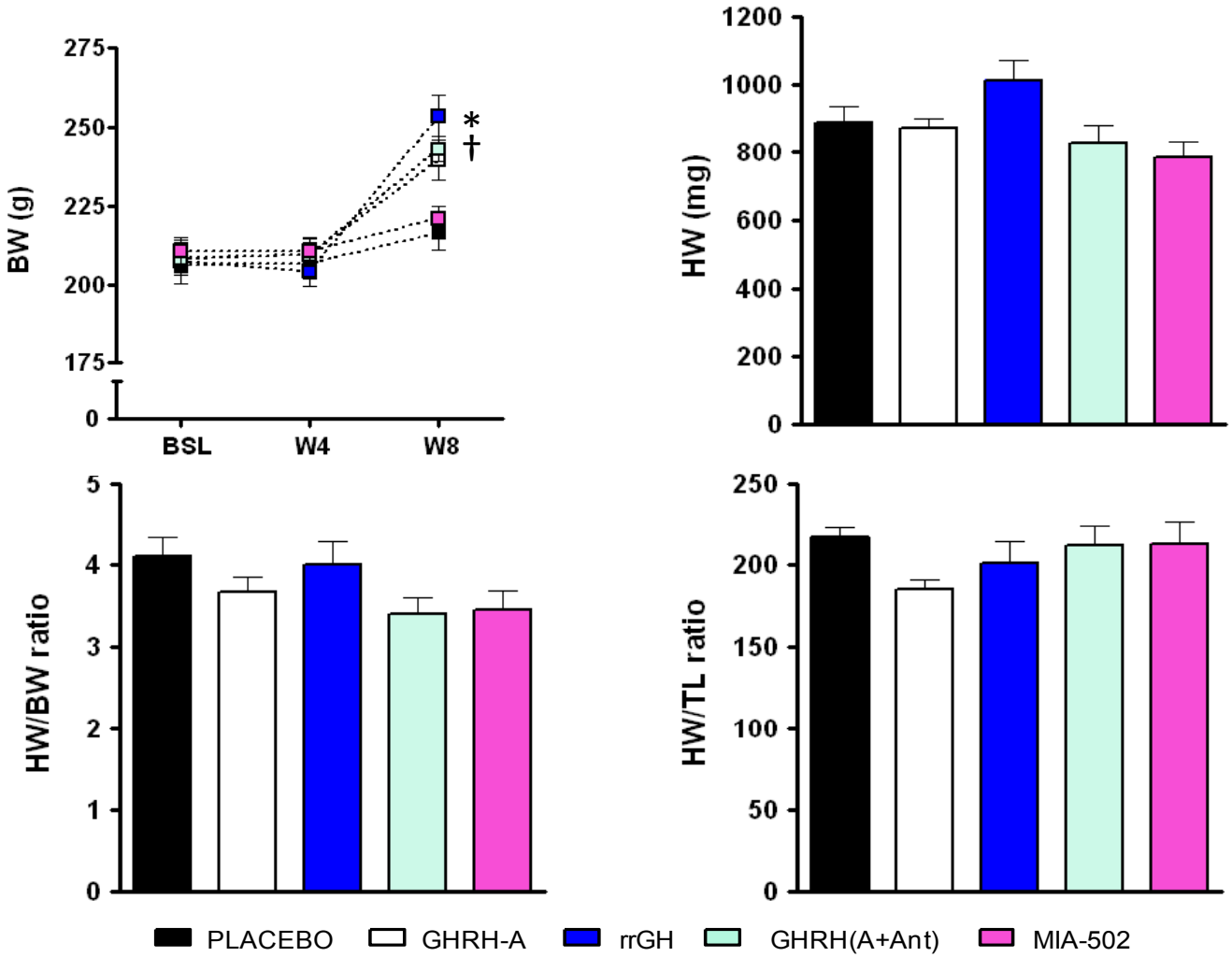
Table S1. Serial echocardiographic analysis of LV chamber size and function. Echocardiographic measurements at baseline (BSL), week 4 (W4) and week 8 (W8) after MI.

Table S2. Hemodynamic parameters and indices of systolic and diastolic function derived from pressure-volume relationships.

Table S3. Antibodies list.

Table S4. TaqMan probes for RT-PCR.

Figure S1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W4
W8
*
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4
3
2
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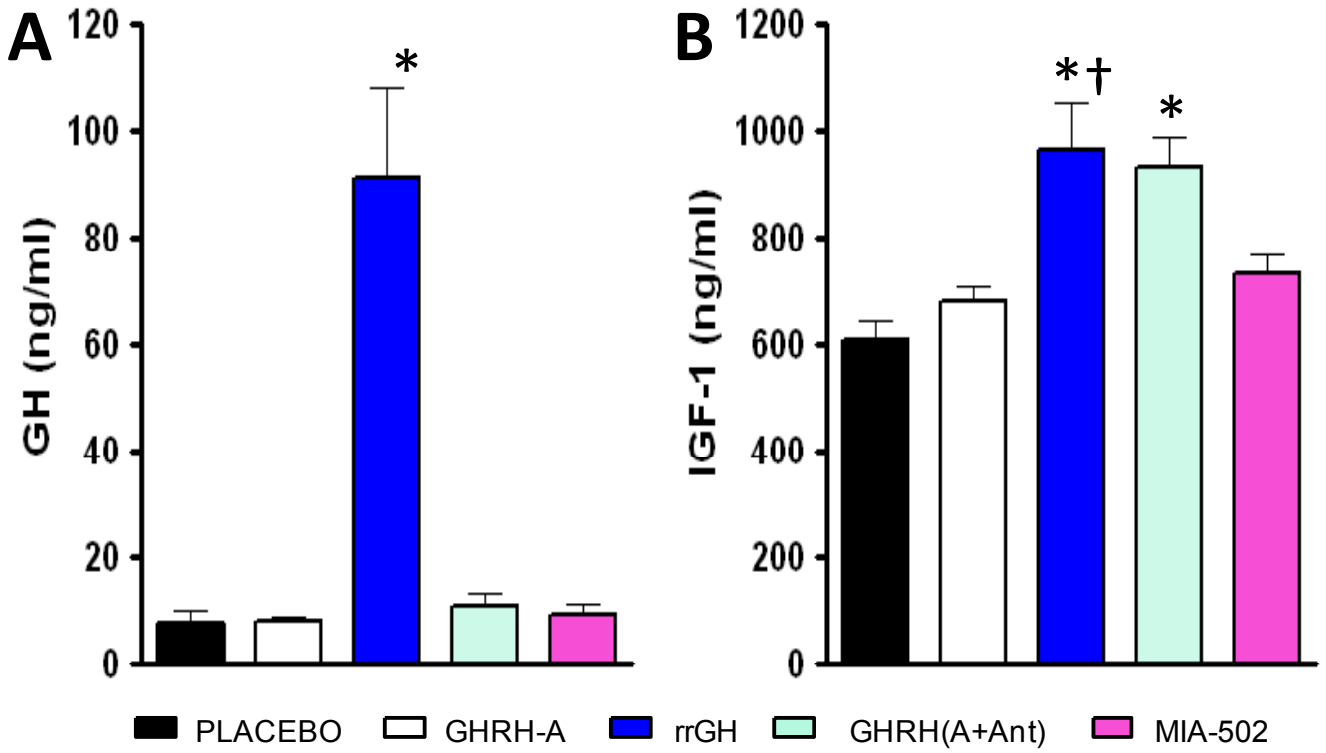
PLACEBO
GHRH-A
rrGH
GHRH(A+Ant)
MIA-502

Figure S2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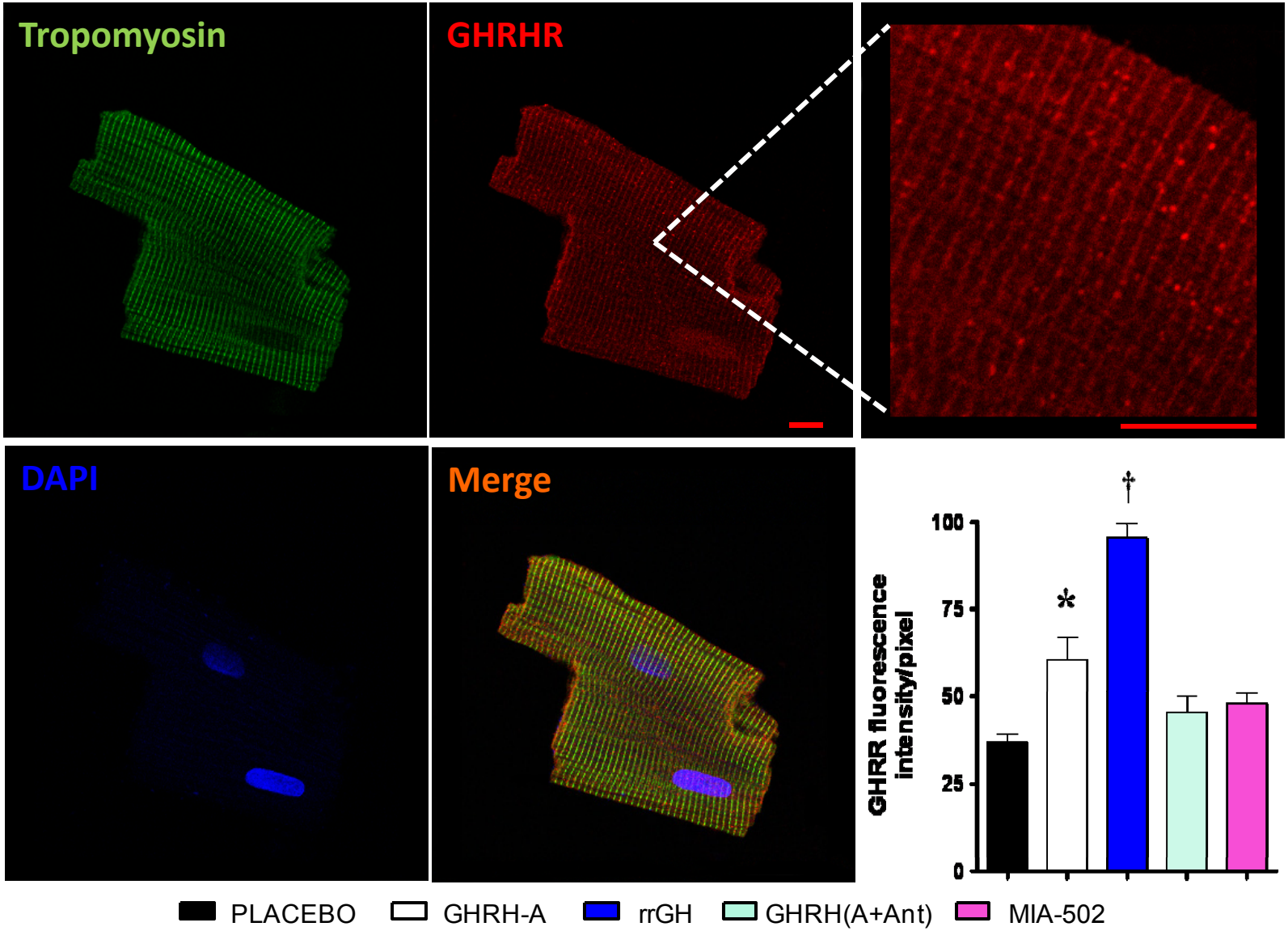
*
PLACEBO
GHRH-A
rrGH
GHRH(A+Ant)
MIA-502

Figure S3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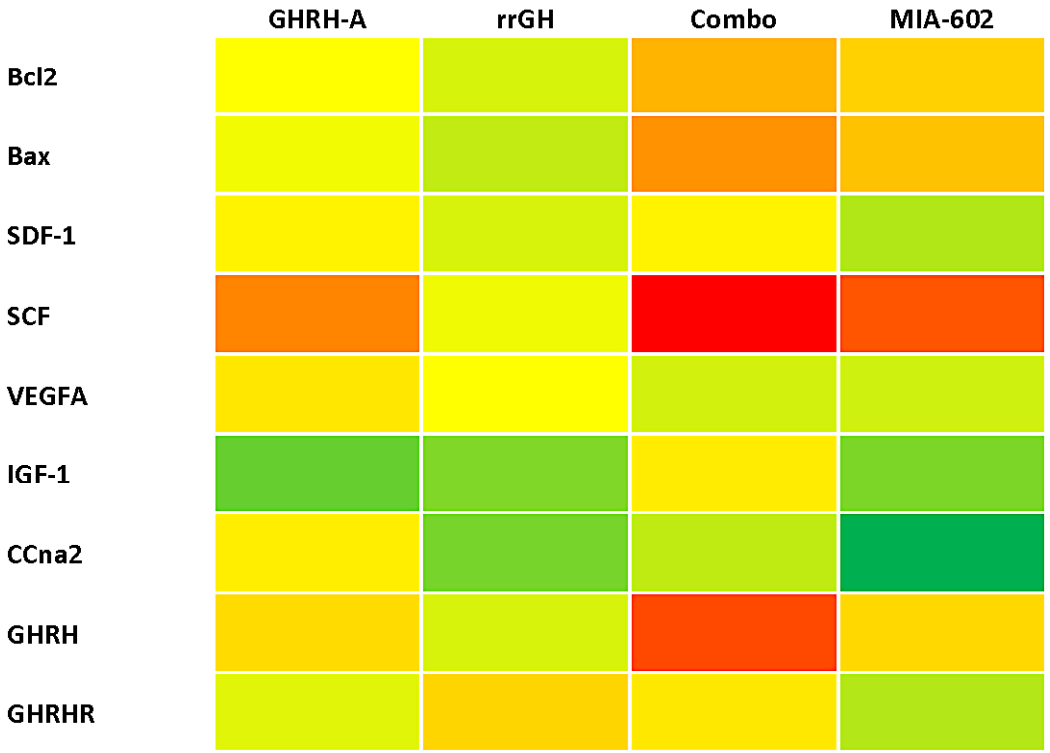
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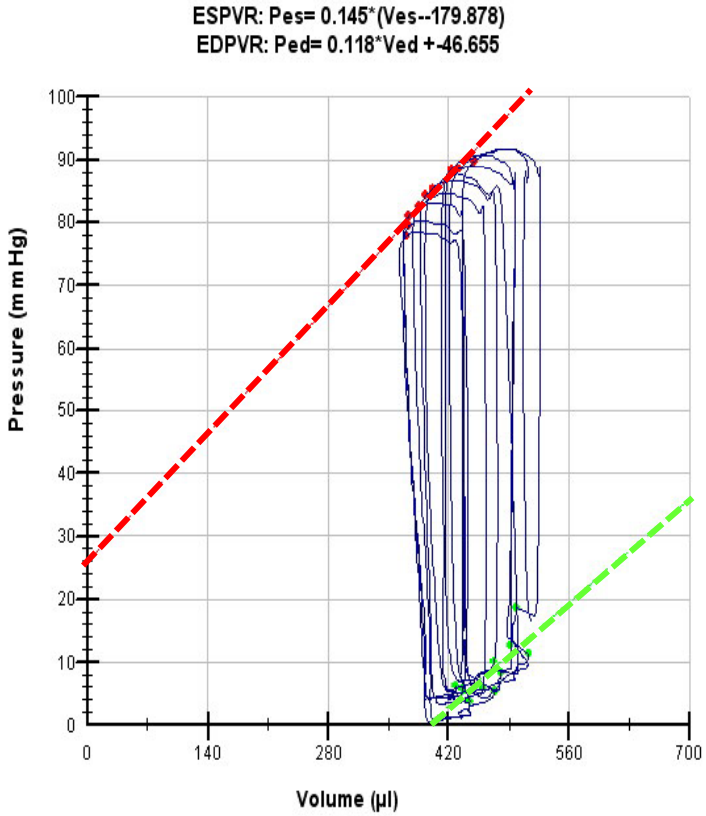
GHRHR

Figure S5

PLACEBO



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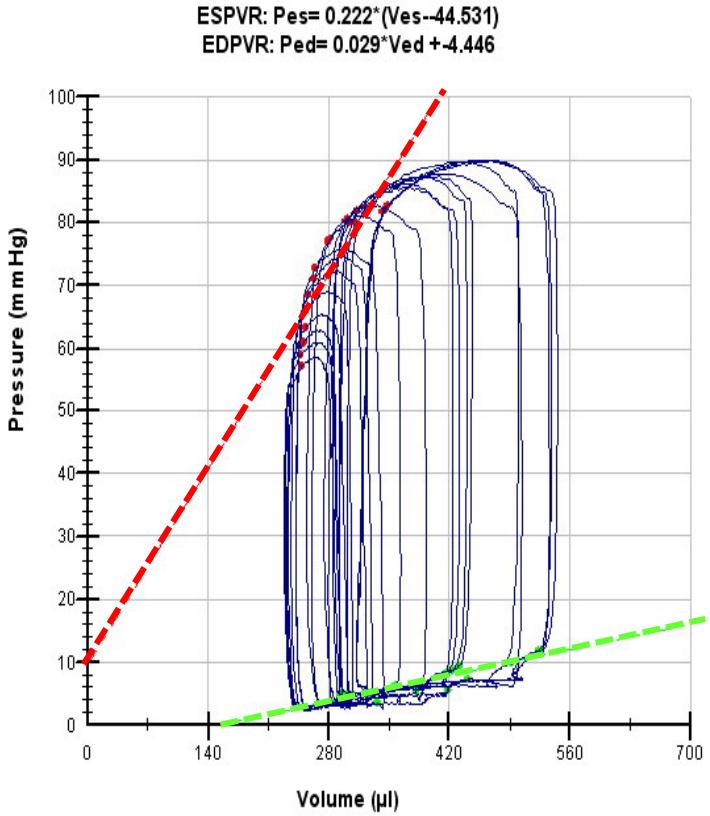

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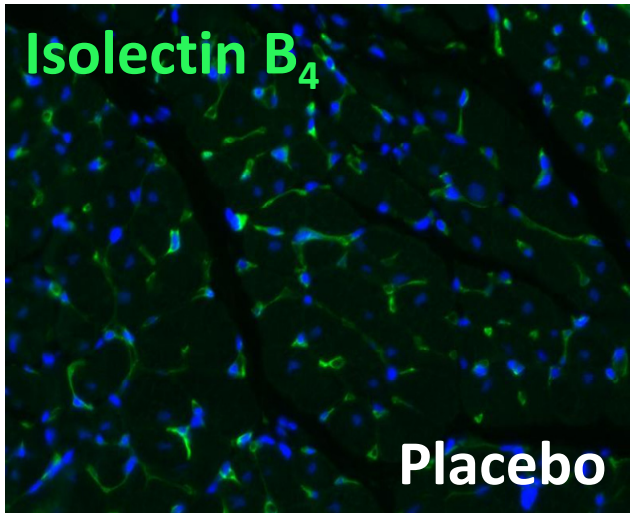
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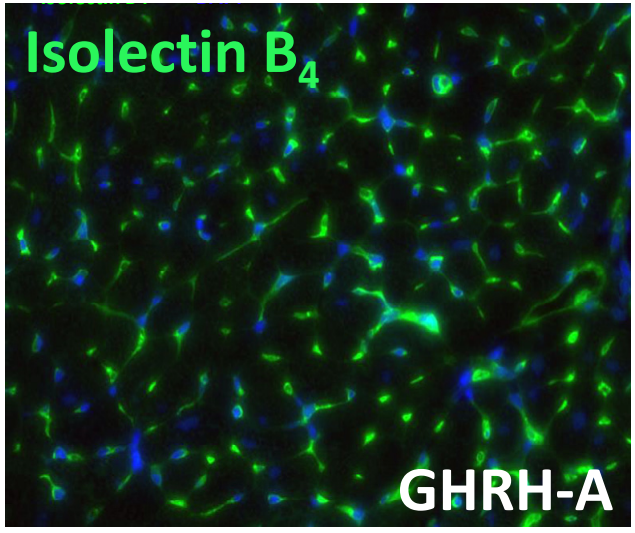
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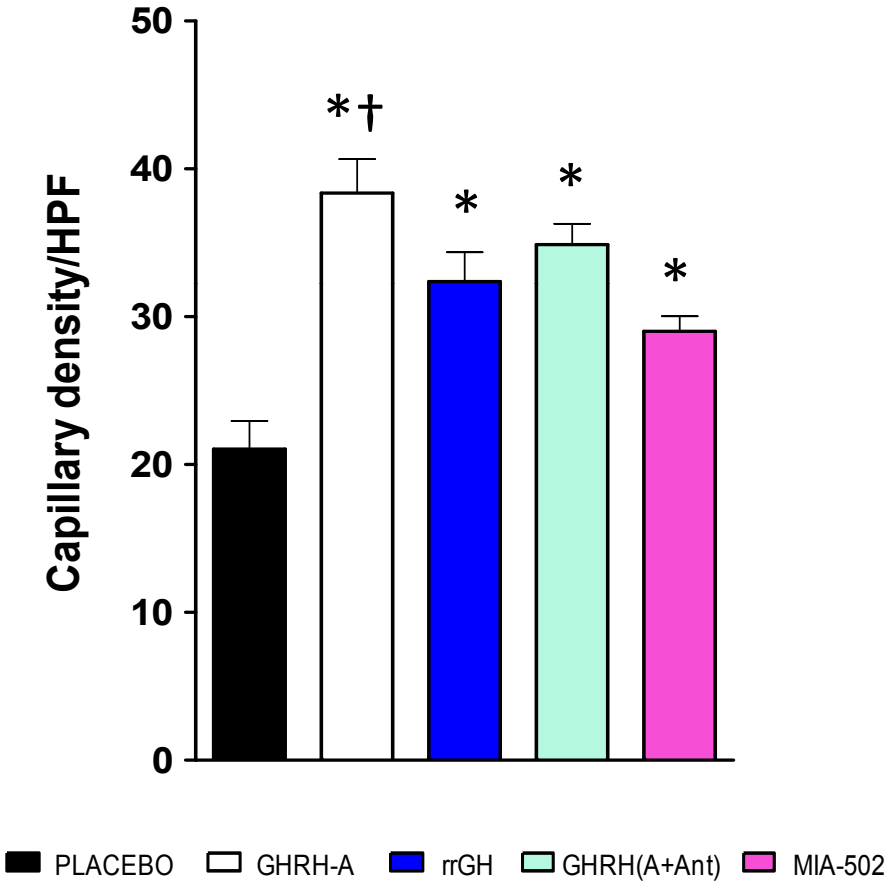
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20
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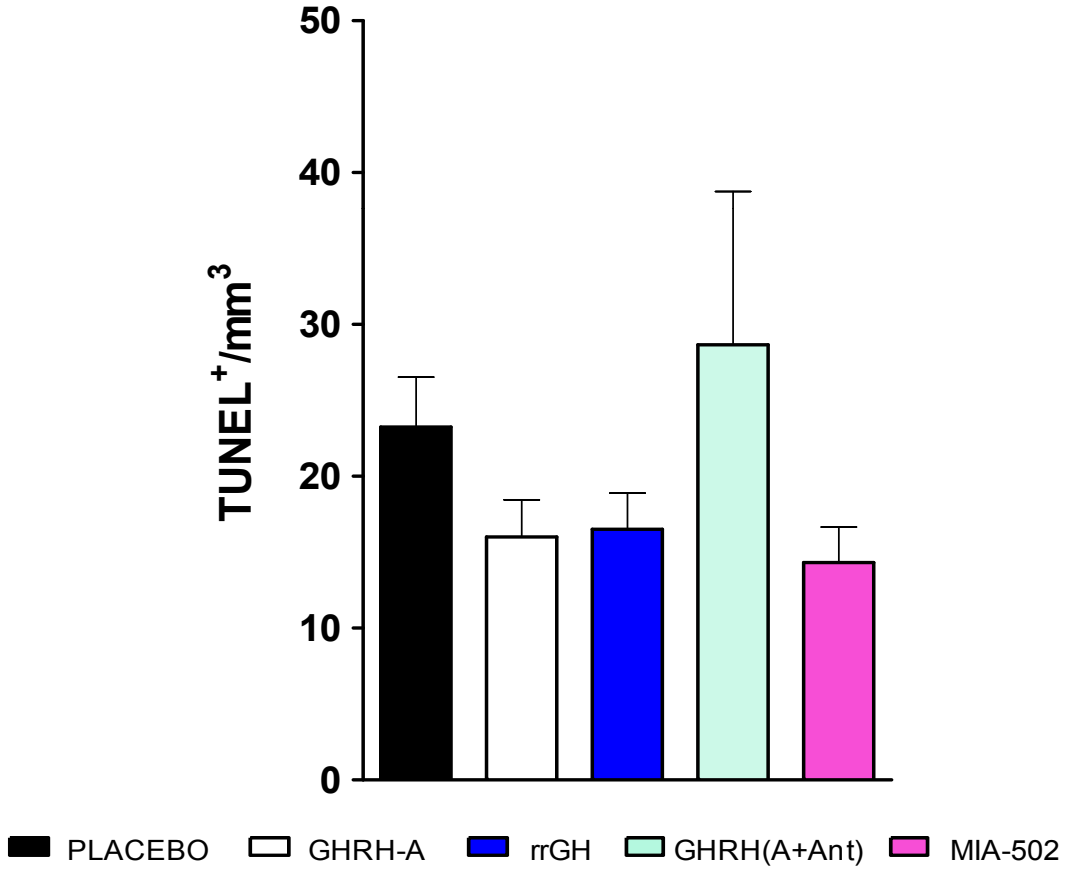
*
*
PLACEBO
GHRH-A
rrGH
GHRH(A+Ant)
MIA-502

Figure S7



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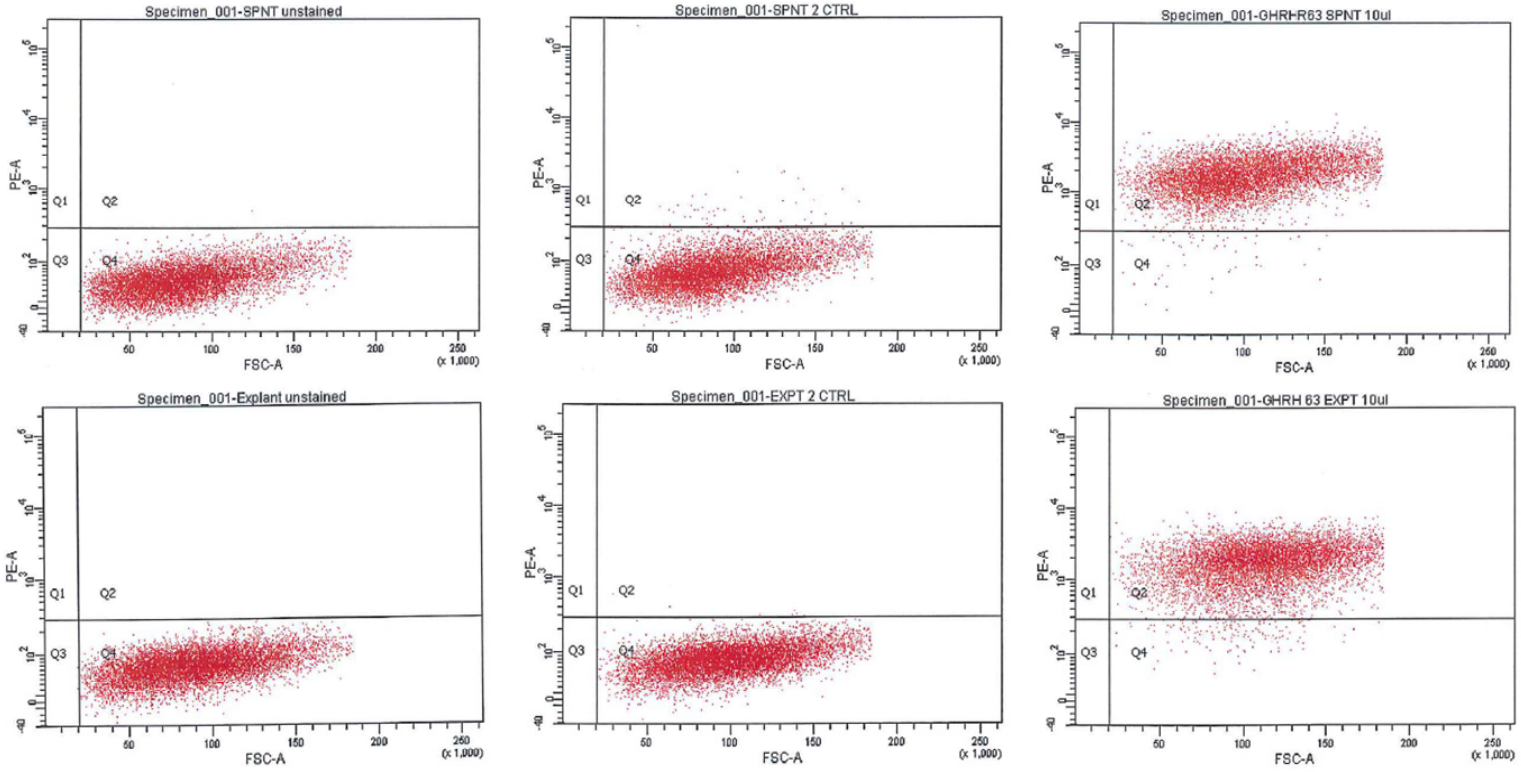
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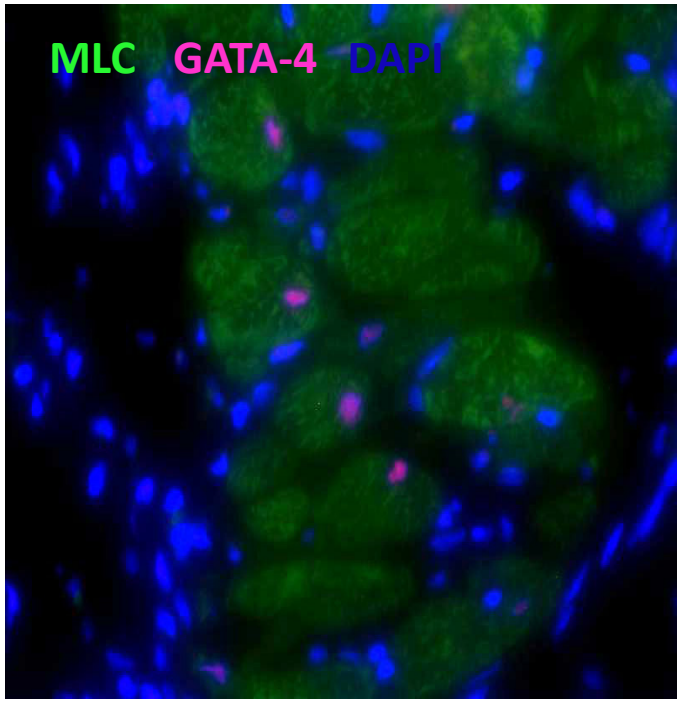
Q1
Q2
Q3
Q4

Figure S9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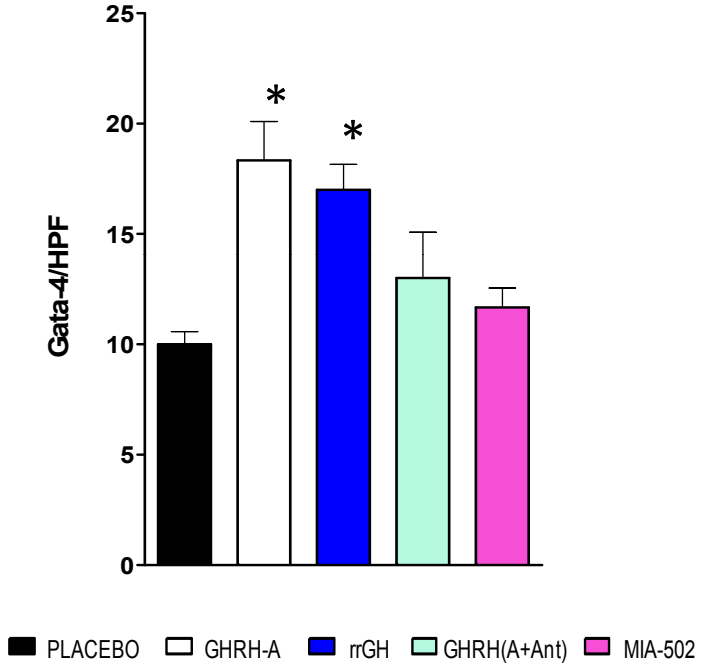
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20
15
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*
*
PLACEBO
GHRH-A
rrGH
GHRH(A+Ant)
MIA-502

Table S1. Serial echocardiographic analysis of LV chamber size and function. Echocardiographic measurements at baseline (BSL), week 4 (W4) and week 8 (W8) after MI.

Parameter	Placebo (8)	GHRH-A (10)	rrGH (9)	GHRH(A+ANT) (7)	MIA-602 (8)
LVEDD (mm)					
BSL	6.0±0.1	6.0±0.1	6.0±0.1	5.9±0.1	6.1±0.1
W4	8.2±0.1 *	8.4±0.1 *	8.4±0.1 *	8.4±0.2 *	8.3±0.2 *
W8	8.7±0.2 *	8.8±0.1 *	9.2±0.2 *†‡	8.9±0.1 *†	8.8±0.3 *
LVESD (mm)					
BSL	2.6±0.1	2.8±0.1	2.7±0.1	2.7±0.1	2.4±0.1
W4	6.8±0.2 *	6.7±0.1 *	6.7±0.2 *	7.0±0.3 *	6.9±0.1 *
W8	7.4±0.2 *†	7.3±0.1 *†	7.8±0.2 *†	7.4±0.1 *	7.4±0.3 *
EF (%)					
BSL	89±0.7	85±0.9	88±1.0	88±0.7	89±0.7
W4	35.4±1.9 *	35.3±2.7 *	38.1±2.5 *	36.0±1.5 *	36.0±2.7 *
W8	32.7±2.7 *	43.0±3.7 *†§	32.3±2.7 *	34.5±2.8 *	31.2±2.5 *†
FS (%)					
BSL	57.2±1.9	53.6±0.7	54.4±1.8	53.4±1.8	54.3±1.1
W4	17.2±1.3 *	19.2±1.0 *	18.4±1.0 *	17.5±1.8 *	17.2±1.4 *
W8	14.6±0.7 *	17.4±0.6 *¶	14.8±1.5 *†	16.4±1.3 *	15.9±1.3 *
VD (µl)					
BSL	254±4.0	264±5.5	257±5.8	281±10.5	260±8.0
W4	369±15 *	387±12 *	388±12 *	426±8 *	428±10 *
W8	523±22 *†	510±17 *†	554±28 *†	525±10 *†	503±30 *†
VS(µl)					
BSL	29±1.7	31±1.8	31±2.0	33±1.4	30±2.6
W4	260±16 *	252±14 *	255±12*	265±9.8*	264±14 *
W8	359±24*†	291±21 *§	376±26 *†	359±31 *†	370±29 *†

* $p < 0.05$ vs. BSL, same group

† $p < 0.05$ vs. W4, same group

‡ $p < 0.05$ vs. all other groups at W8, except GHRH (A+Ant)

§ $p < 0.05$ vs. all other groups

¶ $p < 0.05$ vs. placebo at W8

Table S2. Hemodynamic parameters and indices of systolic and diastolic function derived from pressure-volume relationships.

Parameter	Placebo (5)	GHRH-A (5)	rrGH (5)	GHRH (A+Ant) (7)	MIA-602 (7)
Heart rate (bpm)	266±7.3	241±13	274±12	260±9	295±28
Integrated performance					
EF (%)	22.8±0.9	38.8±4.2 *	29.7±1.9	28.1±1.3	25.1±2.7
SW (mmHgxμl)	5432±277	12572±1685 †	9201±877	8844±426	7328±1027
SV (μl)	94.3±8.5	184.4±19.2 †	137.6±12.4	133.7±3.5	111.4±17.9
CO (ml/ min)	21.9±2.9	40±6.9 †	38.1±3.5	34±2.2	27.2±2.4
Ea/Ees	5.3±0.6	2.7±0.5 ‡	3.5±0.5	4.1±0.2	5.1±1.2
Afterload					
LVESP (mmHg)	89.5±2.9	87.9±3.8	87.7±1.2	89±2.1	89.2±2.2
Ea (mmHg/ μl)	0.9±0.08	0.5±0.06 †	0.6±0.05	0.7±0.04	0.9±0.13
Preload					
LVEDP (mmHg)	19.8±2.3	12.5±1.3 ‡	18±3.1	19.3±3.1	16.2±1.7
Contractility					
dP/dt_{max} (mmHg/s)	6245±363	6642±370	6885±222	6532±429	6813±366
Ees (mmHg/μl)	0.17±0.02	0.19±0.01	0.19±0.02	0.17±0.01	0.19±0.01
PRSW (mmHg)	42±6.2	59±4.4	50±1.4	44±4.7	48±2.9
Lusitropy					
dP/dtmin (mmHg/s)	4314±234	4770±272	4589±300	4372±406	4603±414
TAU (W) (ms)	14.4±05	13.4±0.6	18.2±1.8	16.4±1.5	13.7±1.2

All values represent mean ± SEM. Ejection fraction (EF), stroke work (SW), stroke volume (SV), cardiac output (CO), ratio between arterial elastance and end-systolic elastance (Ea/Ees), left ventricular end-systolic pressure (LVESP), arterial elastance (Ea), left ventricular end-diastolic pressure (LVEDP), peak rate of the pressure rise (dP/dt_{max}), end-systolic elastance (Ees), preload recruitable stroke work (PRSW), peak rate of pressure decline (dP/dt_{min}), relaxation time constant calculated by Weiss method (TAU).

* p<0.05 vs. all other groups. except rrGH.

† p<0.05 vs. placebo and MIA-602

‡ p<0.05 vs.placebo, Student t test

Table S3. Antibody list

Protein	Antibody	Labeling	Fluorochromes
GHRHR	rabbit polyclonal	indirect	F
C-kit	goat polyclonal	indirect	T
Mast cell tryptase	mouse polyclonal	indirect	T
CD45	rabbit polyclonal	indirect	F
Myosin light chain	rabbit monoclonal	indirect	F
Phospho H3	mouse monoclonal	indirect	T
GATA-4	goat polyclonal	indirect	T
Nuclear DNA	DAPI	N/A	
TUNEL	Tdt/dUTP	direct	F

Direct labeling: primary antibody conjugated with the fluorochrome.

Indirect labeling: species-specific secondary antibody with the fluorochrome.

F: fluorescein isothiocyanate

T: tetramethyl rhodamine isothiocyanate,

Dapi: 4',6-diamidino-2-phenylindole.

Table S4. TaqMan® probes for RT-PCR

Gene	TaqMan® probe
Bcl_2	Rn99999125_m1
Bax	Rn02532082_g1
Cxcl12 (SDF-1)	Rn00573260_m1
Kitl (SCF)	Rn00442977_m1
VEGF-A	Rn00582935_m1
IGF-1	Rn00710306_m1
$CCna_2$	Rn01493715_m1