Materials and methods

Human plasma samples

Table S1 Clinical characteristics of patients with non-CAD or AMI

Clinical data	Non-CAD patients (*n*=122)	AMI patients (*n*=121)	*P*-value
Age (years)	63.31±7.31	64.53±13.32	0.380
Males (*n* (percentage))	101 (82.8)	102 (84.3)	0.860
Body mass index (kg/m^2)	23.20±2.71	24.23±3.84	0.017
Current smoker (*n* (percentage))	64 (52.5)	75 (62.0)	0.150
Hypertension (*n* (percentage))	53 (43.4)	72 (59.5)	0.015
Diabetes mellitus (*n* (percentage))	15 (12.3)	40 (33.1)	<0.001
Hyperlipemia (*n* (percentage))	46 (37.7)	70 (57.9)	0.002
Left atrial diameter (cm)	3.54±0.47	3.89±0.56	<0.001
Left ventricular diameter (cm)	4.56±0.40	5.00±0.68	<0.001
LVEF (%)	67.83±5.14	54.61±13.81	<0.001
Plasma IL-15 (pg/mL)	2.43±1.10	11.42±3.53	<0.001

Non-CAD: non-coronary artery disease; AMI: acute myocardial infarction; LVEF: left ventricular ejection fraction. Continuous data are evaluated by unpaired *t*-test and presented as mean±standard error of the mean (SEM). Categorical data are analyzed using a chi-square test.

Table S2 Logistic regression analysis of factors associated with AMI

Clinical data	aOR	95% CI	*P*-value
IL-15	1.320	1.130–1.542	<0.001
BMI	1.033	0.839–1.273	0.757
Hypertension	1.803	0.560–5.803	0.323
Diabetes	1.463	0.429–4.987	0.543
Hyperlipidemia	1.325	0.428–4.104	0.625

aOR: adjusted odds ratio, CI: confidence interval; IL-15: interleukin-15; BMI: body mass index. Continuous data are evaluated by unpaired *t*-test and presented as mean±standard error of the mean (SEM). Categorical data are analyzed using a chi-square test.

Echocardiography

Mice were anesthetized by ventilation with 3%–4% isoflurane, maintained by artificially ventilating with a mixture of O_2 and N_2O (1:2 (volume ratio)) and 1.5%–2.5% isoflurane. Cardiac function of mice was verified at baseline and at 3, 7, and 14 days after AMI by transthoracic echocardiography using a Visual Sonics system with a Ms-400 linear transducer (Vevo 2100, Visual Sonics, Canada). Two-dimensional-guided M-mode echocardiography was used to determine left ventricular chamber volume at systole and diastole and contractile parameters such as left ventricular internal dimension at end-diastole (LVIDd) and systole (LVIDs), left ventricular volume at end-diastole (LVEVd) and systole (LVEVs). Left ventricular fractional shortening (FS) was calculated as follows: FS (%) =(LVIDd−LVIDs)/LVIDd×100%. Left ventricular ejection fraction (EF) was derived as follows: EF (%) =(LVEVd−LVEVs)/LVEVd×100%. All measurements were based on the average of at least three cardiac cycles.

Morphological analysis

Murine hearts were excised and rapidly stored at −80 °C. Four to five heart sections (1 mm) of each mouse were placed in vital dye 2% (0.02 g/mL) triphenyltetrazolium chloride (TTC; Sigma-

Aldrich, USA) at 37 °C in the dark for 30 min and then immersed in 10% formalin at room temperature for 2 d. Image J software (v1.8.0) was used to assess the infarct size and infarct border zone. The final infarct size and infarct border zone for each section were adjusted to the heart section weight.

Culture of primary cardiomyocytes

Ventricles from C57BL/6J mice at 2 d old were dissected and minced, and then digested with collagenase type II (Sigma-Aldrich, USA) for 30 min. Digested tissue was filtered through a 70 μm cell strainer (BD Falcon, USA) and centrifuged at 500*g* for 5 min. Cells were resuspended and pre-plated with 1% (0.01 g/mL) collagen (Thermo Fisher Scientific, USA) for 1.5 h to remove fibroblasts. Subsequently, unattached cells were collected and seeded in high glucose Dulbecco's modified Eagle medium (DMEM) (Thermo Fisher Scientific, USA) containing 10% (volume fraction) fetal bovine serum (FBS; Thermo Fisher Scientific, USA).

Neonatal mouse ventricular cardiomyocytes were subjected to hypoxia. Hypoxia was achieved by placing the cells in a hypoxia incubator chamber (STEMCELL Technologies, Canada) filled with 5% CO_2-nitrogen balanced gas mixture with a final oxygen level of <1% inside the chamber. Cardiomyocytes were divided into 12 groups according to different conditions, either under normoxia or hypoxia, incubating for 24 h with recombinant murine interleukin-15 (rmIL-15) (0, 10, 20, 50, 100, and 200 ng/mL).

Culture of bone marrow-derived macrophages (BMDMs)

Both tibia and femurs were obtained from C57BL/6J mice and flushed with ice-cold PBS. After all bone was rinsed, the suspension was filtered through a 70 μm cell strainer (BD Falcon, USA) to obtain a single-cell suspension. Cells were centrifuged at 500*g* for 5 min and cultured in RPMI1640 (Thermo Fisher Scientific, USA) with 10% FBS containing 10 ng/mL recombinant murine macrophage colony-stimulating factor (MCSF; Peprotech, USA). Cells were incubated and differentiated for 7 d, and medium was replaced every 3 d. Macrophages were subjected to rmIL-15 (50 ng/mL, Peprotech, USA) and gossypin (10 and 50 μmol/L, Sigma-Aldrich, USA) for 24 h with serum-free medium.

Generation of Mer tyrosine kinase (*MERTK*)-overexpressing stable RAW 264.7 cells

RAW 264.7 murine macrophage-like cells (ATCC) were cultured in DMEM. To generate stable *MERTK*-overexpressing cells, lentiviral particles were produced by co-transfecting HEK293T cells (using lipofectamine 3000, Invitrogen) with packaging plasmids and the transfer plasmid. RAW 264.7 cells were transduced with the collected viral supernatants in the presence of polybrene. Stable cell pools were subsequently selected using puromycin (Beyotime, China) for 7–10 d.

Quantitative real-time PCR

Total RNA was extracted from cells or heart tissue using TRIzol reagent (Thermo Fisher Scientific, USA). The RNA samples were reverse-transcribed to complementary DNA (cDNA) using PrimeScript RT Master Mix kit (TaKaRa, Japan) according to the manufacturer's instructions, and the expression of genes was determined by real-time PCR using the SYBR Premix Ex Taq™ kit (TaKaRa, Japan). The relative expression of targeted genes was calculated as a ratio to that of β-actin. The primer sequences are shown as follows:

Gene	mRNA primers
β-actin	TGTGATGGTGGGAATGGGTCAGAA
	TGTGGTGCCAGATCTTCTCCATGT
IL-1α	CGAAGACTACAGTTCTGCCATT
	GACGTTTCAGAGGTTCTCAGAG
IL-1β	AAGGGCTGCTTCCAAACCTTTGAC
	ATACTGCCTGCCTGAAGCTCTTGT
IL-4	GGTCTCAACCCCCAGCTAGT

	GCCGATGATCTCTCTCAAGTGAT
IL-6	CAGAAAACAACCTGAACCTTCC
	GTACTCATCTGCACAGCTCTGG
IL-10	GGCGCTGTCATCGATTTCT
	ATTCATTCATGGCCTTGTAGACAC
IL-15	ACATCCATCTCGTGCTACTTGT
	GCCTCTGTTTTAGGGAGACCT
TNF-α	ACCCTCACACTCACAAACCA
	GGCAGAGAGGAGGTTGACTT
MCP-1	GCTGTAGTTTTTGTCACCAAGC
	AAGGCATCACAGTCCGAGTC
MERTK	CAGGGCCTTTACCAGGGAGA
	TGTGTGCTGGATGTGATCTTC
GAS6	TGCTGGCTTCCGAGTCTTC
	CGGGGTCGTTCTCGAACAC
AXL	ATGGCCGACATTGCCAGTG
	CGGTAGTAATCCCCGTTGTAGA
CD36	ATGGGCTGTGATCGGAACTG
	GTCTTCCCAATAAGCATGTCTCC
FAS	TATCAAGGAGGCCCATTTTGC
	TGTTTCCACTTCTAAACCATGCT
PKM	GCCGCCTGGACATTGACTC
	CCATGAGAGAAATTCAGCCGAG
GLUT1	CAGTTCGGCTATAACACTGGTG
	GCCCCCGACAGAGAAGATG
PGK1	ATGTCGCTTTCCAACAAGCTG
	GCTCCATTGTCCAAGCAGAAT
ENO1	TGCGTCCACTGGCATCTAC
	CAGAGCAGGCGCAATAGTTTTA
HK2	CAGGTGGAGCCCGAGTATTG
	CGTACATCCGTTGGAACGTG

Western blot analysis

Total protein was extracted from homogenized heart tissue using a complete protease inhibitor cocktail and radioimmunoprecipitation assay (RIPA) lysis buffer. Cell pellet was resuspended in RIPA cell lysis buffer containing 50 mmol/L Tris-HCl, 150 mmol/L NaCl, 5 mmol/L ethylenediaminetetraacetic acid (EDTA) (pH 7.4), 0.5% Triton X-100 and protease, and phosphatase inhibitor (Roche Diagnostics, Mannheim, Germany). Protein concentration was measured using bicinchoninic acid (BCA) protein assay with a Thermo Multiskan Spectrum spectrophotometer (Thermo Fisher Scientific, USA). Lysates were subjected to 4%–20% sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) gel electrophoresis and then transferred onto PVDF membranes (Sigma-Aldrich, USA). The membranes were blocked with 5% (0.05 g/mL) milk (Bio-Rad, USA) and then probed overnight at 4 °C with the following primary antibodies: IL-15, IL-15Rα, IL-2Rβ, IL-2Rγ, β-tubulin, β-actin, glyceraldehyde-3-phosphate dehydrogenase (GAPDH; all purchased from Abcam, UK), caspase-3, cleaved caspase-3, histone-H3, nuclear factor-kappa B (NF-κB) p65, phosphorylated NF-κB p65, and MERTK (Cell Signaling Technology, USA). After washing with PBST (3×), the membranes were incubated with HRP-conjugated secondary antibodies for 1 h at room temperature. Protein bands were visualized using chemiluminescent detection reagent Clarity™ ECL western substrate (Bio-Rad, USA), exposed to autoradiography film and finally quantified using Image J software (v1.8.0, NIH).

Immunofluorescence staining

Murine hearts were collected after AMI, embedded in optimal cutting temperature (OCT) compound (Leica, Heidelberg, Germany) and frozen immediately. Frozen tissue sections (5 μm) were thawed and fixed with 4% (0.04 g/mL) paraformaldehyde for 30 min, rinsed in PBS for three

times (5 min each), permeabilized in 0.1% (volume fraction) Triton X-100 (Thermo Fisher Scientific, USA) for 10 min and rinsed in PBS for three times. This was followed by blocking with 5% (0.05 g/mL) bovine serum albumin (BSA)/phosphate-buffered saline (PBS) (Thermo Fisher Scientific, USA) solution for 1 h. Subsequently, the cells were incubated with primary antibodies overnight at 4 °C, washed three times in PBS and afterwards incubated with secondary antibodies. The primary antibodies used were as follows: goat anti-cardiac troponin I (cTnI), mouse anti-vimentin, rabbit anti-IL-15, rat anti-cluster of differentiation 68 (anti-CD68), rat anti-CD3, rat anti-CD19, mouse anti-myeloperoxidase (MPO), rabbit anti-mannose receptor (CD206), rabbit anti-inducible nitric oxide synthase (iNOS; all purchased from Abcam, UK), mouse anti-IL-15R (Santa Cruz Biotechnology, USA), rabbit anti-CD11c and rabbit anti-NF-κB p65 (Cell Signaling Technology, USA). The corresponding secondary antibodies were used subsequently (Abcam, UK).

The terminal-deoxynucleoitidyl transferase-mediated nick end labeling assay (TUNEL, Beyotime Biotechnology, China) was performed according to the manufacturer's instructions. After final washing, the coverslips were mounted on the slides using 50% (volume fraction) glycerin, and the nuclei were further stained with 4',6-diamidino-2-phenylindole eyotime (DAPI; Sigma-Aldrich, USA). The tissues and cells were observed using a fluorescence microscope (Leica, Germany), and analysis was performed by Image-Pro Plus (V6.0, Media Cybernetics Inc., Bethesda, MA, USA). The apoptotic index was expressed as the ratio of terminal-deoxynucleotidyl transferase-mediated dUTP nick-end labeling (TUNEL)-positive staining nuclei to total nuclei under five random high-power fields (100×).

Cell flow cytometry

For the detection of macrophage phagocytosis, bone marrow-derived macrophages were treated with rmIL-15 for 24 h. Their phagocytic activity was assessed using the Phagocytosis Assay Kit (IgG-PE, Cayman, USA).

To investigate the efferocytosis activity of macrophages, apoptotic cardiomyocytes were labeled with PKH26 (MCE, HY-D1451) and co-cultured with rmIL-15-treated macrophages for 4 h. The PKH26 content in macrophages was then analyzed by flow cytometry.

Lactate dehydrogenase (LDH) activity

The LDH assay (Beyotime Biotechnology, China) was performed according to the manufacturer's instructions. LDH activity was measured at 450 nm using a spectrophotometer. Relative LDH activity was calculated as follows: Relative LDH activity= (sample LDH activity−spontaneous LDH activity)/ (max LDH activity−spontaneous LDH activity) ×100%.

Glycolysis assessment

To evaluate changes in macrophage glycolysis, we measured intracellular and extracellular lactate and adenosine triphosphate (ATP) levels. Both lactate (BC2235) (Solarbio, China) and ATP (BC0305) (Solarbio, China) were quantified using commercially available assay kits purchased from Solarbio, following the manufacturer's instructions. Specifically, cell culture supernatants were used to determine extracellular lactate and ATP levels, while cell lysates were used to measure intracellular levels. The results were quantified by measuring absorbance with a microplate reader.

Luminex liquid suspension chip (LISC)

LISC was performed using the Bio-Plex mouse interleukin kit (Wayen Biotechnologies, Shanghai, China) according to the manufacturer's instructions. Briefly, the culture supernatant was incubated in 96-well plates embedded with microbeads for 1 h, and then detected by antibodies for 30 min. Subsequently, samples were added with streptavidin-PE for 10 min to read values by the Bio-Plex MAGPIX System (Bio-Rad, USA).

Enzyme-linked immunosorbent assay (ELISA)

Samples were harvested to perform ELISA according to the manufacturer's instructions. ELISA kits were as follows: human plasma IL-15 (R&D, USA), human serum troponin T (Roche, Germany), C-reaction protein (CRP) and CK-MB (Leadman, Germany), mouse plasma B-type natriuretic peptide (BNP, Mlbio Corporation, Shanghai, China), and mouse tissue lysate IL-15 (R&D, USA).

Sample size estimation

The sample sizes for animal and experimental groups were predetermined by a formal power analysis prior to the study using G*Power (version v3.1.9.7). Animal's analysis was conducted considering our preliminary experimental results for echocardiographic measurement between wide-type (WT), *IL-15* knockout (KO), and *IL-15Rα* KO in mice. Our clinical research analysis set the sample size estimation by G*Power for the logistic regression model based on five risk factors (IL-15, BMI, hypertension, diabetes, hyperlipidemia), with an incidence rate of approximately 50% for myocardial infarction (MI). Both analyses have a desired power of 0.8 ($1-\beta$ error) and α=0.05.

Statistical analysis

Data are expressed as the mean±standard error of the mean (SEM). Statistical analyses were performed using GraphPad Prism v10.1.2 (GraphPad Software, USA). Significant differences between means were analyzed using the unpaired Student's *t*-test or one-way or two-way analysis of variance (ANOVA) followed by Bonferroni post-test. Categorical data were analyzed using a chi-square test. Analysis of survival was performed using log-rank (Mantel-Cox) tests applied to Kaplan-Meier survival curves. To determine the independent association between IL-15 levels and AMI, a multivariable logistic regression model was employed, adjusting for potential confounders including BMI, hypertension, diabetes, and hyperlipidemia. The results are presented as adjusted odds ratios (aOR) with 95% confidence intervals (CIs). Statistical analyses were performed using SPSS v25.0 (SPSS, USA). A *P*-value <0.05 was considered statistically significant.

Table S3 Echocardiographic analysis of mouse models across time points in each KO group

Time of MI	Grouping	HR (BPM)	LVIDd (mm)	LVIDs (mm)	EF (%)	FS (%)
Baseline	Wild type (*n*=12)	476.9±20.3	2.7±0.3	1.3±0.2	85.4±3.3	53.0±3.9
	IL-15 KO (*n*=11)	493.6±27.4	2.4±0.2	1.1±0.2	87.3±4.6	55.8±7.5
	IL-15Rα KO (*n*=7)	496.3±24.2	2.6±0.3	1.2±0.2	84.1±5.7	51.9±7.2
MI 3D	Wild type (*n*=11)	510.4±31.1	3.4±0.3	2.5±0.4	55.3±7.6	28.1±4.7
	IL-15 KO (*n*=7)	511.9±26.3	2.8±0.4*	1.7±0.3*	66.8±6.3*	35.7±4.8*
	IL-15Rα KO (*n*=6)	514.8±25.5	3.0±0.3*	2.0±0.3*	64.5±5.9*	34.2±4.2
MI 7D	Wild type (*n*=9)	493.9±28.3	3.4±0.3	2.6±0.5	48.9±11.1	24.4±6.7
	IL-15 KO (*n*=7)	510.0±26.6	2.9±0.1*	1.9±0.4*	64.2±9.3*	34.1±6.4*
	IL-15Rα KO (*n*=6)	499.3±29.5	3.1±0.3*	2.0±0.2*	66.4±8.6*	36.0±6.7*
MI 14D	Wild type (*n*=6)	502.3±38.5	3.8±0.3	3.0±0.4	44.7±10.3	22.0±5.8
	IL-15 KO (*n*=5)	523.1±26.6	3.4±0.1	2.4±0.2*	57.7±5.4	29.6±3.6
	IL-15Rα KO (*n*=5)	494.9±36.8	3.2±0.4*	2.1±0.3*	60.8±7.8*	31.8±5.6*

HR: heart rate; BPM: beats per minute; LVIDd: left ventricular internal dimension at end-diastole; LVIDs: left ventricular internal dimension at end-systole; EF: ejection fraction; FS: fractional shortening; MI: myocardial infarction. Continuous data are evaluated by unpaired *t*-test and presented as mean±standard error of the mean (SEM). *P<0.05 vs wild-type.

Table S4 Echocardiographic analysis of mouse models across time points in each group (restoring IL-15)

Time of MI	Grouping	HR (BPM)	LVIDd (mm)	LVIDs (mm)	EF (%)	FS (%)
Baseline	Wild type+PBS (*n*=6)	498.2±32.5	2.5±0.6	1.2±0.5	80.7±6.4	47.8±6.3
	Wild type+IL-15 (*n*=8)	499.7±29.3	2.5±0.2	1.2±0.3	84.0±5.9	51.9±7.8
	IL-15 KO+PBS (*n*=8)	495.8±27.2	2.4±0.5	1.2±0.4	82.5±8.5	50.5±9.8
	IL-15 KO+IL-15 (*n*=8)	495.5±28.7	2.5±0.4	1.2±0.3	82.7±6.9	50.5± 8.5
MI 3D	Wild type+PBS (*n*=6)	519.9±46.9	2.6±0.4	1.7±0.3	61.8±9.2	31.9±5.8
	Wild type+IL-15 (*n*=7)	503.9±25.4	3.4±0.2*	2.7±0.3*	44.4±6.4	21.5±3.6
	IL-15 KO+PBS (*n*=7)	501.8±24.1	2.4±0.3	1.5±0.2	72.2±5.2	39.6±4.2
	IL-15 KO+IL-15 (*n*=6)	507.8±27.2	2.9±0.6	2.0±0.4	56.9±6.2*	28.8±4.0*
MI 7D	Wild type+PBS (*n*=6)	496.0±32.7	2.9±0.3	2.1±0.3	53.8±6.3	26.8±4.0
	Wild type+IL-15 (*n*=6)	512.9±38.1	4.1±0.5*	3.5±0.6*	34.8±9.1*	16.5±4.6*
	IL-15 KO+PBS (*n*=6)	508.1±30.3	2.8±0.3	1.9±0.2	62.9±7.7	32.9±5.4
	IL-15 KO+IL-15 (*n*=5)	456.9±17.4	3.3±0.8	2.7±0.9	42.9±14.7*	20.9±8.1*
MI 14D	Wild type+PBS (*n*=6)	530.5±47.9	3.3±0.4	2.6±0.3	42.7±3.9	20.4±2.3
	Wild type+IL-15 (*n*=6)	490.9±24.0	4.9±0.5*	4.4±0.6*	24.3±6.5*	11.3±3.1*
	IL-15 KO+PBS (*n*=5)	510.0±13.8	3.3±0.6	2.4± 0.5	56.3±8.3	28.8±5.3
	IL-15 KO+IL-15 (*n*=5)	505.8±38.5	3.0±0.6	2.4± 0.4	41.1±10.8*	19.6±6.1*

HR: heart rate; BPM: beats per minute; LVIDd: left ventricular internal dimension at end-diastole; LVIDs: left ventricular internal dimension at end-systole; EF: ejection fraction; FS: fractional shortening; MI: myocardial infarction. Continuous data are evaluated by unpaired *t*-test and presented as mean±standard error of the mean (SEM). *P<0.05 vs wild type and PBS.

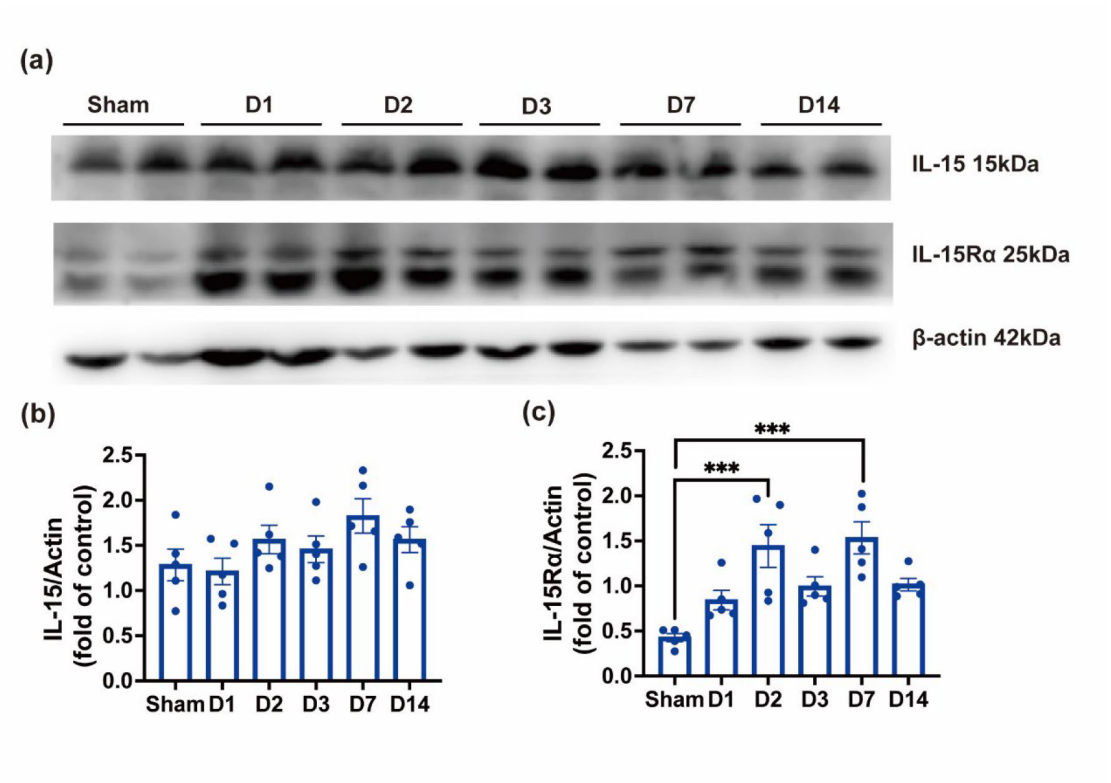

Fig. S1 Interleukin-15 receptor α (IL-15Rα) upregulation without changes in IL-15 levels in remote myocardium. (a) The expressions of IL-15 and IL-15Rα by western blot in the remote area of heart tissue at the different days after acute myocardial infarction (AMI) (*n*=5 for each group). (b, c) Quantitative analysis of expressions of IL-15 (b) and IL-15Rα (c) in the remote area at the different days after AMI by western blot (*n*=5 for each group). * *P*<0.001. Data are expressed as mean±standard error of the mean (SEM).**

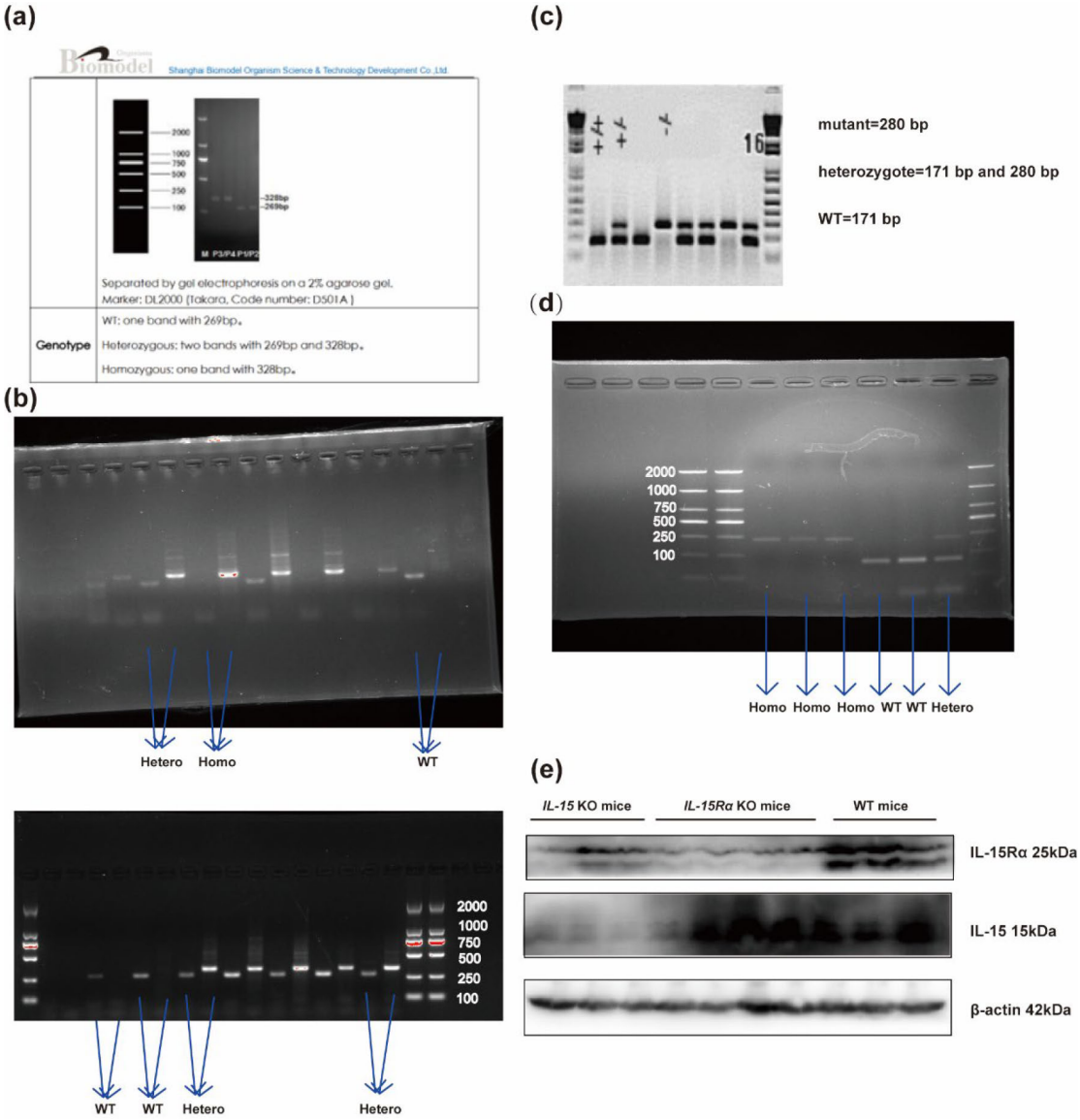

Fig. S2 Genotyping and validation of *interleukin-15* (*IL-15*) and *IL-15 receptor α* (*IL-15Rα*) knockout (KO) mice. (a) Genotyping strategy of *IL-15* KO mice (C57BL/6J). Wild-type (WT) genotype showed a single band at 269 bp, heterozygous (Hetero) genotype displayed two bands at 269 bp and 328 bp, and homozygous knockout (Homo) genotype exhibited a single band at 328 bp. (b) Representative agarose gel electrophoresis confirming genotypes for *IL-15* KO mice. WT, heterozygous, and homozygous mice were labeled accordingly. (c) Genotyping strategy of *IL-15Rα* KO mice (B6/129S). Mutant allele produced a 280 bp band, WT allele generates a 171 bp band, and heterozygous mice display both 171 bp and 280 bp bands. (d) Representative agarose gel electrophoresis confirming genotypes for *IL-15Rα* KO mice. WT, heterozygous, and homozygous mice were labeled accordingly. (e) Western blot analysis validating the knockout efficiency.

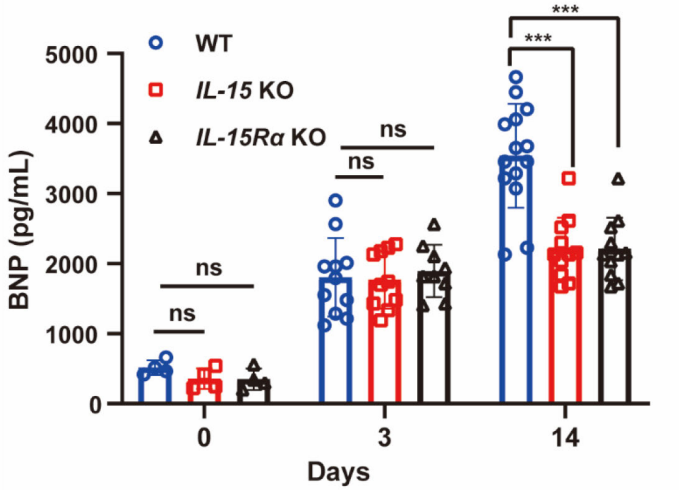

Fig. S3 Plasma B-type natriuretic peptide (BNP) levels in mice over time. Plasma levels of BNP measured in wild-type (WT, blue circles), *interleukin-15* knockout (*IL-15* KO, red squares), and *IL-15 receptor α* (*IL-15Rα*) KO (black triangles) *IL-15* KO (red squares), and *IL-15Rα* KO (black triangles) mice at 0, 3, and 14 d. $^{***}P<0.001$. Data are expressed as mean±standard error of the mean (SEM).

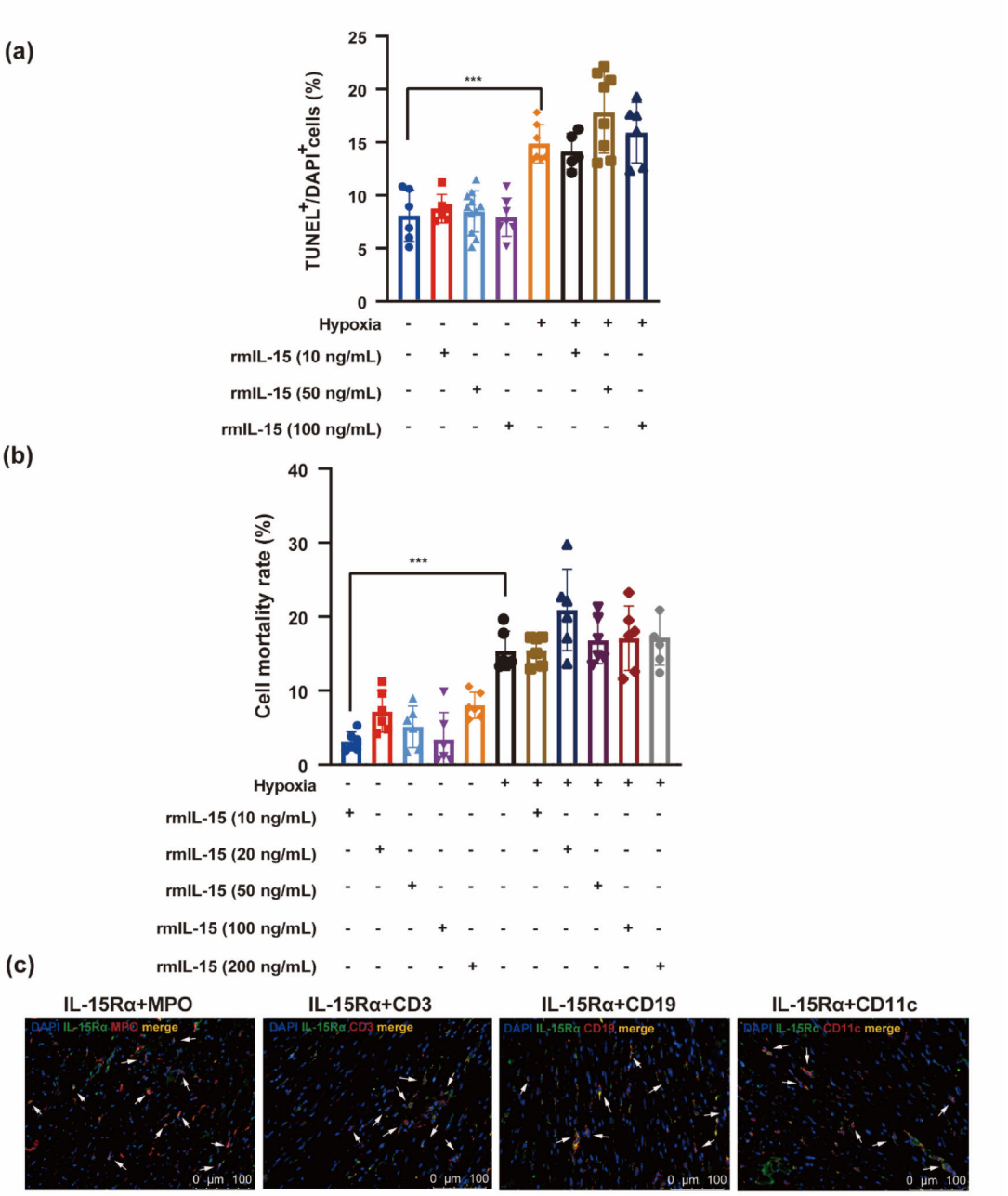

Fig. S4 Impact of interleukin-15 (IL-15) on cardiomyocyte apoptosis and inflammation. (a) Quantification of TUNEL-positive cells under normoxic and hypoxic conditions with or without recombinant murine IL-15 (rmIL-15) treatment. (b) Cell mortality rates (%) under normoxic and hypoxic conditions with rmIL-15 treatment. (c) Representative immunofluorescence staining of IL-15Rα with MPO (neutrophil marker), CD3 (T cell marker), CD19 (B cell marker), CD11c (dendritic cell marker), and DAPI (nuclear) in the heart section of wild-type (WT) mice on the third day after AMI. Positive stained cells are indicated as white arrows. ***P<0.001. Data are expressed as mean±standard error of the mean (SEM).

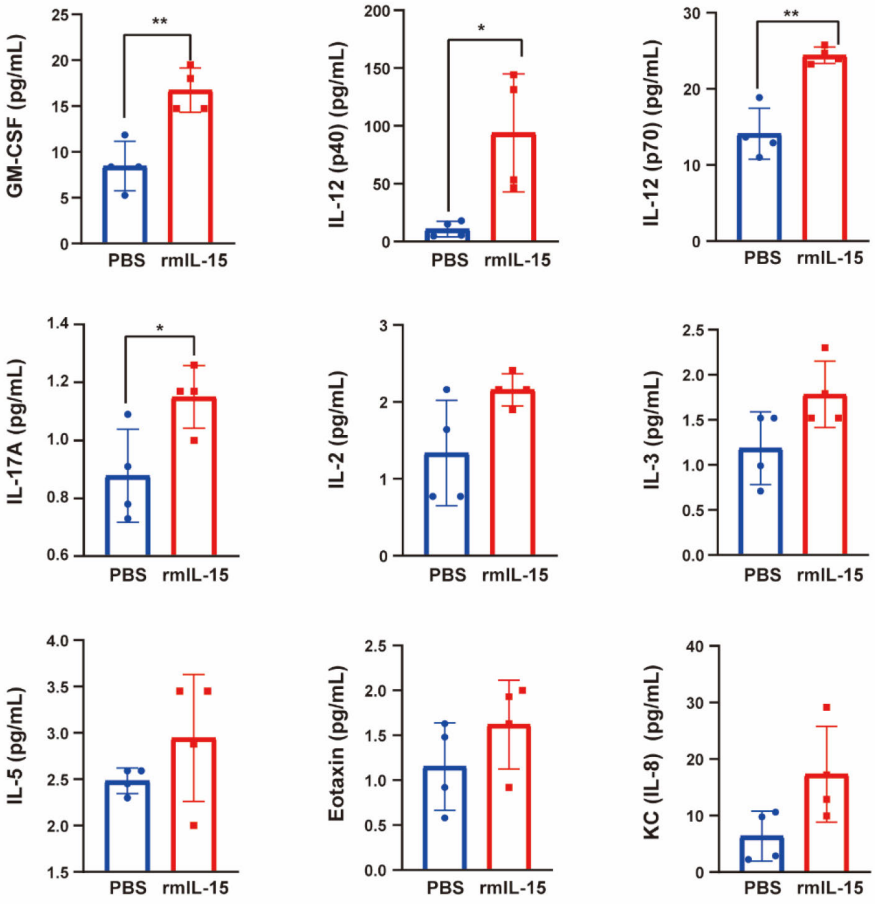

Fig. S5 Cytokine and chemokine profiles in response to recombinant murine interleukin-15 (rmIL-15) treatment. Cytokine and chemokine levels were measured in bone marrow-derived macrophages (BMDMs) treated with PBS or rmIL-15. GM-CSF: granulocyte-macrophage colony stimulating factor; * *P*<0.05, ** *P*<0.01. Data are expressed as mean±standard error of the mean (SEM).