Supplement Materials

Materials and Methods

Human samples

All procedures involving human samples and cells were conducted in compliance with the principles outlined in the Declaration of Helsinki and was approved by the Institutional Review Boards of the Third Military Medical University in Chongqing (No. KY24173). Control lower extremity artery samples were obtained from patients with lower limb trauma who had undergone amputation. Atherosclerotic lower extremity arteries were collected from patients having ischaemia of lower limb due to atherosclerosis who received amputation surgery. Serum samples were collected from patients with vascular disorders, including coronary artery disease (CAD), non-CAD disorders (hypertension, acute aortic syndrome, aneurysm, and cerebrovascular or peripheral vascular diseases), and healthy controls. All participants provided written informed consent. Detailed information on patients with vascular disease and healthy controls is provided in **Table S1**.

Animals

All animal experimental procedures were carried out in accordance with protocols approved by the Third Military Medical University Animal Care and Use Committee (No. AMUWEC20234713). $MG53^{flox/flox}$ mice were purchased from GemPharmatech Co., Ltd., Jiangsu, China (stock number: T005421). SM22-Cre mice were obtained from The Jackson Laboratory (ME, USA; stock number: 004746). MG53 SMC-KO mice were generated by crossing $MG53^{flox/flox}$ mice with SM22-Cre mice. Genomic DNA was extracted from mouse tail-tips for genotyping, and primer sequences for PCR are listed in **Table S2**. Male animals were used in our study based on prior investigations into MG53 function[1, 2]. Sample size was determined with reference to previous studies employing vascular injury models, and all procedures involving animals adhered to randomization and blinding procedures[3, 4].

Rat Carotid Artery Injury Model

Rat carotid artery neointima formation was induced by balloon injury as priorly described[3, 5]. Male Sprague-Dawley rats weighing 220 to 250g were anesthetized by a single intraperitoneal injection (i.p.) of pentobarbital sodium (Sigma-Aldrich, 50 mg/kg body weight), and left common carotid arteries were carefully exposed through a midline cervical incision thereafter. Blood flow transiently stooped by occluding the arteries with vascular clamps, and then a small arteriotomy was made around the external carotid artery. Afterwards, the vessels were completely injured near the carotid bifurcation using a balloon embolectomy catheter (1.5F, Cordis, FL, USA) through the incision hole. Once injured, the wounded skin was closed with a single suture. They were housed under standard laboratory conditions at the Animal Center of the Third Military Medical University. At the indicated time points, animals were euthanized, and carotid arteries were harvested and processed for RT-PCR, Western blot, and immunohistochemistry. Carotid arteries harvested from sham-operated rats served as control. For in vivo rhMG53 treatment studies, rats were treated intravenously with 1mg/kg rhMG53 or saline vehicle every other day. The purification of rhMG53 protein has been reported previously[6]. Carotid arteries were collected two weeks after surgery following euthanasia. For euthanasia, rats received an overdose of pentobarbital sodium (300 mg/kg, i.p.).

Mouse Femoral Artery Injury Model

Mouse femoral artery neointima formation was induced by wire injury as described[5, 7].Male mice aged 8 week were anesthetized with a single injection of ketamine (100mg/kg, i.p.) and xylazine (10 mg/kg, i.p.), and a groin incision was made under a surgical microscope. The left femoral artery was carefully exposed, and vascular clamps were used to interrupt arterial blood flow. A 0.25 mm angioplasty guidewire (Abbott Vascular, CA, USA) was then inserted into the arterial lumen and advanced to the level above the bifurcation of the abdominal aorta. The guidewire was gently pulled back and forth, and the process was repeated 3 times. Once after the guidewire and vascular clamps were removed, the branch of the femoral artery was ligated, and the skin incision was closed. Sham control mice underwent the same procedures

without passage of the guide wire. MG53 SMC-KO mice were orally treated with GSK2837808A (MCE, NJ, USA; HY-100681; 6 mg/kg, once daily) or vehicle[8]. For euthanasia, mice were sacrificed via cervical dislocation while under anesthesia (induced by the same ketamine/xylazine regimen described above).

Cell culture

Primary rat aortic smooth muscle cells (SMCs) were isolated from thoracic aortas of male Sprague-Dawley rats via adherent culture method, as previously described[9]. Briefly, after rats were anesthetized with a single injection of pentobarbital sodium (Sigma-Aldrich, 50 mg/kg body weight, i.p.), thoracic aortas were carefully dissected free from the surrounding tissues under sterile condition, and sequentially rinsed in phosphate buffered saline (PBS) and Hanks balanced salt solution (Gibco, NY, USA; 14175095). The adventitia was carefully removed and endothelial cells were scraped off. Then, the tissue was cut into small pieces (1-2 mm) and digested in collagenase (Sigma-Aldrich [Roche], Mannheim, Germany; 11088858001) for 20 minutes. After discarding the collagenase, the pieces were neutralized with serum (Gibico, A5669701). Using a pipette tip, the tissue pieces were gently and evenly distributed on the bottom of a 25T cell culture flask, after which 4 mL of complete culture medium was added to the opposite side. Following a 2-hour incubation, the flask was carefully inverted so that the medium covered the tissue pieces. The culture was then maintained statically for 1-2 weeks. Once cells had grown to 90% confluence, they were detached and passaged using Ham's F12K (Gibico, 21127022) medium supplemented with 10% FBS and 1% penicillin–streptomycin (Gibco, 15140122). The culture method for MG53 SMC-KO mice and their littermate controls was the same as that described above for rat. The purity of the isolated SMCs was confirmed via immunofluorescence staining for α-smooth muscle actin (Abcam, Cambridge, UK; ab7817). SMCs were used at passages 3 through 7.

Human aortic SMCs obtained from American Type Culture Collection (ATCC) were cultured in SMC medium (ScienCell, CA, USA; 1101), supplemented with 10% FBS, 1% penicillin/streptomycin at 37℃ in 5% CO_2 atmosphere.

Measurement of serum MG53, lactate, and LDHA activity

Peripheral blood samples were collected from patients following clinical diagnosis and centrifuged at 2000 rpm for 20 minutes to isolate serum. Serum samples were immediately aliquoted and stored at -80℃ until further analysis. Serum MG53 concentrations were quantified using a human MG53 enzyme-linked immunosorbent assay (ELISA) kit (Mmbio, Jiangsu, China; MM-64291H1) according to the manufacturer's instructions. Lactate concentrations were determined using a lactate assay kit (Sigma-Aldrich, MO, USA; MAK570) following the manufacturer's instructions. LDHA activity was determined using a commercially available assay kit (Sigma-Aldrich, MAK066) according to the manufacturer's instructions. The assay quantified LDHA-mediated conversion of pyruvate to lactate by monitoring the rate of NADH oxidation spectrophotometrically at 570 nm. Enzyme activity measurements were normalized to total protein content. All samples were analyzed by investigators blinded to patient information.

RNA Isolation and RT-PCR

Total RNA was extracted from tissues and confluent cells using TRIZOL reagent (TaKaRa, Kusatsu, Japan; 9108) following the manufacturer's protocol. Subsequently, RNA (1000 nmol) was reverse transcribed using the PrimeScript RT reagent Kit with gDNA Eraser (TaKaRa, RR047). Real-time PCR based on SYBR Green (TaKaRa, CN830) was conducted in 20 μL reactions using an Applied Biosystems 7500 real-time PCR system (Applied Biosystems, CA, USA) with 10pmol primers from Shangon Biotech (Shanghai, China). Quantitative analysis of expression level changes was performed using the comparative Ct method. Detailed primer sequences are available in **Table S2**.

Western Blot Analysis

Cells were rinsed twice with pre-cold PBS and subsequently collected on ice in RIPA buffer (Beyotime, Shanghai, China; P0013B) containing 1% phenylmethane sulfonyl fluoride (Beyotime, ST506). Tissue samples were powdered in liquid nitrogen, and lysates were prepared as previously outlined[10]. Protein samples were mixed with 5× SDS-PAGE loading

buffer (Beyotime, P0015) and boiled for 5 minutes. Samples including both target proteins and the internal standard were loaded onto the same SDS-PAGE gel to achieve consistent loading across all lanes, which were electrophoresed at a constant voltage (120V running, 80V stacking). Equal sample volumes were applied to each lane, and protein concentrations were determined using BCA assay to verify uniform loading across samples. In cases where target proteins have similar molecular weights, separate Western blots were performed, with a loading control included in each blot to confirm consistent loading across all lanes in each experiment. Following electrophoresis, proteins were transferred onto a polyvinylidene difluoride membrane (PVDF, Millipore, MA, USA), and subsequently blocked with 5% bovine serum albumin (BSA; Boster, Wuhan, China; AR0004) for 1 hour at room temperature. Membranes were stained at 4℃ overnight with primary antibodies as follows: MG53 (Abcam, ab307593, 1:500), IR1 (Proteintech, 20433-1-AP, 1:300), IRS1 (CST, MA, USA; 3407S, 1:500), $p27^{Kip1}$ (CST, 3686S, 1:500), PCNA (CST, 13110S, 1:1000), Cyclin D1 (Abcam, ab134175, 1:1000), MYH11 (Proteintech, IL, USA; 21404-1-AP, 1:500), CNN1 (Proteintech, 13938-1-AP, 1:500), Tagln (Proteintech, 10493-1-AP, 1:500), SMA (Abcam, ab7817, 1:500), GAPDH (CST, 2118S, 1:1000), HK (Proteintech, 22029-1-AP, 1:500), PFK1 (proteintech, 13389-1-AP, 1:500), PKM1 (Proteintech, 15821-1-AP, 1:500), PKM2 (Proteintech, 15822-1-AP, 1:500), LDHA (Proteintech, 19987-1-AP, 1:1000), and β-actin (Proteintech, 66099-1-AP, 1:1000) (**Table S3**). The next day, blots were washed five times for 5 minutes each with Tris-buffered saline containing Tween-20 (Beyotime), followed by incubation for 1 hour at room temperature with the mouse secondary antibodies conjugated to horseradish peroxidase (Abcam, ab205719, 1:5000) or rabbit secondary antibodies conjugated to horseradish peroxidase (Abcam, ab6721, 1:5000). Detection was performed using enhanced chemiluminescence (Amersham International, Buckinghamshire, UK). Quantification of protein bands was carried out using Quantity One software, with normalization to corresponding loading controls.

Immunofluorescence

Prior to serum deprivation, SMCs were cultured on coverslips in 6-well plates at a density of approximately 1.2 × 10^5 cells per well for 24 hours. Cells adhered to coverslips were fixed with 4% ice-cold paraformaldehyde for 15 minutes at room temperature, permeabilized with 1% Triton X-100, and rinsed with PBS. After three PBS rinses, cells were blocked for 20 minutes using 5% bovine serum albumin (BSA). For dual immunofluorescence staining, cells were initially incubated overnight at 4℃ with primary antibodies against MG53 (Abcam, ab307593, 1:100) or LDHA (Abcam, ab135396, 1:100) (**Table S4**). Subsequently, Cy3-conjugated and FITC-conjugated secondary antibodies (Thermo Fisher Scientific) were added. After nuclear counterstaining with 4',6-diamidino-2-phenylindole (DAPI) (Abcam, ab104139), the cells were imaged using an Olympus confocal microscope.

Mitochondrial extracellular acidification rate (ECAR) and oxygen consumption rate (OCR) and measurement

ECAR and OCR of primary SMCs were measured by using an XFe24 Extracellular Flux Analyzer, as priorly described[11]. Primary SMCs were seeded into XFe96 plates at a density of 1.0 × 10^4 cells per well overnight and cultured in Ham's F12K supplemented with 10% FBS. ECAR was measured using Seahorse Glycolysis Stress Test Kit (Agilent, CA, USA; 103020-100). Glycolytic capacity was defined as the difference between the ECAR following oligomycin (2 μM) injection and basal ECAR. Glycolytic reserve was calculated as the increase in ECAR from the glucose (80 mM) injection to the oligomycin (2 μM) injection. OCR was measured using Seahorse Mito Stress Test Kit (Agilent, 103015-100) according to the manufacturer's instructions. Baseline OCR was recorded first, followed by sequential injections of oligomycin (2 μM) to determine ATP-linked respiration, the uncoupler FCCP (2 μM) to assess maximal respiration, and a combination of rotenone (1 μM) and antimycin A (1 μM) to define non-mitochondrial respiration. ECAR and OCR values were normalized to a protein concentration of 100 μg per well.

Mass spectrometry

The slab of the SDS-PAGE gel was rinsed twice with 1000 µL H_2O for 1 hour. Bands were excised with a clean scalpel and cut into cubes. Gel pieces were transfered into a new low binding eppendorf tube and spined down with microcentrifuge. Add 1000 µL decolorization solution to decolorize until the colloidal particles are colorless, remove the decolorization solution. Gels were then incubated with 800 µL of neat ACN for 10 minutes until gel pieces shrink. After removing of all liquids, 600 uL of 5 mM DTT solution was added to completely cover gel pieces for 30 minutes at 55℃. Tubes chilling down to room temperature, incubated with 800 µL of ACN for 10 minutes. For alkylation, 600 µL of 15 mM IAA solution was incubated with gels for 40 minutes at room temperature in the dark. Alkylated protein gels were shrinked with ACN and collected with microcentrifuge. Trypsin digestion was proformed by saturating the gels for 30 minutes with 600 µL trypsin buffer, then additional trypsin buffer covering the gels for 30 minutes, then incubating over night at 37℃. Peptides were extracted with 800 µL of extraction buffer and vaccum dried. Peptides were resuspended with 0.1% formic acid, desalted with C18 tip and stored in 0.1% formic acid for LCMS analysis. For each sample, 5 µL of total peptides were separated and analyzed with a nano-UPLC (EASY-nLC1200) coupled to a Q Exactive HFX Orbitrap instrument (Thermo Fisher Scientific) with a nanoelectrospray ion source. Separation was performed using a reversedphase column. Separation of sample was executed with a 60 minutes gradient at 300 nL/min flow rate. Data dependent acquisition (DDA) was performed in profile and positive mode with Orbitrap analyzer at a resolution of 120000 and m/z range of 350-1600 for MS1; For MS2, the resolution was set to 15000 with a dynamic first mass. The automatic gain control target for MS1 was set to 3E6 with max IT 50 ms, and 1E5 for MS2 with max IT 110 ms. The top 10 most intense ions were fragmented by HCD with normalized collision energy of 27%, and isolation window of 1.2 m/z. The dynamic exclusion time window was 45 seconds, single charged peaks and peaks with charge exceeding 6 were excluded from the DDA procedure.

Coimmunoprecipitation

For immunoprecipitation, a specialized lysis buffer was employed to prepare whole protein

extracts from SMCs. Following centrifugation, protein concentrations were determined using the BCA kit from Millipore. The lysates were gently rocked overnight at 4℃. Next, the lysates were incubated with Protein G Magnetic Beads (CST, 70024S) pre-bound with anti-MG53 (Abcam, ab307593, 1:100), anti-LDHA (Abcam, ab135396, 1:100), or anti-P300 (Abcam, ab275378, 1:50) antibodies for 2 hours. Approximately 3 μL of antibodies was used per 400 μg of total protein. The precipitated proteins underwent 5 washes with cell lysis buffer (CST, 9803S), followed by boiling in 2× sample loading buffer (CST, 7722S). These proteins were separated using 15% or 12% SDS-PAGE gels. Subsequently, the separated proteins were transferred onto a PVDF membrane and immunoblotted with anti-MG53, anti-LDHA (Proteintech, 19987-1-AP, 1:1000), anti-P300 or anti-acetyl-lysine (PTM Biolabs, Chicago, IL, USA; PTM-105) antibodies as required. Normal IgG (CST, 2279S) served as a negative control.

LC-MS/MS analysis

Metabolites were extracted from cellular samples using chilled methanol-based solvents containing internal standards. Solid samples underwent bead-beating homogenization (40 Hz, 4 minutes) and sonication in an ice-water bath (3 cycles), followed by incubation at -40℃ for 1 hour to precipitate proteins. After centrifugation (12000 rpm, 4℃, 15 min), the supernatant was dried under vacuum and reconstituted in methanol:acetonitrile:water (1:1:2, v/v/v) for analysis. Targeted metabolomics was performed on an Agilent 1290 UPLC system coupled with an AB Sciex Triple Quad 6500+ mass spectrometer. Chromatographic separation was achieved using ACQUITY BEH C18 and Atlantis Premier BEH Z-HILIC columns with optimized mobile phases (0.1% FA or ammonium acetate/formate buffers). Data were processed via SCIEX Analyst (1.7.3) and BIOTREE Bio Bud (v2.0.4). The method was validated following FDA guidelines, demonstrating excellent linearity ($R^2 > 0.99$) and a wide dynamic range, with LODs and LOQs spanning 0.49-9375 nmol/L and 0.98-18750 nmol/L, respectively, ensuring robust quantification across diverse biological matrices.

Lentivirus production and transfection

Lentiviral vectors overexpressing MG53 or LDHA, lentiviruses expressing P300-targeted siRNA, and their respective GFP-expressing controls were purchased from Hanbio Biotechnology (Shanghai, China). Cells were seeded in 6-well plates at a density of 2×10^5 cells per well and cultured in ham's F12K medium supplemented with 10% FBS for 24 hours before transfection. Following rinsing with PBS, the virus or corresponding controls, diluted in ham's F12K medium with 10% FBS, were added to cells. After 24 hours, culture medium was replaced with fresh complete growth medium, and transfection efficiency was assessed by measuring luciferase activity (transfection efficiency >95% was considered successful).

Cell proliferation and migration

SMC proliferation was assessed using a 5-ethynyl-2'-deoxyuridine (EdU) assay kit (GeneCopoeia, MD, USA; AB2508A1). Cells were seeded in 6-well plates, and EdU was added during the final 8 hours of stimulation. The extent of EdU incorporation was quantified using flow cytometry according to the manufacturer's instructions. For migration assays, a modified Boyden chamber with a 4μm pore size (Sigma-Aldrich, CLS3422) suitable for 24-well plates was utilized. Prior to seeding in the upper chamber, cells were pre-treated with mitomycin C (Sigma-Aldrich, M5353) for 2 hours to inhibit proliferation. The lower chamber was filled with serum-free F12K medium (800μl) containing PDGF-BB (10ng/mL) (R&D Systems, 520-BB). Following a 24-hour incubation period, non-migrated cells were gently removed using an alcohol-soaked cotton swab, while migrated cells were fixed and stained with 95% alcohol and 1% crystal violet, respectively. Images were captured using an Olympus light microscope.

Statistical Analysis

All data are presented as means±SEM from 3 or more independent experiments. Statistical analyses were conducted using GraphPad Prism 8.3.0 (GraphPad Software, USA). The Shapiro-Wilk test or Kolmogorov-Smirnov test was used to assess data normality. For data following a normal distribution, significant differences were determined using unpaired

Student's t-test (two groups) and analysis of variance (ANOVA) followed by Tukey's multiple comparison test (multiple groups). For non-normally distributed data or experiments with a small sample size ($n<6$), non-parametric tests were used, i.e., Mann-Whitney test to compare two groups, and Kruskal-Wallis test followed by Dunn's multiple comparisons test to analyze data from multiple groups. Correlations were analyzed by Spearman's correlation coefficient (r_S). $P<0.05$ was considered statistically significant.

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Table S1. Clinical characteristics of patients with vascular disorders and controls

Category	Sex	Age (years)	NYHA	LVEF (%)	Diabetes (Y/N)	BNP (pg/ml)	MG53 (pg/ml)
Acute aortic syndrome-1	Male	80	-	50	N	1262	48.91
Acute aortic syndrome-2	Male	75	-	60	N	329	45.98
Acute aortic syndrome-3	Female	56	-	65	N	50.4	38.69
Acute aortic syndrome-4	Male	47	-	55	N	-	33.96
Acute aortic syndrome-5	Male	51	-	58	N	389	69.3
Acute aortic syndrome-6	Male	36	-	61	N	200	45.86
Acute aortic syndrome-7	Male	52	-	63	N	168	45.03
Acute aortic syndrome-8	Male	59	-	61	N	186	51.28
Acute aortic syndrome-9	Male	84	-	64	N	813	56.31
Acute aortic syndrome-10	Male	66	-	59	N	72.3	113.9
Aneurysm-1	Male	81	-	68	N	36.2	44.14
Aneurysm-2	Male	70	-	65	N	27.39	49.08
Aneurysm-3	Male	60	-	67	N	-	109
Cerebrovascular disease-1	Female	71	-	60	Y	138	89.65
Cerebrovascular disease-2	Male	60	-	61	Y	-	82.62
Cerebrovascular disease-3	Female	84	-	-	N	-	103.1
Cerebrovascular disease-4	Female	84	-	74	Y	76	103.1
Cerebrovascular disease-5	Male	76	-	70	N	-	95.75
Cerebrovascular disease-6	Male	78	-	-	N	-	100.3
Cerebrovascular disease-7	Male	65	-	-	N	<50	128.8
Cerebrovascular disease-8	Male	81	-	-	N	-	94.02
Cerebrovascular disease-9	Female	53	-	72	N	-	91.02
Cerebrovascular disease-10	Female	72	-	-	N	-	97.79
Cerebrovascular disease-11	Female	60	-	-	N	-	89.98
Cerebrovascular disease-12	Female	67	-	-	Y	-	98.65
Cerebrovascular disease-13	Female	68	-	66	N	-	140
Cerebrovascular disease-14	Male	59	-	-	Y	-	82.63
Cerebrovascular disease-15	Female	69	-	-	N	-	86.17
Cerebrovascular disease-16	Female	53	-	-	N	-	94.08
Cerebrovascular disease-17	Male	67	-	-	N	-	113.9
Cerebrovascular disease-18	Female	42	-	-	N	-	95.29
Cerebrovascular disease-19	Male	67	-	72	Y	-	84.76
Cerebrovascular disease-20	Female	69	-	76	Y	-	84.09

Cerebrovascular disease-21	Female	84	-	-	N	-	100.1
Cerebrovascular disease-22	Female	83	-	60	N	29.4	105.6
Cerebrovascular disease-23	Male	60	-	-	N	-	79.84
Cerebrovascular disease-24	Female	57	-	59	N	-	111.32
Cerebrovascular disease-25	Male	55	-	67	N	-	79.09
Cerebrovascular disease-26	Female	80	-	-	N	-	87.27
Cerebrovascular disease-27	Male	54	-	-	N	-	105
Cerebrovascular disease-28	Male	75	-	-	N	-	86.52
Cerebrovascular disease-29	Male	39	-	-	N	-	116.7
Cerebrovascular disease-30	Female	64	-	-	N	-	90.53
Cerebrovascular disease-31	Male	70	-	-	Y	-	76.75
Cerebrovascular disease-32	Male	79	-	61	N	619.28	78.31
Cerebrovascular disease-33	Male	77	-	59	Y	-	117.4
Cerebrovascular disease-34	Male	87	-	66	Y	<50	106.3
Cerebrovascular disease-35	Male	80	-	-	N	-	106.7
Cerebrovascular disease-36	Male	72	-	62	N	-	104.8
Cerebrovascular disease-37	Male	49	-	59	Y	-	99.36
Cerebrovascular disease-38	Male	49	-	64	N	-	95.85
Cerebrovascular disease-39	Male	74	-	-	N	-	116
Cerebrovascular disease-40	Female	61	-	65	N	-	50.07
Cerebrovascular disease-41	Male	55	-	-	Y	-	101.7
Cerebrovascular disease-42	Female	54	-	-	Y	-	111.8
Cerebrovascular disease-43	Female	74	-	-	N	-	101.7
Cerebrovascular disease-44	Female	71	-	-	N	-	125.7
Coronary artery disease-1	Male	53	II	59	N	420.85	111.5
Coronary artery disease-2	Male	66	II	65	N	322.93	100.5
Coronary artery disease-3	Female	70	II	57	N	222.47	76.82
Coronary artery disease-4	Male	70	II	-	N	128.49	86.18
Coronary artery disease-5	Male	59	II	69	N	102.85	50.55
Coronary artery disease-6	Female	81	II	59	Y	189.48	81.47
Coronary artery disease-7	Male	71	II	62	Y	807.92	139
Coronary artery disease-8	Female	61	II	62	N	118.83	142.5
Coronary artery disease-9	Male	82	II	68	N	400	65.74
Coronary artery disease-10	Male	43	II	55	N	<50	111.1
Coronary artery disease-11	Male	50	II	66	Y	122.97	100.6
Coronary artery disease-12	Male	60	II	66	N	276.06	77.17
Coronary artery disease-13	Female	77	II	46	Y	213.43	98.11
Coronary artery disease-14	Male	62	II	57	N	198.31	93.48
Coronary artery disease-15	Female	70	II	63	Y	37.67	79.76

Coronary artery disease-16	Male	58	I	58	N	84.5	108.9
Coronary artery disease-17	Male	61	II	61	N	50.42	86.34
Coronary artery disease-18	Male	62	I	58	Y	64.34	124.8
Coronary artery disease-19	Male	72	II	71	N	197.8	83.49
Coronary artery disease-20	Male	82	III	46	Y	3016	88.13
Coronary artery disease-21	Male	55	II	65	Y	38.42	108.8
Coronary artery disease-22	Male	51	II	53	N	65	111.6
Coronary artery disease-23	Male	70	II	71	N	<50	92.57
Coronary artery disease-24	Female	76	II	59	N	64	104.5
Coronary artery disease-25	Male	69	II	61	Y	773.93	100.3
Coronary artery disease-26	Female	57	II	63	Y	128.8	99.79
Coronary artery disease-27	Male	58	II	69	N	72.98	103.9
Coronary artery disease-28	Female	58	III	66	N	226	95.7
Coronary artery disease-29	Male	69	II	72	Y	222.94	132
Coronary artery disease-30	Female	75	II	62	Y	162.19	101.2
Coronary artery disease-31	Male	51	II	63	Y	48.94	127.2
Coronary artery disease-32	Male	62	II	67	Y	-	95.05
Coronary artery disease-33	Male	65	II	57	N	73.68	83.17
Coronary artery disease-34	Male	78	II	64	Y	150.21	61.05
Coronary artery disease-35	Female	78	II	66	N	636.67	111.1
Coronary artery disease-36	Male	78	II	65	N	827.89	108.8
Coronary artery disease-37	Male	53	III	28	Y	5132.52	129.6
Coronary artery disease-38	Female	64	II	70	Y	97.79	142.5
Coronary artery disease-39	Male	60	II	61	N	60.66	119.2
Coronary artery disease-40	Male	45	II	50	Y	580.33	112.6
Coronary artery disease-41	Male	77	II	48	N	600.2	109
Coronary artery disease-42	Male	73	II	77	N	65.96	54.8
Coronary artery disease-43	Male	49	II	34	N	41.9	185.8
Coronary artery disease-44	Male	52	I	70	Y	40.98	107.7
Coronary artery disease-45	Male	59	II	68	Y	102.67	120.1
Coronary artery disease-46	Male	61	I	58	N	127	44.19
Coronary artery disease-47	Male	75	II	57	N	327.87	107.6
Coronary artery disease-48	Male	64	II	59	N	-	66.07
Coronary artery disease-49	Female	62	II	67	N	343.89	68.12
Coronary artery disease-50	Male	81	III	65	N	170.29	110
Coronary artery disease-51	Male	60	II	67	Y	134.44	90.11
Coronary artery disease-52	Female	58	II	67	N	65.97	119.5
Coronary artery disease-53	Male	59	II	35	Y	772.98	82.82
Coronary artery disease-54	Male	54	III	34	N	681.32	69.54

Coronary artery disease-55	Male	68	III	28	Y	682	105.3
Coronary artery disease-56	Male	72	II	73	N	146.92	112.1
Coronary artery disease-57	Male	62	II	63	N	51	89.05
Coronary artery disease-58	Male	61	II	59	Y	<50	86.53
Coronary artery disease-59	Male	69	II	60	N	77.58	107.6
Coronary artery disease-60	Male	77	II	70	N	252.36	103.9
Coronary artery disease-61	Male	50	II	57	Y	107.47	144.2
Coronary artery disease-62	Male	64	I	64	N	66.71	108.1
Coronary artery disease-63	Male	67	III	62	Y	935.37	75.46
Coronary artery disease-64	Male	70	II	63	N	66.74	98.91
Coronary artery disease-65	Male	56	II	42	Y	88.26	130.8
Coronary artery disease-66	Male	46	I	63	Y	29.67	114.3
Coronary artery disease-67	Male	55	II	62	Y	34.55	104.1
Coronary artery disease-68	Male	50	II	50	Y	443.24	82.08
Coronary artery disease-69	Male	71	II	43	N	5755.85	96.99
Coronary artery disease-70	Male	48	III	61	N	67.87	93.26
Coronary artery disease-71	Male	53	II	29	N	5935.93	105.2
Coronary artery disease-72	Male	61	II	61	N	50.42	107.8
Coronary artery disease-73	Male	56	II	56	Y	105.4	104.2
Coronary artery disease-74	Male	76	II	67	Y	410.29	130.3
Coronary artery disease-75	Male	60	II	59	Y	757.96	121.7
Coronary artery disease-76	Male	48	II	65	N	47.1	148.3
Coronary artery disease-77	Male	81	II	66	N	332.01	107.8
Coronary artery disease-78	Male	73	II	63	N	138.76	115.9
Coronary artery disease-79	Male	81	II	67	N	95.14	116.3
Coronary artery disease-80	Male	68	IV	46	Y	31030.71	108.8
Coronary artery disease-81	Male	77	III	46	N	1136.7	133.8
Coronary artery disease-82	Female	65	III	70	Y	137.25	97.73
Coronary artery disease-83	Male	55	II	58	N	273.78	101.3
Coronary artery disease-84	Male	68	II	66	Y	395.18	91.62
Coronary artery disease-85	Female	70	II	62	N	621.53	133.3
Coronary artery disease-86	Male	75	II	46	Y	261.84	110.9
Coronary artery disease-87	Male	52	I	64	N	59.88	89.74
Coronary artery disease-88	Male	61	II	62	N	55.6	146.9
Coronary artery disease-89	Female	57	I	-	N	-	134.2
Coronary artery disease-90	Male	53	II	68	N	77.85	129.1
Coronary artery disease-91	Female	69	II	65	Y	68.75	149.3
Coronary artery disease-92	Male	55	II	66	Y	34.09	140.9
Coronary artery disease-93	Male	63	II	58	Y	60.44	141.4

Coronary artery disease-94	Male	52	II	-	N	<50	146.1
Coronary artery disease-95	Female	70	II	63	N	141.38	128.1
Coronary artery disease-96	Male	66	II	67	N	177.27	148.7
Coronary artery disease-97	Female	77	II	66	N	243	129.7
Coronary artery disease-98	Female	63	II	61	Y	143.27	135.4
Coronary artery disease-99	Male	77	II	67	N	143.47	130
Coronary artery disease-100	Male	71	II	72	Y	424.93	57.02
Coronary artery disease-101	Male	70	II	63	Y	2247.21	192.3
Coronary artery disease-102	Male	55	II	67	N	63.66	194.6
Coronary artery disease-103	Male	72	I	69	Y	142.51	47.58
Coronary artery disease-104	Female	77	III	42	Y	6736.51	127.8
Coronary artery disease-105	Female	82	II	56	Y	83	54.95
Coronary artery disease-106	Female	52	II	62	N	267.39	144.2
Coronary artery disease-107	Male	50	II	73	Y	<50	106.2
Coronary artery disease-108	Male	51	II	69	N	177.24	80.57
Coronary artery disease-109	Male	54	II	60	N	50	45.71
Coronary artery disease-110	Male	66	II	72	N	588.37	101.6
Coronary artery disease-111	Female	62	II	63	Y	138.65	132
Coronary artery disease-112	Female	72	II	77	Y	52.79	153.1
Coronary artery disease-113	Male	58	II	43	Y	175	160.1
Coronary artery disease-114	Male	60	II	70	N	204.93	98.63
Coronary artery disease-115	Male	70	II	70	N	119.24	167
Coronary artery disease-116	Male	67	I	66	Y	156.82	81.71
Coronary artery disease-117	Male	44	II	69	N	125.36	187.9
Coronary artery disease-118	Male	61	III	67	N	<50	131.1
Coronary artery disease-119	Male	71	II	64	N	104.99	158.1
Coronary artery disease-120	Female	70	II	57	N	1406.65	147.8
Coronary artery disease-121	Male	51	II	64	N	124.77	131.7
Coronary artery disease-122	Male	78	II	56	N	257.14	123
Coronary artery disease-123	Male	73	III	33	N	2214.22	168.9
Coronary artery disease-124	Female	79	II	72	Y	428.47	167.7
Coronary artery disease-125	Male	68	II	66	N	79.33	400
Coronary artery disease-126	Female	75	II	-	Y	2028.55	205.1
Coronary artery disease-127	Male	65	II	40	N	1570	154.8
Coronary artery disease-128	Male	55	II	71	Y	75.5	142.1
Coronary artery disease-129	Male	68	II	61	N	176.34	88.95
Coronary artery disease-130	Male	60	II	60	N	105.54	141.8
Coronary artery disease-131	Male	38	III	41	N	809.49	110.2
Coronary artery disease-132	Male	73	II	57	N	390	126.8

Coronary artery disease-133	Male	70	II	61	Y	970	119.6
Coronary artery disease-134	Male	79	II	66	N	375	141.2
Coronary artery disease-135	Male	72	II	63	N	62.74	161
Coronary artery disease-136	Male	30	II	63	N	<50	230.2
Coronary artery disease-137	Male	69	II	60	N	661.96	176.6
Coronary artery disease-138	Female	61	II	67	Y	650.79	180
Coronary artery disease-139	Female	66	II	53	N	413.82	95.87
Coronary artery disease-140	Male	71	II	58	N	428.57	171
Coronary artery disease-141	Female	56	II	66	N	269.03	154.7
Coronary artery disease-142	Male	65	II	63	Y	223.4	149.2
Coronary artery disease-143	Female	72	II	70	N	224.99	145
Coronary artery disease-144	Male	66	II	60	N	353.21	126.6
Coronary artery disease-145	Male	82	I	64	N	105.84	99.35
Coronary artery disease-146	Male	52	II	63	Y	317.2	132.8
Coronary artery disease-147	Male	76	II	61	Y	301.08	110.1
Coronary artery disease-148	Female	66	I	64	Y	171.42	259
Coronary artery disease-149	Male	63	II	59	Y	170.06	173.4
Coronary artery disease-150	Male	58	II	63	N	51	160.9
Coronary artery disease-151	Male	50	II	64	N	3884	177.3
Coronary artery disease-152	Male	75	II	74	N	596	160.6
Coronary artery disease-153	Female	61	II	64	N	<50	126.9
Coronary artery disease-154	Female	72	II	69	N	<50	168.4
Coronary artery disease-155	Male	49	I	62	N	<50	119.9
Coronary artery disease-156	Male	72	II	59	N	5344	134.7
Coronary artery disease-157	Male	87	II	59	N	102.63	89.52
Coronary artery disease-158	Male	61	II	76	N	<50	128.6
Coronary artery disease-159	Male	65	II	60	N	<50	126.8
Coronary artery disease-160	Male	70	II	60	N	4332.6	229
Coronary artery disease-161	Male	62	II	58	N	3547	94.26
Coronary artery disease-162	Male	87	III	41	N	3217	55.26
Coronary artery disease-163	Female	57	II	72	N	44.78	208.1
Coronary artery disease-164	Male	52	II	62	N	56.17	202.5
Coronary artery disease-165	Male	56	II	77	N	76.95	192.4
Coronary artery disease-166	Male	35	II	60	N	155.33	148.5
Coronary artery disease-167	Male	70	I	74	N	82.73	156.6
Coronary artery disease-168	Female	79	II	62	N	489	143.9
Coronary artery disease-169	Male	66	II	53	N	258.15	141.8
Coronary artery disease-170	Male	51	II	69	N	60.5	155.6
Coronary artery disease-171	Male	64	I	60	N	75.96	134

Coronary artery disease-172	Male	83	II	67	N	1444	33.23
Coronary artery disease-173	Female	54	II	64	N	64	293.5
Coronary artery disease-174	Male	53	II	64	N	201	74.83
Coronary artery disease-175	Male	51	II	63	N	<50	58.56
Coronary artery disease-176	Female	73	III	63	N	27.8	54.52
Coronary artery disease-177	Female	55	II	63	Y	<50	79.29
Coronary artery disease-178	Female	71	II	68	Y	102.98	46.32
Coronary artery disease-179	Female	62	II	72	Y	129.62	64.24
Coronary artery disease-180	Female	71	II	66	Y	174.09	59.58
Coronary artery disease-181	Female	57	II	73	Y	78	41.45
Coronary artery disease-182	Male	68	III	61	Y	348	53.8
Coronary artery disease-183	Female	71	II	63	Y	152.96	55.55
Coronary artery disease-184	Male	53	I	65	Y	364	106
Coronary artery disease-185	Male	67	II	64	Y	2916.18	55.41
Coronary artery disease-186	Male	68	II	52	Y	114	42.86
Coronary artery disease-187	Male	41	II	67	Y	54.6	41.99
Coronary artery disease-188	Male	55	III	68	Y	252.89	47.13
Coronary artery disease-189	Female	57	II	63	Y	164.2	46.98
Coronary artery disease-190	Male	53	II	64	Y	69	70.34
Coronary artery disease-191	Male	77	I	66	N	-	97.92
Coronary artery disease-192	Male	63	II	73	N	-	81.03
Hypertension-1	Female	62	-	75	N	-	107.9
Hypertension-2	Male	53	-	63	N	-	101.3
Hypertension-3	Female	71	-	69	Y	261.53	159
Hypertension-4	Female	28	-	65	N	171.5	49.47
Hypertension-5	Male	13	-	66	N	16.09	46.66
Peripheral vascular disease-1	Female	50	-	67	N	253	53.49
Peripheral vascular disease-2	Male	35	-	45	N	518	44.82
Peripheral vascular disease-3	Male	54	-	70	Y	566	67.72
Peripheral vascular disease-4	Male	59	-	56	Y	71	49.07
Peripheral vascular disease-5	Female	86	-	62	N	176	56.97
Peripheral vascular disease-6	Male	70	-	66	N	114	36.63
Peripheral vascular disease-7	Female	40	-	68	N	<50	52.81
Peripheral vascular disease-8	Male	47	-	63	Y	<50	42.36
Peripheral vascular disease-9	Male	69	-	62	Y	88	46.95
Peripheral vascular disease-10	Male	71	-	66	Y	<50	58.88
Peripheral vascular disease-11	Male	77	-	66	N	592.92	88.15
Peripheral vascular disease-12	Female	68	-	-	Y	-	158.2
Peripheral vascular disease-13	Female	50	-	-	Y	-	82.01

Peripheral vascular disease-14	Male	72	-	-	N	-	104.9
Peripheral vascular disease-15	Male	58	-	62	Y	-	126
Peripheral vascular disease-16	Male	68	-	-	N	-	101.4
Peripheral vascular disease-17	Female	61	-	58	N	-	93.5
Peripheral vascular disease-18	Male	77	-	67	N	160.82	93.81
Peripheral vascular disease-19	Female	86	-	68	Y	-	88.39
Control-1	Male	57	-	-	N	-	22.175
Control-2	Female	81	-	-	N	-	171
Control-3	Female	74	-	-	N	-	32.34
Control-4	Female	75	-	-	N	-	34.15
Control-5	Female	75	-	-	N	-	33.55
Control-6	Male	78	-	-	N	-	32.15
Control-7	Male	52	-	-	N	-	32.51
Control-8	Female	82	-	-	N	-	22.96
Control-9	Male	74	-	-	N	-	22.74
Control-10	Male	51	-	-	N	-	44.69
Control-11	Male	68	-	-	N	-	27.73
Control-12	Female	71	-	-	N	-	24.48
Control-13	Male	60	-	-	N	-	19.28
Control-14	Male	67	-	-	N	-	155.7
Control-15	Female	62	-	-	N	-	100.6
Control-16	Female	64	-	-	N	-	120.6
Control-17	Male	61	-	-	N	-	41.74
Control-18	Male	52	-	-	N	-	45.73
Control-19	Male	64	-	-	N	-	24.69
Control-20	Male	53	-	-	N	-	69.8
Control-21	Male	79	-	-	N	-	24.26
Control-22	Male	56	-	-	N	-	25.91
Control-23	Male	73	-	-	N	-	32.24
Control-24	Male	89	-	-	N	-	16.6
Control-25	Male	55	-	-	N	-	43.04
Control-26	Male	66	-	-	N	-	32.77
Control-27	Male	75	-	-	N	-	33.8
Control-28	Male	57	-	-	N	-	37.8
Control-29	Male	56	-	-	N	-	41.35
Control-30	Male	58	-	-	N	-	28.99
Control-31	Male	52	-	-	N	-	25.95
Control-32	Male	56	-	-	N	-	88.34
Control-33	Male	62	-	-	N	-	102.5

Control-34	Female	61	-	-	N	-	39.15
Control-35	Male	56	-	-	N	-	30.5
Control-36	Male	57	-	-	N	-	26.32
Control-37	Male	55	-	-	N	-	336.3
Control-38	Male	56	-	-	N	-	28.89
Control-39	Male	55	-	-	N	-	45.19
Control-40	Male	56	-	-	N	-	30.39
Control-41	Male	53	-	-	N	-	54.88
Control-42	Male	55	-	-	N	-	93.37
Control-43	Male	58	-	-	N	-	26.99
Control-44	Male	64	-	-	N	-	26
Control-45	Male	58	-	-	N	-	28.52
Control-46	Male	56	-	-	N	-	30.41
Control-47	Male	72	-	-	N	-	134.5
Control-48	Female	55	-	-	N	-	24.22
Control-49	Male	70	-	-	N	-	20.92
Control-50	Male	58	-	-	N	-	49.43

NYHA, New York Heart Association functional classification; LVEF, left ventricular ejection fraction; BNP, N-terminal pro-brain natriuretic peptide.

Table S2. Primer sequences

Primer name	Primer sequences
Rat MG53	For: 5’ CTGATGCCAGGTACTGAGAAG 3’ Rev: 5’ CTCCACCTCCCAGTAGTGCT 3’
Rat LDHA	For: 5’ CCGTGTGTTGGAGTTGGTGA 3’ Rev: 5’ AACACCAACTCCACCACCAC 3’
Rat β-actin	For: 5' CGTAAAGACCTCTATGCCAACA 3' Rev: 5' TAGGAGCCAGGGCAGTAATCT 3'
Mouse LDHA	For: 5’ TGTCTCCAGCAAAGACTACTGT 3’ Rev: 5’ GACTGTACTTGACAATGTTGGGA 3’
Mouse β-actin	For: 5' CGTAAAGACCTCTATGCCAACA 3' Rev: 5' TAGGAGCCAGGGCAGTAATCT 3'
MG53 conditional KO mouse	5' arm For:5' GGGTTGCAGACACACACTATCATG 3' Rev: 5' GGGCCTAACCATACTTCATCCTAGT 3' 3' arm For:5' CTGTGCTTAGAGAAGGACCTTTGAG 3' Rev: 5' AAGAGCCAAGAAGCCCAGTTCTG 3'
SM22-Cre mouse	5' arm For:5' TCGATGCAACGAGTGATGAG 3' Rev: 5' TCCATGAGTGAACGAACCTG 3' 3' arm For:5' CAAATGTTGCTTGTCTGGTG 3' Rev: 5' GTCAGTCGAGTGCACAGTTT 3'

Table S3. Antibodies for immunoblot analyses

Antibody	Manufacturer	Catalog	Source of species	MW (kDa)
MG53	Abcam	ab307593	rabbit	53
MG53	Abcam	ab308199	rabbit	53
MG53	proteintech	22151-1-AP	rabbit	53
IR	proteintech	20433-1-AP	rabbit	220
IRS1	CST	3407S	rabbit	180
β-actin	Proteintech	66009-1-Ig	mouse	42
$p27^{Kip1}$	CST	3686S	rabbit	27
PCNA	CST	13110S	rabbit	36
Cyclin D1	Abcam	ab134175	rabbit	36
MYH11	proteintech	21404-1-AP	rabbit	220
Tagln	proteintech	10493-1-AP	rabbit	22
CNN1	proteintech	13938-1-AP	Rabbit	33
SMA	Abcam	ab7817	Mouse	42
GAPDH	CST	2118S	Rabbit	37
HK	proteintech	22029-1-AP	Rabbit	102
PFK1	proteintech	13389-1-AP	Rabbit	86
PKM1	proteintech	15821-1-AP	Rabbit	58
PKM2	proteintech	15822-1-AP	rabbit	58
LDHA	proteintech	19987-1-AP	rabbit	37
P300	Abcam	Ab275378	rabbit	300
Acetyl-lysine	PTM bio	PTM-105	rabbit	
Ub	CST	20326	rabbit	

Table S4. Antibodies for immunofluorescence and immunohistochemistry

Antibody	Manufacturer	Catalog	Source of species
MG53	Abcam	ab307593	rabbit
Alpha-smooth muscle actin	Abcam	ab7817	mouse
Alpha-smooth muscle actin	Abcam	ab5694	rabbit
LDHA	Abcam	ab135396	mouse
PCNA	CST	13110S	rabbit