

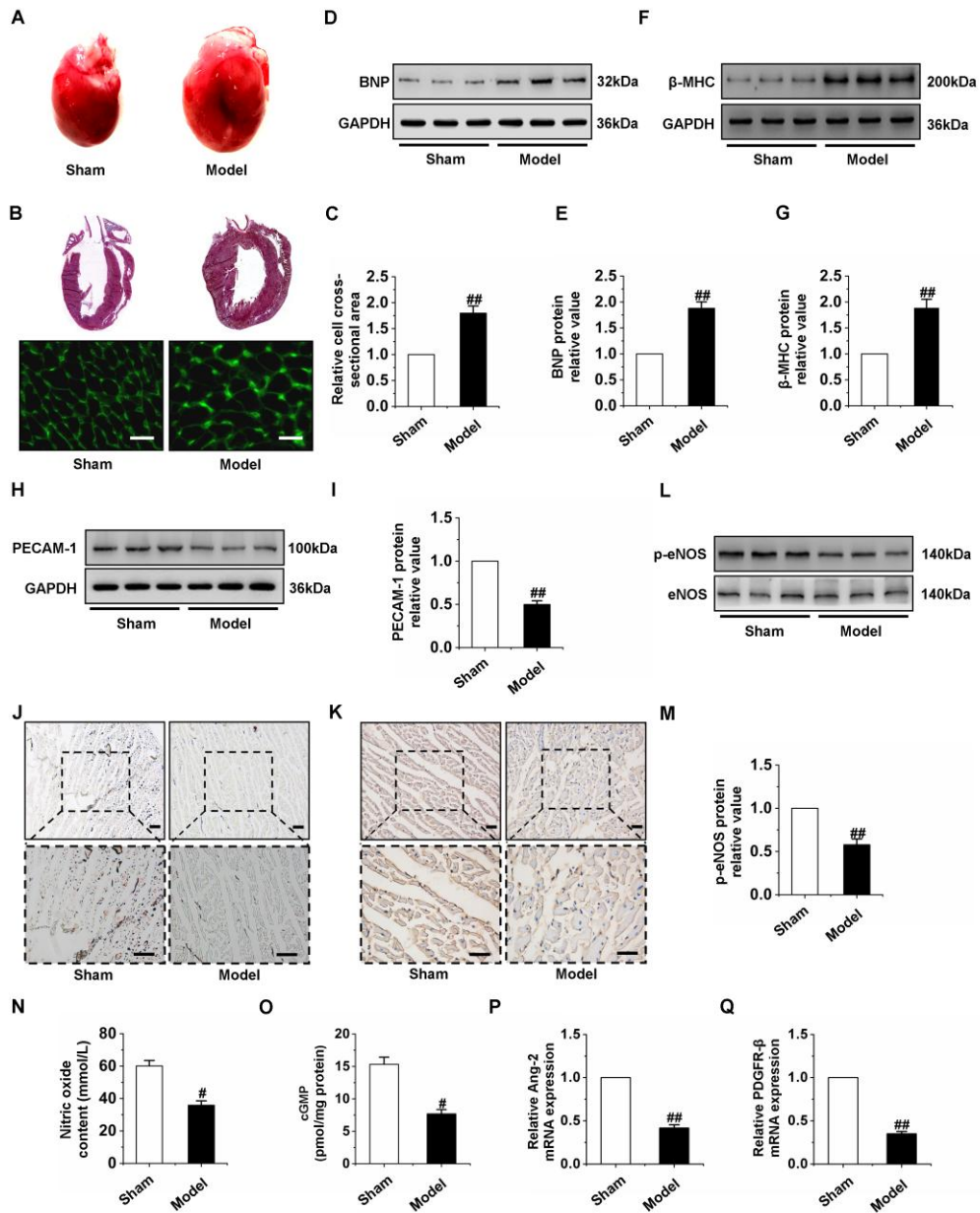
OMTN, Volume 27

## Supplemental information

### **Neutrophil-like cell membrane-coated siRNA of lncRNA *AABR07017145.1* therapy for cardiac hypertrophy via inhibiting ferroptosis of CMECs**

**Pilong Shi, Minghui Li, Chao Song, Hanping Qi, Lina Ba, Yonggang Cao, Meitian Zhang, Yawen Xie, Jing Ren, Jiabi Wu, Ping Ren, and Hongli Sun**

Supplementary figure.1

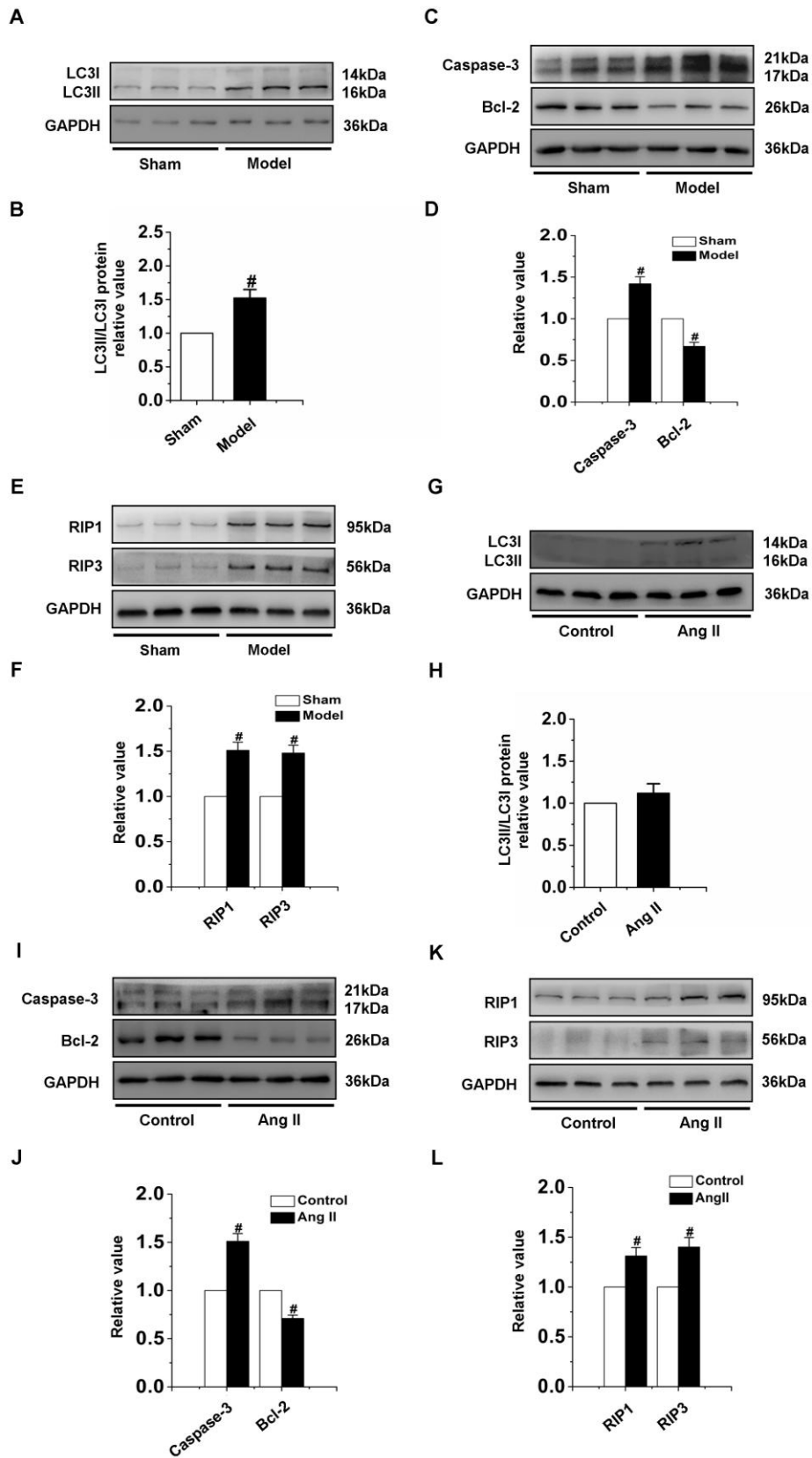


### Supplementary figure S1. Cardiac microvascular damage is induced in hypertrophic hearts

(A) Cardiac volume representative images of rats induced by AAC for 4 weeks. (B-C) Representative sections of heart stained for HE and WGA staining in rats induced by AAC for 4 weeks. (D-E) Detection of *BNP* protein expression by western blot analysis in cardiac tissue. (F-G) Western blot analysis of  $\beta$ -*MHC* expression in cardiac tissue. (H-I) Detection of *PECAM-1* protein expression by western blot analysis in

cardiac tissue. (J) The effect of treatment with AAC for 4 weeks on microvascular perfusion. (K) Representative image of immunohistochemistry of *PECAM-1* in the AAC-induced rat hearts. (L-M) Western blot results of *p-eNOS* protein in cardiac tissue. (N) Statistical analysis of NO content in cardiac tissue of rats. (O) Statistical analysis of cGMP content in cardiac tissue of rats. (P-Q) Quantification of the mRNA expressions of *Ang-2* and *PDGFR-β* was validated by qRT-PCR in cardiac tissue. NO: nitric oxide. AAC: abdominal aorta constriction. WGA: wheat germ agglutinin. Model group: Rats were treated with AAC for 4 weeks. Data were represented by means  $\pm$  SEM (n=6). <sup>#</sup>*P*<0.05 and <sup>##</sup>*P*<0.01 vs. sham group. Scale bars, Figure S1B, 50  $\mu$ m; Figure S1J, 100  $\mu$ m ( $\times$ 100); 100  $\mu$ m ( $\times$ 200); Figure S1K, 50  $\mu$ m ( $\times$ 200); 50  $\mu$ m ( $\times$ 400).

Supplementary figure.2

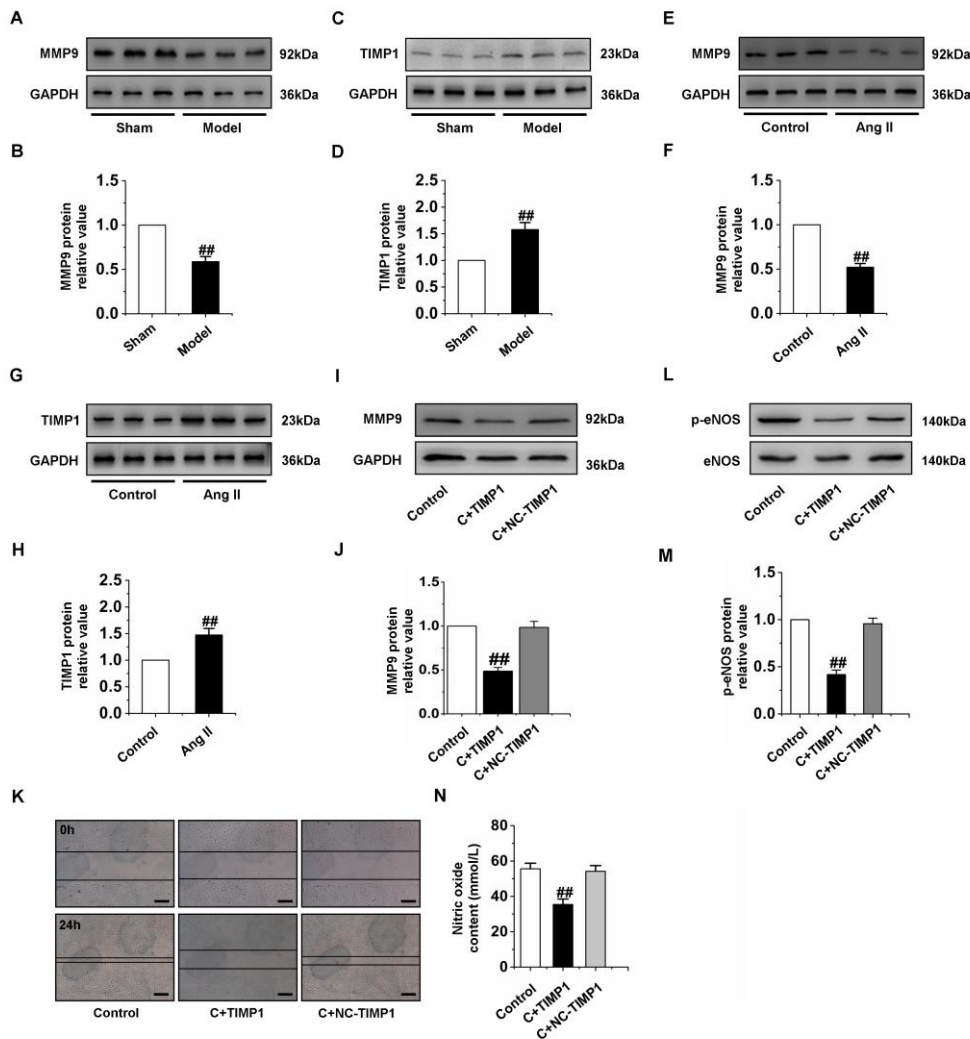


Supplementary figure S2. Autophagy, apoptosis and necroptosis occur in

**myocardial hypertrophy rat hearts, while apoptosis and necroptosis in CMECs**

(A-B) Western blots were performed to analyze the *LC3II* expression in cardiac tissue isolated from rats exposed to AAC for 4 weeks. (C-D) Western blot results of *caspase-3* and *Bcl-2* protein expression in cardiac tissue. (E-F) Western blot results of *RIP1* and *RIP3* protein expression levels in cardiac tissue isolated from rats. Model group: Rats were treated with AAC for 4 weeks. Data were represented by means  $\pm$  SEM (n=6). <sup>#</sup>*P*<0.05 vs. sham group. (G-H) Western blot results of *LC3II* protein expression in CMECs induced by Ang II for 24 h. (I-J) Western blot analysis of *caspase-3* and *Bcl-2* protein expression in CMECs. (K-L) Protein levels of *RIP1* and *RIP3* in CMECs. Ang II group: CMECs were exposed to Ang II for 24 h. Data were represented by means  $\pm$  SEM (n=6). <sup>#</sup>*P*<0.05 vs. control group.

Supplementary figure.3

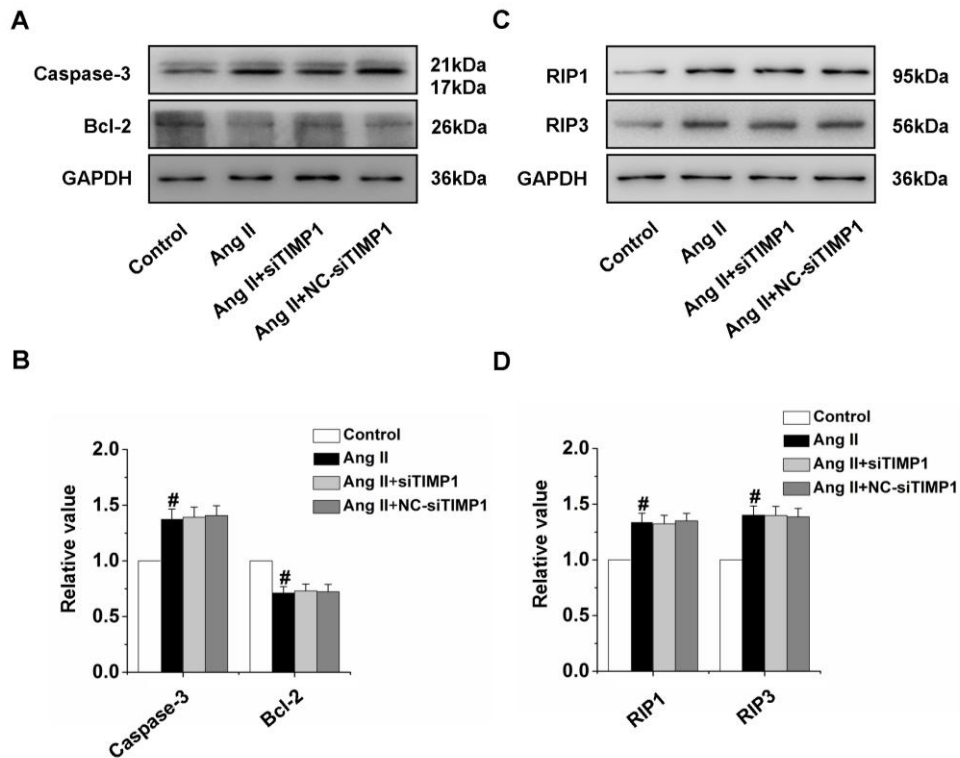


### Supplementary figure S3. Overexpression of *TIMP1* leads to dysfunction of CMECs

(A-D) Detection of *MMP9* and *TIMP1* protein expression of cardiac tissue after AAC treatment for 4 weeks by western blot analysis. Data were represented by means  $\pm$  SEM (n=6).  $^{###}P<0.01$  vs. sham group. (E-H) Detection of *MMP9* and *TIMP1* protein expression in CMECs treated with 100 nM Ang II for 24 h. (I-J) The protein expression of *MMP9* in CMECs were detected by western blot after *TIMP1* or control vectors (NC-*TIMP1*) and x-treme GENE were added to the CMECs and incubated at 37 °C for 24 h. (K) The scratch test was used to detect the effect of overexpression of *TIMP1* on migration ability of CMECs. (L-M) Western blot analysis of *p-eNOS* in CMECs. (N) Statistical analysis chart of NO content in CMECs. C+*TIMP1*: *TIMP1*

and x-treme GENE were added to the CMECs and incubated at 37 °C for 24 h. C+NC-*TIMP1*: pcDNA3.1 empty vector was transfected into CMECs for 24 h. Data were represented by means  $\pm$  SEM (n=6). <sup>##</sup>*P*<0.01 vs. control group. Scale bars: 10  $\mu$ m.

Supplementary figure.4

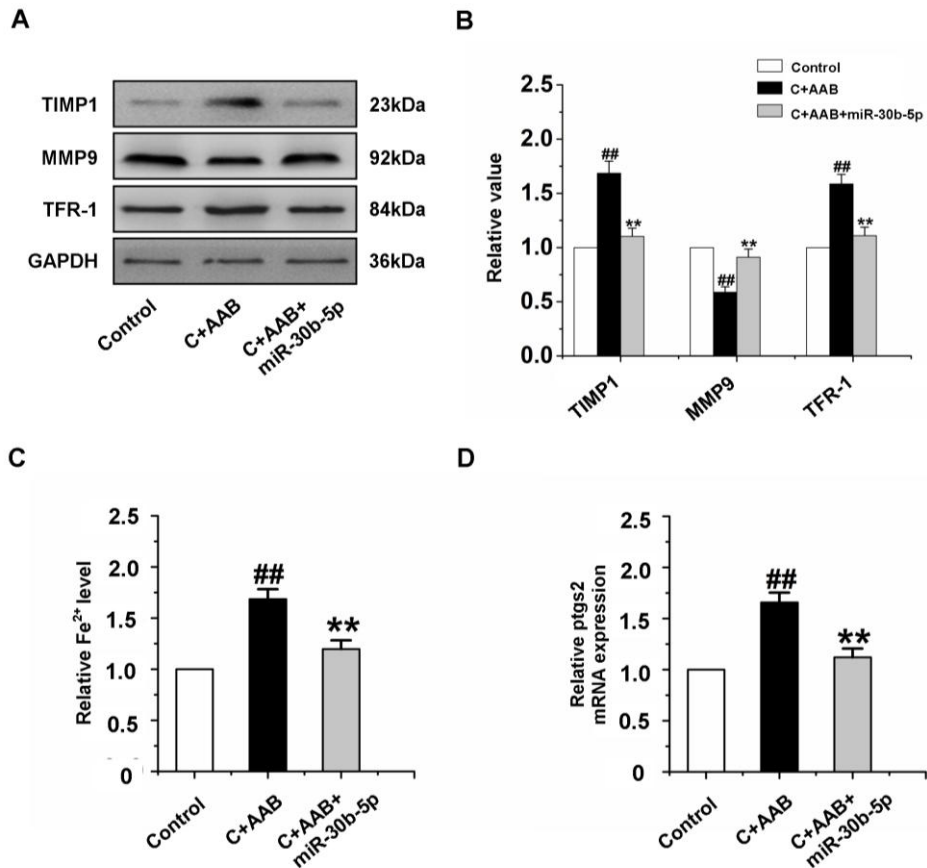


**Supplementary figure S4. Silencing *TIMP1* does not inhibit cell apoptosis and necroptosis**

(A-B) Western blot results of *caspase-3* and *Bcl-2* protein expression in CMECs treated with *siTIMP1* for 24 h and then exposed to Ang II for 24 h. (C-D) Western blot results of *RIP1* and *RIP3* protein expression levels in CMECs. *siTIMP1*: *TIMP1* was silenced. Ang II+*siTIMP1*: CMECs were treated with *siTIMP1* for 24 h and then exposed to Ang II for 24 h. Ang II+NC-*siTIMP1*: CMECs were treated with negative control of *siTIMP1* for 24 h and then exposed to Ang II for 24 h. Data were represented by means  $\pm$  SEM (n=6). #*P*<0.05 vs. control group.



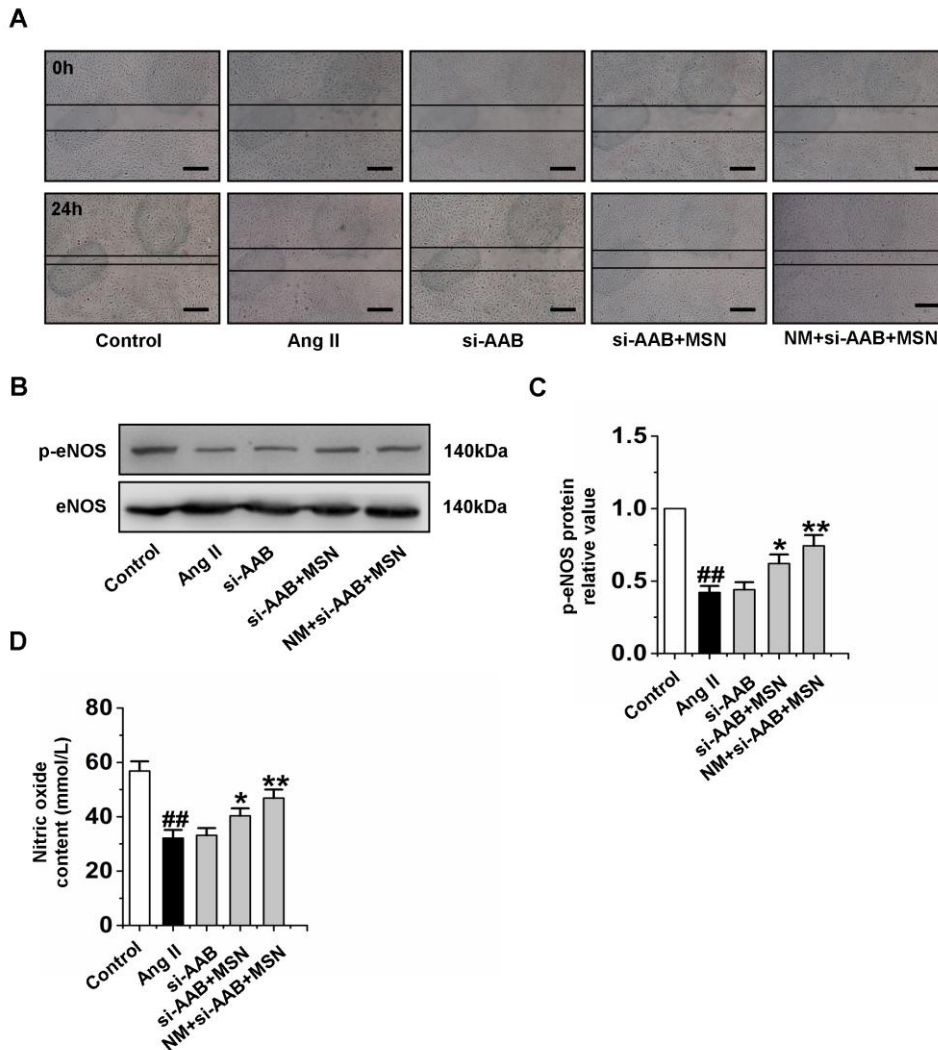
## Supplementary figure.5



### Supplementary figure S5. LncRNA *AAB* regulates ferroptosis via *miR-30b-5p* in CMEC

(A-B) Western blot analysis of *TIMP1*, *MMP9* and *TFR-1* in CMECs co-transfected with lncRNA *AAB* and *miR-30b-5p* for 24 h. (C) Statistical analysis chart of Fe<sup>2+</sup> content in CMECs. (D) qRT-PCR was used to detect the *pts2* mRNA expression in CMECs. *AAB*: lncRNA *AAB*. C+AAB: CMECs were transfected with lncRNA *AAB* for 24 h. C+AAB+*miR-30b-5p*: CMECs were co-transfected with lncRNA *AAB* and *miR-30b-5p* for 24 h. Data were represented by means  $\pm$  SEM (n=6). ##*P*<0.01 vs. control group. \*\**P*<0.01 vs. C+AAB group.

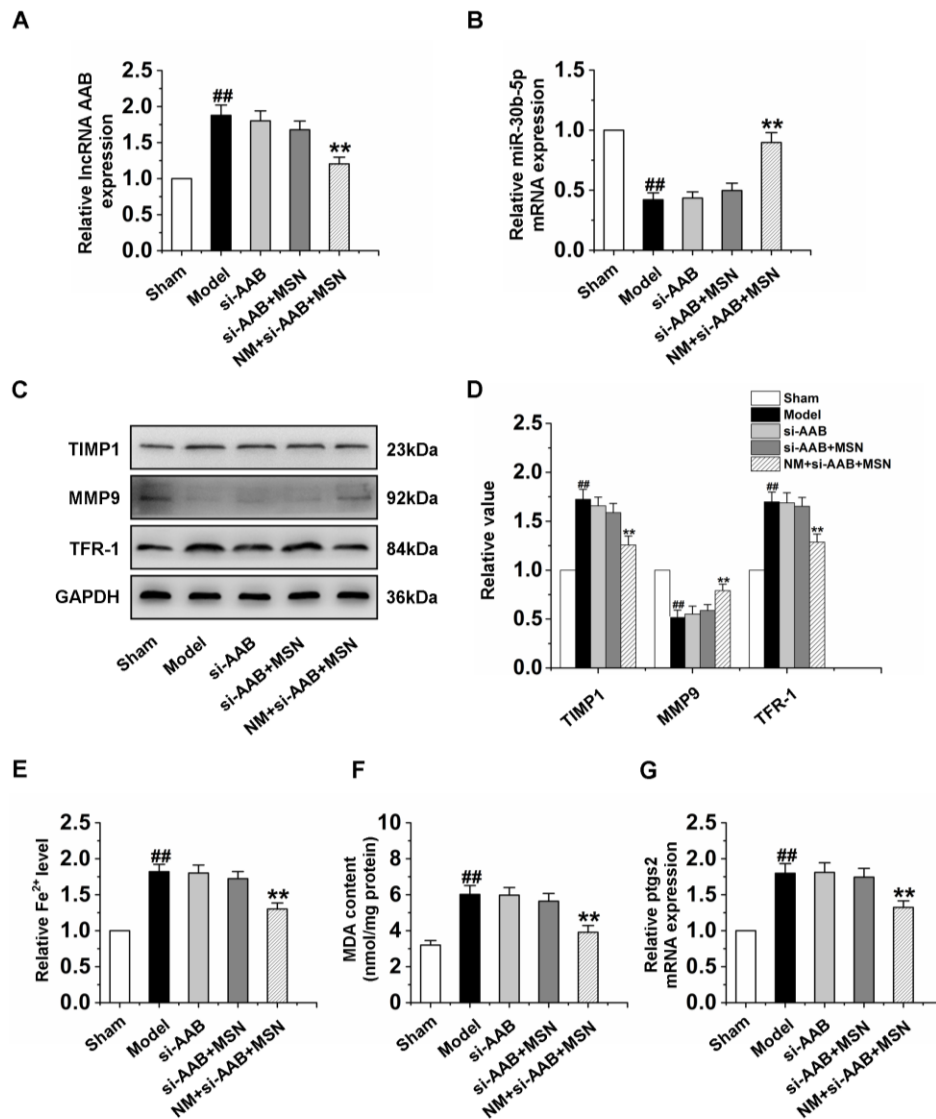
## Supplementary figure.6



### Supplementary figure S6. NM+si-AAB+MSN improves the function of CMECs induced by Ang II

(A) The scratch test was used to detect the effect of si-AAB, si-AAB+MSN and NM+si-AAB+MSN on migration ability of CMECs exposed to Ang II for 24 h. (B-C) Western blot analysis of *p-eNOS*. (D) Statistical analysis chart of NO content. Ang II group: The CMECs were given 100 nM Ang II for 24 h. si-AAB, si-AAB+MSN and NM+si-AAB+MSN groups: The CMECs were given 100 nM Ang II for 24 h following by treatment with si-AAB, si-AAB+MSN and NM+si-AAB+MSN for 48 h, respectively. Data were represented by means  $\pm$  SEM (n=6). <sup>##</sup> $P < 0.01$  vs. control group; <sup>\*</sup> $P < 0.05$  and <sup>\*\*</sup> $P < 0.01$  vs. Ang II group. Scale bars: 10  $\mu$ m.

Supplementary figure.7

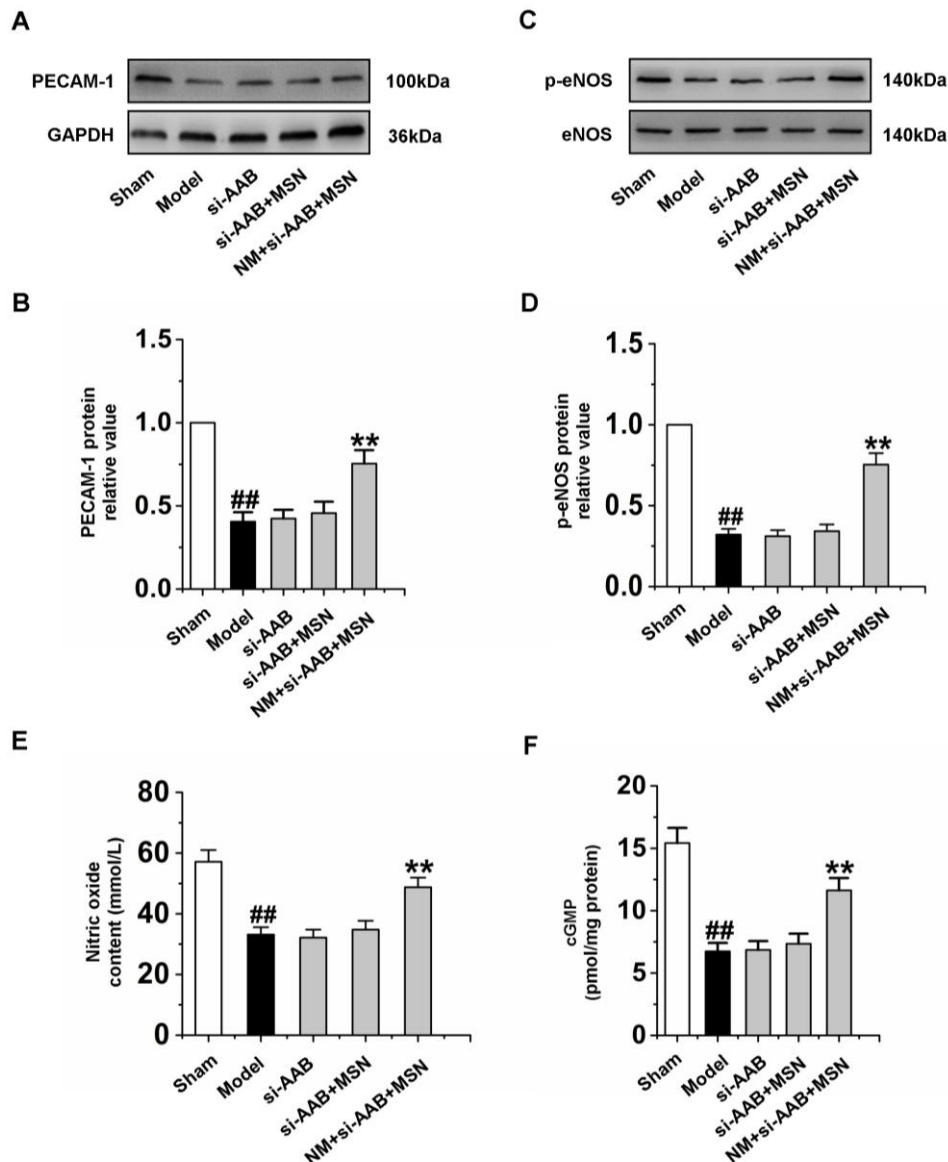


Supplementary figure S7. NM+si-AAB+MSN inhibits the ferroptosis of hypertrophic heart

(A) qRT-PCR analysis of IncRNA *AAB* in the hearts of cardiac hypertrophy rats induced by AAC for 4 weeks. (B) The *miR-30b-5p* mRNA level was detected by qRT-PCR in different groups. (C-D) Western blot analysis of *TIMP1*, *MMP9* and *TFR-1* in cardiac tissue of rats. (E) Statistical analysis chart of iron ions content in hearts of rats. (F) Statistical analysis chart of MDA content in cardiac tissue. (G) mRNA expression of *ptgs2* in cardiac tissue was measured by qRT-PCR. Model group: Rats were subjected to AAC and received vehicle (PBS, caudal vein injection) 24 h later once every 2 days for 4 weeks. si-AAB group: Rats were subjected to AAC and

treated with si-*AAB* via tail vein injection 24 h later every 2 days for 4 weeks. si-*AAB*+MSN group: Rats were subjected to AAC and treated with si-*AAB*+MSN via tail vein injection 24 h later every 2 days for 4 weeks. NM+si-*AAB*+MSN group: Rats were subjected to AAC and treated with NM+si-*AAB*+MSN via tail vein injection 24 h later every 2 days for 4 weeks. Data were represented by means  $\pm$  SEM (n=6).  
## $P$ <0.01 vs. sham group; \*\* $P$ <0.01 vs. model group.

## Supplementary figure.8

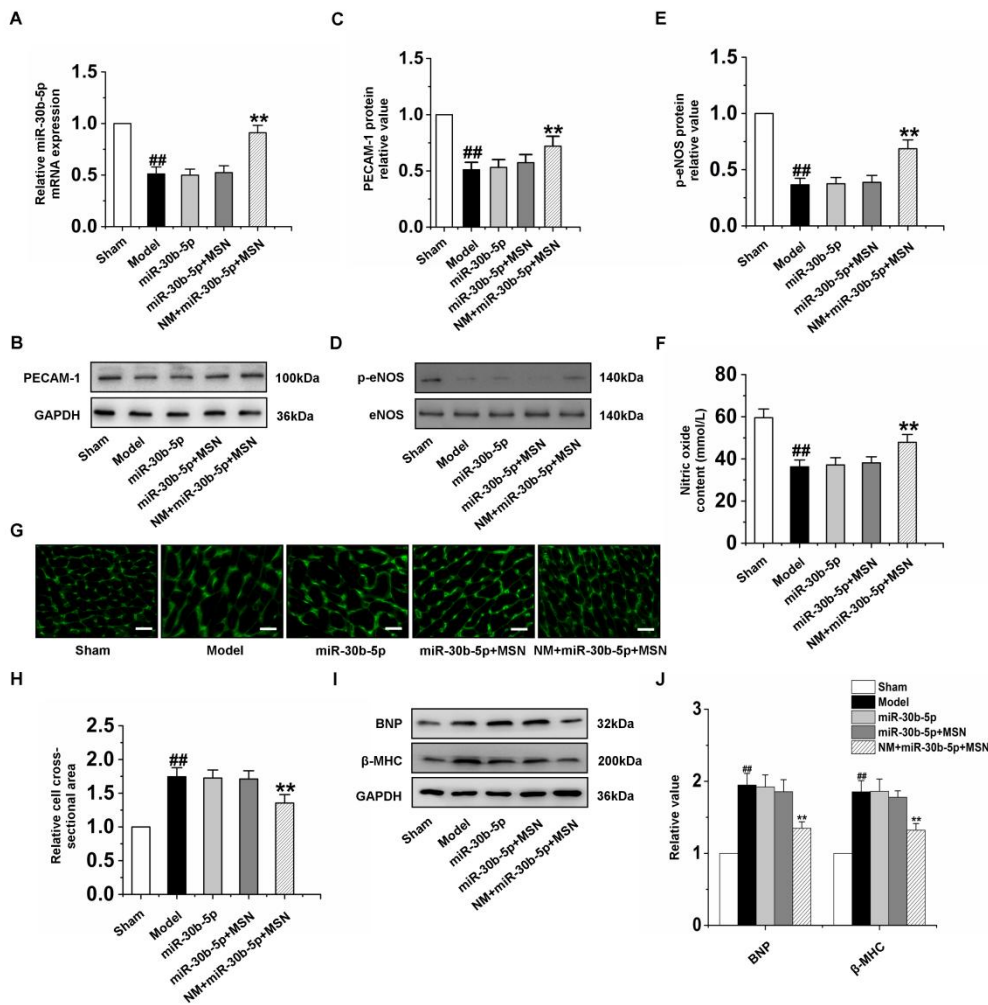


### Supplementary figure S8. NM+si-AAB+MSN plays a protective role in the cardiac microvessels

(A-B) Protein levels of *PECAM-1* in the cardiac hypertrophy rat hearts were assessed by western blot analysis. (C-D) Detection of *p-eNOS* protein expression by western blot analysis. (E) Statistical analysis chart of NO content. (F) Statistical analysis chart of cGMP content. Model group: Rats were subjected to AAC and received vehicle (PBS, caudal vein injection) 24 h later once every 2 days for 4 weeks. si-AAB group: Rats were subjected to AAC and treated with si-AAB via tail vein injection 24 h later every 2 days for 4 weeks. si-AAB+MSN group: Rats were subjected to AAC and

treated with si-AAB+MSN via tail vein injection 24 h later every 2 days for 4 weeks. NM+si-AAB+MSN group: Rats were subjected to AAC and treated with NM+si-AAB+MSN via tail vein injection 24 h later every 2 days for 4 weeks. Data were represented by means  $\pm$  SEM (n=6). <sup>##</sup> $P<0.01$  vs. sham group; <sup>\*\*</sup> $P<0.01$  vs. model group.

Supplementary figure.9



**Supplementary figure S9. NM+miR-30b-5p+MSN inhibits cardiac hypertrophy by improving cardiac microvascular function**

(A) qRT-PCR was used to detect the *miR-30b-5p* expression in cardiac tissue of rats. (B-C) Protein levels of *PECAM-1* in the cardiac hypertrophy rat hearts were assessed by western blot analysis. (D-E) Detection of *p-eNOS* protein expression by western blot analysis. (F) Statistical analysis chart of NO content. (G-H) Representative sections of hearts stained for WGA staining. (I-J) Detection of *BNP* and *β-MHC* protein expression by western blot analysis in cardiac tissue of rats. WGA: wheat germ agglutinin. Model group: Rats were subjected to AAC and received vehicle (PBS, caudal vein injection) 24 h later once every 2 days for 4 weeks. *miR-30b-5p* group: Rats were subjected to AAC and treated with *miR-30b-5p* via tail vein injection 24 h later every 2 days for 4 weeks. *miR-30b-5p*+MSN group: Rats were

subjected to AAC and treated with *miR-30b-5p*+MSN via tail vein injection 24 h later every 2 days for 4 weeks. NM+*miR-30b-5p*+MSN group: Rats were subjected to AAC and treated with NM+*miR-30b-5p*+MSN via tail vein injection 24 h later every 2 days for 4 weeks. Data were represented by means  $\pm$  SEM (n=6). <sup>##</sup> $P<0.01$  vs. sham group; <sup>\*\*</sup> $P<0.01$  vs. model group. Scale bars, 50  $\mu$ m.



Supplementary figure 10

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tctgaa at

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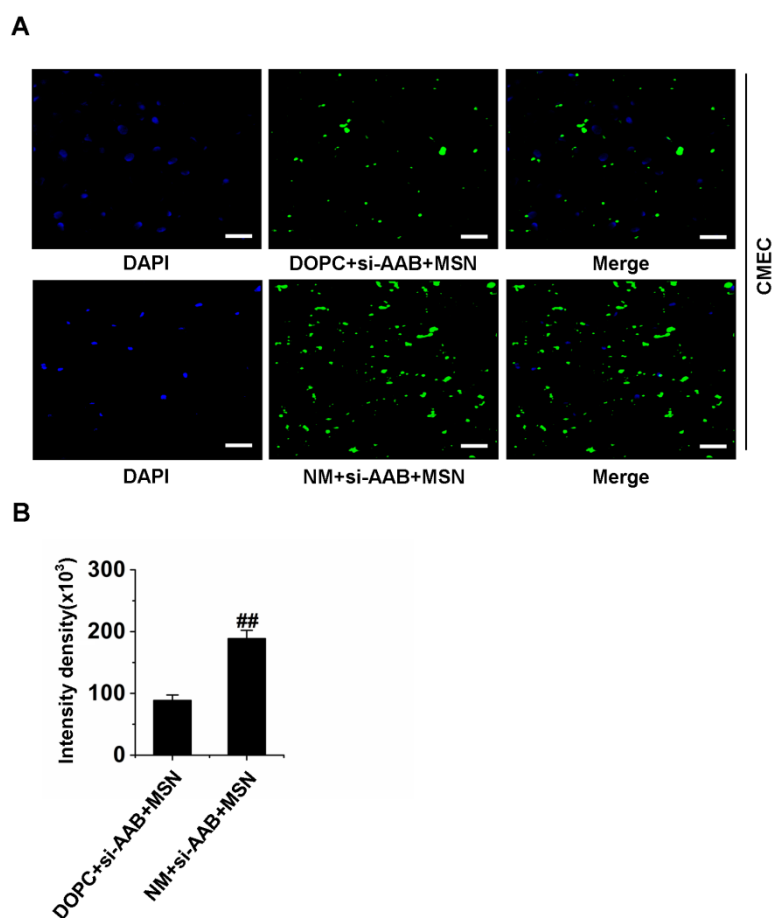
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tt

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Supplementary figure S10. The conserved sites of lncRNA AAB between rat and human

## Supplementary figure.11



**Supplementary figure S11. The cell uptake is evaluated by immunofluorescence**  
(A-B) Cellular uptake of NM+si-AAB+MSN and DOPC+si-AAB+MSN by CMECs exposed to Ang II for 24 h, was observed by fluorescence microscopy. DOPC: dioleoylphosphatidylcholine. Data were represented by means  $\pm$  SEM (n=6). <sup>##</sup> $P < 0.01$  vs. DOPC+si-AAB+MSN group. Scale bars, 50  $\mu$ m.

**Table S1. Changes of cardiac structural and functional indicators in cardiac hypertrophy rat**

Index	Sham	Model
HW/BW (mg/g)	2.69±0.22	3.67±0.29 <sup>###</sup>
LVW/BW (mg/g)	2.03±0.18	3.19±0.21 <sup>###</sup>
HW/TL (mg/mm)	28.45±1.89	39.62±2.25 <sup>###</sup>
LVPWd (mm)	1.21±0.11	1.52±0.12 <sup>#</sup>
LVIDd (mm)	3.82±0.29	5.10±0.28 <sup>#</sup>
LVIDs (mm)	2.11±0.13	3.91±0.24 <sup>###</sup>
LVFS (%)	41.2±2.31	24.87±1.46 <sup>###</sup>
LVEF (%)	78.15±4.52	57.86±3.21 <sup>###</sup>

Data were represented by means ± SEM (n=6). <sup>#</sup>*P*<0.05 and <sup>###</sup>*P*<0.01 vs. sham group.

**Table S2. Changes of hemodynamic index in cardiac hypertrophy rat**

Hemodynamic index	Sham	Model
LVSP (mmHg)	125.36±6.74	81.32±4.30 <sup>###</sup>
LVEDP (mmHg)	5.12±0.24	13.27±1.04 <sup>###</sup>
+dp/dt <sub>max</sub> (mmHg/ms)	4.82±0.27	2.81±0.21 <sup>###</sup>
-dp/dt <sub>max</sub> (mmHg/ms)	-4.58±0.26	-2.78±0.18 <sup>###</sup>

Data were represented by means ± SEM (n=6). <sup>###</sup>*P*<0.01 vs. sham group.

**Table S3. Changes of oxidative stress indexes in cardiac hypertrophy rat**

Oxidative stress index	Sham	Model
SOD (U/mg protein)	130.55 ± 7.63	70.9 ± 4.44 <sup>##</sup>
Mn-SOD (U/mg protein)	19.87 ± 1.34	9.86 ± 0.85 <sup>##</sup>
Cu/Zn-SOD (U/mg protein)	14.80 ± 1.03	7.62 ± 0.56 <sup>##</sup>
CAT (U/mg protein)	42.36 ± 3.17	20.4 ± 2.33 <sup>##</sup>
MDA (nmol/mg protein)	3.2 ± 0.34	5.99 ± 0.45 <sup>##</sup>
GSH (nmol/mg protein)	40.35 ± 2.87	18.0 ± 1.92 <sup>##</sup>
GSH-PX (U/mg protein)	60.21 ± 3.57	35.45 ± 2.31 <sup>##</sup>

Data were represented by means ± SEM (n=6). <sup>##</sup>P<0.01 vs. sham group.

**Table S4. Changes of oxidative stress indexes in CMECs induced by Ang II**

Oxidative stress index	Control	Model
SOD (U/mg protein)	121.36 ± 6.85	88.52 ± 4.22 <sup>#</sup>
Mn-SOD (U/mg protein)	18.22 ± 1.41	9.52 ± 0.92 <sup>##</sup>
Cu/Zn-SOD (U/mg protein)	15.03 ± 1.12	6.99 ± 0.60 <sup>##</sup>
CAT (U/mg protein)	38.96 ± 3.25	22.35 ± 2.38 <sup>##</sup>
MDA (nmol/mg protein)	3.31 ± 0.30	5.55 ± 0.42 <sup>##</sup>
GSH (nmol/mg protein)	38.65 ± 2.62	20.30 ± 1.65 <sup>##</sup>
GSH-PX (U/mg protein)	58.65 ± 3.68	40.35 ± 2.25 <sup>#</sup>

Data were represented by means ± SEM (n=6). <sup>#</sup>P<0.05 and <sup>##</sup>P<0.01 vs. control group.

**Table S5. Eleven lncRNAs shared a highly conserved binding site with miR-30b-5p in rat**

>rno-miR-30b-5p	ENSRNOT00000084731	164	-14.9	2 17	28 49	15	86.67%	86.67%	149.1
>rno-miR-30b-5p	ENSRNOT00000090427	167	-18.42	2 21	947 969	20	75.00%	85.00%	148.58
>rno-miR-30b-5p	ENSRNOT00000086616	162	-14.58	2 21	960 980	19	78.95%	78.95%	147.42
>rno-miR-30b-5p	ENSRNOT00000077548	168	-21.46	2 21	52 71	19	84.21%	89.47%	146.54
>rno-miR-30b-5p	ENSRNOT00000081630	162	-16.5	2 19	102 123	17	76.47%	82.35%	145.5
>rno-miR-30b-5p	ENSRNOT00000079518	162	-17.11	2 16	281 303	15	86.67%	93.33%	144.89
>rno-miR-30b-5p	ENSRNOT00000075897	164	-19.33	2 20	3269 3288	18	83.33%	83.33%	144.67
>rno-miR-30b-5p	ENSRNOT00000084787	163	-19.08	2 20	196 217	18	77.78%	77.78%	143.92
>rno-miR-30b-5p	ENSRNOT00000080464	162	-18.56	2 20	730 752	19	73.68%	84.21%	143.44
>rno-miR-30b-5p	ENSRNOT00000043280	165	-22.17	2 19	714 736	18	83.33%	83.33%	142.83
>rno-miR-30b-5p	ENSRNOT00000087948	160	-18.78	2 21	923 941	19	78.95%	78.95%	141.22

**Table S6. Changes of cardiac structural and functional indicators in cardiac hypertrophy rat**

Indicator	Sham	Model	si-AAB	si-AAB+MSN	NM+si-AAB+MSN
HW/BW (mg/g)	2.64±0.25	3.88±0.28 <sup>###</sup>	3.73±0.27	3.71±0.25	3.06±0.19 <sup>**</sup>
LVW/BW (mg/g)	2.21±0.19	3.22±0.23 <sup>###</sup>	3.26±0.26	3.08±0.18	2.68±0.21 <sup>**</sup>
HW/TL (mg/mm)	28.34±1.91	40.03±2.33 <sup>##</sup>	39.83±2.08	38.88±2.12	32.18±2.35 <sup>**</sup>
LVPWd (mm)	1.22±0.13	1.71±0.14 <sup>#</sup>	1.66±0.12	1.65±0.14	1.34±0.12 <sup>**</sup>
LVIDd (mm)	3.90±0.25	5.48±0.23 <sup>###</sup>	5.19±0.20	5.10±0.22	4.21±0.25 <sup>**</sup>
LVIDs (mm)	2.08±0.12	4.05±0.22 <sup>###</sup>	3.91±0.23	3.82±0.23	2.65±0.20 <sup>**</sup>

Data were represented by means ± SEM (n=6). <sup>#</sup>*P*<0.05 and <sup>##</sup>*P*<0.01 vs. sham group; <sup>\*\*</sup>*P*<0.01 vs. model group.

**Table S7. Primer sequences**

Name	Forward (5'-3')	Reverse (5'-3')
<i>ptgs2</i>	CAACCAGCAGTTCCAGTATCAG	GAGCAAGTCCGTGTTCAAGG
<i>Ang-2</i>	AGCCAGTCTCCCTTCCAG	AGGCAAGCCATTCTCACA
<i>PDGFR-β</i>	GCACCGAAACAAACACACCTT	ATGTAACCACCGTTCGCTCTC
<i>miR-30b-5p</i>	CCAGCAACTGTAAACATCCTACAC	TATGGTTTTGACGACTGTGTGAT
lncRNA <i>AAB</i>	TTCGTCCGCCACTCTCAGGATG	TCGGTCAGCCAGGTGCAGATG
<i>TIMP1</i> siRNA	GGAACGGAAAUUUGCACAUTT	AUGUGCAAUUUCCGUUCCTT
negative control of <i>TIMP1</i> siRNA	UUCUCCGAACGUGUCACGUTT	ACGUGACACGUUCGGAGAATT
<i>miR-30b-5p</i> mimics	UGUAAACAUCCUACACUCAGCU	CUGAGUGUAGGAUGUUUACAUU
negative control of <i>miR-30b-5p</i> mimics	UUGAGCCACAGCUGCAUACTT	ACCCGGAAGGCCGCAAGCTT
lncRNA <i>AAB</i> siRNA	GUCCUGGAUCCACCUUAATT	UUAAGGUGGAAUCCAGGACTT