compared to 62.50% (10/16) in control group (P=0.015). After ischemic and reperfusion, DRP treatment significantly increased ejection fraction in this study (DRP group: 40.1±7.2%; Control group: 27.8±9.1%; Sham group: 53.7±7.6%; P<0.01). Our results showed that DRP decreased LVEDP and increased LVSP in DRP group compared with the control group (LVEDP: DRP group 13.46±8.54 mmHg vs. Control group 26.23±13.12 mmHg vs. Sham group 4.83±5.42 mmHg, P<0.01; LVSP: DRP group 87.71±12.68 mmHg vs. Control group 69.90±11.08 mmHg vs. Sham group 113.24±16.76 mmHg, P<0.05). Myocardial infarct size was significantly decreased in DRP group compared with that in the control group (DRP group: 22.03±8.67%; Control group: 29.54±11.36%; Sham group: 0.00±0.00%; P<0.01).

CONCLUSIONS These results suggested that DRPs had a protective effect on cardiac I/R injury of rat hearts and it may offer a new potential approach for the treatment of acute ischemic heart diseases.

GW26-e4786

Protective effects of naringin on diabetic cardiomyopathy in rats through inhibiting p38MAPK pathway

Hai Jie, Keng Wu
Department of Cardiovasology, The Affiliated Hospital, Guangdong Medical College, Zhanjiang, China

OBJECTIVES To explore the roles of p38MAPK in diabetic cardiomyopathy. To investigate wheather Naringin protects cardiomyocytes against diabetic cardiomyopathy through inhibiting p38MAPK pathway.

METHODS The 60 male SD rats were randomly divided into normal control group (10 rats), model group (50 rats). Diabetic rats induced by high-sugar and high fat diet and intraperitoneal injection of streptozotocin. Naringin (20/40/80 mg·kg-1·d-1) and SB203580 were administered in diabetic rats for six weeks. Blood glucose were detected every 2 weeks, expression of myocardial t-p38MAPK and p-p38MAPK were tested by Western blotting Assay. Serum BNP were assayed using ELISA kit. Morphology of myocardial cell and myocardial structure were observed by light microscopy and electron microscopy.

RESULTS Naringin could lower blood glucose, BNP and heart to weight index in STZ-induced diabetic cardiomyopathy rat. Morphology of myocardial cell and myocardial structure were analyzed through light microscopy and electron microscopy, which were improved by naringin treatment. Compared with the normal control group, the p-p38MAPK expression of myocardial tissue in DCM rats treated by naringin were decreased similar to the inhibitory effect of a p38MAPK inhibitor SB203580.

CONCLUSIONS Naringin could effectively prevent myocardial remodeling and improve cardiac function via inhibiting p38MAPK pathway.

GW26-e5347

Recovery of Mesenchymal Stem Cells Homing to Rabbit Myocardial Ischemic Infarct Area by Cu-microbubble Treatment

Lili Meng,[1] Wen Sun,[1] Xiaorong Sun,[1] Lin Bai,[1] Hongge Wang,[1] Ning Wang,[1] Y. James Kang[1,2]
[1]Regenerative Medicine Research Center, West China Hospital, Sichuan University, Chengdu, Sichuan, China; [2]Department of Pharmacology and Toxicology, University of Louisville School of Medicine, Louisville, Kentucky, USA

OBJECTIVES Mobilization and homing of bone-marrow mesenchymal stem cells (BMSCs) were observed in acute myocardial ischemic injury, but disappeared in a long-term ischemic stress along with depressed copper (Cu) concentrations in the heart. Cu is required for hypoxia-inducible transcription factor-1 (HIF-1) regulation of expression of BMSC homing factors. The present study was to test the hypothesis that Cu supplementation recovers BMSC homing signaling system, leading to BMSC homing to the chronic ischemic infarct area of heart.

METHODS Male adult New Zealand rabbits were subjected to coronary artery ligation to generate myocardial ischemia. Six months after myocardial ischemia, a newly developed ultrasound contrast microbubble composed of Cu-albumin surfaced structure was used to specifically deliver Cu to the infarct area. The autologous BMSCs were labeled with fluorescence and injected via i.v. to the rabbits 24 hours before the heart harvest. AMD3100, the specific SDF-1/CXCR4 axis blocker, was used to treat the labeled BMSCs in one group.

RESULTS BMSCs signaling was observed within 7 days after myocardial ischemia and identified in the ischemic area; but six month after myocardial ischemia, the labeled cells were not found in the ischemic area. The ultrasound-Cu-delivering led to homing of the labeled BMSCs to the chronic ischemic infarct area. AMD3100 blocked the recovery of Cu-microbubble-induced homing of BMSCs.

CONCLUSIONS This study thus demonstrated that Cu supplementation reestablishes the signaling pathways for homing of BMSCs to the chronic ischemic area, which involves the role of Cu-activated HIF-1 transcriptional activity.

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Cardiac ace2/mas expression and cardiac remodeling in hypertensive rats

Junyan Wu, Tingting Chen, Yanling Zhang
Institute of Neurobiology, Taishan Medical University, Taian, China

OBJECTIVES The recent discovery of the new components of the renin-angiotensin system (RAS) suggests the importance of the maintenance of cardiovascular structure and functions. To assess the role of the ACE2-Mas axis in the regulation of cardiac structure and function, the present work investigated the expression of ACE2 and Mas receptor in the heart in the cardiac remodelling that occurs in aortic constricted rats.

METHODS Partial abdominal aortic ligation was carried out in Sprague-Dawley rats. Angiotensin AT1 receptor blockade and ACE inhibition were achieved by losartan and enalapril treatment, respectively.

RESULTS Results showed that aortic constriction increased left ventricular hypertrophy, fibrosis, MAP, plasma renin activity (PRA) and cardiac ACE levels, but decreased the expression of cardiac ACE2 and Mas receptor. Losartan treatment significantly decreased MAP, left ventricle hypertrophy (LVH), fibrosis, and increased cardiac ACE2 and Mas expression. Enalapril also improved the cardiac parameters with a rise in cardiac ACE2, but did not change the Mas level.

CONCLUSIONS Aortic constriction results in cardiac hypertrophy, fibrosis and a rise of cardiac ACE expression. Both AT1 receptor blocker and ACE inhibitor play a cardioprotective role in aortic constriction. However, AT1 receptor blocker particularly promotes cardiac ACE2 and Mas receptor levels. ACE inhibitor is associated with the inhibition of ACE and normalization of cardiac ACE2 activity.

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Low Level electromagnetic field suppresses intermittent hypoxia induced atrial fibrillation through autonomic modulating

Lilei Yu, Xiaoya Zhou, Bing Huang, Zhuo Wang, Songyun Wang, Liping Zhou, Hong Jiang
Department of Cardiology, Renmin Hospital of Wuhan University, Wuhan, China

OBJECTIVES We tested the hypothesis that low level electromagnetic field (EMF) could suppress intermittent hypoxia induced atrial fibrillation (AF) through autonomic modulating.

METHODS Electrode catheters were attached to atria, and all pulmonary veins. Helmholtz coils were powered by a function generator inducing an EMF. The ventilators were adjusted to simulate the intermittent hypoxia for 1 hour as an acute intermittent hypoxia model. Programmed stimulation determined the effective refractory period (ERP) and the window of vulnerability (WOV), a measure of AF inducibility. 40 ms of high-frequency stimulation (HFS; 100 Hz, 0.01 ms pulse width) was delivered 2 ms after atrial pacing (during the refractory period) to determine the AF threshold (AF-TH) at each site. Other electrodes were attached to the superior left ganglionated plexi (SLGP) and left stellate ganglion (LSG) so that HFS (20 Hz, 0.1 ms pulse width) to these sites induced SR slowing and blood pressure (BP) elevation, respectively. Neural activities recorded from the SLGP, LSG and Renal sympathetic nerve (RSN).

RESULTS

(1) Intermittent hypoxia induced a increase in WOV, a decrease in AF-TH and ERP at all sites (all $P < 0.05$).
(2) The SR slowing response induced by SLGP stimulation and BP elevation induced by LSG stimulation were facilitated by 1 hour intermittent hypoxia.