RESULTS (1) compared with the blank control group, LPS group and 0 μ mol/L rosiglitazone group on 6, 12, 24h, HMGB1 secretion increased significantly (P<0.01). Compared with the LPS group, 5 μ mol/Lrosiglitazone group HMGB1 decreased significantly on 6, 12, 24h (P<0.01). Compared with 6h, LPS group and 0 μ mol/L rosiglitazone group HMGB1 secretion increased significantly on 12, 24h; 5 μ mol/L rosiglitazone group decreased significantly on 12, 24h (P<0.05, P<0.01). (2) HMGB1 secretion of 5, 10, 15μ mol/L rosiglitazone group significantly reduced on 24h compared with LPS Group. [(165.77 ± 20.29) ng/ml, (136.63 ± 15.90) ng/ml, (112.25 ± 12.23) ng/ml VS (338.74 ± 18.22) ng/ml, P<0.05]. HMGB1 secretion of 24h in 10, 15 μ mol/L rosiglitazone group is decreased significantly compared with 5 μ mol/L rosiglitazone group (P<0.05, P<0.01).

CONCLUSIONS 1mg/L LPS could obviously stimulate the production of HMGB1 on HUVECs. And it was remarkably inhibited by Rosiglitazone in a concentration and time-dependent manner. The starts up of HMGB1 expression induced by LPS may thereby be one of the mechanism participated in the pathogenesis of atherosclerosis.

GW26-e2389

Ginsenoside Rb1 Attenuates Oxidative Stress and Inflammation in Aged Mice

Shujie Yu,[1] Xiaoxian Qian[1,2]
[1]Department of Cardiology, The Third Affiliated Hospital of Sun Yat-sen University, Guangzhou, Guangdong 510630, China; [2]Institute Integrated Traditional Chinese and Western Medicine, Sun Yat-sen University, Guangzhou, Guangdong 510630, China

OBJECTIVES Our previous studies have shown that Ginsenoside Rb1 (Rb1) prevents endothelial cells from senescence through modulating redox status. Oxidative stress and inflammation are involved in aging and age-related phenomenon. This study was performed to investigate the role of Rb1 in aged mice.

METHODS Female C57/B6 mice were used in the study, which were divided into four groups including control mice (2-3 months), aged mice (22-24 months), Rb1-treated mice (20 mg/kg, 22-24 months). The SOD1 activity and levels of NO and MDA were detected using the corresponding commercially available kits. Levels of ICAM-1 and MCP-1 were examined by ELISA.

RESULTS The oxidative stress was increased in aged mice as shown by decreased SOD1 activity and increased MDA production compared to the young control mice, while administration of Rb1 for 6 weeks was able to reverse the oxidative status significantly. Meanwhile, serum levels of ICAM-1 and MCP-1 were increased in aged mice and were down-regulated after Rb1 treatment. Chronic treatment of Rb1 also partially prevented the loss of body weight in aged mice.

CONCLUSIONS Our study showed that chronic administration of Rb1 was able to attenuate both oxidative stress and inflammation in aged mice. The study may indicate Rb1's potential effect in age-related cardiovascular diseases.

GW26-e3579

Feasible and Controllable Creation of Tricuspid Regurgitation Beagle Dog Model from Tricuspid Leaflet Resection in open beating heart

Xujing Xie,[1] Huanlei Huang[2]
[1]The Third Affiliated Hospital of Sun Yat-sen University; [2]Guangdong Provincial Cardiovascular Institute, Guangdong General Hospital, Guangdong Academy of Medical Sciences

OBJECTIVES To create a controllable and clinical compatible tricuspid regurgitation animal model for studying right heart remodeling caused by TR.

METHODS Fourteen juvenile male Beagle dogs were randomly divided into intervention group (n=11) and sham-operated control group (n=3). Intervention group underwent thoracotomy and right atrium (RA) incision following occlusion of superior and inferior vena cava, anterior leaflet of tricuspid valve was resected in 8 dogs, both anterior and posterior leaflets were resected in 3. Afterward, RA and chest was closed. Animals were planned to be raised for 3 years. Control group underwent the same procedure exception of leaflets resection. One intervention dog and one control dog underwent euthanasia and autopsy at 12m after surgery.

RESULTS All dogs survived the surgery and during 1-year observation. Severe TR immediately occurred after surgery in dogs with anterior leaflets resection, and extremely severe TR in those with two leaflets resection. Duration from thoracotomy to chest closure was 30~40min, and the mean time of superior and inferior vena cava occlusion was 87±10s. CVP increased from baseline 6±1.2mmHg to 13±1.7mmHg (p<0.01). In intervention group, RA area, tricuspid annular diameter and RV index of myocardial performance increased, percent fractional area change of right ventricle and tricuspid annular plane systolic excursion (TAPSE) decreased, and autopsy presented edema, ascites, and cirrhosis morphology.

CONCLUSIONS The protocols of creation of TR were controllable, reproducible, and with high successful and survival rate. Animal model of these surgeries provide opportunities to mechanistically investigate right heart remodeling.

GW26-e5356

Structure-activity Relationships of Flavonoids in Plants of Ampelopsis Grossedentat on Inhibiting hKv1.5 Channels

Xianhong Ou,[1] Xiaohong Bin,[1] Miaoling Li,[1] Yefei Yuan,[2] Yong Ye,[3] Rui Liu,[1] Xinrong Fan,[1] Luzhen Wang,[1] Yan Yang,[1] Xiaorong Zeng[1]
[1]Ministry of Education of China, and the Institute of Cardiovascular Research, Sichuan Medical University, Luzhou 646000, Sichuan, China; [2]College of Pharmacy, Sichuan Medical University, Luzhou, Sichuan, China; [3]College of pharmacy, Guangxi Medical University, Nanning, Guangxi, China

OBJECTIVES To study the structure-activity relationships of Flavonoids in Plants of Ampelopsis Grossedentat on Inhibiting hKv 1.5 Channels.

METHODS To study the structure-activity relationships of Flavonoids in Plants of Ampelopsis Grossedentat on inhibiting hKv 1.5 Channels.

RESULTS Both Myr and DMY could inhibit hKv1.5 Channels. The current density of I_{Kur} was reduced from 215.02 ± 40.59 (pA/pF) to 77.72±17.94 (pA/pF) (n=5, P=0.011 <0.05, VS Control), 116.59±14.30 to 57.23±9.57 pA / pF (n = 14, P < 0.001) respectively by treatment with 10 μM Myr or DMY for 20 min. Moreover, they inhibited I_{kur} in HEK 293 cells in a manner that dose-, time- and frequency-dependence, and inhibited hKv1.5 protein in a dose-dependent manner.

CONCLUSIONS The Inhibition hKv 1.5 Channels activity of Flavonoids in Plants of Ampelopsis Grossedentat is related to the nucleus of flavonoids, but may by not related to 2, 3-flavonone.

GW26-e1056

Qishenyiqi attenuates myocardial fibrosis in rats by inhibiting RAAS-mediated arachidonic acid inflammation

Linghui Lu,[1] Jing Wang,[2] Yan Wu,[3] Jing Han,[3] Yong Wang,[1] Chun Li,[4] Wei Wang[1]
[1]Basic Medical College, Beijing University of Chinese Medicine, Beijing, China; [2]School of Chinese Materia Medica, Beijing University of Chinese Medicine, Beijing, China; [3]Center of Scientific Experiment, Beijing University of Chinese Medicine, Beijing, China; [4]Modern Research Center for Traditional Chinese Medicine, Beijing University of Chinese Medicine, Beijing, China

OBJECTIVES Myocardial fibrosis is a common pathological progress of cardiovascular diseases and treatment of fibrosis may improve clinical efficacy and prognosis of heart failure (HF) patients. Previous study has shown that Qishenyiqi (QSYQ) has definite therapeutic effects on reversing cardiac fibrosis, while its underlying mechanism remains unrevealed. The present study aims to demonstrate that QSYQ treatment can exert anti-fibrosis effects in HF rats mainly by inhibiting Renin-angiotensin-aldosterone System (RAAS) and arachidonic acid (AA) metabolic pathway.

METHODS Sprague-Dawley (SD) rats were randomly divided into 4 groups: sham group, model group (left anterior descending coronary artery ligation), captopril group (LAD ligation and treated with captopril) and QSYQ group (LAD ligation and treated with QSYQ). 28 days after surgery, 2D echocardiography was adopted to detect the heart function. HE and Masson's trichrome staining were performed to assess the degree of myocardial fibrosis. Collagen (I, III) contents and matrix metalloproteinases (MMP2, MMP9) levels were assayed by immunohistochemical method. Western-blot was used to detect expression of critical proteins in RAAS (AT1, AT2) or AA metabolic pathway (PLA2 and COX1/COX2), and their potential signal-transducing proteins (JAK1/STAT3, NFκb and Akt).

RESULTS At 28 days after surgery, echocardiography showed that LVEDd and LVEDs in the model group increased significantly, indicating the induction of ventricular remodeling. HE and Masson's trichrome stain results also demonstrated pathological changes of fibrosis. Expressions of fibrotic markers, such as Collagen (I, III), MMP2 and MMP9, were elevated in model group. Level of AT1 in RAAS