**OBJECTIVES**

Arachidon acid (AA), an omega-6 (n-6) fatty acid, can be converted to inflammatory leukotrienes (LTs) by 5-lipoxygenase (5-LO), which is activated by 5-lipoxygenase-activating protein (FLAP) and then lead to the expression of LTs, which play a role in coronary heart disease (CHD). Omega-3 (n-3) fatty acids are the metabolic counterparts to n-6 fatty acids and have anti-inflammatory effects. Thus, the n-6/n-3 fatty acid ratio may affect LT production. However, whether alteration of tissue n-6/n-3 fatty acid ratio has an effect on 5-LO and FLAP expression is not well known. The fat-1 transgenic mouse, expressing an n-3 fatty acid desaturase, is capable of endogenously converting n-6 fatty acids to n-3 fatty acids, and thereby has a tissue n-6/n-3 fatty acid ratio close to 1:1. The aim of the study was to compare 5-LO and FLAP expression status in immune cells between fat-1 transgenic mice and wild type (WT) littermates.

**METHODS**

Immune cells from fat-1 transgenic mice (n=5) and WT mice (n=5) were harvested from the spleen and cultured for 24h. Mononuclear cells were also isolated from the blood. Cellular n-6/n-3 fatty acid profiles were analyzed using gas chromatography. The mRNA and protein expressions of 5-LO and FLAP in the cells were evaluated using real time RT-PCR and immunoblot (WB) assays, respectively. The expression of LTb4 level was assessed by ELISA assay.

**RESULTS**

He fat-1 transgenic mice showed a lower ratio of n-6/n-3 fatty acids than WT mice in both splenocytes and blood monocytes (AA: 4.53±0.10 vs 2.16±0.09; EPA: 0.13±0.07 vs 0.36±0.15; DHA: 0.49±0.14 vs 2.17±0.76; Total ω-3: 0.68±0.29 vs 2.66±0.70; ω-6/ω-3: 9.49±4.70 vs 1.32±0.36). The mRNA expressions of 5-LO and FLAP were significantly lower in the cells of fat-1 mice than in those of WT mice. Accordingly, the protein levels of 5-LO and FLAP were also markedly lower in fat-1 mice than in WT mice. The LTb4 level was also lower in fat-1 mice than WT mice.

**CONCLUSIONS**

Our findings demonstrate that a decreased tissue n-6/n-3 fatty acid ratio reduces 5-LO and FLAP expression. This study suggests a role for tissue n-6/n-3 fatty acid ratio in the 5-LO pathway of AA metabolism in immune cells, and a new mechanism for the anti-inflammatory effect of n-3 fatty acids, omega-3 fatty acid may play a role in the prevention of CHD.

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**OBJECTIVES**

The present study aims to demonstrate that whether FSXT treatment can ameliorate cardiac function in diabetic rats and to illustrate its effect on energy metabolic mechanism.

**METHODS**

SD rats were randomly divided into 3 groups: normal group, diabetic group, diabetic-FSXT group. 20 weeks after streptozocin induction, FSXT or water was administered for 16 weeks. Cardiac dimensions and function were determined by echocardiography. Adenosine monophosphate-activated protein kinase (AMPK), a critical energy regulator, and sirtuin (PGC1α and sirt1) were measured. The results were associated with the inhibition of the expression of energy metabolic mechanism (CS, AMPK, PGC1α, sirt1) in hearts were detected through PCR.

**RESULTS**

Echocardiography revealed that in the diabetic group, a decrease in E/A and an increase in IVRT were observed in the rats (vs. normal group). Following treatment with FSXT in the diabetic rats, E/A was found to be upregulated, (vs. diabetic group). In the diabetic group, a decrease in EF, FS, LV mass and an increase in LVIDd, LVIDs were observed in the rats (vs. normal group). Following treatment with FSXT in the diabetic rats, EF and FS were found to be upregulated, while LVIDd and LVIDs were markedly decreased (vs. diabetic group).

Real time PCR analysis revealed decreased content of PGC1α, sirt1 and increased levels of CS, AMPK in the diabetic group when compared with the normal group. However, increased levels of CS, AMPK, PGC1α, sirt1 were observed in the FXST-treated group when compared with the diabetic group.

**CONCLUSIONS**

Our study demonstrated that diabetes induced diabetic cardiomyopathy, characterized by both diastolic and systolic dysfunction and metabolic dysfunction in heart. And FSXT protected DCM via attenuating cardiac function. In addition, therapeutic FSXT administration can promote gene levels of energy metabolism. These findings provide evidence as to the cardiac protective efficacy of FSXT to DCM.
induce in vitro cardiomyocytes ischemia-reperfusion injury. Propofol (5-20 μM) was added to the cell culture before and during the OGD/R phases to investigate the underlying mechanism.

RESULTS Our data showed that OGD/R decreased cell viability, increased lactate dehydrogenase leakage, reactive oxygen species and malondialdehyde production in H9c2 cells, all of which were significantly reversed by propofol. Moreover, we found that propofol increased both the activities and protein expressions of superoxide dismutase and catalase. In addition, propofol increased FoxO1 expression in a dose-dependent manner and inhibited p-AMPK formation significantly.

CONCLUSIONS These results indicate that the propofol might exert its antioxidative effect through FoxO1 in H9c2 cells, and it has a potential therapeutic effect on cardiac disorders involved in oxidative stress.

GW26-e2343 Echocardiography-guided percutaneous laser ablation of canine ventricular septum Liwen Liu Department of Ultrasound, Xijing Hospital, Fourth Military Medical University

OBJECTIVES Currently surgical myectomy and ethanol ablation are two established interventions for relieving the left ventricular outflow tract obstruction in hypertrophic cardiomyopathy (HCM) patients. The limitations in safety and efficacy in these interventions call for minimally invasive, potentially safer and more efficacious approach. The aim of this study is to validate the feasibility of echocardiography-guided percutaneous per-ventricular laser ablation of the canine ventricular septum.

METHODS Six domestic dogs were chosen for the study. Laser (Nd: YAG, 800-1064 nm-wavelength, 300 mJ-diameter) that inserted from the right ventricle into X4, Elesta S.R.L., Italy) was used. The laser passed through a needle (2G, PTC, ECOCHIBA, Italy) that inserted from the right ventricle into the targeted septum under the guidance of the echocardiography via a percutaneous route. Laser ablation was performed as follows: 1 W laser for 3 and 5 min at 180 and 300 J, respectively. Echocardiography, serology examination and pathology were performed to assess the results of laser ablation.

RESULTS There was no death or major complication, i.e. tamponade, pericardial effusion or ventricular fibrillation. The real-time echocardiography monitor of M-mode, 2D (LVEF), PW Doppler and TDI presented no significant variation before and after the laser ablation. Contrast echocardiography confirmed the perfusion defects in the ablated septal regions. The laser ablated areas were well demarcated on pathology examination and the diameters of the ablated region were (mm) 4.4 ± 0.57 and 5.28 ± 0.83 for 3- and 5-minute ablation, respectively. Pre- and post-ablation cardiac enzymes (IU/L) were: AST: 39.17 ± 11.23 vs 101.07 (p = 0.02), LDH: 71 ± 33.89 vs 253.33 ± 179.09 (p = 0.007), CK: 488.17 ± 192.42 vs 272.17 ± 130.35 (p = 0.007) and CK-MB: 174.33 ± 92.34 vs 89.17 ± 48.64 (p = 0.03). Microscopically, the ablated myocardium showed contracted coagulative changes. Nuclei disappeared and a zone of vacuoles was formed with red blood cells infiltrating into the widened intercellular space.

CONCLUSIONS Our research showed that percutaneous laser ablation of the septum is feasible, potentially safe and efficacious and may become a viable alternative solution to septum ablation.

GW26-e2407 Protective Effects of Dihydromyricetin and Myricetin against Myocardial Ischemia-Reperfusion Injury In Vivo and In Vitro Yong Ye,1 Xianhong Ou,1 Qiuqie Huang,1 Huagang Liu,1 Yonghong Liang,1 Yefei Yuan,2 Yunfeng Song,2 Xiaorong Zeng2 College of pharmacy, Guangxi Medical University, Nanning 530021, Guangxi, China;1 The Key Laboratory of Medical Electrophysiology, Ministry of Education of China, and the Institute of Cardiovascular Research, Luzhou Medical College;2 College of pharmacy, Guangxi Traditional Chinese Medical University, Guangxi Nanning 530001, China;1 College of pharmacy, Luzhou Medical College 646000, Sichuan, China;2 Postdoctoral R&D Workstations, Guilin Layn Natural Ingredients Corp, Guilin 541100, China

OBJECTIVES The aims of this study were to determine whether Myr or DMY exert any cardioprotective effect against I/R injury and investigate the responsible underlying mechanisms.

METHODS The study utilized an in vitro rat cardiomyocyte H9C2 model of hypoxia/reoxygenation (H/R) injury and an in vivo rat model of MI/R injury. H/R injury was determined by Cell Counting Kit-8 (CCK-8) assay and lactate dehydrogenase (LDH) leakage assay. In the in vivo experiment, histopathology staining was examined. Additionally, cardiac injury markers malondialdehyde (MDA), superoxide dismutase (SOD), glutathione reductase (GSH), myeloperoxidase (MPO), nitric oxide synthase (NOS), Ca2+-Mg2+ATPase and Na+-K+-ATPase were detected. TUNEL assay and caspases activation assays were used to investigate apoptosis. To assess levels of apoptotic regulators, immunochemical staining and real-time PCR were employed.

RESULTS Both Myr and DMY have no cytotoxic effect at the concentrations of 0.5-80 μM Myr or 10-200 μM DMY for 24 hours in H9c2 cells, after being subject to H/R, cellular viability was significantly reduced in the H/R group (P < 0.01 compared to control), and LDH leakage was highly increased (P < 0.01 compared to control (P < 0.01). DMY or Myr post-conditioning significantly increased cell viability compared with the H/R group. DMY (50 and 100 μM) or Myr (20 and 50 μM) markedly reduced H/R-induced cell death and decreased LDH leakage (P < 0.05 or P < 0.01). These results indicate that DMY and Myr significantly preserved cellular viability post-H/R injury in a dose-dependent manner. Both DMY and Myr protected myocardium against I/R (or H/R) injury by increasing NOS, SOD, GSH and ATPases activity, decreasing MDA content and MPO activity at different degrees and attenuating histopathology injury. Meanwhile, DMY and Myr inhibited the cardiomyocyte apoptosis. The level of Bcl-2 protein and mRNA were restored to the normal level by DMY or Myr pharmacological postconditioning. In contrast, the Bax protein level and mRNA level were markedly reduced by DMY and Myr pharmacological postconditioning.

CONCLUSIONS DMY and Myr pharmacological postconditioning could protect against myocardial I/R injury in both in vivo and in vitro models, which are related to apoptosis pathway and antioxidant activity.

GW26-e2435 Role of calreticulin-induced mitochondrial damage in high glucose induced apoptosis in myocardial cells Rui Yan,1,2 Hu Shan,1,2 Lin Lin,1,2 Jiayu Diao,1,2 Ming Zhang,3 Yanhe Zhu,4 Wuhong Tan,1,2 Wei Jin1,2
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OBJECTIVES To observe the effect of high glucose on the protein expression of calreticulin (CRT) and its association with cell apoptosis and mitochondrial dysfunction in the cardiomyocytes.

METHODS AC-16 cardiomyocytes were randomly divided into normal glucose group, high glucose group, high glucose+ CRT siRNA and isotonic control group. The cell apoptotic rate, reactive oxygen species (ROS), mitochondrial membrane potential level, respiratory enzyme activities, and protein expression of CRT were observed.

RESULTS Compared with the cardiomyocytes in normal glucose group, the apoptotic rate of cardiomyocytes and ROS production increased in high glucose groups, accompanying with the decreases in the mitochondrial membrane potential level and enzyme activities of the respiratory chain. The protein expression of CRT was significantly increased in high glucose group. However, compared with high glucose group, high glucose+ CRT siRNA decreased the expression of CRT and attenuated the damage of mitochondrial, but CRT siRNA did not reduce the ROS level in cardiomyocytes.

CONCLUSIONS High glucose brought about CTR over-expression to induce mitocondrial injury, which may be a reason of increasing myoccardial apoptosis.

GW26-e4818 The L-carnitine Ameliorates Pulmonary Arterial Hypertension by Improving Energy Metabolism Dysfunction of Right Ventricular Failure Yan Liu,1 Qinmin Tan,2 Wentao Tang,1,2 Xin Wang,1,2 Zhiyuan Zhang,1,2 Huiqian Song,1,2 Yifan Li,1,2 Yu Jia,1,2 Hui Li,1,2 People's Hospital of Qinhuangdao, Qinhuangdao 063000, China;1 College of pharmacy, Luzhou Medical College 646000, Sichuan, China;2 Postdoctoral R&D Workstations, Guilin Layn Natural Ingredients Corp, Guilin 541100, China

OBJECTIVES L-carnitine is indispensable for energy metabolism and mitochondrial function in the myocardium. Although carnitine


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