12 weeks (model group vs control group: 0.8357 ± 0.0235units/ml vs 0.1870 ± 0.0190units/ml). There were strong positive signals of PKCα both in the intima and in the media closely abutted with the intima. Further in vitro experiment discovered that modified LDL significantly increased cytomembrane PKC activity in HASMC, HAEc and THP-1, and that 100nmol/l PMA (PKC agonist) could synergistically enhance the expressions of ICAM-1 and ezrin in the presence of oxidized LDL (oxLDL, a key modified LDL); however, it reduced the content of I-ßx. Together with oxLDL, PMA stimulated the adhesion ability of endothelial cells with monocytes. 300nmol/l(Calphostin C (PKC inhibitor) could ultimately reverse the effects induced by 100ug/ml oxLDL, that is to say, PKC activity, the expression of ICAM-1 and ezrin, and the adhesion ability of HAEc cells were down-regulated, respectively, whereas the content of I-ßx was increased. Similarly with the effects of oxLDL, enzymatically modified LDL (E-LDL, a key modified LDL) increased the adhesion ability of endothelial cells with monocytes in a dose-dependent manner and the ideal dose of E-LDL is 20-40µg/ml. 200-400nm/l Calphostin C ultimately reverse the effects induced by E-LDL. ICAM-1 and I-ßx may mediate the corresponding pathological process.

CONCLUSIONS The pathway of PMA/Calphostin C - PKC - NF-ßB/I-ßx-ICAM-1-Ezrin/Adherence may be an important one through which the adherent information was conveyed and integrated. In addition, the pathway adhesion molecules-ezrin-cytoskeleton may enhance the adhesion interaction of endothelial cells with monocytes. Calphostin C could effectively inhibited the increase of the adhesion ability of endothelial cells with monocytes induced by modified LDL.

GW26-e0807 Multimodality Molecular Imaging Monitors Bone Marrow Mesenchymal Stem Cells for the Treatment of Ischemic Heart Disease in Rats

Jie Qin, Yuefei Guo, Xiuzhen Chen, Xuelian Liu
Department of Radiology, the Third Affiliated Hospital of Sun Yat-sen University

OBJECTIVES The aim of this study was to explore multimodality molecular imaging to monitor transplanted stem cells with a triple-fused reporter gene in acute myocardial infarction rat models.

METHODS Rat myocardial infarction was established by ligating the left anterior descending coronary artery. A recombinant adenovirus carrying TGF (Ad5-TGF) was constructed. After transfection with 0.8µg/ml Calphostin C (PKC inhibitor) could ultimately reverse the effects induced by 100ug/ml oxLDL, that is to say, PKC activity, the expression of ICAM-1 and ezrin, and the adhesion ability of HAEc cells were down-regulated, respectively, whereas the content of I-ßx was increased. Similarly with the effects of oxLDL, enzymatically modified LDL (E-LDL, a key modified LDL) increased the adhesion ability of endothelial cells with monocytes in a dose-dependent manner and the ideal dose of E-LDL is 20-40µg/ml. 200-400nm/l Calphostin C ultimately reverse the effects induced by E-LDL. ICAM-1 and I-ßx may mediate the corresponding pathological process.

CONCLUSIONS The pathway of PMA/Calphostin C - PKC - NF-ßB/I-ßx-ICAM-1-Ezrin/Adherence may be an important one through which the adherent information was conveyed and integrated. In addition, the pathway adhesion molecules-ezrin-cytoskeleton may enhance the adhesion interaction of endothelial cells with monocytes. Calphostin C could effectively inhibited the increase of the adhesion ability of endothelial cells with monocytes induced by modified LDL.

GW26-e1336 Myocardial Protection by MiR-126 Against Ischemia/Reperfusion Injury Through Suppression of GSK-3β

Qian Wang,1 Jianbing Zhu,2 Lin Wang,1 Wenjun Yu,1 Renfeng Zhang,1 Lihua Qiao,1 Xinyue Ge,1 Jiatao Lou1
1Department of Laboratory Medicine, Shanghai Chest Hospital, Shanghai Jiao Tong University, Shanghai, China; 2Shanghai Institute of Cardiovascular Diseases, Department of Cardiology, Zhongshan Hospital, Fudan University, Shanghai, China

OBJECTIVES miR-126 is considered to play an active role in cardioprotection against ischemia/reperfusion (I/R) injury, as studies suggest that it facilitates vascular regeneration and ameliorates myocardial I/R injury by modulating angiogenic signaling. In this study, the critical role of miR-126 in cardiomyocyte survival and the underlying molecular mechanism were investigated.

METHODS Male Wistar rats were randomly divided into sham operation group and I/R group (in which the left anterior descending coronary artery of the rats were ligated for 30 min and then reperfused for 120 min). Lentivirus expressing miR-126 (LmiR-126) was constructed and transfected into rat through the right common carotid artery. Western blotting and quantitative PCR methods were applied to detect protein and mRNA expressions. Myocardial apoptosis and infarct size were analyzed by in situ apoptosis assay and triphenyltetrazolium chloride (TTC) staining, respectively.

RESULTS Our study demonstrated that the expression of miR-126 was decreased in I/R group compared with sham operation group (p<0.01). Stretch or enforced expression of miR-126 significantly decreased I/R-induced myocardial infarct size by 42% and attenuated I/R-induced myocardial apoptosis, together with down-regulation of glycogen synthase kinase 3 beta (GSK-3β) (p<0.05). The results of luciferase assays suggested that GSK-3β may be a direct target of miR-126. Knockdown of GSK-3β inhibited cell apoptosis and afforded cardioprotection during I/R injury, while its over-expression abolished the effects of miR-126. In addition, miR-126 increased both protein and mRNA levels of β-catenin, which is the downstream effector of GSK-3β in Wnt/β-catenin signaling pathway, and subsequently elevated the expressions of β-catenin targets cyclin D1 and c-Myc.

CONCLUSIONS These findings illustrated that miR-126 played an important role in cardioprotection against I/R injury through targeting GSK-3β and subsequently activating Wnt/β-catenin signaling pathway.

GW26-e1435 Tanshinone IIA Reduces Atrial Fibrillation by Inhibiting Left Atrial Fibrosis Via MMP-9/TIMP-1 Pathway in Isoproterenol-Induced Myocardial Infarction Rats

Jin Ma,1,2 Shiyu Ma,1 Huiliang Qiu,1 Chunhua Ding,1,2 1Guangdong Provincial Hospital of Chinese Medicine Sciences; 2Guangdong Provincial Hospital of Chinese Medicine

OBJECTIVES Atrial fibrillation is a major contributing factor of atrial fibrillation (AF). Tanshinone IIA (TSN) is a lipophilic diterpene extracted from the Chinese herb Salvia miltiorrhiza Bunge with anti-fibrillatory effect. We used isoproterenol-induced myocardial infarction (MI) rats together with transesophageal programmed electrical stimulation (AF) inducing technology to investigate the effects of TSN on AF and the underlying mechanisms.

METHODS MI rat model was induced by isoproterenol. One week after the first injection, 100 mg/Kg/d TSN was gavaged for 4 weeks. AF inducibility and duration were detected by transesophageal programmed electrical stimulation AF inducing technology. The expression of I and III collagen and the change of MMP-9/TIMP-1 balance in left atrial were measured by western blot.

RESULTS After 2 hours of injection isoproterenol, the ST segment elevated and serum CK-MB levels increased. Four weeks after the administration of TSN, the AF inducibility was lower than the MI group (47.1% in TSN vs. 70.6% in MI) and the AF duration was reduced (58.1±25.4s in TSN vs. 441.5±1317.5s in MI, P<0.05). Type I (1.95±0.8 in TSN vs. 169.9±19.4 in MI, P<0.05) and III (1.95±0.20 in TSN vs. 3.19±0.36 in MI, P<0.05) collagen in left atrium were decreased in administration group, compared with MI group. The matrix metalloproteinase (MMP)-9 and tissue inhibitor of metalloproteinase 1 (TIMP)-1 protein levels were lower than MI group, MMP-9/TIMP-1 ratio (0.20 in TSN vs. 0.27 in MI) decreased.

CONCLUSIONS TSN reduces the inducibility rate and duration of AF after MI by inhibiting left atrial fibrillation via regulating MMP-9/TIMP-1 balance.

GW26-e1522 Effect of Egb 761 on the Cardiac Fibrosis in a Rat Model of Myocardial Infarction

Shuqin Li,1 Yuzhou Wu,2 Jiabao Zhu,2 Ru Xing,2 Jiankang Meng2 1Department of Pathophysiology, Hebei Medical University, Shijiazhuang, P.R. China; 2Department of Cardiology, Second Hospital of Hebei Medical University, Shijiazhuang, P.R. China

OBJECTIVES Cardiac fibrosis is a common feature of advanced coronary heart disease and is also a hallmark of heart diseases.

CONCLUSIONS The pathway of PMA/Calphostin C - PKC - NF-ßB/I-ßx-ICAM-1-Ezrin/Adherence may be an important one through which the adherent information was conveyed and integrated. In addition, the pathway adhesion molecules-ezrin-cytoskeleton may enhance the adhesion interaction of endothelial cells with monocytes. Calphostin C could effectively inhibited the increase of the adhesion ability of endothelial cells with monocytes induced by modified LDL.