p<0.01), which were also in a concentration-dependent way. 3. The PAI-1 transcriptional activity were significantly suppressed by fenofibrate (p<0.05), but induced by linoleic acid (p<0.01) in HepG2 cells transfected with PAI-pGL3 total length promoter constructs. 4. When co-transfected with PPARα-pSG5, fenofibrate could suppress the level of PAI-1 transcription further more (p<0.05), while increased y linoleic acid (p<0.01). 5. The PAI-1 transcriptional activity were very inconsistent when transfected with the plasmid containing different length sequences of human PAI-1 gene promoter from −804 to +17 bp.

Conclusions Fenofibrate and linoleic acid could increase the mRNA level of PPARα, and they regulate the synthesis of PAI-1 from transcriptional level, which was concerned with the activated of PPARα by Fenofibrate and linoleic acid. The sequences that could regulate the expression of PAI-1 gene induced by fenofibrate might exist in the areas from −804 to −636 and −636 to −449 of PAI-1 promoter and existed in the areas rom −804 to −636 and −449 to −276 induced by linoleic acid. The effects on expression of PAI-1 were very inconsistently, so there might be other mechanisms involved.

e0148 CHANGES IN EXPRESSION OF ERK1/2, ANGIOTENSIN II RECEPTORS IN HIBERNATING MYOCARDIUM

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Objective To investigate the variation and significance of ERK1/2, Angiotensin II subtype 1 receptor (AT1R) and Angiotensin II subtype 2 receptor (AT_2R) in hibernating myocardium.

Methods 6 little domestic Chinese pigs were implanted a constrictor into the right coronary artery through femoral artery to make a immediate 50%−75% stenosis in the target artery. 1 month later after the operation, NTG $^{99}TC^{m}$-MIBI SPECT (single photon emission CT) was used to detect and locate hibernating myocardium before the animals were killed. Then verify the accuration of SPECT by observing the samples of hibernating myocardium (HM) under electron microscope. Finally assessing the variation of ERK1/2, p-ERK1/2 in normal myocardium and HM by western blot, AT1R and AT_2R were localised by immunohistochemistry and quantified at protein level by western blot respectively.

Results 1. The spatial distribution of AT1R showed no difference among NM and HM. AT1R were found in myocytes and vascular smooth muscle cells (VSMCs); AT_2R were found only in myocytes in NM, while in HM AT_2R could be found not only in myocytes but also in VSMCs. 2. Compared with NM, the relative amount of AT1R significently reduced in HM while AT_2R significently increased in HM. 3. p-ERK1/2 were significantly increased in HM compared with NM.

Conclusion The changes of AT1R and AT_2R may help define the pathophysiological role of the angiotensin system in hibernatine myocardium.

e0149 DIAGNOSTIC IMPLICATIONS OF TG/HDL-C AND PTX-3 IN DIAGNOSIS OF ACUTE CORONARY SYNDROME

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Objective To investigate the value of serum pentraxin-3 (PTX-3) together with triglyceride (TG)/high-density lipoprotein cholesterol (HDL-C) as a predictor risk factor for future acute coronary syndrome (ACS).

Methods Collected and analysed 171 cases of Department of Cardiology of the Second Xiangya Hospital of Central South University in July 2008～December 2009 for elective coronary angiography patients, according to the situation on admission and coronary angiography, patients were divided into three groups: normal control subjects, stable angina pectoris and acute coronary syndrome group, all patients admitted to hospital were extracted fasting venous blood for measuring PTX-3 and lipids (TG, HDL-C) level in the next morning, and Statistically analysed, p<0.05 was considered statistically significant.

Results The serum PTX-3 levels and the ratio of TG and HDL-C in patients with acute coronary syndrome (6.39±3.01 ng/ml; 2.38±2.00) were significantly increased than those in stable angina pectoris (3.87±2.05 ng/ml; 1.70±1.01) and normal control subjects (2.90±1.94 ng/ml, 0.95±0.35), p<0.05.

Conclusions Increased serum PTX-3 and ratio of TG and HDL-C in patients are closely related with acute coronary syndrome, both increase accuracy of early diagnosis of acute coronary syndrome.

e0150 HIGH FREQUENCY OF PERI-STRUT LOW INTENSITY AREA ASSESSED BY OPTICAL COHERENCE TOMOGRAPHY AFTER POLYMER-BASED SIROLIMUS-ELUTING STENTS IMPLANTATION IN PORCINE MODEL

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Objectives To assess the frequency of peri-strut low intensity area (PLIA) in polymeric and nonpolymeric drug-eluting stents by optical coherence tomography (OCT) in porcine model, to analyse the pathological changes in PLIA.

Setting Previous intravascular ultrasound studies showed that black holes were more commonly seen in sirolimus-eluting stent restenosis. Similar phenomenon (peri-strut low intensity area) was also frequently detected by OCT in DES follow-up. However, it is still largely unknown what triggers this uncommon response. Design and interventions: A total of 18 stents (BMS, n=6; polymer-free PES [PF-PES], n=6 and polymer-based SES [PB-SES], n=6) were implanted in six minipigs and OCT was performed at 28 days after stenting. Stented arteries were harvested after terminal OCT imaging for pathological analysis. PLIA was defined as a region around stent struts with a homogenous lower intensity appearance than surrounding tissue on OCT images without significant signal attenuation behind the area.

Results At 28 days, PLIA was more frequently observed around the PBSES struts compared with PFPES and BMS struts (75% vs 33% vs 12%, respectively, p<0.001). Both in DES and BMS group, stents with PLIA showed significantly greater neointimal thickness than stents without PLIA (0.55±0.23 mm vs 0.13±0.08 mm, p<0.001). Histological results showed the existence of fibrin deposition and small amount of inflammatory cells at the site of PLIA.

Conclusions PBSES showed a higher incidence of PLIA compared with BMS and PFPES. PLIA may be related to fibrin deposition and vessel chronic inflammatory response to stent.

e0151 EXPRESSION OF IL-17 IN VIRAL INDUCED DILATED CARDIOMYOPATHY MICE

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Objective To investigate Interleukin 17 (IL-17) levels in viral dilated cardiomyopathy (DCM) mice, aiming at effect of IL-17 in viral DCM.

Methods 40 male BABL/c mice were divided into two groups, the DCM group and the control. The DCM group mice were peritoneal injected Coxsackievirus B3 (CVB3) monthly. After 180 days, all mice were sacrificed and IL-17 mRNA of splenocytes were measured by RT-PCR.
Results In the DCM mice, the heart weight was higher, and the ventricular wall was thinner than the control, and fibrosis in hearts were observed. IL-17 mRNA of splenocytes in DCM mice could be detected and the controls' were zero (0.15±0.04 vs 0.00±0.00, p<0.01).
Conclusion We successfully built murine DCM model by monthly peritoneal injection of CVB3 for 180 days in the DCM group. In the DCM mice, the heart weight was higher, and the ventricular wall was thinner than the control, and fibrosis in heart was observed. The mRNA levels of IL-17 were promoted in Coxsackievirus induced DCM mice. This result suggested that IL 17 which secreted by Th17 subset could be detected in DCM mice, it seems that the Th17 cells might differentiated in DCM mice.

e0152 ENDOGENOUS κ OPIOID PEPTIDE MEDIATES THE CARDIOPROTECTION INDUCED BY ISCHAEMIC POSTCONDITIONING

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Aim Postconditioning is brief cycles of reperfusion and ischaemia during the early phase of reperfusion following a prolonged ischaemic insult. Opioids are well-known endogenous triggers of preconditioning. Because postconditioning shares the protective pathways with preconditioning, G protein–coupled receptor activation may serve as an essential mechanism that triggers protection of postconditioning. Receptor binding studies showed that κ opioid receptor (κ-OR) is a predominant opioid receptor in heart. Therefore, we determined whether endogenous agonist of κ-OR, dynorphin, triggers postconditioning, especially reduces apoptosis of I/R myocardium and to identify its underlying mechanism.
Methods Besides the vehicle, the other SD rats underwent a 30 min left anterior descending occlusion followed by 120 min of reperfusion with or without a postconditioning stimulus (three cycles of 10 s reperfusion and 10 s reocclusion) initiated at the onset of reperfusion. The selective κ opioid receptor antagonist nor-binaltorphimine (Nor-BNI, 2 mg/kg, intravenously), administered 5 min before the reperfusion. The blood plasma was analysed spectrophotometrically for determination of CK and LDH levels. Myocardial apoptosis was quantitatively analysed by detection of TUNEL with an apoptosis detection kit. Six fields from the peri-infarct zone were analysed and the number of TUNEL positive cardiomyocytes was counted on 400 high power fields. Immunoreactive Dynorphin were measured by an antigen competitive ELISA.
Results CK (U/L) and LDH (U/L) were significantly higher in I/R group than those in the control (3401±251 vs 689±76, 2329±216 vs 753±97, p<0.01). Postconditioning significantly reduced the release of CK and LDH from I/R myocardium (2026±268 vs 3401±251, 1543±169 vs 2329±216, p<0.01). These reduction were abolished by nor-BNI (p<0.01). Regional myocardial I/R resulted in a significant increase in cardiomyocyte apoptosis (18.7±2.5 vs 1±0.25, p<0.01). Postconditioning exerted a significant anti-apoptotic effect (10.4±1.3 vs 18.7±2.5, p<0.01). This protective effect was attenuated by pretreatment with Nor-BNI (p>0.05). Immunoreactive dynorphin content (pg/ml) in serum significantly increased after postconditioning (78.5±12 vs 37.3±6.5, p<0.01). Increased dynorphin did not reduced by κ opioid receptor antagonist Nor-BNI (p>0.05).
Conclusions We find that cardiac protection and anti-apoptotic effect of postconditioning is mediated by activating κ opioid receptor. And cardiac protective and anti-apoptosis effect of postconditioning is mediated by enhanced dynorphin express in rats. Recently, clinical use of postconditioning as a treatment for cardiovascular disease has been an increasing attention, and opioid receptor triggers postconditoning, so the study of the relationship between κ opioid receptor and ischaemia reperfusion injury (IRI) may provide a new insight for the curing of IRI.

e0153 EFFECT OF PLATELET MICROPARTILES ON THE EXPRESSION OF CELL ADHESION MOLECULE IN ENDOTHELIAL CELL

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Objective To examine the expressions of cell adhesion molecule (E-selectin.VCAM-1.ICAM-1) in HUVECs (CRL-1730), which is affected by platelet microparticles (PMPs). To investigate the effects of platelet microparticles in coronary heart disease.
Methods 1. PMPs was extracted from anticoagulated blood with sodium citrate. The purity of PMPs was measured by flow cytometry. 2. The prepared PMPs and CRL -1730 cell were co-cultured. The first part was divided into five groups based on the concentration of PMPs. The concentration were: 0, 10 μg/ml, 30 μg/ml, 50 μg/ml, 100 μg/ml, each group contained four wells. Cells in wells were collected after 4 h. The second part was didied into three groups based on the time of co-cultivation: 2 h, 4 h, 24 h, and the concentration of PMPs that were added into each wells was 50 μg/ml. Each group contained four wells. Cells in wells were collected after 2 h, 4 h, 24 h respectively. 3. The RNA of cells was extracted. Semi-quantitate reverse transcription-PCR (SQRT-PCR) was used to detect the relative expression of E-selectin, I CAM-1 and VCAM-1 respectively.
Results In this study, we found that cultured HUVEC (CRL-1730) expressed E-selectin, ICAM-1 and VCAM-1 mRNA in basic states. The expressed levels of E-selectin, ICAM-1 and VCAM-1 were increased when HUVEC (CRL-1730) were interfered by PMPs of centain concentration (p<0.05). But the PMPs stimulated HUVECs (CRL-1730) at different times, the expressions of E-selectin, ICAM-1 and VCAM-1 were of no diffeence (p>0.05).
Conclusions 1. The high purity of PMPs were successfully prepared in the study. 2. The PMPs may increase the expressions of E-selectin, ICAM-1 and VCAM-1 on HUVEC (CRL-1730). It may explain a possible mechanism of PMPs in coronary heart disease.

e0154 EXPRESSION OF TUMOUR NECROSIS FACTOR-α CONVERTING ENZYME AND TUMOUR NECROSIS FACTOR-α IN RATS WITH ALCOHOLIC CARDIOMYOPATHY

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Purpose Chronic excessive consumption of alcohol causes ventricular remodelling and eventually leads to alcoholic cardiomyopahty (ACM). Tumour necrosis factor-α converting enzyme (TACE) has been identified to cleave membrane-bound tumour necrosis factor-α (TNF-α) to soluble TNF-α, which has crucial roles in ventricular remodelling. This study aimed to investigate the expression of TACE and TNF-α, and their impacts on ventricular remodelling in rats with ACM.
Methods 50 healthy male Wistar rats were randomly divided into a control group (n=20) and an ACM group (n=30). Animals in the ACM group were given 10% alcohol ad libitum as the drinking