

preconditioning may strengthen the above function of MSCs, which is correlated with the increase in cytokines induced by hypoxia preconditioning to MSCs.

e0075 EVALUATION THE ROLE OF PRENATAL CHRONIC HYPOXIA ON CARDIAC FUNCTION IN ADULT RABBITS OFFSPRING USING ECHOCARDIOGRAPHY

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Objective To evaluate the role of prenatal chronic hypoxia on cardiac function in adult rabbits offspring via echocardiography.

Methods 16 New Zealand rabbits were divided randomly into two groups: prenatal chronic hypoxia group (12% O₂, n=8) and normal oxygen group (21% O₂, n=8). After delivery, two male offspring of each maternal rabbit were selected and breast-fed for 3 months. Then they were randomly divided into high-fat diet and normal diet respectively. Therefore, four groups were included: Prenatal Chronic Hypoxia with High Fat Diet (n=8), Non-Prenatal Chronic Hypoxia with High Fat Diet (n=8), Prenatal Chronic Hypoxia with Normal Diet (n=8) and Normal Control (n=8). At 6 months of age, the offspring rabbits were undergoing echocardiography examination for left ventricular (LV) dimensions, shortening fraction, ejection fraction and Tei index, and cardiocyte caspase-3 activity detection.

Results Prenatal chronic hypoxia induced a thickening of interventricular septum (main effect is 0.66 mm, $p<0.01$), decrease in ejection fraction of left ventricle (main effect is -4.84%, $p<0.05$), increase of Tei index (main effect is 0.08, $p<0.01$) and cardiocyte caspase-3 activity (main effect is 0.47 unit, $p<0.05$) in 6-month-old prenatal chronic hypoxia offspring. All these effects were aggravated significantly when hyperlipaemia was imposed ($p<0.05$).

Conclusions Echocardiography is a useful tool to evaluate the role of prenatal chronic hypoxia on cardiac function in adult rabbits offspring. Prenatal chronic hypoxia leads to cardiac dysfunction in adult rabbits offspring. This effect is aggravated by hyperlipoidemia.

e0076 SCREENING OXIDATIVE STRESS ASSOCIATED GENES BY GENECHIP ON PERIPHERAL BLOOD MONONUCLEAR CELLS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Objective GeneChip is one of the low-throughput gene screening tools, which is quite suitable for detecting target genes associated with certain pathophysiologic process. Our team has applied such technique into the studying of oxidative stress during the Acute Myocardial Infarction (AMI) and the following ischaemia/reperfusion injury (IRI) after PCI.

Methods 11 patients with confirmed STEMI were admitted to our ER and CCU were involved into our study. 10 healthy volunteers with matching age and sex were set as the controlled group. Blood samples were collected immediately after the diagnosis and the same procedure was done on the 3rd and 7th day. Peripheral blood mononuclear cells (PBMCs) were extracted for RNA extraction. Human Stress & Toxicity Pathway Finder PCR Array was applied for corresponding gene screening. Real Time PCR was applied to confirm the candidate genes mRNA expression.

Results 12 genes were detected with significant changes in the PBMCs of STEMI patients. GADD45A (associated with cell growth/aging), PRDX2 (associated with oxidative stress), HSPD1, DNAJB1, DNAJB2 (associated with heat shock process), RAD50 (associated

with DNA restoration), TNFSF6, TRADD (associated with apoptosis) displayed up-regulated expression. CCNG1 (associated with cell proliferation/cancer), CAT, CYP1A1 (associated with oxidative stress), ATM (associated with DNA restoration) were down-regulated. Further RT-PCR confirmed the previously findings.

Conclusions Sophisticated mechanism was involved during the pathophysiologic development of STEMI and the following IRI after PCI. Oxidative stress, heat shock reaction, cell restoration and apoptosis play an important role in the process of injury and repair.

e0077 EFFECT OF PULSED ALTERNATING MICROCURRENTSTIMULATION ON COMMUNICATION JUNCTION FUNCTION OF COCULTURED RAT MESENCHYMAL STEM CELLS AND CARDIAC MUSCLE CELLS

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Objective This study was to investigate the effect of pulsed alternating micro-current (PAMC) stimulation on the communication junction function of co-cultured rat mesenchymal stem cells (MSCs) and cardiac muscle cells by simulating the physiological electrical environment for cardiac muscle cell growth.

Methods Healthy SD rat bone marrow was subjected to 5-azacytidine induced culture. MSCs not induced (I_{PAMC}) and MSCs cultured for 7 (II_{PAMC}), 14 (III_{PAMC}), and 28 days (IV_{PAMC}) after induction were co-cultured with Hoechst33258 labelled cardiac muscle cells, and stimulated with PAMC. Morphological changes in the gap junction were observed, and the following parameters were determined in each group: intracellular Cx43 distribution and content, the function of intercellular junction communication, the intracellular free calcium concentration and CaMKII expression levels. The results were also compared with those of the group not treated with PAMC.

Results There was a number of typical gap junction structures in group III_{PAMC}. Compared to the groups not subjected to PAMC stimulation, group III_{PAMC} showed a greater increase in the Cx43 expression (78.59 ± 6.72 vs 66.48 ± 9.69 , $p<0.01$), the highest fluorescence recovery rate after photobleaching in the co-culture cells ($50.25\pm4.08\%$ vs $45.89\pm3.94\%$, $p<0.05$), an increase in $[Ca^{2+}]_i$ (101.21 ± 11.36 vs 96.97 ± 9.71 , $p>0.05$), and a significant enhancement of the CaMKII expression (734.35 ± 20.16 vs 596.32 ± 13.45 , $p<0.01$).

Conclusions Appropriate PAMC stimulation will effectively promote the formation of typical intercellular gap junction structures, increase and accelerate the synthesis of gap junction protein, and enhance the intercellular communication junction function.

e0078 THE EFFECT OF ACUTE ATORVASTATIN ON CARDIOPROTECTION OF ISCHAEMIC POSTCONDITIONING IN DIABETES MELLITUS

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Objective This study was to investigate if the low dose of acute atorvastatin treatment could affect the cardioprotection of ischaemic post-conditioning (Ipost) in type 2 diabetes mellitus (T2DM) during ischaemia and reperfusion.

Methods Male Wistar rats and diabetic rats were randomly assigned to four groups: (1) nonconditioning group, (2) Ipost group, (3) acute atorvastatin-treated group (2 mg/kg/day atorvastatin for 3 days),

(4) acute atorvastatin-treated group with Ipost. T2DM were induced with streptozotocin (40 mg/kg, i.p.) after 4-week high-fat diet. All rat hearts were allowed to stabilise for 30 min followed by 30 min of ischaemia and 120 min of reperfusion by the Langendorff technique, and they were divided into regional ischaemia (induced by ligation the left main artery) and global ischaemia (induced by clamping perfusion circuit). Postconditioning was achieved by six cycles of 10 s ischaemia-reperfusion periods after ischaemia. During reperfusion, the functional parameter, the peak rate of pressure development ($+dP/dt_{max}$), was recorded at 5, 30, 60, 90 and 120 min in global-ischaemia protocols, and its recovery was expressed as a percentage of initial preischaemic values. At the end of perfusion, infarct area and risk zone of stained hearts were measured, and standard Western blot analysis was performed.

Results Postconditioning markedly reduced infarct size (the ratio of infarct area and risk zone, %) and improved the values of $+dP/dt_{max}$ (data not shown) in standard-diet group ($22.35 \pm 4.50\%$ vs $44.51 \pm 3.53\%$, $p < 0.05$), but failed in T2DM group ($58.06 \pm 5.37\%$ vs $57.38 \pm 5.11\%$, $p > 0.05$). Acute atorvastatin treatment couldn't decrease ischaemia-reperfusion injury in the healthy and DM rat hearts ($41.65 \pm 4.41\%$ vs $44.51 \pm 3.53\%$, and $55.85 \pm 4.79\%$ vs $57.38 \pm 5.11\%$, $p > 0.05$). However, this short-term statin therapy didn't affect infarct size-limiting and contractile dysfunction-recovering of Ipost in healthy rat hearts ($24.11 \pm 4.08\%$ vs $22.35 \pm 4.50\%$, $p > 0.05$), it restored the protection of Ipost in DM ones ($35.65 \pm 4.93\%$ vs $58.06 \pm 5.37\%$, $p < 0.05$). Western blot analysis revealed that the phosphorylation of Akt Ser⁴⁷³ and eNOS Ser¹¹⁷⁷ increasing was indicated in Ipost group, but not in Ipost T2DM group. Acute atorvastatin treatment slightly increased the phosphorylated expression of Akt and eNOS both in healthy and in T2DM rats. 3-day statin application didn't further increase the phosphorylated Akt and eNOS levels of Ipost in healthy rats, but achieve the largest increasing in T2DM rats.

Conclusions Acute application of atorvastatin show cardioprotective effect in neither healthy rats nor in T2DM ones, and did not interfere with the protection of postconditioning in normal-diet rats, but could restore the infarct size-limiting and contractile dysfunction-reducing of Ipost in the diabetic rats. This study demonstrated that the mechanism was involved in increasing phosphorylation of Akt and eNOS.

e0079 PRESERVATION OF THE CARDIAC FUNCTION IN INFARCTED RAT HEARTS BY THE TRANSPLANTATION OF ADIPOSE-DERIVED STEM CELLS WITH INJECTABLE FIBRIN SCAFFOLDS

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Objective Cell-based therapy can improve cardiac function but is limited by the low cell retention and survival within ischaemic tissues. Injectable cardiac tissue engineering aims to support cell-based therapies and enhance their efficacy for cardiac diseases. Our research is devoted to studying the usefulness of the combination of fibrin glue (as scaffold) and adipose-derived stem cells (ADSCs) to treat myocardial infarction.

Methods The rat ADSCs were isolated from subcutaneous adipose tissues. The surface phenotype of these cells was analysed by flow cytometry. Fibrin glue was then co-injected with ADSCs into the left ventricular wall of rat infarction models. The structure and functional consequences of transplantation were determined by detailed histological analysis and echocardiography.

Results Most cultured ADSCs expressed CD105 and CD90, and negative for CD34 and CD45. After injection, both the 24h-cell retention and 4-week graft size were significantly higher and larger in

the Fibrin+ ADSCs group than those of the ADSCs group alone ($p < 0.01$). The ADSCs could differentiate into cardiomyocyte-like, endothelial and vascular smooth muscle cells in vivo. The heart function improved significantly in the Fibrin+ADSCs group compared to that of the ADSCs group 4 weeks after transplantation ($p < 0.01$). In addition, the arteriole densities within the infarcted area improved significantly in the Fibrin+ADSCs group compared to those in the ADSCs group, 4 weeks after transplantation ($p < 0.01$).

Conclusions The ADSCs with fibrin glue has the therapeutic potential to improve the function of infarcted hearts. The method of in situ injectable tissue engineering combining fibrin glue with ADSCs is promising clinically.

e0080 THE CYP2J2 G50T POLYMORPHISM AND THE RISK OF CORONARY ARTERY DISEASE IN HAN CHINESE POPULATION

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Objective CYP2J2 is the major enzyme responsible for the formation of epoxyeicosatrienoic acids (EETs) in the heart, the EETs are potent endogenous vasodilators and inhibitors of vascular inflammation. The CYP2J2 G-50T polymorphism has been shown to be associated with increased risk of coronary artery disease (CAD) via lower plasma concentrations of EETs, while there are currently some population-based studies which found controversial results. The aim of the study was to assess associations between the G-50T polymorphism of CYP2J2 and CAD via a case-control study in the Han population of China.

Methods There were 249 CAD patients and 243 age-matched control subjects genotyped for the CYP2J2 G-50T polymorphism. The data were assessed for three separate groups: the total subjects, men and women.

Results In total, the distribution of the dominant model of G-50T promoter (G/G versus GT+TT) was significantly lower in CAD patients than control subjects ($p = 0.048$), and simultaneously the T allele was significantly lower in CAD patients than control subjects ($p = 0.037$). However, logistic regression analysis failed to show that the GT+TT genotype was a protective factor for CAD (OR 0.477, 95% CI 0.175 to 1.299; $p = 0.148$). We investigated the synergistic effect between G/T+T/T genotype and no smoking or no DM, finally results showed G/T+T/T genotype and no smoking had a tendency to be a synergistic effect (G/T+T/T genotype+no smoking OR=0.296 vs G/T+T/T genotype+smoking OR=0.730), while lacked sufficient statistical power ($p = 0.127$, $p = 0.663$, respectively).

Conclusions In presence of other risk factors, the CYP2J2 G-50T failed to show a significant association with coronary artery disease in Han population of China. However, since our result is close to the border of significance, further research based on the larger, prospective researches are necessary.

e0081 ASSOCIATION OF GENETIC POLYMORPHISMS OF SAA1 AND SAA2 WITH CORONARY ARTERY DISEASE IN CHINESE HAN POPULATION

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Background Both Low plasma HDL cholesterol (HDL-C) and inflammatory responses are associated with an increased risk of coronary artery disease (CAD). Serum amyloid A protein (SAA) is