

(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