(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 (22.55±4.50% vs 44.51±3.53%, p<0.05), but failed in T2DM group (58.06±5.57% vs 57.38±5.11%, p>0.05). Acute atorvastatin treatment couldn’t decrease ischaemia-reperfusion injury in the healthy and DM rat hearts (41.65±4.41% vs 44.51±3.53%, and 55.85±4.79% vs 57.38±5.11%, p>0.05). However, this short-term statin therapy didn’t affect infarct size—limiting and contractile dysfunction-recov¬ering of Ipost in healthy rat hearts (24.11±4.08% vs 22.55±4.50%, p>0.05), it restored the protection of Ipost in DM ones (55.65±4.93% vs 58.06±5.57%, p<0.05). Western blot analysis revealed that the phosphorylation of Akt Ser175 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 rats 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 ischemic 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 24-h 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.