GW25-e3448
Mitochondrial Transfer from Induced Pluripotent Stem Cells to Ischemic myocardial Cells Protects against Acute Myocardial Infarct in Rats
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Objectives: Induced pluripotent stem cells (iPSCs) protect against acute myocardial infarct and improve infarcted left ventricle function. The stem cells could selectively migrate to peri-infarct area, attenuated MI-induced cell death and restored LV function. The study aimed to determine the role of iPS-MSCs mitochondria in this protection.

Methods: An MI model in rat was created by ligation of left main coronary artery. Female Sprague-Dawley rats were randomized into 3 groups: Control group, AMI group, iPSC-MSCs transplantation group. iPSC-MSCs were injected into LV free wall in the region bordering an infarct in recipient rats following AML 7 days after MI, the EGF donor cells and the transferred DsRed-mitochondria of iPS-MSCs were traced in recipient rats by fluorescence microscopy. We measured myocardium ATP concentration by colorimetric method, respectively. LV function was detected using echocardiography.

Results: Our studies revealed that iPS-MSCs decreased the volume of infarcted myocardium and attenuated MI-induced pathological damages, releasing mitochondrial transferring microvesicles. The iPS-MSCs-derived mitochondria transfer resulted in increased myocardial ATP concentrations. MI-dilated LV with impaired function significantly reduced fractional shortening, cardiac myoocyte peak shortening and relaxation.

Conclusions: This is the first evidence, to our knowledge, that iPS-MSCs could repair tissue damage of AMI and restore LV function by elevated myocardium ATP concentration and their mitochondrial transfer.

GW25-e3465
Relationship between Myocardial Apoptosis and Endoplasmic Reticulum Stress in Excessive Alcohol Consumption Rats
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Objectives: It has been widely recognized that excessive alcohol consumption results in myocardial injury, but the detailed mechanisms need further discussion. Therefore, this study was designed to investigate the relationship between myocardial apoptosis and endoplasmic reticulum stress in excessive alcohol consumption rats.

Methods: Male Wistar rats (n=24) were randomly divided into alcohol consumption group and control group for 12 weeks. General features were collected; morphological changes and myocardial apoptosis were tested by HE staining, TUNEL assay and western blot in hearts of rats, respectively. Expression of ERS activation was detected by immunohistological staining and western blot.

Results: Compared with that in the control group, 12-week alcohol consumption resulted in cardiac LV weight increase (P<0.05), myocardocyte abnormal arrangement, hypertrophy, intermittent fibrosis shown by the data of HE and in the alcohol consumption group. Moreover, it brought about significant apoptosis shown by TUNEL positive results, upregulated expression of Bax and caspase-3 (P<0.001, P<0.01), and downregulated expression of Bcl-2 (P<0.001) in myocardial tissues of rats in alcohol consumption group compared to control group. For further study, the spectrographic analysis of immunohistology and western blot demonstrated the expression of GRP94, CHOP and caspase-12 markedly increased (P<0.01, P<0.01, P<0.001) in myocardial tissues of rats in alcohol consumption group compared with control group.

Conclusions: Excessive alcohol consumption generates myocardial apoptosis probably through activating endoplasmic reticulum stress, which may pave the way for the prophylaxis and treatment of cardiovascular diseases induced by alcohol use.

GW25-e3469
Activation of calcium sensing receptor in CD4+T lymphocyte immune mediated ventricular remodeling
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Objectives: By observing the CaSR on expression of CD4+T lymphocyte and inflammatory cytokines released in different periods after acute myocardial infarction (AMI), in order to Clariﬁy the role of CaSR in CD4+T lymphocyte mediated immune ventricular remodeling, to provide scientiﬁc basis for effective prevention and treatment of heart failure after myocardial infarction.

Methods: Preparation of rat myocardial infarction model, select 40 Wistar rats, weighing200-220g, random is divided into control group, sham group and AMI group, control group without any intervention; sham group, make a slipknot on the left anterior descending coronary artery after thoracotomy; AMI group: ligate the left anterior descending coronary artery after thoracotomy, divided into 3 day and 7 days group, and identification model whether success. Detection of the following indicators: cardiac function was monitored by echocardiography; CaSR expression of CD4+T lymphocytes in heart tissue; cytochrome P450 reductase; levels of CaSR and NLRP3 proteins in CD4+T lymphocyte by immunomagnetic bead extraction were examined by western blot.

Results: Compared with control group and sham group, AMI groups showed declining cardiac function structural, degenerative changes with inflammatory cell infiltration and myocardial fibrosis and robust increases in CaSR and NLRP3, accompanied by expression of downstream signaling, including IL-1β and IL-18 growth by western blot (P<0.05). The degree of the growth is positively related to the time of AML. At the same time, we observed that an increase in CD4+T lymphocytes in myocardial tissue increases expression of CaSR by immunofluorescence.

Conclusions: The results suggest that CaSR induces a robust stimulation in NLRP3 expression to activate CD4+T lymphocyte, which may induces ventricular remodeling after myocardial infarction. Our findings might provide a new insight into scientific evidence for the effective treatment of heart failure.

GW25-e3475
The experimental study of rAAV9 transfecting mouse BMSCs
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Objectives: To investigate the transfection efﬁciency of recombinant adenov- associated virus type 9 (rAAV9) transfecting mouse bone marrow mesenchymal stem cells (BMSCs).

Methods: rAAV9-eGFP (enhanced green fluorescence protein, eGFP) was transfected into BMSCs. eGFP expression was detected by inverted microscopy.

Results: The cells with rAAV9-eGFP transfection at MOI of 1×10^7 began to exhibit eGFP expression at the first day after transfection and the cells transfection at MOI of 1×10^8, 1×10^9 began to exhibit eGFP expression from the second day after transfection. The eGFP expression increased with the time going on. At the fifth day the eGFP expression reached the peak value, and at the same time the eGFP expression of the group MOI=1×10^7 was about 10%.

Conclusions: The recombinant adenov- associated virus type 9 (rAAV9) could not transfect mouse BMSCs effectively.

GW25-e3478
Association of the EGR3 Genetic Polymorphisms and Coronary Artery Disease in the Uygur and Han of China
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Objectives: Endothelial cell activation and dysfunction are the foundation of atherosclerosis including coronary artery disease (CAD). In turn, the level of gene transcription mediates endothelial cell activation. Early growth response 3 (Egr3) is a critical determinant of vascular endothelial growth factor (VEGF) signaling in acti- vated endothelial cells. If endothelial cell excessively activated, it may lead to vasculopathic disease such as pathologic angiogenesis, inﬂammation, and atherosclerosis. The aim of the present study was to assess the association between the polymorphism of the Egr3 gene and CAD.

Methods: Two independent case-control studies, which involved the Han group (409 CAD patients and 351 control subjects) and the Uygur group (299 CAD patients and 303 control subjects), were used to analyze the relation between SNPs (rs1996147 and rs1008949) and CAD. Genotyping was undertaken using the TaqMan SNP geno- typing assay.

Results: For the total sample and men in the Uygur group, an allele frequency of SNP rs1996147 was higher in CAD patients than that in the control subjects (P=0.003 and P=0.005, respectively). Additionally, for the total sample and men, the distribution of the recessive model of rs1996147 (AA vs GG+AG) was significantly different between CAD patients and control participants (P=0.002 and P=0.003, respectively), and the difference remained statistically significant after multivariate adjustment (for total: OR=1.705; 95% CI: 1.166-2.494, P=0.006; for men: OR=1.705; 95% CI: 1.189-2.462, P=0.007). However, for Uygur women, we did not observe the difference of allele frequency or genotype distribution of rs1996147 between CAD patients and control participants. Similarly, the distribution of rs1996147 allele frequency or genotypes had no signiﬁcant difference between patients with CAD and control participants in the Han. The distribution of genotypes, dominant model, recessive model, and allele frequency did not show signiﬁcant difference between patients with CAD and the control subjects in the Han and Uygur groups.

Conclusions: rs1996147 may be a novel polymorphism of the Egr3 gene associated with CAD in men of Chinese Uygur population.