modulation of the AMPK/SIRT1 and TRPA1/NF-κB signaling pathways. Moreover, combined therapy of CCR2B agonist and AD-MSCs has a synergistic effect on cardiac repair and functional improvement after infarction.

**GW26-e2179**  
Genetic Variation in INSIG2 was associated with Coronary Artery Disease in Uygur population in Xinjiang, China  
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**OBJECTIVES**  
Dyslipidemia is a major and independent risk factor for the development of coronary artery disease (CAD). The protein which is encoded by insulin induced gene2 (INSIG2) plays an important role in the mediation of the feedback control of cholesterol synthesis, lipogenesis and glucose homeostasis. However, the relationship between INSIG2 genetic polymorphisms and CAD among diverse ethnicities remains unclear. The aim of the present study was to assess the association between the human INSIG2 gene and CAD in Han and Uygur population of Xinjiang, China.

**METHODS**  
A total of 681 CAD patients (334 Han, 347 Uygur) and 770 controls (346 Han, 424 Uygur) were selected for the present Case–Control tagging SNP (rs17047757, rs18, LRS), rs2161929 in INSIG2 gene were genotyped using TaqMan® assays from Applied Biosystems following the manufacturer’s suggestions and analyzed in an ABI 7900HT Fast Real-Time PCR System.

**RESULTS**  
In the Uygur population, for total, men and women the rs17047757 was associated with CAD by analyses of a recessive model (all, p < 0.001) and additive model (all, p < 0.001) and the difference remained significant after multivariate adjustment in a recessive model (p < 0.001, p = 0.033 and p = 0.002, respectively) and additive model (p < 0.001, p < 0.001 and p = 0.035, respectively), this relationship was also observed in rs2161929 for women by analyses of a recessive model (all, p < 0.001) and additive model (all, p = 0.033), and the difference remained significant after multivariate adjustment in a recessive model (p < 0.001, respectively). However, this relationship was not observed in this three tagging SNPs before and after multivariate adjustment in Han population.

**CONCLUSIONS**  
Our results indicated that both rs17047757 and rs2161929 in the INSIG2 gene was associated with CAD in Uygur population in Xinjiang, China.

**GW26-e2408**  
Left renal sympathetic stimulation and ablation affect ventricular arrhythmia by modulating left stellate ganglion in a cesium-induced long QT canine model  
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**OBJECTIVES**  
Our previous study has shown that left renal sympathetic stimulation (LRS) may facilitate ischemic ventricular arrhythmia (VA) by increasing neural activity of left stellate ganglion (LSG). Furthermore, studies have shown that renal sympathetic ablation (LRA) may be anti-arrhythmia. Therefore, we hypothesized that renal sympathetic intervention may affect VA by modulating LSG activity in a cesium-induced long QT canine model.

**METHODS**  
Twenty-four dogs were randomly divided into three groups, group 1 (n = 8, LRS), group 2 (n = 8, LRA), group 3 (n = 8, LRS followed LSG ablation). Ventricular effective refractory period (ERP), heart rate variability (HRV), serum norepinephrine, BP elevation in response to LSG stimulation and LSG activity were measured before and after autonomic intervention. Following, dose injection of cesium was conducted and then early afterdepolarization amplitude, VA prevalence and tachycardia threshold as measured by dose of CsCl were compared among these groups.

**RESULTS**  
In group 1, 3-hour LRS significantly decreased ventricular ERP at all sites and HRV, increased serum norepinephrine and LSG neural activity, and augmented BP elevation in response to LSG stimulation as compared to group baseline. In group 2, however, LRA resulted in a reverse result. Furthermore, no significant change was shown in ventricular ERP, HRV, serum norepinephrine, BP elevation in response to LSG stimulation or LSG neural activity in group 3. As compared to group 1, the early afterdepolarization amplitude and VA prevalence were significantly reduced, and the tachycardia threshold was significantly higher in group 2 and group 3.

**CONCLUSIONS**  
LRS and LRA might facilitate and prevent VA, respectively, by modulating LSG neural activity in cesium-induced long QT canine model.

**GW26-e2420**  
Danhiog Injection Prevents Nitroglycerin-induced Tolerance in Rat  
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**OBJECTIVES**  
Danhiog Injection (DHI) is a traditional Chinese medicine consisted by two herbal medicines, Radix et Rhizoma Salviae Miltiorrhizae and Rhizoma Flos Carthami, which is used in clinic as a remedy for cardiovascular diseases. The early studies indicated that the mio has protective effect on endothelial cells. This study aimed to investigate the potential effects of DHI on nitroglycerin-induced tolerance in rats.

**METHODS**  
Nitroglycerin-induced tolerance was induced by pretreatment with nitroglycerin (50 mg/kg) once a day for three days on Wistar rats. DHI was co-treated in this period. In addition, the maximal relaxation response curve was drawn and malondialdehyde (MDA) level, nitric oxide synthase (NOS) activity and cyclic guanosine monophosphate (cGMP) level were measured. In vitro, the tolerance was induced by exposure the isolated thoracic aorta obtained from rats to nitroglycerin (10 μM) for 60 min with pretreated of DHI. In addition, nitric oxide synthase inhibitor (L-NAME), ornithine cyclase inhibitor (ODQ) and cyclooxygenase inhibitors (Indo) were used to study the mechanism.

**RESULTS**  
DHI could significantly reduce the MDA content (P < 0.05), increase NO and cGMP (P < 0.05) in comparison with nitroglycerin-induced tolerance. Pre-exposure of aortic rings to nitroglycerin significantly reduced the relaxation to nitroglycerin (P < 0.05) in comparison with controls. Treatment with DHI could increase relaxation’s response compare with nitroglycerin-induced tolerant aortic rings (P < 0.05).

**CONCLUSIONS**  
DHI significantly attenuates nitroglycerin-induced tolerance in vivo and in vitro. The mechanism is at least partly based on endothelium protection and anti-oxidant.

**GW26-e4536**  
The study of asic acid effects on isoprenaline induced cardiac hypertrophy  
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**OBJECTIVES**  
To study whether asic acid (AA) attenuate cardiac hypertrophy through the mitogen-activated protein kinase (MAPK) and phosphoinositide 3-kinase (PI3K) signaling.

**METHODS**  
Cardiac hypertrophy in mice was induced by subcutaneous administration of isoproterenol. 30 mice were divided into three groups (10 mice per group): Sham (saline), ISO (saline) and ISO-AA. ISO has a protective effect on endothelial cells. This study aimed to investigate the potential effects of DHI on nitroglycerin-induced tolerance in rats.

**RESULTS**  
Compared to the ISO group, the HW/BW, HW/TL, CSA were obviously reduced in the ISO group. Compared to the ISO group, the HW/BW, HW/TL, CSA were obviously reduced.

**CONCLUSIONS**  
Our data suggest that AA can attenuate cardiac hypertrophy by modulating LSG neural activity in a cesium-induced long QT canine model.

**GW26-e4771**  
Protective and antiapoptotic effects of luteolin on oxidative injury in H9C2 cardiomyocytes  
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**OBJECTIVES**  
Luteolin, a falconoid compound in many types of plants, plays important cardioprotective roles in cardiovascular...
diseases. But its underlying mechanism needs to be further elaborated. The purpose of this study was to identify the protective and anti-apoptotic effects of luteolin on oxidative injury in H9C2 cardiomyocytes and to clarify the underlying mechanism.

**METHODS**

A model of hydrogen peroxide (H2O2)-induced H9C2 cells oxidative injury was established in vitro. The changes in cell viability were examined with an MTT assay to determine the available concentration of H2O2 and luteolin. 2', 7'-Dichlorofluorescin diacetate (DCFH-DA) and flow cytometry were used to detect the effect of luteolin on ROS level and apoptosis degree respectively. We also used Real time fluorescent quantitative PCR to examine the effect of luteolin on the regulation of caspase-3, bcl-2, bax and the ratio of the latter two.

**RESULTS**

We found that incubation with various concentrations of H2O2 (0.25, 50, 100, 200) for 1h caused dose-dependent loss of cell viability. Treatment with 10μM luteolin effectively decreased the level of H2O2-induced injury. Result of DCFH-DA indicated that 100μM H2O2 also increased the ROS level in H9C2 cells, while luteolin obviously reversed this increase. Moreover, the flow cytometry result suggested that luteolin could effectively inhibit apoptosis induced by H2O2 in H9C2 cells. PCR results further verified that luteolin downregulated the expression of caspase-3 caused by H2O2, and upregulated the ratio of bcl-2 and bax.

**CONCLUSIONS**

Luteolin protects H9C2 cells from H2O2-induced oxidative injury by reducing intracellular ROS level and decreasing apoptosis. The protective and anti-apoptotic effects of luteolin may be related to its regulation on decreasing caspase-3 and increasing the ratio of bcl-2 and bax.

**GW26-e0461**

Exosomes secreted from dendritic cells induce angiogenesis by cardiac microvascular endothelial cells after myocardial infarction

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**OBJECTIVES**

It has been reported that the infiltration of dendritic cells (DCs) significantly increases in infarcted myocardium after myocardial infarction (MI) and DCs ablation impaired angiogenesis post-MI in mice. However, the mechanism of how DCs exert effects on MI is still not completely understood. Exosomes (EX) has been known as the messenger between cells, this study was aimed to clarify whether EXs derived from DCs induce angiogenesis by cardiac microvascular endothelial cells via paracrine signaling post-MI.

**METHODS**

DCs were derived from mouse bone marrow-derived DCs (BMDCs) and primary cultured rat cardiac microvascular endothelial cells (CMECs) were used to form vasculatures. BMDCs suspensions were incubated with the supernatant of necrotic or normal cultured HL-1 myocardial cells for 24 hrs respectively (as necrosis or control group). EXs were then isolated from the supernatant of BMDCs (DC-Exosomes, DC-EXs) and identified by electron micrograph and Western blotting using the exosomal marker. EXs were added to CMECs and the angiogenesis was evaluated by measuring the tube formation and VEGF expression. Finally, the expression profiling of miRNA in splenic DCs of MI mice was analyzed by Affymetrix miRNA 4.0 chip assays and the significantly up-expressed and highly enriched miRNAs were certified both in DCs and EXs by quantitative RT-PCR.

**RESULTS**

Confocal imaging showed EXs could be uptake by CMECs. Compared to the control group DCs, EXs from necrosis group significantly up-regulated the expression of VEGF in CMECs and enhanced the tube formation by CMECs. Some miRNAs including miR-16-5p, 23a-3p, 150-5p, and 126-3p which are associated with angiogenesis were significantly up-regulated and highly enriched in DCs from necrosis group and compared to those from control group.

**CONCLUSIONS**

The current results indicated that exosomal miRNAs especially angiogenenic miRNAs could be secreted from DCs and promote angiogenesis by CMECs post-MI. Our study may present a potent and novel DCs-based therapeutic approach for MI treatment.