modulation of the AMPK/SIRT1 and TLR4/NF-κB signaling pathways. Moreover, combined therapy of Cβ2R agonist and AD-MSc3 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
Dilare Adi,1,2 Zhenyan Fu,1,2 Xiang Xie,1,2 Ying Yang,1,2 Yitong Ma1 Department of Cardiology, First Affiliated Hospital of Xinjiang Medical University, Urumqi, 830054 P.R., China; 2Xinjiang Key Laboratory of Cardiovascular Disease Research, Urumqi, 830054 P.R., China

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, rs2161829 and rs21613329) of 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 rs2161829 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 rs2161829 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
Xiaoaya Zhou, Hong Jiang Renmin Hospital of Wuhan University

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 (LSG) 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.

RESULTS A total of 8 mice were randomly divided into three groups (10 mice per group): Sham (saline), IND (saline) and IND + AAD (saline). In addition, nitric oxide synthase inhibitor (L-NAME), ornithine cyclase inhibitor (ODQ) and cyclooxygenase inhibitors (Indo) were used to study the mechanism. 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 compared 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 aspartic acid effects on isoprenaline induced cardiac hypertrophy
Webin Zhang, Zhenguo Ma, Wenyong Wei, Sichi Xv, Chunxia Wan, Qizhu Tang Department of Cardiology, Renmin Hospital of Wuhan University

OBJECTIVES To study whether aspartic 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. AA has a previous study effect on endothelial cells. This study aimed to investigate the potential effects of DHI on nitroglycerin-induced tolerance in rats.

RESULTS 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 (0-10 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. 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 compared 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.