mRNA in neointima from day 5 to day 14. At day 21, compared with no Electroporation-mediated group, no transfection group and GFP transfection group, AT2R transfection reduced I/M intimal/medial area ratio significantly (0.85±0.1, 1.32±0.19, 1.51±0.19, 1.49±0.25, p <0.01). No significant difference between no Electroporation-mediated, group, GFP group and no transfection group was observed.

Conclusion The results of this study provide evidence that electroporation is an effective means for introducing naked AT2R DNA into the blood vessel wall and gene transfer of AT2R in vessel wall may effectively inhibit VSMC proliferation and neointimal hyperplasia in the rat carotid arteries after balloon angioplasty.

**e0054** RECOMBINANT ADENO-ASSOCIATED VIRUS SEROTYPE 9 TRANSFECTION OF RATS H9C2 CELLS IN VITRO
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Objective To evaluate del transfection efficiency using recombinant adeno-associated virus Serotype 9 mediated enhanced green fluorescent protein (rAAV9-EGFP) to rats H9C2 cells and the impact on growth of H9C2 cells.

Methods rAAV9-EGFP was transfected into H9C2 cells at different multiplicities of infection (MOI=1×105, 1×106, 1×107). EGFP expression in the cells was observed under inverted fluorescence microscope and the EGFP-positive cell percentage determined by flow cytometry. Alamar Blue assay was used to assess the proliferation of the transfected cells.

Results The cells with rAAV9-EGFP transfection at MOI of 1×106 and 1×107 began to exhibit EGFP expression 1 days del after transfection and the cells transfection at MOI of 1×105 began to exhibit EGFP expression 2 days after transfection. The fluorescence intensity increased with the MOI used for transfection. EGFP expression reached the maximum on day 4, at the point of which the transduction efficiency of rAAV9-EGFP in H9C2 cells was (14.1±0.2)%, (35.1±4.8)% and (56.8±0.1)%.

Conclusions rAAV9-EGFP gene can be del transfected in a stable manner and efficiently expressed in H9C2 cells without causing cell growth inhibition. This del The results of this study played foundation (del) (provides a platform) for further research.

**e0055** RELATIONSHIP BETWEEN GENETIC POLYMORPHISM OF CYTOCHROME P4502C19 AND CORONARY ARTERY DISEASE IN XINJIANG UIGUR POPULATION
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Objective To study the relationship between cytochrome P450 family 2C19 subfamily genetic polymorphism and coronary artery disease in Xinjiang Uigur Population.

Methods There are (del) 570 CAD patients and 190 control subjects, who (del) were tested for the genotypes for the (of) CYP2C19 SNPs rs4986893 with the methods (use) of PCR and analysed by restriction fragment length polymorphism. Blood press, (del) () plasma biochemical results and smoking were also included (del) (assessed). Statistic analyses were (del) (analysis) was performed with the SPSS 12.0 software package. The Hardy–Weinberg equilibrium was tested in all the groups.

Results Uigur, the genotypes distribution of the control group and the CHD group were in the Hardy–Weinberg equilibrium the (del), p>0.05(for case p=0.2, for control p=0.068). The frequency of AA, AG and GG were 0.5%, 0.5% and 94.3% in CAD group, compared with the control group that were 0.6%, 5.6% and 93.8% respectively. For x²=0.107, the p value was 0.743. There was no significant difference in between the two groups. But the (amt?level???) blood glucose in the two groups had a difference (del). Smoking was also different in the CAD and the control groups (p=0.0002).

Conclusions The distribution frequency of the CYP2C19 SNPs rs4986893 genotypes in the Uigur population with or without CAS is not significantly different with the blood glucose and smoking both have correlation with CAD (del).

**e0056** CORRELATION BETWEEN VIABLE MYOCARDIUM AND PZF IN SWINE MODEL OF ACUTE MYOCARDIAL INFARCTION
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Objective The aim of this study is to explore the relationship between the viable myocardium and Pzf of related coronary artery in swine model of acute myocardial.

Methods 13 swines, received coronary arteriography after anaesthetic tracheal cannula, the pressure of coronary arteries were measured through guiding wire and the distal flow velocity of left anterior descending coronary arteries measured through doppler wire. Drawing the pressure-flow velocity coordinate graph, then calculated Pzf. Blocked up distal of left anterior descending coronary arteries by angioplasty balloon for 60–90 min to establish the model of acute myocardial infarction. The Pzf were calculated repeatedly. Analysis the Pzf difference between before and after the model of acute myocardial infarction established. Taken out of those swines hearts after executed them, sliced the cardiac muscle to dye with TTC to detect the viable myocardium, then cutted them from the left ventricular myocardium. The percent value of area viable myocardium were estimated by calculated the ratio of weight of viable myocardium to whole left ventricular myocardium Drawing the percent value of area viable myocardium—Pzf after the model of acute myocardial infarction established coordinate graph and observed relationship between them.

Results 11 out of 13 swines were successfully established models of acute myocardial infarction by coronary occlusion. 2 swines died of entricular fibrillation after their coronaries were occluded 45 and 65 min respectively. The viable myocardium could be detected by using TTC dyeing. The result of statistics analysis confirmed that the percent value of area viable myocardium were negative correlation with Pzf.

Conclusion The swine model of acute myocardial infarction can be established successfully by coronary occlusion with angioplasty balloon. The percent value of area viable myocardium were negative correlation with Pzf, which showed that Pzf was a new method to predict the area of viable myocardium after acute myocardial infarction.

**e0057** CORRELATION OF ABCA1 GENE R219K IN THE UIGHUR NATIONAL MINORITY IN XINJIANG WITH LIPIDMETABOLISM AND THE RISK OF MI
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Objective To study the correlation of ATP binding cassette transporter A1 (ABCA1) and the gene polymorphism of R219K in the
Uighur National Minority in Xinjiang patients with myocardiac infarction (MI), and to find the association of ATP binding cassette transporter A1 gene R219K common variant with the risk of the Uighur National Minority in Xinjiang patients with MI.

**Methods** The association of the gene polymorphism of R219K in 150 cases with the Uighur National patients with MI (test group) and 200 cases (control group) were detected by restriction fragment length polymorphism (RFLP) and PCR.

**Results** The 219K site genotype of ABCA1 gene in MI patients was obviously lower than that in control group (p<0.001), and high density lipoprotein (HDL) in all K carriers was obviously higher than non-allele K carriers (p<0.05). Triglyceride (TG) was obviously less than in non-allele K carriers (p<0.05).

**Conclusion** Polymorphism of ABCA1 Gene R219K in the Uighur National is concerned with MI and HDL cholesterol and TG metabolism.

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**Objective** The purpose of the present study was to observe the changes of (in) gene expression and protein level of transient receptor potential channel (TRPC1) in isolated atrial myocardium after heart failure in rabbits.

**Methods** 20 rabbits were randomly divided into two groups: control group (n=10, sham-operation rabbits: 5) and heart failure (n=10) group. (A) Chronic heart failure model was produced by ligating left anterior descending coronary artery. 8 weeks after (the) operation, the mRNA expression and protein level of TRPC1 in atrial myocardia of rabbits were detected by RT-PCR and Western blotting, respectively.

**Results** The TRPC1 mRNA expression in the myocardium of the left auricular appendage was markedly increased in the heart failure group (0.295±0.008) compared to the control group (0.224±0.005) (p<0.05). The TRPC1 protein expression in the myocardium of the left auricular appendage was significantly increased in the heart failure group (0.572±0.011) compared to the control group (0.261±0.007) (p<0.05).

**Conclusion** The mRNA and protein expression of TRPC1 in atrial myocardia of rabbits with heart failure was significantly increased. TRPC1 may play a role in the vulnerability to (of) atrial arrhythmias in dilated atria with heart failure.

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**Objective** To evaluate the role of reduced fetal oxygen supply on morphological impairment in aorta from the adult offspring and further assess its susceptibility to sex-, hyperlipidaemia-, and postnatal hypoxaemia-related differences.

**Methods** Based on a 4×2 full factorial design consisting of four factors (maternal hypoxia, sex, hyperlipidaemia, and postnatal hypoxaemia), pregnant Sprague-Dawley rats were subjected to hypoxia for 3 h in a low pressure cabin with an oxygen concentration of 10%±1% from (for) 7 to 21 days of gestation and their offspring were subjected to high-fat diet feeding (at 10 months of age for 4 months) or hypoxia (at 12 months of age for 4 weeks). The histopathological observation and morphometric analysis of the thoracic aortas were performed in (on a) rat offspring at 16 months of age.

**Results** In a 16-mo-old maternal hypoxia offspring, the thoracic aortas exhibited lesions are similar to early events in atherosclerosis that involved impaired endothelial cells, thickening and fibration of intimas, infiltration of inflammatory cells to the subendothelial space, and migration and proliferation of vascular smooth muscle cells to the intima. In contrast, no detectable pathological changes were observed in the offspring without maternal hypoxia exposure. Morphometric analysis further demonstrated that prenatal hypoxia caused a significant thickening of intima (p<0.001) with a main effect of 5.5 μm, an approximately twofold increase compared with controls. In addition, there was a positive additive relationship between prenatal hypoxia and hyperlipidaemia on the intimal thickness (p<0.05). There were no other main effects or interaction among these four factors.

**Conclusions** Intraterine chronic hypoxia can induce early pathological appearances of atherogenesis in adult offspring. This effect was enhanced with hyperlipaemia but was unaffected by postnatal hypoxia or sex.

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**Objective** Our previous study has shown that resistin, one of the adipokines elevated in metabolic syndrome, is strongly associated with thrombosis complications in patients with metabolic syndrome. The mechanism, however, is far from elucidated. The present study was designed to observe the effect of resistin on the expressions of fibrinolytic factors, including tissue plasminogen activator (t-PA) and plasminogen activator inhibitor-1 (PAI-1) in human vascular endothelial cells, and to investigate the cellular mechanisms.

**Methods** Human umbilical vein endothelial cells (HUVEC) within 3–5 passages were incubated with different concentrations of resistin (25, 50, 100 ng/ml) for various times (durations) (6, 24, 48 h), respectively. Then, HUVEC were preincubated with MAPK activator inhibitor (ERK, p38 MAPK and JNK) inhibitors before resistin treatment. The supernatant protein levels of tPA and PAI-1 were measured by ELISA, (following which the) mRNA levels of tPA and PAI-1 were assayed by real time RT-PCR, The activation of MAPK was (then) characterised by western blot analysis.

**Results** (1) Resistin decreased tPA antigen level and increased PAI-1 antigen level in HUVEC both in time- and concentration-dependent manners (p<0.05), with the maximum effect of 50 ng/ml resistin on tPA and PAI-1 was measured by ELISA, (following which the) mRNA levels of tPA and PAI-1 were assayed by real time RT-PCR, The activation of MAPK was (then) characterised by western blot analysis.

**Conclusions** Resistin may accelerate thrombosis development by impairing fibrinolytic activity in vascular via p38 MAPK dependent pathway in metabolic syndrome. Abbreviations: mitogen-activated protein kinase (MAPK), plasminogen activator (t-PA), plasminogen activator inhibitor-1 (PAI-1), Human umbilical vein endothelial cells (HUVEC), enzyme-linked immunosorbent assay (ELISA), reverse transcription PCR.