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 lipop rotein (HDL) in allel K carriers was obviously higher than non-allel K carriers ($p<0.05$). Triglyeride (TG) was obviously less than in non-allel 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.

e0058 CHANGES OF TRANSIENT RECEPTOR POTENTIAL CHANNELS IN ATRIAL MYOCARDIUM OF RABBITS WITH HEART FAILURE

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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 (0.372 ± 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.

e0059 INTRAUTERINE CHRONIC HYPOXIA LEADS TO MORPHOLOGICAL IMPAIRMENT IN AORTA FROM RATS OFFSPRING

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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 $10\%\pm1\%$ from (for) 7 to 21 days of gestation and their offsprings 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 Intrauterine chromic 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.

e0060 RESISTIN IMPAIRS FIBRINOLYTIC ACTIVITY IN HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS VIA P38 MAPK PATHWAY

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Objective Our previous study has shown that resistin, one of the adipokines elevated in metabolic syndrome, is strongly associated with thrombotic 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 pathway (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 treatment for 48 h ($p<0.01$). Resistin also significantly reduced tPA mRNA level and upregulated PAI-1 mRNA level in HUVEC. (2) Neither PD98059 (ERK1/2 inhibitor) nor SP600125 (JNK inhibitor), but only SB203580 (p38 MAPK inhibitor) blocked the regulation of resistin on t-PA and PAI-1 significantly. (3) Resistin activated the phosphorylation of p38 MAPK in HUVEC.

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.