Conclusion Short-term use of CT-1C-terminal peptide early in reperfusion can reduce myocardial tissue injury and oxidative damage, as well as the extent of cardiomyocyte apoptosis, so that the extension of animal survival time; but the intraperitoneal injection of CT-1C-terminal peptide after a longer period of time reduced the tolerance of SD rats on ischaemia reperfusion injury, the tissue injury and the extent of oxidative damage increased significantly, and cardiomyocyte apoptosis have occurred in the surrounding area of infarction, and the animals have a shorter survival time.

**e0123** THE EFFECT OF DIABETES ON PROTECTION OF ISCHAEMIC POSTCONDITIONING IN MYOCARDIAL ISCHAEMIA-REPERFUSION INJURY

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Objective Study on the effect of diabetes on protection of ischaemia Postconditioning in myocardial ischaemia-reperfusion injury in isolated rat hearts.

Methods The type 2 diabetic rats were induced by the intravenous injection of streptozotocin (STZ) and high caloric diet. 60 Wister rats were divided into three groups randomly: Ischaemia- reperfusion in normal rats (A group), ischaemia postconditioning in normal rats (B group), ischaemia postconditioning in diabetic rats (C group). Rats were used for Langendorff isolated heart perfusion with 50 min of globe ischaemia and 60 min of reperfusion, then the models of ischaemia- reperfusion (A) were made. But to B and C, rat hearts were subjected to six cycles of 10 s of globe ischaemia and 10 s of reperfusion as ischaemia postconditioning during the early minutes of reperfusion. The levels of lactate dehydrogenase (LDH) in the coronary effluent and infarction size was determined by TTC staining. Phosphorylation of akt and gsk-3β were analysed by western blotting and immunohistochemical staining.

Results Ischemic postconditioning reduced LDH, CK and improved the haemodynamic parameters and reduced myocardial infarction size (29.50±3.4% vs 45.65±4.8%), phospho-Akt and phospho-GSK-3β expression increased markedly in B group. But compared A group there were no parenthood difference in C group. The level of LDH, CK didn’t decline and the myocardial infarction size were not reduced. phospho-Akt and phospho-GSK-3β expression in C group is more less than in B group.

Conclusion Ischemic postconditioning may significantly protect myocardium from reperfusion injury in isolated normal rat hearts. But in diabetic rats, the protection of Ischaemic postconditioning has no effect, the mechanism of this phenomenon maybe connected with lower expression of Phosphorylation of Akt and GSK-3β in the condition of diabetic and impaired Reperfusion Injury Salvage Kinase (RISK) signalling pathway (RISK pathway).

**e0124** EFFECTS OF OXIDATIVE STRESS AND GENDER DIFFERENCES IN SD RATS WITH HIGH-SALT HYPERTENSION VIA ACUTE SHORT-TERM COLD EXPOSURE

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Objective To perform high-salt hypertension model in SD rats and observe effects and oxidative stress and gender differences in SD rats, and then evaluate mechanism that blood pressure is affected by oxidative stress induced by cold environment.

Methods The male and female SD rats were randomly divided into four groups (n=8): male control group (MC), female control group (FC), male high-salt group (MS), female high-salt group (FS), MC and FC were fed regularly, MS and FS were fed with diet composed of 8% salt. Feeding period was 8 weeks. Four groups were fed regularly in ninth week; in the first 10 weeks, four groups were put into a 4°C artificial climate box in tenth, 1 h per day. Systolic blood pressure (SBP) in SD rats was measured every other day from 1st week to in first 4 days 9th week with tail cuff. Systolic blood pressure in SD rats was measured daily with tail cuff in late 3 days of 9th week and 10th weeks.24-h urine in each group was collected by biological metabolism, calculated accurately.

Results 1. High-salt diet for 8 weeks, MS group and FS group blood pressure was significantly higher than the control group the same sex (p<0.05). In 10th weekend four sets of blood pressure after cold exposure (BP) were higher, MS group and the FS group blood pressure (∆BP (∆BP=before exposure BP-after exposure BP) significantly higher than the control group the same sex (p<0.01). 2. 8 week FS and MS 24 h urine volume, urinary mALB, urinary RBP, urinary sodium, urinary potassium excretion higher than that of the same sex control group (p<0.01); MS and FS groups showed no change in exposure,3. After cold exposure high salt group 24 h urinary 8-iso-PGF2α excretion compared with before the cold exposure was significantly higher (p<0.01), serum Ang II levels than before the cold increased and serum NO concentration decreased (p>0.05), while no change in the control group. But before and after cold exposure the MS and FS, MC compared with FC no gender differences emerged. 4. After cold exposure NADPH oxidase activity and SOD activity, MS compared with FS, MC compared with FC does not appear gender differences, but the gender of the high salt group was significantly higher (p<0.05).

Conclusion 1. High-salt diet increased blood pressure, and high-salt diet on blood pressure after high salt gender differences emerged; resume normal diet of high salt hypertensive rats have a certain recovery of renal function, blood pressure, but high salt blood pressure, gender differences still exist in blood pressure. Control group with the same sex, short-term acute cold exposure for high-salt hypertensive rats blood pressure increased significantly; 2. Cold high-salt hypertensive rats after exposure, oxidative stress increased; but male and female rats after exposure to cold and oxidative stress between the gender differences are not shown.

**e0125** MMP-9 GENE POLYMORPHISMS CONTRIBUTE TO CORONARY ARTERY DISEASE RISK IN THE UIGHUR POPULATION OF CHINA

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Background Matrix metalloproteinase-9 (MMP-9) plays a pivotal role in early atherosclerosis, vascular remodelling and development of atherosclerotic lesion. The potentially functional MMP-9 polymorphisms may contribute to the susceptibility of coronary artery disease (CAD). We aimed to investigate the association between three SNPs (−1562C>T, R79Q, R668C) of the MMP-9 gene with CAD in the Uighur population of China.

Materials and methods 575 angiographic ally proven patients with coronary artery disease and 417 sex-matched and ethnically matched controls were genotyped for MMP-9 polymorphisms by the PCR-restriction fragment length polymorphism (PCR-RFLP) technique. Genotype/allele frequencies were compared in patients and controls using the χ² test. The relationship between the polymorphism of the MMP-9 gene and the severity of coronary stenosis was analysed also.

Results At MMP-9 -1562 locus, there were significant differences between patients and controls (p<0.05), leading to significant OR for TT genotype (OR=2.93, CI 1.03 to 8.72) and R allele (OR=1.85,
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**e0126 STUDY ON ANTI-OXIDATIVE FUNCTION OF FOUR KINDS OF SCHIZANDRAE LIGNANS**

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**Objective** To study the anti-oxidative function of schisandrin A (SinA), schisandrinB (SinB), schisandrolA (SolA) and schisandrin ester A (SesA).

**Methods** Using the method of the self oxidation method of pyrogallol, Fenton system.

**Results** The results shown that all of four kinds of schizandrae lignans have the inhibition function to Superoxide anion radical ($O_2^{-}$). SinB had the highest inhibition rate which could arrive at 68.74%; They also had the same inhibition to hydroxyl radical (OH) and SinB have the best effect.

**Conclusions** schisandrin A (SinA), schisandrin B (SinB), schisandrolA (SolA) and schisandrin ester A (SesA) can be used as a natural anti-oxidation for human cardiovascular disease treatment and preventive health care.

**e0127 DETERMINATION OF PULMONARY ARTERY PRESSURE AND CARDIAC OUTPUT IN RAT**

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**Objective** To establish a method for determination of pulmonary artery pressure and cardiac output in rat.

**Methods** 20 Sprague-Dawely rats were randomly assigned into two groups: control group and pulmonary arterial hypertension (PAH) group. Rats in PAH group were received a single subcutaneous injection of monocrotaline (60 mg/kg). The hand-made PE-50 catheters were inserted into pulmonary artery via right jugular vein, which we can perform mean pulmonary artery pressure. Similarly, cardiac output was detected through thermodilution method.

**Results** After 21 days, compared with control group, mean pulmonary artery pressure was significantly increased (17.4±1.8 mm Hg in control group vs 61.8±4.3 mm Hg in PAH group, respectively) and cardiac output was significantly decreased (150±5.8 ml/min in control group vs 71±6.7 ml/min in PAH group, respectively) in PAH group.

**Conclusions** This method is a simple and direct method to detect pulmonary artery pressure and cardiac output in rat.

**e0128 ANGIOTENSIN-(1-7) INHIBITS VASCULAR REMODELLING IN RAT JUGULAR VEIN GRAFTS VIA REDUCED ERK1/2 AND P38 MAPK ACTIVITY**

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**Objective** To evaluate the effects of Ang-(1-7) on vascular remodelling in vein grafts.

**Methods** A model of autologous jugular vein grafts in rats was established. With this model system, rats (n=12 per group) underwent autologous jugular vein graft transplantation (Ang-(1-7) and control groups), or a sham operation (sham group) in which grafting was not performed. Three days after operation, minipumps were installed for continuous infusion of Ang-(1-7) (25 µg/kg/h) or normal saline (control and sham groups) through the jugular vein.

**Results** 4 weeks, weight, blood pressure and heart rate were not significantly different between groups. Typical venous-graft hyperplasia, vascular remodelling, ERK1/2 activity, p38 MAPK activity and proliferating cell nuclear antigen (PCNA) and a-smooth muscle actin (a-SMA) expression present in the control group were attenuated by continuous Ang-(1-7) infusion. Tissue angiotensin II expression was increased in the Ang-(1-7) and control groups but was not different between the groups.

**Conclusion** The results of the present study indicate that exogenous Ang-(1-7) interferes with the vascular remodelling of autologous jugular vein grafts and significantly inhibits vein-graft intimal hyperplasia via inhibition of vascular tissue ERK1/2 and p38 MAPK activation. Thus, exogenous Ang-(1-7) improves the outcome of vein grafting via attenuation of vascular remodelling.

**e0129 EFFECT OF TETRANDRINE ON ANOXIA/REOXGENATION-INDUCED RELEASE OF PROINFLAMMATORY FACTORS IN CULTURED CARDIOCYTE OF NEONATE RATS**

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**Objective** To investigate the effect of tetrandrine on anoxia/reoxygenation-induced the release of myocardial enzyme LDH, CK and proinflammatory factors: TNF-$\alpha$, IL-1$\beta$, IL-6 in cultured cardiocytes of neonate rats.

**Methods** After cardiocytes were cultured in vitro successfully, it were divided into four groups: control group (CON), anoxia/reoxygenation group (A/R), tetrandrine group (Tet), simvastatin (Sim) in random. Each group was treated as follow: CON group - not treated anoxia/reoxygenation, continuous incubated 24 h under normal circumstance. A/R group - first anoxia incubate carried, cells were incubated on the non- saccharide non- serum culture medium, which saturate by 95% argon gases 2 h, reoxygenation incubate followed, cells were incubated in normal circumstance 24 h. 0.9% saline were added into culture fluid before the beginning of reoxygenation. Tet group and Sim group —the procedure of anoxia/reoxygenation was same to A/R group, the difference of these two groups was they added Tet (30 µmol/l) or Sim (10 µmol/l) respectively into culture fluid and incubated 60 min before anoxia beginning. LDH, CK, TNF-$\alpha$, IL-1$\beta$, IL-6 were detected after reoxygenation 24 h.

**Result** The LDH and CK were increased significantly in A/R, Tet, and Sim groups compared with CON group (p<0.01). The LDH and CK in Tet and Sim group were lower significant than A/R group (p<0.01). 2. The proinflammatory factors TNF-$\alpha$, IL-1$\beta$ and IL-6 were increased significantly in A/R, Tet, and Sim groups compared with CON group (p<0.01). And it were lower significant than A/R