

CI 1.34 to 2.54). These OR were higher in the sub-sample of smokers (3.87 and 2.06, respectively). Binary logistic regression analysis also confirmed that R allele carriers (CT and TT) have a higher risk of CAD (OR=2.07, CI 1.09 to 2.95). MMP-9 R279Q locus did not show significant differences between patients and controls. But QQ genotype and Q allele were significant risk factors in the smoker group. Q allele carriers (RQ and QQ) were also significantly associated with CAD risk in the smoker group (OR=1.43, CI 1.13 to 1.2.26). The R668Q locus did not show significant differences between two groups. And the MMP-9 polymorphism may not be useful as a predictor of the severity of coronary atherosclerosis.

**Conclusions** MMP-9 -1562T allele and TT genotype are significantly associated with CAD patients from the Uighur Population of China (Xinjiang). This association was stronger in smokers, supporting the conclusion that an interaction between MMP-9 activity and smoking augments CAD risk. Further studies with larger sample size are warranted to confirm these associations in different populations.

#### 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 ( $O_2^{\cdot-}$ ), 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 Sprgue—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 ( $17.4 \pm 1.8$  mm Hg in control group vs  $61.8 \pm 4.3$  mm Hg in PAH group, respectively) and cardiac output was significantly decreased ( $130 \pm 5.8$  ml/min in control group vs  $71 \pm 6.7$  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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**Objects** 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/REOXYGENATION-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 group: 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 26 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