Results One dog in R group died for anaesthesia, another dog in Re group had pacemaker problem that required us to remove them from the study. The SCL shortened after pacing (510±24 ms vs 430±18 ms, 380±26 ms, $p<0.05$), ERP shortened (146±18 ms vs 125±18 ms, 115±19 ms, $p<0.01$), dERP increased. The expression of Cx40 increased significantly in both experiment groups (1017.23±314.46 Int×mm^2 vs 1709.43±429.88 Int×mm^2, 2956.05±829.38 Int×mm^2 $p<0.01$), the expression of Cx43 increased significantly in above groups (915.21±338.93 Int×mm^2 vs 1859.94±412.11 Int×mm^2, 3048.83±931.35 Int×mm^2, $p<0.01$). The length and width of GJ in pulmonary vein sleeves shortened (L: 0.381±0.034 μm vs 0.390±0.117 μm, 0.260±0.069 μm; W: 23.29±3.70 nm vs 22.38±2.46 nm, 18.54±2.56 nm, $p<0.05$), and the Termination/Side (T/S) ratio of Cx40 and Cx43 decreased (Cx40: 1.34±0.11 vs 1.16±0.07, 0.98±0.06; Cx43: 1.27±0.09 vs 1.10±0.07, 0.90±0.09, $p<0.05$).

Conclusions The vagus nerve originated from SVC-Ao fat pad plays a role in pulmonary vein reconstitution, and these are part of basal elements for the development and maintenance of experimental atrial fibrillation.

e0019 STUDY ON THE CHANGES OF MATRIX METALLOPROTEINASE-9 AND HIGH SENSITIVE C-REACTIVE PROTEIN IN PATIENTS WITH CAROTID INTIMA-MEDIA THICKNESS

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Objective To investigate the relationship between matrix metalloproteinase-9, high sensitive C-reactive protein and carotid intima-media thickness.

Methods 180 patients were selected. High-resolution ultrasound was used to scan carotid, brachial arteries of all patients in order to check and measure carotid intima-media thickness (CIMT), plaques of carotid arteries and diameter of brachial arteries at rest. Patients were divided into four groups: the normal carotid intima group, the difficuse proliferative carotid intima group, the stable plaque group and the unstable plaque group. Serum levels of MMP-9, hsCRP were determined with immunological method. The relationship between the results measured by ultrasound instrument and the concentrations of MMP-9, and hsCRP was analysed.

Results The serum levels of MMP-9 and hsCRP in the proliferative carotid intima group were higher than in the normal carotid intima group ($p<0.01$). The serum levels of MMP-9 and hsCRP in the unstable plaque group and in the stable plaque group were higher than in difficuse proliferative carotid intima group ($p<0.05$). The serum levels MMP-9 and hsCRP in the unstable plaque group were higher than in the stable plaque group ($p<0.05$).

Conclusion The increased concentrations of MMP-9 and hsCRP were closely correlated with the increment of carotid intima-media thickness, the unsteady of plaque.

e0020 COMPOUND HETEROZYGOUS NOVEL SPLICING MUTATION D202SP AND MISSENCE MUTATION G272D ON KCNQ1 CAUSED JERVEL AND LANGE-NIELSEN SYNDROME IN A CHINESE FAMILY

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Background Long QT syndrome (LQTS) is an inherited cardiac disorder characterised by QT interval prolongation on ECG, ventricular arrhythmias and sudden death. Two forms have been identified: autosomal dominant Romamo-Ward syndrome (RWS) without deafness and autosomal recessive Jervell and Lange-Nielsen syndrome (JLNS) with deafness.

Methods A Chinese family with JLNS was identified. Family members were diagnosed based on the presence of a QT interval prolongation on 12-lead ECG and a history of syncope, palpatation and deafness. Mutational screening in the KCNQ1 potassium channel gene was performed by direct DNA sequence analysis and Blast with Results: A female 12-y-o proband and her 6-y-o brother were diagnosed with JLNS in May of 2009. The QTc were 0.59 s and 0.60 s for the girl and boy, respectively. Both patients had their first syncope at the age of 2. The QTc for parents was normal. The mutation detection showed two mutations: one is a novel splicing mutation, a A to G change in the position of two bases before exon 3 (A 605-2 A→G), in the acceptor site of intron 2, which is always A followed by G. Another one is a G to A change at position of 815, which is a known missense mutation G272D reported before in a RWS European patient. Both JLNS patients were compound heterozygous for these two mutations D202sp and G272D. The father carries a heterozygous D202sp only, while the mother carries a heterozygous G272D. These two mutation were absent in 100 control alleles. The parents' marriage was not consanguineous. Because of the early age of first syncope and the less effectiveness of b-blocker for JLNS, video-assisted thoracoscopic left cardiac sympathetic denervation (LCSD) was performed successfully in June of 2009. Both patients were syncope free during 1-year follow-up after surgery at the dosage of 2.2 and 1.6 mg/Kg of proparanolol for the girl and boy.

Conclusion Our results suggest that the compound heterozygous mutation D202sp and G272D caused JLNS in the siblings of this Chinese family. To our best knowledge, this is the first report of compound heterozygous splicing and missense mutation to induce JLNS so far. The results expand the spectrum of KCNQ1 mutations causing RWS and JLNS. Further mechanism exploration will deep our understanding of this rare disease.

e0021 A NOVEL APPROACH OF PROTEOMICS TO STUDY THE MECHANISM OF ACTION OF GRAPE SEED PROANTHOCYANIDIN EXTRACTS ON AORTIC ARTERIOSCLEROSIS IN DIABETIC RATS

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Objective Diabetic macrovascular complications are the leading cause of mortality in diabetic patients. To prevent the development of this disease and to improve advanced arteriosclerosis, effective therapies directed towards the key molecular target are required. Grape seed proanthocyanidin extracts (GSPE) have been reported to be effective in treating arteriosclerosis, while little is known about the functional protein changes.

Methods We used streptozotocin to induce diabetic rats. GSPE (250 mg/kg body weight/day) were administrated to diabetic rats for 24 weeks. Serum glycated haemoglobin and advanced glycation end products (AGEs) were determined. Electronic microscope was used to observe the changes of aortic ultrastructure. Immunohistochemistry was used to evaluate the receptor of advanced glycation end products (RAGE) protein expression in aortic tissue. Consequently, 2-D difference gel electrophoresis and AutoFlex martix-assisted laser desorption/ionization time-of-flight mass spectrometry with LIFT technology or liquid chromatography electrospray ionisation mass spectrometry/mass spectrometry were used to investigate aortic protein profiles among the control, untreated, and GSPE treated diabetic rats.

Results GSPE significantly decreased aortic PWV, blood pressures, aortic medial thickness ($p<0.05$), and inhibited the migration of

vascular smooth muscle cells. GSPE significantly reduced the AGEs ($p<0.05$), and the expression of RAGE in aorta of diabetic rats. The expression of 23 proteins was found either up-regulated or down-regulated in the aorta of untreated diabetic rats. Only the expression of 15 proteins was found either down-regulated or up-regulated in the aorta of GSPE treated diabetic rats. Among these proteins, in comparison with the aortic tissue of diabetic rats, the differential proteomic analysis of the aortic tissue of diabetic rats, treated by GSPE further revealed the variation of fifteen proteins, namely, lamin A, ATP synthase alpha chain, proline arginine-rich end leucine-rich repeat protein precursor, LOC500183 protein, heat Shock Protein 27, enoyl-CoA hydratase, glutamate dehydrogenase, protein-L-isoaspartate (D-aspartate) O-methyltransferase 1, lactadherin, leucine amino-peptidase 3, adenylyl cyclase- associated protein 1, apolipoprotein A-I, catalase, Dermcidin, and fibrinogen β chain. In brief, the differentially expressed proteins were related to many important biological functions including metabolism, oxidative stress, signal transduction, cell proliferation, cell growth, apoptosis and heat shock.

Conclusion GSPE plays an important role against diabetic macrovascular complications. Our findings might help to better understanding of the mechanism of diabetic macrovascular complications, and provide novel targets for estimating the effects of GSPE therapy.

e0022 LOSARTAN ATTENUATED CARDIOMYOCYTE APOPTOSIS BY INCREASING AKT ACTIVITY IN AORTIC BANDED RATS WITH CHRONIC HEART FAILURE

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Objective The present study was undertaken to investigate the protective effects of losartan on cardiomyocyte apoptosis in chronic heart failure (CHF) rats induced by banding abdominal aorta.

Methods SD rats underwent abdominal aorta coarctation to induce CHF, confirmed by ultrasound cardiograph and Catheterisation, or sham operation, followed by 8 weeks treatment with Losartan or vehicle. Plasma NE was measured by ELISA, and plasma and tissue Ang II levels were measured by RIA. Cardiomyocyte apoptosis was examined by agarose gel electrophoresis and TUNEL's method. The mRNA levels of Bax and Bcl-2 were determined by RT-PCR and the protein expression of phosphorylated and total Akt were assessed by Western blot.

Results Losartan-treated CHF rats had lower LVEDP ($p<0.01$), higher LVEF ($p<0.05$), lower plasma NE ($p<0.05$) and myocardium Ang II ($p<0.05$), but higher plasma Ang II ($p<0.05$) than vehicle-treated CHF rats. Losartan-treated CHF rats had no obviously "DNA ladder" which was the character of apoptosis, and the apoptosis index was also reduced ($p<0.05$) with a lower expression of Bax/Bcl-2 gene ($p<0.05$) and a higher protein expression of p-Akt ($p<0.05$).

Conclusion Losartan might inhibit cardiomyocyte apoptosis and improve cardiac function in aortic banded rats by blocking Ang II to bind AT1-R and promoting the activation of Akt.

e0023 THE STUDY FOR RELATIONSHIP BETWEEN THE ANGIOTENSIN II TYPE 1 RECEPTOR GENE POLYMORPHISM AND ESSENTIAL HYPERTENSION IN KAZAKANS OF XINJIANG

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Objective To investigate the relationship between the A1166C allele polymorphism of angiotensin II Type 1 Receptor (AT1R) gene and the essential hypertension in Kazakans of Xinjiang.

Methods PCR and restriction fragment length polymorphism methods (PCR-RFLP) were used to detect the A1166C polymorphism of AT1R gene in Kazakans including 321 patients with hypertension and 203 normotensive controls. The frequencies of genotype distribution hypertensives and normotensives were studied.

Results The genotype frequencies of A1166C were AA 0.7664, AC 0.2274, CC 0.0062 in the hypertension group and the corresponding frequencies were 0.7537, 0.2463, 0 in the control group respectively; The A1166 and 1166C allele frequencies were 0.8801, 0.1199 respectively in hypertension group and 0.8768, 0.1232 in normotensive group; The distribution of A1166C-genotype and the allele frequencies of A1166/1166C were not statistically significant in hypertension group as compared to normotensive group ($p>0.05$).

Conclusion There was no significant association found between the A1166C polymorphism of AT1R gene and essential hypertension in Kazakans of Xinjiang. Abbreviations: Angiotensin II Type 1 Receptor (AT1R).

e0024 ALDEHYDE DEHYDROGENASE-2 G1951A GENE POLYMORPHISM AND DRINKING BEHAVIOUR IN MALES

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Objective To study the distribution of genotypes about aldehyde dehydrogenase-2 ($ALDH_2$) and its relationship with drinking behaviours in males.

Methods 226 males were enrolled for collecting blood samples and data about drinking behaviours. $ALDH_2$ genotypes were detected by PCR-restriction fragment length polymorphism (PCR-RFLP).

Results 1. The frequency of $ALDH_2$ G1951A mutant allele was 0.077; 2. The frequency of $ALDH_2$ mutant genotypes (GA+AA) was significantly lower in males whose daily alcohol consumption ≥32.83 g (mean of daily alcohol consumption) than those of <32.83 g ($p<0.05$). Comparing with GG, the subjects with $ALDH_2$ mutant genotypes (GA+AA) were significantly lower in alcohol consumption per occasion (Chinese white liquor), daily alcohol consumption and cumulative alcohol consumption, and had a shorter drinking years ($p<0.05$).

Conclusion $ALDH_2$ G1951A Polymorphism was found in males of Xinjiang, and it was associated with drinking behaviour.

e0025 ALDEHYDE DEHYDROGENASE-2 G1951A GENE POLYMORPHISM AND OBSTRUCTIVE SLEEP APNOEA SYNDROME IN MALE DRINKERS

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Introduction To study the relationship between aldehyde dehydrogenase-2 ($ALDH_2$) G1951A gene polymorphism and Obstructive Sleep Apnoea Hypopnoea Syndrome (OSAHS) in male drinkers.