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 aminopeptidase 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

doi:10.1136/hrt.2010.208967.22

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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

doi:10.1136/hrt.2010.208967.23

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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

doi:10.1136/hrt.2010.208967.24

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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

doi:10.1136/hrt.2010.208967.25

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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.

Methods 226 male drinkers were enrolled in this study with a standard polysomnography. $ALDH_2$ genotypes were detected by PCR-restriction fragment length polymorphism (PCR-RFLP).
Results (1) We detected three kinds of genotypes and two kinds of allele of ALDH2 gene G1951A polymorphism. The frequency of ALDH2 genotype GG, GA, AA were 0.854, 0.137, 0.009 and allele G and A were 0.923, 0.077 respectively in 226 male drinkers.

The genotypic and allelic frequencies were in Hardy—Weinberg equilibrium (=0.3606, p=0.5482). (2) The frequency of ALDH2 genotype GG/(GA+AA) (0.855/0.145) and allele G/A (0.925/0.075) in OSAHS patients had no statistical differences comparing with the control group (p>0.05. Analysing the data stratified with BMI, there was also no significant differences of the frequency of ALDH2 genotype and allele between OSAHS group and the control group either in normoweight or overweight subjects (p>0.05). (3) There were no any differences in AHI, the lowest SaO_2 and the longest apnoea duration between subjects with the two genotypes (GG and (GA+AA)) either in OSAHS patients or the control group.
Conclusion $ALDH_2$ gene G1951A polymorphism was found in male drinkers, it had no any association with OSAHS.

e0026 ASSOCIATION BETWEEN THE M235T, T174M POLYMORPHISM OF THE ANGIOTENSINOGEN GENE AND LEFT VENTRICULAR HYPERTROPHY IN ESSENTIAL HYPERTENSION IN KAZAKANS

doi:10.1136/hrt.2010.208967.26

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Objective To investigate whether the M235T, T174M polymorphisms of the angiotensinogen gene were associated with left ventricular hypertrophy (LVH) in Xinjiang Kazakans with essential hypertension.
Methods 86 patients with essential hypertension and left ventricular hypertrophy and 95 patients with essential hypertension and non-left ventricular hypertrophy. Left ventricular hypertrophy was evaluated by the electrocardiography voltage criteria and the Romhilt-Estespoint-score system. The AGT gene M235T, T174M polymorphisms were amplified by PCR and analysed by RFLP.
Results (1) The genotype distributions of the M235T and T174M in both groups were in agreement with Hardy—Weinberg equilibrium. (2) The frequencies of the AGT genotypes and alleles were not significantly different between LVH and NLVH with hypertension. (3) When analysing the frequencies of genotypes and alleles of M235T according to gender (p>0.05), no significant differences were found between LVH and NLVH. However, we found a significant difference in frequencies of genotypes and alleles of T174M in NLVH group according to gender.
Conclusion There are no associations between M235T genotype and T174M genotype with the presence of LVH in this study.

e0027 GROWTH DIFFERENTIATION FACTOR 15 INDUCE THE PROLIFERATION OF CARDIAC FIBROBLASTS IN A DOSE DEPENDENT MANNER

doi:10.1136/hrt.2010.208967.27

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Objective Growth differentiation factor 15(GDF-15) is a member of Transforming growth factor-β superfamily which is one of the most important profibrotic protein released during Inflammation. It has been suggested that GDF-15 seems to be related to the remodelling processes by anti-hypertrophic in cardiomyocytes. Cardiac fibrosis is one part of the important pathology mechanisms during remodelling. It was still unknown whether GDF-15 could influence remodelling by modulating cardiac fibroblast proliferation and extracellular matrix (ECM) metabolism. Therefore, our study aims to investigate the expression and effects of GDF-15 in the fibroblasts.
Methods Primary myocardial fibroblasts were isolated and cultured from neonatal rats and divided into six groups subjected to different conditions: 10^{-6}mol/l Endothelin-1 (ET-1), 10^{-5}mol/l norepinephrine (NE), 3 ng/ml TGF-β1, 5 pg/ml rhGDF-15, 150 pg/ml rhGDF-15 and no stimuli. The expression of GDF-15 mRNA was detected by Realtime-PCR and the expression of GDF-15 protein was measured by Enzyme-linked Immunosorbent Assay (ELISA). The proliferation of fibroblast induced by rhGDF-15 was measured by 3-(4, 5-dimethylthiazol-2-yl) 2,5-diphenyl tetrazolium bromide (MTT) and Flow cytometry; the migration of fibroblast was shown by Scarification test. The level of phosphor- ERK was determined.
Results GDF-15 is upregulated in ET-1, NE and transforming growth factor (TGF)β1 group; the GDF-15 mRNA is respectively 1.21±0.03, 1.84±0.09, 1.95±0.39 folds of control group (respectively p<0.05, p<0.05, p<0.01); and protein in the supernatant is respectively 3.27±0.81 pg/ml, 3.55±0.20 pg/ml and 3.75±0.70 pg/ml, vs 0.41±0.17 pg/ml of control group (respectively p<0.05, p<0.001, p<0.01). rhGDF-15 (150 pg/ml) increased the OD value as compared to control group (0.56±0.03 vs 0.46±0.02, p<0.05), but 5 pg/ml rhGDF-15 did not (0.43±0.01 vs 0.46±0.02, p>0.05). rhGDF-15 (150 pg/ml) could also raise the cell in S phase nearly three times more than control group (9.62±1.17% vs 3.80±0.78%, p<0.01). The scarification test showed that rhGDF-15 could enhance the migration of fibroblast. The ERK was activated after treatment with rhGDF-15.
Conclusion GDF-15 is upregulated by ET-1, norepinephrine and TGF-β1 in myocardial fibroblasts. rhGDF-15 (150 pg/ml) could enhance the proliferation and migration of fibroblast, which may participate in the progression of myocardial fibrosis and thus was associated with ERK activation. Abbreviations: Growth differentiation factor 15(GDF-15), Extra cellular matrix (ECM), Endothelin-1(ET-1), norepinephrine (NE), Enzyme-linked Immunosorbent Assay (ELISA), 3-(4,5-dimethylthiazol-2-yl) 2,5-diphenyl tetrazolium bromide (MTT), Transforming growth factor (TGF), Extracellular Signal-Regulated Kinase (ERK).

e0028 THE EFFECT OF ACUTE ACTIVATION OF ALDH2 ON MYOCARDIAL ISCHAEMIA/REPERFUSION INJURY IN RAT

doi:10.1136/hrt.2010.208967.28

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Background Our previous study combined several animal experiments conducted recently showed aldehyde dehydrogenase-2 (ALDH2) was correlated with myocardial ischaemia/infarction injury. But whether ALDH2 is beneficial was controversial. We speculated the way in which the ALDH2 activity was changed may be chiefly responsible for the controversy.
Objective To investigate the effect of acute activation of ALDH2 on myocardial ischaemia/reperfusion injury in rat.
Methods 20 male Sprague-Dawley rats were divided into five groups:sham group (n=3); control group (n=4) :occlusion of the left anterior descending coronary artery (30 min) followed by reperfusion (15 min); ethanol group (n=5):intraperitoneal injection of ethanol (0.5 g/kg) 60 min prior to ischaemia; GTN-ON group (n=3):18 h persistent nitroglycerin treatment (0.1 mg/h, delivered by a patch) combined with intraperitoneal injection of ethanol (0.5 g/kg) 60 min prior to ischaemia; GTN-OFF group (n=5):