accumulation of collagen, increased levels of connective tissue growth factor, trans-
forming growth factor β1, tumor necrosis factor-alpha, vascular cell adhesion mole-
cule 1, N-nitrotyrosine and 4-hydroxy-2-nonenal in the aorta.

Conclusions: These findings suggested that chronic IH may lead to aortic damages characterized by oxidative stress and inflammation, and MT may play a pivotal role in the above pathogenesis process.

GW25-e0790
Effects of (P) RR and PLC-beta3 activation on cardiac hypertrophy in hypertensive rats
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Objectives: To investigate the role of cardiac (P) RR activation on the expression of PLC-beta3, PKC and ERK1/2 and on cardiac hypertrophy in hypertensive rats with abdominal aortic ligation.

Methods: Seventy-five SD rats were divided into five groups (n=15 each group) as following: sham operated (SO), rats with the aortic ligation (AL), AL rats were given HRP (4ug kg-1 d-1, SC), AL rats given U73122 (40ug kg-1 d-1, SC) and AL rats given HRP (4ug kg-1 d-1, SC) and U73122. MAP was recorded using a tail-cuff method. After 4 weeks of treatment, levels of (P) RR, PLC-beta3, PKC and ERK1/2 in the heart were examined by RT-PCR and western blot. The surface area of cardiomyocytes was measured.

Results: The expression levels of (P) RR and PLC-beta3 significantly increased in the left ventricle in hypertensive rats (P<0.01, respectively). The surface area of cardiomyocytes and MAP rose markedly (P<0.01). HRR treatment significantly reduced the expression of (P) RR and U73122 suppressed the level of PLC-beta3. The combined treatment of HRP and U73122 significantly decreased levels of PKC-c and ERK1/2 in the heart (P<0.01). Meanwhile, the surface area of cardiomyocytes and MAP were decreased after the treatment (P<0.01).

Conclusions: This is the first report demonstrating that treatment of HRP and U73122 decreased levels of (P) RR, PLC-beta3, PKC-c and ERK1/2 in the heart. Meanwhile, the treatment reduced the surface area of cardiomyocytes and MAP. These findings indicate that cardiac (P) RR may activate PLC-beta3, PKC and ERK1/2 signals and result in hypertension and cardiac hypertrophy.

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GW25-e0850
Effect of acute high altitude exposure on lung function and relationship between lung function and AMS
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Objectives: To investigate the effect of acute high altitude exposure on lung function and the relationship between lung function and AMS.

Methods: We collected the lung function and Lewis Lake data of 73 subjects (age 18 to 26, male) at sea-level and jumumahcang (after five days Exposure to 3000m, 3900m).

Results: Compared with sea-level, lung function decreased in FVC, MMF, V50, V25, White VE1, PEF, V5 did not change, but PEF/PVC, FEV1, PEF, MMF changed after altitude exposure. The relationship between lung function and AMS, there is no difference in FVC between AMS group and NON AMS group at sea-level, lung function of AMS group is statistically significant lower than NON AMS group in PVC, MMF at high altitude; there is differences between AMS group and NON AMS group in the rate of change of PVC, MMF; logistic regression analysis showed that the rate of change of the PVC was independent risk factors, correlation analysis showed that the change of PVC and the change of oxygen saturation is relative.

Conclusions: This study was supported by the National Natural Science Foundation of China (81270336) and Shandong Natural Science Foundation (ZR2009CM074).

GW25-e0876
Role of GRK4 variant 142V in the regulation of renal ETB receptor in hypertension
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Objectives: The endothelin receptor B (ETBR) regulates blood pressure and water and electrolyte balance by engendering natriuresis. In hypertensive states, the ETBR-mediated diuresis and natriuresis is impaired. However, the underlying mechanisms are not clear. G protein-coupled receptor kinase 4 (GRK4), whose locus gene 4p16.3 is linked to essential hypertension, cause sodium retention and increase blood pressure via impairment of renal dopamine receptor and enhancement of renin-angiotensin system functions. Due to the higher activity of GRK4 in kidney from spontaneously hypertensive rats (SHRs) and hypertensive patients, we hypothesize that GRK4 might be the cause of ETBR impairment in hypertension.

Methods: Experiments were carried out in male anaesthetized spontaneously hyper-
tensive rats (SHR) and in normotensive Wistar-Kyoto (WKY) rats. The ETB receptor agonist, BQ-3020 (0.1,0.5,1.0ug/kg/min) were infused via supra-renual artery at a rate of 0.04ml/min for 40 minutes. The same experiments were conducted in GRK4 A142V and GRK4 Wild Type transgenic mice. The ETBR function were also checked in the wild-type and A142V transfected renal proximal tubule (RPT) cells from mice.

Results: We found that diuresis and natriuresis of ETBR agonist, BQ3020, in Wistar-
Kyoto (WKY) rats, which was impaired in SHRs. The GRK4 expression was higher in renal cortex from SHRs as compared with WKY rats. In GRK4 A142V transgenic mice, it resulted that ETBR-mediated diuresis and natriuresis was impaired compared with Wild type. In wild-type transfected cells, activation of ETBR inhibited Na+-K+-ATPase activity; while in A142V transfected cells, the inhibitory effect was lost. There are co-localization and co-immunoprecipitation between ETBR and GRK4 in RPT cells. The linkage of ETBR/GRK4 was higher in wild-type cells than in A142V cells. Similar phenomenon was found in the kidney from WKY and SHRs, SHRs had higher ETBR/GRK4 linkage, accompanied with higher ETBR phosphorylation, which might account for the impaired ETBR function in hypertension.

Conclusions: This study provides a mechanism by which GRK4, via regulation of renal ETBR function, participates in the pathogenesis of hypertension.

GW25-e095
Cardiac Electrical Activity Improved by Overexpression of the Sarcolemmal Reticulum Ca2+-ATPase in Rat Myocardial Failure After Myocardial Infarction Evaluated by Micro electrode Arrays Technology
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Objectives: To explore overexpression recombiant adenovirus (rAd) -mediated sarcoclemmal reticulum Ca2+-ATPase (SERCA2a) for cardiac rhythmicity and conductivity in rat heart failure after myocardial infarction and its possibly electrical mechanisms.

Methods: 26 adult male SD rats were randomly divided into three groups: sham group (n=10), rAd.β-gal group (n=8) and rAd.SERCA2a group (n=8). Sham operation consisted of thoracotomy and cardiac exposure but without coronary artery ligation. RAd.β-gal group and rAd.SERCA2a group were ligated the left anterior descending coronary artery for rat heart failure animal model after myocardial infarction, while the transfecting β-gal and SERCA2a gene into heart respectively. We used ultrasound electrocardiogram for evaluating cardiac diastolic and systolic function, ECG monitoring and microelectrode arrays (MEA) technology for myocardium electrical activity in vitro.

Results: rAd carrying SERCA2a and β-gal gene were successfully transfected in heart failure rats. rAd.SERCA2a group could improve failing heart function, the ventricular end diastolic volume, left ventricular end-systolic volume, left ventricular ejection fraction and fractional shortening. Compared with the sham group, ECG could be found that QT interval prolonged (94.7 ms vs. 74.4 ms, P<0.05) in rAd.β-gal group, and QT interval shortened and the incidence of conduction time was simultaneous in rAd.SERCA2a group, and the cardiac electrocardiogram for evaluating cardiac diastolic and systolic function, ECG monitoring and microelectrode arrays (MEA) technology for myocardium electrical activity in vitro.

Conclusions: rAd.SERCA2a could improve the cardiac function of rat heart failure model after myocardial infarction, and improve electrical activity of the heart. This is possibly due to the rAd.SERCA2a group. Further studies are needed to investigate the underlying mechanisms.