underwent surgical valve replacement. KCNN3 expression was assessed by real-time quantitative PCR analysis.

RESULTS The mRNA expression levels of KCNN3 were obviously increased in persistent atrial fibrillation patients compared with SR patients (p < 0.05). This was consistent with the change of current density of apamin-sensitive SK channels.

CONCLUSIONS Our results demonstrated that SK3 are involved in electrical remodeling of persistent atrial fibrillation. The SK3 channel was detected and redistributed from the peripheral to the whole cell. These findings provide a new insight into mechanisms of electrical remodeling of human persistent atrial fibrillation.

GW26-e1583 N-epsilon-(carboxymethyl)lysine is associated with cardiac diastolic dysfunction in alloxan-induced type 1 diabetic rabbits with early diabetic cardiomyopathy
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OBJECTIVES Elevated diastolic left ventricular (LV) stiffness is an important pathogenesis of cardiac diastolic dysfunction in diabetic cardiomyopathy. It is not fully clarified the contributors to passive diastolic stiffness of fibrosis, N-epsilon-(carboxymethyl)lysine (CML), and cardiac titin in early diabetic cardiomyopathy.

METHODS We established alloxan-induced type 1 diabetic rabbits. After 4 weeks, echocardiography and left ventricular cannulation were performed in diabetic (D, N = 15) and control rabbits (C, N = 12) to measure LV function. Masson stained tissue samples for calculation of collagen volume fraction (CVF). LV collagen solubility was quantified using the hydroxyproline assay. Immunohistochemical examinations were performed to quantify CML deposition. Cardiac total titin and titin N2B/N2BA ratio were measured by gel electrophoresis, and total titin, titin N2B and N2BA mRNA level expression was assessed by Quantitative real-time PCR.

RESULTS Integrated approach identified that 4 weeks of diabetes could induce LV diastolic dysfunction, while systolic function was preserved. There was no difference between diabetic and control rabbits in CVF (P > 0.05), while collagen solubility percentage (%) was reduced in diabetic group (D, 40.43±2.75 vs. C, 50.22±1.99; P < 0.05). Immunohistochemistry CVL scoring (score/mm2) showed increased in diabetic group compared with control (D, 39.46±3.27 vs. C, 18.95±1.78; P < 0.05), as well as there was a linear correlation between myocardial CML content and the LV relaxation parameters (T) (r = 0.797, P < 0.05). Diabetic hearts had significant increases expression of total titin (D, 3.46±0.58 vs. C, 1.0±0.2; P < 0.05), titin N2B/N2BA (D, 3.67±0.75 vs. C, 1.0±0.1; P < 0.05) and N2BA (D, 2.96±0.3 vs. C, 1.0±1; P < 0.05) in gene expression, but not in protein level (P > 0.05).

CONCLUSIONS In a short-standing alloxan-induced diabetic model, fibrosis and cardiac titin are not the necessary contributors to heart diastolic dysfunction. Increased myocardial CML has been definitively linked to cardiac diastolic dysfunction in early diabetic cardiomyopathy.

GW26-e1796 Effect of Ruanmailing containing serum on twinfilin-1 in PDGF-activated vascular smooth muscle cells
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OBJECTIVES It was confirmed previously by our group that Ruanmailing, one of traditional Chinese Medicine, may inhibit migration of vascular smooth muscle cells (VSMCs) in vitro induced by PDGF. In this study, we tested the effect of Ruanmailing containing serum on twinfilin-1 in PDGF-activated VSMCs to further illuminate the possible mechanism of Ruanmailing in anti-vascular remodeling.

METHODS Ruanmailing containing serum was prepared from rats after drug gavage every 12 hours for 5 days. VSMCs were isolated from thoracic aortas of Sprague-Dawley rats. Cells at 90% confluence were passaged by trypsinization and cells between 3-5 passage were used for experiment. Twinfilin-1 in cells was detected by confocal microscope with Alexa647 staining. Structure of actin filament was detected by FITC-phallodine labeling under confocal microscope.

RESULTS In quiescent cells without any stimulants in medium, twinfilin-1 was localized in cytoplasm prominently around the nucleus with no apparent stress fibers in cells. Compared with the control, after PDGF (10ng/ml) stimulation, twinfilin-1 was upregulated and redistributed mainly from peri-nucleus to the whole cytoplasm, especially lamellipodia and actin rich filopodia. Actin cytoskeleton was rearranged with a cluster of stress fibers intensely distributed in cytoplasm. Interestingly however, after treatment with 10% Ruanmailing containing serum, both expression and redistribution of twinfilin-1 induced by PDGF were suppressed. Twinfilin-1 scarcely localized to the lamellipodia and filopodia. The stress fiber was markedly reduced and loosely arranged simultaneously. Treatment of 5μmol/l LY294002 led to the same change of twinfilin-1 and cytoskeleton to that of 10% Ruanmailing containing serum.

CONCLUSIONS PDGF induces expression and redistribution of twinfilin-1 together with the reorganization of cytoskeleton in VSMCs. Ruanmailing containing serum may suppress twinfilin-1 in VSMCs and inhibit the rearrangement of actin cytoskeleton induced by PDGF.

GW26-e2200 Association of a SNP in the CYPI9 gene with risk of coronary heart disease
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OBJECTIVES There is a positive relationship between the imbalance of sex hormone ratio and coronary heart disease(CHD). Aromatase is the main enzyme in the conjugation of androgen and estrogen, and play an important role in the balance of the sex hormone levels. There is little related research. The goal of this study was to investigate the interaction between the SNPs in CYPI9 gene and coronary heart disease.

METHODS We collect 1706 blood samples and use propensity score matching techniques to match the confounding factors between the case and control. Finally, the case-control study including 596 individuals was conducted to identify the association of three SNPs in CYPI9 with CHD by using χ2 test or Fisher exact test and binary Logistic regression analysis. Differences in lipids and the parameters of echocardiography among individuals with different genotypes were assessed by using one way analysis of variance(ANOVA).

RESULTS The distribution of rs2298105 in CYPI9 gene showed a significant difference between CHD and controls(P=0.014) and the heterozygote GT has a significant lower risk than the homozygous GG and TT (GG(P=0.0063) and TT(P=0.057)). ANOVA indicated the blood lipids and the parameters of echocardiography among individuals with different genotypes did not differ from case and control, at the same time, although we find out the distribution of rs4774585 may be associated with CHD in Uygur population, after adjustment of potential confounders, the associations are not statistically significant.

CONCLUSIONS The GT genotype of rs2289105 in CYPI9 gene is associated with CHD and might be a protective genetic marker of CHD.

GW26-e2462 Effects of Rosuvastatin on Aortic Artery and Expression of IL-6 as well as hs-CRP in ApoE-/- Mice
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OBJECTIVES To aim the effects of Rosuvastatin on articular artery and expression of IL-6 as well as hs-CRP in ApoE-/- mice.

METHODS 30 male ApoE-/- mice were randomly divided into model group, interventional group and control group. Model group and interventional group were fed with high-fat diet, while control group were fed with normal diet. Mice in interventional group were administered orally with Rosuvastatin, once a day for 13 weeks. All intervention group were fed with high-fat diet, while control group were fed with normal diet. The GGT genotype frs 2289105 in CYPI9 gene is associated with CHD and might be a protective genetic marker of CHD.

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