cardiac troponin T (CtxtT), myosin heavy chain (MHC), and connexin-43, which relate to myocardial structure.

**Results:** The RNA interference (RNAi) knockdown of HDAC1 gene expression in BMSCs caused significant increases in the mRNA expression levels of genes related to myocardial development and structure in these cells (P<0.05), such as Nkx2.5 (4.89±0.12, 52.10±1.26); Connexin 43 (37.13±3.589), and CtnTf (41.18±3.139). While statistical analysis revealed no significant differences between the normal control groups (1.000±0.000) and the NC group with respect to the mRNA expression levels of the examined genes.

**Conclusions:** The targeted knockdown of the HDAC1 gene promotes the transdifferentiation of BMSCs into cardiomyocyte-like cells.

**GW25-e2286**

Role of ET-1 in the myocardial fibrosis induced by aldosterone in vitro

Li Shuqin1, Wu Yuzhou2, Zhu Jiabao2, Xing Ru2

1Department of Pathophysiology, Hebei Medical University, Shijiazhuang 050007, P.R. China, 2Department of Cardiology, Second Hospital of Hebei Medical University, Shijiazhuang 050000, P.R. China

**Objectives:** Myocardial fibrosis leads to impaired cardiac diastolic and systolic function and is related to adverse cardiovascular events. Cardiac fibroblasts are the most prevalent cell type in the heart and play a key role in the myocardial fibrosis. The endoheitin-1 (ET-1), which is up-regulated during tissue repair and fibrosis, induces lung fibroblasts to produce extracellular matrix. Recent studies showed that aldosterone (Ald) also played an important role in the process of myocardial fibrosis. Therefore, this study was performed to investigate the role of ET-1 in the myocardial fibrosis induced by Ald.

**Methods:** Cardiac fibroblasts were treated with Ald and/or ET-1 receptor antagonist BQ123. Cell cycle, proliferation, ET-1 expression of cells, ET content and collagen content in culture medium were measured by flow cytometry, MTT, immunocytochemistry, radiomunnoassay and hydroxyproline content, respectively.

**Results:** Compared with the control group, Ald group significantly changed cell cycle and increased proliferation of cardiac fibroblasts. Ald significantly increased ET expression and hydroxyproline content in culture medium at Ald group compared to the control group. Basal expression of ET-1 protein in the cardiac fibroblasts could be observed in the control group by immunocytochemical method. Compared to control group, the expression of ET-1 protein was obviously up-regulated after induced by Ald + BQ123 group. The Ald + BQ123 group could improve the cell cycle proliferation and collagen content in culture medium induced by Ald.

**Conclusions:** Our study suggested that ET-1 may play a key role in the myocardial fibrosis induced by Ald in cultured cardiac fibroblasts.

**GW25-e2334**

Purification of Growth Arrest and DNA Damage-inducible Protein GADD45 Alpha and its Expression in Human Platelet and Human Myocardial Tissue

Ye Yujia1, Wang Hailuo1, Ye Qianfang2, Ni Ruich1, Meng Zhaohai2

1Laboratory of Molecular Cardiology, Department of Cardiology, The First Affiliated Hospital of Kunming Medical University, 2Biotherapy Center, the General Hospital of Beijing Military Command, 3Department of Cardiology, Yan An Hospital of Kunming City

**Objectives:** The purpose of the study is to establish strategies of purification of GADD45α and to detect GADD45α’s expression in human platelet and human myocardial tissue.

**Methods:** First, the full-length coding sequence of GADD45α (498bp, GenBank Accession No.: NM_001924.3) was amplified from a human hepatocyte cDNA library using PCR and confirmed by sequencing. The PCR products were digested with BamHI and Xho I, and ligated into BamHI and Xho I digested pGEX-6P-1 vector with GST tag. The recombinant plasmid was transformed into E.coli BL21 (DE3). Positive clones were cultured in LB medium and induced with IPTG. The expression product was purified by affinity, molecular-exclusion and ion exchange chromatography and then condensed in the moderate concentration. Second, the expression of GADD45α was detected in human platelet and human myocardial tissue using RT-PCR.

**Results:** First, We established the strategy so as to clone, expression and the method of purification of GADD45α. Second, we detected GADD45α’s expression in human platelet and human myocardial tissue by RT-PCR.

**Conclusions:** Growth arrest and DNA damage-inducible protein GADD45 alpha is an 18.3 kDa acidic nuclear protein encoded by a DNA-damage-inducible gene, also termed DDB1, GADD45. GADD45α interacts with a number of molecules and play an important role in the regulation of DNA repair; cell cycle, cell proliferation, and apoptosis. GADD45α protein have been implicated in the development of atherosclerosis, coronary heart disease and congenital cardiovascular diseases. Research structure and function of GADD45α protein has a positive and profound significance to elucidate the pathogenesis of multiple diseases and to develop novel and effective treatment strategies. In this study, we established the strategy so as to clone, expression and the method of purification of GADD45α and determined GADD45α’s expression in human platelet and human myocardial tissue by RT-PCR. It provides experiment data for further structure and function study.

**GW25-e3109**

An Improved Method for Isolating Canine Atrial Myocytes in Electrophysiological Study

Wang Luchun, Xian-Hong Ou, Xiao-Hong Bin, Miao-Ling Li, Yan Yang, Xiao-Rong Zeng

Department of Electrophysiology, Institute of Cardiovascular Research, Lachu Medical College

**Objectives:** To establish an efficient and reliable method of isolating canine right atrial myocytes, we describe two steps enzymatic dissociation for isolation of canine atrial myocytes which were suitable for patch-clamp studies.

**Methods:** Firstly, right atrial appendages of canine were chopped into small tissue chunks and washed in Ca2+-free cardioplegic solution, then the tissue chunks were digested in collagenase (Type V) and protease (Type XXIV). Secondly, the isolated myocytes were harvested by centrifuging the tissue suspension. Finally, the cultures such as the ultra-rapid delayed rectifier K+ current (IKur), transient outward K+ current (Ito), and sodium current (INa) were recorded with the whole cell patch-clamp technique.

**Results:** Approximately half of the isolated myocytes had following properties: visible cross striations, well-striated, sharp edges and no spontaneously contracture. The current record was identified as typical Ito, Ito, INa.

**Conclusions:** The two-stage of enzymatic dissociation was a simple and reliable novel method to yield qualified myocytes and provided the ideal myocardial model for the study of the cardiovascular diseases.

**GW25-e3197**

β1-adrenoceptor Autoantibodies Affect Action Potential Duration and Delayed Rectifier Potassium Currents in Guinea Pigs

Zhao Yuehu1, Haisia Huang2, Yinhui Dai1, Xiao Li1, Tingting Li1, Suli Zhang1, Hua Wei1, Junxia Shang1, Ping Liu1, Huirong Liu1

1Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Capital Medical University, Beijing, China, 2Medical Experiment and Test Center, Capital Medical University, Beijing, China, 3Beijing Key Laboratory of Metabolic Disturbance Related Cardiovascular Disease, Beijing, China

**Objectives:** This work aimed to observe the effects of β1-adrenoceptor autoantibodies (β1-AA) on delayed rectifier potassium current and action potential duration and further explore the mechanisms of β1-AA-mediated ventricular arrhythmias.

**Methods:** β1-AAs were obtained from sera of patients with coronary heart disease and nonsustained ventricular tachycardia. With whole-cell patch clamp technique, action potentials and delayed rectifier potassium current were recorded.

**Results:** The results illustrated 0.1 μmol/L, β1-AA autoantibodies shortened action potential duration at 50% (APD50) and 90% (APD90) of the repolarization. However, at 0.01 μmol/L, β1-AA had no effects on either APD50 or APD90 (P>0.05). At 0.001 μmol/L, β1-AA significantly prolonged APD50 and APD90. Moreover, β1-AA (0.001, 0.01, 0.1 μmol/L) dose-dependently increased the slowly activated delayed rectifier potassium current, but similarly decreased the slowly activating delayed rectifier potassium current and increased L-type calcium currents at the different concentrations.

**Conclusions:** Taken together, a significant action potential duration reduction induced by β1-AA concentrations was responsible for the rapidly activating delayed rectifier potassium current increase and would contribute to repolarization changes and trigger the malignant ventricular arrhythmias in coronary heart disease patients.

**GW25-e3225**

Role of Cathespin B in the regulation of cardiac remodeling

We Qingqing1,2, Man Xiao1,2, Yuan Yuan1,2, Fang-Fang Li1,2, Jia Dai1,2, Zheng Yong2,1, Zhou Yan Bian1,2, Wei Deng1,2, Lu Gao1,2, Hongliang Li1,2, Qizhu Tang1,2

1Department of Cardiology, Renmin Hospital of Wuhan University, 2Cardiovascular Research Institute of Wuhan University, 3Department of Cardiology, Institute of Cardiovascular Disease, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology

**Objectives:** Cathespin B (CTSβ), a member of lysosomal cathepsin family that is expressed in the murine and human heart, was previously shown to participate in apoptosis, autophagy and the progression of certain types of cancers. Recently, CTSβ has been demonstrated to be involved in myocardial infarction (MI). We took this study to investigate whether CTSβ plays a critical role in pressure overload-elicted cardiac remodeling.

**Methods:** Aortic banding (AB) was performed to induce cardiac hypertrophy in CTSβ+/− or CTSβ−/− WT mouse. Echocardiography and cather-based measurements of hemodynamic parameters were performed after 8 weeks of AB. The extent of cardiac hypertrophy was also evaluated by pathological and molecular analyses of heart samples. Cardiomyocyte apoptosis was assessed by measuring Bcl-2 family proteins expression, caspase activation and terminal deoxynucelotidyl transferase dUTP nick end labeling staining. In addition, we established H9c2 cell lines with stable