cardiac troponin T (CttT), 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 (4.88, 95% CI: 5.455-6.262, P=0.032) and ankyrin G (37.131, 95% CI: 3.389), and CTD-TRF (41.183, 95% CI:13.193). 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 trans-differentiation of BMSCs into cardiomyocyte-like cells.

GW25-e2286

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

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**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. 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 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. Administration of BQ123 in 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**

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**Objectives:** The purpose of this 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 BamH I and Xho I, and ligated into BamH I and Xho I-digested pGEX-4P-1 vector with GST tag. The recombinant plasmid was transfomed 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, then conditioned in the moderate concentration. Second, the expression of GADD45α 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 DDT1, 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 pronounced 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 research and study.

GW25-e3109

**An Improved Method for Isolating Canine Atrial Myocytes in Electrophysiological Study**

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

**Methods:** Firstly, right atrial appendages of canines were chopped into small tissue chunks and washed in Ca++-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 myocardium such as the ultra-rapid delayed rectifier K+ current (IKur), transient outward K+ current (IKo) 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 IKur, IKo, 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**

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**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 mmol/L β1-AA autoantibodies shortened action potential duration at 50% (APD50) and 90% (APD90) of the repolarization. However, at 0.01 mmol/L β1-AA had no effects on either APD50 or APD90 (P<0.05). At 0.001 mmol/L, β1-AA significantly prolonged APD50 and APD90. Moreover, β1-AA (0.001, 0.01, 0.1 mmol/L) dose-dependently increased the rapidly activating 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 Cathepsin B in the regulation of cardiac remodeling**

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**Objectives:** Cathepsin B (CTSB), 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, CTSB has been demonstrated to be involved in myocardial infarction (MI). We took this study to investigate whether CTSB plays a critical role in pressure overload-elicted cardiac remodeling.

**Methods:** Aortic banding (AB) was performed to induce cardiac hypertrophy in CTSB−/− and CTSB+ + 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 deoxynucleotidyl transferase dUTP nick end labeling staining. In addition, we established H9c2 cell lines with stable