PCR (RT-qPCR). The level of membrane CD37 was measured by Flow Cytometry (FCM). The levels of NF-κB, p65, phospho-NF-κB p65(p-p65) and NFATc1 of the VSMCs were determined by Western blot. TRAF6 was blocked by small interfering RNA (siRNA). MIT was used to observe the cell proliferation. The cell supernatant of IL-2 and IL-6 were tested by ELISA.

**RESULTS**

The level of CD37 was induced by TNF-α in smooth muscle cells after stimulating 24h(40.00±2.85 vs 1.00±0.00, p<0.05). The cells proliferated when treated with the agonist-CD37 mAb. The mRNA level of NFATc1 was increased after stimulated by agonist-CD37 mAb(2.07±0.09 vs 1.00±0.00, p<0.05). The protein of NFATc1 was also increased. Simultaneously, the mRNA level of TRAF6 was also increased(1.39±0.16 vs 1.00±0.00, p<0.05)and p-p65 protein was also increased(p<0.05). When TRAF6 was blocked by TRAF6 siRNA, the expression of p-p65 and NFATc1 was decreased after stimulated by agonist-CD37 mAb compared with the stimulated group(1.15±0.07 vs 2.07±0.09, p<0.05). Pretreated the VSMCs with PDTC(30μmol/L) after 30min, the expression of p-p65 was inhibited in cytoplasm and nucleus, we stimulated the cells by agonist-CD37 mAb at this time, the mRNA level of NFATc1 was also suppressed(1.15±0.07 vs 2.07±0.09, p<0.05) and the NFATc1 protein was inhibited(p<0.05). The cell supernatant of IL-2 and IL-6 went up by agonist-CD37 mAb(0.91±0.1 vs 1.29±0.17, p<0.05, 0.51±0.04 vs 0.61±0.07, p<0.05), and decreased when NFATc1 was silenced(1.29±0.23 vs 1.00±0.00, p<0.05, 0.51±0.04 vs 0.50±0.03, p<0.05).

**CONCLUSIONS**

These results demonstrate that CD37 can be induced by TNF-α in VSMCs, and the CD37 may signaling affect the expression of NFATc1 in mice VSMCs through TRAF6/NF-κB pathway.

**GW26-e0806**

**Intracoronary Cardiomyocyte-Derived Cells for Heart Regeneration After Myocardial Infarction**

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**OBJECTIVES**

We aimed to assess safety of therapy with cardiomyocyte-derived cells (CDCs) in patients with left ventricular dysfunction after myocardial infarction.

**METHODS**

An independent data coordinating center randomly allocated 31 eligible participants of whom 25 were included in a per-protocol analysis. Mean baseline left ventricular ejection fraction (LVEF) was 39% (SD 12) and scar occupied 24% (10) of left ventricular mass. Biopsy samples yielded prescribed cell doses within 36 days (SD 6). No complications were reported within 24 h of CDC infusion. By 6 months, no patients had died, developed cardiac tumors, or MACE in either group. Four patients (24%) in the CDC group had serious adverse events compared with one control (13%; p=1.00). Compared with controls at 6 months, MRI analysis of patients treated with CDCs showed reductions in scar mass (p<0.001), increases in viable heart mass (p<0.001) and regional contractility (p<0.001), and regional systolic wall thickening (p=0.015). However, changes in end-diastolic volume, end-systolic volume, and LVEF did not differ between groups by 6 months.

**CONCLUSIONS**

Intracoronary infusion of autologous CDCs after myocardial infarction is safe and effective, warranting the expansion of such therapy to phase 2 study.

**GW26-e1045**

**ITRAQ-Based Quantitative Proteomic Analysis of Heart in a Rat Model of Exhaustive Training**

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**OBJECTIVES**

To explore the presence of informative protein biomarkers in the rat cardiac between the health group and exhausting group, The purpose of this study is to understand the difference in protein expression patterns between health and after exhaustive swimming and to evaluate the protein contributions to exhaustive training.

**METHODS**

20 male SD rats (male, the weight is 200±12g, SPF) were randomly divided into two groups, health group and exhausting group. The rats of exhausting group were swimming in the artificial pond, get the swimming method result in a model of Single Bout of Exhaustive swimming in Rats. After exhaustive swimming the hearts were collected immediately. We mainly adopted advanced 8-plex ITRAQ coupled with 2D LC-MS/MS technology for proteomics.

**RESULTS**

Analysis of proteomic data found that 122 proteins were identified with quantitative information between the two groups, with the expression level of 69 proteins had significant differences at least, compared with the healthy group(ratio > 1.2 or < 0.7, and P<0.05), of which 50 proteins were up-regulated, 19 proteins down-regulated.

**CONCLUSIONS**

This study provided a global view of potential mechanisms and potential biomarkers of heart, and demonstrated that ITRAQ combined with 2D LC-MS/MS quantitative proteomics is a powerful tool for biomarker discovery.

**GW26-e1069**

**Exendin-4, An Gutagone-like Peptide 1 Analogue, Attenuates Cardiomyocyte Hypertrophy Via AMPK/mTOR Pathway**

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**OBJECTIVES**

Cardiac hypertrophy is the pathological basis of the development of various cardiovascular diseases and is a major independent risk factor of cardiovascular morbidity and mortality. Glucagon-like peptide 1 (GLP-1), an incretin peptide released from the intestine, exerts various cardioprotective actions and is proved to contribute in the regulation of cardiac functions. However, the role of Exendin-4, a stable GLP-1 analogue, in the cardiac hypertrophy remains unclear.

**METHODS**

1. Primary neonatal ventricular cardiomyocytes were cultured to establish the model of cardiomyocyte hypertrophy induced by phenylephrine (PE).
2. RNA isolation and quantitative real-time PCR (q-PCR) were performed to evaluate the transcriptional level of hypertrophic markers such as ANP, BNP and beta-MHC.
3. Western Blotting was carried out to observe the change of the signaling pathway proteins levels.
4. The cardiomyocyte morphological change was manifested by Immunofluorescence staining and measurement of the cell surface area.

**RESULTS**

Our study demonstrated that exendin-4 attenuated cardiac hypertrophy induced by phenylephrine (PE), manifested by decreased hypertrophic markers such as ANP, BNP, beta-MHC and cell surface. Phosphorylated extracellular signal regulated protein kinase (phospho-ERK1/2) and phosphor-p38 mitogen-activated protein kinase (MAPK) protein levels didn’t change in the team treated by exendin-4 and PE. In addition, we discovered that exendin (9-39), a GLP-1 receptor antagonist, can remove the anti-hypertrophic effect of exendin-4, evidently. Moreover, we showed that the anti-hypertrophic effect of exendin-4 was also significantly reversed by compound C, an AMPK inhibitor, and rapamycin, a selective blocker of mTOR.

**CONCLUSIONS**

These results demonstrate that exendin-4 inhibit cardiac hypertrophy induced by PE via AMPK/mTOR pathway.

**GW26-e1380**

**Vascular Adventitia Calcification and Its Underlying Mechanism**

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**OBJECTIVES**

Previous research on vascular calcification has mainly focused on the vascular intima and media. However, we show here that vascular calcification also occur in the adventitia as well. The purpose of this work is to help elucidate the pathogenic mechanisms underlying vascular calcification.

**METHODS**

Mice were fed high fat diets (HFD) for 48 weeks, observing calcified lesions. Also included in this study were human subjects aged 60 years and older that had died of coronary heart disease, heart failure or renal failure, observing calcified lesions. Explant culture of fibroblasts, the primary cell type comprising the adventitia, was...