**GW26-e0093**

Expression of 5-Lipoxigenase and 5-Lipoxigenase-Activating Protein in Immune Cells

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**OBJECTIVES**

Arachidonic acid (AA), an omega-6 (n-6) fatty acid, can be converted to inflammatory leukotrienes (LTs) by 5-lipoxigenase (5-LO), which is activated by 5-lipoxigenase-activating protein (FLAP) and then lead to the expression of LTD4, which play a role in coronary heart disease (CHD). Expression of LTD4 is measured by Elisa assay. Evaluated using real-time RT-PCR and immunoblot (WB) assays, AA: 4.53 ± 0.12 vs. 0.40 ± 0.09; EPA: 0.13 ± 0.02 vs. 0.07 ± 0.01, P < 0.05). Losartan treatment has the similar results with PJS at the dose of 6 g/kg/day.

**CONCLUSIONS** Our data demonstrated that PJS treatment improved cardiac function, and reduced myocardial fibrosis at the early stage of MI. The results were associated with the inhibition of the expression of TGF-β/Smads signaling expression. Our results suggest that PJS may improve left ventricular remodeling possibly via attenuating TGF-β/Smads signaling pathway in the early period of MI.

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**GW26-e1430**

Fufangxueshuantong ameliorates diabetic cardiomyopathy in rats by attenuating cardiac and metabolic dysfunction

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**OBJECTIVES**

Diabetic cardiomyopathy (DCM) can cause diastolic and systolic dysfunction damage, resulting in myocardial ischemia and heart failure. Fufangxueshuantong (FXST), which is composed of Panax notoginseng, Atractylodes lancea and Scrophularia ningpoensis, has been used for the treatment of angina pectoris and diabetic retinopathy for years. However, whether FXST has an effect on DCM is unknown. Metabolic dysfunction occurs in hearts and contributes to DCM. Adenosine monophosphate-activated protein kinase (AMPK), an activator of cell proliferation, is a sensor of nutrient regulation, and AMPK activation may improve left ventricular remodeling possibly via attenuating TGF-β/Smads signaling pathways sense the metabolic demands and impinge on expression of genes encoding for metabolic enzymes (citrate synthase (CS), etc.). So the present study aims to demonstrate that whether FXST treatment can ameliorate cardiac function in diabetic rats and to illustrate its effect on energy metabolic mechanism.

**METHODS**

SD rats were randomly divided into 3 groups: normal group, diabetic group, and FXST group. 20 weeks after streptozocin induction, FXST or water was administered for 16 weeks. Cardiac dimensions and function were determined by echocardiography. LV myocardial (LVIDd) and systolic (LVIDs) were assessed in M-mode, and fractional shortening (FS) and left ventricular mass (LV mass) were calculated with the M-mode measurements. Doppler echocardiography was used to measure IVRT (isovolumic relaxation time) and E/A (Ratio of peak early to late transmitral blood flow velocities). After echocardiographic detection, the indicators of energy metabolism (CS, AMPK, PGC1α, sirt1) in hearts were detected through PCR.

**RESULTS**

Echocardiography revealed that in the diabetic group, a decrease in E/A and an increase in IVRT were observed in the rats (vs. normal group). Following treatment with FXST in the diabetic rats, E/A was found to be upregulated, (vs. diabetic group). In the diabetic group, a decrease in EF, FS, LV mass and an increase in LVIDd, LVIDs were observed in the rats (vs. normal group). Following treatment with FXST in the diabetic rats, EF and FS were found to be upregulated, while LVIDd and LVIDs were markedly decreased (vs. diabetic group).

Real time PCR analysis revealed decreased content of PGC1α, sirt1 and increased levels of CS, AMPK in the diabetic group when compared with the normal group. However, increased levels of CS, AMPK, PGC1α, sirt1 were observed in the FXST-treated group when compared with the diabetic group.

**CONCLUSIONS** Our study demonstrated that diabetes induced diabetic cardiomyopathy, characterized by both diastolic and systolic dysfunction and metabolic dysfunction in heart. And FXST protected DCM via attenuating cardiac function. In addition, therapeutic FXST administration can promote gene levels of energy metabolism. These findings provide evidence as to the cardiac protective efficacy of FXST to DCM.

**GW26-e1454**

Cardioprotective effect of propofol against oxygen glucose deprivation and reperfusion injury in H9c2 cells

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**OBJECTIVES**

The intravenous anesthetic propofol is reported to be a cardioprotective agent against ischemic-reperfusion injury in the heart. However, the regulatory mechanism still remains unclear.

**METHODS**

In the study, we used H9c2 cell line under condition of oxygen glucose deprivation (OGD) followed by reperfusion (OGD/R) to...