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 LTBA4, which play a role in coronary heart disease (CHD). Omega-3 (n-3) fatty acids are the metabolic counterparts to n-6 fatty acids and have anti-inflammatory effects. The n-6/n-3 fatty acid ratio may negatively affect CT lesions. However, whether alteration of tissue n-6/n-3 fatty acid ratio affects expression of 5-LO and FLAP in the cells was not well known.

METHODS
Immune cells from fat-1 transgenic mice (n=5) and WT mice (n=5) were harvested from the spleen and cultured for 24h. Mononuclear immune cells were isolated from the blood. Cellular n-6/n-3 fatty acid profiles were analyzed using gas chromatography. The mRNA and protein expressions of 5-LO and FLAP in the cells were evaluated using real time RT-PCR and immunoblot (WB) assays, respectively. The expression of LTBA4 level was assessed by Elisa assay.

RESULTS
He fat-1 transgenic mice showed a lower ratio of n-6/n-3 fatty acids than WT mice in both splenocytes and blood monocytes (AA: 4.53±0.07 vs. 1.32±0.04; EPA: 0.16±0.01 vs. 0.06±0.02; DHA: 0.59±0.02 vs. 0.31±0.04; FLA: 0.29±0.01 vs. 0.10±0.01; 5-LO: 0.07±0.01 vs. 0.03±0.01). The mRNA expressions of 5-LO and FLAP were significantly lower in the cells of fat-1 mice than in those of WT mice. Accordingly, the protein levels of 5-LO and FLAP were also markedly lower in fat-1 mice than in WT mice. The LTBA4 level was also lower in fat-1 mice than WT mice.

CONCLUSIONS
Our findings demonstrate that a decreased tissue n-6/n-3 fatty acid ratio reduces 5-LO and FLAP expression. This study suggests a role for tissue n-6/n-3 fatty acid ratio in the 5-LO pathway in immune cells, and a new mechanism for the anti-inflammatory effect of n-3 fatty acids, omega-3 fatty acid may play a role in the prevention of CHD.

A prescription of Jiashen reduces postinfarct left ventricular remodeling via inhibition of TGF-β/Smads signaling pathway

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OBJECTIVES
To explore the mechanisms underlying A prescription of Jiashen (PJS)-mediated cardioprotection, we determined whether PJS reduces early left ventricular remodeling via inhibiting TGF-β/Smads signaling pathway in a rat model of myocardial infarction (MI).

METHODS
Male Sprague-Dawley rats were subjected to sham-operation or MI by ligation of the left anterior descending coronary artery. The rats with MI were treated with vehicle, PJS at the dose of 3 g/kg/day, 6 g/kg/day, and gastricly given at the same dosage of normal saline. One weeks later, the rats were randomized into 3 groups: normal group, diabetic group, diabetic + FXST group. 20 weeks after streptozocin induction, FXST or water was administered for 16 weeks. Cardiac dimensions and function were determined by echocardiography. Cardiac enzyme activities (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.

RESULTS
Real time PCR analysis revealed decreased content of PGC1α, sirt 1 and increased levels of CS, AMPK in the diabetic group when compared with the normal group. However, increased levels of CS, AMPK in the diabetic group when compared with the normal group. However, increased levels of CS, AMPK, PGC1α, sirt 1) in hearts were detected through PCR. 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 ratio was found to be upregulated, vs. diabetic group). In the diabetic group, a decrease in EF, LS 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).

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.

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

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OBJECTIVES
The inotropic 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...