(AB). We also detected the expression profile of Sestrin family in the cardiac tissue from patients with HF (HF group: n =3) or with normal left ventricular function (control group: n =3).

RESULTS We found that all three members of Sestrin family had elevated expression in the early stage of cardiac hypertrophy and decreased expression in failing hearts. Sestrin1 had a higher expression level compared to sestrin1 and sestrin2 in mouse heart. As shown in the mouse heart, sestrin1 had the highest expression level and dropped far below the ordinary level at the failing stage, however, the expression of sestrin2 had no significantly difference between normal and failing human heart, while the sestrin 3 in HF group was more than 2 fold increased compare to the normal group.

CONCLUSIONS Sestrin isoform had high expression in hearts especially sestrin1, and presented in a process of dynamic change during the development and transition of cardiac hypertrophy to heart failure. The expression profile in human hearts was different from that in the mouse heart. Further investigation of Sestrin function and regulation may provide new insights in hypertrophic and failing heart.

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Berberine Prevents Atherosclerosis In Apolipoprotein E Knock Out Mice

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OBJECTIVES *Coptis Chinensis*, a Chinese herbal medicine, has been widely used in traditional Chinese medicine for a long time. Berberine, the main alkaloid of *Coptis Chinensis*, has been recently shown to possess extensive cardiovascular pharmacological activities. In present study, we examined the effects of Berberine on aortic atherosclerosis in Apolipoprotein E gene knockout mice (ApoE-/-) and explored the potential underlying mechanisms.

METHODS 30 ApoE-/- mice, fed a high fat diet from 6 weeks of age, were randomized into three groups (n=10): model group (ApoE-/-gourp), Berbrine group (ApoE-/-+Berbrine group) and Simvastatin group (ApoE-/-+Simvastatin group). 10 6-week-old C57BL/6 were treated as the control group, fed a basic diet. After 36 weeks, we sacrificed the mice for various measurements with ELISA, Western blot and Real-time PCR.

RESULTS The results showed that treatment with Berberine significantly reduced blood lipid. Berbrine has the effect of anti-proliferation of Smooth Muscle Cells. It could reduce the level of Hs-CRP, IL-6 and TNF-α in plasma. And it could reduce protein and mRNA expression of NF-κB and MMP-9 in aorta. There is no significant difference between the effects of Berberine and Simvastatin group.

CONCLUSIONS Berberine has the effect of anti-atherosclerosis and anti-inflammation in ApoE-/- mice. Our data have provided some experimental evidences to use Berberine in prevention and cure of atherosclerosis.

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Study on Cross-immunization Protection of Coxsackievirus B3 gene vaccine

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OBJECTIVES Explore the (resulting in myocarditis) cross-protective immunity coxsackievirus B3 gene vaccine for other types of Coxsackie virus infection.

METHODS Using molecular biological method, Competence bacterium were produced and transformed by pcDNA3CVB3VP1 recombinant plasmid.the recombinant plasmids were extracted a little and identified by sect-enzyme, PCR and sequence; the accredited gene vaccine were Proliferated abundantly and BALb/c mice were immuned then. After 4 weeks and 6 weeks, immunization serum was acquired. CVB1, CVB3, CVB3m and CVB5 were Proliferated and titrated by virological experimental method;cross-immunization protection were observed by Neutralization test.

RESULTS After pcDNA3CVB3VP1 gene vaccine were identified, it was showed that aimed CVB3VP1 fragment were conjuncted with plasmid pcDNA3; results of neutralization tests indicate that pathological changes of Hela cells infected by CVB1, CVB3, CVB3m and CVB5 were attenuated due to adding serum from mice bodies inoculated with coxsackievirus B3. Moreover attenuating degree of pathological changes of Hela cells was different which were infected by different types of viruses.

CONCLUSIONS Coxsackievirus B3 gene vaccine plays a protective role in infection of CVB1, CVBV3, CVB3m and CVB5.furthermore the protection is different in infection of CVB1, CVB3, CVB3m and CVB5.

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Monocyte activation in atherosclerosis

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OBJECTIVES Monocyte recruitment in arterial wall is an early event in atherogenesis. The classically activated macrophages (M1 subpopulation) and alternative-activated macrophage (M2 subpopulation) can arise from monocytes in response to different stimuli. In this study, we tried to evaluate the susceptibility to M1 and M2 activation of monocytes circulating in the blood of healthy individuals and patients with asymptomatic carotid atherosclerosis.

METHODS Cross-sectional clinical study was performed, which involved healthy donors, apparently healthy subjects with a predisposition to atherosclerosis, and patients with subclinical atherosclerosis. Study participants did not have clinical manifestations of atherosclerotic disease (ischemic heart disease, myocardial infarction, stroke history), did not take cardiotropic and lipid-lowering drugs, and did not have concurrent chronic diseases that may affect the results of the study (diabetes mellitus, oncopathology, collagenoses, asthma, endocrine diseases). Quantitative diagnostics of pre-atherosclerotic and atherosclerotic states was performed by high-resolution ultrasonography of carotid arteries followed my intima-media thickness (IMT) of common carotid arteries. To identify individual profiles of cell activation, monocytes were isolated from whole blood using magnetic CD14-positive separation. Functional analysis of monocyte activity included the measurement of concentrations of cytokines produced by cells under standardized conditions in response to pro-inflammatory stimulation with interferon-gamma or anti-inflammatory stimulation with interleukin-4. Secretion of TNF-α was considered to be a marker of pro-inflammatory activity of macrophages, while secretion of CCL18 chemokine as a marker of anti-inflammatory activity. Concentrations of TNF-α and CCL18 in the culture medium were determined by ELISA on day 1 or 6 after cell isolation, respectively.

RESULTS Surprisingly, we found a dramatic individual difference in susceptibility to activation between monocytes isolated from the blood of different subjects, regardless of the presence or absence of atherosclerosis. Monocytes in early atherosclerotic lesions may migrate back into the circulation, possibly serving as a lipid clearance system. We attempted to find the relationship between cholesterol in monocytes and their susceptibility to activation. There was an obvious trend towards a reverse association between intracellular cholesterol level and the ability of monocytes to become activated; however, correlation coefficients did not reach statistical significance. To reveal the reason of relationship between intracellular cholesterol level and monocyte susceptibility to activation, we used atherogenic modified LDL from patients with documented atherosclerosis to induce cholesterol accumulation in cultured cells. Although modified LDL induced cholesterol accumulation in cultured monocyte-derived cells neither cytokine secretion nor cytokine genes expression were affected.

CONCLUSIONS We believe these observations are very important because the identified differences may explain the individual features of the immune response in different subjects.

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Endothelial cells dysfunction induced by CD137-CD137L/CyclophilinA activation through oxidative stress via NF-kappaB pathways

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OBJECTIVES Endothelial cell (EC) dysfunction is a key event in the onset and progression of atherosclerosis. Our previous studies showed