smaller neointima areas (0.056±0.037 mm²) versus the model group (0.130±0.049 mm²), so did areas of the whole vessel wall (P < 0.05).

2. Arterial stenosis percentage in the model group and two treatment groups increased compared with the sham group at 28 days after injury. The Yiqihuoxuejiu-treated rats had a lower percentage of stenosis occupied by plaque (10.35±16.53%) than did the model rats (22.85±9.76%).

3. At 7 days after vascular injury, positive z-SMA expression decreased significantly in the media but increased in the neointima and adventitia of the model group and two drug groups (versus the corresponding layer of the sham group P < 0.01). At 28 days after injury, there was positive expression in the media and neointima while there was no expression in adventitia of the model and two drug groups. The Yiqihuoxuejiu and Captopril rats had lower positive areas percentage of z-SMA expression in the neointima than the model rats (P < 0.01). Collagen content of the two drug groups diminished significantly versus the model group (P < 0.01). The Yiqihuoxuejiu formula (10.3±2.43%) reduced collagen hyperplasia more than in the Captopril group (13.1±4.70 %).

4. At 28 days after injury, the ratio of type I/III collagen in the adventitia of the model group (4.26±0.39) obviously increased compared with that of the sham group (1.78±0.13). The ratio in the Yiqihuoxuejiu formula (1.92±0.67) was lower than that of the Captopril group (3.59±0.84) and the model group (P < 0.01).

CONCLUSIONS Yiqihuoxuejiu formula inhibits positive and negative remodeling by reducing hyperplasia in the adventitia in the early stages and suppresses intimal proliferation, reduces the collagen content and elevates adventitial compliance in the later stages. Inhibiting proliferation and secretion of adventitial content and elevates adventitial compliance in the later stages.

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Caloric Restriction Ameliorates Angiotensin Induced Mitochondrial Remodeling and Cardiac Hypertrophy
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OBJECTIVES To discuss Caloric Restriction Ameliorates Angiotensin can induced Mitochondrial Remodeling and Cardiac Hypertrophy.

METHODS We used forty four 4-week-old double transgenic rats (dTGRs) and 16 age-matched normotensive Sprague-Dawley (SD) rats described elsewhere. dTGRs and SD control rats were divided into 4 groups to receive diet regimens for 4 weeks: (1) dTGR controls (n=20); (2) dTGR-CR (60% energy of ad libitum energy intake; n=20); (3) SD controls (n=8); and (4) SD-CR (n=8). Blood pressure recordings measurement of vascular responses, echocardiography, assessment of diastolic dysfunction, tissue harvesting, myocardial morphology. Statistical Analyses Data are presented as mean±SEM. Statistically significant differences in mean values were tested by ANOVA and the Newman-Keuls post hoc test for comparisons of multiple groups. The differences were considered significant when P<0.05.

RESULTS CR effectively prevented Ang II - induced mortality in dTGRs No SD rats died during experiment. CR did not significantly influence blood pressure or heart rate in dTGRs and SD rats CR ameliorated Ang II - induced endothelial dysfunction in dTGRs. CR decreased the body weight of both dTGRs and SD rats The food intake (data not shown) and energy intake of CR rats were 60% of the corresponding ad libitum control groups. Ejection fraction and fractional shortening, 2 indicators of systolic function, were preserved in dTGRs, compared with normotensive SD rats, whereas isovolumic relaxation time was markedly longer in dTGRs, indicating diastolic dysfunction.

CONCLUSIONS CR and decreased nutrient-sensing pathway activity have shown to increase life span and protect against diabetes mellitus, cancer, and cardiovascular disease, and demonstrated that CR improves the recovery of left ventricular function after ischemia/ reperfusion injury and limits infarct size via mechanisms associated with AMPK activation.