After the cells were cultured for 24 hr, 5g/L MTT solution (20μL) was added, then dimethyl sulfoxide was used for vibration and dissolution. Optical density of each hole was detected by using enzyme-linked immunosorbent assay to analyze different concentrations of HCY and Rb1 on the proliferation of human mammary stems cells. Western blot was used to examine the protein expression of the cell inflammatory factors, total inflammatory factors (IL-1β, TNF-α, IL-6, interleukin (IL)-1β, IL-6), macrophage chemotactic protein (MCP)-1, and intercellular adhesion molecule (ICAM)-1. ELISA was used to detect the contents of the cell inflammatory factors.

RESULTS HCY of different doses inhibited the viability of HUVECs and IL-6, MCP-1 and ICAM-1, which were 0.71±0.15, 0.89±0.32, 0.80±0.27, 0.54±0.12 respectively, higher than that of the control group (all P<0.01). Western blotting and ELISA showed that homocysteine significantly increased the expression of TNF-α, IL-6, MCP-1 and ICAM-1, which are 0.71±0.15, 0.89±0.32, 0.80±0.27, 0.54±0.12 respectively, higher than that of 0 hour (all P<0.01). However, the expression levels of TNF-α, IL-6, MCP-1 and ICAM-1 of the 0.50mmol/L HCY-induced HUVECs treatment by Rb1 (20μmol/L) were 0.24±0.04, 0.15±0.03, 0.13±0.04, 0.22±0.06 respectively, not significantly different from those at the 0 hour (all P>0.01).

CONCLUSIONS Rb1 inhibits homocysteine-induced endothelial impairment and inflammatory response, the definite mechanism of which is expecting advanced study.

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Regulation of lipid metabolism and inflammation response by Danqi Pill through PPARα in coronary heart disease
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OBJECTIVES Lipid metabolism disorders and inflammation response play important roles in the pathogenesis of coronary heart disease (CHD). Danqi Pill (DQP) is prescribed routinely in the treatment of CHD. DQP has both anti-inflammatory and lipid-lowering effects. However, its pharmacological mechanisms are not yet fully understood. To provide insights into the pharmacological mechanisms of DQP, we investigated the effects of DQP on PPARα, which regulates lipid metabolism, and inflammatory factors involved in CHD.

METHODS 120 male Sprague-Dawley rats were randomly divided into sham-operated, model group, positive drug-treated group and DQP group. From the day after left anterior descending coronary artery occlusion operation, drugs were administered to rats intra-gastrically for 28 consecutive days. Contents of plasma lipids were evaluated by biochemical examination. Expressions of PPARα, key molecules in lipid uptake-transportation-metabolism pathway (including ApoA1, FABP and CPT1A) and key proteins in inflammatory pathway (PLA2, COX2, NF-κB and STAT3) were examined by western blot.

RESULTS Results showed that plasma levels of TG, LDL and VLDL increased, whereas HDL decreased in model group, indicating that plasma lipid metabolism was deregulated. In lipid uptake-transplantation pathway, protein levels of Apolipoprotein A1(ApoA1) decreased by 12.6% and fatty acid binding protein (FABP) decreased by 10.0% in model group. Expression of carnitine palmitoyl transferase 1A (CPT1A), which is the key enzyme in lipid utilization, was down-regulated by 25.1% and 92.5%, respectively. Expression of FABP and STAT3 were also up-regulated by 35.8% and 113.9% respectively, indicating that inflammatory pathway was activated. PPARα level decreased by 24.3%. In DQP-treated group, lipid disorders were regulated and the indicators returned towards normal level. Compared to model group, expression of ApoA1 was up-regulated by 59.5% and FABP increased by 29.0% in DQP group. Expression of CPT1A was up-regulated by 54.9%. DQP also inhibited PLA2 expression of PLA2 and COX2 were down-regulated by 25.1% and 24.8%, respectively. STAT3 expression showed no statistic difference. However, NF-κB level was down-regulated significantly by 56.2%, indicating that the anti-inflammatory effect of DQP is probably mediated by NF-κB. Expression of PPARα in DQP group also increased significantly by 141.3% compared with model group, demonstrating that DQP is an effective agonist of PPARα.