Abstract

Ischemic heart disease (IHD) is a group of heart diseases which include acute myocardial infarction, atherosclerosis and other coronary heart diseases. Ischemic heart disease is kind of a major chronic noninfectious disease and critical cause of death among chronic noninfectious diseases. 24 million people died of chronic noninfectious disease each year, in which 7.2 million people died of IHD, according to a report from WHO in 1997. The mechanism of Myocardial cell injury during IHD may relate to a series of irreversible pathological alterations. Therefore. the exploitation of naturally occurring drugs may provide new approach that can be effective in the treatment of ischemic heart disease.

Total flavonoids extract from leaves of natural plant Crataegus pinnatifida (hawthorn) possesses a wide range of pharmacological properties. However, little is known about the protective mechanism by which total flavonoids from leaves of Crataegues pinnatifida (TFFLOCP) exert cytoprotective effect in myocaridac cells from oxidative injury. This study focused on the protective mechanism of on H_2O_2-induced injury of H9c2. The appropriate apoptosis-inducing condition was investigated first. We found 150 μM H_2O_2 after incubation of 24hours may achieve the optimized condition for inducing apoptosis of H9c2 cells. Different concentration of TFFLOCP and different incubation time have been investigated by our previous study. Pretreatment of myocardiac H9c2 cells with different concentration groups of TFFLOCP (100μg/ml, 200μg/ml) showed significant effects in preventing oxidative stress injury by exposuring cells in 150umol H_2O_2 for 24 hours vs. control group. Further study indicated TFFLOCP prevented H_2O_2-mediated ROS generation and maintained normal mitochondrial membrane potential. The activity of caspase-3 was also inhibited in comparison to control group. Finally, we proved that high concentration group of TFFLOCP may exert strong protective

effects in myocardic H9c2 cells against H_2O_2-induced oxidative injury via modulation of ROS generation, maintenance of mitochondrial membrane potential and inhibition of caspase-3 activity.