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SIMVASTATIN PREVENT RABBIT ARTERIAL ATHEROSCLEROSIS DEVELOPMENT THROUGH INTERFERING NUCLEAR FACTOR-κB ACTIVATION

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Objectives To explore the effects of simvastatin on NF-κB activation in cholesterol diet rabbit artery and the underlying mechanisms of the beneficial effects of simvastatin on atherosclerosis (AS).

Methods Twenty-four male rabbits were randomly divided into three groups: normal diet groups, cholesterol diet groups and the simvastatin groups which received both cholesterol diet and simvastatin 5 mg/kg/days intragastrically, After 16 weeks rabbits were executed and the aortas were harvested for the pathologic and morphologic observations. Western blot was used to determine cytoplasmic p-I-κB α, I-κB α protein expression and cytoplasmic and nuclear NF-κB p65 protein expression of rabbit aortas.

Results Compared with normal diet groups, cholesterol diet groups demonstrated remarkably atherosclerosis in the arteries. And the expression of cytoplasmic p-I-κBa and nuclear NF-κB p65 expression was obviously increased, but I-κB α was markedly decreased in cholesterol diet groups (n=6; $p<0.01$). However, simvastatin could dramatically inhibit the formation of atherosclerotic plaques, suppress p-I-κB α protein expression, increase I-κB α protein expression, and promote NF-κB p65 translocation from cytoplasm to the nucleus (n=6; $p<0.01$).

Conclusions NF-κB activation might be involved in the process of atherosclerosis in high cholesterol diet rabbits. Simvastatin could ameliorates atherosclerosis (AS) through interfering NF-κB activation and translocation