[gw22-e0805]

NARINGENIN INHIBITS VASCULAR SMOOTH MUSCLE CELL FUNCTION INVOLVING REACTIVE OXYGEN SPECIES PRODUCTION MODULATION AND NF-κB ACTIVITY SUPPRESSION

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10.1136/heartjnl-2011-300867.241

Objective Growth factor and oxidative stress-mediated phenotypic modulation, migration and proliferation of vascular smooth muscle cells (VSMCs) play a pivotal role in the pathogenesis of atherosclerosis and restenosis. Naringenin, a flavonoid from plant foods, was shown to possess antioxidant and antiproliferative activities. We assessed the hypothesis that naringenin could inhibit the function of VSMCs in vitro.

Methods Cultured SD rat VSMCs were treated with naringenin (0, 10, 50, 100 μM) before challenge with thrombin (1 U/ml). Migration, proliferation and reactive oxygen species (ROS) production of VSMCs were assayed by transwell-migration, CCK-8 and reactive oxygen species assay kit, respectively. Differentiation characteristics, smooth muscle (SM)-α-actin and smooth muscle myosin heavy chain (SM-MHC), and nuclear factor (NF)-κB were studied in VSMCs treated with 10% fetal bovine serum (FBS) was studied by western blots.

Results Naringenin elicited a concentration-dependent inhibition of thrombin-stimulated VSMCs migration and proliferation. In the 10% FBS cultured VSMCs, the protein levels of differentiation characteristics, SM-α-actin and SM-MHC, were decreased; while 100 μM naringenin significantly inhibited these changes. Also, naringenin could significantly attenuate the reactive oxygen species production stimulated by thrombin. Meanwhile, treatment of naringenin (100 μM) decreased NF-κB expression in the 10%FBS cultured VSMCs.

Conclusion Naringenin exhibited inhibitory effects on VSMCs phenotypic modulation, migration and proliferation involving reactive oxygen species production modulation and NF-κB activity suppression, which suggests that naringenin may have therapeutic effects in the prevention of atherosclerosis and restenosis.