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Protective effects of honokiol against oxidized LDL-induced cytotoxicity and adhesion molecule expression in endothelial cells.

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Abstract

Honokiol, a compound extracted from Chinese medicinal herb Magnolia officinalis, has several biological effects. However, its protective effects against endothelial injury remain unclarified. In this study, we examined whether honokiol prevented oxidized low-density lipoprotein (oxLDL)-induced vascular endothelial dysfunction. Incubation of oxLDL with honokiol (2.5-20 microM) inhibited copper-induced oxidative modification as demonstrated by diene formation, thiobarbituric acid reactive substances (TBARS) assay and electrophoretic mobility assay. Expression of adhesion molecules (ICAM, VCAM and E-selectin) and endothelial NO synthase (eNOS) affected by oxLDL was investigated by flow cytometry and Western blot. We also measured the production of reactive oxygen species (ROS) using the fluorescent probe 2',7'-dichlorofluorescein acetoxymethyl ester (DCF-AM). Furthermore, several apoptotic phenomena including increased cytosolic calcium, alteration of mitochondrial membrane potential, cytochrome c release and activation of caspase-3 were also investigated. Apoptotic cell death was characterized by terminal deoxynucleotidyl transferase-mediated dUTP nick end-labeling (TUNEL) stain. The results showed that honokiol prevented the copper-induced oxidative modification of LDL. Honokiol also ameliorated the oxLDL-diminished eNOS protein expression and reduced the oxLDL-induced adhesion molecules and the adherence of THP-1 cells to HUVECs. Furthermore, honokiol attenuated the oxLDL-induced cytotoxicity, apoptotic features, ROS generation, intracellular calcium accumulation and the subsequent mitochondrial membrane potential collapse, cytochrome c release and activation of caspase-3. Our results suggest that honokiol may have clinical implications in the prevention of atherosclerotic vascular disease.

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