Effects of hesperidin on cyclic strain-induced endothelin-1 release in human umbilical vein endothelial cells

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摘要

Abstract

1. Hesperidin, a member of the flavanone group offlavonoids, can be isolated in large amounts from the rinds of some citrus species and has been reported to have antihypotensive and vasodilator properties. However, the mechanism of action of hesperidin in the prevention and treatment of vascular diseases remains unclear. 2. The vascular endothelium can produce potent contracting factors, such as endothelin (ET)-1, and endothelium-derived relaxing factors, such as nitric oxide (NO). The aims of the present study were to test the hypothesis that hesperidin may alter strain-induced ET-1 secretion and NO production and to identify the putative underlying signalling pathways in human umbilical vein endothelial cells (HUVEC). 3. Hesperidin (10 and 100 μmol/L) inhibited strain-induced ET-1 secretion. Hesperidin also inhibited strain-induced increases in the formation of reactive oxygen species and extracellular signal-regulated kinase (ERK) phosphorylation. 4. Hesperidin treatment of HUVEC enhanced NO production, endothelial NO synthase (eNOS) activity and the phosphorylation of eNOS and Akt. Furthermore, hesperidin modulated strain-induced ET-1 release and suppressed ERK phosphorylation in part via the NO/protein kinase G pathway. 5. In summary, we have demonstrated that hesperidin inhibits strain-induced ET-1 secretion and enhances NO production in HUVEC.

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