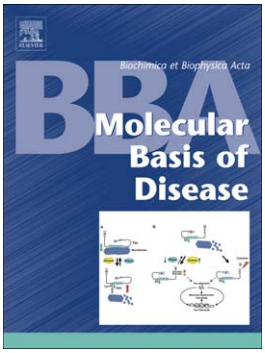

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Elatoside C protects against ox-LDL-induced HUVECs injury by FoxO1-mediated autophagy induction

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Abstract

The vascular endothelial cell injury induced by oxidized low-density lipoprotein (ox-LDL) is a major contributing factor to the pathogenesis of atherosclerosis. Elatoside C (EsC), a natural saponin isolated from Longya *Aralia chinensis* L., possesses anti-oxidative activity; however, there is still no report indicating EsC protects against ox-LDL-induced endothelial cell injury and the exact mechanisms of this protection. Recently, autophagy has attracted extensive attention on basis of its ability to modulate cell survival. Thus, we determined the role of autophagy in the protective effects of EsC against ox-LDL-induced human umbilical vein endothelial cells (HUVECs). Our results demonstrated that EsC pretreatment reduced ox-LDL-induced HUVECs oxidative injury, increased the number of autophagosomes and modulated the expression of autophagy related proteins. Moreover, autophagy inhibitor 3-methyladenine, chloroquine and BECN1 siRNA obviously abolished the anti-oxidative effects of EsC. Furthermore, our data indicated that EsC significantly increased nuclear FoxO1 expression level and FoxO1 siRNA markedly attenuated the protective effects of EsC. In conclusion, EsC attenuated ox-LDL-induced HUVECs injury by inducing autophagy via increasing FoxO1 expression level. EsC is thus considered as a potential drug for the treatment of atherosclerosis.

Keywords: autophagy, apoptosis, Elatoside C, ox-LDL, FoxO1, HUVECs

1. Introduction

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