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ATORVASTATIN DELAY THE SENESCENCE OF VASCULAR ENDOTHELIAL CELL INDUCED BY ANGIOTENSINII THROUGH REGULATING THE EXPRESSION OF BCL-2/BAX PROTEIN

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Objectives To explore the effects of Atorvastatin on the senescence in human umbilical vein endothelial cells (HUVECs) induced by AngiotensinII (AngII) and to study its potential molecular mechanism.

Methods The HUVECs were cultured in vitro and divided into three groups, the control group, AngII group (stimulated with intervened by AngII 10⁻⁶ mol/l for 48 h), Atorvastatin group (10⁻³ mol/l Atorvastatin was added to cell 1 h before 10⁻⁶ mol/l AngII). B-Gal stain and cell cycle analysis were used to identify cell aging status; and the expression of apoptosis-association genes Bcl-2 and Bax were detected by immunocytochemistry, and western-blot.

Results AngII stimulation enhanced the positive cell number of B-gal stained HUVECs, depressed cell proliferation. The AngII group inhibited the expression of Bcl-2 protein expression and increased the expression of Bax protein expression compared with the Atorvastatin group markedly ($p < 0.05$), Bcl-2/Bax was decreased significantly ($p < 0.05$) in the AngII group. The Atorvastatin group increased the expression of Bcl-2 protein expression and decreased the expression of Bax protein expression compared with the AngII group evidently ($p < 0.05$), Bcl-2/Bax was increased significantly ($p < 0.05$) in the Atorvastatin group.

Conclusions Atorvastatin probably delay the senescence of vascular endothelial cell induced by AngII through regulating the expression of Bcl-2/Bax protein.