

Methods Human umbilical vein endothelial cell (HUVEC) were cultured in vitro and intervened by AngII. HUVEC were divided into two groups, the control group, AngII group (stimulated by AngII 10^{-6} mol/l for 48 h). Human HUVECs were cultured in vitro and intervened by AngII. The cell living rate was observed by methyl thiazolyl tetrazolium (MTT), B gal staining and cell cycle analysis were used to identify cell aging status. Cell senescence was used to study by transmission electric microscopy. The expressions of apoptosis-association genes Bcl-2, Bax were detected by immunocytochemistry and ERK1/2 levels were detected by Western-blotting at different time points.

Results 10^{-6} mol/l AngiotensinII stimulation stimulated cell senescence. The cell living rate by AngII-induced cells was ($81.9\% \pm 4.1\%$, $p < 0.01$), the positive cell number of β -gal staining was significantly higher in AngII-induced cells than that in the control cells ($80.10\% \pm 6.81\%$ vs $0.18\% \pm 0.04\%$, $p < 0.01$); the cell cycle was at G0-G1 ($91.36\% \pm 6.45\%$, $p < 0.01$), S phase and G2/M phase were a tendency to disappearance in AngII-induced cells ($6.62\% \pm 0.42\%$ vs $2.12\% \pm 0.33\%$, $p < 0.01$), the senescent cells significantly increased under transmission electric microscopy. Bcl-2mRNA levels were time-dependently decreased, the ratio of Bcl-2/Bax was decreased markedly ($p < 0.05$). Phosphorylation of ERK1/2 began to increase and reach the peak at 24 h ($p < 0.01$).

Conclusions Cell senescence is possibly important factor for atherosclerosis. One of its molecular mechanisms might be associated with decreasing the expression level of Bcl-2 and the ratio of Bcl-2/Bax. There is a probability that activated ERK1/2 signal pathway is involved in the process of pathologic and physiologic reaction in the senescence of endothelial cell induced by AngiotensinII.

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EFFECT OF ERK1/2 SIGNAL TRANSDUCTION PATHWAY IN VASCULAR ENDOTHELIAL CELL APOPTOSIS

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¹Shan Hai-Yan, ¹Bai Xiao-juan, ²Chen Xiang-mei, ¹Bai Xiao-juan. ¹Departments of Gerontology and Geriatrics, The First affiliated Hospital of China Medical University; ²Department of Kidney, General Hospital of Chinese PLA

Objectives To explore the changes in extracellular signal-regulated protein kinase (ERK1/2) in endothelial cell senescence induced by AngiotensinII at the different time courses, and its possible molecular mechanism.