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% ±4.1%, $p<0.01$), the positive cell number of ß-gal staining was significantly higher in AngII-induced cells than that in the control cells (80.10%±6.81% vs 0.18%±0.04%, $p<0.01$); the cell cycle was at G0-G1 (91.36%±6.45%, $p<0.01$), S phase and G2/M phase were a tendency to disappearance in AngII-induced cells (6.62%±0.42% vs 2.12%±0.33%, $p<0.01$), the senescent cells significantly increased under transmission electric microscopy. Bcl-2mRNA levels were time-dependently decreased, the radio 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 radio 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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Objectives To explore the changes in extracellar signal-regulated protein kinase (ERK1/2) in endothelial cell senescence induced by AngiotensinII at the different time courses, and its possible molecular mechanism.