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EFFECT OF ANGIOTENSIN II ON APOPTOSIS AND EXPRESSION OF P38 MITOGEN-ACTIVATED PROTEIN

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Objective To investigate the effects of AngiotensinII (AngII) on apoptosis, and phosphorylation of p38 mitogen-activated protein kinase in endothelial cell, and its possible action mechanism.

Methods Human umbilical vein endothelial cells (HUVEC) were cultured in vitro and intervened by AngII. HUVECs were divided into two groups, the control group, AngII group (stimulated by AngII10^{-6} mol/l for 24 h). The early stage apoptosis was detected by flow cytometery with Annexin V-FITC/PI double staining, morphologic changes and percentage of apoptosis were assayed with acridine orange fluorescence staining. The expression of apoptosis-association gene Bcl-2 was detected by RT-PCR and Western-Blot at different time points. By means of Western-blotting, the activation of p38MAPK was observed at different time points.

Results 10^{-6} mol/l Ang II stimulated cell apoptosis. Bcl-2 mRNA and protein expression decreased markedly ($p<0.05$), the activation of p38MAPK began to increase and reach the peak at 18 h ($p<0.01$).

Conclusion Cell apoptosis is possibly an important factor for atherosclerosis. One of its molecular mechanisms might be associated with decreasing the expression level of Bcl-2. There is a probability that activated p38MAPK signal pathway is involved in the process of pathologic and physiologic reaction in the apoptosis of endothelial cell induced by AngII.