



Zinc finger protein A20 overexpression inhibits monocyte homing and protects endothelial cells from injury induced by high glucose

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ABSTRACT. Diabetes mellitus causes vascular lesions and may ultimately lead to atherosclerosis. One of the earliest steps in the development of atherosclerotic lesions is the adhesion of monocytes to endothelial cells of the vessel wall. It is currently unknown whether zinc finger protein A20 is able to protect endothelial cells from injury caused by high levels of glucose and monocyte homing. In our study, adhesion of monocytes to the vessel wall endothelium was detected by measuring the rolling velocity of monocytes along human umbilical vein endothelial cells (HUVECs). Activation of NF- κ B was analyzed through Western blot. HUVEC apoptosis was monitored by TUNEL *in situ* end-labeling and flow cytometry. High glucose concentrations (25 mM) stimulated monocytes, reducing the velocity at which they roll along HUVECs. Stimulation of monocytes with high levels of

glucose also induced HUVEC apoptosis. Overexpression of the zinc finger protein A20 inhibited monocyte recruitment, NF- κ B activation, P-selectin expression, and HUVEC apoptosis induced by high glucose levels. We conclude that zinc finger protein A20 can protect HUVECs from injury induced by high levels of glucose and potentially could be used to develop treatments against diabetic vascular lesions.

Key words: A20 gene; HUVECs; High glucose; Apoptosis; Monocyte homing