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vascular endothelial growth factor-A (VEGF-A) by vascular smooth muscle cells (VSMCs), but the underlying molecular events are not clear. The present study was designed to test the hypothesis that high glucose-induced VEGF-A expression and secretion from VSMCs are mediated by a proinflammatory transcription factor nuclear factor kappa B (NF-kappaB). Vascular smooth muscle cells were cultured in the presence or absence of high glucose (50 mmol/L) or cortisone (10 micromol/L) plus or minus the proinflammatory agent lipopolysaccharide (LPS, 100 ng/ml). The level of VEGF-A expression was determined by real time polymerase chain reaction and by Western blot analysis of VEGF-A protein; the level of VEGF-A secretion was determined by ELISA. In addition, the effect of NF-kappaB p65 knockdown on high glucose-induced VEGF-A secretion was studied. High glucose alone did not have any direct effect on VEGF-A expression or secretion. High glucose enhanced LPS-stimulated VEGF-A expression and secretion. NF-kappaB transcription activity was significantly increased in high glucose-treated VSMCs exposed to LPS, but not in VSMCs exposed to LPS alone. High glucose induced nuclear translocation of NF-kappaB p65 in VSMCs, which was blocked by the protein kinase A (PKA)

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