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Effect of bradykinin on renal mesangial cell proliferation and extracellular matrix secretion

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ABSTRACT. Recent studies have found that bradykinin (BK) plays a role in delaying glomerulosclerosis, although the mechanism of this phenomenon remains unclear. Mesangial cell proliferation (MCP) and extracellular matrix (ECM) secretion are important mechanisms for glomerulosclerosis. This study investigated the impact of BK on the platelet-derived growth factor (PDGF)-induced proliferation of mesangial cells, and evaluated its correlations with the extracellular signal-related kinase (ERK) signaling pathway. The results showed that on its own, 10-1000 μg/L BK promoted MCP and ECM secretion and induced ERK phosphorylation. However, BK administration after PDGF pre-incubation inhibited PDGF-induced MCP, ECM secretion, and ERK phosphorylation. The BK B2 receptor-specific antagonist, HOE-140, and tyrosine phosphatase inhibitor (OV) effectively blocked the function of BK. In summary, these results demonstrated that BK has a bidirectional effect on MCP and ECM secretion: when used alone, it promoted effects on these phenomena, but these effects were inhibited when combined with PDGF. This suggests that the role of BK might be achieved through inhibiting activation of the PDGF-induced ERK1/2 pathway.

Key words: Bradykinin; Platelet-derived growth factor; Mesangial cells; Extracellular signal-regulated kinase

