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Apamin attenuates PDGF-BB-induced vascular smooth muscle cell proliferation and migration through Akt and Erk signaling pathway

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The increased proliferation and migration of vascular smooth muscle cells (VSMC) are key process in the development of atherosclerosis lesions. Platelet-derived growth factor (PDGF) initiates a multitude of biological effects that contribute to VSMC proliferation and migration. Apamin is a component of bee venom has been known to block the Ca_2^{+-}activated K^+ channels. However, the potential role of apamin in regulation of VSMC proliferation and migration has not been identified. In this study, we investigate the inhibitory effect of apamin on PDGF-BB-induced VSMC proliferation and migration. Apamin suppressed the PDGF-BB-induced VSMC proliferation and migration with no apparent cytotoxic effect. In accordance with these findings, apamin induced the arrest of cell cycle progression at G0/G1 phase. Apamin also decreased the expressions of G0/G1 specific regulatory proteins including proliferating cell nuclear antigen (PCNA), cyclin D1, cyclin-dependent kinases (CDK) 4, cyclin E and CDK2 as well as increased the expression of $p21^{Cip1}$ in PDGF-BB-induced VSMC. Moreover, apamin inhibited PDGF-BB-induced phosphorylation of Akt and Erk1/2. These results suggest that apamin plays an important role in prevention of vascular proliferation and migration through the G0/G1 cell cycle arrest by Akt and Erk1/2 signaling pathway. Thus, apamin may be a promising candidate for the therapy of atherosclerosis.

Biography

Kwan-Kyu Park has completed his MD and PhD from Kyung Pook National University School of Medicine. He has published more than 38 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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