CE-2

Role of Phospholipase C-delta1 in the Bradykinin Receptor-Mediated Signaling in PC12 cells

Yong-Hyun Kim* and Kyong-Tai Kim
Dept of Life Science, Pohang University of Science and Technology

The role of a phosphoinositide-specific phospholipase C, PLC-delta1, in the bradykinin receptor-mediated signaling pathway was investigated using a clone of stably overexpressed PLC-delta1 in rat pheochromocytoma (PC12) cells. Stimulation with bradykinin induced significantly higher $[Ca^{2+}]i$ rise in PLC-delta1-overexpressed cells (PC12-D1) than in the wild type (PC12-W) and the vector-transfected (PC12-V) cells. The maximal effective concentration was similar (5 μM) in the cells. However, the half maximal effective concentration (EC_{50}) was much lower in PC12-D1 cells than in PC12-W or PC12-V cells. On the other hand, bradykinin-induced intracellular Ca^{2+} release from Ca^{2+} stores and IP_3 production didn't show any significant difference in the three kinds of cells. Interestingly, when cells were stimulated with thapsigargin, an inhibitor of Ca^{2+}-ATPase which activates Ca^{2+} release-activated Ca^{2+} channels (CRACs), PC12-D1 cells showed much greater $[Ca^{2+}]i$ rise than PC12-W or PC12-V cells. Furthermore, SK&F 96365, an inhibitor of receptor-mediated Ca^{2+} entry, more significantly decreased bradykinin-induced Ca^{2+} influx in PC12-D1 cells than in PC12-W or PC12-V cells. These results suggest possible involvement of CRACs in the PLC-delta1-mediated bradykinin receptor signaling in PC12 cells.