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ERK Activation Requires CaM Kinases in MCF-7 Breast Cancer Cells

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Abstract

A key signaling pathway involved in regulating cell growth and proliferation throughout the body is the ERK signaling pathway. ERK is activated via numerous pathways including intracellular calcium release downstream of G-Protein Coupled Receptors (GPCRs). Carbachol, a GPCR-agonist, both increases intracellular calcium and ERK activation in MCF-7 cells. ERK activation and control of cell growth may act through the transcription factor Elk-1. Our goal was to elucidate the specific proteins and kinases upstream of ERK in MCF-7 cells treated with carbachol. Secondly, we wanted to investigate the potential involvement of Elk-1 downstream of ERK. Carbachol treatment of MCF-7 cells triggered ERK and Elk-1 phosphorylation within 5 minutes. Interestingly, transfection of cells with shRNAs directed to either CaM KKα or CaM KIγ significantly inhibited carbachol activation of ERK. Phosphorylation of Elk-1, following carbachol stimulation, was also blocked in siERK2 transfected cells, suggesting that ERK2 is required for carbachol's activation of Elk-1. Our results suggest that carbachol treatment of MCF-7 cells activates ERK and cell growth through CaM KK, CaMKI, and an Elk-1-dependent pathway.