2012

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Abstract

The Extracellular Signal-Regulated Kinase (ERK) is part of a key, signaling pathway that regulates both transcription and translation in many cell types. Increases in intracellular calcium levels results in the CaM Kinase-dependent activation of ERK and cell growth in MCF-7 breast cancer cells. ERK has also been shown to play a role in the regulation of MCF-7 cell proliferation through control of downstream transcription factors including Elk-1. The hormone, Vitamin D has been suggested to play an inhibitory role on cancer cells by blocking ERK activation. Our goal was to evaluate the ability of E2 to activate Elk-1, through a CaM Kinase/ERK dependent pathway, in MCF-7 cells. We also examined Vitamin D's inhibitory regulation of ERK and Elk-1 activation. Interestingly, E2 stimulation of MCF-7 cells triggered Elk-1 phosphorylation an effect that was blocked by inhibiting either CaM KK or ERK. Similarly, E2 treatment of MCF-7 cells also triggered a significant increase in Elk-1-dependent luciferase activity. siRNA inhibition of CaM KK or ERK blocked E2-stimulated Elk-1 luciferase activity. Additionally, E2 triggered a sustained increase in ERK and Elk-1 phosphorylation, both of which were blocked by Vitamin D treatment. Vitamin D treatment of cells also inhibited Elk-1 luciferase activity downstream of E2 stimulation. In summary, our data suggests that E2 utilizes both CaM KK and ERK to activate Elk-1 transcriptional activity an effect that is blocked by the hormone Vitamin D.