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John M. Schmitt

George Fox University, jschmitt@georgefox.edu

Jessica N. Magill

George Fox University

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Transcription Factor Regulation by ERK and Estrogen in MCF-7 Cells

Jessica N. Magill and John M. Schmitt

Biology and Chemistry, George Fox University, Newberg, OR

Abstract

ERK is activated by increased intracellular calcium downstream of the hormone estrogen (E2). E2 activates ERK via the CaM Kinases, specifically CaM KK and CaM KI in MCF-7 cells. ERK may control cell growth and proliferation through Elk-1, Rsk, SRF, CREB, and numerous other molecules and nuclear targets. Vitamin D, a hormone, has proven to be an effective antagonist of ERK and MCF-7 breast cancer cell growth. Our goal was to evaluate if the E2 pathway working through CaM KK and ERK regulated the transcription factors Elk-1, CREB, and SRF. We also examined the ability of vitamin D to antagonize ERK activation of its downstream targets. Interestingly, E2 stimulation of MCF-7 cells activated both ERK and Elk-1 an effect that was blocked by inhibiting both CaM KK and ERK. E2 treatment of MCF-7 cells also triggered a significant increase in SRF and CREB phosphorylation in a CaM KK- and ERK-dependent manner. Dimerization of transcription factors may enhance DNA binding and gene expression. E2 stimulation of MCF-7 cells promoted the formation of a molecular complex between endogenous Elk-1 and SRF. Finally, E2 triggered a prolonged increase in ERK and Elk-1 phosphorylation, both of which were blocked by vitamin D treatment. Taken together our data demonstrates several transcriptional targets for E2 working through CaM KK and their inhibition by vitamin D signaling.