

2013

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Recommended Citation

Schmitt, John M. and Rofelty, Angela J., "Estrogen Receptor α Regulates ERK in MCF-7 Cells" (2013). *Faculty Publications - Department of Biology and Chemistry*. Paper 75.
http://digitalcommons.georgefox.edu/bio_fac/75

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Estrogen Receptor α Regulates ERK in MCF-7 Cells

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Abstract

Estrogen (E2) signaling significantly affects breast tumorigenesis by enhancing cell growth and preventing apoptosis. The actions of estrogen in MCF-7 breast cancer cells are mediated through a kinase pathway involving CaM KK, CaM KI, and ERK. Current research is examining the involvement of the Estrogen Receptor (ER) alpha (α) and beta (β) as well as G-Protein Coupled Receptor GPR30 in cell growth and proliferation. ER α is suggested to be responsible for ERK and perhaps CaM Kinase activation. Our goal was to evaluate if ER α , rather than GPR30 or ER β , mediates CaM KI and ERK activation upon treatment of MCF-7 breast cancer cells with E2. E2 treatment of MCF-7 cells led to a significant rise in CaM KI and ERK activation. This effect was blocked by pretreatment with the ER α antagonist MPP; however MPP did not block EGF stimulation of ERK. We then explored the role of ER β in E2-mediated ERK activation by pretreating cells with the ER β inhibitor PHTPP. PHTPP did not reduce ERK phosphorylation, suggesting ER β is not involved in activation of ERK. MCF-7 cells were transfected with siRNA against ER α to further examine the role of ER α in ERK regulation. Knockout of ER α in MCF-7 cells subsequently treated with E2 showed a significant decrease in ERK phosphorylation. To ensure the specificity of the siRNA for ER α , transfected cells were stimulated with EGF. ERK phosphorylation following EGF treatment was not inhibited in ER α -knockout cells. Taken together our data suggests that ER α is capable of rapidly activating both CaM KI and ERK in MCF-7 cells.