

Serine / threonine protein phosphatase 5 (PP5) participates in the regulation of glucocorticoid receptor nucleocytoplasmic shuttling

ABSTRACT

These studies propose a role for PP5 in the control of nucleocytoplasmic shuttling of glucocorticoid receptors, and suggest that the transcriptional activity resulting from nuclear accumulation of highly bindable GR (like binding site) but still needing agonist to trigger maximum transcriptionally.

INTRODUCTION

Serine phosphatase (PP5) is an enzyme that catalyzes the phosphorylation of Ser-1 to Ser-9. Serine phosphatase is a member of the phosphatidylinositol 3-kinase (PI3K)/serine kinase family.

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Serine phosphatase is a member of the phosphatidylinositol 3-kinase (PI3K) family. Most cells contain unliganded glucocorticoid receptors that act as repressors of various cellular functions. The GR, along with other proteins like hsp90/Hop, P23, p38, FKBP42, and FHBP51, is predominantly located in the cytoplasmic cavity and acts as an assembly or stabilizing agent for primary response genes. The molecular mechanisms directing steroid receptor movement through the cytoplasm and into the nucleus remain largely unknown, but recent research suggests that reversible phosphorylation plays a significant role. Qi et al.'s work demonstrated that insufficient nuclear retention of liganded receptors and inadequate cyclic reabsorption of their GRs result in inversely conserved phenotypes. PKA has been shown to influence the intracellular partitioning of GR by acting as a highly reactive protein, which means that PPase is also involved in controlling GR. In vitro, okadaic acid inhibits serine/threonine protein phosphatases (PP1) and 2A from human cells, but due to toxicity and solubility limitations, it is challenging to distinguish between p2A and pp1 with different enzymes. Despite recent research indicating that PP5 associates with the GR-hsp90 complex, there is little evidence of a direct impact from PI3K to cellular functions due to the lack of specific substrates for PP5. To determine if [3H]dexamethasone-mediated suppression of PP5 expression is involved in regulating GR-induced events, binding studies were conducted using data from binding experiments. These studies demonstrated that pp5 does not influence the formation of the high-affinity ligand binding complex or by hormone binding to the GR, suggesting that treatment with ISIS 15534 increases RESISTANCE of GRANTUS, and transient transfection studies using a GLOBAL reporter plasmid showed that both p10 times higher inflammatory. Using a GR-GFP fusion protein and fluorescent microscopy, we were able to observe the movement of GR membranes (GR) in cells treated with dexamethasone and then ISIS 15534 in our study. These studies suggest that this PP5 mediated suppression of [GR]function stems from its ability to suppress the nuclear accumulation of random genes expressed as gram-positive peptide 5.

CONCLUSION

Remarkable conclusions According to the findings, PP5 is involved in controlling nucleocytoplasmic shuttling of GRs, and suppressing its

expression leads to nuclear accumulation of these genitalia without the presence of any hormone. As a result, the previously reported increase in GENI activity after ISIS 15534 induced transcriptional suppression is thought to be primarily due to an increase (since it appears that) selective repression by agonists can still trigger nuclear exportation of both genicogenic and non-human motifs.