Cyclic-AMP-induced protein kinase A (PKA) is a kinase that functions in the regulation of many cellular behaviors, including differentiation, proliferation and migration. Growth factors and their related receptor tyrosine kinases are known to activate protein kinase A in mammalian cells, however the precise mechanisms that link PKA to growth factor signaling remain an area of intense investigation. Our laboratory has recently reported that the catalytic subunit of PKA (PKA-C) is directly phosphorylated on tyrosine 330 by growth factor receptor tyrosine kinases and this phosphorylation enhanced the ability of PKA-C to interact with a peptide substrate (Caldwell et al., 2011). It remains uncertain whether phosphorylation of Y330 influences the interaction between the C subunit and allosteric regulators. The goal of this research is to test the hypothesis that growth factor-induced phosphorylation of PKA-C alters the ability of allosteric inhibitors to modulate kinetic activity. I have tested this hypothesis specifically by 1:determining the effect of phosphorylation on the interaction of the PKA regulatory subunits and 2:protein kinase inhibitor (PKI), both known to allosterically inhibit enzymatic activity. To test this hypothesis, we performed a kinase assay using PKA-C subunits (PKA-C subunits that were previously phosphorylated by protein-derived growth factors and control subunits that were not phosphorylated) and kemptide substrates along with radioactive-ATP tagged with phosphorous-32. We also used increasing concentrations of inhibitor (either PKI or regulatory subunits), which theoretically should decrease the amount of phosphorylation occurring. Thus, when the samples were read on a liquid scintillation counter, we were able to measure the amount of phosphorylation that had occurred in the PKA-kinase assay in a quantitative manner.