The catalytic subunit of cyclic AMP-dependent protein kinase A (PKA-C) becomes phosphorylated on tyrosine Y330 in response to growth factors including platelet-derived growth factor (PDGF). Here we wish to investigate whether this phosphorylation event on PKA-C, results in a new protein-protein interaction important for PDGF-mediated signaling events. As SH2-containing proteins are known to bind to phosphorylated tyrosine residues, we hypothesized that phosphorylated PKA-C would interact with one or more SH2 domain containing proteins. Indeed, a screen conducted by the Deming laboratory identified several potential SH2 containing binding partners, the strongest of which was the regulatory subunit of phosphoinositide 3-kinase (PI3K), p85. PI3K becomes activated during PDGF signaling through an interaction between the p85 regulatory subunit and the PDGF receptor (PDGFR). PI3K then mediates multiple cellular events through the production of the lipid product, phosphatidylinositol (3,4,5)-triphosphate (PIP_3) and our previous work demonstrated a link between PKA and PIP_3 during PDGF signaling. Therefore, the goal of this research was to confirm the preliminary findings that phosphorylated PKA-C interacts with p85 and to determine whether the binding interaction between PKA-C and p85 alters the ability of p85 to interact with the PDGFR. Far western blot analysis using purified p85 confirmed an *in vitro* interaction with tyrosine-phosphorylated PKA-C. Current and ongoing studies will involve *in vitro* binding assays to assess whether the interaction of p85 with phosphorylated PKA-C prevents and/or disrupts its ability to bind to PDGFR. These experiments will aid in understanding the mechanism by which PKA intersects the PDGFR-PI3 K signaling network.