An effective treatment for Parkinson's disease has yet to be developed and a therapy that could prevent neural degeneration in the substantia nigra is greatly needed. An effort to use the PAC1R signaling systems for neuroprotective pharmacology is still in early stages of development, but upregulation of PACAP related survival signaling could counteract pathological neural apoptosis. Drug development based on biased agonism for promoting therapeutic GPCR signaling while minimizing alternative signaling has shown promise; and the difference between PAC1 hop1 and PAC1 null signaling represents one area of PAC1R research requiring further elucidation. Assays were conducted to investigate whether PAC1 receptor pathways and the receptor isoforms differ in their abilities to inhibit apoptosis in HEK cells stressed by the presence of hydrogen peroxide. Additionally, by elucidating the overall temporal differences in ERK activation between the PAC1 receptors I hoped to discover whether one receptor might activate ERK to a different extent at various time points after exposure to PACAP.