Evidence in past research has indicated that the amygdala is critical in the development and expression of conditioned fear (Davis 1992). An increase in amygdala activation is associated with anxiety, while lesions to the amygdala have been shown to eliminate innate fear (Adolphs et al., 2001). One behavior in which the amygdala plays a crucial and clear role in is fear learning. The central nucleus of the amygdala (CE) functions as the main output of the amygdala and makes distinct contributions to fear learning and the mediation of anxious behavior. The bed nucleus of the stria terminalis (BNST) has been implicated in processing responses to stressful stimuli as well as further mediation of anxious behavior. It is well known that the BNST and CE work together as the main output nuclei of the extended amygdala and are reciprocally connected, sharing similar cell types as well as expression of similar efferent targets (Dong et al., 2001). Corticotrophin releasing factor (CRF)-containing neurons in the CE project to the dorsal anterior portion of the BNST. These neurons influence neuronal activity and anxiety-like and drug-seeking behaviors. The reciprocal pathway has not yet been characterized. Neurons that are inhibited in fear learning express the delta isoform of protein kinase C (PKCδ). The present study aims to characterize the neurons in the BNST which project to the CE, and evaluate the circumstances under which this pathway is activated. In the first set of experiments, we will infuse the retrograde tracer FluoroGold (FG) into the CE of male and female rats. Using standard immunocyto-chemistry techniques, we will evaluate whether FG+ neurons within the BNST express the neuropeptide CRF and the delta isoform of protein kinase C (PKC-δ). Since BNST is one of the few sexually dimorphic structures in the rodent brain, differential expression of neuropeptides and genetic markers in BNST neurons which project to the CE, as well as recruitment of this circuitry by behavior in males and females will be investigated.