In vertebrates, the neural crest comprises a population of stem cells that directly gives rise to the peripheral nervous system (PNS), most of the cartilage and bones of the face and skull, the adrenal medulla and also melanocytes. Research in Dr. Bradley’s lab is focusing on NF-protocadherin (NFPC), a cell adhesion molecule required for neural tube and neural crest function, but its specific role still remains to be fully elucidated. Earlier research in Dr. Bradley’s lab has shown that knocking out expression of NFPC inhibits neural crest formation and causes the cells to undergo apoptosis. Because the precise role is still not completely clear, research has focused on making this clear. Preliminary results show that when blocking expression of NFPC by adding an anti-NFPC morpholino, snai2 (slug) is lost and cells undergo apoptosis. This study investigated the role of NFPC in maintaining the expression of snai2 (slug), a transcription factor required for the migration and differentiation of the neural crest. Our objective was to find where rescuing apoptosis rescued the neural crest phenotype. Our hypothesis was that Bcl-xL (a Bcl-2 family anti-apoptotic gene) would rescue slug expression, which would restore the domain of neural crest cells. Our results demonstrated that NFPC is required for slug expression, and that rescuing the cell from apoptosis rescued slug expression, the neural crest phenotype and even extended its expression. This experiment, along with others in the lab, has shown that apoptosis is correlated strongly with the development of the neural crest, as inhibiting it increased the neural crest domain. This is intriguing as apoptosis is largely overlooked in this time of development at the moment. Future experiments will investigate this further as more data is needed. Also, if time allows, these experiments will also include investigating the roles of other apoptotic and anti-apoptotic genes, as each will likely have a specific temporospatial importance.