Effects of a Brain-Derived Neurotrophic Factor Deletion from Oligodendrocytes During Development

Samuel Potenciano[1], Amanda Pan[1], Yangyang Huang[1], Cheryl Dreyfus[1]

[1]Rutgers Robert Wood Johnson Medical School

In the past, the Dreyfus lab found that mature oligodendrocytes synthesize and secrete brain-derived neurotrophic factor(BDNF). The oligodendrocyte-derived growth factor was shown to increase the survival and maturation of neuronal cells in vitro. The purpose of this study was to investigate mature oligodendrocyte-derived BDNF's effect on neuronal axons and myelination during development in vivo using murine models. We hypothesized that BDNF deletion from mature oligodendrocytes would negatively affect the development of axons and mature oligodendrocytes. BDNF deletion from mature proteolipid protein-positive oligodendrocytes was induced using Cre-LoxP technology at Postnatal day (P) 8-9, or P28-32, or P56-60, and results were assessed at P23, or P56, or P91, respectively. Effects were determined using Western blots to detect myelin and axonal proteins in the corpus callosum. Preliminary results indicate that the BDNF deletion examined at P-23 and P-56 resulted in a decrease in myelin-associated glycoprotein (MAG). However, at P91, no effect on MAG was observed. In contrast, vesicular glutamate transporter 1(VGLUT1), a neuronal axonal protein, was reduced at all time points examined. These data suggest that the mature oligodendrocyte-derived BDNF deletion negatively influences early oligodendrocyte development. This influence is lost between P56-P91. On the other hand, effects on proximate axons are evident throughout development and the latest ages investigated. Supported as a Grover Scholar by Dr. and Mrs. Gary Grover.