Volume 12

Washington University
Undergraduate Research Digest

Spring 2017

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Investigating the Role of *actr10* in Schwann Cells of the Peripheral Nervous System

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Myelin is the lipid-rich sheath surrounding axons that promotes the rapid propagation of action potentials. Myelin is made by oligodendrocytes (OLs) in the central nervous system (CNS) and Schwann cells (SCs) in the peripheral nervous system (PNS). Proper myelination of axons is essential for growth and development, and damage to myelin and/or the myelinating glial cells that make myelin can result in neurodegenerative diseases such as multiple sclerosis or Charcot-Marie-Tooth disease. A forward genetic screen performed by the Monk Lab found a mutant, *stl83*, that has reduced myelin in both the CNS and PNS, as assayed by in situ hybridization for *myelin basic protein (mbp)* expression, which marks the mature myelin sheath. The *stl83* phenotype results from a single nucleotide polymorphism (SNP) in the gene *actr10*, which encodes the protein Arp11. Arp11 forms a component of the dynactin motor protein complex, which plays a critical role in cellular transport by directly binding to the motor protein dynein and facilitating retrograde transport of intracellular cargo along microtubules. We hypothesize that the mutation in actr10 prevents proper formation of the dynactin complex, and thus causes a disruption in transport and trafficking. Methods used include the aforementioned *in situ* hybridization, as well as transgene analysis to observe expression of early SC developmental markers such as *foxd3* and *sox10*. Preliminary data involving the drug forskolin, which rescues *mbp* expression, is also included.