**Investigating the role of mitochondria in the protection of neonatal axons from injury.**

First Supervisor: Lyndsay Murray, Centre For integrative Physiology, University of Edinburgh

Second Supervisor: Kosala Dissanayake, Centre for Cognitive and Neural Systems, University of Edinburgh

During both neurodegenerative disease, or after injury, the axonal and synaptic parts of a neuron degenerate very rapidly. In order to study this, we use an ex-vivo model of peripheral nerve injury. In this model, the peripheral nerve and associated muscles are maintained in physiological solutions. In adult mice, this would result in nearly complete degeneration of the axon and synaptic terminal with 24 hours. However, we have recently demonstrated that this process of axon degeneration is much slower in young mice, aged 10 days or less compared to those aged 21 days or more. We performed a large proteomic screen to try to identify proteins and cellular mechanisms which might be responsible for this. This reveal a significant increase in mitochondrial proteins in both muscle and nerve which corresponded to the acceleration in the rate of axon degeneration as the age of the mouse increased. In this project, we aim to understand how the number and activity of mitochondria changes between 10 and 21 days of age. We aim to understand if and how these mitochondrial changes contribute to the acceleration in the rate of axon degeneration over this time period and whether altering the activity of mitochondria affect the rate of axon degeneration in neonatal nerves. This work will give key insight into the mechanisms which regulate axon degeneration, and can help develop ideas about how to slow axon degeneration in disease or after injury.

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