Neuroinflammation is a well-characterized hallmark for Parkinson's disease (PD) and recent evidence from our lab suggests its involvement in L-Dopa induced dyskinesia (LID), a side effect of L-Dopa treatment in PD. The endocannabinoid receptor 2 (CB2) increases in expression in PD and is known to play an important role in modulating inflammation, making this receptor a promising therapeutic target. The current project will aim to examine the neuroprotective and anti-dyskinetic effects of CB2 agonists and to identify the causative role of CB2-expressing cells underlying these effects. For this project, we have created novel genetically modified mice in which CB2 can be removed from specific cell types. These mice are also engineered so that cells express green or red fluorescent proteins, dependent on whether CB2 is present or ablated, respectively. These mice will, with or without CB2 agonist treatment, be analysed for levels of degeneration, inflammation, and dyskinesia severity in our models of PD and LID.

Models and Techniques that you will use

- Genetically modified mice with selective deletion within specific cell types
- Subcutaneous or surgical injection of neurotoxins to induce degeneration
- With or without CB2 agonist treatment

Tissue collection and processing

- Perfusions
- Sectioning
- IHC

Analysis

- Stereology
- Flow cytometry
- Protein analysis

Our Team – 4 Postdocs – 2 Research Assistants – 5 PhD students – 1 Honours student – 2 Internship students