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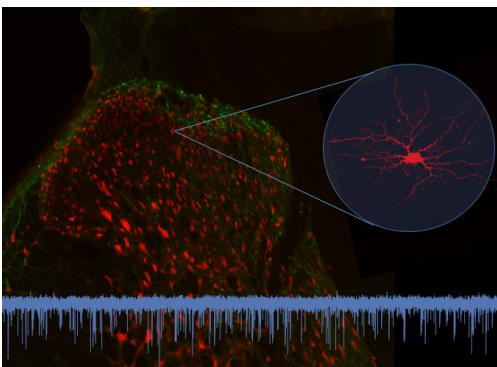
WEB www.imlachlab.com

Chronic pain is a major global health burden, affecting nearly 20% of the Australian population. This condition results in hypersensitivity to sensory input so non-painful stimuli can become painful. Analgesics that are currently in use provide relief in a small proportion of chronic pain patients and there is a great need for more effective therapeutics.

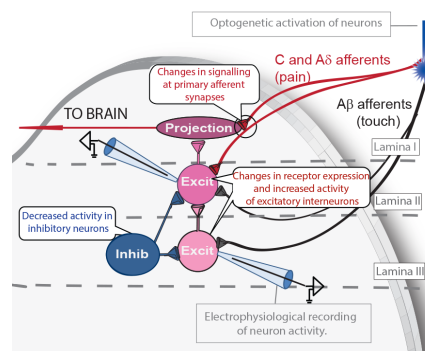
Our lab investigates changes in neuron signalling that happen in pain circuits during the development of chronic pain. Some of these changes can be targeted therapeutically, so the aim of our work is to identify pathological changes and find ways to modify them for the treatment of pain. To understand pain circuitry and to characterize potential analgesics, we use patch-clamp electrophysiology, optogenetics and calcium imaging in brain and spinal cord tissue from animal models. We also use immunohistochemistry and confocal imaging, behavioural assays and genetic profiling.

Research Projects

1. Decoding dysfunctional spinal cord circuitry in chronic pain.
2. Identifying Novel Molecular Targets for Treating Chronic Pain.
3. Characterization of interneuron subtypes in pain pathways.
4. Allosteric modulation of adenosine A1 receptors for the treatment of chronic pain.



Spinal cord dorsal horn with inset showing an interneuron that is part of the nociceptive circuit. Electrophysiological trace in blue shows spontaneous firing of the nociceptive neuron.



Following the development of chronic pain we can see changes in synaptic signalling throughout the spinal cord pain pathways. Some of these changes are potential therapeutic targets. We use patch-clamp electrophysiology and electrical and optogenetic activation to investigate signalling properties.

Selected significant publications:

1. **Imlach WL**, Bhola RF, Mohammadi SA, Christie MJ. 2016. Glycinergic dysfunction in a subpopulation of dorsal horn interneurons in a rat model of neuropathic pain. *Sci Rep.* 6, 37104.
2. **Imlach WL**, Bhola RF, May LT, Christopoulos A, Christie MJ. 2015. A positive allosteric modulator of the adenosine A1 receptor selectively inhibits primary afferent synaptic transmission in a neuropathic pain model. *Mol Pharmacol.* 88, 460-8.
3. Choi BJ, **Imlach WL**, Jiao W, Wolfram V, Wu Y, Grbic M, Cela C, Baines RA, Nitabach MN, McCabe BD. Miniature neurotransmission regulates Drosophila synaptic structural maturation. 2014. *Neuron.* 82, 618-34.
4. **Imlach WL**, Beck ES, Choi BJ, Lotti F, Pellizzoni L, McCabe BD. 2012. SMN is required for sensory-motor circuit function in Drosophila. *Cell.* 151, 427-39.
5. Lotti F, **Imlach WL**, Saieva L, Beck ES, Hao le T, Li DK, Jiao W, Mentis GZ, Beattie CE, McCabe BD, Pellizzoni L. 2012. An SMN-dependent U12 splicing event essential for motor circuit function. *Cell.* 151, 440-54.