manitoba neuroscience network

Circadian rhythms in pain: where neuroscience meets immunology

SEMINAR & VISITING SPEAKER SERIES

DATE Monday, May 27th, 2024

TIME 11:00 AM to 12:00 PM

LOCATION BSMB THEATRE B

SPEAKER

Nader Ghasemlou, PhD

Associate Professor, Department of Anesthesiology, Department of Biomedical & Moledular Sciences, Centre for Neuroscience Studies, Queens University

BIO

Nader completed his PhD at McGill University under the supervision of Dr. Samuel David where he studied the contribution of myeloid cells to secondary damage after spinal cord injury. He then completed a postdoctoral fellowship as a Banting Fellow at Harvard Medical School with Dr. Clifford Woolf, dissecting the contribution of individual immune cells to inflammatory pain. In 2015, Nader established his own research team at Queen's University where he set out to examine the molecular and cellular mechanisms of pain across various injury and disease models, including multiple sclerosis, spinal cord injury, nerve injury and tissue wounds. His research has taken a turn towards studying how circadian rhythms of the immune and nervous systems affect pain, using both preclinical and clinical data. The Pain Chronobiology & Neuroimmunology Lab has been funded by CIHR, the MS Society of Canada, NSERC, and other research organizations. Nader and his team recently established the CircaHealth Research Network to study how chronobiology affects biomarkers of chronic pain.

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ABSTRACT

Circadian (24-hour) rhythms help maintain homeostasis by synchronizing physiological and behavioral functions to daily events (e.g., sleep, feeding) via a feedback loop regulating expression of circadian-controlled genes with the core clock genes Bmal1 and Rev-erbα/β. Feeding and sleep are two well-known functions controlled by these clocks; we posited that pain may have a similar effect given existing clinical observations. To this end, we studied how circadian rhythms affect pain in mice and humans. We began by assessing how circadian rhythms affect pain in mice, and discovered a neuroimmune response controlling the recruitment of immune cells to the skin, where nerve endings reside, and dorsal root ganglia where sensory neuron cell bodies are found. We have identified a link between opioids and their receptors along this neuroimmune axis that mediates basal sensitivity. Moving to the study of chronic pain, we focused our work on a clinical population with chronic low back pain (cLBP), which can arise from both inflammatory/nociplastic or neuropathic etiologies. Mechanisms underlying cLBP are poorly understood and current therapies offer only mild to moderate benefits. We therefore used a bedside-to-bench approach considering biological, psychological, and social facets of chronic pain to develop new therapeutic strategies. By creating a circadian profile of people with chronic pain low back pain, we found that only those with normal circadian gene expression and rhythmic pain were not dependent on opioids and had improved biopsychosocial outcomes, compared to those lacking rhythmic gene expression and pain patterns. Our work identifies several important factors to consider when carrying out preclinical behavioral studies, and holds the promise of moving to the clinic as a treatment option for those living with chronic pain.

OBJECTIVES

1. Define how circadian rhythms, neuroimmunity and pain interact in the basal state.

2. Characterize how peripheral immune cells affect thermal sensitivity via opioids and opioid-receptors.

3. Assess how time-of-day affects pain and gene expression in a clinical population.

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