January 2021

Role of Oxidative Stress and Neuroinflammation Response in Pain Brain Regions after Traumatic Brain Injury

Alyssa Goodwin  
*Wayne State University School of Medicine, fv3566@wayne.edu*

Julia Malewicz  
*Wayne State University, gg5844@wayne.edu*

Scott Lloyd  
*Wayne State University, sclloyd@med.wayne.edu*

Min Wu  
*Wayne State University, gf8552@wayne.edu*

Kelly Bosse  
*Wayne State University School of Medicine, kbosse@med.wayne.edu*

See next page for additional authors

Follow this and additional works at: https://digitalcommons.wayne.edu/som_srs

Part of the Psychiatry and Psychology Commons

Recommended Citation  
Goodwin, Alyssa; Malewicz, Julia; Lloyd, Scott; Wu, Min; Bosse, Kelly; and Conti, Alana, "Role of Oxidative Stress and Neuroinflammation Response in Pain Brain Regions after Traumatic Brain Injury" (2021). *Medical Student Research Symposium. 77.*  
https://digitalcommons.wayne.edu/som_srs/77

This Research Abstract is brought to you for free and open access by the School of Medicine at DigitalCommons@WayneState. It has been accepted for inclusion in Medical Student Research Symposium by an authorized administrator of DigitalCommons@WayneState.
Authors
Alyssa Goodwin, Julia Malewicz, Scott Lloyd, Min Wu, Kelly Bosse, and Alana Conti

This research abstract is available at DigitalCommons@WayneState: https://digitalcommons.wayne.edu/som_srs/77
Abstract

Traumatic brain injury (TBI) affects approximately 3 million people annually, with 70-80% presenting with pain symptoms. Research has shown that increased reactive oxygen species (ROS) and neuroinflammation play a role in both pain and TBI, but the roles and interaction of oxidative stress and inflammation in TBI-related pain remain unclear. The purpose of the current study is to establish molecular data supporting proposed alterations in ROS and neuroinflammation in a mouse model of TBI in pain-related brain regions and to quantify how levels of these mediators change over time. Once the timecourse is determined, it will allow for optimal use of various interventions such as antioxidant and anti-inflammatory treatment. Male (n=54) and female (n=27) mice ages 7-14 weeks were exposed to moderate level TBI or sham control surgery. Microdissections from pain-related brain regions, such as anterior cingulate cortex, amygdala, and periaqueductal gray were taken at 24 hours (n= 26 males, 9 females), 7 days (n= 13 males, 9 females), and 14 days (n= 15 males, 9 females) post-TBI and used to quantify ROS and inflammatory cytokine levels using a cell-based fluorescence assay and an enzyme-linked absorbance assay, respectively. Data demonstrated complex patterns of ROS and cytokine activation that varied with region and time post-injury. These data will provide information leading to optimal intervention strategies to mitigate the increased oxidative stress and neuroinflammation post-TBI.