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## Presentation Abstract

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Title: Signs of central sensitization: inflammation induced by high demand task occurs in peripheral nerve and spinal cord tissue

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Authors: \***M. B. ELLIOTT**<sup>1</sup>, A. E. BARR<sup>2</sup>, M. AMIN<sup>1</sup>, M. F. BARBE<sup>1</sup>;  
<sup>1</sup>Physical Therapy, Temple Univ., Philadelphia, PA; <sup>2</sup>Physical Therapy, Thomas Jefferson Univ., Philadelphia, PA

Abstract: Prolonged or repeated episodes of inflammation in peripheral tissues that alter nociceptor function is a proposed mechanism of central sensitization implicated in chronic pain. Previous studies from our laboratory have shown that performance of high repetition tasks results in musculoskeletal tissue inflammation, median nerve neuritis and fibrosis, decreased nerve conduction velocity, spinal cord neuroplasticity, and substantial motor dysfunction. The present study used our rat model of upper extremity repetitive motion injury to examine whether mediators of peripheral neuritis contribute to changes in the spinal cord indicative of central sensitization, and whether these changes explain maladaptive behavior. Rats were randomly assigned to 2 groups: high repetition, high force forearm handle pulling task (HRHF) or controls with 5-8 rats/group. Controls learned, but did not perform the regimen. HRHF rats were cued to reach and grasp every 15 s at 70% of maximum voluntary pulling force (2hr/d, 3d/wk, for 12 wks). Several behavioral functions, including indices of motor function (reach rate and success, grip strength), sensory function (Von Frey filament testing) and task participation, were monitored throughout the 12 weeks. Immunohistochemistry was used to analyze macrophage and cytokine (TNF and IL-1alpha) expression in the median nerve, and macrophage/microglia, cytokine (TNF and IL-1alpha), and Substance P (SP) expression in the spinal cord. In addition to analyses of

group differences, statistical correlations were performed to assess the relationship between peripheral and central inflammatory markers. Correlations were also performed to examine the influence of peripheral and central markers of inflammation on maladaptive behaviors. We found that performance of the HRHF task induced significant peripheral neuritis that was associated with spinal cord inflammatory markers involved in central sensitization. These changes were associated with behavioral changes in the HRHF rats, such as reduced grip strength, reach success, and task participation. The hypothesis that peripheral inflammation is one mechanism of central sensitization is supported by these results. Furthermore, results from this study suggest that some of the behavioral dysfunction may be due to inflammatory pain. Future studies will target therapeutic strategies to combat inflammation and pain in cases of repetitive motion injury and other insults to the peripheral nervous system.

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