Experimental Pain and Psychological Differences Between Individuals with Early- and End-Stage Carpometacarpal Osteoarthritis

Tamara Ordonez Diaz[1], Terrie Vasilopoulos[2,3], Thomas W. Wright[3], Yenisel Cruz-Almeida[4], Jennifer A. Nichols[1,3]

[1]J. Crayton Pruitt Family Department of Biomedical Engineering, [2]Department of Anesthesiology,

[3]Department of Orthopaedics & Sports Medicine, [4]Department of Community Dentistry and Behavioral Science

Purpose: Carpometacarpal osteoarthritis (CMC OA) is a disabling disease that affects 85% of postmenopausal women. Treating CMC OA is challenging because the interplay of osteoarthritic pain, disease severity, and physical function is poorly understood. Here, as a critical step toward understanding this complex interplay, we focus on understanding CMC OA pain. Understanding alterations in pain mechanisms can highlight to what extent individuals with CMC OA experience localized versus widespread pain, thereby informing personalized treatments. We specifically examined the relationship between OA severity and measures of somatosensory function. We hypothesized that participants with end-stage CMC OA would have higher self-perceived pain, higher disability, and display a loss of sensory function compared to those with early-stage CMC OA.

Methods: Fifteen women with end-stage CMC OA (Eaton-Littler of III or IV) [age 68.1 ± 10.4 years] and sixteen women diagnosed with early-stage CMC OA (Eaton-Littler of I or II) [age: 59.9 ± 11.2 years] participated in this IRB-approved study. Disease severity was classified from radiographs using the Eaton-Littler scale. Measures of somatosensory function using quantitative sensory testing (QST) were assessed at 7 sites (thenar, hypothenar, and brachioradialis on both arms; quadriceps on the affected or dominant side if they had bilateral CMC OA) using standardized procedures. Cognitive and psychological function measures were also collected. T-tests and chi-square statistics were performed for the demographic and clinical variables. QST variables were log-transformed when appropriate and converted to z-scores. To compare cohorts, participants with early-stage CMC OA were used as the control when converting variables to z-scores. ANCOVAs were then used to compare cohorts with age as the covariate in the fully adjusted model.

Results: Participants with end-stage CMC OA had significantly higher self-reported pain ($p=0.02$) and stiffness ($p=0.03$) than those with early-stage CMC OA. However, pain interference and other psychological variables (e.g., stress, anxiety) were similar between cohorts. Somatosensory differences between the two cohorts were only observed for the heat pain threshold when adjusting for age as a covariate (Fig. 1). Specifically, significant differences were identified at the affected hypothenar ($p=0.05$), contralateral brachioradialis ($p=0.02$), and quadriceps ($p=0.02$) testing sites. In the unadjusted model, differences in the cold pain threshold at the affected thenar testing site ($p=0.05$) were detected. Similar differences were observed during the vibratory detection threshold at the contralateral thenar ($p=0.04$) and hypothenar ($p=0.03$) testing sites. Interestingly, women with end-stage CMC OA displayed higher pain sensitivity only during the pressure pain thresholds than women with early-stage CMC OA, although this failed to reach statistical significance.

Conclusion: Our findings suggest that women with end-stage CMC OA display a loss of sensory function when compared to women with early-stage CMC OA. These somatosensory alterations in the affected site likely reflect changes in the peripheral nervous system. Although a prior study showed that patients with CMC OA had lower pressure pain thresholds than healthy controls, no study to our knowledge compared stages of disease severity or used multiple stimuli to comprehensively study pain in individuals with CMC OA. Our results provide no evidence for central nervous system mechanism differences. However, in the future, evaluating a large sample size, comparing affected versus contralateral body sites within participants, or inclusion of healthy age-matched controls may highlight central differences. Despite the limitations, the inclusion of testing the symptomatic area as well as distant pain-free sites provide confidence in our results. The differences in the underlying pathological mechanisms of pain highlighted herein have the potential to inform pain management in individuals with CMC OA.

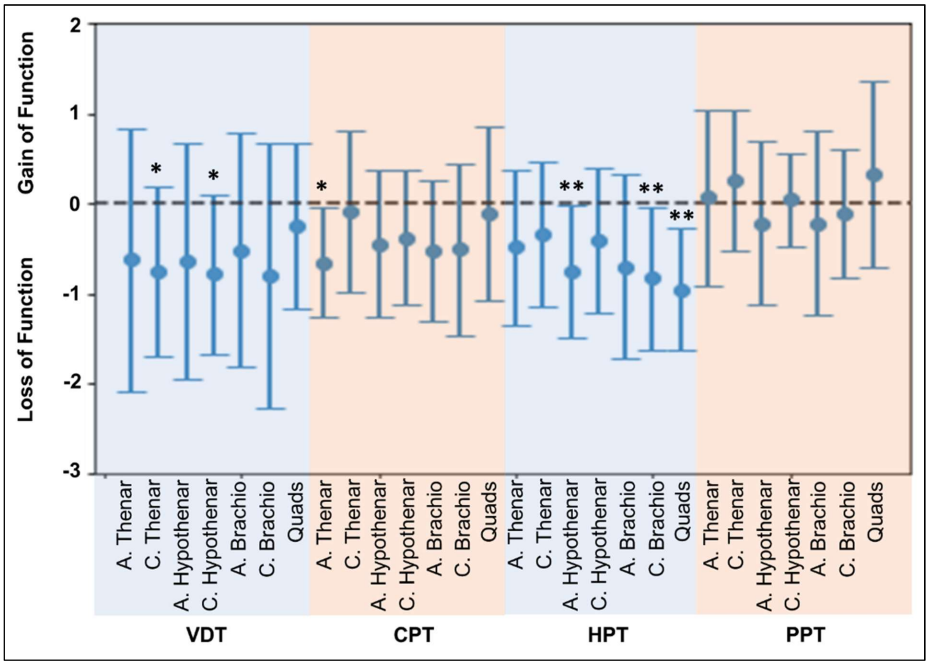

Figure 1. Standardized quantitative sensory testing (QST) scores for participants with end-stage CMC OA. Values are presented as Z-scores using participants with early-stage CMC OA as the control (Z-score = 0). Error bars represent standard deviation. *Abbreviations:* VDT = vibratory detection threshold, CPT = cold pain threshold, HPT = heat pain threshold, and PPT = pressure pain threshold. *$p<0.05$ for the unadjusted model and **$p<0.05$ for the fully adjusted ANCOVA model.