INTRODUCTION: Carpometacarpal osteoarthritis (CMC OA) is a disabling disease that affects 85% of postmenopausal women [1]. CMC OA can lead to severe pain, decreased strength, and decreased range of motion [2]. However, there is a disconnect between self-reported pain and functional disability, highlighting a need to understand the interplay between movement and pain [3-4]. The limited number of studies that report movement and pain either measure pain at rest or fail to differentiate pain during rest versus movement [5]. Yet, movement-evoked pain, the pain experienced due to physical activity, is usually more severe and has a greater impact on clinical outcomes [5]. Integrating movement-evoked pain and movement measurements will elucidate when physical activity alleviates versus evokes pain. This study aimed to examine the relationship between movement and pain by simultaneously measuring pinch force, muscle activity, and movement-evoked pain in individuals with CMC OA and healthy older adults. We hypothesized (1) participants with CMC OA would generate lower forces, (2) activate extrinsic muscles more than intrinsic muscles, and (3) have higher movement-evoked pain than healthy older adults.

METHODS: Five participants diagnosed with CMC OA [female, age: 68.6 ± 11.3 years] and two healthy older adults [female, age: 65.0 ± 5.0 years] participated in this IRB-approved study. Pain, thumb-tip force, and muscle activity were simultaneously recorded from each participant during 4 maximum effort tasks: key pinch, tip pinch, jar grasp, and jar twist. Participants performed each task 3 times and held their maximum force for 3 seconds. Movement-evoked pain ratings were obtained before, during, and after each task using a 101-point visual analog scale (VAS). Forces were collected using a 3D force sensor (ATI), and a set-up similar to prior studies [6]. To examine the effect of fine-wire electromyography (fEMG) on force production, two of the four tasks (maximal key and tip pinch) were measured before and after placing the electrodes (Delays Inc). Muscle activity was recorded from 4 extrinsic muscles [flexor pollicis longus (FPL), abductor pollicis longus (APL), extensor pollicis longus (EPL), extensor pollicis brevis (EPB)] and 4 intrinsic muscles [first dorsal interossei (FDI), adductor pollicis brevis (ADP), opponens pollicis (OPP), and flexor pollicis brevis (FPB)]. Force and fEMG data were time synchronized and sampled at 1,000 and 3,000 Hz, respectively. For each participant, fEMG data were rectified and amplitude normalized to maximum activation across all tasks for that participant. Spider plots were used to compare fEMG across participants and tasks. Independent t-tests for the force and movement-evoked pain measurements across study groups and paired t-tests for before, during, and after measurements within study groups were completed using IBM-SPSS.

RESULTS: Movement-evoked pain measurements were significantly greater during task performance in comparison to before or after task in the CMC OA study group (Fig. 1). Although there was a trend toward higher self-reported pain in the CMC OA versus healthy older adult study group, there were no significant differences in pain measurements between these groups. Participants with CMC OA also generated similar forces as healthy older adults before fEMG with a trend toward greater forces after fEMG (Fig. 2). Healthy older adults demonstrated a greater force reduction during tip pinch (-18.1 N) after fEMG than participants with CMC OA (-6.4 N). Lastly, there were no clear trends in muscle activation patterns across or within study groups (Fig. 3). Interestingly, a participant with CMC OA deactivated some of their intrinsic muscles during maximum jar grasp.

DISCUSSION: Results from this study highlight the need to perform patient-specific analysis to understand the extent to which different levels of force exertion and muscle activity uniquely impact the experience of pain. Contrary to our first hypothesis, participants with CMC OA had higher forces across all tasks, but higher movement-evoked pain ratings than the healthy older adults. These data suggest that despite participants with CMC OA having increased sensitivity to pain, they have also learned to push through the pain and/or compensate through the overuse of muscles to accomplish everyday tasks. Moreover, our movement-evoked pain measurements highlight the differences in pain experience between pain during a task and at rest (before and after task). The heterogeneity of muscle activation patterns within the same tasks demonstrates the presence of muscle redundancy in the upper limb. Yet, participants with CMC OA have found an optimal muscle activation pattern to avoid pain or protect the CMC joint is still unknown. Limitations of this study include the small number of participants and different numbers of participants in each study group. Future work will focus on increasing the sample size and evaluating whether the observed trends in the force measurement hold or shift to match the literature. A larger number of participants will also show whether the heterogeneity, particularly in muscle activation patterns, is still present. To better understand pain, multi-modal quantitative sensory testing will also be performed to quantify painful and non-painful sensory function important for pain and muscle force production (e.g., proprioception, tactile, and pain perception). Merging these results will highlight differences across participants with CMC OA and healthy older adults and enhance our understanding of how multiple mechanisms compensate to alleviate or aggravate CMC OA symptoms.

SIGNIFICANCE/CLINICAL RELEVANCE: This study demonstrates the feasibility of simultaneously collecting kinematics, muscle-activity, and pain data in the same cohort of people. Understanding the interplay of biomechanics and pain associated with CMC OA will allow us to inform and develop targeted treatments that restore full joint mobility and eliminate pain.


ACKNOWLEDGEMENTS: Funding from the University of Florida Graduate Student Preeminence Award and National Institutes of Health (KL2 TR001429).