

APPLYING A SIMULATION PIPELINE TO IDENTIFY KINEMATICS AND KINETIC DIFFERENCES BETWEEN INDIVIDUALS WITH EARLY- AND END-STAGE CARPOMETACARPAL OSTEOARTHRITIS

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INTRODUCTION

Carpometacarpal osteoarthritis (CMC OA) is a multifaceted disease that affects the base of the thumb. Individuals with CMC OA experience pain, stiffness, and reduced range of motion [1-2]. Due to a disconnect between disease severity and symptoms, the early stages of CMC OA often go undiagnosed, and as the disease progresses, the likelihood that individuals will require surgery increases [3].

Biomechanical assessments can provide valuable information about joint movement and forces during functional activities. For individuals with CMC OA, such assessments can help identify compensatory movements and abnormal loading patterns that may contribute to disease progression [4-5]. In this context, the objective of this study is to establish a simulation pipeline that enables identification of biomechanical markers of CMC OA. The pipeline is being designed to provide clinically useful information that can aid in early diagnosis, intervention, and improved patient outcomes. Here, we present the preliminary pipeline and demonstrate how it can be used to evaluate kinematic and kinetic difference between individuals with early- and end-stage CMC OA.

METHODS

Fifteen women with end-stage CMC OA (Eaton-Littler Stage III or IV) [age 68.1 ± 10.4 years] and sixteen women diagnosed with early-stage CMC OA (Eaton-Littler Stage 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.

Motion capture data were collected at 100 Hz using a 12-camera motion capture system. A custom marker set was used that had 31 markers on the hand, including 4 markers on the CMC segment and 3 markers each on the metacarpophalangeal (MP) and interphalangeal (IP) thumb segments. Force data were collected at 3,000 Hz using a multi-axis sensor. Each participant completed three trials of four tasks: three CMC

joint range of motion tasks (flexion/extension, ab/adduction, opposition to base of pinky) and one isometric force task (maximal key pinch).

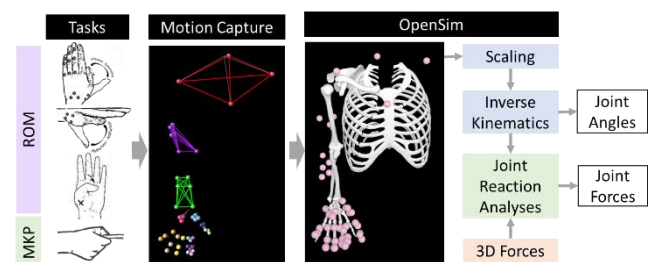


Figure 1. Experimental and simulation pipeline.
ROM = range of motion. MKP = maximum key pinch.

To evaluate kinematic differences between cohorts, motion capture data were processed in OpenSim (v. 4.4). Briefly, an upper limb model [6] was anthropometrically scaled to match each participant. Inverse kinematics was performed to calculate joint angles (Fig 1). Range of motion was calculated as the difference between the maximum and minimum angle of the CMC, MP, and IP joint during each task. Participant data were averaged across trials and cohorts. T-tests were performed to compare range of motion. Significance was defined as $p < 0.05$.

To evaluate kinetic difference between cohorts, force propagation during the key pinch task was analyzed using inverse dynamics and the joint reaction analyzer in OpenSim. A point constraint at the distal end of the thumb was defined to apply the measured external force to the thumb-tip. Prior to analysis, measured force data was centered, filtered, and transformed so that the force, which was recorded in the force sensor's coordinate system, could be applied to the scaled models using the OpenSim coordinate system. Forces reported herein define F_x , F_y , and F_z as distal, dorsal, and ulnar, respectively. As a proof-of-concept, force propagation across the joints was analyzed for one participant from each cohort. Joint reaction forces at the CMC, MP, and IP joints are reported as the average maximum force at each joint for each individual. Percent

differences in joint reaction force for each joint between individuals were also calculated.

RESULTS AND DISCUSSION

The implemented simulation pipeline successfully identified differences in range of motion (Fig. 2) and joint reaction forces (Fig. 3) between the early- and end-stage CMC OA cohorts.

The kinematic analysis indicates that there is larger variability in the range of motion recorded from individuals with end-stage versus early-stage CMC OA (Fig. 2). This suggests that as CMC OA progresses from early- to end-stage disease, there are a variety of effective movement compensations that can be employed to complete a given task. Interestingly, these movement compensations seem to favor increases in range of motion at the IP and MP joints, but not the CMC joint. Compared to the early-stage cohort, there was a significant increase in IP flexion used by individuals with end-stage CMC OA during flexion/extension ($p = 0.03$). With the exception of a few non-physiological outliers, the end-stage cohort also trended toward higher IP and MP flexion during opposition.

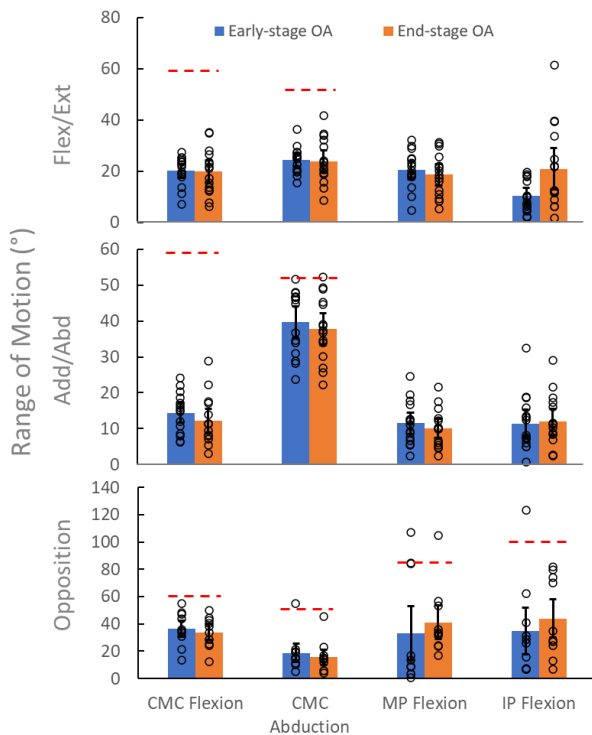


Figure 2. Range of motion of the thumb joints in early- and end-stage CMC OA participants across tasks: flexion/extension ($n = 30$), abduction/adduction ($n = 30$), and base of pinky opposition ($n = 20$). Red dashed line indicates limit of anatomically possible range of motion; red line omitted when limit exceeds magnitude of y-axis.

The proof-of-concept kinetic analysis revealed that differences in the magnitude of the external and internal forces across the thumb can be identified. Specifically, the individual with early-stage CMC OA generated a larger maximum pinch force than the individual with end-stage CMC OA, which resulted in consistent differences in the magnitude

of internal force at each thumb joint (c.f., Fig. 3, magnitude of forces for early-stage is greater than end-stage). Interestingly, the direction of the internal forces across the IP, MCP, and CMC joints differed between the individual with early- versus end-stage CMC OA (c.f., Fig. 3, distribution of color is unique for each bar). These directional difference may relate to known clinical differences in joint stability between early- and end-stage CMC OA. However, analysis of a larger sample is needed before conclusions can be made.

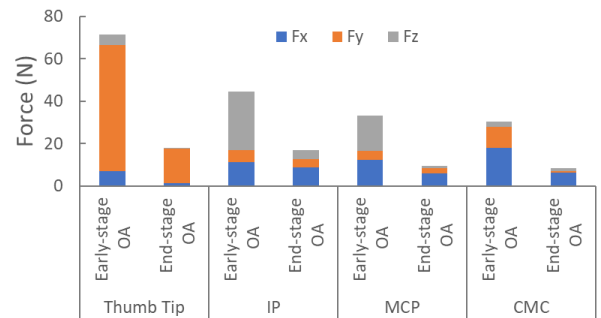


Figure 3. Joint reaction force propagation across the thumb joints during maximal key pinch for representative participants with early- and end-stage CMC OA ($n = 2$).

CONCLUSIONS

Our results highlighted the heterogeneity of movement and joint loading across individuals with CMC OA. This heterogeneity suggests the need for personalized diagnostic and treatment approaches for this disease.

Even with this heterogeneity, our findings also suggest existence of movement compensations and joint instability, particularly in individuals with end-stage CMC OA. Understanding the relationship between biomechanics and disease severity is critical to inform development of effective interventions for CMC OA.

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