INTRODUCTION: Studies have found that active and passive ranges of motion (ROM) are reduced in thumb carpometacarpal (CMC) joint osteoarthritis (OA), yet the underlying causes for this reduction remain unclear [1]. Osteophyte growth and ligament property changes associated with progressive OA have been hypothesized to affect CMC ROM, yet no study has confirmed a causal relationship. An in vitro biomechanical assessment would allow for prescribed, directional load application, shedding light on inherent stabilizing structures. The aim of this work was to determine the in vitro ROM and stiffness in 26 distinct directions of thumb CMC motion for specimens without OA using a musculoskeletal simulator.

METHODS: Ten fresh-frozen human forearms (5M, 5F, 27-62 yrs.) with less than 150 mm² of rimming trapezial osteophytes were sectioned proximally at the midshaft of the radius/ulna. All bones distal to the carpus were removed, except for the first metacarpal (MC1) and the proximal head of the second metacarpal (MC2). An optical motion sensor consisting of 6 infrared markers (NDI) was rigidly mounted via two k-wires to the radial surface of the trapezium (TPM) to provide a reference frame for data reporting. CT scans of all specimens were acquired and post-processed to generate TPM- and MC1-based anatomical coordinate systems, as described previously [2]. Briefly, MC1 and TPM bone coordinate systems (CS) were computed based on directions of principal curvature of the articular surfaces [3] and, for the MC1, its proximal-distal inertial axis. CS axes were directed volarly (+x), proximally (+y), and radially (+z). Each specimen was mounted to a six-axis industrial robot (KUKA KR 6 R700) with the radius and ulna affixed to the robot base and the MC1 to the robot end effector. Specimen-specific CT-generated coordinate systems were registered in the robot space and joint coordinate systems were constructed using simVITRO (Cleveland Clinic) [2]. The flexion-extension axis (Z axis) was fixed in the TPM, the pronation-supination axis (Y axis) was fixed in the MC1, and a floating abduction-adduction rotation axis (X axis) was defined perpendicular to the two body-fixed axes (Fig. 1). All ROM tests began from CMC joint neutral, defined at 1) 0° MC1 rotation in flexion, extension, abduction, adduction; 2) 2 N proximal compression for joint contact, and 0 N volar, dorsal, radial, ulnar joint forces; 3) 0 Nm torque in pronation and supination. Tests were performed in 26 distinct directions of MC1 rotation: pronation, supination, and 24 directions comprising a ROM envelope (the orthogonal anatomically-defined directions of flexion, extension, abduction, and adduction, as well as 20 coupled directions at 15-degree increments from the orthogonal directions). In each of the 26 rotational directions, maximum ROM was determined by rotating the MC1 at 1 ° until a resultant RMS torque of 1 Nm was achieved. Joint forces were fixed at 0 N volarly, dorsally, radially, and ulnarily, and in 2N of joint compression, with translations allowed as necessary to maintain the fixed force state. TPM motion was recorded throughout the duration of each test via the rigidly-attached TPM sensor. Torque-rotation curves were analyzed from 6 DOF kinematics and kinetics of the MC1 with respect to the TPM to determine rotational ROM at 1 Nm. The portion of the torque/rotation curve with a torque greater than 0.5 Nm was fit with a linear regression model. The slope of this model was recorded as the final stiffness K (Nm°). The principal directions of motion were computed as eigenvectors of the envelope of ROM and stiffness.

RESULTS: The major principal axis of the mean rotational envelope for the CMC joint was oriented oblique to the primary axes of flexion-extension and abduction-adduction, angled at 29.2° from pure adduction toward extension (Fig. 2A). The ROM in this principal axis direction was 49.3 ± 13.6°, which was significantly greater than the ROM recorded in the primary directions of extension (28.4±5.2°, p<0.05) and abduction (29.0±10.6°, p<0.01). The major principal axis of the mean rotational stiffness envelope was oriented approximately orthogonal to the mean ROM envelope at 54.1° from pure flexion in the direction of adduction (Fig. 2B). Stiffness was greatest at 45° from flexion toward abduction (0.14±0.03Nm°) and least at 30° from extension toward adduction (0.07±0.03Nm°). Pronation ROM was 49.0 ± 18.8°, supination ROM was 34.2±8.1°, and the combined pronosupination ROM was 82.6± 17.2°. Pronation stiffness was 0.07±0.03 Nm° and supination stiffness was 0.09±0.02 Nm°.

DISCUSSION: We observed that the major principal direction of the thumb CMC range of motion was oriented along the adduction-extension to abduction-flexion axis, which is the path through which the thumb carries out the functional motions of opposition and reposition. Accordingly, the major principal axis of thumb CMC joint stiffness was oriented approximately orthogonal to the ROM principal axis. Pronation and supination stiffness values were consistent with those reported by Shrivastava et al [4]. This study is novel in its presentation of the thumb range of motion envelope in vitro. Traditionally, thumb ROM is characterized by the primary anatomical directions of flexion, extension, abduction, and adduction. However, our data indicate that the greatest ranges of motion of the thumb lie oblique to these primary axes. This pattern is consistent with healthy in vivo thumb circumduction data [1]; however, our ROM values are larger than those reported in vivo [5]. This is likely due to specimen preparation (i.e., missing MC2, musculature) and an externally applied torque that may be greater than the torque at the position a subject would usually consider their maximal range of motion.

SIGNIFICANCE: These results provide healthy CMC biomechanical data that can be used as a benchmark for understanding the mechanics of the pathological and post-operative joint. Additionally, these results support the feasibility of testing CMC biomechanics across a spectrum of osteoarthritides presentation, which would build a more complete understanding of the interplay of pathology and joint mechanics.