Acute Cartilage Compression and Recovery in a Cadaveric Joint Model of Simulated Standing and Walking

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INTRODUCTION: Articular cartilage functions as the biological bearing of joints, supporting load and enabling smooth low friction articulation via poroelastic fluid pressure. Over time, this pressure decreases as fluid exits the cartilage matrix, leading to increased deformation and friction. Maintaining and restoring poroelastic pressure, hydration, deformation, and friction is likely crucial for long-term cartilage health. Ex vivo studies first demonstrated the acute recovery of hydration, deformation, and friction when the cartilage was unloaded, allowing it to swell freely. Later it was observed that sliding could also induce cartilage recovery following a period of static loading induced deformation. While dynamic physiological tasks, such as walking, load the cartilage, they also involve cartilage sliding, unloading, and contact exposure which may act to restore hydration, poroelastic pressure, and mitigate deformation. Currently, the balance between these mechanisms during physiological tasks remains unknown.

While traditional ex vivo studies utilizing excised cartilage have been central to the discovery and isolation of these mechanisms, they lack physiological relevance (excised tissue, altered boundary conditions, rigid non-physiological contacts, and simplified kinematics and kinetics) that preclude our ability to accurately model how everyday tasks affect cartilage deformation. To overcome this limitation, we developed an ex vivo cadaveric joint model that maximizes physiological relevance (intact joints, native contact interfaces, and physiological joint dynamics) while also capturing cartilage deformation. In this work, we utilized our cadaveric joint model to investigate the potential of walking to recover cartilage deformation and demonstrate a balance between the exudative and restorative forces.

METHODS: Specimens and Setup: Five adolescent female Duroc pig stifle joints were procured from a local butcher. The majority of the musculature and soft tissue were removed, with the exception of the collateral ligaments, cruciate ligaments, and menisci. The femoral and tibial shafts were potted in polymethyl-methacrylate and secured to a multi-axis robotic test frame. This system included a KUKA KR160 serial arm robot, two ATI FT44208 six-axis load cells, and an OptiTrack Prime X13 motion capture system. These components were integrated using simVITRO software, enabling precise six-axis control of joint kinematics and kinetics.

Task Sequence: A custom protocol was developed to replicate the task sequence from an ongoing in vivo study. Prior to initiating these tasks, we established a reference state by simulating 5 minutes of standing at -521 N along the superior/inferior (SI)-axis, followed by 560 cycles of walking. The axial force along the SI-axis was then reduced to -50 N with all other axes kinematically locked in the heel-strike position from the ASTM F3141 gait profile. Once the target load was achieved, the joint position along the SI-axis was recorded and defined our reference state (SI_{Ref}). This combined SI force target and joint position was repeated for every measure of cartilage deformation throughout the task sequence. The task sequence (**Fig 1**) began with a 30 min standing period which we simulated by kinematically locking all axes at the heel-strike position except the SI-axis which was loaded to -521 N. Every 100 s the deformation was measured. Next, a 10 min walking period was simulated using the ASTM F3141 gait profile. Since this profile represents bipedal human gait, we scaled the forces to 20%. Every 100 s the deformation was recorded. This protocol was repeated across all biological replicates (N=5). Furthermore, we conducted a resection test (N=1) which included the full task protocol followed by joint distraction for 1-hour and resection of all ligaments and menisci. The task sequence was then repeated.

Data Analysis: Cartilage deformation (δ) was quantified by measuring joint translation along the SI-axis: $\delta = SI_{Task} - SI_{Ref}$, where SI_{Ref} and each subsequent measure (SI_{Task}) were quantified at -50 N along the SI-axis with all other axes kinematically fixed.

RESULTS: Simulated standing resulted in a time-dependent increase in compressive cartilage deformation, and reached an average deformation of -0.77 mm after 30 min. Note that the negative sign indicates that the deformation is compressive. During simulated walking, cartilage recovery was observed. On average 0.23 mm or 30% of the initial deformation was recovered after 10 min of simulated walking. Following resection of the menisci and ligaments we observed a 2.6-fold increase in cartilage deformation during simulated standing (intact = -0.75 mm; resected = -1.93 mm). Interestingly, this increased deformation recovered to the pre-resection values following 10 min of simulated walking (intact = -0.33 mm; resected = -0.33 mm).

DISCUSSION: In this ex vivo cadaveric study, we observed time- and task-dependent changes in joint displacement along the SI-axis, reflecting the deformation and recovery of articular cartilage. While the time-dependent trends were consistent across biological replicates, the magnitudes varied (**Fig 1**), likely due to differences in cartilage thickness or material properties which are known to vary between joints and across a single joint surface. A notable observation of this study was deformation recovery during simulated walking, which was suggested by Moore and Burris in 2017. This work builds upon their theory by applying physiological boundary conditions, kinematics, and kinetics.

This study has a few limitations we wish to acknowledge. First, we applied a human gait cycle to a quadruped stifle joint. While this simplification demonstrates proof of concept, it does not replicate the true anatomy and function of human joints. Second, we assumed cartilage was the sole contributor to the time-dependent deformation; however, other materials like the bone and the potting compound will also contribute to this response. Third, we used a 5 s gait cycle which is over four times the duration of an average human gait cycle (1.16 s). This slower gait cycle reduces the sliding speed which may impact cartilage recovery. Despite these limitations, our findings demonstrate that simulated standing induces compressive deformation, while simulated walking promotes recovery.

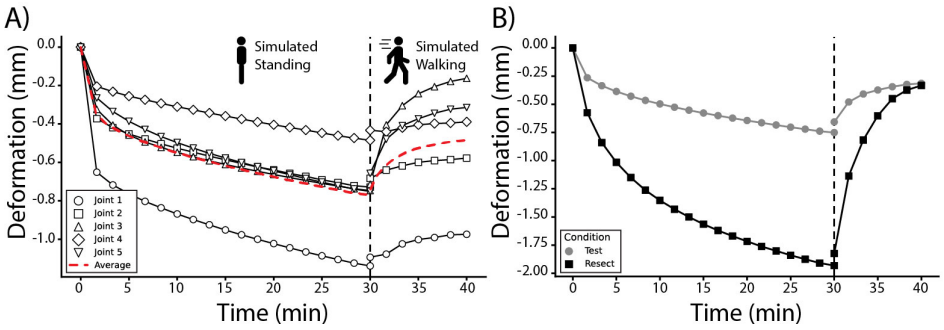

Figure 1. (A) Cartilage deformation during simulated standing and subsequent walking for N=5 biological replicates. (B) A test–resect–retest (N=1) approach was used to evaluate the role of other soft tissues in modulating the deformation response.

SIGNIFICANCE/CLINICAL RELEVANCE: An intact cadaveric joint model was developed to apply physiological kinematics and kinetics and quantify the mechanical function of cartilage in situ. Ultimately, this work will enable us in decoupling the mechanisms that drive cartilage deformation and recovery. This mechanistic understanding will create a path toward clinically actionable PT guidelines and cartilage preservation strategies.