Injurious Impaction to Articular Cartilage Does Not Inhibit Biomechanical Outcomes

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Introduction: Articular cartilage facilitates the load support and friction mitigation necessary for joint articulation. Cartilage is particularly resilient, enduring millions of articulation cycles each year while retaining exceptional mechanical functionality.[1] *Ex vivo*, we have shown that articular cartilages' ability to resist wear is promoted by a sliding/articulation-dependent process that regulates tissue hydration. Using the recently rediscovered convergent stationary contact area (cSCA) explant configuration, which utilizes large (>12mm diameter) convex-surfaced osteochondral explants, we have shown that interstitial fluid lost to compression-induced exudation can be actively pumped back into the cartilage by sliding (Fig. 1A).[2] This mechanism, termed tribological rehydration, results from the curved cartilage surface in the cSCA geometry, promoting the formation of a convergent wedge at the leading edges of contact and allowing for hydrodynamic pressurization of fluid in this wedge during sliding. This pressurized fluid is driven back into the tissue, restoring cartilage hydration, thickness, and pressurization; consistently and reproducibly replicating strain recovery and low friction values seen *in vivo*.[2]

Age-related cartilage degredation and osteoarthritis (OA) are major modern health concerns; however, an accelerated form of secondary OA, post-traumatic OA (PTOA), is an increasing concern in younger patient populations. PTOA is precipitated by injuries such as ligament rupture, meniscal tears, and impacts.[3] Such trauma can immediately change the material structure of articular cartilage, compromising its biomechanical functionality. However, the effects of physical injury on the response of cartilage to physiologically-consistent loading and friction conditions remains unknown. *The goal of this study was to use the physiologically-consistent cSCA configuration to determine the effects of injurious impact on the biomechanical response of articular cartilage to articulation.*

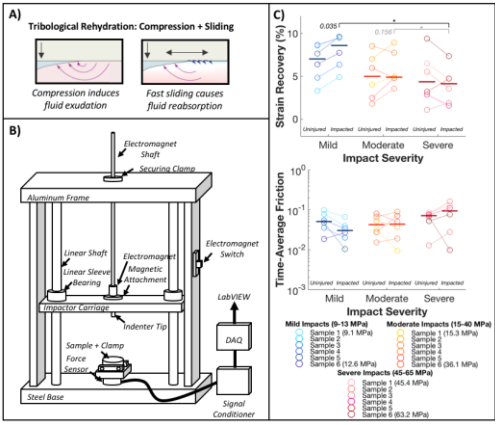

Figure 1. A) Schematic of tribological rehydration, B) diagram of impact drop tower, C) impact did not cause pair-wise changes between before and after impact for strain recovery or time-averaged friction.

Materials & Methods: A custom designed drop tower (Fig. 1B) was used to deliver controlled injurious impacts to osteochondral plugs. Large, 19mm diameter osteochondral explants were harvested from the femoral condyles of bovine stifles and stored in PBS with protease inhibitors at 4°C. Explants were first tested in the uninjured state using a tribological rehydration characterization protocol consisting of 30-min of static compression at 7N ('sedentary period'), followed by 30-min of reciprocal sliding at 80mm/s while maintaining the compressive load ('active period'). Samples free swelled in PBS for >2-hrs before being impacted at the center of the cSCA contact patch and characterized again. Impacts were classified as either low (9-13 MPa peak stress), moderate (15-40 MPa peak stress), or severe (45-65 MPa peak stress) in magnitude.[4]

Results & Discussion: Injurious impact did not cause significant changes in the immediate biomechanical responses of cartilage to compression and sliding compared to their initial un-injured condition. When impacted samples for each group were compared to each other, strain recovery decreased between the mildly and severely impacted samples (Fig. 1C). However, because no differences were observed in the uninjured cartilage among the three testing groups, this finding only served to highlight the subtle nature of the influence of injurious impact on the immediate biomechanical and tribological response of cSCA cartilage explants.

Conclusions: This study speaks to the resilient nature of articular cartilage, demonstrating that impacts well beyond physiological levels do not cause sufficient physical damage to compromise the tribomechanical properties of cartilage in the cSCA. This suggests that physical damage alone may not be the primary mechanism by which articular cartilage degenerates after injurious joint impact, instead implicating cell-mediated responses as a primary

driver of PTOA progression after impact. Future studies will utilize live (viable) cartilage explants to investigate the cellular response to impact, exploring the connection between blunt trauma and the development of PTOA.

References: [1] Eckstein, *Anat Embryo* (1999). [2] Moore, *OA&C* (2016). [3] Buckwalter, *Am J Orthop* (2013). [4] Torzilli, *J Biomech* (1999).