Articular Cartilage Biomechanics and Lubrication Following Mechanical Injury

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Introduction: Healthy articular cartilage maintains extremely low coefficients of friction (~0.02) *in vivo* near 'indefinitely'. Such biomechanical resilience under physiological loads and sliding speeds has only recently been replicated long-term *ex vivo* following our rediscovery of the convergent stationary contact area (cSCA) explant configuration.[1] Using a custom tribometer, we have shown the ability to replicate *in vivo* frictional behaviors *ex vivo* by sliding large, convex cSCA osteochondral explants against a rigid surface. This friction response can be explained by a recently discovered mechanism, tribological rehydration;[1, 2] in which hydrodynamic pressurization of bathing fluid in the convergent wedge of the cSCA (Fig. 1A, top) actively pumps fluid back into the porous cartilage tissue. Tribological rehydration allows applied loads to be primarily supported by the sliding-induced replenishment of cartilage's fluid phase, as opposed to the solid matrix, thus decreasing friction through interstitial lubrication. However, with injury, such articular cartilage tribomechanics may breakdown, causing increased friction/shear and decreased fluid and deformation recovery (Fig. 1B), leading to wear.[3] This increased wear may contribute to joint disease, e.g. osteoarthritis (OA). OA has become more prevalent as younger populations are increasingly developing a post-traumatic form of the disease at earlier ages due to joint injuries, primarily sports-related. Presently, there exists a gap in our understanding of the processes that cause an injured joint to become osteoarthritic, but we predict that altered tribomechanics may accelerate cartilage degeneration, causing earlier onset of OA. Tribological rehydration, which we have shown to regulate the tribomechanical function of cartilage, has been studied in healthy explants[1,4-5] but has not yet been explored in compromised (injured or diseased) cartilage, thus, *the goal of this study is to determine the changes that occur in articular cartilage tribomechanics due to a variety of mechanical injuries.*

Materials and Methods: Large, 19mm dia. osteochondral explants were harvested from the femoral condyles of bovine stifle joints and stored in PBS with protease inhibitors at 4°C between tests. Each explant was first tested in the uninjured state, then again following subsequent injuries of increasing severity (surface vs. full-thickness fissures, or 3mm vs. 6mm dia. chondral defects) (**Fig. 1A**). The testing protocol (30 min static compression at 7N + 30 min reciprocal sliding at 80 mm/s) was used to induce tribological rehydration, mimicking motion during joint articulation.

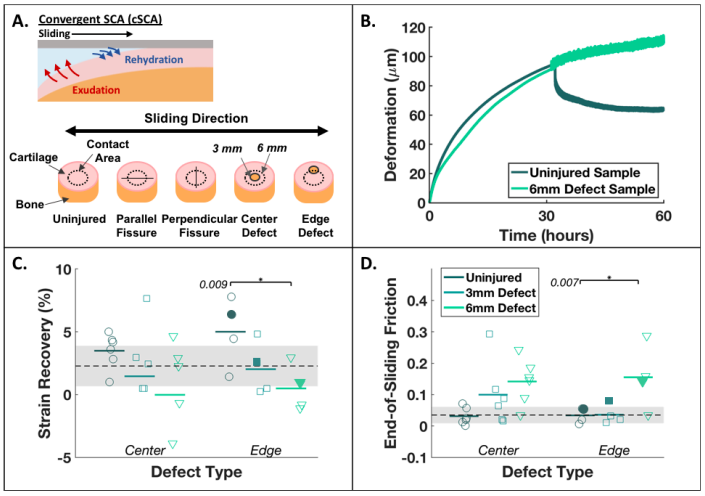

Figure 1. A) Various mechanical injuries investigated in the cSCA configuration, B) sample deformation trace comparing recovery in uninjured state and following severe injury (sample denoted by filled-in markers in C and D), C) recovered strain decreases and D) end-of-sliding friction increases with progressive injury (uninjured, 3mm defect, 6mm defect).

Results and Discussion: Cartilage injury caused a decrease in the degree of strain (and hydration) recovered by sliding (only chondral defects shown, **Fig. 1B, C),** presumably because both fissures and defects interrupt the natural paths for fluid flow and tribological rehydration within cartilage. Friction at the end of sliding also increased with injury (only chondral defects shown, **Fig. 1D**), due to hindered lubrication. Quantification of additional parameters, including shear stress, allowed us to establish the presence of significant changes in cartilage tribomechanics following mechanical injury.

Conclusions: As expected, increasing severity of injury (deeper fissures, larger defects) lead to greater biomechanical dysfunction, elucidating how injury may lead to cartilage degeneration and disease. We're also exploring how injurious cartilage impacts of varying severity (mild: 10 MPa, moderate: 20 MPa, severe: 60 MPa), affect cartilage biomechanics and tribological rehydration. Future studies will also investigate the effect of joint injury on cartilage homeostasis, focusing on the cellular responses to injury, and potential mechanisms for rehabilitation and pharmacological interventions to slow or prevent post-traumatic OA progression.

References: [1] Moore, *OA&C* (2016). [2] Mow, *J Biomech* (1980). [3] Kurz, *Ann Anat* (2005). [4] Burris, *Biotribology* (2017). [5] Graham, *OA&C* (2017). [6] Graham, *J Biomech* (2018).