Sub-Critical Impact Inhibits Cartilage Lubrication Mechanisms

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Introduction: Osteoarthritis (OA) is currently a leading cause of severe disability in the US, and a large proportion of cases (~12%) are attributed to a traumatic injury that may have occurred over a decade earlier, known as posttraumatic OA (PTOA)[1]. Despite the large burden of this disease, its incubation phase is not fully understood or characterized. Both biological influences (e.g. catabolic signaling cascades) and mechanical influences (e.g. disruption of effective lubrication) likely play a role in disease progression. Effective treatment of the disease should inhibit its progression during the incubation phase, but a thorough understanding of the biological and mechanical environment may be necessary to target the most vital aspects altered immediately following an injury [2].

In this study, we probed the lubrication mechanisms of articular cartilage immediately following a sub-critical impact (i.e. an impact that produces surface fissuring but not full thickness defects) to determine what role lubrication may play in the incubation phases of PTOA. To fully characterize the lubrication environment, a framework similar to the Stribeck curve is necessary [3]. Classically, Stribeck curves mapped out the transition from boundary mode lubrication, where friction and wear are high, to hydrodynamic lubrication, where surfaces are separated by a pressurized fluid film and friction is low. By presenting friction as a function of sliding speed, lubricant viscosity, normal force, and contact geometry, the balance between solid contact and lubricant pressurization reveals transitions between lubrication modes. For soft permeable contacts like cartilage, mapping the elastoviscous transition is more appropriate [4]. Similarly to the Stribeck curve, soft permeable contacts transition away from boundary mode but may not achieve full fluid film lubrication due to contact compliance and permeability. In this study, the elastoviscous transition was mapped for both healthy and impacted cartilage, and alterations in this transition reveal specifically which mechanisms of lubrication are altered after injury.

Methods: Using a previously presented, spring-loaded impacting device [5], we impacted neonatal bovine cartilage plugs (6mm diameter by 3mm thick) in unconfined compression to determine impact thresholds resulting in a damaged articular surface but not full-thickness fissuring. Stress and stress rate data were fit to models (Fig. 1A-B) to determine thresholds and correlations with degree of damage. Damage was quantified by converting photographs of impacted surfaces to binary images after application of India ink and measuring the percentage of the surface stained with ink (Fig 1C). After determination of the thresholds, four cartilage plugs were impacted at a level between the thresholds of no damage and full-thickness fissuring (nominally 17MPa peak stress and 24GPa/s peak stress rate). After impact, the samples were tested in a custom-built tribometer. Briefly, samples were compressed to 25% strain and allowed to equilibrate before sliding at a speed sweep between 0.1 and 10 mm/s while bathed in a lubricant. The full elastoviscous transition was mapped by sliding cartilage plugs in three different lubricants with varying viscosities (PBS 1mPas, HA 150mPas, and HYADD 70000mPas).
Friction was presented as a function of the Sommerfield number (viscosity * velocity * contact width / normal force) and fit to a curve to obtain boundary (μB) and minimum (μmin) friction coefficients as well as the Sommerfield number at the midpoint along the transition, referred to as the transition number (St).

**Results:** Significant positive correlations (p<0.05) between stress and stress rate and degree of surface damage provided damage thresholds at 13MPa and 15GPa/s, respectively (Fig 1D-E). Full thickness fissuring was not evident above these thresholds and below 22MPa and 40GPa/s, respectively. The four impacted samples for tribometry had peak stress and stress rate values of 17.2±0.5MPa and 24.8±1.0GPa/s which correlate with ~10% of surface staining for India ink. For healthy cartilage, the tribology curve fit provided values of 0.21, 0.056, and 2.6*10^-6 for boundary friction, minimum friction, and transition number, respectively (Fig 1F). The impacted tribology curve fit provided values of 0.23, 0.084, and 6.9*10^-6 for boundary friction, minimum friction, and transition number, respectively (Fig 1G).

**Discussion:** The threshold analysis provided a range of impact values that result in surface damage without creating a full-thickness fissure in the cartilage samples. Similar injuries that may be difficult to discover arthroscopically may still have a significant effect on the mechanical environment within a joint. Boundary friction increased ~10% indicating the loss of localized lubricants or ineffective lubricant localization with the onset of surface cracking. More dramatically, the minimum friction coefficient increase of 50% and transition number increase of 165% indicate decreased ability to effectively sustain interfacial fluid pressure in superficially cracked cartilage (Fig 1H-J). This finding that damaged tissue would operate comparatively more in boundary mode, which is characteristic of the highest friction coefficients and wear, indicates that superficial tissue damage may predispose a joint to further damage.

**Significance:** This study probed posttraumatic cartilage to determine if lubrication mechanisms are altered after superficial cracking. Using a novel framework, we have shown that both boundary lubrication and, more prominently, the transition away from boundary lubrication are inhibited.

![Figure 1](image-url)