



Cartilage and Joint Lubrication: New Insights Into the Role of Hydrodynamics



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ABSTRACT

Cartilage is slippery when hydrated but it loses hydration and lubricity during loading. However, dehydrated cartilage regains lubricity during high-speed sliding in the stationary contact area; this result is often interpreted as experimental evidence of fluid film lubrication based on theoretical predictions that pre-equilibrated cartilage remains equilibrated during sliding in this testing configuration. In-situ compression measurements were made during variable speed stationary contact area experiments with fully and partially equilibrated osteochondral cores under saline lubrication to test this prediction. Reduced friction coefficients and compression were observed during high speed sliding, at near-physiological speeds. The results suggest that the marked friction reductions observed during high-speed sliding following equilibration were likely consequences of interstitial fluid recovery, a mechanism we term tribological rehydration, rather than the formation of a hydrodynamic fluid film. The results also suggest that hydrodynamic pressurization was the likely driving force underlying the tribological rehydration phenomenon. We propose that external hydrodynamic pressures in the convergent wedge near the leading edge of contact pushed fluid into the porous surface to restore hydration, thickness, mechanical stiffness, and lubrication following periods of static exudation.

1. Introduction

Unlike traditional bearing materials, articular cartilage provides remarkably low friction coefficients under a wide range of physiological load and speed conditions. These unusual tribological properties are primarily attributable to its unique biphasic structure. Cartilage comprises about 80% water and 20% extracellular matrix [1]. During early studies of cartilage against glass, McCutchen observed the same low friction coefficients as those reported for whole joints and thus reasoned that the underlying lubrication mechanism must be the same [2]. However, when testing over longer time scales (~30 min), these low initial friction coefficients increased by more than an order of magnitude as the applied load slowly wrung the interstitial fluid from the tissue (dehydration).

Numerous theories have been proposed to explain this well-established hydration-dependent lubrication phenomenon of cartilage and other biphasic materials [3–6]. The interstitial lubrication theory, in particular, has been widely accepted within the cartilage lubrication community [7]. According to interstitial lubrication theory, load-induced interstitial fluid pressure reduces friction by preferentially

supporting the applied load and reducing the load carried by frictional solid contacts. The mechanism is analogous to hydrostatic lubrication and is effective even without a fluid film. The magnitude of these initial interstitial pressures, which depends largely on tissue hydration and elastic properties [7–10], is typically in the range from 90 to 99% of the contact pressure [7,10–12]. However, these same interstitial pressures also drive fluid from the tissue; the resulting loss of interstitial hydration causes a corresponding loss of interstitial pressure and lubrication [13]. In the absence of interstitial pressure, boundary molecules (e.g. lubricin, hyaluronic acid) are particularly important to the friction and wear mitigation of cartilage and joints [3,14–16].

Given the geometry and speeds (~100 mm/s) of a typical joint, hydrodynamic effects are assumed to play a significant and independent role in the lubrication of joints [6,17–19]. In their seminal paper on ‘boosted’ lubrication, Walker et al. pre-equilibrated large cartilage samples to eliminate interstitial lubrication [6]. The observation that high friction in the slow-speed boundary regime transitioned toward low friction at higher speeds led them to conclude that a hydrodynamic fluid film was responsible for friction reduction. More recently, Gleghorn and Bonassar used a similar pre-equilibration

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approach to eliminate interstitial lubrication before mapping the ‘Strikebeck surfaces’ of cartilage [20]. The boundary friction coefficients they observed in saline and synovial fluid were ~ 0.28 and 0.12 , respectively, and were maintained at speeds below ~ 1 mm/s. At increasing speeds above 1 mm/s (up to the maximum speed of 50 mm/s), they observed monotonically decreasing friction coefficients down to ~ 0.05 , which was interpreted as a shift in load support from the asperities to the fluid film (i.e. mixed mode lubrication).

Although most biomechanics textbooks support the hypothesis that hydrodynamic fluid films provide an important mechanism of joint lubrication [18,19,21], a strong case to the contrary has been made based on the potential flow of fluid into the porous surface [7,22,23]. Upon measuring higher than expected friction at their highest speed condition, Gleghorn and Bonassar proposed that the developing fluid film likely flowed into the porous surface, thus preventing any eventual transition to full film lubrication [20]. Numerous theoretical follow-up studies have shown that surface permeability only hastens the loss of a pre-existing squeeze film by creating an alternate pathway for fluid flow [24,25]. Nonetheless, there have been no direct experimental or theoretical studies investigating the relative extent to which hydrodynamically entrained fluid flows into and between contacting cartilage surfaces.

In our previous study, we interpreted marked sliding-induced thickening of cartilage after partial equilibration as indirect experimental evidence of fluid recovery (rehydration) [26]. We propose here that the ‘tribological rehydration’ phenomenon we observed generally discourages the formation of full film lubrication, as first suggested by Gleghorn and Bonassar [20], and simultaneously restores interstitial pressure/lubrication following pre-equilibration, particularly during sliding at high speeds (high hydrodynamic pressure). In this paper, in-situ displacement and friction measurements were used to clarify the relative contributions of fluid film and interstitial lubrication to the speed-dependent friction response of cartilage to sliding in the typical stationary contact area configuration.

2. Methods

2.1. Specimens

Ten osteochondral cores of 19 mm diameter (ϕ) were removed from the femoral condyles of 5 mature bovine stifles, rinsed, and stored in phosphate buffered saline (PBS). Following a $1 +$ hour equilibration in PBS, samples were removed and prepared for testing. Samples that did not undergo immediate testing were placed in a laboratory refrigerator at 4°C for no > 4 days, a duration shown to be insufficient to cause detectable changes in material and tribological properties [27]. Samples that could not be tested within 4 days were immediately dehydrated after harvest and stored at 4°C for extended periods of time, a practice that has proven equally capable of preserving tissue mechanics [2,28,29]. Dehydrated samples were rehydrated overnight at 4°C in PBS before testing.

2.2. In-situ Tribometer

The in-situ tribometer is shown in Fig. 1 and has been described elsewhere in more detail [26]. Briefly, the sample is clamped via the subchondral bone and loaded to 5 N against a glass microscope slide (Fisher Scientific, $25 \times 75 \times 1.0$ mm) that reciprocates along a 20 mm long track at speeds up to 80 mm/s (2 Hz). The device is instrumented with a 6-channel load cell (ATI Nano 17, 0.005 N resolution) to measure normal and friction forces and a linear variable differential transformer (LVDT, RDP GT2500, 0.1 μm resolution) to measure the distance between the base of the clamped subchondral bone (below the cartilage) and the frame of the instrument as shown in Fig. 1A.

2.3. Tribological Testing

Reciprocating cartilage-on-flat experiments, similar to those of previous studies [6,20,30–32], were used to test our hypothesis that sliding restores interstitial fluid, interstitial pressure, and interstitial lubrication following pre-equilibration of the cartilage. This configuration is commonly referred to as a stationary contact area (SCA) because the contact area never moves relative to the cartilage [32]. As Walker et al. noted, samples large enough to retain the convergent wedge at the leading edge of contact also support the development of external hydrodynamic pressures during sliding [6]. We sub-classify this particular setup as the convergent stationary contact area (cSCA) when the sample diameter is larger than the contact diameter [33]. Preliminary in-situ contact diameter measurements on two representative samples (Fig. 1) demonstrate that a 5 N load generates a contact diameter of ~ 5 mm and a mean contact pressure of ~ 0.25 MPa. Under these conditions the 19 mm diameter sample safely satisfies the requirements of the cSCA configuration.

2.4. Experiments

Each cartilage sample was pre-conditioned by 2 min of static loading at 5 N, followed by 2 min of sliding (60 mm/s) at 5 N, and finally by 2 min of unloaded free swelling in PBS. Tribological experiments, each of which followed this pre-conditioning procedure, followed one of several protocols to test different aspects of the tribological response. The first procedure was designed to reproduce the experiments from Walker et al. [6] as closely as possible. A representative cartilage sample was brought to equilibrium using 14 h of static loading at 5 N of load to eliminate pore pressure [6,13,31,37]. Following equilibration, compression and friction were measured in-situ at increasing sliding speeds from 0.5 to 80 mm/s. In our experience, sliding under equilibrium conditions can quickly damage the articular surface; in an effort to mitigate the effect of damage on the results, sliding at each speed was limited to only four reciprocation cycles. Following testing in the cSCA, the sample was trimmed to $\phi 5$ mm to test the corresponding response to sliding at increasing speeds in the SCA configuration (Fig. 1B demonstrates that the contact diameter is approximately equal to the sample diameter in this case).

A second set of experiments with $N = 5$ samples was designed to probe the equilibrium response as a function of speed. Following pre-conditioning, fully hydrated samples were taken through a high (80 mm/s) to low (0.5 mm/s) speed sweep at the same 5 N load. Unlike the previous study, which was limited to four cycles, sliding was maintained at each speed condition until the friction coefficient and compression reached steady states defined by changes of < 0.01 μmin and < 0.01 mm/min, respectively. The mean steady state value for each speed was calculated over the smaller of 10 cycles and 5 min. The sliding speed was incrementally reduced following steady state at each speed condition. The equilibrium speed sweep test was repeated at 1 N of applied load to study the effect of load on the mechanical and tribological responses of cartilage ($N = 3$ samples).

A third set of experiments was conducted to probe the loss and recovery dynamics. A single sample was loaded statically at 5 N and held for 600 s to simulate the partial equilibration that accompanies intermittent sitting and standing. After this initial exudation event, the sample underwent 3 bouts of intermittent sliding (500 s on, 1000 s off).

When appropriate, one-way ANOVA's were performed using the statistical software package JMP to test for significant differences ($p < 0.05$).

3. Results

Following equilibration at 5 N, the friction coefficient of cartilage in the cSCA ($\phi 19$ mm) increased monotonically with increasing speed from 0.5 to 20 mm/s (Fig. 2). Above 20 mm/s, friction began

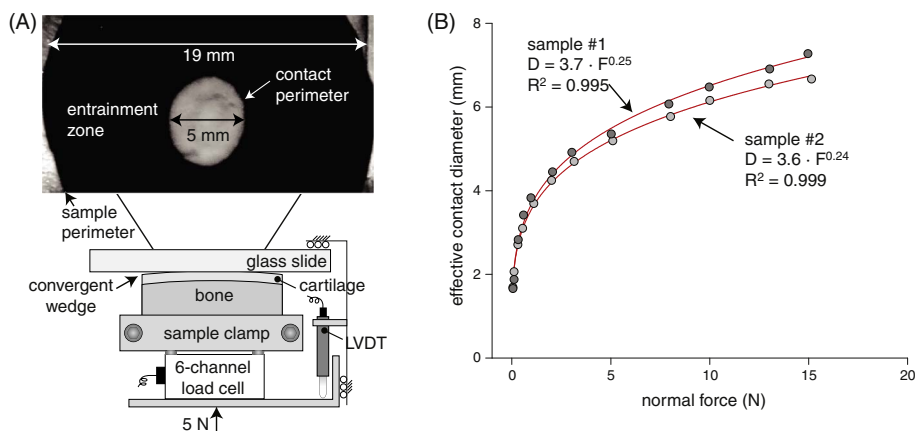


Fig. 1. (A) A schematic of the tribological contact and a representative contact area image as seen through the glass slide. The glass slide can be reciprocated up to 80 mm/s along a 20 mm long track. The contact image was produced through a particle exclusion assay [34] in which the tissue was submerged in India ink and pressed against the glass slide under variable loads. The contact was unloaded in-between each load condition. The growth in contact area with load demonstrates the presence of a convergent wedge. Image is adapted with permission from [26]. (B) The contact diameter (D) is plotted as a function of normal force (F) for $N = 2$ samples. A power law model was fit to the data to determine if Hertzian scaling applied. As demonstrated by the model fits, contact diameter scales with force to the 0.25 power, not the 0.33 power predicted by Hertzian contact mechanics [35]. This difference is attributed to the stiffening effect of the subchondral bone [36].

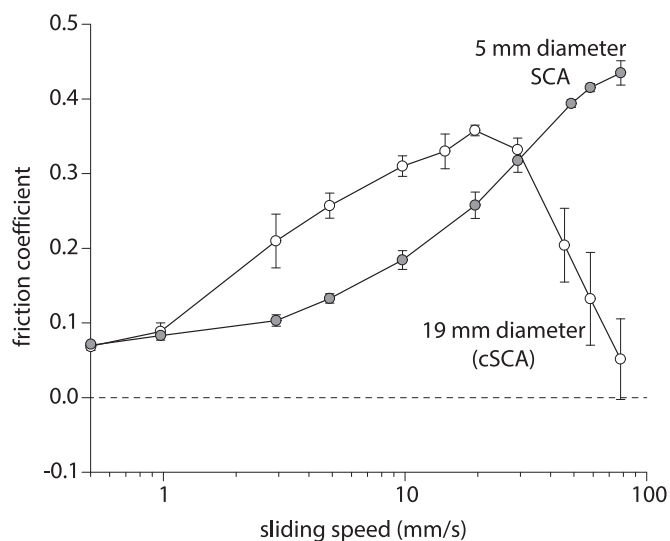


Fig. 2. A single osteochondral core was fully equilibrated at 5 N. A total of 4 sliding cycles were run at each speed (0.5 to 80 mm/s) to mitigate damage accumulation. Friction coefficient is plotted as a function of speed for the original $\phi 19$ mm (open circle) and trimmed $\phi 5$ mm (shaded circle) sample. Error bars represent the standard deviation of 4 cycles.

decreasing with increased speed, which is consistent with the results from previous studies [6,20]. The friction coefficient of the same sample in the SCA (trimmed to $\phi 5$ mm) increased with speed over the entire speed range (0.5 to 80 mm/s). The fact that the transition to low friction following equilibration requires high-speed sliding and a convergent wedge suggests that friction reductions are of hydrodynamic origin as originally proposed by Walker et al. [6].

The steady state friction coefficient and compression responses of cartilage ($N = 5$) to sliding in the cSCA are given as functions of speed in Fig. 3. At speeds below 10 mm/s, the steady state friction coefficients of all five samples were in the range from 0.1 to 0.5 and were largely independent of speed below 10 mm/s. Beyond a critical speed that varied between 10 and 30 mm/s, the friction coefficient of each sample transitioned toward low friction. At 80 mm/s, four of five samples had friction coefficients below 0.03; the sample that did not reach this threshold also had the highest transition speed. These differences in transition speeds are likely driven by differences in cartilage curvature and material properties, both of which vary significantly throughout a joint [27,38,39]. Regardless of these differences, it appears likely that all five samples would have reached physiological friction coefficients (~ 0.02) [40] at physiological sliding speeds (100–150 mm/s) [41]. The steady state compression response in Fig. 3B reveals significant variations in initial compression; this variation is due to large inter-sample

variations in compression modulus and thickness [27]. Compression at steady state decreased with increased speed for each sample. On average, the samples thickened by $160 \pm 70 \mu\text{m}$ from low-speed to high-speed sliding (60–80 mm/s) where all samples reached a mean steady state strain of $5 \pm 3\%$. Whereas small changes in displacement can be attributed to the formation of a fluid film ($< 0.1 \mu\text{m}$) [33] or shear-compression coupling ($\sim 1 \mu\text{m}$) [42], height changes of this magnitude ($> 100 \mu\text{m}$) are almost certainly attributable to the net recovery of interstitial fluid. The fact that the friction coefficient and compression transitioned at comparable speeds suggests that the mechanism of decreased friction was also associated with the recovery of interstitial fluid, i.e. interstitial lubrication, rather than hydrodynamic fluid film lubrication.

Differences between steady state and transient ‘four-cycle’ speed sweeps (Fig. 2) are shown in Fig. 3 (black and red markers respectively) and reveal some interesting trends. First, at low-speed, the first condition tested, the friction coefficients for the first few cycles post-equilibration tended to be lower than those obtained at steady state. Our observations suggest that shear from sliding can wring out additional near-surface water and provides a secondary interstitial lubrication effect just following static equilibration. Second, the transition to low friction occurred at comparable speeds regardless of the number of cycles (four vs. steady state), which suggests that the recovery of interstitial lubrication was not strongly time dependent. Third, the compression recovery from four-cycles ($\sim 30 \mu\text{m}$ over 44 total cycles) was small compared to the compression recovery at steady state (160 μm); whereas friction reflects near-surface hydration, which can be recovered relatively quickly, compression reflects bulk hydration, which takes time to accumulate.

The effects of load on the fluid and lubrication recovery responses of cartilage in the cSCA are illustrated in Fig. 4. At 0.5 mm/s, cartilage produced statistically higher friction coefficients at 1 N than at 5 N, which is consistent with the sublinear dependence of contact area on normal force (Fig. 1B) [43,44]. However, friction and compression recovery required lower speeds at 1 N than at 5 N, which is consistent with a hypothetical competition between load-dependent interstitial pressure (outflow) and speed-dependent hydrodynamic pressure (inflow).

Fig. 5 illustrates the loss and recovery dynamics during intermittent sliding conditions. During contact, the fully hydrated sample exuded fluid as expected until sliding was initiated. During sliding, the compression and friction coefficient decreased toward 0.06 mm and 0.01, respectively, due to the known effects of interstitial fluid recovery on thickness and friction. When sliding stopped, the prior exudation process resumed as before without any obvious influence from the previous sliding condition. All three bouts of sliding produced comparable friction coefficient (0.007 ± 0.0004) and compression (0.058 ± 0.004 mm) results, which demonstrates that tribological

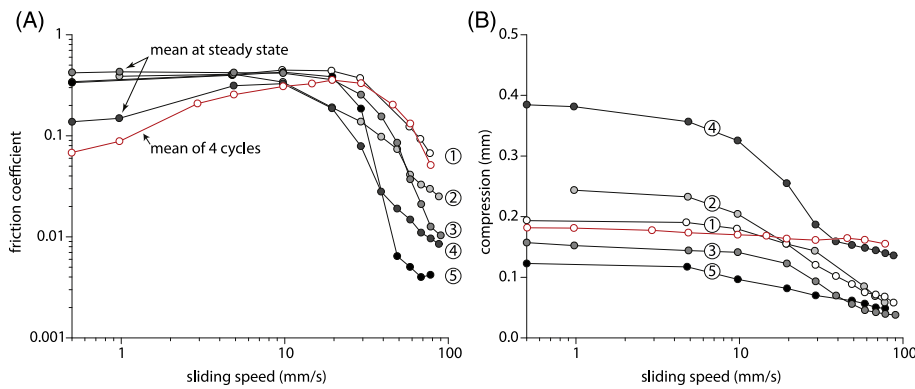


Fig. 3. (A) Steady state friction and (B) compression as a function of sliding velocity under a normal load of 5 N. Data points are connected, shaded and numbered to indicate measures from the same sample. Friction and compression data from the first 4 sliding cycles, from Fig. 2, are shown for comparison.

rehydration is history independent and repeatable. The results illustrate: 1) a reproducible competition between loss and recovery; 2) the first order nature of the loss and recovery processes; and 3) the very different steady state hydration levels associated with active and static loading.

The deformation rates just before and just after initiating sliding (Fig. 5A) provide interesting insights into the competing rates involved in tribological rehydration. The competing rates are quantified for each of the three transitions in Fig. 5B. During static loading, the mean loss rate (change in thickness) due to exudation was 25 nm/s. During subsequent sliding (at the same strain), the recovery rate was 260 nm/s on average. The disproportionately high rates of recovery help explain why relatively brief and infrequent bouts of activity are sufficient to prevent significant joint space thinning due to the loss of interstitial fluid over the long term [45]. They also indicate that the driving force for recovery (e.g. hydrodynamic pressure) must have been far greater than the interstitial pressures driving exudation (~ 0.25 MPa at 5 N).

Fig. 5C illustrates that lubrication and thickness recover over very different time scales. Friction decreased quickly during sliding following static loading with a mean time constant of 16.5 ± 0.1 s; compression took longer to decrease with a mean time constant of 63 ± 8 s. This result is also quite significant in terms of cartilage function. The high friction during startup after inactivity (Fig. 5A) can damage protective boundary films and, eventually, the permanent collagen structures of the cartilage surface; rapid friction reduction helps minimize stresses on the extracellular matrix and the risk of permanent structural damage. This difference in time constants reflects the fact that the surface can be rehydrated faster than the bulk.

4. Discussion

A primary aim of this study was to test a common assumption that pre-equilibration reliably prevents the interstitial lubrication of cartilage, and the dependent conclusion that low friction during high-speed sliding is due to classical hydrodynamic fluid film or mixed-mode

lubrication. The results of this study indicate that cartilage thickened markedly (by ~ 160 μm) as friction decreased during moderate to high-speed sliding; the most reasonable explanation for this magnitude of thickening is the sliding-induced recovery of interstitial fluid, which would be expected to significantly reduce friction with or without hydrodynamic effects. This explains why a sudden transition from high-speed to slow-speed sliding is accompanied by a slow decay in lubrication with a time-constant equal to that of exudation [26].

Fig. 5A demonstrates that the greatest fluid recovery rates accompanied the highest friction coefficients; this observation demonstrates that tribological rehydration preceded any eventual formation of a hydrodynamic fluid film. Even more interestingly, the results in Fig. 5A indicate that flow into the surface (rehydration) is preferred over flow between surfaces (fluid film), particularly in the dehydrated state when fluid films are presumed to be most critical to the lubrication of joints [19]. These results provide compelling evidence that friction decreased as a result of tribological rehydration and the corresponding recovery of interstitial hydration, pressure, and lubrication.

According to interstitial lubrication theory, friction is proportional to the solid contributions to load support [7,13] and it can be shown that the effective friction coefficient has the form [46]:

$$\mu_{\text{eff}} = \mu_{\text{eq}} \cdot \varepsilon \cdot E_{\text{eq}} / \sigma \quad (1)$$

Based on the measured strain ($\varepsilon = 5\%$), equilibrium friction coefficient ($\mu_{\text{eq}} = 0.3$), contact stress ($\sigma = 0.25$ MPa), and equilibrium modulus ($E_{\text{eq}} = 0.5$ MPa [27]), interstitial lubrication is expected to produce a friction coefficient of 0.03, which is consistent with the mean value from all five samples (0.023 ± 0.026) at the highest speed condition. According to the polymer fluctuation lubrication hypothesis [5], friction is independent of the ‘solid’ contribution to load support and is primarily due to the drag of interstitial fluid through the first layer or two of the polymer mesh [47]; in this case, the measured friction coefficients suggest a mesh size of 5–10 nm, which is consistent with the known mesh size of cartilage [2]. Amazingly, these apparently

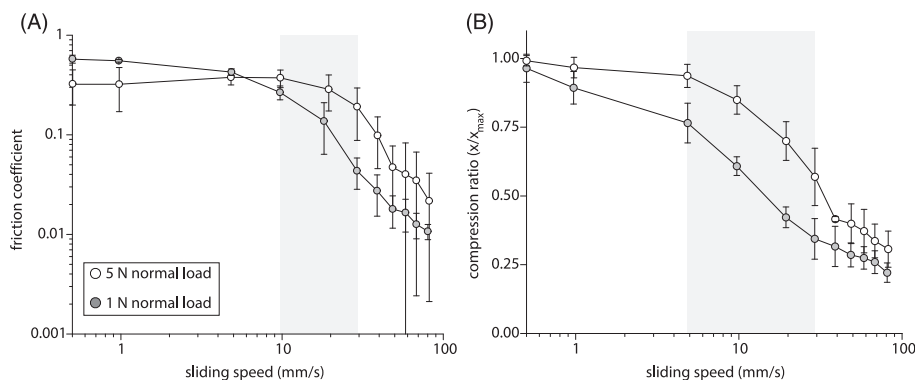


Fig. 4. (A) Steady state friction and (B) compression ratio, x/x_{max} , as a function of sliding speed and load. Compression was normalized to eliminate the load dependence. Data points and error bars represent the mean and standard deviation for $N = 3$ and $N = 5$ independent samples at 1 N and 5 N, respectively. Shaded regions indicate regions of significant difference between the two load conditions. Note that due to the differences in the applied load (1 vs 5 N) that absolute values of tissue compression varied greatly. The mean static equilibrium compression (x_{max}) for 1 and 5 N loads was 0.09 and 0.22 mm, respectively.

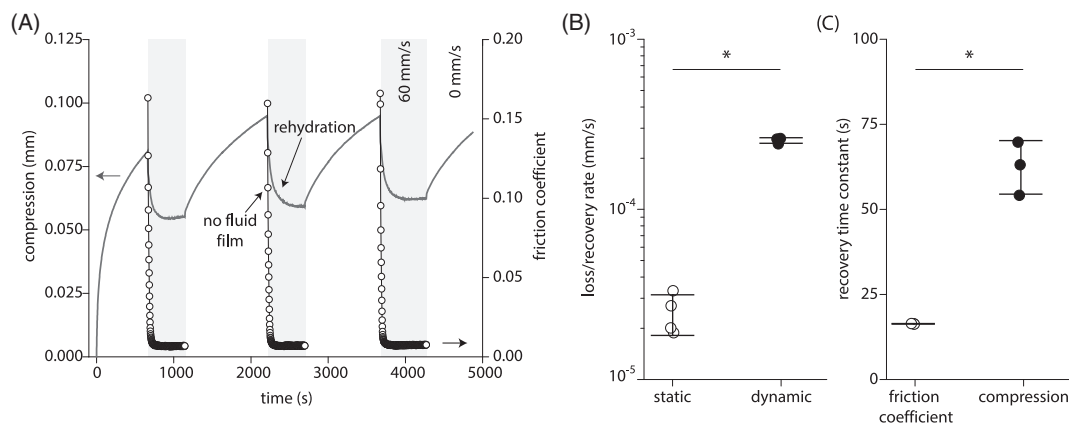


Fig. 5. (A) Compression and friction coefficient as functions of time during intermittent sliding at 60 mm/s. Shading is used to identify regions where sliding occurred. The results demonstrate the repeatable nature of the loss and recovery process, (B) the compression rates during static and dynamic (sliding) contact, and (C) the recovery time constants for friction coefficient and compression. Asterisks denote significant differences.

conflicting theories (interstitial and polymer fluctuation lubrication) agree quantitatively with our measurements. While these analyses provide no insight into which is the correct interpretation, they suggest that the low friction coefficients obtained in this study are more likely attributable to the recovery of near-surface hydration and the resulting recovery of interstitial pressure and mesh size than to hydrodynamic fluid film lubrication in the traditional sense.

Fig. 6A illustrates a model of tribological rehydration that fits our cumulative observations of the phenomenon thus far. During sliding, fluid from the bath to the convergent wedge and pressurized according to well-understood fluid mechanics; the peak hydrodynamic pressure increases with speed and is likely localized to the tip of the convergent wedge. According to Darcy's Law, entrained fluid will flow into the porous surface wherever the external hydrodynamic pressure exceeds the internal interstitial pressure, which is zero at equilibrium; this explains why cartilage thickened even at slow speeds compared to static equilibrium (Fig. 3B). The friction coefficient gradually decreases as the rehydration front propagates across the contact area (Fig. 5A). As Fig. 5B demonstrates, the interface rehydrates quickly, but not instantly, with friction coefficients decreasing from ~ 0.15 to ~ 0.01 with a time constant of ~ 16 s at near-physiological sliding speeds.

Because near-surface hydration is synonymous with lubrication, cartilage can be thought of as a leaky lubrication reservoir (Fig. 6B); the loss, maintenance, and recovery of fluid by the reservoir is governed by the competition between load-induced outflow (exudation) and sliding-induced inflow (recovery). During sitting and standing, the porous tissue leaks at a rate that depends on its permeability, the applied stress, and the strain; under the conditions of these experiments, the 'leak' rate was on the order of 25 nm/s. Sliding served to refill the lubrication reservoir and, as Fig. 5A illustrates, the recovery rate was on the order of 250 nm/s. At steady state, fluid loss balanced recovery at 0.058 mm of compression. The exudation rate at this dynamic steady state was ~ 90 nm/s based on the static exudation curve, which suggests that tribological rehydration rates are also deformation-dependent.

Joints articulate relatively briefly and infrequently, so low leak rates

and high recovery rates are essential to the long-term maintenance of tissue hydration. In the United States, for example, the average person takes ~ 6000 steps per day [48], which corresponds to $\sim 10\%$ activity. As Fig. 5 illustrates, lubrication and hydration are fully restored with time to spare at 33% activity. However, full rehydration becomes less likely as the frequency of activity decreases toward a threshold value that appears to be in the vicinity of 10%. Less frequent activity would increase strain, decrease hydration, and increase friction on average. Our results are consistent with epidemiological studies showing that regular physical activity helps prevent joint-space thinning (indicative of joint disease) over the long-term [49].

Tribological rehydration has equally compelling biological benefits. Interstitial water, which makes up $\sim 80\%$ of cartilage, is vital for controlling the biochemical environment (e.g. pH, ion concentration, and charge density) within the tissue [9]. The loss of significant interstitial hydration during prolonged periods of inactivity (e.g. immobilization) can disrupt homeostasis, cause inflammation, and initiate an enzymatic response that can eventually destroy the extracellular matrix [50]. The detrimental effects of cartilage dehydration are consistent with the prevailing use of joint-space (cartilage thickness) as a clinical assessment of joint health [51]. Additionally, cartilage is an avascular tissue that depends on fluid transport to excrete metabolic waste products and recover nutrients. Whereas fluid film lubrication mitigates friction and wear, tribological rehydration accomplishes the same outcome while simultaneously satisfying the need to maintain hydration and solute transport [52]. It appears to be in the best interest of the tissue to maximize fluid recovery by minimizing flow between surfaces, particularly in low hydration situations.

In this study, the friction coefficient and compression response of five independent samples to high-speed sliding in the cSCA were $\mu = 0.023 \pm 0.026$ and $\delta = 67 \pm 39 \mu\text{m}$, respectively, both of which agree with the results of our previous cSCA study ($\mu = 0.011 \pm 0.007$, $\delta = 73 \pm 35 \mu\text{m}$, $\epsilon = 5.6 \pm 3.7\%$, $N = 26$) [26]. Charnley reported friction coefficients from 0.005 to 0.023 based on $N = 7$ human knees [40] and Linn reported $\mu = 0.011 \pm 0.001$ based on $N = 19$ canine

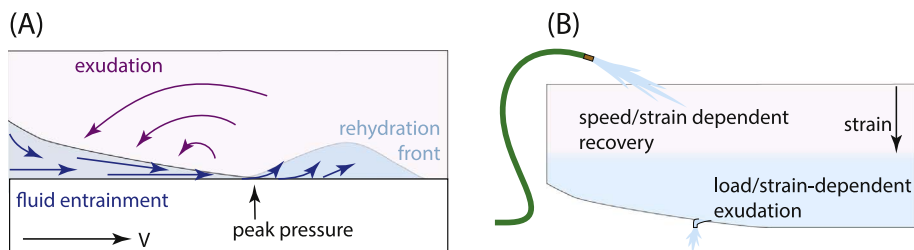


Fig. 6. (A) Hypothesized mechanism of tribological rehydration. (B) The lubrication reservoir model. In this model, lubrication and thickness are proportional to the amount of water in the reservoir, which leaks out over time as a function of load, strain, and permeability. Lubrication is maintained by regularly re-filling the reservoir with sliding (tribological rehydration). The fact that recovery rates greatly exceed ($> 10\times$) exudation rates suggests that external hydrodynamic pressures (recovery) greatly exceeded internal interstitial pressures (exudation).

ankles [53]; both sets of results are in excellent agreement with those of this more controlled study of cartilage on glass. In-vivo joint-space measurements show that human knees maintain strains in the range from 4 to 12% during various physical activities (walking, biking, knee bends) [45,54]. Ex-situ compression measurements from Linn showed that the compression across two joint surfaces was $\sim 150\ \mu\text{m}$ under dynamic equilibrium ($\sim 75\ \mu\text{m}$ each). This consistency suggests that the cSCA experiment described here contains the most important features of the mechanical, tribological, and fluid transport systems in the joint while simultaneously promoting the experimental control of a more traditional cartilage tribology experiment.

5. Conclusions

- 1) Equilibrated cartilage recovered interstitial fluid, interstitial pressure, and interstitial lubrication during high-speed sliding. As a result, we can conclude that pre-equilibration is not a sufficient condition to prevent the interstitial lubrication of cartilage as previously assumed.
- 2) The friction coefficients observed during high-speed sliding were quantitatively consistent with the interstitial and polymer fluctuation lubrication hypotheses based on in-situ compression measurements, measured contact stresses, and the known properties of bovine stifle cartilage. The low friction coefficients measured during high-speed sliding can be attributed to the recovery of near-surface hydration and the resulting increase in interstitial pressure and mesh size rather than the formation of a traditional hydrodynamic fluid film.
- 3) The results suggest that sliding-induced hydrodynamic pressures pushed fluid into the cartilage surface to restore hydration, thickness, mechanical stiffness, lubrication, and nutrient reserves within the tissue. The proposed model treats cartilage as a leaky reservoir: the hydration-dependent mechanical, tribological, and biological functions of the tissue are maintained over the long term if and only if the reservoir is regularly refilled via joint articulation.
- 4) The friction coefficients and deformations observed at steady state during sliding near physiological speeds in the cSCA experiments of this study are indistinguishable from those observed for whole joints. This suggests that the cSCA configuration contains the most important features of the mechanical, tribological, and fluid transport systems in the joint while simultaneously maximizing experimental control over contact pressures, areas, strains, hydration, and lubrication.

Acknowledgements

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