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Cartilage shear dynamics during tibio-femoral articulation: effect of acute joint injury and tribosupplementation on synovial fluid lubrication

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Summary

Objective: To determine the effects of acute injury and tribosupplementation by hyaluronan (HA) on synovial fluid (SF) modulation of cartilage shear during tibio-femoral articulation.

Methods: Human osteochondral blocks from the lateral femoral condyle (LFC) and tibial plateau (LTP) were apposed, compressed 13%, and subjected to sliding under video microscopy. Tests were conducted with equine SF from normal joints (NL-SF), SF from acutely injured joints (AI-SF), and AI-SF to which HA was added (AI-SF + HA). Local and overall shear strain (E_{xz}) and the lateral displacement (Δx) at which E_{xz} reached 50% of peak values ($\Delta x_{1/2}$) were determined.

Results: During articulation, LFC and LTP cartilage E_{xz} increased with Δx and peaked when surfaces slid, with peak E_{xz} being maintained during sliding. With AI-SF as lubricant, surface and overall $\Delta x_{1/2}$ were ~40% and ~20% higher, respectively, than values with NL-SF and AI-SF + HA as lubricant. Also, peak E_{xz} was markedly higher with AI-SF as lubricant than with NL-SF as lubricant, both near the surface (~80%) and overall (50–200%). Following HA supplementation to AI-SF, E_{xz} was reduced from values with AI-SF alone by 30–50% near the surface and 20–30% overall. Magnitudes of surface and overall E_{xz} were markedly (~50 to 80%) higher in LTP cartilage than LFC cartilage for all lubricants.

Conclusion: Acute injury impairs SF function, elevating cartilage E_{xz} markedly during tibio-femoral articulation; such elevated E_{xz} may contribute to post-injury associated cartilage degeneration. Since HA partially restores the function of AI-SF, as indicated by E_{xz}, tribosupplements may be beneficial in modulating normal cartilage homeostasis.

Key words: Synovial fluid, Lubrication, Hyaluronan, Acute injury, Cartilage mechanics, Shear deformation.

Introduction

Within the knee joint, articular cartilage lining the femoral condyle articulates against articular cartilage of the tibial plateau that is uncovered by meniscus [Fig. 1(A)] to facilitate diarthrodial joint movement[1]. Following various physical activities such as knee bending, impact loading, and running, cartilage compresses ~3 to 20%[2–4], with compression typically being higher in cartilage from the tibial plateau than the femoral condyle[3,4]. Under such magnitudes of physiologic compression, tibio-femoral cartilage compression and shear are depth-varying, being highest near the surface and decreasing monotonically with increasing depth, and greater in tibial cartilage than femoral cartilage[5]. However, the shear kinematics as well as the effects of synovial fluid lubrication remains to be elucidated for physiologically apposed cartilage surfaces. Such characterization would further the understanding of cartilage contact mechanics during joint loading and motion by elucidating the boundary conditions at the interface and the intra-tissue shear deformation within physiologically articulating femoral and tibial cartilage surfaces.

In healthy joints, synovial fluid (SF) is present between articulating cartilage surfaces, functioning as an effective boundary lubricant. During cartilage articulation, interstitial fluid pressurization[6,7] and boundary mode lubrication[8] may both mediate friction between articulating surfaces. In boundary lubrication, load is supported by surface-to-surface contact and friction between articular surfaces is thus dictated by bound surface lubricant molecules, which may become increasingly important with prolonged loading as interstitial fluid pressure diminishes[9]. Two major constituents of SF identified to lubricate the articular surface are hyaluronan (HA)[10] and proteoglycan 4 (PRG4)[11,12]. In a configuration to reveal boundary lubrication effects, the surface interactions, as indicated by friction[8] and shear deformation[13,14] of articulating cartilage, are reduced by SF relative to phosphate buffered saline (PBS). The SF lubricant molecules, HA and PRG4, contribute, independently and in combination, to reduce articulating cartilage friction under boundary lubrication conditions[15]. Thus, altered lubricant molecule concentrations may diminish the boundary lubrication function of SF and cause elevated tissue shear, predisposing cartilage to accelerated wear and degradation.

Following acute injury, the friction-reducing function of SF is compromised[16–18], possibly due, in part, to reduced HA

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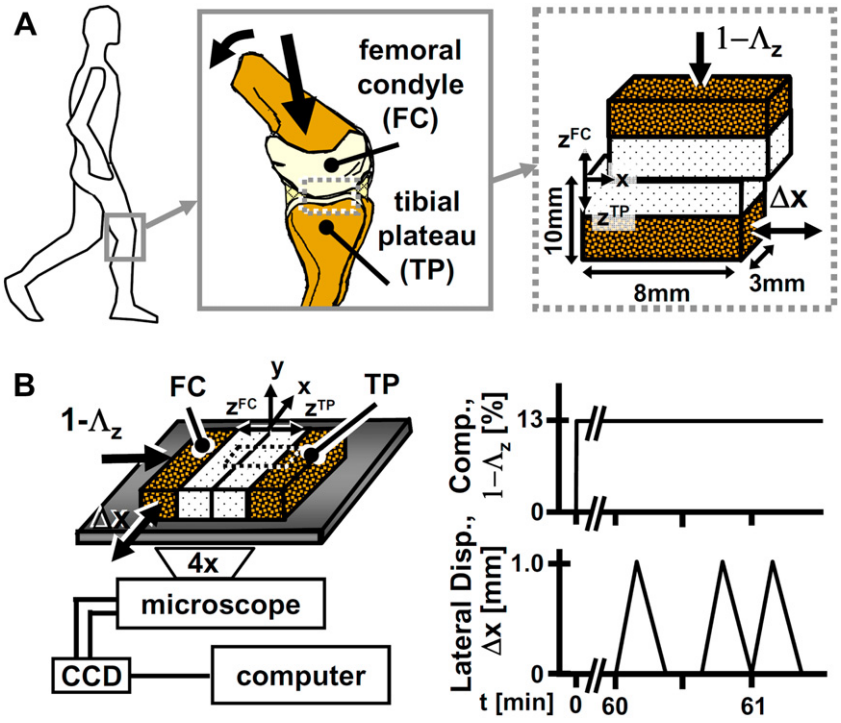

Fig. 1. Schematic of (A) knee joint movements at multiple scales and (B) of experimental setup and loading protocol for microshear testing. Definition of loading variables: compressive axial strain ($1-\Lambda_z$) and lateral displacement (Δx).

concentration. The HA concentration in SF of acutely injured equine joints was decreased from normal (~0.3 mg/ml to 0.2 mg/ml), while PRG4 and surface-active phospholipid (SAPL) concentrations were increased[19]. When SF was tested between articulating cartilage surfaces to reveal boundary mode lubrication, friction was markedly higher with SF from acutely injured joints (AI-SF) than that from contra-lateral normal joints (NL-SF). When AI-SF was augmented with HA (AI-SF + HA), friction coefficient, μ (a dimensionless measure which describes the ratio of frictional force and the normal force between two surfaces), was reduced towards normal values, suggesting lowered HA concentrations reduced SF function. Such alterations in the friction-reducing function of SF following acute injury may alter the normal mechanobiology of articulating cartilage since tissue shear deformation markedly regulates lubricant and matrix metabolism[20–22].

Femoral and tibial human articular cartilage exhibit shear deformation and interaction during tibio-femoral cartilage articulation[5], as revealed by video microscopy[23] to track the displacement of fluorescently labeled cells[24]. With this experimental approach, osteochondral samples from the tibia and femoral condyle were compressed with the cartilage surfaces in apposition and subjected to lateral shearing motion to mimic the biomechanics of tibio-femoral joint articulation [Fig. 1B]. Resultant compressive and shear strains of cartilage were determined locally and overall, with an effective resolution of 40 μm. Using such a configuration, biomechanical microscale analysis can be used to assess the effects of synovial fluid lubrication on local and overall shear deformation of femoral and tibial cartilage during tibio-femoral cartilage articulation.

The hypothesis of this study was that during tibio-femoral articulation, cartilage lubrication by AI-SF elevates tissue shear deformation, while lubrication by AI-SF with augmented HA reduces shear deformation towards normal magnitudes. To test this hypothesis, the objectives of this study were to determine, during tibio-femoral cartilage articulation (1) the effects of acute injury on SF lubricant function and (2) the ability of HA addition to AI-SF to enhance lubricant function, with lubricant function assessed as peak cartilage shear deformation during sliding motion.

Materials and methods

SAMPLE ISOLATION AND PREPARATION

Six osteochondral cores, each with a 10 mm diameter, were isolated, one from each anterior lateral femoral condyle (LFC) of six fresh cadaveric human male ($n=3$) and female ($n=3$) donors (mean ± standard error of the mean (S.E.M) age of 46 ± 1.5 yrs). In addition, six osteochondral blocks (each with a chondral surface area of ~1 cm^2) were harvested from the region of the donor-matched lateral tibial plateau (LTP) not covered by the meniscus. LFC cores with grossly normal surfaces (modified Outerbridge grade of 1[25]) were selected, while all donor-matched LTP blocks displayed mild surface fibrillation and were modified Outerbridge grades of 2–3[25]. The harvested specimens were immersed in phosphate buffered saline (PBS) containing proteinase inhibitors (PIs)[26] and stored at −70°C until use.

The osteochondral specimens were thawed and further processed on the day of testing. The LFC core and LTP block were each trimmed using a low-speed saw with a 0.3 mm thick diamond edge blade (Isomet™, Buehler, Lake Bluff, IL) to yield approximately one rectangular block for biomechanical testing. Each rectangular block had a cartilage surface area of ~3×8 mm^2 and a total thickness of ~1 cm. Blocks were created such that their 8 mm lengths were parallel to the direction of articulation in the joint from which they were isolated from [Fig. 1(A)]. Samples consisted of one LFC and one donor-matched LTP block and were each rinsed with PBS + PI overnight prior to mechanical testing[15].

From macroscopic images[5], thickness measurements of the present samples were made at three separate locations and averaged to yield a full cartilage thickness measurement. For the LTP samples, cartilage thickness (2.88 ± 0.53 mm, mean ± S.E.M, $n=6$ blocks) was somewhat thicker than the cartilage thickness of the LFC samples (2.20 ± 0.15 mm). In addition, the LFC and LTP samples used in this study were characterized by histopathology[5]. LFC cartilage was normal, while LTP cartilage exhibited typical features of very mild degeneration. Both LFC and LTP cartilage exhibited