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The effects of lipid deactivation on repulsive-slippage lubrication: Macroscopic phenomena governed by quantum-level interactions

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Abstract:

Purpose: This study aimed to determine how the integrity and charge state of superficial phospholipid PL bilayers govern the boundary lamellar-repulsive-slippage BLRSL lubrication mechanism in articular cartilage. The work quantified how progressive lipid deactivation affects wettability, electrostatic surface potential ESP, and friction, and explored whether nanoscale proton-dynamics concepts may complement classical interpretations. ***Methods:*** Bovine articular cartilage samples were tested in their native state or after controlled lipid extraction using the Folch method. Measurements included: (i) surface wettability, (ii) friction in a cartilage–cartilage pair under boundary-lubrication conditions (1 mm/s, 15 N), (iii) pH-dependent friction, and (iv) ESP of a DPPE phospholipid calculated via MD simulations (AMBER14) and the APBS method. Delipidated samples served as a model of early osteoarthritic degradation. ***Results:*** Lipid depletion lowered the contact angle from $\sim 100°$ to $\sim 35°$ and increased the friction coefficient from ultra-low values ($f \approx 0.002 - 0.006$) to $\sim 0.02 - 0.023$. Friction displayed a bell-shaped dependence on pH, peaking near the isoelectric point ($\sim pH$ 4.5), consistent with protonation-state changes of PL headgroups. ESP calculations confirmed minimal interfacial stability around the IEP. Interaction of PLs with β_2-Glycoprotein I produced deactivated species unable to form lamellar structures, eliminating the slippage plane. ***Conclusions:*** The transition from ultra-low friction to high boundary friction is governed by the number and integrity of PL bilayers, not by wettability alone. Loss or deactivation of PLs collapses the repulsive-slippage mechanism despite increased hydrophilicity. These findings reveal that macroscopic cartilage lubrication is controlled by molecular-scale PL organization, while proposed quantum-level effects remain a complementary hypothesis requiring further validation.

Keywords: Articular cartilage lubrication, Boundary lamellar-repulsive slippage, Phospholipid bilayers, Surface wettability, Electrostatic surface potential, Biomimetic low-friction surfaces.

I. INTRODUCTION

Boundary lubrication in articular cartilage is strongly influenced by the arrangement and stability of surface phospholipid bilayers. Rather than acting as a simple passive coating, these nanostructures respond dynamically to changes in surface composition, charge distribution, and hydration state, making their lubricating function highly condition-dependent. Stacks of phospholipid layers can support hydration-mediated separation between opposing surfaces, permitting relative motion with minimal resistance. This phenomenon, however, requires well-organized bilayers with adequate surface charge and sufficient hydration; disruption of any of these factors diminishes the ability of the lamellae to sustain repulsive sliding. These features are often oversimplified in classical descriptions of boundary lamellar-repulsive-slippage lubrication (BLRSL) [9], [20], [30], [22], [23], [16], [25].

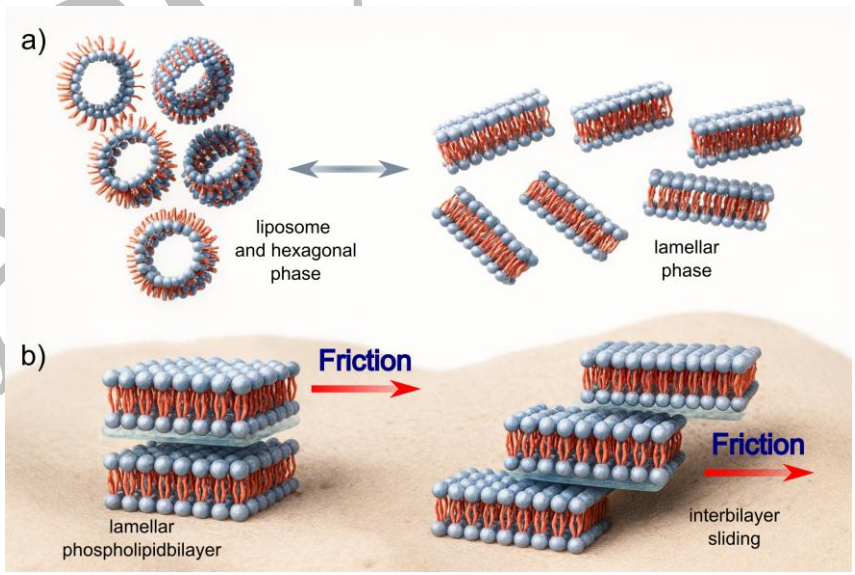

FIG. 1. Self-organized phospholipids: (a) liposomes and hexagonal phases, and lamellar phases under load adsorbed to cartilage surfaces to form bilayers, (b) lamellar repulsive slippage of bilayers.

As illustrated in Fig. 1, only well-organized lamellar phases support efficient interlayer sliding, whereas even modest dehydration or loss of bilayer cohesion leads to substantial increases in friction. This underscores the need to interpret BLRSL behavior within realistic surface conditions rather than idealized textbook scenarios.

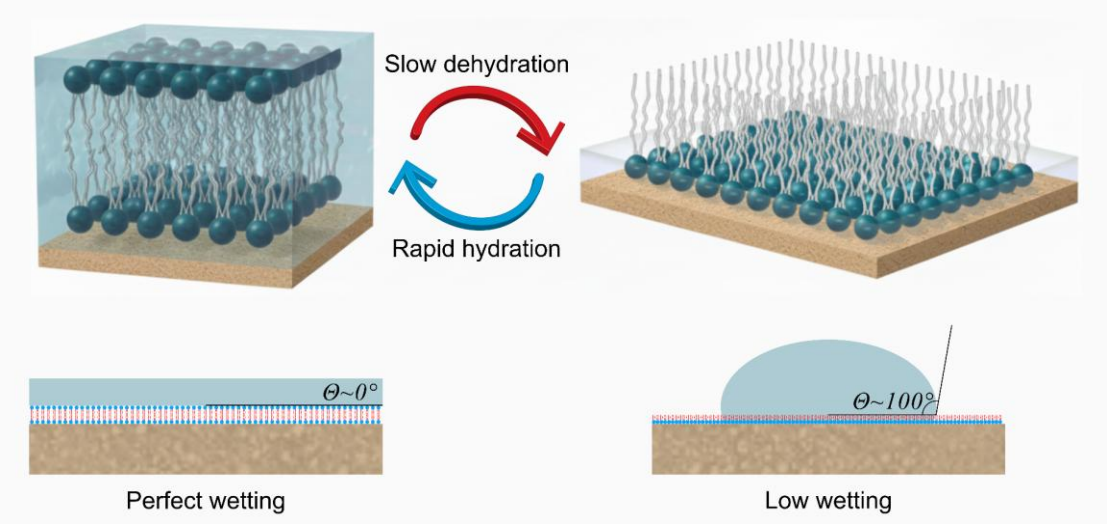

FIG. 2. Schematic representation of the phospholipid arrangement on the articular cartilage surface under hydrated (left) and air-dry (right) conditions. The corresponding wettability changes are illustrated: perfect wetting with a near-zero contact angle ($\theta \approx 0°$) for the hydrated surface and low wetting with a high contact angle ($\theta \approx 100°$) for the dry, hydrophobic surface. The transition between these states involves a flip-flop reorientation of phospholipids within the superficial bilayer.

Figure 2 shows wet AC with hydrophilic and air-dry hydrophobic surface. As the surface dehydrates, phospholipid molecules gradually reorient, leading to a restructuring of the superficial layer and a shift from a highly wettable state to one that is markedly more hydrophobic. The flip-flop rearrangement develops gradually during dehydration, whereas rehydration restores the bilayer structure almost immediately. Consequently, wettability

decreases: low contact angles observed on hydrated surfaces correspond to high hydrophilicity, while high contact angles measured after drying indicate a loss of hydrophilic character and increased hydrophobicity. The dehydration process, along with the accompanying conformational changes within the phospholipid bilayer, proceeds relatively slowly, whereas rehydration and the resulting bilayer reorganization occur rapidly.

In this study, we deliberately restrict our focus to those features of BLRSL that are experimentally verifiable and directly relevant to cartilage-cartilage friction. By comparing native cartilage, which preserves its charged PL bilayers, with delipidated tissue representing an early osteoarthritic phenotype, we expose the degree to which slippage depends critically on the stability of the superficial PL bilayers. Our results demonstrate that the low-friction behavior traditionally attributed to "inherent" cartilage properties is lost almost immediately when the superficial PL structures are disrupted. This finding challenges the common assumption that cartilage lubrication is inherently robust and suggests instead that it is highly vulnerable to biochemical degradation processes characteristic of OA.

Section II extends the discussion by examining whether nuclear quantum effects (NQEs), well established in the behavior of confined and interfacial water, might contribute to the short-range forces implicated in BLRSL. This analysis is intentionally critical and does not claim experimental confirmation at cartilage interfaces. At present, no direct measurements or simulations validate the presence of NQEs in joint lubrication, and any such influence must therefore be regarded as speculative. We present this framework not as an alternative to classical lamellar-repulsive models but as a hypothesis motivated by analogies with other aqueous systems where quantum fluctuations are known to affect hydration forces. This perspective underscores several open questions and points to aspects of interfacial water behavior that are insufficiently captured in current models.

II. A SKETCH OF CLASSICAL-QUANTUM PASSAGE'S EFFECTS ON THE ARTICULAR CARTILAGE SYSTEM

The extremely complex structure of articular cartilage, together with adjacent phospholipid layers and components of synovial fluid, necessitates a multiscale approach to friction phenomena [8]. Although macroscopic friction can be described by continuum models, interfacial interactions at the molecular scale involve electrostatics and quantum effects that cannot be neglected. Experimental studies, including deep inelastic neutron scattering, theoretical models, and simulations indicate that quantum nuclear effects in water can markedly influence its physicochemical properties [6].

From the perspective of facilitated lubrication, the pH of synovial fluid plays a critical role [10], [24]. The friction coefficient of cartilage depends strongly on pH, which itself can be affected by quantum phenomena such as proton tunneling and delocalization. Proton tunneling allows hydrogen nuclei to cross potential barriers lower than their classical energy would permit [6]. Although such behavior is commonly illustrated using simplified one-dimensional barrier models, the relevance of this phenomenon to hydrated biological interfaces remains predominantly conceptual in the present context. The probability T of tunneling through a one-dimensional barrier of height V_0 and width a is given by:

$$T \approx \exp\left[-2a\frac{\sqrt{2m(V_0 - E)}}{\hbar}\right] \quad (1)$$

where: a denotes the width of the potential barrier, m is the mass of the tunneling particle, V_0 signifies the height of the potential barrier, E represents the energy of the tunneling particle and $\hbar$ is the reduced Planck constant.

This effect can influence local proton concentrations and hence the pH of confined water layers between bilayers [3]. Experimental isotope-substitution data demonstrate that nuclear quantum effects measurably shift the autoprotolysis constant of water: the pH of pure H_2O at 298 K increases from 7.0 to about 7.4 in D_2O and to 7.6 in T_2O, consistent with a mass-dependent change in acid dissociation [6], [28]. These data demonstrate that isotope-dependent proton dynamics influence the acid-base properties of water, although the implications for cartilage interfaces remain uncertain. However, direct experimental evidence that proton tunnelling alters the pH of nanometer-thick water films confined between cartilage phospholipid bilayers is not yet available; in this work we therefore treat this link as a qualitative hypothesis motivated by the general behavior of nuclear quantum effects in water.

Non-covalent intermolecular forces, including those collectively described as van der Waals interactions (Keesom: dipole-dipole [14], Debye: dipole-induced dipole [29], and London dispersion: induced dipole-induced dipole [5]), contribute to the cohesion of molecular assemblies within cartilage. Although these forces arise from different modes of dipolar and electronic fluctuations, their combined effect is comparatively weak under physiological hydration, where hydration repulsion and electrostatic interactions dominate the interfacial mechanics. Together with the hydration (water-structuring) force [34], these interactions help maintain nanoscale separation between opposing surfaces, thereby enabling low friction without direct solid-solid contact. Quantum effects contribute through electron-density fluctuations and proton dynamics that influence the structure and stability of the interface.

Recent surface-force measurements between hydrophobic self-assembled monolayers in light and heavy water further support this view: hydrophobic adhesion between fluorinated surfaces is approximately 10% stronger in H_2O than in D_2O, even though the macroscopic contact angles of the two liquids are indistinguishable [32]. This difference is reproduced only

when quantum corrections to the interfacial free energy are included in the analysis, indicating that nuclear quantum effects can modify interfacial forces at nanometer separations without altering macroscopic wettability.

Phospholipid bilayers, being negatively charged, experience strong electrostatic repulsion. Classically this can be described by Coulomb's law, but quantum mechanics reveals a richer picture involving electronic polarization, proton tunneling, and orbital overlap leading to quantum corrections to the potential energy landscape. Such effects extend to wettability, which depends on the interplay between electron density distribution, hydrogen bonding, and van der Waals interactions [27], [11], [31]. At present, quantum tunnelling and electron-density fluctuations are expected mainly to affect the nanometer-scale structure and dynamics of interfacial water and adsorbed lipids, rather than macroscopic contact angles. Their possible influence on the effective contact area and spreading behavior of water at cartilage interfaces should therefore be viewed as a conceptual extension of the known nanoscale effects, rather than a quantitatively established mechanism.

The electrostatic surface potential of phospholipid bilayers is also governed by quantum effects. DFT-based calculations predict charge distributions in the headgroup region with high precision [4]. The ESP of phospholipid bilayers can be derived from the Poisson equation [1]:

$$\nabla^2\phi(\mathbf{r}) = -\frac{\rho(\mathbf{r})}{\varepsilon_0\varepsilon_r} \tag{2}$$

where $\rho(\mathbf{r})$ is the charge density distribution, ε_0 and ε_r are the dielectric constant of vacuum and the relative dielectric constant of the medium, respectively. Variations in $\phi(\mathbf{r})$ across the headgroup region determine interfacial stability and wettability.

Proton tunneling and hydrogen-bond dynamics at the aqueous interface modulate local protonation states and charge organization, influencing bilayer stability and self-assembly. Proton

tunnelling and delocalization in hydrogen-bonded water clusters have indeed been directly observed - for example, in cryogenic scanning tunnelling microscopy (STM) studies of water nanoclusters adsorbed on insulating films, where concerted proton transfer within a cyclic water tetramer was detected [18]. Extrapolating these observations to the specific case of cartilage phospholipid bilayers is, however, non-trivial, and the proposed link to bilayer stability in joints should at this stage be regarded as a working hypothesis that calls for dedicated simulations and experiments.

The amphiphilic character of phospholipids arises from the electronic structure of their headgroups and tails, which determines both charge distribution and dipole moment. The classical-to-quantum passage thus directly affects their interfacial behavior and the structuring of surrounding water.

Different formulations of the classical-quantum transition influence the macroscopic characteristics of phospholipid bilayers - particularly their stability, charge organization, and wettability. A comprehensive understanding of facilitated lubrication in articular cartilage therefore requires elucidation of this transition pathway, which is essential to accurately capture the interplay between electrostatic, dispersive, and tunneling phenomena governing the lubrication mechanism at the quantum level.

Overall, our discussion of nuclear quantum effects is intended to provide a physically plausible framework linking nanoscopic fluctuations of water and phospholipids to the macroscopic tribological response of cartilage. It does not yet constitute a full microscopic derivation of joint lubrication, which remains an open problem.

III. EXPERIMENTAL METHOD

A. Materials

Articular cartilage samples were obtained from bovine knees (~1.5 years old). Osteochondral plugs with diameters of 5 and 10 mm were harvested from the medial and lateral femoral condyles. Each plug was trimmed to a thickness of approximately 3 mm while preserving full attachment to the underlying subchondral bone. The counter samples were prepared as cylindrical disks cut perpendicular to the joint surface to ensure consistent geometry and a reproducible contact area. A total of 50 samples were collected. The specimens were stored at 253 K in 0.155 M NaCl (pH 6.9) and were fully defrosted prior to testing. The bone side of each plug was glued to the stainless-steel holder using a fast-curing adhesive so that the cartilage surfaces remained fully hydrated and aligned. This configuration reproduces the cartilage-cartilage contact geometry used in previous work on articular cartilage lubrication [23]. Friction tests were conducted in 0.155 M NaCl solution. Two sample types were tested: (i) untreated bovine articular cartilage and (ii) bovine articular cartilage subjected to lipid extraction using the Folch reagent [7] (a 2:1 v/v chloroform-methanol mixture). The specimens were immersed in the reagent for 9, 13, or 17 minutes at a constant meniscus level. The delipidization times were chosen to match the partially and completely depleted states, respectively. After extraction, each sample was placed in a saline bath for one hour to remove residual solvent and allow complete rehydration. The delipidated cartilage served as a model of osteoarthritic tissue, as the delipidization process mimics the natural depletion of phospholipid bilayers observed in osteoarthritis.

A radiometer pH-meter with an electrode (Schott-Blue-Line 16 pH type) was used for the experiment.

B. Wettability measurements

A KSV CAM100 computerized tensiometer was used to measure the contact angle of cartilage samples. A drop of 0.155 M saline solution was deposited on to the air-dried cartilage surface. The contact angle measurements of normal, partially and completely depleted PLs cartilage samples were carried out under dry-air condition at 295 ± 2 K and a relative humidity, HR $\sim 50 \pm 5\%$ after 90 min of sample drying. The tests on normal, partial, and completely depleted cartilage samples were repeated five times. These samples were used for surface wettability and friction measurements.

C. Test devices and procedure

The coefficient of friction (f) was measured at room temperature using a sliding friction tester (pin on-disc tribotester manufactured by ITeR, PL). The apparatus was designed to provide a reciprocating sliding motion between two samples of cartilage immersed in a buffer solution. The friction between two discs of cartilage soaked in 0.155 M NaCl (as the lubricating fluid, pH 6.9) and subjected to a load, sliding velocities and time were measured. The tests were performed at a very low speed of 1 mm/s is for 10 min and a load of 15 N (1.2 MPa), which corresponded to lubrication under physiological condition. Schematic diagram of the friction test apparatus is shown in Figure 3. This configuration provides a well-controlled boundary-lubrication contact, but it does not reproduce the time-dependent fluid pressurization present in natural synovial joints.

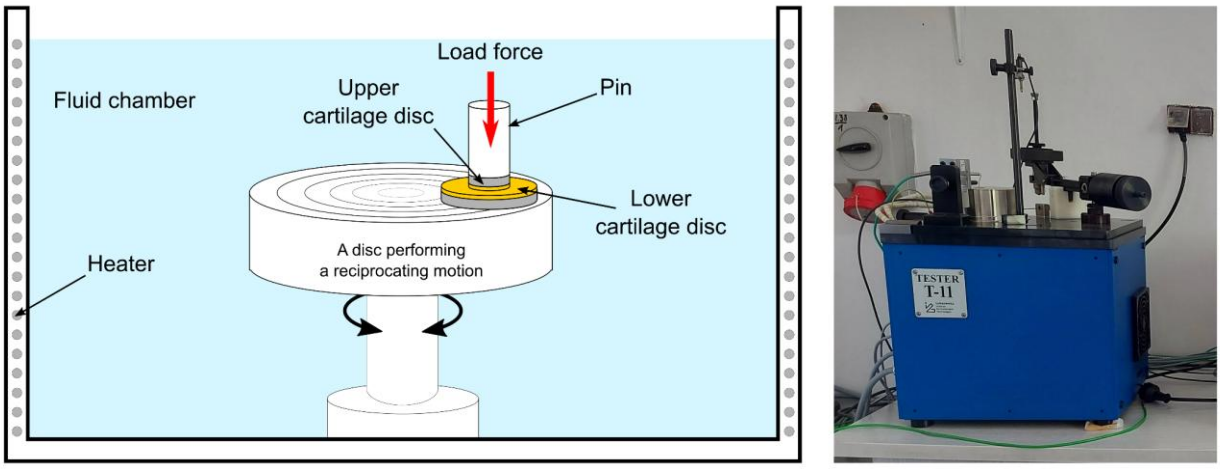

FIG. 3. Schematic diagram of the friction test apparatus (left) and a T-11 sliding friction tester with a pin-on-disk setup, as commonly used in this type of testing (right).

Samples were left for one hour in saline before testing. Friction tests on depleted samples were performed under the same conditions as described above. Across all friction pairs, both the coefficient of friction and the real contact area increased over the course of the test. A total number of five tests were carried out using fresh samples for each experimental set-up with at least three repetitions per specimen pair, from which the mean and standard deviation were calculated.

IV. Results

The natural cartilage surface is capable of simultaneously adsorbing several PLs bilayer nanostructures formation under a friction load. The participation of bilayers in repulsive-slippage lubrication process is expressed by a very low friction coefficient and wear. The multi-lamellar structure of phospholipid bilayers is stabilized by repulsion between bilayers. The most important moment in the repulsive-slippage mechanism is the beginning of the simultaneous movement of all multi-bilayers. The beginning of all multi-bilayers affects wear-less movement, and this adaptive response underlies the effectiveness of joint lubrication. During smart-slippage

lubrication, opposing cartilage surfaces remain separated by hydrated phospholipid bilayers and do not make direct contact. In this study, we use the term "smart-slippage lubrication" to describe a boundary lubrication regime in which the phospholipid-based slip plane at the cartilage surface adapts to changes in pH, hydration and load. It combines the concept of a responsive ("smart") cartilage surface with lamellar and hydration-mediated slippage of phospholipid bilayers. The distance between bilayers is detected by repulsive equilibrium interactions with a distance of 4.5 nm [9].

Electron microscopy image of the articular surface shows amorphous layers (SAL) [9]. The multilamellar structure of phospholipids, namely SAL, covers the natural surface of articular cartilage. The molecular model [21] shows the protection of the cartilage surface with a hydrophilic bi-layer structure with strong adsorption and strong cohesion for the surfactant. Strong adsorption of PLs molecules by their quaternary ammonium positive ion [$(CH_3)_3N^+$–] to the negative cartilage surface (a proteoglycan) and strong cohesion between phosphate ions and calcium (II): (–PO_4^-–Ca–PO_4^-–) results in a close-packed solid layer [21]. Such covered cartilage surfaces had several bilayers. However, considering pH 7.4 condition and properties of phospholipids (PLs), highly self-organized bi-layers are formed and surface is negatively charged. We conclude that a very high porosity (75%) is a critical factor in providing excellent hydration lubrication properties of the articular cartilage.

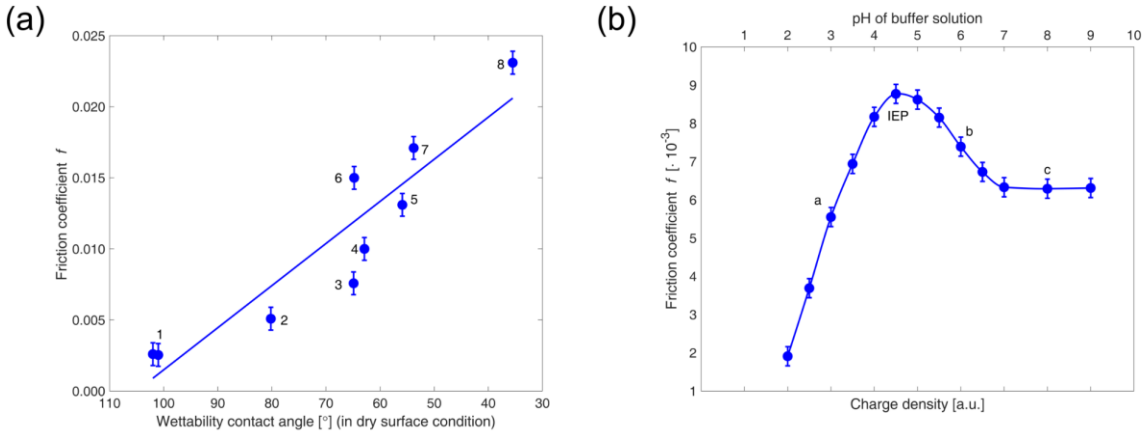

FIG. 4. Friction coefficient of (AC/AC) vs surface wettability (contact angle) (a) and charge density/pH (b). Figure 4a: Normal samples (1) and osteoarthritic samples (2-8). The wettability of cartilage surfaces was measured under air-dry conditions at room temperature but the friction coefficient was measured under wet surface conditions (see Experimental section). Figure 4b: Friction coefficient (f) vs. charge density/pH of bovine cartilage (AC) (obtained in the buffer solution). For isoelectric point IEP, molecule has equal charge distribution ($H_3N^+(CH_2)_nPO_4^-$–R_1R_2). The standard deviation (%) of the friction coefficient (f) ranges from 10 to 15. (Figure reproduced under the journal's Creative Commons license [19].)

Figure 4a shows the friction coefficient of (AC)/(AC) vs changes in wettability for natural healthy cartilage, naturally degenerated articular surface and lipid-depleted cartilage samples (θ°): (1) bovine cartilage surface 101° and 103° [10]; (2) knee 79.7° [13], (3) unhealthy cartilage 65° [22], (4) knee 63° [10], (5) degenerated hip 56.3° [10], and bovine samples for partially depleted (6, 7 this work) 63°, 53° and (8) completely depleted (this work) cartilage samples 35.1°. Samples with higher contact angles ($\theta \approx 80-100°$), corresponding to surfaces covered by intact phospholipid bilayers, exhibit the lowest friction coefficients ($f \approx 0.002-0.006$). Progressive reduction of θ, induced either by natural degeneration or lipid extraction, results in a monotonic increase in friction up to $f \approx 0.02-0.023$ for fully delipidated cartilage. This confirms that wettability alone does not govern frictional behavior; instead, it is the loss of

bilayer structure-and the associated disappearance of the lamellar-repulsive-slip plane-that leads to higher boundary friction.

Figure 4b shows a typical plot of friction test between two normal surfaces (AC/AC), which indicates that the friction on cartilage surfaces is largely dependent on the charge density/pH of the tissue. The friction coefficient initially increases (region a), reaching a maximum close to the isoelectric point, after which it decreases with further sliding (region b). A plateau with nearly constant friction is observed in region (c), corresponding to the pH range of 7-9. This bell-shaped dependence of friction on pH is consistent with earlier experiments on biomimetic phospholipid membranes under varying acid-base conditions, where the interfacial energy and friction coefficient were shown to change strongly around the isoelectric point of the phospholipids [24], [26].

Electrostatic surface potential of a molecule can depend on the pH value owing to changes in the protonation state of ionizable groups within the molecule. As the pH changes the protonation state of these ionizable groups altered, leading to changes in the overall charge distribution of the molecule. These changes modify the electrostatic interactions between the molecule and its environment.

To investigate the effect of pH on the electrostatic surface potential of a single phospholipid, dipalmitoylphosphatidylethanolamine (DPPE), molecular dynamics simulations were conducted using the AMBER14 force field [17]. The simulations were performed in water at a temperature of 310 K (37 °C). The ion concentration was maintained at 0.9% NaCl (physiological solution). The electrostatic surface potential was calculated using the Adaptive Poisson-Boltzmann Solver (APBS) method [1], which indicates weak interactions between PL molecules, leading to instability of the formed micelles, which are prone to disintegration under

external pressure. The ESP values are shown in Figure 5. The lowest value of surface potential was observed at pH corresponding to the isoelectric point (IEP) of the phospholipid.

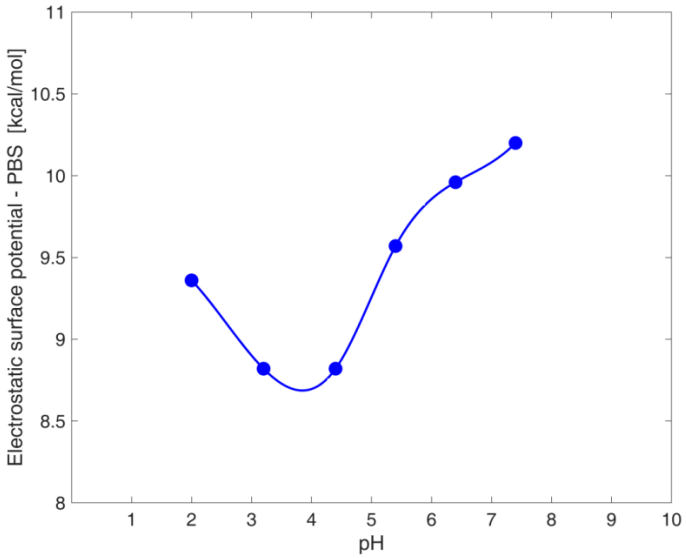

FIG. 5. Impact of pH on the electrostatic surface potential of phospholipids calculated using the Adaptive Poisson-Boltzmann Solver (APBS).

Consequently, this results in an increase in the friction coefficient, as confirmed experimentally and illustrated in Figure 4.

Table 1 summarizes the experimental and computational parameters relevant to boundary lubrication, including the pH-dependent friction response, electrostatic surface potential, and the relationship between contact angle and friction coefficient for native and delipidated cartilage.

Table 1. Quantitative summary of the relationships between friction, wettability, and electrostatic surface potential.

pH	Friction coefficient f	Electrostatic surface potential (kcal/mol)	Contact angle θ (°)	Friction coefficient f
2.0	0.0020	9.35	101.0	0.0026
2.5	0.0038	9.12	79.7	0.0054
3.0	0.0055	8.92	65.0	0.0078
3.5	0.0070	8.70	63.0	0.0096

4.0	0.0081	8.66	56.3	0.0146
4.5 (IEP)	0.0093	8.78	63.0	0.0152
5.0	0.0082	9.24	53.0	0.0176
5.5	0.0088	9.16	35.1	0.0230
6.0	0.0080	9.82		
6.5	0.0070	10.00		
7.0	0.0062	10.08		
8.0	0.0060	10.20		
9.0	0.0060	—		

V. Discussion

The cartilage surface carrying the positive charge, which changes to a negative, can be attributed to the proton transfer reaction. Equation (3) depicts a cartilage surface carrying positive charges. After IEP, while negative charges can be attributed to the proton transfer reaction [13].

$$^{-}NH_3^+ + OH^- \rightarrow {}^{-}NH_2 + H_2O \quad \text{and} \quad {}^{-}PO_4H + OH^- \rightarrow {}^{-}PO_4^- + H_2O \qquad (3)$$

In region (a) friction increase, pH 2 → 4.5: $–NH_3^+ \rightarrow –NH_2$ (surface losing charge). Maximum point, (IEP), pH 4.5, no net electrical charge:

$$H_2N(CH_2)_nPO_4H\,{}^{-}R_1R_2 \Leftrightarrow H_3N^+(CH_2)_n\,PO_4^{-}\,{}^{-}R_1R_2 \qquad (4)$$

In region (b), friction decrease, pH 4.5 → 6.8: $–PO_4H \rightarrow –PO_4^-$ (gaining negative charge). In region (c), constant friction, pH ~7 to 9.0 ($–PO_4^-$), surface is negatively charged.

However, the phospholipids, PLs, in disease a cartilage conditions change status from being active to non-active PLs with blocked phosphate groups (see deactivation Eq. 3). Osteoarthritic cartilage surface deterioration results in the interaction of enzymatically activated β_2-Glycoprotein I [22]. Interaction between the β_2-Glycoprotein I ($–NH_3^+$) group and the phospholipid, $–PO_4^-$ group: $(–NH_3^+) + (–PO_4^-) \rightarrow (–NH_3^+ – PO_4^-)$, is strong enough ($K_{assoc} \sim 10^5$)

[22], [23] to remove deactivated PLs molecules of bilayer surface, Figure 6. The deactivation process of phospholipid in osteoarthritic cartilage at pH ~7.4, Eq. (5):

$$\beta_2 - \text{Glycoprotein I} (-NH_3^+) + \text{Phospholipid} (-PO_4^-) \rightarrow \beta_2 - \text{GP I} (-NH_3^+)(-PO_4^-)\,\text{PL} \quad (5)$$

Deactivated PLs which are the main cartilage lubricants are unable to form a lamellar phase and liposomes and cannot support lubrication. The friction increases and mechanism in this case is the boundary friction mechanism without phospholipids support bilayers.

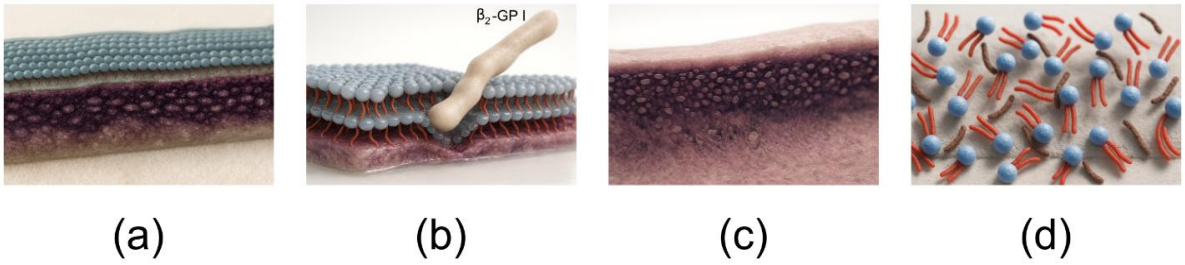

FIG. 6. (a) Healthy cartilage surface with phospholipid bilayers; (b) Degradation of phospholipid bilayers induced by the open conformation of β_2–Glycoprotein I (β_2–GP I); (c) Phospholipid bilayers degraded by the open hockey stick-like conformation β_2–GP I; (d) Phospholipids deactivated by β_2–GP I.

The uppermost layer of the hyaline articular cartilage has many names. This is called the surface amorphous layer (SAL), surface lamina or lamina splendens. The surface amorphous layer, SAL, has an extremely important articular function as it provides: (a) very high hydration of phospholipid bilayers, (b) at pH 7.4 negatively charged surface ($-PO_4^-$), (c) PLs molecules associated with lubricin-PL and hyaluronane-PL are adsorbed to the cartilage surface and support lubrication, (d) most importantly bilayers and lamellar PLs phases lubricate by boundary lamellar-repulsion-slippage mechanism.

SAL bilayers are likely to play a key role in maintaining the mechanical response of the articular cartilage to load. SAL is the first region of cartilage to degrade in OA where PLs are

deactivated PL($–PO_4^-$)($–NH_3^+$)β_2–GP I with no ability to restore the surface. Osteoarthritis is characterized by increase in the concentration of non-active PLs, about three times in synovial fluid was analytically proven [15].

In 2002, Hills [9] examined the natural articular cartilage surface using electron microscopy (EM). The EM imaging technique has sufficient resolution to study the SAL structure in the articular cartilage. SAL formation requires a negatively charged or highly hydrophilic surface.

The low friction and wear-less processes caused by boundary-repulsive-slippage and hydration lubrication mechanisms remain special for biological surfaces with SAL. The structure and composition of the SAL remains a less controversial layer after examining the natural articular cartilage surface by electron microscopy (EM) [9], [15], [30].

Cartilage surfaces have been frequently studied but standardized sample preparation procedures has not been performed. When cartilage samples were stored in formaldehyde, all PLs molecules were dissolved. Because of the charged surface the hydrated SAL provides effective boundary lubrication [9] which is referred to as the boundary lamellar-repulsive-slippage mechanism [20], [22].

At pH 7.4 articular cartilage with a high charge surface density and low contact angle wettability [22], [23] demonstrates that a smart-slippage mechanism exists that is capable to eliminate wear. The boundary repulsive-slippage mechanism was used to generating the load-carrying capacity of the hydrodynamic contact between two parallel plane surfaces. It is crucial that negatively charged ($–PO_4^-$) surfaces (lamellar/lamellar) phase separation where the lamellae slide over each other. The exceptionally low friction of healthy cartilage arises from an adaptive interfacial structure capable of reorganizing under load. The superficial region contains self-assembled phospholipid layers (see Figure 1) that retain fluid-like characteristics, enabling

controlled separation and sliding between opposing surfaces. Disruption of this architecture compromises the tissue's ability to maintain such efficient interfacial motion.

Bilayer structure surfaces with high surface charges at pH 7.4 exhibit very low friction, consistent with adaptive ('smart-slippage') lubrication. In osteoarthritic conditions, PLs bilayers are disintegrated, and repulsive-slippage lubrication transitions to a high-friction regime [2], [12], [13], [33].

A limitation of the present experimental design is that the T-11 tribometer produces sliding under constant nominal contact pressure. In contrast, synovial joints operate under predominantly reciprocating motion with varying load and cyclic recovery of interstitial fluid. As a result, our measurements emphasize boundary-dominated lubrication and likely underestimate hydrodynamic and poroelastic contributions that may arise during physiological gait. The absolute values of the friction coefficient reported here should therefore be interpreted as representative of a controlled boundary regime at the cartilage-cartilage interface, rather than as a full surrogate for in vivo joint mechanics.

VI. Conclusions

In contrast to classical engineering materials, articular cartilage exhibits an inverse relationship between wettability and friction. Higher contact angles indicate the presence of intact phospholipid bilayers, whose multilamellar organization and hydration-mediated repulsive interactions create a stable slippage plane that enables ultra-low friction. As these bilayers are progressively degraded, either through osteoarthritic processes or experimental delipidization, the surface becomes more hydrophilic, yet the lamellar-slippage mechanism collapses. As a result, hydration-repulsion no longer maintains separation between the lamellae. The multilamellar

structure becomes mechanically unstable, and friction rises even though the surface appears more wettable. Friction measurements at different stages of bilayer depletion demonstrate that the integrity of the phospholipid multilayer, rather than wettability alone, dictates the transition from low-friction repulsive slippage to high-friction boundary contact.

Loss of the multilamellar structure alters both the wetting behavior and the chemical state of surface phospholipids. Upon degradation, negatively charged headgroups ($–PO_4^-$) become incorporated into β_2-GPI complexes (β_2-GPI–NH_3^+/–PO_4^-–PL), rendering them ineffective as boundary lubricants. These deactivated species were also detected in the surrounding synovial fluid.

The study highlights a naturally occurring repulsive-slippage lubrication mechanism that may inspire biomimetic strategies for low-friction surface engineering. Ultimately, the repulsive-slippage performance depends on the number and integrity of PL bilayers, underscoring the "smart" and adaptive nature of the cartilage surface.

These findings indicate that the molecular-scale organization of phospholipid bilayers governs the macroscopic frictional response of cartilage. A comprehensive understanding of this behavior therefore requires extending the description beyond classical tribological models toward a framework that also accounts for quantum-level interactions. We emphasize that the role of nuclear quantum effects in joint lubrication discussed here is, at present, predominantly conceptual and based on analogies with well-characterized aqueous and interfacial systems. Direct experimental or simulation evidence at cartilage interfaces is still lacking, and our interpretation should therefore be seen as a hypothesis that complements, rather than replaces, classical lamellar-repulsive lubrication models.

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AUTHOR DECLARATIONS

Conflicts of Interest

The authors have no conflict of interest.

Ethics Approval

Ethical approval is not required.

Author Contributions

Zenon Pawlak: Conceptualization (equal); Data curation (equal); Methodology (equal); Writing - original draft (equal). **Jacek Siódmiak:** Writing - review & editing (equal); Data curation (equal); Investigation (equal); Software; Visualization (equal).

DATA AVAILABILITY

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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