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Cartilage Wear in Healthy and Osteoarthritis Joints

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Abstract

This study is designed to determine whether the outermost layer of articular cartilage is deficient in Osteoarthritis (OA). Phospholipids present in healthy and osteoarthritis (OA) synovial fluid show significant differences in their concentration. While examining the surface properties of OA joints, we found that OA PLs molecules cannot support lubrication, and increased friction was observed. Our lubrication mechanism was based on a surface active phospholipids (SAPL) multibilayer which in OA condition was deactivated and removed from the cartilage surface under OA conditions. Cartilage wettability study clearly demonstrated a significant decrease in hydrophobicity, the contact angle, θ (theta), dropping from 103˚ from bovine healthy cartilage to 65˚ in surface partially depleted and 35.1˚ for completely depleted surface. These results are discussed in the context that surface active phospholipid (SAPL) and lubricin, each has specific roles in a lamellar-repulsive lubrication system. However, deactivated phospholipid molecules are major indicator of cartilage wear (model) introduced in this study.

Keywords

Cartilage Surface, Friction (Cartilage/Cartilage), Osteoarthritis (OA), Surface Active Phospholipids (SAPL)/or Surface Amorphous Layer (SAL), Deactivated Phospholipids, Wear

1. Introduction

The articular cartilage is a few millimeters thick and covers the surfaces of the ends of bones in the knee joint. Its function is to make the joint surfaces very smooth and have low friction to allow smooth joint movements without wear and tear. The knee joint is one of the most common causes of damage owing to

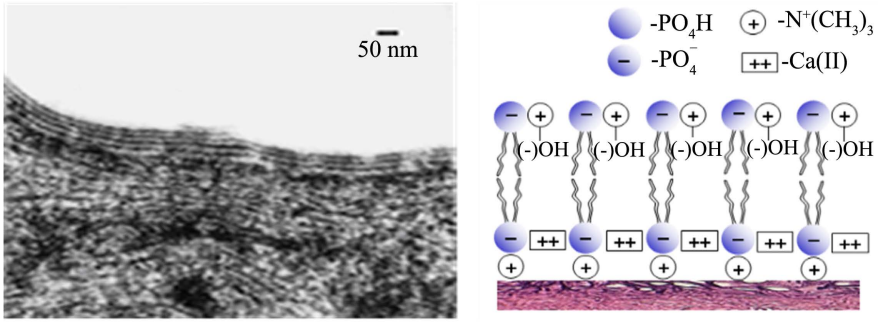
wear and tear (degeneration). If the articular cartilage wears away completely, the bone rubs. Osteoarthritis is often referred to as wear and tear disease. Therefore, if the tissue is damaged, it cannot repair itself or regenerate and grow. Wear and tear on joints can lead to inflammation, breakdown of cartilage and development of osteoarthritis. The friction coefficient significantly increased above the typical value achieved under a normal joint function. Loss of articular cartilage is often the first stage of osteoarthritis [1] [2] [3].

In patients with osteoarthritis (OA), lubricin showed deficiencies in preventing damage to the articular cartilage and was found to be ineffective in reducing friction in arthritic cartilage [4] [5]. The wear of the (cartilage/cartilage) sliding under healthy conditions, which is expected to be in the boundary regime, is almost invisible. Osteoarthritis and inflammation of the human knee are the main causes of wear [6] [7] [8]. Synovial fluid from human joints that have experienced serious traumatic injury and is correlated with joint osteoarthritis disease status has been shown to have two to three times higher concentrations of phospholipids than healthy joints [5]. The insoluble deactivated phospholipids molecules from the cartilage surface to the synovial fluid begin the formation of wear particles.

Change is needed in osteoarthritis medication that addresses the root cause of cartilage damage and depletion. The nuclear receptor RORβ represents a novel therapeutic target to protect cartilage from damage and perhaps turn on cartilage regeneration. RORβ, short for "retinoic acid receptor-related orphan receptor beta," is a nuclear receptor protein. The specific protein RORβ, which manages activities within chondrocytes, is a critical cell type that maintains healthy joint cartilage [9].

The hydrophilic surface of the articular cartilage is attracted to multibilayers of phospholipids to generate a membrane (**Figure 1**). The amphoteric "smart surface" of articular cartilage is covered by bilayers of phospholipids and has ~0˚ wettability when is wet (hydrophilic) and 104˚ wettability when the surface is air-dry and turn hydrophobic [2] [3]. Measurement of the contact angle θ (theta), demonstrated a highly significant decrease in hydrophobicity from 104˚ to 60˚ for the "worn" of arthritic knees [10]. According to Hills and others, the surface amorphous layer (SAL) of cartilage contains surface-active phospholipids: phosphatidylcholines (over 40%) sphingomyelin (~30%) and phosphatidylethanolamines (~30%) which were subsequently identified in the SAL [11]. We observed the importance of surface-active PLs in the lubrication process in the presence of hyaluronan and lubricin macromolecules, osteoarthritis lipids did not appear to further reduce the synergistic reduction in friction by lubricin and hyaluronate.

The objective of this study was to introduce a wear model based on the deactivation of the lubricating layer of SAPL adsorbed to the articular surface, but in OA condition worn-out PLs increased concentration in synovial fluid. In this study, we hypothesized that accelerated wear occurs when phospholipid multibilayers are worn away.



(a) (b)

Figure 1. (a) Microscopic images of the multilamellar lining of adsorbed bilayers of phospholipid of the articular cartilage surface of a human knee [2] [3], and (b) The hydrophilic model of cartilage surface.

2. Material and Methods

Articular cartilage samples used in the atomic force microscopy (AFM) study were obtained from the patellae of 3 - 4 years old bovine animals. The glued sample (5 mm × 5 mm) was submerged in saline solution for AFM imaging using the SMENA® head of the NT-MDT P47 Solver scanning probe microscope (SPM) (NT-MDT). An artificial lipid extraction process (delipidization) was used to simulate the loss of cartilage surface lipids [12].

Articular cartilage samples used determination the phospholipid concentration studies were obtained from humans [4] [5]. Lipids were extracted from cell-free and cell-debris-free SF samples from controls, early- and late-OA. The extracted phospholipid species were quantified by electrospray ionization tandem mass spectrometry (ESI-MS/MS). Most PLs data in this study were adapted from Kosinska *et al.* [4] [5].

Tensiometer. The contact angle between the liquid and the tested cartilage was measured using a KSV CAM100 tensiometer and was between a droplet of a 0.15 M saline solution and a given air-dry cartilage surface. The contact angle test was performed on the normal, partial and completely depleted cartilage samples. Five tests were performed on each specimen and each set-up for surfaces in air-dry condition.

3. Results and Discussion

Our wettability studies demonstrated an important parameter in the characterization of biological surfaces. The wettability surface of cartilage depends on the number of PLs bilayers adsorbed as a solid lubricant; this hypothesis was tested with normal and osteoarthritis (or depleted) cartilage samples. The contact angle, θ, dropped from 103˚ for bovine healthy cartilage to 65˚ in surface partially depleted and 35.1˚ for completely depleted surface. Osteoarthritis surface cartilage deterioration results in the interaction of enzymatically activated β_2-Glycoprotein-I, β_2-GP-I ($-NH_3^+$) [13] [14]. Interaction of the β_2-Glycoprotein-I functional ($-NH_3^+$) group and the phospholipid functional ($-PO_4^-$) group:

($-NH_3^+$) + ($-PO_4^-$ → ($-NH_3^+$ $-PO_4^-$) interaction is strong enough to remove PLs molecules of cartilage surface.

Deactivation process of phospholipid at pH ~ 7.4:

β_2-Glycoprotein-I ($-NH_3^+$) + Phospholipid ($-PO_4^-$) → β_2-GPI($-NH_3^+$)($-PO_4^-$)PL, K_{assoc} ~10^5

Local surface disorganization involves splitting the SAL of the cartilage (**Figure 2**). Continued deterioration of the articular cartilage leads to exposure of the subchondral bone as shown in **Figure 2(b)**. Charged phospholipid bilayers ($-PO_4^-$) protect and maintain intact cartilage surfaces and affect the articular joint lubrication. Chemical interaction between PLs and hydrophilic collagen type II is responsible for its ability to coat articular cartilage with PLs bilayers (**Figure 1(b)**). In osteoarthritis joints, the polar part of the lubricin molecules is not covered by deactivated PLs, and the cartilage surface is naked (**Figure 2(b)**). The surface amorphous layer (SAL), the topmost layer of the cartilage surface was absent. The osteoarthritic deactivated lipids did not appear to further reduce the synergistic reduction in friction caused by lubricin. In patients with osteoarthritis (OA), lubricin is deficient in preventing damage to articular cartilage. Interestingly, lubricin has been found to be ineffective in reducing friction in osteoarthritis articular cartilage [3]. Wear of the articular cartilage is an important symptom of OA [15], and the severity of wear is often used for OA assessment. As OA progresses, proteoglycan starts to deplete, followed by the degradation of collagen II, which results in mechanical failure and ultimately complete erosion of the articular cartilage [16].

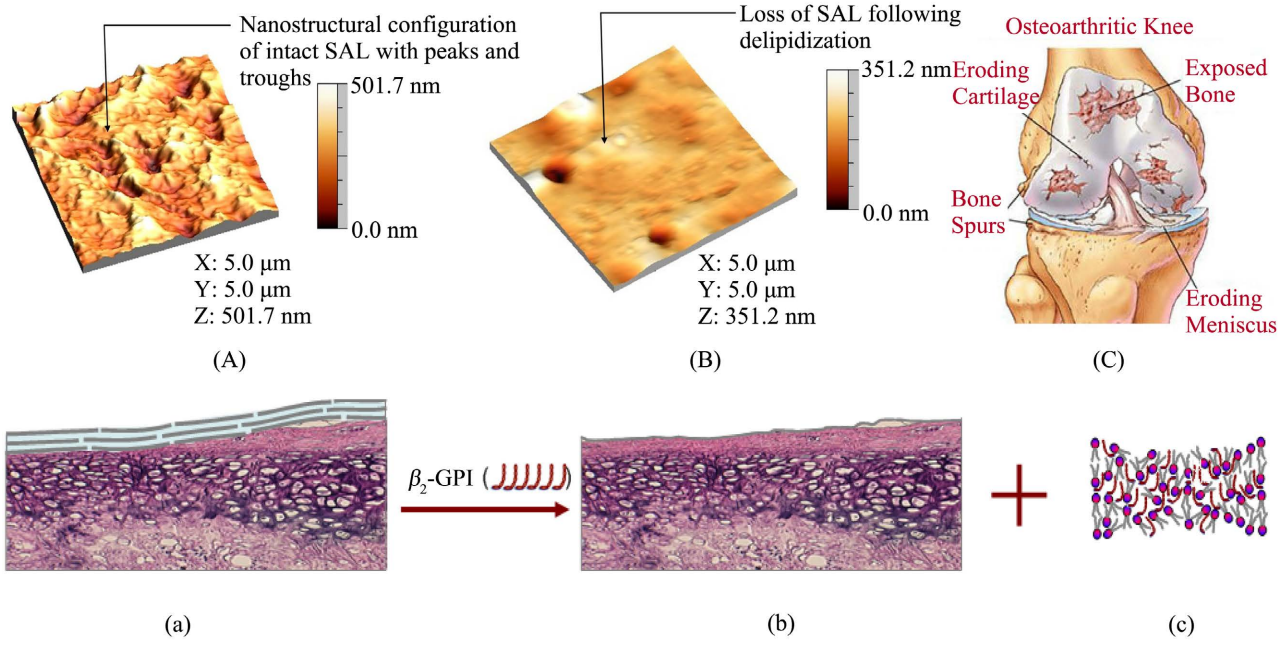


Figure 2. (A) 3D topographical image from atomic force microscopy (AFM) of a normal healthy cartilage surface (103˚ wettability), (B) osteoarthritic cartilage surface (35.1˚ wettability), and (C) knee osteoarthritis. (a) Bilayers of PLs on the cartilage surface, (b) cartilage with degraded surface, and (c) Nanoparticles of deactivated phospholipids (β_2-GPI($-NH_3^+$)($-PO_4^-$)-PL).

Nanoparticles of deactivated phospholipids (β_2-GPI(-NH_3^+)(-PO_4^-)-PL) were extracted from synovial fluid using ferrography and analyzed using field-emission scanning electron microscope (FESEM) and an environmental scanning electron microscope (ESEM) [8]. The particle images were numerically analyzed for shape parameters such as the boundary fractal dimension, shape factor, and area. Wear particles in healthy knee joints are usually small, smooth, and have a simple structure. However, in advanced osteoarthritis, wear particles are chunky, rough, and complex in structure. Irregularly shaped and chunky wear particles have been observed in knee joints with late OA [17] [18].

4. Conclusion

This study presents the results of cartilage surface damage caused by osteoarthritis (OA) via the PLs deactivation mechanism. A deactivated PL molecule has no ability to form liposomes and bilayers or be adsorbed by lubricin molecules, and this process exhibits symptoms of an antiphospholipid syndrome. These results extend our current knowledge of cartilage boundary lubricating molecules. Osteoarthritis lipids did not appear to further reduce the synergistic reduction in friction caused by lubricin. In patients with OA, lubricin showed deficiencies in preventing damage to the articular cartilage and was found to be ineffective in reducing friction in arthritic cartilage. Under osteoarthritic conditions, the immune system produces antibodies that attack phospholipids and produces insoluble macromolecules that aggregate the synovial fluid. In antiphospholipid syndrome, molecules of phospholipids (PLs) and enzymatically activated (β_2-Glycoprotein I) (β_2-GPI) form insoluble compounds. The deactivation mechanism is simply the interaction of the β_2-Glycoprotein-I (-NH_3^+) functional group and the phospholipid (-PO_4^-) functional group: (-NH_3^+) + (-PO_4^-) → (-NH_3^+ -PO_4^-) with an association constant, K_{assoc} 10^5. The interaction was strong enough to remove the PLs molecules from the cartilage surface. Deactivated PLs molecules in osteoarthritis joints increased friction (over 40%) and increased wear by about 7% [19] [20].

Conflicts of Interest

The authors declare no conflict of interest regarding the publication of this paper.

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