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The Phospholipids Activity in Normal and Osteoarthritic Cartilage

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Abstract

The purpose of this paper is to study Phospholipids (PLs) in osteoporotic samples (OA) changes in Synovial Fluid (SF). The (PLs) in (SF) were measured for normal samples, with early and late stages of (OA). During osteoarthritis, enzymatically activated β_2-Glycoprotein I is transformed into antibody conformation. Cartilage degradation of PL bilayers by antibodies (β_2-Glycoprotein I) is considered Anti Phospholipid Syndrome (APS). Deactivated PL molecule has no ability to form bilayers and liposomes. The phospholipid content in Synovial Fluid (SF) in osteoporotic samples is significantly higher (2-3 times) above the normal concentration of PL and has a poor boundary-lubricating ability.

Kewords: Cartilage; Deactivated phospholipid; Osteoarthritis; β_2-Glycoprotein I; Anti Phospholipid Syndrome (APS)

Introduction

A general model of cartilage boundary lubrication has been proposed and is based on a layer of biomolecules that covers the surfaces of articular cartilage and acts as a lubricant [1,2]. Lubricating molecules of Synovial Fluid (SF), such as hyaluronan (A^-) [1], lubricin [3], and surface-active phospholipids [4], adsorb to the surface of articular cartilage and support boundary lubrication [2]. However, according to Hills, PLs is the most important SF component in maintaining efficient joint lubrication [5]. In PL bilayer model the macromolecules (hyaluronan and lubricine) of SF play supportive role. The phospholipid bilayers on the surface of articular cartilage are a main lubricant and during joint inflammation and OA are destroyed. Unexpected elimination of self-organized of PLs in SF is an indication of joint deterioration. A joint disease named Osteoarthritis (OA) or degenerative arthritis is caused by the damage to the cartilage surface tissue in the joint and causes pain and stiffness. Rheumatoid Arthritis (RA) is an autoimmune disease with signs and symptoms that include joint swelling, pain, prolonged morning joint stiffness, fatigue, muscle atrophy, and joint erosions [5]. Osteoarthritis (OA) is a common disease, where the mechanical integrity of articular cartilage is weakened. Two steps occur on surface destruction (a) the first is depletion of phospholipid bilayers and (b) second is proteoglycan loss of damaged cartilage matrix by injurious loading. From our experiment we can predict proteoglycan loss of injured cartilage by decreasing the Fixed Charge Density (FCD) concentration. Electrostatic lubrication of osteoarthritic cartilage is operated by decreasing the Fixed Charge Density (FCD) concentration on surface. This is consistent with experimental findings in the literature. Level of PLs in SF shows a great potential for predicting the progression of (OA) *via* analytical evaluation content of PLs.

Our objective was to evaluate the changes of phospholipid in synovial fluid of cartilages on early (eOA), and late (lOA) stage of osteoarthritic and with Rheumatoid Arthritis (RA) disease. In this work PLs concentration were measured on healthy and pathological joints surfaces.

In this study, we tried to identify degradation bilayers of PLs (surface of AC) as well as deactivation of phospholipid molecules in the Synovial Fluid (SF) from samples either with active Rheumatoid Arthritis (RA) or with early or late stages of Osteoarthritis (OA).

Materials and Methods

Mass spectrometr of phospholipids: The phospholipid species were quantified by ESI-MS/MS on Micromass [6,7]. The research was carried out using synovial fluid derived from undamaged controls and patients with early and late osteoarthritis and rheumatoid arthritis. The authors classified 130 species of lipids. After comparing control synovial fluids, SF of patient with early and late OA had higher levels of most PLs species. Most of the PL data for this paper was taken from Kosinska et al [7].

Results

Hyaluronan, lubricin, and phospholipids contribute to cartilage boundary lubrication that is provided by SF. PL is the most important SF component in maintaining efficient joint lubrication. Phospholipids are believed to cover the surface of articular cartilage, where they form self-organized bilayers of phospholipids, (Figure1A) [4,5] and provide supportive function of hyaluronan and lubricin aqueous hydration in healthy cartilage boundary lubrication. The concentration level of phospholipids increased in SF with early- and late stage OA, and patients with active RA is given in [5] (Figure 1B). Compared to synovial fluid from controls, SF from patients with early (eOA) and those with late (lOA) had higher levels of most PLs species (2 to 3 times) above the normal range (Figure 1B).

Phospholipids are believed to cover the surface of articular cartilage, where they by self-organization form bilayers and contributing to cartilage boundary lubrication [4,5]. Also, the adsorption of PLs by hyaluronan and lubricin and their supportive functions in cartilage boundary lubrication remain accepted. In our

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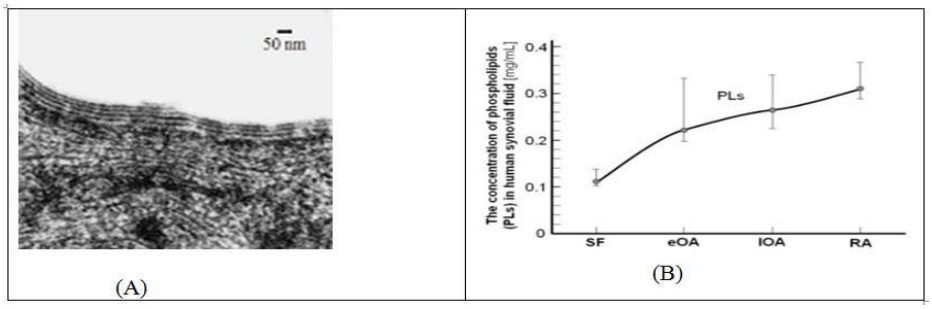

Figure 1: (A) An electron microscopy image of the articular cartilage surface of a human knee demonstrating the oligolamellar lining consisting of phospholipid bilayers. The bar represents 50nm [8,9]. **(B)** Concentration of Phospholipid (PLs) in osteoarthritic Synovial Fluid (SF): controls samples (SF), with early OA (eOA), and late OA (lOA) and (PLs) from Rheumatoid Arthritis (RA) disease.

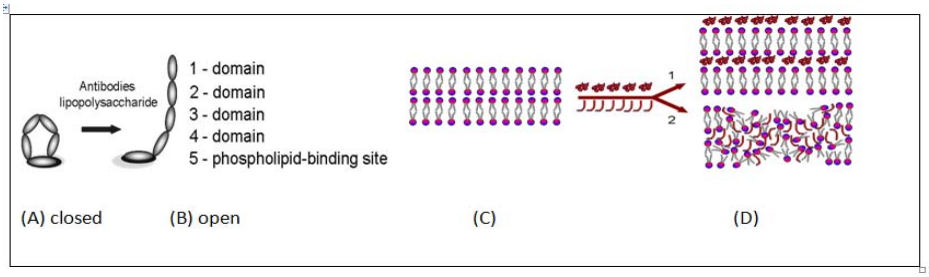

Figure 2: Conversion β_2-Glycoprotein I in closed form (**A**); into an open hockey-stick-like conformation (**B**); each molecule has five domains (1-5). The antibody-binding site (**B5**) is accessible to the autoantibodies. Phospholipid bilayer **(C);** Bilayer and closed (β_2-Glycoprotein I) (**D1**); Deactivated PLs molecules by open hockey-stick (β_2-Glycoprotein I) (**D2**).

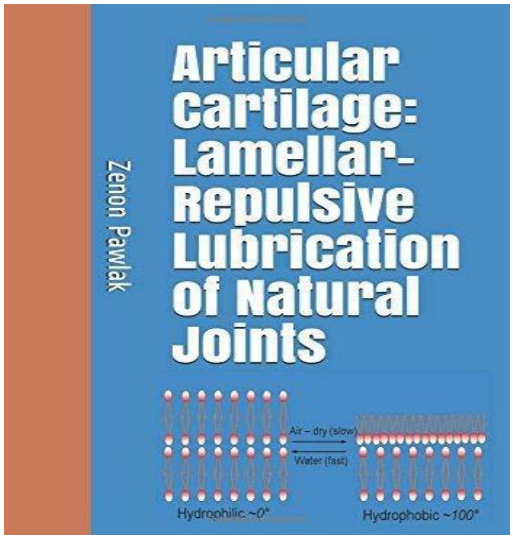

Figure 3: Phospholipidic bilayers of articular cartilage under the wet and air-dry conditions. Variations in surface energy lead to conformational transformations in the surface phospholipids from bilayer (hydrophilic) to monolayer (hydrophobic). Book cover "Articular cartilage: Lamellar-repulsive lubrication of natural joints" [9].

studies a model that was developed by Hills claims that a multilayered PLs membrane-like structure adheres to the surface of articular cartilage was confirmed in our studies [9].

The (β_2-Glycoprotein I)-binding site in domain 5 (Figure 2) contains 326 positively charged amino acids group ($-NH_3^+$). In plasma, it occurs as a closed circular protein in which domain 1 interacts with domain 5. At a pH around 7 amino acids (arginine, lysine, and tryptophan) are positively charged ($-NH_3^+$) an acid-base interaction occurs between the protonated amino acid group ($-NH_3^+$) and the phosphate function group ($-PO_4^-$):

$$PL\text{-}(-PO_4^-) + \beta_2\text{-GPI}(-NH_3^+) \rightarrow \beta_2\text{-GPI}(-NH_3^+)\ (-PO_4^-)\ PL) \quad K_{assoc} \sim 10^5 \quad (1)$$

Electrostatic attractions are strong enough to destroy PLs bilayer on the cartilage surface and deactivate all phospholipids in SF, when association constant has high value, $K_{assoc} \sim 10^5$.

The open hockey stick-like conformation when β_2-GP I is complexed to negatively charged phospholipids ($-PO_4^-$) resulting in the destruction of bilayers and deactivation of phospholipid molecules (D2). B_2-Glycoprotein I, B_2-(-GPI) (MW of 50kDa) circulates in the body and autoimmune disease transforms B_2-GPI in antibody [10,11]. B_2-GPI participates in the Antiphospholipid Antibody Syndrome (APS) through binding of β_2-GPI to the anionic charged phospholipid ($-PO_4^-$) functional group.

Conclusion

Our study has provided insight into composition of SF samples, healthy, with early and late-stage OA and with RA. In conclusion, we have showed that deactivation of PLs by β_2-GPI is believed to totally destroy the cartilage boundary lubrication function of SF and deactivate β_2-GPI($-NH_3^+$) ($-PO_4^-$)PL) and has a poor boundary-lubricating ability. PLs are the foundation in joints boundary lubrication (Figure 3). Hyaluronan and lubricin are unable to take function of lubricant without active phospholipids presence.

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