International Journal of Research Studies in Science, Engineering and Technology
Volume 7, Issue 11, 2020, PP 1-4
ISSN 2349-476X

Antiphospholipid Syndrome in Osteoarthriticarticular Cartilage

Pawlak Z.[1*] Karpinska B.[2]

[1]Tribochemistry Consulting, Salt Lake City, UT 84117, USA and University of Economy, Biotribology Laboratory, Garbary 2, 85-229 Bydgoszcz, PL
[2]University of Economy, Garbary 2, 85-229 Bydgoszcz, PL

***Corresponding Author:** Pawlak Z, Tribochemistry Consulting, Salt Lake City, UT 84117, USA and University of Economy, Biotribology Laboratory, Garbary 2, 85-229 Bydgoszcz, PL*

ABSTRACT

In osteoarthritic condition our immune system makes antibodies that attack phospholipids and produces insoluble macromolecules in blood and joints. In antiphospholipid syndrome molecules of phospholipids(PLs) and enzymatically activated (β2-Glycoprotein I (β2-GPI)formsinsoluble compound. In our blood system veins are clottedand phospholipidsinsynovial fluidare deactivated to be ineffective in reducing friction in arthritic articular cartilage Deactivation mechanism is simply interaction of β2-Glycoprotein I (-NH3+) group and the phospholipid (–PO4-) group: (-NH3+) + (–PO4-) → (-NH3+ -PO4), Association constant, K ~105.Interaction is strong enough to remove PLs molecules from bilayer surface. We examined a bovine cartilage (BC) surface using atomic force microscope.

Keywords: *Cartilage surface; Antiphospholipid syndrome; β2-GlycoproteinI; Atomic force microscopy (AFM); Osteoarthritis.*

INTRODUCTION

Osteoarthritis (OA) is a disease characterized by the destruction of articular cartilage surface as a result of deactivation phospholipid (PLs) showed by increasing friction in joints and increased concentration non-active PLs almost 3 times. This can be related to degradation of the phospholipid bilayers system from surface of articular cartilage. Current OA treatment aim to reduce pain but do not stop the progression of the disease. Phospholipids as boundary lubricant is highly self-organized biomolecules in aqueous media, and their structure allows them to form spontaneously vesicles, lamellar phases, and surface membrane (Fig. 1). The multilamellar structure of phospholipids, namely the surface amorphous layer (SAL), covers the natural surface of articular cartilage found in diarthrodial joints [1, 2].When phospholipid exposed to the water the molecules bilayer spontaneously self-assembles. The phospholipid bilayer on the surface of articular cartilage is a main lubricant and is supported by synovial fluid with the macromolecules hyaluronan, lubricin and phospholipid micelles. Unexpected deactivation of self-organized of PLs in SF is an indication of joint disease, namely osteoarthritis.

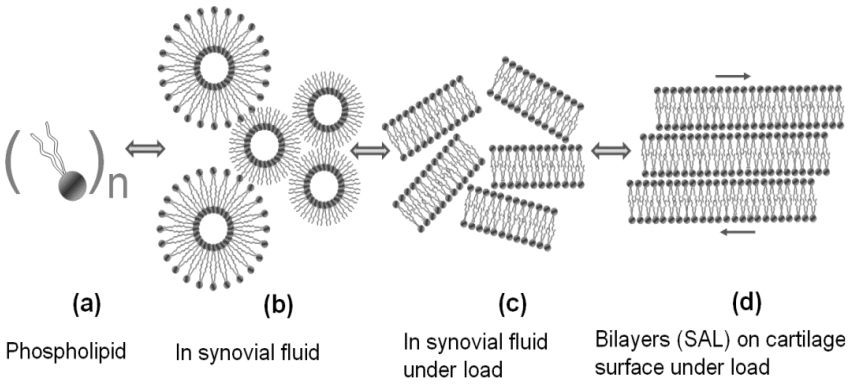


Figure1(a). *Phospholipids, (b) Liposomes and hexagonal phases, (c) Lamellar phases in synovial fluid and (d) lamellar slippage of phospholipid bilayers on the surface of cartilage under load*

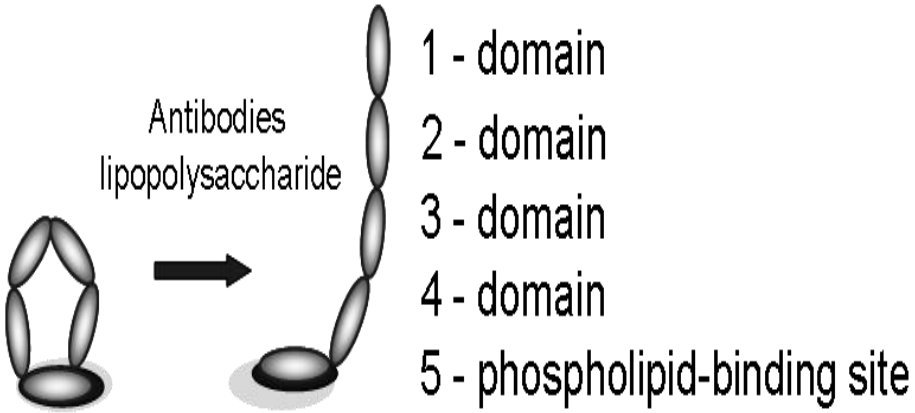
A joint disease named osteoarthritis (OA) or degenerative arthritis iscaused by the damage to the cartilage surface tissue in the joint and SF. In non-osteoarthritic synovial fluid PLs form lamellar phases [1-3] and these structures would interact with the surface of articular cartilage. In osteoarthritic joints we can expect disintegration of self-assembled structures without ability to support lubrication.

Antiphospholipid syndrome occurs when our immune system unexpectedly creates antibodies (β2- GP I) that deactivate PLs on cartilage surface and in synovial fluid (Fig. 2). There's no cure for phospholipid syndrome in our joints. Osteoarthritis is a severe disease start by joint inflammation, surface destruction and synovial fluid deterioration. Deactivated PLs molecules have no ability to participate in lubrication process, no micelles formation, no bilayer formation. Unexpectedly, lubricin was found to be ineffective in reducing friction in arthritic articular cartilage [4].

In this paper, we study the articular surface of natural bovine knee cartilage normal and osteoarthritic samples. To explain PLs increase concentration 2-3 times in synovial fluid we study a bovine cartilage (BC) surface normal and deteriorated using atomic force microscope. We introduced the mechanism of osteoarthritic joint disease and destruction of phospholipid bilayer. The goal of this paper is to show our understanding of mammalian joint lubrication systems, particularly how phospholipid is a major contributor to joint lubrication in OA condition.

Material And Methods

The articular cartilage samples used in this study were obtained from the patellae of 3-4 year old bovine animals harvested from the local abattoir and stored at -20oC until required for testing. The glued sample was submerged in saline solution ready for AFM imaging using the SMENA® head of the NT-MDT P47 Solver scanning probe microscope (SPM) (NT-MDT. The sample preparation and the surface imaging were done using methods described elsewhere in the literature [5]. Phospholipid species were quantified by electrospray-ionization-tandem-mass-spectrometry[6, 7].



*(**A**) Closed molecule* *(**B**) Open hockey-stick molecules with five domains*

Figure 2(A). *β_2-GlycoproteinI molecule in closed form, (**B**) An open hockey stick-like conformation*

Results And Discussion

Molecules of β2-Glycoprotein I, (β2-GPI) (MW of 50 kDa) circulate in the body and autoimmune disease transforms β2-GPI in an antibody, Fig. 2 [8, 9]. The β2-GPI participates in antiphospholipid antibody syndrome (APS) through binding of β2-GPI to the anionic charged phospholipid (-PO4-) group. AtapH around 7, β2-GPI domain 5 has - amino acids (arginine, lysine and tryptophan) are positively charged (-NH3+)and acid-base interaction occurs between the protonated amino acid group (-NH3+) and the phosphate (–PO4-) membrane group: (β2-GPI-NH3+) + (PLs–PO4-) → (-NH3+-PO4-).Interaction and electrostatic attractions is strong enough to destroy the PLs bilayerson the articular surface and deactivate all phospholipids in the synovial fluid (SF). Under the osteoarthritic conditions the phospholipids in the solutions can be deactivated by β2-GPI as well (Fig. 3 A, B) [2]. 3D topographical image of (Fig. 3C) normal healthy cartilage showing the nonstructural arrangement of the surface amorphous layer with several peaks and troughs, (Fig. 3D) showing the loss of the membranous overly (SAL) from surface.

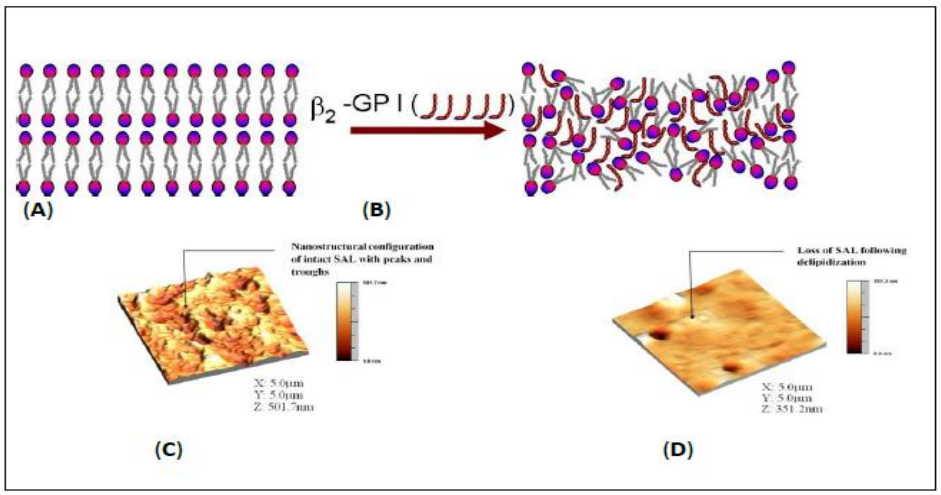


Figure3(A). *Bilayers of phospholipids and the open hockey stick-like conformationβ_2-Glycoprotein I (β_2-GP I), (**B**) (β_2-GPI) moleculesbinding to negatively charged phospholipids. (**C**) Topographical image from atomic force microscopy (AFM) imaging of normal healthy cartilage surface, (**D**) osteoarthritic cartilage surface*

The surface amorphous layer (SAL) is the topmost layer of articular cartilage often in repeated contact can lead to early stages of joint degeneration like osteoarthritis leading to low quality of life in affected patients. Theosteoarthritic surface cartilage deterioration results in the interaction of enzymatically activated β2-Glycoprotein I. Interaction of β2-Glycoprotein I (-NH3+) group and the phospholipid (–PO4-) group: (-NH3+) + (–PO4-) → (-NH3+ -PO4-) interaction is strong enough to remove PLs molecules of bilayer surface.

Deactivation Process of Phospholipid at Ph ~ 7.4

β2-Glycoprotein I (-NH3+) + Phospholipid (–PO4-) → (β2-GP I(-NH3+)(-PO4-) PL, Kassoc ~105

A local surface disorganization involving splitting of the SAL of the cartilage, Fig. 4.Continued deterioration of articular cartilage leads to an exposure of the subchondral bone.

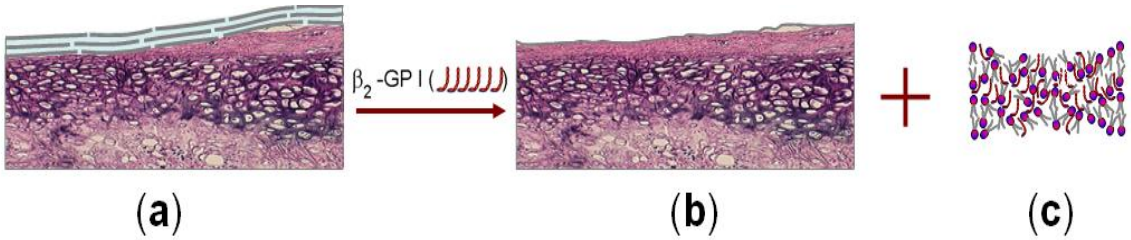


Figure4(a). *Bilayers of PLs on cartilage surface, (**b**) Cartilage with degraded surface, (**c**) Deactivated molecules of phospholipid by (β_2-GPI)*

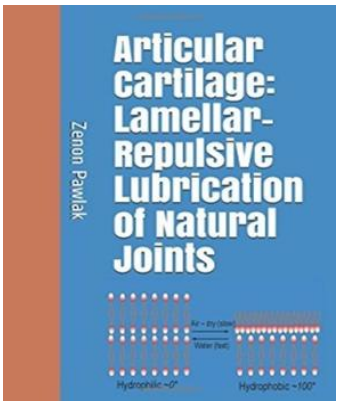


Figure5. *Smart surface of articular cartilage under the wet (hydrophilic) and air-dry conditions (hydrophobic).Book cover "Articular Cartilage: Lamellar-Repulsive Lubrication of Natural Joints" [**2**].*

Charged phospholipidsbi layers (–PO4-) protect and maintain intact cartilage surfaces and affect articular joint lubrication. A chemical interaction between PLs and hydrophilic collagen type II is responsible for its ability to coat articular cartilage by PLs bilayers [2]. Variations in surface energy lead to conformational transformations in the surface phospholipids from bilayer (hydrophilic) to monolayer (hydrophobic) [1, 2],Fig.5.In osteoarthritic joints lubricin molecules a polar part is not cover by deactivated PLs and the cartilage surface is naked (Fig. 4 B). The surface amorphous layer (SAL) the topmost layer of cartilage surface is not present. Osteoarthritic lipids do not appear to further reduce the synergistic reduction in friction by lubricin and hyaluronate. In patients with osteoarthritis (OA) lubricin showed deficiencies to prevent damage to articular cartilage. Interestingly, lubricin was found to be ineffective in reducing friction in osteoarthritic articular cartilage.

CONCLUSION

In conclusion, we found importance of surface active PLs in lubrication process in presence of macromolecules hyaluronan and lubricin. While examining the surface properties of the OA joints, we found that both molecules cannot support lubrication. In healthy joints, lubricin molecules coat the cartilage surface, providing support for boundary lubrication and preventing cell and protein adhesion. Osteoarthritic lipids do not appear to further reduce the synergistic reduction in friction by lubricin and hyaluronate. In patients with osteoarthritis (OA) lubricin showed deficiencies to prevent damage to articular cartilage, was found to be ineffective in reducing friction in arthritic cartilage.

REFERENCES

[1] SchwarzI. M. Hills BA. Synovial surfactant: lamellar bodies in type B synoviocytes and proteolipid in synovial fluid and the articular lining. Br. J. Rheumatol. 35 (1996), 821-827

[2] Pawlak Z. Articular Cartilage: Lamellar-Repulsive Lubrication of Natural Joints, Kindle Direct Publishing, 161pp. Print-book: https://www.amazon.com/dp/B07B42P1JY, e-book: https://www.amazon.com/dp/ 1976760283(2018)

[3] Hills B.A. Surface-active phospholipid: a Pandora's box of clinical applications. Part I. The lung and air spaces.Intern. Med. J. 32 (2002), 170-178

[4] Neu C.P. Reddi AH. K, KomvopoulosK. Schmid T.M. Di Cesare P.E. Increased friction coefficient and superficial zone protein expression in patients with advanced osteoarthritis. Arthritis Rheum. 62 (2010), 2680-2687

[5] Yusuf KQ, Motta N, Pawlak Z, Oloyede A. A micro analytical study of the surfaces of normal, delipidized, and artificially "resurfaced" articular cartilage. Connect. Tissue Res. 2012; 53 (2012) 236-245. doi: 10.3109/03008207.2011.630764.

[6] Kosinska MK, Liebisch G, Lochnit G, Wilhelm J, Klein H, Kaesser U, et al. A lipidomic study of phospholipid classes and species in human synovial fluid. Arthritis Rheum. 65 (2013) 2323–2333. doi: 10.1002/art.38053 PMID: 23784884

[7] Kosinska MK, Ludwig TE, Liebisch G, Zhang R, Siebert C, Wilhelm J, et al. Articular joint lubricants during osteoarthritis and rheumatoid arthritis display altered levels and molecular species. PLoS One.2015; 10 (5), e0125192.

[8] De Groot PG, Meijers JC. β_2-Glycoprotein I: Evolution, structure and function. J. Thromb. Haemost.9 (2011)1275-1284.

[9] de Laat B, Derksen RH, van Lummel M, Pennings MT, de Groot PG. Pathogenic anti-b2-glycoprotein I antibodies recognize domain I of β_2-Glycoprotein I only after a conformational change. Blood.107 (2006) 1916–1924.

Citation: *Pawlak Z., Karpinska B. "Antiphospholipid Syndrome in Osteoarthriticarticular Cartilage", International Journal of Research Studies in Science, Engineering and Technology. 2020; 7(11):1-4.*