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Research Article

The effect of deactivated phospholipids on joints lubrication: Osteoarthritis and lubricating properties

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Received date: May 12, 2021; Accepted date: June 01, 2021; Published date: June 17, 2021

Citation: M. Sojka and Z. Pawlak (2021) The effect of deactivated phospholipids on joints lubrication: Osteoarthritis and lubricating properties *J, Clinical Medical Reviews and Reports.* 3(6); DOI:10.31579/2690-8794/085

Abstract

PLs bilayers coating the major synovial joints such as knees and hips as the lubricant are responsible for the lubrication of articular cartilage. Lamellar-repulsive effect has been considered as a lubrication mechanism but it is likely that lubricin and hyaluronan with PLs participate in the lubrication process. The molecules of lubricin and hyaluronan adsorbed by PLs have a supportive role and provide the efficient lubrication of synovial joints via the hydration mechanism (~80% water content). Lipid profiles of injured and healthy knees' synovial fluids show significant differences. The phospholipid content in synovial fluid (SF) during joint inflammation, osteoarthritis is significantly higher (2 to 3 times) above the normal concentration of PL, and has a poor boundary-lubricating ability because of deactivated PL molecules. Deactivated PL molecule has no ability to form bilayers, lamellar phases, and liposomes.

Keywords: cartilage degradation; phospholipids deactivation; β_2-glycoprotein I, β_2-GP; osteoarthritis; lubricin; hyaluronan

Introduction

Surface-active phospholipids have been experimentally proved to be present in the synovial fluid and on the surface of articular cartilage, surface amorphous layer (SAL) [1, 2, 3]. Moreover, they play an essential role on the surface of articular cartilage in the process of lubrication. However, when the total amount of phospholipids increases in synovial fluid, this raises the question about surface deterioration transforming the hydrophilic layer into the hydrophobic surface [4].

Friction and lubrication are surface processes, only strongly adsorbed moieties to the surface are a primary lubricant and have importance roles in friction (charged macromolecules in synovial fluid). Osteoarthritis is teaching us about the importance of involvement of active phospholipids in the lubrication mechanism of hyaluronan and lubricin [3, 5, 6].

Charged moieties in synovial fluid (or solution)	≠	Charged moieties strongly adsorbed on surface of cartilage

Some researchers proposed a lubrication mechanism based on lubricin and hyaluronan complexes with phosphatidylcholines to provide a remarkable lubrication of synovial joints via hydration mechanism [8, 9]. In mammals, the intact lipid layer of cartilage is lost during degeneration, thus affecting the efficient lubrication of the joint [2, 3, 4].

In this study under osteoarthritic condition deactivated PLs molecules lost ability of bilayers formation and were unable to adsorb to lubricin and hyaluronan molecules. In healthy joints PLs bi-layers provide the low friction ($\mu \approx 0.005$). In osteoarthritic condition surface-attached lubricin with non-active PLs leads to considerably higher friction. In osteoarthritic condition where hyaluronan, non-active PLs and lubricin act separately, this cannot provide good boundary lubrication in articulating joints.

Materials and methods

Mass spectrometry of phospholipids. The phospholipid species were quantified by ESI-MS/MS on Micromass **[**5, 6]. 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. [5, 6]

Results and discussion

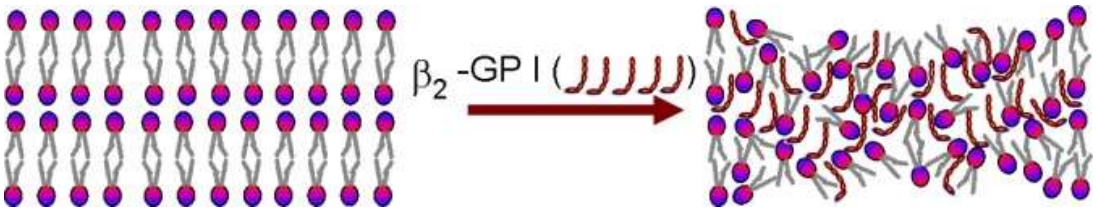

Figure 1. *β2-Glycoprotein I (β2-GPI) (open hockey stick) is complexed to negatively charged phospholipids (-PO4-) resulting in the destruction of bilayers*

Deactivation of a surface-active phospholipid bilayer (SAL). This amphoteric nature of phospholipids allows them to self-assemble into a classic arrangement which represents the basics of all biological membranes. A surface-active phospholipid layer covers normal articular surfaces in a multi-bilayer structure [3]. The bilayers serve to integrate interfacial functions between surfaces and have been a subject of several inquiries due to its tribological features [5, 10]. However, at sites of articular cartilage damage, the SAPL is absent, because a suitable substrate upon which this vital lipid layer can form does not exist [5, 7].

The mechanism of osteoarthritis (OA) is still not fully understood, but it has been established that this debilitating disease is often accompanied by a change in the synovial fluid composition, reduction in viscosity and deterioration of cartilage surface. Well-defined outermost bilayers were clearly visible on healthy cartilage surface, but OA may involve in the depletion of important joint molecules and SAPLs on the articular surface [2, 3]. Further, evidence for SAPL lining depletion was demonstrated by the cartilage wettability contact angle changing from 103 to 65 degrees. This insight led to the hypothesis that the SAPL is deactivated in the pathologic state of OA and remains present in synovial fluid but an inactive state (see Figure. 1). The pathological synovial fluid contains three times more phospholipids (PL), but the cartilage structure changes and its ability to lubricate, is remarkably poor. During normal functioning, the SAPL serves as a sacrificial perturbation bilayer, whereby it can improve by self-assembly mechanisms [10].

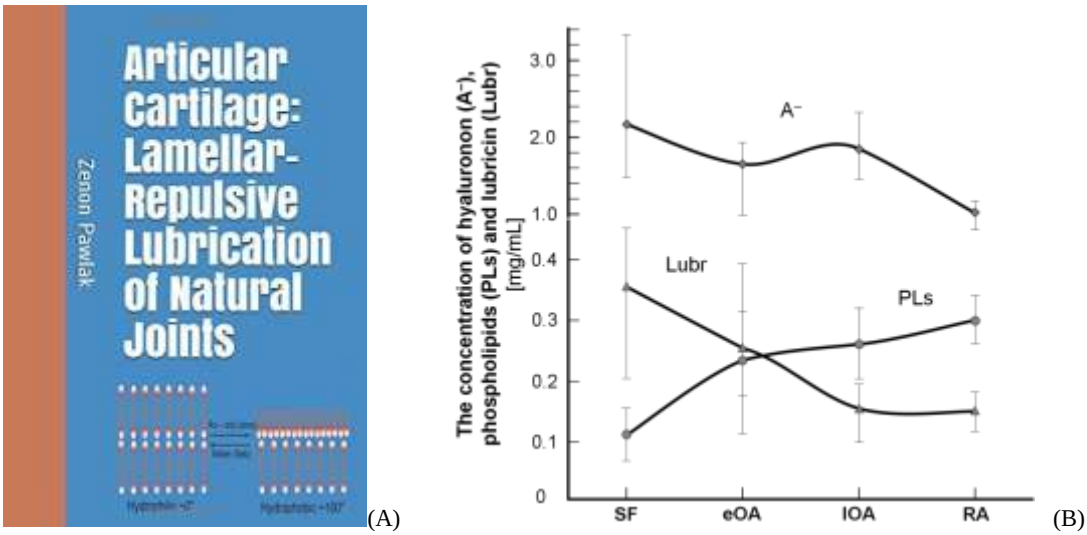

Figure 2. *(A) Phospholipid bilayer of articular cartilage in wet (hydrophilic ~ 0 degree) and air-dry condition (hydrophobic 104 degree) Book cover [3], and (B) the concentration of hyaluronan (A-), phospholipids (PLs) and lubricin (Lubr) in human synovial fluid (SF) in patients with healthy joints control (SF) and joint diseases with early osteoarthritis (eOA), late osteoarthritis (lOA) and rheumatoid arthritis (RA) [5, 6].*

The concentration of components of synovial fluid (SF), such as hyaluronan (A^-), lubricin, and surface-active phospholipids, from unaffected controls (or normal), eOA, lOA, and RA in the human synovial fluid are shown in Fig. 2 (B). During osteoarthritis (OA), and rheumatoid arthritis (RA), contain less hyaluronan (A^-), and lubricin and 2 to 3 times more with phospholipids. Also, the MW distribution of (A^-) shifted toward the lower range in OA and RA SF. These results indicate that activities in OA and RA SF are enhanced, leading to decreased levels of lubricin and high-MW hyaluronan (A).

Cartilage destruction in most rheumatic diseases and osteoarthritis has generally been accepted as a mechanism of deactivation of phospholipid bilayers. An acid-base interaction occurs between protonated amino acid group ($-NH_3^+$) of β_2-Glycoprotein I and the phospholipid ($-PO_4^-$) group: ($-NH_3^+$) + ($-PO_4^-$) → ($-NH_3^+ PO_4^-$) that is strong enough to deactivate the PLs bilayer surface.

β_2-Glycoprotein I (β_2-GPI) is a protein that circulates in blood at variable levels (50–500 μg mL^{-1} with a molecular weight of 50 kDa. β_2-Glycoprotein I (β_2-GP I) can exist in (a) closed conformation and (b) the open hockey stick-like conformation when β_2-GP I. β_2-GP I in its hockey stick-like conformation is a strongly adhesive protein and binds to different receptors on cells. Binding of β_2-GP I to anionic charged phospholipid ($-PO_4^-$) groups at pH ~ 7.4, results in a change in conformation and exposure of the epitope for the autoantibodies [11, 12, 13].

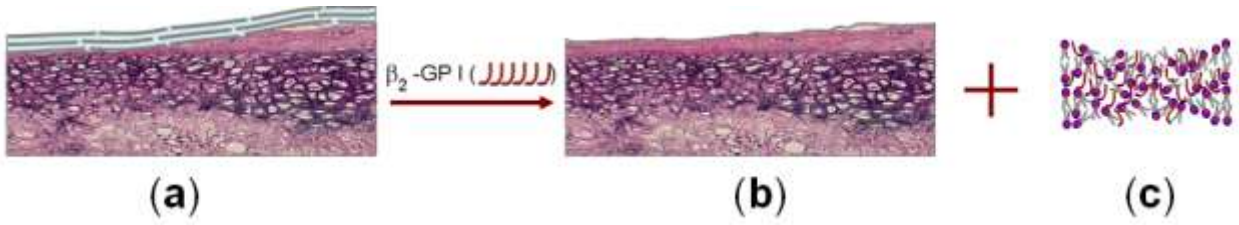

Figure 3. *Articular surface deterioration: (a) The PLs bilayers adsorbed to articular surface; (b) Degraded surface by the open hockey stick β2-Glycoprotein I; (c) Deactivated PLs molecules*

Softening of the cartilage is the first phase of cartilage deterioration [3]. Classic morphological changes of osteoarthritic articular cartilage begin with fibrillation and local surface disorganization involving splitting of the superficial layers of the cartilage, Figure 3. The early splitting is tangential with the cartilage surface, following the axes of' the predominant collagen bundles. Continued deterioration of articular cartilage leads to an exposure of the subchondral bone and more generalized synovial change. To understand the processes leading to cartilage failure is important to look at the cellular processes and biochemical structure of the normal cartilage.

Conclusion

A phospholipid involved in natural articular joints has a layered structure with weak inter-layer forces and low-strength shearing characterizes solid phospholipid lubricants. In this study, it was observed that osteoporosis significantly altered the phospholipid content so that the lipid profile was substantially changed from surface active to being deactivated (β_2-GPI-NH_3^+) + (PLs–PO_4^-) → β_2-GPI (-NH_3^+ PO_4^--PL) with a poor boundary-lubricating ability. Some have postulated or suggested that these lipids indicated "excessive tissue destruction". Deactivated PL molecule is unable to form bilayers, lamellar phases, and liposomes.

Acknowledgments

We gratefully acknowledge Ms Joanna A. Pawlak for editing this manuscript and for the financial supports from the Tribochemistry Consulting (Grant no. 01. 2019)

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DOI: 10.31579/2690-8794/085