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Changes in the structure of synovial fluids between healthy joints and in osteoarthritis

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Abstract

When a mammal's joint is healthy, the nanostructure of phospholipid bilayers is corrugated, and some phospholipids can be observed in the synovial fluid. During illness, like osteoarthritis, β2-Glycoprotein I changes conformation from closed to open hockey-stick-like, resulting in the degeneration of phospholipid bilayers and an increase in phospholipid levels in the synovial fluid. The composition of the synovial fluid in the joints changes since three are upto three times more inactive phospholipids in an inactive state. The unhealthy surface of articular cartilage becomes flatter what can be observed in the three-dimensional image of the articular cartilage.

Introduction

Hills established that the articular cartilage surface is covered by well-organized surface active phospholipids [4,5]. During osteoarthritis, β2-Glycoprotein I, is enzymatically activated, and is transformed into the antibody conformation [2]. Then, the antiphospholipid antibody syndrome, which is the result of it, is probably responsible for the degeneration of articular cartilage in the lubrication process.

One role of the synovial fluid is to redistribute phospholipids to preserve a bilayer lamellate structure which role is facilitating and lubricating the joints [4]. By the "Hills' biological lubrication model" [6] and the lamellar repulsion mechanism [9], phospholipids are the main solid constituents in the joints tribosystems. When the tribological behavior of natural synovial joints is studied, then the main focus is paid to the lubrication mechanism and its three major factors: the characteristics of the articular cartilage surface of articular cartilage, the relationship between wettability, surface energy, and friction, and lubricating mechanism [9].

In the natural lubrication, the role of phospholipids between biological surfaces is explained by some surface parameters: Interfacial energy, charge density, wettability and adsorption and the coefficient of friction. The significant point in the concept of lubrication plays the articular cartilage lubrication mechanism [9] which explains that if the pressure is used, then the drying mechanism is controlled by the synovial fluid flow between articular cartilage surfaces during the motion of a person or animal.

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Materials and methods

Hills' theoretical concept [4,5] was supported by the experimental study with the use of the high-resolution imaging technique called the Atomic Force Microscopy (AFM) [12]. On the 3D topographical images of mammal' articular cartilage, the normal healthy (Figure 1(c)) and depleted cartilage surfaces are presented (Figure 1(e)). It can be easily noticed that nanostructures of these two kinds of cartilages are extremely different. The nanostructural configuration of healthy cartilage has got several peaks and troughs (the average peak height equals 501.7 µm), while depleted cartilage shows the evident loss of the surface amorphous layers (the average peak height is equal to 351.2 µm), what means that 70% of height of average peaks of healthy cartilage.

Results

In a healthy mammal' joint, the articular cartilage is built of phospholipid bilayers, the nanostructure is corrugated, and some phospholipids can be observed in the synovial fluid.

During illness, the conformation of β2-Glycoprotein I changes transforming from closed to open hockey-stick-like, what causes the disintegration of the phospholipid bilayers. Because of that the composition of the synovial fluid in the joint changes, and the level of phospholipids can increase up to three times in the synovial fluid. Moreover, the three-dimensional image of the articular cartilage flattens (the average peak height of surface-active phospholipids becomes around 30% lower) (Figure 1(c), (e)).

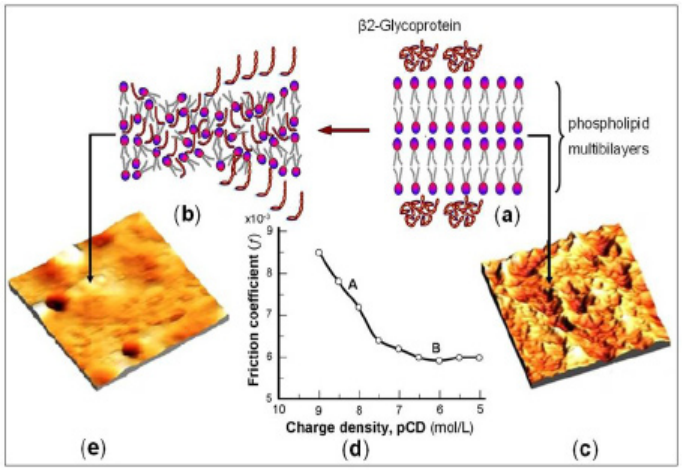

Figure 1: Changes in articular cartilage and synovial fluid between healthy and joint illness (a) phospholipid bilayers in healthy articular cartilage; (b) destructed and deactivated cartilage bilayers in joint diseases; (c) 3D topographical image of the healthy cartilage surface; (d) the friction coefficient f of the cartilage vs. surface charge density: (A) at a low cartilage charge density and (B) at a higher charge density; (e) 3D topographical image of the depleted cartilage surface.

While increasing the surface charge density, it was observed that the friction coefficient decreased rapidly at the beginning of the process and later it stabilizes at about 6.5.

Discussion

β2-Glycoprotein I is a protein, which is in blood at different levels (50–500 µg mL−1 with a molecular weight of 50 kDa) and it can exist in (a) closed and (b) the open hockey stick-like conformations (Figure 2). When β2-GP I is in (b) open – hockey stick-like conformation, it is strongly adhesive and binds to various cells' receptors. While β2-GP I binds to negatively charged phospholipid (–PO4$^-$) groups at pH ~ 7.4, it caused a transition to the second conformation and exposure of the epitope of the auto antibodies [1,2,3,7,10].

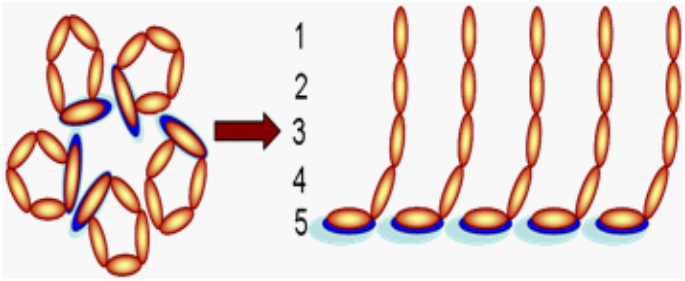

Figure 2: Conformations of β2-Glycoprotein I, (β2-GPI); (a) the circular conformation (closed molecules); (b) a hockey-stick-like conformation (open molecules), each molecule has five domains (1-5) and domain (5) with protonated group (-NH3+)

In the healthy joint, β2-Glycoprotein I, (β2-GPI) circulates in plasma in closed conformation but in the antiphospholipid antibody syndrome, it transforms to the hockey-stick-like conformations and binds to negatively charged phospholipids (-NH_3^+) + (–PO_4^-) → (-NH_3^+ PO_4^--). Moreover, the auto antibodies bind and stabilize β2-Glycoprotein I in its stick-hockey-like conformation. Charge density, -log [OH^-] = p CD. Figure. 1(d) (curve A) the friction coefficient decreases with the increasing charges and not changed density of cartilage (-OH^- + - PO_4H→ H_2O + -PO4$^-$),(curve B) the friction coefficient does not change sensitively with the addition -OH- ions showing up the absence of (- PO_4H) group.

According to Pawlak [9], phospholipids adsorb on the surface of healthy joint cartilage, create bilayers and limit boundary lubrication. When the mammal' joint is healthy, the outer bimolecular layers are clearly visible on the cartilage surface (nanostructure has got peaks and troughs), Figure 1(c), (e). However, some diseases like osteoarthritis may cause the reduction of surface-active phospholipids on the articular surface [6,9]. Moreover, the osteoarthritis often goes together with a change in the structure of the synovial fluid, degeneration of cartilage surface and reduction in viscosity [11]. It can lead to the hypothesis that in this disease, surface-active phospholipids are deactivated, move to the synovial fluid but in an inactive state (see Figure 1 (a), (b)). The structure of articular cartilage changes and the synovial fluid contains up to three times more deactivated phospholipids. Except that, the ability to lubricate unhealthy articular cartilage is remarkably poor, and as it has been shown [8], the depleted surface articular cartilage is less rigid than the healthy one, so losing lipid layers of cartilage causing its destruction and lower rigidness. Hence, the deteriorated articular cartilage is stiffer and flatter.

Acknowledgment

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