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BIOMECHANICS

Biobearings: where mechanics meets biology

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Abstract. This review paper deals with the operation of human joints which, from a mechanical point of view, can be regarded as bearings. The main problem in the studies of biobearings is separate consideration of the matter in different disciplines or areas of research. However, being a highly interdisciplinary field, biomechanics has to collect and apply knowledge from many branches of science. In this paper, the recent advances in tribology, organic chemistry, and tissue biology are reviewed and summarized to give a comprehensive vision of the state of the art in the performance of synovial joints. The emphasis is on the latest findings in lubrication mechanisms and their possible interactions.

Key words: biotribology, synovial joint, articular cartilage, lubrication, lipidic bilayer.

1. INTRODUCTION

The synovial joint is a perfect tribological creation of the nature with low friction and high wear resistance acting without any reparation during service. Thus, the application of tribology in medicine and biology is a growing and rapidly expanding field [1–12]. Biotribology can be defined as the study of friction, lubrication, and wear in biological systems, specifically articular joints. The control of friction and wear is a key to many biological functions. Examples are wide ranging. A well known one is the friction and wear in hip and knee joints, where tribology clearly has a major impact on the reliability and durability, and is a strong function of the tissues cooperation. However, until now there is no comprehensive vision of the mechanisms of the remarkable joint performance. This is why interest has grown over the last decade in identification and characterization of not only the mechanical mechanisms of joint working but also of chemical reactions and biological processes taking place during the life time of the joint. And this is why writing this review was undertaken.

The human joint is a self-acting and dynamic load-bearing structure, which uses a porous and elastic biomaterial (i.e. articular cartilage) as well as a highly non-Newtonian lubricant (i.e. synovial fluid) for its functioning. A simplified scheme of a human hip joint is presented in Fig. 1. The bones are fixed by ligaments and the entire joint is enclosed in a fibrous tissue capsule, the inner surface of which is lined with the synovial membrane that secretes a fluid known as synovial one. Articulating surfaces of the bones at the joint are covered by a hyaline cartilage. The thickness of

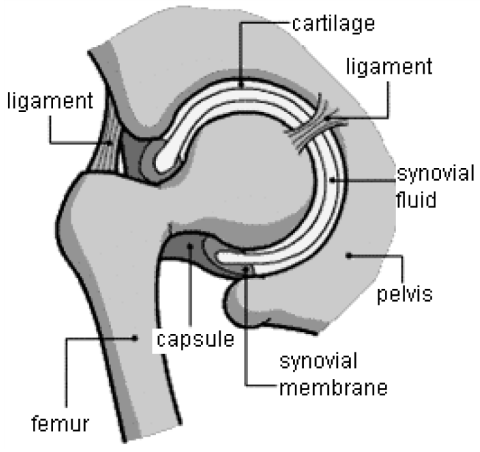

Fig. 1. Schematic view of a hip joint, which may be regarded as a biobearing.

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the cartilage varies with each joint, and may sometimes be uneven. The primary function of the cartilage layer is to minimize contact stresses generated during joint loading and to contribute to lubrication mechanisms in the joints [9,10]; cartilage serves as a kind of damper under dynamic loads.

A healthy joint can withstand a loading up to 24 times the body weight during jumping [11]. Joint diseases are characterized by changes in cartilage and bone, which lead to degradation of joint materials, which, in turn, results in deformation, increase in friction and, finally, wear of cartilage and causes impaired joint motion, pain, and disability. The goal of many studies in biotribology over decades has been to describe joint operation from the tribological point of view to establish the relationships between processes in joints responsible for the functional degradation of materials, their structure and properties, including friction, wear, and lubrication. Below some recent advances in this area of research are reviewed and summarized. The focus is on the mechanics and chemistry of lubrication because in spite of sustained efforts to develop a comprehensive understanding of joint performance there is a large amount of separate data that need generalization.

2. STRUCTURE, PROPERTIES, AND FRICTION OF ARTICULAR CARTILAGE

Cartilage is a highly differentiated porous material filled with synovial fluid. Articular cartilage represents a multi-layered composite (Fig. 2) and is a metabolically active biomaterial. A thin superficial tangential layer provides a smooth surface for two bones to slide against each other. Of all the layers, this thin layer has the highest concentration of collagen (collagen has a great tensile strength), making it very resistant to shear stresses. Below it is an intermediate layer, which is mechanically designed to absorb shocks and distribute the load efficiently. In the middle zone, the collagen fibres are more loosely packed and are randomly orientated. The deepest layer is highly calcified, and fixes the articular cartilage to the bone. In the deep zone, the collagen fibres anatomize forming larger bundles prior to insertion into the calcified zone across the tidemark.

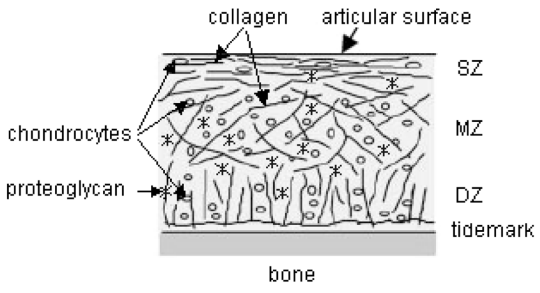

Fig. 2. Schematic representation of the structure of articular cartilage (SZ – superficial tangential zone, MZ – middle zone, DZ – deep zone).

Approximately 65% to 85% of the weight of the whole tissue is water. The remainder is composed primarily of proteoglycans (PG) and collagen. Proteoglycans consist of a protein core to which glycosaminoglycans (chondroitin sulphate and keratan sulphate) are attached to form a bottlebrush-like structure. These PG can bind or aggregate to a backbone of hyaluronic acid to form a huge macromolecule. In an aqueous environment, the PG are poly-anionic; the molecule has negatively charged sites that arise from its sulphate and carboxyl groups [11]. In solution, the mutual repulsion of these negative charges causes an aggregated PG molecule to spread out and occupy a large volume (Fig. 3). In the cartilage matrix, the volume occupied by PG is limited by the entangling collagen framework. The swelling of the aggregated PG molecule against the collagen framework is an essential element in the mechanical response of the cartilage.

The concentration of PG and water content vary through the depth of the tissue as shown in Fig. 2. Near the articular surface, the PG concentration is relatively low, while the water content is the highest in the tissue. In the middle regions of the cartilage, the PG concentration is greatest, and the water content is the lowest near the subchondral bone.

The negative charge of PG is neutralized by positive ions in the surrounding fluid. The higher concentration of ions in the tissue compared to outside the tissue leads to swelling pressures. The exclusion of water raises the density of fixed charge, which in turn raises the swelling pressure and charge–charge repulsion. Thus, the compaction of the PG affects a swelling pressure as well as fluid motion under compression.

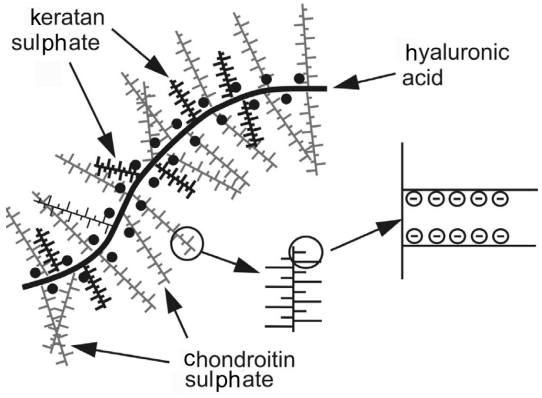

Fig. 3. Structure of the proteoglycan macromolecule: chondroitin sulphate and keratan sulphate aggregated to hyaluronic acid forming a bottlebrush-like structure with negatively charged sites.

The collagen network resists the swelling of the articular cartilage. Collagen accounts for approximately 50% of the dry weight of the tissue. If the collagen network is degraded, as in the case of osteoarthritis, the amount of water in the cartilage will increase because more negative ions are exposed to draw in fluid. The increase in fluid can significantly alter the mechanical behaviour of the cartilage. Collagen fibres provide tensile strength.

In addition, with a pressure gradient or compression, fluid is squeezed out of the cartilage. When the fluid is being squeezed out, there are drag forces between the fluid and the solid matrix, which increase with increasing compression and make it more difficult to exude water. This behaviour increases the stiffness of the cartilage as the rate of loading is increased. Mechanical properties of the cartilage depend on the fluid content.

There are three major factors that contribute to the mechanical behaviour of articular cartilage. First, it is the elastic behaviour of the solid matrix itself. Second, it is the swelling pressure due to the ionic effects in the tissue. And the third one is the fluid–solid interaction in the cartilage under compressive load.

The mechanical behaviour of the solid matrix is determined by the amount, crimp, and orientation of collagen in the matrix. Thus, this matrix follows the classic nonlinear stress–strain curve for soft tissues [10,12] as shown in Fig. 4. The response of cartilage can be vastly different for compressive, tensile, and shearing stresses due to the specialized composition and structural organization of the cartilage layer [10,13]. Furthermore, the response of the tissue to an applied load varies with time, exhibiting viscoelastic behaviours such as creep and stress relaxation. There are two distinct dissipative mechanisms in response to loading: the frictional drag force of interstitial fluid flow through the porous-permeable solid matrix (i.e., the flow-dependent mechanism); and the time-dependent deformations of the solid macromolecules (i.e., the flow-independent mechanism). Because of the charged nature of articular cartilage and the electrolytes dissolved in the interstitial water, articular cartilage also exhibits complex electrochemical phenomena in addition to its mechanical response, including streaming and diffusion potential and charge-dependent osmotic swelling pressures (i.e., the Donnan osmotic pressure). Features of mechanical and mechano-electrochemical behaviour of articular cartilage and modern testing techniques are described in detail in the excellent review paper by Mow and Guo [10].

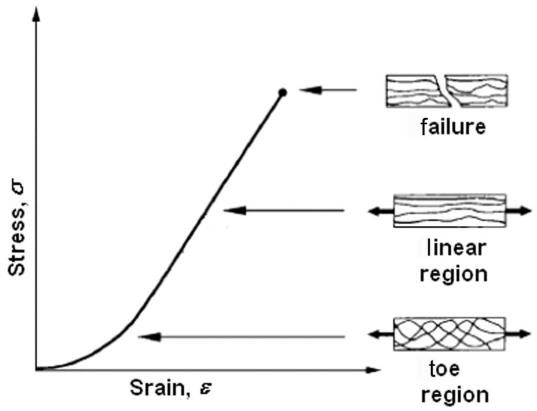

Fig. 4. Characteristic stress–strain relationship of articular cartilage (adapted from [14]).

Time-dependent properties of articular cartilage are related to the interstitial fluid flow and exudation. Fluid movement is governed by the hydraulic permeability of the solid matrix. In turn, the coefficient of permeability is related to the extracellular matrix pore structure, apparent size, and connectivity. Because of the quite low permeability of cartilage (in the range $(1.2–6.2)\times10^{-16}$ m^4/Ns for all types of cartilage materials) [11], large interstitial fluid pressures and dissipations occur in the tissue during loading. Thus, pressurization and high energy dissipation give a possibility of shielding the collagen–PG complex from high stresses and strains associated with joint loading because the pressurized fluid component provides for the major load-bearing function.

It is well known that human synovial joints function with an extremely low friction coefficient. Degradation of either part of the synovial fluid–articular cartilage system leads to increased friction, wear, and reduction of mobility; degeneration of cartilage is characterized by softening and fibrillation that may lead to joint disease.

The biphasic boundary friction model proposed in [14–16] quantifies the load sharing between the solid and fluid phases of the porous-permeable cartilage at the contact interface. The fluid load support W^p/W is defined as the fraction of the total applied normal load W that is supported by the interstitial fluid when it pressurizes. The fluid load support is not measured directly, but its time-dependent behaviour can be inferred from the measured load response $W(t)$ and axial displacement $u(t)$, based on the formulation of biphasic theory [16]:

$$\frac{W^p}{W}=\frac{H_{+A}+\lambda_2}{H_{+A}-\lambda_2}\left[1-\frac{W_0}{W}-\left(\frac{W_{\mathrm{eq}}-W_0}{W}\right)\left(\frac{u-u_0}{u_{\mathrm{eq}}-u_0}\right)\right]$$
$$\equiv\alpha\cdot f(W,u).$$

In this expression, H_{+A} is the tensile aggregate modulus, λ_2 is the off-diagonal modulus, $W_0 = W(t=0)$ is the tare load magnitude, $W_{\mathrm{eq}} = W(t\to\infty)$ is the equilibrium normal load, $u_0 = u(0)$ is the dis-

placement under the tare load, and $u_{eq} = u(t \to \infty)$ is the equilibrium displacement. The function

$$f(W,u) = \left[1 - \frac{W_0}{W} - \left(\frac{W_{eq} - W_0}{W}\right)\left(\frac{u - u_0}{u_{eq} - u_0}\right)\right]$$

is completely determined from experimental measurements; however, without further characterization of the mechanical properties of each sample, the ratio

$$\alpha = \frac{H_{+A} + \lambda_2}{H_{+A} - \lambda_2}$$

is a priori unknown.

The biphasic friction model [15] formulated the following dependence between the transient friction coefficient and interstitial fluid load support:

$$\frac{\mu_{eff}}{\mu_{eq}} = 1 - (1-\varphi)\frac{W^p}{W}.$$

The equation predicts a linear relationship between μ_{eff}/μ_{eq} and $f(W,u)$. From a linear regression performed on this response, the slope $(1-\varphi)\alpha$ can be determined. As a first approximation, $(1-\varphi)$ may be taken as the water content of cartilage at the articular surface; α can then be estimated from the slope of the linear regression, along with W^p/W from the equation.

A representative plot of the transient variation of the coefficient of friction (CoF) and the corresponding fluid load support and a plot for the CoF against the fluid load support is given in Fig. 5. Results strongly suggest that interstitial fluid pressurization is a primary mechanism in the regulation of the friction response of articular cartilage. By supporting the majority of the load transmitted across the contact interface, the interstitial fluid pressurization reduces the load supported by the contacting collagen–PG matrixes, considerably reducing the frictional force relative to the total contact force. As long as the interstitial pressure remains elevated, the effective CoF is small. As the fluid pressure reduces to zero the contact force will increasingly shift to the solid matrix, consequently increasing the CoF.

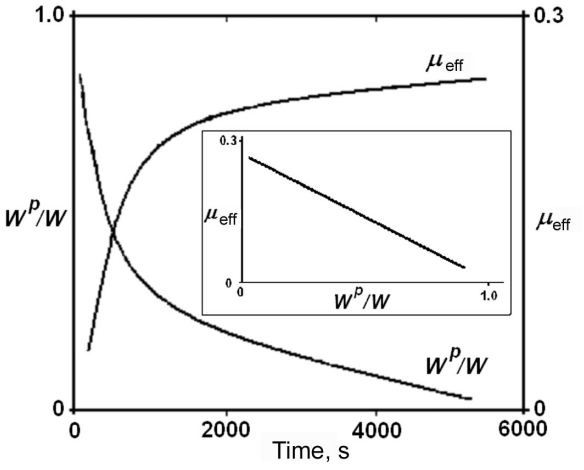

Fig. 5. Effective coefficient of friction and interstitial fluid support vs. time. Inset: effective coefficient of friction vs. interstitial fluid load support, W^p/W.

3. LUBRICATION IN SYNOVIAL JOINTS

Interstitial fluid pressurization seems to be a primary mechanism in the regulation of the frictional response of articular cartilage. However, synovial joints are not continuously separated by a thick fluid layer. It means that boundary lubrication plays an important role in the friction of synovial joints. Boundary lubricants may supplement the key role of interstitial fluid pressurization to help further reduce the CoF of cartilage.

There are numerous studies that compare the frictional response of articular cartilage using synovial fluid versus saline or other lubricants [5,6,14,17–20]. Lower minimum CoFs have been generally demonstrated with the presence of synovial fluid. The findings are consistent with the prevailing hypothesis that the boundary lubricant is present in significant amounts in synovial fluid (in addition to its potential presence in the superficial zone of cartilage).

Experimental testing of soft and highly hydrated biological tissues such as cartilage under conditions approaching those found in vivo poses significant difficulties and technical challenges. Numerical modelling techniques such as finite element analysis have therefore emerged as a useful research tool for investigating such materials. A comprehensive study by Wilson and co-authors [21] gives an overview of different material models developed for articular cartilage and of what they can be used for. One of the advanced tasks to understand and correct the modelling of low-frictional joint behaviour is to consider the amorphous layer on the surface of the sliding cartilages. The existence of such a surface amorphous layer was revealed with the help of a cryo-scanning electron microscope [22].

It was shown [21] that a thin, soft, biphasic surface amorphous layer with lower elastic modulus dramatically alters the load sharing between the solid and liquid phases of articular cartilage, particularly in the near-surface regions of the underlying bulk cartilage and within the surface amorphous layer itself where the fluid load support exceeds 85%. By transferring the load from the solid phase to the fluid phase, the biphasic surface layer improves lubrication and reduces friction, also protecting the underlying cartilage surface by ‘shielding’ the solid phase from elevated stresses. Therefore, the surface

amorphous layer, which is of utmost importance from a lubrication point of view, provides further insights into the boundary lubricating function of 'gels' such as glycosylated surface lubricating proteins and glycosaminoglycans. The increase in lubrication effectiveness has been shown to be greatest during loading scenarios of short duration, such as shock loads.

Such a surface layer may not only play an important role in lubrication but also in protecting articular cartilage in the joint, particularly during shock loading and other transient loading events. The authors are of the opinion that because of its importance in human joint functionality, this surface layer is worth being depicted in cross-sections of articular cartilage as a special zone covering a superficial surface (Fig. 6). Although the exact composition of the amorphous layer is not known, it can be supposed that synovial gel consists of surface active phospholipids (SAPLs), hyaluronic acid (HA), and albumin. For many years, HA was believed to be the lubricant in the joint because it is a major component of synovial fluid and its solutions are very slippery to the touch. However, it is not the boundary lubricant. Basically, it possesses no load-bearing capability unless a SAPL is incorporated [17–24]. Because of that, in the last decade there has been a revived interest in the role of boundary lubricants such as phospholipids, lubricin, and related glycoproteins like superficial zone proteins and PG. The lipid content of cartilage is from 0.3% to 4% or from 1.2% to 10% on a dry-weight basis [23], which was almost totally ignored until quite recently. It was suggested that lipidic molecules could play a role in joint lubrication in the same way that oils and greases are used in engineering. It was found that by rinsing the articular surfaces with a lipid solvent friction increases by 150% [24]. Incubation with a lipase used to digest fat has a similar effect.

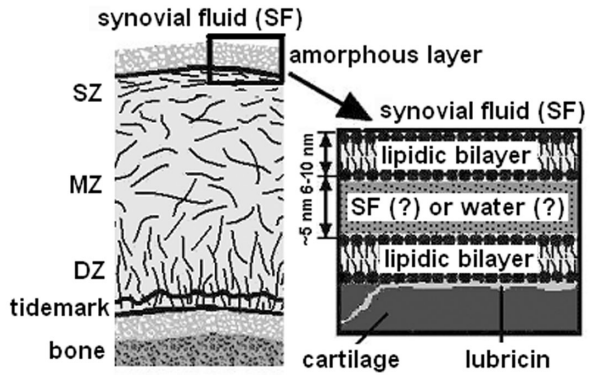

Fig. 6. Schematic representation of cartilage with an additional amorphous layer on its surface and a possible structure of the biphasic surface amorphous layer. *Right*: frictional pair of biobearings: two cartilage surfaces covered with an amorphous boundary layer are separated by a biolubricant (adapted from [26]). SZ – superficial tangential zone, MZ – middle zone, DZ – deep zone.

Lipids are represented by three basic components: cholesterol, triglycerides, and phospholipids. The first two predominate in most sites in which fat is located in the body. However, in a normal joint the major component (61%) is phospholipid. The major sub-fraction of phospholipid is dipalmitoyl phosphatidylcholine (DPPC) [23].

One of the features of joint phospholipids is that they are largely saturated and surface active. Surface activity is essentially derived from a different nature of the two ends of the molecule, one polar that seeks water being hydrophilic and the other non-polar that seeks air or neutral lipids being hydrophobic. The SAPLs form a so-called lipid bilayer, which is composed of two layers of lipids arranged so that their hydrocarbon tails face one another to form an oily core held together by Van der Waals interactions, while their charged heads face the aqueous solutions on both sides of the membrane. The lipid bilayer is a self-assemblying structure with temperature-dependent properties.

An additional feature of the lipid bilayer is that it has a highly positively charged quaternary ammonium (QA) ion as its terminal group. This is an ideal condition for binding these small molecules to the negatively charged PG molecules of cartilage. Thus, with strong adsorption of SAPL molecules and strong cohesion of this adsorbed lining, SAPL satisfies the two fundamental criteria for a high-load-bearing boundary lubricant. The whole system may include not only a single lipidic layer but a stack of several bilayers. Once absorbed, phospholipids create a strong laterally bonded network, thus altering the viscoelastic properties of the interface.

The lipid bilayer may be considered as a two-dimensional fluid of high viscosity allowing some structural mobility within the bilayer. The flow behaviour of lipid bilayer membranes is characterized by surface viscosity for in-plane shear deformations, and an intermonolayer friction coefficient for slip between the two leaflets of the bilayer. Mobility of lipid molecules results in an easy deformability of the layer and a low modulus of elasticity as well as a very low shear modulus exhibiting only viscous resistance to shear. The CoF of two bilayers sliding over each other in a water solution was theoretically estimated to be very low [24]. Thus, an understanding of the mechanical properties of adsorbed lipid layers is vital in a variety of branches of research starting from biology, chemistry, and materials engineering.

Recent research by Gale et al. [25] aimed at analysing the SAPLs found on the surface of retrieved artificial implants showed that unsaturated phospholipids may contribute also to CoF reduction. Therefore, a combination of SAPLs rather than a single SAPL contributes to the boundary lubrication of the joints.

Outstanding experiments presented in [26] showed that the CoF between two hydrogel surfaces imitating

cartilage in the presence of lipid bilayers separated by a physiological salt solution (Fig. 6) but with no HA or albumin is as low as 0.0015. Visualization of the rubbing surfaces revealed that the lipidic surfaces remain almost intact. Low friction could be attributed to the shift of the slip plane from between bilayers to the solution layer. A similar observation of a very low CoF was made by Briscoe et al. [27] when the CoF was tested between two polymer surfaces covered with a surfactant layer in water. The low friction could be due to the fluid hydration layers surrounding the polar head groups attached to the substrate. Lubrication at the surfactant–substrate interfaces may be mediated by the fluid hydration sheaths surrounding the surfactant polar head groups at the substrate. In living systems, lubrication may also be mediated by hydration shells. The hydration layer located between two lipidic bilayers in a synovial joint could probably be thick enough to result in an extremely low CoF.

Moreover, hydrophilic polar groups or phospholipid heads can form either favourable electrostatic interactions or hydrogen bonds with water molecules. This principle was recently used by chemists for obtaining very low friction between bearing surfaces in the presence of specially designed lubricants [27–29]. In particular, it was shown [27] that neutral polymer 'brushes' may lead to a great reduction in sliding friction between the surfaces to which they are attached, whereas hydrated ions can act as extremely efficient lubricants between sliding charged surfaces. Effective CoF with polyelectrolyte brushes in water are lower than 0.001 even at low sliding velocities and at pressures of up to several atmospheres (typical of those in living systems). A similar mechanism may act in the living body and an extremely low CoF may be attributed to the exceptional resistance to the interpenetration of two compressed leaflets of the bilayer together with the fluidity of the hydration layers surrounding the faced water polar head groups of bilayers. In [28] it was shown that the surface attached hydration layers keep the compressed surfaces apart as a result of strongly repulsive hydration forces. Removal of water molecules from the ions to which they are attached is quite an unfavourable process leading to an increase in the energy of the system. Much lower energy may be associated with the diffusion of water molecule within the outer layer. Consequently, the hydrated layer between two bilayers is capable of supporting a large normal load because a rapid exchange of molecules ensures that the surface bond hydration layer remains very fluid. No doubt, fluidity of the bonded water at salt concentrations and pressures typical of those in biological systems has implications in synovial joint functioning.

Also, polyelectrolyte molecules such as lubricin are shown to play a significant role in adhesion on the lipidic membranes [30], which could modify their tribological performance. The synovial joint is a complicated 'living device', and attributing all of its perfect tribo-properties to only a particular mechanism or substance would undoubtedly be a mistake. However, despite their possible influence on the joint performance, interactions between synovial fluid constituents are dealt with in a few studies [26,29–32]. For example, in [31] it is shown that unusual flow characteristics of synovial fluid may be a result of protein aggregation followed by enhanced stress support by entanglement of this tenuous protein network with the long-chain polysaccharide sodium hyaluronate under physiological conditions. Neutron scattering measurements on albumin solutions [31] demonstrated protein aggregation and all measurements were consistent with a weak dipolar attraction energy (about 3 kT) that is most likely augmented by hydrophobic interactions and/or disulphide bond formation between proteins. The findings strongly suggest the formation of a network between HA and phospholipid molecules, which is likely to be present in the synovial fluid and might contribute to determining the unique properties of such a system and its physiological performance, strongly influencing the viscoelastic character of synovial fluid.

In [32] the interactions between HA and phospholipid molecules of DPPC type are studied in vitro and development of membrane-like structures on the substrate and 12-nm thick tubes of HA surrounded by DPPC in a fluid (Fig. 7) are shown. There are good reasons to believe that similar structures may be created in vivo within the joint cavity. Moreover, it was proven that such structures are formed only in the presence of high molecular weight HA while low molecular weight HA induces fragmentation of liposomes.

It is well known that in osteoarthritis affected joints, HA has a reduced molecular weight. Whether this is a result or a cause of osteoarthritis is uncertain; however, it is clear that limited capability of DPPC based layer formation influences the efficiency of boundary lubrication to sustain high loads resulting in increased friction and, as a consequence, wear of cartilage.

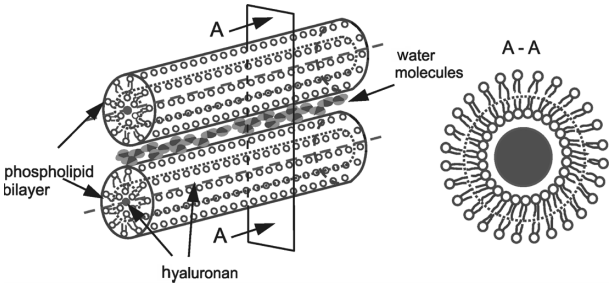

Fig. 7. Possible structure resulting from the interaction of hyaluronic acid and phospholipids.

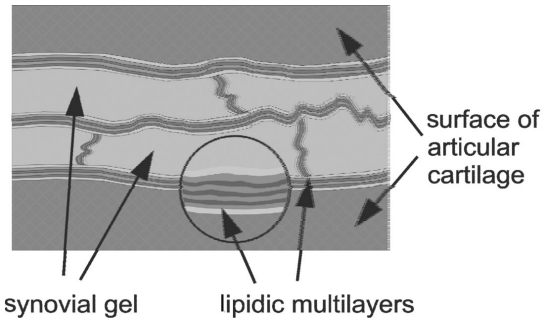

Fig. 8. Sketch of the frictional pair model in biobearing (adapted from [26]).

In addition, the formation of an extended three-dimensional network by intra- and inter-chain bridges contributes to the hydrodynamic and viscoelastic properties of synovia. Some C–H groups of HA are aligned to form regions of hydrophilic character. These hydrophobic zones are repeated along the HA chain and are considered to participate in intra-chain interactions and binding to phospholipids. In this concept the tribological device – synovial joint – may be schematically sketched as shown in Fig. 8. In [26] a similar ex vivo model was used for multiscale analysis of the tribological role of the molecular assemblies of synovial fluid. It was concluded that the presence of lipidic bilayers in the contact area leads to a very low CoF and interaction between biomolecules is of key importance for the tribological performance of the joint.

4. CONCLUDING REMARKS

Within the past decade significant advances have been made in experimental and theoretical studies of the basic sciences related to (human) synovial joint functionality, durability, and diseases. The knowledge obtained from a broad range of researches in mechanical engineering, chemistry, biology, and physics provides enrichment and in-depth understanding of the many processes in the living body and the relationships between any changes in the inner environment and tissue functionality and/or vice versa. Despite the achievements, there are still numerous unanswered questions waiting to be solved. A particular challenge will be realization and investigation of the interactions of the structure–architecture–processes–performance–metabolism: how and why the biomolecules and electrolytes control the interfacial interaction of two (or more) biological surfaces and hence the joint performance and durability; and how these processes can be mimicked using all the attributes of the exceptionally relevant mechanism in tribology – the synovial joint – for the development of new techniques in joint disease treatment and new frictionless mechanical devices and bio-bearings. Now it is fully understood that highly interdisciplinary integrated forces are needed for the studies of the tribology of synovial joints. A further challenge is to quantitatively understand and control the biomolecular 'association' reactions in self-assembly processes and to suitably catalyse these reactions.

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REFERENCES

1. Furey, M. Joint lubrication. In *The Biomedical Engineering Handbook* (Bronzino, J. D., ed.). CRC Press, Boca Raton, 2000, 21–26.

2. Yao, J. Q., Laurent, M. P., Johnson, T. S., Blanchard, C. R., and Crowninshield, R. D. The influences of lubricant and material on polymer/CoCr sliding friction. *Wear*, 2003, **255**, 780–784.
3. Graindorge, S., Ferrandez, W., Jin, Z., Ingham, E., Grant, C., Twigg, P., and Fisher, J. Biphasic surface amorphous layer lubrication of articular cartilage. *Med. Eng. Phys.*, 2005, **27**, 836–844.
4. Fisher, J. Biomedical applications. In *Modern Tribology Handbook, Vol. 2. Materials, Coating and Industrial Applications* (Bhushan, B., ed.). CRC Press, Boca Raton, 2001, 1593–1609.
5. Blewis, M. E., Nugent-Derfus, G. E., Schmidt, T. A., Schumacher, B. L., and Sah, R. L. A model of synovial fluid lubricant composition in normal and injured joints. *Eur. Cells Mater.*, 2007, **13**, 26–39.
6. Elsaid, K. A., Jay, G. D., Warman, M. L., Rhee, D. K., and Chichester, C. O. Association of articular cartilage degradation and loss of boundary-lubricating ability of synovial fluid following injury and inflammatory arthritis. *Arthr. Rheum*., 2005, **52**, 1746–1755.
7. Jin, Z., Williams, S., Tipper, J., Ingham, E., and Fisher, J. Tribology of hip joints from natural hip joints, cartilage substitution, artificial replacements to cartilage tissue engineering. *J. Biomech. Sci. Eng.*, 2006, **1**, 59–79.
8. Peng, Z. Ostearthritis diagnosis using particle analysis technique. *Wear*, 2007, **262**, 630–640.
9. Mow, V. C. and Ateshian, G. A. Lubrication and wear of diarthrodial joints. In *Basic Orthopedic Biomechanics* (Mow, V. C. and Hayes, W. C., eds). 2nd edn. Lippincott-Raven, Philadelphia, 1997, 275–315.
10. Mow, V. C. and Guo, X. E. Mechano-electrochemical properties of articular cartilage: their inhomogeneities and anisotropies. *Annu. Rev. Biomed. Eng.*, 2002, **4**, 175–209.
11. Mansour, J. M. Biomechanics of cartilage. In *Kinesiology: the Mechanics and Pathomechanics of Human Movement* (Oatis, C. A., ed.). Lippincott Williams and Wilkins, Philadelphia, 2003, Ch. 5, 66–79.
12. Mow, V. C., Ateshian, G. A., and Spilker, R. L. Biomechanics of diarthrodial joints: a review of twenty years of progress. *J. Biomech. Eng*., 1993, **115**, 460–467.
13. Mow, V. C., Ratcliffe, A., and Poole, A. R. Cartilage and diarthrodial joints as paradigms for hierarchical materials and structures. *Biomaterials*, 1992, **13**, 67–97.
14. Ateshian, G. A., Wang, H., and Lai, W. M. The role of interstitial fluid pressurization and surface porosities on the boundary friction of articular cartilage. *J. Trib.*, 1998, **120**, 241–251.
15. Krishnan, R., Kopacz, M., and Ateshian, G. A. Experimental verification of the role of interstitial fluid pressurization in cartilage lubrication. *J. Orth. Res.*, 2004, **22**, 565–570.
16. Soltz, M. A. and Ateshian, G. A. A conewise linear elasticity mixture model for the analysis of tension–compression nonlinearity in articular cartilage. *J. Biomech. Eng.*, 2000, **122**, 576–586.
17. Neville, A., Morina, A., Liskiewicz, T., and Yan, Y. Synovial joint lubrication – does the nature teach more effective engineering lubrication strategies? *Proc. Inst. Mech. Eng*. [C]: *J. Mech. Eng. Sci.*, 2007, **221**, 1223–1230.
18. Sawae, Y. and Murakami, T. An experimental investigation of boundary lubrication mechanism with protein and lipid in synovial joint using total internal reflection fluorescence microscopy. *J. Biomech.*, 2006, **39**(1), 476–485.
19. Schwarz, I. M. and Hills, B. A. Surface-active phospholipid as the lubricating component of lubricin. *Brit. J. Rheumatol.*, 1998, **37**, 21–26.
20. Forsey, R. W., Fisher, J., Thompson, J., Stone, M. H., Bell, C., and Ingham, E. The effect of hyaluronic acid and phospholipid based lubricants on friction within a human cartilage damage model. *Biomaterials*, 2006, **27**, 4581–4590.
21. Wilson, W., van Donkelaar, C. C., van Rietbergen, R., and Huiskes, R. The role of computational models in the search for the mechanical behavior and damage mechanisms of articular cartilage. *Med. Eng. Phys.*, 2005, **27**(10), 810–826.
22. Kobayashi, S., Yonekubo, S., and Kurogouchi, Y. Cryo-scanning electron microscopic study of the surface amorphous layer of articular cartilage. *J. Anat.*, 1995, **187**, 429–444.
23. Hills, B. A. and Crawford, R. W. Normal and prosthetic synovial joints are lubricated by surface-active phospholipid: a hypothesis. *J. Arthroplasty*, 2003, **18**(4), 499–505.
24. den Otter, W. K. and Shkulipa, S. A. Intermonolayer friction and surface shear viscosity of lipid bilayer membranes. *Biophys. J.*, 2007, **93**, 423–433.
25. Gale, L. R., Chen, Y., Hills, B. A., and Crawford, R. Boundary lubrication of joints: characterization of surface-active phospholipids found on retrieved implants. *Acta Orthop.*, 2007, **78**(3), 309–314.
26. Trunfio-Sfarghiu, A.-M., Berthier, Y., Meurisse, M.-H., and Rieu, J.-P. Multiscale analysis of the tribological role of the molecular assemblies of synovial fluid. Case of a healthy joint and implants. *Trib. Int.*, 2007, **40**, 1500–1515.
27. Briscoe, W. H., Titmuss, S., Tiberg, F., Thomas, R. K., McGillivray, D. J., and Klein, J. Boundary lubrication under water. *Nature*, 2006, **444**, 191–194.
28. Raviv, U. and Klein, J. Fluidity of bound hydration layers. *Science*, 2002, **297**, 1540–1543.
29. Zappone, B, Ruths, M, Greene, G. W., Jay, G. D, and Israelachvili, J. N. Adsorption, lubrication and wear of lubricin on model surfaces: polymer brush-like behavior of a glycoprotein. *Biophys. J.*, 2007, **92**, 1693–1708.
30. Rhee, D. K., Marcelino, J., Baker, M. A., Gong, Y., Smits, P., and Lefebvre, V. The secreted glycoprotein lubricin protects cartilage surfaces and inhibits synovial cell overgrowth of synovial cell growth. *J. Clin. Invest.*, 2005, **115**(3), 622–631.
31. Oates, K. M. N., Krause, K. E., Jones, R. L., and Colby, R. Rheopexy of synovial fluid and protein aggregation. *J. R. Soc. Interface*, 2003, **3**, 167–174.
32. Pasquali-Ronchetti, I., Quaglino, D., Mori, G., Bacchelli, B., and Ghosh, P. Hyaluronan–phospholipid interactions. *J. Struct. Biol.*, 1997, **120**, 1–10.

Biolaagrid mehaanika ja bioloogia puutepunktis

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Ülevaates on käsitletud inimese liigeseid, mis mehaanika vaatevinklist on laagrid. Koormatud looduslikud biomaterjalid on seotud väga laiade omavaheliste seoste ja protsessidega, mida on vaja uurida mehaanikute, füüsikute, keemikute ning bioloogide ühiste pingutustega. Biolaagriuuringute põhiliseks takistuseks ongi eri teadusharude erinev lähenemine probleemile. Artiklis on käsitletud liigeste triboloogia, orgaanilise keemia ja kudede bioloogia uudset, sünergeetilist arusaama. Ühtlasi on püütud esitada uusi määrdemehhanisme ja nendevahelisi seoseid.