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# Mechanism of Boundary Lubrication and Wear of Frictionless Synovial Joint

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## Abstract

**The natural synovial joints have excellent lubricating ability to provide very low frictional resistance and high wear resistance during various activities of human being. Synovial fluid is having mainly constituents of glycoproteins and phospholipids. Albumin adsorbs over globulin by hydrophobic bonding after formation of globulin coating on hydrophilic cartilage surface and phosphatidylcholine adsorbs over hydrophilic surface of albumin by the formation of phospholipid bilayer. Different interface of adsorbed proteins and phospholipids layer produces very low shear stress and produces the synovial joint highly slippery and frictionless. Still, synovial joints are effected by Osteoarthritis due to degeneration of cartilage surface. Due to very very slippery boundary lubrication of synovial joint, abrasive and adhesive sliding wear of cartilage surface is not possible. Present study describes the damage mechanism of articular cartilage on the basis of asperity fatigue model. As knee joint bear much more load than that of hip joint during walking, knee joint is early effected in Osteoarthritis. As gradually, cartilage surface detoriates loosing ultimate strength and modulus of elasticity, wear rate increases with aging of human being. It indicates ultimate result of Osteoarthritis at old age.**

**Keyword:** Synovial joint, Boundary lubrication, Asperity fatigue wear, Osteoarthritis

## I. Introduction

Synovial joint is an amazing mechanical bearing created by God. The bone ends are covered with articular cartilage within the synovial joint. The synovial joint is sealed from the surrounding tissues by the synovial membranes and the cavity is filled with synovial fluid. The main purpose of the end surface is to generate very good lubrication producing very low frictional resistance so that subchondral bone could be saved from early damage due to excessive wear of bone to bone contact. According to Charnley, animal joints have very low coefficient of friction of the order of $10^{-3}$ [ 1 ] and very low frictional surface of the synovial joint can be compared as skating over icy surface. However, synovial joint of human being produces rolling motion to perform different common activity like sitting, walking and running etc. Generally, during rolling motion of a wheel on rough surface, friction force is generated at the interface which produces translation of the wheel and torque produces rotational motion of the wheel. Now, as interface of cartilage contact is highly slippery and almost frictionless, as usual conventional rolling motion is not possible in any synovial joint. So, cross connected ligaments and muscles of any synovial joint produces rolling motion ( articulation ) satisfying principle of four bar mechanism which happens prominently, in knee joint [ 2 ]. So, highly slippery lubrication of any synovial joint is possible only by boundary lubrication of synovial fluid. First, McCutchen postulated that when two joint surfaces are compressed together, porous cartilage surface weeps (exudes) fluid which behave as a boundary lubricant. They showed that when small specimens of articular cartilage were rubbed against glass in presence of synovial fluid, the co-efficient of friction was lower than in presence of water [ 3 ]. W H Davis et al. have postulated that lubricating glycoprotein (LGP) is adsorbed to the surface and reduction in surface shear accomplished by formation of hydration shells about the polar surface of the adsorbed LGP creating thin layer of viscous structured water at the surface [ 4 ]. Hill have hypothesized that phospholipid, phosphatidylcholine have better lubricating property and it becomes surface active phospholipid after binding with mobile $Ca^{2+}$ and it will adsorbed with negatively charged proteoglycans of the cartilage [ 5 ]. T Murakami et al. have reported the phospholipid, phosphatidylcholine of 0.01 g/dl as physiological concentration and gamma globulin of 1.0g/dl at higher concentration than the physiological level is effective in lower frictional resistance of the synovial joint. The adsorbed film and underlying gel film of proteoglycan have protective roles against severe loading [ 6 ]. M H Naka etall. have reported that water content and substances present on the articular surface play an important role in lubrication through the formation of a layer with a high water content (hydrated layer) [ 7 ]. The mode of boundary lubrication with integration of lubricating effects of all synovia constituents, phosphatidylcholine, albumin, and, gamma globulin is not yet done to understand good boundary lubricating ability of synovial fluid over articular cartilage surface.

As synovial joint is very slippery and frictionless, so sliding wear of cartilage surface is not

possible. Basic mechanism of cartilage wear could be understood from mechanism of different mode of wear. Abrasive wear mode is not possible as hardness of matting surfaces of cartilages are not different. Wear from cartilage rubbing against cartilage surfaces is not the result of adhesive wear mode since cartilage surface do not cold weld together like metal surfaces when their micro-asperities come in contact. So, there is only possibility of fatigue wear mode of cartilage surface by physiological cyclic compressive joint loading. B B Seedhom has hypothesized that cartilage is living tissue, the threshold at which it fails by either mechanism of high stress or fatigue are determined by human activities and life style [ 8 ]. Triona McCormack et al. postulated that the fatigue mechanism of cartilage induces truama in the cartilage causing a weakening of interfibril connections which link collagen fibril in the matrix, leading to reduction in tensile strength [ 9 ]. Still now, fatigue mechanism of cartilage in vivo is not yet reported elsewhere. On the other hand, A Wang et al. have investigated early wear of UHMWPE of artificial joints by microscopic asperity fatigue mechanism considering cyclic tangential motion under constant normal load [ 10 ]. However, as natural joint provides excellent lubrication ($\mu < 0.005$), so, only, cyclic tangential motion could not be effective in the asperity fatigue mechanism of cartilage surface in vivo due to very low frictional resistance. Here, asperity fatigue of cartilage surface in vivo is considered to occur due to cyclic loading and unloading of Hertzian contact zone by compressive normal load during articulation of a synovial joints.

## II. Theoretical mechanism and formulation

### 2.1 Mechanism of boundary lubrication

Synovial fluid is a protein-lipid colloidal solution and it's main boundary lubricating constituents are Albumin, γ globulin and phosphatidylcholine those maintain colloidal stability through their mutual interaction. Phosphatidylcholine is a natural phospholipid that is available in Soyabin oil. When the phopholipids are exposed to water of synovial fluid, they arrange themselves into a spherical two-layer sheet (a bilayer) with all of their tails pointing toward the centre of the sheet. This assembly process is similar to the coalescing of oil droplets in water and is driven by the same force, called the hydrophobic effect. On the other hand, at the molecular level, the hydrophobic effect is important in driving protein folding and aggregation in water. So, Albumin would aggregate with γ Globulin by hydrophobic bonding producing glycoprotein gel in synovial fluid. So, through mutual interaction of phospholipids bilayer and glycoprotein gel, the protein gel would be encapsulated by spherical phospholipids bilayer like cell membrane [ 15 ] producing colloidal stability in synovial fluid. Now, during rolling or articulation of synovial joint under compressive loading, localized protein-lipid adsorption occurs on hydrated cartilage surface. Mechanism of protein adsorption could be explained from nature of end group and secondary structure of proteins. According to CD spectroscopy, BSA is mainly construct from α helix structure (60-67%) and γ globulin is constructed from β sheet structure [ 11 ]. Also, It is known that when protein adsorbs to a surface, it's a helix structure changes to random coil and/or β sheet structure [ 12 ]. So, in between synovia glycoprotein, albumin and γ globulin, as γ globulin is constructed of β sheet secondary structure, so, naturally, it will adsorb first onto cartilage surface by strong hydrophilic bonding as a coating layer initially. Due to high shear strength of γ globulin [ 12 ], it will form a very rigid beta sheets layer over microscopically, rough surface of cartilage [ 13 ] and during articulation of synovial joint under compressive loading, the asperities of cartilage surface will be deformed gradually through γ globulin coating. However, Secondly, albumin will adsorb over coated γ globulin by natural hydrophobic bonding due to presence of more number of hydrophobic group of albumin. Experimentally, it is observed that both of the protein, albumin and γ globulin prefer to adsorb by hydrophobic bonding and it is reviewed by M Malmsten also that most proteins tend to adsorb more extensively at hydrophobic than hydrophilic protein surfaces due to more limited exchange at hydrophobic interaction [ 14 ]. Albumin will form separate protein layer over γ globulin. Thereafter, phosphatidylcholine of synovial fluid will be adsorbed on hydrophilic surface of albumin by hydrophilic bonding through formation of phospholipid bilayer.

### 2.2 Asperity fatigue wear of cartilage surface

According to Greenwood and Williamson model, two rough flat surfaces could be represented by an equivalent rough surface in contact with a smooth flat surface [ 17 ]. Similarly, the contact between two rough spheres can be analyzed by a physical model of a rough sphere with equivalent radius of both spheres pressed against a smooth flat surface. Generally, rolling occurs in any synovial joint under normal compressive loading and Hertzian contact zone of cartilage surface is repeatedly loaded and unloaded. Due to normal cyclic loading and unloading on Hertzian contact zone, asperities deformation of spherical cartilage surface will be elastic, plastic and elastic-plastic with bulk deformation of curve cartilage surface. Let *n* number asperities will be plastically deformed within Hertzian contact zone. Due to cyclic loading and unloading, fatigue strength of asperities will fall down exponentially. When fatigue strength ( $\sigma_u$ ) of a

asperity equals to Hertzian contact pressure ( p ), a hemispherical wear particle will be formed from tip of the asperity ( as considered in adhesive wear law ).

So, $y\sigma_u = p$

or, $y = \frac{p}{\sigma_u}$ (1)

where, *y* is the fatigue strength factor, which determine the number of wear particles formation.

Let, *x* fraction of *n* umber of plastically deformed asperities will produce *x.n* number of wear particle from the elementary Hertzian contact area and volume of wear particle is $v_p$ .

So, elementary wear volume is proportional to number of wear particles × volume of wear particle

$dV \propto xnv_p$

As number of plastically deformed asperities increases with increment of Hertzian contact area so, n ∝ dA and from the equation (1), it implies that number of wear particle increases with increment of fatigue strength fator, so, x ∝ y

Now, $dV \propto ydAv_p$

Substituting, value of *y* in the above equation taking from eq$^n$ (1), it is obtained

$$dV \propto \frac{p}{\sigma_u} dAv_p \quad (2)$$

Volume of asperity deformation over curve cartilage surface would be proportional with bulk deformation of curve cartilage surface because merging of asperities beside asperities would occur. Simultaneously, the volume of asperity deformation over curve cartilage surface would be inversely proportional with surface roughness as tendency of merging of asperities beside asperities would occur much more if surface roughness is small. So, volume of a hemispherical wear particle from asperity tip under Hertzian cyclic fatigue loading could be written as follows.

$$\text{So, } v_p \propto \frac{a^3}{\sigma^2} \quad (3)$$

Now, elementary wear volume could be obtained by substituting value of eq$^n$ (3) in eq$^n$ (2)

$$dV \propto \frac{a^3}{\sigma_u \sigma^2} pdA$$

So, total volume of wear of curve cartilage surface under Hertzian constant compressive loading (ie a = Const)

$$V \propto \frac{a^3}{\sigma_u \sigma^2} \int_A pdA$$

$$\propto \frac{R}{K\sigma_u \sigma^2} N^2 \quad (4)$$

This is the generalized expression for asperity fatigue wear volume of cartilage for a synovial Joints.

## III. Results and Discussion

### 3.1 Boundary lubrication

K Nakashima et al. have hypothesized that 1:2 ratio of glycoproteins, albumin and γ globulin, constituted synergistic adsorb film for wear reduction of PVA surface under severe loading condition and has reported that equal (1:1) ratio of synovia glycoproteins forms separate protein layer of albumin over γ globulin producing very high wear rate [ 16 ]. It indicates that adsorption albumin on γ globulin is the only possibility to produce very low boundary friction on cartilage surface of synovial joint. Hill have hypothesized that only surface active phopholipid, phosphatidylcholine is a very good boundary lubricant of synovial joint producing low frictional resistance [ 5 ]. Also, T Murakami et al. have reported the observation from pendulum test of porcine shoulder joint. The phosphatidylcholine of 0.01 g/dl as physiological concentration and γ globulin of 1.0g/dl at higher concentration than the physiological level were effective in lower frictional resistance of the synovial joint [ 6 ]. So the phosphatidylcholine have better boundary lubricating property of synovial joint due to weak hydrophobic bonding of the phospholipid bilayer. Afterall, boundary lubrication phenomenon of synovial fluid resembles with boundary lubrication of lamellate Graphite. Hydrophobic bonding of adsorbed glycoproteins bilayer and phospholipids bilayer (like bonding of Graphite lamellae layer) are weak shearing interface producing very good boundary lubricating ability of synovial fluid.

### 3.2 Osteoarthritic cartilage damage

From the final expression of wear volume, it imply that wear volume of cartilage surface linearly depends upon dimensional parameters of joint, radius of bone end ( R ), inversely depends material properties parameters of cartilage, modulus of elasticity and ultimate strength of articular cartilage ( K and $\sigma_u$ ), and square of rms surface roughness of the cartilage ( $\sigma^2$ ), and linearly proportional with square of value of compressive normal joint contact force ( $N^2$ ). For a particular subject, material properties parameters, and surface roughness of cartilage could be considered as constant, so, characteristic of fatigue wear of cartilage surface could be understood from the variation of dimensional parameters of and value of compressive normal load of the synovial joint. Generally, lower limb's synovial joints are affected by Osteoarthritis due to cyclic compressive and tensile loading during human walking. In reality, as knee joint works under more load than hip joint during normal human walking [ 18 ] , it is expected that elastic superfacial layer of knee

cartilage will wear out early than that of hip joint. Possibly, that is why, knee cartilage surface is extra protected by meniscus layer whereas hip cartilage surface have no need of such layer. So, meniscus layer over knee cartilage surface plays very important role to protect the joint from osteoarthritis. If somehow, it is damaged early by any unwanted accident, then the knee cartilage surface will wear out very early period of time unexpectedly. In this regard, it should be noted also that obesity, defined as being 20% over one's healthy weight, places people (particularly women) at increased risk for osteoarthritis due to increment of normal joint loading. However, due to gradual loss of articular cartilage by normal fatigue loading, its material parameters, strength and modulus of elasticity will fall down causing further fast wear rate of cartilage surface with aging of human being. Now, the gradual loss of tide superfacial layer cartilage surface ultimately, causes Osteoarthritis of the any synovial joint. After complete loss of tide superfacial layer where collagen fibers are oriented tangentially, middle layer of cartilage surface would be exposed. Thereafter, randomly oriented collagen fibers of cartilage middle layer would be gradually opened by interstitial fluid pressure during cyclic Heartzian compressive loading and eventually, it leads to Osteoarthritis of the synovial joint.

## IV. Conclusion

Theoretical mechanism of boundary lubrication explains why synovial joints are so slippery and almost frictionless. Mainly adsorption of Albumin over Gamma globulin after formation of globulin coating on hydrophilic cartilage surface produces very very low shear stress and it is enhanced by effect of phospholipids. Abrasive and adhesive sliding wear are not possible causing any wear of frictionless cartilage surface. Asperity fatigue wear of cartilage surface under Heatzian cyclic fatigue compressive joint loading produces gradual loss of tide superfacial layer of cartilage surface and eventually, middle layer of cartilage surface is exposed. And gradually, it is opened by interstitial fluid pressure cyclic compressive loading and resulting Osteoarthritis of the synovial joint.

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