A COUPLED CONTACT MODEL OF CARTILAGE LUBRICATION IN THE MIXED-MODE REGIME UNDER STATIC COMPRESSION

JinJing Liao\textsuperscript{a}, David W. Smith\textsuperscript{b}, Saeed Miramini\textsuperscript{a}, Bruce S. Gardiner\textsuperscript{c}, Lihai Zhang\textsuperscript{a*}

\textsuperscript{a} Department of Infrastructure Engineering, The University of Melbourne, Victoria 3010, Australia
\textsuperscript{b} Faculty of Engineering and Mathematical Sciences, The University of Western Australia, WA 6009, Australia
\textsuperscript{c} College of Science, Health, Engineering and Education, Murdoch University, WA 6150, Australia

* Corresponding author (Email: lihzhang@unimelb.edu.au)

Highlights:

- Coupled contact model considering contact gap and cartilage interaction
- Fluid would exude into the gap and extend mixed-mode duration
- Traditional contact model potentially overestimates the interstitial fluid pressure
- Asperity stiffness and synovial fluid viscosity influence the lubrication effect
Abstract.

This study presents a coupled cartilage contact model, in which the contact gap and cartilage tissue are modelled as two poroelastic systems, linked by pressure and normal flux continuity boundary conditions. Using a tibial plug under indentation as a proof-of-concept model, the predictions support the weeping lubrication theory under static compression. Specifically, the interstitial fluid would exude from the underlying cartilage into the contact gap to extend the mixed-mode duration by >20-fold compared to a no fluid exudation counterpart. Moreover, the traditional contact model, that does not consider the contact gap and cartilage fluid exchange, potentially overestimates the interstitial fluid pressure compared to the proposed coupled model. Parametric studies suggest that the increasing viscosity of synovial fluid prolongs the gap fluid pressurisation, while increasing the asperity stiffness reduces the gap fluid pressure but increases contact gap height.

Keywords: cartilage lubrication; coupled contact model; surface roughness; Gap-cartilage interaction.
1 INTRODUCTION

Articular cartilage is the soft connective tissue covering the ends of long bones of vertebrate animals. It serves as a low friction load bearing surface for articular joints. Normal human knee joint cartilage is very thin (~2 mm to 4 mm) but exhibits remarkable lubrication capability and wear resistance. The initial friction coefficient can be as low as 0.001, which outperforms any current man-made bearings [1]. Furthermore, unlike most man-made metal bearings with very smooth contact surfaces, cartilage is a soft and porous bearing, whose surfaces are much rougher than most man-made metal bearings, with peak asperity heights around 10 μm [2].

To explain the joint lubrication, many theories have been proposed. Knowledge gained from engineering tribology has been applied to study this biological problem. For example, it has been argued that two opposing cartilage surfaces are separated by a relatively thin fluid film, and so hydrodynamic lubrication is responsible for the low friction [31]. The classic squeeze-film model analyses the thin film flow by employing Reynold’s equation, while cartilage is modelled using the equations of poroelasticity and their interactions are captured by continuity boundary conditions [3]. Recent developments have incorporated more complexity, such as a non-Newtonian synovial fluid [4]. The squeeze-film model can predict the load carrying capacity of the film and it is generally concluded that the synovial fluid would flow into the cartilage over at least a portion of the contact surface due to high film pressure. One of the limitations of the squeeze-film model is the assumption of smooth surfaces, as it affects both the time taken for the fluid being squeezed out and for the gap to close [6]. The presence of roughness may make the opposing cartilage surface asperities contact sooner (within a fraction of a second) [7], while still having fluid trapped in the contact gap space. Importantly, once the peak
asperities are in contact, the squeeze-film model is no longer applicable. Therefore, although the squeeze-film model addresses the interaction between fluid and cartilage, it may not be the primary lubrication mode due to its short duration.

Once surface asperities are in contact, the randomly distributed asperities together with the confined synovial fluid form an interconnected pore space, here called the “contact gap” at the contact interface [8]. Thereafter, the cartilage contact is in the so-named “mixed-mode” lubrication regime, where there is a co-existence of hydrodynamic and boundary lubrication [9]. Since joints usually operate in the mixed-mode during normal activities [9], studies have turned to the biphasic nature of cartilage and fluid exchange for answers. McCutchen initially proposed a mechanism called “weeping lubrication” [11], in which the interstitial fluid in the consolidating cartilage exudes into the gap space, reducing solid-to-solid contact. Later, an alternative hypothesis called “boosted lubrication” was proposed by Walker et al. [12], which argued that the synovial fluid trapped between the opposing rough cartilage surfaces would move into cartilage, leaving behind a concentrated hyaluronic acid gel (whose molecule sizes are larger than cartilage pore size) as boundary lubricant. Due to the difficulty in performing *in vivo* experiments and the lack of theoretical or computational models, the lubrication processes during mixed-mode are still not fully understood.

This study presents a coupled model that describes the interaction between contact gap flow and cartilage tissue during mixed-model lubrication in a static compression case (e.g. two-legged stance). The coupled model consists of the cartilage contact gap model and the cartilage tissue model, as shown in Fig.1. The contact gap model describes gap fluid flow and local deformation of asperities. The fluid flow within the contact gap is modelled by Darcy’s law. The local deformation of
asperities during contact is captured by a constitutive equation that has an exponential form. The poroelastic model of cartilage tissue includes the aggrecan concentration dependent permeability, the aggrecan concentration dependent compressive modulus and the collagen network tension nonlinearity. The interactions at the interface between the gap model and cartilage model are enforced by the conservation of mass and the continuity of fluid pressure and normal fluid flux. For reference, an overview of the coupled model is shown in Fig.1.

The aim of this coupled model is to promote a further understanding in synovial joint lubrication by investigating the fluid flow direction at cartilage contact interface. We hypothesise that when considering the effect of surface roughness, weeping lubrication shall be the dominate mode during cartilage mixed-mode lubrication regime under static compression. The case study presented below serves as a “proof-of-concept”, with the purpose to demonstrate that the fluid supplement from cartilage will greatly extend the duration of mixed-mode lubrication. For the problem described below, the simplifications that are made to the problem geometry and the idealisations of complex physiological conditions allow us to concentrate on the very basics of the problem, without being distracted by the many secondary factors operating in vivo within real synovial joints.

2 MATERIALS AND METHODS

2.1 Study overview and assumptions

This study computationally simulates an indentation on large tibial cartilage plugs. In the simulation, the cartilage plug is loaded by vertical compressive load only (without sliding or rolling) by a rigid, horizontal impermeable and perfectly smooth indenter while bathed in synovial fluid (see Fig.2). As it is focussed on the mixed-mode lubrication, the study begins when the cartilage surface asperities and the indenter
first contact. The key quantity of interest in this study is the exchange of fluid between
the synovial fluid in the contact gap and the interstitial fluid in the cartilage during
indentation. This is a short-term biomechanical study, and hence it does not consider
changes, either physiological (e.g. synthesis, remodelling, aggrecan transport),
material (e.g. wear) or biochemical (e.g. solute concentration). A few modelling
assumptions are emphasised below

- At the cartilage and gap contact interface, the fluid pressure and normal flux
  are continuous [4],[30].
- The constitutive relationship between local deformation of surface asperities
  and the contact stress is assumed to be exponential.
- For simplicity, the viscosity of synovial fluid is assumed to be constant (i.e.,
  using 1 Pa·s as the base case as per assumptions made in previous studies
  [7],[15]).

The assumption of viscosity value is based on three reasons. Firstly, it is understood
that synovial fluid exhibits shear-thinning effects, that the viscosity decreases with the
increase of shear rates and covers over 4 orders of magnitude [21]. That is for high
shear rates activities (e.g. running) the viscosity is low, while for low shear rate actions
(e.g. standing), the viscosity is expected to be higher. For typical geometries and
speed during a walking cycle, the shear rate is in a range of 10^2-10^4 s^{-1} [36]. In this
study, we investigate the cartilage lubrication a static compression case (i.e.,
standing), the shear rate is expected to be lower that walking, which is assumed to be
one order lower (at 10 s^{-1}) than walking, therefore, the corresponding viscosity is close
to 1 Pa·s.

Secondly, although there are some previous studies [7][8], the method used to
evaluate the gap permeability in Section 2.3.1 is only from mechanical perspective
(i.e., it only considers the obstruction effect of the deformed roughness on the flow).

However, in reality, there are “polymer brushes” (e.g. hyaluronic acid and aggrecan) attached to the surface [39], the polymer brushes have special properties that prevent water being squeezed from between the asperity contacts (protein-adsorbed film in hydration lubrication [39]), which would decrease the gap permeability but it is not captured in the modelling. Therefore, a relatively higher viscosity value may be adopted to arguably capture this effect.

Thirdly, although there are no experiments looking into direct measurements of viscosity and shear rate in the cartilage contact interface, some indirect measurements may shed some light on this issue. Myant and Cann [36] quantified the lubrication film thickness using an optical interferometric tribometer. With an analytical equation for elastic-isoviscous contact derived by Hooke [40], they back estimated the fluid viscosity against the sliding speed. Results showed that compared to the physiological sliding speed for walking (100 mm/s [43]), at relatively low entrainment speed (2-3 mm/s) the viscosity could reach higher than 1Pa·s because the aggregation of protein molecules could form a gel, due to which the in-situ viscosity values in the contact gap could be very different to the bulk solution. By comparison, it is reasonable to assume that under a static compression case (i.e., two-legged stance) which is investigated in this study, the entrainment speed and shear rate would be at low level, and hence a relatively higher viscosity may be assumed. In light of this, 1 Pa·s is adopted as the base case to be consistent with previous study [15]. In addition, a parametric study is performed in Section 3.3.1 to discuss the lubrication effect of different viscosity values.
2.2 Governing equations of cartilage tissue

2.2.1 Poroelastic model

Cartilage is a fully saturated porous material that exhibits biphasic characteristics (fluid and extracellular matrix). Fluid flow inside the cartilage is governed by mass conservation and Darcy’s law shown in Eq. (1) and Eq. (2) respectively [2].

\[
\frac{\partial (\varphi_f \cdot \rho_f)}{\partial t} + \nabla \cdot (\varphi_f \cdot \rho_f \cdot \mathbf{u}_f) = 0,
\]

\[
\frac{\partial (\varphi_s \cdot \rho_s)}{\partial t} + \nabla \cdot (\varphi_s \cdot \rho_s \cdot \mathbf{u}_s) = 0
\]

where \( \mathbf{u}_d \) is the Darcy’s velocity inside the cartilage defined as the relative velocity between the interstitial fluid \( \mathbf{u}_f \) and the solid matrix \( \mathbf{u}_s \). \( \rho_f, \rho_s \) are the intrinsic density for the interstitial fluid and the solid matrix respectively. \( \varphi_f, \varphi_s \) (\( \varphi_f + \varphi_s = 1 \)) are the volume fraction for the interstitial fluid and the solid matrix respectively. \( K_c \) is the hydraulic permeability tensor of cartilage. \( p_c \) is the excess pore pressure of the interstitial fluid in the cartilage.

The force equilibrium state of the cartilage is governed by the momentum conservation equation. Neglecting the body forces and inertia forces, the momentum conservation equation is given by [2],

\[
\nabla \cdot \mathbf{\sigma}_t = 0
\]

where \( \mathbf{\sigma}_t \) is the total applied load. Due to the biphasic nature of cartilage, the total stress in cartilage is resisted by the interstitial fluid excess pore pressure \( p_c \) and the the incremental elastic stress of the solid phase \( \mathbf{\sigma}^e \),

\[
\mathbf{\sigma}_t = \mathbf{\sigma}^e_c + p_c \mathbf{I}
\]
The constitutive equation between the cartilage solid phase effective stress $\sigma^E_s$ (in Cauchy stress form) and the deformation gradient of the solid phase $F^s$ is given below [13],[32],

$$\sigma^E_s = \frac{2}{J^s} F^s : \frac{\partial W^s}{\partial C^s} : F^{sT}$$

(5)

where $W^s$ is the Helmholtz energy per unit volume stored in the solid (i.e., strain energy density). $C^s$ is right Cauchy-Green deformation tensor of the solid phase. $J^s$ is volume ratio of the solid phase, $J^s=\det(F^s)$.

### 2.2.2 Aggrecan and collagen dependent features

Apart from poroelasticity, cartilage also exhibits additional mechanical behaviours, such as aggrecan dependent permeability, aggrecan dependent compressive modulus and tension-compression nonlinearity. The theoretical model developed in Zhang et al. [13] and Miramini et al. [14] has captured these aspects, in which the cartilage cyclic strain of Zhang et al. [13] has been verified against an in vitro measurement [27]. Their models [13],[14] are adopted for the cartilage modelling in this study.

(a). Aggrecan concentration dependent compressive modulus

Aggrecan is negatively charged and hence contributes significantly to the compressive stiffness of the cartilage tissue. A quadratic relationship between the cartilage aggregate modulus $H_{-A}$ and its “actual” aggrecan concertation $\Phi_G$ was established through experimental studies [18] and shown below, in which $\alpha_1$ and $\alpha_2$ are empirical constants taken as 0.25 MPa and 0.0155 MPa respectively [13].

$$H_{-A} = \alpha_1 \Phi_G + \alpha_2 \Phi_G^2$$

(6)

With that the above expression, cartilage elastic compressive modulus $E_c$ can be found using [2],
where \( \nu \) is the aggrecan effective Poisson's ratio.

It should be emphasised that the “actual” aggrecan concentration varies with cartilage temporal volumetric change. More specifically, it is the concentration of the local aggrecan volume excluded by the volume occupied by the collagen, as given by:

\[
\Phi_G(t) = \frac{\Phi_{G0}}{J(t) - \alpha}
\]

where \( \Phi_{G0} \) is the initial “apparent” aggrecan concentration. \( \alpha \) is the volume fraction of the collagen network. Following [14], here \( \alpha \) has the value of 45%, 30% and 25% in the superficial, middle and deep zones, respectively.

The apparent aggrecan concentration is depth dependent and its profiles have been mapped by magnetic resonance imaging (MRI) [28],[29]. It is shown that the concentration is relatively low at the surface of the cartilage and increasing linearly with depth to the deep zone. In this study, aggrecan concentration at \( z=0 \) mm (see in Fig.2) is taken as 25 mg/ml [29] and 120 mg/ml at \( z=-3 \) mm [28]. The values in between are obtained by linear interpolation.

(b). Tension-compression nonlinearity

The collagen network is responsible for the tension and shear resistance of the cartilage. As the fibril orientation and concentration vary with depth, the tensile and shear modulus of the cartilage also vary with depth. In this study, the cartilage is divided into three zones, delineated at 5% and 45% of the cartilage thickness, as detailed in Table 1. The same compressive and shear modulus values used by Miramini et al. [14] are adopted in this study and shown in Table 1.
Table 1 – Summary of cartilage location dependent tensile and shear moduli [14]

<table>
<thead>
<tr>
<th>Cartilage zones</th>
<th>Tensile modulus (MPa)</th>
<th>Shear modulus (MPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial zone (0mm ≤ z &lt; -0.15mm)</td>
<td>100</td>
<td>25</td>
</tr>
<tr>
<td>Middle zone (-0.15mm &lt; z ≤ -1.35mm)</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Deep zone (-1.35mm &lt; z ≤ -3mm)</td>
<td>10</td>
<td>15</td>
</tr>
</tbody>
</table>

(c). Aggrecan concentration dependent permeability

As aggrecans is negatively charged, their resistance to the fluid flow inside the cartilage shall also be modelled. The concept of the “actual” aggrecan concentration in Eq. (8) is also utilised to describe the hydraulic permeability $K_c$. Calibration to the experimental observations by Zamparo and Comper [19] provides,

$$K_c = \frac{n \cdot (\Phi_0)^m}{\eta}$$

where $n$ and $m$ are empirical parameters taken to be $5.4 \times 10^{-22}$ m² and -2.37 respectively [14]; $\eta$ is the viscosity of water at 37°C (0.0007 Pa·s).

2.3 Governing equations of the contact gap

The contact gap model describes gap fluid flow and local deformation of asperities. The fluid flow within the contact gap is modelled using Darcy’s law, in which the gap permeability is numerically estimated for an experimentally measured bovine cartilage surface roughness. The closure of the gap space and fluid exchange between gap and tissue are modelled by the conservation of mass in the contact gap. The local deformation of asperities during contact is captured by a constitutive equation that assumed as an exponential form.

2.3.1 Gap permeability

The fluid flow in the contact gap is modelled with Darcy’s law. As the height of the asperities are very small (i.e. around 10 μm) compared to the dimensions of the
cartilage, it is reasonable to model the flow as a one-dimensional problem. Darcy’s law for the gap is shown below,

\[ v_d^g = -K_g \cdot \nabla p_g \]  \hspace{1cm} (10)

where \( v_d^g \) is the fluid Darcy velocity; \( p_g \) is the fluid pressure of in the contact gap; \( K_g \) is the gap permeability, which is a measure of the ability for the contact gap to allow the fluid to flow laterally. The gap permeability has been numerically evaluated by previous studies [7],[8] by developing a computational fluid dynamic model. Similar methodology is adopted in this study and briefly described below and is depicted in Fig.3.

Firstly, cartilage surface topography (1000×1000 μm) of bovine medial tibia measured by Dektak stylus profilers in our previous study is adopted here [8]. The roughness profile is then used in a computational fluid dynamics (CFD) model to form a micro-scale representative elementary volume (REV) for the gap flow. The CFD models are created in COMSOL Multiphysics (version 5.3, COMSOL, Inc.) by free tetrahedral elements (900816 elements in total including 214616 boundary elements). A mesh sensitivity analysis has been performed and the results indicate that the differences of averaged flow velocity yielded by different mesh size are less than 1%. The asperity level deformation is conveniently approximated by just lowering down the indenter plane (upper surface in Fig. 3) to intersect with the asperities at varies gap heights (i.e., \( h=9, 7, 5, 3, 1 \) and 0.3 μm), which is defined as distance between the indenter and roughness centre line (i.e., \( z=0 \) shown in Fig.2 and Fig.3) that covers the positive asperity part of the cartilage surface roughness, because the Poisson’s ratio of the cartilage extracellular matrix often approaches zero. The fluid flow in the gap micro-scale REV model is assumed to be isothermal, laminar, incompressible and governed by the Navier-Stokes equations. Analysis are
The CFD models are solved at various gap heights to obtain the volume-averaged velocity. Finally, the gap permeability is obtained by equating the volume-averaged velocity of the micro-scale REV model at each height to the macro-scale Darcy velocity in Eq. (10). Since constant viscosity is assumed for the synovial fluid, gap permeability is only related to the gap height (i.e., $K_g = K_g(h)$), their relationship is captured by a trendline approximation function shown in Fig.3.

2.2.2 Fluid exchanges between gap and cartilage

The fluid exchange between the contact gap and the cartilage tissue is modelled by the mass conservation law below,

$$\frac{\partial \varepsilon^{g}}{\partial t} + \nabla \cdot v^g = s$$  

(11)

where $\varepsilon^{g}$ is the volumetric strain of the contact gap (pore space and asperities), as the fluid exits surface asperities deform (i.e., gap closure). $t$ is the time and $s$ is the fluid generation or removal from the gap per unit volume per unit time through exchange with the underlying cartilage.

It should be emphasised that the fluid exchange can be a “source” term (i.e., $s>0$, fluid acquisition for the gap) or a “sink” term (i.e., $s<0$, fluid loss for the gap) depending on the fluid pressure difference between the gap and cartilage tissue. In other words, if the interstitial fluid in the cartilage tissue weeps into the gap, $s$ is a source term. Alternatively, if the pressurised synovial fluid ultrafiltrates into the cartilage tissue, $s$ is a sink term. In this way, the theories of weeping and boosted lubrication can be examined.

The volumetric strain of the gap $\varepsilon^{g}$ is related to the reduction of gap height $h$, a constitutive relationship between the gap closure and the corresponding stress state of the asperities is proposed below.
2.2.3 Constitutive equation for gap height and contact stress

As shown in Fig.2, the total applied loads \( \sigma_t \) in Eq. (3) is resisted by the asperity contact stress \( \sigma_c \), and the fluid pressure in the gap \( p_g \). When contact initiates (indenter touching the highest asperity), \( \sigma_t \) is initially resisted by \( p_g \) only (i.e., \( \sigma_c = 0 \)). As the surface asperities start to deform, the gap space is reduced, squeezing out the fluid, and hence the contact stress \( \sigma_c \) will rise to take up the total applied load as indicated below.

\[
\sigma_t = \sigma_c - p_g
\]  (12)

It is assumed that an exponential constitutive equation can describe the relationship between gap closure and the contact stress, according to the stress-strain (or contact pressure vs. axial displacement) responses obtained from confined and unconfined compression experiments on human cartilages [16],[17]. As shown in Fig.2, this exponential form is trying to account for the expectation that as the contact stress increases, more asperities come in contact to resist the gap closure.

\[
h = h_0 e^{\sigma_c/\beta} = h_0 e^{(\sigma_c + p_g)/\beta}
\]  (13)

where \( h_0 \) is the highest undeformed asperity height from the roughness centre line, which equals to the peak asperity height \( R_q \) of the roughness. In this study, it is taken as \( h_0 = 9 \) μm as shown in 3.

The parameter \( \beta \) represents the stiffness of cartilage asperity. A smaller value of \( \beta \) means the asperities are “softer” and hence deform faster. Due to the small dimensions of the asperities, it is difficult to quantify this parameter experimentally, however, the compressive stiffness is provided by the negatively charged proteoglycan aggregates. The aggregate modulus \( H_A \) represents the stiffness of the tissue at drained state [26] as given in Eq. (6). It is reasonable to assume the asperity stiffness is a fraction (<1) of the aggregate modulus at the upmost layer of the cartilage.
(i.e., contact interface at \(z=0\) mm in Fig.2). Therefore, in this study, \(\beta\) of 20% \(H_A\) is taken as the base case for investigation [38]. In addition, a parametric study of \(\beta\) is presented in Section 3.3.2 to discuss the influence of \(\beta\) on cartilage lubrication.

### 2.3.4 Governing equation in cylindrical coordinates

This study applies the governing equations presented above to investigate a 2D contact gap problem in the cylindrical coordinate system as shown in Fig.2. The integral of \(\nabla \cdot \mathbf{v}_g\) in Eq. (11) over the contact volume represents the rate of fluid volume out of the contact gap from the periphery, with higher order terms neglected, it equals to,

\[
\int_V \nabla \cdot \mathbf{v}_g \cdot dV = \int_V \mathbf{v}_g \cdot dS = (v_{g\theta} + \frac{\partial v_g^r}{\partial r} dr) \cdot (r + dr) d\theta \cdot h_0 - v_g^r \cdot rd\theta \cdot h_0
\]

\[
= v_g^r \cdot dr \cdot d\theta \cdot h_0 + r \frac{\partial v_g^r}{\partial r} \cdot drd\theta \cdot h_0
\]  (14)

The integral of \(\partial \varepsilon^g / \partial t\) in Eq. (11) represents the rate of decrease of pore space volume in the gap,

\[
\int_V \frac{\partial \varepsilon^g}{\partial t} dV = - \frac{rd\theta \cdot dr \cdot dh}{dt}
\]  (15)

The integral of \(s\) in Eq. (11) represents the rate of fluid volume exchanged between the gap and the cartilage,

\[
\int_V s dV = rd\theta \cdot dr \cdot u_{dv}^c
\]  (16)

where \(u_{dv}^c\) is the vertical component of Darcy velocity from the cartilage at the contact interface.

Substitution of Eqs. (14-16) into Eq. (11) gives the governing equation for the contact gap in cylindrical coordinates.

\[
e^{(\sigma + \rho_e)/\rho} \frac{dp_g}{dt} = \frac{\partial}{\partial r} K_g \frac{\partial p_g}{\partial r} + \frac{K_g}{r} \frac{\partial p_g}{\partial \theta} + \frac{u_{dv}^c}{h_0}
\]  (17)
2.4 Boundary and initial conditions

A few boundary conditions are imposed to couple these two sets of governing equations together. The individual lateral/medial contact region of tibia plateau can be roughly assumed to be axisymmetric at \( r = 0 \) mm based on the images of Goodwin et al. [20].

For the fluid flow within the gap, the fluid pressure at the gap periphery is the reference ambient fluid pressure, taken here as zero [4].

\[
p_g(r, z) = p_g(L, 0) = 0
\]

(18)

Similarly, for the sides of cartilage disc, a free flux condition is also assumed at the perimeter edge [5].

\[
p_c(r, z) = p_c(L, z) = 0
\]

(19)

On the indenter, a static uniformly distributed load (UDL) \( \sigma_t \) is applied to both the contact and the cartilage. At the interface between the contact gap and the cartilage top surface, the continuity of fluid pressure [30] and normal fluid velocity [4] are imposed.

\[
p_c(r, 0) = p_g(r, 0)
\]

(20)

The continuity of normal fluid velocity \( u_n \) has already been accounted for as the source term in Eq. (17).

At the bottom surface of the cartilage, a fixed, rigid and impervious subchondral bone substrate is applied to the model.

As shown in Fig.2, the initial conditions for this problem is when the indenter and the peak asperity of cartilage surface roughness are just in contact, at this instant the surface asperities are undeformed with the initial peak height as \( h_0 \) defined in Eq. (13), the total applied loads \( \sigma_t \) is resisted by the fluid pressure in the gap \( p_g \). Therefore, the following initial condition is specified.
At \( t = 0 \), \( p_g = -\sigma_t \), \( h = h_0 \) \hspace{1cm} (21)

2.5 Numerical modelling

The governing equations are implemented in COMSOL Multiphysics (version 5.3, COMSOL, Inc.). The geometry and dimensions of the model are shown in Fig. 2, and is based on MRI images [20]. Cartilage thickness gradually decreases towards the tibia plateau edge. To capture this feature, cartilage thickness at the centre and edge are 3 mm and 1 mm respectively. The cartilage thickness is kept constant close to the centre and curved upwards to the edge at \( r = 2/3L \). The model is meshed by 1896 triangular elements.

The load \( \sigma_t \) is kept constant at 1 MPa (i.e., 314 N) and applied for one hour (3600 s). This is a relatively light load for cartilage, which is equivalent to a person of 64 kg standing for one hour, assuming the load is equally distributed to lateral and medial tibia condyles. The governing equations are solved by the time-dependent implicit solver with a relative tolerance of \( 10^{-3} \).

3 RESULTS AND DISCUSSION

For results, firstly, we use a base case with synovial fluid viscosity \( \eta_0 = 1 \text{Pa}\cdot\text{s} \) and asperity stiffness \( \beta = 20\%H_A \) as an example to investigate the mutual effects of contact gap and cartilage on each other. Then, parametric studies are performed to evaluate the effects of various synovial fluid viscosity and asperity stiffness on lubrication.

3.1 Effects of cartilage tissue on contact gap

This section presents the impact of cartilage tissue deformation on the gap by studying their interactions at the contact interface. The relatively permeability between the gap and cartilage tissue is likely to have a profound impact on the fluid direction under the same pressure condition at the same location. For example, if the gap
permeability is lower than the tissue, synovial fluid could be “pooled” among the surface asperities or even flow into the cartilage [2]. In this study, during the one-hour contact, the tissue permeability at the upmost layer (z=0 mm) has decreased by 62% (from $5 \times 10^{-15}$ m$^2$/Pa·s to around $1.5 \times 10^{-15}$ m$^2$/Pa·s). The ratios of the gap permeability $K_g$ over tissue permeability of cartilage upmost layer $K_c$ at two locations ($r=0$ and 5 mm) are shown in Fig.4 (a). The model predicts that the gap permeability is always greater than the tissue permeability during the contact period, the permeability ratio $K_g/K_c$ drops from initial contact (around 1200) to the steady state (around 40) after 30-40 minutes of contact.

The vertical component of the Darcy velocity of fluid phase in the cartilage's upmost layer (z=0 mm), i.e., through the cartilage surface, is shown in Fig.4 (b) at the locations of $r=0$ and 5 mm. As shown, throughout the contact period the Darcy velocities remain positive, which means that the $s$ term in Eq. (11) remains as a source term, i.e., interstitial fluid flows into the contact gap. This is likely due to the higher permeability in the gap discussed above. Furthermore, it is also noted that at both $r=0$ and 5 mm, curves exhibit peaks at less than 5 min of contact. In each case, the peaks approximately correspond to the same permeability ratio of 300 in Fig.4 (a). When the load is initially applied, there should be negligible fluid pressure gradient between the cartilage top surface and the gap, therefore, the vertical component of Darcy velocity is close to zero. Afterwards, due to the large permeability in the gap, gap fluid will exit at the periphery rapidly and cause the gap fluid pressure gradient to fall, so that the interstitial fluid inside the cartilage at higher pressure begins to weep into the gap to compensate the fluid loss from the contact gap. As the contact gap fluid pressure falls further, the gradients between gap and cartilage steepens, and thus the vertical Darcy velocity increases. It is shown that the vertical Darcy velocity at $r=5$ mm peaks before
This is because the gap fluid at \( r=5 \) mm drains before \( r=0 \) mm. After 30 minutes of contact, the permeability ratio decreases to the steady state and correspondingly the flow from cartilage to the gap slows and remains relatively constant at small magnitudes.

When interstitial fluid flows into the contact gap during contact, it will contribute to the joint lubrication by extending the duration of mixed-mode. Fig.5 (a) compares the fluid pressure in the gap when fluid exchange with the cartilage can and cannot occur. Without fluid supplement, the gap fluid pressure will decay to zero after just 3 minutes, after which the boundary lubrication stage commences (which is usually associated with high friction and surface wear). However, because of the fluid supplied to the contact gap from the permeable cartilage underneath, the duration of mixed-mode lubrication of the contact gap can be substantially extended to at least one hour. This result emphasises the importance of the surface asperities, the porous nature of cartilage, and the process of cartilage expelling fluid and slowly consolidation (usually measured in hours for normal human knee joints). It is this slow process of weeping fluid into the gap that enables a prolonged and fluid dominant mixed-mode lubrication. Therefore, the benefit of weeping lubrication is that it couples the duration of the fluid dominant mixed-mode lubrication to the consolidation time for cartilage [2].

The nominalised total fluid load support and asperity contact force (both integrated over the contact area) are shown in the primary axis of Fig.5 (b). It is seen that the total gap fluid support decreases with time (to 20% after one hour) while asperity contact force increases correspondingly. As the gap layer is also similar to a poroelastic (biphasic) system, the effective start-up friction coefficient \( \mu_{\text{eff}} \) can be estimated by using the theoretical formula derived by Ateshian [10],

\[
\mu_{\text{eff}} = \mu_{\text{eq}} \cdot (1 - W_f) \tag{22}
\]
Where $W_f$ is the fluid load support fraction and $\mu_{eq}$ is the equilibrium friction coefficient (~0.3 [10]). The start-up friction coefficient is plotted in the secondary axis of Fig.5 (b), it is shown that the start-up friction coefficient increases with the static loading time to 0.24 after one hour due to the fluid exuding out of the contact gap. The resulting trend corresponds reasonably well with the experimental observation of Accardi et al. [37], in which cartilage samples were statically loaded (by 1.2-1.8 MPa) at various time before the sliding tests, in which friction coefficient would reach 0.3 after 1 hour of static loading. It is also important to note that the friction coefficient will drop rapidly once the movement (sliding) commences, take the study of Accardi et al. [37] for example, friction coefficient dropped by more than 30% in less than 60 s after the commence of the sliding test. Furthermore, it should be emphasised that phosphate buffered saline was used as lubricant in Accardi et al.'s study [37], while using synovial fluid (bovine) the equilibrium friction coefficient could be much lower (~ 0.1) [45].

Although the results in this study support weeping lubrication, it does not necessarily mean that boosted lubrication does not occur under physiological conditions. It should be emphasised that this study is limited to a static loading condition in the mixed-mode, such as two-legged stance. In different activities, more than one lubrication mechanism may be activated [35], including boosted lubrication. For example, Moore and Burris [34] carried out start-stop sliding tests of cartilage against-glass. They concluded that sliding could enhance the transport of trapped fluid in the contact interface into the underlying cartilage leading to the recovery of the interstitial fluid lost in static loading as the hydrodynamic pressure exceeded the interstitial fluid pressure in sliding condition. Graham et al. [33] also found that sliding could also enhance solute transport in cartilage. In addition, boosted lubrication may also occur in a standing up case known as the “squeeze-film” action [3] before the
asperities are in contact, which has been discussed in the introduction, the sudden applied load due to the action of standing up (from sitting), would give a momentum for the synovial fluid to ultrafiltrate into the cartilage tissue. The ultrafiltration would also increase the concentration of hyaluronic acid and give rise to an increase in viscosity of the remaining synovial fluid [44].

3.2 Effects of contact gap on cartilage tissue

This section discusses the effects of gap flow on cartilage tissue by studying the total fluid exudation volume and interstitial fluid pressure of cartilage. In order to show the impact, two additional cases are compared to the coupled contact model. That is, in addition to having a model of a contact gap due to rough surfaces (i.e., “coupled contact model” case), we introduce two extreme cases, they are “free flux” and “no flux” at contact interface. Specifically, the free flux case allows for a zero pressure boundary at the contact interface (i.e., \( p_c(r,z) = p_c(r,0) = 0 \)), which may be considered as an example of a cartilage with surface defects. The “No flux at interface” case restricts normal fluid outlet at the contact interface (i.e., \( -\mathbf{n} \cdot \mathbf{u}_d = 0 \)), which is traditionally used to model cartilage smooth contacts [14],[22].

The total fluid exudation volume of the three cases are plotted in Fig.6 (a), for the coupled contact model and the free flux model, total fluid exudation is the mathematical summation of top (contact interface) and side (perimeter) surface fluid exudation, while for the no flux model, total fluid exudation equals to the side exudation for it is the only outlet channel. The detail top and side fluid exudation volumes are also summarised in Table 2. It is seen that 91% of the fluid escapes through the top surface in the free flux model. This is because for an isotropic tissue permeability the drainage path to the top surface is much shorter than to the side surface of the cartilage plug. In comparison, the no flux case, which artificially
restrains fluid outlet through top surface, may be unrealistic. The assumption of no
flux at contact interface is made with a premise that the squeeze-film action has
depleted the thin lubrication film between the perfectly smooth cartilage contact
surfaces, and hence there cannot be any fluid outlet through the contact interface
since no lubrication film remains there [22]. However, when considering the surface
roughness, there will always be fluid confined in the interconnected space formed
by the roughness. Therefore, the results of the coupled gap contact model seem to
be more reasonable that certain amount of fluid (19% in this study) would still flow
into the contact interface through cartilage top surface. It should be emphasised
again that this study is a proof-of-concept, and hence the top exudation proportions
would vary with different assumptions. In fact, the percent exuded might even be
greater than predicted here. For example, a recent experimental study has found
out that the cartilage tissue permeability is anisotropic under compression [23].
Specifically, a collapsing trellis-like fibrils of cartilage tissue could decrease the
lateral porosity without altering the axial porosity, which indicates that the lateral
permeability would become ~1000 times smaller than the axial permeability under
compression [23], so that more fluid would go through the top surface and aid the
weeping lubrication.

Table 2 – Summary of fluid exudation of the three models

<table>
<thead>
<tr>
<th>Cases</th>
<th>Side exudation (ml) (Proportion of total)</th>
<th>Top exudation (ml) (Proportion of total)</th>
<th>Total exudation (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free flux at interface</td>
<td>0.012ml (9%)</td>
<td>0.117ml (91%)</td>
<td>0.129ml</td>
</tr>
<tr>
<td>Coupled contact model</td>
<td>0.087ml (81%)</td>
<td>0.020ml (19%)</td>
<td>0.107ml</td>
</tr>
<tr>
<td>No flux at interface</td>
<td>0.098ml (100%)</td>
<td>\</td>
<td>0.098ml</td>
</tr>
</tbody>
</table>

Since the total fluid exudation volume of the proposed coupled contact model
is more than the traditional contact model, the interstitial fluid pressure inside the
cartilage is also reduced. A comparison of the three cases are plotted in Fig.6 (b)
at the location close to the middle of the cartilage (r=0, z=-1.35 mm). In the free
flux case, rapid fluid loss through top surface causes a rapid decay of interstitial fluid pressure, such that it has reached its equilibrium state in less than 30 minutes. On the contrary, the interstitial fluid pressure of the traditional model remains elevated (at 0.49 MPa even after one hour) because the distances of fluid drainage paths are artificially increased (i.e., the diameter of a cartilage disc is greater than the thickness). In comparison, the fluid pressure predicted by the proposed coupled model is 0.38 MPa after one-hour contact.

The interstitial fluid pressure inside the cartilage is important, as biphasic lubrication theory attributes the low friction to the interstitial fluid pressurisation [10]. Biphasic lubrication theory established a linear regression relationship between interstitial fluid pressurisation and friction [24], regardless of the fluid flow direction at contact interface [10]. The biphasic lubrication theory has some similarities to weeping theory for both of them emphasise the importance and dominance of fluid load support in the joint lubrication that minimises direct solid shearing. However, the biphasic lubrication tends to overlook the effects of surface roughness. This study can be seen as a supplement to the biphasic lubrication that when considering the roughness, the two theories (weeping and biphasic) may represent joint lubrication in different contact stages. During the initial contact of surface asperities, the surface roughness forms a soft porous gap space, which is in the fluid and pressure continuum space connected to the porous cartilage tissue. This study shows that when considering the effects of roughness under compression, fluid would exude from the cartilage to the contact gap to maintain the fluid support at contact interface and hence weeping lubrication dominates. However, this is come at a cost that the exudation reduces the consolidation time for the underlying
cartilage. Therefore, the traditional contact model with perfectly smooth surface assumption may potentially overestimate the interstitial fluid pressures.

With the contact continues, gap permeability gradually decreases as the gap closes up to around 250-500 nm [41], the contact reaches a surface amorphous layer (SAL), which is comprised by sulphated sugars, glycoproteins and lipids [35]. This layer is not modelled in this study, but has been studied as a biphasic layer with the assumption of same order of permeability, more water content and lower elastic modulus compared to the underlying cartilage tissue [38]. Their results indicated that the SAL would alter the load carriage of the underlying cartilage that slow down the increase of solid support [38]. Therefore, when it reaches SAL, it is believed that the flow exudation would gradually cease, the interstitial fluid pressure at the upmost layer of cartilage tissue would support the load and hence the biphasic lubrication would dominate until interstitial pressure subsides.

3.3 Parametric Study

3.3.1 Effects of the gap fluid viscosity

This section investigates the effect of gap fluid viscosity on cartilage lubrication. The viscosity of healthy synovial fluid can vary over 3-4 orders of magnitude at different shear rates [21]. Under the same asperity stuffiness ($\beta=20\%H_A$), three orders of constant viscosity magnitudes are considered: $\eta=1, 0.1, 0.01$ Pa·s, corresponding to measured viscosities at shear rates order of 10, 100, 1000 s$^{-1}$ respectively, which covers a wide range of physiological activities [21]. The gap fluid pressure at the axis of symmetry of contact ($z=0$ mm, $r=0$ mm) are plotted in Fig.7. This figure shows that synovial fluid viscosity plays an important role in cartilage lubrication, with higher synovial fluid viscosity prolonging hydrodynamic lubrication. For example, with $\eta=1$ Pa·s as reference, a 10-fold decrease in gap viscosity would
cause a more rapid decay in the gap pressure, only being 18% of the reference value after one hour of contact (i.e., from 0.38MPa of $\eta=1$ Pa·s to 0.07MPa of $\eta=0.1$ Pa·s).

For the case of $\eta=0.01$ Pa·s, the gap fluid pressure drops to almost zero in less than half an hour.

### 3.3.2 Effects of asperity stiffness

A parametric study is performed in this section to investigate the effect of asperity stiffness on cartilage lubrication. In this model, the initial aggregate modulus at the upmost layer is 0.69MPa, which is within the typical range of human cartilage (0.5-0.9MPa) [25]. Three cases are considered: $\beta=20\%$, 50% and 100% of $H_A$, under the same viscosity (1 Pa·s). It should be noted that once coupled, the asperity stiffness will also be affected by the “actual” aggrecan concentration such that it will become increasingly stiffer under further compression.

The nominalised gap height and gap pressure at contact centre ($z=0$ mm, $r=0$ mm) are shown in Fig.8. As expected, the stiffer asperities result in greater equilibrium gap heights ($h=4.3$, 2.2 and 0.52 μm for $\beta=20\%$, 50% and 100%$H_A$ respectively). However, a reversed trend is obtained for gap fluid pressure (and so the lubrication capability), with the gap fluid pressure of stiffer asperities falling much more quickly than softer asperities. For example, although the gap height of $\beta=100\%H_A$ is only reduced by half at 30 minutes, the corresponding gap pressure has decreased by 93%. This is because the majority of the applied load will always be distributed to the components with higher stiffness. Therefore, it is suggested that a relatively soft asperity may aid weeping lubrication better.

The above results may also shed some light on the question that why the cartilage surface is so rough. The presence of permeable and deformable roughness together with synovial fluid forms something similar to a very thin porous “cushion bearing” (i.e.,
the porous contact gap) above the cartilage tissue. This bearing to some degree also
has the biphasic characteristics that fluid phase will initially carry the applied load, and
the fluid supply from the cartilage below helps to extend the duration of mixed-mode
lubrication of the bearing.

3.4 Limitations

There are some important limitations in this study that are noted here. From
physiological point of view, firstly, the problem geometry is simplified and idealised.
For example, a real knee joint is not perfectly axisymmetric and flat at the top surface.
Secondly, this study simulates a static in vitro indentation, however, for a real joint with
a meniscus, the static contact loading tends to be parabolically-distributed (rather than
uniformly distributed) when standing still. Furthermore, when walking and running, the
joints work in a reciprocal motion that results in a combined dynamic compression and
shear loading on the cartilage surfaces. Therefore, more realistic loading conditions
will need to be considered in future studies. Thirdly, as stated earlier, this study
assumes constant viscosity for synovial fluid, but in fact the non-Newtonian behaviour
of the synovial fluid will also affect the fluid exchanges during cartilage contact. Lastly,
the geometry of fluid flow model in the contact gap is also very elementary. In reality
there may exist more than one flow path, i.e. between the contact surfaces of femur
and tibia cartilages, and between of femur and meniscus and tibial surface and
meniscus.

For the theoretical modelling, it should be noted that two key assumptions are
made for the uncertainties of synovial fluid viscosity and asperity stiffness. For the
viscosity, a relatively high value is assumed here because the focus of this study is to
investigate the joint lubrication in a static compression condition (e.g., standing), where
shear rate is expected to be low. However, under high sliding speed the viscosity may
decrease to close to water, in which the load effects (impulse compression and shear) and the responses of cartilage tissue could also change. As a combination of various lubrication modes could exist during physiological conditions, further experimental and theoretical studies are required. Synovial fluid is a complex solution that makes it difficult to model mathematically, before it reaches the steady state, the shear-thinning effects are spatially and time dependent in the contact interface (i.e., viscosity varies with locations and time as well). In addition, the protein aggregation mechanism (entanglement of the tenuous protein network with the long-chain hyaluronate [42]) during contact would make the in-situ viscosity higher than its bulk phase values obtained from viscometer [36]. Therefore, more advanced synovial fluid model is required. For the asperity stiffness, it is reasonable to assume the asperities exhibit biphasic characteristics similar to that of cartilage tissue. However, further experimental works are required to determine the time and spatially dependent asperities stiffness during the contact process. This study presents a first step towards understanding the effects of cartilage surface roughness on the lubrication behaviour of joints under static loading.

4 CONCLUSIONS

The present study numerically investigates the lubrication behaviour of cartilage in the mixed-mode by modelling fluid flow exchange between two poroelastic systems, one representing the cartilage tissue and the other representing the fluid flow in the contact gap formed by surface asperities. The following are major findings:

- As a proof of concept, the results of this study support weeping lubrication significantly extending the duration of the mixed-mode regime. Without the fluid supplement, the mixed-mode lubrication could only maintain a short period of
time. In contrast, the fluid supplement from cartilage into the contact gap could significantly prolong the mixed-mode lubrication duration at least 20-fold.

- Under the sustained compression, there is an initial increase in the flow flux from cartilage into contact gap in the early stage of loading, reaching a peak flow rate within 5 minutes, and then the fluid flux into the gap decreases gradually. In addition, the time for peak flow rate from the cartilage into the contact gap is spatially dependent, i.e., longer time is required when approaching the load centre.

- A model not considering the fluid exudation from cartilage tissue into the contact gap (i.e., no flux at contact interface), would underestimate the total fluid exudation volume and hence overestimate the temporal cartilage interstitial fluid pressure, and the consolidation time for the cartilage.

- A higher asperity stiffness would result in a larger equilibrium contact gap height but also cause a significant and rapid decay in gap fluid pressure, and hence reduce the duration of mixed-mode lubrication significantly. In contrast, a lower asperity stiffness would result in a higher gap fluid pressure but early collapse of gap height and an increase the contact area and boundary lubrication.

- The increase of viscosity of synovial fluid can significant prolong the gap fluid pressure.

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Declarations of interest: none
REFERENCES


[27] Barker M, Seedhom B. The relationship of the compressive modulus of articular cartilage with its deformation response to cyclic loading: does cartilage optimize its modulus so as to minimize the strains arising in it due to the prevalent loading regime? Rheumatology 2001; 40:274–284.


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Fig. 1 - An overview of the coupled contact model.
Fig. 2 - Schematic diagram of the cartilage contact in mixed-mode regime.

**Macro-structure of cartilage contact model**

**Micro-structure of contact gap**
Fig. 3 - Evaluation of gap permeability $K_g$.

- Surface a: flow inlet;
- Surface b: flow outlet;
- Surface c: lateral walls parallel to flow (symmetrical boundary);
- Surface d: upper wall, rigid impermeable indenter (no-slip boundary);
- Surface e: lower wall, cartilage surface roughness (no-slip boundary).
Fig. 4 - Gap and cartilage interaction at the contact interface (z=0 mm): (a). Ratio of gap and tissue permeability (r=0 mm and 5 mm); (b). Vertical component of Darcy velocity through cartilage surface (r=0 mm and 5 mm).
Fig. 5 - Load balancing between fluid and asperity in the contact gap: (a). Comparison of fluid pressure in the contact gap (with and without cartilage); (b). Nominalised fluid and asperity load support in the contact gap and start-up friction coefficient.
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Fig. 7 - Parametric study of the gap fluid viscosity: gap fluid pressure at contact centre 
\((r=0 \text{ mm}, \ z=0 \text{ mm})\).
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Author/s:
Liao, JJ; Smith, DW; Miramini, S; Gardiner, BS; Zhang, L

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