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The spatio-temporal mechanical environment of healthy and injured human cartilage during sustained activity and its role in cartilage damage

Saeed Miramini[a,*], David W. Smith[b], Lihai Zhang[c], Bruce S. Gardiner[d]

[a] Department of Infrastructure Engineering, The University of Melbourne, Victoria 3010, Australia
[b] Faculty of Engineering, Computing and Mathematics, The University of Western Australia, WA 6009, Australia
[c] Department of Infrastructure Engineering, The University of Melbourne, VIC 3010, Australia
[d] School of Engineering and Information Technology, Murdoch University, WA 6150, Australia

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ABSTRACT

Recently we presented a computational model of articular cartilage calibrated for normal human tissue explants. This model was able to capture the transient deformation of cartilage experiencing a cyclic load. The model takes into account the tension-compression nonlinearity of the cartilage and incorporates the dependency of the compressive stiffness and fluid permeability of cartilage on the deformation-dependent aggrecan concentration in cartilage tissue. As such it represents a leading constitutive model of articular cartilage. Here we build on the previous study to develop an experimentally validated computational model to simulate mechanical consolidation response of intact and previously injured cartilage under sustained static loading, to develop our understanding of the implications for rates of tissue damage. We see that the type of prior injuries compromise the cartilage function in different ways. Relatively rapid consolidation is predicted for cartilage with a complete meniscectomy and that with a full thickness defect, indicating the inability of cartilage with such injuries to sustain interstitial fluid pressurisation for long periods of time, as does uninjured cartilage. By comparing the consolidation response of articular cartilage predicted by computational model against experimental measurements of the apparent friction coefficient following static loading, we find a strong linear positive correlation exists between cartilage degree of consolidation (DoC) and friction coefficient at the joint. As the DoC of articular cartilages can be estimated *in vivo* via medical imaging, the DoC can be used as an index to non-invasively evaluate the apparent friction coefficient between opposing cartilage surfaces, and so estimate the likelihood of frictional surface wear and/or cartilage damage.

1. Introduction

Articular cartilage is a multiphasic material. However it is often treated mathematically using two phases: a solid phase comprising collagen, proteoglycans, other proteins, and chondrocytes and fluid phase comprising water and electrolytes (Gardiner et al., 2007; Kwan et al., 1984; Mow and Huiskes, 2005; Pierce et al., 2013; Soltz and Ateshian, 2000; Zhang et al., 2007). When cartilage is subjected to compressive loading, the load is initially carried by the fluid phase. As interstitial fluid exudes through the tissue surface, load is transferred to the solid matrix. Time dependent fluid drainage leads to a time dependent deformation of the cartilage tissue known as consolidation (Verruijt, 2013).

As a material with multiple biological functions, articular cartilage faces several difficult biomechanical challenges. It experiences high loads, stresses and deformations during normal use. The human knee cartilage experiences contact forces up to 5 times of body weight during stair climbing (Taylor et al., 2004). Yet at the same time as experiencing high contact stresses, it achieves very low friction. Cartilage on cartilage friction coefficients are in the range 0.005–0.02 (Longmore and Gardner, 1975; Merkher et al., 2006). This friction coefficient is lower than all manufactured 'slippery' surfaces (McNary et al., 2012; Mow et al., 1992). Low friction is important, as friction is the principal cause of wear at contacting surfaces. But it is important to recognise that the (apparent) friction coefficient increases with time, and so a long period of static loading with attendant consolidation of the cartilage followed by movement, can result in much larger friction coefficients (Forster and Fisher, 1996). The consolidation deformations associated with the larger apparent friction coefficients may lead to greatly increased surface wear and cartilage tissue damage. This implies

* Corresponding author.
E-mail addresses: s.miramini@unimelb.edu.au (S. Miramini), david.smith@uwa.edu.au (D.W. Smith), lihzhang@unimelb.edu.au (L. Zhang), b.gardiner@murdoch.edu.au (B.S. Gardiner).

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that the details of the time-dependent load patterns are a crucially important consideration when attempting to assess the likely impact of activities on joint health (Gardiner et al., 2016).

In this study, we hypothesize that the duration of cartilage loading relative to the characteristic time for cartilage consolidation plays an important role in determining the friction coefficient (and so the rate of cartilage damage and wear). For example, a long duration of continuous usage may significantly compromise cartilage's famously low friction coefficient, thereby exposing it to increased shear stresses and rates of wear, while larger volumetric and shear deformations increase the rate of extracellular matrix damage which may potentially lead to cell death. A corollary of this hypothesis is that the degree of consolidation (DoC) of the cartilage should also be related to the apparent friction coefficient. We speculate that the DoC is strongly correlated with damage potential of cartilage tissue. This is potentially of great practical interest not only conceptually, but because the DoC can be estimated via *in vivo* imaging based on the time dependent closure of the joint space. In contrast, excess interstitial fluid pressure, exudate fluid volume and effective contact stresses cannot be practically measured *in vivo*.

The objective of this paper is to compare the mechanical consolidation response of articular cartilage to sustained periods of static loading, contrasting the consolidation response for intact and compromised cartilage or joint tissue to investigate the relationship between characteristic time for cartilage consolidation and friction coefficient. Specifically we will numerically study human tibial cartilage (lateral tibial plateau) for 5 different cases: (1) intact cartilage; (2) cartilage with partial meniscectomy; (3) cartilage with complete meniscectomy; (4) cartilage with a partial thickness defect; and (5) cartilage with a full thickness defect. In addition, we will investigate the possible correlation between DoC and apparent friction coefficient of articular cartilages.

2. Materials and methods

We have recently published a non-linear poroelastic model of human knee articular cartilage under cyclic loading (Zhang et al., 2015) and validated it against *in vitro* lab testing of osteochondral explants (Barker and Seedhom, 2001). We further develop our model to investigate the mechanical consolidation of cartilage for the 5 prototypical cases. We briefly summarise the model below.

2.1. Poroelastic cartilage model

The cartilage extracellular matrix is treated as a fully saturated porous media, composed of interstitial fluid and solid phase. The total stress tensor $\boldsymbol{\sigma}$ inside the cartilage tissue is the sum of solid and fluid Cauchy stress tensors as follows:

$$\boldsymbol{\sigma} = \boldsymbol{\sigma}^s + \boldsymbol{\sigma}^f = \boldsymbol{\sigma}_E^s - p\mathbf{I} \quad (1)$$

where p is the excess interstitial fluid pressure generated by loading the tissue, $\mathbf{I}$ is the identity matrix and $\boldsymbol{\sigma}_E^s$ is the incremental effective stress associated with the deformation of the solid phase. It should be noted that the analysis is an incremental analysis from the initial state. Excess interstitial fluid pressure, is the current interstitial fluid pressure minus the initial interstitial fluid pressure. The initial interstitial fluid pressure is assumed to be zero by definition. However, this ignores very small contributions to pressure that are in fact present e.g. gravitational.

Under the absence of body and inertial forces, the momentum equation can be expressed as:

$$\nabla . \boldsymbol{\sigma} = 0. \quad (2)$$

The cartilage solid matrix can be modelled as an elastic solid with different properties in tension and compression (Soltz and Ateshian, 2000). This behaviour is due to the fact that collagen network governs cartilage's tensile properties while it does not support compression, whereas the aggrecan dominates the cartilage behaviour under slow or static compressive loads. Experimental studies have shown that a quadratic relationship exists between compressive stiffness of cartilage solid matrix and its aggrecan volume fraction (Treppo et al., 2000):

$$H_{-A} = a_1 \varnothing_G + a_2 \varnothing_G^2 \quad (3)$$

where H_{-A} is cartilage aggregate (osmotic) modulus (MPa), $\varnothing_G$ (1) is the 'actual' aggrecan volume fraction of cartilage tissue and a_1 (MPa) and a_2 (MPa) are empirical constants. The cartilage aggregate modulus can be used to find the compressive elastic modulus of cartilage based on the following relation:

$$E_c = 3H_{-A}(1 - 2\nu) \quad (4)$$

where ν is aggrecan effective Poisson's ratio (i.e. Poisson's ratio of aggrecan matrix without excess interstitial fluid pressure) which is usually found to be small, and is taken zero in this study (Schinagl et al., 1996).

It should be noted that, generally, the laboratory measurements of aggrecan volume fraction is an 'apparent' volume fraction defined as aggrecan volume over total cartilage volume. The 'apparent' volume fraction of aggrecan varies along the cartilage depth (see Table 1). However because a significant portion of the volume of articular cartilage is occupied by collagen fibrils and aggrecan can reside only within the extra-fibular domain, the aggrecan actually exists within cartilage at a higher local concentration or 'actual' volume fraction. In other words, the collagen volume fraction influences the 'actual' aggrecan volume fraction of cartilage and subsequently compressive stiffness. For example, if the collagen volume fraction (collagen volume over total cartilage volume) and the 'apparent' aggrecan volume fraction in the cartilage superficial region is $\alpha = 50\%$ and 60 mg/ml respectively, the 'actual' aggrecan volume fraction would be $60/\alpha = 120$ mg/ml.

In addition the aggrecan volume fraction varies due to cartilage volumetric deformation, based on the following mathematical equation:

$$\varnothing_G(t) = \frac{\varnothing_{G0}}{J^s(t) - \alpha} \quad (5)$$

where $\varnothing_G(t)$ is 'actual' aggrecan volume fraction at time t, $\varnothing_{G0}$ is initial 'apparent' aggrecan volume fraction, $J^s(t)$ is cartilage solid phase volume ratio (i.e. Jacobian determinant of the deformation gradient of solid phase $= det(\boldsymbol{F})$) and α is collagen volume fraction of cartilage.

The cartilage collagen network is responsible for tensile stiffness of the cartilage matrix. The stiffness of collagen network in tension in a healthy cartilage is relatively high compared to aggrecan in compression. The tensile stiffness of cartilage matrix also varies with depth and orientation, corresponding to variation in the collagen volume fraction and fibre orientation. Table 2 shows the depth and orientation dependent tensile modulus of cartilage matrix applied in this study.

The continuity equation for the cartilage porous medium can be written as:

$$\nabla \bullet (\boldsymbol{v}_d + \boldsymbol{v}^s) = 0 \quad (6)$$

Table 1
Material parameters applied in the computational model in this study.

Parameter	Value	Reference
$\varnothing^f$	0.8	(Bonassar et al., 2000)
$\varnothing_{G0}(z = 0)$	60 mg/ml	(Wedig et al., 2005)
$\varnothing_{G0}(z = -5mm)$	100 mg/ml	(Wedig et al., 2005)
a_1	0.01 MPa	(Treppo et al., 2000)
a_2	0.075 MPa	(Treppo et al., 2000)
m	−2.37	(Zamparo and Comper, 1989)
n	5.4 e-22 m^2	(Zamparo and Comper, 1989)
μ (at 37 °C)	7 e-4 Ns/m^2	(Kestin et al., 1978)