Materials Science and Engineering C 78 (2017) 79–87

Contents lists available at ScienceDirect

Materials Science and Engineering C

journal homepage: www.elsevier.com/locate/msec

On mechanical mechanism of damage evolution in articular cartilage

Yu-tao Men *, Yan-long Jiang, Ling Chen, Chun-qiu Zhang *, Jin-duo Ye

Tianjin Key Laboratory of the Design and Intelligent Control of the Advanced Mechatronical System, PR China

ARTICLE INFO

Article history:
Received 2 August 2016
Received in revised form 20 December 2016
Accepted 30 March 2017
Available online 1 April 2017

Keywords:
Biological materials
Damage mechanics
Cartilage
Crack
Numerical analysis

ABSTRACT

Superficial lesions of cartilage are the direct indication of osteoarthritis. To investigate the mechanical mechanism of cartilage with micro-defect under external loading, a new plain strain numerical model with micro-defect was proposed and damage evolution progression in cartilage over time has been simulated, the parameter were studied including load style, velocity of load and degree of damage. The new model consists of the hierarchical structure of cartilage and depth-dependent arched fibers. The numerical results have shown that not only damage of the cartilage altered the distribution of the stress but also matrix and fiber had distinct roles in affecting cartilage damage, and damage in either matrix or fiber could promote each other. It has been found that the superficial cracks in cartilage spread preferentially along the tangent direction of the fibers. It is the arched distribution form of fibers that affects the crack spread of cartilage, which has been verified by experiment. During the process of damage evolution, its extension direction and velocity varied constantly with the damage degree. The rolling load could cause larger stress and strain than sliding load. Strain values of the matrix initially increased and then decreased gradually with the increase of velocity, and velocity had a greater effect on matrix than fibers. Damage increased steadily before reaching 50%, sharply within 50 to 85%, and smoothly and slowly after 85%. The finding of the paper may help to understand the mechanical mechanism why the cracks in cartilage spread preferentially along the tangent direction of the fibers.

1. Introduction

Articular cartilage is a biological material consisting of a solid phase extracellular matrix and a porous liquid phase. The extracellular matrix mainly consists of proteoglycans that are reinforced by a tension-resistant fiber network [1]. A unique mechanical property of the extracellular matrix is its ability effectively resist joint impact force with reduced friction and wear between joints [2–4]. However articular cartilage is in frequent use, which generates chronic mechanical stress. For example, knee joint cartilage is susceptible to injury due to the mechanical stress. Moreover, impaired articular cartilage commonly accompanied by increased joint mechanical stress is suggested to contribute to degenerative osteoarthritis [5–7]. If articular cartilage is damaged, repair to pre-injury levels is challenging [8,9]. Thus, it is important to understand processes related to injury susceptibility and/or pathophysiology of diseases associated with damaged articular cartilage.

In our previous experiments, crack propagation of cartilage did not follow the normal direction of the boundary, but follow the direction of an angle with normal (Fig. 1). There was a branch at the tip of the split. The crack direction observed wasn't always straight forward, but changed constantly at the different layer of the cartilage. The phenomenon of the experiment could not be explained at that time. It is important to understand damage evolution mechanism of cartilage for curing cartilage injury diseases. We hypothesized that the filbers of the cartilage may affect the crack propagation of damage cartilage and the process of the damage evolution of the cartilage.

To date, there have been some studies on mechanical mechanism of damaged AC. Thambyah et al. [10] studied the failure of AC following creep loading induced by impact load with fracture mechanics method. They found the creep-loaded cartilage matrix exhibited a substantial radial collapse or compaction of the fibrillar network in its primary radial zone. The increase of crack length in the prior creep-loaded cartilage was consistent with a reduction in its dissipative properties as indicated by a reduction in rebound velocity. Hosseini et al. [11] established the finite element composite model of cartilage matrix and collagen, their study found that the matrix damage and fiber fracture had different impact on the cartilage damage progress. Ground substance softening and collagen damage had distinct effects on cartilage mechanopathology, and damage in either one of them may promote each other. Kaplan [12] performed a study on the mechanical fatigue of cartilage with the test of healthy human tissue in vivo. Research indicated there was progressively more damage to the microstructure of matrix and collagen network under the 20 N 1.44 Hz through 200 N 2.88 Hz loading. There was significant physical damage under the higher loading conditions, particularly 200 N at 2.88 Hz. Kenneth [13] found defect presence had dramatic effects on dynamic cartilage deformation and defects district

* Corresponding authors at: School of Mechanical Engineering, Tianjin University of Technology, Tianjin 300384, PR China.
E-mail address: yutaomen@163.com (Y. Men).

http://dx.doi.org/10.1016/j.msec.2017.03.289
0928-4931/

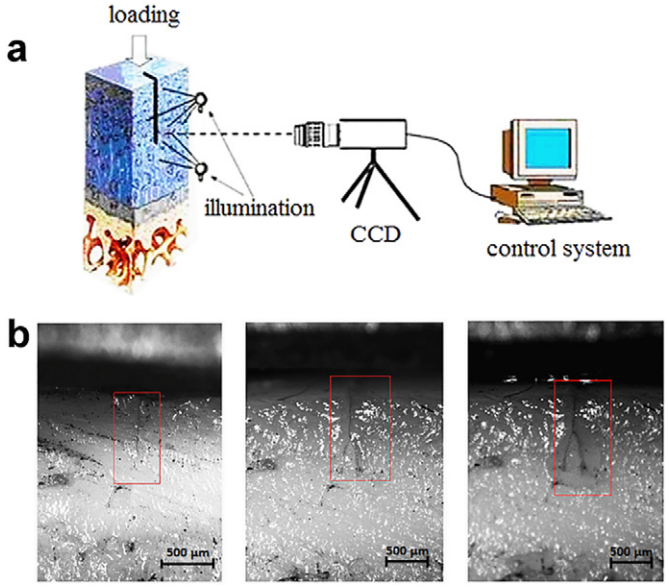

Fig. 1. Schematic diagram of experimental system under rolling and sliding loads (a), the experimental images (b) of crack extension direction in different time.

affected strain distributions markedly. Tissue adjacent to a defect experienced increased magnitudes of axial, lateral, and shear strain. These studies demonstrated that as damage to the articular cartilage progresses, more severe immediate and adjacent structural consequences occur such as fibrillation and deep cracks penetrating into soft tissue. These and other maladaptations are impactful activities of daily living because they contribute to reduced tissue stiffness [14] under tension, which is paralleled by increased compression and shear stress [15–18] followed by gradual decreases in cartilage carry capacity [19]. Lastly, although the exact responsible mechanisms remain unclear, several histological studies have shown that chondrocyte death is a maladaptive response to exacerbated mechanical loading of cartilage [20–23]. In addition, the tissue engineering repair therapy is closely related to the mechanical environment in damage zone [24–26]. The primary aim of this study was to assess the mechanical mechanism of cartilage damage evolution using a fiber-reinforced viscoelastic finite element model with profound consideration of the hierarchical structure and fiber distribution characteristics of cartilage. With these data, we aim to demonstrate a new model that will be able to predict the cartilage response to damage under several loading conditions.

2. Materials and methods

2.1. Cartilage finite element model

ANSYS 12.0 was used to establish 2D fiber-reinforced finite element model [16,20,27,28]. Illustrated in Fig. 2, the cartilage model (6 mm long, 2 mm thick) was divided into surface (10%), middle (40%) and deep layers (50%). The plane183 elements for the matrix and link1 elements for the fibrils were chosen. The viscoelastic response of the matrix was taken into account in this study. Relaxation modulus was

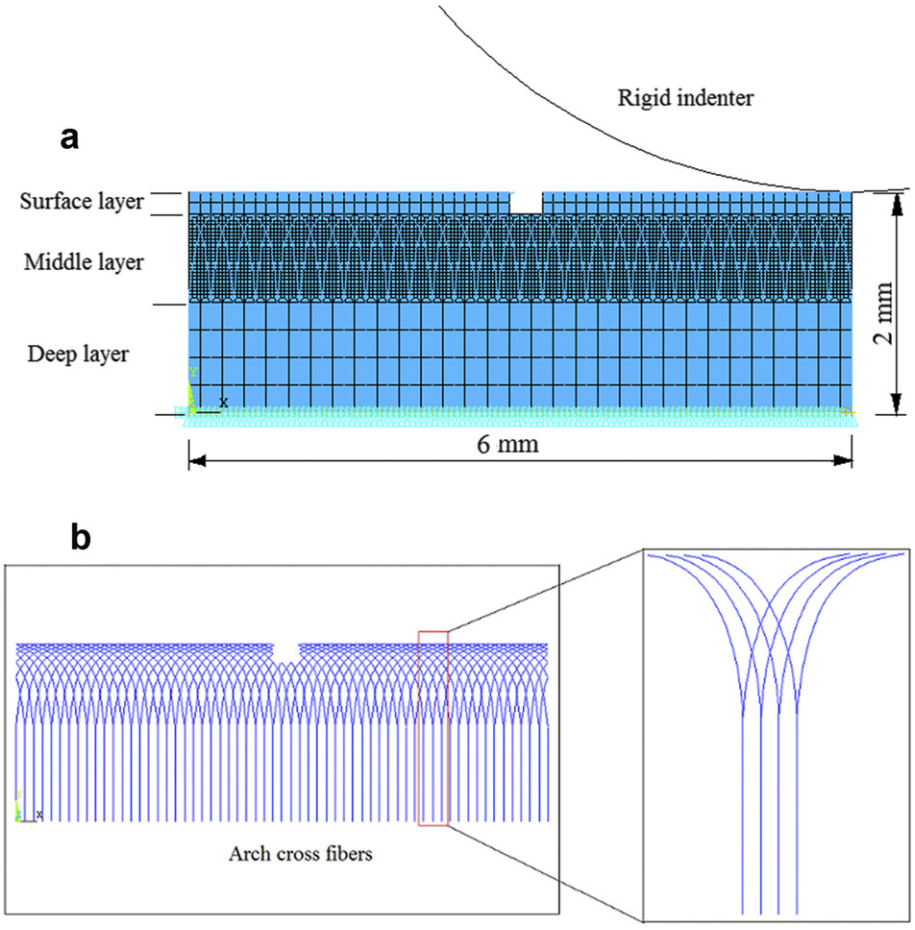

Fig. 2. The cartilage model (6 mm long, 2 mm thick) was divided into surface, middle and deep layers. The matrix chose plane strain quadrilateral elements with 8-node and the fibrils chose the link elements (a). The fiber orientation of the surface layer was nearly parallel with the upper surface layer (0° to 30°), the middle fibers crossed each other (30° to 90°), and the deep fibers were perpendicular to the subchondral bone (90°) (b).