The Role of Collagen Fibrils Networks in the Strain and Stiffness of Articular Cartilage during Indentation Probing

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INTRODUCTION

The reduction of the indentation stiffness of articular cartilage is one of the earliest detectable signs of degeneration [1]. With the development of arthroscopic indentation probes, objective measurements of cartilage stiffness to gauge the extent of tissue degeneration and repair have become possible. During cartilage indentation, 2D imaging techniques have demonstrated nonhomogeneous strains along the radius and depth from the probe site [2,3]. Collagen fibrils networks play an important role in transient cartilage mechanics [4,5] and as such their disruptions likely affect strains and stiffness of cartilage. Specifically, tensile modulus of the superficial layer of cartilage, where fibrils are in the test direction, decrease substantially with fibrillation and osteoarthritis [6,7]. The objective of this study was set to use a microstructurally-based 3D finite element model to elucidate the relative role of collagen fibrils networks at different zones in the biomechanical response (i.e., intra-tissue strains and stiffness) of cartilage to indentation compression [2].

METHODS

Incompressible elastic (identical to transient poroelastic [8]) analyses were carried out to study the effects of collagen networks structure on the short-term indentation response of cartilage. Articular cartilage was modeled as having two phases: a non-fibrillar matrix represented by incompressible continuum brick elements with isotropic elastic modulus varying nonlinearly from 0.3 MPa at the superficial layer to 1.2 MPa at the deepest layer, and fibrillar networks, represented by either membrane (for superficial and deep fibrils) or continuum brick (for middle fibrils) elements [4,8]. Thickness of fibrillar elements in different regions of cartilage was computed based on fibrillar volume fraction, increasing from the articular surface to cartilage-bone junction. Bone was considered rigid due to its high modulus compared to cartilage. The bottom surface of cartilage at bone junction was fixed.

The model simulated a micro-scale indentation [2] with the geometry of cartilage as 10×2.5×1.85 mm^3 (W×L×H). The cartilage/bone surface of osteochondral blocks was against a glass slide through which 2-D deformation was imaged [2]. In accordance with experiments, indentation along a centerline (Fig.1, dotted line on the plane of symmetry) was modeled under both a 1-mm wide rectangular-prismatic indenter (case a) and a 1-mm diameter cylinder-ended indenter (cases b-f), both impermeable to a depth of 190 μm over 1.57s. Friction coefficient (μ) of 0.1 was assumed at the indenter-tissue interface, while μ=0 at the glass wall-tissue interface.

To examine the effect of mechanical integrity of collagen fibrils at different layers of cartilage on the response to indentation with cylindrical tip, the volume fraction (i.e. stiffness) of fibrils networks (at superficial, middle, and deep layers corresponding to cases c, d and e, respectively) was reduced to 1/3 of its reference value (case b). In an additional case (f), random fibrils were extended to the deep zone at a small volume fraction (3%) to represent non-vertical cross-links fibrils.

RESULTS

Irrespective of indenter type, strains along the radius and depth were inhomogeneous. Compared to indentation by the cylinder-ended indenter (case b), indentation by the rectangular indenter (case a) caused greater strain concentrations at top layer near its tip (Fig. 1, showing the glass-specimen interface for a length of 1.2 mm over 5 mm) as well as much larger reaction force (Fig. 2) and strain variations along the thickness under the indenter itself (Figs. 1 and 3, with the latter showing depth-varying strains along the centerline).

Reaction forces diminished only slightly with the decreased fibril volume fraction in the superficial (-8%, case c) or deep (-2%, case e) layers, compared to the reference case b. The lateral and axial strains along the depth were also minimally altered (Fig. 3). In contrast, reaction force decreased markedly (-45%) when random fibrils were disrupted (case d) and increased substantially (85%) when random fibrils were extended into the deep zone with a small volume fraction (case f) (Fig. 2). The lateral and axial strains along the centre-line were also markedly affected in both magnitude and spatial pattern when random fibrils network was modified. Finally, apart from the free end of the specimen (opposite to that confined by the glass), the strain field remained nearly the same all along the length of the sample (orthogonal to glass-cartilage plane of Fig.1), which was due to the identical length (2.5 mm) of the indenter and specimen.

DISCUSSION

Predicted principal strains (maximum of 14% and -15% for cylindrical indenter and 15% and -19% for rectangular indenter in the plane of image underneath the indenter) (Fig. 1) were in good agreement (in magnitude and trend) with experimental results [2] under both indenter shapes. Results of this study suggest important role of random fibrils network in the middle layer in mechanical integrity of cartilage under indentation probing. Alterations in the volume fraction of random fibrils by a uniform reduction to 1/3 of its initial value substantially influenced the transient indentation force and strain field. Similar changes in horizontal and deep vertical fibrils networks, however, had much smaller effects on overall tissue stiffness and strain fields.

This study also predicted that extension of random fibrils to the deep zone, though at only 3% volume fraction, markedly increases the overall tissue stiffness and alters strain distribution. The presence of such fibrils has indeed been observed at the deep zone [9]. Results indicate that early-stage degenerative cartilage that is histologically marked by disruption of collagen fibrils can be detected by arthroscopic probing. They could hence be of great help in refinement of indentation tests.

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