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The critical size of focal articular cartilage defects is associated with strains in the collagen fibers

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ABSTRACT

The size of full-thickness focal cartilage defect is accepted to be predictive of its fate, but at which size threshold treatment is required is unclear. Clarification of the mechanism behind this threshold effect will help determining when treatment is required. The objective was to investigate the effect of defect size on strains in the collagen fibers and the non-fibrillar matrix of surrounding cartilage. These strains may indicate matrix disruption. Tissue deformation into the defect was expected, stretching adjacent superficial collagen fibers, while an osteochondral implant was expected to prevent these deformations.

Finite element simulations of cartilage/cartilage contact for intact, 0.5 to 8 mm wide defects and 8 mm implant cases were performed. Impact, a load increase to 2 MPa in 1 ms, and creep loading, a constant load of 0.5 MPa for 900 s, scenarios were simulated. A composition-based material model for articular cartilage was employed.

Impact loading caused low strain levels for all models. Creep loading increased deviatoric strains and collagen strains in the surrounding cartilage. Deviatoric strains increased gradually with defect size, but the surface area at which collagen fiber strains exceeded failure thresholds, abruptly increased for small increases of defect size. This was caused by a narrow distribution of collagen fiber strains resulting from the non-linear stiffness of the fibers. We postulate this might be the mechanism behind the existence of a critical defect size. Filling of the defect with an implant reduced deviatoric and collagen fiber strains towards values for intact cartilage.

1. Introduction

Full-thickness focal cartilage defects are common (Hjelle et al., 2002; Widuchowski et al., 2007), and are associated with pain and disability (Heir et al., 2010; Solheim et al., 2014). The size of a focal defect is assumed to be predictive of its fate; small defects remain stable or even heal spontaneously, whereas larger defects may progress towards osteoarthritis (Gelber et al., 2000; Hunziker, 2002; Messner and Gillquist, 1996). Attempts to identify a critical defect size leading to osteoarthritis in animal models resulted in diameters of 3, 5, 7 and 9 mm for rats, rabbits, sheep and horses respectively (Convery et al., 1972; Mizuta et al., 2004; Moyer et al., 2010; Otsuka et al., 1997; Schinhan et al., 2012; Shapiro et al., 1993) implying that the defect size is correlated with joint size and/or with body weight. For humans, surface pressure analyses using pressure sensitive film in cadaveric joints with focal defects of various sizes, showed peak pressures close to the rim at defects 10 mm and larger (Guettler et al., 2004; Hunt et al., 2012; Papaioannou et al., 2010). Such stress concentrations were confirmed by finite element studies (Papaioannou et al., 2010; Pena et al., 2007), thus it seems plausible to state that these increased contact pressures are associated with, or predictive for, the progression of defects into OA. However, contact pressure is a mechanical parameter acting at the surface of cartilage, and it is unknown how contact pressure is related to the mechanical conditions of the internal structural components, which become damaged. Thus, the failure mechanism of structural components of articular cartilage surrounding a sufficiently large defect remains unclear.

The nature of the stresses and strains inside the tissue opposing and adjacent to the defect might provide more insight into the mechanism of damage progression or stabilization surrounding a defect. When loaded, neighbouring tissue may bulge into the defect, which alters the natural deformation of the tissue. Consequently, internal or superficial collagen fibrils may become overstrained, and excessive shear strain in the proteoglycan rich non-fibrillar matrix may develop. Both parameters have been proposed to be associated with fiber and matrix damage (Hosseini et al., 2014). In this way these adverse strain patterns may affect the local degenerative cascade eventually resulting in osteoarthritis. Further quantitative evaluation of internal mechanical

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conditions may reveal whether these altered conditions are indeed likely to damage the structural components of cartilage.

The objective of the current study therefore was to quantify the effect of focal defect size in articular cartilage on the strains in the collagen fibers and the non-fibrillar matrix of the cartilage layers adjacent to and opposing the defect. Further, it was evaluated where these strains are sufficiently excessive to potentially cause matrix disruption. Finally, we questioned whether placing an osteochondral metal implant, which is one of the current treatment methods for focal cartilage defects (Martinez-Carranza et al., 2013), would protect the tissue against the development of excessive internal strains.

To monitor internal tissue strains during loading is cumbersome experimentally. Therefore, this study adopted a finite element modelling approach while using a composition-based material model for articular cartilage, which has been validated with experimental data during a number of previous studies (Wilson et al., 2004; Wilson et al., 2005; Wilson et al., 2006a, 2006b). The advantage of using this model is not only that the internal mechanical conditions of the collagen fibers and the proteoglycan-rich ground substance can be quantified over time, but it also provides the possibility to study effects of various defect sizes while all other factors, e.g. geometrical, material and loading, are kept constant. Excluding biological variability of tissue quality and composition makes it easier to derive conclusions on effects of defect size alone.

2. Methods

2.1. Geometry and mesh

A plane strain 2D geometry was developed to represent simplified cartilage/cartilage contact in a joint (Fig. 1). A 2D plane strain geometry does not have strains in the 3rd direction, thus it can be considered as a geometry that is infinitely thick. The contact width between the opposing cartilage was 25 mm. Due to symmetry, only half of the geometry was considered, thus a 12.5 mm contact width. Cartilage thickness was set to 1.5 mm. The cartilage edges were rounded, to prevent high deformations outside of the area of interest. Complete and frictionless contact was assumed between all contacting areas. In the defect models, a full depth cartilage defect was created at the symmetry axis. The width of the defect ranged from 0 to 8 mm, with increments of 0.5 mm. The corresponding loss in contact area ranged from 2% to 32%, which was assumed to cover the range of defects observed clinically. In the implant case, an 8 mm wide defect (32% loss of contact area) was filled with rigid material, representing the metal implant. A perfect fit of the implant into the defect was assumed.

The upper cartilage part was meshed finer than the bottom part, which was required for the contact calculations. Element size varied between 0.1 mm to 0.16 mm. The element type was a 4-node pore pressure plane strain element with bilinear displacement and hybrid formulation with constant pressure stress (element type CPE4PH, ABAQUS v6.14, Dassault Systèmes, Vélizy-Villacoublay, France).

2.2. Material models

Healthy articular cartilage was described by a previously developed and validated composition-based, fiber-reinforced, poroviscoelastic biphasic swelling model (Wilson et al., 2006a, 2006b). In this material model, cartilage tissue is modeled as a porous solid matrix saturated with water. The solid consists of a proteoglycan-rich ground substance and a fibrillar part, which represents the collagen network. The proteoglycan-rich ground substance contains fixed charges, i.e. the negative charges of the proteoglycans and the fixed charges induce Donan-Gibbs osmotic swelling. The viscoelastic fiber network is implemented as a collection of two primary and seven secondary fiber directions per integration point. The primary fibers make up the arcade-like organization proposed by Benninghoff (1925) (Fig. 1). The primary fiber directions are oriented such that they are perpendicular to the cartilage/bone interface in the deep zone, and closer to the articular surface they bend in opposite directions towards a direction parallel to the surface. A less dense network of fibrils in random directions in the cartilage tissue is modeled by the seven secondary fibers. The secondary fiber directions are distributed evenly in the tissue. The total stress ($\boldsymbol{\sigma}_{tot}$) in the cartilage is determined by the combination of hydrostatic, non-fibrillar and fibrillar matrix stresses and osmotic pressure ($\Delta\pi$) (Wilson et al., 2006a, 2006b):

$$\boldsymbol{\sigma}_{tot} = -\mu_f \boldsymbol{I} + n_{s,0}\left(\left(1 - \sum_{i=1}^{totf} \rho_c^i\right)\boldsymbol{\sigma}_{nf} + \sum_{i=1}^{totf} \rho_c^i \boldsymbol{\sigma}_f^i\right) - \Delta\pi \boldsymbol{I},$$

with μ_f the fluid pressure, $n_{s,0}$ the initial solid volume fraction, $\rho_c{}^i$ the volume fraction of the collagen fibers in the ith direction with respect to the total volume of the solid matrix, $\boldsymbol{\sigma}_{nf}$ the stress in the non fibrillar part, *totf* the number of fibers directions per integrationpoint, σ_f^i the

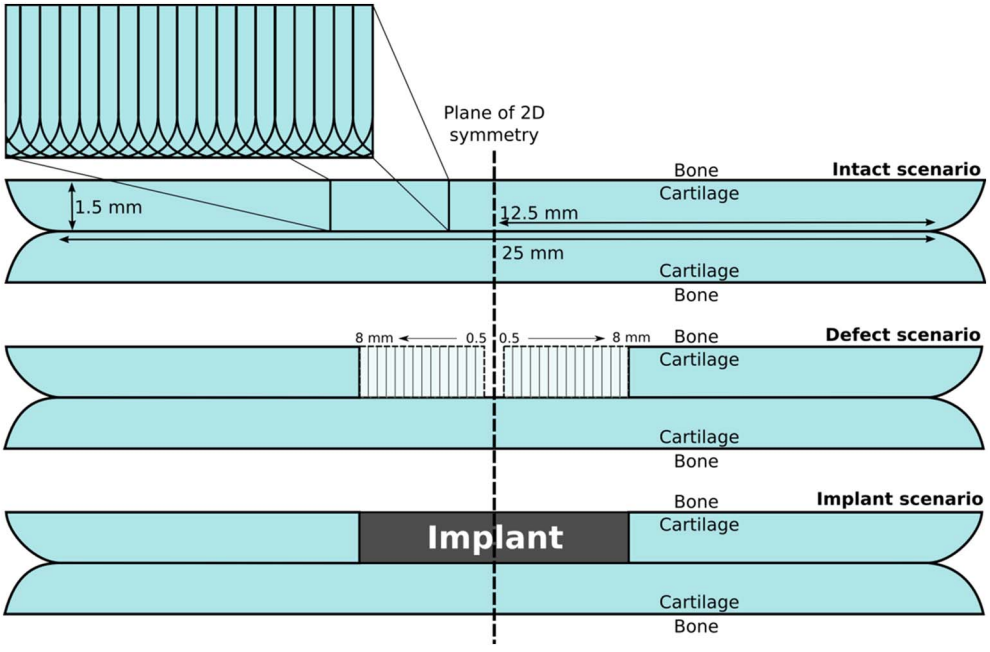

Fig. 1. A schematic representation of the 2D finite element geometry for intact, defect and implant scenarios, including a zoomed area showing a representation of the modeled collagen fibers.