

INTERSTITIAL FLUID LOAD SUPPORT OF BOVINE ARTICULAR CARTILAGE IN UNCONFINED COMPRESSION FOLLOWING COLLAGENASE DIGESTION

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INTRODUCTION

Theoretical and experimental studies have shown that interstitial fluid pressurization plays a fundamental role in the load support mechanism of articular cartilage, supporting more than 90% of the load applied at the articular surface [1,2]. As demonstrated by theory [3,4], the maximum fluid load support is dependent on the ratio of tensile to compressive moduli. In articular cartilage, this ratio can be in excess of 20:1, which helps explain our recent experimental findings [5] that showed maximum fluid load support as high as 95% in unconfined compression of bovine cartilage. The tensile properties of articular cartilage are mainly due to the presence of collagen fibrils in the extracellular matrix, while the compressive behavior is regulated mostly by the proteoglycans [6]. An alteration in the content of either component would likely change the ratio between tensile and compressive moduli, therefore affecting the maximum fluid load support. Changes in the composition of articular cartilage can be induced by enzymatic digestion, and alterations in its mechanical properties have been reported after different enzymatic treatments [7,8]. The objective of this study was to determine the peak fluid load support in enzymatically treated bovine articular cartilage samples under unconfined compression. Our hypothesis is that the maximum fluid load support in cartilage will be reduced by enzymatically degrading the collagen matrix.

MATERIALS AND METHODS

Sixteen cylindrical cartilage plugs were harvested from the femoral condyles of three 2-4 month-old calf knee joints (diameter 6 mm, thickness 1.72 ± 0.20 mm) and stored at -25°C until ready for use. On the day of testing, specimens were thawed at room temperature and the deep zone was microtomed to ensure a uniform thickness. The testing apparatus (Fig. 1) was similar to that of our recent study [5]. Each cartilage sample was placed in a testing chamber having a \varnothing 4.78 mm \times 1.5 mm recess holding a free-draining porous filter (Fig. 1). A piezoresistive microchip pressure transducer (Lucas Novasensor NPC-1210-100G-3N, max. 100 psi), was bonded to the bottom of the chamber. Loading was applied via an impermeable glass platen using

a voice coil load actuator (Model LA17-28-00A, BEI Kimco Magnetics Division) and measured with a load cell (Model 8523, Burster, $\pm 200\text{N}$).

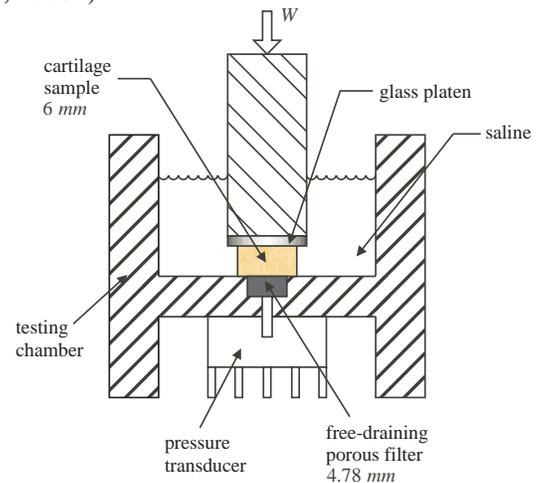


Figure 1.- Diagram of the testing chamber for unconfined compression

The articular surface of the specimen faced the porous filter. Following the application of a tare load that reached 15 N in 20 s, a total compressive strain of 8% was imposed at a rate of 0.2%/s at the top platen, followed by unloading at the same rate. The load supported by interstitial fluid at the articular surface was determined from the pressure transducer measurement, assuming a trapezoidal profile to account for the smaller diameter of the porous filter relative to the specimen [5]. Following this first test, eight of the samples were enzymatically digested with 1 unit/mg of tissue wet weight of type IV collagenase (Sigma) for 18 hours at 37°C on a rotating shaker (treated group). The remaining eight samples were incubated in PBS under similar conditions and used as control. At the end of the treatment, the

samples were rinsed twice with PBS. Fluid load support was again measured using the same protocol. Two-way ANOVA ($\alpha=0.05$) with repeated measures between the first and second test, was used to detect statistical differences between tests, and between control and treated groups.

RESULTS

Typical responses for the total load (W) and fluid load (W^p) before and after collagenase treatment are shown in Fig. 2. Fluid load support was determined from the slope dW^p/dW during the loading phase (Fig. 3) using linear regression. Mean values for the first and second test of the control and treated groups, along with statistical differences, are presented in Fig. 4.

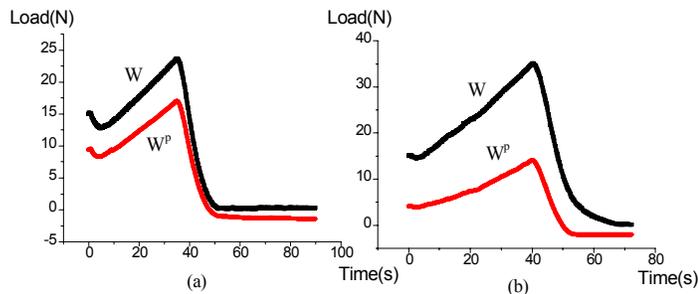


Figure 2.- Typical response of W and W^p , (a) before and (b) after collagenase treatment

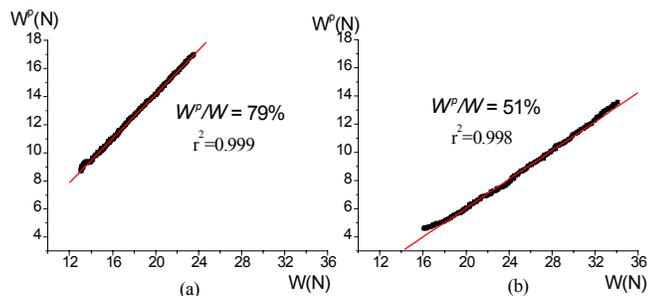


Figure 3.- Plot of W^p vs W during the loading phase, (a) before and (b) after collagenase treatment

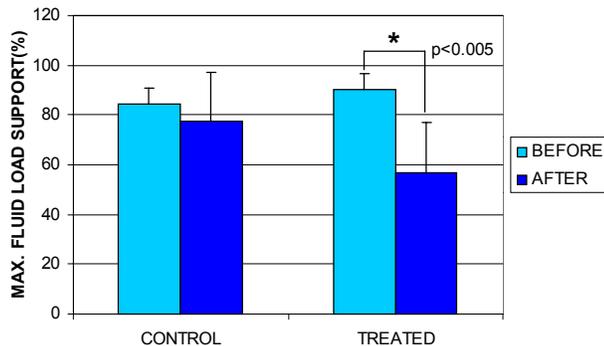


Figure 4.- Change in maximum fluid load support

DISCUSSION

The objective of this study was to investigate experimentally whether the maximum fluid load support in articular cartilage in unconfined compression could be altered by enzymatic degradation of the tissue

matrix. The results shown in Fig. 4 indeed establish that the peak fluid load support of articular cartilage is significantly reduced by collagenase degradation, from a mean value of $90\% \pm 6\%$ down to $57 \pm 20\%$. This result may be attributed to a concomitant decrease in the ratio of tensile to compressive stiffness of the tissue, though direct measurements of these properties need to be performed to verify this hypothesis. The implication of this finding is that tissue degradation, as may occur in osteoarthritis, leads to a loss of interstitial fluid load support and correspondingly greater stresses and strains in the solid matrix of cartilage. This elevation in strains and stresses may then lead to further tissue degeneration. Furthermore, our recent studies have found a direct inverse correlation between the friction coefficient of cartilage and its interstitial fluid load support [9]. The loss of interstitial fluid pressurization with collagenase digestion would imply that the cartilage friction coefficient will increase with increasing degradation [10]. Future studies will include measurement of the tensile and compressive properties of the tissue samples before and after digestion, as well as biochemical analyses to characterize collagen and GAG changes with enzymatic treatment. Such measurements would make it possible to directly correlate the drop in peak fluid load support with the change in tissue composition and material properties.

ACKNOWLEDGMENTS

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