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Effect of frequency on crack growth in articular cartilage

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ABSTRACT

Cracks can occur in the articular cartilage surface due to the mechanical loading of the synovial joint, trauma or wear and tear. However, the propagation of such cracks under different frequencies of loading is unknown. The objective of this study was to determine the effect of frequency of loading on the growth of a pre-existing crack in cartilage specimens subjected to cyclic tensile strain. A 2.26 mm crack was introduced into cartilage specimens and crack growth was achieved by applying a sinusoidally varying tensile strain at frequencies of 1, 10 and 100 Hz (i.e. corresponding to normal, above normal and up to rapid heel-strike rise times, respectively). These frequencies were applied with a strain of between 10–20% and the crack length was measured at 0, 20, 50, 100, 500, 1000, 5000 and 10,000 cycles of strain. Crack growth increased with increasing number of cycles. The maximum crack growth was 0.6 ± 0.3 (mean ± standard deviation), 0.8 ± 0.2 and 1.1 ± 0.4 mm at frequencies of 1, 10 and 100 Hz, respectively following 10,000 cycles. Mean crack growth were 0.3 ± 0.2 and 0.4 ± 0.2 at frequencies of 1 and 10 Hz, respectively. However, this value increased up to 0.6 ± 0.4 mm at a frequency of 100 Hz. This study demonstrates that crack growth was greater at higher frequencies. The findings of this study may have implications in the early onset of osteoarthritis. This is because rapid heel-strike rise times have been implicated in the early onset of osteoarthritis.

1. Introduction

Osteoarthritis (OA) is a degenerative, multifactorial disease. The most recognized symptom of this disease is pain that drives individuals to seek medical attention (Ayis and Dieppe, 2009). Approximately 27 million US adults and 8.5 million UK adults have clinical OA defined on the basis of symptoms and physical findings (National Collaborating Centre for Chronic Conditions, 2008; Lawrence et al., 2008). The significant disability associated with this disease is a great physical burden for affected individuals and an economic burden on the health-care system (Woolf and Pfleger, 2003). Although OA is considered to be a disease of the joint (Loeser et al., 2012), articular cartilage is central to the disease and its progression (Creamer and Hochberg, 1997). The disease involves a decrease in thickness and volume of the tissue (Cicuttini et al., 2004), in addition to an increase in the number and size of cartilage defects (Ding et al., 2005). This can be observed in both animal and human tissue (Clark, 1991). An important element in the disease is the fracture of cartilage, because once cartilage fractures, it has a limited ability to heal the cracks (Buckwalter et al., 1987). It has been hypothesised that these cracks grow with time as an important constituent of the development and progression of OA (Meachim, 1972).

Most testing of cartilage mechanical failure has been undertaken through quantifying the tensile strength of cartilage tissue (Akizuki et al., 1986) or one-dimensional tensile testing of cartilage samples (Roberts et al., 1986). However, cartilage fails by crack formation and fibrillation (Clark and Simonian, 1997). Qualitative measurements of the crack growth in slices of cartilage samples have introduced the concept of cartilage fracture as an important process in the degeneration of cartilage (Broom, 1986). Previous studies (Chin-Purcell and Lewis, 1996; Stok and Oloyede, 2003) have also suggested methods to measure the fracture toughness of cartilage. These studies came to the conclusion that cartilage failure *in vivo* involves the progressive growth of defects.

Rapid heel-strike rise times during gait have been implicated in the early onset of OA in lower limb joints (Radin et al., 1986, 1991). Heel strike rise times in the normal population have been determined to be typically 100–150 ms (Shepherd and Seedhom, 1997). However, Radin et al. (1986) have shown that at heel-strike, some people exhibit a very high rate of loading with a distinct impulsive peak. These rapid heel-strikes take only 5–25 ms to reach a maximum force (Simon et al., 1981). The duration of the heel-strikes corresponds to loading frequencies of 3–5 Hz for normal and up to 90 Hz for impulsive heel-strike rise times (Fulcher et al., 2009). The effect of rapid heel-strike rise times on crack growth in articular cartilage can be investigated by subjecting cartilage specimens with an initial crack to frequencies representative

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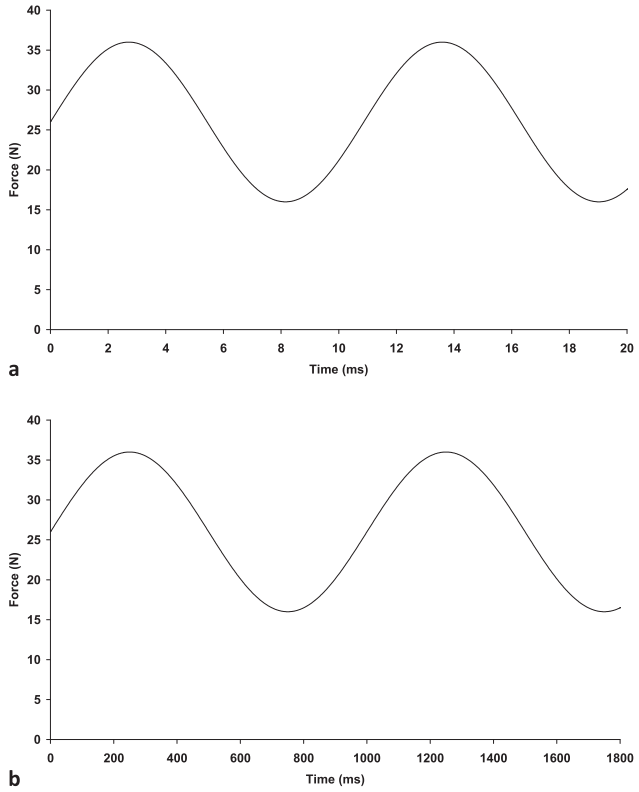

Fig. 1. Sinusoidally varying force a) at 92 Hz (rise time 5.4 ms) b) at 1 Hz (rise time 500 ms).

to such rise times, as shown in Fig. 1. The rise time of the force is approximated by the time taken from the trough to the peak of the sine wave (Fulcher et al., 2009). That is

$$t = \frac{1}{2f} \tag{1}$$

where f is the frequency of the sine wave. Thus, a sinusoidally varying force with a frequency of 92 Hz has been estimated as being representative of a rise time of 5.4 ms, and a frequency of 1 Hz as representative of a heel-strike with a rise time of 500 ms (Fulcher et al., 2009). It has been argued that rapid heel-strike rise times (e.g. 5 ms) lead to impulsive loading (Radin et al., 1991), which might be associated with a predisposition to OA.

Previous studies, on the fracture propagation, of articular cartilage have been focused on the effect of split line direction in tension (Sasazaki et al., 2006) or the effect of impact (Borrelli et al., 1997). However, the effect of frequency has been ignored. Previous studies (Fulcher et al., 2009; Sadeghi et al., 2015a; Espino et al., 2014) have hypothesised that the possibility of cartilage failure would increase with loading frequency because at higher frequencies the ability of the tissue to store energy increased. Therefore, at higher frequencies more energy is available to damage cartilage (Fulcher et al., 2009). Damage caused by increasing the loading frequency (or rate of loading) has been suggested to be different to the damage by increasing load only following comparisons between failure patterns from static loading tests (Fick and Espino, 2011, 2012). This has been demonstrated experimentally by increasing the loading frequency, from relevant gait (1 Hz) to an impulsive frequency (100 Hz), which resulted in more failure of the cartilage-on-bone specimen samples subjected to cyclic compression (Sadeghi et al., 2015b) and bending (Sadeghi et al., 2017). However, these studies focused on compression and bending, rather than tensile strains which have been implicated in the growth of superficial cartilage cracks during impact loading (Kelly and O'Connor, 1996). Therefore, it is currently unknown whether frequencies associated with rapid heel-strikes might also predispose articular cartilage to increased crack growth under purely tensile conditions.

This study aimed to investigate the effect of the variation of loading frequencies associated with relevant gait (1 Hz), above gait (10 Hz) and impulsive loading frequencies (100 Hz) on crack growth in bovine articular cartilage specimens subjected to tensile strains.

2. Methods

2.1. Specimen preparation

Ten bovine shoulder joints, aged between 18 and 24 months, were obtained from Dissect Supplies (King's Heath, Birmingham, UK). Bovine cartilage was used because it is an accepted model for human cartilage (Taylor et al., 2012) and the frequency-dependent viscoelastic trends of bovine articular cartilage have been shown to be consistent with those of human articular cartilage; this includes a similar frequency dependency and high-frequency plateau (Temple et al., 2016). Upon arrival in the laboratory, the humeral head, of each joint, was isolated. The humeral head was wrapped in tissue soaked in Ringer's solution (Sigma-Aldrich, Dorset, UK), sealed in a plastic bag and stored at –40 °C. The influence of freeze-thaw treatment on the mechanical properties of articular cartilage was assumed to be negligible (Stok and Oloyede, 2003, 2007). On the day of testing, humeral heads were removed from storage, and allowed to thaw at room temperature. India ink (Loxley Art Materials, Sheffield, UK) was applied to the humeral heads to identify surface lesions (Meachim, 1972). The India ink was rinsed off and regions, without surface damage, were selected for testing. Three rectangular 40 × 20 mm specimens, which comprised of both bone and cartilage, were cut using a saw from the central load-bearing region of each humeral head.

The underlying bone was approximately 60 mm in thickness and was used to grip the specimens. A mandoline slicer (Mastrad inc., Paris, France) with a 1 mm gap was used to remove cartilage slices while it was still attached to the bone. Cartilage specimens had a maximum of 1 mm depth from the articulating surface towards the bone. In total 30 test specimens, consisting only of cartilage, were obtained from ten humeral heads.

The final cartilage specimens for testing were then produced with dimensions of 20 × 10 mm using a 15 blade medical scalpel (Swann-Morton, Sheffield, UK). A digital Vernier calliper (Fisher Scientific, Leicestershire, UK) was used to measure and highlight an area of 10 × 10 mm and a 2.26 mm crack was cut into the middle of the specimen using a scalpel blade (Fig. 2). The length of the crack was based on the work of McCormack and Mansour (1998), where the initial crack length was 22.6% of the specimen width.

2.2. Mechanical testing

Testing was performed using a Bose ElectroForce 3200 testing machine (Bose Corporation, Minnesota, USA; now, TA Instruments, New Castle, DE, USA) running WinTest 4.1 Software. Two custom-made grips were attached to the testing machine. Emery paper (120 grit) was fixed to the grips (McCormack and Mansour, 1998) and the cartilage specimen was secured by the tightening of screws (Fig. 3). A preload of 0.1 N was applied to the cartilage specimens to prevent the twisting of the specimens about the axial length (aligned with loading axis) while being tested. The actuator of the testing machine applied a sinusoidally varying tensile strain to the tissue specimen with a minimum of 10% and a maximum of 20% of the specimen length (10 mm) for 10,000 cycles. A block command function was used, to initially displace specimens to 15% of their gauge length; specimens were then held in this position for 5 s while an image was acquired. Images were taken using an Apple iPhone 6 Plus (Apple Inc, California, USA) operated under iOS 8 with Sony Exmor RS camera (8 megapixels, 1.5 focus pixels). A scale-bar was included in each image, positioned in the field of view. Images