BREAKING BONES: THE EFFECT OF MICRODAMAGE ON THE FATIGUE BEHAVIOUR OF COMPACT BONE

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ABSTRACT

Secondary compact bone is often compared to a composite material and to metals as discontinuities within the material may provide stress concentration sites for crack initiation but also serve as barriers to crack growth. However, little experimental data exists to back up this hypothesis. Fluorescent agents were applied at specific intervals to bone specimens fatigue tested in cyclic compression and tests were carried out to failure. Microcracks were identified using UV epifluorescence microscopy, their densities recorded and cracks in transverse sections were also described in terms of location to secondary osteons. Microcrack length at the time of encountering the cement line surrounding an osteon was also recorded. Microcracks were shown to develop rapidly during the first 10,000 cycles of testing, but no significant increase took place between 10,000 cycles and 50,000 cycles. A further increase in microcrack density then took place between 50,000 cycles and failure. Microcracks greater than 100 μm in length were found to be more likely to continue to grow after encountering a cement line surrounding an osteon. Only microcracks greater than 300μm in length were found to be capable of penetrating osteons. This work provides experimental data to back up the hypothesis that secondary osteons act as barriers to crack propagation in compact bone and thus support the concept of a microstructural barrier effect in bone. However, it shows that this microstructural barrier effect is dependent on the crack length at the time of encountering an osteon. It shows that for the vast majority of cracks, osteons act as barriers to growth but for the small number of cracks that are actually long enough and do break through the cementline, an osteon may actually act as a weakness in the bone and encourage further propagation.

1 INTRODUCTION

Fatigue damage in bone occurs in the form of microcracks due to the regular day to day activities of normal life in healthy human beings. This damage acts as a stimulus for bone remodelling (Martin and Burr [1], Burr et al [2], Burr and Martin [3], Mori and Burr [4], Lee et al [5], Martin [6]). Bones, therefore, have an advantage over most engineering structures in that they have an inherent ability to repair damage. However if this damage accumulates at such a rate that the capacity for bone repair is exceeded, stress fractures result. These fractures occur commonly in athletes and soldiers engaged in high intensity, repetitive activities such as marching or running. If, on the other hand, damage accumulates at 'normal' rates but the bone's repair mechanism is deficient, fragility fractures result which occur commonly in aging bone (Schaffler et al [7]).

Osteonal bone can be compared to a composite material and to metals whereby discontinuities within the material (e.g. fibres, laminae, voids) may provide sites for crack initiation, but they also serve as barriers to crack growth which may slow down or even halt crack propagation completely. Some reseachers have proposed the possibility of a microstructural barrier concept existing in bone (Martin and Burr [8], Taylor and Prendergast [9], Akkus and Rimnac [10]) whereby the morphology of osteonal bone provides barriers to crack growth in the form of cement lines; currently, insufficient experimental work has been carried out to support this hypothesis.

A technique has been developed by the authors (Lee et al [11], O'Brien et al [12]) which allows microcrack growth to be monitored during the course of a mechanical fatigue test by the application of a series of fluorescent chelating agents. These are as effective as the standard method, basic fuchsin, in identifying microcracks but are also site specific as they bind to calcium ions lining the crack walls. Each agent fluoresces a different colour under UV light and so individual agents can be distinguished when viewed using UV epifluorescence microscopy. Using this technique, it was proposed to label microcracks and monitor microcrack development during fatigue testing to look at the process by which microcracks propagate and interact with the bone's microstructure ultimately bringing about failure.

2 MATERIALS AND METHODS

Specimens were taken from fresh bovine tibiae and machined into typical, waisted, "dog-bone" type of circular cross section using an established protocol (Taylor et al [13], O'Brien et al, [14]). Compressive fatigue tests was carried out in an INSTRON 8501 servo-hydraulic testing machine used in load control to apply an axial force to the specimens, which were enclosed in a small plastic bath to which the dyes could be added and removed. All tests were carried out at room temperature, at a frequency of 3 Hz, and at a stress range of 80 MPa.

The fluorochromes were applied using the established sequence and concentrations (O'Brien et al [12]). The specimens were stained before testing with alizarin complexone in order to label pre-existing damage. They were then tested in another agent, xylenol orange for the first 10,000 cycles of testing. This agent was then replaced with calcein to 50,000 cycles and finally calcein blue between 50,000 cycles and failure. Failure was defined using established criteria; a 10% reduction in stiffness (Taylor et al, 1999) which generally coincided with the appearance of a large crack. Following testing, the gauge length of the specimens was removed using a diamond saw. 250 µm thick sections were cut, handground to between 100 and 150 µm and mounted under a glass coverslip. They were examined using epifluorescence microscopy, their cross sectional areas obtained and microcracks identified and measured using the established criteria (Lee at al [15], Lee at al [16], O'Brien et al [17]). Numerical crack density: Cr.Dn (number of cracks occurring per mm^2) was also measured.

Identified microcracks were split up into five distinct categories (i) microcracks which were located in interstitial bone and did not encounter secondary osteons (ii) microcracks which initiated in interstitial bone but when they encountered secondary osteons, they stopped growing outright (iii) microcracks which initiated in interstitial bone but when they encountered secondary osteons, they continued to grow but their path was deflected around the cement line surrounding the osteon and they did not propagate into the circumferential lamellae of the osteon (iv) microcracks which initiated in interstitial bone but when they encountered secondary osteons, they penetrated the cement line and propagated into the osteon and (v) microcracks which encountered secondary osteons at either end of the crack and did not propagate at either end. The relationship between microcrack length and location using the five distinct categories was then analysed.

3 RESULTS

Thirteen successful fatigue tests were carried out. The chelating agents allowed a clear distinction to be made between pre-existing microcracks, microcracks formed in the first part of the test (up to 50,000 cycles) and microcracks formed in the remainder of the test (up to failure). Only 6% of microcracks were pre-existing and these did not propagate during testing. Fig. 1 illustrates the pattern of microcrack accumulation during the course of a test.

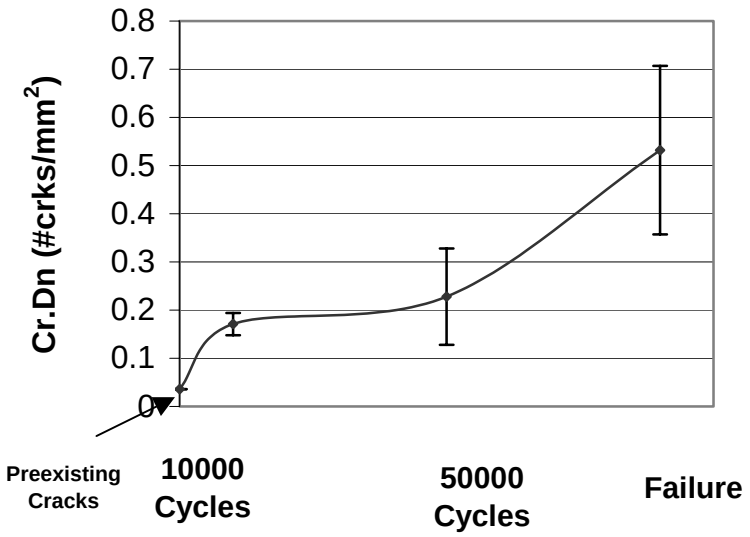

Fig. 1 Accumulated microcrack density v time. Microcrack density was increased rapidly during the first 10,000 cycles but then there was a reduced rate of accumulation until 50,000 cycles have elapsed after which there is another rapid rate of accumulation.

Microcracks were shown to develop rapidly during the first 10,000 cycles, but no significant increase took place between 10,000 cycles and 50,000 cycles. A further increase in microcrack-density then took place between 50,000 cycles and failure with the average Nf being 88,380 (S.D. 22,400) cycles to failure. No significant difference was found in microcracks formed at individual

periods during testing (0-10,000 cycles, 10,000- 50,000 cycles, 50,000 cycles to failure). The mean crack length of cracks formed during these periods was found to be 170 μm (S.D. 56 μm). Pre-existing microcracks were short in comparison to the other types (56 μm S.D. 50 μm) and were found close to the surface of the specimen and were not found to propagate during testing. Propagating microcracks were found to be longer than microcracks formed at individual periods during testing (281 μm S.D. 119 μm).

The majority of microcracks were located in interstitial bone (85%) and did not penetrate the cement lines surrounding secondary osteons. Table 1 shows mean crack length at the time of encountering an osteon for each of the five categories described.

Table 1. Mean crack length at the time of encountering an osteon for each of the five categories described.

CRACK TYPE	Cr.Le (μm)	S.D.
Did not hit osteon	198.1	75.3
Hit osteon but stopped	95.3	25.7
Hit osteon and was deflected around cement line	173.9	47.1
Hit osteon and penetrated cement line	312.6	116.2
Hit osteons on both sides of crack	105.7	37.1

One-way analysis of variance (ANOVA) showed that there was a significant difference between all groups ($p<0.05$), indicating that crack length at the time of encountering an osteon significantly affected its ability to propagate. This table illustrates the influence of crack length on its ability to penetrate cement lines and propagate through an osteon. The first category indicates that in regions of the bone with a low osteon density where cracks did not encounter these features (lateral side of the tibia in this study) cracks grew to an average length of 198 μm before growth was halted. The second category illustrates that if cracks were less than 100 μm when they encountered a cement line surrounding an osteon, they stopped growing outright. However the third category shows that if a crack was in region of 150-200 μm in length when it encountered a cement line surrounding an osteon, it did not penetrate the cement line but did continue to grow and was deflected around the osteon. These cracks were observed to stop growing soon after encountering the cement line. The fourth category shows cracks which did actually manage to penetrate osteons, these were significantly longer ($p<0.05$) than the other categories. The fifth category illustrates cracks which encountered osteons at either tip. This usually happened in areas of high osteon density (medial side of the tibia in this study). Cracks in this category stopped growing outright when they encountered cement lines and their average length of 106 μm is also comparable with the average spacing of approximately 100 μm between osteons.

4 DISCUSSION

This study provides new information on how microcracks accumulate during a bone specimen's fatigue life and eventually cause failure. Crack accumulation during the life of a test specimen follows a characteristic curve in which many cracks initiate early during the specimen's life (first 10,000 cycles) but then accumulation of more cracks is suppressed with only a slight increase occurring between 10,000 and 50,000 cycles before microcracks rapidly accumulate after 50,000 cycles eventually resulting in failure. Some authors have mentioned the possibility of a microstructural barrier concept governing the fatigue behaviour of bone (Taylor and Prendergast, 1997; Taylor, 1998; Akkus and Rimnac, 2001) with the bone's microstructure allowing microcracks to initiate rapidly but because of the morphology of osteonal bone, microcracks encounter barriers which suppress further growth until late in a bone's life. Fig 1 adds further evidence to back up this theory.

The vast majority of microcracks in bone do not grow as they encounter barriers to growth such as cement lines surrounding secondary osteons. In this study, the vast majority of microcracks were found in interstitial bone. Table 1 shows the the relationship between microcrack length and their ability to grow. Microcracks shorter than 100 μm in length were likely to stop growing if they encountered an osteon while cracks in the range 150-300 μm may continue to grow after encountering cement lines surrounding secondary osteons but they are likely to be deflected and often cease growing soon afterwards. Only microcracks greater than 300 μm in length as they encounter osteons were shown to have any real potential to grow to critical lengths and cause failure.

No significant difference in length was found in microcracks formed at individual periods during testing, however, propagating microcracks which grew during at least two stages of the specimen's life were found to be significantly longer ($p<0.05$) than microcracks formed at individual periods during testing which not continue growing in a second stage during the test. This length is similar to that of microcracks which were found to penetrate cementlines indicating that microcracks which were formed at least as early as the second stage of the specimen's life and then continued to propagate have a greater chance of breaking through osteons. Interestingly however, although microcracks which did not encounter osteons (this usually occurred in regions of the bone with a low osteon density) continued to grow to reasonable lengths (198 μm S.D. 75 μm), they did not grow to critical lengths and never caused failure even though they did not encounter cementlines which might have acted as barriers to growth. This suggests that another mechanism may exist which causes certain cracks to propagate to failure.

Failure was always observed to occur with the propagation of one or very few long cracks to critical lengths rather than the coalescence of numerous small microcracks. However, an interesting observation was that these cracks always penetrated a cementline at some stage on the path on the path to failure. Fig. 2 shows a typical example of two large cracks that were involved in failure of a specimen.

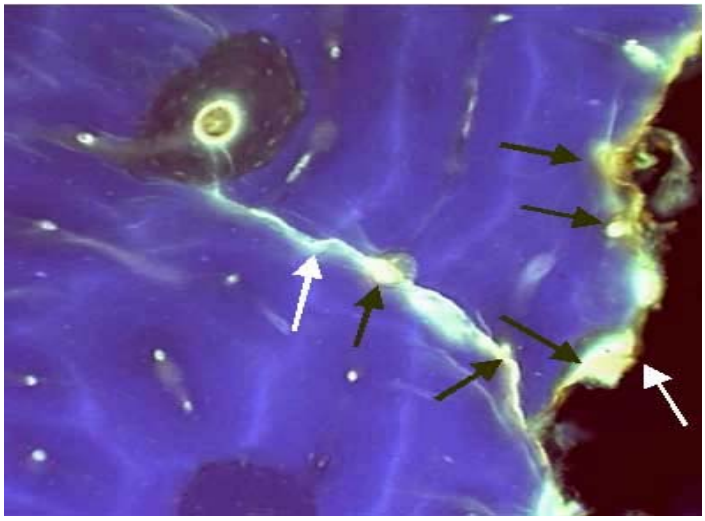

Fig. 2 This illustrates a typical example of two large cracks (white arrows) that were involved in failure of a specimen (the second crack is the main failure surface). As these cracks grew to macrocrack size, they managed to penetrate the cement lines of numerous osteons and used the Haversian canals (black arrows) as weaknesses in which to further propagate.

It can be seen clearly that as these cracks grew to macrocrack size, they managed to penetrate the cement lines of numerous osteons. This was a recurring theme when the fracture surfaces were studied, all failure surfaces showed splitting of osteons usually at the Haversian canals in the centre. As failure tended to occur with the critical growth of cracks which had actually managed to penetrate cement lines rather than the growth of cracks which were found in regions of the bone with few secondary osteons, this would suggest that in the event of microcracks growing to lengths which allowed them to penetrate cement lines, then these canals acted as weaknesses in the bone and allowed a pathway for further propagation and eventually failure.

In conclusion, this study further demonstrates the concept of a microstructural barrier effect existing and having a major effect on the fatigue behaviour of bone. It shows that for the vast majority of cracks, osteons act as barriers to growth but for the small number of cracks that are actually long enough and do break through the cement line, an osteon may actually act as a weakness in the bone and encourage further propagation.

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