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Short communication

# Propagation of surface fatigue cracks in human cortical bone

J.J. Kruzic[a,1], J.A. Scott[b], R.K. Nalla[b], R.O. Ritchie[b,*]

[a]*Department of Mechanical Engineering, Oregon State University, Corvallis, OR 97331, USA*
[b]*Materials Sciences Division, Lawrence Berkeley National Laboratory, and Department of Materials Science and Engineering, University of California, Berkeley, CA 94720, USA*

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**Abstract**

An understanding of how fatigue cracks grow in bone is of importance as fatigue is thought to be the main cause of clinical stress fractures. This study presents new results on the fatigue-crack growth behavior of small surface cracks (~75–1000 μm in size) in human cortical bone, and compares their growth rates with data from other published studies on the behavior of both surface cracks and many millimeter, through-thickness large cracks. Results are obtained with a cyclically loaded cantilever-beam geometry using optical microscopy to examine for crack growth after every 100–500 cycles. Based on the current and previous results, small fatigue cracks appear to become more resistant to fatigue-crack growth with crack extension, analogous to the way the fracture resistance of cortical bone increases with crack growth. Mechanistically, a theory attributing such behavior to the development of bridges in the wake of the crack with crack growth is presented. The existence of such bridges is directly confirmed using optical microscopy.
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*Keywords:* Cortical bone; Fatigue-crack growth; Small cracks; Fracture; Bridging

## 1. Introduction

Stress fractures in human bone are believed to result from continued repetitive (cyclic fatigue) loading, rather than a single traumatic loading event (Burr, 1997; Taylor, 2003). Accordingly, there is interest in understanding the mechanisms governing the initiation and growth of fatigue cracks in cortical bone. Traditional fatigue life (or $S/N$) testing, where nominally flaw-free specimens are cycled at various stress or strain levels ($S$) in order to determine the number of cycles to failure ($N$), is limited in this regard as it is difficult to separate the factors that govern the crack initiation and growth portions of the fatigue lifetime. Consequently, a better approach is for crack initiation and growth to be studied separately in order to gain a full mechanistic understanding of fatigue behavior.

In general, fatigue-crack propagation is characterized by the crack-growth rate per cycle, $dc/dN$, as a function of the linear-elastic (mode I) stress-intensity range, $\Delta K = K_{max} - K_{min}$,[2] where $K_{max}$ and $K_{min}$ are the maximum and minimum stress intensities during the loading cycle (Paris and Erdogan, 1963). For cortical bone, there have only been a few studies on such crack-propagation behavior on macroscopic (millimeter-sized) through-thickness cracks (Wright and Hayes, 1976; Gibeling et al., 2001; Nalla et al., 2005a), and on small (<1 mm) surface cracks (Akkus and Rimnac, 2001).

*Corresponding author. Tel.: +1 510 486 5798; fax: +1 510 486 4881.
*E-mail address:* roritchie@lbl.gov (R.O. Ritchie).

[1]Formerly with Materials Sciences Division, Lawrence Berkeley National Laboratory, Berkeley, CA 94720, USA.

[2]The stress-intensity factor, $K$, is a global parameter which fully characterizes the local stress and deformation fields in the immediate vicinity of a crack tip in a linear-elastic solid, and thus can be used to correlate to the extent of crack advance. It is defined for a crack of length $c$ as $K = Q\sigma_{app}(\pi c)^{1/2}$, where $\sigma_{app}$ is the applied stress and $Q$ is a geometry factor of order unity (Knott, 1976).

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Table 1
Summary of small-crack experiments

| Sample number[a] | Crack size range ($2c$) (mm) | Test temperature (°C) | Maximum stress (MPa) |
|---|---|---|---|
| 41FL1 | 0.7–1 | 25 | 26 |
| 37ML1 | 0.5–1 | 37 | 24 |
| 37ML2 | 0.3–0.4 | 37 | 15 |
| 37ML3 | 0.4–0.5 | 37 | 8, 20[b] |
| 34FL1a | 0.72–0.87 | 37 | 9, 14[b] |
| 34FL1b | 0.15–0.28 | 37 | 8, 13[b] |
| 34FL2 | 0.076–0.21 | 37 | 13, 20[b] |

[a]The notation reads as follows: Age (years), Sex (M = Male, F = Female), Arm (L = Left, R = Right), with the number being a unique identifier when more than one specimen were taken from the same donor, and any following letter differentiating if more than one crack was monitored on a sample.
[b]The stress was increased in order to reinitiate crack growth; dashed lines in Fig. 1 indicate when this occurred.

However, there are several studies on microcrack accumulation (Carter and Hayes, 1977; Zioupos et al., 1996; Fazzalari et al., 2002; O'Brien et al., 2003), the imaging of such microcracks (O'Brien et al., 2002), descriptions of their growth (Taylor, 1998) and models of microcrack bridging (Yeni and Fyhrie, 2001). Mechanistically, such crack growth in bone has been ascribed to a fatigue-related mechanism of alternating crack blunting and re-sharpening at low crack-growth rates ($<5\times10^{-7}$ m/cycle), with quasi-static fracture mechanisms controlling crack growth at higher rates (Nalla et al., 2005a).

The issue of crack size in cortical bone is important due to the fact that bone is extrinsically[3] toughened principally by crack bridging (Pezzotti and Sakakura, 2003; Nalla et al., 2004, 2005b). As cracks advance, intact regions of material are left in the crack wake, termed uncracked-ligament bridges; these bridges act to resist crack opening and sustain part of the applied load, which otherwise would go towards crack propagation. This results in rising fracture resistance with crack extension, and thus smaller cracks are able to propagate more easily. A similar effect occurs in fatigue; indeed, at the same nominal crack-driving force, small fatigue cracks typically grow faster than large cracks in materials where extrinsic mechanisms are present (Suresh and Ritchie, 1984; Ritchie and Lankford, 1986; Ritchie, 1988). In the context of the present study, "large cracks" are those that have a steady-state extrinsic toughening contribution with properties that are insensitive to crack size, while "small cracks" refers to cracks smaller than or comparable to the extent of the crack-bridging zone in the crack wake. For cortical bone, where crack bridges may be up to 100 μm in size (Nalla et al., 2004, 2005b), traditional microcracks, as well as cracks on the meso-scale, are expected to fall within this "small crack" definition, although this needs to be confirmed experimentally. It is then implied that small cracks may slow down with crack extension under constant cyclic load, as suggested by Taylor (1998) and confirmed by Akkus and Rimnac (2001) and O'Brien et al. (2003). Unfortunately, due to the difficulty in conducting crack-growth experiments with small surface cracks, there are only limited crack-growth data available (8 cracks total), and no direct comparisons of large and small fatigue-crack growth rates for human cortical bone. Accordingly, it is the objective of this paper to present new results for the growth of small cracks in human cortical bone and to compare these results with existing fatigue data, with the goal of understanding what effects, if any, the formation of bridges in the crack wake has on fatigue cracking in bone.

[3]Crack propagation can be considered in terms of both *intrinsic* mechanisms, which operate ahead of the crack tip to promote microstructural damage and hence crack advance, and *extrinsic* mechanisms, which operate principally in the wake of the crack tip and act to "shield" the crack from the applied driving force (Ritchie, 1988).

## 2. Experimental procedures

Fresh frozen, human cortical bone (humeral, donor ages: 34, 37 and 41 years, no known skeletal pathologies) was machined, using a slow speed diamond saw (TechCut II, Allied High-Tech Products, Inc., Rancho Dominguez, CA), into rectangular cross-section bend beams ($1.3–2.0\times1.3–2.0\times10–15$ mm) by sectioning (with continuous irrigation) the medial cortices of the mid-diaphyses of humeri taken from the three donors. Specimens ($N=6$, details in Table 1) were cyclically loaded (sine wave, $R=K_{\min}/K_{\max}=0.1$) in cantilever-bending (inset Fig. 1) using a mechanical test frame (ELF® 3200, EnduraTEC Inc., Minnetonka, MN) at a test frequency of 1 Hz while continuously irrigated ($N=1$, 25 °C) or submerged ($N=5$, 37 °C) in Hanks' Balanced Salt Solution, HBSS (Sigma-Aldrich, St. Louis, MO) with gentamicin added to prevent bacterial degradation. The beams were oriented for surface crack propagation along the long axis of the osteons (and

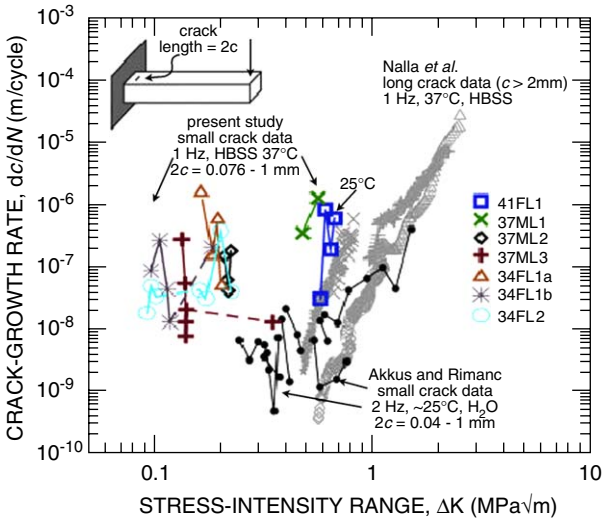


Fig. 1. Comparison of fatigue-crack growth behavior in human humeral bone for small surface cracks (~75–1000 μm in length) from the present study and large through-thickness cracks (several mm in length) from (Nalla et al., 2005a), as well as small surface cracks in 37–40 year old human femoral bone (Akkus and Rimnac, 2001). Crack-growth rates for the smallest surface cracks tended to be higher than those for large cracks; however, data for the bigger surface cracks were closer to the large, through-thickness, crack data, consistent with traditional concepts of small fatigue-crack growth.

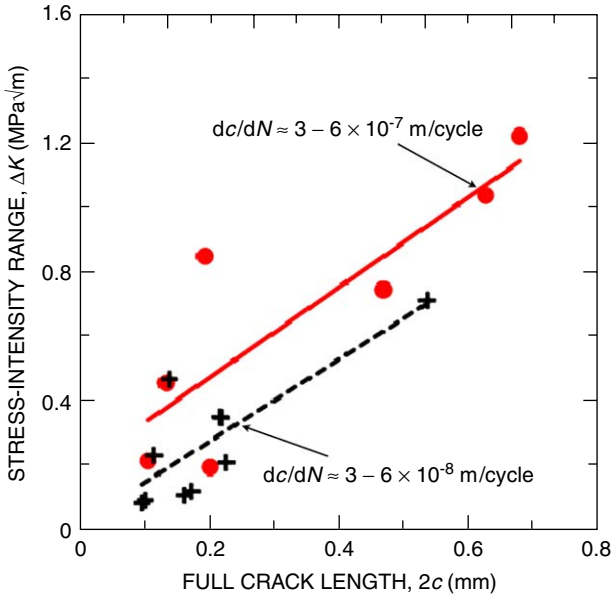


Fig. 2. Plot of the stress-intensity range, $\Delta K$, needed to propagate cracks at growth rates of $3–6\times10^{-7}$ and $3–6\times10^{-8}$ m/cycle as a function of crack length. Data for all seven cracks are shown; however, for each growth rate range only cracks that exhibited growth at the appropriate rates were used, five cracks for the former, and six cracks for the latter. Note the trend of increasing $\Delta K$ with crack length in both cases.

humeri) in order to directly compare with previous large-crack data (Nalla et al., 2005a) for the same cracking direction, donor set, and anatomical location. Cycling was periodically interrupted, after intervals of 100–500 cycles, during which the sample was removed from the machine and the tensile side examined for cracking using bright-field optical microscopy (Olympus STM-UM Measuring Microscope, Olympus America Inc., Melville, NY; 0.5 μm resolution). When a crack or cracks were identified, which typically occurred within 1000 cycles, the surface stress at each crack location was computed (Table 1) for each cycling interval using elementary beam theory. The stress-intensity range was calculated using solutions for surface cracks in bending (Newman and Raju, 1981), assuming $a/c \sim 1$ ($a$ is crack-depth, $c$ is half surface-length) based on subsurface observations by synchrotron X-ray computed tomography (Beamline 8.3.2, Advanced Light Source, Berkeley, CA; 5 μm resolution) ($N = 3$); details of this technique are given elsewhere (Kinney and Nichols, 1992). One crack was monitored on each of five samples, and two cracks on the other sample, for a total of seven cracks ($N = 7$). Crack-growth rates, $dc/dN$, were computed by dividing the crack extension by the number of cycles for each cycling interval. Data were not collected where $2c > 0.5B$ ($B$ is specimen thickness) as this is outside the validity of the stress-intensity solution. Statistical differences between the stress intensities for large (Nalla et al., 2005a) and small cracks were examined at various fixed crack-growth rates using $t$-tests.

## 3. Results and discussion

New results for fatigue-crack growth of small ($2c \sim 75-1000$ μm) surface cracks, initiated near the maximum tensile stress location (see inset Fig. 1), are compared with large-crack results for the same donor set of human humeri (Nalla et al., 2005a) and with small-crack data for human femora (Akkus and Rimnac, 2001) in Fig. 1. Typically, small cracks are expected to grow at lower stress intensities than large cracks until they reach a critical size where the extrinsic toughening contribution reaches a steady state. In bone, that critical size is expected to be related to the development of the bridging zone in the wake of the crack, i.e., as bridges develop, resistance to cracking is enhanced until a steady-state bridging zone is achieved. Although results are limited, this concept is consistent with the data seen here, where higher stress intensities are needed to achieve a given growth rate as the cracks extend (Fig. 2). This was also the trend seen by Akkus and Rimnac (2001), where rising crack-growth resistance was observed with crack extension, to an extent that all but one of their fatigue cracks arrested. Four cracks in the present study arrested, and the load had to be raised to induce further growth (as shown by the

data-points connected by dashed lines in Fig. 1). Two cracks[4] (41FL1 and 37ML1) did not arrest throughout the valid data collection range (i.e., up to $2c = 0.5B$); note that both of these had relatively large starting crack sizes (i.e., $2c > 500\,\mu m$). Statistically, the small cracks in the present study were observed to grow at stress-intensity ranges (at fixed $dc/dN$) which were significantly lower ($p < 0.01$) than large cracks grown in specimens from the same donors (Nalla et al., 2005a). While the small crack data of Akkus and Rimnac (2001) overlaps considerably with the long crack data of Nalla et al. (2005a), several of their cracks also exhibited growth at stress intensities below what is needed for long cracks. Unfortunately, differences in anatomical location (femur versus humerus), donor set, and test frequency are confounding variables in this case, although, based on long crack results (Nalla et al., 2005a), frequency effects are most likely negligible for such small differences.

Similar to fracture behavior in cortical bone (Nalla et al., 2004, 2005b), it is anticipated that the rising crack-growth resistance with crack extension is due to the development of crack bridging, although as Schaffler et al. (1995), Taylor (1998), Akkus and Rimnac (2001), O'Brien et al. (2003) pointed out, local crack arrest may also be related to interactions with microstructural heterogeneities, such as cement lines. These concepts are not inconsistent, however, since one possible mechanism for bridge formation is local arrest, followed by re-initiation of cracking ahead of the original crack tip, leaving intact bridges of material in the crack wake. Furthermore, extensive crack bridging has been observed in the wake of large, through-thickness, fatigue cracks in human cortical bone (Nalla et al., 2005a). For the present study, crack bridging was observed to form as the cracks grew larger in all cases. Indeed, representative optical micrographs are shown in Fig. 3, where one of the smallest cracks (34FL2) has no visible bridging at the beginning of data collection (Fig. 3a), while there is clearly crack bridging on the surface of sample 37ML1 at the end of data collection, i.e., when $2c \sim 1\,mm$ (Fig. 3b). Fig. 3 provides support for the notion that small cracks propagate more easily in bone due to a lessened effect of crack bridging, and that their increasing resistance with crack extension can be associated with the formation of such bridges. It is also possible that, in addition to crack bridging, local crack deflection can also lead to a lower crack-driving force, as suggested by Akkus and Rimnac (2001), although we observed little systematic evidence to support this explanation in the present study.

It must be acknowledged that, to date, the extent of available data on small-crack growth in bone is extremely limited, and further studies using larger donor and sample sets are needed to make firm conclusions. However, based on present and previously published data, it appears that small surface cracks in bone demonstrate increased resistance to fatigue-crack growth with extension, akin to behavior in structural materials (Suresh and Ritchie, 1984; Ritchie and Lankford, 1986; Ritchie, 1988); moreover, the development of bridging ligaments in the crack wake appears to play an important role in this behavior.

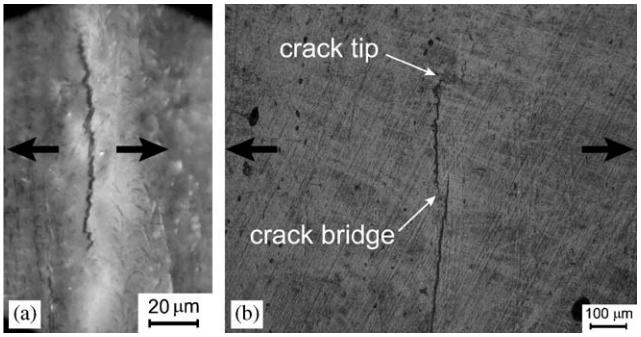


Fig. 3. Optical micrographs of (a) a bridgeless surface crack (34FL2) and (b) bridging in crack 37ML1 after propagating to a length of $2c = 1000\,\mu m$. Note the evidence of crack bridging for the larger sized crack in (b); such micrographs are representative of what was observed in all cases. Black arrows indicate the directions of tensile stress for each micrograph.

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[4]The arrest behavior of the seventh crack is unknown since the sample failed due to an inadvertent overload.

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