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Mechanistic aspects of in vitro fatigue-crack growth in dentin

J.J. Kruzic[a], R.K. Nalla[a], J.H. Kinney[b], R.O. Ritchie[a,*]

[a]*Department of Materials Science and Engineering, Materials Sciences Division, Lawrence Berkeley National Laboratory, University of California, 381 Hearst Mining Building, Berkeley CA 94720, USA*
[b]*Lawrence Livermore National Laboratory, Livermore, CA 94550, USA*

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Abstract

Although the propagation of fatigue cracks has been recognized as a problem of clinical significance in dentin, there have been few fracture mechanics-based studies that have investigated this issue. In the present study, in vitro cyclic fatigue experiments were conducted over a range of cyclic frequencies (1–50 Hz) on elephant dentin in order to quantify fatigue-crack growth behavior from the perspective of understanding the mechanism of fatigue in dentin. Specifically, results obtained for crack extension rates along a direction parallel to the dentinal tubules were found to be well described by the stress-intensity range, ΔK, using a simple Paris power-law approach with exponents ranging from 12 to 32. Furthermore, a frequency dependence was observed for the crack-growth rates, with higher growth rates associated with lower frequencies. By using crack-growth experiments involving alternate cyclic and static loading, such fatigue-crack propagation was mechanistically determined to be the result of a ''true'' cyclic fatigue mechanism, and not simply a succession of static fracture events. Furthermore, based on the observed frequency dependence of fatigue-crack growth in dentin and observations of time-dependent crack blunting, a cyclic fatigue mechanism involving crack-tip blunting and re-sharpening is proposed. These results are deemed to be of importance for an improved understanding of fatigue-related failures in teeth.

Keywords: Dentin; Fatigue; Fracture mechanics; Crack propagation

1. Introduction

Dentin is the most abundant calcified tissue in the human tooth. Located between the exterior enamel and the interior pulp, dentin is a hydrated nano-composite of hydroxyapatite mineral crystallites (~5 nm thick, ~45% by volume) distributed in a scaffold of type-I collagen fibrils (~50–100 nm diameter, ~30% by volume), with fluid and non-collagenous proteins making up the remaining ~25%. The collagen scaffold is aligned perpendicular to the dentinal tubules, which are the paths taken by the odontoblasts during dentinogenesis. In general terms, the inorganic mineral in dentin is believed to provide the strength and stiffness, and the organic collagen to provide the toughness [1–3].

As fracture is a critically important issue with human teeth, an understanding of the structural performance of dentin is crucial. This is reflected in the literature where there have been extensive reports of clinical studies of the fracture of teeth, particularly following dental restoration (e.g., [4–11]). The mechanisms of fracture gain added significance when viewed in conjunction with a wide range of clinical observations. For example, exposed root surfaces often exhibit non-carious notches in the dentin just below the enamel–cementum junction. It is believed that the etiology of such lesions could involve a combination of erosion due to chemical action, abrasion from foreign-body (e.g., food particles, improper dentifrice, etc.) induced wear, and abfraction from tooth flexure [12,13]. These notches are often the

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

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sites of failure of the tooth due to fracture. While it is plausible that the actual fracture is a result of a single catastrophic loading event, it is generally believed that such failures are the end result of subcritical cracking induced by repetitive occlusal stresses, i.e., due to cyclic fatigue damage [14]. In addition, another commonly encountered example is crack growth from damage induced during reparative dental procedures; indeed, there are many reports that suggest that restored teeth are more likely to experience fracture over time (e.g., [5,7,9]). Again, such reparative damage could result in failure due to prolonged cyclic masticatory stresses [15].

Given the compelling clinical need to understand the fatigue properties of dentin, it is perhaps surprising to note that this has been the topic of only a few relevant studies [15–19] to date, especially with respect to the mechanisms of fatigue. Tonami et al. [16] used the classical "Stress-Life" (S/N) approach[1] to fatigue and utilized a "staircase" method to determine the 10^5-cycle endurance strength[2] of bovine dentin. The present authors recently reported stress-life (S/N) behavior in human dentin, with apparent fatigue limits at 10^6–10^7 cycles for a range of frequencies and load ratios (minimum/maximum load) [15,19]. In addition, the first fatigue-crack growth data for human dentin was obtained in that work by measuring specimen stiffness loss in the S/N samples [15]. Fatigue-crack growth rate behavior has also been measured by Arola et al. [17,18] for various orientations in bovine dentin. These authors found that fatigue-crack propagation rates were highest for crack growth perpendicular to the tubules, but did not attempt to discern the mechanisms associated with the fatigue process.

From a mechanistic viewpoint, a critical issue is whether fatigue in dentin is time or cycle dependent (or both). In this regard, Nalla et al. [15,19] examined the effect of time vs. cycles on the S/N fatigue behavior in human dentin by varying the test frequency. They observed that by changing the frequency from 20 to 2 Hz, the fatigue limit and fatigue lifetimes (in terms of cycles) were both lowered, although when such S/N data were plotted on the basis of time, this frequency effect was much reduced. The inference from this is that the mechanisms of fatigue-related failures in dentin may be predominantly time-, rather than cycle-, dependent, i.e., there may not be a true cyclic fatigue mechanism, although further experiments clearly revealed that the process of crack initiation was far easier under cyclic, as compared to static loads. Since S/N experiments include both the crack initiation and crack growth stages, which may have different time and cycle dependencies, these results cannot be easily interpreted. Consequently, to reveal mechanisms, it is generally more instructive to distinguish between these two stages of the fatigue life. It is for this reason that the current work is focused solely on the propagation of fatigue cracks in dentin, which is characterized using a fracture mechanics-based approach.

While mechanisms of fatigue-crack growth are generally well understood for most common engineering materials, e.g., by alternating crack blunting and resharpening in ductile metals, crazing in polymers, and static fracture in brittle ceramics [20], no equivalent mechanisms (to the authors' knowledge) have ever been proposed for dentin. However, previous studies on hydrated dentin tested under a sustained (non-cyclic) load clearly revealed significant time-dependent crack blunting [21]. Correspondingly, crack growth in dentin under cyclic fatigue loading could be envisioned to occur via alternating crack-tip blunting, as in metals, where each blunting event during loading serves to create new crack surface area upon unloading [22]. However, as time-dependent crack propagation has also been reported in dentin under static loading (over short time spans of less than 1 h) [21], fatigue-crack growth could conversely be the result of individual (static) cracking events, as in brittle ceramics [23], driven by the peak/mean load during each cycle.

To investigate whether fatigue in dentin involves such cycle-dependent (fatigue-dominated) or time-dependent mechanisms, or whether (as in cortical bone [24–26]), there is a transition from cycle-dependent processes at low growth rates to time-dependent processes at high growth rates, we specifically examine in this study the effect of (i) cyclic frequency and (ii) holding at peak load, on fatigue-crack propagation behavior in dentin over a wide spectrum of crack velocities ($\sim 10^{-10}$–10^{-5} m/cycle). The objective of this research is to gain insight into the mechanisms of fatigue damage, with the ultimate goal of providing the clinician with tools for predicting fracture risk.

2. Materials and methods

2.1. Materials

Due to specimen size limitations imposed by human dentin, fatigue-crack growth behavior in the current work was examined in elephant dentin. Fatigue test

[1] The traditional "Stress-Life" or "Strain-Life" (S/N) approach to fatigue design and life prediction involves cycling (nominally flaw-free) specimens at various load/stress/strain levels in order to determine the number of cycles to failure. By contrast, the more conservative "damage tolerant" approach utilizes a fracture mechanics methodology; it assumes the presence of existing damage, e.g., the presence of flaws, and computes the number of cycles to failure for the largest of these flaws to grow subcritically to failure [20].

[2] The *endurance strength* is defined as the applied stress amplitude to yield a specific life, generally 10^6 cycles or above. If the S/N curve is horizontal at this point, as in many steels, this is referred to as a *fatigue limit*, because below this stress amplitude, the material, in principle, will never fail by fatigue.

samples were machined from recently harvested fractured shards of elephant tusk from an adult male elephant (*Loxodonta africana*), obtained from the Oakland Zoo, Oakland, CA; the bulk of this material, which is commonly referred to as ivory, is composed of dentin and is microstructurally quite similar to human dentin. While extensive microstructural studies on elephant dentin have not been performed, Raubenheimer et al. [27,28] noted that the microstructure was similar to human dentin, although the dentinal tubules had a slightly larger diameter. Our own observations indicated that the tubules in elephant dentin are more elliptical in shape and the peritubular cuffs are much smaller or non-existent, although the tubule density and mineral content appear to be similar to human dentin.

In vitro fatigue-crack propagation tests were performed using compact-tension, $C(T)$, specimens; eleven ($N = 11$) specimens were machined with specimen thicknesses of $B \sim 1.8 - 3.2$ mm, widths of $W \sim 14.4 - 17$ mm and initial notch lengths of $a_o \sim 3.4 - 4.4$ mm. The nominal crack-growth direction was perpendicular to the long axis of the tubules, and the crack plane was in the plane of the tubules. To permit observations of the crack path, these specimens were polished to a 1200 grit finish, followed by polishing steps using a 1 μm alumina suspension and finally a 0.05 μm alumina suspension. The notch was introduced with using a slow speed saw, and finally carefully sharpened to a consistent root radius of $\rho \sim 15$ μm using a razor-micronotching technique, where the sharp notch was created by repeatedly sliding a razor blade over the saw-cut notch using a custom made rig, while continually irrigating with a 1 μm diamond slurry.[3]

All specimens were maintained in a hydrated condition by continuous irrigation throughout preparation. In addition, specimens were hydrated prior to actual testing by soaking in Hanks' Balanced Salt Solution (HBSS) for at least 40 h at room temperature. Although it is possible that there may have been some degradation of the collagen phase and/or leaching of the mineral phase into solution during this phase, which can result in changes in the mechanical properties with time, no such changes could be detected following short time storage of dentin under similar conditions [29].

2.2. Mechanical testing

All fatigue-crack growth testing was conducted in HBSS (with gentamicin to prevent bacterial action) at 37(±0.5) °C on an MTS electro servo-hydraulic testing machine (MTS 810, MTS Systems Corporation, Eden Prairie, MN) in general accordance with ASTM Standard E-647 [30]. Specifically, fatigue cycling was performed under load control at a constant load ratio (ratio of minimum to maximum load) of $R = 0.1$, at three different sinusoidal frequencies of 1, 10 and 50 Hz ($N = 3$ for each frequency). While 1 Hz is a frequency of clinical significance for human dentin, higher frequencies were used to understand the mechanistic aspects of fatigue-crack growth; specifically, variations in growth rates with change in frequency, if any, can aid in developing a mechanism for fatigue, as will be discussed later.

Crack lengths, a, were monitored in situ using measurements of the elastic unloading compliance of the sample, i.e., the slope of the load-line displacement/load curve. Load-line displacements were measured to within a quoted accuracy of ±1 μm using a linear variable-displacement transducer (LVDT) mounted in the load frame, from which crack lengths were deduced using the linear-elastic compliance solutions given in Ref. [31]. The experimental set-up is shown schematically in Fig. 1; further details on compliance crack-length monitoring methods as applied to dentin are described elsewhere [21]. Periodic optical microscopy measurements were used to verify the actual crack lengths, and to correct for small errors (typically 5–10%) due to "bridges" of intact material that can form and span the wake of the cracks, thereby affecting the compliance. Such corrections were made by assuming that errors accumulate linearly with crack extension. Fatigue-crack growth rates, da/dN, were characterized in terms of the stress-intensity range, $\Delta K = K_{max} - K_{min}$, where the maximum and minimum stress intensities, K_{max} and K_{min} respectively, were computed from the maximum and minimum loads of the loading cycle using standard handbook linear elastic stress-intensity solutions [32].

In order to test over a range of ΔK levels, the loads were continually adjusted such that the ΔK was varied with a controlled K-gradient, C', defined by the relationship:

$$\Delta K = \Delta K_{initial} \exp[C'(a - a_{initial})]. \quad (1)$$

The majority of the testing was performed under decreasing ΔK conditions, with C' nominally set at $-0.08\,mm^{-1}$; however, for growth rates exceeding $\sim 5 \times 10^{-7}$ m/cycle, increasing ΔK conditions were utilized, with a K-gradient of $C' \approx +0.08\,mm^{-1}$. Crack growth-rate data spanning ~2–3 decades between 10^{-10} and $\sim 10^{-5}$ m/cycle were measured at each test frequency. The fatigue-crack propagation data thus obtained was expressed in terms of a simple Paris power-law expression:

$$da/dN = C(\Delta K)^m, \quad (2)$$

[3]The technique of razor micronotching is used to permit easy initiation of a fatigue precrack. Actual fatigue-crack growth rate data are then collected once the precrack has grown ~50–100 μm so that subsequent measurements are not affected by the strain field of the notch.

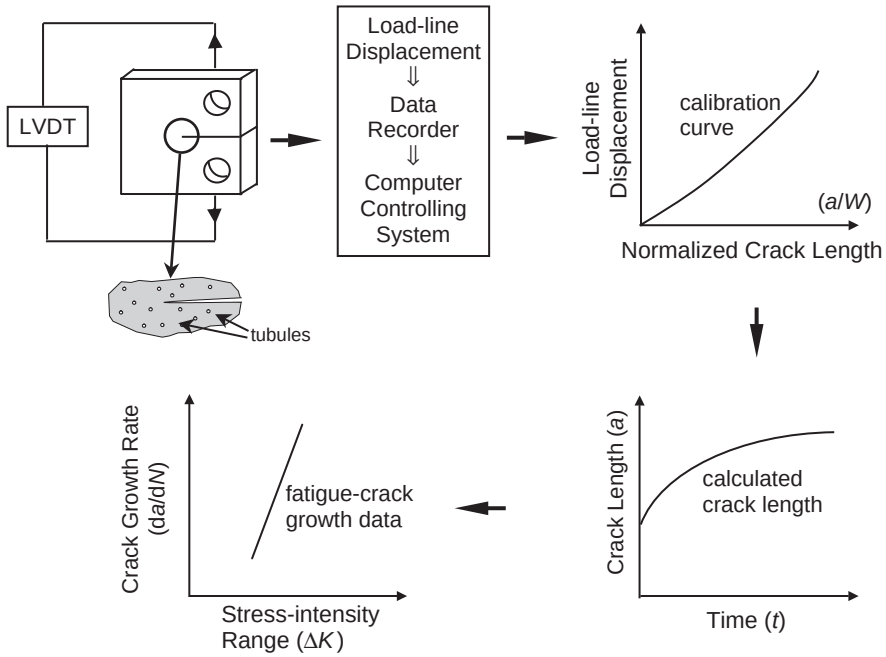

Fig. 1. Schematic illustration of the experimental setup used to measure crack lengths using sample compliance and to obtain the in vitro fatigue-crack growth data in this study. The orientation of the notch/crack with respect to the dentinal tubules is also shown.

where C and m are experimentally determined scaling constants.

In addition to the fatigue-crack propagation testing, experiments involving transitions between static and cyclic loading were performed using the remaining two $C(T)$ specimens in order to ascertain the role of static vs. cyclic fatigue mechanisms on subcritical crack growth. Such "fatigue/sustained load/fatigue" tests were performed in HBSS at $37(\pm 0.5)\,^{\circ}\mathrm{C}$ over a range of applied loads and crack sizes to maintain a constant maximum stress intensity ($K_{\max} = 1\,\mathrm{MPa\,m^{1/2}}$) and involved a three-step loading regimen:

- fatigue at a constant ΔK of $0.9\,\mathrm{MPa\,m^{1/2}}$ ($R = 0.1, 50\,\mathrm{Hz}$),
- sustained loading at the same $K_{\max}$ and
- fatigue under identical conditions to the first block.

During these tests, crack extension was again monitored using the sample compliance as measured by the LVDT, with corrections being made based on optical measurements at the end of each loading block.

Crack paths and fracture surfaces were subsequently examined using optical and scanning electron microscopy (SEM). SEM imaging was performed in the secondary electron mode after coating the specimens with a gold-palladium alloy to minimize specimen charging.

3. Results

3.1. Fatigue-crack growth

The variation in fatigue-crack propagation rates, $\mathrm{d}a/\mathrm{d}N$, with the stress-intensity range, ΔK, for dentin in 37 °C HBSS is shown in Fig. 2a from the results of the nine $C(T)$ specimens. Data spanning growth rates from $\sim 4 \times 10^{-9}$ to $\sim 9 \times 10^{-6}\,\mathrm{m/cycle}$ at 1 Hz, from $\sim 2 \times 10^{-10}$ to $\sim 1 \times 10^{-7}\,\mathrm{m/cycle}$ at 10 Hz, and from $\sim 1 \times 10^{-10}$ to $\sim 1 \times 10^{-8}\,\mathrm{m/cycle}$ at 50 Hz were obtained. Differences in the range of growth rates collected for each frequency reflect a balance between time constraints for slow growth rates and ease of controlling the test such that cracks did not rapidly go unstable causing catastrophic failure for high growth rates. However, all three ranges of data overlapped at ΔK levels between ~0.7 and $0.9\,\mathrm{MPa\,m^{1/2}}$, which permits a direct comparison of behavior at different frequencies.

Fatigue-crack propagation data in dentin were expressed in terms of the Paris relationship (Eq. (2)); values of the scaling constants, C and m, for the data in Fig. 2a are listed below in Table 1. The Paris exponents, m, for elephant dentin lay between 12 and 32; these values are higher than those reported in previous work on human and bovine dentin (and cortical bone) [15,18,26,33,34].

From the results in Fig. 2, it is clear that in vitro fatigue-crack growth rates in dentin are markedly sensitive to frequency; at a given stress-intensity range,

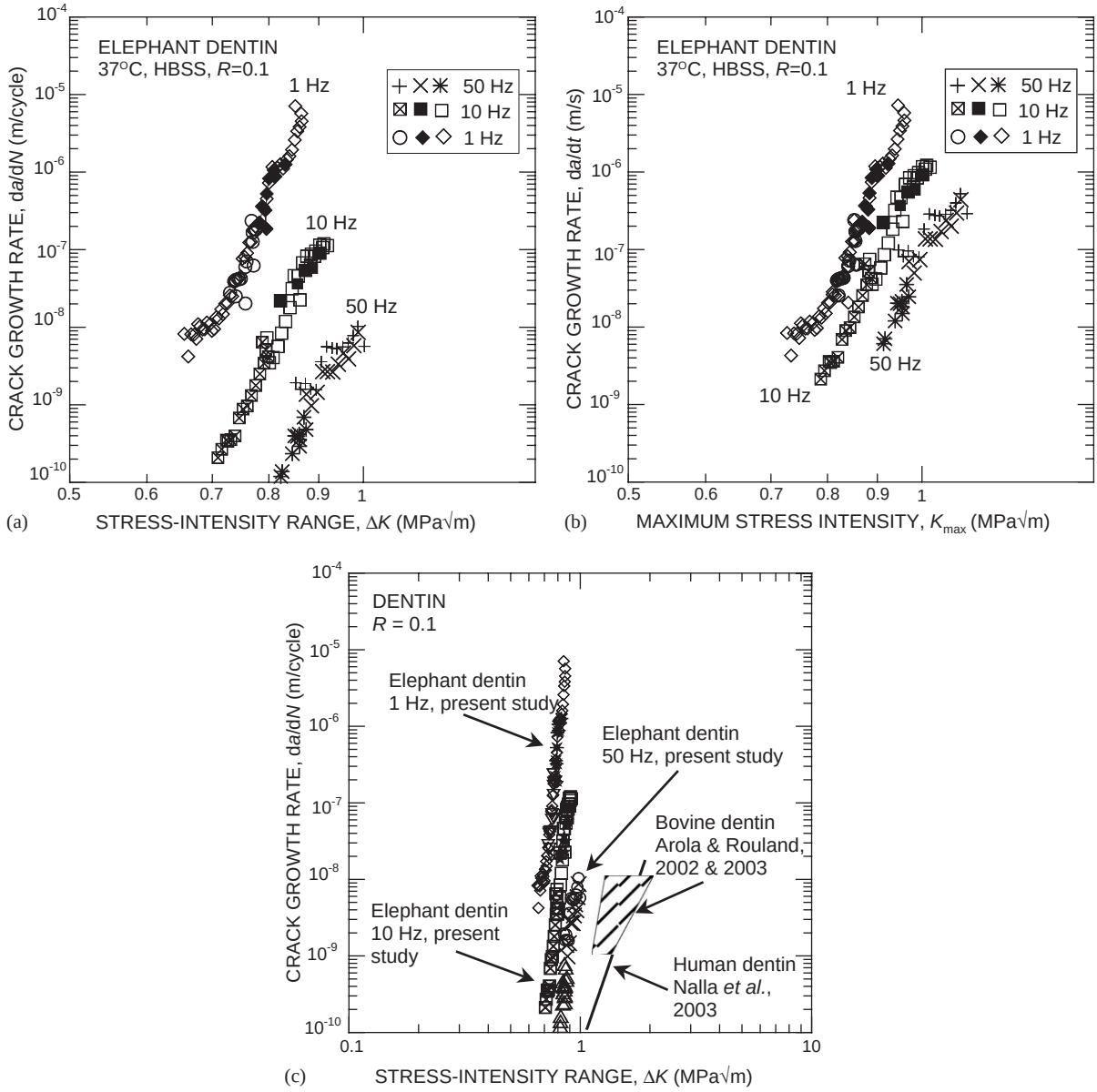

Fig. 2. (a) In vitro fatigue-crack growth data for elephant dentin are shown as plots of the crack-growth rates per fatigue cycle, da/dN, as a function of the stress-intensity range, ΔK, for the three test frequencies (1, 10 and 50 Hz) investigated. Tests were performed on compact-tension specimens at $R = 0.1$ in an environment of 37 °C HBSS. (b) The data in (a) are replotted in terms of crack-growth rates with respect to time, da/dt, as a function of the maximum stress intensity, K_{max}. (c) The data in (a) are compared with results from other fatigue-crack growth studies in dentin, by Nalla et al. [15] (human dentin, 2 Hz, $R = 0.1$, crack growth orientation perpendicular to tubules) and by Arola et al. [17,18] (bovine dentin, 5 Hz, $R = 0.1$, various orientations).

growth rates are increased with decreasing frequency. To assess the effect of frequency, these data were replotted in Fig. 2b in terms of the growth rates with respect to time, da/dt, as a function of the maximum stress intensity, K_{max}. It is apparent that characterizing on the basis of time, rather than cycles, does not normalize the growth-rate data at the different frequencies, implying that crack growth in dentin is definitely not purely time-dependent. However, the effect of frequency is definitively reduced, suggesting that crack growth in dentin is principally cycle-dependent, but with a smaller time-dependent component, similar to behavior in many structural materials [20].

A comparison of the present fatigue-crack propagation data on elephant dentin with the only other results reported in the literature, for human [15] and bovine dentin [17,18], is shown in Fig. 2c. It is apparent that growth rates are fastest, fatigue thresholds the lowest, and m values the highest, in the elephant dentin. Care, however, should be exercised in drawing too many conclusions on inter-species variations from this comparison as clearly only limited results are currently

Table 1
Constants in the Paris law for fatigue-crack growth in elephant dentin

Test frequency (Hz)	Constant C^a	Exponent n^a	Coefficient of determination (R^2)
1	1.8×10^{-4}	26.5	0.96
1	5.1×10^{-4}	32.1	0.65
1	6.4×10^{-4}	32.1	0.70
10	3.0×10^{-6}	28.5	0.94
10	1.8×10^{-6}	26.5	0.91
10	4.7×10^{-7}	16.7	0.87
50	9.6×10^{-9}	12.1	0.81
50	8.6×10^{-9}	15.2	0.91
50	1.1×10^{-8}	22.0	0.60

[a]Units: m/cycle $(\text{MPa m}^{1/2})^{-m}$.

available at this time and the three different studies reflect differing test conditions (temperature, environment, testing frequency), sample dimensions and orientation, all variables whose specific influence on cyclic crack-growth rates have yet to be fully documented.

3.2. Fatigue/sustained load/fatigue tests

The results of the constant-K_{max} "fatigue/sustained load/fatigue" tests, as described in Section 2.2, are shown in Fig. 3; typical crack extension (Δa) vs. time (t) data obtained for one of these tests are shown in the top panel. Crack growth at a velocity of approximately 5×10^{-8} m/s can be seen to occur readily under cyclic loading in the first block; however, at point A where the load is held steady to maintain the same K_{max} value (but with no cycling), crack growth becomes practically undetectable, until the fatigue cycling is resumed at point B. This experiment strongly implies that it is not the maximum stress itself, but the process of fatigue cycling, i.e. repeated loading and unloading between the maximum and minimum stresses, that primarily drives crack growth in dentin. The optical micrographs in Fig. 3 (bottom panels) show the near-tip crack profiles at various stages of the test. It is apparent that significant crack blunting occurs, as evidenced by the increased crack-opening displacements, during the sustained load portion of the test (Fig. 3b). There also appears to be evidence of crack growth in Fig. 3b and while it is plausible that this is the result of unloading from the sustained load prior to actual imaging, previous studies have shown similar small amounts of crack extension under sustained loads before the crack arrests, generally within the first hour [21]. Crack growth resumes once fatigue cycling is resumed, as is demonstrated in Fig. 3c.

Fracture surfaces representative of the fatigue and overload failure regions are shown in Fig. 4. Similar to human dentin [15,35], there was no evidence of fatigue striations on the fatigue fracture surface, akin to those seen commonly in many metallic alloys and polymers [20]. Furthermore, few differences exist between the observed morphology of the overload fractures (Fig. 4b) and those associated with cyclic fatigue-crack growth (Fig. 4a), although macroscopically the overload fracture surfaces are somewhat rougher. However, the inherent complexity of the underlying microstructure masks any subtle distinctions.

4. Discussion

4.1. Fatigue-crack propagation behavior

The principal results of this work are that in vitro fatigue-crack propagation rates in dentin (i) can be characterized in terms of a power-law function of the stress-intensity range, (ii) are sensitive to cyclic frequency, and (iii) mechanistically involve a cycle-dependent process. The latter result, which is of importance as it establishes a true fatigue mechanism in dentin, can be readily appreciated from the "fatigue/sustained load/fatigue" results in Fig. 3. By examining the transition from fatigue to sustained loading at the same K_{max}, i.e., at the transition A in Fig. 3, it is clear that the unloading portion of the fatigue cycle is essential to crack growth; without such unloading, the growth rate is dramatically reduced. However, the fact that propagation rates are sensitive to frequency also implies the presence of a time-dependent contribution to cracking, although akin to most metallic materials, this is clearly secondary.

These results are consistent with previously reported stress-life data for human dentin [15], although unlike the S/N data they *do* establish the clear role of cycle-dependent mechanisms. Indeed, the frequency effect seen on fatigue-crack growth (Fig. 2), where (at the same ΔK) an order of magnitude increase in growth rates can be associated with an order of magnitude reduction in cyclic frequency, is in agreement with the S/N data which demonstrated shorter fatigue lifetimes at lower frequencies [15]. It is also consistent with many results on fatigue-crack propagation rates in the literature on metallic materials [20], where for a given ΔK level, growth rates per cycle tend to be faster at lower frequencies as there is more time during each cycle for time-dependent processes, such as environmentally induced cracking, to contribute to crack advance.

While the present results give conclusive evidence regarding fatigue-crack propagation behavior, they provide little insight into the role cycling frequency or test time versus number of cycles on the ease of *initiating* a dominant crack. Previous in vitro studies in human dentin [15] have clearly indicated how crack initiation at a specific applied stress is significantly easier under cyclic loading. It is thought that this may be related to the crack-tip blunting which is observed in hydrated dentin. Such behavior is seen in lower strength metallic

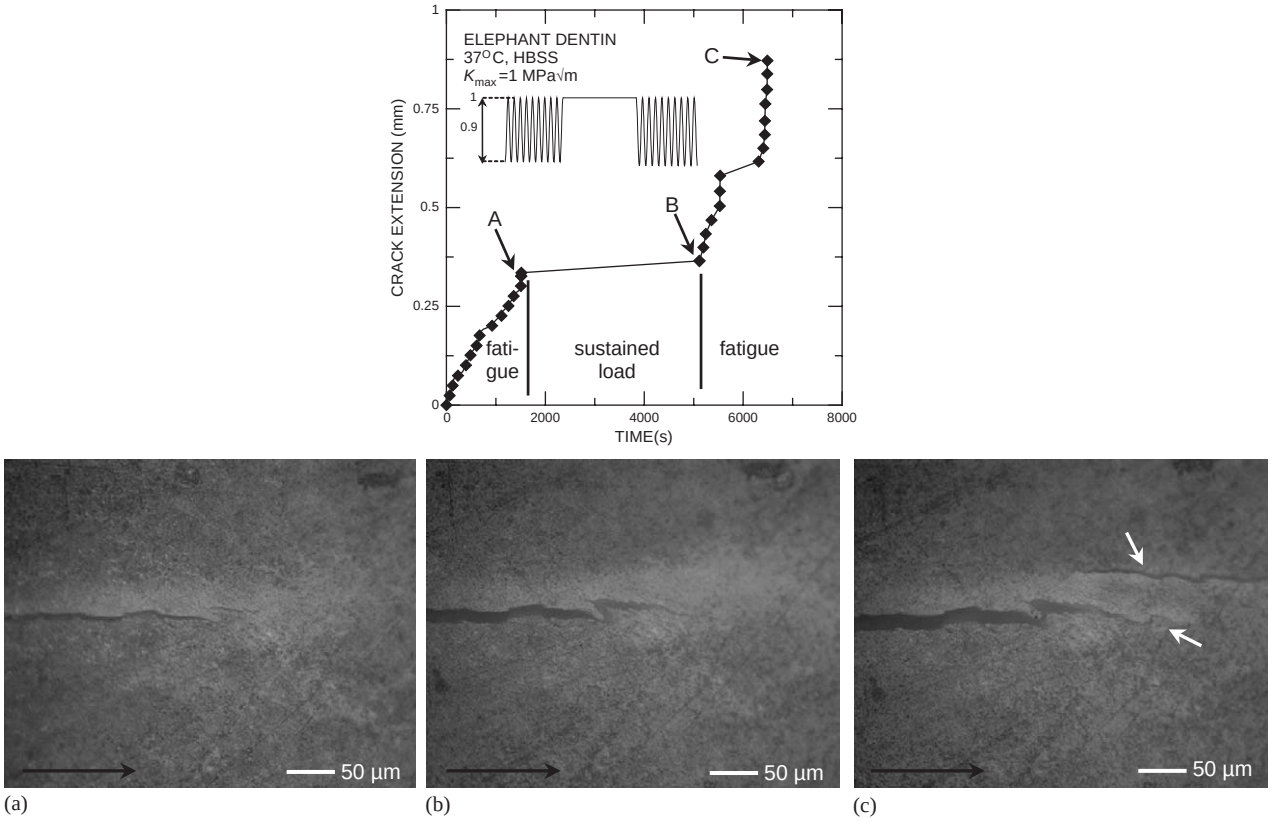

Fig. 3. Results of the "fatigue-sustained load-fatigue" tests at a fixed K_{max} level of 1 MPa $m^{1/2}$ (see inset), presented as plots of crack extension as a function of time, together with optical micrographs of the crack path at the end of each loading block. Note the substantial crack blunting during the sustained load block (b), and the subsequent crack extension during the second fatigue block (indicated by white arrows in c). The black arrow in each micrograph indicates the direction of nominal crack growth.

materials, where cyclic loading can maintain the relative sharpness of any initial cracks that would otherwise macroscopically blunt (and often arrest) under sustained loads. However, from a perspective of understanding the structural life of dentin in a tooth, issues of initiation may not be so important, as preexisting flaws and notches will inevitably exist in the tooth. Accordingly, we believe that a damage-tolerant approach to fatigue, where the useful life is estimated solely in terms of time or loading cycles to cause an existing crack to propagate to failure, is more relevant clinically. Thus, this is a primary reason why the current work is focused on fatigue-crack propagation.

4.2. Fatigue-crack growth mechanisms

The cycle-dependent fatigue-crack growth behavior for hydrated dentin reported in this study (Figs. 2 and 3) together with direct observations of crack-tip blunting (Fig. 3) provide support for the notion that cyclic crack extension occurs as a result of alternating blunting and resharpening of the crack tip, despite the fact that fatigue striations were not observed. As shown schematically in Fig. 5, this mechanism, by which fatigue cracks grow in ductile materials such as metals [22], involves blunting of the crack during the loading portion of the cycle due to permanent plastic deformation[4] (Fig. 5b). With such plastic deformation limited to the vicinity of the crack tip, upon unloading the surrounding linear elastic material compresses the deformed near-tip region (e.g., [37]), resulting in resharpening of the crack tip, with the new crack area contributing to crack advance. Previous work on dentin [21] has shown that the degree of crack blunting is time-dependent, with increased blunting occurring with longer times at load. This implies that at slower cycling frequencies, which extends the time near the peak load during each cycle, more blunting would occur during each cycle, giving rise to more crack advance per cycle than with higher frequencies, i.e., consistent with the results in Fig. 2.

While in pure metals and many ductile alloys visible fatigue striations often provide evidence for the accepted

[4]The precise mechanisms of plasticity, or more appropriately, inelasticity, in mineralized tissues such as dentin and bone are not clearly understood but are believed to involve such processes as microcracking and microdamage, and deformation of the collagen fibrils [36].

blunting and resharpening mechanism [20,22], no evidence of striations was found on the fatigue surfaces of dentin in the present study, nor in our previous study on human dentin [15]. This perhaps is not too surprising considering that ductile fatigue striations seen in metals are often the result of a specific deformation mechanism, e.g., dislocation emission on alternate slip planes at the crack tip [20], which would not occur in dentin. Additionally, striations are rarely found in materials with complex microstructures as their formation and visibility can be masked by the underlying structure [20].

Finally, since it is known that crack propagation in dentin is associated with the formation of bridges, by uncracked ligaments or individual collagen fibrils, which act in the wake to impede the opening of the crack [21], it is also plausible that the mechanism of fatigue may additionally involve the cycle-dependent degradation of such bridging elements, akin to the mechanism seen in brittle materials such as toughened ceramics [20]. However, careful visual inspection with optical microscopy of the crack wake of cracks grown under both cyclic and monotonic loading revealed no evidence of smaller bridging zones in fatigue, suggesting that this mechanism may not be relevant in dentin.

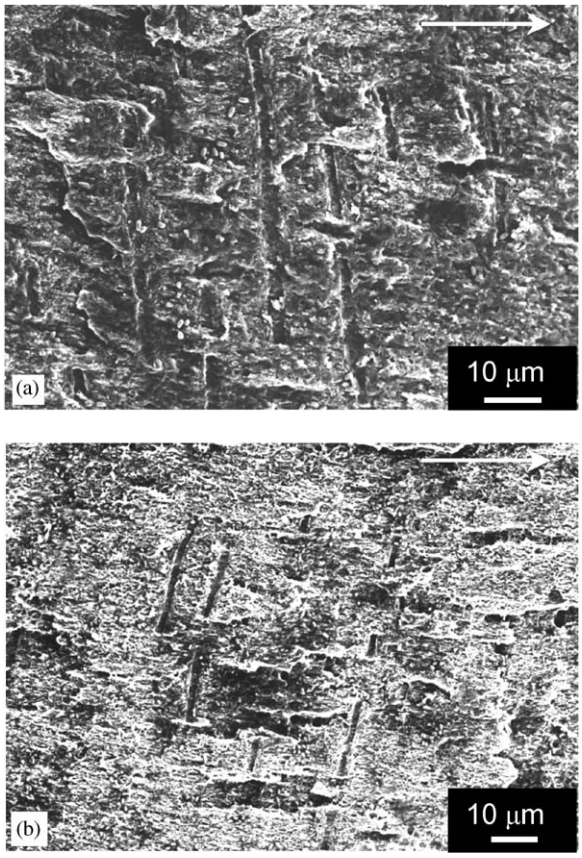

Fig. 4. Typical scanning electron micrographs of the (a) fatigue and (b) overload fracture regions. Note that the overload surface is macroscopically rougher. The tubules appear as "tire tracks" on the fracture surfaces. The white arrow at the top of each micrograph indicates the direction of nominal crack growth.

5. Conclusions

Based on an in vitro investigation of the fatigue-crack growth behavior of hydrated elephant dentin in 37 °C Hanks' Balanced Salt Solution, the following conclusions can be made:

1. Fatigue-crack propagation rates in dentin, in a nominal direction perpendicular to the long axis of the tubules with the crack plane in the plane of the tubules, can be expressed in terms of a Paris power-law, with growth rates over the range $\sim 10^{-10}$–10^{-5} m/cycle characterized by the range in stress intensity, ΔK, with power-law exponents between $m = 12$ and 32.
2. Such fatigue-crack growth was found to be markedly sensitive to the cyclic frequency (in the range 1–50 Hz) with faster growth rates (at a given ΔK level) occurring at lower frequency.

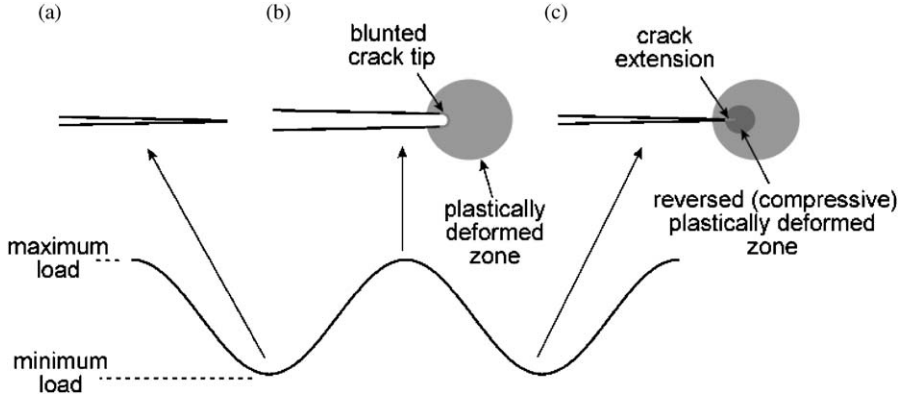

Fig. 5. Schematic illustration of the proposed mechanism of alternating crack-tip blunting and resharpening for fatigue-crack growth in dentin, showing (a) the crack at the beginning of the loading cycle, (b) the blunted crack at the peak of the loading cycle, and (c) the resharpened, extended crack after unloading.

3. Experiments at constant K_{max} where the loading is changed from cyclic to sustained and then back to cyclic provide strong evidence that a ''true'' cyclic fatigue-crack growth mechanism exists in dentin, i.e., fatigue-crack growth may not be considered as simply a succession of static fracture events in dentin (akin to ceramics).
4. The effect of frequency on crack growth, together with direct observations of crack blunting, suggest that the cycle-dependent mechanism of fatigue-crack advance in dentin occurs via a mechanism of alternating crack-tip blunting and resharpening, although no confirming evidence of fatigue striations could be detected on fatigue fracture surfaces. Because such blunting is time-dependent, a slower frequency would be expected to permit more blunting, and correspondingly a larger crack increment per cycle, consistent with the higher crack-growth rates observed at lower frequencies.

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