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Contributions of Aging to the Fatigue Crack Growth Resistance of Human Dentin

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Abstract

An evaluation of the fatigue crack resistance of human dentin was conducted to identify the degree of degradation that arises with aging and the dependency on tubule orientation. Fatigue crack growth was achieved in specimens of coronal dentin through application of Mode I cyclic loading and over clinically relevant lengths (0 to 2 mm). The study considered two directions of cyclic crack growth in which the crack was either in-plane (0°) or perpendicular (90°) to the dentin tubules. Results showed that regardless of tubule orientation, aging of dentin is accompanied by a significant reduction in the resistance to the initiation of fatigue crack growth, as well as a significant increase in the rate of incremental extension. Perpendicular to the tubules, the fatigue crack exponent increased significantly (from $m=14.2\pm1.5$ to 24.1 ± 5.0), suggesting an increase in brittleness of the tissue with age. For cracks extending in plane with the tubules, the fatigue crack growth exponent does not change significantly with patient age (from $m=25.4\pm3.03$ to 22.9 ± 5.3), but there is a significant increase in the incremental crack growth rate. Regardless of age, coronal dentin exhibits the lowest resistance to fatigue crack growth perpendicular to the tubules. While there are changes in the cyclic crack growth rate and mechanisms of cyclic extension with aging, this tissue maintains its anisotropy.

Keywords

anisotropy; dentin; fatigue crack growth; fracture

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INTRODUCTION

Dentin, the hard tissue occupying the majority of the human tooth, is an interesting structural material. This tissue is approximately 45% mineral, 33% organic material (primarily type I collagen) and 22% fluid by volume [1], a composition similar to that of cortical bone. Perhaps the most distinct feature of the microstructure is the network of microscopic channels (i.e. tubules) that extend outward from the pulp towards the Dentin-Enamel Junction (DEJ) and cementum. The tubule geometry is dependent on location within the tooth and distance from the pulp chamber. Overall, the lumens exhibit an average diameter exceeding one micrometer (ranging from approximately 1 to 2.5 μm) and a density from roughly 10,000 to 60,000 per mm^2 [2, 3]. Each tubule lumen is bordered by a highly mineralized cuff of peritubular dentin having thickness ranging from 0.5 to 1 μm . The region between the tubules, regarded as intertubular dentin, consists of a collagen fibril mesh that is supported by both inter- and intra-fibrillar apatite crystallites [4, 5].

Both cortical bone and dentin are commonly regarded as hierarchical structural materials, a characterization that originates from the constituents, the multiplicity in scale and their complex integrated relationship [6–9]. Nevertheless, microscopic evaluations of dentin are dominated by the dentin tubules and their arrangement. Due to their appearance as reinforcing fibers, dentin is often perceived to possess mechanical anisotropy. Surprisingly, dentin exhibits only a small degree of elastic anisotropy with the largest elastic modulus obtained for loading perpendicular to the tubules [10–12]. The tensile strength of human dentin exhibits a greater degree of orientation dependence, with the largest strength obtained in the direction perpendicular to the tubules [13–15]; the root exhibits the greatest degree of anisotropy [16, 17]. Though the tubules and the peritubular cuffs are the most obvious structural components, the differences in strength have been perceived to result from the collagen fibril orientation rather than the tubules [5].

Due to the cyclic loading that develops during mastication and the potential for flaws to be introduced within teeth via the practice of restorative dentistry, the fatigue and fracture properties of dentin are critically important. The importance of tubule orientation on these measures of mechanical behavior and apparent anisotropy has been explored to some extent. In response to cyclic loading, the lowest fatigue strength is obtained for loads applied parallel to the tubules [18] and cracks prefer to grow perpendicular to the tubule axis during fatigue crack growth [19]. Consistent with that observation, an evaluation of the work of fracture showed that dentin exhibits the largest resistance to fracture for cracks extending in-plane (i.e. parallel) with the dentin tubules [20]. Iwamoto and Ruse [21] reported that the fracture toughness of human dentin was approximately twice as large for cracks extending in-plane and parallel to the tubules, when compared to fracture that occurs perpendicular to the tubules. The importance of tubule orientation to the fracture resistance has been attributed to differences in the toughening mechanisms and participation of the collagen fibrils [22].

While cortical bone and dentin have nearly equivalent composition, two qualities set these tissues apart. Aside from the cytoplasmic extensions within the tubule lumens, the majority of the dentin is acellular. As such, dentin does not have the ability to undergo remodeling and/or repair defects (e.g. damage or cracks) in the same manner as bone. Secondly, bone undergoes a reduction in both density and degree of mineralization with aging [23–25], which contribute to an increase in bone fragility with patient age. In contrast, aging of dentin is accompanied by an increase in mineral content as a result of the deposition of mineral within the lumens [26]. This process begins in the third decade of life and continues progressively until complete filling of the lumens [27], at which point the tissue becomes transparent and is regarded as “sclerotic”. The influence of this gradual change in

microstructure on the mechanical behavior of dentin is an important topic to the practice of restorative dentistry. Studies performed using nanoindentation have shown that there are minimal changes in the hardness and elastic modulus of dentin with age [28], except for the mantle region where both the hardness and elastic modulus increase [29]. Yet, there is a significant reduction in the strength of dentin with patient age under both monotonic and cyclic loading [26, 30] as well as a reduction in the resistance to fatigue crack growth [26, 31]. Furthermore, independent studies of the R-curve behavior also have shown that dentin undergoes a significant reduction in the fracture toughness with age [32–34].

The aging process causes distinct changes to the microstructure of dentin, which appear to be detrimental to the mechanical behavior. In young dentin the inherent anisotropy of the tissue deters cracks from extending axially along the tubules and towards the vital pulp, a natural defense to tooth loss via fracture. In the case of old dentin, fracture more commonly occurs along the tooth's long axis and in-plane with the tubules [35–37]. No study has addressed whether aging results in diminution of the anisotropy in crack growth resistance that is exhibited by young dentin. Therefore, the primary objective of this study was to evaluate the changes in the fatigue crack growth behavior of coronal dentin as a function of patient age and to assess if these changes are dependent on the tubule orientation. The null hypothesis of the investigation was that the microstructural changes in human dentin that occur with aging do not cause reduction in anisotropy of the fatigue crack growth resistance.

MATERIALS AND METHODS

Non-carious third molars were obtained from dental practices in Maryland according to an approved protocol issued by the Institutional Review Board of the University of Maryland Baltimore County (Approval Y04DA23151). The teeth were obtained from patients ranging from 17 to 89 years of age and stored in Hanks Balanced Salt Solution (HBSS) with record of patient age and gender. Two simple age groups were formed with the collected teeth, which are described conveniently as the young (17 age 33) and old (55 age) age groups.

All teeth were sectioned within one month of extraction using a numerical controlled slicer/grinder (Chevalier, smart H818II), diamond abrasive slicing wheels and copious water-based coolant. Serial sections were made either parallel or perpendicular to the tooth's axis to obtain specimens from the coronal region with desired tubule orientation. Secondary sections and other features were introduced as necessary to develop the Compact Tension (CT) specimen geometry as described in Figure 1(b). Holes were introduced to enable cyclic loading and a well-defined notch was prepared for initiating a crack using methods presented previously [31, 38]. Note that only one specimen was prepared from each tooth. The two approaches to sectioning resulted in specimens with tubules parallel (0° orientation) or perpendicular (90° orientation) to the fracture surface as evident in Figure 1. In the 0° orientation the tubules are in-plane with the fracture surface, but perpendicular to the direction of crack extension. For the 90° orientation a total of 47 specimens were evaluated including young (N=32) and old (N=15) specimens. For the 0° orientation, 38 specimens were evaluated, including N=28 young and N=10 old specimens.

Fatigue loading of the CT specimens was performed using a BOSE ElectroForce 3200 universal testing system using routine methods described elsewhere [31, 39]. Briefly, the specimens were subjected to cyclic loading under load control at 5 Hz testing frequency while immersed within an HBSS bath at room temperature (22°C). The frequency of loading is higher than that of normal occlusion (~ 1 Hz), but was chosen to balance concerns related to clinical relevance and the duration of time required to complete the individual tests. The testing frequency was also chosen to maintain consistency with previous studies [31, 40, 41]. Prior to loading the notch tip of each specimen was sharpened using a razor blade and 1

mm diamond paste, resulting in a notch-tip radius of approximately 20 μm . Crack initiation was achieved from the sharpened notch by cyclic loading with a stress ratio (R = ratio of minimum to maximum load) of 0.5, which served to accelerate the initiation process [42]. After identification of a well-defined tip, the crack was extended approximately 0.5 mm, i.e. outside the region of stress gradient posed by the notch. As crack initiation was often evident through an increase in the specimen's compliance, the actuator displacement was monitored visually after the beginning of cyclic loading. If after a period of loading (e.g. 40kcycles or larger) there was no evidence of crack extension from changes in compliance, the crack tip was visually examined using a digital microscope (Navitar IEEE 1394, T series) to identify growth. In short, a comparison of the crack characteristics before and after the loading increment was performed. In the case that crack extension was not achieved, cyclic loading was resumed using $R=0.1$ and the increasing ΔK approach to determine the ΔK_{th} value. However, if visual inspection revealed the onset of growth, then cyclic loading was continued using the conventional incremental evaluation with constant ΔK .

The incremental crack growth rates (da/dN) were computed by dividing the measured incremental crack extension (Δa) by the increment of loading cycles (ΔN). Crack length measurements were achieved using a digital microscope (Navitar IEEE 1394) at magnifications of 36X and 60X. The number of cycles between measurements (ΔN) was chosen according to the observed crack growth rate and typically ranged between 5 and 20 kcycles; the average increment of crack extension was between 50 to 200 μm .

The incremental fatigue crack growth rate (da/dN) within the region of steady state response (Region II) was quantified using a power law model after Paris et al [43] according to

$$\frac{da}{dN} = C(\Delta K)^m \quad (1)$$

where ΔK is the stress intensity range, and the quantities C and m are the fatigue crack growth coefficient and exponent, respectively. The stress intensity range (ΔK) is determined from the difference in stress intensity at the minimum and maximum loads according to [31]. Using the incremental crack length measurements and the corresponding stress intensity, the fatigue crack growth rate (da/dN) was plotted in terms of ΔK to estimate the quantities m and C for each specimen. In addition, the apparent stress intensity threshold (ΔK_{th}) was estimated from the responses at the stress intensity range at which cyclic crack growth ensued. Thus, results for each specimen included quantitative estimates of ΔK_{th} , m and C . Cumulative fatigue crack growth responses were obtained in terms of these parameters for each group comprised of specific age (young and old) and tubule orientation (0° and 90°). In addition, results for the young samples with 0° orientation were further subdivided in terms of relative distance from the pulp based on differences in the tubule density and the mineral to collagen ratio [44]. A one-way analysis of variance was used to analyze the fatigue crack growth parameters obtained for each group of specimens. A $p < 0.05$ was considered significant.

The fracture surfaces for each CT specimen were evaluated using a JEOL Model JSM-5600 scanning electron microscope in secondary electron imaging mode. Micrographs were also obtained from the viewing surface monitored during fatigue crack growth for the 0° orientation specimens to enable convenient measurement of the tubule density and microstructural features. An image analysis software in the public domain (NIH Image J) was used for quantifying the average lumen dimensions.

RESULTS

Representative fatigue crack growth responses for young coronal dentin in both orientations of cyclic crack extension are shown in Figure 2. The parameters used in quantifying the behavior, including the stress intensity threshold (ΔK_{th}) and the steady-state fatigue crack growth parameters (m and C) are highlighted for clarity. Note the difference in apparent resistance to the initiation of cyclic crack growth, and the overall lower incremental growth rate of the specimen with 0° orientation. The fatigue crack growth responses for all specimens with 0° tubule orientation are shown as a function of the stress intensity range in Figure 3(a). Due to the potential importance of location, the responses for young dentin are divided into three relative regions including near the pulp, midway from the pulp and nearest the DEJ. These three regions of the crown are regarded as the inner, central and peripheral regions, respectively. The fatigue crack growth responses obtained for young and old dentin for samples with 90° orientation are shown in Figure 3(b). As a result of the methods of preparation and limited tissue available in the crown, the evaluation for this tubule orientation was limited to central dentin.

When examining the responses in Figure 3, both tubule orientation and patient age appear to have important contributions to the nature of cyclic crack extension. Average values for the fatigue crack growth parameters corresponding to the initiation and steady-state growth behavior were determined from the cumulative results and are presented in Table 1. For young dentin, tubule orientation was important to all aspects of the fatigue crack growth behavior. The initiation of cyclic extension perpendicular to the tubules occurred at a significantly lower ($p < .0005$) stress intensity range ($\Delta K_{th} = 0.83 \pm 0.11 \text{ MPa} \cdot \text{m}^{0.5}$) than for cyclic crack growth in-plane with the dentin tubules ($\Delta K_{th} = 1.03 \pm 0.06 \text{ MPa} \cdot \text{m}^{0.5}$). Within the regions of steady-state cyclic extension, the cyclic crack growth exponent (Fig. 4(b)) for the 0° orientation ($m = 25.47 \pm 3.03$) was significantly greater ($p < .0005$) than that for the 90° orientation ($m = 14.15 \pm 1.55$). There was also a significant difference in the cyclic crack growth coefficient with respect to the tubule orientation for young dentin ($p < .0005$). The significant difference in the responses obtained for the two tubule orientations (Table 1) confirms that the fatigue crack growth resistance of young dentin is anisotropic.

Also evident from the results in Figure 3, age and relative depth of the tissue from the DEJ were important to the fatigue crack growth responses. A comparison of the fatigue crack growth parameters that includes the three regions of evaluation for the 0° orientation is presented in Figure 4. In the 0° orientation there was a significant difference between the average stress intensity threshold obtained for the old dentin and that for the peripheral and central regions of young dentin ($p < .0005$ and $p < .0005$, respectively) as evident in Figure 4(a). However, the difference between ΔK_{th} of the young inner dentin and that of the old age group was not significant ($p > .05$). In comparing the parameters associated with steady-state fatigue crack growth the results were equally interesting. There was no significant difference ($p > .05$) in the fatigue crack growth exponents between the young and old dentin for the 0° orientation (Table 1). This statement is true whether pooling the fatigue crack growth responses for inner, central and peripheral dentin, or simply evaluating the three regions separately as evident in Figure 4(b). However, there was a significant difference ($p < .0005$) between the average fatigue crack growth coefficient obtained for the old dentin and that for the central and peripheral regions of young dentin (Figure 4(c)). The average value of C for the old dentin with 0° orientation is more than three factors of magnitude larger than that of the young dentin in the central and peripheral regions, indicating the higher incremental fatigue crack growth rates in the older tissue. In contrast, the tissue closest to the pulp (inner dentin) performed similar to that of old dentin (Fig. 4(c)) and did not exhibit a significant difference in C from that of the old age group.

Similar to the changes in initiation behavior for cracks oriented in plane with the tubules, there was a significant ($p = .0005$) reduction in the stress intensity threshold with age for cyclic crack growth in the 90° orientation as well; the average ΔK_{th} reduced from 0.83 ± 0.11 to $0.60 \pm 0.08 \text{ MPa}\cdot\text{m}^{0.5}$ (Table 1). In comparing the steady state fatigue crack growth responses, there were significant differences ($p = .0005$) between the values of m and the values of C obtained for the young and old dentin (Table 1). The average value of the fatigue crack exponent nearly doubled from an average of 14.15 ± 1.55 to 24.16 ± 4.33 (Fig. 4(b)). The increase in m with age signifies that there is a rise in sensitivity to the stress intensity range, which denotes an increase in brittleness. There was also a significant difference in the crack growth coefficient between the two age groups as evident in Table 1.

The results presented in Figures 3 and 4 reveal the impact of aging on the fatigue crack growth responses in each tubule orientation considered, but they do not clearly distinguish if the old dentin exhibits anisotropy. A comparison of the results for the two different tubule orientations limited to old dentin is shown in Figure 5. In examining the initiation behavior, the difference in ΔK_{th} for the two orientations was significant ($p = .0005$) as noted in Table 1. For the steady-state responses, there was no significant difference in m between the responses of old dentin with 0° and 90° tubule orientations. However, there was a significant difference ($p = .0005$) in the crack growth coefficients. The value of C obtained for the 90° orientation was more than two factors of magnitude greater than that obtained for the 0° orientation. Over the stress intensity range associated with cyclic extension, the higher incremental fatigue crack growth rates occurred perpendicular to the tubules in the old dentin. This difference in response between the two orientations reveals that old dentin exhibits anisotropy in both the initiation of fatigue crack growth and the average rate of cyclic extension.

To understand contributions of the microstructure to fatigue crack growth, sequential images were obtained to document the nature of cyclic extension for each tubule orientation. Figure 6(a) shows a sequence of images corresponding to the path of cyclic extension in a young dentin sample with 0° tubule orientation. As evident from these images, the crack extended through fracture of adjacent peritubular cuffs along a path consistent with the highest local lumen density and within the region of maximum opening mode stress. At the viewing surface, the crack did not undergo extension in each cycle, but typically remained at an open lumen for a short period of cyclic loading (hundreds of cycles), and then continued to an adjacent lumen or along a sequential set of lumens to the next location. Consistent with previously reported observations [45], occasionally fluid or bubbles were noted escaping from a lumen during loading, which must arise from water movement within the tubule. In regions of non-uniform density the crack proceeded along the path with highest apparent lumen density, or adjusted its path to that with higher number of sequential lumens. A similar sequence of crack growth images for the 90° orientation is shown in Figure 6(b). In this orientation the Mode I crack path appeared less tortuous and was not as clearly influenced by microstructural features evident from the viewing surface.

Occasionally the crack would undergo branching, or a satellite crack (sometimes referred to as a “daughter crack”) developed above or below the apparent crack as highlighted in Figure 6(b), which was not connected with the primary crack. Previous studies have reported similar observations [7, 46]. Akin to cyclic extension in-plane with the tubules, crack extension did not occur every cycle of loading, but rather consisted of an increment of extension, a period of dwell with cyclic loading, and then extension to another location with advance of often between 5 to 10 μm .

An evaluation of the fracture surfaces using electron microscopy provided additional details regarding the mechanisms of cyclic crack extension and the importance of aging.

Micrographs of the fracture surfaces for the 0° tubule orientation are shown in Figure 7(a). Surfaces generated by fatigue crack growth in young dentin show that the crack proceeded through fracture of the peritubular cuffs as evident from the fractured cuffs and exposed lumens. In the old dentin some of the interior lumens were exposed as a result of cuff fracture, but that was limited to those that had not undergone complete filling with mineral. There was also evidence where the crack proceeded about the peritubular cuff boundary, rather than through the mineralized region. Overall, the fatigue crack growth surfaces of the old samples appeared less tortuous than those for young dentin. A comparison of representative fracture surfaces for young and old samples with 90° orientation is shown in Figure 7(b). Most evident is the marked reduction in lumen diameter of the tubules in the old sample, which resulted from the deposition of mineral within. While there was no evidence of debonding between the intertubular and peritubular dentin in the young samples, there was some degree of interfacial debonding observed in the old group. Consistent with observations made for the 0° orientation, the fracture surfaces appeared to be less tortuous, due primarily to the reduction in number of ligaments that were noted to develop along the fracture surface.

DISCUSSION

According to the experimental results, in both young and old dentin the fatigue cracks oriented perpendicular to the tubules underwent initiation and cyclic extension at the lowest stress intensity range. This behavior is consistent with the nature of anisotropy identified in earlier studies limited to young dentin, which focused on the fatigue strength [18, 39] and fracture toughness [21, 22]. With confirmation that the fatigue crack growth resistance of both young and old dentin is anisotropic, the remaining question is whether there is a difference in the degree of anisotropy between these two age groups. Results showed that there was a significant reduction in ΔK_{th} with age for both tubule orientations (Table 1). For the initiation regime, the degree of anisotropy can be characterized in terms of the ratio in ΔK_{th} for the 0° and 90° orientations. In comparing responses for the 0° orientation, the nature of cyclic extension in-plane with the tubules is highly dependent on the distance from the pulp (Fig. 3(a)). Therefore, the degree of anisotropy could be subject to spatial variations and should be considered. For young coronal dentin, the degree of anisotropy identified by the ratio of ΔK_{th} is indeed a function of location, with the values ranging from 1.48 (peripheral region) to 1.0 (inner dentin). The average of these values for young dentin is 1.24 and is essentially equivalent to that for the old dentin (1.28), which was computed using all results, (i.e. tissue from the entire crown). Thus, when applied to the initiation behavior of dentin the null hypothesis must be accepted. Despite the microstructural changes occurring with age (Figure 7), old human dentin exhibits a higher resistance to the initiation of fatigue crack extension in-plane with the tubules.

Quantifying anisotropy in the steady-state regime of fatigue crack growth is less objective. However it is possible to estimate the degree of anisotropy by the ratio of stress intensity range (ΔK) necessary to achieve a specific fatigue crack growth rate. This value of ΔK could be considered the necessary driving force to achieve a specific incremental rate of cyclic crack extension. According to the responses in Figures 3 and 5, a growth rate of 1×10^{-5} mm/cycle is an acceptable choice as it lies within the range of growth rates achieved in all regions of evaluation. It is also consistent with the rate estimated from fatigue crack growth surfaces of cracks found in restored teeth [40]. Thus, the degree of anisotropy for steady state cyclic extension is the ratio of ΔK for the 0° and 90° orientation that is necessary to achieve a growth rate of 1×10^{-5} mm/cycle. A previous investigation focused on fatigue crack growth in-plane with the tubules [41] showed that the driving force necessary for steady state fatigue crack growth was depth dependent and largest for the peripheral dentin. That could suggest that degree of anisotropy decreases with depth from the

peripheral region to inner dentin. The ratio of necessary driving forces for the 0° and 90° orientations ranged from 1.57 within the peripheral dentin to 1.06 within the inner dentin; the average degree of anisotropy for the three regions is 1.32. Thus, consistent with the initiation behavior the degree of anisotropy for the young dentin is location dependent. Note that these estimates incorporated the average value of driving force obtained for central dentin in the 90° samples as the importance of depth was not explored for this orientation. For the old dentin the degree of anisotropy distinguished by the ΔK ratios for the two orientations is 1.25, which is within 5% of the average value obtained for young dentin (1.32). Using the average values for central dentin in the two orientations reveals that the degree of anisotropy does not decrease with age. Thus, when applied to the steady-state fatigue crack growth responses, the null hypothesis must be accepted as well. Coronal dentin exhibits the highest incremental fatigue crack growth rate for crack extension perpendicular to the tubules, regardless of age.

Although the degree of anisotropy exhibited by tissue of the young and old age groups was consistent, there were differences in how age influenced the fatigue crack growth responses for each tubule orientation. For cracks extending in-plane with the tubules, there was essentially no change in the fatigue crack growth exponent with age, but a marked increase in the fatigue crack growth coefficient. In contrast, for the 90° there was a significant change in both parameters describing the steady-state region of crack growth. This difference can be described in terms of the unique mechanisms of cyclic extension in the two orientations. In the 0° orientation, cyclic crack extension took place along a path of adjacent peritubular cuffs within the K-dominant region that were most favorably positioned in front of the crack tip (e.g. Fig. 6(a)). There were no differences in this mechanism from the inner to the peripheral region, only the distance to the nearest neighboring lumen. Indeed, the fatigue crack growth exponents for young dentin in the three regions of characterization were essentially equal (Fig. 4(b)), indicating that the mechanisms of cyclic extension were equivalent over the range in tubule density. With aging, that process of cyclic crack extension was unchanged, i.e. a path established by microfracture of peritubular cuffs. But there were variations that developed within the areas of tissue in which the lumens were largely filled with mineral. In these regions the stress concentration acting on the peritubular cuff was reduced, or even eliminated in cases where the lumen was completely filled. Crack advance by microcracking of the peritubular cuffs is less likely or suppressed in regions of occluded lumens, thereby forcing the crack to proceed about the cuff and along the boundary of the intertubular and peritubular dentin (e.g. Fig. 7(a)), or through the collagen matrix. Note that crack extension within old dentin of this orientation results in a larger path length within the intertubular space. Thus, the increase in C with aging suggests that there is a reduction of the relative crack growth resistance of the intertubular dentin. Recognizing that changes to the apatite crystals within the intertubular dentin with age are limited to only small variations in size [26, 47], the increase in C could arise from a modification of the collagen matrix or the relationship between the organic and the inorganic constituents. This hypothesis is suggestive and not conclusive. For example, an alternate explanation might stem from the reduction in number of open lumens with aging, which causes a reduction in the opportunity for crack tip blunting in old dentin when the crack tip has advanced within the peritubular cuff.

For fatigue crack growth occurring perpendicular to the tubules, both parameters (m , C) associated with the rate of incremental fatigue crack growth increased significantly with patient age (Table 1). The primary mechanism of crack growth resistance in the young dentin was the development of unbroken ligaments that bridged the crack and reduced the available energy for cyclic extension (Figure 6(b)). This is a relatively well-documented mechanism of crack growth toughening that participates in fracture processes in many hard tissues [7, 48]. In dentin, the ligaments developed as a result of the formation of satellite or

“daughter” cracks in front of the primary crack tip, undoubtedly at intrinsic defects within the K-dominant region of the crack front, and potentially in localized regions of high lumen density that cause formation of a distributed stress concentration or flaw. The formation of unbroken ligaments was not identified in the old dentin samples, which is expected to result from the lower intrinsic fracture toughness of the material [34] and the movement of mineral into the lumens. Filling of the lumens reduces the tissue’s capacity for formation of ligaments due to the absence of the near-field stress concentration of open lumens, i.e. the primary origin of satellite cracks. Therefore, the significant increase in the crack growth exponent (m) can be attributed to the discontinuation of ligament bridging in old dentin. The corresponding increase in C with age for the 90° orientation could be related to the reduction in intrinsic toughness of the intertubular dentin, as inferred for the 0° orientation. Though speculative, there is some support for the proposed theory. Note that there was an increase in the crack growth coefficient of approximately 3 factors of magnitude over the age range spanned between the young and old age group. Results for the 0° orientation also showed the equivalent change in C over the age range, i.e. an increase by 3 factors of magnitude as well. Admittedly, further evaluation is required to increase the strength of that argument.

Results of this investigation have provided additional understanding of the effects from aging on the mechanical behavior of coronal dentin. These findings are of substantial importance to the field of restorative dentistry as they potentially identify the need for treating senior patients with an approach unique from that of younger patients. Foremost, it is essential to minimize the introduction of defects (i.e. damage or cracks) within dentin during the restoration of teeth in senior patients due to the reduction in crack growth resistance and greater potential for tooth fracture. One critical finding is that there for the 0° orientation, there is no significant difference between the fatigue crack growth behavior of young inner dentin and old dentin. Thus, inner coronal dentin behaves like old dentin and requires the same special care concern when cutting deep preparations and placing restorations. A recent study of coronal dentin showed that there is a significant reduction in tensile strength of dentin from the DEJ to inner dentin, with the ratio in strength between these two regions approaching a factor of two [44]. Though the ratio of stress intensity threshold values (Fig. 4a) for these two regions is lower (a factor of 1.6), the ratio of average apparent incremental fatigue crack growth rates exceeds a factor of 100! Therefore, flaws that serve as the stimulus for crack initiation are extremely detrimental within deep coronal dentin. There are spatial variations in the natural flaw distribution of coronal dentin that are related to the microstructure [49] and that are undoubtedly important in the probability of tooth fracture. But the most dominant flaws are expected to be those introduced during restorative dentistry [50]. Owing to the substantial reduction in fatigue crack growth resistance with depth, the potential for restoration failure attributed to fracture of the supporting hard tissue foundation is far more likely in teeth requiring deep restorations, regardless of patient age.

Although this investigation makes a fundamental contribution to understanding the detrimental effects of aging on the mechanical behavior of dentin, there are some recognized limitations. Due to the larger volume of tissue available from the tooth crown and the greater percentage of restorative processes that take place there, the evaluation was limited to coronal dentin. Vertical root fractures within endodontically restored teeth become more frequent with increasing patient age [51–53] and are more likely to result in extraction [54]. It is not known whether the aging process that takes place in the crown is different from that in the root. The root and crown exhibit different tubule density [55] and mineral/collagen ratios [17]. Thus, it may not be possible to apply the findings of the present study directly to radicular dentin. Future studies should explore aging of radicular dentin and potentially the aging process post-endodontic therapy as well. There are other factors that may contribute to the fatigue behavior of dentin with age as well, including frequency of loading [46] and

hydration [31] which were not examined in this study and could be considered. Due to the difficulties in obtaining sections of tissue with specific tubule orientation, the estimation of spatial variations in fatigue crack growth was limited to the 0° orientation. As such, the estimated degree of anisotropy for the inner, central and peripheral regions were developed using results for the central dentin in the 90° orientation. That could result in an overestimate of the gradient in the degree of anisotropy from the inner to the peripheral dentin. Also relevant to orientation, fatigue crack growth in-plane to the tubules was not conducted parallel to the tubule axis, which is the primary direction of crack growth active during tooth splitting. Yet, prior studies on the fracture toughness of dentin have reported that there is no significant difference in apparent growth resistance for cracks in-plane with the tubules and extending parallel or perpendicular to the tubule axis [21, 22].

While it was found that the differences in fatigue resistance between the adopted definitions of old and young dentin were significant, it remains unknown at what age the tissue should be considered “over the hill”. It would be desirable to identify at what age the greatest degradation in fatigue resistance takes place and identify the physiological factors responsible at this time period in life. Further studies are underway to address the aforementioned issues and to help support the development of improved approaches for treating the senior dentate in the pursuit of lifelong oral health.

CONCLUSIONS

An experimental evaluation of the fatigue crack growth resistance of human coronal dentin was performed. On the basis of the results obtained, the following conclusions may be drawn:

1. Aging results in a significant reduction in the resistance of coronal dentin to the initiation of fatigue crack growth, regardless of the tubule orientation. The stress intensity threshold (ΔK_{th}) of old dentin was approximately 25% lower than that of the young dentin for cracks extending in-plane or perpendicular to the dentin tubules.
2. For crack growth perpendicular to the tubules (i.e. 90° orientation), the old dentin exhibited significantly larger fatigue crack growth exponent and coefficient than for the young dentin. Although there are a variety of mechanisms that could contribute to the reduction in fatigue crack growth resistance, these changes appeared to predominately result from a marked reduction in the formation of unbroken ligaments during crack growth in old dentin.
3. For the 0° orientation there was no significant difference between the fatigue crack growth exponents of the young and old dentin, indicating that similar toughening mechanisms were active for both age groups. However, aging resulted in a significant increase in the fatigue crack growth coefficient (C). The increase in C may have resulted from the reduction in unfilled tubules available for crack tip blunting or changes in the resistance of the intertubular dentin to crack growth. Further studies are required to establish their individual contributions to the reduction in crack growth resistance.
4. The fatigue crack growth resistance of young dentin is anisotropic, which is attributed to the difference in fatigue crack growth mechanisms and relative influence of the microstructure. Dentin exhibits the lowest fatigue crack growth resistance for cracks oriented perpendicular to the dentin tubules. Although there are changes in the fatigue crack growth mechanisms with age that occurs with the evolution in microstructure, the fatigue crack growth resistance remains

anisotropic. The degree of anisotropy of young and old dentin was found to be equivalent.

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REFERENCES

1. Ten Cate, AR. Oral histology: Development, structure and function. Vol. Vol. 5. Mosby: St. Louis; 1998.
2. Garberoglio R, Brännström M. Scanning electron microscopic investigation of human dentinal tubules. *Arch Oral Biol*. 1976; 21(6):355–362. [PubMed: 1066114]
3. Pashley DH. Dentin: a dynamic substrate—a review. *Scanning Microsc*. 1989; 3(1):161–174. discussion 174–176. [PubMed: 2662395]
4. Marshall GW Jr, Marshall SJ, Kinney JH, Balooch M. The dentin substrate: structure and properties related to bonding. *J Dent*. 1997; 25:441–458. [PubMed: 9604576]
5. Kinney JH, Marshall SJ, Marshall GW. The mechanical properties of human dentin: a critical review and re-evaluation of the dental literature. *Crit Rev Oral Biol Med*. 2003; 14(1):13–29. [PubMed: 12764017]
6. Weiner S, Wagner HD. The material bone: structure–mechanical function relations. *Annu Rev Mater Sci*. 1998; 28:271–298.
7. Kruzic JJ, Ritchie RO. Fatigue of mineralized tissues: cortical bone and dentin. *J Mech Behav Biomed Mater*. 2008; 1(1):3–17. [PubMed: 19627767]
8. Rho JY, Kuhn-Spearing L, Zioupos P. Mechanical properties and the hierarchical structure of bone. *Med Eng Phys*. 1998; 20(2):92–102. [PubMed: 9679227]
9. Currey JD. Materials science. Hierarchies in biomineral structures. *Science*. 2005; 309(5732):253–254. [PubMed: 16002605]
10. Miyazaki M, Inage H, Onose H. Use of an ultrasonic device for the determination of elastic modulus of dentin. *J Oral Sci*. 2002; 44(1):19–26. [PubMed: 12058866]
11. Kinney JH, Balooch M, Marshall GW, Marshall SJ. A Micromechanics model of the elastic properties of human dentin. *Arch Oral Biol*. 1999; 44(10):813–822. [PubMed: 10530914]
12. Kinney JH, Gladden JR, Marshall GW, Marshall SJ, So JH, Maynard JD. Resonant ultrasound spectroscopy measurements of the elastic constants of human dentin. *J Biomech*. 2004; 37(4):437–441. [PubMed: 14996555]
13. Lertchirakarn V, Palamara JEA, Messer HH. Anisotropy of tensile strength of human dentin. *J Dent Res*. 2001; 80(2):453–456. [PubMed: 11332531]
14. Carvalho RM, Fernandes CA, Villanueva R, Wang L, Pashley DH. Tensile strength of human dentin as a function of tubule orientation and density. *J Adhes Dent*. 2001; 3(4):309–314. [PubMed: 11893046]
15. Inoue S, Pereira PN, Kawamoto C, Nakajima M, Koshiro K, Tagami J, Carvalho RM, Pashley DH, Sano H. Effect of depth and tubule direction on ultimate tensile strength of human coronal dentin. *Dent Mater J*. 2003; 22(1):39–47. [PubMed: 12790295]
16. Wang R. Anisotropic fracture in bovine root and coronal dentin. *Dent Mater*. 2005; 21(5):429–436. [PubMed: 15826699]
17. Arola D, Ivancik J, Majd H, Bajaj D, Zhang X, Fouad AF. On the microstructure and mechanical behavior of radicular and coronal dentin. *Int Endod J*. 2011 (accepted) Invited article.
18. Arola DD, Reprogel RK. Tubule orientation and the fatigue strength of human dentin. *Biomaterials*. 2006; 27(9):2131–2140. [PubMed: 16253323]
19. Arola DD, Rouland JA. The effects of tubule orientation on fatigue crack growth in dentin. *J Biomed Mater Res A*. 2003; 67(1):78–86. [PubMed: 14517864]
20. Rasmussen ST, Patchin RE, Scott DB, Heuer AH. Fracture properties of human enamel and dentin. *J Dent Res*. 1976; 55(1):154–164. [PubMed: 1060651]

21. Iwamoto N, Ruse ND. Fracture toughness of human dentin. *J Biomed Mater Res.* 2003; 66A(3): 507–512.
22. Nalla RK, Kinney JH, Ritchie RO. Effect of orientation on the in vitro fracture toughness of dentin: the role of toughening mechanisms. *Biomaterials.* 2003; 24(22):3955–3968. [PubMed: 12834591]
23. Riggs BL, Wahner HW, Seeman E, Offord KP, Dunn WL, Mazess RB, Johnson KA, Melton LJ 3rd. Changes in bone mineral density of the proximal femur and spine with 23 aging. Differences between the postmenopausal and senile osteoporosis syndromes. *J Clin Invest.* 1982; 70(4):716–723. [PubMed: 7119111]
24. Riggs BL, Wahner HW, Dunn WL, Mazess RB, Offord KP, Melton LJ 3rd. Differential changes in bone mineral density of the appendicular and axial skeleton with aging: relationship to spinal osteoporosis. *J Clin Invest.* 1981; 67(2):328–335. [PubMed: 7462421]
25. Heaney RP. Is the paradigm shifting? *Bone.* 2003; 33(4):457–465. [PubMed: 14555248]
26. Kinney JH, Nalla RK, Pople JA, Breunig TM, Ritchie RO. Age-related transparent root dentin: mineral concentration, crystallite size, and mechanical properties. *Biomaterials.* 2005; 26(16): 3363–3376. [PubMed: 15603832]
27. Arola, D. Fracture and Aging of Dentin. In: Curtis, R.; Watson, T., editors. *Dental Biomaterials: Imaging, Testing and Modeling.* Cambridge, UK: Woodhead Publishing; 2007. p. 314–340.
28. Zheng L, Nakajima M, Higashi T, Foxton RM, Tagami J. Hardness and Young's modulus of transparent dentin associated with aging and carious disease. *Dent Mater J.* 2005; 24(4):648–653. [PubMed: 16445030]
29. Senawongse P, Otsuki M, Tagami J, Mjör I. Age-related changes in hardness and modulus of elasticity of dentine. *Arch Oral Biol.* 2006; 51(6):457–463. [PubMed: 16426564]
30. Arola D, Reprogl RK. Effects of aging on the mechanical behavior of human dentin. *Biomaterials.* 2005; 26(18):4051–4061. [PubMed: 15626451]
31. Bajaj D, Sundaram N, Nazari A, Arola D. Age, dehydration and fatigue crack growth in dentin. *Biomaterials.* 2006; 27(11):2507–2517. [PubMed: 16338002]
32. Koester KJ, Ager JW 3rd, Ritchie RO. The effect of aging on crack-growth resistance and toughening mechanisms in human dentin. *Biomaterials.* 2008; 29(10):1318–1328. [PubMed: 18164757]
33. Koester KJ, Ager JW 3rd, Ritchie RO. Aging and Fracture of Human Cortical Bone and Tooth Dentin. *JOM.* 2008; 60(6):33–38. [PubMed: 20617106]
34. Nazari A, Bajaj D, Zhang D, Romberg E, Arola D. Aging and the reduction in fracture toughness of human dentin. *J Mech Behav Biomed Mater.* 2009; 2(5):550–559. [PubMed: 19627862]
35. Cohen S, Berman LH, Blanco L, Bakland L, Kim JS. A demographic analysis of vertical root fractures. *J Endod.* 2006; 32(12):1160–1163. [PubMed: 17174672]
36. Tamse A. Iatrogenic vertical root fractures in endodontically treated teeth. *Endodontics and Dental Traumatology.* 1988; 4(5):190–196.
37. Testori T, Badino M, Castagnola M. Vertical root fractures in endodontically treated teeth: a clinical survey of 36 cases. *Journal of Endodontics.* 1993; 19(2):87–91. [PubMed: 8509743]
38. Zhang D, Nazari A, Soappman M, Bajaj D, Arola D. Methods for examining the fatigue and fracture behavior of hard tissues. *Experimental Mechanics.* 2007; 47(3):325–336.
39. Arola D, Reid J, Cox ME, Bajaj D, Sundaram N, Romberg E. Transition behavior in fatigue of human dentin: structure and anisotropy. *Biomaterials.* 2007; 28(26):3867–3875. [PubMed: 17553559]
40. Bajaj D, Sundaram N, Arola D. An examination of fatigue striations in human dentin: in vitro and in vivo. *J Biomed Mater Res B Appl Biomater.* 2008; 85(1):149–159. [PubMed: 17853417]
41. Ivancik J, Neerchal NK, Romberg E, Arola D. The reduction in fatigue crack growth resistance of dentin with depth. *J Dent Res.* 2011; 90(8):1031–1036. [PubMed: 21628640]
42. Arola D, Zheng W, Sundaram N, Rouland JA. Stress ratio contributes to fatigue crack growth in dentin. *J Biomed Mater Res A.* 2005; 73:201–212. [PubMed: 15744763]
43. Paris PC, Gomez MP, Anderson WP. A rational analytic theory of fatigue. *Trend Eng.* 1961; 13:9–14.

44. Ryou H, Amin N, Ross A, Eidelman N, Wang DH, Romberg E, Arola D. Contributions of microstructure and chemical composition to the mechanical properties of dentin. *J Mater Sci Mater Med.* 2011; 22(5):1127–1135. [PubMed: 21455677]
45. Kahler B, Swain MV, Moule A. Fracture-toughening mechanisms responsible for differences in work to fracture of hydrated and dehydrated dentine. *J Biomech.* 2003; 36(2):229–237. [PubMed: 12547360]
46. Kruzic JJ, Nalla RK, Kinney JH, Ritchie RO. Mechanistic aspects of in vitro fatigue-crack growth in dentin. *Biomaterials.* 2005; 26:1195–1204. [PubMed: 15451639]
47. Porter AE, Nalla RK, Minor A, Jinschek JR, Kisielowski C, Radmilovic V, Kinney JH, Tomsia AP, Ritchie RO. A transmission electron microscopy study of mineralization in age-induced transparent dentin. *Biomaterials.* 2005; 26(36):7650–7660. [PubMed: 16005961]
48. Arola D, Bajaj D, Ivancik J, Majd H, Zhang D. Fatigue of biomaterials: hard tissues. *Int J Fatigue.* 2010; 32(9):1400–1412. [PubMed: 20563239]
49. Staninec M, Marshall GW, Hilton JF, Pashley DH, Gansky SA, Marshall SJ, Kinney JH. Ultimate tensile strength of dentin: Evidence for a damage mechanics approach to dentin failure. *J Biomed Mater Res.* 2002; 63:342–345. [PubMed: 12115767]
50. Staninec M, Meshkin N, Manesh SK, Ritchie RO, Fried D. Weakening of dentin from cracks resulting from laser irradiation. *Dent Mater.* 2009; 25(4):520–525. [PubMed: 19062088]
51. Testori T, Badino M, Castagnola M. Vertical root fractures in endodontically treated teeth: a clinical survey of 36 cases. *J Endod.* 1993; 19(2):87–91. [PubMed: 8509743]
52. Tamse A. Iatrogenic vertical root fractures in endodontically treated teeth. *Endod Dent Traumatol.* 1988; 4(5):190–196. [PubMed: 3073952]
53. Mireku AS, Romberg E, Fouad AF, Arola D. Vertical fracture of root filled teeth restored with posts: the effects of patient age and dentine thickness. *Int Endod J.* 2010; 43(3):218–225. [PubMed: 20158533]
54. Pitts DL, Natkin E. Diagnosis and treatment of vertical root fractures. *J Endod.* 1983; 9(8):338–346. [PubMed: 6579193]
55. Mjör IA, Nordahl I. The density and branching of dentinal tubules in human teeth. *Arch Oral Biol.* 1996; 41(5):401–412. [PubMed: 8809302]

a)

b)

Figure 1. Specimen preparation and tubule orientations. a) specimens prepared for the 90° (left) and the 0° tubule orientations, b) schematic diagram of the specimen geometry. All dimensions are in millimeters ($W=5.1\text{mm}$).

Figure 2. Typical fatigue crack growth responses for the two tubule orientations and distinction of the Paris Law parameters used in characterizing the cyclic crack growth behavior.

a)

b)

Figure 3.

Cumulative fatigue crack growth responses for the two tubule orientations and the differences in behavior between the two age groups. a) 0° tubule orientation. Note that the young dentin is divided into the responses of tissue as a function of relative location. The old samples are primarily central dentin, b) 90° tubule orientation

a)

b)

c)

Figure 4.

Comparison of the average fatigue crack growth parameters obtained from the experimental evaluation. The results are presented in terms of the mean (white) and standard deviation (gray). a) stress intensity threshold (ΔK_{th}), b) fatigue crack growth exponent (m), c) fatigue crack growth coefficient (C). Columns with different letters are significantly different. Note that the values for C are presented using the Log base(10) form to accommodate for the large differences in magnitude.

Figure 5.

A comparison of the fatigue crack growth responses for the old dentin representing the two different tubule orientations. Note that all old data are from the Central location of the coronal dentin.

Figure 6.

Mechanisms of cyclic crack extension in young coronal dentin. Each sequence of images shows the progression of cyclic crack growth in a selected sample over a period of cyclic loading. a) 0° orientation. Note the locations highlighted with black arrows (top) designate where crack branching occurred as a result of the crack changing paths to follow a segment with higher tubule density. The sequence of photos (bottom) designated i through iii present snapshots of the crack path documented over a series of measurement increments where the crack tip is identified by the open white arrows; the number of cycles between the crack extension increments from i–ii is 6780 cycles and from ii–iii is 3323 cycles, respectively. b) 90° orientation. Black arrows (top) highlight unbroken ligaments that have formed as a

result of the variation in the crack plane. Similar to (a), the sequence of photos (bottom) present snapshots of the crack path documented over a series of measurement increments where the crack tip is identified by the open white arrows. The number of cycles between the crack extension increments from i-ii is 20,000 cycles and from ii-iii is 11,620 cycles, respectively.

a)

b)

Figure 7.

Scanning electron micrographs of the fracture surfaces resulting from fatigue crack growth. Each sets of micrographs compares representative surfaces for young (left) and old (right) dentin. The crack path was from left to right in each of these figures. a) 0° orientation with samples from young (male 28 yo) and old (male 72 yo) patients. Note the regions where the crack grew around the lumens rather than through as highlighted by the asterisks.; b) 90° orientation with samples from young (male 22 yo) and old (male 58 yo) patients. Note the combination of open lumens (black arrows) and closed lumens (white arrows) in this cross-section.

Table 1

Results from the One-way ANOVAs for the fatigue crack growth parameters. Note that all old data are from the Central location and Y and O refer to the Young and Old groups, respectively.

Orientation	Age	Parameter*	0° (n=11)	90° (n=32)	F	p
0°	Y	ΔK_{th}	1.03±0.1	0.83±0.1	35.80	0.0005
		m	25.47±3.0	14.15±1.6	284.32	0.0005
		C	4.79×10 ⁻⁸	2.69×10 ⁻⁵	138.14	0.0005
	O		0° (n=10)	90° (n=15)		
		ΔK_{th}	0.77±0.1	0.60±0.1	17.26	0.0005
		m	23.11±5.1	24.16±4.3	0.31	0.585
90°	Young (n=32)	C	6.61×10 ⁻⁵	1.58×10 ⁻²	17.21	0.0005
			Young (n=11)	Old (n=10)		
		ΔK_{th}	1.03±0.1	0.77±0.1	50.12	0.0005
	Old (n=15)	m	25.47±3.0	23.11±5.1	2.06	0.21
		C	4.79×10 ⁻⁸	6.61×10 ⁻⁵	31.31	0.0005
			Young (n=32)	Old (n=15)		
90°	Young (n=32)	ΔK_{th}	0.83±0.1	0.60±0.1	49.54	0.0005
		m	14.15±1.6	24.16±4.3	136.49	0.0005
		C	2.69×10 ⁻⁵	1.58×10 ⁻²	114.04	0.0005

* Units for ΔK_{th} and C are MPa•m^{0.5} and (mm/cycle)(MPa•m^{0.5})^{-m}, respectively.