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The effect of aging on crack-growth resistance and toughening mechanisms in human dentin

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Abstract

Crack-growth experiments in human dentin have been performed *in situ* in an environmental scanning electron microscope to measure, for the first time, the crack-growth resistance curve (*R*-curve) for clinically relevant (<250 μm) crack extensions and to simultaneously identify the salient toughening mechanisms. ''Young'' dentin from donors 19–30 years in age and ''aged'' dentin from donors 40–70 years in age were evaluated. The ''young'' group had 0–4% of its tubules filled with apatite; the ''aged'' group was subdivided into ''opaque'' with 12–32% filled tubules and ''transparent'' with 65–100% filled tubules. Although crack-initiation toughnesses were similar, the crack-growth resistance of ''young'' dentin was higher by about 40% compared to ''aged'' dentin. Mechanistically, this behavior is interpreted in terms of three phenomena: (i) gross crack deflection of the growing crack, (ii) microcracks which initiated at unfilled tubules in the high stress region in the vicinity of a propagating crack (no microcracks formed at filled tubules), and (iii) crack propagation which followed a *local* trajectory *through* unfilled tubules and *deflected around* filled tubules. The higher toughness of the ''young'' dentin was related to enhanced microcracking (at unfilled tubules) ahead of the growing crack, which (i) shields the crack by activating multiple crack tips and by reducing the local stress intensity through crack deflection and (ii) leads to the formation of crack bridges from ''uncracked ligaments'' due to the incomplete coalescence of these microcracks with the main crack tip. With age, the role of these toughening mechanisms was diminished primarily to the lower fraction of unfilled, and hence microcracked, tubules.

Keywords: Human dentin; Aging; Fracture mechanics; Resistance curves; Toughness; *In situ* electron microscopy

1. Introduction

Dentin is the mineralized tissue that comprises the bulk of the human tooth and therefore determines its structural integrity. It is a hydrated composite of mineralized collagen fibers and nanocrystalline hydroxyapatite, with ~45% hydroxyapatite, 35% collagen and 20% water by volume. The mineralized collagen fibrils form the intertubular dentin matrix and are arranged in a felt-like structure oriented perpendicular to a series of channels known as the tubules. These tubules are 1–2 μm in diameter, and extend from the pulp cavity to the exterior of the tooth; they are lined with a highly mineralized cuff of peritubular dentin [1,2]. During aging, human dentin sclerosis causes the tubules to become occluded through deposition of carbonated apatite [3,4] leading to transparency to visual light of the dentin and a change in the mechanical properties, most notably a loss in ductility, toughness and cyclic fatigue resistance [4–8].

Dentin has the desirable quality of increasing toughness with crack extension, i.e., it displays resistance-curve

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toughening behavior.[1] Fracture resistance studies of dentin have identified several toughening mechanisms, including constrained microcracking [10,11], crack bridging [10,11], crack blunting [6,12], and associated viscoelastic flow [13,14]. The most effective toughening mechanism, crack bridging, results from the formation of microcracks at the tubules ahead of a growing crack; until these microcracks are able to join the main crack tip, they leave regions (which are micrometers to tens of micrometers in size) of uncracked material (termed ''uncracked ligaments'' as described in Refs. [9,15,16][2]) spanning (i.e., bridging) the crack, which carry load that would be otherwise used to further propagate the main crack.

The majority of these mechanistic studies, however, have been conducted on elephant and bovine dentin, rather than human dentin, and although the microstructures of these mineralized tissues are similar, the size-scales over which cracks can propagate are much smaller in human dentin. This is important as dentin, like cortical bone [17], primarily derives its fracture toughness *extrinsically* during crack growth, and thus when cracks are small compared to characteristic microstructural dimensions, the full effect of this toughening is not felt.

Although the strength, fracture energy, and fatigue resistance of human dentin have all been examined, to date there have been no measurements of the toughness of human dentin in terms of the full crack-resistance (*R*-curve) behavior. Studies on mechanical properties in general, however, have shown that they vary with the orientation of the tubules; specifically, the strength and fracture energy are lowest when the long axis of the tubules is parallel to the crack front and highest when the long axis of the tubules is perpendicular to the crack front [18,19]. Moreover, dentin displays a clear deterioration in strength, toughness and fatigue resistance with age [7,18].

In light of this, in the current study we employed *in situ* mechanical testing inside a scanning electron microscope to determine, for the first time, the fracture toughness crack-resistance curve behavior for human dentin over physiologically relevant crack extensions ($\Delta a < 250\ \mu m$), while simultaneously imaging in real time the salient toughening mechanisms, explicitly in terms of how the crack path interacts with the dentin microstructure. We examine the effect of aging on dentin and find that mineral deposition within the tubules leads to changes in the crack path and a consequent degradation in toughness over size-scales relevant to the behavior of human teeth.

2. Materials and methods

Human molars ($N = 7$), extracted according to protocols approved by the University of California San Francisco, Committee on Human Research, were used in this study. Rectangular-beam bend samples (4 mm long, 1 mm wide, 0.5 mm thick), two or three per tooth, were wet sectioned from the central portion of the crown and root (Fig. 1) using a low-speed saw, and stored in Hanks' Balanced Salt Solution (HBSS) at 25 °C. The molars were divided into three groups, as determined by the fraction of the occluded tubules (Table 1):

- ''young'' dentin (19–30 years old) with 3–7% filled tubules ($N = 4$);
- ''aged/opaque'' dentin (40–70 years old) with 12–32% filled tubules ($N = 5$); and
- ''aged/transparent dentin'' (40–70 years old) with 65–100% filled tubules such that they are transparent to visible light ($N = 5$).

The fraction of filled tubules was determined by obtaining scanning electron microscopy images for four samples from each group.

The bend specimens were notched to roughly one-half of their width, and the notch was then sharpened to a radius of ~10 μm with a micro-notching technique using a razor blade irrigated with 1 μm diamond suspension. The orientation of the notch was such that the intended direction of crack propagation was perpendicular to the long axis of the tubules. It should be noted, however, that it is never possible to orient samples precisely with respect to the direction of the tubules; in addition, the tubules may not run straight through the beam sample. For these reasons, the direction of crack propagation can only be specified approximately. All of the samples were wet polished using increasingly fine grits to a final polish of 0.05 μm diamond suspension and were subsequently immersed in HBSS for 18–24 h before testing. *In situ* mechanical testing to determine the toughness behavior was performed for stable crack extensions less than ~250 μm in a Hitachi S-4300SE/N environmental scanning electron microscope (ESEM) (Hitachi America, Pleasanton, CA) using a Gatan Microtest 2 kN mechanical three-point bending stage (Gatan UK, Abingdon UK); images of the crack path were obtained simultaneously at 15 kV using backscattering mode at a pressure of 35 Pa and a temperature of 25 °C.

Fracture toughness *R*-curves were determined in terms of the crack-driving force (the stress intensity K)[3] as a function of crack extension (Δa). As growing cracks often follow a trajectory dictated by the local microstructure and any crack deflection can enhance the toughness, where the cracks grew at an angle to plane of maximum tensile stress, deflected crack solutions [20] were used to calculate the stress intensity. Specifically, crack deflection induces local mixed-mode loading conditions at the crack tip, *e.g.*, mode I (tensile opening) plus mode II (shear) for in-plane deflections,[4] such that standard mode I stress-intensity solutions become inapplicable. However, the cracks grew away from the notch at a roughly constant angle such that standard crack-deflection solutions could be used to compute a mixed-mode

[1] Crack propagation can be considered as a mutual competition between two classes of mechanisms: *intrinsic* mechanisms, which are microstructural damage mechanisms that operate ahead of the crack tip, and *extrinsic* mechanisms, which act principally in the wake of the crack tip to ''shield'' the crack from the applied driving force [9]. In dentin, both constrained microcracking and crack bridging are examples of extrinsic toughening mechanisms as they act behind the crack tip to reduce the local stress intensity experienced at the crack tip. As these mechanisms are primarily active *during crack growth*, a natural consequence of their presence is resistance-curve (*R*-curve) toughening behavior, where the resistance to cracking increases with crack extension.

[2] Uncracked-ligament bridging refers to the process where cracks initiated ahead of a growing crack leave uncracked regions, which can act as bridges, between them and the main crack tip before they link up. This toughening mechanism is well documented in ceramics, composites and rocks and has been identified as a major toughening mechanism in dentin and bone [9,15,16]. The word ''ligament'' here is not used in the anatomical sense.

[3] The stress intensity K represents a field parameter which characterizes the strength of the stress and displacement field at a crack tip; it is generally defined as $K = Q\sigma_{app}(\pi a)^{1/2}$, where σ_{app} is the applied stress, a is the crack length, and Q is a function (of order unity) of crack size and geometry. The critical value of K for unstable fracture at a pre-existing crack is referred to as the fracture toughness, K_c. Alternatively, the toughness can be expressed as a critical value of the strain energy release rate, G_c, defined as the change in potential energy per unit increase in crack area, i.e., when $G_c = K_c^2/E'$, where E' is the appropriate elastic modulus.

[4] Where cracks additionally deflect through the thickness of the sample, mode III (anti-plane shear) displacements also are possible.

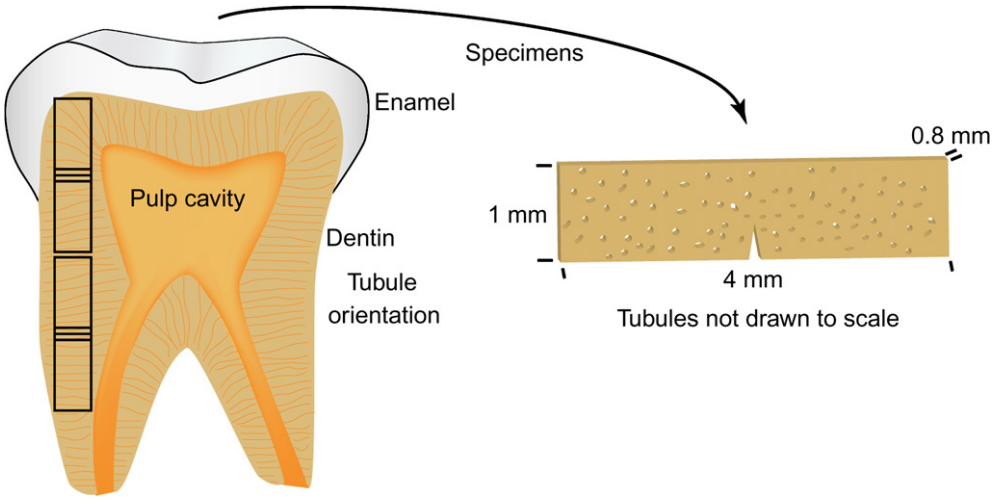

Fig. 1. Schematic diagram of a human tooth and the location from which the three-point bend specimens were cut from each tooth. The diagram shows the orientation of the notch and the tubules. The samples were fabricated so that the long axis of the tubules was approximately parallel to the crack front. Note that for clarity, the size of the tubules is not to scale, relative to the specimen size.

driving force. Assuming primarily an in-plane deflection of the crack path through an angle α from the plane of the notch, the *local* mode I and II linear-elastic stress intensities at the deflected crack tip, k_1 and k_2, respectively, are given by [10,20]:

$$k_1 = c_{11}(\alpha)K_I + c_{12}(\alpha)K_{II},$$

$$k_2 = c_{21}(\alpha)K_I + c_{22}(\alpha)K_{II}, \quad (1)$$

where $c_{ij}(\alpha)$ are mathematical functions of the deflection angle [20] and K_{I} and K_{II} are the applied (*global*) mode I and II stress intensities, respectively. For the present three-point bending configuration, $K_{\mathrm{II}} = 0$ and K_{I} is given by handbook solutions [21]:

$$K_I = \frac{PS}{BW^{3/2}} f\left(\frac{a}{W}\right), \quad (2)$$

where P is the applied load, S is the loading span length, W is the sample width, B is the sample thickness, a is the crack length obtained from backscattered images, and $f(a/W)$ is the geometry function tabulated in Ref. [21]. To compute a mixed-mode driving force, k_1 and k_2 can be summed as strain energy release rates ($G = k^2/E'$), the mixed-mode G is then reconverted back to an effective stress intensity, K_{eff}, for the deflected crack [10], *viz.*:

$$K_{\mathrm{eff}} = \left(k_1^2 + k_2^2\right)^{1/2}. \quad (3)$$

Where crack paths remained relatively linear and did not undergo large deflections, i.e., they extended along an expected nominal path of the maximum tensile stress (i.e., where $K_{\mathrm{II}} = 0$), the crack-deflection angle is zero and the deflected crack solution reduces to the standard handbook linear-elastic mode I solution for cracks in the single-edge notched three-point bending geometry [21].

Differences of the means of the measured parameters (crack-growth toughness, unfilled and microcracked tubule fractions) between the groups were tested with one-way ANOVA and, when appropriate, one-tailed t tests were used to make comparisons between the groups. Differences between the groups were considered to be significant at the $p < 0.05$ level.

Table 1
Tubule density and the filled tubule fraction (mean ± standard deviation) for the three dentin groups

	Tubules/ 10,000 μm^2	Filled tubule fraction
Young	129 ± 60	0.04 ± 0.03
Aged/opaque	106 ± 29	0.20 ± 0.08
Aged/transparent	128 ± 48	0.87 ± 0.19

3. Results

R-curves for human dentin, computed from measured load-displacement data for stably growing cracks (up to $\Delta a \sim 250$ μm), are shown in Fig. 2. The crack-initiation toughness, given by intercept on the ordinate axis at $\Delta a \rightarrow 0$, is of the order of 1 MPa$\sqrt{\mathrm{m}}$ and appears to be similar for all groups (it was not possible to obtain an accurate extrapolation of the data for each sample to $\Delta a = 0$, so further analysis of the initiation toughness differences was not undertaken). It

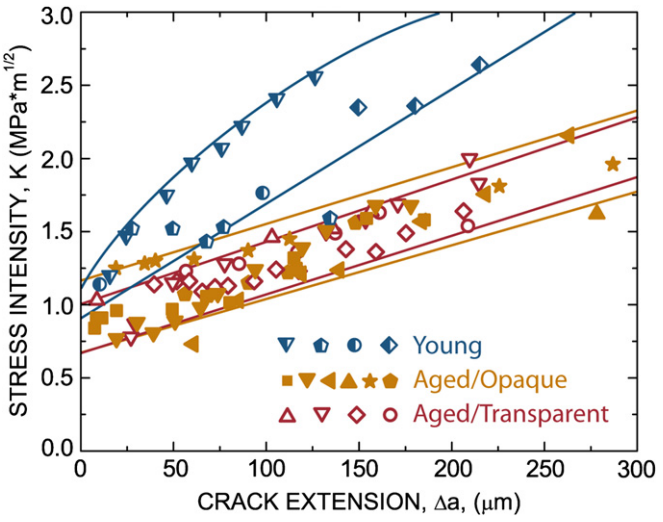

Fig. 2. Fracture toughness *R*-curves for moist "young", "aged/opaque", and "aged/transparent" human dentin. The sample groups have different fractions of occluded tubules (Table 1). The crack-growth toughness is degraded by aging, an effect that we attribute to lessened contribution from microcracking, crack deflection and crack bridging. The lines are guides to the eye indicating the range of data for each data set.

is clear, however, that the toughness of the ''young'' samples is higher than that of the ''aged'' samples after ~100 μm or more of crack extension. This increase in toughness was quantified by the crack-growth toughness which we define here as the slope (least-squares fit) of the resistance curve. Growth toughness results are shown in Fig. 3a. Young dentin had a significantly higher ($p < 0.05$) growth toughness than aged/opaque and aged/transparent dentin. For the aged dentin, the opaque group also had a significantly higher ($p < 0.05$) growth toughness than the older transparent group.

The decreased growth toughness with age is paralleled by a similar decrease in the fraction of unfilled and microcracked tubules involved in the crack path (Fig. 3), as discussed below. Unfilled tubule and microcracked tubule fractions were calculated by counting the number of filled tubules, unfilled tubules, and microcracked tubules that were intersected by the propagating crack in the ESEM images of crack propagation and normalizing by the total number of tubules. Typically there were 7–15 tubules in the ~200 μm of crack wake that was examined. The results are shown in Fig. 3b. We note that not all of the samples used to generate the crack-growth resistance curves were suitable for examining the growth toughness and tubule fractions in the crack profile; typically 2–5 samples per group were employed to generate these data.

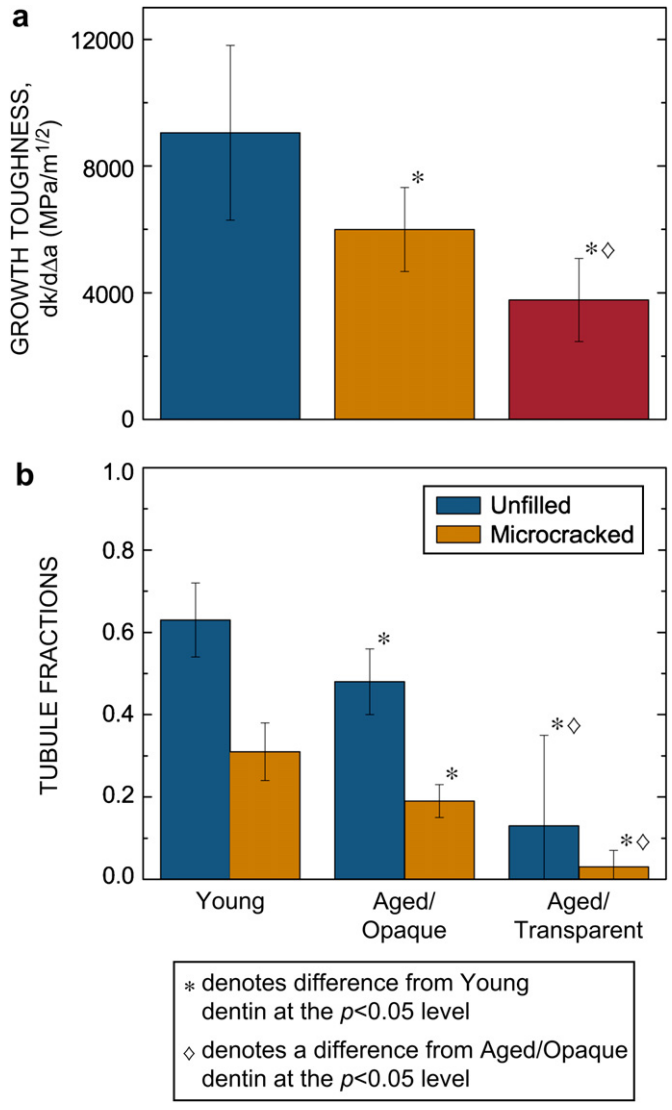

Fig. 3. Plots of the crack-growth toughness and tubule fractions along the crack paths. The crack-growth toughness, defined as slope of the resistance curve, can be seen to decrease as the fraction of unfilled tubules decreases.

It should be noted here that since the tubule density and diameter can vary both from person to person and with location within the tooth, these factors could be confounding variables for these experiments. However, examination of the tubule density for each sample in each of the normal, aged/opaque and aged/transparent groups revealed that the average tubule density ranged from 10,530 to 12,879 tubules/mm^2 for the different groups; i.e., it did not differ by more than 20% from group to group. Similarly, the tubule size was found to be consistent between the different groups, with an average tubule diameter ranging from 1.9 to 2.2 μm, i.e., varying by less than 15% from group to group.

ESEM images of typical fracture surfaces, shown in Fig. 4, clearly show the presence of the tubules in the crack path. There were no noticeable differences in fracture morphology, and by inference fracture mechanisms, between the surface and interior of the dentin specimens; moreover, the crack fronts were essentially straight. This suggests that our *in situ* SEM observations of fracture behavior are likely to be representative of the bulk.

One problem with solely examining fracture surface morphology is that it does not readily reveal how the crack actually interacts with the salient microstructural features. For this reason, most of our analyses are based on sections through propagating cracks, obtained from the *in situ* ESEM images; these are presented in Figs. 5–8.[5] As discussed in Section 4, several toughening mechanisms can be identified; these include gross crack deflection, uncracked-ligament bridging, crack branching, and local crack deflection. The nature of these mechanisms is discussed in the following section.

4. Discussion

In this work, we have shown that the aging-related sclerosis in human dentin, which causes the filling of dentinal tubules with carbonated apatite, results in a deterioration of the fracture toughness of the dentin. However, there is only a negligible effect on the crack-initiation toughness, whereas the toughness associated with growing cracks, i.e., the crack-resistance curve, is significantly degraded.

The variations in toughness can be related to changes in the crack path, specifically how the crack interacts with the local microstructure in dentin. Examination of *in situ* SEM images associated with this age-related deterioration in toughness revealed important differences in the way the propagating crack interacted with the tubules, which are predominantly

[5] Some of these images do not include the starter notch and show only the propagating crack. Any ''misalignment'' of the micronotches from the mode I direction is small and is visible only because of the high resolution associated with *in situ* testing in the ESEM.

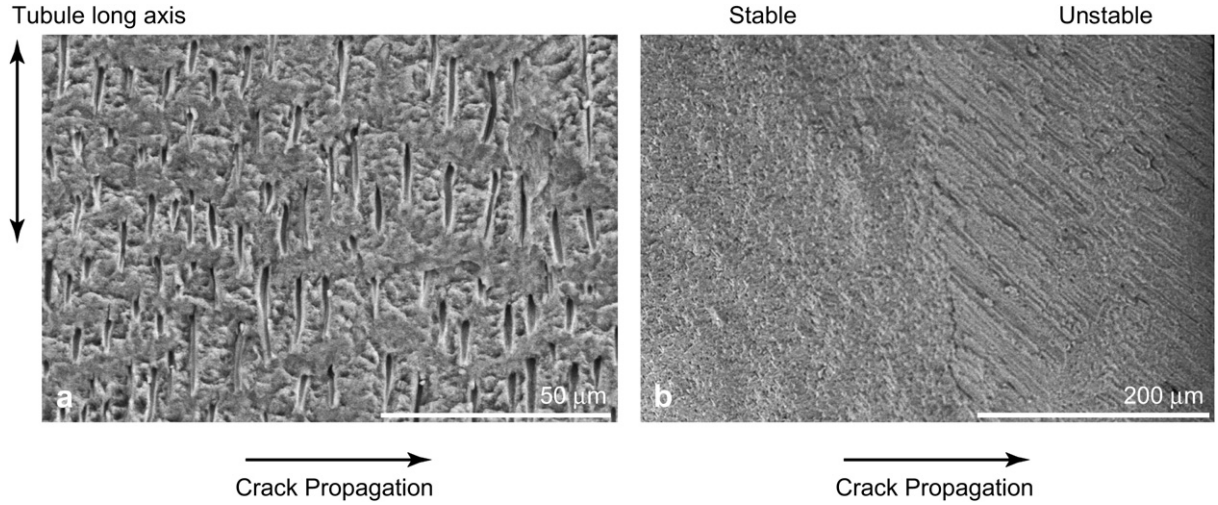

Fig. 4. SEM images of the fracture surfaces in aged/opaque dentin. Image (a) shows the orientation of the tubules was such that the advancing crack front was approximately parallel to the long axis of the tubules. The transition from stable crack growth to unstable crack growth is shown in (b). It was found through fractography that the crack fronts were uniform throughout the thickness of the sample; moreover, the nature of the fracture surface morphology was essentially identical. This suggests that crack-path profiles taken using *in situ* SEM on the surface are likely to be representative of fracture behavior throughout the thickness of the sample.

unfilled in the young group and increasingly filled with apatite in the opaque and transparent groups. Fig. 5 shows representative crack paths in young dentin; here the majority of the tubules are unfilled and the crack path tends to preferentially follow these tubules as it would in any low-modulus phase (the unfilled tubules can be thought of as a phase with a modulus of zero). We note that in the high stress region ahead of a growing crack, these unfilled tubules tend to initiate microcracks (Fig. 5b) prior to the main crack actually ''penetrating'' the tubule, as in Fig. 5c. Also, initiation of new crack tips can occur simultaneously at multiple sites around an unfilled tubule resulting in crack branching and multiple crack tips (Fig. 5e). The proportions of unfilled and microcracked tubules in the crack wake for all three groups are summarized in Fig. 3. It was observed that young dentin had both significantly more unfilled tubules and also more microcracked tubules in the crack wake, as compared to the other groups ($p < 0.05$). While the former is not surprising (young dentin has more unfilled tubules to begin with), the latter suggests a relationship between apatite filling and microcracking.

Fig. 6 shows that the same mechanisms can occur in aged/opaque dentin, but less frequently due to the lower proportion of empty tubules. The unfilled tubules are intimately linked with many of the toughening mechanisms seen in young dentin. Because the filled tubules do not microcrack, they do not result in crack branching. This is not to say that branching cannot occur in the aged dentin, but it is diminished as there are far fewer empty tubules at which branching can occur. The cracks in aged dentin still follow the empty tubules. However, since there are fewer unfilled tubules, this does not dominate the global direction of crack growth to the same extent as in young dentin. The filled tubules in aged dentin result in local deflections as the crack propagates around their interface with the matrix and does not penetrate the tubules.

Fig. 7 shows the interaction of a crack tip with a filled tubule in transparent dentin. The crack did not penetrate the filled tubule but instead followed a path around the hypermineralized peritubular cuff (Fig. 7b), which may be due to the difference in mechanical properties between peritubular and intertubular dentin, before arresting [18]; the crack then propagated from a new crack initiated from a different location on the cuff. Examination of many such images led to the following observation. If the crack only grazed the edge of a filled tubule, it did not penetrate it. If a crack was incident on a filled tubule in such a manner that it would have to either penetrate the tubule or undergo a deflection through a large angle, it would propagate along the boundary between the matrix and the peritubular cuff. However, as the crack grows around the tubule along this interface it must deflect again in order to continue to propagate along the interface. This additional deflection of the crack can be less favorable than the initiation of a new crack on the surface of the filled tubule. This sequence of events results in a short-term bridging of the crack tip. After initiating at a new site, the crack continues to open, eventually leading to the destruction of the uncracked region, i.e., the filled tubule, in the crack wake. However, due to the specific set of circumstances required for this phenomenon to happen, the penetration of filled tubules by cracks was not commonly observed.[6]

[6] It is interesting to note that this scenario is also seen in many brittle materials toughened by grain bridging. In situations where a crack is growing intergranularly in process of debonding a grain, it similarly may need to back-propagate towards the open crack mouth in order to follow the grain boundary. This is an extreme form of crack deflection known as the ''stall'' condition. As it requires a considerable increase in crack-driving force to continue propagation under these circumstances, often a crack will reinitiate elsewhere, generally ahead of the original crack tip, leading to the creation of uncracked-ligament bridges. Since this mechanism can occur over size-scales on the order of a single grain size, it is believed that it is primarily responsible for the toughening associated with the rapid initial increase in the *R*-curve seen in many brittle materials, *e.g.*, monolithic structural ceramics [22].

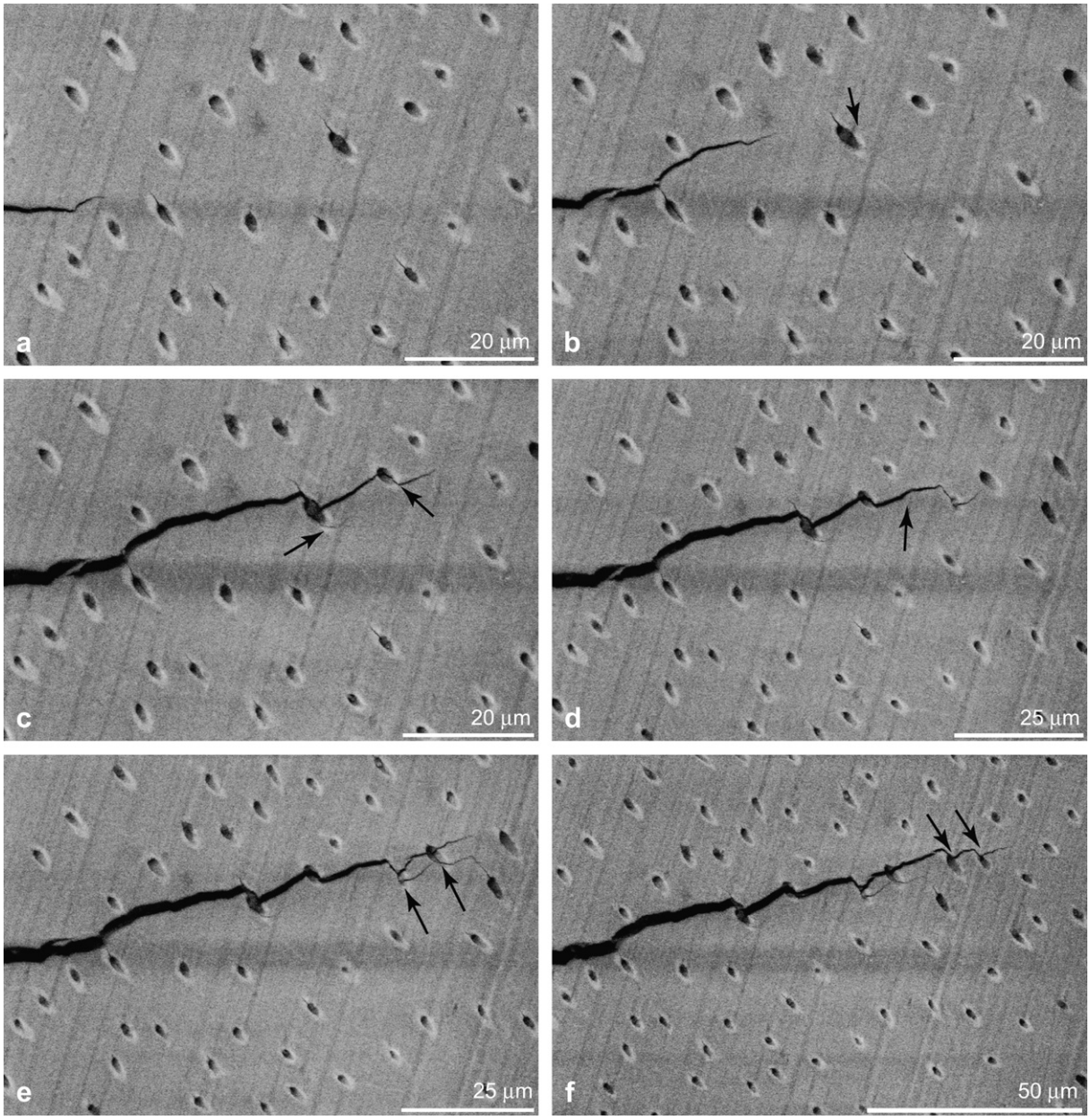

Fig. 5. Series of ESEM images of a growing crack in moist young dentin demonstrating how cracked/unfilled and uncracked/unfilled tubules interact with a growing crack. Image (a) shows the initial crack prior to loading and the crack length increases sequentially to (f). The propagating crack extends through the microcracks to form deflections and branching in the crack wake. The arrows in the images indicate regions of interest where microcracking, branching, and the growth of microcracks occur during crack propagation.

An important difference between the normal and aged groups was the prevalence of crack deflection. As shown in Fig. 8a, b, crack propagation tended to be straighter in young dentin as compared to aged/transparent dentin. Crack bridging was found in both groups, but the mechanism was different. Comparison of Fig. 8c and d indicates the difference in bridge formation in young *vs*. aged dentin, the distinction between the two being that in young dentin, the bridges form between the tubules and tend to be larger than that in the aged dentin where the occluded tubule itself forms the bridge. Also, although crack branching was found both in young and aged dentin, it only occurred at the sites of unfilled tubules. As the fraction of unfilled tubules is much higher in young dentin (Table 1, Fig. 3), toughening from crack branching is more prominent in this condition. The mechanisms associated with such crack-growth toughness in dentin are shown schematically in Fig. 9; Table 2 summarizes our observations of these toughening mechanisms for short crack extensions in human dentin.

In many respects, the prominence of crack-growth toughening is to be expected as dentin, like other mineralized tissues such as cortical bone [23], derives its toughness principally from extrinsic mechanisms during crack growth, most specifically from crack bridging and to a lesser extent from microcracking and crack deflection [10,16]. Toughening

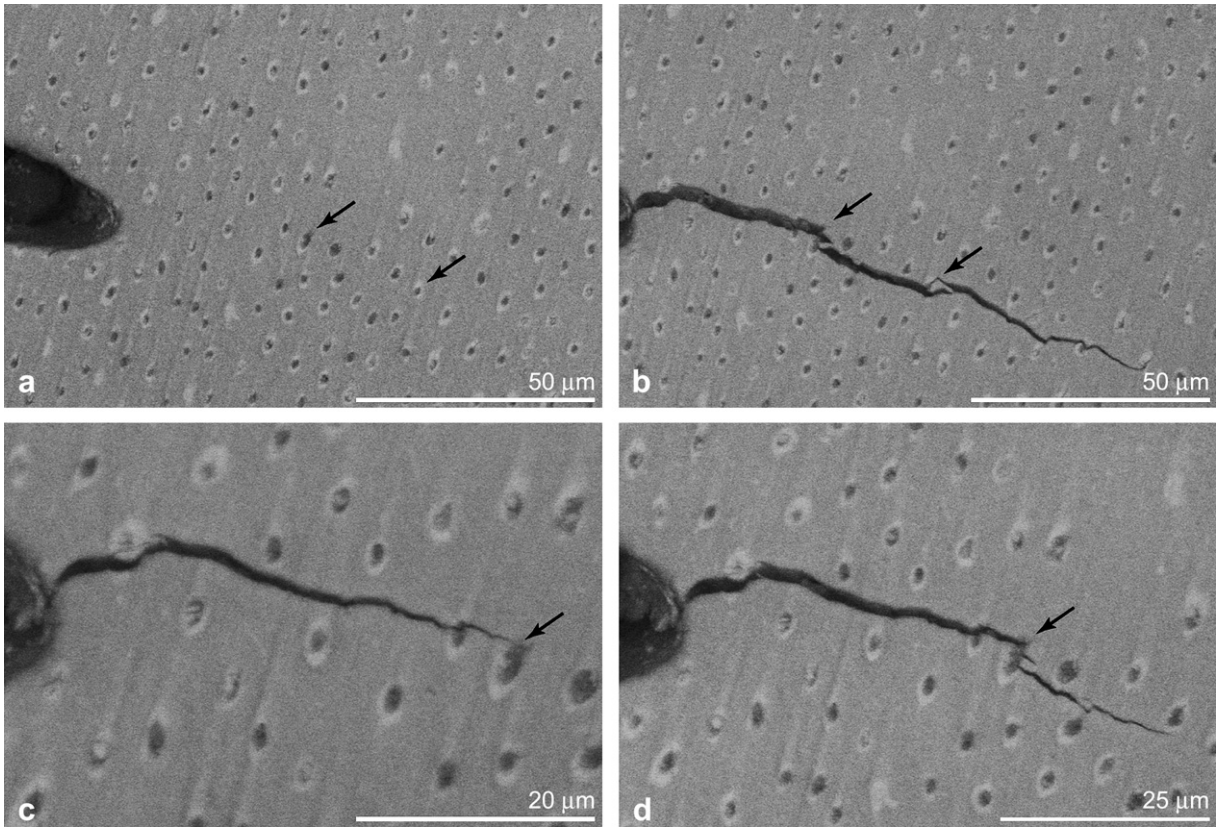

Fig. 6. ESEM images of crack propagation in moist aged/opaque dentin showing the sequence of events that occur when a growing crack propagates through unfilled and uncracked tubules in human dentin. Image (a) is of the area of interest prior to loading and (b) is of the same region after crack extension. Images (c) and (d) are of the same sample showing the intermediate crack extension between images (a) and (b) at high magnification. The images show the penetration of a tubule by the crack and subsequent microcracking and bridge formation that result, respectively. The arrows indicate the unfilled tubules of interest that microcrack and result in bridge formation and branching.

mechanisms such as bridging and deflection depend critically on the nature of the crack path with respect to the microstructure, and the ''embrittlement'' of dentin with age can be traced to this effect. Indeed, it is clear that the crack path, and hence the (crack-growth) toughness, are controlled by how the primary crack interacts with the tubules (the principal microstructure feature) and whether these tubules are unfilled, microcracked or filled. We had argued in our earlier work on elephant dentin [12,23] that the age-related degradation in toughness from occluded tubules could be explained by the fact that filled tubules would be less likely to microcrack (due to the absence of the stress concentration). In young dentin, the linkage of the much higher fraction of microcracks to the main crack tip can result in regions of uncracked material which can act as crack-bridging ligaments (so-called uncracked-ligament bridging) [15]; less microcracking in aged dentin would mean less post-yield ductility and reduced uncracked-ligament bridge formation, both of which we reported for transparent elephant dentin. However, stable crack growth (and hence *R*-curve behavior) extend over far larger dimensions in elephant dentin, typically as much as 5–6 mm, which maximizes the effect of such extrinsic toughening; this is to be contrasted with human dentin where realistic crack extensions are over an order of magnitude smaller and are measured more in the hundreds of micrometers, as shown in the present study.

Over the smaller size-scales associated with human dentin, the influence of aging and transparency on the toughness is clearly similar to elephant and bovine dentin although somewhat more complicated. To be certain, the filling of the tubules with age does cause a reduction in the fracture resistance which can be associated, at least in part, with a reduced role of crack bridging. Microcracked tubules located ahead of a growing crack do result in the formation of uncracked-ligament bridges by becoming active before the crack actually penetrates the tubule; however, unlike the larger-crack behavior seen with bovine and elephant dentin [10,11], these events are not particularly common. The presence of large uncracked ligaments, tens of micrometers in size, which can be seen spanning cracks in these other types of dentin [10,11], are simply not observed in human dentin for the studied tubule orientations.

It is clear that the origins of (crack-growth) toughness in human dentin, and its degradation with age, are associated with additional mechanisms. Arola and Reprogel [6] suggest that the empty tubules in human dentin enhance fatigue resistance by blunting the crack tip; i.e., when a propagating crack penetrates a tubule, its crack-tip radius is increased from near atomistic dimensions to the radius of the tubule, thereby markedly reducing the crack-tip stress field. Transparency would thus degrade the toughness as such blunting would

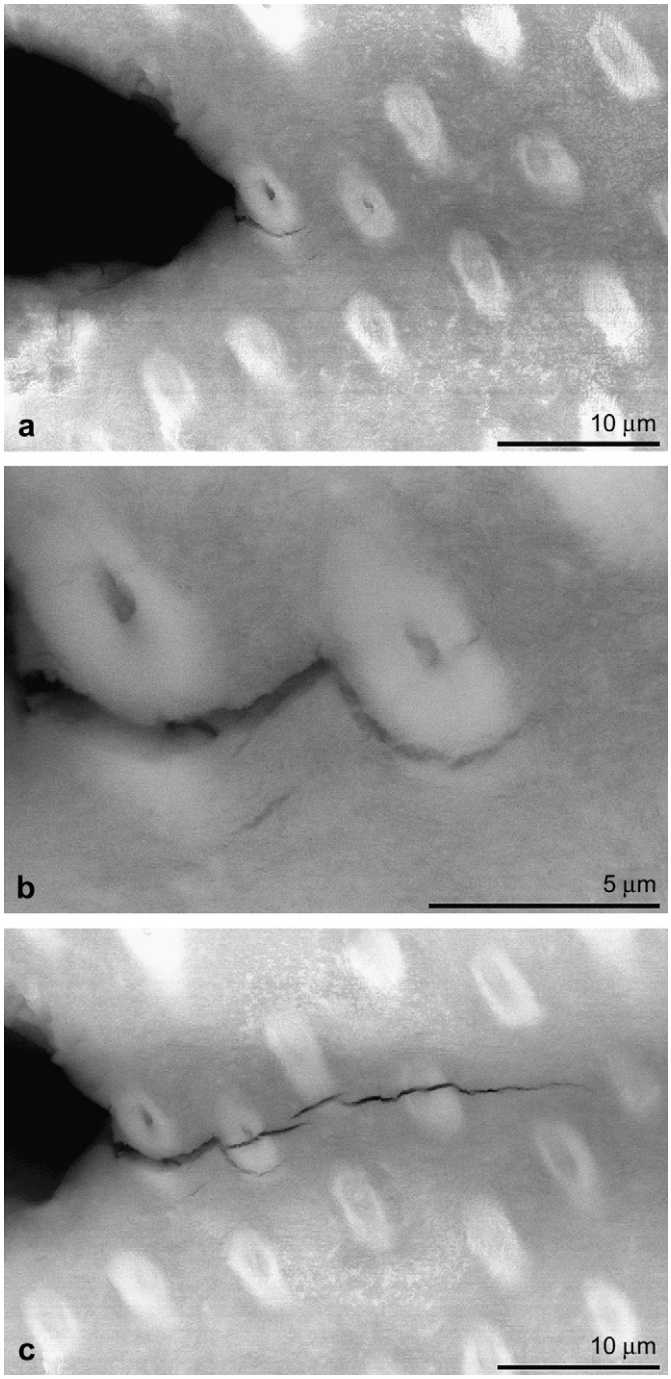

Fig. 7. Sequence of ESEM images of a crack intersecting with a filled tubule in transparent dentin. Initially as the crack approaches the tubule, it deflects and propagates around the hyper-mineralized peritubular cuff (a, b). At some point the crack ceases to follow the interface between the peritubular cuff and the matrix a new crack is initiated elsewhere on the tubule. This newly initiated crack then becomes the main crack that advances.

not occur with filled tubules. Whereas this mechanism may be effective at the lower crack-driving forces associated with fatigue, our observations that the unfilled tubules in the vicinity of the growing crack are microcracked (Figs. 4 and 5) precludes the notion of crack-tip blunting being responsible for crack-growth resistance in dentin.

In light of this and the diminished role of crack bridging (as compared to larger-crack behavior in elephant and bovine dentin), the current work demonstrates that in human dentin the toughening, and its age-related degradation, can be related more to crack deflection and crack branching. The deflection of the crack to follow the microcracked and empty tubules in dentin, away from the expected $K_{II} = 0$ crack path of maximum tensile stress, represents an extrinsic toughening mechanism as the crack will experience a reduced driving force, as compared to the undeflected crack (see Eq. (1)). In the present study, the average crack-deflection angles observed for young, aged/opaque, and transparent dentin (± 1 standard deviation) were, respectively, $\alpha = 37^\circ \pm 12^\circ$, $27^\circ \pm 17^\circ$, and $5^\circ \pm 9^\circ$. Using Eqs. (1) and (3), these deflection angles result in a decrease in the crack-driving force on the order of 20%, 5%, and 0% for young, aged/opaque, and transparent dentin, respectively.[7] Although significant, these crack-deflection angles are still relatively small compared to transverse cracking in cortical bone where globally meandering cracks can be deflected at osteon boundaries (cement lines) with angles on the order of 90°, resulting in reductions in crack-driving force approaching a factor of two [24].[8] However, the microcracked tubules also serve to toughen human dentin by promoting crack branching, i.e., by allowing multiple crack tips to be active which then shield each other. This can be seen in Fig. 8e, f, where the existence of a branched crack means that the global stress intensity is now shared by more than one crack tip, thereby reducing the driving force for any one of them to grow.

Overall, it is clear that the degradation in the crack-growth resistance of human dentin with age results primarily from the decreased fraction of unfilled tubules and hence microcracked tubules (filled tubules do not microcrack), as these microstructural features play a prominent role in all the significant toughening mechanisms in dentin. This is apparent from Fig. 3 where the change in toughness between the three groups is mirrored by a change in the fraction of unfilled tubules involved in the crack path. As there is little uncracked-ligament bridging in either of the young or aged groups, the toughening results primarily from gross crack deflection and crack branching in human dentin although, as noted above, deflection only appears to result in $\sim$20% decrease in crack-growth resistance between young and transparent dentin. Crack branching requires the presence of unfilled tubules that can microcrack ahead of the crack tip to be active. This leads to a diminished branching with age as there are fewer unfilled tubules and thus fewer tubules that can microcrack, as shown in Fig. 3. These mechanisms, however, do not vanish completely with age as there are always some unfilled tubules even in aged/

[7] These crack deflections are referred to as *gross* deflections because they involve global excursions of the crack away from the $K_{II} = 0$ path, rather than local deflections around it.

[8] The *R*-curve toughness of human cortical bone can be over an order of magnitude higher in the transverse orientation, due to gross crack deflections and delamination along cement lines and interstitial interfaces, when compared to corresponding toughness behavior in the longitudinal orientations where crack paths are essentially linear [24].

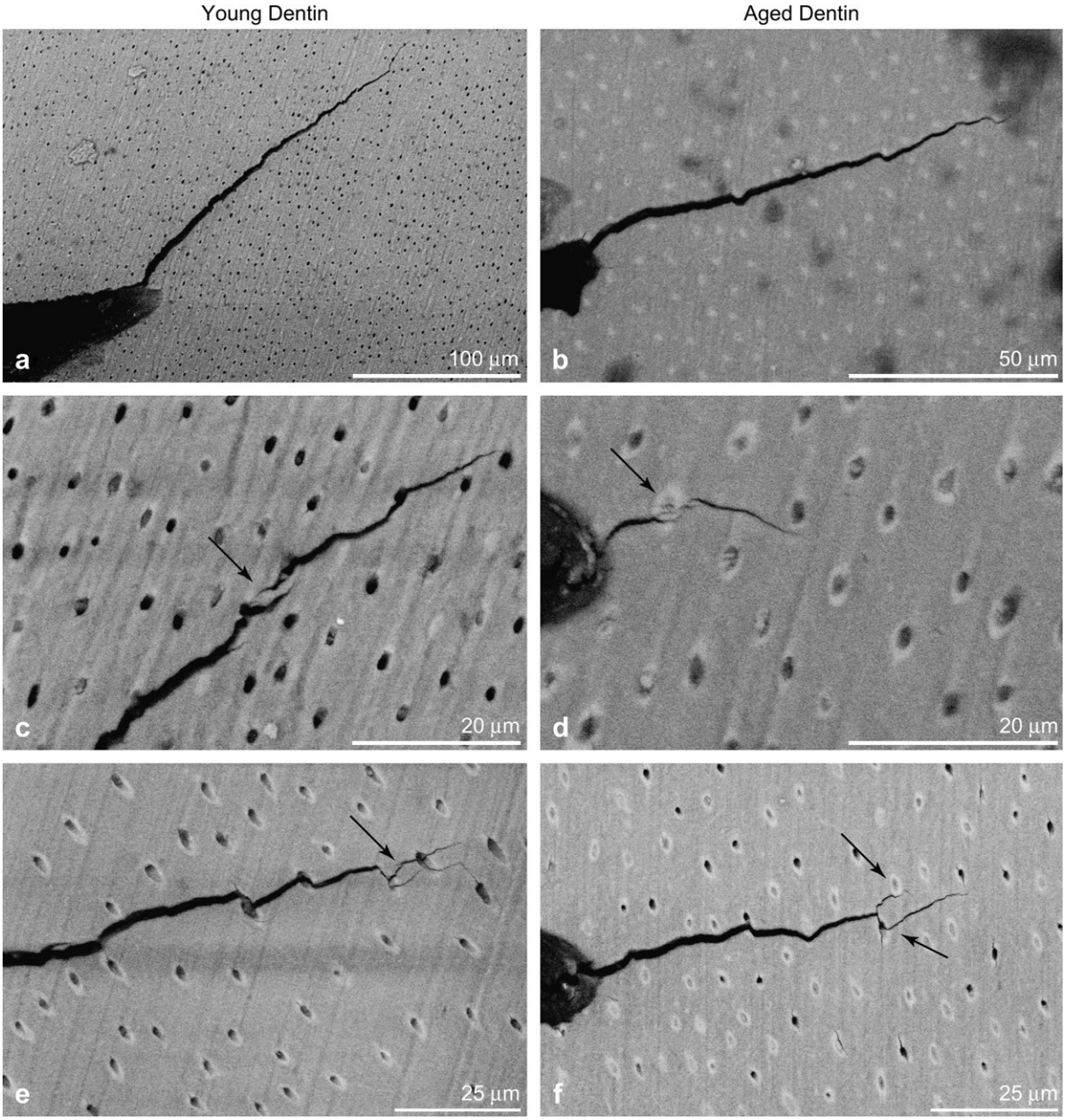

Fig. 8. ESEM images displaying the deterioration of the toughening mechanisms in moist human dentin with age. Images (a) and (b) show the gross crack deflection in young and aged/transparent. Crack bridging in young dentin (c), and aged/opaque dentin (d), was present in both conditions but formed via different mechanisms. The arrows in (c) and (d) indicate the bridges in these images. Crack branching, shown in images (e) and (f) and highlighted with the black arrows, was observed in all types of dentin.

transparent dentin. As can be seen in Fig. 3b, in all three groups, approximately half of the unfilled tubules, that the cracks intersect, are subject to microcracking.

In magnitude, the increase in crack-growth resistance for small ($\sim$250 μm) crack extensions in human dentin is not particularly large (less than a factor of 2.5–3), but toughnesses are comparable to other mineralized tissues such as human cortical bone, *in the longitudinal orientation*, for similar extents of cracking. This is logical as both tissues are comprised of type I collagen and hydroxyapatite and for short crack extensions the major extrinsic toughening mechanisms in the crack wake, such as crack bridging, will have not fully developed. Crack extensions in human dentin are short due to physiological size-scales; as a corollary, previous studies on elephant and bovine dentin (*e.g.*, Refs. [10,11]) have examined its fracture properties at length-scales that are not physical for humans. The disadvantage of this is that the crack deflection and branching mechanisms discussed herein, which may be the most important for human teeth, become dwarfed at larger length-scales by potentially more potent toughening mechanisms such as uncracked-ligament bridging. It is clear from the present work that this latter mechanism is far less

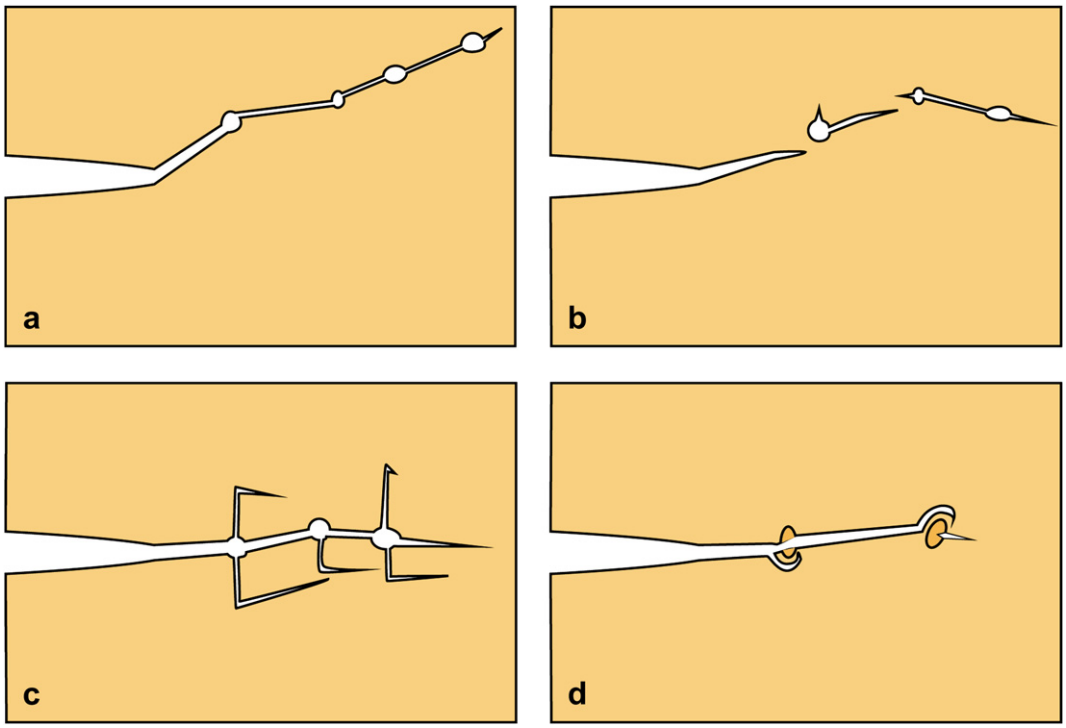

Fig. 9. Schematic diagrams of four toughening mechanisms in human dentin. (a) Gross crack deflection occurs as the crack initially grows from the sharpened notch at an angle and continues to grow at this angle following a path of open tubules ahead of the crack. (b) Uncracked-ligament bridges are formed on the micrometer scale by the growth of microcracks in the tubule network. (c) Crack branching occurs via the initiation of crack growth from multiple microcracks on the same tubule resulting in multiple active crack tips that shield each other. (d) Filled tubules also cause local crack deflection and result in the formation of bridges in the crack wake, although this mechanism was not commonly observed. All of the mechanisms, except (d), were observed in all three types of dentin; consequently, the difference in fracture toughness can be rationalized in terms of the reduced unfilled tubule fractions in aged dentin.

common at the relevant, sub-millimeter crack extensions associated with human dentin.

5. Conclusions

Based on an *in situ* environmental scanning electron microscopy study of the fracture toughness (from crack-growth resistance curves) of human dentin and its degradation with aging, the following conclusions can be made.

1. Human dentin derives its toughness primarily during crack growth via mechanisms that involve with the interaction of the crack path with the tubules. Indeed, in young dentin (from donors 19–30 years in age), the stress intensity required to sustain cracking was observed to be increased by up to a factor of two, as compared to aged dentin (from donors 40–70 years in age), over the first ~250 μm of stable crack extension (rising *R*-curve behavior).
2. Aging in dentin, which results in the filling of tubules with carbonated apatite, can diminish the crack-growth toughness by a factor of two but has little effect on the crack-initiation fracture toughness.
3. Microcracked tubules act as the ‘‘catalyst’’ for the generation of crack deflection, crack branching and

Table 2
Observations on the prevalence of the toughening mechanisms in human dentin and how they differ between the two extremes of young and aged/transparent dentin

Mechanism	Young	Aged/transparent	Empty tubule dependent	Corresponding figure
Gross crack deflection	Nearly all of the cracks grew at an angle	Uncommon and at an angle smaller than in young dentin	Maybe	Fig. 7a, b
Uncracked-ligament bridging between tubules	Uncommon	Not observed	Yes	Fig. 7c
Uncracked-ligament bridging through tubules	Very uncommon	Uncommon	No	Figs. 6 and 7d
Crack branching	Common	Uncommon	Yes	Figs. 4 and 7e, f

The table also refers to the figures that illustrate the differences between young and aged/transparent dentin.
Note: aged/opaque lies between these two extremes for all of the mechanisms.

uncracked-ligament bridging; their diminished presence in aged dentin means that these toughening mechanisms must become far less effective.

4. Due to the small crack extensions which are limited by the physical size of human teeth, toughening by bridging was far less prevalent in human dentin as compared to that observed at much larger-crack extensions in elephant and bovine dentin.

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