Available online at www.sciencedirect.com
ScienceDirect
Biomaterials 29 (2008) 1318–1328
Biomaterials
www.elsevier.com/locate/biomaterials

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

Kurt J. Koester [a,b], Joel W. Ager III [a], Robert O. Ritchie [a,b,*]

[a] Materials Sciences Division, Lawrence Berkeley National Laboratory, Berkeley, CA 94720, USA
[b] Department of Materials Science and Engineering, University of California, Berkeley, CA 94720-1760, USA

Received 12 September 2007; accepted 4 December 2007
Available online 31 December 2007

**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

* Corresponding author. Department of Materials Science and Engineering, University of California, 216 Hearst Mining Building, MC 1760, Berkeley, CA 94720-1760, USA. Tel.: +1 510 486 5798; fax: +1 510 643 5792.
*E-mail address:* roritchie@lbl.gov (R.O. Ritchie).

0142-9612/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.biomaterials.2007.12.008

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 ($\Delta a$). 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 $\sigma_{app}$ 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.