Contribution of Root Canal Treatment to the Fracture Resistance of Dentin

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Abstract

Introduction: Although the strength and toughness of dentin decrease with age, no study has explored if restorative treatments are a contributing factor. **Methods:** Multiple extracted teeth were obtained from randomly selected donors and categorized according to donor age and prior root canal treatment. The microstructure and chemical composition of radicular dentin were evaluated using scanning electron microscopy and Raman spectroscopy, respectively, and the strength was evaluated in 4-point flexure to failure. Data were compared using the Student *t* test. **Results:** Dentin from the root canal–restored teeth exhibited significantly lower strength ($P < .05$) than tissue from age- and donor-matched unrestored tooth pairs. Although there was no significant difference in the mineral-to-collagen ratio between the 2 groups, dentin obtained from the root canal–treated teeth exhibited more extensive collagen cross-linking and lower tubule occlusion ratios than the unrestored tooth pairs. **Conclusions:** There is a decrease in the strength of radicular dentin with aging, but prior root canal treatment increases the extent of degradation. *(J Endod 2019;45:189–193)*

Key Words

Aging, collagen, dentin, fracture, root

Significance

There is a decrease in the strength of radicular dentin in teeth that have received endodontic treatment followed by clinical function. This degradation exceeds that which results from natural aging and increases the potential for root fractures over time.

With the growth in the number of dentate seniors, the dental profession is facing new challenges (1). One of the more frequent problems is tooth fracture (2). There are detrimental changes to the mechanical properties of teeth with aging, particularly in dentin (3, 4).

There is a gradual reduction in the diameter of the dentin tubule lumens with increasing age because of an accumulation of minerals (5–7). As a result, the tissue becomes more translucent, commonly known as dentin sclerosis. This change in microstructure is accompanied by a decrease in resistance to fracture (7–10). Age-related degradation is equally prominent in the crown and the root (11); yet, no study has evaluated if the age-related degradation is more extensive in restored teeth.

Vertical root fracture (VRF) is among the most common forms of tooth failure and involves cracks originating from the root apex and extending into the occlusal-cervical plane. Patients diagnosed with VRF often experience discomfort, but there are no consistent signs or symptoms, making a diagnosis difficult (12). There is no universal approach for repairing teeth with VRF, which often leads to extraction (13). Most importantly, VRFs are more common in seniors (14, 15).

Teeth that have received root canal therapy experience VRF more often than unrestored teeth (15, 16). Survival rates can be initially high after treatment (17), but the risk of fracture increases with time (18). The greater risk of fracture has been attributed to the increase in stress with loss of tooth structure (19). The treatment itself is also a concern. Although instrumentation does not generate defects that cause tooth failure (20), rotary and reciprocating instruments can lead to dentinal defects (21) that undergo mechanical cycling during apical filling of the root (22) and thereafter during function. It is unclear if this leads to tooth fracture.

Degradation after root canal treatment is also a possibility. Carter et al (23) reported a significant difference in the shear strength and toughness of dentin between vital and root canal–treated teeth; yet, Cheron et al (24) found that the elastic modulus and hardness of radicular dentin are not different before and after root canal treatment. Similarly, Missau et al (25) reported that treated teeth exhibit the same fatigue resistance as untreated teeth.

In this study, the chemical composition, microstructure, and strength of multiple teeth from selected donors were compared as a function of donor age and prior root

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canal treatment. The null hypothesis was that there is no difference in the fracture resistance of unrestored teeth and those that have undergone treatment followed by clinical function.

Materials and Methods

A total of 55 human teeth were obtained from participating oral surgeons according to an exempt protocol approved by the Institutional Review Board of the University of Washington, Seattle, WA. An inspection of the teeth was conducted for caries or lesions, structural defects, or prior restorations. Those with evidence of caries or defects were discarded. Between 6 and 13 teeth were obtained from each donor (Fig. 1*A*), and 5 of the donors possessed root canal–treated teeth. The teeth were immediately stored in Hank's balanced salt solution (HBSS), with record of tooth position, donor age, and gender.

Those teeth selected for evaluation were cast in a polyester resin foundation and sectioned axially in the mesial-distal direction using slow-speed diamond abrasive wheels with continuous water irrigation. One half of each tooth was subjected to further sectioning following established methods (11) to obtain rectangular beams from the roots in the buccal-lingual quarters (Fig. 1*B*). After machining, the beams were stored in HBSS at 22°C for fewer than 2 days.

The dentin beams were subjected to 4-point flexure to failure according to established methods (3). The flexure loading apparatus consisted of a 1/3 span arrangement (Fig. 1*C*). The experiments were conducted using a universal testing system (EnduraTEC Elf Model 3200; TA Instruments, New Castle, DE) with specimens immersed in an HBSS bath (22°C) to maintain hydration during testing. Quasi-static flexure was performed under displacement control loading to failure with a crosshead rate of 0.001 mm/s. The strength of each beam was determined from the maximum flexure stress to failure, which was calculated according to conventional beam theory. The strengths of the root canal–treated and untreated dentin from the 6 donors were checked for normality and compared using a paired Student *t* test with the critical value (alpha) set to 0.05.

The microstructure of the radicular dentin was examined using a scanning electron microscope (Model JSM- 6010PLUS/LA; JEOL, Peabody, MA) and image processing. The remaining half of each sectioned tooth was embedded in cold-cured epoxy resin exposing the root canal and longitudinal section according to established methods (11). The exposed dentin in the resin mount was polished using silicon carbide abrasive paper from #800–#4000 mesh until the dentinal lumens became evident. Further polishing was performed using diamond particle suspensions to 3 μm in size. The total number of open lumens and the number of occluded lumens were counted using commercial software (ImageJ 1.8.0; National Institutes of Health, Bethesda, MD). Results were expressed as the occlusion ratio, which is the number of occluded lumens to the total number of lumens according to Montoya et al (7).

The chemical composition was analyzed using Raman spectroscopy (Renishaw InVia, West Dundee, IL) with scans performed over the spectral range of 400–1900 cm^{-1} and acquired at distances of 4 mm away from the root apex. The spectra were baseline corrected for fluorescence using WiRE 3.4 (Renishaw, West Dundee, IL). The cross-linking ratio was calculated from the ratio of the area under the pyridinoline (Pyr) peak at 1660 cm^{-1} and the dehydrodihydroxylysinonorleucine (deH-DHLNL) peak at 1690 cm^{-1} according to Yan et al (11). The mineral-to-collagen ratios, collagen cross-linking ratios, and the occlusion ratios for the root canal–treated and untreated dentin were checked for normality and compared using an unpaired Student *t* test with the critical value (alpha) set to 0.05.

Results

The average flexure strength of the radicular dentin samples is presented for both the unrestored and root canal–restored teeth as a function of donor age in Figure 2*A*. The average strength of radicular dentin reported by Yan et al (11) for young donor teeth is also shown for reference (199 ± 36 MPa, age ≤30 years). All of the teeth from donors ≥55 years of age exhibited significantly lower strength (P < .0001) than that of young dentin. The average flexure strength of the unrestored dentin for the 5 donors identified as "old" is 150 ± 16 MPa, respectively. There is a general decrease in strength with increasing donor age for this group, and the strength for the oldest donor teeth was the lowest overall (130 ± 1.4 MPa).

For the root canal–treated teeth (Fig. 2), there is no trend in the strength of dentin with donor age. There was no significant difference in the strength (P > .05) of dentin from the root canal–treated teeth among the 5 donors. The overall average flexure strength of the root canal–restored dentin is 123 ± 18 MPa, which is significantly lower ($P \leq$ .0005) than that of dentin from all the unrestored teeth, and also significantly lower than that of dentin from the unrestored teeth within the donor-matched dentition ($P \leq$ .05).

A

Age	Location		Treatment		Total
	A	P	Endo treated	Non-endo treated	
46	1	7	0	8	8
56A	5	8	5	8	13
56B	1	5	2	4	6
60	5	6	3	8	11
63	3	7	4	4	8
74	3	3	3	3	6
Total	18	36	17	35	52

B C

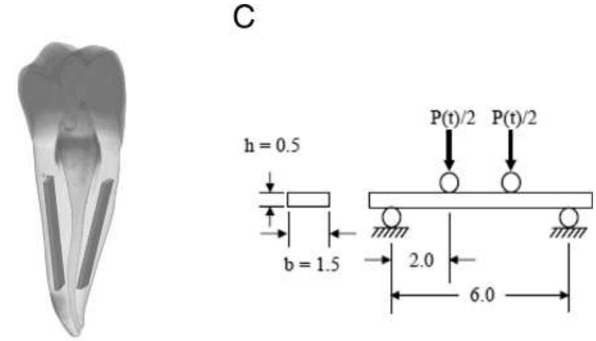

Figure 1. Details concerning the samples and methods of evaluating the strength and fatigue resistance. (*A*) The investigation involved 52 total teeth obtained from 6 donors with age spanning from 46 to 74 years. A, anterior tooth; P, posterior tooth. Note that 56A and 56B represent the teeth of 2 different donors with the same age. (*B*) The teeth were divided into anterior and posterior groups and root canal–treated and non–root canal–treated teeth (control). (*C*) Beams were obtained from the buccal-lingual and mesial-distal regions of the teeth, and (*D*) the beams were subjected to 4-point flexure according to a 1/3 point loading arrangement.

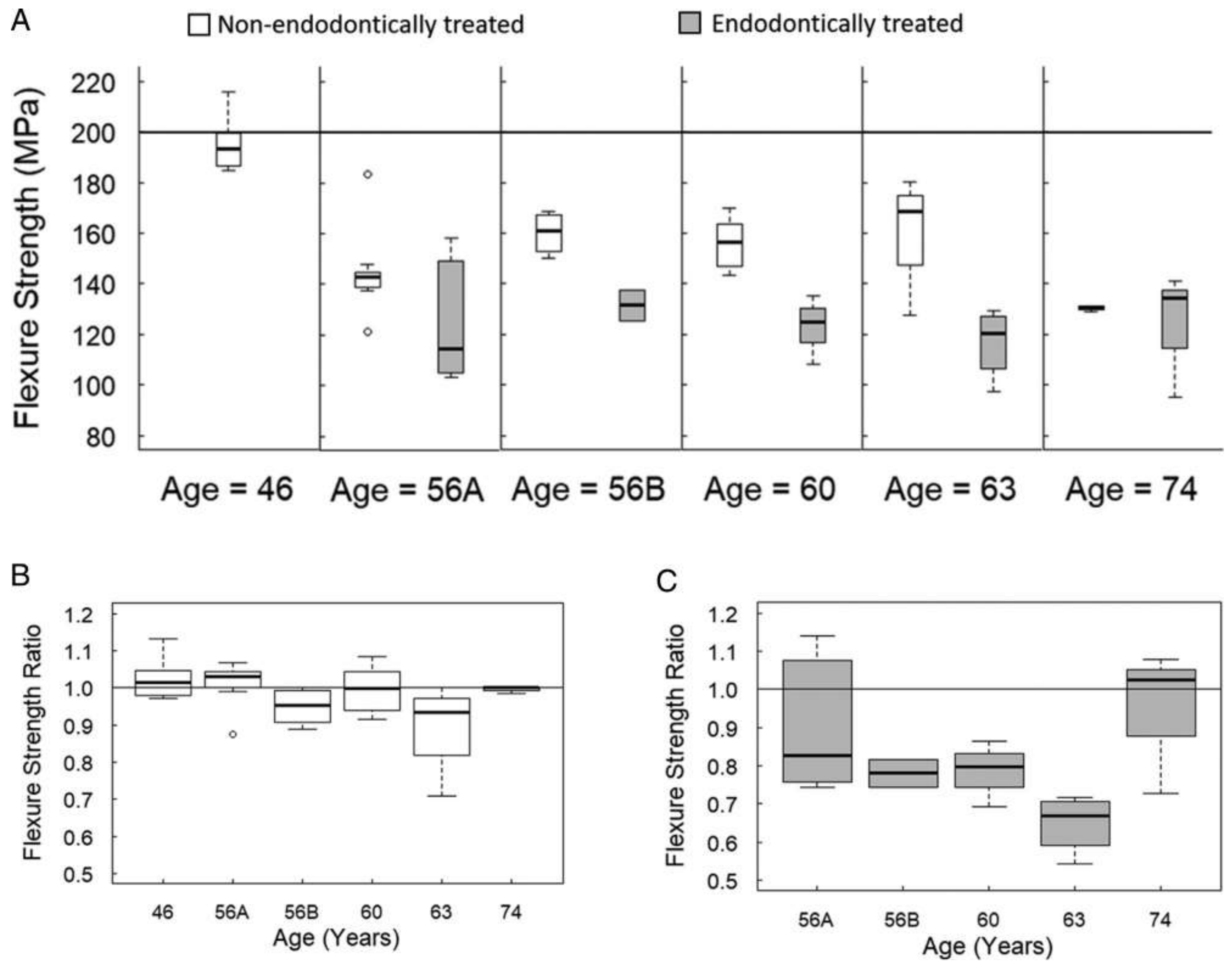

Figure 2. Results for the flexure strength of dentin from the various teeth. (*A*) Flexure strength of root canal–treated and non–root canal–treated teeth in relationship to young radicular dentin. The average flexure strength of young radicular dentin (199.7 MPa) is shown for comparison based on Yan et al (11). The ratio of flexure strength of dentin from the (*B*) unrestored and (*C*) root canal–restored teeth to the strength of radicular dentin from untreated incisors of the same donor.

A normalization of the flexure strength was performed for further analysis. Specifically, the flexure strength of dentin from all teeth evaluated was normalized by that from a non–root canal–treated incisor (including tooth numbers 8, 9, 24, and 25) from the same donor. Results for the unrestored and the root canal–treated teeth are presented in Figure 2*B* and *C*, respectively. In Figure 2*B*, the strength of the unrestored posterior teeth is not significantly different from the incisor control. The overall average flexure strength ratio of root canal–treated teeth was 0.82 ± 0.17; the smallest ratio was 0.65 ± 0.08 and was obtained for the 63-year-old donor. For 4 of the 5 donors (56A, 56B, 60, and 63), the strength ratios of the posterior teeth were significantly lower ($P \leq .05$) in comparison with the healthy incisors. Interestingly, the root canal–treated teeth from the oldest donor did not show significant differences in strength from the unrestored incisor ($P > .05$) as evident in Figure 2*C*.

Results of the chemical composition and microstructural analysis are shown in terms of the mineral-to-collagen ratio, the collagen cross-linking ratio, and the occlusion ratio in Figure 3*A*–*C*, respectively. There was no significant difference in the mineral-to-collagen ratio between the dentin of root canal– and non–root canal–treated teeth. However, dentin from the root canal–treated teeth exhibited a significantly greater collagen cross-linking ratio ($P \leq .005$) and a significantly lower occlusion ratio ($P \leq .025$).

Discussion

In this investigation, the chemical composition, microstructure, and strength of radicular dentin were evaluated from multiple teeth of randomly selected donors. A comparison of the properties performed using age- and donor-matched tooth pairs enabled an assessment of the importance of prior root canal treatment.

The strength of radicular dentin from the unrestored teeth (150 ± 16 MPa, Fig. 2) was significantly lower than that reported for young radicular dentin (199 ± 36 MPa). Apart from the average strengths of dentin from teeth of the donor identified as 56A, there was also a general trend indicating a reduction in strength with increasing age, as expected. The maximum reduction in strength (35%) with regard to young dentin was exhibited by teeth from the 74-year-old donor. The variation in strength of the unrestored teeth was also the smallest for the 74-year-old donor. For radicular dentin, Yan et al (11) found the rate of degradation in strength is approximately

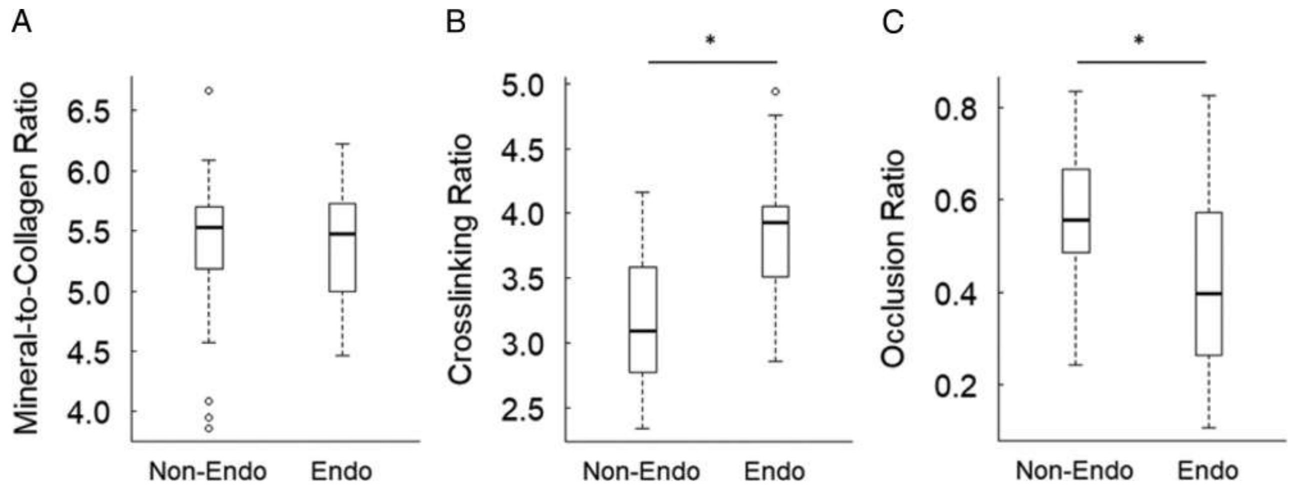

Figure 3. Results of Raman spectroscopy and microscopic analysis of dentin from the buccal-lingual quadrants and 4 mm from the root apex. (*A*) The mineral-to-collagen ratio, (*B*) the collagen cross-linking ratio, and (*C*) the occlusion ratio are presented in terms of prior root canal treatment. Endodontically treated teeth have a significantly higher cross-linking ratio ($P < .005$) and lower occlusion ratio ($P < .025$) than untreated teeth.

25 MPa per decade until reaching age ≥55 years, after which it reaches a plateau. That trend is also reflected in coronal dentin (3).

In contrast to the unrestored group, dentin from the restored teeth did not show age dependence (Fig. 2*A* and *C*). Nevertheless, the average strength of the treated group was almost 20% lower than that for the unrestored group. A comparison of results in Figure 2 show that, regardless of age, the strength of dentin from root canal–treated teeth is lower than that of unrestored teeth, which requires rejection of the null hypothesis. Thus, independent of the loss of tooth structure with instrumentation (19), the strength of the root tissue decreases after root canal treatment. This is the first investigation to provide clear evidence of the changes in strength of dentin occurring after treatment and clinical function.

A major concern in previous studies on the properties of tooth tissues is the importance of patient/donor-specific oral conditions and health status. Indeed, this concern is highlighted in the strength of dentin for 2 different 56-year-old donors (Fig. 2). The average flexure strength for the unrestored teeth for these 2 donors is significantly different ($P \leq .05$). Microstructural characteristics such as lumen density (26) and collagen fibril diameter (27) are important. Dietary habits, use of medications, prior trauma, and even differences in masticatory habits are also plausible contributions. There is a reduction in the strength of dentin with age, but the extent of reduction is clearly patient specific.

Normalizing the strength measurements with a reference from the same patient reduced the influence of patient-specific and potentially confounding factors as previously discussed. The merit of this approach is evident in Figure 2*B* and *C* where all the unrestored and root canal–restored teeth are normalized to a healthy unrestored incisor. The unrestored teeth from 5 of the 6 donors have an average strength within 5% of that of the healthy incisor, indicating a very small variation in properties "within" the patient's dentition. For the root canal–restored teeth, 4 of the 5 donors possessed strength that is 15% lower than that of the unrestored teeth, independent of the degradation related to age.

Carter et al (23) found that root canal–treated dentin is weaker and more brittle than vital dentin but did not establish the principle cause. Prior studies suggest that root canal–treated teeth are more brittle because of dehydration (28, 29), regardless of instrumentation. This is a controversial subject. For instance, Huang et al (30) conducted compression, indirect tensile testing, and impact testing on human dentin and showed no significant difference between the properties of dentin from treated and untreated teeth. Additional evidence suggests that dehydration does not necessarily reduce the resistance to fracture (31) or fatigue crack growth (8) of dentin. Future work should focus on the changes in free and bound water in the dentin of root canal–treated teeth and their contribution to its mechanical durability.

Collagen cross-linking is considered a primary contributor to the degradation in fracture resistance of bone with age (32). In comparing the root canal–treated teeth with their unrestored reference, there was no difference in the mineral-to-collagen ratio (Fig. 3*A*), and, although statistically significant, there was a small difference in the occlusion ratio (Fig. 3*C*). However, the root canal–treated teeth exhibited a significantly greater collagen cross-linking ratio, above that associated with aging. That signals an increase in the nonreducible hydroxypyridinium cross-links Pry and deoxypyridinoline relative to the reducible divalent cross-links deH-DHLNL and dehydrohydroxylysinonorleucine. An increase of nonreducible cross-links has been observed in dentin with aging (33) and contributes to its fragility (11, 34). The ratio of Pyr and deH-DHLNL characterizes collagen maturity (35). The higher cross-linking ratio of the root canal–treated dentin (Fig. 3*B*) suggests that it is the primary contribution to the reduction in strength relative to the unrestored teeth. Because this is superposed with the effects of natural aging, root canal treatment appears to accelerate the aging process, which causes an increase in tooth fragility.

There are several limitations to this study. Root canal treatment is often applied to teeth that have been exposed to bacteria and have undergone inflammation of the pulp. Although this could be a factor in the lower strength, the mineral-to-collagen ratio did not show any differences based on treatment (Fig. 3*A*). A related concern is that the period between treatment and extraction was unknown, and the treated teeth undoubtedly have unique periods of posttreatment clinical function. This topic is important and requires further exploration.

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