Effect of Prolonged Direct and Indirect Peroxide Bleaching on Fracture Toughness of Human Dentin

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ABSTRACT

Statement of the Problem: The effects of prolonged exposure to peroxide bleaching agents on dentin structural integrity are uncertain.

Purpose: To evaluate the effect of in vitro prolonged tooth bleaching on the fracture toughness (K_{1C}) of human dentin.

Materials and Methods: Dentin from recently extracted molar teeth was *directly* or *indirectly* treated to simulate a prolonged at-home (10% carbamide peroxide or 3% hydrogen peroxide, 6 hours/day, 5 days/week for 8 weeks) or in-office (30% hydrogen peroxide, 1 hour/week for 8 weeks) bleaching regimen ($N = 8$/group). Placebo gel and distilled water acted as control materials. Compact tension test specimens (approximately $4.60 \times 4.50 \times 1.60$ mm) were prepared from coronal dentin and tensile loading was applied at a rate of 10 mm/min 24 hours after the last bleaching session. Results were analyzed using analysis of variance and Tukey's test ($p < 0.05$). For *direct* bleach application, the treatment materials were applied onto dentin that was already prepared as compact tension specimens. For *indirect* bleach application, bleach was applied to the enamel of intact teeth prior to specimen preparation.

Results: There was a significant decrease in dentin K_{1C} after 8 weeks of *direct* bleach treatment ($p < 0.05$). There were no significant differences between the bleach and control groups after 8 weeks of *indirect* bleach treatment ($p = 0.19$).

Conclusions: The in vitro fracture resistance of dentin was reduced after the prolonged use of bleach products that were applied *directly* to dentin.

CLINICAL SIGNIFICANCE

Caution should be considered when using bleach for prolonged treatment times in clinical cases where there is dentin exposure such as occlusal attrition or gingival recession.

(*J Esthet Restor Dent* 19:100–110, 2007)

INTRODUCTION

Tooth bleaching methods generally include the application of 10% (or higher) carbamide peroxide (CP) or hydrogen peroxide (HP) equivalent products onto the tooth surface for several hours daily over a few weeks in an *at-home* tooth bleaching regimen, and the application of 30% (or higher) HP onto the tooth surface for several minutes over a few appointed sessions

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DOI 10.1111/j.1708-8240.2007.00075.x

in an *in-office* tooth bleaching regimen. These regimens can be repeated many times to maintain tooth whitening. A wide range of bleaching products is available on the market. These products are often classified as cosmetics and there is, therefore, no detailed requirement to ensure material safety and effectiveness. It is important to determine the potential adverse effects of these widely used products, particularly when used repeatedly.

Tooth bleaching treatments have been associated with possible negative effects on dental hard tissues, including decreased bonding ability,[1–3] changes to enamel and dentin surface morphology,[4–9] decreased surface hardness,[10–11] and decreased abrasion resistance.[12] In a recent study, direct exposure to 10% CP caused a significant decrease in the flexural strength and flexural modulus of elasticity of bovine dentin.[13] This effect appeared to be time related, showing a greater decrease in mechanical properties after the 2-month period over the 2-week period of bleach application and no difference when the shorter application times of HP were used. Chng and colleagues reported significant decreases in the ultimate tensile and micropunch shear strengths of dentin after an intracoronal bleach application of 30% HP.[14] In these studies, the bleach was applied *directly* onto the dentin surface. These studies characterized structural changes that occurred as a result of direct bleach application to dentin and are relevant to the clinical cases of bleaching where there is dentin exposure such as occlusal attrition or gingival recession. The intended clinical application of bleach, however, is onto the enamel. The permeation of bleach through the enamel then exposes the dentin to bleach *indirectly*. The effect of *indirect* application of bleach to dentin is unknown.

The use of a fracture toughness (K_{1C}) test better quantifies the tooth's resistance to fracture than conventional strength tests such as the flexural strength test.[15] The application of fracture mechanics principles to dental biomaterials is invaluable in evaluating structural integrity and in failure analysis.[16] Plane-strain K_{1C} is an intrinsic material property and K_{1C} values are true measures of structural integrity that do not change with different specimen geometries or loading conditions.

This study proposed the use of the K_{1C} parameter to study further the effects of tooth bleach on dentin mechanical properties in a *direct* application (bleach applied directly to dentin) and in a more clinically relevant experiment using an *indirect* application of bleach (bleach applied directly onto enamel using a whole-tooth model and hence, indirectly to dentin). As a starting point for detecting possibly small differences and to better understand bleaching effects, a prolonged exposure time (8 weeks) was selected for this study. An 8-week cumulative exposure time can be encountered in patients dealing with whitening regression. Even longer treatment times are recommended for patients with tetracycline staining.[17–18] The objective of this study was to determine the effects of *direct* and *indirect* dental bleaching on the K_{1C} of human dentin using a prolonged bleaching protocol. The null hypothesis for this study was bleaching has no effect on the K_{1C} of dentin.

MATERIALS AND METHODS

Eighty human molar teeth, extracted within the previous 3 months and immediately stored in 4°C distilled water (DW) containing 1% chloramine, were collected from local dentists' and oral surgeons' offices. The protocol for the collection and use of teeth was approved by the institutional Research Ethics Board (protocol reference #18028). Teeth with visible signs of decay, fractures, or cracks were discarded. No attempt was made to determine the age, sex, or site of the extracted teeth. The teeth were randomly divided into material (10% CP, 3% HP, 30% HP, placebo, or DW) and application mode (*direct* or *indirect*) treat-

ment groups ($N = 8$) and subjected to bleach or control treatments as outlined in Table 1. A bleach treatment consisted of an application of a 1- to 2-mm-thick layer of fresh bleach gel to the tooth surface or immersion into fresh bleach liquid for a prescribed period of time. During this time, the specimens were stored in an incubator at 37°C, > 80% relative humidity. At the end of each bleach treatment, the specimens were rinsed with tap water to remove all external traces of bleach and the specimens were stored in an incubator in 37°C DW until the next bleach application.

To simulate a prolonged at-home bleaching regimen, the dentin was directly or indirectly treated with 10% CP (Opalescence, Ultradent Products, Inc., South Jordan, UT, USA) or 3% HP (prepared using 30% HP liquid [Wiler-PCCA, London, Ontario, Canada] and placebo gel [Ultradent Products, Inc.]) for 6 hours daily, 5 days a week for 8 weeks. A simulation of the prolonged in-office bleaching regimen was achieved by immersing the dentin directly or indirectly into 30% HP liquid (Wiler-PCCA) 1 hour per week for 8 weeks. Placebo gels (Ultradent Products, Inc.) and DW were used as control materials and a fresh supply surrounded the tooth surface of each control specimen in a direct or indirect application mode for 6 hours daily, 5 days a week for 8 weeks.

Compact tension test specimens (Figure 1), as described by El Mowafy and Watts[19] and nominally conforming to ASTM E399,[20] were prepared from the coronal dentin for K_{1C} testing. A water-cooled low-speed diamond saw (Buehler Ltd., Lake Bluff, IL, USA) was used to cut a rectangular slice with approximate dimensions of 4.60 × 4.50 × 1.60 mm from the dentin below the occlusal enamel. The orientation of the slice was parallel to the occlusal surface. Only one slice was obtained from one tooth. A central notch of specific length was then made with diamond discs (ThinFlex x929.7, [Abrasive Technology] Premier Products Co, Plymouth Meeting, PA, USA) and sharpened with a razor blade. This notch acted as a stress concentrator and crack propagation originated from this notch. The specimen and notch dimensions were measured using a micrometer and confirmed with a microscope.

TABLE 1. MATERIALS USED FOR THE BLEACH AND CONTROL GROUPS.

Group	Materials	Treatment Protocols
At-home		
10% carbamide peroxide (CP) gel	Opalescence (Ultradent Products, Inc., South Jordan, UT, USA). Containing 10% CP, glycerine, carbopol, water, pH 6.4.	6 hours daily, 5 days per week for 8 weeks
3% hydrogen peroxide (HP) gel	Prepared using 30% HP (Wiler-PCCA, London, Ontario, Canada) and placebo gel (Ultradent Products, Inc.), pH 6.0.	
Placebo gel (pH 6.8)	Ultradent Products, Inc. Containing glycerine, carbopol, water, pH 6.8.	
Distilled water (pH 7.4)	—	
In-office		
30% HP liquid	HP 30% (Wiler-PCCA). pH 4.0.	1 hour per week for 8 weeks

The treatment protocols apply to both the *direct* and the *indirect* bleaching application methods. The pH values were recorded using a pH meter prior to application to the teeth (Accumet 620 pH/mV meter, Fisher, Pittsburgh, PA, USA).

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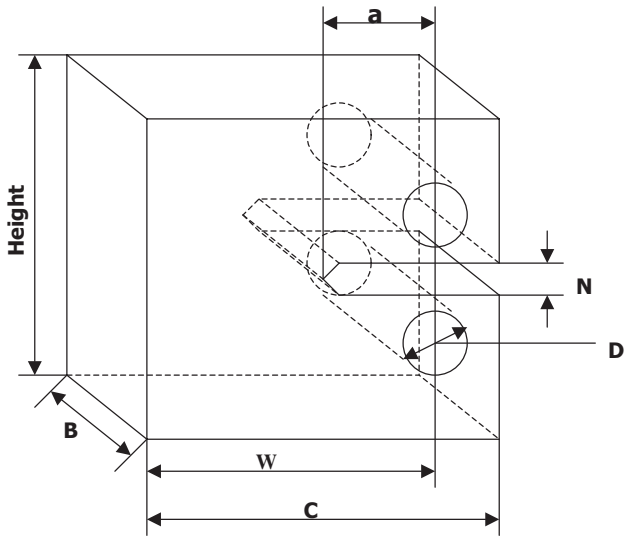

Figure 1. Compact tension test geometry. Height is approximately 4.5 mm.
C = total width, approximately 4.6 mm; B = thickness, approximately 1.6 mm; a = effective notch length (to fulfill the criteria of a/W between 0.45 and 0.55); W = net width; N = notch width, approximately 0.4 mm; D = hole diameter, approximately 0.8 mm.

For *direct* dentin bleaching, the treatment materials were applied directly onto dentin that was already prepared as compact tension specimens. For *indirect* bleaching, the treatment materials were applied onto the coronal aspect of the intact molar prior to compact tension specimen preparation. For the 10% CP, 3% HP, and placebo groups, the molar roots were suspended through a 1-mm-thick sheet of wax that was adapted to each molar around the cementoenamel junction. The roots were immersed in DW beneath the wax lid while the treatment materials were applied to the coronal enamel. For the 30% HP and DW groups, the molar crowns were inverted directly into the treatment solution. Care was taken to avoid dehydration of the root surface by maintaining specimen storage in the humidity chamber. Areas of enamel in close proximity to the root surface were left unbleached rather than risk inadvertent contact of the bleach material with the root surface. Compact tension test specimens were prepared from the indirectly treated molars approximately 20 hours after the last bleaching session.

Twenty-four hours after the last bleaching session, the specimens were mounted on an Instron universal testing machine (Model 4301, Instron Corp., Canton, MA, USA) for K_{1C} testing. Two round wires with a diameter of 0.5 mm were utilized for attachment. Tensile loading was applied at a rate of 10 mm/min. The K_{1C} results were analyzed using analysis of variance (ANOVA) and Tukey's test ($p < 0.05$).

The K_{1C} of each specimen was calculated using the following formula:

$$K_{1C} = P \cdot Y_2 / B \cdot W^{1/2},$$

where:
P = maximum load required to fracture specimen (MPa);
$Y_2 = f(a/W)$; a tabulated function of (effective notch length, a)/W;
B = specimen thickness (m); and
W = specimen net width (m).

The specific compact tension test specimen criteria were verified for each specimen. The specimens were stored immediately after fracture in 100% ethanol. Two specimens were randomly selected from each group for evaluation of the fracture surface using scanning electron microscopy (SEM) (Hitachi S-2500, Hitachi Ltd., Tokyo, Japan). These specimens were critical point dried (Polaron CPD-7501, Fisons Instruments, Sussex, England) using ethanol and liquid CO_2 as the intermediate and final dehydration fluids. The critical point method of

drying helps avoid the deformation and collapse of vulnerable surface structures resulting from surface tension effects by never allowing a liquid/gas interface to develop. The specimens were then mounted on a 12-mm aluminum stub using a cyanoacrylate adhesive and sputter-coated with platinum (Polaron SC515 SEM Coating Systems, Fisons Instruments).

RESULTS

The ANOVA showed that there were significant differences among all the treatment groups ($p < 0.0001$). There was a significant interaction among the treatment factors, material, and application mode ($p = 0.002$).

The K_{1C} results for the *direct* application of bleach and control treatment groups are shown in Figure 2. Compared with the control groups, the mean dentin K_{1C} was significantly lower in the 10% CP, 3% HP, and 30% HP groups after 8 weeks of direct bleach treatment ($p < 0.05$). The mean K_{1C} for the 10% CP and 30% HP direct treatment groups was significantly lower than the mean K_{1C} for the 3% HP group ($p < 0.05$). There was no significant difference between the control placebo and DW material groups.

The K_{1C} results for the *indirect* application of at-home and in-office treatment groups are shown in Figure 3. There were no significant differences among the bleach and control material groups after 8 weeks of indirect bleach treatment ($p = 0.19$).

SEM examination of the fracture surfaces generally revealed no marked differences between the bleach and control groups. The dentin fracture surface displayed the length of the dentinal tubules surrounded by peritubular dentin and intertubular dentin. Localized regions of exposed collagen fibers and voids, an appearance consistent with demineralization in the intertubular dentin, were detected near the fracture surface edges that were directly exposed to bleach. Localized regions of exposed collagen fibers and voids were also detected near the fracture surface edges that were directly exposed to control materials but to a lesser degree. Such regions were also sporadically detected in the central portion of the dentin fracture surface (remote from the edge) of the direct bleach

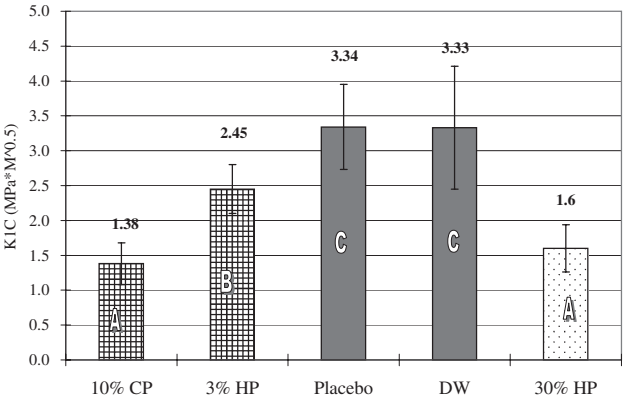

Figure 2. Fracture toughness (K_{1C}) of human dentin after direct application of 10% carbamide peroxide (CP), 3% hydrogen peroxide (HP), 30% hydrogen peroxide using simulated at-home (hatched) and in-office (dotted) wear pattern of bleaching, or control placebo and distilled water (DW) materials (shaded). Groups denoted by the same letter do not differ significantly. The K_{1C} values of the 10% CP and 30% HP groups were significantly lower than that of the 3% HP group, and all values were significantly lower than that of the control (placebo and DW) groups.

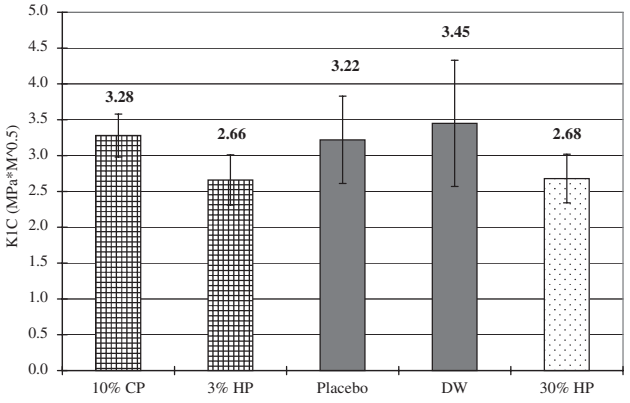

Figure 3. Fracture toughness (K_{1C}) of human dentin after indirect application of 10% carbamide peroxide (CP), 3% hydrogen peroxide (HP), 30% hydrogen peroxide using simulated at-home (hatched) and in-office (dotted) wear pattern of bleaching, or control placebo and distilled water (DW) materials (shaded). There were no significant differences in K_{1C} among the groups.

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groups (Figure 4) but not in the direct control groups. No such regions were detected in the indirectly treated bleach and control groups (Figure 5).

DISCUSSION

This was the first study to utilize the K_{1C} test to evaluate the effect of tooth bleaching on dentin. The compact test specimen is a standard configuration used for K_{1C} testing. The specimen preparation aspects of this study were time consuming and required great care.

The mean K_{1C} for the control dentin groups ranged from 3.2 to 3.5 MPa.m$^{1/2}$ in this study. Few studies in the literature have measured dentin K_{1C}. The mean K_{1C} reported for human dentin at different temperatures, measured by using a compact tension specimen geometry, ranged from 2.9 to 3.1 MPa.m$^{1/2}$,[19] and in another study of elephant dentin using different tubule orientations, from 1.5 to 2.6 MPa.m$^{1/2}$.[21] Another study used fatigue-precracked three-point bend bar samples and measured K_{1C} values in the range of 1.73 to 1.85 MPa.m$^{1/2}$.[22] That study

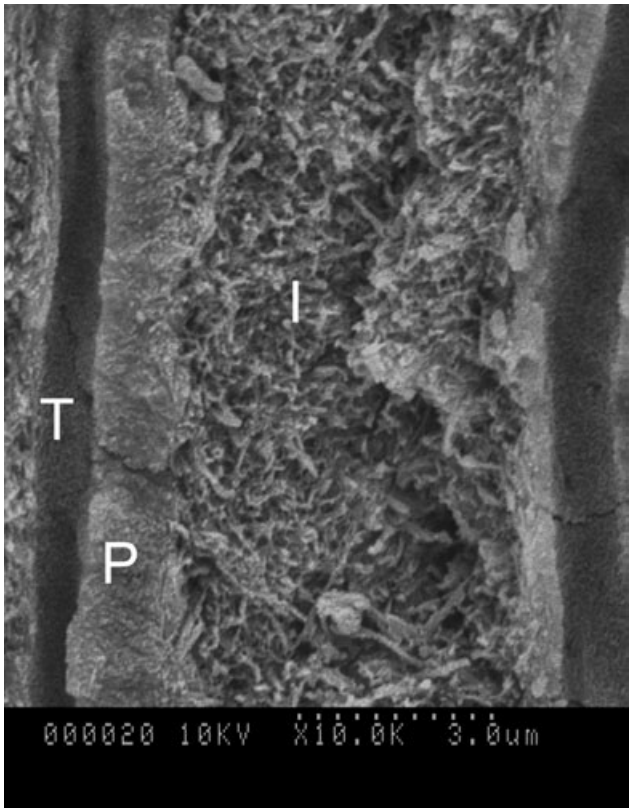

Figure 4. Scanning electron microscopy photomicrograph of dentin fracture surface after 8 weeks of direct bleach treatment (3% hydrogen peroxide). The location of this photomicrograph is near the middle of the fracture surface. Voids and collagen fibers are visible in the intertubular dentin.
T = dentin tubule lumen; P = peritubular dentin; I = intertubular dentin.

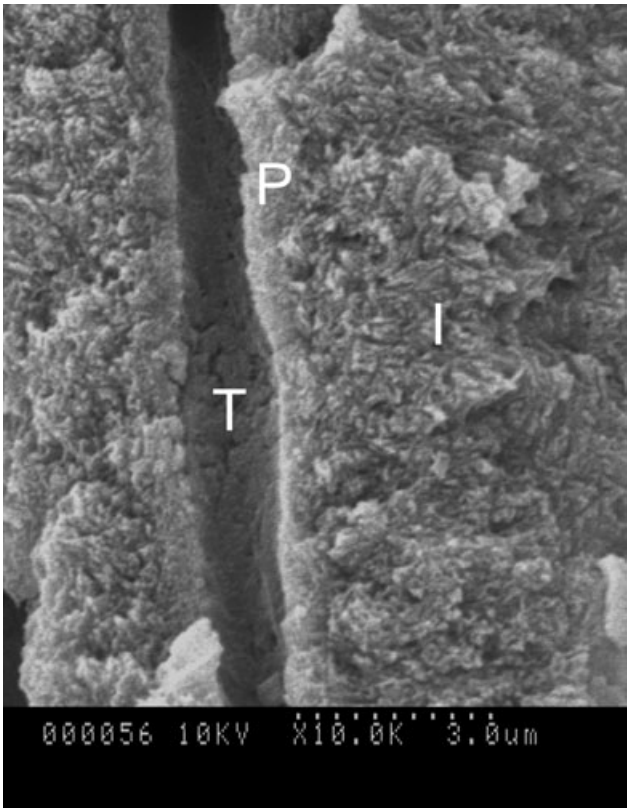

Figure 5. Scanning electron microscopy photomicrograph of a typical dentin fracture surface after 8 weeks of indirect bleach treatment (3% hydrogen peroxide). The location of this photomicrograph is near the middle of the fracture surface.
T = dentin tubule lumen; P = peritubular dentin; I = intertubular dentin.

attributed the lower K_{1C} values to a difference in the orientation of the dentinal tubules and primarily to the effect of notch acuity on the K_{1C} value. The orientation of the dentinal tubules for the specimens in this study was "in-plane parallel" to the notch plane for all the specimens. This orientation was associated with a higher dentin K_{1C} than a "perpendicular" orientation.[21,23]

In this study, the K_{1C} specimens fractured along the midplane and the fracture initiated from the tip of the central notch. The tip of the central notch in this study was sharpened with a fine blade but was not fatigue precracked. The lack of a precrack in the specimens may also have contributed to higher K_{1C} values for this study. The use of a precrack may be theoretically correct but is difficult to produce and measure. For composite resin materials, it was concluded that the use of a precrack was somewhat impractical and that the use of sharply notched specimens provided precise data that are indicative of the K_{1C} of the materials.[24]

According to ASTM E399,[20] a state of plane strain is achieved when the sample thickness is $> 2.5\ (K_c/\sigma_y)$ (where σ_y = yield strength). For dentin, the minimum sample thicknesses for plane-strain conditions was estimated to be approximately 0.44 to 0.61 mm[19] or 1.4 mm,[22] depending on the σ_y value selected for use in the study calculations. Using the more conservative limit, a total of eight specimens did not meet the plane-strain K_{1C} specimen criteria. The difficulty in achieving the minimum thickness is due to the small size of available human dentin. It has been suggested that K_{1C} values measured with similar specimens are indicative of fracture resistance because the minimum thickness criterion is generally quite conservative and because the plastic or damage zone of fracture is well contained within the specimen boundaries.[21,22]

In this study, the mean K_{1C} of human dentin after direct 10% CP application for 8 weeks was approximately half of that of the control groups. This was similar to the relative degree of flexural strength reduction reported previously as a result of direct CP application to bovine dentin for an equivalent length of time.[13] It was postulated that the observed reduction in dentin structural integrity could be related to changes in the water content of the dentin[25] or to changes in either the organic or inorganic component of the dentin[13] occurring as result of bleach treatment. In one study, bleaching caused changes in the Ca/P ratio of dental hard tissues.[26,27] The amount of calcium loss, however, has been reported as small and possibly clinically insignificant.[28] In another study, bleaching decreased the organic content of the dentin and increased the relative inorganic content of the dentin and cementum.[29] A 30% decrease in the apparent K_{1C} of enamel after bleaching for a period of 12 hours with 10% CP was attributed to an alteration in the organic matrix of enamel.[12] The relative contributions of changes to either the organic (collagenous or noncollagenous proteins) or inorganic (mineral) components of dentin to the observed decrease in dentin K_{1C} after bleaching needs investigation.

The qualitative SEM examination did not provide sufficient evidence to conclude that there are significant differences in morphology between bleached and unbleached specimen fracture surfaces. However, areas of dentin demineralization were more evident near the surfaces that were directly exposed to bleach. The pH of the at-home 10% CP and 3% HP bleaches was slightly less than neutral, and the prolonged length of exposure to these slightly acidic materials could explain this observed dentin demineralization. Dentin demineralization was not observed to a greater degree in the lowest pH group (30% HP) than in the higher pH groups. However, the 30% HP group had the shortest exposure time. The finding of more demineralization occurring near the surface of direct bleach exposure and the

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observed lack of dentin demineralization in the indirectly exposed specimens suggest that the bleach affects the morphology of the exposed dentin surface most. Some penetration of the bleach through the dentinal tubule and lateral canals could contribute to the dentin demineralization that was observed deep to the exposed dentin surface. Further SEM investigations are necessary.

Until the specific cause for reduced dentin structural integrity as a result of bleaching is determined, therapies for the prevention of dentin weakening or for the recovery of dentin fracture resistance after bleaching are unknown. Topical fluoride treatment and specimen storage in artificial saliva for 2 weeks after bleach or control material application did not improve the flexural strength and modulus of bleached specimens over control specimens.[25] A better simulation of clinical conditions would have been to store the specimens in an artificial saliva or calcium phosphate solution rather than in DW during the intervals between the daily bleach treatment times. The authors are currently studying the effect of the simultaneous use of fluoride and bleach on the K_{1C} of dentin that is stored daily in an artificial saliva solution.

There was a lesser reduction in dentin K_{1C} after direct 3% HP application compared with the equivalent-concentration 10% CP group. Possible reasons for this lesser reduction in K_{1C} include a reduced stability of the HP compared with the CP over 6 hours application time, and a lack of urea, a known protein denaturing agent, in the 3% HP. The direct 30% HP group significantly reduced the dentin K_{1C} more than the 3% HP group despite a reduced overall treatment time. The higher active bleach concentration, as well as a possible increased permeability of the 30% HP that was used in liquid (as opposed to gel) form, may explain this. In clinical practice, gel formulations are generally used for in-office bleach applications.

The application of bleach to the enamel, as opposed to the dentin, is the common intended clinical practice. A "whole-tooth" model, with varying thickness of enamel and remaining pulpal tissue, was used to mimic the clinical situation in this first in vitro approximation of indirect bleaching effects on human dentin. The effects of the pulp and pulp degradation products on the dentin during bleach treatment were not investigated. In vivo, the pulp would provide outward fluid pressure within the dentinal tubules. This could potentially reduce the degree of bleach permeation into the dentin in the clinical situation. Dentinal fluid in vital teeth could act as a buffer as well.

When the bleach treatment was applied to dentin indirectly through intact enamel in this study, there were no significant differences in dentin K_{1C} between the bleach and control groups. This suggested that, although it has been shown that peroxide penetrates through the enamel and dentin through a capillary rise in enamel interprismatic spaces, convective mass transfer, or classic molecular diffusion properties[30–32] and forms measurable amounts of bleach within the tooth pulp, a variable thickness of the enamel and dentin could reduce the effects of CP and HP on dentin mechanical properties.

It would be of interest to determine if there is a minimum critical thickness of enamel beyond which deleterious effects of indirect bleach application start to occur. During the experiment, we erred on the side of caution and preferred to keep the bleach away from the thin cervical enamel in an effort to avoid bleach application to the root surface. If the bleach were applied to a thin enamel thickness or to root dentin, it is possible that there would have been a significant decrease in dentin K_{1C}. Further investigations are required to determine the barrier effects of both the enamel and dentin to bleach.

CONCLUSIONS

Direct in vitro application of 10% CP, 3% HP, and 30% HP bleach to

dentin for 8 weeks in a prolonged simulated at-home and in-office bleach treatment caused a significant decrease in dentin K_{1C}. A similar but *indirect* in vitro application of 10% CP, 3% HP, and 30% HP bleach to the dentin for 8 weeks caused no significant decrease in dentin K_{1C}. This K_{1C} study confirmed the results of a previous flexural strength and modulus study, which showed a significant decrease in dentin mechanical properties after prolonged *direct* tooth bleaching treatment, and evaluated the effects of tooth bleaching in a more clinically relevant manner by using an *indirect* application method as well as a *direct* application method. Further studies are necessary to characterize the mechanism of bleach permeation through enamel and dentin, as well as the actual mechanism of tooth bleaching.

DISCLOSURE AND ACKNOWLEDGMENTS

The authors do not have financial interests in the companies whose materials are mentioned in this article. We would like to thank the following: Monica McFarlane, Jian Wang, and Jennifer Chen. This study was supported by a Dentistry of Canada Fund research grant and a Canadian Institutes of Health Research Health Professional Summer Research Award. The bleach and placebo products were provided by Ultradent Products, Inc.

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