©Operative Dentistry, 2011, **36-2,** 126-132

Clinical Research

Oxalic Acid Under Adhesive Restorations as a Means to Reduce Dentin Sensitivity: A Four-Month Clinical Trial

C Barrientos • G Xaus • C Leighton
J Martin • VV Gordan • G Moncada

Clinical Relevance

Oxalic acid may offer a novel approach to improve the reduction of dentin postoperative sensitivity after placement of resin-bonded restorations.

SUMMARY

The aim of this double-blind randomized controlled clinical trial was to evaluate the reduction of dentin sensitivity using an oxalate-based compound, placed under adhesive restorations, during a four-month period. One hundred three preoperatively sensitive teeth, on 36 patients aged 25 to 66 years (mean, 40.3±7), were included in the study. Group A (experimental) was treated with oxalic acid (BisBlock) before resin-based composite (RBC) restorations (n=52), and group B (control) was treated with distilled water before RBC restorations (n=51). The first tooth in each patient was randomly assigned to group A, and the second tooth received group B. Clinical evaluation was made by a thermal/evaporation test with an air syringe and measurement by visual analog scale (VAS) at baseline and four months after treatment. The results showed sensitivity reduction during the evaluation period (expressed in VAS values): group A, 7.6 to 0.8; group B, 7.3 to 2.6. We concluded from this study that both treatments reduced dentin sensitivity during the

Claudia Barrientos, DDS, University of Chile, Operative Dentistry, Santiago, Chile

Gloria Xaus, DDS, University of Chile, Operative Dentistry, Santiago, Chile

Catherine Leighton, DDS, University of Chile, Operative Dentistry, Santiago, Chile

Javier Martin, DDS, University of Chile, Operative Dentistry, Santiago, Chile

Gustavo Moncada, DDS, University of Chile, Operative Dentistry, Santiago, Chile

*Valeria V. Gordan, DDS, MS, MSCI, University of Florida, Operative Dentistry, Gainesville, FL, USA

*Reprint request: University of Florida, Operative Dentistry, PO Box 100415, Gainesville, FL 32610, USA; e-mail: vgordan@dental.ufl.edu

DOI: 10.2341/09-364-C

evaluation period, with group A showing significantly less dentin sensitivity after four months ($p<0.05$).

INTRODUCTION

Dentin sensitivity could be defined as "pain arising from exposed dentin, typically in response to either a chemical, thermal, tactile or osmotic stimuli that cannot be explained by other dental defect or pathology."[1] There are other dental conditions such as caries, tooth and/or restoration fracture, and cracked-tooth syndrome that may produce the same short, sharp tooth pain. Thus, a careful dental history and precise clinical and radiographic examinations are necessary to establish a differential diagnosis of the clinical condition.[1,2]

The highly subjective nature of dentin sensitivity and the large variability among individual responses makes it difficult for an accurate assessment of this disorder.[3] Indeed, studies show extreme variations in the prevalence of this condition, ranging from 3% to 57% of dentin sensitivity in individuals with no previous dental diagnosis to as much as 72% to 98% of patients affected by periodontal conditions.[2]

According to Brännstrom, the mechanism of dentin sensitivity is considered to be related to a hydrodynamic theory in which fluid moves, within or through the dentinal tubules, at a rate that activates mechanoreceptor nerves in the dental pulp, causing pain.[4-6] The unit of measurement for the movement of fluids through the dentinal tubules is called dentin hydraulic conductance.[5] The occlusion of the dentinal tubules prevents fluid movement and therefore avoids dentinal sensitivity.[4-6] Agents such as oxalates, which promote the occlusion of the dentinal tubules, aim to cause the precipitation of crystals, thereby reducing the movement of the dentinal fluids. Ideally, these agents must be resistant to pH variations that take place in the oral environment, such as variations in diet and saliva quality and quantity.[7] Dentin desensitizers, based on oxalates, effectively reduce the dentin hydraulic conductance[6,8] by forming calcium oxalate crystals that precipitate over the dentin surface, occluding dentinal tubules. These desensitizers are available in gels or solutions that contain a low concentration of oxalic acid at low pH (Table 1).[8-10]

Gillam and others[11] evaluated *in vivo* the effects of ferric oxalate solution on the reduction of dentin sensitivity during a four-week time period. They reported a significant reduction in sensitivity five minutes after the solution was applied. However, the dentin sensitivity came back to its baseline level four weeks later. Another study demonstrated that the application of potassium oxalate has a relatively low life span for occlusion of dentinal tubules, as a significant loss of the oxalate precipitate was observed only one week after its application.[12]

Different authors have associated oxalate-based desensitizing agents and adhesive restorations, in *in vitro* studies, with improving the long-term occlusion of the dentinal tubules, and therefore reduction of dentin sensitivity, in teeth with cervical loss of tissue.[7,8,11-13] This treatment combines the occlusion of dentin tubules with the oxalate-based compounds and the use of an adhesive system.[13]

The aim of this randomized controlled clinical trial was to determine the performance of oxalic acid as a desensitizer when used under adhesive restorations during a four-month period. The hypothesis to be tested was that oxalic acid, placed under resin-based composite (RBC) restorations in cervical lesions, reduces dentin sensitivity compared with resin-only restoration.

METHODS AND MATERIALS

This double-blind randomized controlled clinical trial recruited 36 patients, aged 25 to 66 years (mean, 40.3±7 years), with 103 sensitive teeth. Dentin sensitivity was defined as having a minimum score of 1.5 on a visual analog scale (VAS), in response to air being applied directly to exposed dentin using an air syringe.

The sample size was determined *a priori* using G*Power 3,[14] with a population effect size of 0.4, a significance level α of 0.05, and a power (1-β) of 0.8 (Figure 1).

Inclusion criteria were as follows:

a. Patients older than 18 years having at least two sensitive teeth.
b. Teeth that have evidence of buccal cervical loss of tissue by abrasion or erosion, with the indication of resin-based restoration
c. Teeth without cavities or defective restorations in the buccal hypersensitive area

Patients presenting any of the following conditions were excluded from the study:

a. Chronic inflammatory systemic diseases
b. Chronic pain
c. Pregnancy

Table 1: *Examples of Oxalate-Based Dentin Desensitizers*

Product	Manufacturer	Composition	Concentration	pH
Protect Drops	John O Butler Co, Chicago IL, USA	Monohydrogen monopotassium oxalate solution	3%	NA
Oxagel	Art Dent, Sao Paulo, Brazil	Oxalic acid gel	3%	4
MS Coat	Sun Medical Co Ltd, Shiga, Japan	Oxalic acid gel	2%	1 approx
Bisblock	Bisco Inc, Schaumburg, IL, USA	Oxalic acid	<5%	1,5-1,8

d. Recent periodontal surgery, orthodontic treatment, or desensitizing treatments within the past three months

All patients signed the informed consent approved by the Ethics Committee (Dental School, Chile University).

At baseline, a clinical evaluation was made by thermal/evaporation test with an air syringe. The test was carried out using an air syringe with direct air pressure for one second (Siemens, Sirona, at 60 psi, from 19°C to 24°C and with an air flow of about 2 mm diameter) applied perpendicularly, 1 cm from the tooth's surface.[11,15–17] Dentin sensitivity was scored using a VAS. Patients quantified responses to the air stimulus by placing a mark on a 100-mm-length line anchored by word descriptors at each end: *no pain* at the left end and *very severe pain* at the right end. The VAS score was determined by measuring in millimeters from the left-hand end of the line to the point indicated by the patient.[18,19] All assessments were done by the same evaluator (G.X.).

Patients were recruited continuously over the course of five months, the period at which the desired sample size was reached. Once teeth were evaluated for sensitivity, they were assigned to one of the groups using a coin-toss method to randomly assign one tooth to either the experimental or control group. The next tooth was automatically assigned to the other group, and if a third tooth was involved, the coin was used again (Pocock Method).[18,20]

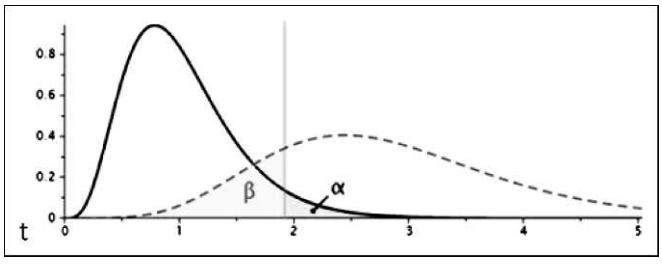

Figure 1. *Distribution-based approach of the two-way analysis of variance test, with effect size of 0.4, α error probability of 0.05, and power (1-β error probability) of 0.80 (G*Power).*

- **Group A:** Experimental group, n=52 teeth (13 molars, 39 premolars) treated with BisBlock (Bisco Inc, 1100 W Irving Park Rd, Schaumburg, IL 60193, USA)
- **Group B:** Control group, n=51 teeth (15 molars, 36 premolars) treated with distilled water

Each patient participated in both groups (experimental and control) with at least one tooth in each category, so both groups were composed of patients of identical age and gender: 24 women (66.66%) and 12 men (33.33%).

The exposed dentin of the teeth of both groups was etched with Uni-Etch (Bisco Inc) 32% orthophosphoric acid for 15 seconds, rinsed with water, and air dried to remove the excess water while retaining slight moisture on the surface. Teeth in group A were treated with BisBlock, over the dentin surface, for one minute and then rinsed with water for 15 seconds. Teeth in group B were treated with distilled water, over the dentin surface, for one minute and then rinsed with water for 15 seconds. To ensure the double-blind aspect of the trial, a different operator applied the experimental/control treatment prior to the adhesive/restoration placement. The dentin surface was left slightly moist before applying two layers of one-step dentin adhesive (biphenyl dimethacrylate >10%, hydroxyethyl methacrylate >10%, acetone >40%)[21] according to the manufacturer's instructions (Bisco Inc). After the adhesive application, the surface was polymerized for 15 seconds. The area was then restored with Aelite Composite (Bisco Inc) by applying the material in two small increments. The material was polymerized after each application for 40 seconds. During all procedures, moisture control was accomplished by using cotton

roll isolation, gingival cord, and low- and high-power suction. All treatments, other than the desensitizing step, were done by the same operator (C.B.).

Four months after treatment, a visual clinical examination of the restorations confirmed that they remained intact during the period of the study, and another evaluation using the thermal/evaporation test and the original rater (G.X.) was performed.

Two-way analysis of variance (ANOVA) test was applied with dentin sensitivity as the dependant variable and treatments and time as factors (SPSS 14.0, Chicago, IL, USA).

RESULTS

Mean VAS scores, standard deviation (SD), and comparison between the initial and final VAS score of each group (represented as mean sensitivity reduction) for dentin sensitivity at baseline and four months after treatment are presented in Table 2 and Figure 2.

Sensitivity reduction for each sample (each tooth) was obtained using the following formula:

$$(VAS\,at\,Baseline - VAS\,four\,months\,after\,treatment) \div (VAS\,at\,Baseline) \times 100$$

Mean sensitivity reduction and SD were obtained by averaging the data obtained by each tooth.

At baseline, both groups presented similar scores for sensitivity (p=0.289).

Four months after treatment, group A showed an 88.99% ± 23.42% reduction in dentin sensitivity, whereas the dentin sensitivity reduction in group B was 61.01% ± 35.71%.

Two-way ANOVA showed significant sensitivity reduction associated with the following variables: time (p=0.0001), oxalate application (p=0.003), and when the two variables were combined (p=0.0001; Table 3).

DISCUSSION

In this clinical study, both treatments showed reduction of dentin sensitivity as reported by participating patients after four months. However, the experimental group showed a significantly higher reduction in dentin sensitivity when compared with the control group over a four-month period. These results are in agreement with those obtained by Prati and others[22] in a four-week clinical evaluation study. In that study, it was noted that an oxalate-based product applied over sensitive teeth provided a reduction in dentin sensitivity in the range of 19% to 68%. Another study showed dentin permeability reduction in the range of 65% to 95% with ferric oxalate solutions,[23] and an *in vitro* study showed a significant reduction of the dentin hydraulic conductance with the use of potassium oxalate.[13] Although several of these studies reported reduction of dentin sensitivity, long-term results and permanent tooth desensitization remain a clinical problem. Later studies demonstrated that the tubular occlusion achieved by oxalate compounds lasted only a short period of time, and it was surmised that this was probably due to its solubility in the oral environment.[12]

Gillam and others,[24] in a scanning electron microscopy study, observed that four oxalate solutions (aluminium oxalate, potassium oxalate, ferric oxalate e, and oxalic acid) applied onto dentin discs occluded the dentin tubules to different degrees. However, radiographic diffraction techniques were unable to identify oxalate salts within the tubules. It was suggested that some oxalates appear to occlude the dentinal tubules through the formation of crystals. It is possible that the oxalate's components are important in the initial formation of those crystals.[24] Two possible mechanisms explaining the action of potassium oxalate have been proposed.[25] First, the formation of calcium oxalate crystals blocks dentinal tubules and prevents dentin fluid loss. This mechanism would be responsible for the long-term desensitizing action. The second mecha-

Table 2: *Dentin Sensitivity Expressed in VAS Score and SD at Baseline and Four Months After Treatment, Separated by Group*

	N	VAS Score and SD for Sensitivity at Baseline	VAS Score and SD for Sensitivity 4 Mo After Treatment	Mean of Sensitivity Reduction and SD Between Both Periods
Group A	52	7.66 ± 1.58	0.82 ± 1.60	88.99% ± 23.42%
Group B	51	7.32 ± 1.63	2.65 ± 2.19	61.01% ± 35.71%

Abbreviations: SD, standard deviation; VAS, visual analog scale.

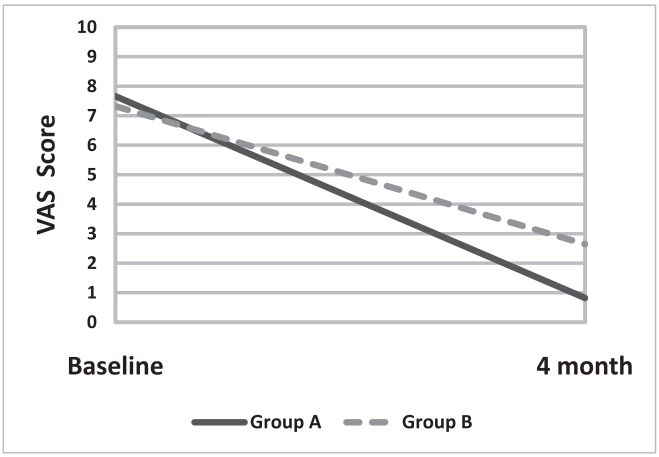

Figure 2. *Sensitivity response to thermal evaporative stimulus.*

nism suggests that high levels of potassium would increase the extracellular K^+ concentration around the dentinal terminal nerves, causing depolarization and causing them to become less excitable, which would explain the temporary reduction in tooth sensitivity.[25]

A previous *in vitro* study evaluated desensitizing agents and dentin bonding agents with and without placement of RBCs. That study concluded that placement of primers without etching reduced permeability more than any other treatment modality, indicating that etching a sensitive dentin area may be appropriate only if an RBC is to be placed in the area.[26]

Some studies have reported that oxalate-based products react with the calcium ions available in the dentin surface and dentin fluid, transforming the labile cover in an acid-resistant structure by replacement of the smear layer with a layer of calcium oxalate crystals.[7,24,27] The smear layer provides a great source of calcium ions that would be available in the formation of the crystals, and an *in vitro* study reported a reduction in the numbers of crystals that were formed on surfaces treated with oxalate when the smear layer had been removed with chemical agents. Therefore, without the smear layer, the number of calcium ions available is decreased and so is the precipitation of crystals. The low pH of the oxalate solution causes the dissolution of the smear layer. This phenomenon gradually increases the pH because of the dissolution of the hydroxyapatite and other dentin components, which neutralize the hydrogen ions, causing the precipitation of the calcium oxalate and ferric phosphate. Although the oxalate crystals resist the acid attack, they do not completely block the tubules and therefore produce only a replacement for the smear layer.[23]

The reduction of dentin permeability is obtained by a superficial tubular occlusion, which does not

Table 3: *Two-Way ANOVA Summary Table*[a]

Source	Type III Sum of Squares	*df*	Mean Square	*F*	Sig
Corrected model	1800.819[b]	3	600.273	191.786	0.0001
Intercept	4379.207	1	4379.207	1399.151	0.0001
Treatments	28.422	1	28.422	9.081	0.003
Time	1705.831	1	1705.831	545.011	0.0001
Treatments*Time	60.177	1	60.177	19.226	0.0001
Error	632.240	202	3.130		
Total	6805.830	206			
Corrected total	2433.059	205			

Abbreviation: ANOVA, analysis of variance.
[a] *Test of between-subjects effects dependent variable: dentin sensitivity.*
[b] R^2=*0.740 (adjusted* R^2=*0.736).*

interfere with the adhesive technique, as demonstrated by previous studies.[7,8,11-13] The use of dentin adhesives over oxalate salts provides better tubular occlusion of the exposed dentin without affecting the adhesive resistance of the restoration.[7,8,11-13]

One limitation of the study is the well-known existence of a placebo response by a subject knowingly participating in a sensitivity study.[28] We hope that with the double-blind randomized design of the study and the use of a control group, the placebo effect over the study was diminished.

CONCLUSIONS

The results of this randomized clinical trial show that the application of oxalate acid under RBC restorations significantly reduces dentin sensitivity, compared with resin-only restorations, over a period of four months. However, further studies are needed to evaluate the long-term stability of this technique as a permanent treatment for dentin sensitivity.

(Accepted 8 November 2010)

REFERENCES

1. Addy M (1990) Etiology and clinical implications of dentine hypersensitivity *Dental Clinics of North America* **34(3)** 503-514.
2. Addy M (2002) Dentine hypersensitivity: New perspectives on an old problem *International Dental Journal* **52(5)** 367-375.
3. Gillam DG, Aris A, Bulman JS, Newman HN & Ley F (2002) Dentine hypersensitivity in subjects recruited for clinical trials: Clinical evaluation, prevalence and intraoral distribution *Journal of Oral Rehabilitation* **29(3)** 226-231.
4. Brannstrom M (1986) The hydrodynamic theory of dentinal pain: Sensation in preparations, caries, and the dentinal crack syndrome *Journal of Endodontics* **12(10)** 453-457.
5. Pashley DH (1990) Mechanisms of dentin sensitivity *Dental Clinics of North America* **34(3)** 449-473.
6. Pashley DH (1986) Dentin permeability, dentin sensitivity, and treatment through tubule occlusion *Journal of Endodontics* **12(10)** 465-474.
7. Pereira JC, Segala AD & Gillam DG (2005) Effect of desensitizing agents on the hydraulic conductance of human dentin subjected to different surface pre-treatments—An in vitro study *Dental Materials* **21(2)** 129-138.
8. Tay FR, Pashley DH, Mak YF, Carvalho RM, Lai SC & Suh BI (2003) Integrating oxalate desensitizers with total-etch two-step adhesive *Journal of Dental Research* **82(9)** 703-707.
9. Santiago SL, Pereira JC & Martineli ACBF (2006) Effect of commercially available and experimental potassium oxalate-based dentin desensitizing agents in dentin permeability: Influence of time and filtration system *Brazilian Dental Journal* **17(4)** 300-305.
10. Bisco Inc. (2008) BisBlock material safety data sheet. Retrieved online April 2010 from: http://www.bisco.com/instructions/BisBlock.asp
11. Gillam DG, Newman HN, Davies EH, Bulman JS, Troullos ES & Curro FA (2004) Clinical evaluation of ferric oxalate in relieving dentine hypersensitivity *Journal of Oral Rehabilitation* **31(3)** 245-250.
12. Kerns DG, Scheidt MJ, Pashley DH, Horner JA, Strong SL & Van Dyke TE (1991) Dentinal tubule occlusion and root hypersensitivity *Journal of Periodontology* **62(7)** 421-428.
13. Pashley DH, Carvalho RM, Pereira JC, Villanueva R & Tay FR (2001) The use of oxalate to reduce dentin permeability under adhesive restorations *American Journal of Dentistry* **14(2)** 89-94.
14. Faul F, Erdfelder E, Lang AG, Buchner A. (2007) G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences *Behavior Research Methods* **39(2)** 175-191.
15. Ide M, Morel AD, Wilson RF & Ashley FP (1998) The role of a dentine-bonding agent in reducing cervical dentine sensitivity *Journal of Clinical Periodontology* **25(4)** 286-290.
16. Gillam DG, Coventry JF, Manning RH, Newman HN & Bulman JS (1997) Comparison of two desensitizing agents for the treatment of cervical dentine sensitivity *Endodontics & Dental Traumatology* **13(1)** 36-39.
17. Morris MF, Davis RD & Richardson BW (1999) Clinical efficacy of two dentin desensitizing agents *American Journal of Dentistry* **12(2)** 72-76.
18. Holland GR, Narhi MN, Addy M, Gangarosa L & Orchardson R (1997) Guidelines for the design and conduct of clinical trials on dentine hypersensitivity *Journal of Clinical Periodontology* **24(11)** 808-813.
19. Gould D, Kelly D, Goldstone L & Gammon J (2001) Examining the validity of pressure ulcer risk assessment scales: Developing and using illustrated patient simulations to collect the data *Journal of Clinical Nursing* **10(5)** 697-706.
20. Pocock SJ (1983) *Clinical Trials: A Practical Approach* Wiley, Chichester [West Sussex]; New York.
21. Bisco Inc. (2008) ONE-STEP material safety data sheet. Retrieved online April 2010 from: http://www.bisco.com/instructions/One-Step.asp
22. Prati C, Cervellati F, Sanasi V & Montebugnoli L (2001) Treatment of cervical dentin hypersensitivity with resin adhesives: 4-week evaluation *American Journal of Dentistry* **14(6)** 378-382.
23. Dragolich WE, Pashley DH, Brennan WA, O'Neal RB, Horner JA & Van Dyke TE (1993) An *in vitro* study of dentinal tubule occlusion by ferric oxalate *Journal of Periodontology* **64(11)** 1045-1051.
24. Gillam DG, Mordan NJ, Sinodinou AD, Tang JY, Knowles JC & Gibson IR (2001) The effects of oxalate-containing products on the exposed dentine surface: An SEM

investigation *Journal of Oral Rehabilitation* **28(11)** 1037-1044.

25. Muzzin KB & Johnson R (1989) Effects of potassium oxalate on dentin hypersensitivity *in vivo Journal of Periodontology* **60(3)** 151-158.
26. Jain P, Reinhardt JW & Krell KV (2000) Effect of dentin desensitizers and dentin bonding agents on dentin permeability *American Journal of Dentistry* **13(1)** 21-27.
27. Pashley DH & Galloway SE (1985) The effects of oxalate treatment on the smear layer of ground surfaces of human dentine *Archives of Oral Biology* **30(10)** 731-737.
28. Yates RJ, Newcombe RG & Addy M (2004) Dentine hypersensitivity: A randomised, double-blind placebo-controlled study of the efficacy of a fluoride-sensitive teeth mouthrinse *Journal of Clinical Periodontology* **31(10)** 885-889.