Research Article

# Occlusion of dentin tubules by desensitizing agents

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ABSTRACT: ***Purpose***: To evaluate the occluding effect of five desensitizing agents on human dentin tubules. ***Methods***: 30 buccal and lingual surfaces were prepared from 15 extracted intact third molars. Each surface was polished with aluminum oxide abrasive papers to remove enamel and to expose the underlying dentin in cervical area. The flat dentin surfaces were treated with 0.5 M EDTA for 2 minutes to expose dentin tubule orifices. The samples were randomly divided into six groups: AS - immersed in artificial saliva at 37°C for 2 weeks (control); OX – Oxagel (monopotassium oxalate), DU – Duraphat (sodium fluoride), DE – Desensibilize (strontium chloride), OD – Odahcam (acidulated phosphate fluoride) and SE – Sensodyne (strontium chloride + calcium carbonate abrasive). Dentin desensitizers were applied during 2 weeks and after each application the samples were kept in artificial saliva at 37°C. The samples were prepared according to the scanning electron microscope procedures and were examined at x2000 magnification. ***Results***: The results were expressed in percent (%) of tubule occlusion and analyzed by ANOVA and Duncan's multiple range test ($P < 0.05$): AS- $45.41 \pm 11.65^{a}$; OX- $42.65 \pm 11.79^{a}$; DU- $47.25 \pm 8.59^{ab}$; DE- $49.36 \pm 18.27^{ab}$; OD- $64.43 \pm 15.55^{b}$ and SE- $65.44 \pm 10.93^{b}$. Results suggest that the dentin surfaces treated with OD and SE showed higher tubule occlusion when compared to AS and OX, but were not different compared to DU and DE treatments. (*Am J Dent* 2004;17:368-372).

CLINICAL SIGNIFICANCE: Open dentin tubules exposed to the oral environment, which are implicated in the etiology of dentin hypersensitivity, can be treated by desensitizing agents with varying degrees of tubule occlusion.

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## Introduction

Dentin hypersensitivity is a common painful condition in response to intraoral stimuli.[1-4] It is estimated that 8-30% of the adult dentate population experiences occasional pain due to dentin hypersensitivity.[5-9] Morphologic and physiologic studies suggest that dentin hypersensitivity results from exposure of dentin surfaces to the oral environment.[10-14] The differences between sensitive and non- sensitive exposed dentin seem to be related to the diameter, patency and number of open dentin tubules.[9,11,12,15-17]

Dentin can be exposed when the enamel covering the crown and cementum are lost. Removal of enamel may result from abfraction, abrasion or erosion[18,19] and denudation of the root surface can occur as a result of gingival recession or periodontal therapy.[20] Interstitial fluid of movement within the dentin tubules is the basis for the transmission of sensations according to the hydrodynamic theory.[21-23] The dentin fluid in the tubules may be set in motion by external stimuli such as thermal, mechanical or chemical, resulting in activation of the nerve fibers and causing pain.[10]

A method of interrupting this transmission of pain stimuli and reducing the flow of interstitial fluid is to block the dentin tubules.[3,24,25] Tubules can be sealed on the dentin surface, occluded within the orifices of the tubules or in the subsurface dentin within the tubules.[4] The treatment of dentin hypersensitivity comprises a variety of regimens and desensitizing agents, including dentist-applied in-office treatments and patient self-applied, desensitizing dentifrices.[2,16,26]

This study evaluated, with scanning electron microscopy, the tubule occlusion on the dentin surface by desensitizing agents: fluorides, potassium oxalate and strontium chloride plus potassium nitrate.

## Materials and Methods

The specimens were prepared from 15 extracted sound human third molars stored in 10% formalin (pH 7.0) at room temperature no longer than 1 month prior to their use.

*Dentin specimen preparation* - The teeth were sectioned in mesio-distal direction using a water-cooled diamond saw[a] to obtain 30 buccal and lingual surfaces. Each surface was ground (600-grit) flat on a polishing machine[b] to remove enamel and to expose the underlying dentin in the cervical area. The exposed dentin surfaces were polished with wet 1000- and 1200-grit aluminum oxide abrasive paper,[c] simulating the dentin hypersensitivity in cervical regions. All dentin surfaces were treated with 0.5 M EDTA (pH 7.4) for 2 minutes and rinsed with air-water for 30 seconds to remove smear layer and smear plugs, and expose dentin tubule orifices (Fig. 1).[14,27,28]

*Sample assignment and treatment modalities* - The samples were randomly assigned to six groups, each consisting of five samples:

*Group 1* - The samples were kept in artificial saliva[29] (1 g sodium carboxymethylcellulose, 4.3 g xylitol, 0.1 g potassium chloride, 0.1 g sodium chloride, 0.02 mg sodium fluoride, 5 mg magnesium chloride, 5 mg calcium chloride, 40 mg potassium phosphate, 1 mg potassium thiocyanate and 100 g distilled deionized water) at 37°C for 14 days as controls.

*Group 2* - Oxagel[d] was applied for 2 minutes on every fourth day (Days 1, 5, 9 and 13). After the last treatment, the samples were kept in artificial saliva until Day 14 to be evaluated.

*Group 3* - Duraphat[e] was applied for 2 hours on every fourth day (Days 1, 5, 9 and 13). After the last treatment, the samples

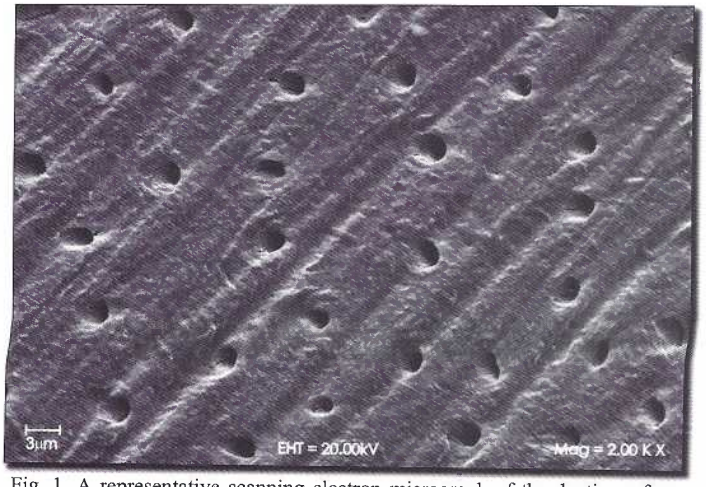


Fig. 1. A representative scanning electron micrograph of the dentin surface treated with EDTA to remove the smear layer.

Table 1. Components of the dentin desensitizers used in this study.

| Material | Type | Composition |
|---|---|---|
| Oxagel | Gel | 3% monopotassium oxalate |
| Duraphat | Varnish | 2.26% F (sodium fluoride in alcohol solution of resins) |
| Desensibilize | Solution | Strontium chloride + potassium nitrate |
| Odahcam | Gel | 1.23 % F (acidulated phosphate fluoride) |
| Sensodyne | Dentifrice | 10% strontium chloride + calcium carbonate abrasive |

were kept in artificial saliva until Day 14 to be evaluated.

*Group 4* - Desensibilize[f] was applied 10 minutes on every fourth day (Days 1, 5, 9 and 13). After the last treatment, the samples were kept in artificial saliva until Day 14 to be evaluated.

*Group 5* - Odahcam[g] was applied every fourth day for 4 minutes on every fourth day (Days 1, 5, 9 and 13). After the last treatment, the samples were kept in artificial saliva until Day 14 to be evaluated.

*Group 6* - Sensodyne[h] was brushed with a soft toothbrush[i] for 1 minute twice a day for 14 days.

After each treatment, the samples were incubated in artificial saliva in a shaker[j] at 37°C, which was changed every day. While Groups 2 to 5 were submitted to the treatments described above, the samples remained stored at 100% relative humidity, followed by 15-second washing with distilled deionized water and artificial saliva storage. In Group 6, the samples were brushed for 1 minute in 20 mL of 33% (w/v) dentifrice/water twice a day for 2 weeks (the only group that was submitted to daily application), washed for 15 seconds and kept in artificial saliva. A brushing machine[k] was used to brush dentin surfaces at 125 strokes/minute and a 200-g weight was placed on the heads of each toothbrush. The composition of five commercially available agents for treatment of hypersensitive dentin is shown in Table 1.

*SEM observations and measurements* - The samples were prepared according to the scanning electron microscope procedures and examined at x2000 magnification.[l] Photomicrographs were taken from each dentin surface examined and, from these, the total number of dentin tubules presented compared to the percent of tubules that were occluded per area was determined. A single investigator who was unaware of the specimen treatments performed the measurements. The tubules density was calculated according to the formula used by Garberoglio & Brännström.[30] The results were expressed as the percentage of tubule occlusion per area and analyzed by one-way ANOVA and Duncan's multiple range tests at 5% level of significance.


Fig. 2. SEM micrograph of the dentin surface after storage in artificial saliva. Note reduction of the tubule lumen (arrow).

Table 2. Means of tubule density (number of dentin tubules per square millimeter) and percentage of occluded dentin tubules.

| Treatment | Tubule density | Occluded tubules (%) |
|---|---|---|
| Artificial saliva | 19459 ± 1523 a | 45.4 ± 11.7 a |
| Oxagel | 17978 ± 2214 a | 42.7 ± 11.8 a |
| Duraphat | 19332 ± 2770 a | 47.3 ± 8.6 ab |
| Desensibilize | 16433 ± 782 a | 49.4 ± 18.3 ab |
| Odahcam | 16823 ± 3472 a | 64.4 ± 15.6 b |
| Sensodyne | 15206 ± 3166 a | 65.4 ± 10.9 b |

Groups in any columm identified with different lower case letters are significantly different.

## Results

The means of the tubule density (number of tubules per area) and the means of the percentage of tubule occlusion for the six groups are summarized in Table 2. One-way ANOVA revealed no statistically significant difference in total tubule density ($P > 0.05$), but a significant difference the percentage of tubule occlusion ($P < 0.05$). Duncan's multiple range test showed that the percentage of tubule occlusion produced by Odahcam and Sensodyne treatments were higher than those found in the Oxagel and artificial saliva groups ($P < 0.05$). Odahcam, Sensodyne, Duraphat and Desensibilize exhibit similar percentage of tubule occlusion ($P > 0.05$). The samples treated with artificial saliva, Oxagel, Duraphat and Desensibilize Dentin Desensitizers showed no differences in percentage of tubule occlusion ($P > 0.05$).

No values were given for the number of tubules open on EDTA-treated dentin that had not been incubated in artificial saliva for 2 weeks, because it was just used to illustrate EDTA-treated dentin surface (Fig. 1). Figures 2 - 4 show the SEM appearance of the dentin surfaces after various treatments. Saliva immersion resulted in 45.41 ± 11.65% of dentin tubules occluded, moreover, the tubule diameters in Fig. 2 are smaller than Fig. 1. SEM photomicrographs of acidulated phosphate fluoride and toothbrushing-treated dentin (Figs. 3, 4) revealed the formation of precipitates that occluded dentin tubules. A granular morphology on dentin

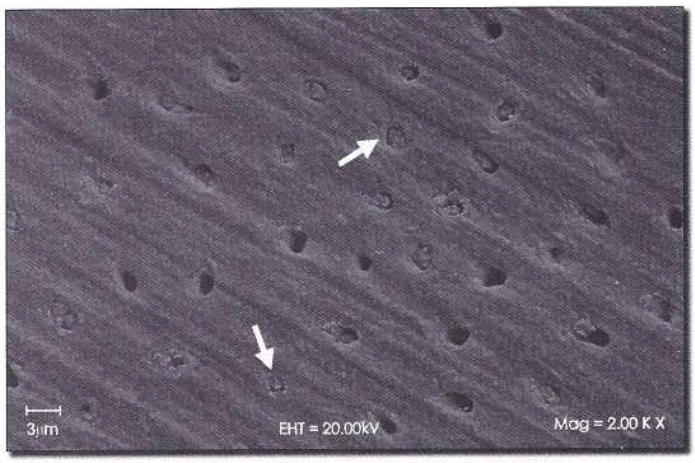


Fig. 3. SEM micrograph of the dentin surface treated with Odahcam (acidulated phosphate fluoride). The arrow indicates a tubule occluded by a precipitate. This was probably due to the formation $CaF_2$.

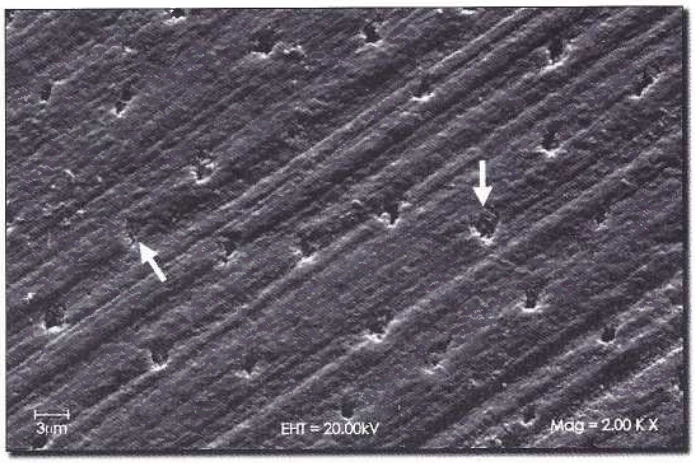


Fig. 4. SEM micrograph of the dentin surface after toothbrushing with Sensodyne dentifrice. Note toothbrush scratches and occluded tubules due to the abrasive action of the dentifrice (arrow).

treated with acidulated phosphate fluoride may indicate that it is somehow associated with the $CaF_2$-like formation (Fig. 3). Some particles that could block dentin tubules during toothbrushing can be noted in Fig. 4.

## Discussion

Studies[30-33] report that the range of tubule densities in superficial dentin varies from 17,000 to 20,000 tubules/mm$^2$, which was similar to the values obtained on the simulation of cervical hypersensitivity in this study. The scanning electron microscopic examination of human dentin at the cervical areas found no statistical difference in the means of tubule density among the groups. Therefore, dentin surfaces investigated from all groups started with similar tubule densities.

Sensodyne dentifrice containing 10% strontium chloride plus abrasives, and Odahcam, 1.23% F (pH 3.5), showed more effectiveness on superficial occlusion of dentin tubules than the control samples maintained in artificial saliva or those treated with Oxagel, which contains 3% monopotassium oxalate. Duraphat varnish with 2.26% F, and Desensibilize, which contains strontium chloride and potassium nitrate in solution, were as effective as Sensodyne and Odahcam.

The samples stored in artificial saliva showed 45.41 ± 11.65% tubule occlusion. Artificial saliva is supersaturated with respect to hydroxyapatite, and, thus, precipitation of calcium phosphate would seem to have occurred on the dentin surface, leading to the occlusion of dentin tubules.[34] Natural occlusion of exposed dentin tubules on the surfaces of hypersensitive dentin can occur through formation of calculus, intratubular crystals from salivary mineral and dentin fluid, progressive formation of peritubular dentin or intratubular deposition of collagen or plasma proteins.[12-14,25,34]

Dentin treated with potassium oxalate revealed crystals of calcium oxalate in dentin tubules. The oxalate from potassium oxalate reacts with the ionized calcium in the dentin tubule and forms insoluble calcium oxalate crystals. This reaction of soluble oxalate salts with dentin seems to occur within seconds[35,36] and is limited due to the concentration of the ionized calcium.[1] The crystals block the dentin tubule and prevent fluid flow in the tubule, providing reductions in dentin permeability.[35,37] However, studies demonstrated that applications of potassium oxalate are relatively short-lived procedures for occluding dentin tubules[27] due to the dissolution of calcium oxalate in the artificial saliva.[34] The dissolution of calcium oxalate crystals over the 2-week period may explain the low percentage of occluded tubules with Oxagel in this study. However, the tubules may have remained occluded with intratubular calcium oxalate crystals below the surfaces.[35,39]

Reports of the action of strontium chloride demonstrated that strontium ions penetrate deep into dentin.[39,40] Strontium chloride is a desensitizing agent that has been used in dentifrices to reduce dentin hypersensitivity; indeed, clinical evaluations indicate effectiveness in controlling hypersensitivity.[40-43] However, topical application of 10% strontium chloride solution to the dentin surface has shown little effect on reducing hypersensitivity[34] or the hydraulic conductance of dentin.[35]

Two dentin desensitizers containing strontium chloride, Desensibilize and Sensodyne, were investigated in the present study. Desensibilize was topically applied and produced occlusion of 49.36 ± 18.27% of the total number of dentin tubules. Potassium nitrate, the other component from Desensibilize, was unable to close dentin tubules, thus, its desensitizing role was related to depolarizing the pulpal sensory nerves causing interruption of pain transmissions.[44] Although Desensibilize produced a lower percentage of tubule occlusion than Sensodyne toothpaste, there was no statistically significant difference between them. However, Sensodyne toothpaste produced a higher percentage of tubule occlusion than Oxagel or control specimens stored in artificial saliva. This additional blockage may occur by directly occluding the tubules with calcium carbonate abrasive or indirectly by the formation of a smear layer during toothbrushing.[35,45,46] Thus, the desensitizing effect of the strontium chloride dentifrice can also be attributed to the abrasive fillers beyond the proposed active agent.

Dental professionals can prescribe or apply fluoride in some forms as a way to manage hypersensitive dentin.[26,47-49] Topical fluoride applications have been used successfully for the treatment of hypersensitive dentin.[50-52] In this study, topical fluoride was used in the form of acidulated phosphate fluoride gel and as a varnish. Duraphat is an alcoholic solution of natural resins in which 1 ml of the varnish contains 50 mg sodium fluoride. The mechanism of action from Duraphat in

desensitizing dentin probably rests in its ability to form calcium fluoride and to some extent fluorapatite, in the outer surface of dentin, thus, blocking transmission of stimuli to the pulp.[49] The effectiveness of sodium fluoride varnish has been demonstrated when it was used in conjunction with a dentifrice[49] or Nd:YAG laser irradiation.[53] Duraphat used alone in Group 3 had no effect on superficial occlusion of dentin tubules related to Group 1.

Acidulated sodium fluoride reduced the rate of fluid flow through dentin *in vitro* when compared to neutral sodium fluoride.[35] The 0.1 M phosphoric acid that is present in the composition of Odahcam probably etched the dentin, raising the ionized calcium concentration to a level where it exceeded the solubility product constant for calcium fluoride, which then precipitated on and in the tubules.[34,52] Investigators have reported precipitations similar to Fig. 3, which were observed in scanning electron microscope micrographs physically blocking dentin tubules.[52]

This *in vitro* experiment screened a number of products used to treat dentin hypersensitivity. The results suggest that blockage and reduction of the tubule lumen can occur as a result of deposition of fluoride, insoluble salts or abrasive action of the dentifrice.[2,3,54,55]

a. Buehler Ltd., Lake Bluff, IL, USA.
b. Arotec S/A Ind. e Com., Cotia, SP, Brazil.
c. 3M do Brasil, Sumaré, SP, Brazil.
d. Art-Dent Ind. e Com. de Produtos Odontológicos Ltda., Araraquara, SP, Brazil.
e. Rhône-Poulenc Rorer GmbH, Cologne, Germany.
f. FGM Produtos Odontológicos, Joinville, SC, Brazil.
g. Herpo Produtos Dentários Ltda., Rio de Janeiro, RJ, Brazil.
h. Stafford-Miller Ind. Ltda., Rio de Janeiro, RJ, Brazil.
i. Oral B – Gillette do Brasil Ltda., Manaus, AM, Brazil.
j. Tecnal Equip. Lab., Piracicaba, SP, Brazil.
k. Equilabor, Piracicaba, SP, Brazil.
l. Zeiss, Oberkochen, Germany.

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## References

1. Muzzin KB, Johnson R. Effects of potassium oxalate on dentin hypersensitivity *in vivo*. *J Periodontol* 1989; 60: 151-158.
2. Collaert B, Fischer C. Dentine hypersensitivity: A review. *Endod Dent Traumatol* 1991; 7: 145-152.
3. Addy M, West N. Etiology, mechanisms, and management of dentine hypersensitivity. *Periodontol* 1994; 34: 71-77.
4. Schüpbach P, Lutz F, Finger WJ. Closing of dentinal tubules by Gluma desensitizer. *Eur J Oral Sci* 1997; 105: 414-421.
5. Abel I. Study of hypersensitive teeth and a new therapeutic aid. *Oral Surg* 1958; 11: 491-495.
6. Jensen AL. Hypersensitivity controlled by iontophoresis: Double blind clinical investigation. *J Am Dent Assoc* 1964; 68: 217-225.
7. Krauser JT. Hypersensitive teeth. Part I: Etiology. *J Prosthet Dent* 1986; 56: 153-156.
8. Flynn J, Galloway R, Orchardson R. The incidence of "hypersensitive" teeth in the West of Scotland. *J Dent* 1985; 13: 230-236.
9. Addy M. Etilogy and clinical implications of dentine hypersensitivity. *Dent Clin North Am* 1990; 34: 503-514.
10. Berman LH. Dentinal sensation and hypersensitivity. A review of mechanisms and treatment alternatives. *J Periodontol* 1984; 56: 216-222.
11. Absi EG, Addy M, Adams D. Dentine hypersensitivity. A study of the patency of the dentinal tubules in sensitive and non-sensitive cervical dentine. *J Clin Periodontol* 1987; 14: 280-284.
12. Yoshiyama M, Masada J, Uchida A, Ishida H. Scanning electron microscopic characterization of sensitive *vs.* insensitive human radicular dentin. *J Dent Res* 1989; 68: 1498-1502.
13. Yoshiyama M, Noiri Y, Ozaki K, Uchida A, Ishikawa Y, Ishida H. Transmission electron microscopic characterization of hypersensitive human radicular dentin. *J Dent Res* 1990; 69: 1293-1297.
14. Cuenin MF, Scheidt MJ, O'Neal RB, Strong SL, Pashley DH, Horner JA, van Dyke TE. An *in vivo* study of dentin sensitivity: The relation of dentin sensitivity and the patency of dentin tubules. *J Periodontol* 1991; 62: 668-673.
15. Pashley DH. Mechanisms of dentin sensitivity. *Dent Clin North Am* 1990; 34: 449-473.
16. Trowbridge HO, Silver DR. A review of current approaches to in-office management of tooth hypersensitivity. *Dent Clin North Am* 1990; 34: 561-581.
17. Kuroiwa M, Kodaka T, Kuroiwa M, Masayuki A. Dentin hypersensitivity. Occlusion of dentinal tubules by brushing with and without an abrasive dentifrice. *J Periodontol* 1994; 65: 291-296.
18. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. *J Prosthet Dent* 1984; 52: 374-380.
19. Owens BM, Gallien GS. Noncarious dental "abfraction" lesions in an aging population. *Compend Contin Educ Dent* 1995; 16: 552-554.
20. Cox CF. Etiology and treatment of root hypersensitivity. *Am J Dent* 1994; 7: 266-270.
21. Brännström M, Lindén L, Johnson G. Movement of dentinal and pulpal fluid caused by clinical procedures. *J Dent Res* 1968; 47: 679-682.
22. Brännström M. Sensitivity of dentine. *Oral Surg Oral Med Oral Pathol* 1966; 21: 517-526.
23. Brännström M, Åstrom A. The hydrodynamics of the dentine; its possible relationship to dentinal pain. *Int Dent J* 1972; 22: 219-229.
24. Pashley DH, Livingston MJ, Reeder OW, Horner JA. Effects of the degree of tubule occlusion on the permeability of human dentine *in vitro*. *Arch Oral Biol* 1978; 23: 1127-1133.
25. Pashley DH. Dentin permeability, dentin sensitivity, and treatment through tubule occlusion. *J Endod* 1986; 12: 465-474.
26. Plagmann H, König J, Bernimoulin J, Rudhart AC, Deschner J. A clinical study comparing two high-fluoride dentifrices for the treatment of dentinal hypersensitivity. *Quintessense Int* 1997; 28: 403-408.
27. Kerns DG, Scheidt MJ, Pashley DH, Horner JA, Strong SL, van Dike TE. Dentinal tubule occlusion and root hypersensitivity. *J Periodontol* 1991; 62: 421-428.
28. Pashley DH, Ciucchi B, Sano H, Horner JA. Permeability of dentin to adhesive agents. *Quintessence Int* 1993; 24: 618-631.
29. Cavalli V, Reis AF, Giannini M, Ambrosano GMB. Bond strength to enamel relative to the elapsed time from bleaching with various concentrations of carbamide peroxide. *Oper Dent* 2001; 26: 597-602.
30. Garberoglio R, Brännström M. Scanning electron microscopic investigation of human dentinal tubules. *Arch Oral Biol* 1976; 21: 355-362.
31. Pashley DH, Okabe A, Parham P. The relationship between dentin microhardness and tubule density. *Endod Dent Traumatol* 1985; 1: 176-179.
32. Fosse G, Saele PK, Eide R. Numerical density and distributional pattern of dentin tubules. *Acta Odontol Scand* 1992; 50: 201-210.
33. Olsson S, Öilo G, Adamczak E. The structure of dentin surfaces exposed for bond strength measurements. *Scand J Dent Res* 1993; 101: 180-184.
34. Suge T, Ishikawa K, Kawasaki A, Yoshiyama M, Asaoka K, Ebisu S. Duration of dentinal tubule occlusion formed by calcium phosphate precipitation method: *In vitro* evaluation using synthetic saliva. *J Dent Res* 1995; 74: 1709-1714.
35. Greenhill JD, Pashley DH. The effects of desensitizing agents on the hydraulic conductance of human dentin *in vitro*. *J Dent Res* 1981; 60: 686-698.
36. Pashley DH, Galloway SE. The effects of oxalate treatment on the smear layer of ground surfaces of human dentine. *Arch Oral Biol* 1985; 30: 731-737.
37. Jain P, Reinhardt JW, Krell KV. Effect of dentin desensitizers and dentin bonding agents on dentin permeability. *Am J Dent* 2000; 13: 21-27.
38. Gillam DG, Khan N, Mordan NJ, Barber PM. Scanning electron microscopy (SEM) investigations of selected desensitizing agents in the dentine disc model. *Endod Dent Traumatol* 1999; 15: 198-204.
39. Penney DA, Karlsson UL. Fast desensitization of tooth roots by topically applied $SnF_2$ and $SrCl_2$ in dogs. *Arch Oral Biol* 1976; 21: 339-347.
40. Uchida A, Wakano Y, Fukuyama O, Miki T, Iwayama Y, Okada H. Controlled clinical evaluation of a 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. *J Periodontol* 1980; 51: 578-581.

41. Meffert RM, Hoskins SW. Effect of a strontium chloride dentifrice in relieving dental hypersensitivity. *J Periodontol* 1964; 35: 232-235.
42. Collins JF, Perkins L. Clinical evaluation of the effectiveness of three dentifrices in relieving dentin sensitivity. *J Periodontol* 1984; 55: 720-725.
43. Minkoff S, Axelrod S. Efficacy of strontium chloride in dental hypersensitivity. *J Periodontol* 1987; 58: 470-474.
44. Kim S. Hypersensitive teeth: Desensitization of pulpal sensory nerves. *J Endod* 1986; 12: 482-485.
45. Pashley DH, O'Meara JA, Kepler EE, Galloway SE, Thompson SM, Stewart FP. Dentin Permeability. Effects of desensitizing dentifrices *in vitro*. *J Periodontol* 1984; 55: 522-525.
46. Pearce NX, Addy M, Newcombe RG. Dentine hypersensitivity: A clinical trial to compare two strontium desensitizing toothpastes with a conventional fluoride toothpaste. *J Periodontol* 1994; 65: 113-119.
47. Hoyt WH, Bibby BG. Use of sodium fluoride for desensitizing dentin. *J Am Dent Assoc* 1943; 30: 1372-1376.
48. Minkov B, Marmari I, Gedalia I, Garfunkel A. The effectiveness of sodium fluoride treatment with and without iontophoresis on the reduction of hypersensitive dentin. *J Periodontol* 1975; 46: 246-249.
49. Clark DC, Hanley JA, Geoghegan S, Vinet D. The effectiveness of a fluoride varnish and a desensitizing toothpaste in treatment dentinal hypersensitivity. *J Periodont Res* 1985; 20: 212-219.
50. Everett FG. Desensitization of hypersensitive exposed root surfaces. *Dent Clin North Am* 1964; 3: 221-230.
51. Murthy KS, Talim ST, Singh I. A comparative evaluation of topical application and iontophoresis of sodium fluoride for desensitization of hypersensitive dentin. *Oral Surg* 1973; 36: 448-458.
52. Tal M, Oron M, Gedalia I, Ehrlich J. X-ray diffraction and scanning electron microscope investigations of fluoride-treated dentine in man. *Arch Oral Biol* 1976; 21: 285-290.
53. Lan W, Liu H, Lin C. The combined occluding effect of sodium fluoride varnish and Nd:YAG laser irradiation on human dentinal tubules. *J Endod* 1999; 25: 424-426.
54. Pashley DH, Carvalho RM, Pereira JC, Villanueva R, Tay FR. The use of oxalate to reduce dentin permeability under adhesive restorations. *Am J Dent* 2001; 14: 89-94.
55. Baysan A, Lynch E. Treatment of cervical sensitivity with a root sealant. *Am J Dent* 2003; 16: 135-138.