

Effects of a new desensitizing paste containing 8% arginine and calcium carbonate on the shear bond strength of orthodontic brackets

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Objective: The purpose of this study was to evaluate shear bond strength (SBS) and failure site location of brackets bonded to enamel with or without desensitizer application. **Methods:** Sixty-six freshly extracted human premolar teeth were randomly divided into 3 groups of 22. Group 1 served as the control. Desensitizer was applied to the remaining teeth at two time intervals (Group 2, bonded immediately after Pro-Relief™ (Colgate-Palmolive Co., New York, NY, USA) application and Group 3, bonded 30 days after Pro-Relief™ application with the teeth stored in artificial saliva during the 30 days). Orthodontic brackets were bonded with a light cure composite resin and cured with a halogen light. After bonding, the SBS of the brackets was tested using a universal testing device. Adhesive remnant index (ARI) scores were determined after the brackets failed. Data were analyzed with analysis of variance, Tukey's HSD, and G tests. **Results:** The SBS was significantly lower in Group 2 than in Groups 1 ($p = 0.024$) and 3 ($p = 0.017$). Groups 1 and Group 3 did not differ ($p = 0.991$). ARI scores did not differ significantly among groups. **Conclusions:** The Pro-Relief™ desensitizer agent applied immediately before bonding significantly reduces bond strength, but the SBS values still exceed the minimum 5.9 - 7.8 MPa required for adequate clinical performance. Immersing the teeth in artificial saliva for 30 days after applying the Pro-Relief™ desensitizer agent and before bonding increased the SBS to control levels. (*Korean J Orthod* 2011;41(2):121-126)

Key words: Dentin desensitizing agents, Orthodontic brackets, Shear strength

INTRODUCTION

Dentinal hypersensitivity is a common condition and is often a chief concern among patients. The pain associated with dentinal hypersensitivity is caused by various types of external stimuli and the intensity of the sensitivity varies between patients. Dentinal hypersensitivity is characterized by short, sharp pain arising from exposed dentin in response to stimuli, typically thermal, evaporative, tactile, osmotic, or chemical, in the absence of any other dental defect or disease.¹ The most widely accepted mechanism of dentinal sensitivity is the hydrodynamic theory, first described by Brannstrom.² According to this theory, the movement of fluids within the dentinal tubules due to temperature or

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physical osmotic changes stimulates pressure-sensitive nerve receptors, leading to transmission of the stimuli.³⁻⁵

Dentinal hypersensitivity is sometimes observed in adolescence, but it is more typically found in the adult population.⁶ The prevalence of dentinal hypersensitivity is as high as 14.3% of all dental patients, between 3.8% and 57% of the adult dentate population, and up to 30% of adults at some time during their lifetime.⁷ The major portion of sufferers is in the age range of 20 to 49 years, with a peak incidence between 30 and 39 years.¹ Buccal cervical regions of the permanent teeth are most commonly affected, and canine, premolar, and incisor teeth are more frequently affected than the molar teeth.¹

Successful management of dentin hypersensitivity is often very challenging for dental professionals. Although some of the traditional methods provide some relief to patients, more effective, faster acting, and longer lasting treatments for dentinal hypersensitivity are in demand. In 2002, Kleinberg⁸ reported the development of new anti-sensitivity technology based upon the role that saliva plays in naturally reducing dentinal hypersensitivity. This technology, called Pro-Argin, physically plugs and seals exposed dentinal tubules and effectively relieves hypersensitivity.⁸ In 2007, the Colgate-Palmolive Company introduced a new Pro-Argin technology for the treatment of hypersensitivity and in early 2009, Colgate[®] Sensitive Pro-Relief[™] (Colgate-Palmolive Co., New York, NY, USA) in-office desensitizing paste was introduced. This product contains 8% arginine and calcium carbonate, and mimics the natural process of plugging and sealing the patient's dentinal tubules.³

The need for orthodontic treatment in the adult population is high, comprising 50% to 60% of young adults.⁹ Orthodontists may apply bonding brackets to hypersensitive teeth that have been treated with desensitizers.⁷ The effect of desensitizers on the bond strength of adhesives to dentin is well documented,^{10,11} and a consensus has been reached that these agents significantly affect bond strength.⁷ To our knowledge, however, there are few studies of the effects of desensitizer agents on the shear bond strength (SBS) of orthodontic brackets to human enamel.^{7,12,13} Colgate[®]

Sensitive Pro-Relief[™] in-office desensitizing paste is a new material and there is no literature investigating the effects of this desensitizing agent on the SBS of orthodontic brackets to human enamel.

The purpose of this *in vitro* study was to determine the effect of Pro-Relief[™] in-office desensitizer paste on the SBS and to determine the adhesive remnant index (ARI) of metallic brackets bonded with orthodontic composite at two time intervals (bonded immediately after desensitizer paste application and bonded 30 days after desensitizer paste application).

MATERIAL AND METHODS

Sixty-six non-carious maxillary premolars, extracted for orthodontic indications, were used in this study. Teeth with hypoplastic areas, cracks, and enamel structure irregularities were excluded. The criteria for tooth selection included no pretreatment with a chemical agent such as alcohol, formalin, or hydrogen peroxide. Immediately after extraction, the teeth were scraped of any residual tissue tags and washed under running tap water. The teeth were stored in distilled water, and the water was changed weekly to avoid bacterial growth. The sample was randomly divided into three groups of 22 teeth each. Each tooth was mounted vertically in a self-cure acrylic block to expose the crown. The buccal surfaces were cleaned and polished with a rubber cup and slurry with pumice and water, followed by rinsing with a water spray and drying with compressed air.

Specimens were prepared for bracket bonding according to one of the following procedures.

Group 1 (Control group): A 37% phosphoric acid gel (3M Dental Products, St Paul, MN, USA) was used to acid-etch the premolars for 15 seconds. The teeth were rinsed with water for 20 seconds and dried with an oil-free source for 20 seconds. In all etched samples, the enamel appeared frosty white. Standard edgewise premolar stainless steel brackets (G&H Wire Company, Greenwood, IN, USA), with a base surface area of 10 mm² (according to the manufacturer's specification), were bonded to the teeth using standard protocols according to the manufacturer's instructions. Transbond XT primer (3M Unitek, Monrovia, CA, USA) was applied to the etched surface in a thin film.

Transbond XT adhesive paste (3M Unitek, Monrovia, CA, USA) was applied to the bracket base, and the bracket was positioned on the tooth and pressed firmly into place. Excess resin was removed with an explorer before it was polymerized. Then, a light-emitting diode (Blue Swan Digital, Dentanet, Istanbul, Turkey) was used to cure the specimens for 20 seconds.

Group 2: Colgate[®] Sensitive Pro-Relief[™] in-office desensitizing paste was applied to the surface for 15 seconds using a rubber cup with a slow speed hand-piece at 3000 rpm using moderate to light pressure.¹⁴ After polishing, the samples were rinsed in tap water and then bonding procedure was applied as in Group 1.

Group 3: This group was treated the same as Group 2, but the teeth were stored in artificial saliva for 30 days at room temperature after applying the desensitizing paste and before bonding. The artificial saliva was changed every day.

Debonding procedure

After completion of the procedures, the embedded specimens were secured in a jig attached to the base plate of an Instron Universal Testing Machine (Instron Corp., Norwood, MA, USA). A chisel-edge plunger was mounted in the movable crosshead of the testing machine and positioned so that the leading edge was aimed at the enamel-adhesive interface. A crosshead speed of 0.5 mm/min was used, and the maximum load necessary to debond the bracket was recorded. The force required to remove the brackets was measured in Newtons (N), and the SBS (1 MPa = 1 N/mm²) was then calculated by dividing the force values by the bracket base area (10 mm²).

ARI scores

After debonding, all teeth and brackets were examined under 10X magnification. Any adhesive remaining after bracket removal was assessed using the ARI.^{15,16} The criteria were as follows: score 0 = no adhesive remaining on the tooth; score 1 = less than half of the adhesive remaining on the tooth; score 2 = more than half of the adhesive remaining on the tooth; and score

3 = all adhesive remaining on the tooth with a distinct impression of the bracket mesh.

Statistical methods

All statistical analyses were performed with the Statistical Package for the Social Sciences software package (SPSS for Windows 13.0, SPSS, Chicago, IL, USA) and Applet "Frequency Matrix Applet" Version 3.1. The Shapiro-Wilks normality test and Levene's variance homogeneity test were applied to the data. The data were normally distributed, and there was homogeneity of variance among the groups. Thus, the statistical evaluation of SBS values among test groups was performed using parametric tests.

Descriptive statistics, including the mean, standard deviation, and minimum and maximum values were calculated for the three groups of teeth tested. Comparisons of means of SBS values were made using an analysis of variance (ANOVA). Post-hoc multiple comparisons were done by Tukey's HSD test. The G-test was used to determine significant differences in the ARI scores among the groups.

RESULTS

The descriptive statistics for the SBSs of the three groups tested are presented in Table 1. The results of the ANOVA indicated statistically significant differences in the SBS among the three groups ($p < 0.01$). Tukey's HSD test showed that the SBS of Group 1 (control group, mean: 17.9 ± 5.4 MPa) and Group 3 (bonded 30 days after Pro-Relief[™] application, mean: 18.1 ± 4.5 MPa) were similar ($p = 0.991$), whereas the SBS of Group 2 (bonded immediately after Pro-Relief[™] application, mean: 13.8 ± 3.4 MPa) was significantly lower (Group 1 vs. Group 2: $p = 0.024$ and Group 2 vs. Group 3: $p = 0.017$). There were no significant differences between Groups 1 and 3 ($p = 0.991$).

The ARI scores for the different groups tested are listed in Table 2. The results of the G test comparisons indicated no significant differences among the three groups.

Table 1. Descriptive statistics and the results of ANOVA comparing the SBS of the three groups tested

Group tested ^a	Bond strength (Mpa)					ANOVA comparison	Tukey's HSD	
	N	Mean	Standard deviation	Minimum	Maximum		Group 2	Group 3
1	22	17.9	5.4	8.2	26.9	$p = 0.009^{\dagger}$	$p = 0.024^*$	$p = 0.991$ NS
2	22	13.8	3.4	8.4	28.4			$p = 0.017^*$
3	22	18.1	4.5	10.9	27.8			

^aGroup 1, Control; Group 2, bonded immediately after desensitizer paste application; Group 3, bonded 30 days after desensitizer paste application. NS, Not significant; SBS, shear bond strength. * $p < 0.05$; $^{\dagger}p < 0.01$.

Table 2. Adhesive remnant index (ARI) scores (%)

Group tested ^a	N	ARI score ^b				G-Test
		0	1	2	3	
1	22	2 (9.1%)	9 (40.9%)	6 (27.3%)	5 (22.7%)	$p = 0.9071$, NS
2	22	2 (9.1%)	8 (36.4%)	1 (4.5%)	11 (50%)	
3	22	2 (9.1%)	5 (22.7%)	6 (27.3%)	9 (40.9%)	

^aGroup 1, Control; Group 2, bonded immediately after desensitizer paste application; Group 3, bonded 30 days after desensitizer paste application. ^bARI scores: Score 0, No adhesive remaining on the tooth; Score 1, less than half of the adhesive left on the tooth; Score 2, more than half of the adhesive left on the tooth; Score 3, all adhesive left on the tooth with a distinct impression of the bracket mesh. NS, Not significant.

DISCUSSION

Dentin hypersensitivity is an uncomfortable and unpleasant condition that affects up to 57% of patients within a dental practice setting.¹⁷ A variety of products and methods are available for the treatment of dentin hypersensitivity. Treatment for dentinal hypersensitivity can involve occlusion of the dentinal tubules through the application of sedative agents, cavity varnishes, anti-inflammatory agents, dentin bonding agents, or restorative resin along with promotion of dentin remineralization.¹⁸ Several desensitizer agents have been used to provide desensitization of the natural teeth. In the present study, a new desensitizer paste was used prior to bonding and its effect on the SBS of orthodontic brackets was compared at two time intervals.

From a clinical perspective, orthodontists do not routinely desensitize teeth. Rather, general dentists do the desensitizing and orthodontists apply the brackets sometime later. Thus, the time span between desensi-

tizer application and bracket bonding should be considered a possible factor in the effect of the desensitizer on bond strength. For this reason, the brackets in Group 2 were immediately bonded to enamel treated with desensitizer and the brackets in Group 3 were bonded 30 days after application of the desensitizer. Group 2 had the lowest SBSs.

Power analysis using the G*Power Ver. 3.0.10. (Franz Faul, Universität Kiel, Germany) software, based on a 1 : 1 ratio among groups, indicated that a sample size of 21 teeth would give more than 80% power to detect significant differences with a 0.35 effect size at a significance level of $\alpha = 0.05$.

Several theories have been suggested to explain the mechanism of tooth sensitivity, but the "hydrodynamic theory" is widely accepted.³⁻⁵ According to this theory, the aspiration of odontoblasts into the dentinal tubules, as an immediate effect of physical stimuli applied to exposed dentin, results in the outward flow of the tubular contents (dentinal fluids) through capillary action.

Saliva provides calcium and phosphate, which over time occludes and blocks open dentinal tubules from external stimuli associated with dentinal hypersensitivity.^{8,19} The mechanism providing the clinical effectiveness of Pro-Relief™ desensitizer agent utilizes arginine, an amino acid; bicarbonate, a pH buffer; and calcium carbonate, a source of calcium. The technology is proposed to block dentinal hypersensitivity pain by occluding dentinal tubules with arginine, which is positively charged at a physiologic pH of 6.5 to 7.5 to bind to the negatively charged dentin surface, and helps attract a calcium-rich layer from the saliva to infiltrate and block the dentinal tubules.¹⁹

Türkkahraman and Adanir⁷ evaluated the effects of potassium nitrate and oxalate desensitizer agents on the SBS of orthodontic brackets and reported significantly lower SBS values in the groups receiving potassium nitrate and oxalate desensitizers. In the present study, SBS values were significantly lower in Group 2 (bonded immediately after Pro-Relief™ application) than in the other groups. We found no statistically significant differences in the bond strength between Group 1 (control group) and Group 3 (teeth bonded 30 days after Pro-Relief™ application).

Garcia-Godoy et al.¹⁴ investigated the effect of a desensitizing paste containing 8% arginine and calcium carbonate on the surface roughness of dental enamel. In that study, the 3D non-contact profilometry images showed slight roughness after using the desensitizing paste but these changes were not statistically significant. Covering the surface with desensitizing agents and remnants may affect adhesive bonding. Malkoc et al.¹² reported a remarkably decreased bond strength of orthodontic adhesives used to attach the bracket to the etched enamel surface after application of a desensitizer. Alterations in bond strength might be significant with regard to clinical operative procedures that involve composite resin bonding, such as bonding orthodontic brackets, porcelain veneers, composite veneers, or future composite restorations.²⁰

Yip et al.⁹ demonstrated that application of arginine-calcium carbonate in office desensitizing paste to teeth exhibiting sensitivity following a dental prophylaxis resulted in instant relief from discomfort and that the relief lasted for 28 days after a single application.

Schiff et al.²¹ applied this product immediately following scaling and 4 weeks later. In that study, the arginine-calcium carbonate paste group demonstrated statistically significant reductions in dentin hypersensitivity with respect to baseline adjusted mean air blast and mean tactile hypersensitivity scores, and no statistically significant differences were exhibited between paste groups at the post-scaling and 12-week examinations.

Reynolds²² determined the clinically acceptable minimum bond strength values in direct orthodontic bonding systems to be 5.9 to 7.8 MPa. All bond strength values of composites used in this study were greater than this minimum requirement and fell within the clinically acceptable range. Clinical conditions may significantly differ, however, from an *in vitro* setting. Moreover, the oral cavity is a complex environment with variations in temperature, stresses, humidity, acidity, and plaque.²³ Because of the probable differences between *in vivo* and *in vitro* conditions, a direct comparison cannot be made with the findings of the other studies.

Most orthodontic bonding studies have shown a mixed or cohesive-type failure.^{15,16} In those studies, after bond strength testing, a part of the composite resin remained either on the enamel surface or the bracket base, causing cohesive failure rather than adhesive failure between enamel and composite resin. Bond failure at the bracket-resin interface or within the resin is more desirable than at the resin-enamel interface because enamel fractures and cracks have been reported during bracket debonding.²⁴ For mechanically retained brackets, the most common failure site was the bracket-resin interface, and, on average, more than 50% of resin remains on teeth after debonding.²⁵ The ARI score comparisons in the present study indicated no significant differences among the three groups tested.

There are no published data about comprehensive observations or intraoral applications concerning Pro-Relief™ in-office desensitizer paste and the effects on the bond strength of brackets. Thus, the clinical significance of this new desensitizer paste should be further clarified in detail under *in vivo* conditions.

CONCLUSION

The use of a Pro-Relief™ desensitizer agent immediately before bonding significantly reduces the SBS, but the SBS still exceeds the minimum 5.9 to 7.8 MPa required to expect adequate clinical performance.

Immersion of teeth applied with Pro-Relief™ desensitizer agent in artificial saliva for 30 days before bonding increased the SBS value to that of controls.

The use of desensitizer procedures with arginine and calcium carbonate immediately before bonding orthodontic brackets is not recommended.

REFERENCES

1. Addy M. Dentine hypersensitivity: new perspectives on an old problem. *Int Dent J* 2002;52:367-75.
2. Brannstrom M. Dentin sensitivity and aspiration of odontoblasts. *J Am Dent Assoc* 1963;66:366-70.
3. Cummins D. Dentin hypersensitivity: from diagnosis to a breakthrough therapy for everyday sensitivity relief. *J Clin Dent* 2009;20:1-9.
4. Walters PA. Dentine hypersensitivity: a review. *J Contemp Dent Pract* 2005;6:107-17.
5. Swift EJ Jr. Causes, prevention, and treatment of dentin hypersensitivity. *Compend Contin Educ Dent* 2004;25:95-106.
6. West NX. Dentine hypersensitivity. In: Lussi A editor. *Dental erosion*. Basel: Karger; 2006. p. 173-89.
7. Türkahraman H, Adanir N. Effects of potassium nitrate and oxalate desensitizer agents on shear bond strengths of orthodontic brackets. *Angle Orthod* 2007;77:1096-100.
8. Kleinberg I. SensiStat. A new saliva-based composition for simple and effective treatment of dentinal sensitivity pain. *Dent Today* 2002;21:42-7.
9. Yip CK. The need and demand of orthodontics among Chinese adults in Hong Kong (dissertation). Hong Kong: Univ of Hong Kong, 1993.
10. Sengun A, Koyuturk AE, Sener Y, Ozer F. Effect of desensitizers on the bond strength of a self-etching adhesive system to caries-affected dentin on the gingival wall. *Oper Dent* 2005;30:430-5.
11. Aranha AC, Siqueira Junior Ade S, Cavalcante LM, Pimenta LA, Marchi GM. Microtensile bond strengths of composite to dentin treated with desensitizer products. *J Adhes Dent* 2006; 8:85-90.
12. Malkoc S, Demir A, Sengun A, Ozer F. The effect on shear bond strength of different antimicrobial agents after acid etching. *Eur J Orthod* 2005;27:484-8.
13. Holzmeier M, Ernst CP, Willershausen B, Hirschfelder U. In-vitro shear bond strength of self-etching versus traditional adhesives for orthodontic luting. *J Orofac Orthop* 2006;67: 244-59.
14. Garcia-Godoy F, Garcia-Godoy A, Garcia-Godoy C. Effect of a desensitizing paste containing 8% arginine and calcium carbonate on the surface roughness of dental materials and human dental enamel. *Am J Dent* 2009;22:21A-4A.
15. Artun J, Bergland S. Clinical trials with crystal growth conditioning as an alternative to acid-etch enamel pretreatment. *Am J Orthod* 1984;85:333-40.
16. Oliver RG. The effect of different methods of bracket removal on the amount of residual adhesive. *Am J Orthod Dentofacial Orthop* 1988;93:196-200.
17. Addy M. Etiology and clinical implications of dentine hypersensitivity. *Dent Clin North Am* 1990;34:503-14.
18. Trowbridge HO, Silver DR. A review of current approaches to in-office management of tooth hypersensitivity. *Dent Clin North Am* 1990;34:561-81.
19. Panagakos F, Schiff T, Guignon A. Dentin hypersensitivity: effective treatment with an in-office desensitizing paste containing 8% arginine and calcium carbonate. *Am J Dent* 2009;22:3A-7A.
20. Josey AL, Meyers IA, Romaniuk K, Symons AL. The effect of a vital bleaching technique on enamel surface morphology and the bonding of composite resin to enamel. *J Oral Rehabil* 1996;23:244-50.
21. Schiff T, Delgado E, Zhang YP, Cummins D, DeVizio W, Mateo LR. Clinical evaluation of the efficacy of an in-office desensitizing paste containing 8% arginine and calcium carbonate in providing instant and lasting relief of dentin hypersensitivity. *Am J Dent* 2009;22:8A-15A.
22. Reynolds IR. A review of direct orthodontic bonding. *Br J Orthod* 1975;2:171-8.
23. Zachrisson YO, Zachrisson BU, Büyükyılmaz T. Surface preparation for orthodontic bonding to porcelain. *Am J Orthod Dentofacial Orthop* 1996;109:420-30.
24. Bishara SE, Olsen ME, Von Wald L. Evaluation of debonding characteristics of a new collapsible ceramic bracket. *Am J Orthod Dentofacial Orthop* 1997;112:552-9.
25. Forsberg CM, Hagberg C. Shear bond strength of ceramic brackets with chemical or mechanical retention. *Br J Orthod* 1992;19:183-9.