Restor Dent Endod. 2023 May;48(2):e13
https://doi.org/10.5395/rde.2023.48.e13
pISSN 2234-7658·eISSN 2234-7666

Restorative Dentistry & Endodontics RDE

Research Article

Check for updates

Epigallocatechin-3-gallate prior to composite resin in abfraction lesions: a split-mouth randomized clinical trial

Luísa Valente Gotardo Lara Alves,* **Lisiane Martins Fracasso**, **Thiago Vinicius Cortez**, **Aline Evangelista Souza-Gabriel**, **Silmara Aparecida Milori Corona**

Department of Restorative Dentistry, Ribeirão Preto School of Dentistry, University of São Paulo, Ribeirão Preto, SP, Brazil

OPEN ACCESS

Received: Oct 9, 2022
Revised: Nov 23, 2022
Accepted: Dec 1, 2022
Published online: Mar 20, 2023

Alves LVGL, Fracasso LM, Cortez TV, Souza-Gabriel AE, Corona SAM

***Correspondence to**
Luísa Valente Gotardo Lara Alves, DDS
Department of Restorative Dentistry, School of Dentistry of Ribeirão Preto, University of São Paulo, Ribeirão Preto, São Paulo, Brazil.
Avenida do Café, S/ No, Ribeirão Preto, SP 14040-904, Brazil.
Email: luisavalente_@hotmail.com

Conflict of Interest
No potential conflict of interest relevant to this article was reported.

Author Contributions
Conceptualization: Alves LVGL, Fracasso LM, Souza-Gabriel AE; Corona SAM; Data curation: Alves LVGL, Fracasso LM, Cortez TV; Formal analysis: Alves LGVL, Fracasso LM, Souza-Gabriel AE; Investigation: Alves LVGL; Fracasso LM; Cortez TV; Methodology: Alves, LVGL, Fracasso LM; Souza-Gabriel AE;

ABSTRACT

Objectives: Natural extracts have been investigated as a biomimetic strategy to mechanically strengthen the collagen network and control the biodegradation of extracellular matrix. This study evaluated the effect of epigallocatechin-3-gallate (EGCG) on abfraction lesions prior to the composite resin.
Materials and Methods: The sample consisted of 30 patients (aged between 28 and 60 years) with abfraction lesions located in 2 homologous premolars. The teeth were randomly assigned according to dentin treatment: 0.02% EGCG solution or distilled water (control). After enamel acid etching, the solutions were applied immediately for 1 minute. The teeth were restored with Universal Adhesive (3M) and Filtek Z350 XT (3M). Analyzes were done by 2 independent examiners using modified USPHS (retention, secondary caries, marginal adaptation, and postoperative sensitivity) and photographic (color, marginal pigmentation, and anatomical form) criteria at baseline (7 days) and final (18 months). The data analysis used Friedman and Wilcoxon signed-rank tests ($\alpha = 0.05$).
Results: At baseline, all restorations were evaluated as alpha for all criteria. After 18 months, restorations were evaluated as alpha for secondary caries, color, and marginal pigmentation. There was significant difference between baseline and 18 months ($p = 0.009$) for marginal adaptation and postoperative sensitivity ($p = 0.029$), but no significant difference were verified between treatments ($p = 0.433$). The EGCG group had a restoration retention rate of 93.3%, while the control group had 96.7%.
Conclusions: The application of EGCG solution on abfraction lesions did not significantly influence the survival of the restorations based on clinical and photographic criteria.

Keywords: Composite resin; Dentin; Tooth wear; Solution

INTRODUCTION

Abfraction is a tooth lesion caused by the pathological loss of tissues at the cervical third of the crown and root surface, disconnected to dental caries [1]. These lesions are noted mainly on the buccal surfaces of premolars and anterior teeth and are typically wedged-shaped lesions with defined internal and external limits [2].

Corona SAM; Project administration: Alves LGVL; Fracasso LM; Souza-Gabriel AE; Corona SAM; Resources: Alves LGVL, Fracasso LM; Corona SAM; Software: Souza-Gabriel AE; Supervision: Corona SAM; Validation: Alves LGVL; Corona SAM ; Visualization: Alves LGVL; Fracasso LM; Souza-Gabriel AE; Corona SAM; Writing - original draft: Alves LVGL; Fracasso LM; Writing - review & editing: Alves LVGL; Fracasso LM; Cortez TV; Souza-Gabriel AE; Corona SAM.

ORCID iDs

Luísa Valente Gotardo Lara Alves
https://orcid.org/0000-0003-2058-7657
Lisiane Martins Fracasso
https://orcid.org/0000-0002-2913-469X
Thiago Vinicius Cortez
https://orcid.org/0000-0002-1490-1884
Aline Evangelista Souza-Gabriel
https://orcid.org/0000-0002-9280-2945
Silmara Aparecida Milori Corona
https://orcid.org/0000-0002-1733-3472

Abfraction has a multifactorial etiology, but the formation process remains unclear [3]. Probably the development mechanism of cervical lesions involves a complex interaction of endogenous (parafunction, occlusion, and deglutition) and exogenous (mastication, habits, occupations, and dental appliances) factors combined with fatigue stress [4]. The biomechanically-based theory suggests that lesions have been attributed to excessive loading cusp forces that cause flexure and micro-crack of the hydroxyapatite crystals of the enamel and dentin in the cervical area [2,4]. Laboratory studies have evidenced that when teeth are charged horizontally, fatigue stress becomes focused in the cervical area [5]. However, the clinical studies available are insufficient to confirm this theory [6,7].

Another possible theory is associated with bending forces arising during bruxism and an electrical response in enamel and dentin [8]. Enamel and dentin are insulators and, therefore, when interacting with a beam of electrons their ability to store electrical charge is different, which leads to an investigation of other phenomena in human teeth, such as piezoelectricity [9]. A piezoelectric episode occurs by the increased surface polarization charge due to mechanical stress [9]. Furthermore, bruxism cannot always be associated with abfraction lesions because not all patients with the lesions demonstrate occlusal wear and not all patients with severe occlusal wear exhibit abfraction lesions [2].

Abfraction is often asymptomatic, but these lesions may progress to esthetic problems, dentinal hypersensitivity, discomfort to the patient, functional impairment, and tooth fracture [10]. No guidelines support when abfraction conditions should be restored, so this decision involves the esthetic demands, dentinal hypersensitivity, and clinical judgment [3]. Composite restorations of cervical abfraction lesions have a higher percentage of failure and represent a challenge to the dental profession [7].

The longevity of composite resin restorations depends on the integrity of the resin-dentin interfacial components [11]. Ideally, the exposed collagen matrix of the dentin should be completely resin-infiltrated and polymerized, the so-called hybrid layer [12]. However, organic dentin matrices contain proteolytic enzymes such as matrix metalloproteinases (MMPs) and cysteine cathepsins secreted as latent proenzymes [12,13]. The areas of resin-sparse collagen are the threshold for the resin–dentin-bond degradation since the activity of MMPs might accelerate cathepsins and endopeptidases [14]. During the adhesion steps, the hydrolytic and enzymatic degradation of the collagen matrix always occurs, and an unstable adhesive interface can be degraded through water sorption [13].

The modification of the collagen matrix of dentin by natural-derived substances has been considered to maintain the long-term collagen entirety in the hybrid layer [15]. Epigallocatechin-3-gallate (EGCG) is monomeric catechin found in polyphenol-rich natural extracts and has antibacterial characteristics and an inhibitory effect against both MMPs and cysteine cathepsins, thus preserving the dentin bond strength. EGCG extracts do not exert cytotoxicity against odontoblast-like cells while keeping an antibacterial effect and antiproteolytic activity against MMP-2 and MMP-9 enzymes [16-19].

Although there are few studies evaluating EGCG at 0.02% concentration, various concentrations of EGCG ranging from 0.0065% to 5% have been used in dentistry [17,20-22]. In this range, 0.02% and 0.1% EGCG/water solution can effectively facilitate dentin bonding [20]. Besides, 0.02% EGCG can stabilize collagen depending on hydrogen bond and hydrophobic interactions with collagenases and stabilization of collagen may be one of the mechanisms that inhibit MMPs [23].

Considering that the bioactive constituent of EGCC can modify the dentin surface, protecting the collagen fibers and improving the adhesion over time, this longitudinal study evaluated the influence of EGCG solution prior to composite resin on teeth with abfraction lesions at baseline (7 days) and after 18 months [15].

MATERIALS AND METHODS

Sample size calculation and ethical aspects

The sample size calculation was done using G*power software (University of Düsseldorf, Düsseldorf, Germany) and based on a previous clinical trial of our research group involving noncarious cervical lesions and natural agents to treat dentin [24]. The following parameters were used: α= 5%, power 90%, success percentage of 98% (control x experimental), reaching a minimum sample of 27 restorations *per* group. Twenty percent of patients were added to the sample because of the possible losses, reaching 32 individuals. The clinical study was reviewed and approved by the local Research Ethics Committee (CAAE: 5791719.0.0000.5419) and was registered at the Registry of Clinical Trials (RBR-772thc). Patients were instructed on the conditions and objective of the study, and each patient signed an informed consent form and authorization to participate in the study.

Patient's selection

During 6 months, 420 patients were examined at our School of Dentistry. Among them, 32 patients (28 to 60 years old, mean age = 45.35, 20 women and 12 men) were selected for the study. The inclusion criteria included good oral hygiene, no periodontal disease, and at least 2 abfraction lesions on the buccal surface of homologous vital premolars. All lesions were between 2 mm and 3 mm deep and positively related to hypersensitivity (stimulated pain resulting from exposed dentin). Patients with non-vital teeth, temporomandibular dysfunction, bruxism, spontaneous acute pain, fistula, or edema were excluded from the study. Tooth vitality was tested with the thermal test with Endofrost (Roeko, Langenau, Germany). The medical history and dental charts were completed, and patients received oral hygiene instructions. Sixty percent of the restorations were located in the upper arch, while 40% were in the lower arch. Patients not selected for the study that requires restorative treatment were referred to our dental clinic.

Treatment of the abfraction lesions

Prophylaxis with pumice paste (SS White, Rio de Janeiro, RJ, Brazil) and a rubber cup or Robinson brush (Jon, São Paulo, SP, Brazil) was performed using a low-speed handpiece (Gnatus, Ribeirão Preto, SP, Brazil). Waxed dental floss (Hillo – Aperifio, Aperibé, RJ, Brazil) was used on the proximal surfaces. No cavity preparation was carried out. Initial photographs of the teeth were taken (Canon EOS Rebel T5i 18.0 Megapixels Canon, Tokyo, Japan).

The 2 premolars of each patient received 1 of the dentin pre-treatments (experimental - 0.02% EGCG solution or control - distilled water) according to a split-mouth randomized design (**Figure 1**). A computer-generated randomization list was used to assign patients. The EGCG solution was prepared by dissolving 2 mg of EGCG powder (≥ 95 purity; Sigma-Aldrich, St. Louis, MO, USA) in 100 mL of deionized water for 1 hour under stirring (pH = 7.5) made fresh every time before each application.

Shade selection was made using a shade guide (Ivoclar Vivadent, Schaan, Liechtenstein). The isolation procedure was done using a rubber dam with a stainless steel clamp positioned

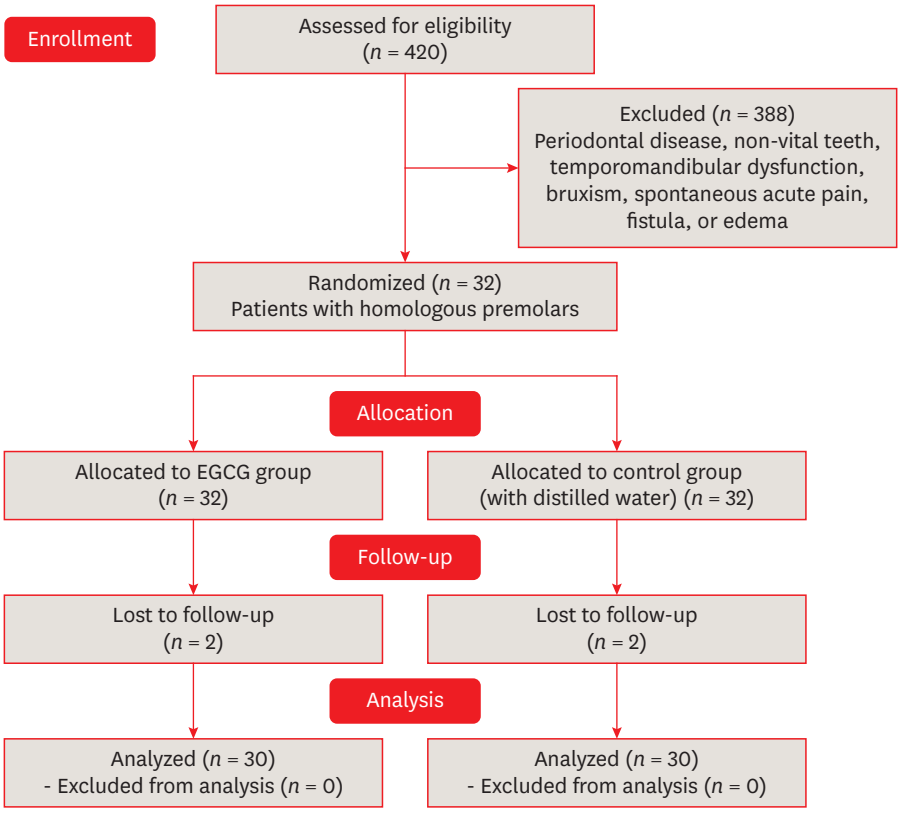

Figure 1. Study CONSORT diagram.
EGCG, epigallocatechin-3-gallate.

2 teeth behind the tooth to be restored. The rubber dam was cut between dental papilla, whenever necessary, to allow adaptation of the cervical margin of the restoration. After enamel etching with 35% phosphoric acid gel (Total Etch, Ivoclar Vivadent), the surface was washed with water for 1 minute, the excess water was removed with the suction cannula, and the surface dried with absorbent paper.

The EGCG solution was actively applied to the dentin with a disposable microbrush (KGBrush; KG Sorensen, Cotia, SP, Brazil) for 1 minute, followed by an airstream of 5 seconds. The excess solution was removed with absorbent paper, leaving the dentin surface moist. In the teeth of the control group, distilled water was applied as described for the experimental group. Two coats of the adhesive system (Scotchbond Universal Adhesive; 3M ESPE, St Paul, MN, USA) were actively applied with a disposable microbrush (KGBrush; KG Sorensen) for 10 seconds. An air blast was applied, and the adhesive was light-cured for 20 seconds (Gnatus). Resin composite (Filtek Z350; 3M ESPE) increments were inserted with a resin spatula and light-cured for 20 seconds using a calibrated light-curing unit. The resin excess was removed using a #12 blade and diamond burs (KG Sorensen, Cotia, SP, Brazil). Polishing disks (Sof-Lex polishing disks; 3M ESPE) were used for finishing. Seven days later, final polishing was performed using abrasive cups (Enhance; Dentsply Sirona, Schaan, Liechtenstein) and pumice paste (SS White).

https://doi.org/10.5395/rde.2023.48.e13

Clinical evaluation

Three independent and calibrated clinicians were responsible for the clinical and photographic analyzes of restorations following modified USPHS clinical (retention, secondary caries, marginal adaptation, and postoperative sensitivity) and photographic (restoration color, marginal pigmentation, and anatomical form) criteria at baseline and 18 months. The baseline rating was carried out 1 week after restoration, immediately after finishing and polishing the procedure.

A single dentist performed all restorations after training and calibrating the clinical protocol (**Figure 2**). The restorations were scored into Alpha—when the evaluated criterion had no problems, and the restoration was in perfect condition; Bravo—when the evaluated criterion had minor but clinically acceptable failures and Charlie— when the evaluated criterion had relevant failures and the restoration needed to be replaced (**Table 1**). Intraoral photographs were taken at both time-points. The photographs were analyzed under the same environment and light conditions through a laptop's screen.

Data analysis

Data analysis was performed using the Statistical Package for the Social Sciences software (SPSS version 25.0; IBM Corp., Armonk, NY, USA) with a significance level of 5% and was based on descriptive and inferential statistics. Descriptive statistics were used to describe the frequency of distribution of modified USPHS scores, including the percentage of failed restorations. The inferential statistical analysis used Friedman's nonparametric analysis of variance (ANOVA) test and the Wilcoxon test signed-rank of repeated measurements for the different periods of analysis (baseline and 18 months) and interactions between treatment and analysis period. The Cohen Kappa test was used to compare intra- and inter-examiner agreements.

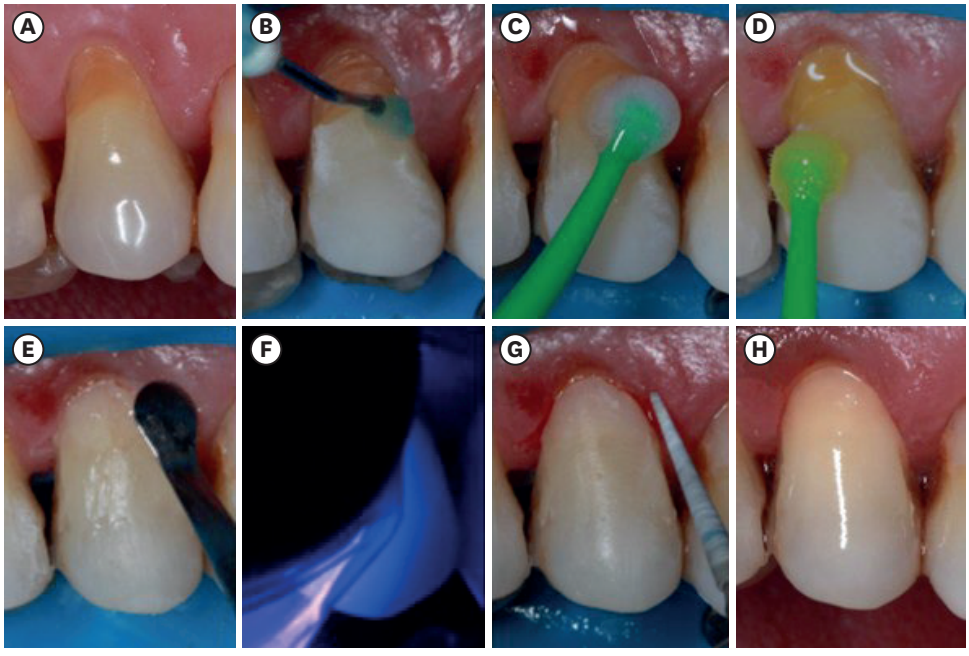

Figure 2. Restorative procedures steps of the experimental group. (A) Initial aspect of the abfraction lesion; (B) Enamel etching with 35% phosphoric acid gel; (C) Active application of epigallocatechin-3-gallate (EGCG) solution on the dentin; (D) Universal Adhesive system application; (E) Composite resin increments inserted in the cavity; (F) Resin light-curing for 20 seconds; (G) Diamond bur used for the finishing step; (H) Final aspect of the restoration.

https://doi.org/10.5395/rde.2023.48.e13

Table 1. Modified USPHS criteria used for the clinical and photographic analysis of restorations

Clinical criteria	Score	Photographic criteria	Score
Retention	A. No loss of restorative material	Restoration color	A. Corresponds to adjacent dental structure in terms of color and translucency
	B. Partial loss of restorative material		B. Slight change in color, shade, or translucency between restoration and adjacent tooth
	C. Total loss of restorative material		C. Clear color change and translucency
Secondary caries	A. No recurrence of caries	Marginal Pigmentation	A. No pigmentation along the margin between restoration and adjacent tooth
	B. With the recurrence of superficial caries		B. Slight pigmentation along the margin between the restoration and adjacent tooth
	C. With the recurrence of deep caries		C. Pigmentation present along restoration margin
Marginal adaptation	A. Perfectly adaptable with no visible margins	Anatomic form	A. Restoration in continuity with existing anatomical form
	B. Visible but clinically acceptable margin		B. Restoration in discontinuity with anatomical form of tooth
	C. Marginal mismatch, clinical failure		C. Loss of material by exposing dentin or restoration base
Postoperative sensitivity	A. Missing stimulated sensitivity		
	B. Present and localized sensitivity		
	C. Present and diffuse stimulated sensitivity		

A, Alpha; B, Bravo; C, Charlie scores.

RESULTS

The intra-examiner Kappa index was 1.0 for examiner A and 0.98 for examiner B. The inter-examiner index (A and B) was 0.94. At the end of the study, 30 patients return to the 18-month follow-up (recall rate = 93.75%, mean age = 45.35). **Tables 2** and **3** displayed the clinical and photographic analyses of the tested protocols at baseline and 18 months.

For the retention criteria, the teeth that received pre-treatment with EGCG solution had a restoration retention rate of 93.3%, while the control group (without EGCG) had 96.7% restoration retention. Therefore, there was no significant difference between treatments (p = 0.433) after 18 months.

Regarding postoperative sensitivity, it was verified that in the EGCG-treated group, 4 teeth (13.3%) remained stimulated sensitive at the baseline, while in the control group, the symptom was present in 5 teeth (16.7%). After 18 months, sensitivity was present in 2 teeth (6.7%) in the experimental group, and only 1 tooth (3.3%) remained sensitive in the control group. There was no significant difference between treatments (p = 0.593), but there was a significant difference between baseline and 18 months (p = 0.029).

Table 2. Clinical analyzes at baseline (7 days) and final (18 months) periods after the restorative procedure

Treatment	Evaluation period	Values	Retention			Secondary caries			Marginal adaptation			Postoperative sensibility		
With 0.02% EGCG	Baseline (7 days)	n = 30 (%)	A	B	C	A	B	C	A	B	C	A	B	C
			30	-	-	30	-	-	30	-	-	26	4	-
			100	-	-	100	-	-	100	-	-	86.7	13.3	-
	Final (18 mon)	n = 30 (%)	A	B	C	A	B	C	A	B	C	A	B	C
			28	-	2	30	-	-	28	2	-	28	2	-
			93.3	-	6.7	100	-	-	93.3	6.7	-	93.3	6.7	-
With distilled water	Baseline (7 days)	n = 30 (%)	A	B	C	A	B	C	A	B	C	A	B	C
			30	-	-	30	-	-	30	-	-	25	5	-
			100	-	-	100	-	-	100	-	-	83.3	16.7	-
	Final (18 mon)	n = 30 (%)	A	B	C	A	B	C	A	B	C	A	B	C
			29	-	1	30	-	-	28	2	-	29	1	-
			96.7	-	3.3	100	-	-	93.3	6.7	-	96.7	3.3	-

EGCG, epigallocatechin-3-gallate.

https://doi.org/10.5395/rde.2023.48.e13

Table 3. Photographic analyzes at baseline (7 days) and final (18 months) periods after the restorative procedure

Treatment	Evaluation period	Values	Color			Marginal pigmentation			Anatomic form		
With 0.02% EGCG	Baseline (7 days)	*n* = 30 (%)	A	B	C	A	B	C	A	B	C
			30	-	-	30	-	-	30	-	-
			100	-	-	100	-	-	100	-	-
	Final (18 mon)	*n* = 30 (%)	A	B	C	A	B	C	A	B	C
			30	-	-	30	-	-	29	-	1
			100	-	-	100	-	-	96.6	-	3.3
With distilled water	Baseline (7 days)	*n* = 30 (%)	A	B	C	A	B	C	A	B	C
			30	-	-	30	-	-	30	-	-
			100	-	-	100	-	-	100	-	-
	Final (18 mon)	*n* = 30 (%)	A	B	C	A	B	C	A	B	C
			30	-	-	30	-	-	29	-	1
			100	-	-	100	-	-	96.6	-	3.3

EGCG, epigallocatechin-3-gallate.

Both groups (experimental and control) had 6.7% failure in the marginal adaptation after 18 months but were clinically acceptable (bravo score). There was a significant difference between baseline and 18 months ($p = 0.009$), but no significant differences were found between treatments ($p = 0.566$).

For secondary caries, color and marginal pigmentation criteria, a 100% alpha score was verified when teeth were treated with EGCG or distilled water (control). There was no significant difference between treatments ($p = 0.533$) and time ($p = 0.075$) for the anatomic form criteria.

DISCUSSION

Noncarious cervical lesions are challenging to restore because they include the cervical third of the tooth, and the most significant part of the lesion is in the dentin [4,7]. The cervical area has been established as a weak region because the enamel is thin with lower mineral content and low density of Hunter-Schreger bands [25]. The water absorption and hydrolytic biodegradation of unprotected collagen matrix have been considered responsible for dentin/resin bond degradation, decreasing the longevity of composite restorations [11].

Currently, the green tea solution might emerge as a powerful tool for preventing the progression of non-carious cervical lesions because the ECGC and other catechins present in the solution have a chelating effect on metallic ions, resulting in a wear protective layer [26].

In our study, after 18 months, the application of a low concentration of EGCG, at 0.02%, did not influence the clinical retention and appearance of restorations based on the USPHS criteria. This result follows the clinical study of Costa *et al.* [27] that evaluated a higher concentration of EGCG, at 0.1%, in an aqueous solution as a dentin pretreatment on the clinical performance of restorations of non-carious cervical lesions after 24 months.

However, when considering bond strength analysis, the concentration of EGCG seems to influence differently compared to the clinical and photographic analysis of restorations. A previous study demonstrated that ECGC acts as a crosslinker and enhances the bond strength of adhesive systems [20]. However, EGCG at higher concentrations could interrupt the adhesive polymerization, thereby affecting the bond strength [21]. Even at a concentration of 0.1%, EGCG did not show promising results [28]. EGCG has a free radical depletion

effect, with can interfere with the polymerization of the adhesive, and this effect is directly proportional to the EGCG concentration [29]. The compromise in bond strength can be explained by the fact that the EGCG is probably entrapped within the linear chains after curing, thereby interfering with the monomer conversion and bond strength. These findings corroborate the results described in the meta-analysis by Hardan *et al.* [30].

The difference between using EGCG as a pretreatment or incorporating it into the adhesive seems to affect the protocol's success. Positive results were observed in our study, considering that the EGCG was applied as a pretreatment in dentin. However, the interaction of the photoinitiator and ECGC when incorporating it into the adhesive may explain the worst results found in another study, with a loss in the retention of composite resin restorations [31].

Besides, the effect of EGCG pretreatment on adhesive–dentin bonds is concentration-dependent [32]. Hiraishi *et al.* [22] evaluated the impact of various plant-derived agents on the stability of collagen matrix to resist collagenase degradation, and a dose-dependent effect of EGCG was found. However, Vidal *et al.* [17] reported that, even at a very low concentration (0.0065%), EGCG could inhibit MMP-9. A 0.02% EGCG has already been shown to have higher immediate and aged bonding strength than 0.1% EGCG, while 0.02% EGCG presented less nanoleakage expression than 0.1% EGCG [32]. Therefore, more clinical studies are needed to evaluate the performance of EGCG at the 0.02% concentration.

The high retention rates after 18 months showed the bond success of Scothbond Universal Adhesive. This adhesive system is stable and contains 10-methacryloyloxydecyl dihydrogen phosphate (10-MDP), an acidic phosphate monomer able to bond to hydroxyapatite [33]. 10-MDP molecules are involved in the micromechanical and chemical bonding to the dentin, which may have directly impacted the longevity of restorations.

For marginal adaptation, 94.3% of restorations were scored as alpha, and 6.7% were scored as visible but clinically acceptable after 18 months in both groups. These results can be associated with selective enamel etching previous to EGCG, providing an effective etching pattern in enamel. The selective enamel etching can dissolve prism cores and boundaries, increasing the surface free energy [34].

The EGCG concentration of our study can explain the slight decrease in retention and marginal adaptation after 18 months. In the previous *in vitro* studies, a 0.02% EGCG showed better results in preserving the bond strength over time, but this protocol could not influence the longevity of restorations [20,32]. Probably, the concentration was insufficient to inhibit collagenase activity and promote adequate hydrogen bonding between phenolic compounds and dental proteins to alter the longevity of restorations [23].

The relationship between the adhesive and MMPs' activity is not yet well established. The literature demonstrates that MMPs activity depends on the adhesive system because of its pH and chemical composition [35]. For example, 2-hydroxyethyl methacrylate (HEMA) is a monomer that inhibits MMPs and can, in theory, contribute to a more stable bond. However, HEMA was found in adhesives that showed bond stability but also in adhesives where lower bond strength was observed [35-37]. Therefore, the relation between HEMA and MMP-2 should be interpreted cautiously because adhesive systems have other components, such as the acidic types capable of counteracting HEMA's inhibitory effect on MMP-2 [35].

https://doi.org/10.5395/rde.2023.48.e13

Overall, we found no occurrence of second caries in the experimental or control groups after 18 months of treatment. These results can be explained by the selection of patients for the study, who had good oral hygiene and did not have caries. Considering the antibacterial effect of EGCG against several bacterial pathogens, with an effect equal to chlorhexidine the use of EGCG in high-risk caries patients, on the caries-affected dentin can be the object of long-term studies [15,20].

Dentin biomodifiers alter the structure of collagen fibrils improving their degradation resistance, and when used as pre-treatment prior to the bonding procedures aid in increasing the bond strength values [38,39]. However, Fialho *et al.* [37] showed that dentin pre-treatment with EGCG at 0,02%, 0,2% and 0,5% concentration did not prevent loss of bond strength over time, however, they decreased nanoleakage on caries-affected dentin. In opposition, Nivedita *et al.* [38] concluded that chitosan and proanthocyanidin, when used as biomodifiers agents, improved bond strength to dentin, but showed no improvement in nanoleakage. Therefore, further studies are needed to evaluate different dentin biomodifiers as well as their influence on composite resin restorations.

CONCLUSIONS

The high retention rates, absence of second caries and marginal pigmentation suggest that the benefits of EGCG solution on abfraction lesions can be further explored in clinical studies. At 18 months, the EGCG used as dentin pre-treatment in abfraction lesions did not influence the clinical performance of composite resin restorations.

ACKNOWLEDGEMENTS

The authors would like to thank the Brazilian National Council for Scientific and Technological Development (CNPq) (Processo: 304366/2020-0).

REFERENCES

1. Francisconi LF, Graeff MS, Martins LM, Franco EB, Mondelli RF, Francisconi PA, Pereira JC. The effects of occlusal loading on the margins of cervical restorations. J Am Dent Assoc 2009;140:1275-1282.
PUBMED | CROSSREF
2. Badavannavar AN, Ajari S, Nayak KU, Khijmatgar S. Abfraction: Etiopathogenesis, clinical aspect, and diagnostic-treatment modalities: a review. Indian J Dent Res 2020;31:305-311.
PUBMED | CROSSREF
3. Michael JA, Townsend GC, Greenwood LF, Kaidonis JA. Abfraction: separating fact from fiction. Aust Dent J 2009;54:2-8.
PUBMED | CROSSREF
4. Peumans M, Politano G, Van Meerbeek B. Treatment of noncarious cervical lesions: when, why, and how. Int J Esthet Dent 2020;15:16-42.
PUBMED
5. Rees JS, Hammadeh M. Undermining of enamel as a mechanism of abfraction lesion formation: a finite element study. Eur J Oral Sci 2004;112:347-352.
PUBMED | CROSSREF
6. Romeed SA, Malik R, Dunne SM. Stress analysis of occlusal forces in canine teeth and their role in the development of non-carious cervical lesions: abfraction. Int J Dent 2012;2012:234845.
PUBMED | CROSSREF

7. Rusu Olaru A, Popescu MR, Dragomir LP, Rauten AM. Clinical study on abfraction lesions in occlusal dysfunction. Curr Health Sci J 2019;45:390-397.
PUBMED

8. Wood I, Jawad Z, Paisley C, Brunton P. Non-carious cervical tooth surface loss: a literature review. J Dent 2008;36:759-766.
PUBMED | CROSSREF

9. Reyes-Gasga J, Galindo-Mentle M, Brès E, Vargas-Becerril N, Orozco E, Rodríguez-Gómez A, García-García R. Detection of the piezoelectricity effect in nanocrystals from human teeth. J Phys Chem Solids 2020;136:109140.
CROSSREF

10. Grippo JO, Chaiyabutr Y, Kois JC. Effects of cyclic fatigue stress-biocorrosion on noncarious cervical lesions. J Esthet Restor Dent 2013;25:265-272.
PUBMED | CROSSREF

11. Matuda LS, Marchi GM, Aguiar TR, Leme AA, Ambrosano GM, Bedran-Russo AK. Dental adhesives and strategies for displacement of water/solvents from collagen fibrils. Dent Mater 2016;32:723-731.
PUBMED | CROSSREF

12. Yiu CK, King NM, Pashley DH, Suh BI, Carvalho RM, Carrilho MR, Tay FR. Effect of resin hydrophilicity and water storage on resin strength. Biomaterials 2004;25:5789-5796.
PUBMED | CROSSREF

13. Prakki A, Xiong Y, Bortolatto J, Gonçalves LL, Bafail A, Anderson G, Stavroullakis AT. Functionalized epigallocatechin gallate copolymer inhibit dentin matrices degradation: mechanical, solubilized telopeptide and proteomic assays. Dent Mater 2018;34:1625-1633.
PUBMED | CROSSREF

14. Breschi L, Mazzoni A, Ruggeri A, Cadenaro M, Di Lenarda R, De Stefano Dorigo E. Dental adhesion review: aging and stability of the bonded interface. Dent Mater 2008;24:90-101.
PUBMED | CROSSREF

15. Zhang Z, Yu J, Yao C, Yang H, Huang C. New perspective to improve dentin-adhesive interface stability by using dimethyl sulfoxide wet-bonding and epigallocatechin-3-gallate. Dent Mater 2020;36:1452-1463.
PUBMED | CROSSREF

16. Kato MT, Leite AL, Hannas AR, Calabria MP, Magalhães AC, Pereira JC, Buzalaf MA. Impact of protease inhibitors on dentin matrix degradation by collagenase. J Dent Res 2012;91:1119-1123.
PUBMED | CROSSREF

17. Vidal CM, Aguiar TR, Phansalkar R, McAlpine JB, Napolitano JG, Chen SN, Araújo LS, Pauli GF, Bedran-Russo A. Galloyl moieties enhance the dentin biomodification potential of plant-derived catechins. Acta Biomater 2014;10:3288-3294.
PUBMED | CROSSREF

18. Zarella BL, Buzalaf MA, Kato MT, Hannas AR, Salo T, Tjäderhane L, Prakki A. Cytotoxicity and effect on protease activity of copolymer extracts containing catechin. Arch Oral Biol 2016;65:66-71.
PUBMED | CROSSREF

19. Mankovskaia A, Lévesque CM, Prakki A. Catechin-incorporated dental copolymers inhibit growth of Streptococcus mutans. J Appl Oral Sci 2013;21:203-207.
PUBMED | CROSSREF

20. Santiago SL, Osorio R, Neri JR, Carvalho RM, Toledano M. Effect of the flavonoid epigallocatechin-3-gallate on resin-dentin bond strength. J Adhes Dent 2013;15:535-540.
PUBMED

21. Du X, Huang X, Huang C, Wang Y, Zhang Y. Epigallocatechin-3-gallate (EGCG) enhances the therapeutic activity of a dental adhesive. J Dent 2012;40:485-492.
PUBMED | CROSSREF

22. Hiraishi N, Sono R, Sofiqul I, Yiu C, Nakamura H, Otsuki M, Takatsuka T, Tagami J. *In vitro* evaluation of plant-derived agents to preserve dentin collagen. Dent Mater 2013;29:1048-1054.
PUBMED | CROSSREF

23. Jackson JK, Zhao J, Wong W, Burt HM. The inhibition of collagenase induced degradation of collagen by the galloyl-containing polyphenols tannic acid, epigallocatechin gallate and epicatechin gallate. J Mater Sci Mater Med 2010;21:1435-1443.
PUBMED | CROSSREF

24. de Souza JC, Tedesco AC, Takahashi LA, Curylofo-Zotti FA, Souza-Gabriel AE, Corona SA. Influence of nanoparticulated chitosan on the biomodification of eroded dentin: clinical and photographic longitudinal analysis of restorations. J Mater Sci Mater Med 2021;32:11.
PUBMED | CROSSREF

25. Lynch CD, O'Sullivan VR, Dockery P, McGillycuddy CT, Sloan AJ. Hunter-Schreger Band patterns in human tooth enamel. J Anat 2010;217:106-115.
PUBMED | CROSSREF

26. De Moraes MD, Passos VF, Padovani GC, Bezerra LC, Vasconcelos IM, Santiago SL. Protective effect of green tea catechins on eroded human dentin: an *in vitro*/*in situ* study. Braz Oral Res 2021;35:e108.
PUBMED | CROSSREF

27. Costa CA, Passos VF, Neri JR, Mendonça JS, Santiago SL. Effect of metalloproteinase inhibitors on bond strength of a self-etching adhesive on erosively demineralized dentin. J Adhes Dent 2019;21:337-344.
PUBMED

28. Neri JR, Yamauti M, da Silveira FD, Mendonca JS, de Carvalho RM, Santiago SL. Influence of dentin biomodification with epigallocatechin-3-gallate on the bond strength of self-etch adhesive: twelve-month results. Int J Adhes Adhes 2016;71:81-86.
CROSSREF

29. Epasinghe DJ, Yiu CK, Burrow MF, Tay FR, King NM. Effect of proanthocyanidin incorporation into dental adhesive resin on resin-dentine bond strength. J Dent 2012;40:173-180.
PUBMED | CROSSREF

30. Hardan L, Daood U, Bourgi R, Cuevas-Suárez CE, Devoto W, Zarow M, Jakubowicz N, Zamarripa-Calderón JE, Radwanski M, Orsini G, Lukomska-Szymanska M. Effect of collagen crosslinkers on dentin bond strength of adhesive systems: a systematic review and meta-analysis. Cells 2022;11:2417.
PUBMED | CROSSREF

31. de Souza LC, Rodrigues NS, Cunha DA, Feitosa VP, Santiago SL, Reis A, Loguercio AD, Matos TP, Saboia VP, Perdigão J. Two-year clinical evaluation of proanthocyanidins added to a two-step etch-and-rinse adhesive. J Dent 2019;81:7-16.
PUBMED | CROSSREF

32. Yang H, Guo J, Deng D, Chen Z, Huang C. Effect of adjunctive application of epigallocatechin-3-gallate and ethanol-wet bonding on adhesive-dentin bonds. J Dent 2016;44:44-49.
PUBMED | CROSSREF

33. Costa C, Albuquerque N, Mendonça JS, Loguercio AD, Saboia V, Santiago SL. Catechin-based dentin pretreatment and the clinical performance of a universal adhesive: a two-year randomized clinical trial. Oper Dent 2020;45:473-483.
PUBMED | CROSSREF

34. Souza-Junior EJ, Prieto LT, Araújo CT, Paulillo LA. Selective enamel etching: effect on marginal adaptation of self-etch LED-cured bond systems in aged Class I composite restorations. Oper Dent 2012;37:195-204.
PUBMED | CROSSREF

35. Carvalho RV, Ogliari FA, de Souza AP, Silva AF, Petzhold CL, Line SR, Piva E, Etges A. 2-hydroxyethyl methacrylate as an inhibitor of matrix metalloproteinase-2. Eur J Oral Sci 2009;117:64-67.
PUBMED | CROSSREF

36. Gerhardt KM, Oliveira CA, França FM, Basting RT, Turssi CP, Amaral FL. Effect of epigallocatechin gallate, green tea extract and chlorhexidine application on long-term bond strength of self-etch adhesive to dentin. Int J Adhes Adhes 2016;71:23-27.
CROSSREF

37. Fialho MP, Hass V, Nogueira RP, França FM, Turssi CP, Basting RT, Amaral FL. Effect of epigallocatechin-3- gallate solutions on bond durability at the adhesive interface in caries-affected dentin. J Mech Behav Biomed Mater 2019;91:398-405.
PUBMED | CROSSREF

38. Nivedita L, Prakash V, Mitthra S, Pearlin Mary NS, Venkatesh A, Subbiya A. Evaluation of the effect of collagen stabilizing agents like chitosan and proanthocyanidin on the shear bond strength to dentin and microleakage of resin composite at enamel and cemental walls: an *in vitro* study. J Conserv Dent 2019;22:483-489.
PUBMED | CROSSREF

39. Srinivasulu S, Vidhya S, Sujatha M, Mahalaxmi S. Shear bond strength of composite to deep dentin after treatment with two different collagen cross-linking agents at varying time intervals. Oper Dent 2012;37:485-491.
PUBMED | CROSSREF