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# A long-term evaluation of experimental potassium oxalate concentrations on dentin hypersensitivity reduction: A triple-blind randomized clinical trial

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ABSTRACT

*Objective:* The aim of this split-mouth, triple-blind, randomized clinical trial was to evaluate the long-term clinical efficacy of experimental potassium oxalate concentration (10%) in relieving dentin hypersensitivity (DH), after a four-session application protocol.

*Methods:* Potassium oxalate gels with different concentrations (5 and 10%) were randomly assigned to half of the 31 patients from the sample in a split-mouth design. The desensitizers were applied following a four-session protocol, one session every 48 h. The primary outcome was the assessment of pain level with the visual analog scale (VAS, 0–10), at baseline, immediately after each desensitizing session, and also after the seventh day and along 1-,3-, 6-, 9- and 12-months follow-ups. Statistical analyses were performed using Friedman repeated measures and Wilcoxon signed rank tests ($\alpha = 0.05$).

*Results:* For both groups, the minimum of three sessions were required for the achievement of lower DH levels. Regardless of the concentration, the desensitizing effect was maintained all the way to the end of the 6-month follow-up. The 10%-potassium oxalate group was more effective for both 9 and 12-months follow-up periods ($p < 0.001$). No complications and adverse effects were observed.

*Conclusions:* When a four-session protocol is applied, both concentrations of potassium oxalate (5 and 10%) proved to be effective on DH reduction for up to six months. However, the higher concentration promoted better long-term results.

Clinical Significance: The DH is an increasing condition in clinical practice, which affects the patient's life quality. This study provides primary clinical evidence, suggesting that multiple application sessions and higher concentrations of potassium oxalate may result in maintenance of the desensitizing effect for more extended periods.

Trial registered under number: ClinicalTrials.gov NCT03083496.

## 1. Introduction

Over the last decades, the public health strategies and technological developments have led to the improvement of individuals' quality of life and to an increase in life expectancy [1,2]. This situation, associated with the awareness of the population concerning of oral hygiene, is promoting the maintenance of natural teeth in the oral cavity for a longer period [1,2]. In addition, the reduction in the incidence of caries (due to successful oral health prevention strategies), daily stressful routine and new eating habits (acidic and industrialized products) have led patients to seek treatment for diseases that are not related to microorganisms, such as noncarious cervical lesions and dentin hypersensitivity (DH) [3].

The DH is characterized as a brief and sharp pain caused by thermal, chemical, tactile, and evaporative stimuli. To date, several data supports a theory (hydrodynamic) that these types of stimuli can induce the flow of fluids inside the dentinal tubules, which triggers receptors near the pulp (mainly A-delta fibers) and results in painful sensations for the patient [4,5]. The prevalence of DH is considerably wide in adult populations, ranging from 1.3% [6] to 92.1% [7,8] and its etiology is

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multifactorial, which involve an association of factors: tension (promoted by parafunctional habits and traumatic occlusion), friction (by attrition or abrasion), and corrosion (chemical, biochemical and electrochemical degradation caused by acid of intrinsic and extrinsic sources) [3,9].

Plenty of agents with different mechanisms of action have been described and evaluated in DH management-related literature. The dentin desensitizers can be classified, according to their action, as neural (blocking neural responses, e.g. potassium nitrate and low-power lasers), tubule-blocking (obliterating the dentinal tubules, e.g. oxalates, glutaraldehyde, and high-power lasers) and, agents with both actions (e.g. potassium oxalate) [10–12].

Among the desensitizers, the potassium oxalate has been widely used in clinical practice, presenting satisfactory results, with no side effects [13–15]. This agent's mechanism of action is based on the obliteration of exposed dentin tubules (through the precipitation of calcium oxalate crystals) [16] and depolarization of the nerve endings [10]. Although a few studies have been carried out evaluating different potassium oxalate concentrations [17,18], to the best of the author's knowledge, no information is currently available regarding the long-term effect of this agent when a multiple-session protocol is applied.

The DH is a clinical condition that affects the population's quality of life [19,20]. Even though there is a large number of DH relief therapies, there is still no established protocol for an effective lasting treatment [21,22]. Therefore, this study aimed to evaluate the long-term efficacy of experimental potassium oxalate (10%) in the reduction of DH, after a four-session protocol application.

## 2. Materials and methods

### 2.1. Study design and ethics approval

This study was designed as an interventional, single-center, triple-blind (operators, patients and evaluator), split-mouth randomized clinical trial. The clinical investigation was approved (protocol #108076) by the local university's ethics committee. The research protocol was also registered at clinicaltrials.gov (#NCT03083496) and carried out according to the Consolidated Standards of Reporting Trials (CONSORT) guidelines with an extension for within-person designs [23]. The research was carried out at the NonCarious Cervical Lesions and Cervical Dentin Hypersensitivity Ambulatorial Rehabilitation Clinic of the local School of Dentistry from March 2017 to August 2018.

### 2.2. Recruitment and eligibility criteria

The recruitment occurred by means of a written advertisement posted at the local university's bulletin board. Patients were recruited in the order they showed up for screening, thus establishing a convenience sample. Before being enrolled in the study, all individuals were informed regarding its nature and objectives, and signed a written consent form.

Participants included in this study should be at least 18 years old, in good overall and oral health, and were required to have two teeth with DH in contralateral quadrants. Teeth with dental caries, restorations or fractures were excluded. Participants who underwent recent periodontal surgery or desensitizing treatment in the last three months and those with dental prostheses and orthodontics appliances or with symptoms of pulpitis were not included. Also, pregnant and lactating women, individuals with bruxism or any systemic/psychological disease, users of anti-inflammatory or analgesic medication, smokers, and patients undergoing tooth-whitening procedures were excluded.

### 2.3. Sample size calculation

The primary result obtained in this study was the DH level. A sample size calculation was performed on the website (www.sealedenvelop.com), using an alpha of 0.05 and 80%-power. Thus, the minimum sample size in this equivalence trial was 31 patients in order to detect a 30% difference in DH level between groups [24]. The sample size calculation was performed without accounting for the potential correlation coefficient between the paired treatment outcome. Published within-person trials do not report this correlation coefficient, and for this reason, the authors decided to adopt a conservative strategy.

### 2.4. Randomization and allocation concealment

The randomization process (simple random scheme) was carried out using computer-generated reports through www.sealedenvelope.com, which was operated by a researcher, not involved in the intervention and evaluation processes. The identification of the treatment to be applied on the right side of the patients' arches were kept concealed in sequentially-numbered, opaque and sealed envelopes, which were opened by the operator immediately before the procedures. The left side of the mouth was assigned to receive the other concentration of desensitizer. No negative control group was allowed by the ethics committee [25,26].

### 2.5. Blinding

This study consisted of a triple-blind clinical trial in which the patient, the operator and the and the evaluator were blinded to the group assignment. The randomization process, delivery and guidance on the administration of the gel were carried out by a researcher who was not involved neither in the application, nor the evaluation procedures. Both desensitizing gels (potassium oxalate 5 and 10%) had similar color and consistency and were delivered from identical tubes labeled as "A" and "B" so that it could not be identified by operators and patients. The coding structure was known by the research coordinator only.

### 2.6. Study intervention

The desensitizing procedure was carried out in all patients by one researcher with clinical experience. The desensitizers used in this study were synthesized as 5 and 10% potassium oxalate gels (Homeocenter, Ribeirão Preto, Brazil) and the protocol for the gel application was the same for both agents (Table 1). The teeth were cleaned with pumice and a rubber cup coupled in a slow-speed handpiece. The operating field was isolated with cotton rolls, suction, and a retraction cord #000 (Ultrapak, Ultradent, South Jordan) inserted into the gingival sulcus of the hypersensitive tooth. The operator placed the experimental

**Table 1**
Composition and application methods of the desensitizer agents.

| Agents | Composition | Application Method |
|---|---|---|
| 5%-Potassium Oxalate | Potassium oxalate monohydrate 2.266 g; lauryl sulfate 0.299 g; glycerin 4.989 g; sorbitol 7.484; benzoate sodium 0.125; sucralose 0.050 g; aristoflex gel 36.947 g | 1 Prophylaxis with an oil-free product<br>2 To apply the agent uniformly on the cervical region of the tooth (using a micro-applicator), rub for 10 s and wait for 10 min.<br>3 To remove the gel from the teeth with cotton and plenty of water |
| 10%-Potassium Oxalate | Potassium oxalate monohydrate 5.252 g; lauryl sulfate 0.284 g; glycerin 4.972 g; sorbitol 7.090; benzoate sodium 0.118; sucralose 0.047 g; aristoflex gel 35.003 g | |

concentration (10%) on one side of the arch, and the other side received the gel (5%). The side of the product application was randomly defined.

Each gel was applied to the cervical area of the teeth during one minute with friction movements and left undisturbed in contact with the dental structure for ten minutes. The gel was removed with a suction tip and water. Four desensitizing sessions were performed every 48 h. All participants received oral hygiene recommendations and were requested not to use desensitizing or bleaching toothpaste during the study.

### 2.7. Dentin hypersensitivity level assessment

An evaporative stimulus (controlled air blast), generated by a three-way syringe, was used to determine dentin hypersensitivity levels. The air was directed perpendicularly to the cervical buccal surface of the hypersensitive tooth for two seconds from a distance of 1 cm. Adjacent teeth were protected with cotton rolls to avoid false positive results. After the stimulus, the evaluator requested the participants to quantify their pain, according to a visual analog scale (VAS) [27]. The used VAS scale was a 10-cm horizontal line, where 0 = no sensitivity and 10 = severe sensitivity. To ensure the application of the same stimulus during the study, the three-way syringe was constantly calibrated at 25–28 psi [28] and the evaluator was previously instructed to apply the same pressure during the assessments. The efficacy of agents was measured at baseline and immediately after each application session. The participants attended appointments after one week and 1, 3, 6, 9 and 12 months from treatment and the DH levels were measured using the same previously described procedures. All measuring was carried out by the same evaluator.

### 2.8. Statistical analyses

Data distribution normality was checked with the Kolmogorov-Smirnov test. In this study, nonparametric tests were used, as the data did not present normal distribution. The analyses at different periods in each group were conducted using the Friedman repeated measures test and *posthoc* Tukey test. For comparisons among groups at each assessment point, the Wilcoxon sign-rank test was applied. The data analysis was performed by using the statistical software Sigma Plot version 12.0 (Systat Software Inc, San Jose, California), and the level of significance was determined as $\alpha = 0.05$.

## 3. Results

Thirty-one subjects were enrolled in this study, and their baseline characteristics are presented in Table 2. All participants attended the recall visits and completed the 12-month trial period (Fig. 1). For both groups, only one session was necessary to promote a significant DH reduction in comparison with the baseline. At least three sessions were required to achieve the lower levels of DH found in the study. No significant difference in the DH was observed between the groups when the number of application sessions was compared (Table 3). Regardless of potassium oxalate concentration, the desensitizing effect was maintained until the 6-month follow-up (Table 4). On the other hand, the group treated with 10% potassium oxalate showed better desensitizing effects for both 9 and 12-months timepoints when compared with 5% ($p < 0.001$) (Table 4). No complications, such as the presence of spontaneous pain and allergic reactions, were observed throughout the study.

**Table 2**
Characteristics of the participants and the distribution of teeth.

| Characteristic | Total |
|---|---|
| Sex | |
| Male | 12 |
| Female | 19 |
| Age (years) | |
| 18-25 | 10 |
| 26-35 | 9 |
| 36-45 | 4 |
| > 45 | 8 |
| Tooth type | **Group A/B** |
| Central incisors | 3/7 |
| Lateral incisors | 4/3 |
| Canines | 3/2 |
| First premolars | 10/10 |
| Second premolars | 3/5 |
| First molars | 5/3 |
| Second molars | 3/2 |

## 4. Discussion

This trial was designed to evaluate the clinical behavior of a different potassium oxalate concentration in an attempt to find an effective long-term treatment for DH relief.

Oxalate-based agents were introduced as desensitizing agents between the 1970s and the 1980s [5,29–31] and since then, they have been well accepted by practitioners [32], demonstrating satisfactory results in the reduction of DH [17,30,31,33,34]. Several *in vitro* studies have reported significant decreases in hydraulic conductance across dentinal tubules treated with oxalates, suggesting that this kind of desensitizer limits fluid flow in exposed dentin due to their ability to promote the precipitation of insoluble calcium oxalate crystals on the surface and inside dentin tubules walls [35–37]. The oxalates can reduce more than 98% of the dentin permeability [38,39] and to promote the formation of calcium oxalate crystals 30 s after their application [31], which leads to immediate relief of DH levels [17,18]. When oxalate (oxalic acid) is associated with potassium (potassium hydroxide) [40], it becomes a combined agent, with mixed action. Therefore, the potassium oxalate presents the capabilities of a neural as well as tubule-blocking agent in a single product. In this situation, the oxalate acts initially as a carrier, enabling the potassium to contact and promotes the depolarization of the odontoblast endings, favoring the long-term effectiveness of the agent [10].

In this study, 5 and 10% potassium oxalates were selected to be evaluated. The 5% was chosen because it is the highest concentration of potassium oxalate desensitizing gel available on the market, and the experimental 10% was tested as an attempt to improve the efficacy of this agent. Both concentrations of potassium oxalate presented a desensitizing effect until the 6-month evaluation, which can be explained by the action mechanism described above. Also, it is worthy of note that calcium oxalate crystals appear among the less soluble salts, with relative insolubility in acid and solubility almost comparable with the one of dentin hydroxyapatite [16], which makes them resistant to dissolution [16,18].

At least three sessions were necessary to achieve the lowest levels of DH in the study. Probably, only a single application may not be sufficient to induce the adequate precipitation of calcium oxalate crystals, which suggest that a multiple sessions approach might result in maintenance of the desensitizing effect for more prolonged periods [28,41,42]. Even though a four-session protocol has been applied, for 9 and 12 months timepoints, 10% potassium oxalate promoted better results when compared with 5%. The literature reported that the size and area of precipitated crystals depends on the active agent concentration, which may subsequently affect the desensitizer's occlusive power in a long-term evaluation, supporting the results found in this study [17,30].

In order to assess the DH levels, at the baseline and follow-up sessions, an evaporative stimulus (air blast) was applied. This type of stimulus has been recommended in literature for years [26,41,43–45] and acts promoting the evaporation of the fluid from the inside the tubules and reducing the dentin surface temperature. This fact reduces the

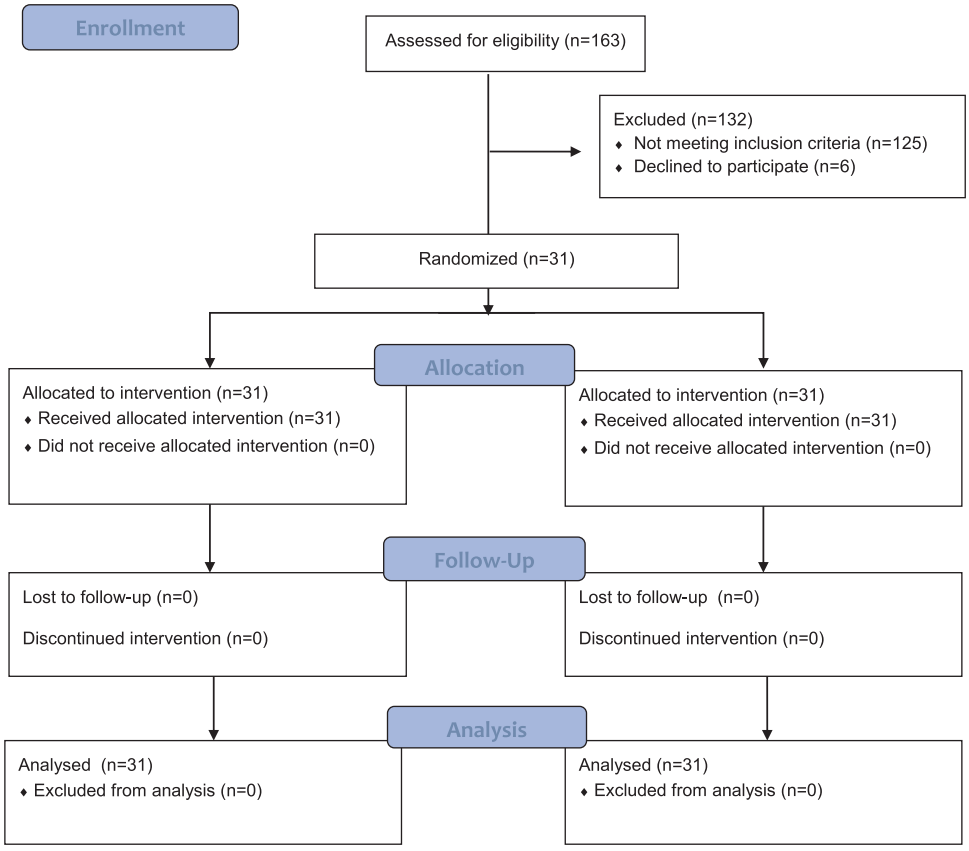


**Fig. 1.** CONSORT flow diagram of the clinical trial.

**Table 3**
Dentin hypersensitivity levels for baseline and after each application session.

| Treatment/ Assessment point | Potassium oxalate 10% | | Potassium oxalate 5% | | *p*-value** |
|---|---|---|---|---|---|
| | Mean ( ± SD) | Median (interquartile range) | Mean ( ± SD) | Median (interquartile range) | |
| **Baseline** | 8.16 ± 1.65 | 8 (7-10) Aa | 7.93 ± 1.73 | 8 (7-10) Aa | 0.46 |
| **Session 1** | 5.03 ± 2.54 | 5 (4-8) Ab | 4.65 ± 3.14 | 5 (2-7) Ab | 0.43 |
| **Session 2** | 3.00 ± 2.72 | 3 (0-6) Abc | 3.03 ± 3.27 | 2 (0-6) Abc | 0.95 |
| **Session 3** | 1.77 ± 2.34 | 1 (0-3) Acd | 2.32 ± 3.07 | 0 (0-5) Ac | 0.45 |
| **Session 4** | 0.83 ± 1.69 | 0 (0-2) Ad | 1.80 ± 2.82 | 0 (0-3) Ac | 0.12 |
| ***p*-value*** | < 0.001 | | < 0.001 | | |

* Friedman Repeated Measures and Tukey test for comparison of pain levels between assessment points, for the same treatment group. Values followed by the same lower-case letter (columns) are statistically similar ($p > 0.05$).

** Wilcoxon sign-rank test for comparison of pain levels between treatments groups, in each assessment point. Values followed by the same upper-case letter (lines) are statistically similar ($p > 0.05$).

difference of pressure inside the tubule and consequently triggers the receptors in the pulp, causing the painful sensation [46]. After the stimulus, the DH level was determined using VAS. This evaluation type was employed due to being regarded as an adequate and reproducible method that is easily understood by patients [47,48]. The advantage of this method is that it consists of a continuous numerical scale with easy application, allowing the conversion of the subjective response into objective data [44].

This RCT was performed under rigorous randomization control, avoiding conscious and subconscious interventions and preventing allocation bias. Adequate blinding avoided performance and detection bias by both operators and participants. A split-mouth design was selected so that within-patient and tooth-related variables could be controlled. Within-paired designs allow the use of robust statistical methods of analysis which take advantage of repeated measures and promote a reduction in the variability within a subject [49] as each individual serves as their own control [50]. The carry-across effect is the most worrisome aspect for this type of design. For this reason, in this study, only teeth located in contralateral quadrants were included, retraction cords were used to isolate the field during the desensitizing procedure [51] and agents with great thixotropy were applied in order to avoid cross over possibilities. In addition, due to the professional application of the agents, the desensitizers' topical action, and the fact that patients can differentiate pain in both sides of the mouth, the cross over possibility were not a concern for this trial.

In this study, only one type of desensitizer (potassium oxalate) was tested. Future studies evaluating the number of application sessions with larger sample sizes for different agents and concentrations are required in order to reconfirm the findings of this study and to clarify the stability and longevity of each agent.

**Table 4**
Dentin hypersensitivity levels for each follow-up timepoint, according to each group.

| Treatment/ Assessment point | Potassium oxalate 10% | | Potassium oxalate 5% | | *p*-value** |
|---|---|---|---|---|---|
| | Mean ( ± SD) | Median (interquartile range) | Mean ( ± SD) | Median (interquartile range) | |
| **AT** | 0.83 ± 1.69 | 0 (0-2) Aa | 1.80 ± 2.82 | 0 (0-3) Aa | 0.12 |
| **1 week** | 0.90 ± 1.70 | 0 (0-2) Aa | 2.19 ± 2.99 | 0 (0-5) Ab | 0.045 |
| **1 month** | 1.00 ± 1.73 | 0 (0-2) Aa | 2.25 ± 3.07 | 0 (0-5) Abc | 0.074 |
| **3 months** | 1.01 ± 1.73 | 0 (0-2) Aa | 2.26 ± 3.07 | 0 (0-5) Ac | 0.074 |
| **6 months** | 1.01 ± 1.73 | 0 (0-2) Aa | 2.26 ± 3.07 | 0 (0-5) Ac | 0.074 |
| **9 months** | 2.74 ± 1.21 | 2 (2-3) Ab | 4.93 ± 1.80 | 5 (4-6) Bb | < 0.001 |
| **12 months** | 3.32 ± 1.35 | 3 (2-4) Ab | 6.80 ± 1.74 | 7 (5-8) Bb | < 0.001 |
| ***p*-value*** | < 0.001 | | < 0.001 | | |

**AT.** After treatment.

* Friedman Repeated Measures and Tukey test for comparison of pain levels between assessment points, for the same treatment group. Values followed by the same lower-case letter (columns) are statistically similar ($p > 0.05$).

** Wilcoxon sign-rank test for comparison of pain levels between treatments groups in each assessment point. Values followed by the same upper-case letter (lines) are statistically similar ($p > 0.05$).

## 5. Conclusion

Within the limitations of this study and in the absence of a negative control group, it may be concluded that when a four-session protocol is applied, both 5 and 10%-potassium oxalates can be considered effective in the DH reduction for at least six months. However, the highest concentration promoted better results in the long-term evaluation.

## Declaration of Competing Interest

The authors declare no conflict of interest.

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## References

[1] A. Lussi, T.S. Carvalho, Erosive tooth wear: a multifactorial condition of growing concern and increasing knowledge, Monogr. Oral Sci. 25 (2014) 1–15.
[2] R. Orchardson, L.P. Gangarosa, G.R. Holland Sr., D.H. Pashley, H.O. Trowbridge, F.P. Ashley, I. Kleinberg, U. Zappa, Dentine hypersensitivity-into the 21st century, Arch. Oral Biol. 39 (Suppl) (1994) 113S–119S.
[3] J.O. Grippo, M. Simring, T.A. Coleman, Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20-year perspective, J. Esthet. Restor. Dent. 24 (1) (2012) 10–23.
[4] M. Brannstrom, The surface of sensitive dentine. An experimental study using replication, Odontol. Revy 16 (4) (1965) 293–299.
[5] D.H. Pashley, M.J. Livingston, O.W. Reeder, J. Horner, Effects of the degree of tubule occlusion on the permeability of human dentine in vitro, Arch. Oral Biol. 23 (12) (1978) 1127–1133.
[6] C.T. Bamise, A.O. Olusile, A.O. Oginni, O.O. Dosumu, The prevalence of dentine hypersensitivity among adult patients attending a Nigerian teaching hospital, Oral Health Prev. Dent. 5 (1) (2007) 49–53.
[7] M.B. Chabanski, D.G. Gillam, J.S. Bulman, H.N. Newman, Clinical evaluation of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department: a pilot study, J. Oral Rehabil. 24 (9) (1997) 666–672.
[8] L.F. Zeola, P.V. Soares, J. Cunha-Cruz, Prevalence of dentin hypersensitivity: systematic review and meta-analysis, J. Dent. 81 (2019) 1–6.
[9] H. Hajizadeh, A. Nemati-Karimooy, S. Majidinia, A. Moeintaghavi, M. Ghavamnasiri, Comparing the effect of a desensitizing material and a self-etch adhesive on dentin sensitivity after periodontal surgery: a randomized clinical trial, Restor. Dent. Endod. 42 (3) (2017) 168–175.
[10] J.S. Assis, L.K. Rodrigues, C.S. Fonteles, R.C. Colares, A.M. Souza, S.L. Santiago, Dentin hypersensitivity after treatment with desensitizing agents: a randomized, double-blind, split-mouth clinical trial, Braz. Dent. J. 22 (2) (2011) 157–161.
[11] A. Davari, E. Ataei, H. Assarzadeh, Dentin hypersensitivity: etiology, diagnosis and treatment; a literature review, J. Dent. 14 (3) (2013) 136–145.
[12] E. Oncu, S. Karabekiroglu, N. Unlu, Effects of different desensitizers and lasers on dentine tubules: an in-vitro analysis, Microsc. Res. Tech. 80 (7) (2017) 737–744.
[13] D.G. Gillam, H.S. Seo, H.N. Newman, J.S. Bulman, Comparison of dentine hypersensitivity in selected occidental and oriental populations, J. Oral Rehabil. 28 (1) (2001) 20–25.
[14] J. Pereira, A. Martineli, S. Santiago, Treating hypersensitive dentin with three different potassium oxalate-based gel formulations: a clinical study, J. Appl. Oral Sci. 9 (2001) 123–130.
[15] S. Sauro, M.G. Gandolfi, C. Prati, R. Mongiorgi, Oxalate-containing phytocomplexes as dentine desensitisers: an in vitro study, Arch. Oral Biol. 51 (8) (2006) 655–664.
[16] E.M. Varoni, T. Zuccheri, A. Carletta, B. Palazzo, A. Cochis, M. Colonna, L. Rimondini, In vitro efficacy of a novel potassium oxalate hydrogel for dentin hypersensitivity, Eur. J. Oral Sci. 125 (2) (2017) 151–159.
[17] K.B. Muzzin, R. Johnson, Effects of potassium oxalate on dentin hypersensitivity in vivo, J. Periodontol. 60 (3) (1989) 151–158.
[18] J.C. Pereira, A.D. Segala, D.G. Gillam, Effect of desensitizing agents on the hydraulic conductance of human dentin subjected to different surface pre-treatments–an in vitro study, Dent. Mater. 21 (2) (2005) 129–138.
[19] O.V. Boiko, S.R. Baker, B.J. Gibson, D. Locker, F. Sufi, A.P. Barlow, P.G. Robinson, Construction and validation of the quality of life measure for dentine hypersensitivity (DHEQ), J. Clin. Periodontol. 37 (11) (2010) 973–980.
[20] D.W. Douglas-de-Oliveira, G.P. Vitor, J.O. Silveira, C.C. Martins, F.O. Costa, L.O.M. Cota, Effect of dentin hypersensitivity treatment on oral health-related quality of life - A systematic review and meta-analysis, J. Dent. 71 (2018) 1–8.
[21] V. Moraschini, L.S. da Costa, G.O. Dos Santos, Effectiveness for dentin hypersensitivity treatment of non-carious cervical lesions: a meta-analysis, Clin. Oral Investig. 22 (2) (2018) 617–631.
[22] I.C. Porto, A.K. Andrade, M.A. Montes, Diagnosis and treatment of dentinal hypersensitivity, J. Oral Sci. 51 (3) (2009) 323–332.
[23] N. Pandis, B. Chung, R.W. Scherer, D. Elbourne, D.G. Altman, CONSORT 2010 statement: extension checklist for reporting within person randomised trials, BMJ 30 (2017) 357–j2835.
[24] M.E. Wewers, N.K. Lowe, A critical review of visual analogue scales in the measurement of clinical phenomena, Res. Nurs. Health 13 (4) (1990) 227–236.
[25] A.O. Lopes, C. de Paula Eduardo, A.C.C. Aranha, Evaluation of different treatment protocols for dentin hypersensitivity: an 18-month randomized clinical trial, Lasers Med. Sci. 32 (5) (2017) 1023–1030.
[26] C.R. Torres, T.M. Silva, B.M. Fonseca, A.L. Sales, P. Holleben, R. Di Nicolo, A.B. Borges, The effect of three desensitizing agents on dentin hypersensitivity: a randomized, split-mouth clinical trial, Oper. Dent. 39 (5) (2014) E186–94.
[27] L.C. Gentile, S.L. Greghi, Clinical evaluation of dentin hypersensitivity treatment with the low-intensity Gallium-Aluminum-Arsenide laser - AsGaAl, J. Appl. Oral Sci. 12 (4) (2004) 267–272.
[28] G.F. Moura, L.F. Zeola, M.B. Silva, S.C. Sousa, F.R. Guedes, P.V. Soares, Four-session protocol effectiveness in reducing cervical dentin hypersensitivity: a 24-Week randomized clinical trial, Photobiomodul. Photomed. Laser Surg. 37 (2) (2019) 117–123.
[29] J.D. Greenhill, D.H. Pashley, The effects of desensitizing agents on the hydraulic conductance of human dentin in vitro, J. Dent. Res. 60 (3) (1981) 686–698.
[30] D.H. Pashley, S.E. Galloway, The effects of oxalate treatment on the smear layer of ground surfaces of human dentine, Arch. Oral Biol. 30 (10) (1985) 731–737.
[31] D.H. Pashley, J.A. O'Meara, E.E. Kepler, S.E. Galloway, S.M. Thompson, F.P. Stewart, Dentin permeability. Effects of desensitizing dentifrices in vitro, J. Periodontol. 55 (9) (1984) 522–525.
[32] J. Cunha-Cruz, J.C. Wataha, L. Zhou, W. Manning, M. Trantow, M.M. Bettendorf, L.J. Heaton, J. Berg, Treating dentin hypersensitivity: therapeutic choices made by dentists of the northwest PRECEDENT network, J. Am. Dent. Assoc. 141 (9) (2010) 1097–1105.
[33] S.L. Santiago, J.C. Pereira, A.C. Martineli, Effect of commercially available and experimental potassium oxalate-based dentin desensitizing agents in dentin permeability: influence of time and filtration system, Braz. Dent. J. 17 (4) (2006)

300–305.
[34] T. Suge, A. Kawasaki, K. Ishikawa, T. Matsuo, S. Ebisu, Effects of pre- or post-application of calcium chloride on occluding ability of potassium oxalate for the treatment of dentin hypersensitivity, Am. J. Dent. 18 (2) (2005) 121–125.
[35] M.F. Cuenin, M.J. Scheidt, R.B. O'Neal, S.L. Strong, D.H. Pashley, J.A. Horner, T.E. Van Dyke, An in vivo study of dentin sensitivity: the relation of dentin sensitivity and the patency of dentin tubules, J. Periodontol. 62 (11) (1991) 668–673.
[36] H.J. Shiau, Dentin hypersensitivity, J. Evid. Dent. Pract. 12 (3 Suppl) (2012) 220–228.
[37] O. Thanatvarakorn, S. Nakashima, A. Sadr, T. Prasansuttiporn, M. Ikeda, J. Tagami, In vitro evaluation of dentinal hydraulic conductance and tubule sealing by a novel calcium-phosphate desensitizer, J. Biomed. Mater. Res. B Appl. Biomater. 101 (2) (2013) 303–309.
[38] M.F. Morris, R.D. Davis, B.W. Richardson, Clinical efficacy of two dentin desensitizing agents, Am. J. Dent. 12 (2) (1999) 72–76.
[39] F.L. Pillon, I.G. Romani, E.R. Schmidt, Effect of a 3% potassium oxalate topical application on dentinal hypersensitivity after subgingival scaling and root planing, J. Periodontol. 75 (11) (2004) 1461–1464.
[40] R. Orchardson, D.G. Gillam, Managing dentin hypersensitivity, J. Am. Dent. Assoc. 137 (7) (2006) 990–998 quiz 1028-9.
[41] S. Freitas Sda, L.L. Sousa, J.M. Moita Neto, R.F. Mendes, R.R. Prado, Dentin hypersensitivity treatment of non-carious cervical lesions - a single-blind, split-mouth study, Braz. Oral Res. 29 (2015) 45.
[42] S. Joshi, A.S. Gowda, C. Joshi, Comparative evaluation of NovaMin desensitizer and Gluma desensitizer on dentinal tubule occlusion: a scanning electron microscopic study, J. Periodontal Implant Sci. 43 (6) (2013) 269–275.
[43] S. Miglani, V. Aggarwal, B. Ahuja, Dentin hypersensitivity: recent trends in management, J. Conserv. Dent. 13 (4) (2010) 218–224.
[44] A.H. Vieira, V.F. Passos, J.S. de Assis, J.S. Mendonca, S.L. Santiago, Clinical evaluation of a 3% potassium oxalate gel and a GaAlAs laser for the treatment of dentinal hypersensitivity, Photobiomodul. Photomed. Laser Surg. 27 (5) (2009) 807–812.
[45] X. Yu, B. Liang, X. Jin, B. Fu, M. Hannig, Comparative in vivo study on the desensitizing efficacy of dentin desensitizers and one-bottle self-etching adhesives, Oper. Dent. 35 (3) (2010) 279–286.
[46] T. Pamir, H. Dalgar, B. Onal, Clinical evaluation of three desensitizing agents in relieving dentin hypersensitivity, Oper. Dent. 32 (6) (2007) 544–548.
[47] C. Corral, P.V. Grez, M. Letelier, E.A. Dos Campos, A.L. Dourado, G.E. Fernandez, Effect of oxalic acid-based desensitizing agent on cervical restorations on hypersensitive teeth: a triple-blind randomized controlled clinical trial, J. Oral Facial Pain Headache 30 (4) (2016) 330–337.
[48] L. Wang, A.C. Magalhaes, L.F. Francisconi-Dos-Rios, M.P. Calabria, D. Araujo, M. Buzalaf, J. Lauris, J.C. Pereira, Treatment of dentin hypersensitivity using nano-hydroxyapatite pastes: a randomized three-month clinical trial, Oper. Dent. 41 (4) (2016) E93–E101.
[49] A.A. Antczak-Bouckoms, J.F. Tulloch, C.S. Berkey, Split-mouth and cross-over designs in dental research, J. Clin. Periodontol. 17 (7 Pt. 1) (1990) 446–453.
[50] R. Shetty, A.N. Bhat, D. Mehta, W.J. Finger, Effect of a calcium phosphate desensitizer on pre- and postcementation sensitivity of teeth prepared for full-coverage restorations: a randomized, placebo-controlled clinical study, Int. J. Prosthodont. 30 (1) (2017) 38–42.
[51] J. Cunha-Cruz, J.R. Stout, L.J. Heaton, J.C. Wataha, N. PRECEDENT, Dentin hypersensitivity and oxalates: a systematic review, J. Dent. Res. 90 (3) (2011) 304–310.