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Etiology and Management of Whitening-induced Tooth Hypersensitivity

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ABSTRACT Tooth hypersensitivity has long been, and continues to be, the most commonly reported adverse effect of vital tooth whitening with peroxide gels. The complex etiology of whitening-induced tooth hypersensitivity has been a major obstacle in developing a definitive strategy for its prevention. This article reviews the multiple etiologic factors implicated in whitening-induced tooth hypersensitivity and the evidence for efficacy of various strategies for its management.

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At-home vital tooth bleaching — the dentist-dispensed/dentist-supervised use of a carbamide peroxide gel in a custom-fitted tray to whiten teeth — was first described in 1989.[1] Since that time, an extensive body of evidence validating the safety and efficacy of this procedure has been established.[2] As widespread use of the technique became commonplace, however, reports/descriptions of the side effects of gingival irritation and tooth hypersensitivity became commonplace as well.[3-8] Sensitivity is the most frequently reported side effect for home tray whitening.[9] It will commonly manifest itself as generalized hypersensitivity to cold stimuli, but often also occurs as a spontaneous sharp shooting pain or "zinger" limited to one or a few teeth.[10]

Gingival irritation during tray tooth whitening is typically caused by prolonged contact of the peroxide gel with gingival tissues and can be prevented by proper trimming of the tray such that contact with the gel is limited to hard tooth surfaces.[11] A definitive understanding of the tooth hypersensitivity issue however, has been far more elusive, and, as such remains the most common adverse event associated with whitening of vital teeth.[9,12] Reports and estimates of whitening-induced tooth hypersensitivity incidence range from 0 percent to 100 percent, but are more commonly in the 60 percent range, and the degree of hypersensitivity in these reports ranges from very mild to intolerable.[5,8,13,14,16,17] Results of a clinical trial of a 15 percent CP gel by Jorgensen and Carroll were more specific, concluding that about half of all patients who undergo whitening will experience mild sensitivity; 10 percent will have moderate sensitivity; and 4 percent will have severe sensitivity.[18] Schulte et al. reported that 14 percent of subjects in their clinical trial discontinued treatment due to intolerable sensitivity.[5] Despite the fact that the hypersensitivity is transient (sensitivity levels typically return to normal upon completion of, if not during, the whitening treatment), it continues to render peroxide vital tooth whiten-

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ing unavailable to patients who experience severe, intolerable hypersensitivity during the procedure.[8,16,19,20] For other patients experiencing less severe symptoms, their discomfort during whitening may nonetheless adversely effect their compliance with the procedure, and thus produce suboptimal results. Efforts to fully elucidate the etiology of and develop strategies to manage or eliminate this side effect thus continue at robust levels.

Etiology

Current understanding of the etiology of peroxide whitening-induced tooth hypersensitivity is constructed around three fundamental concepts: Brännström's hydrodynamic theory of tooth pain; dentinal fluid outflow caused by osmotic stimuli; and permeation during whitening of peroxides through enamel and dentin and into the pulp. Brännström's well-known theory posits that dentinal fluid expands, contracts, or flows within dentinal tubules under the influence of thermal, evaporative, or osmotic changes and stimulates pressure-sensitive nerve receptors (A-δ fibers) that transmit the stimulus and produce the perception of pain.[21,22] Cold is reportedly the most common stimulus for dentin hypersensitivity.[22,23] While this conceptual construct is widely accepted, it is important to remember that tooth sensitivity during whitening is a multifactorial phenomenon and not exclusively dependent on the use of a whitening product.[12]

An understanding of the chemical entities present during vital tooth whitening is essential in order to consider the potential of each as an etiologic agent in tooth hypersensitivity. Key ingredients in whitening gels are typically carbamide peroxide ($CH_6N_2O_3$) or hydrogen peroxide (H_2O_2), glycerin or propylene glycol as the carrier, carbopol — a water-soluble polymer thickening agent common in household products such as toothpaste, shampoo, and flavorings. A slightly acidic pH improves stability (shelf life) of these products, so small amounts of phosphoric or citric acid are commonly added as well.[24] Carbamide peroxide dissociates in saliva into hydrogen peroxide and urea ($Ca[NH_2]_2$). Urea breaks down into ammonia (NH_3) and carbon dioxide (CO_2). Hydrogen peroxide, considered the active whitening ingredient, penetrates into tooth structure and breaks down into oxygen (O_2) and water (H_2O). The released oxygen oxidizes pigment molecules in enamel and dentin to produce the whitening effect.[24]

PENETRATION OF PEROXIDE

through enamel and dentin and into the pulp during tooth whitening has been reported.

Penetration of peroxide through enamel and dentin and into the pulp during tooth whitening has been reported.[25-27] Within five to 15 minutes after application of whitening gel, peroxide penetrates to the pulp where it irritates nerves and essentially produces a reversible pulpitis.[10,27] This etiology, then, can function independently of any osmotic effects on dentin (discussed later in this article) by whitening products, and may account for the common occurrence of sensitivity in patients with no gingival recession or other sites of exposed dentin.

Amounts of peroxide measured in pulps of extracted teeth after simulated 14-day overnight treatment with 3.5 percent, 6 percent, and 12 percent hydrogen peroxide were microgram quantities, with no differences by peroxide concentration or by four-hour versus seven-hour/night regimens.[28] Inasmuch as milligram amounts of peroxide in the pulp are necessary to inhibit pulpal enzymes, available evidence strongly indicates that peroxide from tooth whitening will, at worst, cause only irreversible pulpal irritation.[29]

Histological features of peroxide-whitened teeth further corroborate the common clinical observation that any whitening-induced pulpal changes are reversible.[30-32] Pulpal irritation evident histologically during treatment typically resolves within two weeks of terminating treatment.[32] Studies of long-term (six-month) treatment of tetracycline-stained teeth with at-home tray whitening similarly do not reveal nonresolving pulpal changes or sensitivity symptoms.[33] Furthermore, sensitivity levels reported for tetracycline-stained teeth during long-term whitening are not greater than those for teeth in normal whitening protocols.[33]

Carbamide peroxide allows for a time-release application of hydrogen peroxide and as such has been suggested as having less potential to produce sensitivity.[10] This necessitates, however, longer tray application times for carbamide peroxide versus hydrogen peroxide home whitening products. Ten percent and 20 percent carbamide peroxide break down to release 3.35 percent and 7 percent hydrogen peroxide, respectively. Carbamide peroxide releases 50 percent of its hydrogen peroxide in the first two to four hours of tray application, and the remainder over next two to six hours.[34] Instructions for carbamide peroxide products thus commonly recommend the options of applying the gel in

the tray for two hours during the day or overnight while asleep. While several studies report no significant difference in sensitivity when using 16 percent versus 10 percent carbamide peroxide, these same studies acknowledge a trend toward higher incidence of sensitivity with higher concentrations of carbamide peroxide, and others demonstrate a correlation between sensitivity levels and carbamide peroxide concentration.[20,35,36] It is also noteworthy that while the higher concentration products may shorten overall treatment time by a few days, this relationship is not linear (e.g., 20 percent does not whiten twice as fast at 10 percent).[10]

Dentist-dispensed whitening gels containing 5 percent to 10 percent hydrogen peroxide are marketed with the purported advantage of shorter application time per dose versus carbamide peroxide. Hydrogen peroxide releases all of its oxygen in 30 to 60 minutes of tray application, hence the shorter application times in instructions for use for hydrogen peroxide-containing products.[37] This rapid perfusion of peroxide into the pulp, however, is cited as producing higher sensitivity levels than an equivalent dose of carbamide peroxide.[10]

The aforementioned effects of peroxides on teeth would also account for sensitivity observed with over-the-counter whitening products, which typically contain 5 percent to 6 percent hydrogen peroxide.[38,39] Like dentist-dispensed gels containing hydrogen peroxide, OTC adhesive whitening strips have comparable whitening efficacy with less contact time compared to 10 percent carbamide peroxide in a custom tray.[40] Greater sensitivity has been reported with strips containing higher versus lower concentrations of hydrogen peroxide despite the shorter application time for the former.[41] Betke et al. also reported dentin desiccation by an OTC paint-on whitener containing ethanol but no glycerin.[42]

The actual whitening process, oxidation of organic pigments, may contribute to sensitivity via dehydration. It has been hypothesized that some of the apparent whitening effect perceived immediately upon completion of treatment is due to saturation of the tooth with oxygen altering the refractive index of enamel and dehydration of the tooth from the oxidative process.[24,43] As residual oxygen dissipates over the two weeks following completion of treatment, the actual lightened shade becomes apparent.[44] Rehydration during this period, in addition to restoring the normal optical properties of the teeth and contributing to stabilization of the lightened shade, likely facilitates the re-establishment of osmotic equilibrium in the teeth and resolution of hypersensitivity.

THE ACTUAL WHITENING process, oxidation of organic pigments, may contribute to sensitivity via dehydration.

Numerous studies investigating the efficacy of at-home whitening report sensitivity among subjects in placebo groups ranging from 20 percent to 35 percent, suggesting that peroxide is not the sole etiologic agent.[20,45,46] Placebo formulations typically consist of the whitening gel minus any peroxides. Gel composition and tray fit have thus also been implicated and investigated as possible contributors to whitening-induced sensitivity.

The potential of whitening gel ingredients to act as osmotic stimuli on dentin has been extensively studied. While carbopol is a benign substance in this regard, glycerin — also a common ingredient in many whitening gels — is a desiccant that can cause pain via dentinal fluid outflow.[18] Because of its anhydrous nature and potential for tooth dehydration, glycerin was implicated as the causative agent in whitening-induced tooth sensitivity. Several manufacturers have thus replaced glycerin with water-based solutions or formulations with added water, or have substituted propylene glycol for glycerin as gel base inasmuch as this may also reduce sensitivity.[20,47,48] These strategies were initially based on a presumed desiccating effect of glycerin on dentin, but this effect was eventually directly demonstrated in vitro.[42]

Tray pressure as a cause of sensitivity has been suggested by findings such as those by Leonard et al. who reported that 20 percent of participants in a clinical trial experienced sensitivity when wearing the tray alone.[20] A tightly adapted tray can theoretically apply active orthodontic forces effecting small degrees of tooth movement and thus giving rise to mild levels of sensitivity. Trays are less rigid now versus early years of tray whitening and are less likely to exert orthodontic forces on teeth.[15] Additionally, a tray design using reservoirs on the labial surfaces was presumed to both reduce the tray-induced sensitivity via a relaxed fit and to improve efficacy by holding a larger volume of whitening gel against each tooth. Reported evidence of these effects by reservoirs is equivocal, however, with several studies finding no difference in sensitivity or color change when comparing trays with and without reservoirs.[20,49]

Crim reported that a carbamide

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peroxide whitening gel adversely affected the marginal seal of class V restorations at the cementoenamel junction in vitro.[50] Evidence of increased microleakage or accelerated failure of restorations subjected to at-home whitening is otherwise absent in the literature as well as anecdotally. Available clinical data similarly do not report differences in whitening-induced hypersensitivity between restored and unrestored teeth. Teeth with carious lesions and failing/leaking restorations are nonetheless at high risk for whitening-induced hypersensitivity as relatively large volumes of gel can directly contact deep dentin and produce sensitivity via the mechanisms described previously. It is thus prudent and widely recommended that such sites be restored at least on an interim basis, e.g., with glass ionomer, prior to initiating whitening procedures.[51,52]

Management

Just as the understanding of mechanisms at work in whitening-induced hypersensitivity remains incomplete, no definitive strategy for prevention of its occurrence has emerged. Strategies for management of hypersensitivity associated with tooth whitening such as they currently exist include those conducted prior to whitening (reduction/elimination of pre-existing hypersensitivity or reduction of hypersensitivity risk) and in conjunction with whitening ("desensitizing" additives to whitening gels, and adjunctive desensitizer products for home use). Clinicians typically utilize a product-based approach as a first-line treatment for hypersensitivity, but evidence suggests that pretreatment assessment and increased focus on preventive strategies are equally, if not more, effective.

Pre-existing hypersensitivity and a whitening regimen of more than one application of product per day are the best predictors of moderate-to-severe hypersensitivity during whitening.[11,13,15,51] Gingival recession, on the other hand, is at best a contributing factor to pre-existing sensitivity, but not a predictor — most studies have found no association between gingival recession and tooth whitening.[15,18] Leonard et al. ruled out patient age and gender as predictors of sensitivity during whitening, but a subsequent study by Matis et al. reported higher incidence of sensitivity with females and patients under age 40 versus males and patients over 40.[11,49]

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Prevalence of pre-existing dentinal hypersensitivity has been reported to vary from 4 percent to 57 percent in the general adult population, and from 60 percent to 98 percent in periodontal patients.[23,53-56] It has been suggested this broad range reflects the differences in the populations studied and the investigation methods, and may also reflect structural differences — more and larger tubule openings — in sensitive versus nonsensitive dentin.[57,58] It is nonetheless clear that a careful screening for hypersensitivity prior to home whitening procedures is appropriate.

Several reports suggest that dentin hypersensitivity is probably underdiagnosed and undertreated.[47] Orchardson, in an excellent general review of dentin hypersensitivity, pointed out that strategies for its prevention are perhaps underutilized.[57,59] Specifically, the role of agents that increase dentin surface permeability (dietary and gastric sources of acid, abrasive dentifrices) must not be overlooked. Patient screening should include queries that identify potential etiologic agents in the diet (acidic foods/beverages such as carbonated soft drinks — especially colas — sports drinks, fruits and fruit juices, white wine, and yogurt); digestive tract (gastric reflux, regurgitation); and oral hygiene habits (dentifrice type, toothbrush type, toothbrushing technique). These factors can be effectively managed by increasing patients' awareness of their role in tooth hypersensitivity and thus motivating patients toward modifications in diet and oral hygiene, and in seeking medical management of gastrointestinal disorders. Leonard et al. also recommended asking patients before whitening if their teeth are sensitive to hot and cold, or after a prophy, and use gentle blast of air to assess.[11]

Acid erosion and abrasion can reportedly combine to potentiate each other's individual effects as etiologic agents in dentin hypersensitivity.[60] It has therefore been recommended that patients delay toothbrushing until at least two hours after ingesting acidic foods or drinks to reduce any combined harmful effects on dentin permeability.[56,57,59,60] The recommendation presumes remineralization of the acid-softened dentin by salivary calcium, phosphate, and fluoride over the two hours, thus restoring the dentin's abrasion resistance. Studies attempting to validate this recommendation, however, have had varied results, including no improvement in abrasion resistance after one to two hours salivary remineralization.[61,62] Rinsing for one minute with a 2,000 ppm sodium fluoride solu-

tion immediately before brushing was shown to significantly reduce abrasion of acid-eroded dentin in vitro.[63] Use of a fluoridated mouthrinse in lieu of brushing after ingesting acidic food or drinks to augment the saliva's remineralization capacity thus appears to be prudent.

A common recommendation for treating whitening-induced tooth hypersensitivity is to reduce the frequency or duration of whitening applications.[8,16] This approach can render the whitening procedure tolerable for some patients who would otherwise find the procedure too uncomfortable, but it does lengthen total treatment time significantly.

Products used to manage dentin hypersensitivity fall into two broad categories: agents that reduce dentin permeability either by occluding tubule openings on exposed dentin surfaces or by hypermineralization, and those that block nerve conduction.

Tooth pain from dentin hypersensitivity is caused by depolarization, then repolarization resulting in activity of dentinal sensory nerves. Potassium nitrate acts by preventing nerve repolarization after initial depolarization, reducing nerve excitability and the ability of the nerve to transmit pain.[64,65] Like hydrogen peroxide, potassium nitrate also rapidly (within minutes) filters through enamel and dentin and in to the pulp.[63]

Over-the-counter "desensitizing" dentifrices containing 5 percent potassium nitrate have long been available and have been extensively studied. Several clinical trials have reported the efficacy of potassium nitrate as a dentifrice additive.[67-69] Brushing with a potassium nitrate toothpaste was shown to reduce sensitivity in two weeks.[67] Toothbrushing with potassium nitrate dentifrice must be done twice a day as part of an ongoing brushing routine in order to produce a desensitizing effect.[23] Overall, however, evidence addressing the efficacy of potassium nitrate as a desensitizer is equivocal. A 2001 meta-analysis of four randomized clinical trials failed to produce conclusive evidence of significant efficacy for potassium nitrate in reducing cervical hypersensitivity.[67]

It is also noteworthy that most research into the desensitizing efficacy of potassium nitrate has been examined with respect to its use as a dentifrice additive to desensitize cervical dentin. The degree to which it reduces sensitivity during tooth whitening when used as an additive to whitening gels remains in question.[9,26] Tam examined the efficacy of 3 percent potassium nitrate and 0.11 fluoride ion wt/vol in reducing sensitivity when added to a 10 percent carbamide peroxide whitening gel.[16] The additives were reported to significantly reduce, but not eliminate sensitivity. The study also did not attempt to differentiate between effects of one versus the other additive.

PREBRUSHING WITH a potassium nitrate dentifrice for two weeks was reported to reduce sensitivity during whitening.

Adequate evidence exists, nonetheless, to state that potassium nitrate-containing dentifrices and desensitizer gels are indicated for management of mild of moderate hypersensitivity.[56] It is non-invasive, treats multiple teeth simultaneously, and inexpensive compared to in-office treatment such as sealing sensitive areas with adhesive resin.[47]

The efficacy of a dentist-dispensed desensitizer gel containing 5 percent potassium nitrate + 1,000-ppm fluoride ion for use in bleaching tray has also been investigated.[12,71] Haywood et al. concluded that 10 to 30 minutes wearing time of the gel in the tray before or after whitening may reduce sensitivity in more than 90 percent of patients and make whitening tolerable.[71] Leonard et al. assessed efficacy of the gel to prevent or decrease sensitivity compared to placebo in an at-risk population, i.e., patients reporting pre-existing sensitivity and risk factors.[12] They reported efficacy — 41 percent in the treatment group had at least one day of sensitivity versus 78 percent in the placebo group. The gel can also be used as needed during the day or worn in the tray on alternating nights when an overnight whitening regimen is used.[10]

Prebrushing with a potassium nitrate dentifrice for two weeks was reported to reduce sensitivity during whitening, and the efficacy of an OTC potassium nitrate dentifrice worn in a tray has been investigated as well.[72] Tray application for several hours/day for one week was shown to provide relief from tooth sensitivity, and 10 to 30 minutes of tray application immediately prior to a dental prophylaxis reduced discomfort during and after the procedure.[51,73] However, some toothpaste ingredients such as sodium lauryl sulfate (a foaming ingredient) or flavorings may give rise to gingival irritations (apthous ulcers) when applied in a tray for extended periods.[10]

Haywood nonetheless recommended initially using an OTC dentifrice as a tray-applied desensitizer due to its cost-effectiveness and convenience, then switching to a dentist-dispensed desensitizer gel if gingival irritation occurs.[10]

Agents for desensitization via block-

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age of patent dentinal tubules include fluoride, oxalate compounds, and derivatives of resin adhesive restorative products. Tubule blockers have been reported to have varying levels of efficacy on general dentin hypersensitivity, and their potential to prevent whitening-induced hypersensitivity is limited to reduction in osmotic stimulation on exposed dentin surfaces. These agents have no effect on peroxide diffusion to the pulp and as such should not be expected to completely eliminate whitening-induced hypersensitivity.

Neutral sodium fluoride works by occlusion of dentinal tubules with fluoride precipitates; calcium fluoride is produced after exposure of dentin to high concentrations of topical fluoride, and fluorapatite is produced after exposure to lower concentrations.[16] Brushing with a fluoride gel for four weeks was shown to reduce cervical hypersensitivity.[74] A single application of a 5 percent sodium fluoride varnish has been shown to reduce cervical dentin hypersensitivity within two weeks.[75]

Anecdotally, the author has observed that brushing twice daily with a 5000 ppm F dentifrice for one week prewhitening reduced discomfort during whitening. Evidence suggested that tubule blockage by fluoride has limited efficacy in treating whitening-induced hypersensitivity, however, since the peroxide molecule is small enough to pass through the interstitial spaces between tubules.[10] Betke et al. demonstrated that a fluoride varnish failed to inhibit dentin dehydration by whitening agents.[28] It was postulated that any calcium-fluoride precipitate formed by the varnish was inadequate inhibit dehydration.

Oxalate salts form a crystalline precipitate of calcium oxalate to occlude dentinal tubules immediately upon application, and thus reduce or prevent fluid movement.[76] Swift described the following categories of these dentist-dispensed products: potassium oxalate (Protect, Butler; Therma-Trol, Premier; Super Seal, Phoenix), ferric oxalate + acp + potassium phosphate (Quell, Pentron); potassium phosphate + potassium carbonate + calcium chloride + strontium chloride (D/Sense 2, Centrix).[47] Efficacy of these products for general management of cervical hypersensitivity has been demonstrated.[77,78] A variant of this product type (Pain-Free, Parkell) contains 3 percent oxalic acid + 5 percent polymethyl methacrylate-co-p-styrenesulfonic acid to produce a resin seal on exposed dentin along with tubule blocking via the oxalate precipitate.[47]

THE NEWEST PRODUCTS aimed at hypersensitivity management are those containing amorphous calcium phosphate as the active ingredient.

Resin-based desensitizing "varnishes" are essentially the hydrophilic resin primer component (2-hydroxyethyl methacrylate, or HEMA) common to many adhesive resin bonding products with the addition of an antibacterial compound. Examples include Gluma Desensitizer (Heraeus Kulzer), originally sold as a dentin primer, an aqueous solution of 35 percent HEMA and 5 percent gluteraldehyde 26; HurriSeal (Beutlich) — 35 percent HEMA + benzalkonium chloride+ NaF; Hemaseal and Cide (Advantage Dental Products) — HEMA + 4 percent chlorhexidine).[26]

Several reports cite the efficacy of this approach: An assessment of several such products by Clinical Research Associates, however, found that while all reduced sensitivity, none provided relief in 100 percent of cases.[79-82] It should be noted that one of the varnishes in this study, an acetone-containing dentin adhesive, produced significant dehydration of the dentin samples during its application despite creating a protective seal against dehydration by whitener products. Limitations of resin varnish desensitization include thin layers subject to eventual loss via abrasion, techniques sensitivity (care must be taken to prevent moisture contamination and prevent pooling of resin into thick layers), limited access to interproximal root surfaces, and expense. Additionally, and as mentioned previously, these products have limited efficacy in reducing whitening-induced sensitivity inasmuch as they do not prevent diffusion of peroxide to the pulp.

The newest products aimed at hypersensitivity management are those containing amorphous calcium phosphate as the active ingredient. ACP forms hydroxyapatite in enamel and increases enamel hardness, hence its primary application as an agent to remineralize carious lesions, as well as reduce susceptibility to their formation. An effective delivery system for ACP has been developed by combining it with casein phosphopeptide, a milk protein derivate. The resultant complex — CPP-ACP, aka Recaldent, stabilizes the ACP such that it provides a reservoir of calcium and phosphate that remain bioavailable in saliva for several hours after application. Topical application of a CPP-ACP-containing paste (Prospec, MI Paste, GC America) has been shown to rapidly reduce dentin hypersensitivity, suggesting its applicability in managing whitening-induced hypersensitivity.[83,84] ACP (without CPP) is now present as

an additive in some whitening products as well.[85] Reports of efficacy in this regard are promising to date, with one clinical trial citing sensitivity remaining at levels similar to baseline.[86]

Summary and Conclusions

Whitening-induced tooth hypersensitivity is a complex, multifactorial phenomenon and as such, continues to defy attempts to develop definitive preventive therapies. The best currently available evidence suggests the following:

- Pretreatment assessment of baseline tooth hypersensitivity is key to the effective management of whitening-induced hypersensitivity. Thorough screening for risk factors (acidic foods, gastric reflux, abrasive dentifrices) facilitates the elimination of these factors and reduction of baseline sensitivity before whitening treatment is initiated.
- Carious lesions and failing/leaking restorations should be addressed prior to whitening treatment. Interim restoration of these sites with a glass ionomer material provides an excellent microleakage-resistant seal to potentially reduce sensitivity during whitening, arrest lesion progression, and stabilize involved teeth until postwhitening shade matching and definitive restoration can be conducted.
- Increased frequency of whitening gel application is associated with higher levels of sensitivity during treatment. Instructions to patients should stress adherence to appropriate protocols for the products being used.
- Carbamide peroxide products tend to produce less sensitivity than those containing hydrogen peroxide, and should be the first choice for patients with significant levels of pre-existing hypersensitivity.
- Topically applied potassium nitrate is an effective method for reducing whitening-induced hypersensitivity. Brushing twice daily with an OTC dentifrice containing potassium nitrate for two weeks prior to whitening can reduce baseline sensitivity and risk of discomfort during whitening. The dentifrice can also be used in the tray for up to 30 minutes before and/or after whitening gel application to manage sensitivity during treatment. Dentist-dispensed gels containing potassium nitrate for tray application have also demonstrated efficacy in sensitivity reduction.
- Products containing amorphous calcium phosphate show promise for management of sensitivity via topical application. Whitening gels containing ACP may also prove to be effective in this regard. ■■■■

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