Current and Novel Clinical Approaches for the Treatment of Dentin Hypersensitivity

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Brief Overview of the Chapter

According to Orchardson and Gillam (2006), the management of dentin hypersensitivity (DH) has been traditionally based on products or procedures (in-office [professionally-applied] and over-the-counter (OTC)/at-home) that may either block the dentin tubules on the exposed root surface or desensitise the nerves in the pulp (see Chap. 10). The clinician however is often faced with an array of products from the manufacturers all claiming to be effective in terms of both immediate and long-lasting relief from pain. Whether there is an ideal treatment or a so-called gold standard for DH, it is still a challenge. (Orchardson and Gillam 2006; Gillam et al. 2013). Furthermore several investigators have reported on the wide range of in-office and OTC products and procedures that have been used by clinicians in daily dental practice, and it was evident from these reports that there is considerable confusion for the clinician with regard to which of these products actually are effective in reducing DH (Gillam et al. 2002; Cunha-Cruz et al. 2010).

One of the problems that a clinician may face when examining a patient complaining of oral pain is the clinical diagnosis of the condition. This has been addressed in the earlier chapters of this book (see Chaps. 4 and 5), and it is evident that without a definite diagnosis of DH, the clinician cannot effectively manage the condition successfully. DH as previously defined is essentially a diagnosis of exclusion, and this will clearly take the clinician sometime in order to record the necessary information during the screening process and subsequent clinical examination (see Chap. 5). Once the clinician is confident that the correct diagnosis of DH has been made, then the management of the condition can be undertaken. However as indicated in Chap. 10, the provision or prescription of a particular in-office procedure or OTC product in itself without the removal of the essential components of the aethiological and predisposing factors that initiated the problem in the first place by the clinician, together with

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counselling and educating the patient (dietary changes/toothbrushing techniques, etc.) and subsequent monitoring the condition over time, will result in ineffective and unsuccessful treatment. The aim of this chapter therefore is to review the various products and procedures that have been recommended for the treatment of DH and any novel products that have been recently introduced, together with some recommendations for future development of desensitising strategies.

Classification of Desensitising Agents

The classification of the various treatments for DH can be a challenging process particularly if the precise mode of action is unknown. Grossman (1935) and Gillam (1997) reported that an ideal product for DH should have specific criteria in order to be both effective and acceptable to the patient and clinician. The ideal desensitising agent must be fast acting, be effective for long periods, be easy to apply, not dangerous to the pulp, not cause pain and not stain the teeth. There have been a number of previous classifications published in the literature, some relatively simply and others more complex in nature. For example, a simple classification of products into in-office and OTC/at-home products (Orchardson and Gillam 2006), or based on the mechanism of action (Gillam 2002; Miglani et al. 2010); or whether the desensitising products for in-office procedures (1) do not polymerise (varnishes/precipitants/primers containing HEMA), (2) undergo setting or polymerisation reactions (conventional glass ionomer cements, or resin-reinforced glass ionomers/compomers, adhesive resin primers, adhesive resin bonding systems); or whether these agents are used in conjunction with (3) the use of mouthguards, (4) iontophoresis combined with fluoride pastes or solutions or (5) lasers (Pashley 2000) (see also Table 8.1). One of the problems, however, when recommending or evaluating these approaches for the treatment of DH is that clinicians appear not only to be uncertain as to the most successful way in which to manage DH but also expressed

a level of dissatisfaction with the various products and techniques available (Cunha-Cruz et al. 2010) (see Chap. 10).

Current Therapies for Dentin Hypersensitivity (DH)

Currently, many of the dentin desensitisers that have been used in treating DH have supported evidence from in vitro, in situ or in vivo studies (Orchardson and Gillam 2006). The mechanism of action for the majority of the desensitising agents in these studies was by tubule occlusion. There are however a number of problems with the application of both in-office and OTC/at-home products in that the tubular occlusion was not permanent in nature, particularly following the impact of an acid challenge to the surface deposit (see Chap. 6). Furthermore the resin or toothpaste precipitate in the dentin tubule may only provide a chemical-physical blockage rather than induce the initiation of new dentin (Calabria et al. 2012). Ideally a bioactive/biomimetic material able to modify its composition and/or induce the precipitation of insoluble fluorapatite would be a major advantage in the treatment of DH (see Chap. 6). According to Orchardson and Gillam (2006), the management of DH should be based on a stepwise approach based on the extent and severity of the condition. An inherent component of this approach was the importance of the removal of any aetiological or predisposing factors, counselling the patient with regard to dietary intake (e.g. excessive consumption of acidic drinks) and toothbrushing techniques and appropriate monitoring by the clinician (see Chap. 10). A more recent UK Guidelines publication on the management of DH by Gillam et al. (2013) also stressed the importance of not using a single treatment philosophy to treat DH but recommended management strategies based on the aetiology and predisposing factors of specific presenting conditions, namely, (1) patients with good oral hygiene associated with DH, (2) patients with toothwear and associated DH and (3) periodontally related DH together with an awareness of the potential impact of DH on the patient's quality of life (see Chaps. 9 and 10).

Table 8.1 Classification of dentin desensitisers and procedures according to the mechanism of action

Mechanism of action	Desensitising agents
Nerve desensitisation	Potassium nitrate, chloride, citrate, oxalate,
Protein precipitation	Glutaraldehyde, silver nitrate, zinc chloride, strontium chloride hexahydrate
Tubule occlusion	Stannous and sodium fluoride
	Strontium chloride
	Ferric oxalate
	Potassium oxalate
	Amorphous calcium phosphate
	Calcium phosphate
	Calcium carbonate
	Calcium hydroxide
	Calcium sodium phosphosilicate
	Tricalcium phosphate (TCP)
	Dental varnish (Clinpro™ 3M™ ESPE™) containing 5 % sodium fluoride with tricalcium phosphate (TCP)
	Prophylaxis polishing paste containing 1.23 % fluoride
	Prophylaxis polishing paste containing 1.1 % sodium fluoride +5 % NovaMin
	Sensodyne NUPRO (NovaMin® – Dentsply)
	1.1 % Sodium fluoride anticavity toothpaste (Clinpro™ 3M™ ESPE™ 5000)
	Vanish™ XT Extended Contact Varnish (light-cured resin-modified glass ionomer (RMGI) based on the patented methacrylate-modified polyalkenoic acid)
	Bioactive glasses (SiO_2–P_2O_5–CaO–Na_2O)
	Aluminium
	Gingival surgery – recovering the exposed dentin area
Neural action and tubules occlusion	Laser
	Neodymium-doped yttrium aluminium garnet (Nd:YAG), laser GaAlAs (gallium-aluminium-arsenide laser), erbium-YAG laser
Anti-inflammatory effect	Homeopathic medication – propolis
	Corticoid steroids
Covering the dentin surface	Tooth restoration
	Periodontal surgery

Adapted from Miglani et al. (2010)

In-Office (Professionally Applied) Treatment

Following a successful diagnosis of DH, the clinician will base the treatment on the extent and severity of the condition. For example, where the problem is mild to moderate in nature (see Chap. 5), then an OTC/at-home approach may be initiated with the caveat that if the pain does not resolve before the next appointment (or if this approach was ineffective), then the patient may return for a more invasive in-office (professionally applied) procedure. If the pain is localised to one or two teeth and is severe in nature, the clinician may opt for an in-office procedure using one of the restorative materials as suggested by

Pashley (2000). There is a tendency however to apply an in-office treatment following several attempts to resolve the patient's pain following the unsuccessful use of an OTC/at-home product. There are however a vast array of desensitising products currently available which may make the choice of the most appropriate product both confusing and difficult for the clinician (Cunha-Cruz et al. 2010).

Fluoride Application

According to Cummins (2010), fluoride in the form of gels, varnishes and resins has been recommended for an in-office treatment for DH

(Gaffar 1999; Orchardson and Gillam 2006; Ritter et al. 2006; Merika et al. 2006; Hoang-Dao et al. 2008; Yilmaz et al. 2011; Trushkowsky and Oquendo 2011; Petersson 2013). High-level fluoride-based desensitisers (solutions, gels and varnishes) have also been reported to provide an instant and relative long-term relief of DH (Al-Sabbagh et al. 2009; Gaffar 1999; Ozen et al. 2009; Petersson 2013; Pradeep et al. 2012; Ritter et al. 2006; Schmidlin and Sahrmann 2013). For example, a single topical application was reported to relieve DH for 3–6 months (Al-Sabbagh et al. 2009; Glockner 2013; Ritter et al. 2006). The combined use of fluoride with iontophoresis has also been reported in the literature (Gangarosa and Park 1978; Brough et al. 1985; Gangarosa 1994; Singal et al. 2005; Gupta et al. 2010; Aparna et al. 2010; da Rosa et al. 2013; Patel and Langalia 2014); however, the clinical effectiveness of this technique (with or without fluoride) has been questioned (Gillam and Newman 1990; Pashley 2000). The mode of action of topically applied fluorides is by tubule occlusion in the form of calcium fluoride (Table 8.1) although the resultant layer is generally not resistant to the frequent acid challenges in the oral environment, resulting in reapplication in order to maintain any efficacy (Cummins 2010; Calabria et al. 2012). Fluoride compounds however have been reported to demonstrate low permeability values in vitro in laboratory studies, which may suggest that the transient effect in reducing DH observed in clinical trials may be as a result of the other properties of the varnish rather than the physical precipitation of crystals inside the dentin tubules. A combination approach using a fluoride mouthrinse has also been recommended by several investigators (Glockner 2013; Marinho et al. 2004). Although currently there does not appear to be a systematic review exclusively addressing the role of professionally applied fluoride varnishes in DH in randomised clinical trials (Ozen et al. 2009; Sharif et al. 2013; Thrash et al. 1994), there was some evidence reported in the published literature that indicated that professionally applied fluoride varnishes were effected in relieving pain associated with DH (Orchardson and Gillam 2006; Sharif et al. 2013).

Potassium Salts

Potassium salts have also been used for the treatment of DH (Hodosh 1974; Pereira et al. 2005; Sauro et al. 2006; Cummins 2010) and have been reported to react with the exposed dentin section with a precipitation of calcium compound crystals onto the dentin surface and sub-superficially into the dentin tubules to effectively occlude the dentin tubules (Pereira et al. 2005; Calabria et al. 2012; Cummins 2010; Markowitz and Pashley 2008) (see Chap. 2). Potassium has also been combined with an oxalate formulation (Cunha-Cruz et al. 2011). Three types of oxalate formulations (6 % ferric, 30 % dipotassium and 3 % monohydrogen monopotassium) have also been classically indicated to occlude the dentin tubules and reduce DH (Al-Sabbagh et al. 2009; Pashley et al. 1987, 2001; Schmidlin and Sahrmann 2013). For example, 3 % Potassium oxalate solutions have been used to treat DH, and the resultant oxalate crystals have been reported to be more resistant to an acid challenge compared to the precipitated fluoride crystals (Pereira et al. 2005; Calabria et al. 2012). The in vitro testing of desensitising agents has been traditionally through the observation of the surface and subsurface of the dentin tubules using a number of techniques, for example, SEM scanning electron microscopy and CLSM confocal laser scanning microscopy, and comparing images before and after conditioning and treatment application (Fig. 8.1a, b) (see Chap. 6). By comparing the images of potassium oxalate-treated dentin before and after an acid challenge (Figs. 8.2b and 8.3a), one can observe the greater precipitation of crystals inside the dentin tubules following an acid challenge. This observation may also be seen with other formulations based on crystal precipitation, except for fluorophosphates.

Several investigators have reported that any relief from DH may be transient in nature as the precipitated crystals may be dissolved or removed over time (Al-Sabbagh et al. 2009; Cooley and Sandoval 1989; Kerns et al. 1991; Knight et al. 1993). According to Al-Sabbagh et al. (2009), oxalate-containing phytocomplexes however may be an alternative to the traditional oxalate preparations. For example, Sauro et al. (2006)
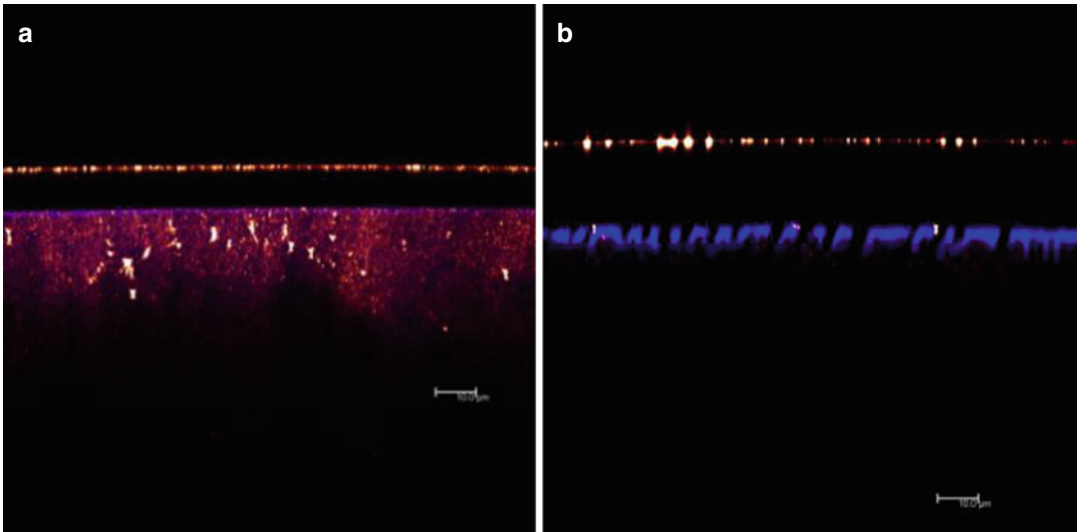
Fig. 8.1 Confocal laser scanning microscopy (CLSM, *XZ* axis) of mineralised sound dentin. (**a**) and after dentin conditioning with phosphoric acid (**b**)
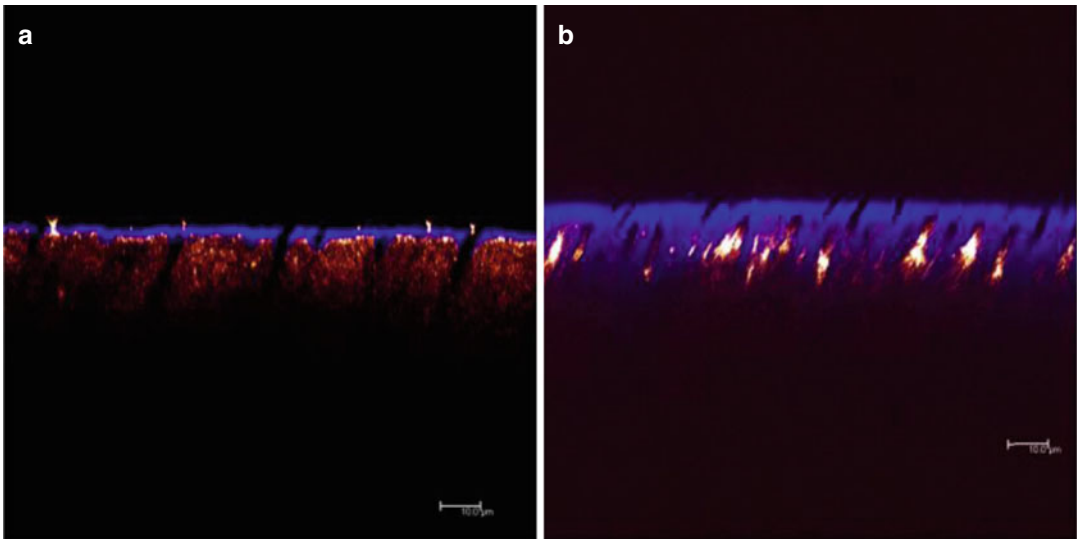

Fig. 8.2 Confocal laser scanning microscopy (CLSM, *XZ* axis) of demineralised dentin (**a**) treated with 1.23 % acidulated fluorophosphate (APF) (**b**) and 3 % potassium oxalate (KOx). Reflexive deposits can be seen inside dentin tubules of the specimen treated with KOx; in the APF specimen reflexive particles appear infiltrated in the body of intertubular dentin, similar to untreated dentin in Fig. 8.1a

reported that these complexes reduced dentin permeability following a single application in an in vitro study. These investigators also postulated that these complexes may protect the smear layer formed by scaling procedures on the root dentin from an acid challenge (Sauro et al. 2007).

A number of reviews recognised that, with the possible exception of the 3 % monohydrate monopotassium, oxalate preparations were not considered to be effective desensitising products based on evidence-based clinical criteria (Al-Sabbagh et al. 2009; Cunha-Cruz et al. 2011;
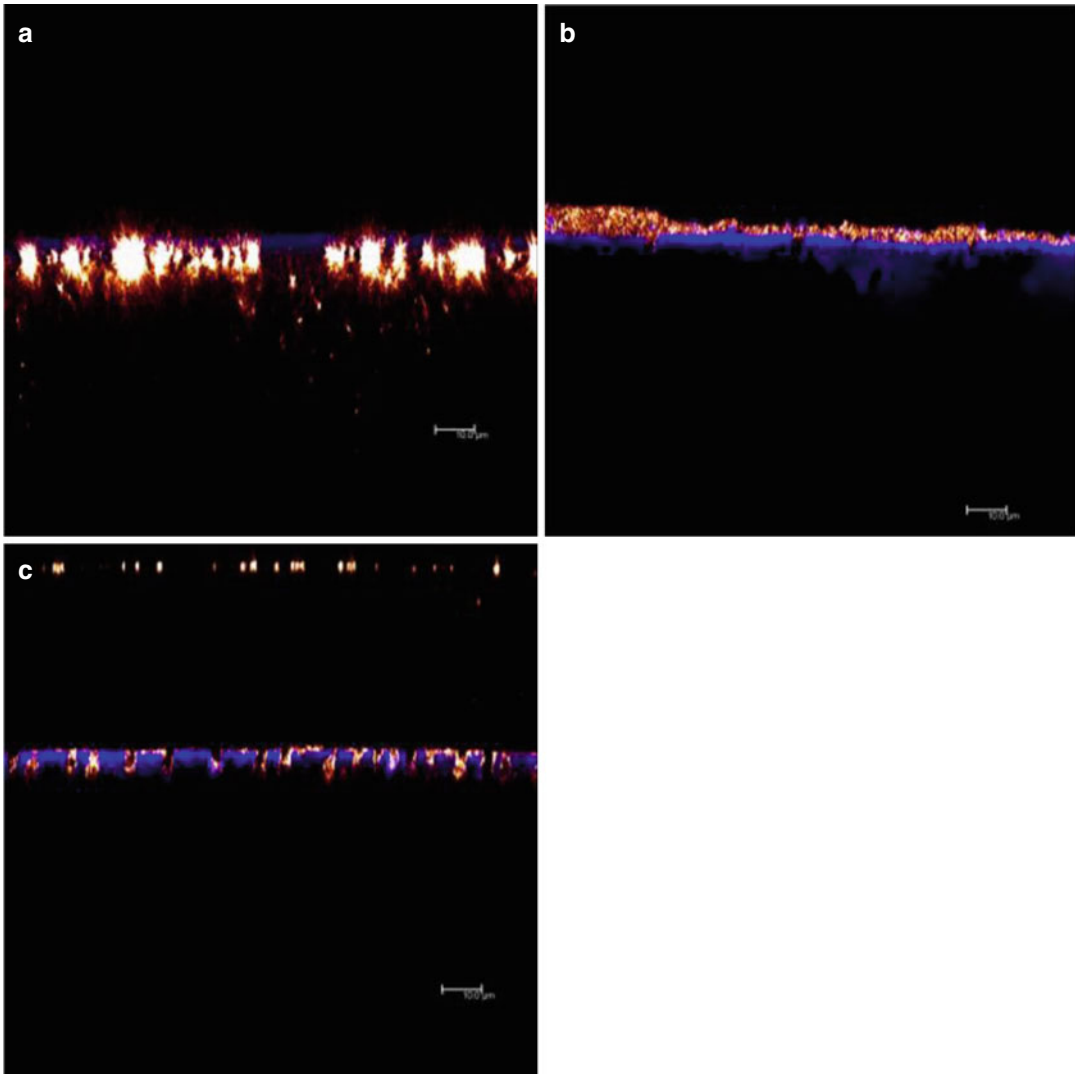
Fig. 8.3 CLSM *XZ* axis images of specimens before and after acid challenge. (**a**) Compared with Fig. 8.2b, the specimens treated with KOx challenged with citric acid show greater concentration of crystals inside the dentin tubules. (**b**) Specimen treated with Desensibilize NanoP® with clusters of crystals on dentin surface. (**c**) Specimen also treated with Desensibilize NanoP® shows crystals displaced into the dentin tubules after an acid challenge

Gillam et al. 1997; Orchardson and Gillam 2000; Schmidlin and Sahrmann 2013; Sharif et al. 2013).

Calcium Phosphate Compounds

Currently there appears to be limited data on the clinical effectiveness of calcium compounds in reducing DH although calcium hydroxide has been reported to immediately reduce DH in over

90 % of treated teeth in clinical studies (Al-Sabbagh et al. 2009; Levin et al. 1973). Calcium phosphate has also been reported in in vitro studies to be more effective than potassium oxalate in occluding the dentin tubules (Al-Sabbagh et al. 2009; Suge et al. 2005).

Recently, nano-hydroxyapatite-based agents (e.g. Desensibilize NanoP® and Clinpro white varnish [Clinpro]) with bioactive and biocompatibility potential have been reported to

be effective both as a remineralising and desensitising agent (Calabria et al. 2012; Hanning and Hanning 2010; Tschoppe et al. 2011). These agents contain nano-sized particles, which act in a similar manner to dental apatite in regard to their morphology, structure and crystallinity arrangement (Vandiver et al. 2005). These novel approaches were based on technology that explored the bioactivation with natural compounds with a view to improve the stability of the surface precipitate on the tooth surface following an acid challenge (Fig. 8.3). These images are consistent with the results of dentin permeability following dentin treatment with both potassium oxalate and Desensibilize NanoP®-FGM (Calabria et al. 2012).

One of the problems, however, with most of the products utilising an occlusive strategy was that the precipitate was unable to withstand the continual impact of the acid challenge in the oral environment. This issue has subsequently led investigators to develop innovative bioactive formulations to enhance the remineralising or tubule-occluding properties of the marketed products (Cummins 2010; Markowitz and Pashley 2008; Reynolds 1997; Reynolds et al. 2008; Rusin et al. 2010; Calabria et al. 2012; Nongonierma and Fitzgerald 2012; Pei et al. 2013) (see Chap. 6).

Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), a milk derivative, has also been reported to be effective in remineralising enamel and dentin (Reynolds 1997; Kumar et al. 2008; Reynolds et al. 2008; Agnihotri et al. 2012; Nongonierma and Fitzgerald 2012). CPP is a bioactive peptide released from caseins, which has been shown to enhance bivalent mineral solubility and also optimise binding ability to minerals, as calcium. Amorphous calcium phosphate (ACP) is the precursor of hydroxyapatite and in an oral environment in the presence of saliva was converted to hydroxyapatite. At the tooth surface interface, both calcium and phosphate are able to precipitate as stable hydroxyapatite (Nongonierma and Fitzgerald 2012). According to investigators, however, the effectiveness of ACP was only comparable to that of a placebo after approximately 3 months (Al-Sabbagh et al. 2009; Yates et al.

1998). According to Reynolds et al. (2008), CPP-ACP may also be successfully incorporated into commercial toothpaste products for the treatment of DH to enhance the occlusion of the dentin tubules through 'remineralised dentin' technology.

A number of recent products containing tricalcium phosphate (TCP) has been recently developed for the dental market (3M™ ESPE™ St Pauls Minneapolis, USA), namely, (1) a dental varnish (Clinpro™ 3M™ ESPE™) containing 5 % sodium fluoride with tricalcium phosphate (TCP), (2) a prophylaxis polishing paste containing 1.23 % fluoride, (3) prescription toothpaste 1.1 % sodium fluoride anticavity toothpaste (Clinpro™ 5000) and (4) Vanish™ XT Extended Contact Varnish, which is a light-cured resin-modified glass ionomer (RMGI) based on the patented methacrylate-modified polyalkenoic acid. The indications for use are based on remineralising of the enamel and blocking the open dentin tubules (tubule occlusion) (Vanichvatana and Auychai 2013; Asaizumi et al. 2013).

Several investigators have, however, reported conflicting results when comparing either an ACP (Schemehorn et al. 2011) or an MI Varnish with RECALDENT™ containing CPP-ACP (Cochrane et al. 2014) with 5 % ACP with TCP or CPP-ACP with a functionalised tricalcium phosphate (fTCP). According to these investigators, varnish ACP and MI Varnish had the highest release of calcium and fluoride ions compared to the TCP varnish.

Other calcium-containing desensitisers designed for in-office use are formulations containing arginine and calcium carbonate. These formulations have been reported to effectively occlude the dentin tubules and to provide immediate and lasting relief for DH (Pei et al. 2013; Hamlin et al. 2009; Schiff et al. 2009). According to the manufacturers, arginine is an amino acid naturally available in saliva, which presents a positive charge. Since dentin is negatively charged, arginine may play an important role, in association with calcium, favouring its deposition on the dentin surface. This mechanism is attributed to the deposition of an arginine, calcium carbonate and phosphate complex that physically occlude the dentin tubules and was resistant to normal
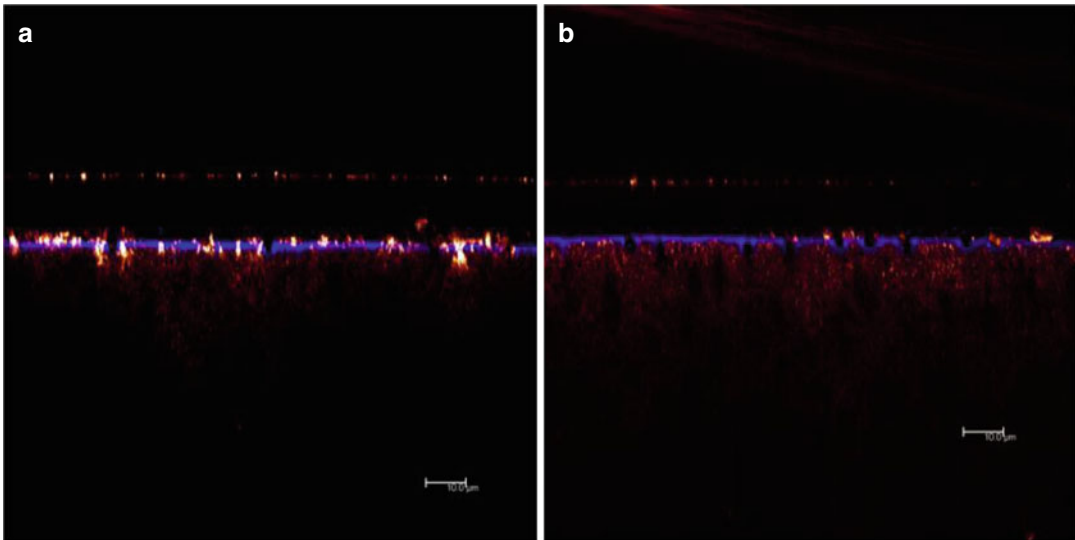
Fig. 8.4 CLSM *XZ* axis of dentin specimens treated with Pro-Argin™ before (**a**) and following an acid challenge (**b**)

pulpal pressures and to an acid challenge (Cummins 2010; Hamlin et al. 2009; Schiff et al. 2009; Pei et al. 2013). Pro-Argin™ technology is currently available as an in-office and as an OTC/at-home toothpaste that can be used in association with in-office treatment, for example, scaling and polishing procedures (Hamlin et al. 2009). According to Calabria et al. (2012), there is evidence from CLSM techniques that polishing with a Pro-Argin™ prophylaxis paste was effective in depositing these crystals in covering the dentin surface and occluding the subsurface of the dentin (Fig. 8.4a). Arginine-based products have also been reported to promote relief of DH compared to a negative control in clinical studies (Kapferer et al. 2013). However, there is some concern that these formulations are not able to resist to an acid challenge, as shown in Fig. 8.4b.

Calcium Sodium Phosphosilicate (Bioactive Glass)

Calcium sodium phosphosilicate, for example, NovaMin®, based on the original 45S5 Bioglass® formulation by Larry Hench (2006) was previously used for as a synthetic bone grafting material (PerioGlas®) (US Biomaterials Corp., Jacksonville, FL, USA; now NovaBone®).

According to the manufacturer, a bioactive glass material (e.g. NovaBone) has the ability to release Si, Ca and phosphate ions and initiate the upregulation and activation of a family of genes in osteoprogenitor cells, a process defined as osteostimulation (Price et al. 1997; Xynos et al. 2000; Loty et al. 2001; Sollazzo et al. 2010). Both PerioGlas and NovaBone products have been reported to be effective as a synthetic bone graft material in a number of dental indications, for example, in periodontal intrabony defects (Zamet et al. 1997; Park et al. 2001; Sculean et al. 2002), bone regenerative procedures (Yukna et al. 2001), bone augmentation prior to implant placement (Norton and Wilson 2001), furcation defects (Anderegg et al. 1999; Giusto 2005) and extraction sockets (Kates et al. 1998). The original 45S5 Bioglass® formulation was later adapted for use in toothpaste formulations and prophylaxis polishing pastes for the treatment of DH (NovaMin Technology Inc., Alachua, FL, USA; now GSK). According to Lynch et al. (2012), the proposed mode of action for both the toothpaste and the polishing paste was considered to be through the precipitating of hydroxycarbonate apatite (HCA) onto the dentin surface and subsequently occluding the dentin tubules.

In-office prophylaxis pastes containing both arginine/calcium carbonate and bioactive glasses

(e.g. calcium sodium phosphosilicate) have subsequently been reported to provide effective clinical pain relief from DH following application (Cummins 2010; Gendreau et al. 2011; Hughes et al. 2010; Pradeep et al. 2012; Pradeep and Sharma 2010). According to Sharif et al. (2013), however, there is currently insufficient evidence on its clinical efficacy in reducing DH following in-office procedures.

Dentin Bonding Agents/Adhesives and Adhesive Restorations

The application of dentin adhesive materials for relieving DH has been also reported by several investigators (Bruton et al. 2000; Mehta et al. 2014). Previous adhesive systems were based on the application of phosphoric acid to promote the interlocking of the resin material to the dentin surface. However, the need of an acidic dentin conditioning before the application of an adhesive may theoretically increase pulpal sensitivity. Although there were concerns that these organic matrix-enriched materials may fail to resist the repeated impact of an acid challenge in the oral environment, a number of glutaraldehyde-based agents (e.g. Gluma Desensitizer™) have been historically indicated for DH treatment (Schmidlin and Sahrmann 2013; Vora et al. 2012). Although the concern about its biocompatibility still persists, there is evidence of its effectiveness in treating DH (Orchardson and Gillam 2006; Schmidlin and Sahrmann 2013; Vora et al. 2012). The introduction of self-etching agents, however, has reactivated interest in applying these adhesive systems as dentin desensitisers. According to Pei et al. (2013), these products are relatively easy to apply and appear to be less aggressive to the dentin-pulp complex than the other etch-and-rinse systems. These agents also have been reported to promote a micro-mechanical interlocking through a hybridisation process, but due to the presence of functional monomers in mild self-etch adhesives, they have the potential to interact chemically with the calcium ions from the residual hydroxyapatite that remains available within the submicron hybrid layer (Pei et al. 2013; Inoue et al. 2005).

The functional monomers may also interact with the calcium ions from the smear layer, potentially resulting in additional chemical bonding.

The long-lasting effectiveness of any kind of adhesive system as a dentin desensitiser, for example, the self-etching adhesives, alone or in association with restorative materials, however, remains unclear. For example, it is recognised that dentin in a non-carious lesion is hypermineralised, and theoretically this may be an obstacle to dentin hybridisation. However any superficial layer of any type of material on the exposed dentin may not be able to resist to any outward fluid movement. According to Francisconi et al. (2009), the tooth may be subjected to significant stress concentration at the cervical region during either mastication or during toothbrushing which may present a challenge to the ability of an adhesive material to resist these forces. Although a strong interaction with the dentin substrate and the occlusion of the dentin tubules are mandatory for a long-lasting protective effect of the exposed dentin by a desensitising agent, this does not appear to occur on the exposed dentin of a cervical non-carious lesion. As illustrated in Fig. 8.5b, the hybrid layer is barely seen, and the resin tags only superficially penetrate the tubule openings in a dentin specimen from a non-carious cervical lesion. In Fig. 8.5a, however, in a sound cervical young dentin specimen, there was the presence of long resin tags and a well-defined hybrid layer (Calabria et al. 2010).

Adhesives may also be indicated for severe cases of DH that failed to respond to the other treatment modalities (Al-Sabbagh et al. 2009; Glockner 2013; Schmidlin and Sahrmann 2013; Swift et al. 2001). It is evident from the published literature that there is a vast array of products claiming to be effective in reducing DH. For example, dentin bonding agents (e.g. Scotchbond, Gluma Desensitizer, etc.) have been reported to be effective in reducing DH for up to 18 months post application (Al-Sabbagh et al. 2009; Brahmbhatt et al. 2012; Copeland 1985; Dondidall'Orologio et al. 1999; Ianzano et al. 1993; Kakaboura et al. 2005; Sharif et al. 2013; Schüpbach et al. 1997; Sethna et al. 2011; Yu
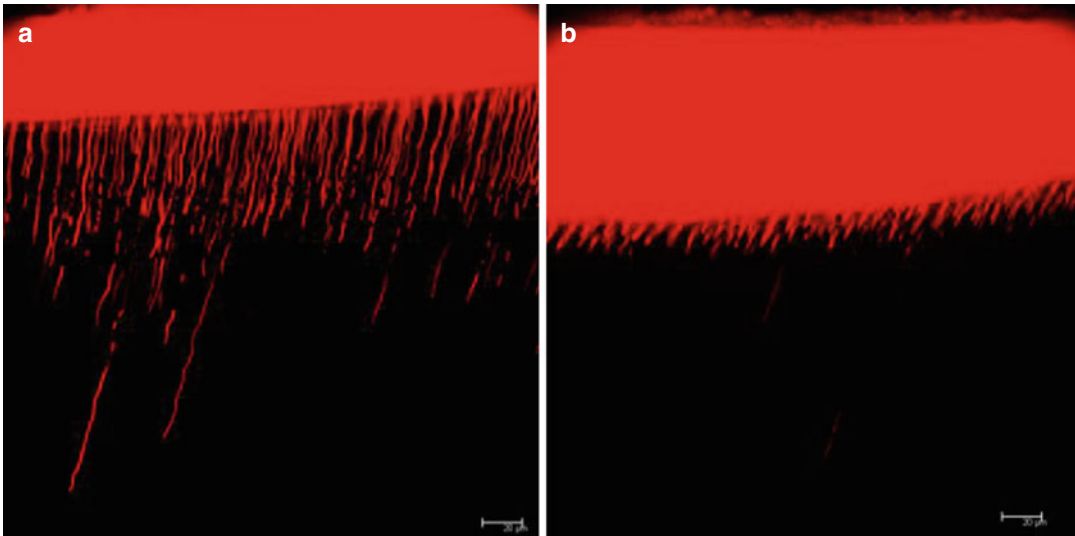
Fig. 8.5 CLSM images from sound cervical young dentin (**a**) and from a non-carious cervical lesion (**b**)

et al. 2010). According to several investigators, however, the available in vitro and well-conducted clinical studies have provided only limited data on the effectiveness of these products (Al-Sabbagh et al. 2009; Sharif et al. 2013; Tay et al. 1994). A further problem with some of these products is the lack of a filler material which may possibly favour wear from the surface of the material (Schmidlin and Sahrmann 2013).

Glutaraldehyde-based products have also been reported to reduce DH by protein precipitation, and these products have been reported to be effective in treating DH in the clinical environment (Duran et al. 2005; Glockner 2013; Kakaboura et al. 2005; Schmidlin and Sahrmann 2013; Schüpbach et al. 1997). Although several investigators have reported immediate pain relief to DH following application (Aranha et al. 2009; Lopes and Aranha 2013), reapplications are subsequently required in order to maintain the reduction of DH (Lopes et al. 2013). According to several investigators, however, there are biocompatibility hazards associated with these products that cannot be ignored (Arenholt-Bindslev et al. 1987; Schmidlin and Sahrmann 2013; Schweikl and Schmalz 1997). With respect to the glutaraldehyde-containing adhesives, several investigators have reported immediate and long-term relief of pain

associated with DH (Dondidall'Orologio et al. 2002; Lopes and Aranha 2013; Qin et al. 2006; Schüpbach et al. 1997).

Although a minimal invasive approach is preferred when treating DH with restorative materials (Orchardson and Gillam 2006), the clinician may be confronted with significant loss of the tooth structure associated with DH which would require restoration of the tooth shape in order to maintain its aesthetic and function. In this situation, restorative intervention may be recommended using glass ionomer cement (GICs) and resin-based materials which are generally indicated when a more resilient material is desirable. For example, where a class V restoration is necessary, an adhesive restoration is a valid option (Schmidlin and Sahrmann 2013). Large and deep cervical non-carious lesions, regardless of the degree of pain, must be restored in order to recover dental anatomy and preserve health of gingival tissue. GICs or composite restorations have been used for these purposes, but evidences supporting their success is weak (Al-Sabbagh et al. 2009; Polderman and Frencken 2007). Several investigators have also used GICs in association with periodontal procedures, including root coverage procedures, where it has been reported that the placement of a GIC may favour the attachment of the periodontal fibres and release fluoride

(Tantbirojn et al. 2006; Polderman and Frencken 2007; van Dijken and Pallesen 2008; Francisconi et al. 2009; Santos 2014)

Laser Technology

According to a number of investigators (Al-Sabbagh et al. 2009; Kimura et al. 2000; Moritz et al. 1998; Schmidlin and Sahrman 2013; Bader et al. 2014), the clinical effectiveness of laser technology (e.g. Er:YAG, Er, Cr:YSGG, CO_2, GaALAS and low-level diode) in treating DH ranged from 5.2 to 100 %, depending on the laser type and parameters used in the clinical studies. For example, an Nd:YAG laser has been reported to minimise discomfort immediately following application (Al-Sabbagh et al. 2009; Kara and Orbak 2009; Lan and Liu 1996; Liu et al. 1997; Lopes and Aranha 2013). Furthermore, according to Lin et al. (2013), a Nd:YAG laser may also have a supplementary analgesic effect when compared with the other high-power lasers. The clinical efficacy of GaAlAs laser has been reported to be based on tubule occlusion and possibly by interrupting neural transmission (Tengrungsun and Sangkla 2008), but this reported success may be less evident than that reported by the application of a dentin bonding agent. The long-term effectiveness of lasers compared to the other types of treatment approaches has also been favourably reported (Aranha et al. 2005; Birang et al. 2007; Gholami et al. 2011; Kara and Orbak 2009; Lopes and Aranha 2013). However, the reported clinical effectiveness of lasers in the treatment of DH has also been challenged by Schmidlin and Sahrmann (2013) and Sgolastra et al. (2011). According to several investigators, there were no significant reductions in DH in comparison to the other types of laser and placebo therapies (Al-Sabbagh et al. 2009; Lier et al. 2002; Martens 2013). Furthermore, lasers in general are less effective in cases of severe DH (Al-Sabbagh et al. 2009; Kimura et al. 2000). There do not however appear to be any major adverse effects when using lasers if they are used in accordance with the manufacturers' instructions (Lopes and Aranha 2013).

The mechanisms involved in the laser treatment of DH are relatively unknown (Kimura et al. 2000). The mechanism of the high potency laser devices appears to promote the closure of the exposed dentin tubules, primarily, by producing a melting effect of the dentin. According to Bader (2014), there appears to be a degree of uncertainty as to whether there is also a synergic effect if lasers are combined with other occlusive pretreatments, which, in turn, may help to promote a more stable layer to obliterate the dentin tubules. The low-potency laser has been reported to diminish the neural response to hypersensitive stimuli (Bader et al. 2014), although there does not appear to be any strong evidence to support the use of laser therapy for this purpose due, in part, to the lack of consensus of the type of laser and the parameters that should be incorporated into standardised and efficient protocols.

Combined Treatment of Lasers with Other Treatment Approaches

The combination of techniques (e.g. laser technology with a fluoride varnish) may also be a strategy in the treatment of DH. The rationale for this approach is the association of the combined effects, which may result in significant better outcomes (Duran and Sengun 2004; Erdemir et al. 2010; Ipci et al. 2009; Lin et al. 2013; Lopes and Aranha 2013). Dentin desensitisers with an occluding potential may be used in association with low-level lasers in order to simultaneously block the dentin tubules and decrease the level of neural stimulation which may in turn initiate the formation of tertiary dentin (Farmakis et al. 2012; Lopes and Aranha 2013; Lopes et al. 2013). A similar indication of this combined use has been in the recommendation of an OTC product (e.g. toothpaste) following an in-office treatment (Rösing et al. 2009).

Although the evidence from both randomised controlled trials and systematic reviews supports the clinical use of lasers in treating DH (Deng et al. 2011; He et al. 2011; Sharif et al. 2013), further well-conducted studies with appropriate clinical protocols comparing the use of both low

or high frequencies and any association with the other alternatives discussed for treating DH should be implemented (Al-Sabbagh et al. 2009; Ipci et al. 2009; Petersson 2013; Schmidlin and Sahrmann 2013).

Other Restorative-Based Procedures

Periodontal Plastic Surgery (Root Coverage Procedures)

Periodontal disease may lead to changes in the periodontal tissues, and the consequent recession of the marginal tissues (gingival recession) has been reported to be associated with DH as a result of the exposure of the root surface to the oral environment. Both nonsurgical and surgical periodontal procedures together with the effects of periodontal disease on the teeth and their supporting structures may also impact on the patient's aesthetics and quality of life due to the pain associated with DH (see Chap. 3). The clinician should be aware of the various etiological and precipitating factors associated with gingival recession (e.g. anatomical, plaque, restorative procedures, patient habits, smoking, etc.) (see also Chap. 4). Furthermore the clinician should also consider how to manage both the patient's and his/her own expectation when proposing a treatment plan for resolving the problem of the aesthetics associated with gingival recession (e.g. posttreatment, presence of the so-called black triangles, etc.) (Fig. 8.6).

This may present a problem if both the perception and expectations expressed by the patient are either ignored or not met (by the clinician) to their satisfaction. Root coverage procedures associated with root scaling and the covering of the exposed root through periodontal surgical procedures have been reported to reduce or completely abolish DH over time (Orchardson and Gillam 2006; Lin and Gillam 2012). According to Douglas de Oliveira et al. (2013a), root coverage procedures (both partial and complete coverage) have been reported to decrease pain and improve a patient's quality of life although currently there appears to be insufficient scientific evidence to associate root

Fig. 8.6 Clinical photograph of patient with so-called black triangles which may cause aesthetic concerns

coverage procedures with the complete suppression of DH (Douglas de Oliveira et al. 2013b).

As indicated above, gingival recession may also occur in the healthy tissues and has been related to a combination of anatomical and physiological aspects of each individual tooth. For example, gingival recession was more often observed on the buccal surfaces compared with other surfaces of the teeth as a result of anatomical factors. According to Kassab and Cohen (2003), more than 50 % of the population in their study had at least one or more sites with gingival recession of 1 mm or more (see Chap. 3).

Gingival recession may be also associated with the periodontal biotype, and the analysis of this factor is essential for the planning of surgical procedures for root coverage. Three different periodontal biotypes have been described in the published literature, namely, the thin scalloped, the thick flat and the thick scalloped (Seibert and Lindhe 1989; Zweers et al. 2014). The teeth in the thin scalloped category have the characteristics of a thin gingival tissue with a more pronounced contour of the gingival margin, with a greater height of the interproximal papilla and with an underlying thin buccal bone that can present with fenestrations. The thick biotype category is characterised by thicker bone tissue surrounding the teeth and covered by a thick and smooth contour gingival tissue, which results in a small height of the papilla. The thick scalloped category displays the characteristics of the two other patterns (Fig. 8.7a–c).

It is important for the clinician to have sufficient knowledge of the relevant characteristics of each of

Fig. 8.7 Clinical pictures of (**a**) normal, (**b**) thin and (**c**) thick gingival biotypes

the biotypes, both in health and disease, particularly when planning root coverage procedures, as the different biotypes may respond differently both during the surgery and, subsequently, in the wound healing process. For example, thin biotypes may tear easily during probing and surgical intervention and root coverage may not be optimum, whereas in a thick biotype, the thickness of the flap may be related to a higher mean gain of complete root coverage (Seibert and Lindhe 1989; Müller et al. 2000; Hwang and Wang 2006). Due to the higher incidence of gingival recession in the thin biotype, the aim of the planned surgical procedures should not only consider the root coverage aspect but also the resultant aesthetics together with the treatment of DH (Ahmedbeyli et al. 2014; Cairo et al. 2014; Polack and Mahn 2013; West et al. 2013).

According to Gillam (2014), several classifications for treating gingival recession have been recommended in order for the clinician to facilitate both a diagnosis and a template for the correction of gingival recession defects. One of the classifications that has been favoured over the last three

Table 8.2 Miller's classification of gingival recession (GR)

| Classification	Criteria
Class I	Marginal tissue recession that does not extend to the mucogingival junction (MGJ) with no interproximal tissue
Class II	Marginal tissue recession that extends to or beyond the MGJ, with no periodontal attachment loss (bone or soft tissue) in the interdental area
Class III	Marginal tissue recession that extends to or beyond the MGJ with periodontal attachment loss in the interdental area or malpositioning of teeth
Class IV	Marginal tissue recession that extends to or beyond the MGJ with severe bone or soft tissue loss in the interdental area, to a level apically to the buccal/labial soft tissue margin and/or severe malpositioned teeth

Acknowledgement modified from Kassab and Cohen (2003)

decades is the Miller classification system ([I–IV] (Miller 1985) (Table 8.2, Fig. 8.8a, b) which enabled the clinician to predict successful outcomes of the surgical procedure based on type of
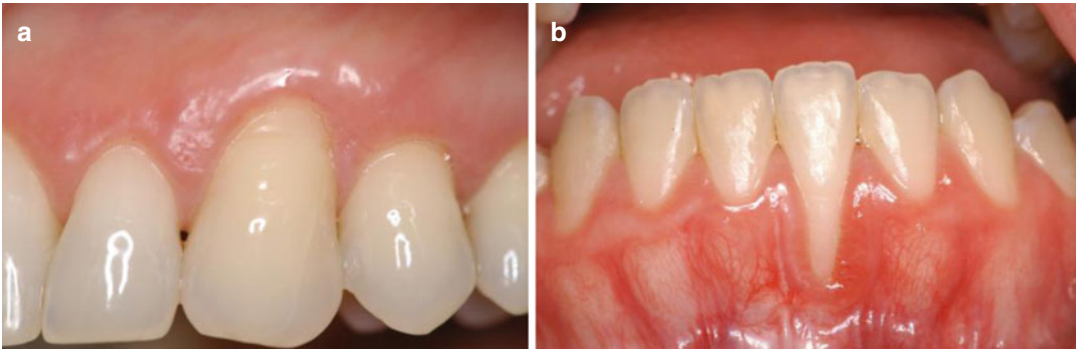
Fig. 8.8 (**a**, **b**) Examples of a Miller' Class I (**a**) and Class II marginal defect (**b**)

Fig. 8.9 (**a**, **b**) Example of a root coverage procedure using free gingival and connective tissue grafts (**a**) pretreatment and (**b**) posttreatment connective tissue graft: Acknowledgement for [Courtesy of D. Chatzapoulou]

Connective Tissue Graft

Free Gingival Graft
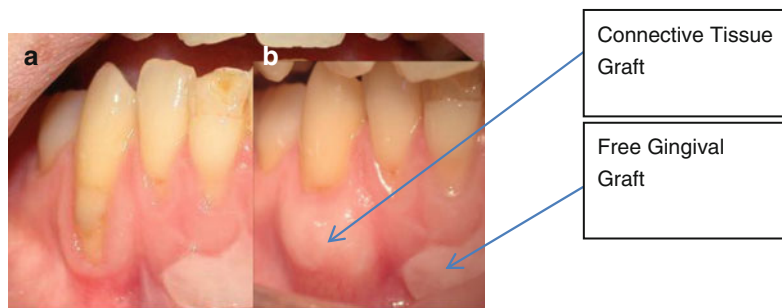
defect, taking into account the anatomical features. A number of materials and techniques have been reported in the published literature, and these include guided tissue regeneration (GTR), coronally advanced flap and Emdogain (CAF + EMD), connective tissue graft (CTG) and free gingival graft (acellular dermal matrix allograft/Mucograft ADM) (Gillam 2014) (Fig. 8.9).

Irrespective of the surgical procedures, associated techniques, flap design or materials, the desired outcome for treating gingival recession defects is that the resulting gingival tissue covering the root surface must have the characteristics of a thick keratinised mucosa that will enable optimum plaque control without any change in the graft's position (e.g. apical direction). When the root coverage along with an increase in thickness is desired, a systematic review of studies indicates an association of repositioned flap with subepithelial connective tissue graft as the best option, and this association can be considered as the gold standard surgery for root coverage (Chambrone et al. 2008).

Several investigators however have reported that in some patients, the clinician may not be able to treat the gingival recession defect by the conventional surgical root coverage procedures described above, and therefore, the remaining exposed root dentin may require a restorative approach, for example, the provision of a partial laminate porcelain veneer, composite material or glass ionomer or contouring of the defect with a finishing bur (Zalkind and Hochman 1997; Tugnait and Clerehugh 2001; Drisko 2002; Özgünaltay and Önen 2002; Santamaria et al. 2007). In patients where there has been extensive periodontal destruction resulting in open gingival embrasures (so-called black triangles), which may be aesthetically unacceptable for the patient (Sharma and Park 2010), a relatively simpler option to the techniques described above may be utilised, for example, a prosthetic gingival veneer (Greene 1998).

Gingival Augmentation

The various methods of soft tissue augmentation for root coverage procedures may also be an option depending on the condition of the hard and soft

tissues (Al-Sabbagh et al. 2009; Chambrone et al. 2012; Schmidlin and Sahrmann 2013). According to Al-Sabbagh et al. (2009), however, there are limited clinical data to support this technique as part of a treatment approach in treating DH.

Over-the-Counter (OTC) Products

Individuals with mild to moderate local or generalised DH may decide to resolve any pain associated with DH without going to see a clinician by purchasing desensitising products that have been advertised in the media or through recommendation by a close friend or family member. According to Mantzourani and Sharma (2013), there are three main reasons that contribute to this behaviour: the need of a fast and urgent relief, the easy availability of these products in the market and their low cost, as this does not include the expenses of professional appointments and subsequent remedies. Desensitising products that are available through pharmacies or commercial outlets are called OTC or at-home care products in the form of toothpaste, gel or mouthrinse formulations. These OTC products are available without a medical prescription, but their indications for use are limited in that if the individual continues to have pain for longer than 2–4 weeks, they are advised to see a clinician. These OTC/at-home products may also be recommend by the clinician in association with in-office treatments (see Chap. 10), when the pain is associated with generalised DH, which is mild to moderate in severity.

OTC consumer products are generally based on formulations with the same active ingredients as for in-office products although the concentration permitted in OTC products may be considerably lower than those used by the clinician, for example, fluoride concentrations used in the office compared to fluoride concentrations used in OTC toothpastes. The constituents of these OTC products are usually formulated using a variety of salts (with different concentrations depending on whether the formulation is in a toothpaste, gel or mouthrinse) with the objective of reducing pain by dentin tubule occlusion or nerve desensitisation.

Most of the OTC products available to the consumer are toothpaste products, although there is a number of mouthrinses currently available in the market. Although these rinses may deliver the therapeutic agent to all tooth surfaces in the oral cavity, one disadvantage would appear to be the necessity to deliver lower concentrations of a therapeutic agent in the mouthrinse compared to a toothpaste formulation (Markowitz 2013). The mechanism of action of the mouthrinses however is similar to a toothpaste formulation depending on the formulation used to deliver the therapeutic agent. Examples of these salts are potassium nitrate, potassium chloride, potassium citrate, sodium fluoride, strontium chloride, strontium acetate, dibasic sodium citrate, formaldehyde, sodium monofluorophosphate, stannous fluoride, amine fluoride, arginine, hydroxyapatite, calcium carbonate, calcium sodium phosphosilicate (bioactive glass) and amorphous calcium phosphate (Miglani et al. 2010; Orchardson and Gillam 2006; Karim and Gillam 2013; Talioti et al. 2014) (Table 8.3).

Fluoride-Containing Toothpaste, Gel or Mouthrinse Formulations

According to Cury and Tenuta (2014), fluoride is the most important therapeutic substance used in toothpaste products and may provide a reduction in dental caries and together with the other beneficial therapeutic substances incorporated into toothpastes (e.g. triclosan/copolymer or zinc) may also have additional benefits in reducing the dental biofilm, gingivitis, periodontitis, calculus formation and halitosis (malodour). Although conventional fluoride-containing toothpaste formulations have been suggested for the treatment of DH, there are limited data to support this claim (Orchardson and Gillam 2006; West 2008). Toothpaste and mouthrinse formulations containing sodium, stannous or amine fluoride (NaF, MFP, AmF and SnF_2) have been used as OTC/at-home remedies to alleviate DH (Morris et al. 1999; Plagmann et al. 1997; Pradeep et al. 2012; Rösing et al. 2009). Nevertheless, inconsistent data are available on its clinical effectiveness (Pradeep et al. 2012). Earlier investigators

Table 8.3 Examples of selected toothpaste formulations for the treatment of DH available either on prescription or OTC

Mechanism of action	Commercial toothpaste (and mouthrinses)	Compounds
Dentin tubule occlusion	Colgate Sensitive Pro-Relief (Pro-Argin technology)	Calcium carbonate and arginine – an amino acid naturally found in saliva – 0.8 % arginine, PVM/MA copolymer, pyrophosphates and 0.05 % sodium fluoride in an alcohol-free base mouthwash
	Sensodyne Repair and Protect (NovaMin® technology)	Fluoride, calcium and phosphate ions
	Sensodyne Pronamel	Fluoride and potassium nitrate
	Sensodyne Rapid Relief	8 % Strontium acetate in a silica base, 1,040 ppm sodium fluoride
	SootheRx (NovaMin® technology)	Calcium sodium phosphosilicate (new hydroxyapatite)
	AMFLOR (Pharmaceuticals)	3.85 % Amine fluoride
Clinician-prescribed fluoride toothpaste formulations	Fluoridex (Philips Oral Healthcare)	5,000 ppm Fluoride
	Colgate Gel Kam Sensitive	Stannous fluoride 0.4 % w/w, 1,000 ppm F
	Colgate Duraphat	2,800 ppm Fluoride
Neural desensitisation	Potassium-containing products	Potassium chloride, citrate, nitrate and oxalate and fluoride compounds
Dentin tubule occlusion and neural desensitisation (other products)	Sensitive toothpaste (Himalaya herbals)	Spinach – natural oxalate compound

acknowledged that fluoride containing toothpaste formulations may have a beneficial desensitising effect (Kanouse and Ash 1969; Orchardson and Gillam 2000) and in some studies provided similar results to those reported for the specific desensitising toothpaste formulations containing nerve desensitising or dentin tubule-occluding agents (Gillam et al. 1997; Orchardson and Gillam 2000; West et al. 1997). Several investigators have used fluoride-containing formulations in clinical trials as a placebo control rather a desensitising product per se, when evaluating desensitising products (Gillam et al. 1996b; West et al. 1997; Yates et al. 2004; Pradeep et al. 2012; Rösing et al. 2009).

The mode of action of fluoride in anticariogenic, remineralising and DH strategies was based on its ability to react with hydroxyapatite and form fluorapatite, which was to be considered less susceptible to acid dissolution than hydroxyapatite due to its lower critical pH (Featherstone 2000). Calcium fluoride may also be deposited on the dentin surface when fluoride is incorporated in a toothpaste, mouthrinse or varnish, subsequently blocking the dentin tubules in vitro. With respect to these fluoride compounds, the fluoride ions apparently do not contribute effectively to dentin tubule occlusion (Petersson 2013), probably due to the small size of calcium content precipitated crystals (Calabria et al. 2012). Figure 8.2 shows the pattern of calcium fluoride crystal precipitation (a) in comparison with calcium oxalate crystals (b) from both fluoride-containing formulations and potassium oxalate formulations (in-office products). As demonstrated by these pictures, fluoride precipitates predominantly in the body of the intertubular dentin. Relatively small number of particles can be observed inside the tubules, whereas in the potassium oxalate sample, a large number of dentin tubules are occluded below the surface of the dentin. These CLSM images can also be compared with mineralised and acid conditioned dentin (a and b – in Fig. 8.1). This apparently ineffective action observed in in vitro studies does not however correspond to the clinical studies using fluoride formulations, and this may be

due to the difficulties in completely mimicking the oral environment in the laboratory (see Chap. 6). A number of these studies have demonstrated that DH may be reasonably well controlled by using a fluoride-containing toothpaste (1,450 ppm) during the duration of the study, especially in the combination with the various tubule-occluding agents discussed in this chapter.

The incorporation of metal ions (e.g. zinc, tin, strontium and potassium) and abrasive components in a toothpaste or mouthrinse formulation (e.g. alumina, silica, calcium carbonate, zinc, etc.) may also have a synergistic effect with the active ingredient on the dentin surface and block the dentin tubules (Gillam 1992; Petersson 2013). For example, a F-toothpaste with 0.454 % stannous fluoride (SnF_2) has been reported to be more effective in reducing DH following 4 weeks of daily use when compared to a toothpaste with 0.76 % MFP (Schiff et al. 2000). He et al. (2011) also reported a significant improvement in both significantly immediate and ongoing relief from a 0.45 % SnF_2 toothpaste compared to a 0.76 % sodium monofluorophosphate (MFP) toothpaste utilised as a negative-control group. Other desensitising agents in combination with fluoride and associated metal ions (e.g. strontium, 8 % arginine and calcium carbonate) have also been reported to be effective in clinical studies (Orcahrdson and Gillam 2006). Amine fluorides (AMFLOR™, Pharmaceuticals) have also been used in toothpaste and mouthrinse formulations to alleviate pain associated with DH, although currently there are limited data on its effectiveness as a desensitising agent. A recent study by Pradeep et al. (2012) however reported that a amine fluoride toothpaste formulation had a similar effect on DH, compared to a calcium sodium phosphosilicate and potassium nitrate containing toothpaste.

The adjunctive use of a fluoridated mouthrinse therapy in controlling DH in conjunction with the regular use of a desensitising toothpaste may also be recommended (e.g. potassium and amine fluoride) (Yates et al. 2004; Petersson and Kambara 2004; Glockner 2013; Petersson 2013; Pradeep et al. 2012), although currently there are limited data on their effectiveness (Petersson 2013). Other fluoridated mouth rinse formulations incorporat-

ing the various desensitising agents mentioned in this chapter may also enhance the effectiveness of fluoride toothpaste formulations (Petersson and Kambara 2004) and perhaps favour the remineralisation of dentin (Glockner 2013).

Irrespective of the actual active agent, the effectiveness of OTC products is dependent of the individual (patient or consumer) maintaining regular and effective oral hygiene practices by applying the toothpaste and/or mouthrinse on a twice daily basis (Petersson 2013). However, depending on the nature of these OTC products, its active component may take up to 2–4 weeks to achieve relief of DH (Talioti et al. 2014) (see also Chap. 10). One of the problems in evaluating the claims of these desensitising products in clinical studies is the lack of heterogeneity of the published studies which makes the results difficult to understand (Karim and Gillam 2013; Talioti et al. 2014). Furthermore, as discussed in Chap. 7, placebo and non-placebo effects may also impact on the results from these studies, although Lin et al. (2013) in a systematic review reported that despite these observed effects, both in-office and OTC products, techniques and treatment approaches may have a real effect on DH.

With regard to the effectiveness of the regular use of a fluoride-containing toothpaste in reducing DH, it was observed that the prevalence was higher in regions where individuals regularly used fluoride toothpaste and lived in fluoridated water area. This may, to some extent, question the apparent effectiveness of fluoride in minimising DH (Rösing et al. 2009; West 2008). According to Rösing et al. (2009), there are, however, limited data supporting the sole use of OTC fluoride products for treating DH, but fluoride in combination with tubule-occluding toothpaste and mouthrinse formulations may be a valuable option in terms of preventing and controlling other oral conditions, namely, plaque, caries, gingivitis, malodour, etc. (Cummins 2010; Pradeep et al. 2012; Glockner 2013; Petersson 2013). There is no evidence supporting the efficacy of these anticaries claims for specific desensitising toothpaste and mouthrinse formulations per se, but only appended claims of fluoride efficacy from the anticaries studies (so-called piggyback claims) (see Chap. 1). Whether high concentration of fluoride in

in a toothpaste, for example, 2,800/5,000 ppm (Duraphat) (see Table 8.2), may be effective as desensitisers has not been fully explored, although they have a recognised anticaries effect and may be effective in preventing and controlling root caries (Bizhang et al. 2009; Nordström and Birkhed 2010; Walsh et al. 2010; Srinivasan et al. 2014).

Strontium-Containing Products

Although strontium (historically in the form of strontium chloride and more recently as strontium acetate) in toothpaste formulations have been extensively evaluated in clinical studies over the last 50–60 years and reported to be effective in treating DH compared to placebo controls (Kobler et al. 2008; Liu and Hu 2012; Sharif et al. 2013; Markowitz 2009; Mason et al. 2010), nevertheless there was controversy over whether these formulations were effective in treating DH (Schiff et al. 1994; Rösing et al. 2009; Cummins 2010; Karim and Gillam 2013). Furthermore, Jackson (2000) in a review on the studies on strontium containing toothpaste formulations reported that none of these studies demonstrated a consistent, significant improvement in the participants' symptoms from DH when compared with the negative-control toothpaste. There was also, according to Jackson (2000), no supportive evidence from the published literature that the strontium salts enhanced the deposition of the toothpaste ingredients or increased the durability of this deposit on the tooth surface. The main mode of action of strontium formulations (acetate/chloride) was considered to be one of tubular occlusion based on in vitro studies (Ling and Gillam 1996; West 2008) although the actual effect attributed to strontium in clinical studies has yet to be defined (Rösing et al. 2009). Recently, the original strontium acetate-based toothpaste was reformulated as Sensodyne(®) Rapid Relief containing 8 % strontium acetate in silica base and 1,040 ppm sodium fluoride. There have been a number of in vitro, in situ and in vivo studies that have demonstrated that the mode of action is by tubule occlusion and that the surface deposit is more resistant to an acid challenge compared to the arginine-based products (Hughes et al. 2010; Mason et al. 2010; Parkinson and Willson 2011; Olley et al. 2012; Seong et al. 2013). The two in vivo studies by Hughes et al. and Mason et al. (2010) reported that the newly formulated strontium acetate toothpaste was effective in reducing DH compared to a fluoride control toothpaste (Mason et al. 2010) and to a marketed control toothpaste containing 8 % arginine, calcium carbonate, and 1,450 ppm sodium monofluorophosphate (Hughes et al. 2010). However Schiff et al. (2009) in a 16-week crossover design study comparing Colgate Sensitive Pro-Relief (also marketed as Elmex Sensitive Professional) and Sensodyne Rapid Relief reported that following 8 weeks of brushing with Colgate Sensitive Pro-Relief provided significant reduction of mean DH values compared to the Sensodyne Rapid Relief toothpaste. A further observation from this study was that the Colgate Sensitive Pro-Relief subjects achieved significantly improved reductions in DH following the crossover from the initial use of brushing with the Sensodyne Rapid Relief toothpaste. However those subjects who initially brushed with the Colgate Sensitive Pro-Relief toothpaste did not benefit from the crossover to the Sensodyne Rapid Relief.

Potassium-Containing Products

Potassium-containing products (nitrate, chlorine and citrate) including both toothpaste and mouthrinse formulations have been reported to be effective in reducing DH compared to placebo controls (Orchardson and Gillam 2000; Frechoso et al. 2003; Markowitz 2009). Of these formulations, 5 % potassium nitrate toothpaste formulations have been the most extensively evaluated desensitising toothpaste (Rösing et al. 2009). Overall, the majority of the published studies have reported better results with the use of potassium-containing toothpaste formulations, in comparison to controls (Chesters et al. 1992; Collins et al. 1984; Nagata et al. 1994; Orchardson and Gillam 2000; Pradeep et al. 2012; Schiff et al. 1994, 1998; Silverman 1985; Silverman et al. 1996; Tarbet et al. 1980, 1982) or compared to other desensitising toothpaste

formulations (10 % strontium chloride (Orchardson and Gillam 2000; Silverman et al. 1996; Tarbet et al. 1982), 2 % sodium citrate and or 1.4 % formaldehyde (Orchardson and Gillam 2000; Tarbet et al. 1982). However there was an ongoing debate as to whether the actual clinical effect was due to the potassium ion per se (Orchardson and Gillam 2006; Poulsen et al. 2001, 2006; Rösing et al. 2009; Karim and Gillam 2013; Lin et al. 2013; Schmidlin and Sahrmann 2013). For example, there has been a degree of controversy as a result of its proposed mode of action (nerve desensitisation) based on an animal model (Markowitz et al. 1991; Markowitz and Kim 1992) which postulated that potassium ions are able to diffuse along the dentin tubules and decrease the excitability of the intra-dental nerve fibres (A fibres) by blocking the axonic action, and subsequently reduce DH (Schmidlin and Sahrmann 2013; Boneta et al. 2013). Several investigators have however challenged this mode of action as indicated above and have suggested that these formulations may instead be through tubule occlusion and attributed to other constituents in the formulation (e.g. abrasives) than the potassium ion per se (Addy and Mostafa 1989; Ling et al. 1997; Gillam et al. 1996a, b; Pashley et al. 1984; West et al. 1997; Orchardson and Gillam 2000). According to Jackson (2000) and Panagakos et al. (2009), a potassium-containing toothpaste formulation was, no more effective than regular fluoride containing toothpaste.

As described above, the focus in the majority of the earlier desensitising studies was on toothpaste formulations, and relatively few studies have been conducted examining the effectiveness of desensitising agents containing potassium mouthrinse formulations. Several investigators have reported that these mouthrinse formulations containing potassium nitrate and sodium fluoride (Gillam et al. 1996a; Pereira and Chavas 2001), potassium citrate or sodium fluoride (Yates et al. 1998) or a mixture of fluorides (Yates et al. 2004) may reduce DH. Gillam et al. (1996a) have demonstrated that a 3 % potassium nitrate and sodium fluoride mouthrinse significantly reduced DH compared to a sodium fluoride mouthwash, after 2 and 6 weeks of use. Pereira and Chavas (2001) also demonstrated that after 2 weeks, there were

no statistically significant differences between the two groups using tactile and thermal stimuli. At 6 weeks, however, the 3 % potassium nitrate and 0.2 % sodium fluoride mouthwash demonstrated a significant difference in DH when stimulated by cold air, as compared to the control 0.2 % sodium fluoride mouthwash.

More recently, a potassium oxalate mouthrinse containing 1.4 % potassium oxalate (Listerine Advanced Defence Sensitive (LADS)) has been developed by Johnson & Johnson Consumer and Personal Products Worldwide, Skillman and Morris Plains, NJ, USA. There are however limited data on its long-term effectiveness on DH with only two randomised clinical studies (RCTs) of 5 days and 4 weeks, respectively (Sharma et al. 2013a, b). The investigators reported that in both studies, the potassium oxalate mouthrinse significantly reduced DH to the negative-control groups (Sharma et al. 2013a, b). Further RCTs however would be required before making any further comment on the efficacy of this product in reducing DH. Although it may be postulated that with the combination of potassium and oxalate there would be dual mode of action in reducing DH (namely, nerve desensitisation [potassium] and tubule occlusion [oxalate]), the two published in vitro studies used a tubule-occluding model to demonstrate the mode of action by the oxalate deposit. Two studies demonstrated that the LADS mouthrinse was significantly more effective in tubule occlusion (intratubular penetration was more evident) (Eliades et al. 2013; Sharma et al. 2013c) and in reducing dentin permeability than the other test products. Furthermore according to these investigators, the tubule occlusion associated with LADS was substantially more stable in resisting an acid challenge compared to the other test products.

Calcium-Containing Compounds

Casein Phosphopeptide-Amorphous Calcium Phosphate (CPP-ACP)

Casein phosphopeptide (CPP) (Recaldent™ technology, GC Tooth Mousse) was primarily developed for both anticaries and remineralisation

strategies rather than for the treatment of DH per se. Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) was previously marketed as GC Tooth Mousse (Recaldent™), which was a water-based, sugar-free dental topical crème. According to Reynolds et al. (1998), the CPP component (casein derived from the milk protein) was proposed by Reynolds (1997) to provide bioavailable calcium and phosphate ions at the tooth surface (e.g. enamel). The stabilisation of ACP component by the CPP ensured the delivery of both Ca and PO₄ ions onto the enamel surface for remineralisation to CPP, and formulated as CPP-ACP, this subsequently prevented the dissolution of both calcium and phosphate ions that supersaturate saliva (Reynolds 1997). A number of studies have reported that CPP-ACP can effectively remineralise the enamel subsurface lesions (Lata et al. 2010; Cai et al. 2003). The manufacturers suggested that CPP-ACP would be ideal to treat DH on the basis of its remineralising potential, and a number of in vitro and in vivo studies were conducted which demonstrated that calcium phosphate preparations deposit a mineral precipitate onto the dentin. surface, block dentin tubules and reduce dentin permeability in the dentin disk model and reduce DH in patients (Ebisu 2002; Suge et al. 2002; Cherng et al. 2004; Geiger et al. 2003; Azarpazhooh and Limeback 2008; Gandolfi et al. 2010; Walsh 2010). According to Wegehaupt et al. (2011), however, it is questionable that the casein CPP-ACP formulation would demonstrate any long-lasting effect on DH. A recent systematic review by Talioti et al. (2014) reported that although the CPP-ACP containing products may provide some benefit in reducing DH, there was insufficient data from the published studies to support any claims of efficacy in reducing DH.

Functionalised Tricalcium Phosphate (fTCP)

According to Vanichvatana and Auychai (2013), fTCP is produced by the solid-state ball milling of beta-tricalcium phosphate and sodium lauryl sulphate and has been demonstrated by several investigators to prevent calcium ions from prematurely interacting with ionic fluoride and

forming calcium fluoride, thus delivering more fluoride and calcium ions to the enamel surface (Karlinsey et al. 2010, 2012). fTCP has also been reported to have remineralising effects in both in vitro and in situ studies (Karlinsey et al. 2009a, b; Mensinkai et al. 2012; Vanichvatana and Auychai 2013). fTCP has recently been commercially developed and is now available as a 950 ppm fluoride toothpaste (Clinpro Tooth Creme; 3 M ESPE, Saint Paul, MN, USA). Asaizumi et al. (2013) also assessed the effect on 0.21 or 1.1 % NaF toothpaste containing functionalised tricalcium phosphate (fTCP) on the density of white spots in vitro, and Mensinkai et al. (2012) in an in situ remineralisation study of white-spot enamel lesions compared a nonfluoride toothpaste with 500 and 1,100 ppm F-containing fTCP toothpaste. Although there appears to be data on file (technical reports) on the effects (tubule occlusion and dentin permeability) of TCP products on DH (e.g. Vanish™ XT Extended Contact Varnish), there does not appear to be any data currently available in the published dental literature.

Pro-Argin-Based Toothpaste and Mouthrinse formulations

Pro-Argin, a calcium carbonate and arginine complex based on Kleinberg's original formulation Sensistat (Kleinberg 2002), has been introduced into the consumer market as a desensitising toothpaste (Colgate® Sensitive Relief with Pro-Argin™ technology) and more recently as a desensitising mouthrinse (Colgate-Palmolive Co, New York, NY) (Mello et al. 2013a, b; Hu et al. 2013; Boneta et al. 2013) for the treatment of DH. Arginine is an amino acid found naturally in saliva. The combination of arginine and calcium carbonate mimics the saliva's ability to occlude the open dentin tubules, subsequently reducing the fluid flow and making the tooth surface more resistant to an acid challenge and thermal stimulus (Petrou et al. 2009) (see also section on strontium-based toothpaste). Pro-Argin™ toothpaste have also been reported to reduce DH in randomised clinical studies when compared to either placebo or comparator products (Petrou et al. 2009; Ayad et al. 2009; Docimo et al. 2009a, b;

Que et al. 2010; Fu et al. 2010; Boneta et al. 2013). Several clinical studies have also reported that a Pro-Argin™ mouthrinse containing 0.8 % arginine, PVM/MA copolymer, pyrophosphates and 0.05 % sodium fluoride in an alcohol-free base mouthrinse was effective compared to control mouthrinses in reducing DH (Hu et al. 2013; Boneta et al. 2013). Several investigators have suggested a role for a arginine-based toothpaste in managing DH (Fu et al. 2010; Que et al. 2010; Sharif et al. 2013) although Sharif et al. (2013) has suggested that more robust and well-designed randomised clinical studies should be conducted to determine their clinical efficacy in reducing DH. Furthermore according to Sharif et al. (2013), larger sample sizes are required in the clinical studies with a longer study duration included in the study design.

Calcium Sodium Phosphosilicate (Bioactive Glass)

As previously described, bioactive glasses (calcium sodium phosphosilicate), for example, NovaMin® developed by NovaMin Technology Inc. (Alachua, FL, USA) based on the original 45S5 Bioglass® formulation by Larry Hench (US Biomaterials Corp., Jacksonville, FL, USA; now GSK) (Hench 2006) has been incorporated into toothpaste products for the treatment of DH. The proposed mode of action is by the precipitating of hydroxycarbonate apatite (HCA) onto the dentin surface and subsequently occluding the dentin tubules (Litkowski et al. 1998; Gillam et al. 2002; Forsback et al. 2004; Tai et al. 2006; Vollenweider et al. 2007; Burwell 2006; Burwell et al. 2009; Wang et al. 2010; Pradeep and Sharma 2010).

One advantage of the precipitated HCA layer was that it was chemically and structurally similar to both natural enamel and dentin (Burwell 2006; Gendreau et al. 2011). There have however been concerns over the long-term durability of HCA in the oral environment, and it has been postulated that the formation of fluorapatite (FAp) rather than HCA was preferable as this layer may be more resistant to acid attack and would less likely to dissolve when the teeth are exposed to an acid challenge. Brauer et al. (2010) recently demonstrated that fluoride-containing bioactive glasses form FAp rather than HCA in physiological solutions.

As described previously, the original 45S5 Bioglass® formulation was marketed as a desensitising toothpaste for the treatment of DH, namely, NovaMin™ (NovaMin Technology Inc., Alachua, FL, USA; now GSK Consumer HealthCare, UK). The new NovaMin™ toothpaste is based on an anhydrous toothpaste formulation with silica and amorphous calcium sodium phosphosilicate with fluoride (not currently with fluoride in the USA). The toothpaste has been demonstrated to be effective in occluding the dentin tubules in vitro and reported to be resistant to an acid challenge as well as demonstrating clinical efficacy compared to other desensitising products (Andersson and Kangasniemi 1991; Forsback et al. 2004; Du Min et al. 2008; Pradeep and Sharma 2010; Litkowski and Greenspan 2010; Narongdej et al. 2010; Dong et al. 2011; Wang et al. 2011a, b; Gendreau et al. 2011; Rajesh et al. 2012; Pradeep et al. 2012; Sharif et al. 2013; Petersson 2013). However one of the problems in evaluating the results from the clinical studies is the lack of homogeneity in the methodology and designs of the studies which makes the analysis of these results difficult (Talioti et al. 2014) (see Chap. 1).

Innovative Developments in the Treatment of Non-carious Cervical Lesions as a Strategy for the Management of DH

Protease Inhibitors

According to Kleter et al. (1993), once dentin is exposed to oral environment, the collagen layer is also exposed to the variations in pH which may subsequently result in a mineral depleted layer which may hamper the ionic diffusion of minerals from the saliva into the demineralised area (the degree of collagen loss is based on the demineralisation rate). In order to preserve the demineralised organic matrix, it would therefore be reasonable to use products that would be expected to reduce the progression of dentin loss

(Kato et al. 2012). The exposed organic matrix is relatively resistant to mechanical forces but was reported to be susceptible to biochemical degradation by proteases, for example, metalloproteinases (MMPs) from the saliva and from the dentin matrix itself (Tjäderhane et al. 1998).

In order to potentially preserve the organic dentin (collagen) matrix, several investigators have examined the role of known protease inhibitors, for example, polyphenol epigallocatechin-3-gallate (Demeule et al. 2000), chlorhexidine (Gendron et al. 1999; Carrilho et al. 2009; Scaffa et al. 2012) and ferrous sulphate (Kato et al. 2010). Initially the results are promising as the amount of dentin loss was reduced by 39–100 % with a number of these protease inhibitors in association with different vehicles (Kato et al. 2009, 2010; Magalhães et al. 2009). According to Kato et al. (2012), the data from these studies would suggest that the mechanism of action of these substances in preventing dentin erosion was due to their ability to inhibit the proteases, thus preserving demineralisation of the dentin matrix. These investigators also studied the protective effect of 400 μM polyphenol epigallocatechin-3-gallate, 0.012 % chlorhexidine, 1 mM ferrous sulphate solution and 1.23 % NaF solution, before submitting the dentin specimens to collagen degradation with *Clostridium histolyticum*, confirmed by the assaying of hydroxyproline in artificial saliva. The results demonstrated that the concentrations of hydroxyproline were significantly lower for the groups treated with polyphenol epigallocatechin-3-gallate, chlorhexidine and ferrous sulphate, respectively, when compared to the groups of sodium fluoride, placebo or with the untreated group. These results strongly suggested that the preventive effect of the protease inhibitors tested against dentin erosion was due to their ability to reduce the degradation of demineralised organic matrix (Kato et al. 2012).

Recently, Calabria et al. (2014) investigated the effect of a titanium fluoride (TiF4) solution on the hydraulic conductance of dentin, in comparison with a potassium oxalate gel, a sodium fluoride varnish and solution. The investigators reported that the TiF4 solution reduced dentin permeability by approximately 35 % (a similar percentage to

that of a sodium fluoride solution). The permeability of the dentin specimens treated with potassium oxalate and a sodium fluoride varnish was reduced 85 and 65 %, respectively, in the same experiment. However, the association of these salts with TiF4 or with any other protease inhibitors has not been currently investigated in a clinical study.

Although there appears to be limited data available on these protease inhibitors, it may be speculated that if these inhibitors were able to reduce the subsurface demineralisation of organic dentin matrix either as a product or in association with the traditional desensitisers, then they may be relevant for the prevention of dental erosion and the treatment of DH.

Clinical Evidence of the Efficacy of OTC and In-Office Products

After almost 100 years of research, the clinical management of DH continues to be largely empirical in nature and very challenging to the clinician (Glockner 2013; Orchardson and Collins 1987; Pradeep et al. 2012). In view of the wide range of agents and procedures used to treat DH (Ling and Gillam 1996; Orchardson and Gangarosa 1994; Orchardson and Gillam 2000; Cunha-Cruz et al. 2010), it would be unreasonable for the clinician to expect that one particular product would resolve every aspect of the problem or that these particular treatment strategies would be appropriate for every individual (Lin et al. 2013; Gillam et al. 2013; Schmidlin and Sahrmann 2013) (see Chap. 10).

In general, in-office treatments are successful in treating DH in patients with a localised severe problem in one or more teeth, depending on the desensitiser used to treat the problem. OTC products which are recommended for mild to moderate generalised DH usually demonstrate a reduction in DH over time (Brauer et al. 2010; Wang et al. 2010). The advantage of using products available for home use is that they are immediately available for treatment when compared with those products applied by the professional. One disadvantage however is that more time is required for the remission of symptoms

(2–4 weeks), while those applied in-office promote immediate relief (Lynch et al. 2012; Pradeep et al. 2012) (see Chap. 10).

Conclusions

The continual introduction of new desensitising materials and treatment approaches would appear to suggest that there is no ideal desensitising product or clinical approach that can fully satisfy the original postulates of Grossman or fully address the impact of DH on the quality of life of patients (Grossman 1935; Gillam 1997) (see Chap. 9). It may however be unreasonable to expect that any one desensitising product or treatment approach would successfully resolve all the clinical factors associated with DH, and therefore the search for the ideal desensitising agent must be implemented (Gillam et al. 2013; Schmidlin and Sahrmann 2013). Furthermore there does appear an existing problem in both terms of the confidence of the clinician in successfully treating DH and in identifying an ideal desensitising product and treatment approach (Cunha-Cruz et al. 2010; Schmidlin and Sahrmann 2013; van Loveren 2013). There is, therefore, a need for the establishment of evidence-based clinical guidelines that may be used in daily practice (Al-Sabbagh et al. 2009). The recent publication by Gillam et al. (2013) attempted to address this particular problem, but there is clearly a need for such guidelines to be simple and pragmatic in order for the clinician to implement them successfully in clinical practice. The importance of a simple stepwise approach that may be supplemented with both diagnosis and preventive (counselling) measures, as well as long-term monitoring, is also imperative for any successful management strategy (Orchardson and Gillam 2006; Gillam et al. 2013; Lussi and Hellwig 2013) (see Chap. 10).

Future Trends

Clinicians in their daily practice must therefore consider two different aspects regarding the patient's experience of DH. One is related to the patient's discomfort and their need for an immediate pain resolution. The other is in regard to the entire spectra of events that may lead to a non-pathological exposure of dentin tissue in the oral environment. Both of these aspects demand that the clinician has not only a wide range of knowledge and clinical experience in order to offer the best treatment and the corrective strategies to their patients. Furthermore it is essential that well-controlled randomised clinical studies are undertaken in order to investigate new materials and their properties under rigorous clinical conditions. Currently there does not appear to be an ideal desensitising product (which successfully mimics the natural tissue) that can be effectively utilised in both in-office and in OTC/at-home environments. It is therefore imperative that researchers responsible for the development of new materials direct their attention into areas of research with targeted formulations that can mimic and restore the natural structures of the tooth.

References

Addy M, Mostafa P (1989) Dentine hypersensitivity. II. Effects produced by the uptake in vitro of toothpastes onto dentine. J Oral Rehabil 16(1):35–48

Agnihotri Y, Pragada NP, Patri G, Thajuraj PK (2012) The effect of CPP-ACP on remineralization of artificial caries like lesions: an in vitro study. Indian J Multidiscip Dent 2(1):366

Ahmedbeyli C, Ipçi ŞD, Cakar G, Kuru BE, Yılmaz S (2014) Clinical evaluation of coronally advanced flap with or without acellular dermal matrix graft on complete defect coverage for the treatment of multiple gingival recessions with thin tissue biotype. J Clin Periodontol 41(3):303–310. doi:10.1111/jcpe.12211, Epub 2014 Jan 16

Al-Sabbagh M, Brown A, Thomas MV (2009) In-office treatment of dentinal hypersensitivity. Dent Clin N Am 53(1):47–60, viii

Anderegg CR, Alexander DC, Freidman M (1999) A bioactive glass particulate in the treatment of molar furcation invasions. J Periodontol 70(4):384–387

Andersson OH, Kangasniemi I (1991) Calcium phosphate formation at the surface of bioactive glass in vitro. J Biomed Mater Res 25(8):1019–1030

Aparna S, Setty S, Thakur S (2010) Comparative efficacy of two treatment modalities for dentinal hypersensitivity: a clinical trial. Indian J Dent Res 21:544–548

Aranha AC, Domingues FB, Franco VO, Gutknecht N, Eduardo CP (2005) Effects of Er:YAG and Nd:YAG

lasers on dentin permeability in root surfaces: a preliminary in vitro study. Photomed Laser Surg 23(5):504–508

Aranha AC, Pimenta LA, Marchi GM (2009) Clinical evaluation of desensitizing treatments for cervical dentin hypersensitivity. Braz Oral Res 23(3):333–339

Arenholt-Bindslev D, Hörsted-Bindslev P, Philipsen HP (1987) Toxic effects of two dental materials on human buccal epithelium in vitro and monkey buccal mucosa in vivo. Scand J Dent Res 95(6):467–474

Asaizumi M, Karlinsey RL, Mackey AC, Kato T, Kuga T (2013) In vitro assessments of white-spot lesions treated with NaF plus tricalcium phosphate (TCP) toothpastes using microtomography (micro-CT). J Dent Oral Hyg 5(7):68–76

Ayad F, Ayad N, Delgado E, Zhang YP, DeVizio W, Cummins D, Mateo LR (2009) Comparing the efficacy in providing instant relief of dentin hypersensitivity of a new toothpaste containing 8.0% arginine, calcium carbonate, and 1450 ppm fluoride to a benchmark desensitizing toothpaste containing 2% potassium ion and 1450 ppm fluoride, and to a control toothpaste with 1450 ppm fluoride: a three-day clinical study in Mississauga, Canada. J Clin Dent 20(4):115–122

Azarpazhooh A, Limeback H (2008) Clinical efficacy of casein derivatives: a systematic review of the literature. J Am Dent Assoc 139(7):915–924

Bader J, Balevi B, Farsai P, Flores-Mir C, Gunsolley J, Matthews D, Vig K, Zahrowski J (2014) Lasers may reduce pain arising from dentin hypersensitivity. J Am Dent Assoc 145(4):e1–e2

Birang R, Poursamimi J, Gutknecht N, Lampert F, Mir M (2007) Comparative evaluation of the effects of Nd:YAG and Er:YAG laser in dentin hypersensitivity treatment. Lasers Med Sci 22(1):21–24

Bizhang M, Chun Y-HP, Winterfeld M-T, Altenburger MJ, HM Raab WHMand Zimmer S (2009) Effect of a 5,000 ppm fluoride toothpaste and a 250 ppm fluoride mouth rinse on the demineralisation of dentin surfaces. BMC Res Notes 2:147

Boneta ARE, Ramirez K, Naboa J, Mateo LR, Stewart B, Panagokos F, De Vizio W (2013) Efficacy of a mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride compared to a commercial mouthwash containing 2.4% potassium nitrate and 0.022% sodium fluoride and a control mouthwash containing 0.05% sodium fluoride on dentine hypersensitivity: a six-week randomized clinical study. J Dent 41:S34–S41

Brahmbhatt N, Bhavsar N, Sahayata V, Acharya A, Kshatriya P (2012) A double blind controlled trial comparing three treatment modalities for dentin hypersensitivity. Med Oral Patol Oral Cir Bucal 17(3):e483–e490

Brauer DS, Karpukhina N, O'Donnell MD, Law RV, Hill RG (2010) Fluoride-containing bioactive glasses: effect of glass design and structure on degradation, pH and apatite formation in simulated body fluid. Acta Biomater 6(8):3275–3282

Brough KM, Anderson DM, Love J, Overman PR (1985) The effectiveness of iontophoresis in reducing dentin hypersensitivity. J Am Dent Assoc 111(5):761–765

Brunton PA, Kalsi KS, Watts DC, Wilson NH (2000) Resistance of two dentin-bonding agents and a dentin desensitizer to acid erosion in vitro. Dent Mater 16(5):351–355

Burwell A (2006) Tubule occlusion of a novamin-containing dentifrice compared to Recaldent-containing dentifrice – a Remin/Demin study in vitro. Novamin Research Reports

Burwell AK, Litkowski LJ, Greenspan DC (2009) Calcium sodium phosphosilicate (NovaMin): remineralization potential. Adv Dent Res 21(1):35–39

Cai F, Shen P, Morgan MV, Reynolds EC (2003) Remineralization of enamel subsurface lesions in-situ by sugar free lozenges containing casein phosphopeptide – amorphous calcium phosphate. Aust Dent J 48:240–243

Cairo F, Pagliaro U, Nieri M (2008) Treatment of gingival recession with coronally advanced flap procedures: a systematic review. J Clin Periodontol 35(8):136–162

Cairo F, Nieri M, Pagliaro U (2014) Efficacy of periodontal plastic surgery procedures in the treatment of localized facial gingival recessions. A systematic review. J Clin Periodontol 41(15):S44–S62

Calabria MP, Silva LM, Francisconi LF, Modena KCS, Casas-Apayco LC, Nahsan FPS, Wang L, Atta MT, Pereira JC (2010) A comparative study of morphological adhesive interface between NCCL and sound teeth with CLSM and SEM. Bauru, 2010. J Appl Oral Sci Bauru 18(5):12, Sept. Oct. 2010. Supplement. (1678–7757)

Calabria MP, Dantas LM, Wang L, Magalhães AC, Lauris JRP, Graeff M, Pereira JC (2012) Effect of different desensitizing agents on dentin hydraulic conductance. J Dent Res 91:393, Sp. Issue B, abstr

Calabria M, Porfirio R, Fernandes S, Wang L, Buzalaf M, Pereira J, Magalhães A (2014) Comparative in vitro effect of TiF4 to NaF and potassium oxalate on reduction of dentin hydraulic conductance. Oper Dent 39(4):427–432

Carrilho MR, Tay FR, Donnelly AM, Agee KA, Tjäderhane L, Mazzoni A et al (2009) Host-derived loss of dentin matrix stiffness associated with solubilization of collagen. J Biomed Mater Res B Appl Biomater 90:373–380

Chambrone L, Chambrone D, Pustiglioni FE, Chambrone LA, Lima LA (2008) Can subepithelial connective tissue grafts be considered the gold standard procedure in the treatment of Miller class I and II recession-type defects? J Dent 36(9):659–671

Chambrone L, Pannuti CM, Tu YK, Chambrone LA (2012) Evidence-based periodontal plastic surgery. II. An individual data meta-analysis for evaluating factors in achieving complete root coverage. J Periodontol 83(4):477–490

Cherng AM, Chow LC, Takagi S (2004) Reduction in dentin permeability using mildly supersaturated calcium phosphate solutions. Arch Oral Biol 49(2):91–98

Chesters R, Kaufman HW, Wolff MS, Huntington E, Kleinberg I (1992) Use of multiple sensitivity measurements and logit statistical analysis to assess the effectiveness of a potassium-citrate-containing dentifrice in reducing dentinal hypersensitivity. J Clin Periodontol 19(4):256–261

Cochrane NJ, Shen P, Yuan Y, Reynolds EC (2014) Ion release from calcium and fluoride containing dental varnishes. Aust Dent J 59(1):100–105

Collins JF, Gingold J, Stanley H, Simring M (1984) Reducing dentinal hypersensitivity with strontium chloride and potassium nitrate. Gen Dent 32(1):40–43

Cooley RL, Sandoval VA (1989) Effectiveness of potassium oxalate treatment on dentin hypersensitivity. Gen Dent 37(4):330–333

Copeland JS (1985) Simplified remedy for root sensitivity. Northwest Dent 64(6):13–14

Cummins D (2010) Recent advances in dentin hypersensitivity: clinically proven treatments for instant and lasting sensitivity relief. Am J Dent 23(Spec No A):3A–13A

Cunha-Cruz J, Wataha JC, Zhou L, Manning W, Trantow M, Bettendorf MM, Heaton LJ, Berg J (2010) Treating dentin hypersensitivity: therapeutic choices made by dentists of the northwest PRECEDENT network. J Am Dent Assoc 141(9):1097–1105

Cunha-Cruz J, Stout JR, Heaton LJ, Wataha JC, Northwest PRECEDENT (2011) Dentin hypersensitivity and oxalates: a systematic review. J Dent Res 90(3):304–310

Cury JA, Tenuta LMA (2014) Evidence-based recommendation on toothpaste use. Braz Oral Res São Paulo 28(Spec Iss 1):1–7

da Rosa WL, Lund RG, Piva E, da Silva AF (2013) The effectiveness of current dentin desensitizing agents used to treat dental hypersensitivity: a systematic review. Quintessence Int 44(7):535–546

de Oliveira DW D, Marques DP, Aguiar-Cantuária IC, Flecha OD, Gonçalves PF (2013a) Effect of surgical defect coverage on cervical dentin hypersensitivity and quality of life. J Periodontol 84(6):768–775. doi:10.1902/jop.2012.120479, Epub 2012 Aug 16

de Oliveira DW D, Oliveira-Ferreira F, Flecha OD, Gonçalves PF (2013b) Is surgical root coverage effective for the treatment of cervical dentin hypersensitivity? A systematic review. J Periodontol 84(3):295–306. doi:10.1902/jop.2012.120143, Epub 2012 May 1

Demeule M, Brossard M, Page M, Gingras D, Beliveau R (2000) Matrix metalloproteinase inhibition by green tea catechins. Biochim Biophys Acta 1478:51–60

Deng Y, Mahdian M, George AT, Blake B, Huang D, Shi Z (2011) Laser therapy for dentinal hypersensitivity. Cochrane Database Syst Rev (11):CD009434

Docimo R, Montesani L, Maturo P, Costacurta M, Bartolino M, DeVizio W, Zhang YP, Cummins D, Dibart S, Mateo LR (2009a) Comparing the efficacy in reducing dentin hypersensitivity of a new toothpaste containing 8.0% arginine, calcium carbonate, and 1.450 ppm fluoride to a commercial sensitive toothpaste containing 2% potassium ion: an eight-week clinical study in Rome, Italy. J Clin Dent 20(1):17–22

Docimo R, Montesani L, Maturo P, Costacurta M, Bartolino M, Zhang YP, DeVizio W, Delgado E, Cummins D, Dibart S, Mateo LR (2009b) Comparing the efficacy in reducing dentin hypersensitivity of a new toothpaste containing 8.0% arginine, calcium carbonate, and 1.450 ppm fluoride to a benchmark commercial desensitizing toothpaste containing 2% potassium ion: an eight-week clinical study in Rome, Italy. J Clin Dent 20(4):137–143

Dondidall'Orologio G, Lorenzi R, Anselmi M, Opisso V (1999) Dentin desensitizing effects of gluma alternate, health-dent desensitizer and scotchbond multi-purpose. Am J Dent 12(3):103–106

Dondidall'Orologio G, Lone A, Finger WJ (2002) Clinical evaluation of the role of glutardialdehyde in a one-bottle adhesive. Am J Dent 15(5):330–334

Dong Z, Chang J, Deng Y, Joiner A (2011) Tricalcium silicate induced mineralization for occlusion of dentinal tubules. Aust Dent J 56(2):175–180

Drisko C (2002) Dentine hypersensitivity: dental hygiene and periodontal considerations. Int Dent J 52:385–393

Du Min Q, Bian Z, Jiang H, Greenspan DC, Burwell AK, Zhong J, Tai BJ (2008) Clinical evaluation of a dentifrice containing calcium sodium phosphosilicate (novamin) for the treatment of dentin hypersensitivity. Am J Dent 21(4):210–214

Duran I, Sengun A (2004) The long-term effectiveness of five current desensitizing products on cervical dentine sensitivity. J Oral Rehabil 31(4):351–356

Duran I, Sengun A, Yildirim T, Ozturk B (2005) In vitro dentine permeability evaluation of HEMA-based (desensitizing) products using split-chamber model following in vivo application in the dog. J Oral Rehabil 32(1):34–38

Ebisu S (2002) Calcium phosphate precipitation method for the treatment of dentin hypersensitivity. Am J Dent 15:220–226

Eliades G, Mantzourani M, Labella R, Mutti B, Sharma D (2013) Interactions of dentine desensitisers with human dentine: morphology and composition. J Dent 41(4):S28–S39

Boneta ARE, Ramirez K, Naboa J, Mateo LR, Stewart B, Panagokos F, De Vizio W (2013) Efficacy in reducing dentine hypersensitivity of a regimen using a toothpaste containing 8% arginine and calcium carbonate, a mouthwash containing 0.8% arginine, pyrophosphate and PVM/MA copolymer and a toothbrush compared to potassium and negative control regimens: an eight-week randomized clinical trial. J Dent 41(1):S42–S49

Erdemir U, Yildiz E, Kilic I, Yucel T, Ozel S (2010) The efficacy of three desensitizing agents used to treat dentin hypersensitivity. J Am Dent Assoc 141(3):285–296

Farmakis ET, Kozyrakis K, Khabbaz MG, Schoop U, Beer F, Moritz A (2012) In vitro evaluation of dentin tubule occlusion by Denshield and Neodymium-doped yttrium-aluminum-garnet laser irradiation. J Endod 38(5):662–666

Featherstone JD (2000) The science and practice of caries prevention. J Am Dent Assoc 131(7):887–899

Forsback AP, Areva S, Salonen JI (2004) Mineralization of dentin induced by treatment with bioactive glass S53P4 in vitro. Acta Odontol Scand 62:14–20

Francisconi LF, Graeff MS, Martins Lde M, Franco EB, Mondelli RF, Francisconi PA, Pereira JC (2009) The effects of occlusal loading on the margins of cervical restorations. J Am Dent Assoc 140(10):1275–1282

Frechoso SC, Menéndez M, Guisasola C, Arregui I, Tejerina JM, Sicilia A (2003) Evaluation of the efficacy of two potassium nitrate bioadhesive gels (5% and 10%)

in the treatment of dentine hypersensitivity. A random-ized clinical trial. J Clin Periodontol 30(4):315–320

Fu Y, Li X, Que K, Wang M, Hu D, Mateo LR, DeVizio W, Zhang YP (2010) Instant dentin hypersensitivity relief of a new desensitizing dentifrice containing 8.0% arginine, a high cleaning calcium carbonate system and 1.450 ppm fluoride: a 3-day clinical study in Chengdu, China. Am J Dent 23(Spec No A):20A–27A

Gaffar A (1999) Treating hypersensitivity with fluoride varnish. Compend Contin Educ Dent 20(1):27–33; quiz 35

Gandolfi MG, Taddei TA, Prati C (2010) Apatite-forming ability (bioactivity) of ProRoot MTA. Int Endod J 43(10):917–929

Gangarosa LP Sr (1994) Current strategies for dentist-applied treatment in the management of hypersensitive dentine. Arch Oral Biol 39(Suppl):101S–106S

Gangarosa LP, Park NH (1978) Practical considerations in iontophoresis of fluoride for desensitizing dentin. J Prosthet Dent 39(2):173–178

Geiger S, Matalon S, Blasbalg J, Tung M, Eichmiller FC (2003) The clinical effect of amorphous calcium phosphate (ACP) on root surface hypersensitivity. Oper Dent 28:496–500

Gendreau L, Barlow AP, Mason SC (2011) Overview of the clinical evidence for the use of NovaMin in providing relief from the pain of dentin hypersensitivity. J Clin Dent 22(3):90–95

Gendron R, Grenier D, Sorsa T, Mayrand D (1999) Inhibition of the activities of matrix metalloproteinases 2, 8, and 9 by chlorhexidine. Clin Diagn Lab Immunol 6:437–439

Gholami GA, Fekrazad R, Esmaiel-Nejad A, Kalhori KA (2011) An evaluation of the occluding effects of Er;Cr:YSGG, Nd:YAG, CO_2 and diode lasers on dentinal tubules: a scanning electron microscope in vitro study. Photomed Laser Surg 29(2):115–121

Gillam DG (1992) The assessment and treatment of cervical dentinal sensitivity – DDS Thesis, University of Edinburgh

Gillam DG (1997) Clinical trial designs for testing of products for dentine hypersensitivity – a review. Periodontal Abstr 45:37–46

Gillam DG (2014) Treatment approaches for dentin hypersensitivity. In: Taha S, Clarkson BH (eds) Clinician's guide to the diagnosis and management of tooth sensitivity. Springer, Berlin/Heidelberg, pp 51–80, Chapter 5

Gillam DG, Newman HN (1990) Iontophoresis in the treatment of cervical dentinal sensitivity–a review. J West Soc Periodontol Periodontal Abstr 38(4):129–133

Gillam DG, Bulman JS, Jackson RJ, Newman HN (1996a) Efficacy of a potassium nitrate-based mouthwash in alleviating cervical dentine sensitivity (CDS). J Clin Periodontol 23:993–997

Gillam DG, Bulman JS, Jackson RJ, Newman HN (1996b) Comparison of 2 desensitizing dentifrices with a commercially available fluoride dentifrice in alleviating cervical dentine sensitivity. J Periodontol 67(8):737–742

Gillam DG, Coventry JF, Manning RH, Newman HN, Bulman JS (1997) Comparison of two desensitizing agents for the treatment of cervical dentine sensitivity. Endod Dent Traumatol 13(1):36–39

Gillam DG, Bulman JS, Eijkman MA, Newman HN (2002) Dentists' perceptions of dentine hypersensitivity and knowledge of its treatment. J Oral Rehabil 29:219–225

Gillam D, Chesters R, Attrill D, Brunton P, Slater M, Strand P, Whelton H, Bartlett D (2013) Dentine hypersensitivity–guidelines for the management of a common oral health problem. Dent Update 40(7):514–516, 518–520, 523–524

Giusto TJ (2005) Bioactive glass and bioabsorbable membrane in the treatment of a maxillary class II furcation defect: case report with 6-month re-entry. Compend Contin Educ Dent 26(1):41–42, 44, 46 passim; quiz 52-3

Glockner K (2013) What are the unmet needs in the dental office/at home to treat dentin hypersensitivity? Clin Oral Investig 17(1):S61–S62

Greene PR (1998) The flexible gingival mask: an aesthetic solution in periodontal practice. Br Dent J 184:536–540

Grossman LI (1935) A systematic method for the treatment of hypersensitive dentine. J Am Dent Assoc 22:592–602

Gupta M, Pandit IK, Srivastava N, Gugnani N (2010) Comparative evaluation of 2% sodium fluoride iontophoresis and other cavity liners beneath silver amalgam restorations. J Indian Soc Pedod Prev Dent 28(2):68–72

Hamlin D, Williams KP, Delgado E, Zhang YP, DeVizio W, Mateo LR (2009) Clinical evaluation of the efficacy of a desensitizing paste containing 8% arginine and calcium carbonate for the in-office relief of dentin hypersensitivity associated with dental prophylaxis. Am J Dent 22(Spec No A):16A–20A

Hanning M, Hanning C (2010) Nanomaterials in preventive dentistry. Nat Nanotechnol 5:565–569

He S, Wang Y, Li X, Hu D (2011) Effectiveness of laser therapy and topical desensitising agents in treating dentine hypersensitivity: a systematic review. J Oral Rehabil 38(5):348–358

Hench LL (2006) The story of bioglass. J Mater Sci Mater Med 17(11):967–978

Hoang-Dao BT, Hoang-Tu H, Tran-Hung L, Camps J, Koubi G, About I (2008) Evaluation of a natural resin-based new material (Shellac F) as a potential desensitizing agent. Dent Mater 24(7):1001–1007

Hodosh M (1974) A superior desensitizer–potassium nitrate. J Am Dent Assoc 88(4):831–832

Hu D, Stewart B, Mello S, Arvanitidou L, Panagakos F, De Vizio W, Zhang YP, Mateo LR, Yin W (2013) Efficacy of a mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride compared to a negative control mouthwash on dentin hypersensitivity reduction. A randomized clinical trial. J Dent 41:S26–S33

Hughes N, Mason S, Jeffery P, Welton H, Tobin M, O'Shea C, Browne M (2010) A comparative clinical study investigating the efficacy of a test dentifrice containing 8% strontium acetate and 1,040 ppm sodium

fluoride versus a marketed control dentifrice containing 8% arginine, calcium carbonate, and 1,450 ppm sodium monofluorophosphate in reducing dentinal hypersensitivity. J Clin Dent 21(2):49–55

Hwang D, Wang HL (2006) Flap thickness as a predictor of root coverage: a systematic review. J Periodontol 77(10):1625–1634

Ianzano JA, Gwinnett AJ, Westbay G (1993) Polymeric sealing of dentinal tubules to control sensitivity: preliminary observations. Periodontal Clin Investig 15(1):13–16

Inoue S, Koshiro K, Yoshida Y, De Munck J, Nagakane K, Suzuki K, Sano H, Van Meerbeek B (2005) Hydrolytic stability of self-etch adhesives bonded to dentin. J Dent Res 84(12):1160–1164

Ipci SD, Cakar G, Kuru B, Yilmaz S (2009) Clinical evaluation of lasers and sodium fluoride gel in the treatment of dentine hypersensitivity. Photomed Laser Surg 27(1):85–91

Jackson RJ (2000) Potential treatment modalities for dentine hypersensitivity: home use products. In: Addy M, Edgar WM, Orchardson R (eds) Tooth wear and sensitivity. Martin Dunitz, London, pp 328–338

Kakaboura A, Rahiotis C, Thomaidis S, Doukoudakis S (2005) Clinical effectiveness of two agents on the treatment of tooth cervical hypersensitivity. Am J Dent 18(4):291–295

Kanouse MC, Ash MM Jr (1969) The effectiveness of a sodium monofluorophosphate dentifrice on dental hypersensitivity. J Periodontol 40(1):38–40

Kapferer I, Pflug C, Kisielewsky I, Giesinger J, Beier US, Dumfahrt H (2013) Instant dentin hypersensitivity relief of a single topical application of an in-office desensitizing paste containing 8% arginine and calcium carbonate: a split-mouth, randomized-controlled study. Acta Odontol Scand 71(3 – 4):994–999

Kara C, Orbak R (2009) Comparative evaluation of Nd:YAG laser and fluoride varnish for the treatment of dentinal hypersensitivity. J Endod 35(7):971–974

Karim BF, Gillam DG (2013) The efficacy of strontium and potassium toothpastes in treating dentine hypersensitivity: a systematic review. Int J Dent 2013:573258

Karlinsey RL, Mackey AC, Stookey GK (2009a) In vitro remineralization efficacy of NaF systems containing unique forms of calcium. Am J Dent 22(3):185–188

Karlinsey RL, Mackey AC, Stookey GK et al (2009b) In vitro assessments of experimental NaF dentifrices containing a prospective calcium phosphate technology. Am J Dent 22(3):180–184

Karlinsey RL, Mackey AC, Walker ER et al (2010) Surfactant-modified beta-TCP: structure, properties, and in vitro remineralization of subsurface enamel lesions. J Mater Sci Mater Med 21(7):2009–2020

Karlinsey RL, Mackey AC, Schwandt CS (2012) Effects on dentin treated with eluted multi-mineral varnish in vitro. Open Dent J 2012(6):157–163

Kassab MM, Cohen RE (2003) The etiology and prevalence of gingival recession. J Am Dent Assoc 134:220–225

Kates JJ, Greskovich MS, Smith WK, Ariel DR, Diamond MW (1998) Long-term evaluation of the use of PerioGlas in alveolar ridge augmentation following

tooth extraction: a prospective human study. Dental Cadmos

Kato MT, Magalhães AC, Rios D, Hannas AR, Attin T, Buzalaf MA (2009) Protective effect of green tea on dentin erosion and abrasion. J Appl Oral Sci 17:560–564

Kato MT, Leite AL, Hannas AR, Oliveira RC, Pereira JC, Tjäderhane L et al (2010) Effect of iron on matrix metalloproteinase inhibition and on the prevention of dentine erosion. Caries Res 44:309–316

Kato MT, Leite AL, Hannas AR, Calabria MP, Magalhães AC, Pereira JC, Buzalaf MAR (2012) Impact of protease inhibitors on dentin matrix degradation by collagenase. J Dent Res 91:1119–1123

Kerns DG, Scheidt MJ, Pashley DH, Horner JA, Strong SL, Van Dyke TE (1991) Dentinal tubule occlusion and root hypersensitivity. J Periodontol 62(7):421–428

Kimura Y, Wilder-Smith P, Yonaga K, Matsumoto K (2000) Treatment of dentine hypersensitivity by lasers: a review. J Clin Periodontol 27(10):715–721

Kleinberg I (2002). SensiStat. A new saliva-based composition for simple and effective treatment of dentinal sensitivity pain. Dent Today 21(12):42–47

Kleter GA, Damen JJ, Everts V, Niehof J, ten Cate JM (1993) The influence of the organic matrix on demineralization of bovine root dentin in vitro. J Dent Res 73:1523–1529

Knight NN, Lie T, Clark SM, Adams DF (1993) Hypersensitive dentin: testing of procedures for mechanical and chemical obliteration of dentinal tubuli. J Periodontol 64(5):366–373

Kobler A, Kub O, Schaller HG, Gernhardt CR (2008) Clinical effectiveness of a strontium chloride- containing desensitizing agent over 6 months: a randomized, double-blind, placebo-controlled study. Quintessence Int 39(4):321–325

Kumar VL, Itthagarun A, King NM (2008) The effect of casein phosphopeptide-amorphous calcium phosphate on remineralization of artificial caries-like lesions: an in vitro study. Aust Dent J 53(1):34–40

Lan WH, Liu HC (1996) Treatment of dentin hypersensitivity by Nd:YAG laser. J Clin Laser Med Surg 14(2):89–92

Lata S, Varghese NO, Varughese JM (2010) Remineralization potential of fluoride and amorphous calcium phosphate-casein phospho peptide on enamel lesions: An in vitro comparative evaluation. J Conserv Dent 13:42–46

Levin MP, Yearwood LL, Carpenter WN (1973) The desensitizing effect of calcium hydroxide and magnesium hydroxide on hypersensitive dentin. Oral Surg Oral Med Oral Pathol 35(5):741–746

Lier BB, Rösing CK, Aass AM, Gjermo P (2002) Treatment of dentin hypersensitivity by Nd:YAG laser. J Clin Periodontol 29(6):501–506

Lin YH, Gillam DG (2012) The prevalence of root sensitivity following periodontal therapy: a systematic review. Int J Dent 2012:407023. doi:10.1155/2012/407023, Epub 2012 Oct 31

Lin PY, Cheng YW, Chu CY, Chien KL, Lin CP, Tu YK (2013) In-office treatment for dentin hypersensitivity: a systematic review and network meta-analysis. J Clin Periodontol 40(1):53–64

Ling TY, Gillam DG (1996) The effectiveness of desensitizing agents for the treatment of cervical dentine sensitivity (CDS)–a review. J West Soc Periodontol Periodontal Abstr 44(1):5–12

Ling TY, Gillam DG, Barber PM, Mordan NJ, Critchell J (1997) An investigation of potential desensitizing agents in the dentine disc model: a scanning electron microscopy study. J Oral Rehabil 24(3):191–203

Litkowski L, Greenspan DC (2010) A clinical study of the effect of calcium sodium phosphosilicate on dentin hypersensitivity — proof of principle. J Clin Dent 21(3):77–81

Liu H, Hu D (2012) Efficacy of a commercial dentifrice containing 2% strontium chloride and 5% potassium nitrate for dentin hypersensitivity: a 3-day clinical study in adults in China. Clin Ther 34(3):614–622

Liu HC, Lin CP, Lan WH (1997) Sealing depth of Nd:YAG laser on human dentinal tubules. J Endod 23(11):691–693

Lopes AO, Aranha AC (2013) Comparative evaluation of the effects of Nd:YAG laser and a desensitizer agent on the treatment of dentin hypersensitivity: a clinical study. Photomed Laser Surg 31(3):132–138

Lopes AO, de Paula Eduardo C, Aranha AC (2013) Clinical evaluation of low-power laser and a desensitizing agent on dentin hypersensitivity. Lasers Med Sci [Epub ahead of print]

Loty C, Sautier JM, Tan MT, Oboeuf M, Jallot E, Boulekbache H, Greenspan D, Forest N (2001) Bioactive glass stimulates in vitro osteoblast differentiation and creates a favorable template for bone tissue formation. J Bone Miner Res 16(2):231–239

Lussi A, Hellwig E (2013) Diagnosis and management of exposed cervical dentin. Foreword. Clin Oral Investig 17(1):S1–S2

Lynch E, Brauer DS, Karpukhina N, Gillam DG, Hill RG (2012) Multi-component bioactive glasses of varying fluoride content for treating dentin hypersensitivity. Dent Mater 28(2):168–178

Magalhães AC, Wiegand A, Rios D, Hannas A, Attin T, Buzalaf MA (2009) Chlorhexidine and green tea extract reduce dentin erosion and abrasion in situ. J Dent 37:994–998

Mantzourani M, Sharma D (2013) Dentine sensitivity: past, present and future. J Dent 4I(4):S3–S17

Marinho VC, Higgins JP, Sheiham A, Logan S (2004) Combinations of topical fluoride (toothpastes, mouthrinses, gels, varnishes) versus single topical fluoride for preventing dental caries in children and adolescents. Cochrane Database Syst Rev (1):CD002781

Markowitz K (2009) The original desensitizers: strontium and potassium salts. J Clin Dent 20(5):145–151

Markowitz K (2013) A new treatment alternative for sensitive teeth: a desensitizing oral rinse. J Dent 4I:S1–S11

Markowitz K, Kim S (1992) The role of selected cations in the desensitization of intradental nerves. Proc Finn Dent Soc 88(1):39–54, Review

Markowitz K, Pashley DH (2008) Discovering new treatments for sensitive teeth: the long path from biology to therapy. J Oral Rehabil 35(4):300–315

Markowitz K, Bilotto G, Kim S (1991) Decreasing intradental nerve activity in the cat with potassium and divalent cations. Arch Oral Biol 36(1):1–7

Martens LC (2013) A decision tree for the management of exposed cervical dentin (ECD) and dentin hypersensitivity (DHS). Clin Oral Investig 17(1):S77–S83

Mason S, Hughes N, Sufi F, Bannon L, Maggio B, North M, Holt J (2010) A comparative clinical study investigating the efficacy of a dentifrice containing 8% strontium acetate and 1040 ppm fluoride in a silica base and a control dentifrice containing 1450 ppm fluoride in a silica base to provide immediate relief of dentin hypersensitivity. J Clin Dent 21(2):42–48

Mehta D, Gowda VS, Santosh A, Finger WJ, Sasaki K (2014) Randomized controlled clinical trial on the efficacy of dentin desensitizing agents. Acta Odontol Scand 72(8):936–941

Mello SV et al (2013a) Mode of action studies of a new desensitizing mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride. J Dent 4IS:S12–S19

Mello SV, Arvanitidou E, Vandeven M (2013b) The development of a new desensitising mouthwash containing arginine, PVM/MA copolymer, pyrophosphates, and sodium fluoride—A hydraulic conductance study. J Dent 4IS:S20–S25

Mensinkai PK, Ccahuana-Vasquez RA, Chedjieu I, Amaechi BT, Mackey AC, Walker TJ, Blanken DD, Karlinsey RL (2012) In situ remineralization of white-spot enamel lesions by 500 and 1,100 ppm F dentifrices. Clin Oral Investig 16(4):1007–1014

Merika K, HeftitArthur F, Preshaw PM (2006) Comparison of two topical treatments for dentine sensitivity. Eur J Prosthodont Restor Dent 14(1):38–41

Miglani S, Aggarwal V, Ahuja B (2010) Dentin hypersensitivity: recent trends in management. J Conserv Dent 13(4):218–224

Miller PD Jr (1985) A classification of marginal tissue recession. Int J Periodontics Restorative Dent 5(2):8–13

Moritz A, Schoop U, Goharkhay K, Aoid M, Reichenbach P, Lothaller MA, Wernisch J, Sperr W (1998) Long-term effects of CO2 laser irradiation on treatment of hypersensitive dental necks: results of an in vivo study. J Clin Laser Med Surg 16(4):211–215

Morris MF, Davis RD, Richardson BW (1999) Clinical efficacy of two dentin desensitizing agents. Am J Dent 12(2):72–76

Müller HP, Heinecke A, Schaller N, Eger T (2000) Masticatory mucosa in subjects with different periodontal phenotypes. J Clin Periodontol 27(9):621–626

Nagata T, Ishida H, Shinohara H, Nishikawa S, Kasahara S, Wakano Y, Daigen S, Troullos ES (1994) Clinical evaluation of a potassium nitrate dentifrice for the treatment of dentinal hypersensitivity. J Clin Periodontol 21(3):217–221

Narongdej T, Sakoolnamarka R, Boonroung T (2010) The effectiveness of a calcium sodium phosphosilicate desensitizer in reducing cervical dentin hypersensitivity: a pilot study. J Am Dent Assoc 141(8):995–999

Nongonierma AB, Fitzgerald RJ (2012) Biofunctional properties of caseinophosphopeptides in the oral cavity. Caries Res 46(3):234–267

Nordström A, Birkhed D (2010) Preventive effect of high-fluoride dentifrice (5,000 ppm) in caries-active adolescents: a 2-year clinical trial. Caries Res 44(3):323–331

Norton MR, Wilson J (2001) Bone augmentation with bioactive ceramics before insertion of endosseous dental implants: histories and human histology on seventeen consecutive cases. Key Eng Mater 192 – 195:869–872

Olley RC, Pilecki P, Hughes N, Jeffery P, Austin RS, Moazzez R, Bartlett D (2012) An in situ study investigating dentine tubule occlusion of dentifrices following acid challenge. J Dent 40(7):585–593

Orchardson R, Collins WJ (1987) Clinical features of hypersensitive teeth. Br Dent J 162(7):253–256

Orchardson R, Gillam DG (2000) The efficacy of potassium salts as agents for treating dentin hypersensitivity. J Orofac Pain 14(1):9–19

Orchardson R, Gillam DG (2006) Managing dentin hypersensitivity. J Am Dent Assoc 137(7):990–998; quiz 1028–1029

Orchardson R, Gangarosa LPS (1994) Hypersensitive dentine; biological basis of therapy. Arch Oral Biol 39(Suppl):1S–153S

Ozen T, Orhan K, Avsever H, Tunca YM, Ulker AE, Akyol M (2009) Dentin hypersensitivity: a randomized clinical comparison of three different agents in a short-term treatment period. Oper Dent 34(4):392–398

Özgünaltay G, Önen A (2002) Three-year clinical evaluation of a resin modified glass–ionomer cement and a composite resin in non-carious class V lesions. J Oral Rehabil 29:1037–1041

Panagakos F, Schiff T, Guignon A (2009) Dentin hypersensitivity: effective treatment with an in-office desensitizing paste containing 8% arginine and calcium carbonate. Am J Dent 22(Special Issue):3A–7A

Park JS, Suh JJ, Choi SH, Moon IS, Cho KS, Kim CK, Chai JK (2001) Effects of pretreatment clinical parameters on bioactive glass implantation in intrabony periodontal defects. J Periodontol 72(6):730–740

Parkinson CR, Willson RJ (2011) An in vitro investigation of two currently marketed dentin tubule occlusion dentifrices. J Clin Dent 22(1):6–10

Pashley DH (2000) Potential treatment modalities for dentine hyper sensitivity: in-office products. In: Addy M, Embery G, Edgar WM, Orchardson R (eds) Tooth wear and sensitivity. Martin Dunitz, London, pp 351–361

Pashley DH, O'Meara JA, Kepler EE, Galloway SE, Thompson SM, Stewart FP (1984) Dentin permeability. Effects of desensitizing dentifrices in vitro. J Periodontol 55(9):522–525

Pashley DH, Leibach JG, Horner JA (1987) The effects of burnishing NaF/kaolin/glycerin paste on dentin permeability. J Periodontol 58(1):19–23

Pashley DH, Carvalho RM, Pereira JC, Villanueva R, Tay FR (2001) The use of oxalate to reduce dentin permeability under adhesive restorations. Am J Dent 14(2):89–94

Patel N, Langalia A (2014) Iontophoresis in conservative dentistry and endodontics: a review. J Res Adv Dent 3(1):140–143

Pei D, Liu S, Huang C, Du X, Yang H, Wang Y, Deng D (2013) Effect of pretreatment with calcium-containing desensitizer on the dentine bonding of mild self-etch adhesives. Eur J Oral Sci 121(3 Pt 1):204–210

Pereira R, Chavas VK (2001) Efficacy of a 3 % potassium nitrate desensitizing mouthwash in the treatment of dentinal. Clin Oral Investig 17(1):S63–S71, S69 hypersensitivity. J Periodontol, 2001;72(12):1720–25

Pereira JC, Segala AD, Gillam DG (2005) 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):129–138

Petersson LG (2013) The role of fluoride in the preventive management of dentin hypersensitivity and root caries. Clin Oral Investig 17(1):S63–S71

Petersson LG, Kambara M (2004) Remineralisation study of artificial root caries lesions after fluoride treatment. An in vitro study using electric caries monitor and transversal micro-radiography. Gerodontology 21(2):85–92

Petrou I, Heu R, Stranick M, Lavender S, Zaidel L, Cummins D, Sullivan RJ, Hsueh C, Gimzewski JK (2009) A breakthrough therapy for dentin hypersensitivity: how dental products containing 8% arginine and calcium carbonate work to deliver effective relief of sensitive teeth. J Clin Dent 20:23–31

Plagmann HC, König J, Bernimoulin JP, Rudhart AC, Deschner J (1997) A clinical study comparing two high-fluoride dentifrices for the treatment of dentinal hypersensitivity. Quintessence Int 28(6):403–408

Polack MA, Mahn DH (2013) Biotype change for the esthetic rehabilitation of the smile. J Esthet Restor Dent 25(3):177–186. doi:10.1111/jerd.12029, Epub 2013 May 3

Polderman RN, Frencken JE (2007) Comparison between effectiveness of a low-viscosity glass ionomer and a resin-based glutaraldehyde containing primer in treating dentine hypersensitivity–a 25.2-month evaluation. J Dent 35(2):144–149

Poulsen S, Errboe M, Hovgaard O, Worthington HW (2001) Potassium nitrate toothpaste for dentine hypersensitivity. Cochrane Database Syst Rev (2):CD001476

Poulsen S, Errboe M, Lescay Mevil Y, Glenny AM (2006) Potassium containing toothpastes for dentine hypersensitivity. Cochrane Database Syst Rev (3):CD001476

Pradeep AR, Sharma A (2010) Comparison of clinical efficacy of a dentifrice containing calcium sodium phosphosilicate to a dentifrice containing potassium nitrate and to a placebo on dentinal hypersensitivity: a randomized clinical trial. J Periodontol 81(8):1167–1173

Pradeep AR, Agarwal E, Naik SB, Bajaj P, Kalra N (2012) Comparison of efficacy of three commercially available dentifrices [corrected] on dentinal hypersensitivity: a randomized clinical trial. Aust Dent J 57(4): 429–434

Price N, Bendall SP, Frondoza C, Jinnah RH, Hungerford DS (1997) Human osteoblast-like cells (MG63) proliferate on a bioactive glass surface. J Biomed Mater Res 37(3):394–400

Qin C, Xu J, Zhang Y (2006) Spectroscopic investigation of the function of aqueous 2-hydroxyethylmethacrylate/glutaraldehyde solution as a dentin desensitizer. Eur J Oral Sci 114(4):354–359

Que K, Fu Y, Lin L, Hu D, Zhang YP, Panagakos FS, DeVizio W, Mateo LR (2010) Dentin hypersensitivity reduction of a new toothpaste containing 8.0% arginine and 1450 ppm fluoride: an 8-week clinical study on Chinese adults. Am J Dent 23(Spec No A): 28A–35A

Rajesh KS, Hedge S, Arun Kumar MS, Shetty DG (2012) Evaluation of the efficacy of a 5% calcium sodium phosphosilicate (Novamin) containing dentifrice for the relief of dentinal hypersensitivity: a clinical study. Indian J Dent Res 23(3):363–367

Reynolds EC (1997) Remineralization of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions. J Dent Res 76:1587–1595

Reynolds EC (1998) Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. Spec Care Dentist 18(1):8–16

Reynolds EC, Cai F, Cochrane NJ, Shen P, Walker GD, Morgan MV, Reynolds C (2008) Fluoride and casein phosphopeptide-amorphous calcium phosphate. J Dent Res 87(4):344–348

Ritter AV, de L Dias W, Miguez P, Caplan DJ, Swift EJ Jr (2006) Treating cervical dentin hypersensitivity with fluoride varnish: a randomized clinical study. J Am Dent Assoc 137(7):1013–1020; quiz 1029

Rösing CK, Fiorini T, Liberman DN, Cavagni J (2009) Dentine hypersensitivity: analysis of self-care products. Braz Oral Res 23(1):56–63

Rusin RP, Agee K, Suchko M, Pashley DH (2010) Effect of a new desensitizing material on human dentin permeability. Dent Mater 26(6):600–607

Santamaria MP, Suaid FF, Nociti FH Jr, Zaffalon M et al (2007) Periodontal surgery and glass ionomer restoration in the treatment of gingival recession associated with a non-carious cervical lesion: report of three cases. J Periodontol 78(6):1146–1153

Santos MJ, Ari N, Steele S, Costella J, Banting D (2014) Retention of tooth-colored restorations in non-carious cervical lesions-a systematic review. Clin Oral Investig 18(5):1369–1381

Sauro S, Gandolfi MG, Prati C, Mongiorgi R (2006) Oxalate-containing phytocomplexes as dentine desensitisers: an in vitro study. Arch Oral Biol 51(8):655–664

Sauro S, Mannocci F, Watson TF, Piemontese M, Sherriff M, Mongiorgi R (2007) The influence of soft acidic drinks in exposing dentinal tubules after non-surgical periodontal treatment: a SEM investigation on the protective effects of oxalate-containing phytocomplex. Med Oral Patol Oral Cir Bucal 12(7):E542–E548

Scaffa PM, Vidal CM, Barros N, Gesteira TF, Carmona AK, Breschi L, Pashley DH, Tjäderhane L, Tersariol IL, Nascimento FD, Carrilho MR (2012) Chlorhexidine inhibits the activity of dental cysteine cathepsins. J Dent Res 91(4):420–425

Schemehorn BR, Wood GD, McHale W, Winston AE (2011) Comparison of fluoride uptake into tooth enamel from two fluoride varnishes containing different calcium phosphate sources. J Clin Dent 22(2):51–54

Schiff T, Dotson M, Cohen S, De Vizio W, McCool J, Volpe A (1994) Efficacy of a dentifrice containing potassium nitrate, soluble pyrophosphate, PVM/MA copolymer, and sodium fluoride on dentinal hypersensitivity: a twelve-week clinical study. J Clin Dent 5(Spec No):87–92

Schiff T, Dos Santos M, Laffi S, Yoshioka M, Baines E, Brasil KD, McCool JJ, De Vizio W (1998) Efficacy of a dentifrice containing 5% potassium nitrate and 1,500 PPM sodium monofluorophosphate in a precipitated calcium carbonate base on dentinal hypersensitivity. J Clin Dent 9(1):22–25

Schiff T, Bonta Y, Proskin HM, DeVizio W, Petrone M, Volpe AR (2000) Desensitizing efficacy of a new dentifrice containing 5.0 % potassium nitrate and 0.454 % stannous fluoride. Am J Dent 13(3):111–115

Schiff T, Delgado E, Zhang YP, Cummins D, DeVizio W, Mateo LR (2009) Clinical evaluation of the efficacy of an in-office desensitizing paste containing 8% arginine and calcium carbonate in providing instant and lasting relief of dentin hypersensitivity. Am J Dent 22(Spec No A):8A–15A

Schmidlin PR, Sahrmann P (2013) Current management of dentin hypersensitivity. Clin Oral Investig 17(1): S55–S59

Schüpbach P, Lutz F, Finger WJ (1997) Closing of dentinal tubules by Gluma desensitizer. Eur J Oral Sci 105(5 Pt 1):414–421

Schweikl H, Schmalz G (1997) Glutaraldehyde-containing dentin bonding agents are mutagens in mammalian cells in vitro. J Biomed Mater Res 36(3):284–288

Sculean A, Barbe G, Chiantella GC, Arweiler NB, Berakdar M, Brecx M (2002) Clinical evaluation of an enamel matrix protein derivative combined with a bioactive glass for the treatment of intrabony periodontal defects in humans. J Periodontol 73(4):401–408

Seibert JS, Lindhe J (1989) Esthetics and periodontal therapy. In: Lindhe J (ed) Textbook of clinical periodontology. Munksgaard, Copenhagen

Sensistat KI (2002) A new saliva-based composition for simple and effective treatment of dentinal sensitivity pain. Dent Today 21:42–47

Seong J, Macdonald E, Newcombe RG, Davies M, Jones SB, Johnson S, West NX (2013) In situ randomised trial to investigate the occluding properties of two desensitising toothpastes on dentine after subsequent acid challenge. Clin Oral Investig 17(1):195–203

Sethna GD, Prabhuji ML, Karthikeyan BV (2011) Comparison of two different forms of varnishes in the treatment of dentine hypersensitivity: a subject-blind randomised clinical study. Oral Health Prev Dent 9(2):143–150

Sgolastra F, Petrucci A, Gatto R, Monaco A (2011) Effectiveness of laser in dentinal hypersensitivity

treatment: a systematic review. J Endod 37(3): 297–303

Sharif MO, Iram S, Brunton PA (2013) Effectiveness of arginine-containing toothpastes in treating dentine hypersensitivity: a systematic review. J Dent 41(6): 483–492

Sharma AA, Park JH (2010) Esthetic considerations in interdental papilla: remediation and regeneration. J Esthet Restor Dent 22:18–30

Sharma D, McGuire JA, Amini P (2013a) Randomized trial of the clinical efficacy of a potassium oxalate-containing mouthrinse in rapid relief of dentin sensitivity. J Clin Dent 24(2):62–67

Sharma D, McGuire JA, Gallob JT, Amini P (2013b) Randomised clinical efficacy trial of potassium oxalate mouthrinse in relieving dentinal sensitivity. J Dent 41(4):S40–S48

Sharma D, Hong CX, Heipp PS (2013c) A novel potassium oxalate-containing tooth-desensitising mouthrinse: a comparative in vitro study. J Dent 41(Suppl 4):S18–S27

Silverman G (1985) The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. Compend Contin Educ Dent 6(2):131–133, 136

Silverman G, Berman E, Hanna CB, Salvato A, Fratarcangelo P, Bartizek RD, Bollmer BW, Campbell SL, Lanzalaco AC, Mackay BJ, McClanahan SF, Perlich MA, Shaffer JB (1996) Assessing the efficacy of three dentifrices in the treatment of dentinal hypersensitivity. J Am Dent Assoc 127(2):191–201

Singal P, Gupta R, Pandit N (2005) 2% sodium fluoride-iontophoresis compared to a commercially available desensitizing agent. J Periodontol 76(3):351–357

Sollazzo V, Palmieri A, Scapoli L, Martinelli M, Girardi A, Pezzetti F, Morselli PF (2010) PerioGlas® acts on human stem cells isolated from peripheral blood. Dent Res J Isfahan 7(1):28–34

Srinivasan M, Schimmel M, Riesen M, Ilgner A, Wicht MJ, Warncke M, Ellwood RP, Nitschke I, Müller F, Noack MJ (2014) High-fluoride toothpaste: a multicenter randomized controlled trial in adults. Community Dent Oral Epidemiol 42(4):333–340

Suge T, Ishikawa K, Kawasaki A, Suzuki K, Matsuo T, Noiri Y, Imazato S (2002) Calcium phosphate precipitation method for the treatment of dentin hypersensitivity. Am J Dent 15:220–226

Suge T, Kawasaki A, Ishikawa K, Matsuo T, Ebisu S (2005) Comparison of the occluding ability of dentinal tubules with different morphology between calcium phosphate precipitation method and potassium oxalate treatment. Dent Mater J 24(4):522–529

Swift EJ Jr, May KN Jr, Mitchell S (2001) Clinical evaluation of Prime & Bond 2.1 for treating cervical dentin hypersensitivity. Am J Dent 14(1):13–16

Tai BJ, Bian Z, Jiang H, Greenspan DC, Zhong J, Clark AE, Du MQ (2006) Anti-gingivitis effect of a dentifrice containing bioactive glass (NovaMin®) particulate. J Clin Periodontol 33(2):86–91

Talioti E, Hill R, Gillam DG. The Efficacy of Selected Desensitizing OTC Products: a systematic review. Hindawi Publishing Corporation ISRN Dentistry 2014, Article ID 865761, 14 pages. http://dx.doi.org/10.1155/2014/865761

Tantbirojn D, Feigal RJ, Ko CC, Versluis A (2006) Remineralized dentin lesions induced by glass ionomerdemonstrate increased resistance to subsequent acid challenge. Quintessence Int 37(4):273–281

Tarbet WJ, Silverman G, Stolman JM, Fratarcangelo PA (1980) Clinical evaluation of a new treatment for dentinal hypersensitivity. J Periodontol 51(9):535–540

Tarbet WJ, Silverman G, Fratarcangelo PA, Kanapka JA (1982) Home treatment for dentinal hypersensitivity: a comparative study. J Am Dent Assoc 105(2):227–230

Tay FR, Gwinnett AJ, Pang KM, Wei SH (1994) Structural evidence of a sealed tissue interface with a total-etch wet-bonding technique in vivo. J Dent Res 73(3):629–636

Tengrungsun T, Sangkla W (2008) Comparative study in desensitizing efficacy using the GaAlAs laser and dentin bonding agent. J Dent 36(6):392–395

Thrash WJ, Dodds MW, Jones DL (1994) The effect of stannous fluoride on dentinal hypersensitivity. Int Dent J 44(1):107–118

Tjäderhane L, Larjava H, Sorsa T, Uitto V-J, Larmas M, Salo T (1998) The activation and function of host matrix metalloproteinases in dentin matrix breakdown in caries lesions. J Dent Res 77:1622–1629

Trushkowsky RD, Oquendo A (2011) Treatment of dentin hypersensitivity. Dent Clin North Am 55(3):599–608

Tschoppe P, Zandim DL, Martus P, Kielbassa AM (2011) Enamel and dentine remineralization by nanohydroxyapatite toothpastes. J Dent 39:430–437

Tugnait A, Clerehugh V (2001) Gingival recession-its significance and management. J Dent 29:381–394

van Dijken JW, Pallesen U (2008) Long-term dentin retention of etch-and-rinse and self-etch adhesives and a resin-modified glass ionomer cement in non-carious cervical lesions. Dent Mater 24(7):915–922

van Loveren C (2013) Exposed cervical dentin and dentin hypersensitivity summary of the discussion and recommendations. Clin Oral Investig 17(1):S73–S76

Vandiver J, Dean D, Patel N, Bonfield W, Ortiz C (2005) Nanoscale variation in surface charge of synthetic hydroxyapatite detected by chemically and spatially specific high-resolution force spectroscopy. Biomaterials 26:271–283

Vanichvatana S, Auychai P (2013) Efficacy of two calcium phosphate pastes on the remineralization of artificial caries: a randomized controlled double-blind in situ study. Int J Oral Sci 5(4):224–228

Vollenweider M, Brunner TJ, Knecht S, Grass RN, Zehnder M, Imfeld T, Stark WJ (2007) Remineralization of human dentin using ultrafine bioactive glass particles. Acta Biomater 3:936–943

Vora J, Mehta D, Meena N, Sushma G, Finger WJ, Kanehira M (2012) Effects of two topical desensitizing agents and placebo on dentin hypersensitivity. Am J Dent 25(5):293–298

Walsh LJ (2010) The effects of GC tooth mousse on cervical dentinal sensitivity: a controlled clinical trial. Int Dent 12(1):4–12

Walsh T, Worthington HV, Glenny AM, Appelbe P, Marinho VC, Shi X (2010) Fluoride toothpastes of different concentrations for preventing dental caries in children and adolescents. Cochrane Database Syst Rev 1:CD007868

Wang Z, Sa Y, Sauro S, Chen H, Xing W, Ma X, Jiang T, Wang Y (2010) Effect of desensitising toothpastes on dentinal tubule occlusion: a dentine permeability measurement and SEM in vitro study. J Dent 38(5): 400–410

Wang Z, Jiang T, Sauro S, Pashley DH, Toledano M, Osorio R, Liang S, Xing W, Sa Y, Wang Y (2011a) The dentine remineralization activity of a desensitizing bioactive glass-containing toothpaste: an in vitro study. Aust Dent J 56(4):372–381

Wang Z, Jiang T, Sauro S, Wang Y, Thompson I, Watson TF, Sa Y, Xing W, Shen Y, Haapasalo M (2011b) Dentine remineralization induced by two bioactive glasses developed for air abrasion purposes. J Dent 39(11):746–756

Wegehaupt FJ, Tauböck TT, Stillhard A, Schmidlin PR, Attin T (2011) Influence of extra- and intra-oral application of CPP-ACP and fluoride on re-hardening of eroded enamel. Acta Odontol Scand 25:1–7

West NX (2008) Dentine hypersensitivity: preventive and therapeutic approaches to treatment. Periodontol 2000 48:31–41

West NX, Addy M, Jackson RJ, Ridge DB (1997) Dentine hypersensitivity and the placebo response. A comparison of the effect of strontium acetate, potassium nitrate and fluoride toothpastes. J Clin Periodontol 24(4): 209–215

West NX, Lussi A, Seong J, Hellwig E (2013) Dentin hypersensitivity: pain mechanisms and aetiology of exposed cervical dentin. Clin Oral Investig 17(1):S9–S19

Xynos ID, Hukkanen MV, Batten JJ, Buttery LD, Hench LL, Polak JM (2000) Bioglass 45S5 stimulates osteoblast turnover and enhances bone formation in vitro: implications and applications for bone tissue engineering. Calcif Tissue Int 67(4):321–329

Yates R, Owens J, Jackson R, Newcombe RG, Addy M (1998) A split-mouth placebo-controlled study to determine the effect of amorphous calcium phosphate in the treatment of dentine hypersensitivity. J Clin Periodontol 25(8):687–692

Yates RJ, Newcombe RG, Addy M (2004) Dentine hypersensitivity: a randomised, double-blind placebo-controlled study of the efficacy of a fluoride-sensitive teeth mouthrinse. J Clin Periodontol 31(10):885–889

Yilmaz HG, Kurtulmus-Yilmaz S, Cengiz E (2011) Long-term effect of diode laser irradiation compared to sodium fluoride varnish in the treatment of dentine hypersensitivity in periodontal maintenance patients: a randomized controlled clinical study. Photomed Laser Surg 29(11):721–725

Yu X, Liang B, Jin X, Fu B, Hannig M (2010) Comparative in vivo study on the desensitizing efficacy of dentin desensitizers and one-bottle self-etching adhesives. Oper Dent 35(3):279–286

Yukna RA, Evans GH, Aichelmann-Reidy MB, Mayer ET (2001) Clinical comparison of bioactive glass bone replacement graft material and expanded polytetrafluoroethylene barrier membrane in treating human mandibular molar class II furcations. J Periodontol 72(2):125–133

Zalkind M, Hochman N (1997) Alternative method of conservative aesthetic treatment for gingival recession. J Prosthet Dent 77:561–563

Zamet JS, Darbar UR, Griffiths GS, Bulman JS, Bragger U, Burgin W, Newman HN (1997) Particulate bioglass as a grafting material in the treatment of periodontal intrabony defects. J Clin Periodontol 24(6):410–418

Zweers J, Thomas RZ, Slot DE, Weisgold AS, Van der Weijden GA (2014) Characteristics of periodontal biotype, its dimensions, associations and prevalence-a systematic review. J Clin Periodontol. doi:10.1111/jcpe.12275