Prevention and treatment of demineralisation during fixed appliance therapy: a review of current methods and future applications

IN BRIEF

- Highlights the incidence of white spot lesions (WSL) in orthodontic patients.
- Stresses the importance of making patients aware of this risk during the consent process.
- Explains the methods clinicians have to help prevent and treat WSL.
- Suggests a new manufacturing process allowing material characteristics to be tailored for purpose, which may help prevent demineralisation in the future.

PRACTICE

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VERIFIABLE CPD PAPER

Orthodontic treatment, like all aspects of dentistry, exposes the clinician to the risk of malpractice and litigation. Demineralisation of tooth enamel is still one of the main complications of orthodontic treatment and it is essential patients are made aware of this risk during the consent process. There are a variety of fluoride delivery systems (mouthrinse, varnish, bonding system, and elastics), which can be used to prevent white spot lesion (WSL) formation. Glass-ionomer bonding cements (GIC) have also been shown to reduce WSL formation and have the benefit of not relying on patient compliance. However, these materials have not found widespread acceptance, possibly due to handling characteristics. A number of new technologies, principally fillers and coatings, have recently become available with potential antimicrobial and antibiofilm properties. Coatings can be applied to brackets and wires, which prevent bacterial adhesion. However, the longevity of these coatings is questionable. There are a number of methods available aimed at reducing the incidence of WSL, but they all have limitations. Capitalising on technological advances will enable the production of tailor made orthodontic brackets and adhesive systems, which provide long-term protection against WSL without relying on patient compliance.

BACKGROUND

Orthodontic treatment, like all aspects of dentistry, exposes the clinician to the risk of malpractice and litigation. Users of healthcare services are becoming ever more demanding and critical of their treatment, which has resulted in an increase in the number of malpractice claims against clinicians. Demineralisation of tooth enamel is still one of the main complications of orthodontic treatment, particularly with fixed appliances.[1] It is therefore essential that patients are made aware of this risk as part of the consent process and given the information necessary to minimise the chances of its occurrence.

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Why is orthodontic treatment associated with demineralisation? It is known that caries can develop in the presence of dental plaque, a susceptible tooth surface and a fermentable carbohydrate, such as sucrose. Fixed appliances not only make conventional oral hygiene procedures more difficult, they also increase the number of plaque retentive sites. This can lead to a rapid shift in the bacterial composition of dental plaque[2,3] and in the number of acidogenic bacteria, most notably *Streptococcus mutans* (*S. mutans*).[4] The first sign of demineralisation may be the development of white spot lesions (WSLs) on the enamel surface around the bracket margins, which if left unchecked can progress to cavitation. This whole process occurs more rapidly in orthodontic patients when compared to the development of similar lesions in non-orthodontic patients (Fig. 1).[5,6]

Preventing enamel demineralisation and the formation of WSL is an important consideration for orthodontists, as the lesions are not aesthetically pleasing and potentially irreversible. One cross- sectional

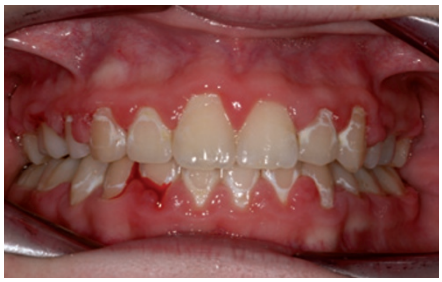

Fig. 1 Demineralisation caused by fixed orthodontic appliances resulting in early debond

study showed that up to 50% of individuals undergoing fixed appliance therapy had non-developmental WSL compared with just 25% of controls.[7] Even five years after the completion of treatment, orthodontic patients have been shown to have a significantly higher incidence of WSL compared to that of non-orthodontic patients.[8] When individual teeth within the arch are considered, it would seem the maxillary lateral incisor emerges as the tooth most at risk of developing a WSL (23%). This is closely followed by the mandibular canine (18%) and first premolar (17.5%).[9] This comes as no surprise, as these are the regions where

hooks and elastics are attached, making it more difficult for patients to keep these areas clean.

Although the progression from WSL to cavitation is low (cavitation occurs in only 2% of WSL), the high incidence of WSLs is a significant factor where the orthodontic treatment is being performed to improve aesthetics.[10]

Orthodontic treatment not only elevates the level of cariogenic bacteria, but also increases biofilm accumulation and levels of periodontal bacteria. The dental biofilm is a complex structure, which adheres to the tooth/bracket surface and is comprised of colonies of bacteria. The salivary pellicle that forms on the tooth surface acts as an adhesive, which facilitates this bacterial attachment. These early colonisers (mainly Gram +ve streptococci for example, *S. mutans*, *S. gordonii*) secrete a mucilaginous protective coating, in which they become encased. These bacteria encourage other free-floating bacteria to attach on to the surface and also produce an extracellular protective matrix (biofilm). Micro colonies then form within the biofilm and over time the bacterial composition shifts to an increase in concentration of Gram -ve anaerobic bacteria (for example, *F. nucleatum* – middle coloniser and *A. actinomycetemcomitans* – late coloniser). These bacteria not only produce acidic waste metabolites, which can cause demineralisation, but if left undisturbed can also extend into the gingival sulcus, resulting in gingival inflammation. Gram -ve bacteria flourish in this anaerobic environment and their presence can ultimately result in periodontal disease, particularly in adults and susceptible patients.[11,12]

The relatively high incidence of WSL and risk of gingival inflammation arising as a result of orthodontic treatment has driven research into methods aimed at eliminating these bacteria and preventing their attachment to the appliance and surrounding tooth surfaces.

PREVENTION OF DEMINERALISATION

Although enamel demineralisation can be prevented by fastidious attention to maintaining a high standard of oral hygiene, it is often the case with child and teenage patients that the standard of oral hygiene falls below an acceptable level during a prolonged course of orthodontic treatment. Adjuncts such as antibacterial mouthwashes, sprays or varnishes containing fluoride (F^-) or chlorhexidine gluconate have been proposed in order to help control bacterial colonisation of teeth and orthodontic appliances.[13,14]

FLUORIDE

Fluoride has been proven to be particularly effective at reducing demineralisation of dental hard tissue. It becomes incorporated into the tooth structure and the hydroxyl group (OH^-) of the hydroxyapatite is replaced by F^-. This results in the creation of a fluoroapatite lattice that is more resistant to acid dissolution at or below the critical pH of 4.5. While there is clearly some protection from tooth bound 'intrinsic' fluoride, extrinsically provided fluoride has been shown to be considerably more effective in preventing decay.

Bacterial growth and metabolism can also be reduced by fluoride at concentrations exceeding 10 ppm. However, fluoride levels in the oral cavity are generally relatively low as fluoride is cleared from the mouth due to salivary secretion and swallowing. Therefore the effect of fluoride after using oral care products on bacteria is limited. Such fluoride levels are limited to a very short period after using a fluoride product.[15]

Today's consensus regarding the main mode of action of fluoride in the prevention of caries is by enhancing the remineralisation of initial caries defects and by inhibiting the demineralisation that would lead to caries initiation or progression. Fluoride thus shifts the 'demin-remin balance' from net demineralisation, in the case of caries active patients, towards net remineralisation.[16] Hence frequent exposure to fluoride products is key to preventing decay in individuals of all ages.[17]

Currently around 10% of the UK's population benefit from a water supply where the fluoride content, either naturally or artificially, is at the optimum levels for dental health. However, the risk of dental fluorosis is higher from the use of topical fluorides in these areas. It is important clinicians and patients are aware of the fluoride status of their water supply to ensure the appropriate topical fluoride regime can be applied to avoid this side effect. A particularly useful resource on this subject is *Delivering better oral health: an evidence-based toolkit for prevention* produced by the Department of Health.[18] This document clarifies what fluoride supplements are suitable for children of varying age groups and the concentration of the fluoride supplements available on the market.[18]

Benson *et al.*[19] carried out a Cochrane review to assess the effectiveness of fluoride and its delivery mechanism (mouthrinse, varnish, bonding system, and elastics) in the prevention of WSL formation; some of which is discussed further in the non-compliance section of this article. Overall the authors conclude there is evidence to support the use of daily 0.05% sodium fluoride mouthrinses in order to reduce the prevalence and severity of WSL.

Since this review a well-constructed randomised control trial has been carried out investigating the effectiveness of fluoride varnish. Children receiving orthodontic treatment with fixed appliances were randomly allocated to receive a fluoride varnish or a placebo varnish every sixth week during the treatment period. The study found the incidence of WSL was 7.4% in the group receiving fluoride varnish and 25.3% in the placebo group. This indicates there is a clinically significant benefit to applying fluoride varnish to orthodontic patients at each appointment. However, this will inevitably result in increased appointment times and other methods that do not increase the burden on clinicians may be more acceptable.[20]

Fluoride-releasing chewing gum is also available, which has been shown to release low concentrations of fluoride into saliva.[21] This has the benefit of combining salivary stimulation with active caries prevention. However, this relies on patient compliance and there is no control over the fluoride release mechanism. Patients may not stick to the recommended prescription and consume large quantities of fluoride, which has the potential to be toxic.

CHLORHEXIDINE

Chlorhexidine is a positively charged molecule that binds to negatively charged sites on bacterial cell walls. The uptake of chlorhexidine is very rapid

and can destabilise bacterial cell walls within 20 seconds. Once the cell wall is damaged, chlorhexidine then crosses into the cell itself and attacks the cytoplasmic membrane. Damage to the delicate semi-permeable membrane causes leakage of components, which leads to cell death.[22] Many antimicrobial agents struggle to eliminate organisms in a biofilm. However, chlorhexidine can inhibit adherence of microorganisms to a surface, thereby preventing growth and development of biofilms. This is of particular use in the oral cavity as biofilms protect and facilitate bacterial attachment to tooth surfaces.[23]

The Cochrane review carried out by Benson *et al.*[19] also compared the efficacy of F-varnish relative to alternating F^- and antimicrobial varnishes (for example, Cervitec 1% chlorhexidine varnish) in the prevention of WSL formation. There was only one study suitable for inclusion, Ogaard *et al.*[24] This trial reported that antimicrobial varnish significantly reduced the levels of *S. mutans* for up to 48 hours after bonding, but resulted in no significant reduction in WSL formation. This finding is surprising, considering the potent action of chlorhexidine against *S. mutans* and the importance of these bacteria in the initiation of WSL development. Another clinical trial by Attina *et al.*[25] found that the inhibitory effect of chlorhexidine is shorter lived than previously thought with *S. mutans* levels returning to baseline levels within two weeks of varnish application. Other studies[26,27] have both supported and contradicted these findings. However, on balance, the evidence indicates chlorhexidine varnish and mouthrinses are unlikely to be an efficient method of preventing demineralisation during orthodontic treatment.

In addition to the lack of evidence supporting its use, chlorhexidine use has a tendency to stain both the glass-ionomer cements and composites used to bond brackets, producing a particularly non-aesthetic effect that is extremely difficult to remove. Considering the poor compliance with simple F^- mouthwashes, it is reasonable to assume the compliance would be even lower with poor tasting chlorhexidine mouthwash and gel.

Relying on patients or clinicians to remember to apply fluoride or chlorhexidine between and during appointments is not an efficient method of preventing WSL formation. It is often the case that patients at the highest risk of developing WSL are those least likely to comply with oral hygiene and mouthrinsing regimes. Therefore less patient dependent modes of delivery are required and a number have been investigated.

NON-COMPLIANCE RELIANT MODES OF PREVENTION

Glass-ionomer cements (GIC) have been used in dentistry for a number of years partly as a result of the ability to release fluoride ions. The initial release of fluoride ions from the cement is due to polyacid attack on its alumino silicate glass. Subsequently the glass ionomer is also able to absorb fluoride from toothpastes and mouthwashes, and it then acts as a reservoir, releasing the fluoride back to the mouth in a continuous manner. As a result of this fluoride releasing effect and also because acid etching of enamel is not required before use, glass-ionomer cements have also been adopted as orthodontic bonding agents.

When used in orthodontics, their ability to reduce the incidence of WSL formation when compared to the use of conventional diacrylate bonding agents has been demonstrated by Marcusson *et al.*[28] In their clinical trial there were significantly fewer WSL on teeth bonded with GIC (24%) compared to those bonded with diacrylate (40.5%). The findings of this study and many others were included in the 2004 Cochrane review[19] into the prevention of white spot lesions, which concluded that the use of glass-ionomer cement for bracket bonding reduces the prevalence and severity of white spot lesions. However, studies have shown GIC to have a significantly lower shear bond strength compared to resin bonding systems[29] and in an attempt to increase fluoride release and to improve bond strength, resin-modified glass-ionomer cements (RMGIC) were developed.[30]

Dual- and light-cured RMGICs have been used by clinicians to bond orthodontic brackets. However, depending on the material used and pre-treatment of the teeth before bonding, the achieved bond strengths can vary.[31,32]

Most studies have concluded that RMGICs have comparable shear bond strengths to resin bonding systems if acid etching of the tooth surface is carried out before bonding.[33]

More recent studies, including a trial by clinical trial by Choo *et al.*[34] have found that RMGIC is capable of achieving the same or greater bond strength compared to resin bonding systems, even if the enamel has not been acid etched or is not contaminated with water before bonding.[35] However, etching with 15% phosphoric acid for 15 seconds and without moisture contamination of the RMGIC appears to produce optimal bond strengths.[35]

Even so, glass-ionomer-based materials have not found widespread acceptance as orthodontic bonding agents. Possibly because clinicians are more familiar and comfortable with the acid etch technique and the handling characteristics of composite resins. As an evidence-based profession it is clear more consideration needs to be given to the widespread use of RMGICs as orthodontic bonding agents in order to help prevent this common side effect of fixed appliance treatment.

Fluoride has also been incorporated into composite bonding systems in an attempt to reduce the risk of developing WSL. Although laboratory studies have shown that fluoride does not compromise the measured force to debond, the initial high fluoride release is short-lived and cannot be relied upon to prevent WSL over a full course of orthodontic treatment.[36]

Another orthodontic bonding agent capable of releasing fluoride ions during service and therefore reducing WSL formation compared with composite resin bonding agents is compomer. One study reported the prevalence of WSL at debond to be 20% in the case of patients treated using a compomer, compared to 26% in the case of the composite bonding agent.[37] Compomers comprise approximately 50% resin and 50% glass-ionomer cement. They also have not gained widespread acceptance as orthodontic bonding agents, possibly because the literature only reports small and clinically insignificant reductions in demineralisation. Their use is little known and many orthodontists are unfamiliar with their use for orthodontic bonding.

Chlorohexidine and zinc oxide have also been successfully incorporated into glass-ionomer bonding agents. These provide improved antimicrobial properties when

compared to the original GIC compound, without significantly altering the measured force to debond.[38,39] This may help reduce demineralisation and gingival inflammation around orthodontic bands, but, for the reasons stated previously, glass ionomer is not routinely used in orthodontic bonding.

Elastomeric ligation remains the most commonly used method of ligation and elastomers containing stannous fluoride (SnF) particles have been made commercially available (for example, Fluor-I-Ties, Ortho Arch Co. Inc., USA). Tinsley *et al.*[40] showed that they not only leach fluoride into the oral cavity, but they are also able to imbibe fluoride from the surrounding environment for later release. Fluoride releasing elastomerics are capable of providing a statistically significant and clinically worthwhile reduction in enamel decalcification during fixed appliance therapy.[41,42] However, a Cochrane review on the prevention of white spot lesions highlighted concerns regarding the methodology and bias, including a lack of blinding in the studies. The authors concluded there was insufficient evidence to advocate their use for prevention of WSL.[19] These elastomerics have not found widespread acceptance due to reported compromised force delivery and inconsistent fluoride release.[43,44]

Slow release fluoride devices have also been developed, which can elevate intra-oral fluoride levels of plaque and saliva for prolonged periods of up to two years. These resemble small glass beads, which can be bonded to posterior teeth or attached to orthodontic wires. Toumba *et al.*[45] carried out a randomised clinical trial, which demonstrated 76% fewer new carious surface lesions in high caries-risk children after two years. However, these are inherently plaque retentive and like any attachment in the mouth, they can detach and therefore require regular monitoring.

NEWER TECHNOLOGIES

Coatings

A number of new technologies, principally fillers and coatings, have recently become available with potential antimicrobial and antibiofilm properties. Silver-platinum (Ag-Pt) coatings have been applied to stainless steel orthodontic brackets to provide antimicrobial activity and resistance to biofilm formation as a result of silver ion (Ag^+) release. Their mode of action is through ionisation of the Ag when it comes into contact with saliva to produce Ag^+ ions. These ions then interact with the bacterial cell membranes leading to pitting, an increase in cell membrane permeability, the leaching of the cell contents and eventual cell death.[46]

Although such coatings have the potential to be effective, they are also likely to be abraded and lost over time, or may potentially corrode within the oral environment. To date no trials have been undertaken using such brackets and it is unlikely any coating would be maintained on the bracket surface for the duration of a course of orthodontic treatment. However, if antimicrobial particles could be incorporated directly within the bulk of the bracket material or adhesive, then longevity may no longer be an issue. In light of this, experimental composite adhesives have also been manufactured with Ag particles embedded within the matrix. Although they demonstrate reduced bacterial adhesion, the antimicrobial effect is short-lived and the resin produced is darker in colour as result of the innate colour of the silver particles.[47] High concentrations of Ag would render the adhesive unsuitable for use under aesthetic orthodontic brackets.

Antimicrobials

Other low molecular weight antimicrobials, such as antibiotics and iodine, have also been incorporated into dental composites to reduce or prevent bacterial adhesion. However, they are also potentially toxic since there is a lack of control over the release mechanism.[48] Cetylpiridinium chloride (CPC), a cationic quaternary ammonium compound often found in mouthwashes and toothpastes, has been incorporated into a commercially available dental adhesive resin. In laboratory tests it has been shown to exhibit long-term antimicrobial activity (up to 196 days), although as the concentration increases beyond 2.5% by weight the diametric strength of the composite reduces. No clinical trials have been carried out to see if the antimicrobial properties would persist for any length of time in the mouth, or whether the resin would be suitable for orthodontic bonding.[49]

Clearfil Protect Bond (Kuraray Medical, Okayama, Japan), a new fluoride-releasing, composite bonding agent, also containing a similar antibacterial monomer (12 methacryloyloxydodecylpyridinium bromide, or MDPB) is commercially available for clinical use. A split mouth design clinical trial carried out to compare the failure rate, plaque accumulation and occurrence of WSL between this material and Transbond plus (3 M Unitek, Seefeld Germany) found the bond failure rate with Clearfil Protect Bond to be significantly higher and with no additional benefit with regard to plaque accumulation and prevention of demineralisation.[50]

THE FUTURE?

Nanotechnology

Many scientists believe Dr Richard Feynman was the visionary who first theorised the possibilities of nanotechnology in his series of lectures 'Plenty of room at the bottom' (1958). Here he discussed how biological systems are exceedingly small and made up of even smaller active cells. These cells can manufacture various substances and store and transfer information, all on a very small scale. Feynman stated, '*Consider the possibility that we too can make a thing very small which does what we want. That we can manufacture an object that manoeuvres at that level*!'

Nanoparticles are of particular interest because their surface-to-mass ratio is far greater than other particles, improving their ability to absorb and carry compounds such as drugs and proteins. Entrapment of drugs in this way can enhance delivery to, or uptake by, target cells and thereby reduce the toxicity of the drug to non-target organs. This will produce an increase in the therapeutic index and reduce toxicity to other organ systems.

Titanium dioxide nanoparticles

Titanium dioxide (TiO_2), particularly in its nanoparticle format, has generated a great deal of interest over recent years as it has numerous potential applications. A number of studies have since demonstrated TiO_2 to be an effective light driven photocatalyst with strong bactericidal activity. It is now considered to be one of the most promising antimicrobial materials because of its chemical stability, low cost,

non-toxic nature, long-term stability and high activity.[51,52]

In dentistry TiO_2 has been tested as a coating on both dental implant instruments in order to help reduce bacterial colonisation and improve disinfection.[53]

More recently, *in vitro* pilot studies have been carried out to assess if TiO_2 coatings applied to orthodontic brackets, both steel[54] and ceramic,[55] and also to orthodontic wires[56] could reduce bacterial colonisation. Although such coatings can successfully prevent bacterial adhesion, their longevity in the clinical setting is questionable. An alternative approach to get over the problems of wear would be to incorporate TiO_2 nanoparticles into commercially available dental adhesives resins in volumes sufficient to achieve a good bactericidal effect, but without compromising the bond strength.[57]

However, just as with coatings, the full antimicrobial effect of such a TiO_2 containing resin cannot be achieved unless the nanoparticles are exposed to UV-A light on a regular basis. Not only does UV-A form only a very small fraction of the solar spectrum (<5%), meaning that its activity is severely reduced under darker conditions, such as those found within the oral cavity,[58] but the levels required would be so high as to cause damage to human cells.

In an attempt to overcome the problem of UV-light researchers have conducted extensive studies on doping, sensitisation and covering the surface of the particles with dyes in order to extend the light absorption to within the visible spectrum and also increase the photocatalytic activity. This has been found to be possible by doping with elements, such as silver (Ag) and platinum (Pt) from group VIII in the Periodic Table.[59]

Ag^+ doped TiO_2 has become the most suitable material for industrial and biomedical applications, and it is cheap and easy to prepare. Ag^+ modification of TiO_2 induces a decrease in the band gap energy, allowing visible light to activate the material's photocatalytic activity. This opens the possibility that such technology could be adopted to help to prevent bacterial colonisation of orthodontic brackets and reduce the occurrence of WSL over a full course of orthodontic treatment. If the particles are retained within the bulk of any material (such as a bonding resin or polymeric bracket) as well as at the surface, then any wear would simply expose more particles and thereby confer an antimicrobial effect throughout the duration of treatment.

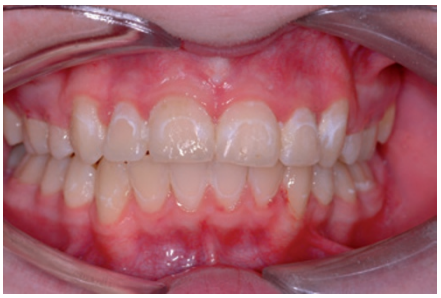

Fig. 2a Orthodontic demineralisation at debond; courtesy of Pamela Ellis

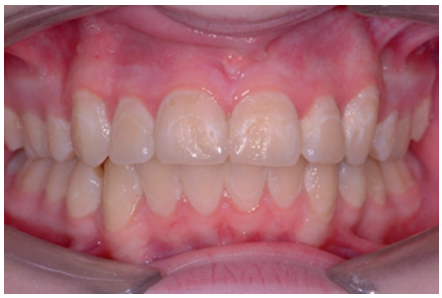

Fig. 2b Natural remineralisation of WSL at 3-month review; courtesy of Pamela Ellis

Zinc oxide nanoparticles

As well as TiO_2, studies have shown that metal oxide nanoparticles such as zinc oxide (ZnO_2) can lower bacterial counts on the surface of bonding composites by up to 80%. In addition they do not appear to compromise the mechanical or aesthetic properties of the composite. Once again there is a potential for the effects to be maintained indefinitely, since the nanoparticles are incorporated within the bulk resin rather than just at the surface.[60,61]

NEW MANUFACTURING PROCESSES

As existing dental materials reach their performance limits, one of the major scientific challenges for the future is the development of dental and orthodontic materials, which incorporate and retain these antimicrobial nanoparticles. This is a task that can only be met by developing new manufacturing processes, which allow material characteristics to be tailored for purpose. One such potential technology is freeze casting. Also known as ice-templating, this is a method by which custom made porous ceramic structures can be constructed using a frozen ceramic powder slurry. By means of a temperature controlled gradient it is possible to develop a ceramic structure with varying degrees of porosity. The temperature gradient results in the formation of ice crystals within the material, which are subsequently evapourated by placing the frozen cast within a freeze drier.[62] The resulting porosity within the ceramic structure can then be infiltrated with a variety of materials such as polymer, and these in turn may contain antimicrobial nanoparticles. In this way it may be possible to produce a true hybrid ceramic/polymeric bracket, which is graduated from almost 100% ceramic at the slot and tie wings to 100% antimicrobial polymer at the bonding surface. This might provide long term antimicrobial protection to the areas of the tooth most prone to demineralisation, adjacent to the bracket base and with minimal need for patient compliance. In addition such a truly customised hybrid bracket would enable easy debonding of the polymeric bracket while retaining the essential properties of the ceramic bracket in the body a nd slot area.

Pilot research is currently underway at Bristol University using this new technology.

REMINERALISATION

Fortunately the process known as remineralisation can, in many cases, reverse WSL. Demineralisation and remineralisation of the dentition is a daily occurrence and when the equilibrium shifts in favour of remineralisation, WSL can reduce in size or disappear completely. This involves the penetration of the enamel surface to the subsurface lesion by ions of calcium, phosphate and fluoride.[63] These ions combine to form hydroxyapatite, or fluoroapatite in the presence of fluoride. As the mineral is formed, it is deposited on the margins of the lesion, resulting in a progressive reduction in size towards the centre. There is considerable evidence in the literature to demonstrate that on removal of fixed orthodontic appliances, WSL regression may occur provided the intraoral environment is suitable (Figs 2a and b).[63,64]

TREATMENT OF DEMINERALISED LESIONS FOLLOWING ORTHODONTIC TREATMENT

It would seem logical that rapid remineralisation of WSL by the application of high concentrations of fluoride would be the treatment of choice after the removal of fixed orthodontic appliances. However, this is not the case and it has been shown that the application of high concentrations of fluoride leads to cessation of both remineralisation and demineralisation of WSL subsurface lesions due to hypermineralisation of the surface of the enamel.[65] This surface hypermineralisation inhibits diffusion of ions into deeper parts of the lesion, essentially fixing its size, shape and appearance.[66] Therefore, high fluoride should be avoided and patients should be advised to brush as normal with 1,500 ppm toothpaste, twice a day, to maintain the benefits of fluoride, while limiting the possibility of causing hypermineralisation.

Increased levels of saliva and its circulation around the mouth have been shown to aid remineralisation and the use of sugar–free chewing gum has been recommended to promote this effect.[67] A regime of chewing gum for 20 minutes, five times per day for three weeks has been shown to promote remineralisation in non-orthodontic patients. Furthermore, the use of xylitol containing gum may aid remineralisation due to its potential anti-caries properties. Xylitol is taken up by many strains of *S. mutans* and *S. sanguis*, even though these organisms are unable to metabolise it, which results in inhibition of the glycolysis pathway and reduces the production of acidic waste metabolites.[68]

Casein phosphopeptide amorphous calcium phosphate

Reynolds *et al.*[69] first reported casein phosphopeptide amorphous calcium phosphate (CPP-ACP) as a novel product that may be beneficial in aiding remineralisation of non-cavitated lesions. This has since been marketed as GC Tooth Mousse. Its mechanism of action is believed to be the localisation of ACP on the tooth surface by binding to pellicle, supra and subgingival plaque.[70] A supersaturated state is then maintained at the enamel surface allowing calcium and phosphate ions to diffuse down concentration gradients into the subsurface lesions and promote remineralisation. This ensures the activity gradient is favourable for remineralisation, and because calcium hydrogen phosphate ($CaHPO_4$) is uncharged, it can diffuse freely into the body of the WSL. As it dissociates in the subsurface lesion, charged ions are formed and are rapidly incorporated into hydroxyapatite or fluorapatite crystals, promoting the continued diffusion of $CaHPO_4$. The reverse will occur at the surface as the charged ions form neutral $CaHPO_4$, driven by the same equilibrium. This constant cycle maintains the activity gradient until the lesion has remineralised. Both electron microprobe and transverse microradiography studies have shown conclusively that remineralised enamel in the form of hydroxyapatite following CPP-ACP application to be present in the body of the lesion, with a higher calcium to phosphate ratio than normal.[71] The majority of these products also contain low concentrations of fluoride and it is not clear if this has a synergistic effect.

Numerous *in vitro* and *in vivo* studies have demonstrated that CPP-ACP containing sugar-free chewing gum can retard caries development and accelerate remineralisation.[72,73]

Bleaching

An alternative treatment for WSL is vital tooth bleaching with hydrogen peroxide or carbamide peroxide, which is used to increase the whiteness of the surrounding enamel to match that of the WSL, thus disguising the appearance of the lesion. This is a non-invasive procedure, which patients and clinicians are often happy to adopt. However, it merely camouflages the lesion and does not affect its size or depth.

Although these methods of remineralising or masking WSL no doubt have their place, they are a reactionary mode of treatment and prevention of WSL is clearly more beneficial to both patients and clinicians.

CONCLUSIONS

Demineralisation is still one of the major complications of orthodontic treatment, leading to unsightly WSL formation. There are a number of methods available aimed at reducing the incidence of WSL, but they all have limitations. Recent technological advances have enabled ceramics and polymers to be combined, producing a hybrid material where the antimicrobial properties can be tailored for purpose. Capitalising on these advances will enable the production of aesthetic orthodontic brackets, which provide long-term protection against WSL without relying on patient compliance.

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