Review Article

Prevention and Treatment of White Spot Lesions in Orthodontic Patients

Abstract

Decalcification of enamel, appearing as white spot lesions (WSLs), around fixed orthodontic appliances is a major challenge during and after fixed orthodontic treatment by considering the fact that the goal of orthodontic treatment is to enhance facial and dental esthetic appearance. Banded or bonded teeth exhibit a significantly higher rate of WSLs compared to the controls with no braces as fixed appliances and the bonding materials promote retention of biofilms. These lesions are managed in the first step by establishing good oral hygiene habits and prophylaxis with topical fluorides, including high-fluoride toothpastes, fluoride mouthwashes, gels, varnishes, fluoride-containing bonding materials, and elastic ligatures. Recently, other materials and methods have been recommended, including the application of casein phosphopeptides-amorphous calcium phosphate, antiseptics, probiotics, polyols, sealants, laser, tooth bleaching agents, resin infiltration, and microabrasion. This article reviews the currently used methods to manage enamel demineralization during and after orthodontic treatment and the risk factors and preventive measures based on the latest evidence.

Keywords: *Demineralization, oral hygiene, orthodontic treatment, remineralization, white spot*

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Introduction

Enamel decalcification is a sequela of orthodontic treatment, which is aggravated by poor oral hygiene. Incipient enamel caries results in subsurface demineralization beneath an intact surface layer of enamel. Light is reflected differently from demineralized enamel surfaces compared to the adjacent sound enamel, giving rise to a chalky white appearance.[1] White spot lesions (WSLs) appear as small lines around the brackets; in some patients, they are visible as large decalcified areas with or without cavitation. Detection of WSLs after the removal of orthodontic appliances is absolutely discouraging.[2] This article reviews the frequency, distribution, and initiation of WSLs during and subsequent to orthodontic treatment and evaluates the current methods used to manage enamel demineralization resulting from orthodontic treatment, in addition to risk factors and preventive measures involved.

Prevalence of White Spot Lesions and Risk Factors

Clinically, WSLs might develop rapidly, appearing on the 4th week after initiating treatment in the presence of poor oral hygiene.[3] These decalcifications have been reported to be more common in patients undergoing fixed orthodontic treatment. However, their frequency has been reported to be widely variable, from 2% to 97% in different epidemiological studies,[4-7] which might be explained by the techniques used to detect and characterize them, including visual inspection, photographs, fluorescent methods, and optical modalities such as diagnodent, quantitative light-induced fluorescence, and digital image fiber-optic transillumination.[6,8] Methods using quantitative laser techniques are more sensitive, yielding a higher prevalence rate than the simple visual technique. On average, such decalcifications are found in 15.5%–40% of patients before orthodontic treatment and in 30%–70% during the treatment.[6] Based on a recent meta-analysis, in the 14 studies evaluated for WSLs, the incidence rate of new carious lesions that developed during orthodontic treatment was 45.8%, with a prevalence rate of 68.4% in patients under orthodontic treatment. It was concluded that the incidence and prevalence rates of WSLs are quite high and alarming in patients receiving orthodontic treatment, necessitating the attention of both patients and caregivers to effective caries prevention measures.[9]

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Therefore, before undertaking orthodontic treatment, these lesions should be diagnosed and recorded by means of standardized photographic plates, taking into account magnification, exposure time, lighting, etc.[4] WSLs before orthodontic treatment are considered a risk factor for the development of new lesions,[5] with poor oral hygiene, excessive drinking, frequent use of fermentable carbohydrates, excess bonding, long etching time (>15 s), decayed/treated molars, and the duration of treatment being considered other risk factors.[5,10] Richter *et al*. reported the development of three new lesions in 22 months, with at least five injuries in 33 months of treatment.[11]

Formation and Distribution of White Spot Lesions

There is a major change in the bacterial flora of the plaque in the oral cavity after orthodontic fixed appliances are introduced into the oral cavity, with higher concentrations of acidogenic bacteria in the plaque, the most important of which are *Streptococcus mutans* and *Lactobacilli*. High concentrations of bacteria lower the plaque in orthodontic patients to a greater extent compared to that in other patients,[12] resulting in more rapid progression of caries in patients with a full set of orthodontic appliances. WSLs might appear within 1 month of bracket placement around the brackets; regular carious lesions normally take at least 6 months to develop. WSLs commonly appear on the buccal aspects of teeth around the brackets, especially in the gingival area,[13] with the labiogingival area of lateral incisors as the most common and the maxillary posterior segments as the least common site for WSL; males are affected at a higher rate in comparison to females.[3] A significant increase was reported in the prevalence of these lesions around the brackets bases or between the brackets/bands and in the gingival margins in the cervical areas and the middle thirds of the teeth under orthodontic wires[14] and also with full-coverage rapid maxillary expanders.[15] Tufekci *et al*. reported a sharp increase in the number of WSLs during the first 6 months of treatment, increasing at a slower rate up to 12 months. Therefore, it is necessary to carry out critical evaluations of oral hygiene during the 1st months of treatment.[16]

Prevention and Management of White Spot Lesions

WSLs should be managed using a multifactorial approach. The most important strategy is to prevent demineralization and biofilm formation, and use of methodologies for remineralization of lesions, thinning, microabrasion, erosion-infiltration, adhesive composite resin restorations, and the bonded facets.[17]

Oral hygiene control

Prevention should first begin by educating and motivating the patient for compliance with a noncariogenic diet and observation of oral hygiene. Effective oral hygiene is the bedrock of prophylactic measures in fixed orthodontic patients. Mechanical plaque control and removal by proper brushing of the tooth surfaces, at least twice daily, with fluoride-containing toothpaste, especially in biofilm retention areas, is strongly recommended. During the recall visits, patient motivation should be reevaluated and if deemed necessary, the tooth surfaces should receive a professional cleaning and oral hygiene and dietary instructions should be repeated.[7,18] Use of a power toothbrushes or daily irrigation with water in association with manual tooth brushing might prove more effective in decreasing accumulation of plaque compared to manual toothbrushing alone.[19] Professional prophylactic cleaning reduces the bacterial load, increases the efficacy of brushing, and facilitates cleaning by the patient. Professional tooth cleaning two or three times a year helps maintain a healthy mouth, decreasing the risk of dental caries and the number of teeth with carious lesions. Fluoridated pastes with progressively finer particle sizes can be used to polish coronal surfaces; furthermore, elastomeric polishing cups or brushes help prevent mechanical retention of bacteria.[20] Along with the brushing frequency, patient age, time past from appliance removal, length of treatment, type of the tooth (central or lateral incisor), and WSL surface area had also affect WSL improvement.[21]

Fluoride products

The favorable role of fluoride in preventing WSL has been documented with the use of the following: fluoride mouthwashes, fluoride gels, fluoride toothpastes, fluoride varnishes, fluoride in bonding agents, and fluoride in elastomers. The fluoride ion prevents dental caries, by modifying bacterial metabolism in dental plaque through inhibition of some enzymatic processes, by inhibiting production of acids by altering the composition of bacterial flora and/or the metabolic activity of microorganisms, and by decreasing demineralization and promoting remineralization of carious lesions at early stages through a remineralization effect, especially at low concentrations.[22]

Fluorinated toothpastes

The fluoride concentration of toothpastes (in the form of sodium fluoride, monofluorophosphate, stannous fluoride) should be over 1000 ppm; toothpastes with higher fluoride concentrations are most effective.[2,23,24] The use of a dentifrice with a high fluoride concentration (5000 ppm), twice daily, by patients at high risk for WSL is more effective than conventional formulations;[25-27] however, such a toothpaste (Duraphat) cannot be prescribed for patients under 16 years of age. Heymann and Grauer[5] recommend this toothpaste for brushing in the evenings only. Nonetheless, use of a fluoride toothpaste alone is not effective in preventing WSL in the majority of patients, even with good oral hygiene.[11] Therefore, it is recommended that other fluoride sources be used.[4,18,28-31]

Fluoridated mouthwashes

Daily use of fluoridated mouthwashes containing sodium fluoride has been shown to result in a significant decrease in the development of carious lesion around and beneath bands. Antibacterial agents have been incorporated into these mouthwashes, including chlorhexidine, triclosan, or zinc to promote their cariostatic effects.[2] Benson carried out a systematic review and recommended the daily use of 0.05% NaF mouthwash to prevent enamel demineralization during fixed orthodontic treatment.[32] A daily mouthwash containing NaF (0.05% or 0.2%) and/or weekly rinse containing alpha-1-fetoprotein (1.2%) have been demonstrated to decrease the incidence of enamel demineralization during fixed orthodontic treatment.

Fluoride varnishes

Fluoride varnishes (2–4 applications yearly) have proved effective in decreasing the incidence of caries in both deciduous and permanent dentitions.[33] Fluoride varnishes have proved a safe method of fluoride application. Advantages of fluoride varnishes over other topical fluoride regimens include protection of enamel in the absence of patient compliance and continuous fluoride release over a long period of time. The application of a fluoride varnish resulted in a 44.3% decrease in enamel demineralization in patients undergoing orthodontic treatment.[2] Azarpazhooh and Limeback[34] reported after a 3-year follow-up period that application of a fluoride varnish every 6 months proved most cost-effective technique for high- and medium-risk groups. They also concluded that Durafluor and Duraphat released fluoride at a slow rate for up to 6 months, with the greatest release observed during the first 3 weeks, followed by a more gradual delivery. Therefore, they supported the recommendation of a biannual application of single-dose preparations. However, some studies have recommended an application every 90 days (every 3 months) to promote adequate protection.[35] The application of a fluoride varnish every 6 weeks during orthodontic treatment has been shown to effective in some other studies.[29] Recently, an *in vivo* study by Perrini *et al*. showed that periodic application of fluoride varnishes in patients undergoing fixed orthodontic treatment can provide some protection against WSLs, which might not be statistically significant if the patients exhibit excellent oral hygiene.[36]

A one-time application of a fluoride varnish, just before the initiation of orthodontic treatment, did not provide any additional preventive advantage over good dental hygiene with the use of fluoride toothpastes in terms of the development of WSLs and gingivitis in patients at a low to moderate caries risk. Patients often undergo an application of fluoride varnish just before orthodontic treatment with fixed appliances. However, the efficacy of this technique is yet to be elucidated.[37]

Considering the low efficacy of patient-applied measures, there have been attempts to use the benefits of materials that release fluoride over time, including continuous release of fluoride from the bonding system around the bracket base, which can be very advantageous. Fluoride-containing adhesives have not proved effective in decreasing demineralization,[38,39] but compomers[40] and glass-ionomer cements[41,42] have been promising in this context. Glass-ionomer cements are less strong than composite resins; therefore, there are more bracket failures when they are used for orthodontic bonding procedures.[43] In recent decade, ever-increasing attention has been devoted to the use of "smart" bioactive materials in the dental field, especially to remineralize dentin, with bioactive glass (BAG), BAG-ionomer, being incorporated into gastrointestinal to enhance bioactivity, tooth regeneration, and reconstruction capacity in some studies.[44]

The release of fluoride from elastomeric ligatures might help decrease demineralization prevalence;[45] however, incorporating fluoride into elastics might affect their physical properties, resulting in their faster deterioration in the oral cavity.[46,47] The latest Cochrane review[29] on the role of fluoride in preventing WSL as a result of orthodontic treatment did not lead to recommendations for the use of adhesives or ligatures that release fluoride since it does not fulfill the inclusion criteria of studies (randomized controlled trial-clinical trials on comparison of fluoride product with no use of such products or the use of a nonfluoridated control-assessment of remineralization of enamel at the beginning and at the end of orthodontic treatment). A recent study suggested that orthodontic cements with microcapsules release bioavailable fluoride, calcium, and phosphate ions near the tooth surface, with the capacity to be recharged with fluoride and with no effect on the adhesion of the material to enamel.[48] Incorporation of microcapsules into dental materials might promote remineralization. Various intraoral fluoride slow-release devices,[49] including copolymer membrane device, glass device containing fluoride, hydroxyapatite-Eudragit RS 100 diffusion-controlled fluoride system and slow-release tablets for intrabuccal use have been introduced in recent years, with the capacity to release small amounts of fluoride over a long period of time, possibly for up to 6 months, before being replaced.[49,50]

It was reported that light cured pit and fissure sealants placed on the buccal surfaces near bonded orthodontic brackets were very effective (80%) in preventing demineralization *in vitro*, requiring no patient compliance.[51] However, these sealants cannot be removed easily and require meticulous polishing after removal. Application of a fluoride-containing sealant to the buccal aspects of bovine incisors to prevent the development of carious lesions around orthodontic brackets showed that ProSeal sealant alone or in association with brushing and/or brushing and the use of a fluoride-containing mouthwash was more efficacious in protecting enamel compared to brushing alone.[52]

Strategies to enhance or boost the anticariogenic properties of fluoride

The remineralization strategies used to boost fluoride increase the concentration of calcium and phosphate ions delivered to carious lesions and/or increase their concentrations in the plaque and saliva. Nonetheless, fluoride-based strategies are the gold standard for prevention and management of caries, with no evidence available in relation to any strategy that can effectively replace use of fluoride.[53]

Use of casein phosphopeptides amorphous calcium phosphate

Demineralization of enamel might be prevented by products containing casein phosphopeptides-amorphous calcium phosphate (CPP-ACP), with Reynolds reporting that CPP-ACP, which is derived from milk casein, was absorbed through the enamel surface and affected the demineralization-remineralization processes.[54] Recent research has shown that this is accomplished by a part of the casein protein referred to as CPP, which carries calcium and phosphate ions "stuck" to it, in the form of APP.[55] This complex of CPP-ACP delivers the bioavailable calcium and phosphate ions. It has been suggested that the anticariogenic activity of CPP-ACP relies on the incorporation of nanocomplexes into the dental plaque and on the tooth surface, thereby serving as a calcium and phosphate reservoir. CPP-ACP binds to the bacterial wall and tooth surfaces.[56] In case of an intraoral acid attack, the calcium and phosphate ions are released, reaching a supersaturated state of ions in the saliva and then precipitating a calcium-phosphate compound on the exposed tooth surface.[57] In addition, the breakdown of the CPP can help increase the pH (buffer) by producing ammonia; in addition, it might prevent bacterial adhesion to tooth surfaces and delay formation of biofilms.[58] There is no Cochrane review available on the role of CPP-ACP in demineralization and remineralization. Nonetheless, several *in vitro* and *in situ* studies have shown that CPP-ACP-containing products decrease demineralization and support remineralization.[17,59-61] CPP-ACP might be incorporated into chewing gums, lozenges, or creams.[18] It is marketed by GC as a cream for application on tooth surfaces twice a day after brushing the teeth, refraining from drinking or eating for 30 min subsequent to application (Tooth Mousse, Tooth Mousse Plus) (Fluor 900 ppm).

Some clinical studies demonstrating the efficacy of CPP-ACP in the prevention and regression of WSLs during orthodontic treatment are referred to here. Robertson *et al*.[62] showed that CPP-ACP + fluoride (Tooth Mousse Plus) had a preventive effect compared to placebo. Two other studies showed no difference between the CPP-ACP, a fluoride gel (5% NaF)[63] or a fluoride varnish (Fluor Protector).[64] Lesions developed during orthodontic treatment are good candidates for studying remineralization strategies because treating such lesions with agents containing concentrated fluoride can mineralize the surface but not the lesion body, making the arrested lesions, depending on their location, an esthetic concern over time.[65] It is believed that the mechanism of action of CPP-ACP paves the way for deeper penetration of ions, resulting in remineralization of the entire body of the lesion rather than only the surface layer; this improves the esthetic appearance. In such studies, the duration of intervention is relatively short because it is believed that the bulk of regression of postorthodontic WSLs occurs immediately after debonding of brackets.[66] A recent study showed that application of CPP-ACP-containing varnish to bovine incisors, with or without brushing and use of a mouthwash, decreased the depth of carious lesions around orthodontic brackets.[67] Some studies have shown that daily application of a remineralizing cream was more effective in reversing the severity and visual appearance of postorthodontic WSLs compared to fluoride toothpaste. Application of CPP-ACP might be more effective than the fluoride rinse for remineralization postorthodontic treatment WSLs.[68-72] Therefore, the ability of the CPP-ACP to prevent the formation of orthodontic WSLs in the long term is yet to be elucidated. Clinical studies are not sufficiently strong and conclusive to end in reliable recommendations.[17]

Probiotics

Probiotics are live microorganisms with health benefits when they are administered in adequate numbers. It is hypothesized that probiotic strains interfere with or inhibit other microorganisms, especially pathogens. Probiotic bacteria might enhance to the effect of fluoride in preventing dental caries.[73]

Polyols

Polyols are sweeteners that are weakly metabolized (sorbitol) or not metabolized (xylitol) by cariogenic bacteria. Evidence supports that xylitol is noncariogenic, exhibits a dose- and frequency-dependent effect on dental plaque and mutans streptococci, and is safe. Chewing gum with xylitol (2 g of xylitol/socket) or polyols is recommended after each meal (three times daily) for 10–20 min.[60,74-76] Sengun reported that xylitol lozenges significantly decreased the acidity of dental plaque in fixed orthodontic appliance patients.[77] The xylitol lozenges helped neutralize the acidity of dental plaque after the consumption of sucrose in patients undergoing fixed orthodontic treatment.

Antiseptics

Listerine

No clinical studies are available on the effect of Listerine on prevention of WSLs during orthodontic treatment.[17]

Chlorhexidine

Chlorhexidine is the most commonly used antiseptic in dentistry and has proved very effective in the control and management of biofilms in gingivitis. It is available as mouthwashes, gels, or varnishes. It affects cariogenic flora and decreases mutans streptococci counts. Chlorhexidine varnishes are more effective than its gels and mouthwashes. Some studies have shown the efficacy of chlorhexidine varnishes in decreasing the prevalence of caries during orthodontic treatment while others have not shown the efficacy of a varnish of 40% chlorhexidine.[78-80]

Lasers

Laser irradiation for its acid resistance might be an invaluable adjunct to conventional acid etching at susceptible sites in patients at high caries risk, including those with rampant caries, those with disabilities unable to follow oral hygiene instructions, or those receiving orthodontic treatment with attachments on their teeth that retain plaque.[81] Application of lasers to prevent caries dates back to 1972. Laser beams increase enamel microhardness and resistance to acid attack. The principal lasers that are used in preventive dentistry include the argon lasers, CO_2, Nd-YAG, and erbium YAG.[82,83]

Irradiation of enamel with argon laser beams decreases the amount of demineralization up to 30%–50%. Fox[84] reported that, apart from decreasing enamel demineralization, laser beams lowered the dissolution threshold pH value. Laser beams resulted in changes in surface morphology but maintained an intact enamel surface. Several mechanisms have been suggested to explain increased resistance of enamel to caries after laser irradiation, but the exact mechanism is yet to be elucidated. The most likely mechanism appears to be through the formation of microspaces within the enamel after exposure to laser beams. These microspaces trap the released ions and serve as sites for remineralization within the enamel surface. Application of argon laser beams (488 nm) significantly decreased the mean lesion depth compared to visible light controls, supporting the fact that irradiation with argon laser beams might prevent the development of WSLs during treatment.[85,86]

There are conflicting reports on the effects of lasers in preventing WSLs associated with orthodontic treatment,[87-91] highlighting the need for randomized clinical trials.

After the Orthodontic Treatment

After the orthodontic appliances are removed, it is common to see a regression appearance of WSLs due to natural remineralization by saliva and abrasion due to brushing in the presence of oral and food hygiene.[92] This improvement depends on the severity of lesions and occurs in the order of 6 months of the debonding process; however, it is not sufficient and these WSLs should be treated. As a result, Guzmán-Armstrong *et al*.[92] recommend a delay of 6 months before treating these lesions.

Remineralization

The first choice for the elimination of WSLs is remineralization which, apart from strict oral hygiene measures, involves repeated applications and the compliance of a motivated patient and might take a long time. Several professionally and home applied products are available in different forms for such a purpose: solutions, varnishes, creams, pastes, and chewing gums. These products contain fluorides and/or casein phosphopeptide-amorphous calcium phosphate, and there is evidence with varying degrees of success in the dental literature.[4,26,28,93,94] Denis *et al*. advocated these measures for score of 0 and 1 of these lesions based on the ICDAS classification.[95] However, from the score 2, these measures were unable to remineralize the lesions in all their depth and it was necessary to consider more invasive techniques such as erosion–infiltration,[95] bleaching, and microabrasion.[92] Products with high concentrations of fluoride are not recommended for the treatment lesions in incisors and canines because they lead to tooth discoloration.[17] It should be considered that there is a lack of reliable scientific data to support remineralizing or camouflaging approaches to manage postorthodontic WSLs and further well-designed trials are needed.[96]

Bleaching

The esthetic results of bleaching procedures are limited and they might give rise to tooth sensitivity and a decreased enamel microhardness.[97] However, a recent study showed that bleaching incipient enamel caries with 10% carbamide peroxide could camouflage WSLs with no effect on the chemical and mechanical properties of the enamel; in addition, application of casein phosphopeptide-amorphous calcium phosphate was considered an adjunct treatment for promotion of mineral gain in the subsurface lesion.[98] Khoroushi *et al*. showed in an *in vitro* study that a gentle, noninvasive bleaching procedure by incorporating three different biomaterials, including nano-BAG, nano-hydroxyapatite, and nano-amorphous calcium phosphate, into bleaching agents might mitigate the negative effects of tooth bleaching and prevent the irreversible changes in the enamel surface.[99,100] This treatment modality should be reserved for patients with good oral hygiene to mask inactive lesions when natural remineralization is not complete.[92]

Microabrasion

Microabrasion consists of a chemical and mechanical processing of the enamel surface by applying an abrasive slurry of 6.6% (Opalustre) or 6% (Whiteness RM) hydrochloric acid with a brush. As microabrasion is relatively more invasive in nature, it was believed that delayed application was beneficial given improvements of lesions through saliva-based remineralization and spontaneous surface abrasion subsequent to debonding.[101] This is a useful method for the treatment of postorthodontic WSLs, but the depth of the lesion should be under 0.2 mm[4,5] and it might be associated with the bleaching technique.[92,102]

Erosion-infiltration

A minimally invasive treatment modality has been introduced in recent years, in which the WSL is infiltrated with the use of a low-viscosity resin. HCl etching is used to transform the outer surface into a more permeable, and the underlying porous structure is infiltrated with the use of a triethylene glycol dimethacrylate-based resin.[101]

Infiltration of proximal carious lesions (micropores) is initiated with a very low-viscosity resin, manufactured by dimethylglycine (icon). The procedure involves the penetration of the resin through etching with 15% hydrochloric acid for 20 s, followed by rinsing, drying, and dehydration of the enamel surface with ethanol. This resin stops the progression of caries and the other, with a refractive index close to that of sound enamel, camouflages the WSL in addition to reinforcing the compromised enamel prism structure.[95] The camouflage effect of this technique has been demonstrated both *in vitro* and *in vivo*. This camouflage effect varies depending on the depth of the lesion. The treatment is more effective esthetically in the early stages when it is in the active rather than in the inactive stage.[5,103] Since this is a new technique, there is insufficient clinical experience available in relation to orthodontic WSLs. Although 1-year follow-up study demonstrated that the method can create an enduring esthetic improvement of postorthodontic WSLs.[104] Senestraro *et al*.,[97] Knösel *et al*.,[105] Feng and Chu[27] did not observe color changes after 2, 6, and 12 months, respectively. However, Tirlet *et al*. reported good clinical outcomes 19 months after the treatment of nonorthodontic WSLs such as fluorosis after trauma.[106] An *in vitro* study by Yetkiner evaluated the color improvement and stability of WSLs following infiltration, fluoride, or microabrasion treatments and reported that infiltration and microabrasion decreased the whitish appearance of WSLs. Only infiltrated WSLs were stable after a discoloration challenge.[101]

Conclusion

Enamel decalcification around fixed orthodontic appliances is a common complication during and after orthodontic treatment. These lesions are managed by educating and motivating the patient to observe good oral hygiene. In addition, prophylaxis should be carried out with topical fluoride. Other materials and methods including CPP-ACP, antiseptics, probiotics, polyols, sealants, lasers, tooth bleaching, resin infiltration, and microabrasion have also been recommended. However, good oral hygiene is the most important prophylactic measure in fixed orthodontic patients to prevent WSLs.

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Conflicts of interest

There are no conflicts of interest.

References

1. Kleter GA. Discoloration of dental carious lesions (a review). Arch Oral Biol 1998;43:629-32.
2. Zabokova-Bilbilova E, Popovska L, Kapusevska B, Stefanovska E. White spot lesions: Prevention and management during the orthodontic treatment. Pril (Makedon Akad Nauk Umet Odd Med Nauki) 2014;35:161-8.
3. Chapman JA, Roberts WE, Eckert GJ, Kula KS, González-Cabezas C. Risk factors for incidence and severity of white spot lesions during treatment with fixed orthodontic appliances. Am J Orthod Dentofacial Orthop 2010;138:188-94.
4. Bishara SE, Ostby AW. White spot lesions: Formation, prevention, and treatment. Semin Orthod 2008;14:174-82.
5. Heymann GC, Grauer D. A contemporary review of white spot lesions in orthodontics. J Esthet Restor Dent 2013;25:85-95.
6. Julien KC, Buschang PH, Campbell PM. Prevalence of white spot lesion formation during orthodontic treatment. Angle Orthod 2013;83:641-7.
7. Sangamesh B, Kallury A. Iatrogenic effects of orthodontic treatment – Review on white spot lesions. Int J Sci Eng Res 2011;2:2-16.
8. Pretty IA. Caries detection and diagnosis: Novel technologies. J Dent 2006;34:727-39.
9. Sundararaj D, Venkatachalapathy S, Tandon A, Pereira A. Critical evaluation of incidence and prevalence of white spot lesions during fixed orthodontic appliance treatment: A meta-analysis. J Int Soc Prev Community Dent 2015;5:433-9.
10. Khalaf K. Factors affecting the formation, severity and location of white spot lesions during orthodontic treatment with fixed appliances. J Oral Maxillofac Res 2014;5:e4.
11. Richter AE, Arruda AO, Peters MC, Sohn W. Incidence of caries lesions among patients treated with comprehensive orthodontics. Am J Orthod Dentofacial Orthop 2011;139:657-64.
12. Lundström F, Krasse B. Streptococcus mutans and lactobacilli frequency in orthodontic patients; the effect of chlorhexidine treatments. Eur J Orthod 1987;9:109-16.
13. Mattousch TJ, van der Veen MH, Zentner A. Caries lesions after orthodontic treatment followed by quantitative light-induced fluorescence: A 2-year follow-up. Eur J Orthod 2007;29:294-8.
14. Mizrahi E. Enamel demineralization following orthodontic treatment. Am J Orthod 1982;82:62-7.
15. Yagci A, Korkmaz YN, Buyuk SK, Yagci F, Atilla AO. White spot lesion formation after treatment with full-coverage rapid maxillary expanders. Am J Orthod Dentofacial Orthop 2016;149:331-8.
16. Tufekci E, Dixon JS, Gunsolley JC, Lindauer SJ. Prevalence of white spot lesions during orthodontic treatment with fixed appliances. Angle Orthod 2011;81:206-10.
17. Morrier JJ. White spot lesions and orthodontic treatment. Prevention and treatment. Orthod Fr 2014;85:235-44.
18. Sudjalim TR, Woods MG, Manton DJ. Prevention of white spot lesions in orthodontic practice: A contemporary review. Aust Dent J 2006;51:284-9.
19. Harvey WJ, Powell KR. Care of dental enamel for the orthodontic patient. Aust Orthod J 1981;7:70-6.

20. Øgaard B. Oral microbiological changes, long-term enamel alterations due to decalcification, and caries prophylactic aspects. Orthodontic Materials Scientific and Clinical Aspects. Stuttgart: Thieme; 2001. p. 123-42.
21. Kim S, Katchooi M, Bayiri B, Sarikaya M, Korpak AM, Huang GJ. Predicting improvement of postorthodontic white spot lesions. Am J Orthod Dentofacial Orthop 2016;149:625-33.
22. Davidson CL, Bekke-Hoekstra IS. The resistance of superficially sealed enamel to wear and carious attack *in vitro*. J Oral Rehabil 1980;7:299-305.
23. Marinho VC. Cochrane reviews of randomized trials of fluoride therapies for preventing dental caries. Eur Arch Paediatr Dent 2009;10:183-91.
24. Walsh T, Worthington HV, Glenny AM, Appelbe P, Marinho VC, Shi X. Fluoride toothpastes of different concentrations for preventing dental caries in children and adolescents. Cochrane Database Syst Rev 2010;1:CD007868.
25. Al-Mulla A, Karlsson L, Kharsa S, Kjellberg H, Birkhed D. Combination of high-fluoride toothpaste and no post-brushing water rinsing on enamel demineralization using an *in-situ* caries model with orthodontic bands. Acta Odontol Scand 2010;68:323-8.
26. Bergstrand F, Twetman S. A review on prevention and treatment of post-orthodontic white spot lesions – Evidence-based methods and emerging technologies. Open Dent J 2011;5:158-62.
27. Feng CH, Chu XY. Efficacy of one year treatment of icon infiltration resin on post-orthodontic white spots. Beijing Da Xue Xue Bao 2013;45:40-3.
28. Bahoum A, Bahije L, Zaoui F. Enamel demineralization in orthodontics. Systematic use of fluoride in prevention and treatment. Schweiz Monatsschr Zahnmed 2012;122:937-47.
29. Benson PE, Parkin N, Dyer F, Millett DT, Furness S, Germain P. Fluorides for the prevention of early tooth decay (demineralised white lesions) during fixed brace treatment. Cochrane Database Syst Rev 2013;12:CD003809.
30. Benson PE, Parkin N, Millett DT, Dyer FE, Vine S, Shah A. Fluorides for the prevention of white spots on teeth during fixed brace treatment. Cochrane Database Syst Rev 2004;3:CD003809.
31. Srivastava K, Tikku T, Khanna R, Sachan K. Risk factors and management of white spot lesions in orthodontics. J Orthod Sci 2013;2:43-9.
32. Benson PE, Pender N, Higham SM. Quantifying enamel demineralization from teeth with orthodontic brackets – A comparison of two methods. Part 2: Validity. Eur J Orthod 2003;25:159-65.
33. Marinho VC, Worthington HV, Walsh T, Clarkson JE. Fluoride varnishes for preventing dental caries in children and adolescents. Cochrane Database Syst Rev 2013;7:CD002279.
34. Azarpazhooh A, Limeback H. Clinical efficacy of casein derivatives: A systematic review of the literature. J Am Dent Assoc 2008;139:915-24.
35. Vivaldi-Rodrigues G, Demito CF, Bowman SJ, Ramos AL. The effectiveness of a fluoride varnish in preventing the development of white spot lesions. World J Orthod 2006;7:138-44.
36. Perrini F, Lombardo L, Arreghini A, Medori S, Siciliani G. Caries prevention during orthodontic treatment: *In-vivo* assessment of high-fluoride varnish to prevent white spot lesions. Am J Orthod Dentofacial Orthop 2016;149:238-43.
37. Kirschneck C, Christl JJ, Reicheneder C, Proff P. Efficacy of fluoride varnish for preventing white spot lesions and gingivitis during orthodontic treatment with fixed appliances-a prospective randomized controlled trial. Clin Oral Investig 2016;20:2371-8.
38. Turner PJ. The clinical evaluation of a fluoride-containing orthodontic bonding material. Br J Orthod 1993;20:307-13.
39. Banks PA, Burn A, O'Brien K. A clinical evaluation of the effectiveness of including fluoride into an orthodontic bonding adhesive. Eur J Orthod 1997;19:391-5.
40. Millett DT, McCluskey LA, McAuley F, Creanor SL, Newell J, Love J. A comparative clinical trial of a compomer and a resin adhesive for orthodontic bonding. Angle Orthod 2000;70:233-40.
41. Marcusson A, Norevall LI, Persson M. White spot reduction when using glass ionomer cement for bonding in orthodontics: A longitudinal and comparative study. Eur J Orthod 1997;19:233-42.
42. Vahid-Dastjerdi E, Borzabadi-Farahani A, Pourmofidi-Neistanak H, Amini N. An *in-vitro* assessment of weekly cumulative fluoride release from three glass ionomer cements used for orthodontic banding. Prog Orthod 2012;13:49-56.
43. Norevall LI, Marcusson A, Persson M. A clinical evaluation of a glass ionomer cement as an orthodontic bonding adhesive compared with an acrylic resin. Eur J Orthod 1996;18:373-84.
44. Khoroushi M, Keshani F. A review of glass-ionomers: From conventional glass-ionomer to bioactive glass-ionomer. Dent Res J (Isfahan) 2013;10:411-20.
45. Banks PA, Chadwick SM, Asher-McDade C, Wright JL. Fluoride-releasing elastomerics – A prospective controlled clinical trial. Eur J Orthod 2000;22:401-7.
46. Mattick CR, Mitchell L, Chadwick SM, Wright J. Fluoride-releasing elastomeric modules reduce decalcification: A randomized controlled trial. J Orthod 2001;28:217-9.
47. Miethke RR. Comment on determination of fluoride from ligature ties. Am J Orthod Dentofacial Orthop 1997;111:33A.
48. Burbank BD, Slater M, Kava A, Doyle J, McHale WA, Latta MA, *et al.* Ion release, fluoride charge of and adhesion of an orthodontic cement paste containing microcapsules. J Dent 2016;45:32-8.
49. Pessan JP, Al-Ibrahim NS, Buzalaf MA, Toumba KJ. Slow-release fluoride devices: A literature review. J Appl Oral Sci 2008;16:238-46.
50. Marini I, Pelliccioni GA, Vecchiet F, Alessandri Bonetti G, Checchi L. A retentive system for intra-oral fluoride release during orthodontic treatment. Eur J Orthod 1999;21:695-701.
51. Garcia-Godoy F. Shear bond strength of a resin composite to enamel treated with an APF gel. Pediatr Dent 1993;15:272-4.
52. Pithon MM, Santos Mde J, de Souza CA, Leão Filho JC, Braz AK, de Araujo RE, *et al.* Effectiveness of fluoride sealant in the prevention of carious lesions around orthodontic brackets: An OCT evaluation. Dental Press J Orthod 2015;20:37-42.
53. Fontana M. Enhancing fluoride: Clinical human studies of alternatives or boosters for caries management. Caries Res 2016;50 Suppl 1:22-37.
54. Reynolds EC. Casein phosphopeptide-amorphous calcium phosphate: The scientific evidence. Adv Dent Res 2009;21:25-9.
55. Shen P, Cai F, Nowicki A, Vincent J, Reynolds EC. Remineralization of enamel subsurface lesions by sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. J Dent Res 2001;80:2066-70.
56. Cross K, Huq N, Stanton D, Sum M, Reynolds E. NMR studies of a novel calcium, phosphate and fluoride delivery vehicle-α S1-casein (59-79) by stabilized amorphous calcium fluoride phosphate nanocomplexes. Biomaterials 2004;25:5061-9.
57. Iijima Y, Cai F, Shen P, Walker G, Reynolds C, Reynolds EC. Acid resistance of enamel subsurface lesions remineralized by a sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. Caries Res

2004;38:551-6.
58. Cochrane NJ, Cai F, Huq NL, Burrow MF, Reynolds EC. New approaches to enhanced remineralization of tooth enamel. J Dent Res 2010;89:1187-97.
59. Cochrane NJ, Reynolds EC. Calcium phosphopeptides – Mechanisms of action and evidence for clinical efficacy. Adv Dent Res 2012;24:41-7.
60. Frencken JE, Peters MC, Manton DJ, Leal SC, Gordan VV, Eden E. Minimal intervention dentistry for managing dental caries – A review: Report of a FDI task group. Int Dent J 2012;62:223-43.
61. Lynch RJ, Smith SR. Remineralization agents – New and effective or just marketing hype? Adv Dent Res 2012;24:63-7.
62. Robertson MA, Kau CH, English JD, Lee RP, Powers J, Nguyen JT. MI paste plus to prevent demineralization in orthodontic patients: A prospective randomized controlled trial. Am J Orthod Dentofacial Orthop 2011;140:660-8.
63. Uysal T, Amasyali M, Koyuturk AE, Ozcan S. Effects of different topical agents on enamel demineralization around orthodontic brackets: An *in vivo* and *in vitro* study. Aust Dent J 2010;55:268-74.
64. Wen-Dan H, Ying-Zhi L, Yuan-Yuan X, Dong C. Study on application of CPP-ACP on tooth mineralization during orthodontic treatment with fixed appliance. Shanghai J Stomatol 2010;19:140-3.
65. Beerens MW, van der Veen MH, van Beek H, ten Cate JM. Effects of casein phosphopeptide amorphous calcium fluoride phosphate paste on white spot lesions and dental plaque after orthodontic treatment: A 3-month follow-up. Eur J Oral Sci 2010;118:610-7.
66. Bröchner A, Christensen C, Kristensen B, Tranæus S, Karlsson L, Sonnesen L, *et al.* Treatment of post-orthodontic white spot lesions with casein phosphopeptide-stabilised amorphous calcium phosphate. Clin Oral Investig 2011;15:369-73.
67. Pithon MM, Dos Santos MJ, Andrade CS, Leão Filho JC, Braz AK, de Araujo RE, *et al.* Effectiveness of varnish with CPP-ACP in prevention of caries lesions around orthodontic brackets: An OCT evaluation. Eur J Orthod 2015;37:177-82.
68. Andersson A, Sköld-Larsson K, Hallgren A, Petersson LG, Twetman S. Effect of a dental cream containing amorphous cream phosphate complexes on white spot lesion regression assessed by laser fluorescence. Oral Health Prev Dent 2007;5:229-33.
69. Ellwood R, Fejerskov O, Cury J, Clarkson B, Kidd E. Dental Caries: The Disease and Its Clinical Management. 2nd ed. Oxford; Ames, Iowa: Blackwell and Munksgaard Oxford; 2008.
70. Keçik D, Cehreli SB, Sar C, Unver B. Effect of acidulated phosphate fluoride and casein phosphopeptide-amorphous calcium phosphate application on shear bond strength of orthodontic brackets. Angle Orthod 2008;78:129-33.
71. Uysal T, Amasyali M, Ozcan S, Koyuturk AE, Akyol M, Sagdic D. *In vivo* effects of amorphous calcium phosphate-containing orthodontic composite on enamel demineralization around orthodontic brackets. Aust Dent J 2010;55:285-91.
72. Lopatiene K, Borisovaite M, Lapenaite E. Prevention and treatment of white spot lesions during and after treatment with fixed orthodontic appliances: A systematic literature review. J Oral Maxillofac Res 2016;7:e1.
73. Twetman S, Keller MK. Probiotics for caries prevention and control. Adv Dent Res 2012;24:98-102.
74. Fontana M, González-Cabezas C. Are we ready for definitive clinical guidelines on xylitol/polyol use? Adv Dent Res 2012;24:123-8.
75. Milgrom P, Söderling EM, Nelson S, Chi DL, Nakai Y. Clinical evidence for polyol efficacy. Adv Dent Res 2012;24:112-6.
76. Pickett FA. Nonfluoride caries-preventive agents: New guidelines. J Contemp Dent Pract 2011;12:469-74.
77. Sengun A, Sari Z, Ramoglu SI, Malkoç S, Duran I. Evaluation of the dental plaque pH recovery effect of a xylitol lozenge on patients with fixed orthodontic appliances. Angle Orthod 2004;74:240-4.
78. Jenatschke F, Elsenberger E, Welte HD, Schlagenhauf U. Influence of repeated chlorhexidine varnish applications on mutans streptococci counts and caries increment in patients treated with fixed orthodontic appliances. J Orofac Orthop 2001;62:36-45.
79. Kronenberg O, Lussi A, Ruf S. Preventive effect of ozone on the development of white spot lesions during multibracket appliance therapy. Angle Orthod 2009;79:64-9.
80. Øgaard B, Larsson E, Henriksson T, Birkhed D, Bishara SE. Effects of combined application of antimicrobial and fluoride varnishes in orthodontic patients. Am J Orthod Dentofacial Orthop 2001;120:28-35.
81. Karandish M. The efficiency of laser application on the enamel surface: A systematic review. J Lasers Med Sci 2014;5:108-14.
82. Olivi G, Genovese MD, Caprioglio C. Evidence-based dentistry on laser paediatric dentistry: Review and outlook. Eur J Paediatr Dent 2009;10:29-40.
83. Verma SK, Maheshwari S, Singh RK, Chaudhari PK. Laser in dentistry: An innovative tool in modern dental practice. Natl J Maxillofac Surg 2012;3:124-32.
84. Fox JL, Yu D, Otsuka M, Higuchi WI, Wong J, Powell GL. Initial dissolution rate studies on dental enamel after CO2 laser irradiation. J Dent Res 1992;71:1389-98.
85. Noel L, Rebellato J, Sheats RD. The effect of argon laser irradiation on demineralization resistance of human enamel adjacent to orthodontic brackets: An *in vitro* study. Angle Orthod 2003;73:249-58.
86. Meeran NA. Iatrogenic possibilities of orthodontic treatment and modalities of prevention. J Orthod Sci 2013;2:73-86.
87. Ahrari F, Poosti M, Motahari P. Enamel resistance to demineralization following Er:YAG laser etching for bonding orthodontic brackets. Dent Res J (Isfahan) 2012;9:472-7.
88. Esteves-Oliveira M, Pasaporti C, Heussen N, Eduardo CP, Lampert F, Apel C. Rehardening of acid-softened enamel and prevention of enamel softening through CO2 laser irradiation. J Dent 2011;39:414-21.
89. Liu JF, Liu Y, Stephen HC. Optimal Er:YAG laser energy for preventing enamel demineralization. J Dent 2006;34:62-6.
90. Rodrigues LK, Nobre dos Santos M, Pereira D, Assaf AV, Pardi V. Carbon dioxide laser in dental caries prevention. J Dent 2004;32:531-40.
91. Steiner-Oliveira C, Nobre-dos-Santos M, Zero DT, Eckert G, Hara AT. Effect of a pulsed CO2 laser and fluoride on the prevention of enamel and dentine erosion. Arch Oral Biol 2010;55:127-33.
92. Guzmán-Armstrong S, Chalmers J, Warren JJ. Ask us. White spot lesions: Prevention and treatment. Am J Orthod Dentofacial Orthop 2010;138:690-6.
93. Benson PE, Shah AA, Millett DT, Dyer F, Parkin N, Vine RS. Fluorides, orthodontics and demineralization: A systematic review. J Orthod 2005;32:102-14.
94. Mota SM, Enoki C, Ito IY, Elias AM, Matsumoto MA. Streptococcus mutans counts in plaque adjacent to orthodontic brackets bonded with resin-modified glass ionomer cement or resin-based composite. Braz Oral Res 2008;22:55-60.

95. Denis M, Atlan A, Vennat E, Tirlet G, Attal JP. White defects on enamel: Diagnosis and anatomopathology: Two essential factors for proper treatment (part 1). Int Orthod 2013;11:139-65.
96. Sonesson M, Bergstrand F, Gizani S, Twetman S. Management of post-orthodontic white spot lesions: An updated systematic review. Eur J Orthod 2016. pii: Cjw023.
97. Senestraro SV, Crowe JJ, Wang M, Vo A, Huang G, Ferracane J, *et al.* Minimally invasive resin infiltration of arrested white-spot lesions: A randomized clinical trial. J Am Dent Assoc 2013;144:997-1005.
98. Kim Y, Son HH, Yi K, Ahn JS, Chang J. Bleaching effects on color, chemical, and mechanical properties of white spot lesions. Oper Dent 2016;41:318-26.
99. Khoroushi M, Mazaheri H, Saneie T, Samimi P. Fracture toughness of bleached enamel: Effect of applying three different nanobiomaterials by nanoindentation test. Contemp Clin Dent 2016;7:209-15.
100. Khoroushi M, Shirban F, Doustfateme S, Kaveh S. Effect of three nanobiomaterials on the surface roughness of bleached enamel. Contemp Clin Dent 2015;6:466-70.
101. Yetkiner E, Wegehaupt F, Wiegand A, Attin R, Attin T. Colour improvement and stability of white spot lesions following infiltration, micro-abrasion, or fluoride treatments *in vitro*. Eur J Orthod 2014;36:595-602.
102. Sundfeld RH, Croll TP, Briso AL, de Alexandre RS, Sundfeld Neto D. Considerations about enamel microabrasion after 18 years. Am J Dent 2007;20:67-72.
103. Kim S, Kim EY, Jeong TS, Kim JW. The evaluation of resin infiltration for masking labial enamel white spot lesions. Int J Paediatr Dent 2011;21:241-8.
104. Eckstein A, Helms HJ, Knösel M. Camouflage effects following resin infiltration of postorthodontic white-spot lesions *in vivo*: One-year follow-up. Angle Orthod 2015;85:374-80.
105. Knösel M, Eckstein A, Helms HJ. Durability of esthetic improvement following Icon resin infiltration of multibracket-induced white spot lesions compared with no therapy over 6 months: A single-center, split-mouth, randomized clinical trial. Am J Orthod Dentofacial Orthop 2013;144:86-96.
106. Tirlet G, Chabouis HF, Attal JP. Infiltration, a new therapy for masking enamel white spots: A 19-month follow-up case series. Eur J Esthet Dent 2013;8:180-90.