European Journal of Orthodontics 29 (2007) 294–298
doi:10.1093/ejo/cjm008
Advance Access publication 5 May 2007

Caries lesions after orthodontic treatment followed by quantitative light-induced fluorescence: a 2-year follow-up

T. J. H. Mattousch*, M. H. van der Veen*,** and A. Zentner*
*Department of Orthodontics, Academic Centre for Dentistry Amsterdam and **Inspektor Research Systems BV, Amsterdam, The Netherlands

SUMMARY The aim of this study was to monitor, by means of quantitative light-induced fluorescence (QLF), the natural behaviour of white spot lesions detected immediately after orthodontic treatment and 2 years post-treatment. The buccal tooth surfaces of 51 subjects (≥12 years), 24 males and 27 females, were examined with QLF for the presence of caries immediately after debonding (T0), and 6 weeks (T1), 6 months (T2), and 2 years (T3) thereafter. The fluorescence loss [ΔF (per cent)] and area [A (mm^2)] of any lesions were determined using dedicated software. The lesion development and influence of gender were determined by a general linear model (Friedman repeated-measures analysis and two-way repeated-measures analysis of variance).

Using QLF, 370 carious surfaces were recorded at T0. During the study, 19 lesions were lost from QLF analysis of which 16 lesions (ΔF_0 = 7.6–39.2 per cent) in two subjects were restored and three teeth with lesions were extracted or crowned. This resulted in 351 lesions that were included in this study with a median ΔF at T0 of 8.5 per cent (quartiles 6.6 per cent; 11.9 per cent). The lesions varied from incipient (ΔF < 10 per cent, n = 227) to advanced (ΔF > 25 per cent, n = 6). Overall, the lesions showed improvement between T0 and T2 (P < 0.01) but no further significant improvement at T3. Thirty-five lesions became significantly worse after 2 years. The majority of lesions (n = 171) were considered to be stable, and 145 lesions improved significantly of which only 10 lesions improved to such an extent that they disappeared.

White spot lesions developed during orthodontic treatment have very limited ability to improve after appliance removal. Further research to investigate the potential of preventive measures to enhance lesion improvement is necessary.

Introduction

White spot lesions remain a serious problem in orthodontics (Gorelick *et al.*, 1982; Øgaard *et al.*, 1988; Boersma *et al.*, 2005). In the literature, reports of long-term monitoring of white spots are limited, as most research stops in the first year after debonding (Al-Khateeb *et al.*, 1998). The general belief that these lesions disappear after removal of the fixed appliances is controversial, and in many cases these lesions remain visible as a permanent enamel scar. Therefore, the use of extra preventive measures has been advocated (Benson *et al.*, 2005). For a better understanding of whether and which preventive measures will be most efficacious, knowledge is required about the beginning and natural behaviour of these orthodontic-related white spots.

The reported prevalence of white spots after fixed appliance treatment varies between 2 and 96 per cent (Gorelick *et al.*, 1982; Mitchell, 1992). This large variation might be a result of the difficulty in standardizing clinical examinations, the variety of detection methods, or the presence of white spots before the start of orthodontic treatment (Kanthathas *et al.*, 2005). White spot formation during orthodontic treatment has been attributed to the effect of prolonged accumulation and retention of bacterial plaque. Fixed appliances make conventional oral hygiene for plaque removal more difficult, and adjacent to the brackets the clearance of plaque by saliva and cheeks is also reduced. There seems to be a difference in progression rate between traditional caries formation and white spot lesions induced by deficient oral hygiene combined with fixed orthodontic appliances (Øgaard and ten Bosch, 1994). The latter has a rather superficial and more rapid character and can become apparent within 1 month after placement of fixed appliances. The formation of a 'normal' caries lesion is usually a slower process, which takes at least 6 months (Ekanayake and Sheiham, 1987). There have been only a few studies (Øgaard and ten Bosch, 1994; Al-Khateeb *et al.*, 1998) related to the possibility of improvement or even 'cure' of white spots after debonding, with or without extra measures.

When a tooth becomes carious, the fluorescence radiance at the location of the caries lesion decreases. The fluorescence image of enamel with incipient lesions can be digitized and the fluorescence loss in the lesion can then be quantified in relation to the fluorescence radiance level of sound enamel (Bjelkhagen *et al.*, 1982; de Josselin de Jong *et al.*, 1995). The amount of fluorescence radiance loss has been validated by the use of transverse and longitudinal micro-radiography

and is very closely (r = 0.97) correlated with the mineral loss in the lesion (Hafström-Björkman *et al.*, 1992; Emami *et al.*, 1996; Ando *et al.*, 2001). Recent studies indicate that quantitative light-induced fluorescence (QLF) is suitable for *in vivo* monitoring of mineral changes in incipient enamel lesions (van der Veen and de Josselin de Jong, 2000; Al-Khateeb *et al.*, 2002). Furthermore, the use of QLF as a method of following caries development during orthodontic treatment has been suggested and encouraged by the results of several *in vitro* studies (Benson *et al.*, 2003a,b; Pretty *et al.*, 2003).

The aim of the present investigation was to monitor, by means of QLF, the natural behaviour of white spot lesions detected directly after fixed appliance treatment as well as 6 weeks, 6 months, and 2 years later.

Subjects and methods

Approval of the Medical Ethical Committee of the Academic Medical Centre of the University of Amsterdam was obtained (MEC01/099#01.17.594) for this study. The participants were recruited consecutively from patients treated with a fixed appliance at the Department of Orthodontics at the Academic Centre for Dentistry Amsterdam. The inclusion criteria were age 12 years or older, good general health, a treatment period with fixed appliances of at least 1 year at the debonding appointment, and an informed consent signed by the participant and for those under 18 years, signed in addition by their parents or guardians. The participants' dentists were informed that their patients were participating in the study and asked not to administer extra fluoride to these subjects while they were participating and to contact the study investigator in case restorations were required.

No special attention was given to oral hygiene measures for subjects participating in this study immediately after debonding or at the retention visits. At the start of fixed appliance treatment, the patients were advised to brush their teeth three times a day, were shown how to clean their mouth with the appliance *in situ*, and were given a leaflet with brushing instructions.

The presence and extent of lesions on the buccal surfaces of all teeth, except second and third molars, were assessed by means of QLF at the debonding visit (T0) and at three retention visits scheduled for 6 weeks (T1), 6 months (T2), and 2 years (T3) after debonding. Thus, a maximum of 24 buccal surfaces per subject were assessed at each time point. No special measures were taken to remove plaque from the surfaces, apart from normal cleaning as part of the debonding procedure. The examination intervals were according to the retention protocol of the department.

QLF images were captured using an intraoral fluorescence camera (Inspektor Research Systems BV, Amsterdam, The Netherlands) on a personal computer using the image capturing software (QLF Patient version 3.0.0.4) delivered with the system. To ensure that the images of the tooth surfaces are always captured with the same camera position and from the same angle, the software uses video-repositioning techniques. The video-repositioning technique displays the baseline and live image simultaneously and computes their correlation based on similar geometry of the fluorescence intensities (Buchalla *et al.*, 2001). Images are stored in a list when the correlation is higher than 0.90 and the system automatically stops 'grabbing' when the correlation reaches 0.98. In this way, images captured at T0, T1, T2, and T3 should show the tooth surface from the same angle and at the same magnification, except for other changes such as, for instance, differences in gingival swelling.

QLF images were examined visually for signs of demineralization, which appear as dark areas surrounded by bright green fluorescing sound tooth tissue (de Josselin de Jong *et al.*, 1995). The presence or absence of lesions was scored for each surface base for each patient. If lesions were detected, the fluorescence loss and lesion area were determined using the system's analysis software to determine the lesion extent (de Josselin de Jong *et al.*, 1995). For this purpose, a patch was drawn surrounding the lesion site with its borders on sound enamel. Inside this patch, the fluorescence levels of sound tissue were reconstructed using the fluorescence radiance of the surrounding sound enamel. Subsequently, the percentage difference between the reconstructed and original fluorescence levels was calculated. Pixels inside the patch were considered part of the lesion when the relative fluorescence loss exceeded the 5 per cent threshold (Al-Khateeb *et al.*, 2002). To ensure that the same area of a tooth surface was analysed at each time point, the analysis patch and surface contour were copied and then matched for size, orientation, and location to this tooth surface in all consecutive images.

The fluorescence loss and corresponding lesion area were obtained for each lesion at four time points. The total fluorescence loss and corresponding lesion area were calculated for each subject and then normalized to 24 surfaces by correcting for the number of missing and filled surfaces. Lesion development, such as progression or regression for the whole group of subjects, was determined using Friedman repeated-measures analysis of variance (ANOVA) on ranks. To determine change in individual lesions, first the relative standard deviation (SD) was calculated, after which a change equivalent to 2SD was considered as significant. This was carried out both for fluorescence loss (ΔF in per cent) and for area (A in mm^2). In addition, the potential influence of gender was examined using two-way repeated-measures ANOVA followed by all pairwise comparison procedures according to the Holm–Sidak method (Lautenberger *et al.*, 2000).

Results

From the total of 64 participants recruited into the study, 13 dropped out between T0 and T3. Fifty-one participants (age 12–56 years, median 16.0 years), of whom 24 were male and 27 female, completed the study.

As assessed by QLF, the buccal surfaces of two female subjects were considered entirely caries free. In the remaining 49 subjects, 370 carious surfaces were recorded immediately after debonding. During the study, 19 lesions were lost from analysis: 16 lesions in two subjects, one male and one female, were restored and three lesions were not analysed because of tooth extraction or crown restoration. Thus, a total of 351 lesions were followed over the time of this investigation (Table 1). These lesions had a median fluorescence loss at debonding (ΔF_0) of 8.5 per cent (quartiles 6.6 per cent; 11.9 per cent, threshold 5 per cent), varying from incipient ($\Delta F_0 < 10$ per cent, $n = 227$) to advanced ($\Delta F_0 > 25$ per cent, $n = 6$).

Overall, on a subject level, lesion improvement became significant at T2 (Table 1; $P_{\Delta F_0 \rightarrow \Delta F_2} = 0.014$, $P_{\Delta F_1 \rightarrow \Delta F_2} = 0.043$). No differences were found between debonding and T1 ($P = 0.063$) or between T2 and T3 ($P = 0.25$). The median value for total fluorescence loss per subject, corrected for the number of missing and filled surfaces, was 55.7 per cent (quartiles 29.4 per cent; 101.5 per cent) at T0, which changed to 51.4 per cent (quartiles 26.7 per cent; 101.5 per cent) at T1, 48.3 per cent (quartiles 25.9 per cent; 97.4 per cent) at T2, and 43.9 per cent (quartiles 22.4 per cent; 99.2 per cent) at T3. The lesion areas showed a similar, however, less strongly pronounced pattern (Table 2). A significant difference was observed between T2 and T0 ($P = 0.045$) as well as between T3 and T0 ($P = 0.015$) or T1 ($P = 0.021$); yet, no differences were found between any of the consecutive visits. The median value for total lesion area per subject, corrected for the number of missing and filled surfaces, was on average 3.5 mm² (quartiles 1.0 mm²; 8.1 mm²) at T0, which changed to 2.5 mm² (quartiles 0.7 mm²; 6.4 mm²) at T1, 2.2 mm² (quartiles 0.6 mm²; 6.1 mm²) at T2, and 1.7 mm² (quartiles 0.4 mm²; 5.7 mm²) at T3.

Despite the statistically significant difference between the numbers of lesions in males (24) and females (27) found with QLF at all four time points, no difference between genders was observed with regard to lesion behaviour during the 2-year retention phase. When the data from the individual lesions were examined over time with respect to fluorescence loss (Table 3), 35 lesions were identified as significantly worse after 2 years. The majority of lesions ($n = 171$) were considered to be stable, and 145 lesions improved significantly of which only 10 lesions improved to such an extent that they were no longer detectable. Regressing and progressing lesions were found in the same subjects.

Discussion

This study showed that the generally expected improvement of white spot lesions after the removal of fixed appliances and restoration of oral hygiene does not apply. Sixteen lesions were so severe that they required restoration. Only 10 of the 370 lesions remineralized completely after 2 years, while the majority of lesions found after orthodontic treatment were considered stable after 2 years of retention. These findings are in contrast with the general belief that lesions regress once the appliance is removed and oral hygiene is restored. The overall lesion improvement seen in the current study was

Table 1 Median sum of fluorescence loss (ΔF) per subject and median ΔF per lesion.

	T0 = debonding			T1 = 6 weeks retention			T2 = 6 months retention			T3 = 2 years retention		
	ΔF (%)	25%	75%	ΔF (%)	25%	75%	ΔF (%)	25%	75%	ΔF (%)	25%	75%
51 subjects	55.7[a]	29.4	101.5	51.4[a]	26.7	101.5	48.3[b]	25.9	97.4	43.9[b]	22.4	99.2
351 lesions	8.5	6.6	11.9	8.0	6.4	10.8	7.6	6.1	10.9	7.0	5.7	10.7

Superscript characters indicate the statistical differences between the times. $P < 0.05$ was considered statistically significant.

Table 2 Median sum of lesion area (A) per subject and median area per lesion.

	T0 = debonding			T1 = 6 weeks retention			T2 = 6 months retention			T3 = 2 years retention		
	A (mm²)	25%	75%	A (mm²)	25%	75%	A (mm²)	25%	75%	A (mm²)	25%	75%
51 subjects	3.5[a]	1.0	8.1	2.5[a,b]	0.7	6.4	2.2[b,c]	0.6	6.1	1.7[c]	0.4	5.7
351 lesions	0.4	0.1	1.1	0.3	0.1	0.9	0.3	0.1	0.9	0.2	0.0	0.8

Superscript characters indicate the statistical differences between the times. $P < 0.05$ was considered statistically significant.

Table 3 The behaviour of lesions categorized by fluorescence loss (ΔF) and area (A) at the four measurement points: 0 = at debonding, 1 = 6 weeks retention, 2 = 6 months retention, and 3 = 2 years of retention.

Lesions	$\Delta F_0 \rightarrow \Delta F_3$	$\Delta F_0 \rightarrow \Delta F_1$	$\Delta F_2 \rightarrow \Delta F_3$	$A_0 \rightarrow A_3$	$A_0 \rightarrow A_1$	$A_2 \rightarrow A_3$
Worsened	35	47	39	27	21	19
Improved	145	75	88	71	33	40
Stable	171	229	224	250	297	292
Restored	19	3	11	19	3	11

accounted for by only two-fifths of the lesions. This finding and the loss of 16 lesions from the study because of restorative treatment make clear that removal of plaque stagnation sites by appliance removal alone is insufficient for adequate lesion remineralization.

The magnitude of changes in mineralization was greatest in the first 6 months after debonding. White spots still visible after this time did not disappear in the remaining retention period.

The patients in this study did not receive extra prevention measures either before, during, or after orthodontic treatment, except routine oral hygiene instruction and use of fluoride toothpaste. Despite the presence of white spot lesions at debonding, the subjects were not given extra fluoride as it has been suggested that high doses of fluoride used on porous white spot lesions affect only the outermost surface, thereby inhibiting complete remineralization of lesions (Øgaard *et al.*, 1988; Willmot, 2004). Recent evidence suggests that the use of extra fluorides during fixed appliance treatment reduces the occurrence and severity of white spot lesions (Øgaard *et al.*, 1988; Benson *et al.*, 2004; Derks *et al.*, 2004). Nevertheless, prevention of white spot lesions is better than repairing lesions once they exist. Therefore, research and orthodontic treatment have focussed mainly on prevention of white spots, for example by starting fixed appliance treatment only in plaque-free patients, providing a strict oral hygiene protocol during treatment, and discontinuing orthodontic treatment as soon as incipient white spot lesions become visible. Despite these preventative measures, there will be patients with unsightly white spot lesions after debonding presenting a challenge for restorative treatment. It seems, for instance, that low doses of fluoride in mouth rinses after debonding do not improve these lesions (Willmot, 2004). Allowing remineralization by saliva and, if necessary, the use of acid microabrasion 10 weeks after debonding is suggested by Bishara *et al.* (1987) and Welbury and Carter (1993). The approach of natural lesion improvement in the present study proved unsuccessful.

Conclusions

Many patients develop white spots during fixed appliance treatment. This study shows that these lesions do not disappear after debonding. Although two-fifths of the lesions showed some improvement, the majority of lesions were considered to be stable, while 15 per cent had worsened after 2 years of retention.

Given the high number of lesions found at debonding in this study population, investigations focussing on remineralizing strategies for these types of lesions are necessary together with ongoing research to find more efficacious prevention treatments that can be used during fixed appliance treatment.

Address for correspondence

Monique H. van der Veen
Department of Cariology, Endodontology, Pedodontology
Academic Centre for Dentistry Amsterdam
Louwesweg 1
1066 EA Amsterdam
The Netherlands
E-mail: m.vd.veen@acta.nl

References

Al-Khateeb S, Forsberg C M, de Josselin de Jong E, Angmar-Månsson B 1998 A longitudinal laser fluorescence study of white spot lesions in orthodontic patients. American Journal of Orthodontics and Dentofacial Orthopedics 113: 595–602

Al-Khateeb S, Exterkate R A M, Angmar-Månsson B, ten Cate J M 2002 Light-induced fluorescence studies on dehydration of incipient enamel lesions. Caries Research 36: 25–30

Ando M, van der Veen M H, Schemehorn B R, Stookey G K 2001 Comparative study to quantify demineralized enamel in deciduous and permanent teeth using laser- and light-induced fluorescence techniques. Caries Research 35: 464–470

Benson P E, Pender N, Higham S M 2003a Quantifying enamel demineralization from teeth with orthodontic brackets—a comparison of two methods. Part 1: repeatability and agreement. European Journal of Orthodontics 25: 149–158

Benson P E, Pender N, Higham S M 2003b Quantifying enamel demineralization from teeth with orthodontic brackets—a comparison of two methods. Part 2: validity. European Journal of Orthodontics 25: 159–165

Benson P E, Parkin N, Millett D T, Dyer F E, Vine S, Shah A 2004 Fluorides for the prevention of white spots on teeth during fixed brace treatment. Cochrane Database Systematic Review CD003809

Benson P E, Shah A A, Millett D T, Dyer F, Parkin N, Vine R S 2005 Fluorides, orthodontics and demineralization: a systematic review. Journal of Orthodontics 32: 102–114

Bishara S E, Denehy G E, Goepferd S J 1987 A conservative postorthodontic treatment of enamel stains. American Journal of Orthodontics and Dentofacial Orthopedics 92: 2–7

Bjelkhagen H, Sundström F, Angmar-Månsson B, Ryden H 1982 Early detection of enamel caries by the luminescence excited by visible laser light. Swedish Dental Journal 6: 1–7

Boersma J G, van der Veen M H, Lagerweij M D, Bokhout B, Prahl-Andersen B 2005 Caries prevalence measured with QLF after treatment with fixed orthodontic appliances: influencing factors. Caries Research 39: 41–47

Buchalla W, de Josselin de Jong E, Ando M, Eggertsson H, Lennon A, Stookey G K 2001 Video-repositioning—increased repeatability for QLF caries monitoring method. Journal of Dental Research 80: 115 (abstract)

de Josselin de Jong E, Sundström F, Westerling H, Tranaeus S, ten Bosch J J, Angmar-Mansson B 1995 A new method for *in vivo* quantification of changes in initial enamel caries with laser fluorescence. Caries Research 29: 2–7

Derks A, Katsaros C, Frencken J E, van't Hof M A, Kuijpers-Jagtman A M 2004 Caries-inhibiting effect of preventive measures during orthodontic treatment with fixed appliances. A systematic review. Caries Research 38: 413–420

Ekanayake L S, Sheiham A 1987 Reducing rates of progression of dental caries in British schoolchildren. A study using bitewing radiographs. British Dental Journal 163: 265–269

Emami Z, Al-Khateeb S, de Josselin de Jong E, Sundström F, Trollsas K, Angmar-Mansson B 1996 Mineral loss in incipient caries lesions quantified with laser fluorescence and longitudinal micro-radiography. A methodologic study. Acta Odontologica Scandinavica 54: 8–13

Gorelick L, Geiger A M, Gwinnett A J 1982 Incidence of white spot formation after bonding and banding. American Journal of Orthodontics 81: 93–98

Hafström-Björkman U, Sundström F, de Josselin de Jong E, Oliveby A, Angmar-Mansson B 1992 Comparison of laser fluorescence and longitudinal microradiography for quantitative assessment of *in vitro* enamel caries. Caries Research 26: 241–247

Kanthathas K, Willmot D R, Benson P E 2005 Differentiation of developmental and post-orthodontic white lesions using image analysis. European Journal of Orthodontics 27: 167–172

Lautenberger J A, Stephens J C, O'Brien S J, Smith M W 2000 Significant admixture linkage disequilibrium across 30 cM around the FY locus in African Americans. American Journal of Human Genetics 66: 969–978

Mitchell L 1992 Decalcification during orthodontic treatment with fixed appliances—an overview. British Journal of Orthodontics 19: 199–205

Øgaard B, ten Bosch J J 1994 Regression of white spot enamel lesions: a new optical method for quantitative longitudinal evaluation *in vivo*. American Journal of Orthodontics and Dentofacial Orthopedics 106: 238–242

Øgaard B, Rölla G, Arends J 1988 Orthodontic appliances and enamel demineralization. Part 1: lesion development. American Journal of Orthodontics and Dentofacial Orthopedics 94: 68–73

Pretty I A, Pender N, Edgar W M, Higham S M 2003 The *in vitro* detection of early enamel de- and re-mineralization adjacent to bonded orthodontic cleats using quantitative light-induced fluorescence. European Journal of Orthodontics 25: 217–223

van der Veen M H, de Josselin de Jong E 2000 Application of quantitative light-induced fluorescence for assessing early caries lesions. Monographs in Oral Science 17: 144–162

Welbury R R, Carter N E 1993 The hydrochloric acid-pumice microabrasion technique in the treatment of post-orthodontic decalcification. British Journal of Orthodontics 20: 181–185

Willmot D R 2004 White lesions after orthodontic treatment: does low fluoride make a difference?. Journal of Orthodontics 31: 235–242; discussion 202