CLINICAL INVESTIGATIONS

Gap Size and Wall Lesion Development Next to Composite

N.K. Kuper^{1*}, N.J.M. Opdam¹, J.L. Ruben¹, J.J. de Soet², M.S. Cenci³, E.M. Bronkhorst¹, and M.C.D.N.J.M. Huysmans¹

Abstract: *This in situ study investigated whether there is a relationship between gap size and wall lesion development in dentin next to 2 composite materials, and whether a clinically relevant threshold for the gap size could be established. For 21 days, 14 volunteers wore a modified occlusal splint containing human dentin samples with 5 different interfaces: 4 gaps of 50 μ m, 100 μ m, 200 μ m, or 400 μ m and 1 non-bonded interface without a gap. Eight times a day, the splint with samples was dipped in a 20% sucrose solution for 10 minutes. Before and after caries development, specimens were imaged with transversal wavelength-independent microradiography (T-WIM), and lesion depth (LD) and mineral loss (ML) were calculated at the 5 different interfaces. After correction for the confounder location (more mesial or distal), a paired t test clustered within volunteers was performed for comparison of gap widths. Results showed no trend for a relationship between the corrected lesion depth and the gap size. None of the differences in lesion depth for the different gap sizes was statistically significant. Also, the composite material (AP-X or*

Filtek Supreme) gave no statistically significant differences in lesion depth and mineral loss. A minimum gap size could not be established, although, in a non-bonded interface without a measurable gap, wall lesion development was never observed.

Key Words: secondary caries, recurrent caries, lesion depth, mineral loss, Transversal Wavelength Independent Microradiography, composite resin restorations.

Introduction

Secondary caries is a frequently encountered problem in dental practice and has been widely recognized to be one of the most important reasons for restoration replacement (Mjör, 1997). Secondary caries is defined as a new caries lesion at the margins of an existing restoration (Mjör and Toffenetti, 2000). These lesions have 2 parts: an outer lesion, which is histologically similar to a primary lesion and formed on the outer surface of the tooth next to the restoration; and a wall lesion, which is a lesion that develops at the interface between the restoration and the tooth

(Hals *et al.*, 1974). One of the possible factors in the development of secondary caries is the existence of a gap or defect at the cavity wall. With this considered a weak point, the presence of defective margins leads to many clinical decisions to repair or replace restorations (Gordan *et al.*, 2009), even where caries has not yet developed.

There are 2 different theories for explaining a possible relationship between gaps and wall lesion development. First, in what we shall call the 'microleakage theory', leakage of bacterial acids into a small gap is considered to be sufficient to cause demineralization and a wall lesion. According to this theory, a wall lesion can develop in any gap, but the wider the gap, the higher the risk. Second, recent changes in the theory of cariology put more emphasis on the importance of the biofilm in driving the caries process in gaps. Clinical and microbiological studies have indicated that microleakage alone does not lead to active demineralization beneath a restoration (Kidd and Fejerskov, 2004). Only where a biofilm can establish itself along the tooth-restoration interface wall lesions may develop. The width of the

DOI: 10.1177/0022034514534262. ¹College of Dental Sciences, Department of Preventive and Restorative Dentistry, Radboud University Medical Center, the Netherlands;

²Department of Preventive Dentistry, Academic Centre for Dentistry Amsterdam (ACTA), University of Amsterdam and VU University, Amsterdam, the Netherlands; and

³Federal University of Pelotas, School of Dentistry, Gonçalves Chaves, 457, 5th floor, Pelotas, 96015560, Brazil; *corresponding author, Nicolien.Kuper@radboudumc.nl

A supplemental appendix to this article is published electronically only at <http://jdr.sagepub.com/supplemental>.

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gap needed for such a cariogenic biofilm has been roughly estimated to be 400 micrometers (Kidd *et al.*, 1995) or 225 micrometers (Thomas *et al.*, 2007a).

The literature provides no conclusive answer to the question whether a relationship between gap size and wall lesion development exists and, if so, the minimum gap size needed. Results of non-bacterial *in vitro*, bacterial *in vitro*, and clinical (*in situ*) studies are conflicting. Nassar and González-Cabezas (2011), in their sucrose cycling microbial caries model, showed that in uniform gaps, the size of the gap was positively correlated with the size of dentinal wall lesions, but they gave no threshold for the size of the gap. Diercke *et al.* (2009), in their bacterial-based *in vitro* model, found a statistically significant increase in lesion depth in enamel between a 50- μ m gap and a 250- μ m gap and in dentin between a 50- μ m and a 100- μ m gap. Totiam *et al.* (2007) showed *in vitro* a trend toward bigger wall lesions associated with larger gaps in both enamel and dentin, but did not establish a threshold for gap size. Finally, the clinical *in situ* study by Thomas *et al.* (2007a) gave an average gap size of 225 μ m (range, 80-560 μ m) needed for the development of wall lesions.

Secondary caries lesion development has been observed to be more of a problem for composite restorations than for amalgam restorations (Opdam *et al.*, 2010; Kuper *et al.*, 2012). This has been suggested as the main reason for the reduced life expectancy of composite (National Institutes of Health, 2009). Composite restorative materials, however, may differ in their relative susceptibility to secondary caries lesion development. In this study, therefore, we evaluated 2 composite materials.

The aim of this *in situ* study was to investigate whether there is a relationship between gap size and wall lesion development next to 2 composite materials, and whether a clinically relevant threshold for the gap size can be established. The null hypothesis tested was that neither the gap size nor the composite material would influence

the lesion depth or mineral loss of wall lesion development.

Materials & Methods

Study Participants

Fourteen volunteers (six men, eight women, aged 20-57 yr, mean age = 30.4 yr) were recruited within the Dental School in Nijmegen, the Netherlands. Exclusion criteria were active caries, periodontitis (DPSI > 2), ASA>2, or the wearing of a removable prosthetic appliance in the mandibular jaw.

The study design and protocol were approved by the local ethics committee, METC (CMO file nr. 2011/248, NL33528.01.11).

Preparation of Specimens

Sound human molars were ground flat until all the enamel was removed (Fig. 1). The roots were cut off, and the remaining crowns were perpendicularly cut into 56 dentin bars with a fixed width of 3.2 mm and various lengths. The dentin bars were manually ground with 400-grit paper (Siawat, Abrasives, Frauenfeld, Switzerland) to a height of 2.0 mm. The dentin bars were gas-sterilized with ethylene oxide (Isotron Nederland B.V., Venlo, the Netherlands) (Thomas *et al.*, 2007b).

For each sample, 2 dentin bars (occlusal surface downward) were placed in a rectangular putty mould with dimensions of 15 x 3.2 x 2.5 mm. On the pulpal side, a self-etching primer and bonding agent (SE Bond, Clearfil, Kuraray, Okayama, Japan) were applied according to the manufacturer's instructions, and 0.5 mm composite was applied, fixing the 2 dentin bars. For the purpose of the microradiographic method used, utmost care was taken to keep the bars perfectly straight with rectangular angles and to position the top surface of the dentin in such a way that when placed in the microradiography holder, it was parallel to the central ray of the x-ray beam.

In each composite-dentin bar, 5 slots were made parallel to the dentin tubuli with a 012 cylindrical bur with a depth of 1.9 mm (Fig. 1A). While the bar was fixed in a mould, 4 slots were filled with 1 of

the 2 chosen composites, Filtek Supreme XTE (A1 body shade, 3M ESPE, St. Paul, MN, USA) or AP-X PLT (A2, Clearfil, Kuraray, Okayama, Japan), without any adhesive procedures, and with matrices of different thicknesses (50, 100, 200, and 400 μ m) between the dentin and the composite. One slot was filled completely with composite (also not bonded) without any matrix. One interface of this slot was randomly chosen as the control interface without a gap (non-bonded, no gap interface = NG). The order of the different gap sizes was changed *per* volunteer, starting with the largest gap of 400 μ m at the most mesial position (order 400-200-100-50-NG) for the first volunteer and starting with the 200- μ m gap at the most mesial position (order 200-100-50-NG-400) for the second volunteer and so forth. The samples were polished to remove excess composite material, and the final rectangular composite-dentin bars with gaps had dimensions of 15 mm (length), 3.2 mm (width), and 2.2 mm (height).

Each volunteer received a modified occlusal splint for the mandibular jaw (Fig. 1C), with buccal flanges holding 4 embedded metal slots of 20 mm x 3.2 mm x 2.5 mm. Only the 2 lower slots were used for this study. The 2 composite materials were placed at the left or right side alternately *per* volunteer.

Experimental Protocol

The occlusal splints were worn for 3 wk for 24 hr a day, being removed only during eating, drinking, or oral hygiene, with the device kept in a physiologic salt solution. Volunteers were instructed to dip the device with the samples in a 20% sucrose solution 8 times a day for 10 min. They were asked to keep intervals between different sucrose dippings around an hour or more, but with a minimum of 30 min. They were given a diary to record the exact moments of sucrose-dipping. After being dipped in sucrose, the device was rinsed with tap water.

Volunteers were asked to apply some fluoride toothpaste/saliva slurry to the samples once a day (when they brushed their teeth). They were explicitly

Sample preparation and sample holder. **(A)** Schematic representation of sample preparation. **(B)** Microscopic view of gap samples, 10 $\times$ magnification. **(C)** Modified occlusal splint with gap samples *in situ*.

With a linear mixed-effects model, a first analysis was carried out to identify possible confounders that would prohibit comparison of effects within a patient. Included in this analysis were left/right effect, material effect (APX or Filtek Supreme composite), and effect of the

Figure 2.

T-WIM images of the dentin with composite and the different interfaces (gaps of 50, 100, 200, and 400 μm , and no gap = NG) at baseline **(A)** and after 21 days with caries development **(B)**. The area of measurement (red rectangle) starts at a 400- μm distance from the surface to prevent overlap with the surface lesion.

Table 1.

Gap Width and Lesion Depth *per* Group

Group	Gap Measurements (μm)			Uncorrected Lesion Depth (μm)		Corrected Lesion Depth (μm)	
	Minimum	Maximum	Mean	Mean	SD	Mean	SD
No gap	N.A.	N.A.	N.A.	2.4	5.7	<i>Not corrected</i>	
50 gap	68	202	121	74.4	45.7	47.2	47.0
100 gap	110	275	169	93.8	49.4	66.1	48.9
200 gap	166	312	257	89.3	58.2	58.5	52.2
400 gap	406	587	462	95.9	45.9	63.3	45.6

location of the gap. Where necessary, a correction for confounding factors was performed. Subsequently, gaps within patients were compared by paired *t* tests. A Bonferroni correction was applied to correct for multiple testing ($\alpha = 0.05/6 = 0.008$).

Results

Ten volunteers completed the study successfully in 3 wk. Two volunteers dropped out of the study too early for data analysis ($n = 1$) or lost their sample device ($n = 1$). Two volunteers completed only 2 wk, but since they did show caries development and effects were evaluated within patients, they were included in the analysis.

Gaps were, on average, 60 to 70 μm larger than intended (Table 1).

First analysis of the data with a linear mixed-effects model showed that there was no effect for left/right position ($p = .765$) or composite material ($p = .119$). However, the location of the gap was statistically significant, since the most mesial position showed significantly less demineralization than the other more distally located positions. Therefore, lesion depth values were corrected for location. The difference in lesion depth between the 2 most extreme locations (most mesial *vs.* most distal) was 45.5 μm ($p = .0001$). The changes in lesion depth with location can be seen in Appendix Table 1a (online). Corrected lesion depths are shown in Table 1, and corrected mineral loss is shown in Appendix Table 1b (online).

After correcting for this location effect, we performed paired *t* tests comparing

gaps within volunteers for all gap sizes (Table 2). Since the correlation between lesion depth and mineral loss was high ($r = 0.795$), only the analysis for lesion depth is shown in Table 2; results for mineral loss are shown in Appendix Table 2 (online). None of the differences in lesion depth and mineral loss for the different gap sizes was statistically significant.

Discussion

In this *in situ* study, the null hypothesis could not be rejected, since we could not find a relationship between gap size and wall lesion development; no clear trend for increasing lesion progression with wider gaps could be observed. Neither could a minimum gap size be established, although in non-bonded

Table 2.Paired *t* Test Results of Comparison of Lesion Depths among Gap-width Groups

Pair	Mean Difference* ($\mu\text{m} \pm \text{SD}$)	<i>p</i> value	95% Confidence Interval of the Difference	
			lower	upper
LD 50 - LD 100	-18.8 (43.3)	.044	-37.1	-0.6
LD 50 - LD 200	-11.3 (38.1)	.161	-27.3	4.8
LD 50 - LD 400	-12.0 (40.0)	.165	-29.3	5.3
LD 100 - LD 200	7.6 (41.5)	.380	-9.9	25.1
LD 100 - LD 400	4.5 (38.8)	.587	-12.3	21.2
LD 200 - LD 400	-0.7 (47.0)	.944	-21.0	19.6

*All lesion depth values are corrected for distance from the mesial location.

interfaces without measurable gaps, wall lesions never occurred.

In situ studies approximate clinical situation best, but unfortunately they cannot completely mimic the *in vivo* situation. An unexpected confounder in this *in situ* study was location effect. The most mesial location showed significantly less demineralization than the more distally located positions. We suspect that this was caused by reduced oral clearance and more plaque accumulation in the back of the buccal vestibule.

Also, compliance of volunteers was challenging. Many volunteers were not used to wearing an occlusal splint (increased height), and since this splint had to be worn 24 hr a day, speech and oral comfort were significantly compromised. One volunteer reported that she unconsciously removed the splint at night during her sleep. Other volunteers had difficulties fulfilling the strict schedule of '8 × 10 min' sucrose dipping each day, because of work or other obligations.

In all volunteers, wall lesions developed in 1 or more gaps, although the extent ranged from minimal to substantial, but surface lesions developed only in 4 AP-X samples and 5 Filtek supreme samples of the 12 volunteers. This shows that the wall lesions in this study developed independently of the outer surface and can be seen as an entity on their own. In the 'no gap' surfaces, wall lesions never developed at the interface between dentin and composite. Given the

relatively short period of time, and since we know that secondary caries in such a model can develop in 21 days, our results show that secondary caries is probably not caused by microleakage as long as there is no hydrodynamic flow due to restoration loading (Kuper *et al.*, 2013).

In the 'no gap' samples, outer lesions were sometimes seen to extend slightly deeper next to the composite. Such lesions were also observed in the *in situ* study by Barata *et al.* (2012), and we have observed them both *in vitro* (Kuper *et al.*, 2013; non-bonded samples) and *in situ* (Thomas *et al.*, 2007a; bonded samples). In our opinion, those lesions, while being directly associated with the tooth-composite interface, are not indisputable wall lesions, since they do not obviously progress from the interface (Hals *et al.*, 1974). Although there is a possibility that the curved lesion extensions are the result of microleakage (of acid) in the interface, the fact that such lesion shapes were also found next to bonded samples, where microleakage may be present but only to a very limited degree, points more toward an explanation by the enhanced diffusion of dissolution products at the edge of the tooth surface, accelerating demineralization locally. This is in agreement with the results of an earlier *in vitro* study wherein different windows were exposed with the use of tape, as opposed to the use of a restorative material with an interface (Ruben *et al.*, 1999).

Unfortunately, all gaps turned out to be wider than intended. The smallest gap measured was 68 μm , in which a lesion of 79.4 μm developed (not corrected for location). The 'gaps' of the non-bonded surfaces could not be measured, since these were too small for our measurement software to distinguish. The authors emphasize that there is not yet a standard for measuring gaps on T-WIM images.

It is known that patient risk factors play an important role in restoration survival (van de Sande *et al.*, 2013). A large patient factor was also seen in this study. Despite the very highly standardized cariogenic exposure, which would make all volunteers 'high caries risk' as far as the appliance was concerned, we still observed volunteers (with established good compliance) with very little caries development, whereas other volunteers had considerable caries development. Caries progression is probably also influenced by other patient factors, *e.g.*, saliva components, biofilm composition, or other oral conditions.

We conclude that gap size is probably not relevant as long as the caries risk is low, but when caries risk is high, even a gap size of 68 μm may allow for secondary wall lesion development. These new findings undermine the accepted theory that only in large gaps (> 250 μm ; Mjör, 2005), which enable a cariogenic biofilm to thrive, wall lesions can develop. Since the 'no gap' interfaces never showed wall lesions, and minimal

gaps of 68 µm did, it may be assumed that the threshold for secondary wall lesion development lies in the range of about 0 to 70 µm.

Acknowledgments

This study was funded by the National Institutes of Health (246 NIH), grant number 1R01DE021383-01, under call RFA-DE-10-005, 'Increasing the service life of dental resin composites'. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. The authors declare no potential conflicts of interest with respect to the authorship and/or publication of this article.

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