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Wall-lesion development in gaps: The role of the adhesive bonding material

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ABSTRACT

Objectives: This study evaluated the caries wall lesion development in different composite–dentin interfaces to investigate if the presence and location of two bonding materials in the gaps influence wall caries lesion development.

Methods: Fourteen volunteers wore a modified occlusal splint containing samples with four different interfaces: perfect bonding/no gap, or with a fixed gap (234 ± 30 μm) with either no bonding material, bonding material (Clearfil Protect Bond–PB and Clearfil SE Bond–SE) on dentin or on composite. Eight times a day, the samples were dipped in 20% sucrose solution for 10 min, during 3 weeks. The samples were imaged with microradiography (T-WIM), and lesion depth (LD) and mineral loss (ML) were measured. The data were analysed with paired *t*-test.

Results: The perfect bonding group did not show any caries wall lesion development, whereas all other interfaces did. The interface with bonding on dentin did not show significantly different wall lesion development from the interface with no material. However, when bonding was present on composite, both LD and ML were significantly higher than both other gap conditions (p-values < 0.05). A difference between the bonding material was only seen when applied on composite: PB showed less ML than SE ($p = 0.01$).

Conclusions: The presence of bonding on the composite side of a composite–dentin gap increased wall lesion development *in situ*.

Clinical significance: The presence and location of an adhesive bonding material in the composite–dentin gaps plays a role on the wall caries lesion development.

1. Introduction

Secondary caries refers to caries lesions affecting the margins of existing restorations[1] and has been widely demonstrated to be a common reason for repair and replacement of posterior failed bond restorations, regardless of the type of restorative material.[2] Secondary caries has been reported to develop in two locations: at the tooth surface adjacent to a filling, similar to primary caries, but also in the interfacial gap between

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restoration and tooth.[3] The latter, often called wall lesions, have been implied in the reported higher susceptibility of composite restorations to secondary caries, as compared to amalgam restorations.[4]

Composite resin is a popular filling material bonded to the tooth structure using bonding agents, creating a composite–tooth interface. This interface is reported as the most vulnerable structure of the adhesive restorations.[5] Since the composite–dentin interface is instable and fragile, even small defects at the cavosurface angle (detectable) and at the inner part of the cavity (undetectable) might present voids. These might be created by incomplete filling of the cavity (particularly in areas of difficult access), by polymerisation shrinkage of resin composites and weak bonding to dentin, by presence of excessive residual water left from the etching and washing procedures, and by others defects from the hybridisation process.[6,7] It was shown that it is almost impossible to prevent creating such voids when using minimally invasive techniques.[8]

Caries wall lesions next to composite restorations have been studied recently both *in vitro* and *in situ*.[7,9–11] These studies used artificially produced interfacial gaps of standardised dimension, but none reported using adhesive bonding material in creating the composite restorations (as the gap made bonding superfluous). In a clinical situation, where a void has been created or an adhesive bond has failed, however, adhesive bonding material will always be present at some location in the interface. Restorative materials may influence the secondary caries development in numerous ways. A recent *in vitro* study reported that the type of bonding material could influence wall lesion development in gaps, with a protective effect of an antibacterial bonding agent on caries lesion development.[12] Those bonding agents were developed with the promise of having anti-caries properties through the presence of an bacterial inhibitor monomer in its composition.

There are different types of *in vitro* caries-like lesion induction models that do not present a standard pattern of caries development.[13] However, *in situ* models seem to be more conclusive in predicting clinical behaviour.[14] Therefore, the objective of this *in situ* study was to evaluate the caries wall lesion depth and mineral loss of different composite–dentin interfaces to investigate if the presence and location of two adhesive bonding materials (with or without an antibacterial monomer) in the gaps influence wall caries lesion development. The null hypothesis tested was that caries development would be similar for all the adhesive interfaces.

2. Materials and methods

2.1. Study design

This was a mono-centre study, randomised (regarding the sequence/location of the tested conditions) with split-mouth design with respect to gap conditions and bonding materials. Two bonding materials with (Clearfil Protect Bond–PB, Kuraray, Okayama, Japan) or without (Clearfil SE Bond–SE, Kuraray) antibacterial monomers were investigated and applied according the manufacturer's recommendations. The outcome variable was wall caries lesion depth (LD) and mineral loss (ML). Results from a parallel study evaluating the effect of gap size on wall lesion development were reported previously.[11]

2.2. Study participants

The study design and protocol were approved by the Local Ethics Committee, METC (CMO file nr. 2011/248, NL33528.01.11). All the participants agreed and signed the written informed consent. Fourteen volunteers (six men and eight women, aged 20–57 year, mean age = 30.4 year) were recruited within the Dental School in Nijmegen, the Netherlands, following the inclusion criteria of subjects between the ages of 18 and 60 yr and with good general health. Exclusion criteria were active caries, periodontitis (DPSI > 2), ASA > 2, and the wearing of orthodontic or a removable prosthetic appliance in the mandibular jaw.

2.3. Preparation of samples

Sound human molars were ground flat with 180-grit Sic paper until complete occlusal enamel removal and dentin exposure was reached (Fig. 1a). The roots were cut off, and the remaining crowns were perpendicularly cut into four dentin sections with a fixed width of 3.2 mm and ±2.5 mm of length. The dentin sections were ground with 600-grit Sic paper to achieve a height of 2.2 mm. The dentin sections were gas-sterilised with ethylene oxide (Isotron Nederland B.V., Venlo, the Netherlands).[14]

For each sample, two dentin sections were placed in a rectangular putty mould with dimensions of 15 mm × 3.2 mm × 2.5 mm. On the pulpal side of the dentin sections, a self-etching primer and bonding agent of the adhesive system used for that group (either SE or PB) were applied on dentin according to the manufacturer's instructions, and 0.5 mm composite resin paste (AP-X PLT, shade A2, Clearfil, Kuraray) was inserted and cured in order to fix the two dentin sections (composite bar, Fig. 1b). 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 of the X-ray beam.

2.4. Bonding procedure

In each composite–dentin bar, three spaces were made (one in each side of the two dentin sections) roughly parallel to the dentin tubule direction with a 012 cylindrical bur with a depth of 1.9 mm (bur, Fig. 1b). While the bar was fixed in a mould, the spaces were filled with the composite resin (AP-X PLT) creating different composite–dentin interfaces:

1) Composite–adhesive–dentin perfect bonding/no gap: the space was filled completely by composite (positive control). The composite and dentin were bonded without any gap between them and with the adhesive systems (PB and SE) applied following the manufacturer's instructions;

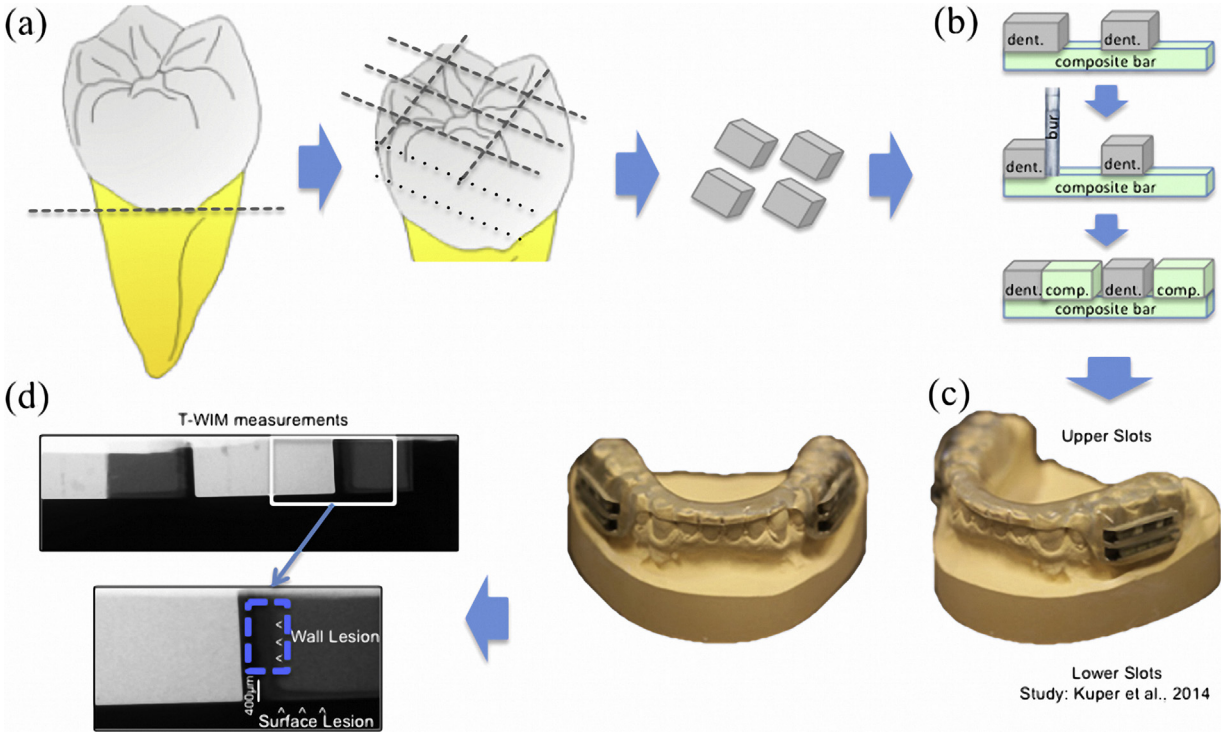

Fig. 1 – Schematic design of samples preparation and analysis. (a) Preparation of the teeth. (b) Preparation of the samples and composite–dentin interfaces. (c) Occlusal splints with the samples positioned into the samples-holder (metallic slots). (d) T-WIM picture and measurement details.

In the remaining groups fixed gaps were created using a plastic matrix of standard thickness of 200 μm.

2) Bonding material on dentin/gap: the adhesive systems (PB and SE) were applied just on dentin side and the composite resin was placed with a fixed gap between them;
3) Bonding material on composite/gap: the bonding agents of the adhesive systems (PB and SE) were applied on the composite side with a fixed gap between them: the plastic matrix was placed in contact with the dentin, bonding material was applied on the other side of the matrix and light-cured, and then the composite was placed and cured against the matrix;
4) No bonding material/gap: no adhesive system was applied in the interface with a fixed gap between composite and dentin (negative control). The data of this group was obtained from a parallel study evaluating the effect of gap size, described elsewhere.[11]

The position of the different composite–dentin interfaces on the composite–dentin bar was changed per volunteer, following a random sequence generated by computer software (Excel Program). The two adhesive systems were placed at the left or right side of the occlusal splint alternately per volunteer also following a random sequence. The samples were polished (600-grit Sic paper) to remove the excess composite, and the final rectangular composite–dentin bars 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 four embedded metal slots of 20 mm × 3.2 mm × 2.5 mm. Both upper and lower slots were used for this study. The samples on the uppers slots were prepared specific for the present study, and the samples on the lowers slots were used for the parallel study, described in detail elsewhere.[11]

2.5. *Experimental protocol*

The occlusal splints (device) were worn 24 h per day for 3 weeks, being removed only during eating, drinking (keeping their normal diet) or for oral hygiene, with the device kept in a physiologic salt solution during those periods. Additionally, volunteers were instructed to dip the device with the samples in a 20% sucrose solution eight times a day for 10 min. They were instructed to observe intervals between sucrose dippings of at least 1 h. 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 and replaced in the mouth. Volunteers were asked to apply fluoride toothpaste/saliva slurry on the samples once a day (when they brushed their teeth). They were explicitly requested to not clean or brush the samples at all. All instructions were given both orally and in writing by a researcher involved in the study.

2.6. *Transversal wavelength independent microradiography (T-WIM)*

T-WIM pictures were made at baseline (T0) and after 21 days (T21) according to a method previously described.[15] The settings for the microradiography were 60 kV, 30 mA and an

exposure of 8 s. A stepwedge with the same absorption coefficient as tooth material (94% Al/6% Zn alloy) was used for proper quantitative measurement of LD and ML.

2.7. Film processing and image measurements

After exposure, films were developed (10 min), fixed (7 min), rinsed, and dried. Digital images of each sample were recorded with a light microscope (Leica Microsystems, Wetzlar, Germany) with a magnification of 10× and a CMOS camera (Canon EOS 50D, Tokyo, Japan). The digital T-WIMs were edited in Adobe Photoshop CS3 (version 10.0; Adobe Systems, San Jose, CA, USA). The contour of the interfaces on the baseline picture was selected and copied to the T21 picture to standardise it. The selected contour was coloured black so that in case of caries development, LD could be easily distinguished from gap width.

From each sample, the wall lesions in the dentin facing were measured with a software program (T-WIM calculation program, version 5.25, J. de Vries, Groningen, NL) at a fixed area of 400 μm distance from the outer surface (dashed blue line at Fig. 1d) in order to prevent overlap with the surface lesion. Baseline measurements (T0) were subtracted from measurements after 3 weeks (T21), to estimate true LD (μm) and ML (μm vol%). The subtracted values were used in the statistical analysis.

To obtain the real gap sizes, the gaps were measured on the baseline T-WIM image using the same software. Since gaps were not always perfectly straight, but slightly tapered, the distance between restoration material and dentin was always measured at the entrance of the gap.

2.8. Statistical analysis

The data were analysed through paired *t*-tests to investigate the effect gap conditions and adhesive bonding material ($\alpha = 0.05$) (*Statistical Package for Social Sciences*, version 20, Chicago, IL, USA).

3. Results

Ten volunteers completed the study successfully. Three volunteers completed only 2 weeks, but since they did show caries development and effects were evaluated within patients, they were included in the analysis. One volunteer lost their sample device and was excluded from the analysis.

The actual gap size between the composite and dentin was 234 μm (±30 μm). For all interface conditions with a gap, the correlation between gap size and LD/ML was not statistically significant, with *p* values ranging between 0.103 and 0.965. Therefore the gap size was not included in the analysis as a factor.

The perfect composite–adhesive–dentin bonding did not present a development of caries wall lesions during the 3 weeks of cariogenic challenge at all. All other composite–dentin interfaces (adhesive bonding on dentin and on composite, for both bonding materials, and for the no bonding material group) presented caries wall lesion development (Figs. 2 and 3).

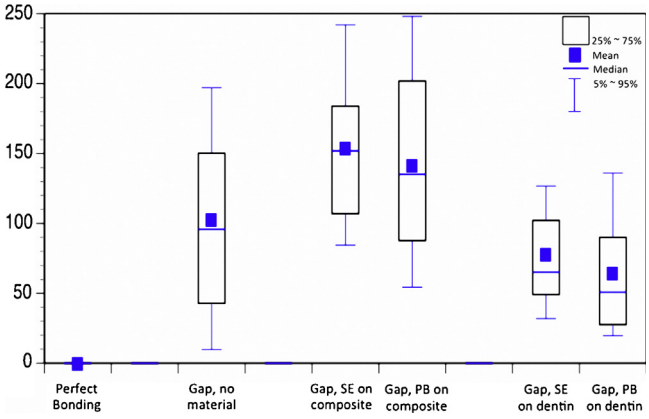

Fig. 2 – Boxplot display of lesion depth (μm) values for each group, considering the interfaces conditions and bonding materials (SE and PB), showing inter-quartile ranges.

The paired *t*-test results of comparisons among the gap conditions groups are presented in Table 1. When comparing the gap conditions with adhesive to the no adhesive group, a different effect was seen for the two adhesive locations: where adhesive was present on dentin no significant difference could be shown (*p*-values ranging between 0.74 and 0.15), whereas where adhesive was present on the composite, both LD and ML were significantly increased (*p*-values ranging between 0.05 and <0.01). For both adhesive bonding materials together, adhesive presence on composite resulted in significantly more LD and ML than adhesive presence on dentin ($p < 0.01$). For both adhesive locations analysed together, the PB bonding material showed less ML than SE ($p = 0.01$), no difference was found for LD ($p = 0.32$).

4. Discussion

This present study investigated the effects of different bonding conditions in caries development on dentin adjacent to simulated failed bond restorations, showing that the condition

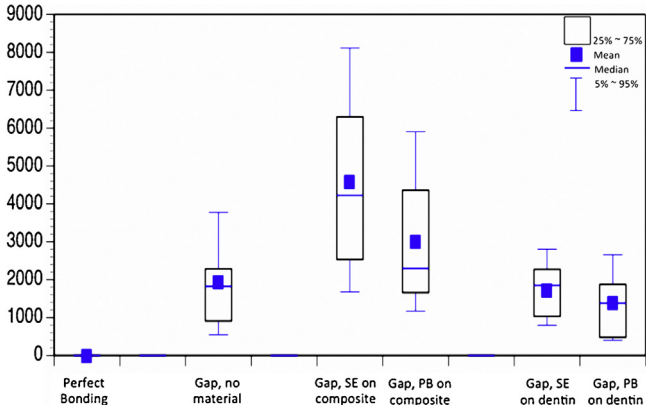

Fig. 3 – Boxplot display of mineral loss (μm vol%) values for each group, considering the interfaces conditions and bonding materials, showing inter-quartile ranges.

Table 1 – Paired *t*-test results of comparisons of lesion depth and mineral loss among the composite–dentin interface conditions Pair Mean Difference* (μm ± SD) *p* value.

	Pair	Mean difference	*p* value	95% Confidence interval of the difference	
				Lower	Upper
Lesion depth	B comp–B dent	–76.7	$p < 0.01$	–99.2	–54.1
	PB–SE	–12.9	$p = 0.32$	–39.2	13.2
	PB comp–NB dent	43.3	$p = 0.03$	5.5	81.2
	SE comp–NB dent	56.3	$p = 0.04$	1.4	111.3
	PB dent–NB dent	–35.6	$p = 0.14$	–85.5	14.4
	SE dent–NB dent	–23.7	$p = 0.36$	–78.5	30.9
Mineral loss	B comp–B dent	–2236.1	$p < 0.01$	–2983.9	–1488.2
	PB–SE	–944.9	$p = 0.01$	–1643.5	–246.3
	PB comp–NB dent	1163.1	$p = 0.05$	10.6	2315.5
	SE comp–NB dent	2922.8	$p < 0.01$	1670.9	4174.7
	PB dent–NB dent	–458.9	$p = 0.38$	–1562.4	644.6
	SE dent–NB dent	–156.7	$p = 0.74$	–1185.7	872.3

B, bonding on; comp, composite; dent, dentin; PB, Clearfil Protect Bond; SE, Clearfil SE Bond; NB, no bonding.

that the composite–dentin interface presents when there is a gap plays a role on the LD and ML. Thus, the null hypothesis should be rejected as the presence of bonding material in the gaps influenced the wall caries lesion development.

While it is widely recognised that the characteristics of the bonding substrate plays a role on the quality of adhesion, the issue of bond behaviour and durability has dominated most current research in composite–dentin bonding.[16] The breakdown of interfacial sealing poses a challenge to the longevity of restorations.[2] If longevity is mainly affected by leakage of oral fluids and bacteria along the interface, studies on this phenomenon should be more clinically relevant to better predict the clinical performance of adhesive restorations.[17] In this context, the present study was carried out, as the presence of different bonding interfaces of the restoration could influence the caries development at those interfaces.

To the authors' knowledge there are no previous studies that have investigated the effect of different interface conditions, simulating the situation after a failed bond, on mineral loss in adjacent dentin, making direct comparisons impossible. In this study the presence of bonding material on composite side of composite–dentin gaps significantly increased lesion development as compared to bare composite. This negative effect could be explained by the presence of bonding material acting as a retention factor for the biofilm. Contrary to expectations, the presence of bonding on dentin portion did not reduce wall caries lesion development, compared to condition without bonding. This finding highlights the fragility of the adhesive interface and that the interaction between the adhesive monomer and the etched dentin is an instable structure when exposed to a cariogenic challenge.[18,19] However, in this study caries wall lesions only developed when there was a defect/gap on the adhesive interface. The presence of a perfect bonding prevented wall lesions, confirming earlier reports that the presence of a gap is a crucial factor in wall lesion development.[7,11]

The use of *in situ* models provides standardised conditions, simultaneously maintaining the individual variability of the oral cavity complexity.[20] During the 3-week period, the subjects immersed the samples into a sucrose solution, ensuring a standard baseline cariogenic challenge that was able to promote mineral loss and caries develop in the dentin. The use of fluoride-containing dentifrice was included because of its widespread use and to model more closely the *in vivo* situation. The split-mouth set-up of the study allowed direct comparison between materials. The adhesive system with antibacterial component (PB) resulted in less ML when it was applied on composite resin, which may be attributed to the antibacterial properties of the adhesive. A similar effect was found recently in an *in vitro* study.[11] Nevertheless, the presence of PB on dentin did not offer any additional protection to wall caries lesion development, showing similar results to the material without the antibacterial monomer (SE). In the conditions of this study, secondary caries progression may ultimately depend on individual habits and different patterns of oral pathogens prevalence within the biofilm,[20] and the material composition may not have had an effect large enough to promote significant differences in lesion depth. Therefore, the clinical effect of antibacterial composites on secondary caries lesion progression remains uncertain.

5. Conclusion

In conclusion, within the limitations of the present study, the presence and location of an adhesive bonding material in the

gaps influences wall caries lesion development. Its presence on the composite side of a composite–dentin gap increased wall lesion development *in situ*, and only at this location the bonding materials had different effects, with the antibacterial adhesive showing less mineral loss, but not less lesion depth.

Conflict of interest

The authors declare no conflicts of interest.

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