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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;

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