DENTAL MATERIALS 32 (2016) 11–25

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.intl.elsevierhealth.com/journals/dema

Review

Secondary caries and microleakage

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ARTICLE INFO

Keywords:
Biofilms
Corrosion
Dental caries
Dental materials

ABSTRACT

Objectives. To critically appraise experimental ex vivo research that has focused on secondary caries, and to offer possible explanations for the seemingly poor correlation to clinical observations.

Methods. The literature relating to the etiopathogenesis or prevention of secondary caries gained from experimental ex vivo research was reviewed, with particular emphasis on microleakage and artificial caries-like lesions.

Results. It is doubtful whether a caries wall lesion can exist independently of an outer enamel caries lesion. Microleakage experiments apparently continue to emerge regardless of multiple reviews questioning the reliability and validity of the method. Several of the approaches used to generate artificial caries-like lesions are very aggressive. Remarkably little discussion has evolved about how these aggressive approaches create microenvironments that do not occur in reality. Corrosion- and biodegradation products may influence the biofilm qualitatively and quantitatively and it is difficult to replicate these variables in any ex vivo environment. Clinical data sampling method, patient demography as well as study methodology influences the incidence and prevalence estimates of secondary caries. Clinical results based on clinical work in settings where cost per unit time is of nominal concern do not provide any indications on how the restorative material will perform when placed by the average dentists in the mouths of their spectrum of patients during a busy workday.

Significance and recommendations. The term "wall lesion" including its variants is ill defined, has been, and is still being used indiscriminately. Stakeholders should avoid using this ambiguous label due to its connotation to an entity that does not exist per se.

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http://dx.doi.org/10.1016/j.dental.2015.09.006
0109-5641/

1. Introduction

Secondary caries is the most commonly reported reason for re-restoration of teeth, regardless of restorative material. This conclusion has been consistent in multiple narrative and systematic reviews on the clinical performance of dental restorations published over the last few decades [1–3].

For obvious ethical reasons, it is not feasible to conduct clinical trials to monitor the progress of initial secondary caries adjacent to restoration margins, with the objective of studying etiopathogenesis and/or to identifying potential prognostic factors. Prognostic factors are likely associated with the patient, the operator and restorative material, including the structure of the tooth-restoration interface following optimal, as well as suboptimal handling and placement of the restorative material [2,3].

Consequently, stakeholders with a strategy to decrease rates of secondary caries by improving restorative materials or material handling procedures are forced to statistically correlate as best as possible one, two or combinations of specific properties of existing restorative materials or handling procedures with reported rates of secondary caries in different clinical studies with various methodological qualities. Such statistics can be deceptive, because of a range of likely biases and possible confounding of both the independent and dependent variables in the majority of existing clinical studies.

Planning, conducting and reporting relevant outcomes of clinical comparative trials is logistically challenging, costly and potentially unpredictable if the study participant attrition is so high that adequate study power cannot be maintained. Moreover, correctly handled restorative materials placed under optimal conditions remain intact for an extensive time. Manufacturers may question a prioritizing of limited research funds to conduct clinical studies to evaluate the degrees of flawlessness of restorations monitored for anything less than 3 years. Moreover, to the author's knowledge, there is no evidence that a satisfactory clinical performance after 1 or 2 years is predictive of good long-term performance. It is therefore debatable whether the results of any clinical study of less than 3 years should have any impact at all on consideration of change of existing material compositions or material handling procedures other than to reject materials exhibiting an unacceptably high early failure rate.

Stakeholders have therefore pursued alternative strategies to improve our understanding of how to develop innovative restorative materials which reduce the risk of secondary caries formation. Both academia and industry have designed numerous ingenious laboratory models and protocols for in situ experimental studies with the hoped objective of minimizing adhesion of cariogenic biofilms, preventing the occurrence of secondary caries adjacent to restorations, and elucidating the reasons for the deterioration of the restorative materials including the tooth-restoration interface.

Unfortunately, the correlation between microleakage around restorations or artificial caries-like lesions adjacent to restorations generated in vitro *or* in situ is poor versus tooth-restoration interface qualities measured in vitro or in vivo. It is also poor versus reported incidences of secondary caries observed in clinical efficacy or effectiveness studies [4–10]. There are probably multiple reasons for the incongruence between experimental data and clinical observations of secondary caries. The objective of this review is to critically appraise the existing experimental research with a focus on etiopathogenesis or prevention of secondary caries, and to explore possible explanations for the seemingly poor correlation to clinical observations.

2. Dental caries

A full review of the continuum of dental caries is outside of the scope of this article, but some features of this disease warrants a brief review in the context to the secondary caries puzzle.

Caries develops first in the enamel, a tissue with densely packed uniaxial crystallites with inter- and intra-prismatic micropores that are 1–30 nm wide. The tissue structure display a type of molecular sieve behavior and is anisotropic to light [11,12]. The anisotropy is due to both its intrinsic (or crystalline) birefringence, as well as a form (or textural) birefringence (also known as structure anisotropy). When the enamel demineralizes, the intrinsic birefringence changes, but the form birefringence persists because pores between the enamel prisms remain oriented and they have a diameter and separation that is much smaller than the wavelength of visible light (390–700 nm).

Transilluminated ground sections of enamel with caries demonstrate in most situations four distinct zones within the caries lesion when viewed in an optical microscope. The zones represent different optical properties of the tissue and are particularly discernible when the ground section is imbibed in a medium with a refractive index (R.I.) similar to intact enamel in a polarized light microscope (PLM). Investigators have used either water (R.I. = 1.33), alcohols (R.I. = 1.33–1.43), Thoulet's solution, consisting of potassium mercuric chloride (R.I. = 1.41–1.62) or quinoline (R.I. = 1.62) to enhance the separation of the enamel lesion zones in the microscope as well as estimating pore sizes as a function of molecular size of the imbibition media. A narrow superficial zone appears intact, probably because of precipitation of mineral ions interfacing the pellicle, saliva and biofilms in vivo, alternatively some fluid in vitro. Immediately below