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Minimal intervention dentistry for managing dental caries – a review

Report of a FDI task group*

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This publication describes the history of minimal intervention dentistry (MID) for managing dental caries and presents evidence for various carious lesion detection devices, for preventive measures, for restorative and non-restorative therapies as well as for repairing rather than replacing defective restorations. It is a follow-up to the FDI World Dental Federation publication on MID, of 2000. The dental profession currently is faced with an enormous task of how to manage the high burden of consequences of the caries process amongst the world population. If it is to manage carious lesion development and its progression, it should move away from the 'surgical' care approach and fully embrace the MID approach. The chance for MID to be successful is thought to be increased tremendously if dental caries is not considered an infectious but instead a behavioural disease with a bacterial component. Controlling the two main carious lesion development related behaviours, i.e. intake and frequency of fermentable sugars, to not more than five times daily and removing/disturbing dental plaque from all tooth surfaces using an effective fluoridated toothpaste twice daily, are the ingredients for reducing the burden of dental caries in many communities in the world. FDI's policy of reducing the need for restorative therapy by placing an even greater emphasis on caries prevention than is currently done, is therefore, worth pursuing.

Key words: *Minimal intervention dentistry, caries lesion detection, caries risk assessment, caries preventive measures, restorative care, plaque control, repaired restoration*

Minimal Intervention Dentistry (MID) is a response to the traditional, surgical manner of managing dental caries, that is based on the operative concepts of G.V. Black of more than a century ago. MID is a philosophy that attempts to ensure that teeth are kept functional for life. This term, therefore, is not restricted to the management of dental caries but is also applicable to other areas of oral health; such as periodontology, oral rehabilitation and oral surgery.

Dental caries is the most prevalent of the oral diseases worldwide. This paper presents the rationale and content of MID for managing dental caries, using evidence-based information whenever available.

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RATIONALE OF THE MID PHILOSOPHY

Without doubt, the many studies assessing the effect of water fluoridation on the progression of carious lesions have contributed greatly to the development of the MID philosophy. The one study that stands out in terms of importance is the Tiel-Culemborg study from the Netherlands[1]. This study, like many others, showed that the fluoridation of water reduced the prevalence of cavitated dentine lesions by approximately 50%. It also showed that the main long-term action of fluoride is retarding the progression of a carious lesion, rather than prevention of its development[2]. This outcome became evident as ample time was spent assessing not only cavitated dentine lesions, but also enamel carious lesions. The secondary study outcome was confirmed in later studies that researched the effectiveness of fluoride in varnishes, gels and mouth rinses[3]. These data led to a change in the cariology paradigm: fluoride appears not to act pre-eruptively, as was thought, but mostly post-eruptively by changing the mineral saturation characteristics at the tooth surface[4].

Another topic that was researched extensively in the 1960–1980 period was dental plaque. The outcomes resulted in the wide acceptance of the fact that dental plaque or dental biofilm, as it is sometimes more correctly termed, should at least be disturbed or at best be removed from the tooth surfaces daily, if carious lesion development is to be minimised. In combination with fluoride toothpaste, plaque removal with a toothbrush has become a major cornerstone in managing carious lesion development for communities worldwide[5].

An important concept, that governed the development of MID, is the 'Repeat Restoration Cycle'. Elderton *et al.*[6] clearly demonstrated, on the basis of studying the survival of amalgam restorations, that 'eliminating' carious lesions in order to improve oral health, through restorative procedures based on the G.V. Black concept, does not keep teeth functional for life for all individuals. The concept reitterated that preventive or non-operative actions should go hand-in-hand with restorative care, and that assessment of carious lesion development and progression plays a vital part in the provision of adequate oral health care. The development of various adhesive materials and adhesive systems has contributed greatly to attaining the primary aim of MID. The ability to reduce the need for cutting away healthy tooth tissues when using adhesive materials, relative to the traditional restorative concepts, has led to smaller and less destructive cavity preparations and therefore, smaller restorations[7]. Retaining sound tooth structure, and thus increasing the chance for maintaining its vitality and function, was further increased as a result of the work done by colleagues like Massler[8] and Fusayama[9]. They showed that only the 'infected' ('outer carious' or 'decomposed') dentine needed to be removed as part of the cavity preparation process, and that the 'affected' ('inner carious' or 'demineralised') dentine could remain. This demineralised dentine would remineralise under a well placed, well sealed and well maintained restoration[10–12].

By early 1990, research had shown that managing dental carious lesions should depart from a traditional surgical approach and move to a 'biological' or 'medical' approach. The research pointed to a completely new approach to the management of the carious lesion. As far as we know, it was Mount[13] who first cited the need for 'Minimal Treatment' of dental caries. Further elaboration of this new approach was published by Dawson and Makinson[14,15], who first termed 'Minimal Intervention Dentistry' in the literature. The first International Association for Dental Research (IADR) symposium on minimal intervention techniques for dental caries was held in 1995 and was almost entirely devoted to the developments of one of the MID approaches, namely Atraumatic Restorative Treatment (ART)[16].

As mentioned earlier, the aim of MID is to keep teeth healthy and functional for life. A most important element is achieved through implementing the important strategies for keeping teeth free from carious lesions. These strategies are considered to be: (i) early caries detection and risk assessment; (ii) remineralisation of demineralised enamel and dentine; (iii) optimal caries preventive measures; (iv) minimally invasive operative interventions and; (v) repair rather than replacement of restorations[17]. It is self-evident that MID does not equate to cutting smaller cavities than before, as many dentists thought[18,19].

The first three MID aspects (early caries detection and caries risk assessment; remineralisation of demineralised enamel and dentine; optimal caries-preventive measures) should be employed throughout a person's life and only when oral health maintenance has failed and a cavity has developed should a minimally invasive operative intervention be undertaken. The authors are aware that the implementation of the MID philosophy will vary in different countries for a number of reasons, which include: professional dental training, access to the internet and printed dental literature, availability and type of dental equipment and dental materials and oral health remuneration systems.

The remainder of this paper will discuss in detail the five strategies that make up the MID philosophy using, as much as possible, evidence-based information available in the peer-reviewed literature.

EARLY CARIES DETECTION AND CARIES RISK ASSESSMENT

Detection devices

The oldest device used for detecting carious lesions, apart from the probe, is the X-ray machine. Radiography is reliable for detecting carious lesions in approximal tooth surfaces but considered unreliable in occlusal surfaces, particularly for diagnosing carious lesions in enamel and in the outer one-third to one half of the dentine[20–22]. Fibre-Optic Trans-Illumination (FOTI) appears to be a very reliable device for detecting carious lesions in approximal surfaces, particularly in anterior teeth[23]. In contrast, an infrared laser fluorescence device (e.g. DIAGNOdent; Kavo GmbH, Bibberach, Germany) has been reported to be invalid in detecting carious lesions in occlusal surfaces because it not only detects organic carious tissues, and putatively, the porphyrins from bacterial metabolism, but also other organic material such as plaque, calculus, stain and food remnants[24,25]. Its validity is further compromised by the presence of enamel hypomineralisation of origin other than that of dental caries[24]. Similar disadvantages apply to quantitative light-induced fluorescence (QLF; Inspektor, Amsterdam, the Netherlands), which uses the fluorescence differences between sound and demineralised enamel to detect and quantify enamel carious lesions, although its reliability appears to be higher than that of the infrared laser fluorescence-based devices[26]. A new system using light fluorescence technology (Sopralife; Acteon, Bordeaux, France) utilises a different wavelength than QLF to detect carious lesions, in conjunction with a camera. Currently, the value of QLF systems for carious lesion detection in clinical practice seems to be limited. Other methods, such as electrical impedance (CarieScan PRO™; CarieScan Ltd, Dundee, UK) and photothermal radiometry (Canary System™; Quantum Dental Technologies, Toronto, ON, Canada) have recently been developed. However more research is required before they can be advised.

It appears that both X-ray and FOTI devices are suitable for use for carious lesion detection on approximal surfaces and that infrared laser fluorescence and light-induced fluorescence devices are not sufficiently reliable for assessing carious lesions in pits and fissures of occlusal surfaces[27]. This also applies to the deciduous dentition, in which newer technology-based systems have not been found to be reliable for the accurate detection of carious lesions on approximal surfaces[28]. Therefore, different techniques should be used for assessing carious lesions on occlusal and smooth tooth surfaces. One such technique employs visible-tactile methods.

Visual-tactile methods

Perhaps because of the absence of a properly validated and reliable carious lesion detection device, early enthusiasm dimmed, and the emphasis shifted back to visual-tactile detection methods in the second half of the 1990s. The World Health Organisation (WHO) had propagated its method, which was based on a 'yes/no' clearly cavitated dentine lesion, as a reliable data base was required for comparison of decayed, missing and filled (DMF) teeth scores among member countries and because DMF data from decades earlier were available[29]. This very crude cut-off point, and the fact that caries prevalence and carious lesion development had declined in many industrialised countries, were reasons for epidemiologists to subsequently include the assessment of enamel lesions in caries assessment indices. One such group of epidemiologists developed the International Caries Detection and Assessment System (ICDAS)[30]. This two-digit enamel and dentine carious lesion scoring system has recently received much attention. It was developed for use in epidemiological surveys, research, dental education and in practices. The index, when used in epidemiological surveys, has received some criticism[31], could not be applied correctly[32] and was unable to properly allow the reporting of findings[33]. Prior to the launch of ICDAS, Nyvad[34] published her 'Nyvad-index', which permits the assessment of enamel and dentine carious lesions as well as the activity/inactivity of enamel carious lesions. The index has been used recently in a number of studies[35,36] and appears to be valid. Monse *et al.*[37] introduced the 'Pulpal Involvement Ulcerations Fistula Abscess' (PUFA index) with the intention of alerting the dental/medical/educational communities about the poor state of dentitions of children in the Philippines. A novel visual one-digit caries assessment index was reported recently[38]. It includes non-cavitated and cavitated carious lesions, pulpally involved and abscessed teeth, as well as sealed, restored and lost teeth. In developing the index, experience gained from applying the ICDAS II[31] and PUFA[39] indices in the field were essential. The index is termed Caries Assessment Spectrum and Treatment (CAST). It has been validated for face and content[40], while construct validity and reliability testing is on-going.

Carrying out an oral investigation on the basis of assessing teeth with cavitated dentine lesions only (DMF) should be considered a screening exercise. If the investigation is conducted for healthcare planning purposes, enamel carious lesions should be assessed as well, whether in clinical practice or when conducting an epidemiological survey. The ICDAS II and Nyvad-index may be suitable in a clinical practice setting, although the number of studies supporting this assumption is low. The same caveat applies to the recently developed epidemiological indices PUFA and CAST, which appear to be promising, but need further research.

Caries risk assessment

The caries disease process is dynamic and multi-factorial in nature. Caries risk is defined as 'the probability of future caries disease development'. Disease development includes both primary disease (new carious lesions) and secondary disease (lesion progression or reactivated carious lesions). Risk assessment for such a dynamic disease is complex as it is only able to provide a snapshot at that particular moment and risk factors may change over time. Most importantly, for assessing lesion activity accurately in one session, using a combination of indicators (visual appearance, location, tactile sensation and gingival health) might still provide the best way to determine lesion activity. Moreover, activity criteria are not designed to quantify lesion progression, with regard to either size or depth. Despite its current shortcomings, however, risk assessment and caries prediction is a crucial part of contemporary clinical decision-making that dental professionals apply on a daily basis. It serves as the foundation for the patients' prognosis for caries and is embodied in the individually tailored oral health management plan provided to the patients.

A strong body of evidence exists that at all ages the 'past and present caries experience' (and in particular the presence of active carious lesions) is still the most accurate and powerful, single predictor of risk of future carious lesion development[41–43]. This conclusion, however, is unfortunate. It does not provide much guidance for a 'whole disease prevention approach': one would hope to prevent even the earliest onset of a carious lesion, and intervene through interruption of the caries process and healing of affected tooth tissues after the fact. Risk factors may also change over time. A patient may have numerous restorations but, for whatever reason, the risk as assessed objectively may now be low (e.g. all risk factors have been determined and resolved). To continue with full-blown caries management strategies would be overtreatment. On the other hand, a patient's caries risk may also change rapidly to extreme risk if, e.g., medications have been prescribed that affect salivary glands and lead to hyposalivation.

Caries risk prediction is still a work-in-progress. A recent publication provides an excellent concise and thorough overview of the evidence related to patient caries risk assessment[44]. The authors concluded that 'it is more important that an assessment is carried out, incorporating the best available evidence, than that no attempt is made due to the lack of firm evidence. The risk should be documented in a patient's chart and be used to influence a treatment plan.' One of the tools that assists clinicians worldwide in motivating patients is the 'Cariogram', an interactive validated program for patient motivation[45]. This informative program illustrates caries risk in an instructive but simple graphical way, including the interaction between the various caries related factors. The Cariogram demonstrates the 'chance to avoid new carious lesion development' in the near future and to what extent the various factors will affect this chance. The software is available in 13 languages and can be downloaded as 'shareware'[46].

'Whole-population' approach and 'risk-based' strategy

Caries-risk assessment is usually described at the level of the individual patient[47]. It provides information needed to determine the most appropriate management decision for an individual patient. Risk prediction in a group is also pursued to enhance healthcare efficacy by focusing on those individuals with the largest risk, thus aiming to prevent or reduce a disease burden in the near future. This provides the oral healthcare professional with both individualised and population-based strategies for improving oral health. Although this may seem dichotomous, managing carious lesions calls for both approaches.

The 'whole population' approach is appropriate for the prevention of oral diseases and applying it is the only way to reduce the burden of these diseases and the cost of oral care[48]. In today's healthcare debates, often initiated by changing economic situation, cost-effectiveness seems equally important as quality of care. Oral healthcare discussions are often complicated by the non-proportional distribution of the burden of the preventable disease called dental caries. Whether a risk-based caries management plan is cost-effective and ultimately leads to improved oral health needs to be investigated.

In a balanced view, the advantages and disadvantages of both the 'high-risk' strategy (seeks to protect susceptible individuals) and 'population' approach (seeks to control the causes of incidence) have been considered[49]. This led to the conclusion that 'the "high-risk" strategy was an interim phase, only needed as long as the underlying causes of a disease were not yet clear or couldn't be controlled. If causes could be removed, susceptibility ceases to matter'[50]. The causes of dental disease are known but – at present – cannot be completely controlled[51].

A significant reduction in caries increment was shown when caries preventive measures were targeted at children with active non-cavitated lesions[52]. Inclusion of individual risk prediction as a basis for targeted prevention will increase efficacy of targeted measures and thus improve cost-effectiveness. Similarly, caries risk status used to determine a personalised recall interval[50,52,53] allows for enhanced recall periods, resulting in more effective use of oral healthcare professionals' time. The personalised recall interval, directed towards optimal oral health, can be adjusted to the person's compliance with preventive and maintenance advice.

It may be concluded that although the caries risk prediction may guide the best use of available funds to support preventive caries management, the dwindling financial means for the same, or even increasing, needs continue to call for the 'high-risk' strategy as well as the 'whole-population' approach. While the dental profession needs to embrace a more primary preventive approach to caries management based on common risk factors, secondary prevention and management will continue to focus on patient-centred caries management, including both preventive and minimally invasive tissue-preserving operative interventions[53]. These interventions will be discussed later in this paper.

REMINERALISATION OF ENAMEL AND DENTINE CARIOUS LESIONS

Dental plaque and dental caries

Dental caries is a complex process of cyclical enamel de- and re-mineralisation. *Streptococcus mutans* and *Streptococcus sobrinus* are two putatively important bacteria in the initiation of enamel demineralisation, with *Lactobacillus caseii* assuming greater importance after initial progression of the carious lesion. This is Loesche's so-called 'specific plaque hypothesis'[54,55]. Dental caries occurrence is due to organic acids produced by *mutans streptococci* and *Lactobacilli* as by-products of the metabolism of sugars, namely lactic, formic and acetic acids[56,57]. However, certain researchers have promulgated a mixed bacterial ecological theory in which the previously mentioned cariogenic bacteria are but a few of several potentially cariogenic bacteria present in plaque, with several species not until recently having been identified, due to the difficulty in culturing these bacteria under normal laboratory conditions[58,59]. Some authors believe that many bacterial species have the potential to cause carious lesion development, depending on the characteristics of the diet and the acidogenicity and aciduricity of the commensal oral bacteria, which lead to ecological shifts in the plaque bacterial community and subsequently the caries risk[59–61].

Repeated consumption of readily fermentable carbohydrates, especially sucrose, leads to the proportional overgrowth of cariogenic bacteria such as *S. mutans*. These changes in the biofilm increase the potential for enamel mineral loss, the subsequent production of organic acids, and an amphibiotic change in the oral microflora leading to increased risk of carious lesion development[4,62].

An individual is never free of dental caries[63]. The process of enamel demineralisation and remineralisation cycles constantly moves between net loss and net gain of mineral. It is only when the balance leans towards net loss for some time that clinically identifiable signs of the process become apparent. The long-term outcome of this cyclic process is determined by the composition and amount of plaque, sugar consumption frequency and timing, fluoride exposure, salivary flow and quality, enamel quality and individual immune response[64–66]. In summary, the disease is manifested as an interplay between environmental, behavioural and genetic factors[4].

Mechanisms of action of fluoride in enamel

The presence of fluoride during the remineralisation/demineralisation cycle leads to its incorporation into the crystalline structure of the carbonated hydroxyapatite, which not only decreases crystal solubility, but also increases the precipitation rate of enamel mineral in the presence of calcium and phosphate due to the lower solubility of fluorapatite[67,68]. The fluoride decreases enamel solubility in two ways: (i) the fluoride ion is more stable in the crystal lattice than the hydrogen ion and (ii) it interacts with the calcium ions on the crystal surface, interacting closely and binding strongly[69].

The effect and penetration of fluoride into the biofilm on the tooth surface is dependent on the type of fluoridated product and the time of exposure. When a clinical biofilm was exposed to 1,000 ppm (0.22%) sodium fluoride solution, exposure of up to 120 seconds increased plaque surface fluoride concentrations only, while 30-minute exposure allowed penetration of more than 1,000 ppm (0.22%) fluoride up to 900 μm into the plaque[70]. The clinical relevance or practicality of a 30-minute exposure is questionable, apart from placement of high concentration (22,600 ppm F^-; 5% NaF) fluoride varnish. Thus, the efficacy of intermittently applied professional topical gels and foams is questionable, and the use of high concentration fluoridated varnishes should be encouraged, even in children[3].

Role of calcium and phosphate

The pre-eminent role of fluoride in preventive dentistry remains valid. However, the effectiveness of fluoride to remineralise enamel and obtain net mineral gain is limited by the bio-availability of calcium and phosphate ions[71–73]. If the acid challenge to the enamel is extensive, the salivary calcium and phosphate reservoir is quickly depleted and a net loss of enamel mineral can occur[71,73].

The intrinsic sources of calcium and phosphate are saliva, dissolved tooth mineral and to a lesser degree, gingival crevicular fluid. To gain net remineralisation, the action of fluoride is limited by the amount of calcium derived from saliva, without extrinsic bioavailable sources of calcium and phosphate[68,73]. Increased concentrations of calcium would also increase the retention of fluoride in the plaque biofilm by increasing calcium-bridging[74].

Therefore, for remineralisation to occur during increased caries risk, an increase in bioavailable calcium and phosphate is fundamental to improving the effectiveness of the agent. Increased calcium and phosphate can be stabilised by macromolecules inherent in the saliva and plaque. However, the concentration of these proteins and peptides is limited. Therefore a method for improving the effectiveness of calcium and phosphate stabilisation in the oral environment is required[73,75].

Casein phosphopeptide-amorphous calcium phosphate complexes

Ongoing research over the past 25 years has isolated and purified peptides from casein, a multi-phosphorylated protein present in milk. Casein phosphopeptide-amorphous calcium phosphate complexes (CPP-ACP) have been demonstrated to have anti-cariogenic activity in laboratory, animal and human *in situ* and clinical experiments[75–80]. Casein phosphopeptides (CPP) have the ability to stabilise high concentrations of calcium and phosphate in metastable solution[81]. The CPP complexes bind to form clusters with calcium and phosphate, preventing the growth of seed crystals to the critical size required for nucleation and phase transformation/crystallisation, providing a ready source of ionic calcium and phosphate[82,83]. The complexes are bound in plaque and buffer the calcium and phosphate ion activities in the plaque fluid and at the tooth surface, establishing an environment supersaturated with calcium and phosphate, inhibiting demineralisation and driving remineralisation. Therefore, the ability to provide supersaturated levels of ionic calcium and phosphate at the tooth surface would increase the efficacy of remineralisation.

In order to manage caries lesion development through minimising the solubility of enamel during an acid attack, the individual's tooth surfaces should be exposed to supersaturated levels of calcium, phosphate and fluoride that are available in products containing these ions in a bio-available form.

OPTIMAL CARIES PREVENTIVE MEASURES

Different measures have been proposed for preventing and arresting carious lesions. It is the task of the dental professional to select, based on evidence and on the patient's profile, which preventive measure(s) is most appropriate for a specific clinical situation. In many cases, more than one preventive measure needs to be applied. The whole population approach and individual caries risk assessment are essential activities, alongside with the provision/usage of personalised preventive measure(s) that will ultimately determine the level of reduction of carious lesions in patients and populations.

Dental caries is a preventable disease. Therefore the best strategy for managing the disease is to intervene before its signs and symptoms are clinically detected. Disturbance of the biofilm (dental plaque) by brushing teeth with a sufficiently-fluoridated toothpaste on a daily basis is an effective measure which contributes to the control of enamel carious lesion development[84]. Even disturbing the biofilm from cavitated dentine lesions appears to arrest further progression of such lesions[85]. The effectiveness of different measures for preventing and/or arresting carious lesions for use in MID will be discussed below, and summarised in Table 1.

Effectiveness of caries-preventive measures

Diet counselling and sugar substitutes

The assumed relationship between carious lesion development and consumption of fermentable sugars used to be stronger in the past than currently. The extensive exposure to different kinds of fluoride vehicles is considered the main reason for this situation[86]. Diet control, in terms of intake of sugars and other fermentable carbohydrates, is still an important factor in managing carious lesion development. Individuals at high caries risk, and/or those that do not use fluoride agents, will benefit from dietary control measures. The interplay between consumption of cariogenic food, oral hygiene, availability of saliva and fluoride is nicely modeled by Van Loveren and Duggal[87]. They state that as long as saliva and fluoride are available in the mouth in abundance, and if biofilm control is performed properly at the same time, the detrimental effect of cariogenic food consumption on demineralising enamel and dentine can be considered low.

The use of sugar substitutes is a preventive measure that assists individuals in reducing total cariogenic sugar intake. Xylitol and sorbitol are the sugar alcohols most frequently added to 'sugar-free' products[88]. In general, evidence suggests that the use of sugar-free chewing gum immediately after meals reduces carious lesion progression[89,90]; that the use of chewing gum containing xylitol should be part of a strategy for carious lesion control in schools[90] and that the provision of xylitol-containing gummy bear snacks is feasible[91]. The last can be implemented, with good compliance from both children and parents, in a caries-control regimen at schools[91]. Although the consumption of xylitol-based candies and lozenges favours a reduction in carious lesion increment, in general, this effect is not seen on approximal tooth surfaces[92]. A side-effect of polyol-based sugar-free products is their potential to cause dental erosion if containing acidic flavouring agents[93]. An empirical rule is that children should be advised to restrain their sugar-containing food intake to a frequency of not more than five times daily[94].

Fluoridated agents

Fluoride can be provided via water, milk or salt, or be administered topically by professionals and through self-application devices (toothpaste, gel, varnish and mouthwash). Fluoride is found naturally in the environment (water and plants) and can be added to consumer products, such as infant formulas and beverages.

Water fluoridation is a method of making fluoride accessible to an entire community without requiring individuals to change their behaviour in order to obtain the benefits of fluoridation[95]. It is still considered the best public health strategy for reducing carious lesion development and progression in many societies[96–98]. The safety and efficacy of fluoridated drinking water have been assessed, mainly in child populations[99]. Results show a dose-dependent relationship between carious lesion reduction and severity of dental fluorosis. Adults also benefit from water fluoridation[100].

With respect to milk fluoridation, the Cochrane Collaboration review[101] concluded that there is insufficient evidence to show the effectiveness of fluoridated milk in controlling dental caries, despite a beneficial effect for school children, mainly observed in the permanent dentition. Controversy exists regarding the effectiveness of salt fluoridation[102]. A systematic review on the topic favoured salt fluoridation *versus* no exposure to fluoride for caries prevention in permanent teeth[103]. However, the number of confounding factors, observed methodological bias and overall quality of the papers included in the review stressed the need for more high quality studies to determine conclusively the efficacy of salt fluoridation. Owing to the lack of sufficient evidence of the efficacy and effectiveness of salt fluoridation, caution should be taken before this fluoride vehicle can be safely recommended as part of a strategy aimed at reducing carious lesions in a community.

A series of Cochrane reviews on self- and professionally applied fluoride agents has been published during recent years. The main results have been summarised by Marinho[3] and showed that the use of fluoride toothpaste, fluoride mouthrinses, fluoride gels and fluoride varnishes are able to reduce the incidence of dental carious lesions, irrespective of whether other fluoride vehicles are being used at the same time. The use of fluoridated toothpaste is the most widespread method used for maintaining a constant level of fluoride in the oral cavity. It is considered to be one of the major factors that has contributed to the decline of the prevalence of dental caries in high-income countries[104,105]. The higher the fluoride concentration in toothpaste, the higher its caries-preventive effect[106]. If the risk of dental fluorosis is of concern, the fluoride level of toothpaste for young children (under 6 years of age) is recommended to be lower than 1,000 ppm[107].

Chlorhexidine-containing agents

Chlorhexidine is available in mouth rinses, gel and varnish. A systematic review was aimed at determining the carious lesion-inhibiting effect of chlorhexidine varnishes on the permanent dentition of children, adolescents and young adults. Chlorhexidine varnish showed a moderate caries-inhibiting effect when applied every 3–4 months, but this effect had diminished 2 years after the last application. Studies that test chlorhexidine effectiveness with longer application intervals are required[108]. There is also weak evidence that in the absence of regular professional tooth cleaning and oral hygiene instruction, chlorhexidine varnish provides a beneficial effect in special needs patients[109].

The overall conclusion about chlorhexidine as a carious lesion control agent is that evidence of its effectiveness in mouth rinses and gel products is lacking[110]. Chlorhexidine varnish can be considered a short term option for caries control in individuals at high caries risk who have high bacteria counts[111–113].

Silver diammine fluoride

Silver diammine fluoride (SDF) is a combination of silver nitrate and sodium fluoride ($Ag(NH_3)_2F$) that, when applied to carious tissues, inhibits carious lesion progression by its interaction with bacteria[114]. Very few studies assessing the effect of SDF as a carious lesion control agent in non-cavitated lesions have been conducted. Braga *et al.*[115] investigated the effect of SDF in arresting enamel carious lesions in pits and fissures of permanent molars for up to 30 months. The results were no different from those achieved by plaque control through tooth brushing and the use of glass-ionomer sealant; two approaches which are largely used for enamel carious lesion management. In another study, the effectiveness of an annual application of SDF solution and of quarterly application of sodium fluoride varnish and chlorhexidine varnish were tested on sound and carious root surfaces in an institutionalised elderly population[116]. After 3 years there was no difference in carious lesion incidence between the three preventive measures observed but all three measures reduced carious incidence better than plaque control alone. It appears that evidence for the effectiveness of SDF solution in preventing carious lesion development is weak. Its effectiveness in cavitated carious lesions is presented later on.

Casein phosphopeptide-amorphous calcium phosphate agents

CPP-ACP is usually incorporated in chewing gum or in prophylactic dental paste with or without fluoride added, but tests have been also carried out on hard candy confections, sports drinks and milk incorporating CPP-ACP in their formulation. The effectiveness of such products in remineralising enamel is still being investigated, but results from *in-situ* and clinical studies show that CPP-ACP has a short-term remineralisation effect and a promising caries control effect for long-term clinical use[117].

Many laboratory and *in-situ* studies on the effectiveness of CPP-ACP have been published in the last two decades. As part of the process of obtaining clinical evidence, the number of clinical studies is currently still low. Studies show different outcomes, ranging from a superior effect of CPP-ACP to the control group(s)[79,80,118–120] to no additional effect over the control group[121,122].

Ozone

Ozone gas, the tri-atomic state of di-oxygen, was proposed as an antimicrobial agent that could reduce the number of micro-organisms on tooth surfaces. It is naturally produced in the presence of light or by different industrial processes. In dentistry, ozone is claimed to have a sterilising effect, killing cariogenic bacteria and subsequently leading to the arrest of carious lesions[123]. However, clinical studies have not achieved the same efficacy found in laboratory studies. Three systematic reviews concluded that there is no reliable evidence that the application of ozone gas to the surface of cavitated teeth arrests or reverses carious lesions. It does not appear to be a cost-effective additional step to the existing carious lesion management approaches[123–125].

Infiltration method

Caries infiltration has been proposed as an alternative for management of non-cavitated enamel carious lesions on approximal and buccal surfaces[126,127]. The infiltration concept is based on the penetration of a low-viscosity resin material (‘infiltrant’) in the subsurface enamel porosities of the lesion, the surface of which is previously etched and eroded with hydrochloric acid. As such, the infiltrant creates a diffusion barrier for hydrogen ions preventing lesion progression[128]. Laboratory and *in situ* studies have shown that infiltrants are capable of inhibiting the progression of natural carious lesions[129,130], and this has been confirmed by clinical studies. Resin infiltration combined with fluoride varnish application was superior in arresting superficial carious lesions in approximal surfaces of primary molars, compared to only fluoride varnish application, after 3 years[131]. The progression of enamel and dentine carious lesions on distal surfaces of first primary molars in young children after 2.5 years was lower for resin infiltration (46.4%) than for flossing these surfaces (71.4%)[131]. However, comparing the infiltration technique with sealing carious lesions in approximal surfaces of permanent teeth with a resin-bonding material did not show a significant difference in carious lesion progression after 3 years[132].

In summary, the evidence currently available indicates that resin infiltration of enamel lesions is a promising micro-invasive method for reducing the progress of enamel lesions[133]. Nevertheless, more randomised clinical trials are required for conclusive findings to be reached.

Pits and fissure sealants

Pits and fissures of permanent molars are particularly vulnerable to carious lesion development during and after tooth eruption[42,134]. The morphology of pits and fissures has been reported to be one of the main caries risk factors[135], with molars being more frequently affected than premolars[136]. Sealing aims to modify patent pits and fissures into smooth surfaces that are protected from bacterial colonisation and exposure to fermentable substrate and can be cleaned easily. The strategy is effective not only as a preventive measure, but also in arresting non-cavitated enamel carious lesions in pits and fissures[137]. The superiority of pit and fissure sealants over fluoride varnish application in the prevention of occlusal carious lesions has been reported[138].

Resin composites and glass-ionomer cements are the dental materials generally used to seal pits and fissures. A high-viscosity glass-ionomer is indicated for use with the ART sealant technique. It is generally accepted that resin composite sealants are retained longer than low- to medium viscosity glass-ionomer sealants[139,140]. However, which of the two types of sealant is more effective in inhibiting carious lesion development is not clear. Three systematic reviews comparing the carious lesion preventive effect of resin composite and low- and medium-viscosity glass-ionomer sealants have been conducted. However, these reviews did not provide evidence that either one of these materials was superior to the other[141–143]. Furthermore, there was no conclusive evidence that resin-modified glass-ionomer is superior to resin-based material used as fissure sealants in preventing dental caries[144].

Using high-viscosity glass-ionomer as the material for sealing pits and fissures according to the ART technique showed higher retention rates than low- and medium viscosity glass-ionomers[145]. The preventive effect of these ART sealants was high; the annual mean dentine lesion incidence rate over the first 3 years was 1%[146]. This finding appears to be better than reported for resin-based sealants in a systematic review[147]. Compared to resin composite sealants, the high-viscosity glass-ionomer ART sealants were more successful after 5 years in one of the two comparative studies available[148]. In the other study, the carious lesion preventive effect was equal after 2 years[149]. No resealing was performed in either study.

The preventive effect of the glass-ionomer sealant could be clarified by the presence of the material in the bottom of the fissures, even though the material could not be detected clinically[150–153]. Based on extensive evidence, the use of dental sealants is strongly recommended for all at-risk surfaces and shows good results for both resin composite material and high-viscosity glass-ionomer use with the ART approach. The latter can also be used in situations where electricity and running water are unavailable.

MINIMALLY INVASIVE OPERATIVE APPROACHES FOR MANAGING CAVITATED DENTINE CARIOUS LESIONS

Despite the plea made by WHO, the FDI World Dental Federation (FDI) and IADR to reduce the use of restorative materials, especially amalgam, through placing much greater emphasis on caries preventive measures, the need for treating cavitated teeth will remain into the foreseeable future.

Remineralisation of demineralised dentine

Continued presence of cariogenic plaque is the principal aetiological factor for demineralisation of both enamel and dentine. It seems obvious that depleting or reducing the cariogenic potential of dental plaque/biofilm is the most important activity for the maintenance of a healthy dentition. Whether this activity is being achieved at the plaque development site, through reduction of the frequency of sugar intake, or at the plaque destruction level through disturbance or removal of it, or by increasing the acid resistance of tooth tissues through mineralising agents, or by reducing the micro-organisms in plaque through disinfecting agents, tooth surfaces ought to be free from cariogenic plaque. This fact is also applicable to the tooth surfaces that give a dentinal cavity its shape. Clearly, a major reason for restoring a tooth cavity, from a cariology and preventive point of view, is to seal it and allow easy removal of dental plaque from the restored surfaces of the tooth. Concurrently, cavities are also restored to alleviate toothache and to restore form, function and aesthetics.

Similar to remineralisation of enamel carious lesions, remineralisation of dentine carious lesions is possible. The evidence for this phenomenon in open cavities is still very weak, but evidence for closed cavities that had remnants of dental plaque and retained decomposed dentine, and were filled with a restorative material, is abundant[11,12,154–158].

Within MID for dental caries, the principle guideline for managing a cavitated tooth is to remove decomposed (previously named ‘infected’) dentine, to leave demineralised (previously named ‘affected’) dentine behind and to restore the cleaned cavity with a restorative that has optimum biological and physical properties. Demineralised dentine has the ability to remineralise, as Fusayama *et al.*[159] showed decades ago. Remineralisation of demineralised dentine occurs through: (i) the function of the odontoblast process, providing calcium and phosphate from the vital pulp[159]; (ii) diffusion of ions (fluoride, calcium and phosphate) from materials placed on the floor of a restored cavity[10,12] and; (iii) contact of saliva with the carious lesion, providing calcium and phosphate, notably in root dentine in conjunction with oral hygiene measures[87].

Inadvertent retention of decomposed dentine and remnants of cariogenic plaque in a cavity skillfully restored with a well-manufactured restorative material, which seals the cavity, leads to the depletion of the cariogenic potential of those remnants of dental plaque. Systematic reviews have reported that micro-organisms left behind in cavities sealed over have no further ability to drive the caries process once they are cut off from the oral cavity, thus depriving micro-organisms of the source of metabolic nutrition required for their survival and for the production of acid that demineralises tooth surfaces. This situation leads to a change in the environment of cariogenic micro-organisms and inhibits their metabolic ability[160–163].

The adage ‘the seal is the deal’ should be adopted if oral healthcare professionals are to assist the ever-growing number of people with a functional natural dentition to keep their teeth healthy from youth into old age. In order to achieve ‘teeth for life for all’, it may even imply that under certain circumstances small dentine cavities can be sealed instead of receiving a restoration. It also calls for a redefinition of dental caries, away from it being labelled an infectious disease. Dental caries is an example of a behavioural disease with a bacterial component, particularly if it is accepted that de- and remineralisation cycles take place at tooth surfaces in all of us many times every day, without causing irreversible destruction. The actions taken by professional bodies such as the FDI and WHO, to have dental caries included in the United Nations list of non-communicable diseases, support this change. Calling dental caries a behavioural disease is in support of the principle that behavioural actions cause the disease, and as such, diet control and oral hygiene measures are required in order to manage carious lesion development as well as to avoid the onset of periodontal disease.

Ways of removing decomposed dentine from tooth cavities in the context of cavity preparation according to MID and evidence-based survival results of restored teeth are presented in the following section.

Appropriate excavation methods

According to the concept of Minimal Intervention Dentistry (MID), only the decomposed dentine needs to be removed from within the cavity. This then poses the question: which method removes decomposed dentine most effectively? In aiding this process, caries-detector dyes were introduced decades ago[9]. The dyes are very popular in certain parts of the world. However, as opposed to the initial intention to stain micro-organisms in decomposed dentine, subsequent studies have demonstrated that the dyes do not stain micro-organisms but rather stain the organic matrix of less mineralized dentine[164,165]. The dye also stained the enamel-dentine junction of freshly extracted caries-free teeth, because of the higher proportion of organic matrix present[165]. Thus, in removing dye stained dentine, the dental professional removes potentially remineralisable dentine, which is contrary to the intention of Minimal Intervention Dentistry.

A number of laboratory studies, using different detection techniques and endpoints to delineate decomposed dentine, have investigated the efficacy and effectiveness of methods for its removal. Considering the variation in study designs, it appeared that rotating round metal burs have the tendency to over-prepare cavities[166,167] and that laser and oscillation techniques under-prepare cavities[167–169]. Self-limiting burs made of polymer and ceramic material have been introduced but found to under-prepare cavities[167,168]. The most appropriate decomposed dentine removal methods had used either a chemo-mechanically applied gel (Carisolv, Sweden) or a metal hand excavator[166–170]. The efficacy of hand excavation in comparison to chemo-mechanical removal of decomposed dentine was tested in a clinical study which showed no difference between the two methods; 94% of chemo-mechanical and 89% of hand excavated cavities were free of visible decomposed dentine[171].

Whilst the use of a chemo-mechanical gel seems most effective in removing decomposed dentine adequately, the fact that its excavation takes a relatively long time cannot be ignored, and neither can the comparative increased cost. This then leaves hand excavation with a sharp metal excavator as a very effective method for removing decomposed carious dentine prior to restoration. The time involvement in cleaning the cavity may be an issue and most likely will, among others, depend on the operators' experience in using hand excavators. Study findings vary from no significant difference for cavity preparation in primary teeth between rotary instrumentation and hand excavation[172] to rotary instrumentation's being faster (2 minutes) than hand excavation[173].

Disinfecting excavated cavities

Earlier it has been stated that micro-organisms, retained under a well-sealed restoration, are reduced in numbers over time and have no potential to further demineralise the dentine, provided that the seal remains secured. It is often noticed that in such a situation the demineralised dentine remineralises over time[10,157]. The question that then arises is: what is the added advantage of applying a disinfectant to a cavity after it has been adequately cleaned? Common disinfection agents are 2% chlorhexidine solution and, more recently, ozone gas. Cavity disinfection by chlorhexidine solution is only superficial and not-effective as has been shown in restoration survival studies using glass-ionomers after 2 years[174] and 5-years[175]. Studies investigating the effect on the restoration survival rate of cavity disinfection with ozone gas prior to restoration, are lacking. No data are available to support cavity disinfection prior to restoration.

Restorative materials

In past decades amalgam and silicate cement were the two most popular dental materials used for restoring cavities in posterior and anterior teeth respectively, and these materials have been superseded to a large extent by resin-based and glass-ionomer-based materials. Both types of adhesive material are constantly being modified to mimic the physiological (behavioural) and physical characteristics of enamel and dentine. In particular, glass-ionomer restorative materials have undergone major changes during the last decades. Medium-viscosity glass-ionomer was recommended initially for non-stress bearing surfaces. However, the latest systematic review on restoration comparison concluded that the survival rates of high-viscosity glass-ionomer restorations placed in stress-bearing surfaces in both deciduous and permanent dentitions were equal to or higher than those of comparable amalgam restorations[176].

Minimally invasive operative treatment approaches and adhesive materials and systems go hand-in-hand.

Resin-based and glass-ionomer-based materials have their advantages and disadvantages. The dental practitioner ought to know the chemistry, characteristics and handling features of the restorative material that (s)he is using. Proper application of that knowledge in clinical practice is the basis for a long lasting restoration.

Restorative therapy

Deciduous dentition

According to a systematic review, covering studies carried out between 1988 and 2003, the mean annual failure rate for Class I and Class II amalgam restorations in deciduous posterior teeth was 6.6% and 7.6%, respectively[177]. The mean annual failure rate for comparable resin composite restorations (Class I and Class II combined), assessed according to the Ryge and USPHS criteria, varied between 0% and 15%[177]. Many of the included studies had assessed restorations placed in cavities designed according to the principle of 'extension for prevention'.

It goes without saying that those cavity designs, proposed by G.V. Black, have no place in MID. On the contrary, the contemporary design principles is tissue-saving: 'prevention of extension'[7]. In addition to the traditional techniques of excavation with a round bur or hand excavator and restoration of the cavity with preformed crowns, amalgam, resin-based or glass-ionomer-based materials in a tooth tissue-preserving manner, minimally invasive treatment approaches that do not use electrically driven equipment and running water are available. These are presented below.

Atraumatic restorative treatment (ART)

This approach uses hand instruments for opening cavities further, only to the extent required for removing decomposed carious dentine. The cavity is then cleaned, restored with a high-viscosity glass-ionomer and adjacent pits and fissures are sealed concurrently[178]. Evidence-based studies have shown ART restorations in single-surface cavities in deciduous posterior teeth to survive as long as comparable amalgam restorations[176]. The mean annual failure rate of these ART restorations over the first two years was 3.5%[146]. Multiple-surface ART restorations in deciduous posterior teeth have a lower survival rate than single-surface ART restorations but they appear not to differ from either comparable resin-composite restorations[174] or amalgam restorations[172,179]. In addition to the high survival rates, other advantages of the ART approach include the absence of noise and vibration and the reduced need to administer local anaesthesia[180,181].

Because electricity and running water are not required, ART is a proven caries management approach for use in outreach situations such as in schools and in rural areas. The survival rates of ART restorations produced in office-based practice and those produced in primary schools do not differ[146]. Using the ART approach, both preventive and restorative care can be provided to a larger number of people than is possible through use of the traditional restorative therapy.

In comparing restoration survival rates of ART high-viscosity glass-ionomer and those produced through use of the traditional therapy using either amalgam or resin-composite, one has to take into account the evaluation criteria that have been used in these studies. ART restorations have predominantly been evaluated according to the ART restoration criteria, while traditionally produced restorations have mainly been evaluated according to the United States Public Health Service criteria or the FDI criteria. The ART restoration criteria turned out to be more stringent than both the FDI criteria[182] and the USPHS criteria[183] for restorations in permanent teeth. The difference in restoration survival rates, assessed by both the ART and the USPHS criteria was 22% and 27% for single- and multiple surfaces, respectively being higher for restorations assessed using the USPHS criteria over a 10-year period. This large difference in outcome shows that evaluating a treatment, whether it concerns a restoration, a crown or a sealant, should be done by internationally accepted appropriate criteria. Furthermore, restoration survival rates of different restorative materials should only be compared if the same assessment criteria have been used.

Hall technique

Another minimally invasive restoration therapy that may be helpful in reducing the treatment burden of cavitated dentine carious lesions is the Hall technique[184]. A prefabricated metal crown is cemented over the cavitated tooth, using a low-viscosity glass-ionomer after removing debris but without removal of decomposed carious dentine. However few studies have been carried out. A 5-year practice-based study, in which cavitated teeth treated with the Hall technique were compared with those restored using common practice in Scotland showed significantly better performance for the Hall-treated teeth than those treated by general dentists using standard restorations[185]. More studies are required before the Hall technique can be recommended for general use.

Non-restorative therapy

Plaque removal from cavities in deciduous teeth

The vast majority of cavitated carious lesions in deciduous teeth are being neither restored nor extracted, a finding which is prevalent in all countries in the world. Figures based on the WHO data base vary from, on average, 80% in high-income countries to 95% in low income countries[186]. This unwanted situation should make the dental profession consider whether the 'cure' for cavitated carious lesions in deciduous teeth should always be the placement of a traditional restoration. Alternative restorative procedures have been discussed in the previous section. The important question then arises: what about guiding a cavitated deciduous tooth towards exfoliation without restoring it while ensuring infection free, symptom free, general anaesthesia free exfoliation? 'A clean tooth surface doesn't decay' is the slogan. What would happen if the inner cavity surface were kept free from cariogenic plaque by brushing the cavity clean with a fluoride-containing toothpaste? Surely, the caries process would most probably cease. It is obvious that not all cavities are suitable for 'internal' cleaning with a brush and paste, as pulp degeneration in deciduous teeth is far more rapid than in permanent teeth, especially with approximal lesions. The larger ones can be cleaned more easily than the smaller ones, and this approach has been investigated. A treatment protocol based on restoring small cavities, in this case with ART, and cleaning medium and large cavities with a toothbrush and toothpaste compared with the traditional treatment protocol using amalgam and the full ART protocol, showed no difference in survival rates of teeth treated by the three protocols[187]. This outcome seems to be consistent with the outcome from a retrospective practice-based study which reported that 84% of untreated cavitated teeth exfoliated symptom-free[188]. It is expected that more results on this approach will appear in future but until they are available, caution should be taken when cleaning cavities is part of a non-restorative treatment protocol for management of cavitated carious lesions.

Application of silver diammine fluoride

Consistent with the rationale for stopping the demineralising effect of cariogenic plaque in cavities by removing it, 38% silver diammine fluoride (SDF) has been used in cavitated carious lesions. Three studies have been published, all with different application frequencies[189–191]. It seems reasonable to conclude from these studies that twice-yearly application of 38% SDF may be able to arrest the carious process in the cavity. A single application of 38% SDF within an interval of 2 years, as suggested for use in deprived communities, appears to have a lesser effect in arresting carious lesions over 2 years compared to tooth brushing alone[191]. This non-invasive treatment has the advantage that trained dental auxiliary personnel can apply the solution, thus reaching more children than is achieved with the traditional restorative treatment. Nevertheless, this approach requires additional investigation before it can be recommended for general use.

Permanent dentition

Carious lesions in anterior teeth should preferably be restored using a proven anterior resin composite because of its superior aesthetic performance. According to a systematic review regarding effectiveness of adhesive materials bonded to enamel and dentine in non-carious cervical lesions, the glass-ionomers were superior to resin-based adhesives[192], the buccal and cervical carious lesions in the posterior area are best restored using a (resin-modified) glass-ionomer, while three-step etch-and-rinse adhesive and a resin composite was the second best. The use of a hand excavator for removing decomposed carious tissues near the gingival margin may cause less bleeding than may occur when a rotary instrument is used and may increase the survival rate of the restoration.

According to a systematic review, the mean annual failure rate for single-surface amalgam and resin composite restorations in permanent posterior teeth, evaluated according to the USHPS criteria, are 2.1% and 1.8%, respectively[193]. According to a meta-analysis, the use of the stringent ART restoration criteria showed the mean annual failure rate for high-viscosity glass-ionomers in comparable tooth surfaces to be 4%[146]. It is safe to conclude that, had these glass-ionomer restorations been evaluated according to the USPHS criteria, the mean annual failure rate would be lower. They would, most probably, be comparable with the survival rate of amalgam and resin composite restorations.

Restoring multiple-surfaces in posterior teeth is best done using amalgam or resin composite materials following 'the box only' cavity design[17]. Evidence regarding the success of tunnel restorations has not been increased since a previous publication on this minimal intervention approach[17]. Therefore, this procedure cannot be recommended for general use. Studies that have assessed multiple-surface high-viscosity glass-ionomer restorations in permanent teeth are negligible and evidence for a predictive outcome, if this material is used in multiple-surfaces, is not available.

EXAMINING RESTORATIONS, AND WHEN REPAIR OR REPLACEMENT IS REQUIRED. HOW SUCCESSFUL ARE REPAIRED RESTORATIONS?

Examining restorations

The presence of defective restorations or restorations with the clinical diagnosis of secondary caries is one of the most frequent problems encountered by general practitioners today. The diagnosis is inconsistent among dental practitioners and often does not rely on objective criteria[194–196]. If in doubt, most general dental practitioners choose replacement as opposed to options of non-surgical treatment, including systematic restoration monitoring. Restoration replacement is especially common for restorations not originally placed by the evaluating practitioner[197–199]. A dental practice-based research study involving 197 clinicians from the USA and Scandinavian countries, and close to 10,000 restorations, indicated that practitioners chose replacement over repair of defective dental restorations in over 75% of cases[200]. The same study confirmed that practitioners who did not place the original restoration were more likely to replace it than practitioners who did.

In summary, replacement of restorations constitutes over 50% of the work performed by general dental practitioners in their practices[201,202] and it has contributed to the perpetuation of the 'Repeat Restoration Cycle'[6]. Consequently, the diagnostic finding 'defective' for an existing restoration is a critical step in treatment planning and it invariably affects the longevity of the restored tooth.

Secondary caries and staining of the margins of existing restorations are the most common reasons for restoration replacement in permanent and primary teeth[201,203,204]. Without objective criteria, it is difficult to differentiate secondary caries from marginal staining clinically[205]. Despite the fact that some studies have associated microleakage with secondary carious lesion formation[206,207], the majority of the evidence[208–210] demonstrates no relationship between the development of secondary carious lesions and the size of the leakage or gap alongside an amalgam restoration, except in cases in which the crevice exceeds 400 μm[210]. Although the criteria for the diagnosis of a defective restoration may be based solely on visual and tactile examination, the subsequent management plan for this restored tooth should be based on the caries risk assessment of the patient as well.

When is repair or replacement required?

Laboratory and clinical studies have shown that removal of the existing restoration will remove significant healthy tooth structure, subsequently resulting in larger dental restorations[211–213]. The removal of existing restorations may also cause additional stress on the tooth, with possible pulp reaction to thermal, chemical, bacterial, or mechanical stimuli[214,215], depending on the size and depth of the existing restoration. Therefore, the decision to replace existing restorations should be taken cautiously, as it may significantly affect the remaining tooth structure and, consequently, impact the longevity of future restorations and the lifespan of the tooth. Studies have demonstrated that replacing an existing restoration will not necessarily guarantee that the new restoration will surpass the clinical performance of other alternative treatments such as repair, sealing or monitoring[216–218].

Long-term clinical studies have also shown that when alternative treatments fail, the failure usually takes place within 24 months[216,217]. When the clinician is evaluating an existing restoration with one or more localized clinical features that deviate from ideal and the restoration is considered defective, the clinician should assess whether the tooth in question will truly benefit from a new restoration. When the practitioner is faced with a borderline situation, the patient's past dental history and current caries risk status[219,220], and the best treatment for the tooth in question should be considered. If the practitioner is unsure whether the defective area can be removed by polishing or by sealing the affected area, another conservative and predictable approach would be to repair the restoration by removing the deteriorated area and re-restoring this area only. Generally, replacement should only take place if the practitioner cannot properly manage the defective areas without removing the entire restoration, or if there are pulpal symptoms.

How successful are repaired restorations?

Minimal Intervention Dentistry aimed to limit unnecessary removal of healthy tooth structure, and repair of defective restorations is one of its strategies. Although the repair of resin composite restorations has been investigated extensively and found successful[221–223], dental practitioners do not routinely consider this treatment option in the management of defective restorations.

The repair of resin composite restorations is taught in most, but not in all dental schools in North America, the United Kingdom, Ireland, Germany and Scandinavia[224–227]. Although considered a long lasting treatment by the schools teaching this practice, a practice-based research study showed that only practitioners who practiced in non-fee service settings, practitioners with fewer years since graduation from dental school, and practitioners who assessed caries risk, chose preventive treatment options more often than replacement when assessing defective restorations[228]. The preference for replacement of restorations may be the result of a complex interplay between the lack of clear standards for replacing restorations and lack of an existing reimbursement for these treatments. That same study reported that general practitioners would most likely intervene surgically in a defective resin composite restoration but not in a defective amalgam restoration[228].

So far, prospective studies have shown that repaired restorations in permanent teeth have the same or increased longevity as restorations that were replaced completely[217,218,228]. Repair treatment remained stable over a 7-year observation period[216,217]. Additionally, the reason that repaired restorations may even outlast those that were replaced probably relates to the fact that most of the restoration's original form is kept intact, limiting the introduction of new elements that can affect the success of the restoration. When other restoration stress factors are considered, such as stress on the tooth, post-operative sensitivity, and re-exposure of the dentinal tubules with possible pulpal reactions to thermal or mechanical stimulus[214,215], damage to the adjacent tooth and the possibility of more complex restorations, it makes perfect sense to pursue the repair of defective restorations as a predictable and conservative approach to preserving tooth structure. A recent overview regarding restoration margins concludes that margin defects, without visible evidence of soft dentine on the wall or base of the defect, should be monitored, repaired or resealed, in lieu of total restoration replacement[229].

Besides being a successful treatment, restoration repair is also practical. Defective restorations can be repaired more quickly and with lower operational costs than replacement. Therefore, repaired restorations could present a reduction in patient and/or the third party payers' expenses which would potentially increase the number of individuals who could afford dental care. The cost of care and oral health are severely impacted by the replacement of existing restorations. Examining outcomes of alternative treatment to the replacement of restorations and establishing consistent criteria that will affect general practitioners' treatment decisions is a critical issue

Table 1 Summary of carious control measurements and their evidence-based effectiveness

Measurements	Effectiveness	Reference
Fluoride agents		
Community-based methods	Water fluoridation: associated with a decrease in carious lesions for both children and adults. Salt fluoridation: there is an indication that it can be effective in caries prevention when compared to no fluoride exposure. Nevertheless, this finding cannot be considered conclusive, as there is a lack of quality studies regarding the topic. Milk fluoridation: insufficient evidence that proves its effectiveness	MacDonagh *et al.*[99], Griffin *et al.*[100], Armfield[98], Yeung *et al.*[101], Ellwood *et al.*[102], Yengopal *et al.*[103]
Professional-based methods	Fluoride gels and varnish are able to reduce the incidence of dental carious lesions	Marinho[3]
Individual methods (toothpaste, mouthwash)	Fluoride toothpaste and mouthwashes are effective in caries prevention. Fluoride toothpaste is one of the major factors related to the decline of caries prevalence in high-income countries	Carvalho *et al.*[105], Petterson and Bratthall[104], Marinho[3]
Silver Diamine Fluoride	Lack of evidence showing that SDF is effective as a caries control agent in non-cavitated lesions. The current information shows that it is as effective as plaque control and sealants	Braga *et al.*[115]
Non-Fluoride agents		
Sugar Substitutes	Xylitol and sorbitol are the most frequently used sugar substitutes. The use of sugar-free dental chewing gum had proved to be effective for carious lesion control on school premises	Ly *et al.*[90,91]
Chlorhexidine	Evidence regarding chlorhexidine gel and varnish for carious control is inconclusive, and there is a lack of evidence that chlorhexidine rinses are useful as a preventive measure against dental caries	Zhang *et al.*[108]
Casein Phosphopeptide amorphous calcium phosphate	CPP-ACP has a short-term remineralization effect and a promising caries control effect for long-term clinical use. More clinical studies are needed to confirm the last	Yengopal and Mickenautsch[117]
Ozone therapy	No reliable evidence that the application of ozone gas stops or reverses carious lesions	Rickard *et al.*[124], Brazzelli *et al.*[123], Azarpazhooh and Limeback[125]
Infiltration	Current evidence indicates that resin infiltration of non-cavitated lesions is a promising therapy to avoid carious lesion progression in enamel and dentine	Paris and Meyer-Lueckel[129,130], Ekstrand *et al.*[131], Martignon *et al.*[132]
Pit and fissure sealants	It is an effective measure for both carious prevention and arresting non-cavitated carious lesions. Although resin-based sealants are retained longer than those performed with glass-ionomer cements, none of them were shown to be superior in terms of caries prevention	Beiruti *et al.*[141], Griffin *et al.*[137], Yengopal *et al.*[117], Yengopal[103]

Table 2 Survey results on MID application in undergraduate curricula

Country	University	Education year	Department	Starting year
Australia	Melbourne	2–4–5	Restorative, Community, Cariology	2000
Brasil	Brasilia	2	Paediatric	2008
Israel	Jarusalem	4	Restorative	2004
Italy	Pavia	Master course	Paediatric	1999
Japan	Osaka	4/6	Restorative	?
Kuwait	Kuwait	4–5–6	Paediatric, Restorative, Preventive	1998
Lebanon	Beirut	4–5	Paediatric, Restorative	2005
Netherlands	Nijmegen	1–6	Cariology, Restorative, Paediatric	1998
Romania	Iasi	3	Cariology, Restorative	2007
Sweden	Malmö	2–3–4–5	Cariology Paediatric, Restorative, Prosthodontics	2001
Turkey	Ege	3–4–5	Paediatric, Restorative	2006
UK	Dundee	2–3–4–5	Paediatric, Restorative	2005

that may profoundly change the over-treatment of existing restorations.

In summary, dental practitioners should consider repairing truly defective restorations, an appropriate minimal invasive operative intervention worth pursuing.

INTEGRATION OF MID IN THE DENTAL CURRICULUM

If MID is to make an impact in supporting the aim of 'Teeth for Life', it ought to be included in the dental curriculum. As a literature search did not reveal sufficient information on the state of integration of Minimal Intervention Dentistry into dental curricula, a survey was carried out amongst 50 dental schools in 50 countries via the internet. Unfortunately, the response rate was rather low: only 12 schools responded (*Table 2*). This reveals that MID has been introduced to students mainly during their clinical education years in the subjects 'restorative dentistry' and/or 'paediatric dentistry', and/or 'preventive dentistry' and/or 'cariology'. It was not possible to obtain reliable data on the content of the lectures or on whether MID was effectively taught and had made a difference.

It is suggested that Policy Statements of the FDI and those of other major dental (educational) institutions should support and advocate the incorporation of the principles of MID across the entire dental curriculum. It is important that faculty lecturers and clinical instructors are open to accepting changes in patient care based on evidence-based research findings. Current and future dental professionals should recognize themselves as oral physicians and counselors rather than only dental surgeons.

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Conflict of interest

All authors declare to have no conflict of interest.

REFERENCES

1. Backer Dirks O, Houwink B, Kwant GW. The results of 6 1/2 years of artificial fluoridation of drinking water in the Netherlands. The Tiel-Culemborg experiment. *Arch Oral Biol* 1961 5: 284–300.
2. Groeneveld A, Van Eck AA, Backer Dirks O. Fluoride in caries prevention: is the effect pre- or post-eruptive? *J Dent Res* 1990 69: 751–755.
3. Marinho VC. Evidence-based effectiveness of topical fluorides. *Adv Dent Res* 2008 20: 3–7.
4. Fejerskov O. Changing paradigms in concepts on dental caries: consequences for oral health care. *Caries Res* 2004 38: 182–191.
5. Frencken JE, Holmgren CJ, van Palenstein Helderman WH. *Basic Package of Oral Care*. Nijmegen: WHO Collaborating Centre for Oral Health Care Planning and Future Scenarios; 2002.
6. Elderton R. Principles in the management and treatment of dental caries. In: Elderton R, editor. *The Dentition and Dental Care*. Oxford: Heinemann Medical Books; 1990. p. 237–262.
7. Peters MC, McLean ME. Minimally invasive operative care. I. Minimal intervention and concepts for minimally invasive cavity preparations. *J Adhes Dent* 2001 3: 7–16.
8. Massler M. Pulpal reactions to dental caries. *Int Dent J* 1967 17: 441–460.
9. Fusayama T. The process and results of revolution in dental caries treatment. *Int Dent J* 1997 47: 157–166.
10. Ngo HC, Mount G, Mc Intyre J *et al.* Chemical exchange between glass-ionomer restorations and residual carious dentine in permanent molars: an in vivo study. *J Dent* 2006 34: 608–613.
11. Alves LS, Fontanella V, Damo AC *et al.* Qualitative and quantitative radiographic assessment of sealed carious dentin: a 10-year prospective study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2010 109: 135–141.
12. Peters MC, Bresciani E, Barata TJ *et al.* In vivo dentin remineralization by calcium-phosphate cement. *J Dent Res* 2010 89: 286–291.

13. Mount GJ. Minimal treatment of the carious lesion. *Int Dent J* 1991 41: 55–59.
14. Dawson AS, Makinson OF. Dental treatment and dental health. Part 1. A review of studies in support of a philosophy of minimum intervention dentistry. *Aust Dent J* 1992a 37: 126–132.
15. Dawson AS, Makinson OF. Dental treatment and dental health. Part 2. An alternative philosophy and some new treatment modalities in operative dentistry. *Aust Dent J* 1992b 37: 205–210.
16. Horowitz AM. Introduction to the symposium on minimal intervention techniques for caries. *J Public Health Dent* 1996 56: 133–134.
17. Tyas MJ, Anusavice KJ, Frencken JE *et al.* Minimal intervention dentistry–a review. FDI commission project 1-97. *Int Dent J* 2000 50: 1–12.
18. McIntyre J. Minimal intervention dentistry. *Ann R Australas Coll Dent Surg* 1994 12: 72–79.
19. Burke FJT. Minimal intervention isn't just small cavities! *Dent Update* 2008 35: 509.
20. Ricketts DN, Kidd EA, Smith BG *et al.* Clinical and radiographic diagnosis of occlusal caries: a study in vitro. *J Oral Rehabil* 1995 22: 15–20.
21. Ricketts DN, Whaites EJ, Kidd EA *et al.* An evaluation of the diagnostic yield from bitewing radiographs of small approximal and occlusal carious lesions in a low prevalence sample in vitro using different film types and speeds. *Br Dent J* 1997 182: 51–58.
22. Angnes V, Angnes G, Batisttella M *et al.* Clinical effectiveness of laser fluorescence, visual inspection and radiography in the detection of occlusal caries. *Caries Res* 2005 39: 490–495.
23. Davies GM, Worthington HV, Clarkson JE *et al.* The use of fibre-optic transillumination in general dental practice. *Br Dent J* 2001 191: 145–147.
24. Hamilton JC, Gregory WA, Valentine JB. DIAGNOdent measurements and correlation with the depth and volume of minimally invasive cavity preparations. *Oper Dent* 2006 31: 291–296.
25. Neves AA, Coutinho E, De Munck J *et al.* Does DIAGNOdent provide a reliable caries-removal endpoint? *J Dent* 2011 39: 351–360.
26. Zandona AF, Zero DT. Diagnostic tools for early caries detection. *J Am Dent Assoc* 2006 137: 1675–1684.
27. Diniz MB, Boldieri T, Rodrigues JA *et al.* The performance of conventional and fluorescence-based methods for occlusal caries detection: an in vivo study with histologic validation. *J Am Dent Assoc* 2012 143: 339–350.
28. Chawla N, Messer LB, Adams GG *et al.* An in vitro comparison of detection methods for approximal carious lesions in primary molars. *Caries Res* 2012 46: 161–169.
29. World Health Organization. *Oral Health Surveys. Basic Methods*, 2nd ed. Geneva: WHO; 1977.
30. Pitts N. ICDAS – an international system for caries detection and assessment being developed to facilitate caries epidemiology, research and appropriate clinical management. *Community Dent Health* 2004 21: 193–198.
31. de Amorim RG, Figueiredo MJ, Leal SC *et al.* Caries experience in a child population in a deprived area of Brazil, using ICDAS II. *Clin Oral Investig* 2012 16: 513–520.
32. Cadavid AS, Lince CM, Jaramillo MC. Dental caries in the primary dentition of a Colombian population according to the ICDAS criteria. *Braz Oral Res* 2010 24: 211–216.
33. Eggertsson H, Agustsdottir H, Gudmundsdottir H *et al.* Caries prevalence of permanent teeth: a national survey of children in Iceland using ICDAS. *Community Dent Oral Epidemiol* 2010 38: 299–309.
34. Nyvad B, Machiulskiene V, Baelum V. Reliability of a new caries diagnostic system differentiating between active and inactive caries lesions. *Caries Res* 1999 33: 252–260.
35. Nyvad B, Machiulskiene V, Fejerskov O *et al.* Diagnosing dental caries in populations with different levels of dental fluorosis. *Eur J Oral Sci* 2009 117: 161–168.
36. Séllos MC, Soviero VM. Reliability of the Nyvad criteria for caries assessment in primary teeth. *Eur J Oral Sci* 2011 119: 225–231.
37. Monse B, Heinrich-Weltzien R, Benzian H *et al.* PUFA – an index of clinical consequences of untreated dental caries. *Community Dent Oral Epidemiol* 2010 38: 77–82.
38. Frencken JE, de Amorim RG, Faber J *et al.* The Caries Assessment Spectrum and Treatment (CAST) index: rational and development. *Int Dent J* 2011 61: 117–123.
39. Figueiredo MJ, de Amorim RG, Leal SC *et al.* Prevalence and severity of clinical consequences of untreated dentine carious lesions in children from a deprived area of Brazil. *Caries Res* 2011 45: 435–442.
40. de Souza AL, van der Sanden WJM, Leal SC *et al.* Caries assessment spectrum and treatment (CAST) index: face and content validation. *Int Dent J* 2012 62: 270–276.
41. Demers M, Brodeur JM, Simard PL *et al.* Caries predictors suitable for mass-screenings in children: a literature review. *Community Dent Health* 1990 7: 11–21.
42. Powell LV. Caries prediction: a review of the literature. *Community Dent Oral Epidemiol* 1998 26: 361–371.
43. Harris R, Nicoll AD, Adair PM *et al.* Risk factors for dental caries in young children: a systematic review of the literature. *Community Dent Health* 2004 21: 71–85.
44. Twetman S, Fontana M. Patient caries risk assessment. *Monogr Oral Sci* 2009 21: 91–101.
45. Bratthall D, Hansel Petersson G. Cariogram–a multifactorial risk assessment model for a multifactorial disease. *Community Dent Oral Epidemiol* 2005 33: 256–264.
46. Bratthall D, Hänsel Petersson G, Stjernswärd JR. *CARIOGRAM Manual*. 2004-04-02 ed. Malmö, Sweden: Malmö University; 2004.
47. Fontana M, Zero DT. Assessing patients' caries risk. *J Am Dent Assoc* 2006 137: 1231–1239.
48. Batchelor PA, Sheiham A. The distribution of burden of dental caries in schoolchildren: a critique of the high-risk caries prevention strategy for populations. *BMC Oral Health* 2006 6: 3.
49. Rose G. Sick individuals and sick populations. *Int J Epidemiol* 1985 14: 32–38.
50. Rose G. Sick individuals and sick populations. *Int J Epidemiol* 2001 30: 427–432.
51. Page J, Weld JA, Kidd EA. Caries control in health service practice. *Br Dent J* 2010 208: 449–450.
52. Hausen H, Seppa L, Poutanen R *et al.* Noninvasive control of dental caries in children with active initial lesions. A randomized clinical trial. *Caries Res* 2007 41: 384–391.
53. Selwitz RH, Ismail AI, Pitts NB. Dental caries. *Lancet* 2007 369: 51–59.
54. Loesche WJ. Clinical and microbiological aspects of chemotherapeutic agents used according to the specific plaque hypothesis. *J Dent Res* 1979 58: 2404–2412.
55. Kidd EAM, Fejerskov O. What constitutes dental caries? Histopathology of carious enamel and dentin related to the action of cariogenic biofilms. *J Dent Res* 2004 83:C35–C38.
56. Geddes DA, Jenkins GN. Intrinsic and extrinsic factors influencing the flora of the mouth. *Soc Appl Bacteriol Symp Ser* 1974 3: 85–100.

57. Geddes DA. Diet patterns and caries. *Adv Dent Res* 1994 8: 221–224.
58. van Houte J, Sansone C, Joshipura K *et al.* Mutans streptococci and non mutans streptococci acidogenic at low pH, and in vitro acidogenic potential of dental plaque in two different areas of the human dentition. *J Dent Res* 1991 70: 1503–1507.
59. Takahashi N, Nyvad B. Caries ecology revisited: microbial dynamics and the caries process. *Caries Res* 2008 42: 409–418.
60. Modesto M, Biavati B, Mattarelli P. Occurrence of the family Bifidobacteriaceae in human dental caries and plaque. *Caries Res* 2006 40: 271–276.
61. Haukioja A, Söderling E, Tenovuo J. Acid production from sugars and sugar alcohols by probiotic Lactobacilli and Bifidobacteria in vitro. *Caries Res* 2008 42: 449–453.
62. Ruby J, Goldner M. Nature of symbiosis in oral disease. *J Dent Res* 2007 86: 8–11.
63. Kidd E. The implications of the new paradigm of dental caries. *J Dent* 2011 39(Suppl 2): S3–S8.
64. Aoba T, Fejerskov O. Dental fluorosis: chemistry and biology. *Crit Rev Oral Biol Med* 2002 13: 155–170.
65. Featherstone JDB. The continuum of dental caries–evidence for a dynamic disease process. *J Dent Res* 2004 83: C39–C42.
66. Vale GC, Tabchoury CPM, Arthur RA *et al.* Temporal relationship between sucrose-associated changes in dental biofilm composition and enamel demineralization. *Caries Res* 2007 41: 406–412.
67. ten Cate JM. Current concepts on the theories of the mechanism of action of fluoride. *Acta Odontol Scand* 1999 57: 325–329.
68. Yamazaki H, Litman A, Margolis HC. Effect of fluoride on artificial caries lesion progression and repair in human enamel: regulation of mineral deposition and dissolution under in vivo-like conditions. *Arch Oral Biol* 2007 52: 110–120.
69. de Leeuw NH. Resisting the onset of hydroxyapatite dissolution through the incorporation of fluoride. *J Phys Chem B* 2004 108: 1809–1811.
70. Watson PS, Pontefract HA, Devine DA *et al.* Penetration of fluoride into natural plaque biofilms. *J Dent Res* 2005 84: 451–455.
71. Featherstone JD. The caries balance: contributing factors and early detection. *J Calif Dent Assoc* 2003 31: 129–133.
72. Featherstone JD. Caries prevention and reversal based on the caries balance. *Pediatr Dent* 2006 28: 128–132.
73. Reynolds EC. Calcium phosphate-based remineralization systems: scientific evidence? *Austr Dent J* 2008 53: 268–273.
74. Kato K, Nakagaki H, Arai K *et al.* The influence of salivary variables on fluoride retention in dental plaque exposed to a mineral-enriching solution. *Caries Res* 2002 36: 58–63.
75. Reynolds EC. Casein phosphopeptide-amorphous calcium phosphate: the scientific evidence. *Adv Dent Res* 2009 21: 25–29.
76. Cochrane NJ, Saranathan S, Cai F *et al.* Enamel subsurface lesion remineralisation with casein phosphopeptide stabilised solutions of calcium, phosphate and fluoride. *Caries Res* 2008 42: 88–97.
77. Reynolds EC, del Rio A. Effect of casein and whey-protein solutions on caries experience and feeding patterns of the rat. *Arch Oral Biol* 1984 29: 927–933.
78. Reynolds EC, Cai F, Cochrane NJ *et al.* Fluoride and casein phosphopeptide-amorphous calcium phosphate. *J Dent Res* 2008 87: 344–348.
79. Morgan MV, Adams GG, Bailey DL *et al.* The anticariogenic effect of sugar-free gum containing CPP-ACP nanocomplexes on approximal caries determined using digital bitewing radiography. *Caries Res* 2008 42: 171–184.
80. Bailey D, Adams G, Tsao C *et al.* Regression of post-orthodontic lesions by a remineralizing crème. *J Dent Res* 2009 88: 1148–1153.
81. Reynolds EC, Cai F, Shen P *et al.* Retention in plaque and remineralization of enamel lesions by various forms of calcium in a mouthrinse or sugar-free chewing gum. *J Dent Res* 2003 82: 206–211.
82. Holt C, Wahlgren NM, Drakenburg T. Ability of a b-casein phosphopeptide to modulate the precipitation of calcium phosphate by forming amorphous dicalcium phosphate nanoclusters. *Biochem J* 1996 314: 1035–1039.
83. Reynolds EC. Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. *J Spec Care Dentist* 1998 18: 8–16.
84. Peters MC. Strategies for non-invasive demineralized tissue repair. In: Young DA, Fontana M, Wolff MS, editors. *Current Concepts in Cariology. Dent Clin North Am* 2010 54. p. 507–525.
85. Nyvad B, Fejerskov O. Active root surface caries converted into inactive caries as a response to oral hygiene. *Scand J Dent Res* 1986 94: 281–284.
86. Burt BA, Pai S. Sugar consumption and caries risk: a systematic review. *J Dent Educ* 2001 65: 1017–1023.
87. van Loveren C, Duggal MS. The role of diet in caries prevention. *Int Dent J* 2001 51: 399–406.
88. Ly KA, Milgrom P, Rothen M. Xylitol, sweeteners and dental caries. *Pediatr Dent* 2006 28: 154–163.
89. Mickenaustch S, Leal SC, Yengopal V *et al.* Sugar free chewing gum and dental caries: a systematic review. *J Appl Oral Sci* 2007 15: 83–88.
90. Ly KA, Milgrom P, Rothen M. The potential of dental protective chewing gum in oral interventions. *J Am Dent Assoc* 2008a 139: 553–563.
91. Ly A, Riedy CA, Milgrom P *et al.* Xylitol gummy bear snacks: a school-based randomized clinical trial. *BMC Oral Health* 2008b 8: 20.
92. Antonio AG, Pierro VS, Maia LC. Caries preventive effects of xylitol-based candies and lozenges: a systematic review. *J Public Health Dent* 2011 71: 117–124.
93. Nadimi H, Wesamaa H, Janket SJ *et al.* Are sugar-free confections really beneficial for dental health? *Br Dent J* 2011 211: E15.
94. Kalsbeek H, Verrips GH. Consumption of sweet snacks and caries experience of primary school children. *Caries Res* 1994 28: 477–483.
95. Centers for Disease Control and Prevention. 2012. Available from: http://www.cdc.gov/fluoridation. Accessed 06 October 2012.
96. National Health and Medical Council Research. *A Systematic Review of the Efficacy and Safety of Fluoridation. Part A: Review of Methodology and Results*. Canberra, Australia: National Health and Medical Council Research; 2007.
97. Kumar J. Is water fluoridation still necessary? *Adv Dent Res* 2008 20: 8–12.
98. Armfield JM. Community effectiveness of public water fluoridation in reducing children's dental disease. *Public Health Rep* 2010 125: 655–664.
99. McDonagh MS, Whitining PF, Wilson PM *et al.* Systematic review on water fluoridation. *Br Med J* 2000 321: 855–859.
100. Griffin SO, Regnier E, Griffin PM *et al.* Effectiveness of fluoride in preventing caries in adults. *J Dent Res* 2007 86: 410–415.

101. Yeung A, Hitchings JL, Macfarlane TV *et al.* Fluoridated milk for preventing dental caries. *Cochrane Database Syst Rev* 2005 In Cochrane Library, Art. No. CD 003876. doi: 10.1002/14651858.

102. Ellwood R, Fejerskov O, Cury JA *et al.* Fluorides in caries control. In Fejerskov O, Kidd E, editors. *Dental Caries. The Disease and its Clinical Management*. Oxford: Blackwell Munksgaard Ltd; 2008. p. 308.

103. Yengopal V, Chikte UM, Mickenautsch S *et al.* Salt fluoridation: a meta-analysis of its efficacy for caries prevention. *SADJ* 2010 65: 60–67.

104. Petersson GH, Bratthall D. The caries decline: a review of reviews. *Eur J Oral Sci* 1996 104: 436–443.

105. Carvalho JC, Nieuwenhuysen JP, D'Hoore W. The decline in dental caries among Belgian children between 1983 and 1998. *Community Dent Oral Epidemiol* 2001 29: 55–61.

106. Walsh T, Worthington HV, Glenny AM *et al.* Fluoride toothpaste of different concentrations for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev* 2010 20: CD007868.

107. Wong MC, Clarkson J, Glenny AM *et al.* Cochrane reviews on the benefits/risks of fluoride toothpastes. *J Dent Res* 2011 90: 573–579.

108. Zhang Q, van Palenstein WH, van't Hof MA *et al.* Chlorhexidine varnish for preventing dental caries in children, adolescents and young adults: a systematic review. *Eur J Oral Sci* 2006 114: 449–455.

109. Slot DE, Vaandrager NC, van Loveren C *et al.* The effect of chlorhexidine varnish on root caries: a systematic review. *Caries Res* 2011 45: 162–173.

110. James P, Parnell C, Whelton H. The caries-preventive effect of chlorhexidine varnish in children and adolescents: a systematic review. *Caries Res* 2010 44: 333–340.

111. Whelton H, O'Mullane D. The use of combinations of caries preventive procedures. *J Dent Educ* 2001 65: 1110–1113.

112. Du MQ, Tai BJ, Jiang H *et al.* A two-year randomized clinical trial of chlorhexidine varnish on dental caries in Chinese preschool children. *J Dent Res* 2006 85: 557–559.

113. Amorim RG, Leal SC, Bezerra AC *et al.* Association of chlorhexidine and fluoride for plaque control and white spot lesion remineralization in primary dentition. *Int J Paediatr Dent* 2008 18: 446–451.

114. Knight GM, McIntyre JM, Craig GG *et al.* Differences between normal and demineralized dentine pretreated with silver fluoride and potassium iodide after an in vitro challenge by Streptococcus mutans. *Aust Dent J* 2007 52: 16–21.

115. Braga MM, Mendes FM, De Benedetto MS *et al.* Effect of silver diammine fluoride on incipient caries lesions in erupting permanent first molars: a pilot study. *J Dent Child* 2009 76: 28–33.

116. Tan HP, Lo EC, Dyson JE *et al.* A randomized trial on root caries prevention in elders. *J Dent Res* 2010 89: 1086–1090.

117. Yengopal V, Mickenaustch S. Caries preventive effect of casein phosphopeptide-amorphous calcium phosphate (CPP-ACP): a meta-analysis. *Acta Odontol Scand* 2009 21: 1–12.

118. Andersson A, Sköld-Larsson K, Hallgren A *et al.* Effect of a dental cream containing amorphous calcium phosphate complexes on white spot lesion regression assessed by laser fluorescence. *Oral Health Prev Dent* 2007 5: 229–233.

119. Cai F, Shen P, Walker GD *et al.* Remineralizing of enamel subsurface lesions by chewing gum with added calcium. *J Dent* 2009 37: 763–768.

120. Rao SK, Bhat GS, Aradhya S *et al.* Study of the efficacy of toothpaste containing casein phosphopeptide in the prevention of dental caries: a randomized controlled trial in 12- to 15-year-old high caries risk children in Bangalore, India. *Caries Res* 2009 43: 430–435.

121. Bröchner A, Christensen C, Kristensen B *et al.* Treatment of post-orthodontic white spot lesions with casein phosphopeptide-stabilised amorphous calcium phosphate. *Clin Oral Investig* 2011 15: 369–373.

122. Beerens MW, van der Veen MH, van Beek H *et al.* Effects of casein phosphopeptide amorphous calcium fluoride phosphate paste on white spot lesions and dental palque after orthodontic treatment: a 3-month follow-up. *Eur J Oral Sci* 2010 118: 610–617.

123. Brazzelli M, McKenzie L, Fielding S *et al.* Systematic review of the effectiveness and cost-effectiveness of HealOzone for the treatment of occlusal pi/fissure caries and root caries. *Health Technol Assess* 2006 10: 16.

124. Rickard GD, Richardson RJ, Johnson TM *et al.* Ozone therapy for the treatment of dental caries. *Cochrane Database Syst Rev* 2004 Art. No.: CD004153. doi: 10.1002/14651858.CD004153.pub2.

125. Azarpazhooh A, Limeback H. The application of ozone in dentistry: a systematic review of the literature. *J Dent* 2008 36: 104–116.

126. Robinson C, Hallsworth AS, Weatherell JA *et al.* Arrest and control of carious lesions: a study based on preliminary experiments with resorcinol-formaldehyde resin. *J Dent Res* 1976 55: 812–818.

127. Paris S, Meyer-Lueckel H, Kielbassa AM. Resin infiltration of natural caries lesions. *J Dent Res* 2007 86: 662–666.

128. Meyer-Lueckel H, Paris S, Kielbassa AM. Surface layer erosin of natural caries lesions with phosphoric and hydrochloric gels in preparation for resin infiltration. *Caries Res* 2007 41: 223–230.

129. Paris S, Meyer-Lueckel H. Infiltrants inhibit progression of natural caries lesions in vitro. *J Dent Res* 2010a 89: 1276–1280.

130. Paris S, Meyer-Lueckel H. Inhibition of caries progression by resin infiltration in situ. *Caries Res* 2010b 44: 47–54.

131. Ekstrand KR, Luna LE, Promisiero L *et al.* The reliability and accuracy of two methods for proximal caries detection and depth on directly visible proximal surfaces: an in vitro study. *Caries Res* 2011 45: 93–99.

132. Martignon S, Ekstrand KR, Gomez J *et al.* Infiltrating/sealing proximal caries lesions: a 3-year randomized clinical trial. *J Dent Res* 2012 91: 288–292.

133. Kielbassa AM, Muller J, Gernhardt CR. Closing the gap between oral hygiene and minimally invaisve dentistry: a review on the resin infiltration technique of incipient (proximal) enamel lesions. *Quintessence Int* 2009 40: 663–681.

134. Carvalho JC, Ekstrand KR, Thylstrup A. Dental plaque and caries on occlusal surfaces of first permanent molar in relation to stage of eruption. *J Dent Res* 1989 68: 773–779.

135. Disney JA, Graves RC, Stamm JW *et al.* The University of North Carolina Caries Risk Assessment study: further developments in caries risk prediction. *Community Dent Oral Epidemiol* 1992 20: 64–75.

136. Feigal RJ. The use of pits and fissure sealants. *Pediatr Dent* 2002 24: 415–422.

137. Griffin SO, Oong E, Kohn W *et al.* The effectiveness of sealants in managing caries lesions. *J Dent Res* 2008 87: 169–174.

138. Hiiri A, Ahovuo-Saloranta A, Nordblad A *et al.* Pit and fissure sealants versus fluoride varnishes for preventing dental decay in children and adolescents. *Cochrane Database Syst Rev* 2010 17: CD003067.

139. Simonsen RJ. Pit and fissure sealants: review of the literature. *Pediatr Dent* 2002 24: 393–414.

140. Locker D, Jokovic A, Kay EJ. Prevention. Part 8: the use of pit and fissure sealants in preventing caries in the permanent dentition of children. *Br Dent J* 2003 195: 375–378.

141. Beiruti N, Frencken JE, van't Hof MA *et al.* Caries preventive effect of resin-based and glass ionomer sealants over time: a systematic review. *Community Dent Oral Epidemiol* 2006 34: 403–409.

142. Yengopal V, Mickenautsch S, Bezerra AC *et al.* Caries-preventive effect of glass ionomer and resin-based fissure sealants on permanent teeth: a meta-analysis. *J Oral Sci* 2009 51: 373 –382.

143. Mickenautsch S, Yengopal V. Caries-preventive effect of glass ionomer and resin-based fissure sealants on permanent teeth: an update of systematic review evidence. *BMC Res Notes* 2011 4: 22.

144. Yengopal V, Mickenautsch S. Resin-modified glass-ionomer cements versus resin-based materials as fissure sealants: a meta-analysis of clinical trials. *Eur Arch Paediatr Dent* 2010 11: 18–25.

145. van't Hof MA, Frencken JE, van Palenstein WH *et al.* The ART approach for managing dental caries: a meta-analysis. *Int Dent J* 2006 56: 345–351.

146. de Amorim RG, Leal SC, Frencken JE. Survival of atraumatic restorative treatment (ART) sealants and restorations: a meta-analysis. *Clin Oral Investig* 2012 16: 429–441.

147. Ahovuo-Saloranta A, Hiiri A, Nordblad A *et al.* Pit and fissure sealants for preventing dental decay in the permanent teeth of children and adolescents. *Cochrane Database Syst Rev* 2008 4: CD001830.

148. Beiruti N, Frencken JE, van't Hof MA *et al.* Caries-preventive effect of a one-time application of composite resin and glass-ionomer sealants after 5 years. *Caries Res* 2006 40: 52–59.

149. Chen X, Du MQ, Fan M *et al.* Caries preventive effect of sealants produced with altered glass-ionomer materials after 2 years. *Dent Mater* 2012 28: 554–560.

150. Mejàre I, Mjör IA. Glass ionomer and resin-based fissure sealants: a clinical study. *Scand J Dent Res* 1990 98: 345–350.

151. Övrebö RS, Raadal M. Microleakage in fissures sealed with resin or glass ionomer cement. *Scand J Dent Res* 1990 98: 66 –69.

152. Williams B, Laxton L, Holt RD *et al.* Fissure sealants: a 4-year clinical trial comparing an experimental glass polyalkenoate cement with a bis glycidyl methacrylate resin used as fissure sealants. *Br Dent J* 1996 180: 104–108.

153. Frencken JE, Wolke J. Clinical and SEM assessment of ART high-viscosity glass-ionomer sealants after 8–13 years in 4 teeth. *J Dent* 2010 38: 59–64.

154. Bjørndal L, Larsen T, Thylstrup A. A clinical and microbiological study of deep carious lesions during stepwise excavation using long treatment intervals. *Caries Res* 1997 31: 411–417.

155. Mertz-Fairhurst EJ, Curtis JW Jr, Ergle JW *et al.* Ultraconservative and cariostatic sealed restorations: results at year 10. *J Am Dent Assoc* 1998 129: 55–66.

156. Ribeiro CC, Baratieri LN, Perdigão J *et al.* A clinical, radiographic, and scanning electron microscopic evaluation of adhesive restorations on carious dentin in primary teeth. *Quintessence Int* 1999 30: 591–599.

157. Massara MLA, Alves JB, Brandao PR. Atraumatic restorative treatment: clinical, ultrastructural and chemical analysis. *Caries Res* 2002 36: 430–436.

158. Santiago BM, Ventin DA, Primo LG *et al.* Microhardness of dentine underlying ART restorations in primary molars: an in vivo pilot study. *Br Dent J* 2005 199: 103–106.

159. Fusayama T. *A Simple Pain-Free Adhesive Restorative System by Minimal Reduction and Total Etching*. Tokyo: Ishiyaku EuroAmerica Inc Tokyo; 1993. p. 1–21.

160. Mertz-Fairhurst EJ, Schuster GS, Williams JE *et al.* Clinical progress of sealed and unsealed caries. Part I: depth changes and bacterial counts. *J Prosthet Dent* 1979 42: 521–526.

161. Handelman SL, Leverett DH, Espeland M *et al.* Retention of sealants over carious and sound tooth surfaces. *Community Dent Oral Epidemiol* 1987 15: 1–5.

162. Weerheijm KL, Groen HJ. The residual caries dilemma. *Community Dent Oral Epidemiol* 1999 27: 436–441.

163. Oong EM, Griffin SO, Kohn WG *et al.* The effect of dental sealants on bacteria levels in caries lesions: a review of the evidence. *J Am Dent Assoc* 2008 139: 271–278.

164. Boston DW, Graver HT. Histological study of an acid red caries-disclosing dye. *Oper Dent* 1989 14: 186–192.

165. Yip HK, Stevenson AG, Beeley JA. The specificity of caries detector dyes in cavity preparation. *Br Dent J* 1994 176: 417 –421.

166. Banerjee A, Kidd EA, Watson TF. In vitro evaluation of five alternative methods of carious dentine excavation. *Caries Res* 2000 34: 144–150.

167. Celeberti P, Francescut P, Lussi A. Performance of four dentine excavation methods in deciduous teeth. *Caries Res* 2006 40: 117–123.

168. Neves Ade A, Coutinho E, De Munck J *et al.* Caries-removal effectiveness and minimal-invasiveness potential of caries-excavation techniques: a micro-CT investigation. *J Dent* 2011 39: 154–162.

169. Fluckiger L, Waltimo T, Stich H *et al.* Comparison of chemomechanical caries removal using Carisolv or conventional hand excavation in deciduous teeth in vitro. *J Dent* 2005 33: 87–90.

170. Magalhães CS, Moreira AN, Campos WR *et al.* Effectiveness and efficiency of chemomechanical carious dentin removal. *Braz Dent J* 2006 17: 63–67.

171. Nadanovsky P, Cohen Carneiro F, Souza de Mello F. Removal of caries using only hand instruments: a comparison of mechanical and chemo-mechanical methods. *Caries Res* 2001 35: 384–389.

172. de Amorim RFSG. Caries epidemiology and appropriate oral care in schoolchildren from Paranoá, Brazil. PhD thesis. Radboud University Nijmegen, Nijmegen, the Netherlands; 2012. p. 121–138.

173. Eden E, Topaloglu-Ak A, Frencken JE *et al.* Survival of self-etch adhesive Class II composite restorations using ART and conventional cavity preparations in deciduous teeth. *Am J Dent* 2006 19: 359–363.

174. Ersin NK, Uzel A, Aykut A *et al.* Inhibition of cultivable bacteria by chlorhexidine treatment of dentin lesions treated with the ART technique. *Caries Res* 2006 40: 172–177.

175. Farag A, van der Sanden WJ, Abdelwahab H *et al.* 5-Year survival of ART restorations with and without cavity disinfection. *J Dent* 2009 37: 468–474.

176. Mickenautsch S, Yengopal V, Banerjee A. Atraumatic restorative treatment versus amalgam restoration longevity: a systematic review. *Clin Oral Investig* 2010 14: 233–240.

177. Hickel R, Kaaden C, Paschos E *et al.* Longevity of occlusally-stressed restorations in posterior primary teeth. *Am J Dent* 2005 18: 198–211.

178. Frencken JE, Pilot T, Songpaisan Y *et al.* Atraumatic restorative treatment (ART): rationale, technique and development. *J Public Health Dent* 1996 56: 135–140.

179. Taifour D, Frencken JE, Beiruti N *et al.* Effectiveness of glass-ionomer (ART) and amalgam restorations in the decidu-

ous dentition: results after 3 years. *Caries Res* 2002 36: 437–444.

180. Lenters M, van Amerongen WE, Mandari GJ. Iatrogenic damage to the adjacent surfaces of primary molars, in three different ways of cavity preparation. *Eur Arch Paediatr Dent* 2006 7: 6–10.
181. Leal SC, Abreu DM, Frencken JE. Dental anxiety and pain related to ART. *J Appl Oral Sci* 2009 17: 84–88.
182. Farag A, van der Sanden WJ, Abdelwahab H *et al.* Survival of ART restorations assessed using selected FDI and modified ART restoration criteria. *Clin Oral Investig* 2011 15: 409–415.
183. Zanata RL, Fagundes TC, Freitas MC *et al.* Ten-year survival of ART restorations in permanent posterior teeth. *Clin Oral Investig* 2011 15: 265–271.
184. Innes NP, Evans DJ, Stirrups DR. The hall technique: a randomized controlled clinical trial of a novel method of managing carious primary molars in general dental practice: acceptability of the technique and outcomes at 23 months. *BMC Oral Health* 2007 20: 7–18.
185. Innes NP, Evans DJ, Stirrups DR. Sealing caries in primary molars: randomized control trial, 5-year results. *J Dent Res* 2011 90: 1405–1410.
186. Baelum V, van Palenstein Helderman W, Hugoson A *et al.* The role of dentistry in controlling caries and periodontitis globally. In: Fejerskov O, Kidd E, editors. *Dental Caries. The Disease and its Clinical Management*. Oxford, UK: Blackwell Munksgaard; 2008. p. 576–605.
187. de Amorim RFSG. Caries epidemiology and appropriate oral care in schoolchildren from Paranoá, Brazil. PhD thesis. Radboud University Nijmegen, Nijmegen, the Netherlands; 2012. p. 139–157.
188. Levine RS, Pitts NB, Nutgent ZJL. The fate of 1,587 unrestored carious deciduous teeth: a retrospective general dental practice based study from northern England. *Br Dent J* 2002 193: 99–103.
189. Chu CH, Lo EC, Lin HC. Effectiveness of silver diamine fluoride and sodium fluoride varnish in arresting dentin caries in Chinese pre-school children. *J Dent Res* 2002 81: 767 –770.
190. Llodra JC, Rodriguez A, Ferrer B *et al.* Efficacy of silver diamine fluoride for caries reduction in primary teeth and first permanent molars of schoolchildren: 36-month clinical trial. *J Dent Res* 2005 84: 721–724.
191. Yee R, Holmgren C, Mulder J *et al.* Efficacy of silver diamine fluoride for arresting caries treatment. *J Dent Res* 2009 88: 644–647.
192. Peumans M, Kanumilli P, De Munck J *et al.* Clinical effectiveness of contemporary adhesives: a systematic review of current clinical trials. *Dent Mater* 2005 21: 864–881.
193. Manhart J, Chen H, Hamm G *et al.* Buonocore memorial lecture. Review of the clinical survival of direct and indirect restorations in posterior teeth of the permanent dentition. *Oper Dent* 2004 29: 481–508.
194. Kay E, Watts A, Paterson R *et al.* Preliminary investigation into the validity of dentists' decisions to restore occlusal surfaces of permanent teeth. *Community Dent Oral Epidemiol* 1988 16 91–94.
195. Noar SJ, Smith BGN. Diagnosis of caries and treatment decisions in approximal surfaces of posterior teeth in vitro. *J Oral Rehabil* 1990 17: 209–218.
196. Bader JD, Shugars DA. Agreement among dentists' recommendations for restorative treatment. *J Dent Res* 1993 72: 891–896.
197. Elderton RJ, Nuttall NM. Variation among dentists in planning treatment. *Br Dent J* 1983 154: 201–206.
198. Davies JA. The relationship between change in dentist and treatment received in the general dental service. *Br Dent J* 1984 157: 322–324.
199. Bader JD, Shugars DA. Understanding dentists' restorative treatment decisions. *J Public Health Dent* 1992 52: 102–110.
200. Gordan VV, Riley JL III, Geraldeli S *et al.* Repair or replacement of defective restorations by dentists in The Dental PBRN. *J Am Dent Assoc* 2012, Accepted.
201. Mjör IA, Moorhead JE, Dahl JE. Reasons for replacement of restorations in permanent teeth in general dental practice. *Int Dent J* 2000 50: 360–366.
202. Mjör IA, Shen C, Eliasson ST *et al.* Placement and replacement of restorations in general dental practice in Iceland. *Oper Dent* 2002 27: 117–123.
203. Pink FE, Minden NJ, Simmonds S. Decisions of practitioners regarding placement of amalgam and composite restorations in general practice settings. *Oper Dent* 1994 19: 127–132.
204. Deligeorgi V, Wilson NH, Fouzas D *et al.* Reasons for placement and replacement of restorations in student clinics in Manchester and Athens. *Eur J Dent Educ* 2000 4: 153–159.
205. Mjör IA, Toffenetti F. Secondary caries: a literature review with case reports. *Quintessence Int* 2000 31: 165–179.
206. Kidd EA. Microleakage: a review. *J Dent* 1976 4: 199–206.
207. Kidd EA. Caries diagnosis within restored teeth. In Anusavice KJ, editor. *Quality Evaluation of Dental Restorations*. Chicago, IL: Quintessence; 1989. p. 111–121.
208. Soderholm KJ, Antonson DE, Fishlschweiger W. Correlation between marginal discrepancies at the amalgam tooth interface and recurrent caries. In: Anusavice KJ, editor. *Quality Evaluation of Dental Restorations*. Chicago, IL: Quintessence, 1989. p. 85–108.
209. Kidd EA, O'Hara JW. Caries status of occlusal amalgam restorations with marginal defects. *J Dent Res* 1990 69: 1275–1277.
210. Kidd EAM, Joyston-Bechal S, Beighton D. Marginal ditching and staining as a predictor of secondary caries around amalgam restorations: a clinical and microbiological study. *J Dent Res* 1995 74: 1206–1211.
211. Gordan VV. In vitro evaluation of margins of replaced resin based composite restorations. *J Esthet Dent* 2000 12: 217–223.
212. Gordan VV. Clinical evaluation of replacement of Class V resin based composite restorations. *J Dent* 2001 29: 485–488.
213. Gordan VV, Mondragon E, Shen C. Evaluation of the cavity design, cavity depth, and shade matching in the replacement of resin based composite restorations. *Quintessence Int* 2002 32: 273–278.
214. Bissada NF. Symptomatology and clinical features of hypersensitive teeth. *Arch Oral Biol* 1994 39: 31S–32S.
215. Hirata K, Nakashima M, Sekine I *et al.* Dentinal fluid movement associated with loading of restorations. *J Dent Res* 1991 70: 975–978.
216. Gordan VV, Garvan CW, Blaser PK *et al.* A long-term evaluation of alternative treatments to replacement of resin-based composite restorations: results of a seven-year study. *J Am Dent Assoc* 2009 140: 1476–1484.
217. Gordan VV, Riley JL III, Garvan CW *et al.* 7-Year results of alternative treatments to defective amalgam restorations. *J Am Dent Assoc* 2011 142: 842–849.
218. Moncada G, Martin J, Fernández E *et al.* Sealing, repair and refurbishment of class I and class II defective restorations: a three-year clinical trial. *J Am Dent Assoc* 2009 140: 425–432.
219. Campain AC, Morgan MV, Evans RW *et al.* Sugar-starch combinations in food and the relationship to dental caries in low-risk adolescents. *Eur J Oral Sci* 2003 111: 316–325.

220. Mettes TG, van der Sanden WJ, Mokkink HG *et al.* Routine oral examinations in primary care: which predictors determine what is done? A prospective clinical case recording study. *J Dent* 2008 36: 435–443.

221. Tezvergil A, Lassila LV, Yli-Urpo A *et al.* Repair bond strength of restorative resin composite applied to fiber-reinforced composite substrate. *Acta Odontol Scand* 2004 62: 51–60.

222. Gordan VV, Shen C, Mjor IA. Marginal gap repair with flowable resin-based composites. *Gen Dent* 2004 52: 390–394.

223. Shen C, Mondragon E, Mjör IA *et al.* Effect of mechanical undercut on the strength of composite repair. *J Am Dent Assoc* 2004 135: 1406–1412.

224. Blum IR, Lynch CD, Wilson NHF. Teaching of direct composite restoration repair in undergraduate dental schools in the United Kingdom and Ireland. *Eur J Dent Educ* 2012 16: e53–e58.

225. Blum IR, Lynch CD, Wilson NHF. Teaching of the repair of defective composite restorations in Scandinavian dental schools. *J Oral Rehabil* 2012 39: 210–216.

226. Blum IR, Lynch CD, Schreiver A *et al.* Repair versus replacement of defective composite restorations in German dental schools. *Eur J Prosthodont Restor Dent* 2011 19: 56–61.

227. Gordan VV, Mjör IA, Blum I *et al.* Teaching students the repair of resin based composite restorations: a survey of North American dental schools. *J Am Dent Assoc* 2003 134: 317–323.

228. Gordan VV, Garvan CW, Richman JS *et al.* How dentists diagnose and treat defective restorations: evidence from the dental practice-based research network. *Oper Dent* 2009 34: 664–673.

229. Dennison JB, Sarrett DC. Prediction and diagnosis of clinical outcomes affecting restoration margins. *J Oral Rehabil* 2012 39: 301–318.

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