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Published in:
British Dental Journal

DOI:
[10.1038/s41415-020-2220-4](https://doi.org/10.1038/s41415-020-2220-4)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2020

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Banerjee, A., Splieth, C., Breschi, L., Fontana, M., Paris, S., Burrow, M., Crombie, F., Foster Page, L., Gatón-Hernández, P., Giacaman, R. A., Gugni, N., Hickel, R., A. Jordan, R., Leal, S., Lo, E., Tassery, H., Thomson, W. M., Manton, D. J., & Schwendicke, F. (2020). When to intervene in the caries process? A Delphi consensus statement. *British Dental Journal*, 229(7), 474-482. <https://doi.org/10.1038/s41415-020-2220-4>

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When to intervene in the caries process? A Delphi consensus statement

Avijit Banerjee,^{*1} Christian Splieth,² Lorenzo Breschi,³ Margherita Fontana,⁴ Sebastian Paris,⁵ Michael Burrow,⁶ Felicity Crombie,⁷ Lyndie Foster Page,⁸ Patricia Gatón-Hernández,⁹ Rodrigo A. Giacaman,¹⁰ Neeraj Gugrani,¹¹ Reinhard Hickel,¹² Rainer A. Jordan,¹³ Soraya Leal,¹⁴ Edward Lo,⁶ Hervé Tassery,¹⁵ William Murray Thomson,⁸ David J. Manton^{7,16} and Falk Schwendicke⁵

Key points

Provides much needed guidance for the primary care practitioner to help in their management of dental caries.

Considers the quality of the evidence available to provide pragmatic guidelines for caries management interventions.

Comprehensive diagnosis is the basis for systematic decision-making on when to intervene in the caries process and existing lesions

Abstract

Objectives To define an expert Delphi consensus on when to intervene in the caries process and existing carious lesions.

Methods Non-systematic literature synthesis, expert Delphi consensus process and expert panel conference.

Results Lesion activity, cavitation and cleansability determine intervention thresholds. Inactive lesions do not require treatment (in some cases, restorations may be placed for form, function, aesthetics); active lesions do. Non-cavitated carious lesions should be managed non- or micro-invasively, as should most cavitated lesions which are cleansable. Cavitated lesions which are not cleansable usually require minimally invasive management. In specific circumstances, mixed interventions may be applicable. Occlusally, cavitated lesions confined to enamel/non-cavitated lesions extending radiographically into deep dentine may be exceptions. Proximally, cavitation is hard to assess tactile-visually. Most lesions extending radiographically into the middle/inner third of dentine are assumed to be cavitated. Those restricted to the enamel are not cavitated. For lesions extending radiographically into the outer third of dentine, cavitation is unlikely. These lesions should be managed as if they were non-cavitated unless otherwise indicated. Individual decisions should consider factors modifying these thresholds.

Conclusions Comprehensive diagnosis is the basis for systematic decision-making on when to intervene in the caries process and existing lesions.

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Refereed Paper.

Accepted 22 April 2020

<https://doi.org/10.1038/s41415-020-2220-4>

This manuscript has been modified with permission from Schwendicke F, Splieth C, Breschi L *et al.* Consensus statement: When to intervene in the caries process? *Clin Oral Invest* 2019; 23: 3691–3703, Springer. <https://doi.org/10.1007/s00784-019-03058-w>

Building an expert Delphi consensus on caries intervention thresholds

There is an ever-increasing number of strategies available to manage the caries process and its outcome, the carious lesion (ranging from very early signs and symptoms through to extended cavitated lesions with pulp involvement). These strategies are applied to avoid pain, prevent loss of tooth tissues or entire teeth, retain functionality and aesthetics. Daily, oral health practitioners are faced with the decision of 'if and when' to intervene using one of the many approaches available. This decision should be based on the available evidence, the characteristics, needs and wishes of the specific patient, the features of the tooth or carious lesion to be managed (as will be discussed below) and the operator's experience.¹

Expert consensus can also support decision-making, especially if existing systematic

evidence is limited or of too narrow scope. There have been recent attempts in dentistry and specifically cariology, to assist clinical decision-making by expert consensus; for example, on minimum intervention dentistry and carious tissue removal.^{2,3,4,5} The presented paper describes the consensus reached by an expert consensus panel who met in July 2018 in London, UK and used a structured online Delphi process before and after the meeting to systematically collate expert opinion and come to an agreement. The consensus focused specifically on when to intervene in the caries process and on existing carious lesions, rather than caries prevention. A detailed description of the methods can be found in Appendix 1, also including the guidance on conducting and reporting Delphi studies (CREDES).⁶

Firstly, the contemporary understanding of the caries process and the characteristics of carious lesions will be described. Secondly,

the different levels of interventions will be presented and brief examples given. Thirdly, factors determining the decision of when to intervene will be outlined. Finally, consensus recommendations, which emerged from a 2-round Delphi process and a consensus conference involving a panel of more than 20 international experts, will be presented. The panel comprised members of the European Organisation for Caries Research (ORCA) and delegates of the European Federation of Conservative Dentistry (EFCDD) and international experts from around the world. Note that this document and the resulting consensus recommendations do not aim to update or replace existing (often broader) guidance. The consensus on minimum intervention dentistry, for example, discussed caries detection and risk assessment, remineralisation and other preventive measures, minimally invasive operative interventions and re-treatments.⁴ Instead, this document specifically aims to assist decision-making on when to intervene in the caries process and on existing carious lesions.

Dental caries and carious lesions

Dental caries is one of the most prevalent and ubiquitous non-communicable diseases affecting humankind today.⁷ It was first understood to be an infectious disease, requiring removal of all plaque (biofilm) from the teeth or from affected carious hard tissues (specific plaque hypothesis). This concept, whilst debated (that is, biofilm being cariogenic under certain conditions; non-specific plaque hypothesis), was later modified, suggesting that the mere presence of biofilm is not sufficient for the pathogenesis of caries, but that an overlapping interaction between the host/teeth, substrate and microbiota is needed. Despite being 'infected' (or contaminated) with cariogenic bacteria, a cavitated carious lesion will not develop without a cariogenic diet.^{8,9} Marsh [1994] introduced the ecological plaque theory. The microbial composition of the biofilm is stable unless 'environmental perturbations' occur which can affect microbial homeostasis, leading to dysbiosis.^{10,11} With respect to caries, diet (mainly free sugars), oral hygiene and salivary factors are the contributing drivers of dysbiosis, leading to a shift in the microbiota towards acidogenic and aciduric microorganisms. There are also wider influences on caries experience,¹² but consideration of those is beyond our remit. The original composition of an individual's

dental biofilm is influenced by both hereditary and environmental factors, but, as time goes on, the types and proportions of micro-organisms acquired early in life are modified by environmental influences.¹³ Currently, the extended ecological plaque hypothesis is accepted as an explanation of the pathogenesis of caries.¹⁴

This pathogenesis involves organic acids, the by-product of microbial metabolism of dietary free sugars. As the pH of the biofilm decreases, it reaches a point where the biofilm fluid at the surface of the tooth is under-saturated with respect to tooth mineral, and dissolution occurs to maintain equilibrium.¹⁵ Initially, the dissolution occurs at the surface of the tooth, but, if conditions persist, and the lesion becomes more extensive, mineral from deeper in the enamel (and subsequently dentine) will be lost. Caries has an indirect genetic component, with influencing factors including enamel quality and quantity, immune response, dietary preferences and salivary characteristics.^{16,17} In summary, dental caries is a disease characterised by a process of demineralisation of the dental hard tissues, caused by frequent free sugars exposure to the dental biofilm, which shifts the ecological balance towards a cariogenic dysbiosis. For dentine and root caries, cleavage of collagen by bacterial or mainly dentine enzymes follows early mineral loss and contributes to the loss of the hard tissue.^{18,19}

Management of the caries process/ carious lesions

The former management of the caries process and carious lesions was influenced by an understanding that caries was a purely infectious disease and could be managed invasively/restoratively by removing all demineralised and 'contaminated'/infected tissue. This was grounded in (1) a lack of understanding that the caries process and carious lesions are separate, but related; (2) the incorrect assumption that once a lesion had established and the tooth was 'infected', eradication of microbiota was needed; (3) the erroneous concept that lesion progression was inevitable; and, (4) the fact that the majority of carious lesions dentists encountered in the past were truly 'decayed', that is, cavitated dentine lesions. Therefore, the professional education of dental surgeons concentrated on mechanistic surgical procedures instead of that of dental physicians who manage the disease and patient

as a whole. Remuneration systems incentivised such invasive/restorative therapies.^{20,21} Notably, the shift away from this approach was initiated decades ago, but consistent updated data from all over the globe demonstrates that it has not been fully adopted.²²

Building on the evidence accrued over several decades, it is clear that (1) the caries process can be controlled by modifying the patient's caries risk/susceptibility, depending on his/her adherence to behavioural modifications and not only by intervening operatively on carious lesions, yet success/understanding of behavioural interventions on caries control has been limited;²³ (2) the caries process and carious lesions can be managed without removing microorganisms, but by rebalancing the dysbiosis within the tooth surface biofilm and arresting those within the depths of the tissues; (3) active (progressing) lesions can be inactivated;²⁴ (4) in many high-income countries the spectrum of carious lesions has been and is shifting, especially in younger people, as there are now more non-cavitated lesions being detected;^{25,26} and, (5) while dental education in some countries now involves the contemporary understanding of caries and its subsequent carious lesions, remuneration systems for oral healthcare delivery have largely not been adjusted accordingly (there may be exceptions, and admittedly the uptake of a more contemporary approach towards caries and carious lesions has improved slowly over the last three decades or so).

Hence, the conventional restorative/invasive approach towards managing the caries process and carious lesions is not grounded in current understanding of the disease and it is also not appropriate for managing the broad spectrum of lesions found in many individuals (from very early to large cavitated lesions). It should also be considered that subsequent interventions on restored teeth are often necessary.^{27,28,29} This is classically known as the 'restorative death spiral'.^{30,31,32} Given these alternative arguments, there is consensus that invasive/restorative interventions alone are not beneficial for managing the caries process and lesions in all situations. Instead, invasive/restorative interventions represent a late step in the management puzzle, repairing the gross tissue damage and restoring form, function, aesthetics and cleansability, thereby allowing the control of risk of future loss of function. Invasive strategies may also be used to approach acute carious lesions.²⁶ Invasive/restorative interventions are an

Fig. 1 Overview of different intervention levels and strategies. All strategies should be provided following the principles of minimally invasive dentistry

important and relevant tool, but they should be complemented by other (non- or micro-invasive) management strategies. These 'other' strategies aim to control the caries process and the activity of carious lesions (as will be discussed below). If they are successfully implemented, the presence of bacteria is no longer a problem, since the cariogenic activity and the resulting mineral loss from dental hard tissues can be controlled concomitantly. In order to implement these alternative approaches successfully, there needs to be clear communication between the oral healthcare professional team and the patient, with the latter appreciating their role in valuing and respecting their own oral health.²⁰

Intervention strategies: levels of invasiveness

In the present document, we distinguish three levels of invasiveness to classify intervention strategies for 'treating' existing carious lesions. They are based on the degree of tissue removal associated with each strategy (Fig. 1):

- Non-invasive strategies do not remove dental hard tissue and involve, for example, fluorides and other chemical strategies for controlling mineral balance, biofilm control measures and dietary control
- Micro-invasive strategies remove the dental hard tissue surface at the micrometre level, usually during an etching step, such as sealing or infiltration techniques
- Minimally invasive strategies remove gross dental hard tissue, such as through use of

hand excavators, rotary instruments or other devices. In most cases, this process is associated with the placement of restorations.

Note that some interventions can be regarded as 'mixed', not clearly falling into one of these categories. In the following sections, an overview of what falls into the three different levels of invasiveness will be offered. The individual details pertaining to each strategy will not be discussed, since the aim of this consensus paper was not to give recommendations on how to specifically intervene, but when.

Non-invasive interventions

To arrest existing lesions, several strategies are available, many of which have been developed originally for preventing the development of carious lesions and are now also applied for arresting lesions. These include diet control, biofilm control and mineralisation control.

Diet control

With free sugars being the driver of biofilm dysbiosis and cariogenic activity, both prevention and lesion arrest should, theoretically, be possible if sugar intake (mainly free sugars) is restricted/regulated.³³ Sugar replacement may also be a valid option. Despite having biological plausibility, diet control measures, however, have been tested mainly for their preventive efficacy, as opposed to lesion arrest.

Biofilm control

Dental caries results from dysbiosis in the dental biofilm on a susceptible tooth surface. Therefore, restoring balance within that biofilm (through the use of mechanical biofilm control, antimicrobials, probiotics, etc.) has been advocated.¹¹ Regular toothbrushing, interdental hygiene and antimicrobial strategies (including chlorhexidine and polyols) are the most common examples. Toothbrushing in combination with regular provision of fluoride has been investigated specifically for arresting existing active lesions. Most other therapies have been tested mainly for their preventive effect, not necessarily to arrest existing carious lesions.³⁴

Mineralisation control

Fluoride has been shown to reduce dental caries occurrence consistently in both the primary and permanent dentitions, with the most current evidence strongly suggesting that its effect is primarily topical (that is, post-eruptive). Examples include dentifrices with fluoride concentrations above 1,000 ppm,^{35,36,37} 5,000 ppm fluoride dentifrices,^{38,39,40,41} and fluoride rinses.⁴² Professionally-applied fluoride products, such as gels and varnishes, as well as silver fluoride products (such as silver diamine fluoride) have also been tested for arresting non-cavitated lesions.^{35,38,43,44} Moreover, a variety of products containing calcium in different forms (for example, calcium stabilised by casein derivatives, calcium sodium phosphosilicate, etc) or self-assembling peptides⁴⁵ have been introduced to aid remineralisation. The evidence supporting the clinical efficacy of these products is currently limited.^{34,46,47}

Micro-invasive interventions

There are two main strategies falling into this level of invasiveness; sealing and infiltration.

Sealing

A sealant places a diffusion barrier on the susceptible tooth surface and, hence, impedes acid diffusion into and mineral loss from the tooth tissues. It can also help re-contour the surface so aiding mechanical biofilm control. Sealing non-cavitated carious lesions on proximal, occlusal or smooth surfaces has been assessed in a range of studies.^{48,49,50} There has been a limited assessment of the sealing of cavitated surfaces^{49,50,51} and the current data imply a greater risk of sealant failure due

to fracture or loss of retention, but further research needs to be undertaken to enable definitive conclusions to be drawn.^{51,52,53} The relevance of sealant loss for lesion progression may further differ between different sealant materials (that is, resin versus glass-ionomer cement-based sealants).⁵⁴

Infiltration

Lesion infiltration involves the penetration of incipient enamel carious lesions by low-viscosity resins after removal of the surface layer (by etching with hydrochloric acid) and drying (using ethanol and air).⁵⁵ A diffusion barrier is created within the dental hard tissue sub-surface, impeding acid diffusion into and mineral loss from the tooth tissue, so inactivating the lesion. There is some evidence supporting infiltration of non-cavitated proximal lesions.^{34,48} However, there is currently only one product available on the market for caries infiltration and nearly all related studies have been sponsored by the manufacturer of that product, with the associated potential for bias in the reported findings.

Mixed interventions

Non-restorative cavity control

Non-restorative cavity control (NRCC) aims to reinstate the cleansability of cavitated lesions by chiselling, cutting or grinding away overhanging, biofilm-trapping enamel or dentine.⁵⁶ Subsequently, patients are encouraged to clean the opened 'saucerised' cavity and apply fluoride toothpaste/products (in addition to professional fluoride varnish application). Currently, the technique has been applied only in primary teeth or root surface lesions. Under optimal conditions, NRCC seems to be a feasible option to manage cavitated lesions, especially in uncooperative children who may not tolerate other treatments^{57,58,59,60} or dependent and systemically compromised older adult patients.

Hall technique

The Hall technique involves 'sealing' of cavitated carious lesions in primary teeth using preformed metal crowns without any tooth preparation. It combines the biological management of carious lesions (via sealing bacteria and depriving them of nutrition) and the restorative advantages of preformed metal crowns.^{58,61,62}

Minimally invasive interventions

Minimally invasive operative caries management involves placement of a restoration after selective removal of carious tissue, done to create conditions for long-lasting restorations and maintaining the tooth-restoration complex. Modern bio-interactive adhesive materials have allowed for minimally invasive restorations that provide a seal and focus on preserving healthy and repairable/remineralisable tissue and maintaining pulp health. An International Caries Consensus Collaboration presented recommendations on terminology and on carious tissue removal and restorative management of cavitated carious lesions. These recommendations support less invasive carious lesion management, delaying entry to, and slowing down, the restorative cycle by preserving tooth tissue, being selective on tissue removal and retaining teeth long-term.⁵

Factors affecting caries intervention thresholds

A number of key factors help to determine caries intervention thresholds. These are activity, cavitation, and cleansability of the lesion.

Activity

The term 'lesion activity' reflects the ongoing mineral loss or gain of a lesion. It indicates how likely a lesion is to progress. An inactive (arrested) lesion may be considered as a 'scar' and does not require any treatment. In some cases, restorations might be placed for reasons of form, function and/or aesthetics. As discussed, restorations may also be provided in cases of pain to reduce pulp stress and improve symptoms. Lesion activity is often determined visually; tactile assessment should only be performed gently so as to not damage the surface, using a rounded/ball-ended explorer (forceful probing with a sharp dental explorer is not recommended). For root surfaces, gentle probing may be undertaken to determine the texture.⁶³

Since there is currently no technology available that allows an objective longitudinal measurement of lesion activity, the following clinical signs can be used to estimate lesion activity: (1) the presence of biofilm covering the lesion may indicate activity, especially, in the presence of high and frequent consumption of sugars; (2) the condition of the gingiva

(local gingivitis in proximity to the lesion) can also serve as a proxy measure to determine whether biofilm has been present over time; (3) lesion characteristics, such as texture, hardness and appearance. A smooth enamel lesion surface indicates inactivity, while rough surfaces may indicate activity. Colour-wise, a white chalky matt lesion colour may indicate activity, but shiny or dark lesions may indicate inactivity.^{63,64,65,66}

Notably, not all clinical criteria will be always available, for example, in proximal surfaces or for micro-cavitated lesions. However, in many circumstances, one criterion or more will be assessable and may be complemented with (4) longitudinal recall data, if available (for example, via repeated radiographs, or those from fluorescence-assisted systems, visual scales, or clinical photographs). These can also be used for activity assessment. Lesion activity is the first main factor to decide intervention thresholds, although such evidence is limited and more research is strongly advocated.

Cavitation

Cavitated lesions are those with a surface breach that is clearly detectable to the naked eye or a rounded dental explorer. Often, this may also involve dentine exposure. Cavitation increases the likelihood of lesion progression,^{50,67} because the dental biofilm is protected from self-cleaning and oral hygiene procedures. Moreover, cavitation encourages a more rapid diffusion of acids and carbohydrates, as well as greater bacterial contamination of tissues. In cavitated lesions, the involved dentine is demineralised and the outer portion of the lesion is bacterially contaminated/infected.

A subgroup of cavitated lesions are the micro-cavitated lesions. Sometimes, magnification is needed to detect cavitation in these lesions. Micro-cavitated lesions can show enamel breakdown without visible dentine exposure.

Note that cavitation can be assessed tactile-visually on accessible smooth surfaces (buccal, lingual). Occlusally, their assessment is more complicated. Occlusal carious lesions which radiographically extend deep into the dentine (middle or inner dentine third, D2/3) are usually heavily bacterially contaminated and demineralised. These lesions may require a different management than lesions without such extensive dentine involvement.

On proximal surfaces, detecting cavitation using visual-tactile means is nearly impossible

when adjacent teeth are present (except for clear cavitation in more extensive lesions). Orthodontic separators may be used to gain access over some days, but this is not practical in most settings. Hence, most clinicians will rely on further diagnostic aids, mainly bitewing radiography, to gauge the likelihood of cavitation. While radiographs seldom permit accurate detection of cavitation itself, the lesion depth serves as a proxy of how likely cavitation is; deeper radiographic lesions come with a higher chance of being cavitated. A number of studies have assessed the relationship between the radiographic lesion depth and the presence of cavitation. These have been summarised in a recent review.⁶⁸ Lesions confined radiographically to the enamel (outer or inner enamel half, E1/E2) are seldom cavitated, while those into the middle or inner dentine third (D2/D3) are usually cavitated. Some of the lesions radiographically extending into the outer dentine third (D1) may be cavitated, others not. Further detection aids such as near-infrared light transillumination or fluorescence-based systems⁶⁹ can be used to corroborate radiographic lesion depth assessment.

Cleansability

Cavitation is a major factor for deciding whether and how to intervene, because it serves as an indicator of cleansability and, as described, activity. Nevertheless, sometimes even cavitated lesions can be cleansable, for example in primary anterior teeth with open smooth surface lesions, or in root surface lesions.

There are further factors which can modify caries intervention thresholds: the patient's caries risk/susceptibility, age and the dentition.

Caries risk/susceptibility and behavioural adherence

The term caries risk/susceptibility refers to the chances of an individual developing new carious lesions in the future. There are a number of possible aspects to be integrated into caries susceptibility assessment, such as past caries experience (assuming past caries experience to be a robust indicator of risk factors on the behavioural and the genetic level) and factors directly or indirectly related to caries pathogenesis (diet, oral hygiene, saliva) or lesion development (fluoride intake). Past caries experience has been found to have a good predictive value,⁷⁰ while most other factors have only moderate or low predictive

value. Many further factors suggested for caries risk assessment (salivary buffering capacity, bacterial number/concentration in the saliva, to name but two) show limited predictive value.⁷¹ For root caries, the number of surfaces at risk (exposed roots) seems to be a useful robust parameter for risk assessment.⁷²

There are some established caries risk/susceptibility assessment systems which integrate these factors and weight them. These systems have been found useful to predict coronal and root caries to some degree, but with limited generalisability.^{73,74,75}

Caries susceptibility assessment helps to identify patient-specific factors, which are of value. These should be managed and re-evaluation performed regularly throughout any episode of care. If risk factor modification is not successful or longitudinal re-evaluation not possible, this should be taken into consideration in determining intervention thresholds.

Age

There are several unique considerations that are important in the oral health management of children's and older adults (or vulnerable groups with special needs).⁷⁶ The oral health and behaviour of the carer has a marked influence on the oral health of younger children or other dependent individuals, such as the elderly and institutionalised individuals. The factors affecting patient behaviour in such groups, as in all patients, must be considered, that is capability, opportunity and motivation. Very young children may show only limited cooperation and behavioural adherence.⁷⁷ In some of these individuals, sedation or general anaesthesia are needed to provide care. Interventional thresholds may be lowered and more invasive treatments chosen in some of these instances.

The dentition

The structure of primary teeth differs from that of permanent teeth. The enamel is thinner and slightly less mineralised. The pulp space is proportionally larger and the shape of the teeth is different. The contact areas of the molar teeth, especially between the mandibular molars, are flatter than permanent molars and premolars. This predisposes to carious lesions developing below the wider contact area, close to the gingival margin. Given this anatomy, conventional restorative approaches (including carious tissue removal and direct restorations) have higher risks of

complications (more pulp exposure and restorative complications) in primary than permanent teeth.^{61,78} Moreover, visual-tactile lesion detection on proximal surfaces of primary teeth is difficult because of these wider contact areas.⁷⁹ Also, and most importantly, primary teeth exfoliate. The time to exfoliation and the speed of progression of the carious lesion can influence the decision affecting treatment for a lesion. Pulp therapies for primary molars (especially those with necrotic pulp tissue) may be successful, but are technically demanding.⁸⁰ In a few cases, removing primary molars and, if needed, maintaining the space may be a valid option to avoid pain or sepsis. In contrast, for permanent teeth, the overarching therapeutic aim is long-term retention of teeth in a functional, pain-free and depending on the location in the mouth, aesthetically acceptable condition. Any intervention threshold should consider these aspects on an individual patient basis.

When to intervene in the caries process?

Based on the contemporary understanding of the caries process, the available interventions and the factors determining or modifying intervention thresholds, all discussed in this article, the consensus group agreed on a number of recommendations for when to intervene non-invasively, micro-invasively or minimally invasively/restoratively in the caries process and for existing carious lesions. These recommendations were the subject of the Delphi process, with experts voting on the verbatim recommendations below.

The recommendations should be adapted to each individual patient and setting and need to be applied with each oral healthcare professional's individual expertise and practising context in mind. Also note that nearly all recommendations made are not based on strong empirical evidence, but mainly on expert opinion and experience. Hence, clinical judgment remains a key element in deciding the threshold for intervention. The recommendations are set out below, together with the level of agreement (mean values on a scale from 1 [do not agree at all] to 10 [fully agree] and standard deviations [SD]).

1. Lesion activity should be assessed. An inactive (arrested) lesion is like a 'scar' and does not require any treatment, but should be reviewed. In some cases, restorations

- might be placed for reasons of form, function or aesthetics. An active lesion needs management (agreement 9.4, SD 1.1) (Fig. 2)
2. Cavitation increases the likelihood of activity and lesion progression, as the dental plaque biofilm is protected from self-cleaning and oral hygiene procedures. Moreover, cavitation encourages a more rapid diffusion of sugars and acids. Cavitation can be assessed on visible/accessible surfaces (9.2, SD 0.8)
 3. As a general principle:
 1. Inactive, non-cavitated and cavitated lesions do not require any treatment (except for reasons of form, function or aesthetics) (8.8, SD 1.4)
 2. Active, non-cavitated carious lesions should be managed non- or micro-invasively (9.1, SD 0.9) (Fig. 2)
 3. Cavitated carious lesions which are cleansable but active, can also be managed non- or micro-invasively (except for reasons of form, function or aesthetics) (8.8, SD 1.4) (Fig. 2)
 4. Cavitated carious lesions which are not cleansable and active, should be managed using minimally invasive/restorative strategies. In specific circumstances, mixed interventions may be applicable (9.1, SD 1.0) (Fig. 2).
 4. On occlusal surfaces, two specific scenarios arise (Fig. 3):
 - A. Micro-cavitated lesions extending only into enamel. These can be successfully managed using micro-invasive or mixed interventions (8.8, SD 1.5).
 - B. Micro-cavitated lesions radiographically extending deep into dentine (middle or inner dentine third, D2/3). These are often bacterially contaminated/infected, demineralised and also cavitated, but the cavitation cannot be detected/accessed given the specific anatomy of the occlusal surface. Lesion arrest using non-invasive means may be unlikely here. Also, the stability of any kind of sealant material placed over these lesions appears to be limited. Hence, such lesions should be managed minimally invasively/restoratively in the majority of cases (9.1, SD 0.8).
 5. On proximal surfaces, cavitation of early lesions is usually hard to assess tactile-visually. Orthodontic separators may be used to gain visible access, or lesion depth ascertained from bitewing radiography can be used as proxy to

Fig. 2 Factors involved in determining caries intervention thresholds. Activity, cavitation status and cleansability are the main factors determining intervention thresholds (whether and when to intervene). Thresholds need to be adapted to each individual patient and setting, and need to be applied with each dental professional's individual expertise in mind. There are specific scenarios (Fig. 3, note that as described, not all interventions are applicable in both dentitions or all patients)

Fig. 3 Factors determining intervention thresholds for occlusal lesions. Activity and cavitation status are the main factors determining intervention thresholds. In addition, radiographic dentine involvement should be considered. Note that as described, not all interventions are applicable in both dentitions or all patients

determine the likelihood of cavitation (Fig. 4). Lesions extending radiographically into the middle or inner third of the dentine (D2/3) can be assumed to be cavitated, whilst those restricted to the enamel (E1/2) are usually not cavitated. These lesions should be managed accordingly. For lesions radiographically extending into the outer third of the dentine (D1), a decision problem exists. If possible, these lesions should be managed as if they were non-cavitated, as they are likely to be non-cavitated (that is, non- or micro-invasively) (8.9, SD 1.1)

6. The caries risk/susceptibility of a patient should be assessed. Identified patient-specific risk factors should be, if possible, managed, and re-evaluation performed regularly. If risk factor modification is not successful or longitudinal re-evaluation not possible, this should be taken into consideration and may influence subsequent interventional thresholds (9.0, SD 0.8)
7. Interventional thresholds may be lowered and more invasive treatments chosen for treatment requiring sedation or general

anaesthesia (for example, children, special care patients) (9.2, SD 1.1)

8. In permanent teeth, the overarching therapeutic aim is retaining teeth in a functional, pain-free and – depending on the location in the mouth – aesthetically acceptable condition. In primary teeth, maintaining the space of primary molars and avoiding pain or sepsis is the overarching therapeutic aim. Intervention thresholds should consider these aims accordingly (9.0, SD 1.3).

Conclusions

A consensus on when to intervene in the caries process and for existing carious lesions using non- or micro-invasive, minimally invasive/restorative or mixed interventions has been presented. Lesion activity, cavitation and cleansability are the key factors to be considered to determine intervention thresholds. Inactive lesions do not usually require any treatment (in some cases, restorations might be placed for reasons of form, function, aesthetics); active lesions do. Non-cavitated carious lesions should be managed non- or micro-invasively, as should cavitated carious lesions which are cleansable. Cavitated carious lesions which are not cleansable usually require minimally invasive/restorative management, also to restore form, function and aesthetics of the tooth. In specific circumstances, mixed interventions may be applicable. On occlusal surfaces, cavitated lesions confined to enamel and non-cavitated lesions radiographically extending deep into dentine (middle or inner dentine third, D2/3) may be exceptions to that rule. On proximal surfaces, cavitation is usually hard to assess tactile-visually. Hence, radiographic lesion depth is used to determine the likelihood of cavitation. Lesions extending radiographically into the middle or inner third of the dentine (D2/3) can be assumed to be cavitated, while those restricted to the enamel (E1/2) are usually not cavitated. For lesions extending radiographically into the outer third of the dentine (D1), cavitation status remains unclear. These lesions should be managed as if they were non-cavitated unless otherwise indicated. Individual decisions and clinical judgment should consider factors modifying the described intervention thresholds. Comprehensive diagnosis is the basis for systematic decision-making on when to intervene in the caries process and on existing carious lesions. Patients should be

Fig. 4 Factors determining intervention thresholds for proximal lesions. Activity and cavitation status are the main factors determining intervention thresholds. However, cavitation status is clinically often difficult to assess. Hence, radiographic lesion depth is usually used as a proxy to estimate the chances of cavitation. For lesions confined to enamel (E1, 2), cavitation is unlikely. For lesions extending into the middle or inner third of the dentine (D2, 3), cavitation is likely. Lesions extending into the outer third of the dentine (D1), are more likely non-cavitated than cavitated. If a cavitation cannot be detected clinically, dentists should prefer non- or micro-invasive means if possible. Only under specific circumstances, minimally invasive (or mixed) interventions should be applied. Note that as described, not all interventions are applicable in both dentitions or all patients

informed fully about treatment options and should provide informed consent accordingly and thorough, contemporaneous records maintained.

Conflict of interest

The corresponding author formally requested a declaration of possible conflicts of interest from each of the consensus panel members. No relevant conflicts of interest at the organisational and individual levels related to this consensus document were identified.

Funding

The conference was kindly sponsored by DMG (Hamburg, Germany). This included travel, accommodation and conference costs for panel members. The sponsor had no role in design or conduct of the conference or the content of this manuscript and were not present during the conference. No honoraria were given to any of the panel members.

Ethical approval

This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent

For this type of study, formal consent is not required.

References

1. Sackett D L, Rosenberg W M C, Gray J A M, Haynes R B, Richardson W S. Evidence based medicine: what it is and what it isn't. *BMJ* 1996; **312**: 71–72.
2. Frencken J E, Innes N P, Schwendicke F. Managing Carious Lesions: Why Do We Need Consensus on Terminology and Clinical Recommendations on Carious Tissue Removal? *Adv Dent Res* 2016; **28**: 46–48.
3. Innes N P, Frencken J E, Schwendicke F. Don't Know, Can't Do, Won't Change: Barriers to Moving Knowledge to Action in Managing the Carious Lesion. *J Dent Res* 2016; **95**: 485–486.
4. Frencken J E, Peters M C, Manton D J, Leal S C, Gordan V V, Eden E. Minimal intervention dentistry for managing dental caries – a review: report of a FDI task group. *Int Dent J* 2012; **62**: 223–243.
5. Schwendicke F, Frencken J E, Bjørndal L *et al*. Managing Carious Lesions: Consensus Recommendations on Carious Tissue Removal. *Adv Dent Res* 2016; **28**: 58–67.
6. Junger S, Payne S A, Brine J, Radbruch L, Brearley S G. Guidance on Conducting and Reporting Delphi Studies (CREDES) in palliative care: Recommendations based on a methodological systematic review. *Palliative Medicine* 2017; **31**: 684–706.
7. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* 2019; **393**: e44.
8. Keyes P H. The infectious and transmissible nature of experimental dental caries. Findings and implications. *Arch Oral Biol* 1960; **1**: 304–320.
9. Keyes P H, Fitzgerald R J. Dental caries in the Syrian hamster. IX. *Arch Oral Biol* 1962; **7**: 267–277.
10. Marsh P D. Dental plaque as a biofilm and a microbial community – implications for health and disease. *BMC Oral Health* 2006; **6**: S14.
11. Marsh P D. In Sickness and in Health – What Does the Oral Microbiome Mean to Us? An Ecological Perspective. *Adv Dent Res* 2018; **29**: 60–65.

12. Fisher-Owens S A, Gansky S A, Platt L J *et al.* Influences on children's oral health: a conceptual model. *Pediatrics* 2007; **120**: e510–520.
13. Gomez A, Espinoza J L, Harkins D M *et al.* Host Genetic Control of the Oral Microbiome in Health and Disease. *Cell Host Microbe* 2017; **22**: 269–278.
14. Takahashi N, Nyvad B. The role of bacteria in the caries process: ecological perspectives. *J Dent Res* 2011; **90**: 294–303.
15. Dawes C. What is the critical pH and why does a tooth dissolve in acid? *J Can Dent Assoc* 2003; **69**: 722–724.
16. Vieira A R, Gibson C W, Deeley K, Xue H, Li Y. Weaker dental enamel explains dental decay. *PLoS One* 2015; **10**: e0124236.
17. Weber M, Bogstad Sovik J, Mulic A *et al.* Redefining the Phenotype of Dental Caries. *Caries Res* 2018; **52**: 263–271.
18. Valdebenito B, Tullume-Vergara P O, Gonzalez W, Kretz J, Giacaman R A. *In silico* analysis of the competition between *Streptococcus sanguinis* and *Streptococcus mutans* in the dental biofilm. *Mol Oral Microbiol* 2018; **33**: 168–180.
19. Takahashi N, Nyvad B. Ecological Hypothesis of Dentin and Root Caries. *Caries Res* 2016; **50**: 422–431.
20. Banerjee A. 'Minimum intervention' – MI inspiring future oral healthcare? *Br Dent J* 2017; **223**: 133–135.
21. Schwendicke F, Gostemeyer G. Understanding dentists' management of deep carious lesions in permanent teeth: a systematic review and meta-analysis. *Implementation Sci* 2016; **11**: 142.
22. Innes N, Schwendicke F. Restorative Thresholds for Carious Lesions: Systematic Review and Meta-analysis. *J Dent Res* 2017; **96**: 501–508.
23. Albino J, Tiwari T. Preventing Childhood Caries: A Review of Recent Behavioral Research. *J Dent Res* 2016; **95**: 35–42.
24. Domejean S, Banerjee A, Featherstone J D B. Caries risk/susceptibility assessment: its value in minimum intervention oral healthcare. *Br Dent J* 2017; **223**: 191–197.
25. Ismail A. Diagnostic levels in dental public health planning. *Caries Res* 2004; **38**: 199–203.
26. Kassebaum N J, Bernabe E, Dahiya M, Bhandari B, Murray C J, Marcenes W. Global burden of untreated caries: a systematic review and metaregression. *J Dent Res* 2015; **94**: 650–658.
27. Raedel M, Hartmann A, Bohm S *et al.* Four-year outcomes of restored posterior tooth surfaces: a massive data analysis. *Clin Oral Invest* 2017; **21**: 2819–2825.
28. Burke F J, Lucarotti P S, Holder R L. Outcome of direct restorations placed within the general dental services in England and Wales (Part 2): variation by patients' characteristics. *J Dent* 2005; **33**: 817–826.
29. Schwendicke F, Gostemeyer G, Blunck U, Paris S, Hsu L Y, Tu Y K. Directly Placed Restorative Materials: Review and Network Meta-analysis. *J Dent Res* 2016; **95**: 613–622.
30. Elderton R J. Clinical studies concerning re-restoration of teeth. *Adv Dent Res* 1990; **4**: 4–9.
31. Brantley C, Bader J, Shugars D, Nesbit S. Does the cycle of rerestitution lead to larger restorations? *J Am Dent Assoc* 1995; **126**: 1407–1413.
32. Tyas M J, Anusavice K J, Frencken J E, Mount G J. Minimal intervention dentistry – a review. FDI Commission Project 1–97. *Int Dent J* 2000; **50**: 1–12.
33. Moynihan P J, Kelly S A M. Effect on Caries of Restricting Sugars Intake: Systematic Review to Inform WHO Guidelines. *J Dent Res* 2014; **93**: 8–18.
34. Slayton R L, Urquhart O, Araujo M W B *et al.* Evidence-based clinical practice guideline on nonrestorative treatments for carious lesions: A report from the American Dental Association. *J Am Dent Assoc* 2018; **149**: 837–849.
35. Marinho V C, Worthington H V, Walsh T, Clarkson J E. Fluoride varnishes for preventing dental caries in children and adolescents. *The Cochrane database of systematic reviews* 2013; **7**: Cd002279.
36. Walsh T, Worthington H V, Glenny A M *et al.* Fluoride toothpastes of different concentrations for preventing dental caries in children and adolescents. *The Cochrane database of systematic reviews* 2010; **10**: Cd007868.
37. Wolff M S, Schenkel A B. The Anticaries Efficacy of a 1.5% Arginine and Fluoride Toothpaste. *Adv Dent Res* 2018; **29**: 93–97.
38. Wierichs R J, Meyer-Lueckel H. Systematic Review on Noninvasive Treatment of Root Caries Lesions. *J Dent Res* 2015; **94**: 261–271.
39. Baysan A, Lynch E, Ellwood R *et al.* Reversal of primary root caries using dentifrices containing 5,000 and 1,100 ppm fluoride. *Caries Res* 2001; **35**: 41–46.
40. Ekstrand K, Martignon S, Holm-Pedersen P. Development and evaluation of two root caries controlling programmes for home-based frail people older than 75 years. *Gerodontology* 2008; **25**: 67–75.
41. Ekstrand K R, Poulsen J E, Hede B *et al.* A randomized clinical trial of the anti-caries efficacy of 5,000 compared to 1,450 ppm fluoridated toothpaste on root caries lesions in elderly disabled nursing home residents. *Caries Res* 2013; **47**: 391–398.
42. Marinho V C, Chong L Y, Worthington H V, Walsh T. Fluoride mouthrinses for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev* 2016; **7**: Cd002284.
43. Gao S S, Zhang S, Mei M L, Lo E C, Chu C H. Caries remineralisation and arresting effect in children by professionally applied fluoride treatment – a systematic review. *BMC Oral Health* 2016; **16**: 12.
44. Fung M H T, Duangthip D, Wong M C M, Lo E C M, Chu C H. Randomized Clinical Trial of 12% and 38% Silver Diamine Fluoride Treatment. *J Dent Res* 2018; **97**: 171–178.
45. Alkilzy M, Santamaria R M, Schmoeckel J, Splieth C H. Treatment of Carious Lesions Using Self-Assembling Peptides. *Adv Dent Res* 2018; **29**: 42–47.
46. Fontana M. Enhancing Fluoride: Clinical Human Studies of Alternatives or Boosters for Caries Management. *Caries Res* 2016; **50 Suppl 1**: 22–37.
47. Alkilzy M, Tarabai A, Santamaria R M, Splieth C H. Self-assembling Peptide P11–4 and Fluoride for Regenerating Enamel. *J Dent Res* 2018; **97**: 148–154.
48. Krois J, Gostemeyer G, Reda S, Schwendicke F. Sealing or infiltrating proximal carious lesions. *J Dent* 2018; **74**: 15–22.
49. Schwendicke F, Jager A M, Paris S, Hsu L Y, Tu Y K. Treating pit-and-fissure caries: a systematic review and network meta-analysis. *J Dent Res* 2015; **94**: 522–533.
50. Griffin S O, Oong E, Kohn W *et al.* The Effectiveness of Sealants in Managing Caries Lesions. *J Dent Res* 2008; **87**: 169–174.
51. Fontana M, Platt J A, Eckert G J *et al.* Monitoring of sound and carious surfaces under sealants over 44 months. *J Dent Res* 2014; **93**: 1070–1075.
52. Hesse D, Bonifacio C C, Mendes F M *et al.* Sealing versus partial caries removal in primary molars: a randomized clinical trial. *BMC Oral Health* 2014; **14**: 58.
53. Bakhshandeh A, Qvist V, Ekstrand K. Sealing occlusal caries lesions in adults referred for restorative treatment: 2–3 years of follow-up. *Clin Oral Invest* 2012; **16**: 521–529.
54. Mickenausch S, Yengopal V. Validity of sealant retention as surrogate for caries prevention – a systematic review. *PLoS One* 2013; **8**: e77103.
55. Paris S, Hopfenmuller W, Meyer-Lueckel H. Resin Infiltration of Caries Lesions. *J Dent Res* 2010; **89**: 823–826.
56. Gruythuysen R. Non-Restorative Cavity Treatment. Managing rather than masking caries activity. *Nederlands tijdschrift voor tandheelkunde* 2010; **117**: 173–180.
57. Mijan M, de Amorim R G, Leal S C *et al.* The 3.5-year survival rates of primary molars treated according to three treatment protocols: a controlled clinical trial. *Clin Oral Invest* 2014; **18**: 1061–1069.
58. Santamaria R M, Innes N P T, Machiulskiene V *et al.* Alternative Caries Management Options for Primary Molars: 2.5-Year Outcomes of a Randomised Clinical Trial. *Caries Res* 2017; **51**: 605–614.
59. Hansen N V, Nyvad B. Non-operative control of cavitated approximal caries lesions in primary molars: a prospective evaluation of cases. *J Oral Rehab* 2017; **44**: 537–544.
60. Lo E C, Schwarz E, Wong M C. Arresting dentine caries in Chinese preschool children. *Int J Paed Dent* 1998; **8**: 253–260.
61. Hickel R, Kaaden C, Paschos E *et al.* Longevity of occlusally-stressed restorations in posterior primary teeth. *Am J Dent* 2005; **18**: 198–211.
62. Innes N P, Evans D J, Stirrups D R. Sealing caries in primary molars: randomized control trial, 5-year results. *J Dent Res* 2011; **90**: 1405–1410.
63. 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.
64. Braga M, Mendes F, Martignon S, Ricketts D, Ekstrand K. *In vitro* comparison of Nyvad's system and ICDAS-II with lesion activity assessment for evaluation of severity and activity of occlusal caries lesions in primary teeth. *Caries Res* 2009; **43**: 405–412.
65. Braga M M, Martignon S, Ekstrand K R, Ricketts D N, Imperato J C, Mendes F M. Parameters associated with active caries lesions assessed by two different visual scoring systems on occlusal surfaces of primary molars – a multilevel approach. *Comm Dent Oral Epidemiol* 2010; **38**: 549–558.
66. Nyvad B, Fejerskov O. Assessing the stage of caries lesion activity on the basis of clinical and microbiological examination. *Comm Dent Oral Epidemiol* 1997; **25**: 69–75.
67. Ferreira Zandona A, Santiago E, Eckert G J *et al.* The natural history of dental caries lesions: a 4-year observational study. *J Dent Res* 2012; **91**: 841–846.
68. Wenzel A. Radiographic display of carious lesions and cavitation in approximal surfaces: Advantages and drawbacks of conventional and advanced modalities. *Acta Odontol Scand* 2014; **72**: 251–264.
69. Tassery H, Levallois B, Terrer E *et al.* Use of new minimum intervention dentistry technologies in caries management. *Aust Dent J* 2013; **58**: 40–59.
70. Broadbent J M, Foster Page L A, Thomson W M, Poulton R. Permanent dentition caries through the first half of life. *Br Dent J* 2013; **215**: E12. <https://doi.org/10.1038/sj.bdj.2013.991>.
71. Mejare I, Axelsson S, Dahlen G *et al.* Caries risk assessment. A systematic review. *Acta Odontol Scand* 2013; **72**: 81–91.
72. Lopez R, Smith P C, Gostemeyer G, Schwendicke F. Ageing, dental caries and periodontal diseases. *J Clin Perio* 2017; **44**: S145–S152.
73. Bratthall D, Hansel Petersson G. Cariograma multifactorial risk assessment model for a multifactorial disease. *Comm Dent Oral Epidemiol* 2005; **33**: 256–264.
74. Hayes M, Da Mata C, McKenna G, Burke F M, Allen P F. Evaluation of the Cariogram for root caries prediction. *J Dent* 2017; **62**: 25–30.
75. Domejean S, White J M, Featherstone J D. Validation of the CDA CAMBRA caries risk assessment – a six-year retrospective study. *J Calif Dent Assoc* 2011; **39**: 709–715.
76. Innes N P, Manton D J. Minimum intervention children's dentistry – the starting point for a lifetime of oral health. *Br Dent J* 2017; **223**: 205–213.
77. Leal S C. Minimal intervention dentistry in the management of the paediatric patient. *Br Dent J* 2014; **216**: 623–627.
78. Manhart J, Chen H, Hamm G, Hickel R. 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.
79. Ribeiro A A, Purger F, Rodrigues J A *et al.* Influence of contact points on the performance of caries detection methods in approximal surfaces of primary molars: an *in vivo* study. *Caries Res* 2015; **49**: 99–108.
80. Smail-Faugeron V, Glenny A M, Courson F, Durieux P, Muller-Bolla M, Fron Chabouis H. Pulp treatment for extensive decay in primary teeth. *Cochrane Database Syst Rev* 2018; **5**: Cd003220.

Appendix 1 A detailed description of the methods used, including the guidance on conducting and reporting Delphi studies

The expert group represented members of the European Association for Caries Research (ORCA) and the European Federation of Conservative Dentistry (EFCD) as well as (mainly overseas) non-members. The group was organised and the process led by two members, FS and DJM. These members also organised financial support for the meeting. The members of the expert group were chosen based on their clinical and scientific expertise, allowing sufficient breadth of experience, as well as geographic aspects. All experts were familiar to one or both organisers. Note that some experts came from the same institution; no weighting or adjustment during the consensus was performed for this, as any kind of possible bias introduced by this was assumed to be limited and was accepted, but also as no valid rules for such weighting or adjustment are available.

Both ORCA and EFCD approved and supported the initiative, its aims and the meeting, and the then-president-elect of ORCA and the then-acting-president of EFCD were members of the group. As described, all members of the group provided a conflict of interest declaration and no member was found to be subject to relevant conflict of interest related to the consensus statement.

Prior to the meeting, a working paper, which also formed the basis for the present consensus document, was drafted by a smaller group of members, whose task it was to sum up and synthesise the available evidence for the different levels of invasiveness (NI, MI, invasive) as well as the evidence base towards possible intervention thresholds. Note that no systematic review process was performed, but existing reviews were considered. The compiled draft was sent to the overall group, who commented on it extensively, in two rounds. The resulting manuscript was the basis of the following steps and included consensus recommendations. Only these recommendations were voted on during the subsequent Delphi process; the text itself (excluding the recommendations) was not submitted to any further consensus process as we felt the core of the consensus was the recommendations.

A two-staged confidential e-Delphi survey was then undertaken. Between the two Delphi rounds, the consensus panel meeting was held. The reporting for this Delphi follows the Guidance on conducting and reporting Delphi studies (CREDES),⁶ with all points being laid out below once more for reasons of clarity.

Rationale for the choice of the Delphi technique

1. Justification: A stepwise approach of coming to a consensus on a set of evidence-based statements, after discussion first via email/text, then in a form of a meeting, was decided to be built on the Delphi technique. This technique is transparent, anonymous in voting and accepted by the community. Further, it was feasible and fitted to the specific design of this consensus process. By combining an open-ended approach with a Delphi, a systematic, but nevertheless comprehensive approach was encouraged.

Planning and design

2. Planning and process. The consensus rules (see below) were agreed to by the panel via email before starting the Delphi process. Modifications are described below. The Delphi asked for an agreement to each consensus statement (as can be found in the consensus recommendations section of the main paper), with a scale of 0–10 (do not at all agree to agree fully) being used. A multi-stage Delphi was planned, without removal of any items prior to concluding at maximum three rounds. Each round closed after a 4-week period. One reminder via email was sent for each round. Panellists were allowed to comment on each item. The survey was conducted via Delphi Manager 3.0, University of Liverpool, UK, and Surveyjet (Calibrium, <https://calibrium.com>), and survey data was analysed descriptively.
3. Definition of consensus. The following consensus rules applied: (1) Agreement to an item was defined by marking grades 7–10 on a scale from 1–10; (2) Minimum 70% of all participants needed to agree to an item for this to be consensually accepted. Items which did not meet these criteria after the planned two rounds were to be dropped (no item was eventually dropped). For reasons of transparency, we additionally report on the mean agreement and the standard deviation

Study conduct

4. Informational input: The material provided to the panel is described in the main text. Its attainment has been described above.
5. Prevention of bias: To identify possible risk of bias, all members filled out a conflict of interest form. To prevent bias, a systematic, evidence-grounded approach was chosen. Note that the topic itself does only limitedly lend itself for financial/commercial bias. The planning and conduct were performed independent from the sponsor.
6. Interpretation and processing of results: There was, as discussed stable agreement to all items after the second round.
7. External validation: No external validation was sought.

Reporting

8. Purpose and rationale: These have been provided.
9. Expert panel: The criteria for the selection of experts was provided.
10. Description of the methods: Preparatory steps, synthesis of the evidence, piloting of the statements, survey rounds and conference have been described.
11. Procedure: The Delphi steps have been described.
12. Definition and attainment of consensus: The following consensus rules applied: (1) Agreement to an item was defined by marking grades 7–10 on a scale from 1–10; (2) Minimum 70% of all participants needed to agree to an item for this to be consensually accepted.
13. Results: The results are reported in the main text. Note that between steps, at the panel meeting, a discussion on all items was held; these discussions had not been planned a priori but found necessary after the first round and the revision of the manuscript. Some items, mainly those showing low agreement in round 1, were revised in language or content, and all items provided to the group in the second round. A consensus was reached on all items in the second Delphi round. All panellists except one took part in both Delphi rounds.
14. Discussion of limitations: A limited group of people have been invited and came to this consensus, which is a limitation. Moreover, and as laid out, most statements are not supported by strong evidence, as this is missing.
15. Adequacy of conclusions: The conclusions reflect the outcomes of the Delphi and aim for applicability of the deduced guidance points.
16. Publication and dissemination: The consensus paper will be translated in various languages and published in national journals for dissemination.