Beyond *Streptococcus mutans*: clinical implications of the evolving dental caries aetiological paradigms and its associated microbiome

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Key points

- Provides an update on the current understanding of the aetiological paradigms of the dental caries process.
- Emphasises the importance of maintaining a healthy diverse oral microbiome for long-term caries control.
- Discusses oral care implications of the new aetiological concepts of dental caries.

Aetiological concepts of dental caries have evolved over the years from being considered as a disease initiated by nonspecific microorganisms, to being regarded as an 'infectious' disease caused by specific bacteria, to the current paradigms that emphasise a 'mixed bacterial-ecological approach' as being responsible for lesion initiation and pathogenesis. These aetiological paradigms are not just intellectual concepts but have important implications on how clinicians manage this age-old disease in the twenty-first century. Despite evidence-backed recommendations for adopting more biological measures to counter the disease, a significant proportion of dentists continue following traditional caries management guidelines in their daily clinical practice. This paper will review the evolving dental caries aetiological concepts and highlight the current evidence for adopting a more ecological approach to caries prevention, risk assessment, and treatment.

Introduction

Dental caries remains the most common chronic disease in children (being five times as common as asthma), and is a major contributor to tooth loss in adults.[1] In fact, the most recent Global Burden of Disease report revealed that untreated caries in permanent teeth was the most prevalent human disease condition worldwide, with untreated caries in primary teeth being the tenth most prevalent disease.[2] Dental caries is now recognised to belong to a group of diseases like cancer or diabetes that are considered 'complex' or 'multifactorial' with no single causation pathway.[3] There have been important paradigm shifts in the aetiological concepts of the disease and it is now widely recognised that the earlier singular focus on *Streptococcus mutans* to assess caries risk status or success of preventive measures and treatment interventions is no longer a viable strategy. This article highlights the evolution of dental caries aetiological theories over the years and how this continues to have important implications for caries prevention, risk assessment, and treatment.

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Evolving paradigms of caries

The role of microbes in the development of caries lesions was suggested as early as 1890 with the chemoparasitic theory of Miller,[4] and by the end of the nineteenth century it was commonly accepted that dental diseases were caused by the nonspecific overgrowth of bacteria in dental plaque.[5] According to this nonspecific plaque hypothesis (NSPH) it was the quantity of plaque that determined levels of pathogenicity without discriminating between different levels of bacterial virulence. Applying the NSPH, it was recommended that the best way for preventing caries would be the mechanical removal of as much plaque as possible by tooth brushing or flossing.[6]

By the mid-twentieth century, Keyes' and Fitzgerald's famous animal model experiments revolutionised caries aetiological concepts by demonstrating that dental caries was an 'infectious and transmittable' disease.[7,8] In 1976, Loesche announced the specific plaque hypotheses (SPH), postulating that dental caries was an 'infection' caused by specific bacteria within dental plaque.[9] The specific bacteria that have for long been the *cause célèbre* for caries initiation and progression belonged to a group of Gram-positive acidogenic and aciduric bacterial species, now designated as mutans streptococci (MS), of which *Streptococcus mutans* and *Streptococcus sobrinus* are the most common in humans. For decades most diagnostic, preventive and therapeutic interventions were directed against these microorganisms. For example, the SPH proposed the use of antibiotics against specific bacterial species as a method to prevent and treat dental caries.[10]

However, it is now acknowledged that Robert Koch's postulates on infectious diseases that focused on specific pathogens as causative agents of disease are not applicable for microbial community-based diseases like dental caries or periodontitis. The bulk of the data that supports a relation between MS and dental caries can be considered associative rather than causative.[11] There is also evidence showing that individuals with high MS levels do not necessarily develop caries lesions, while

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lesions have been detected even in the absence of MS.[12,13] Furthermore, the specific bacteria suggested to be responsible for caries are actually part of the indigenous microflora and unlike foreign pathogens cannot be eliminated for long from the oral cavity by use of antibiotics. Thus, while dental caries is of course microbially induced, the important point is that it is caused by endogenous bacteria that are a normal part of the resident microbiome, and not by specific exogenous bacteria acquired from outside the host, as in other infectious diseases.[3]

The bacterial-ecological approach

Contemporary concepts of dental caries aetiology and pathogenesis emphasise a 'mixed bacterial-ecological approach' as being responsible for lesion initiation and progression.[13] Rather than being considered an 'infectious disease' caused by a specific organism, dental caries is now understood to be a biofilm-mediated disease.[14] Caries lesions develop due to a catastrophic ecological shift in the plaque biofilm microbial flora, instigating an imbalance in the physiologic equilibrium between tooth mineral and biofilm fluid, and ultimately tipping the caries balance towards demineralisation and lesion formation.[15] According to this ecological plaque hypothesis (EPH), the critical factors that trigger an upsurge in the acidogenic/aciduric component of the oral microbiome are local environmental conditions like frequent dietary sugar exposures or salivary dysfunction. Dental caries can thus be considered to be an endogenous infection which may occur when members of resident flora obtain a selective ecological advantage over other species, disrupting the homeostatic balance of the biofilm, and thereby initiating the disease process.[16]

Takahashi and Nyvad proposed an extension of the caries EPH to explain the relation between the dynamic changes in the phenotypic/genotypic properties of plaque bacteria and the de/remineralisation equilibrium of the caries process.[17,18] They suggested that the oral plaque biofilm is a dynamic microbial ecosystem with different microbial communities associated with the three reversible stages of the caries process (dynamic stability stage, acidogenic stage, and aciduric stage). Mutable bacterial ecological succession takes place in the plaque biofilm at each stage of the caries process, depending on the severity and frequency of biofilm acidification.[18] It is only when the acidogenic environment is prolonged that highly aciduric bacteria, like MS or lactobacilli, begin to dominate and replace the 'low pH' non-MS, *Actinomyces* spp., or *Veillonella* spp. that are the main species populating the plaque microflora in the early stages of incipient (non-cavitated) carious lesions. Even at the highly aciduric stage, microbial composition and mineral loss can be reversed, provided the acidogenic/aciduric properties of the biofilm are modified by adopting effective preventive measures (for example, by restricting sugar exposures).[17] The significance of the extended EPH model is that it is not the bacterial genotype *per se,* but the phenotypic characteristics (acidogenic and aciduric properties) and their regulatory parameters that are more relevant for causing a microbial ecological shift leading to caries.[18] There is now a consensus that any bacterial species can participate in the caries process as long as they are aciduric and dominant.[19]

These caries ecological concepts have been confirmed by recent DNA- and RNA-based molecular studies that have uncovered an extraordinarily diverse microbial ecosystem, where *S. mutans* accounts for a very small fraction (0.1%–1.6%) of the bacterial community implicated in the caries process.[20] Oral microbiologists have now expanded the principal bacterial species in caries-associated microbiomes from the traditional culture-isolated cariogenic bacteria like *S. mutans*, *S. sobrinus*, *Lactobacillus,* and *Actinomyces* to include bacterial species like non-MS, *Bifidobacterium*, *Scardovia wiggsiae*, *Prevotella* spp., *Selenomonas*, *Olsenella* spp., *Atopobium* spp., *Capnocytophaga* and many more.[21–23] In addition, bacterial-fungal associations can also synergistically enhance cariogenic biofilm virulence, with *Candida albicans* frequently detected in high numbers in dental plaque of children with early childhood caries (ECC).[24,25] This was an intriguing observation as *Candida* does not normally associate well with *S. mutans*, nor colonise teeth or metabolise sucrose effectively on its own.[26] However, recent studies have demonstrated that a symbiotic relationship exists between *S. mutans* and *C. albicans,* mediated through the influence of bacterially-derived glucosyltransferases, contributing to the increased severity of ECC.[27–29] These findings prompt the possibility of incorporating anti-*Candida* therapy also in the treatment of virulent ECC.[26]

The polymicrobial nature of carious lesions implies that consortia formed by multiple microorganisms act collectively, probably synergistically, to initiate and advance the disease.[20] The RNA-based estimates of microbial diversity over carious lesions also show different microbial consortia are formed in the dental plaque of different individuals. These microbial combinations have essentially the same functional profile, indicating that focusing on metabolic output of microbial communities would be more useful in controlling the disease regardless of the specific microbial compositions involved in the process.[18,20]

Health-associated microbial communities

Another evolving paradigm in our understanding of the caries associated microbiome is the role that health-promoting oral microbial communities can play in mitigating acid produced by cariogenic bacteria.[30] Many oral commensal bacteria can counter the low pH produced by acidogenic organisms by using arginine or urea to generate ammonia.[31] Such alkaline metabolites produced by 'healthy' bacteria can play a major role in maintaining biofilm homeostasis to beneficially alter the de/remineralisation equilibrium. Research has also shown microbial biodiversity to be crucial to health, with plaque and saliva of healthy individuals revealed to be much more diverse than originally hypothesised.[32] Results from clinical studies have shown that oral microbiomes of children with ECC were significantly less diverse than those of children without ECC, confirming that diversity in plaque microbial communities is essential for maintaining oral health.[33] From the applied viewpoint, the use of broad-spectrum biocides like chlorhexidine for long-term caries control may not be desirable as they would cause an indiscriminate suppression of even healthy oral microbiota.[34]

Genetic factors

Genetic factors can also be responsible for microbial ecological shifts that lead to disease. An individual's genotype can potentially prevent the existence of certain beneficial bacteria or allow pathogenic species to reside and contribute to its unique microbiome.[35] The criticism of the caries EPH is that it does not consider host genetic susceptibility to caries.[6] A salient observation from the unethical Vipeholm study was that about 20% of the individuals who received high frequency cariogenic snacks for two years did not develop dental caries, and this genetic resistance to caries was further supported by the observation that parents and siblings of these individuals also showed lower caries prevalence

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than the rest of the population.[36,37] There is now additional recent evidence that certain at-risk individuals and population groups are genetically more susceptible to dental caries,[38–40] implying that hereditary factors should also be considered while planning individual caries management protocols.

In summary, the current biofilm model of dental caries presents a complex picture of a multifactorial, pH-driven disease whose onset and progression is influenced by multiple pathogens, systemic effects, and hereditary components layered on interactions of diet, behavioural, environmental, socioeconomic, and physiological risk factors. The caries process can be considered as a model system of dynamic amphibiosis, where under 'normal' environmental conditions the biofilm micro-organisms live in a symbiotic relationship with the host, characterised by commensalism and mutualism.[41] The nature of this symbiosis may shift under changing local environmental conditions with mutualism becoming parasitism, and this dynamic adaptation is the basic principle that underlines our current understanding of endogenous disease processes like dental caries.

Oral care implications

The current aetiological paradigms of dental caries have important consequences for oral care. Complex diseases like dental caries that arise from the concerted action of biofilm-embedded polymicrobes, risk-conferring behaviours, environmental influences, and genes present clinical challenges on how these multiple contributing factors can be assessed in a way that can be translated into effective approaches for caries prevention, risk assessment, and therapy. Contemporary evidence-backed recommendations for management of dental caries, based on the ecological-biofilm concepts of the disease, have revolutionised many conventional caries management philosophies, and clinicians need to be aware of the emerging evidence with regards to prevention, risk-assessment and treatment of dental caries.

Dental caries prevention

With the mechanism of dental caries now well established, new strategies are being sought for better targeted caries prevention based on a scientific understanding of the processes involved. The earlier focus on determining the specific causative agents is giving way to ecology-based propositions where the disease is seen as the output of a skewed microbial community caused by environmental changes.[20] While fluoride will remain the cornerstone of any dental caries preventive protocol, a consensus is emerging that preventive measures should also focus on remediation of environmental pressures responsible for the plaque biofilm dysbiosis, while maintaining the resident microbiota at levels compatible with health.[42]

Advising patients to reduce dietary exposure to fermentable carbohydrates is clearly the most effective approach to prevent acidification and the detrimental ecological shift of the plaque biofilm. However, requiring individuals to modify their dietary behaviour as part of caries prevention is usually the most difficult advice for patients to adhere to.[43] Additional preventive measures that are synergistic with fluoride have thus been advocated for high caries-risk groups.[44] Ecological caries preventive approaches are probably the next frontier in the long-standing attempts to obtain control over this ubiquitous disease. These preventive measures are based on the current caries aetiological paradigms, where an ecologically balanced and diverse microbiome is seen as the key to long-term control over the disease. Ecological preventive approaches can broadly be divided into measures that either enhance the growth of health-promoting microbial communities within the plaque biofilm, or antimicrobial strategies that can weaken cariogenic biofilm virulence properties without eliminating health-associated oral microflora. Examples include prebiotics, probiotics, antimicrobial peptides (AMP), sugar polyols, phytochemicals, quorum-sensing (QS) targets, and genetically-modified 'designer' bacteria.

The ecological preventive approach that is probably the most evidence-backed is the use of prebiotics like arginine or urea. Prebiotics are nutritional substrates that commensal bacteria can breakdown to alkalise the biofilm, thus preventing the overgrowth of acidogenic/aciduric bacteria. The nutritional stimulation of endogenous oral flora to restore microbial balance and promote oral health has been validated in mixed species models.[45] A substantial body of evidence from microbiological, genetic, and biochemical analyses suggests that alkali generation in dental biofilms contributes to inhibition of dental caries.[46] Additionally, human *in situ* studies, double-blinded RCTs, and systematic reviews have demonstrated that a 1.5% arginine-containing fluoride toothpaste (Colgate Maximum Cavity Protection plus Sugar Acid Neutraliser) provided significantly greater protection against caries than a fluoride dentifrice alone.[47–51] Although other reviews have questioned the dentifrice's preventive efficacy and its comparative higher costs,[52,53] the preponderance of evidence does seem to suggest that arginine-fluoride dentifrices offer a new standard of care for caries prevention in high-risk patients.[54] An alternative to arginine dentifrices may be a toothpaste containing enzymes and proteins (Zendium), that was recently shown to boost oral defences by promoting a beneficial shift in the ecology of the oral microbial community over time.[55] More comprehensive randomised control trials (RCTs) regarding its caries preventive effects are required before firm clinical recommendations can be made.

The use of probiotic bacteria (lactobacilli and bifidobacteria) to support health-associated microbes or restore diversity in the oral plaque biofilm is enjoying growing popularity as an ecological method to control dental caries. However, while probiotics may indeed improve surrogate caries markers (for example, reduced MS counts),[56] there are doubts on whether this actually results in any reduction in individual caries experience.[57] Another drawback of using traditional probiotics for caries prevention is the potentially harmful acidogenic effects of many commonly used probiotic bacterial strains.[58] Furthermore, the traditional use of gut-associated lactobacilli and bifidobacteria probiotic species to promote oral health may not succeed as these non-oral bacterial strains cannot efficiently colonise the oral niche, which is vital for the long-term success of probiotics. A more promising breakthrough in the use of oral probiotics for caries prevention is the recent identification of two *S. mutans*-antagonistic oral probiotic strains, *Streptococcus dentisani* and *Streptococcus* A12. Both these bacteria are natural oral commensal species having double probiotic action, as they can not only inhibit the growth of major oral pathogens but can also moderate plaque pH through their arginolytic actions.[59,60] However, clinical recommendations would be premature at this stage without evidence from RCTs showing significant reductions in caries increment on using these oral probiotics.

Another long-standing approach to promote healthy biofilms is the use of non-fermentative sugar polyols (for example, xylitol, sorbitol, erythritol) that are believed to inhibit virulent bacteria allowing healthy bacteria to dominate in the biofilm. Despite an immense body of

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literature, the caries preventive effects of xylitol oral products remains inconclusive, with more recent data concluding that there is limited evidence to show that xylitol provided any additional benefit beyond other preventive measures.[61–63] Among the sugar polyols, erythritol may be the one that holds most promise, as new studies indicate that it may be more effective than xylitol and sorbitol, and critically, its anticaries effects were shown to persist for up to three years after the intervention ended.[64–66]

The other side of the ecological preventive approach is to use antimicrobial agents to modify the plaque biofilm. It is logical to assume that caries prevention can potentially be improved by combining fluoride, which predominantly has physiochemical effects on the hard dental tissues, with biological agents that can reduce the severity of the bacterial attack on teeth. However, many of the commonly available toothpastes and mouthwashes use broad-spectrum antimicrobials which cause an undesirable suppression of even the healthy plaque microflora. There is now a consensus that antimicrobials used for dental caries prevention should be more subtle in their antiplaque actions, aiming to undermine bacterial virulence properties (for example, glucan synthesis or acid production) rather than bacterial viability.[67] Promising antimicrobial measures with biofilm-modifying cariostatic action include specifically targeted antimicrobial peptides (STAMP),[68] natural phytochemicals,[69,70] genetically-engineered *S. mutans* strains,[71,72] and QS-targeting compounds.[73,74] Although promising, the ultimate success of these antimicrobial ecological approaches will depend on their ability to effectively act in highly cariogenic oral environments, often found in high-risk individuals, where there is frequent biofilm acidification due to multiple sugar exposures.

The current paradigms of dental caries has implications for dental public health programmes too. A consequence of dental caries being a multifactorial disease is that a particular preventive programme that is successful in one population group, need not be so in another population group with different cultural and behavioural habits. The complexity of the disease also implies that dental caries can never be 100% preventable at the individual or societal level.[3] What this implies is that a well-trained dentist should regularly monitor patients for any change in their oral environmental conditions to avoid new lesions.

Caries-risk assessment

Caries-risk assessment (CRA) is fundamental to modern caries management protocols, assisting the clinician in making decisions regarding the type of diagnostic procedures, preventive/treatment interventions, and recall intervals required for each patient. When high caries-risk patients are effectively identified, more resources and intensive preventive interventions can be directed towards them, while avoiding redundant diagnostic procedures and therapy in patients at low-risk of developing caries lesions. Past caries experience has been demonstrated to be the best risk predictor of the disease in every age group.[75,76] However, this metric does not provide an explanation for the heightened risk, and thus may not suggest the best avenue for prevention.

Microbiological tests on the levels of MS or lactobacilli in plaque or saliva have traditionally been used to predict an individual's risk of developing caries lesions. However, with an entire consortium of acidogenic/aciduric bacterial species now known to be involved in the caries process, such microbial tests have limited applicability in assessment of caries activity and in risk prediction.[77] For instance, eating habits and the socio-economic status of children and their caregivers have been shown to be better predictors of ECC than MS titre levels.[78] The high MS levels detected in patients with severe ECC can be considered an incidental consequence of the frequent acidification of plaque by poor eating habits, rather than an early risk predictor.[18] The fact that *S. wiggsiae* has been shown to have the same prevalence as *S. mutans* in ECC lesions, or that *Bifidobacterium dentium* was found in cavitated lesions but never in healthy plaque, suggests that assessing combined levels of several indicator aciduric bacterial taxa may be more useful for risk prediction.[79] Microbiological tests should only be considered as one component of the patient's caries risk history, establishing a norm for the individual patient, with any deviations from this indicating a change in the oral environment.[77]

The recognition of the multifactorial aetiology of dental caries has led experts and professional bodies to develop multivariate CRA systems accounting for each of the disease-associated factors, assessing them not in isolation but with regards to their unique and dynamic interactions. Examples of some of the currently recommended CRA protocols include the cariogram model,[80] the caries management by risk assessment (CAMBRA) system,[81,82] the American Academy of Paediatric Dentistry's Caries-Risk Assessment Tool (CAT),[83] systems of the American Dental Association (ADA),[84,85] the traffic light matrix system (TLM),[86] and the Dundee Caries Risk Assessment Model (DCRAM).[87]

The clinical utility of these protocols to guide the design of precise personalised care has been questioned with a systematic review finding that the validity of some the commonly used CRA protocols (cariogram, CAMBRA, CAT, and ADA) to accurately predict caries risk is limited.[88] This has been confirmed with the Cariogram model shown to be not 'particularly useful in identifying high caries risk patients in a low-caries community',[89] while the CAMBRA protocol was not able to significantly distinguish between the low and moderate caries-risk groups.[90] A more recent critical review suggested that it was fallacious to transfer and apply population risk estimates to assess individual caries risk.[91] For example, ECC risk factors shown to be consistently correlated with caries prevalence in large population studies are poor predictors of individual lesion occurrence.

However, many of these CRA tools still have excellent pedagogical value, facilitate communication with patients, and have the potential to enhance oral care by identifying the specific causes for the caries activity, allowing the clinician to customise the treatment plan according to individualised needs of patients.[91,92] For a more comprehensive risk assessment and personalised caries care plan, clinicians may be advised to adopt the international caries classification and management system (ICCMS)[93] or the system for total environmental management (STEM)[94] protocol, as these include explicit patient-level assessments as part of the overall disease management.

Dental caries treatment

Caries treatment approaches have progressed over an extended timeframe from the early extractive phase to the commonly practised restorative phase, through to the currently recommended preventive/preservative phase.[95] The evidence for the evolution of caries management philosophies from a restorative-only approach to a preventive/preservative approach has now been available for at least a couple of decades.[96,97] Unfortunately, caries management continues to be skewed to 'drilling' and 'filling', with a recent systematic review and meta-analysis finding that a significant proportion of dentists continue to intervene

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invasively (restoratively) on carious lesions where clinical recommendations indicate that less invasive therapies should be used.[98] The largely technical and mechanistic approaches to tooth restoration ignores the emerging evidence strongly recommending more biological approaches to controlling disease causative factors and the need for clinicians to adopt the principles of minimum intervention dentistry (MID) for treating non-cavitated and cavitated lesions.[99] The overarching approach to caries management in the twenty-first century should be to 'preserve the tooth structure and restore only when necessary'.[100]

The rationale underpinning many of the current recommendations for managing caries lesions are based on the aetiological paradigms of EPH. Fundamental to the EPH is that unless there is an attempt to interfere with the environmental factors driving the biofilm dysbiosis, the patient is likely to suffer from repeated episodes of the disease and the clinician will encounter frequent failure of any restorative or preventive treatment rendered. Most restoration failure is due to secondary caries associated with restorations and sealants (currently referred to as CARS), highlighting that patient risk factors should be regularly monitored and actively managed.[92] While repairing cavitations (the end result of the disease process) is important, these are merely symptoms of the disease, and alleviating symptoms by restorations alone cannot proffer a 'final solution' to the disease.[94] It is wrong to assume that drilling out a caries lesion and placing a restoration eliminates bacteria and stops the disease process.[101] With the extended EPH establishing a firm association between plaque microbiome diversity and the de/remineralisation caries equilibrium, a biological understanding of the disease process and its associated microbiome is vital for successful clinical management of dental caries.

There are now excellent papers detailing the contemporary guidelines for operative management of caries lesions in permanent and primary teeth based on the recommendations of the International Caries Consensus Collaboration (ICCC).[102,103] These consensus recommendations underscore the pathological basis of dental caries as a biofilm disease and emphasise that both the prevention of new lesions and management of existing lesions should primarily focus on biofilm management rather than tissue removal. Although some of these recommendations may seem counterintuitive to decades of clinical practice and dental training, they are in keeping with our current understanding of the dental caries disease process. For instance, in teeth with deep cavitated lesions, it is recommended that carious tissue be removed only to create conditions for long-lasting, tightly sealed restorations, and all the bacterially contaminated (soft/infected dentine) or demineralised tissue close to the pulp need not be removed.[103] This makes sense once it is accepted that dental caries is tissue destruction caused by bacterial metabolism in the biofilm – if the disease process can be arrested by modifying the biofilm, the symptoms of the disease (demineralised dentine) is removed purely to create a sufficiently large surface to bond to and optimise the longevity of a restoration. Once bacteria are sealed into the tooth, the biofilm is physically prevented from accessing nutrition and an actively carious lesion becomes an arrested lesion.

A number of studies have clearly shown that the microbiological load in infected dentine is progressively reduced when it is sealed off from the oral environment.[97,104–106] The gradual reduction in lesion activity allows time for the pulp-dentine complex to lay down tertiary and peri-tubular dentine providing further protection to the pulp complex and reducing risk of pulp exposure.[107] Multiple RCTs and systematic reviews have not found any detrimental effects to the pulp by sealing in bacteria.[97,107–112] These include the classical studies of the Mertz-Fairhurst group[97] which showed that bonded and sealed composite restorations placed over frank cavitations arrested the clinical progress of these lesions even at ten years, profoundly changing our concepts of how much demineralised dentine may be left during cavity preparation.[113] A similar rationale supports the use of the Hall Technique, where preformed stainless steel crowns are placed directly over asymptomatic carious primary molar teeth without removing any carious tissue (and hence not requiring any local anaesthesia or tooth preparation). This radical treatment option gave rise to a great deal of controversy and questions in the past,[114,115] but as authors of a recent seminal paper on the Hall Technique assert 'emotion, misinformation, and outdated ideas have been used in arguments against Hall crowns rather than logic, understanding or evidence'.[116] There is now robust evidence showing that the Hall Technique, when appropriately used in indicated teeth (clear band of dentine between carious lesion and pulp), has success rates superior to comparator treatment.[109,110,117–119] Given the high success rate and patient acceptability of the Hall Technique, clinicians still reluctant to offer this treatment option, need to examine why they are treating a child more invasively when a less invasive option is available.[120]

Many of the core principles of MID elucidated by Walsh *et al.*[121] are also in keeping with the current aetiological and preventive/preservative paradigms of dental caries. These MID principles include: recognition (of disease contributory factors); re-orientation (of the contributory lifestyle factors); remineralisation (of both cavitated and non-cavitated lesions); repair (only when other solutions are not possible); and review (to ensure healthy oral and life environment is maintained).[120,121] The chance for MID to be successful is increased if dental caries is not considered as an infectious disease but rather a behavioural disease with a bacterial component.

By approaching the clinical situation of caries from a biological standpoint, the vicious cycle of treatment and retreatment of CARS can be terminated. This is done most effectively by evaluating and tackling the risk factors that fuel the disease process and adopting one of the several biological management options available. These contemporary caries management philosophies are consistent with the ICCMS[93] or STEM,[94] whose integral aims are to preserve tooth structure with non-operative biological care at the initial stages, and conservative tooth-preserving operative care at more extensive stages of the carious lesion.

Conclusions

About 700–800 bacterial species have been identified from the human oral microbiome making the mouth the most microbiologically diverse environment in the body. Both traditional as well as newly identified bacterial species have an important role in the caries process, and from an ecological point of view it may be more important to describe what the bacteria are doing in the biofilm community rather than which bacteria are present. Given the polymicrobial nature of dental caries, it is predicted that diagnostic, preventive, and treatment strategies directed toward specific bacterial species will not be universally effective.[20] Another important change in perspective is the realisation that there are beneficial members of the oral microbiome and an understanding of health and disease requires knowledge of all microorganisms, not just a select few pathogens.

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Dental caries affects all age groups, and can have particularly devastating, life-long consequences in young children, while in the elderly it can compromise an individual's ability to eat leading to malnutrition and expediting mortality. It is thus critical that dentists recognise the evolving aetiological paradigms of the dental caries process and its clinical implications. Oral healthcare professionals need to incorporate contemporary evidence-based guidelines into their routine practice for the benefit of their patients. Further education and training for dental practitioners, coupled with fairer remunerative schemes, would encourage a change in favour of the provision of more biological approaches towards prevention and treatment of dental caries.[122]

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