New paradigms for assessing caries risk and lesion activity

By Professor Laurence J. Walsh

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When assessing a periodontal patient, we take into account the level of attachment loss in relation to the patient's age and their oral hygiene to identify patients who are susceptible to destructive periodontitis. Likewise, we look at the individual site and assess the presence or absence of bleeding on probing in the absence of marginal inflammation to gauge the disease activity at that site. Today, we understand that some 20% of the population are highly susceptible to destructive periodontitis because of the combination of genetic factors (responsible for 50% of the risk) acting in concert with predisposing factors (which enhance the accumulation of dental plaque or hinder its removal) and modifying factors (which alter the response of the host).

These concepts have obvious parallels to management of dental caries, but it is all too easy for the similarities (both being plaque-related diseases) to be lost in translation. In terms of susceptibility, one could say that only some patients are inherently susceptible to destructive periodontitis despite poor oral hygiene, because of their genetically-determined favourable host immune response. In dental caries, there is a response to dental plaque bacteria but this is not strongly protective for the host and the extent of variation from one individual to the next is very subtle and of little clinical significance. Similarly, there are small individual differences in salivary mucins which show a modest correlation to caries risk, but overall the role of genetic factors is minimal in most patients. Those patients with missing major salivary glands or those suffering from auto-immune attacks on salivary glands show clear genetic patterns, but fortunately such conditions are not commonly encountered.

There is good evidence that the dental plaque biofilm in normal healthy adults harbours a broad range of potentially cariogenic bacteria. For example, it is normal for adults to have three or more strains of mutans streptococci (MS) in their dental plaque at low numbers despite having no active dental caries. This is the case because these are part of the normal biofilm. Transmission of these and other oral bacteria, from mother to child by salivary transfer, has been demonstrated in various studies, through behaviours such as kissing, sampling of food and sharing spoons and other utensils. By the age of 24 months, most infants harbour this organism as part of their normal oral flora.

Today, most approaches to caries microbiology align with the biofilm catastrophe concept of dental caries, as originally proposed by Phillip Marsh in 1995. The "ecological concept" of dental caries is based on the concept that a catastrophic change in the normal plaque biofilm is responsible for disease. This approach recognizes the possibility that bacteria yet to be cultured can participate in the caries process. It also aligns with a comprehensive approach to caries as a disease which results from the interplay of host, microbial, lifestyle and behavioural factors.

Perhaps most importantly, this concept supports "caries risk management throughout life", rather than focusing only on narrow "windows of infection" or assumed high risk periods. This recognizes the clinical reality that caries can develop in a tooth at any time after tooth eruption.

A risk assessment approach - understanding patient susceptibility

Recognizing that many organisms can contribute to acid production in addition to the MS, one can clearly conclude the risk of dental caries at any one point in time may be low, moderate or high - but it is never zero. For this reason, we need to consider management of risk as a better concept than simple prevention of disease. Any individual can develop caries if the environment changes - a fact demonstrated amply in those who develop lesions during orthodontics or in the later years of life but who believed incorrectly that they were "immune" to dental caries because of little or no disease being experienced at other times in their life.

For this reason, as clinicians we need to use a composite or multi-parameter approach to risk assessment for dental caries. Examples of some factors we would consider are listed in Table 1, however for ease of use, a number of clinical tools have been developed such as the Traffic Light Matrix (Figure 1) and the "wheel of misfortune" (Figure 2) so that the patient can be engaged in the process and their understanding of this multi-factorial lifestyle disease improved. The process is not sufficiently robust to give statistically reliable odds, despite many attempts to achieve this end. Said another way, we should never give a patient a false sense of security and have them believe that they NEVER WILL get caries in the future - when the reality is that they CAN. This emphasizes the rationale for ongoing attention to lifestyle factors, oral hygiene and fluoride exposure across the lifespan rather than only in the childhood years.

Behavioural and lifestyle factors change during the passage of time and many of these can contribute to heightened caries risk, including grazing eating patterns, with a higher frequency of intake of sucrose and other fermentable carbohydrates and frequent intake of acidic fruit juices, cordials and soft drinks. In addition to serving as a source of substrate, these exert an aciduric influence on the oral microflora. The use of a medicine which alters salivary parameters is a further common and important example.

MS and other bacteria implicated in caries are facultative microorganisms, which can tolerate oxygen levels in normal mouth air and in the general atmosphere, but prefer oxygen-poor

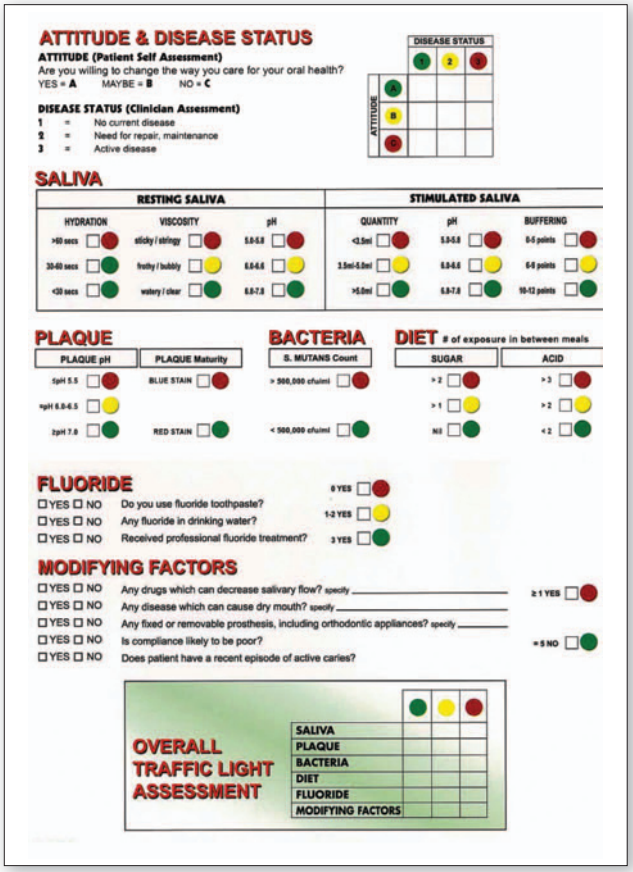

Figure 1. The Traffic Light Matrix combines the traffic light results from salivary test results with those from dental plaque biofilm assessments and qualitative determinations of patient motivation and compliance. From Ngo H and Gafney S (2005) in: Preservation and Restoration of Tooth Structure, 2nd edition.

growth conditions. This feature is essential not only to their survival deep within the plaque biofilm but is also critical in terms of favoured growth sites such as fissures and interproximal spaces. This facultative nature also explains in part the interaction between smoking and caries risk. Smoking creates anaerobiosis within the oral cavity and the low oxygen environment favours the growth of MS.

Table 1. Predictors of caries risk
Past caries – restored surfaces and tooth loss
Depressed resting salivary output and pH
Depressed stimulated salivary flow and buffer capacity
Total sucrose consumption
Frequency of fermentable carbohydrate
Frequency of acidic foods and drinks
Standard of oral hygiene
Levels of mutans streptococci in saliva
Levels of lactobacilli in saliva

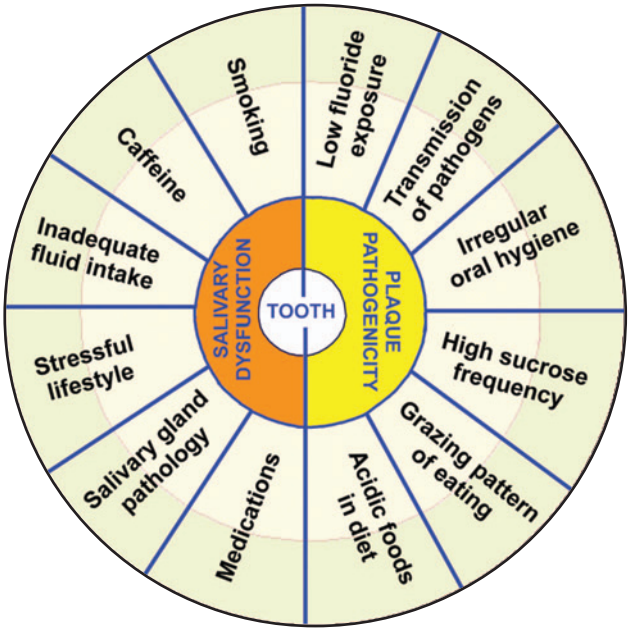

Figure 2. The "wheel of misfortune" which divides caries risk factors into those which primarily work via saliva (on the left) and those which alter the biofilm (on the right). From Walsh LJ (2005) in: Preservation and Restoration of Tooth Structure, 2nd edition.

Table 2. Characteristics of active carious lesions (ICDAS 2)

Overlying plaque

- Thick tenacious dental plaque biofilm
- Spontaneous acid production from stored substrate
- Rapid acid production after sucrose/glucose challenge

Enamel lesions

- Topographical association with dental plaque and stagnation
- Located in protected areas with little saliva contact
- Increased opacity
- Loss of surface reflection
- Appearance more obvious with drying
- Rough surface when the tip of a blunt probe is slid gently across the surface

Root surface lesions

- Topographical association with dental plaque and stagnation
- Location in risk regions - e.g. buccal of mandibular posterior teeth
- Located in protected areas away from stimulated saliva
- Softening of the surface - a leathery characteristic on probing
- Light colouration of the root surface

There are two additional components involved in the impact of smoking on dental caries. The first is salivary dysfunction, which is the result of the pharmacological effects of nicotine on salivary gland flow. Reduced flow is linked with reduced pH, which is also a favourable parameter for growth. The second factor is the direct effect of nicotine in the salivary milieu on MS. There is emerging evidence that concentrations of nicotine of some 1.0 mmol/L stimulate the growth of MS - a level similar to that achieved in saliva with many tobacco products.

The tendency for MS to occupy an anaerobic ecological niche has an interesting consequence in terms of periodontal therapy. It is well known that both attachment loss per se and root surface exposure from surgical and non-surgical periodontal treatment are associated positively with root surface caries. The susceptibility for root surfaces for caries after periodontal therapy has been attributed to a loss of the fluoride-rich outer layers of cementum and dentine, however the microbial effects of periodontal therapy should also be considered. Through soft tissue changes such as recession, the available supragingival area for dental plaque biofilm is increased. There is clear evidence that following periodontal therapy, the severity of root surface caries is associated strongly with high salivary counts of MS. This effect is compounded in very elderly patients, in whom polypharmacy causes reductions in the resting salivary flow rate and salivary pH and corresponding increases in both sugar clearance time and levels of MS.

It follows that a key preventive strategy is reducing the frequency of cariogens in the dental plaque biofilm. This could be achieved by a number of methods used singly or in combination, such as restriction of fermentable substrates; inhibiting acid production, e.g. by using high concentrations of fluoride; using non-fermentable sugar substitutes, e.g. xylitol; using alkalinizing mouthrinses; reducing acidic drinks in the diet; and promoting alkali generation, e.g. from arginine or urea supplements.

Disease activity at the site

As well as considering the patient as a whole and their likelihood of experiencing dental caries in the future, one inevitably is drawn to look at what is easier to see and understand - the tooth surface and the lesions which may be present there. Once again, a composite of clinical indicators is used to assess lesions, whether they are located on enamel or root surfaces (Table 2). Because the development of surface cavitation is a late stage in the caries process, there are opportunities to intervene in the process to arrest and reverse the lesion, rather than committing to

Table 3. Steps in using plaque biofilm diagnostics

1. Explain to the patient what the test can show and how it is done
2. Conduct the test
3. Show the patient the results
4. Explain what the results mean
5. Record the results
6. Give practical advice and lifestyle counselling
7. Follow-up with a repeat of the test at the next visit
8. Recognize and reward compliance

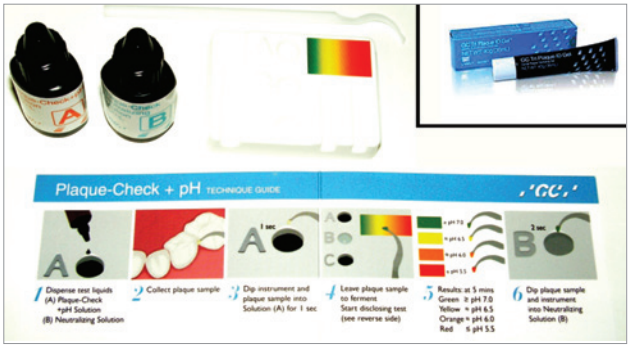

Figure 3. Two generations of tests for dental plaque fermentation - the GC PlaqueCheck+pH chairside diagnostic kit uses sucrose as a challenge to plaque taken from specific sites in the mouth. The GC TriPlaque ID kit (inset) has the challenge and dye technology in the one liquid which is used in the same fashion as a disclosing dye.

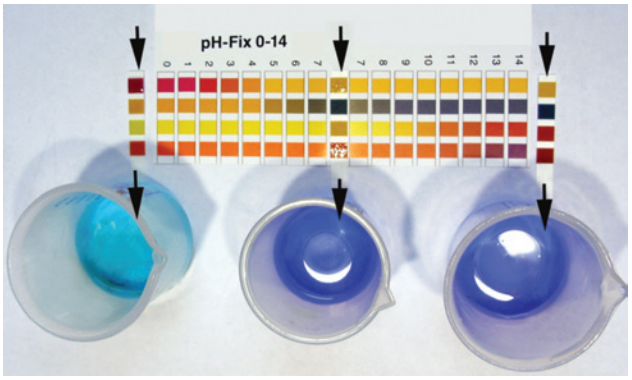

Figure 5. The pH selective response of the dye combination. The three beakers show the dye mixture at acidic, neutral and alkaline pH. The arrows show the pH indicator strips taken from the three beakers and compares them to the manufacturer's calibration chart. An aqua green colour develops at acidic pH values, which provides the third colour of the test.

restorative procedures at the outset. The number of pre-cavitation early carious lesions typically exceeds the number of clinically detectable cavitated lesions by a considerable margin. This is true both for enamel and root surfaces, although the latter are even more difficult to detect unless radiographs are also used. One needs to have a high index of suspicion when discovering a frank cavitation, as it often represents the "tip of the iceberg" in terms of other sites with disease present.

Lesions which show 'distinct visual change' are termed ICDAS code 2. At this stage, the lesion is non-cavitated and can be seen when the tooth surface is wet with saliva. Ideally, one would want to identify and recognize lesions at an even earlier timepoint, ICDAS code 1. At this earliest stage of caries, the tooth surface shows its first visual change. In enamel, a code 1 lesion cannot be seen when the tooth surface is wet with saliva, but is evident after air drying for 5 seconds. Such lesions can be visualized using blue-green light induced fluorescence, for example using a blue LED curing light and orange filter shows these as dark areas within a fluorescent background of yellow tooth structure.

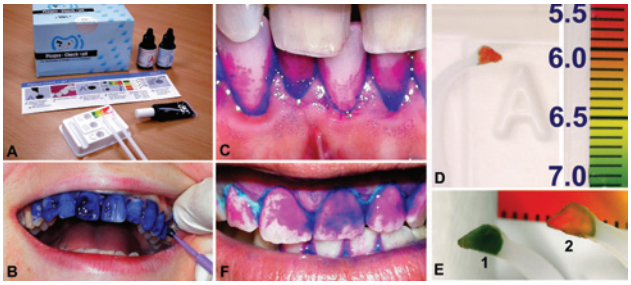

Figure 4. A - GC PlaqueCheck+pH chairside diagnostic kit for assessing plaque biofilm; B - Applying a 2-tone plaque disclosing gel with a micro-brush; C - Disclosing of the lower incisor teeth shows both thin (pink) plaque deposits as well as areas of mature (blue) plaque at the cervical aspects and interdentally; D - The plaque fermentation test result after 5 minutes can be compared to a pH scale, which corresponds to a "traffic light" sequence in terms of caries risk; E - Two plaque samples from the same site, immediately after exposing the plaque sample to the test reagent (1), and after 5 minutes (2). The latter shows clearly the colour change which has occurred, from green to red; and F - Disclosing using the new GC TriPlaque ID solution. Areas of acid production are seen as bright green-blue areas cervically and interdentally, which are superimposed on the same pattern of pink-blue staining. Compare the staining pattern to panel C, immediately above.

Thinking of caries as a disease process rather seeing a lesion as outcome, it is clear that demonstration of the disease-causing potential of the dental plaque biofilm at a particular site would be powerful both for diagnosis and patient education. Applying this "ecological catastrophe" concept to the issue of pH within the dental plaque biofilm, the low pH environment generated from carbohydrate metabolism is the major factor responsible for the shifts observed in the oral microflora with high carbohydrate diets.

There is now available technology to achieve this diagnostic objective, literally a visible Stephan curve (Figure 3). The first generation test for plaque acid production in response to sucrose challenge was the GC Plaque-Check+pH™ fermentation test, invented by the author. This test is based on the causal association between caries activity and the production of strong acids from plaque in response to sucrose, which was first identified by the work of Stephan more than 65 years ago. The Plaque-Check+pH test (Figure 4) recognizes that many dental plaque bacteria can ferment carbohydrate substrates and this leads to the production of numerous organic acids (of varying potency for demineralization). Thus, the test assesses the intact plaque biomass at a given site and documents the net result of fermentation, in terms of plaque pH, using a colourimetric test to show the pH-drop which occurs in intact plaque samples when exposed to an excess supply of sucrose. As fermentation occurs, the greatest reduction in pH occurs after 5 minutes and this can be seen as a colour change in a pH indicator. This test also includes a separate reagent with two dyes, using erythrosin and fast green to stain "young" plaque red and "old" plaque blue, respectively.

In March 2011, the second generation of this technology was released commercially. The GC Tri-Plaque ID test takes the same

Figure 6. Two clinical examples of real-time acid production in dental plaque. Panels A and B, before and after staining, showing thin pink and thick dark blue plaque deposits with an intense local area of acid production (light blue) on the gingival margin of tooth 43. Case courtesy of Dr Andrew Brostek. Panels C and D, before and after staining, showing thick dark blue plaque deposits with multiple areas of acid production (aqua green). Case courtesy of Ms Carol Tran.

concept of acid production but incorporates the assessment of plaque ecology at multiple sites simultaneously rather than as separate steps. This technology, also invented by the author, relies on the pH selective response of different dyes (Rose Bengal and Brilliant Blue FCF) (Figure 5) included in a glucose-containing disclosing liquid, to show intra-orally both plaque age and plaque acid production following a substrate challenge, within 2 minutes. This latest test answers at the same time the two key questions: Is there thin or thick mature plaque? Is the plaque dangerous in terms of acid production - are there white spots already forming on the enamel beneath these plaque deposits? (Figure 6).

The use of simple chairside tools such as 2-tone plaque disclosing and plaque fermentation tests, and 3-tone disclosing helps the clinician to assess caries activity at a specific site as well as caries risk for the patient (the latter when the test results are used in combination with other measures such as assessments of lifestyle and salivary factors). The visible results also help to educate patients regarding the production of acid as a major event in the caries process and springboard a discussion of whether their plaque biofilm is “young/good” or “old/bad”. Repeating these tests allows compliance with oral hygiene and lifestyle changes designed to reduce the cariogenic potential of their plaque to be assessed by the dental professional (Table 3).

About the author

Professor Laurence J. Walsh is the technology editor of Australasian Dental Practice magazine. He is also a noted commentator on and user of new technologies and is the Head of The University of Queensland School of Dentistry.