Contending with CARIES

A clinical perspective

Graeme Milicich, BDS

Graeme Milicich, BDS, graduated from the University of Otago, New Zealand, in 1976 and has since been in private group practice. His fields of interest include cosmetic dentistry, occlusal rehabilitation, implant prosthetics, minimal intervention, and lasers. He is a Fellow, Diplomate, and founding board member of the World Congress of Minimally Invasive Dentistry. He also is a founding board member of the NZ Institute of Minimal Intervention Dentistry. Dr. Millicich has several peer-reviewed publications in the fields of Minimal Intervention Dentistry and on the glass-ionomer cement Co-cure Technique. E-mail him at gwmilicich@xtra.co.nz, or visit his Web site, *www.advancedental-ltd.com*.

Caries and periodontal disease are the two primary diseases facing our profession. Looking back at my 30 years in practice, periodontitis has been much easier to manage and treat than caries. Additionally, a significantly larger proportion of my patients suffer from the ravages of caries than periodontitis. So caries, by far, has been the greater challenge.

It's the bacteria

Both periodontitis and caries basically are caused by an imbalance in the bacterial populations of what are natural and normally healthy biofilms.[1] The complexities of the disease we know as caries are the multiple factors[2] that are associated with the evolution of a healthy bacterial biofilm population to one that is pathological.

Caries is an infectious and transmissible disease, and the primary infection often can come from family members or caregivers.[2,3] Even once all these factors are understood, it is still a significant challenge for many patients to be able to modify their risk factors to create an oral environment that will lead to a re-establishment of a healthy bacterial population within the oral biofilm.[4] The understanding of the behavior and complexities of biofilms (**Fig. A**) helps explain the difficulties we are often faced with when treating caries at the clinical level.[1]

part 1:

Diagnostic and treatment philosophies are shifting to a medical model, based on evidence that caries is a disease.

By Graeme Milicich, BDS

More than drill and fill

The surgical excision of demineralized and infected tooth structure does nothing to change the primary caries infection. The pathological biofilm is still present, and unless it is addressed, the patient is going to return in a year or two with further cavities. Treating caries with a focus on risk assessment and management has been shown to be more effective compared to simple restoration of cavities.[4,5]

A healthy biofilm can be made up of more than 700 bacterial species, and there can be less than 1% of potentially pathogenic bacteria within the biofilm, which acts as a protective mechanism to help protect the mouth from infection by pathogenic bacteria. Biofilms by their nature are very resistant to change, and when they do change, it usually takes time for the evolution of bacterial species to occur. A change can be caused by modifying pressures from constant overload from pathogenic organisms, external risk factors and risk behaviors. These can all lead to environmental changes within the biofilm that favor the proliferation of aciduric and acidogenic pathogenic species like *mutans streptococci* and *lactobacilli*[6] that help them to take over the biofilm.[7,8] A cariogenic biofilm can then be made up of more than 95% pathological bacteria, compared to less than 1% in a healthy biofilm. When all the factors that may contribute to a biofilm evolution are examined, it appears the primary driver is an acidic pH shift that either can be extrinsic or intrinsic to the dental biofilm, or both.[9-11]

Depending on the patient's contributing risk factors, shifting a biofilm population from pathological back to healthy can take considerable time and effort. Brushing and flossing breaks up the biofilm, which is an essential factor in caries control. However, this does nothing to change the bacterial species that are present, as the biofilm re-establishes itself over the next 12 to 24 hours. As an analogy I use with my patients, simply mowing a weed-filled lawn does nothing to change the proportion of weeds in the lawn; they are just a bit shorter. Equally, spraying the weeds (using a simple antibacterial mouthrinse) with a weed killer does not prevent new weeds from growing straight back again. We have to do more, like fertilizing the lawn, to help promote the growth of healthy grasses. When treating caries, this analogy means using mechanical debridement, anti-

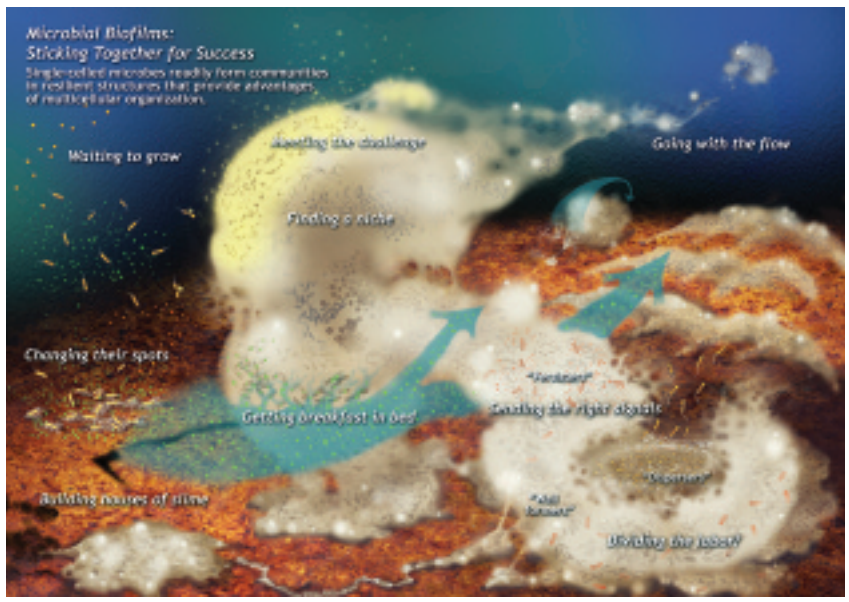

Fig. A Image from the Centre for Biofilm Research, Montana University. This is an excellent source of educational information on the complexities of biofilms and how they behave. For more information on Microbial biofilms and the image shown here, see the Web site: *www.erc.montana.edu/MultiCellStrat/default.html*

Photo: P. Dirck, MSU Center for Biofilm Engineering

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bacterial rinses (preferably ones that help promote the growth of healthy bacteria), management of foods that promote the production of acid from aciduric bacteria, and the use of rinses that help challenge the acidic environment of a cariogenic biofilm.

The early bird…

The ideal in helping our patients prevent damage to their teeth from a caries infection would be to diagnose the presence of a pathological biofilm *before* it has done damage to the teeth. Our current diagnostic model relies primarily on the detection of the signs and symptoms of a caries infection. The first observable sign is a white spot lesion in the enamel, probable damage in a fissure, or early radiographic evidence of demineralization. This is the equivalent of waiting for angina to develop and then telling the patient that they have cardiovascular disease, rather than assessing patients for risk factors associated with the development of cardiovascular disease.

The ideal would be to screen patients to test their biofilm for the presence of an imbalance in the bacterial flora. This would then give us a chance to help the patient address the issues that are leading to this bacterial population shift, before damage has even occurred. In reality, this is no different from many screening procedures we expect from the healthcare sector to help us identify our risk for heart disease, some cancers, diabetes, etc. However, this approach requires a philosophical change in how a practice is managed.

Fig. B CRT test for *Mutans Streptococci* (MS) and *Lactobacilli* (LB) from a very high-risk patient.

What is the reality of instigating a medical approach to diagnosing and treating a biofilm disease, rather than waiting for damage to occur to the teeth?

Test and educate

First, we need a quick and effective way of clinically testing the dental biofilm for potential pathogenicity. Second, we need to effectively educate the patient on the potential consequences of a positive result. Finally, we have to offer patients an effective treatment and management program that they can take home with them.

A common caries management pathway taken at the moment is to detect the symptoms of the disease (cavities), and then simply restore them.[12] However, a patient with high risk factors but no current clinical expression of these factors, which may also include a cariogenic biofilm, is simply a patient with a disease that is yet to express its symptoms. Patients much prefer the concept of treating the infection before it has led to the need for a tooth to be drilled. However, this is a complete reversal of the systems that are commonly in place in a practice. To successfully make a change requires planning along with education of the staff.

Speaking as one who recently has been down this path, I have found that the easiest way to change is to start with the end point in mind and work back through the plan to work out how to integrate this into the patient treatment flow. Not only

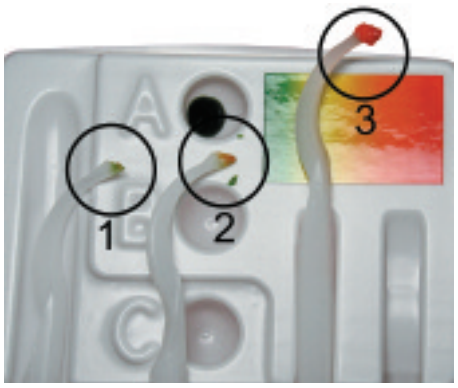

Fig. C Three plaque pH tests from very low-risk (**1**) low-risk (**2**) and very high-risk (**3**) patients. As acid production from the plaque bacteria increases, pH drops, causing the litmus liquid to change color. The very deep red from patient 3 indicates a plaque pH < 5.5. Patient No. 2 has a plaque pH of 6.5, and patient No. 1, a plaque pH of 7.

does the dentist need to understand the concepts, but so does the staff, and it is important that the staff have a good knowledge base, because the dentist will not have the time to educate all the patients. However, it takes significant time spent on education and systems development to be able to make a successful change in a practice.

One of the biggest time-consumers can be educating the patients. To this end, it is essential that the staff members are well trained, as they become an additional source of information transfer. Another very effective way of educating patients can be via a practice newsletter that is sent out as the next examination recall.

This can be used to explain a change in the practice's philosophy, and to let patients know what to expect that will be different on their next visit. Experience has shown that this is a very effective way to get detailed information across, because most patients do read their dentist's newsletters. The more information given to patients prior to their visits, the less chair-time will be needed to explain caries risk assessment and its benefits to them.

This article is in no way meant to be an "advertorial," but from personal experience, I have found that changing systems

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Fig. D The CariScreen test requires a swab (**1**) to be taken of the buccal surfaces of tooth Nos. 11 and 16. The swab then is placed into the tube, and the required chemicals are released by snapping the vial on the end of the swab. After agitating, the tube is placed into the light meter (**2**). The relative light units are displayed after 15 seconds (**3**). RLUs below 1500 are low caries risk; RLUs between 1500 and 2500 are moderate risk; and RLUs greater than 2500 are patients with a high caries risk.

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or introducing new concepts into a busy practice can be very difficult. Oregon-based Oral BioTech has developed the CariFree caries screening and treatment system that is easily implemented; all the normal "sticking points" in making big changes have been recognized and systematized to help the practice integrate an effective caries management program into its busy schedule.

It was the simple "plug and play, caries management in a box" concept that so attracted me to the concept. I had been trying to develop an effective caries management program, but was not having great success in integrating the concepts into my daily routines. There are three aspects to the Oral BioTech CariFree system: dentist and staff education, a simple biofilm screening test, and a basic biofilm treatment program that can be modified with additional products as required, to target certain risk factors in high- and extreme-risk patients.

Fig. E A 24-hour culture of *Mutans streptococci* (MS) from plaque gathered from the dental biofilm. This culture was done because the original CariScreen ATP test indicated the presence of a high-risk biofilm. This result is typical for a high-risk patient. Unlike the CRT test, the CariFree culture tube does not have to be opened to read the results, meaning staff members are not exposed to the highly unpleasant odors associated with plaque cultures.

Identifying pathological biofilm

Risk assessment requires standardized risk assessment forms; educational material to inform patients on risk factors and protective factors and how a disturbance of the balance can lead to development of a cariogenic biofilm; and finally, a simple screening test. There currently are three products available for assessing the cariogenicity of a biofilm.

Ivoclar Vivadent's CRT bacterial culture kit

This is a 48-hour bacterial culture kit to measure the colony forming units of planktonic (free-floating) *Mutans Streptococci* (MS) and *Lactobacilli* (LB) in a patient's saliva. This requires the patient to chew on wax for five minutes, then spit into a cup to collect the saliva. The sample is flowed over the double-sided agar plate, which then is incubated for 48 hours (**Fig. B**).

GC America's Plaque-Check+pH test kit

This is a relatively simple five-minute chairside test kit that measures the change in plaque pH when it is exposed to sugar. The change in plaque pH after five minutes gives an indication of the potential cariogenicity of the plaque bacteria (**Fig. C**). This test gives a more accurate indication of biofilm cariogenicity because it allows different areas of biofilm to be tested; whereas the CRT test measures salivary levels of planktonic MS and LB bacteria shed from the overall oral biofilm.

Oral Biotech's CariScreen test

This is a simple screening test of the dental biofilm that takes less than a minute. It utilizes a completely different concept to measure the potential pathogenicity of dental plaque. Acidophilic and aciduric bacteria are able to survive in a low pH environment because of their ability to maintain a neutral intracellular pH via an efficient cell wall hydrogen ion pump that removes hydrogen ions as they diffuse from the extra-cellular, high pH environment, back through the cell wall. This protective mechanism requires significant amounts of energy that is derived from mitochondrial ATP. The CariScreen test measures dental biofilm ATP levels by mixing the bacterial ATP with luciferin, which then produces a quantifiable level of light. The light output (Relative Light Units) has been calibrated to known pathogenic bacterial standards. The object of the test is to be able to screen a patient's plaque in real-time (**Fig. D**). If a positive result is obtained, the screening test is then confirmed using a 24-hour bacterial culture for *Mutans Streptococci* (**Fig. E**). CariScreen has a sensitivity and specificity in excess of 90%.

Now that you know…

However, simply diagnosing a cariogenic biofilm is of little significance if a practical solution cannot be offered to the patient. Next month, Part 2 of this article will cover treatment options for at-risk patients. DPR

Disclosure

I was so impressed with the ease that I was able to introduce caries risk assessment and management into my practice using the Oral BioTech CariFree system that I purchased shares in the company. –G.M.

References

1. Teles RP, Haffajee AD, and Socransky SS, Microbiological goals of periodontal therapy. Periodontol 2000, 2006;42:180-218.
2. Florio FM, et al. Time of initial acquisition of mutans streptococci by human infants. J Clin Pediatr Dent 2004;28(4):303-8.
3. Berkowitz RJ. Acquisition and transmission of mutans streptococci. J Calif Dent Assoc 2003;31(2): 135-8.
4. Featherstone JD, et al. Caries management by risk assessment: consensus statement, April 2002. J Calif Dent Assoc 2003; 31(3):257-69.
5. Featherstone, JD. The caries balance: contributing factors and early detection. J Calif Dent Assoc, 2003;31(2):129-33.
6. Harper DS, Loesche WJ. Growth and acid tolerance of human dental plaque bacteria. Arch Oral Biol 1984;29(10):843-8.
7. Busscher HJ, Evans LV. Oral biofilms and plaque control. Amsterdam: Gordon and Breach/Harwood Academic Publishing, 1998.
8. Marsh PD. Host defenses and microbial homeostasis: role of microbial interactions. J Dent Res 1989;68:1567-75.
9. Bradshaw DJ, McKee AS, Marsh PD. Effects of carbohydrate pulses and pH on population shifts within oral microbial communities in vitro. J Dent Res 1989;68:1298-1302.
10. Tenuta L, et al. Effect of sucrose on the selection of Mutans streptococci and Lactobacilli in dental biofilm formed in situ. Caries Research 2006;40(6):546-9.
11. Marsh P. Dental plaque as a biofilm and a microbial community - implications for health and disease. BMC Oral Health 2006. 6(Suppl 1):S14.
12. Fejerskov O, Kidd E. Dental Caries: The disease and its clinical management. Oxford, UK: Blackwell Munksgaard, 2003.

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A clinical perspective

part 2:

Diagnostic and treatment philosophies are shifting to a medical model, based on evidence that caries is a disease.

By Graeme Milicich, BDS

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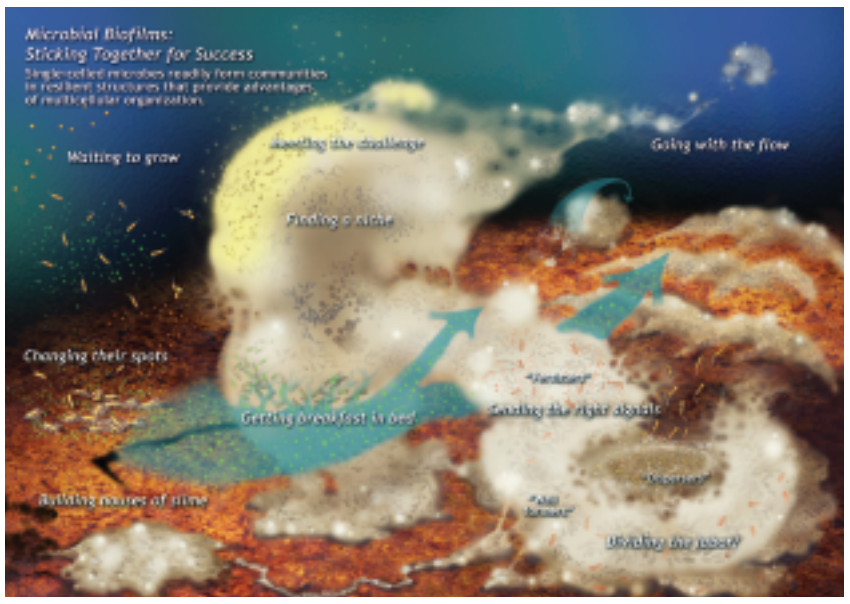

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A detailed discussion of cariogenic biofilm and its diagnosis were covered last month in Part 1 of this article (page 100). The "complexities" of biofilm are outlined in Fig. A (right). However, simply diagnosing a cariogenic biofilm is of little significance if a practical solution is not available to the patient.

The treatment of a cariogenic biofilm can be very complex due to the multi-factorial aspects of the disease. The protocols presented to patients, based on their diagnosed needs, must be simple and practical—otherwise very few patients will persevere to the point that they have success.

Managing biofilm: disruption

The first concept in managing a biofilm disease is physical disruption of the biofilm mass. If this is not done, antibacterial rinses will have little or no effect on the biofilm, which develops in such a way that it can resist serious attack from antibacterial agents. Ideally, the rinse should also be able to attack the physical structure of a dental biofilm, which is made up of approximately 85% extracellular mucopolysaccharides, to help expose the bacteria to the antibacterial agent.

There are several effective broad-spectrum antibacterial agents—isopropyl alcohol, gluteraldehyde, sodium hypochlorite, ozone, and chlorine dioxide, to name a few. However, alcohol, gluteraldehyde, and ozone cannot be used safely as a total mouthrinse. Sodium hypochlorite is very effective in its effects on a biofilm because it challenges the bacteria as well as the physical mucopolysaccharide structure of the biofilm. A further desirable attribute of a mouthrinse would be to have a pH greater than 7.[1,2]

We discourage patients from consuming low pH drinks and foods that help create a low pH oral environment and, in turn, aid in the development of a cariogenic biofilm. Ironically, however, we can get patients to use oral rinses that have a significantly low pH. Some rinses are as low as pH 4, and very few are above pH 7. High-risk patients should rinse regularly with water containing baking soda to help raise the intraoral pH, so it makes sense that an antibacterial rinse would also have this ability.

As discussed in Part 1 of this article last month, the sodium hypochlorite used in the treatment phase of Oral Biotech's CariFree system (*www.carifree.com*) is not only strongly antibacterial and broad spectrum, but it also has a pH of 10.3.

Trading bacteria

When we accept that we all must have a biofilm in our mouths, the concept of trying to permanently kill off the bacteria makes no sense. We have to work with Mother Nature, rather than against her, in an unwinnable fight. One conceivable approach would be to seriously challenge the bacteria in a pathological biofilm for a short period, and then create an environment that would be conducive to the re-establishment of a biofilm containing more non-pathogenic bacteria. This is done using several strategies.

Modify the risk

The first is the modification of patient risk factors and risk behaviors, including reduction in sugar and acid exposure to decrease the frequency of acid attacks on the enamel.[3-5] Without risk modification, nothing else will succeed, so it is essential patients are well educated about it.

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Photo: P. Dirck, MSU Center for Biofilm Engineering

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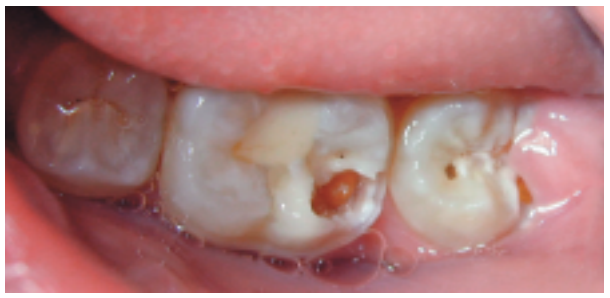
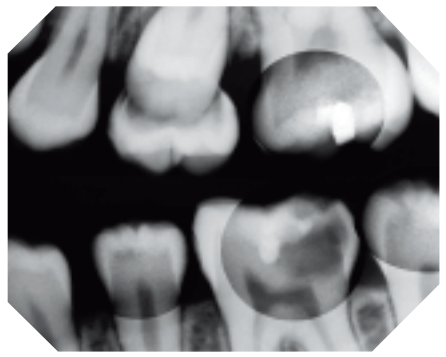
Raise the pH

Following a strong antibacterial challenge for several days, the next step would be to create an oral environment with a pH above 7 that also is conducive to the proliferation of non-pathogenic bacteria. The use of Xylitol,[6-8] fluoride,[9-11] and naturally occurring antibacterial agents like polyphenols[12-13] and anthocyanidins[14-15] in a rinse with a pH 8 is formulated to do just this. In the case of high-risk patients, particularly those who exhibit low resting salivary pH, a mouth spray containing fluoride, and Xylitol with a pH 9 associated with CaOH, can be used on a regular basis throughout the day. The goal is to make it as easy as possible for patients to comply with our recommendation. I have yet to find many patients who find it convenient to carry around a liter of water mixed with baking soda, so they can sip on it in a regular basis. In high-risk patients, the addition of high-fluoride toothpaste and CPP-ACP paste can further enhance the pressure on a pathological biofilm.





Figs. B and C Clinical presentation of 14-year-old high-risk patient. The occlusal surface of tooth No. 18 was even cavitated under the operculum.

The use of fluoride and chlorhexidine in a caries control regime is difficult because patients have to use the products at different times due to the problems associated with combining cationic and anionic agents at the same time. As soon as a management regime becomes complicated, patient compliance diminishes. The CariFree system does not have the problems associated with the combination of various products and is essentially compatible with any other ancillary products that may be required for high- and extreme-risk patients. These may include the use of chewing gums containing Xylitol and CCP-ACP, fluoride varnish, MI Paste (GC America, *www.gcamerica.com*), and high-fluoride dentifrice.

Case study

When I first gained access to the CariFree treatment and maintenance rinses in 2004, I used them in conjunction with the Vivadent CRT test to assess their efficacy in helping modify a cariogenic biofilm.

A 14-year old female presented with 14 cavities in her posterior dentition; some were near exposures (Figs. B and C, above).

A base line CFU for MS and LB was established using the CRT test (Fig. D, facing page).

This patient was then placed on the CariFree treatment rinse twice a day for 2 weeks, followed by the maintenance rinse twice a day for 3 weeks. This cycle was then repeated.

Her risk factors were identified via a standardized questionnaire, and she was then educated on what she needed to do

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to minimize her risk. Her risk factors were relatively simple—primarily poor oral hygiene and excessive exposure to sugar between meals via drinks and sweets. She was taught how to clean and floss well.

Following three months on the rinse cycle and completion of three of the quadrants of dentistry, the CRT test was still "moderate" in terms of the CFU score (Fig. E, below). This possibly was due to continual recontamination of the mouth from the cavities in the unrestored quadrant. Following completion of the restorations, the patient was placed on a final cycle of the CariFree treatment and maintenance rinses and the CFUs were then reassessed (Fig. F, below).

This low risk result with a CFU score of less than 10^5 was very encouraging, indicating she was successfully addressing her risk factors and oral hygiene. In conjunction with the CariFree rinses, her biofilm had recovered to a healthy state. She continues to maintain this state.

Using this system, I have had encouraging success in helping many of my high-risk patients, who in the past have not been able to control their infection based on the use of chlorhexidine, fluoride, diet control and good oral hygiene.

Conclusion

A semantics tangle in dentistry has made the discussion of caries very difficult. We use the term "caries" to synonymously describe a biofilm disease and cavities in teeth.

Caries the biofilm disease cannot be treated surgically, which is what the primary focus has been in the past. As a profession, we need to make a conscious effort to address the disease as well as the symptoms. We are experts at treating caries symptoms and their ongoing consequences, and now we need to become as effective and efficient at managing the actual disease.

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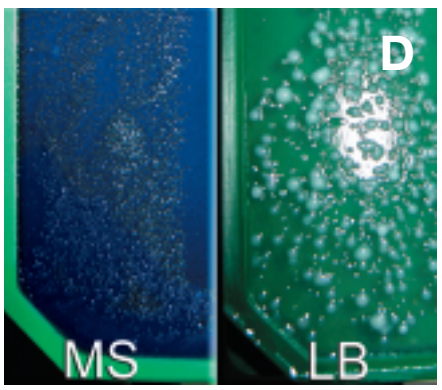

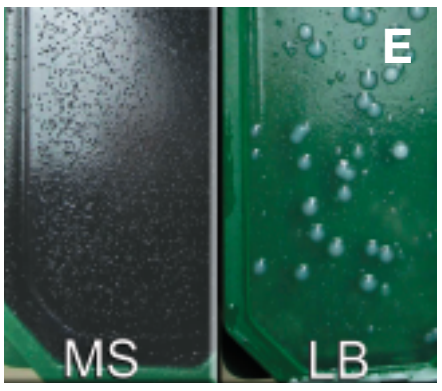

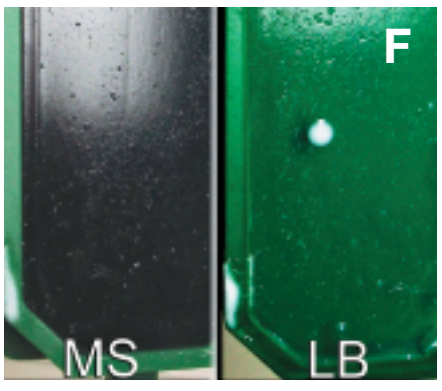

Fig. D Pre-treatment CRT culture indicating very high risk. **Fig. E** CRT test 3 months after the commencement of treatment; some reduction in CFUs. **Fig. F** Post-treatment CRT results with the CFUs indicating the patient is now at low risk.

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The challenge for practitioners today is that there remains no known, documented universal formula for treating dental caries. The simple one-size-fits-all therapy may work well with a single-pathogen disease model, but may have only limited effectiveness with a multifactorial/multipathogenic biofilm-based disease model. As our understanding of the complexities of the disease process improves, new techniques and materials are becoming available to aid in improving our ability to help our patients manage their disease, focusing on treatment strategies targeted to specific risk factors uniquely designed for each individual patient.

As caregivers, we all respond to change, and are motivated most when it is in the best interest of the people we serve. Once effective caries management is in place, both the dentist and the patient feel more comfortable with the prospect of accepting advanced restorative procedures, because there is a confidence that recurrent cavitation associated with an untreated pathological biofilm will not compromise the longevity of the restorative work.

The hardest part has been making the change from a surgical model to a medical model of caries management and treatment. DPR

Disclosure

I was so impressed with the ease that I was able to introduce caries risk assessment and management into my practice using the Biotech Carifree system that I have purchased shares in the company.

References

1. Blake-Haskins JC, et al. The effect of bicarbonate/fluoride dentrifices on human plaque pH. J Clin Dent 1997;8(6):173-7.
2. Bradshaw JD, Marsh PD. Analysis of pH driven disruption of oral microbial communities in vitro. Caries Res 1998;32:456-62.
3. Dong YM, et al. Plaque pH and associated parameters in relation to caries. Caries Res 1999;33(6):428-36.
4. Carlsson J. Microbial aspects of frequent intake of products with high sugar concentrations. Scand J Dent Res 1989;97(2):110-4.
5. Cury JA, et al. Biochemical composition and cariogenicity of dental plaque formed in the presence of sucrose or glucose and fructose. Caries Res 2000;34(6):491-7.
6. Kakuta H, et al. Xylitol inhibition of acid production and growth of mutans streptococci *in* the presence of various dietary sugars under strictly anaerobic conditions. Caries Res 2003;37(6):404-9.
7. Trahan L. Xylitol: A review of its action on mutans streptococci and dental plaque—its clinical significance. Int Dent J 1995;45(1 Suppl 1):77-92.
8. Maehara H, et al. Synergystic inhibition by combination of fluoride and Xylitol on glycolysis by mutans streptococci and its biochemical mechanism. Caries Res 2005;6(39):521-8.
9. Wahab FK, Shellis RP, Elderton RJ. Effects of low fluoride concentrations on formation of caries-like lesions in human enamel in a sequential-transfer bacterial system. Arch Oral Biol 1993;38(11):985-95.
10. Bowden GH. Effects of fluoride on the microbial ecology of dental plaque. J Dent Res 1990;69 Spec No:653-9;discussion 682-3.
11. Bradshaw DJ, McKee AS, Marsh PD. Prevention of population shifts in oral microbial communities in vitro by low fluoride concentrations. J Dent Res 1990;69(2):436-41.
12. Li JY, et al. Effect of tea polyphenol on demineralization and remineralization of enamel in vitro. Sichuan Da Xue Xue Bao Yi Xue Ban 2004;33(3):364-6.
13. Liu T; Chi Y. Experimental study on polyphenol antiplaque effect in humans. Zhonhua Kou Qiang Yi Xue Za Zhi 2000;35(5):383-4.
14. Steinberg D, et al. Effect of a high-molecular-weight component of cranberry on constituents of dental biofilm. J Antimicrobial Chemother 2004;54(1):86-9.
15. Weiss EI, Kozlovsky A, et al. A high molecular mass cranberry constituent reduces mutans streptococci level in saliva and inhibits in vitro adhesion to hydroxyapatite. FEMS Microbiol Lett 2004;232(1):89-92.