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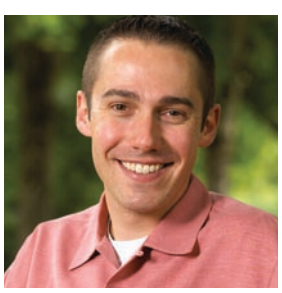

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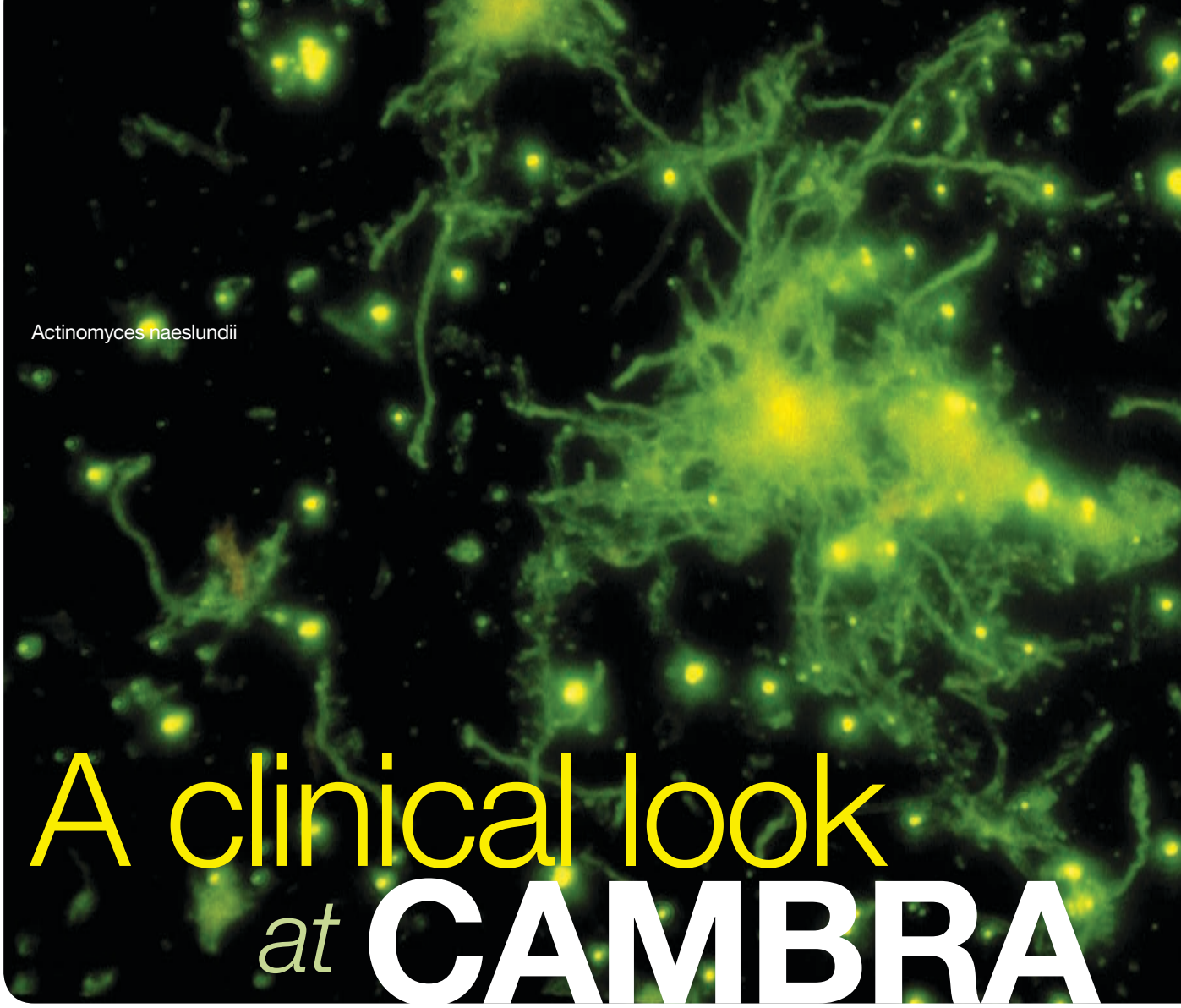


Actinomyces naeslundii

A clinical look at CAMBRA

Management and treatment of the biofilm aspect of caries disease.

By Drs V. Kim Kutsch, Carson L. Kutsch, and B.C. Nelson

Although much has been written about dental caries as an infectious and transmissible bacterial disease,[1] coupled with growing scientific evidence that supports treating dental caries with a medical model,[2] the majority of dentists still treat this disease primarily by using surgical methods. Drilling and filling does restore the teeth to function and reduce pain, but it does nothing to eliminate the disease cause.[3] There seems to be a missing link between the scientific information and the application of this information to normal clinical practice. Even dentists performing routine caries risk-assessment-based diagnosis struggle with how to successfully treat the biofilm component of the disease. Furthermore, dentists and patients alike seem confused on reasonable treatment outcomes and expectations for success. Treating the biofilm aspect of caries disease presents a serious clinical challenge, and there simply are no easy answers or magic bullets.

A broader landscape

Early dental caries research tried to fit the disease into the period's existing, traditionally understood disease model. Then, as microbiology emerged, the profession focused on identifying specific pathogen(s) as the cause of dental caries. From the time they were initially isolated and identified, *Mutans streptococci* (MS) and *Lactobacillus* (Lb) have been thoroughly researched as the probable etiologic agents for caries.[4]

However, more sophisticated biofilm research in conjunction with the development of modern disease models suggests that the MS/Lb model is simplistic and incorrect. Recent research has seriously challenged previous theories and has begun painting a broader landscape for this disease.[5-8] Dental caries is found in the absence of MS, and patients with high MS levels do not always have dental caries. The same is true with Lb. Although these bacteria offer good markers for the disease, there is only an associative—not cause-and-effect—relationship.[9] Independent research from multiple sites, using a reverse checkerboard analysis of DNA sequencing combined with population studies, have accumulatively identified up to 23 different bacterial species now significantly implicated in the caries process (See Table, "Bacteria implicated in dental caries," page 63). While MS is present as a pathogen, numerous other streptococci also are acidogenic/aciduric and often outnumber MS in the studies. Additionally, several species of Lb have been routinely identified at high levels in healthy, low caries-risk individuals. Relying on these two pathogens as the diagnostic predictors for the disease in clinical practice may provide only limited results, as non-specific diagnostic tests emerge that identify levels of aciduric bacteria.

Therefore, as a biofilm disease, dental caries is much more complicated than previously thought because it involves multiple species interacting together as an ecosystem.[10] This makes caries treatment significantly more complex, and it may require long-term repeated cycles of strategic therapies to achieve good results.

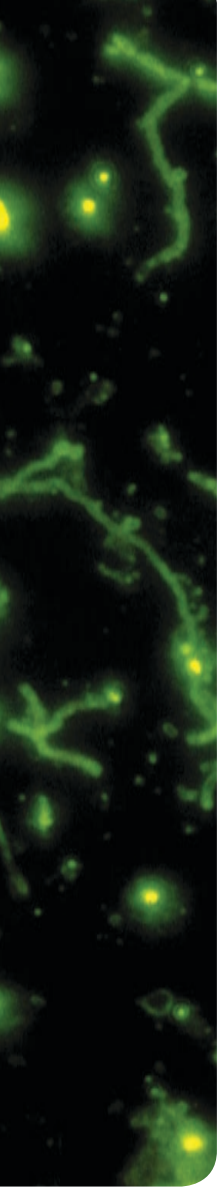
Linking science with application

As the science develops, newer disease models that more appropriately fit the growing body of evidence are emerging. The following models help illustrate the missing link between science (theory) and its application to normal clinical practice (caries risk assessment).

1. Ecological plaque theory

Proposed by Marsh, this theory contends that selection pressure, primarily low pH, causes a shift in the bacterial ecology of the mouth from healthy bacteria to primarily acidogenic/aciduric/cariogenc bacterial species. These species may account for less than 1% of a healthy biofilm, but

Bacteria photos: The Centers for Disease Control and Prevention, www.cdc.gov



in a high caries-risk individual they may account for more than 96% of the bacteria population. Multiple studies have clearly demonstrated that the selection pressure for this population shift is low pH, favoring the multiple species of acidogenic/aciduric bacteria responsible for the cariogenic biofilm.[11]

2. Caries balance model

Dr. John D.B. Featherstone has expanded the ecological plaque theory in his caries balance model to include identifying all factors contributing to or preventing the bacterial population shift from primarily healthy bacteria to cariogenic bacteria in the patient's oral biofilm.[12] Identifying risk versus protective factors provides broader evidence as the basis for sound diagnosis and also results in logical direction for specific therapeutic measures targeted at these specific factors for individual patients.

3. Caries risk assessment

This caries balance model for disease has led to the development of a caries risk assessment form, which is used to evaluate and identify these known factors with each patient. Routinely using a standardized caries risk assessment form has become a rapidly growing standard of care in dental schools—one that should be integrated into clinical practice. Many forms are available and can be downloaded from these Web sites: The California Dental Association (CDA), *www.cdafoundation.org*; or Oral Biotech's CariFree system, *www.carifree.com* (see sample form, below left). Information gathered from these questionnaires becomes integral in the caries diagnosis and treatment process.

Dental caries diagnosis and risk assessment is made from a combination of data; no single piece of data is used as a standalone for caries diagnosis. Instead, the more information, the better the decision process. The assessment should include results from the clinical exam, radiographic exam, caries risk assessment form, a patient's decay history and experience over time, and direct bacterial or biofilm culturing or metrics including saliva testing. Examining all of the pertinent information results in a clearer picture of the disease status and future risk assessment.[13]

CARIES RISK ASSESSMENT FORM – ADULTS/CHILDREN OVER AGE 6

Patient Name: _____ Age: _____

Instructions: **Circle the answers that apply:**

FACTORS	HIGH	MODERATE	LOW
1. Disease Factors			
* Visible cavitations	yes		no
* Cavity in last 3 years	yes		no
* Radiographic lesions	yes		no
* White spot lesions	yes		no
2. Risk Factors			
Deep pits/fissure		yes	no
Inadequate saliva flow		yes	no
Exposed roots		yes	no
Appliances present		yes	no
GERD	yes		no
Sjogren's syndrome	yes		no
Hyposalivary meds	yes		no
Radiation Therapy	yes		no
Snacks between meals	>3 times	1-3 times	infrequent
Regular Soda	yes	infrequent	no
Recreational drugs	yes		no
3. Protective Factors			
Fluoridated water	no		yes
Fluoridated toothpaste	no		yes
Adequate saliva flow	no		yes
Fluoride mouthrinse		no	yes
Xylitol gum/mints		no	yes
CariFree rinse		no	yes
Other Rx rinse		no	yes
4. Laboratory Tests			
Screening	recommended	results	
Culture	recommended	results	

CARIES RISK ASSESSMENT	HIGH	MODERATE	LOW
PROGNOSIS	POOR	MODERATE	GOOD

I have been given the recommendation to have a CARICULT to determine my bacterial counts as a part of my overall caries risk assessment. I understand the risks and benefits of the test and I decline, releasing my dentist(s) of any liability associated with declining the test.

Release signature_____ Date_____

Sample caries risk assessment form

From causes to care

In addition to assessing causative factors and caries risk using updated caries disease models, we can apply some post-diagnostic treatment specifics and present clinical examples of treatment strategies for a low caries-risk patient, a moderate caries-risk patient, and a high caries-risk patient. An outline of this treatment process, from screening to re-care, is presented in "Dental caries decision tree," page 64.

After a caries diagnosis is made, steps should be taken to provide effective treatment—not just for the existing end stages of the disease, such as cavitations and pulp death, but also in identifying therapies and strategies aimed at reversing the biofilm to favor healthy bacterial species and reducing future caries risk.

The American Dental Association (ADA) offers the parameters of caries risk categories, grouped by ages, for children through adults[14] (see "ADA caries risk guidelines," page 66). After completing risk assessment and determining a diagnosis, the next practical step is to identify appropriate treatment and therapeutic strategies tailored to each patient's individual risk factor(s). This treatment process can be broken down logically into several steps. Because patient compliance is crucial in successful caries treatment, simplified treatment strategies combined with fewer patient compliance issues increase chances for successful treatment outcomes.

Beyond traditional models

Advances in biofilm research have moved clinical thinking about the possible causes of caries beyond just the traditional MS and Lb disease models and into linked considerations including the ecological plaque theory, caries balance model, and caries risk assessment. Caries diagnosis should not be based upon a single piece of data—instead, one should rely on multiple sources, including post-diagnostic steps and treatment frequency and fees.

Going after the biofilm: GIC and fluoride

After a diagnosis of caries disease, the biofilm needs to be managed. When treating this biofilm aspect, consistent with

BACTERIA IMPLICATED IN DENTAL CARIES

BACTERIAL SPP.	AUTHORS										
	Beighton	Hayes	van Houte	Becker	Loesche	Hamada	Kleinberg	Bunting	Tanner	Hoshino	Sissons
Streptococcus salivarius	X			X							
S. parasanguinis				X							X
S. constellatus				X							
S. mutans		X	X	X	X	X	X				X
S. sobrinus			X		X	X	X				
S. oralis	X		X				X				X
S. milleri			X								
S. mitis			X								X
S. gordonii			X								
S. anginosus			X								
S. cricetus							X				
S. intermedius									X		X
Lactobacillus fermentum				X							X
L. plantarum										X	X
L. acidophilus							X	X			
L. casei							X				
Candida albicans						X					X
Actinomyces israelii	X										X
A. gerensceriae	X			X							
A. naeslundii	X										
Veillonella				X							
Veillonella parvula							X				
Bifidobacterium			X	X							
Neisseria sicca							X				
Fusobacterium animalis				X							
Capnocytophaga gingivalis											X
S. vestibularis											X

**These bacterial species are related to caries as per the author's interpretation after reading articles by the listed authors.

"The worst-case scenario is not the patient who drinks
drive to work, but the one who sips that same drink co

Dental caries DECISION TREE

A simple decision tree for the sequence from screening to re-care looks like this:

1. **Screening tests:**
   - Self-assessment test
   - pH determination
   - Caries susceptibility test
2. **Validation tests:**
   - Caries risk assessment form
   - Clinical exam
   - Radiographic exam
   - Motivational interview
   - Bacterial metric/culture or biofilm metric
3. **Diagnosis:**
   - ADA Council on Scientific Affairs definitions
4. **Corrective strategies:**
   - **Reparative:**
     - Restorative
     - Remineralization
   - **Therapeutic:**
     - Antimicrobial
     - pH
     - xylitol
   - **Behavioral:**
     - Oral hygiene instructions
     - Dietary counseling
   - **Non-modifiable factors:**
     - Special needs
     - Xerostomia
     - Medication-induced xerostomia
     - Ongoing preventive
5. **Re-screening/Re-evaluation/ Re-care cycle**

the traditional surgical treatment of the disease, all existing cavitations should be restored. In a high caries-risk individual it may make good sense to restore these areas of decay using glass ionomer cement (GIC) sealant as an interim or provisional material while other strategies are implemented to reverse the biofilm. Such a strategy provides two benefits: It reduces the likelihood of recurrent decay locally and provides a short-term fluoride reservoir in the mouth.

Fluoride therapy has proven highly effective in treating dental caries, and it also can be considered a reparative step in attempting to remineralize non-cavitated or white spot lesions. Although different vehicles exist for fluoride delivery, the best method appears to be fluoride varnish. Both the ADA and CDA recommend this therapy for moderate- and high-risk caries therapy. Recent research indicates significant caries reduction and benefits increasing from one application to up to four applications per year.[15]

The CDT VII fee codes (see "Fee codes," page 67) now feature fee code D1206 for this specific procedure on moderate and high caries-risk patients. Fluoride varnish supplies high yet safe levels of fluoride, demonstrates good substantivity, and removes patient compliance from the equation. The greatest benefit may be the latter, because the success of this therapy does not depend on patient cooperation—the fluoride is applied professionally and simultaneously to all teeth.

Additional treatments

Additional therapies directed at the biofilm disease include antimicrobial, xylitol, pH, and behavioral strategies.

Antimicrobials

Numerous attempts have been made to provide antimicrobial treatment for dental caries—the cariogenic biofilm chlorhexidine, in addition to povidone iodine, ethyl alcohol, sodium hypochlorite, chlorine dioxide, triclosan, cetylpyridium chloride, and even essential oils. These all have been used in mouth rinses and other oral care products with varying degrees of success. Fluoride also has been successfully used at high levels as an antimicrobial. Bacteria in biofilm are known to be more resistant to antibiotics, antibodies, and antimicrobial agents, increasing the challenge of creating good treatment outcomes.

Xylitol

In addition to antimicrobial agents, using xylitol has shown some bacteriostatic-type properties with cariogenic bacteria; plus it is a sugar/alcohol that MS cannot metabolize. Scientific studies further indicate that xylitol significantly reduces transmission of these bacteria from mother to child, reduces the ability of the bacteria to stick to the teeth, and potentiates the effect of even low levels of fluoride.[16] Xylitol is available in chewing gum, mints, and some oral rinses and gels. It is advisable to include some form of xylitol in the overall anti-caries treatment plan.

Managing pH

pH strategies play an important role in reversing the selection pressure on the diseased biofilm. While cariogenic bacteria are both acidogenic and aciduric, many healthy oral bacteria have the ability to elevate the oral pH after acid development in the classic Stephan curve from dietary events. This is accomplished through several vehicles—primarily salivary bicarbonate, urea and arginine. Many bacteria possess enzymes to break down the arginine and urea into ammonia, carbon dioxide, and acetates, which function to elevate the oral pH. This helps maintains a normal pH range in the mouth, reducing the acidic pH selection pressure caused by cariogenic organisms. Dental caries can be described as a dynamic dysfunction of the movement of calcium (Ca+) and phosphate (PO4) ions from the tooth to the plaque to the saliva, with the bacterial plaque biofilm acting as the intermediary. The bacteria are negatively charged on the cell wall and co-aggregate and are bridged together by the positively charged calcium ions.

At normal pH range (from 6-8), there is no selection pressure on the biofilm, and the healthy bacteria are stable and resist pathogenic attack. As short-term episodes of acidic pH occur with eating, any demineralization that occurs is quickly reversed, as the healthy bacteria help raise oral pH to a normal or neutral level.

However, when prolonged episodes of low pH occur, there is rapid selection for the cariogenic organisms. At pH below 5.5, Ca+ and PO4 ions move from the tooth to the biofilm, become lost into the saliva, and demineralization occurs. Prolonged demineralization leads to cavitation and bacterial invasion of the tooth surface.

Conversely, elevated pH situations favor remineralization of enamel as Ca+ and PO4 ions move back into the tooth, and excess ions precipitate in the biofilm to act as a reservoir for future low pH events. At high pH levels (from 8-10), the calcium ions are not readily available to bridge the bacteria, thereby challenging co-aggregation and plaque development. Not surprisingly, there is a clinically inverse relationship of dental caries and calculus in the remineralization cycles. Therefore, therapeutic strategies targeted at raising the oral pH provide several benefits: the elevated pH drive remineralization; cariogenic selection pressure is reduced; and co-aggregation and plaque development are challenged. Strategies to elevate oral pH have been indicated as even more significant than fluoride therapy, according to one population study.[17]

Behavioral strategies

Behavioral strategies include good oral

the ‘Big Gulp’-sized cup of Mountain Dew during the
ontinuously in small amounts all day long.” —Dr. B.C. Nelson

hygiene instruction combined with appropriate dietary counseling. It is important to reduce the overall bacterial load in the mouth, but in the hands of most patients, brushing and flossing alone cannot effectively modify or treat this disease. Without modifying the environment and the pH selection pressure, the biofilm that reforms after oral hygiene measures is still predominated by the existing cariogenic bacteria. Therefore, it’s not realistic to simply brush and floss the disease away.

Patient dietary habits should be considered a mainstay of behavioral modification. Dietary counseling is important, for it centers around frequency of consuming refined carbohydrates. Interestingly, the frequency of snacking is more important than the exact nature of the snack food. Snacking more than three times per day between meals significantly raises a patient’s caries risk. Refined carbohydrates—foods including white bread, white rice, pasta from white flour, and some sugary cereals—are best consumed during a meal.

The worst-case scenario is **not** the patient who drinks a “Big Gulp”-sized cup of Mountain Dew during the drive to work, but the patient who sips that same drink continuously in small amounts all day long. Recommendations for dietary changes often play an important role in treatment success. But dentists and patients must develop realistic expectations when the success depends upon patient behavioral changes.

Some behavioral factors may not be modifiable. This may be true with a special needs patient who is not capable of complying with all of the suggested therapies. Some patients are xerostomic, which creates additional challenges in disease treatment and control. Many senior patients commonly exhibit some level of medication-induced xerostomia or salivary reduction. Often, combining multiple medications compounds this problem, and for many this issue cannot be modified. Therefore, clinicians must direct additional effort at helping these patients keep their mouths hydrated while maintaining a healthy oral pH balance. For all patients, ongoing preventive measures should be addressed, including home care habits, dietary habits, fluoride therapy, pH strategies, and appropriate frequency of recall exams and radiographs.

Treatment frequency and fees

The ADA recommends frequency of radiographs based on the patient’s car-

ADA caries RISK GUIDELINES

The ADA Council on Scientific Affairs has developed a series of definitions or parameters for each of the caries risk categories for age groups 0 to 5 years old and 6 to adult.

- Low caries risk (all age groups): no incipient or cavitated primary or secondary lesions in the past 3 years and no risk factors.
- Moderate caries risk (0 to 5 years): no cavitated lesions in the past 3 years but at least one risk factor.
- Moderate caries risk (6 years to adult): one or more cavitated lesions in the past 3 years or at least one risk factor.
- High caries risk (0 to 5 years): Any of the following, including any cavitated lesion in the past 3 years, multiple risk factors, low socioeconomic status, suboptimal fluoride exposure, xerostomia.
- High caries risk (6 years to adult): Any of the following, including three or more cavitated lesions in the past 3 years, multiple risk factors, suboptimal fluoride exposure, xerostomia.

Source: ADA Council on Scientific Affairs[18]

Fig. 1 Low caries-risk patient treatment is centered on maintaining primary oral health.

Fig. 2 Moderate caries-risk patient treatment focuses on keeping healthy biofilm and on remineralizing, repairing, and preventing white spot lesions.

CLINICAL TREATMENT EXAMPLES

The following are examples of clinical treatment strategies for patients who have low caries risk, moderate risk, and high risk.

Fig. 3 High caries-risk patient treatment is a multiphased approach involving repair, dietary counseling, and increased dental care.

ies risk assessment.[18] The sidebar "ADA caries risk guidelines," (upper left) gives a glance at specifics per patient risk factor. However, all patients should be screened and examined at least once a year, as one's caries risk can change over time. The low caries-risk patient of today could become tomorrow's high-risk patient.

As discussed in this article, besides the focus on frequency, the level of a patient's caries risk determines selection of the proper clinical treatment method—that is, the application of CAMBRA principles determine a patient's clinical implications. We will now present some clinical examples involving patients with various caries risk factors

Clinical treatment examples

The following are examples of clinical treatment strategies for patients who have low caries risk, moderate risk, and high risk.

1. Low caries-risk patient (Fig. 1)

The patient is a 51-year-old female with no significant findings in the medical history. There is no recent history of decay and there are no visible lesions. The caries risk assessment form indicates no risk factors, and there are no radiographic lesions. The patient's Caries Susceptibility Test and aciduric bacterial culture both score low. The patient has no apparent active disease and meets the ADA criteria for low caries risk.

Treatment for this patient is centered on maintaining primary oral health. The patient is educated on the cause of dental caries and the concept of risk factors, and is scheduled for re-screening and re-care annually with radiographs every 2-3 years. Good dietary habits and oral hygiene are re-enforced, along with daily use of fluoride, pH strategies, and xylitol as optional preventive measures. The patient is given additional information about the benefits of sealants and fluoride varnish therapies. It is important to note that diagnoses for periodontal health and occlusal disease are made independent from the caries diagnosis. Recommendations for appropriate oral care are given and prioritized. Elective care for cosmetic or replacement treatment can proceed on this patient with no restriction on material selection.

2. Moderate caries-risk patient (Fig. 2)

The patient is a 24-year-old male with no significant findings in his medical history. There is no history of

Photos: Figs. 1-3 courtesy of Dr. V. Kim Kutsch.

decay or restorations in the past 3 years and there are no visible cavitated lesions. The patient's caries risk assessment form indicates white spot lesions as the only risk factor, and there are no radiographic lesions. The patient's caries susceptibility test and aciduric bacterial culture score in the low-to-moderate levels. The patient has potentially active disease in the form of white spot lesions and meets the ADA criteria for moderate caries risk.

Treatment is focused on reparative strategies targeted at remineralizing and repairing the white spot lesions, in addition to preventive strategies for future lesions and encouraging a healthy biofilm. The patient is given an initial fluoride varnish treatment along with daily-use xylitol/fluoride rinse and pH neutralizing dentifrice. He is encouraged to chew several pieces of xylitol gum per day and refrain from frequent refined carbohydrates in his diet between meals.

With a goal of promoting primary oral health, the patient is educated on the cause of dental caries and the concept of risk factors. He is scheduled for re-screening and re-care twice a year, with radiographs every 18-24 months. The appointments stress proper dietary habits and oral hygiene, including information on benefits of sealants and fluoride varnish therapies. Scheduling additional fluoride varnish therapy will be discussed at his re-care appointment.

With a patient who exhibits moderate caries risk, once again note that diagnoses for periodontal health and occlusal disease are conducted separately from the caries diagnosis. After recommendations for appropriate care are given and prioritized, any elective care for cosmetic or replacement treatment can proceed with caution as long as the patient understands his moderate risk for future disease. As it stands now, this patient's current risk may warrant delaying an elective treatment or it can influence restorative material selection.

3. High caries-risk patient (Fig. 3)

This 47-year-old male patient has a history of hypertension and takes daily medication. He has an extensive history of carious lesions, with multiple current lesions visible. The caries risk assessment form identifies multiple caries risk factors—visible cavitations, radiographic lesions, white spot lesions, restorations within the past 3 years, inadequate saliva flow, and excessive snacking between meals. The patient scores high on the caries susceptibility and aciduric bacterial culture tests. The patient has a high level of active disease and meets the ADA criteria for high caries risk.

Treatment for this patient has multiple approaches and multiple phases. The patient is educated on the cause of dental caries and the concept of risk factors, and undergoes several treatment phases.

The first phase of treatment is directed at reparative strategies to restore the existing cavitations, using GIC as an interim material. The patient also is given an immediate fluoride varnish treatment, with re-applications planned at 3 month intervals and is placed on daily use of antimicrobial mouth rinses and fluoride/xylitol rinses. He undergoes oral hygiene instruction (OHI) and dietary counseling to change his snacking behavior and improve his home care.

In addition to chewing five pieces of xylitol gum per day, the patient is given pH neutralizing dentifrice and oral spray for daily use. He is scheduled for rescreening and re-care in 3 months, when the biofilm will be recultured or re-evaluated. He is scheduled for radiographic exams every 6-12 months, depending on the clinical exam and presence of visible cavitations.

As with the other patients, diagnoses for periodontal health and occlusal disease are offered separate from the caries diagnosis. After receiving and prioritizing recommendations for appropriate care, the patient's long-term restorative care must be weighed and planned based upon his risk for caries disease. The final restorative phase should be postponed until a low caries-risk level is documented. This patient should be re-examined and followed actively at least every 3 months during the caries treatment phase to monitor treatment success levels. Additional therapies may prove necessary for this patient to accomplish a healthy biofilm and reduce caries risk.

FEE CODES

CDT VII Fee codes for the screening, diagnosis, and treatment of dental caries are as follows. Note the recent addition of code D1206:

- **D0415** Bacteriology studies
- **D0425** Caries susceptibility test
- **D0145** Oral evaluation <3 years of age and counseling with primary caregiver
- **D1206** Topical fluoride varnish; therapeutic application for moderate-to high-risk caries patients

Beyond drill 'n fill

Dental caries is a complex multifactorial biofilm disease. Successful treatment poses a tremendous challenge and requires addressing all factors involved in the disease process. In addition to educating patients about the cause of dental caries and identifying their specific risk factors, it's also important to help them develop appropriate, reasonable expectations for treatment outcomes.

But the clinician also should remember that a lifetime of developing disease cannot simply be wiped out in a week or even a month of therapy. The more significant the disease history, the more likely that the patient will require longer term strategies to help control the disease and reduce future risks. DPR

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