

Disease Prevention: Caries Risk Assessment

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Is it possible to prevent cavities before they start? This is a complex question and it is one that is getting some serious attention as the dental profession begins to understand the science of biofilms. In the past, the phrase preventive dentistry has been an overused buzzword utilized by both educators and clinicians alike, so much so that it has lost meaning and importance in the mind of the patient. In addition, the promise of prevention has never resulted in preventing disease for most people. So the phrase has lost credibility, and all the brushing and flossing, fluoride, dietary changes, etc, have not prevented as much disease as promised.

Now patients turn a deaf ear when they hear the term. They have been sold that same bill of goods too many times with too much disappointment to listen anymore, while the manufacturers have been complicit as well. Take a stroll down the dental care aisle of any pharmacy department, and the shelves are filled with products that promise everything you can think of, from cavity fighting, to cavity preventing, to whiter/sexier smiles, to promoting health. Yet in spite of these products' sales and use, patients continue to get cavities. Now when we say "prevention" to most patients, they give us that glassy-eyed, deer-in-the-headlights look and probably just hear "blah, blah, blah!" Not to be cynical, but for the most part, we shouldn't blame them.

At the turn of the 20th century there was a rapid age of discovery in dental caries as James Leon Williams first demonstrated the demineralization of enamel under a gelatinous plaque mass.¹ Within 3 years, G.V. Black concluded that the demineralization was caused by the lactic acid dissolving the calcium and phosphate salts from the enamel structure.² These discoveries led to an attempt to make dental caries a pathogen-specific disease that fit our understanding of the disease process at the time. Mutans streptococci (Ms) and Lactobacilli (Lb) became the usual suspects in future scientific studies. During the 1980s, research began to demonstrate that levels of Ms and Lb were directly linked to caries incidence. Early work by Zickert, et al concluded that not only was the incidence of dental caries related to levels of Ms and Lb by themselves and in combination, but children with high levels of these bacteria developed significantly more new cavitations than children with low levels.^{3,4} During the scope of their study, children with high bacterial levels developed 20.8 new cavitations versus 3.4 for children with low bacterial levels.⁵ These findings continued to support the earlier bodies of work implicating 2 pathogens in the caries process. Further studies began examining the effect of controlling the bacterial levels and a resulting reduction in caries incidence. More recently, we have described dental caries as a bacterial infection of these pathogens, and as such demonstrated transmission from mother to child,⁶ and in fact transmission to and from every member of a family unit. Primary caregivers have taken the place of mothers for working moms, and they, too, can transmit the disease. Studies indicate that the age and severity of exposure to these bacteria are the greatest predictors in the childhood caries experience. Reduction in the Ms and Lb levels in mom reduces the caries incidence in a child.⁷

Based on the work by Zickert, et al, diagnostic tools made their way to the market to culture Ms from the saliva and extrapolate the colony-forming units on the bacterial plaque.⁸ Clinicians began using fluoride rinses and chlorhexidine rinses to treat the bacterial infection. (More recently, povidone iodine has been used as an antimicrobial mouthrinse to fight this disease.⁹) Scientific studies under strict conditions demonstrated improved results, but trying to duplicate and maintain these conditions in clinical practice was difficult, if not impossible. Private practices continued to witness patients developing new cavitations despite their best efforts. The plaque theory just didn't hold water, and our early cultures and antimicrobial therapies didn't produce the results we were looking for. Once again, the 'prevention' word failed to deliver on its promises.

We continued to refine our understanding of dental caries and began looking at risk assessment models during the 1990s. Suddenly, the traditional approach of surgically removing the decay was being called into question, and many authors and opinion leaders in dentistry proposed treating dental caries like an infection with a medical management model.¹⁰ Too late we discovered that drilling and filling, even at a great pace, had little to do with treating the disease, although it is known that it offered temporary relief from pain and restoration of the teeth to function.¹¹ But dental caries continued, and this was not the end of the story.

It was determined that dental caries is a multifactorial disease, and research continued to examine how each of the factors affects the disease process. We next examined individual risk factors and the specific role they play in dental caries.¹² What role did fluoride really play? Where did diet, xylitol, and saliva fit into the equation? Too often dentists opined that if the patients just brushed and flossed, or used the fluoride gel, or eliminated sugar from their diet, or stopped drinking sodas, everything would just take care of itself. This was not the case at all.

Richard Simonsen first described the preventive resin restoration back in 1978 and put the wheels in motion to progress slowly on this concept of minimal surgical invasion of the tissue.¹³ At the turn of the 21st century, minimally invasive dentistry (MID) became a new phrase focused on saving healthy tissue, and this philosophy spawned the World Congress of Minimally Invasive Dentistry (WCMID.com). This group of educators, scientists, and clinicians from across the globe meet once per year to review research, education models, and clinical experiences with MID. Members of the WCMID are practicing caries risk assessment as a standard of care clinically and getting results.

In the past couple of years, John Featherstone introduced the concept of the caries balance,¹⁴ and demonstrated that managing dental caries by caries risk assessment does work. In a landmark study he demonstrated a significant reduction in new cavitations for patients who were treated with caries risk assessment and a medical model versus patients who were treated with the traditional surgical model of drill and fill. Medical management of dental caries is possible, and it provides better treatment outcomes than surgical intervention alone. So, we have established that dental caries is a multifactorial, transmissible bacterial infection that can be treated and prevented with a multifactorial, antimicrobial approach.

An Age of Discovery



Figure 1. CariScreen (Oral BioTech) Caries Susceptibility Testing Meter and Swab.



Figure 2. Caries Risk Assessment Form (CariFree [Oral BioTech]).

Fast-forward to 2006. We are once again in an age of rapid discovery as we now recognize that dental caries is not only a disease of bacterial origin, but it is a biofilm disease.¹⁵ When the oral environment favors these bacteria, the biofilm population shifts from the normal healthy flora to the acidogenic and aciduric bacteria associated with dental caries.^{11,16-19} A biofilm is present whenever there is fluid, a surface, and bacteria present. Species of bacteria attach to the surface and convert from planktonic to sessile. In the process they undergo up to 84 gene changes and are difficult to recognize as the same bacterium.

The biofilm is a sophisticated ecosystem with its own infrastructure. There are metabolic and waste channels, and bacteria share genetic information and communicate with each other. Within the biofilm the bacteria may now become up to 1,000 times more resistant to antibodies, antibiotics, and antimicrobial materials. The biofilm disease model represents a significant new challenge in treating dental caries. Previous scientific studies that examined the cariogenic bacteria as planktonic organisms in a Petri dish now may not translate to a biofilm disease model. The clinical studies, however, were treating a biofilm disease, whether the researchers were aware of it or not, and they are significant.

As we look at research that has taken place on biofilms outside of the mouth, from river streams to petroleum pipelines, we have identified several effective methods to treat a biofilm. Mechanical debridement, heat, and a strong oxidizing agent are effective therapies to disrupt the biofilm. These all face some limits, however, as we apply them to the mouth. In the mouth, it is nearly impossible to debride the cariogenic biofilm away completely. The biofilm reforms within hours after it is removed, and bacteria are ubiquitous in the mouth. The Ms are attached to the pellicle and are not removed by brushing and flossing.¹⁷ Heat can be applied to living tissue, but temperatures required to denature bacteria also denature human tissue. Strong oxidizers are used in different areas of dentistry, but they must be applied to the mouth with limitation and under strict conditions to be utilized safely.

The pathogenesis of the biofilm itself becomes a pH issue. Ms and Lb are acidogenic and aciduric.^{16, 17} These bacteria are able to metabolize sugars and produce lactic and acetic acids. They thrive in this acidic environment because they have the unique ability to pump the acid H⁺ ions back out of their cells to maintain intracellular neutrality in an acidic environment.¹⁹ This hydrogen ion pump mechanism is shared with all acidogenic/aciduric bacteria, and many more bacteria may play a role in the dental caries process that we have yet to identify successfully. So, these cariogenic/acidogenic/aciduric bacteria share this unique trait and produce and expend tremendous amounts of ATP to

maintain intracellular neutrality. In fact the magnitude of their ATP use is so much greater than other bacteria that this becomes a good metric to measure in determining whether a patient's oral bio-film is acidogenic or healthy. It is now possible to perform a caries susceptibility test (Figure 1) to determine the degree to which the patient's biofilm is cariogenic. While that much is straightforward, dealing with the cariogenic biofilm, treating it, and replacing it with a healthy biofilm becomes a challenge that requires an accurate diagnosis of the factors that led to the diseased biofilm and factors that may help reverse the situation. As a multifactorial disease, caries risk assessment then plays a significant role in helping the clinician routinely identify the known risk factors for dental caries. Use of a standardized caries risk assessment form for all patients adds a scientific measure to the diagnostic process. Such a form was published in the Journal of the California Dental Association in March 2003²⁰ and is also available from the CDA foundation, the WCMID, and CariFree.com (Figure 2).

The Science of Prevention

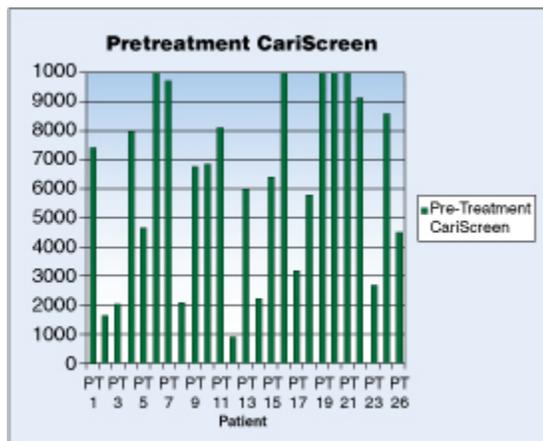


Figure 3. Data of caries susceptibility test preoperative results.

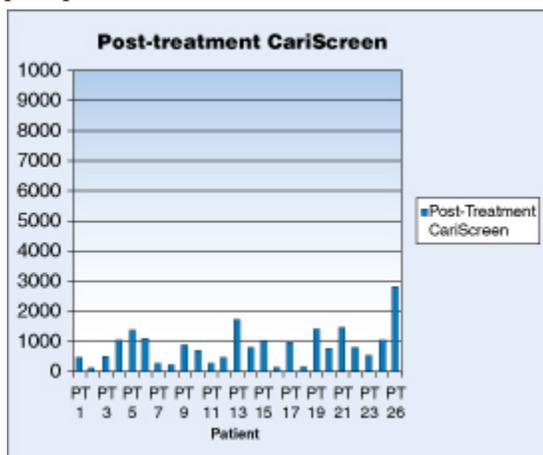


Figure 4. Data of caries susceptibility test treatment outcomes.

The science of prevention now includes identifying, documenting, treating, and validating treatment outcomes of the known caries risk factors in addition to the probiotic approach to treating the cariogenic biofilm and regenerating a healthy biofilm in its place.^{21,22} This is in addition to surgically removing the areas of decay and restoring the teeth to function. Ultimately, the underlying problem in all of this for clinicians is that scientific research supports the premise that we should be diagnosing and treating dental caries from a medical model, but there has been little scientific direction to describe exactly how to do so. There has been no accepted, standardized treatment regimen for treating the bacterial biofilm disease that causes dental caries. While the science is compelling, abundant, and remarkably clear that we should be doing this, clinicians' opinions are wide-ranging based on their previous paradigms, education, and experiences.

Can we prevent cavities before they start? The answer is finally yes! But it's important to recognize that this is still a complex question and the answer is complex, because dental caries is a multifactorial biofilm disease. We now counsel all patients and

give them hope for finally controlling their cavity issues. In our practices, for every patient, we should routinely provide caries risk assessment and screen with a caries susceptibility test at least once annually. Risk factors are known to change over time with changes in environment, saliva, systemic health, diet, and medications, so it makes sense to screen all patients once a year. The graph in Figure 3 represents the results from the caries susceptibility test for patients before treatment, and the graph in Figure 4 demonstrates the results postoperative to caries management by risk assessment and antimicrobial/probiotic therapy. The bottom line is, when a dental practice implements caries risk assessment and an antimicrobial/probiotic approach to treating the disease on all patients, it is possible to control the disease process, restore the mouth to health, and prevent cavities before they start. Our patients have newfound confidence and trust in their health and in the longevity of any fine restorative dentistry that has been done or is about to be done. We can now have the same level of confidence.

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Disclosure: Dr. V. Kim Kutsch serves as chief executive officer and chief technical officer of Cosmetic Dental Materials and Oral BioTech, which manufactures CariScreen and the CariFree System for Oral Health.

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Disclosure: Dr. Carson L. Kutsch is a minor shareholder in Oral Biotech, the manufacturer of CariFree.

