

Dental caries: A new look at an old disease

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Dental caries is an infectious transmissible biofilm disease of the teeth driven by protracted periods of low pH that results in net mineral loss of the calcified tissues.¹ Ultimately, if left untreated it results in pulpal death and loss of teeth. Dental caries is the number one childhood disease in the United States.² Although dental caries (tooth decay) is largely preventable, it remains the most common chronic disease of children aged 5 to 17 years – 5 times more common than asthma (59% versus 11%).² In addition, it affects adults as well, 27% of adults aged 35 to 44 years have untreated dental caries.² Thirty percent of senior adults over the age of 65 also have untreated dental caries.² This very common disease is perhaps the most difficult to diagnose and the most difficult to treat. There are four recognized disease indicators and multiple risk factors for the disease.³ These risks may also be specific by site or tooth surface, individual tooth location, tooth anatomy, individual, age, population and socioeconomic demographic strata.⁴ It is a very complex disease with a rapidly emerging understanding from the development of the biofilm scientific evidence.⁵ It is a disease that is not understood by the public and, for the most part, is not well understood by the profession dedicated to treating it.

Just the term dental caries by itself can be confusing. While the CDC lists the demographic data by untreated “dental caries,” what they most typically refer to is the presence of untreated caries lesions or cavities.⁶ (A list of terms and abbreviations is included to facilitate the reading and interpretation of the diagnostic criteria and results. Dental examiners were trained to use modified Radike’s criteria⁵ to diagnose dental caries and its sequelae (missing teeth [due to disease] and filled teeth). The modification consisted of eliminating the “extraction indicated” code.

Dental examiners were asked to dry the

tooth surfaces with compressed air and use a non-magnifying mirror and a No. 23 dental explorer to assess for the presence of carious and restored (filled) lesions. To be consistent with the NHANES 1988–1994 protocols and diagnostic criteria, pits and fissures were coded as carious if the explorer would catch after insertion with moderate, firm pressure, accompanied with either softness at the base of the lesion or an opacity adjacent to or evidence of undermining enamel. Four surfaces of incisors and canines and five surfaces, including the occlusal surface, of premolars and molars were examined. No radiographs were taken. Detailed diagnostic and coding guidelines were included in the procedures manuals for dental examiners and recorders available at the NHANES web site.¹)

To effectively understand this disease we must separate the signs and symptoms from the etiology and diagnosis of the disease process proper. The CDC data provides a clear picture of the number of untreated cavities in a population group, which creates meaningful data to track over time, but assumes the cavity is the disease, which is not the case.⁷ Dental caries is the infectious transmissible biofilm disease that causes the cavities. The presence of this disease and/or risk factors among these populations is not well accounted for simply by identifying cavities. The presence of other disease factors, such as enamel white-spot lesions, radiographic interproximal lesions, and a history of caries lesions, provides a much more accurate profile of this disease in any population.³ The dental profession is beginning to recognize the significance between identifying lesions and diagnosing the disease process.⁸ Simply restoring the caries lesions does not effectively treat the disease process. To effectively treat dental caries requires an understanding of the local, microbial, behavioral, environmental, and socioeconomic factors contributing to the disease.⁹

DISEASE MODELS

Until recently, dental caries was thought to be a fairly simple disease. It was caused primarily by two bacteria, **Mutans streptococci** and *Lactobacillus*^{10,11} and required only refined sugar and tooth structure to occur. The traditional disease model was supported by an abundance of scientific studies linking *Mutans streptococci* and *Lactobacillus* levels to caries risk in children.^{12,13} But as the field of biofilm research developed, a broader, more complex picture became apparent. More bacteria were implicated in the disease process by different researchers worldwide.¹⁴ Now oral biofilms can be studied with forensic-type precision by identifying the bacteria with the 16S gene sequence of their rRNA.¹⁵ This has added additional species to the growing list of implicated dental caries pathogens.¹⁶⁻²⁸ As early as 1989, Marsh demonstrated that the selection pressure for the acidogenic/aciduric bacteria in a mixed culture biofilm was a function of pH and not sugar availability.²⁹ This early research later led to his description of the Ecological Plaque Hypothesis.³⁰ In this biofilm disease model, the environmental factor of low pH drives the selection of cariogenic bacteria in a patient's dental biofilm, causing mineral loss and cavitation of the teeth.³¹ Studying the effects of different risk factors, Featherstone introduced the concept of the caries balance in 2004. He demonstrated that dental caries and health are also a function of a balance – or rather an imbalance – between the pathologic and the protective factors for the disease.³² So the understanding of the disease model became more complex: one of disease indicators and risk factors, protective factors, and numerous pathogens present in a biofilm behaving based on ecological principles.

Recent biofilm research based on 16S gene sequence DNA evidence is also broadening the picture of dental caries.⁵ It is clear now that some of the previous paradigms on the microbiology of dental caries were wrong.^{5,28} The mouth represents a unique environment in the body for biofilms. The teeth are the only non-shedding surfaces in the body, so the biofilms on the teeth tend to be more complex and microbiologically diverse than previously thought.³³ While more than 700 bacterial phylotypes could potentially be found in the human mouth, a healthy individual will only have around 113 different bacterial species, while a high caries risk

individual will have an average of 94, presumably because fewer bacteria are capable of surviving the low pH conditions consistent with the disease.³⁴ There is also an inverse relationship between the bacteria present and absent in healthy versus high caries risk individuals.³⁵ The bacteria are also site-specific on the teeth, with individual sites containing only 20 to 30 different phylotypes for an individual.³⁴ Some bacteria are common to all sites, like *Gemella*, *Granulicatella*, *Streptococcus*, and *Veillonella*, while others are more site-specific. The occlusal fissures are predominated by *Mutans streptococci*, but the smooth surfaces contain mostly *Actinomyces* and other streptococcal species. The interproximal areas are predominated by anaerobic and periodontal species and the cervical regions demonstrate a strong presence of gingival related bacterial.³⁴ The concept of dental caries being a disease of *Mutans streptococci* and *Lactobacilli* needs to permanently be put to rest, as the biofilm scientific evidence strongly suggests otherwise.

Most recently, Takahashi and Nyvad provided an even clearer picture of this disease.⁵ They compiled evidence from a broad range of scientific sources and combined the current DNA biofilm evidence with other known concepts in the caries process. Research indicates that additional streptococcal species are potentially cariogenic, and they described these as low-pH non-*Mutan streptococci*.⁵ This group includes *Streptococcus mitis*, *oralis*, *gordonii*, and *anginosus*. Under prolonged periods of low environmental biofilm pH, these bacteria actually adapt and become acidogenic/aciduric, creating a pH similar to *Mutans streptococci* and producing acid equally as fast. These bacteria

are able to adapt to this environment with four strategies: their cell wall becomes more impervious to the H⁺ ions, they up regulate ATP-ase activity to increase their metabolism and ability to transport the H⁺ ions out of their cell, they induce the arginine deaminase system to increase the pH, and they produce stress proteins to protect their intracellular enzymes and DNA.⁵ The significance of this research is that *S gordonii* is an early colonizer and, along with *S oralis*, has previously been widely considered as a healthy member of the biofilm.^{5,34} Takahashi and Nyvad point out that it is no longer just a consideration of which specific bacteria are present, but rather what those bacteria are doing. Are they behaving as healthy bacteria in a neutral and balanced biofilm, or are they behaving as acidogenic/aciduric bacteria contributing to the dental caries disease process? The question becomes: are they good bacteria or bad bacteria? And the complex answer is yes, both. Takahashi and Nyvad present a broadened view of this disease, the Extended Caries Etiological Hypothesis. In this theory they conclude that because of the complexities of this disease model, traditional treatment methods will fall short. The best approach will not be targeting a specific group of organisms like *Mutans streptococcus* through gene therapy, vaccine or antimicrobial treatment, but rather environmental measures to be implemented to stimulate the bacteria such as non-*Mutans streptococcus* and *Actinomyces* by avoiding acidification of the biofilm. Logical treatment strategies would include pH neutralizing techniques. While perhaps more challenging, this disease model represents the best current understanding of dental caries.

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The dental profession is beginning to recognize the significance between identifying lesions and diagnosing the disease process. Simply restoring the caries lesions does not effectively treat the disease process.

Caries Risk Assessment Form
Children under 6 Years

Name: _____ Date: _____
Instructions: Circle all answers that apply.
If 1 or more DISEASE INDICATORS or 2 or more RISK FACTORS are identified, then the patient is AT RISK and preventative therapeutic intervention is recommended to reduce the risk of dental caries (tooth decay).

	AT RISK	LOW RISK
1 SCREENING		
Biometric/Bacterial Test	Yes	No
2 DISEASE INDICATORS		
Mother/Caregiver active caries	Yes	No
Socio-Economic risk	Yes	No
Visible cavitations	Yes	No
Cavities in last 2 years	Yes	No
White spot lesions	Yes	No
RISK FACTORS		
Visible plaque	Yes	No
Gingiva bleeds easily	Yes	No
Inadequate saliva flow	Yes	No
Appliance present	Yes	No
No dental home/Episodic care	Yes	No
Developmental problems	Yes	No
Medications	Yes	No
Continuous bottle feeding (not water)	Yes	No
Sleeps with bottle	Yes	No
Nurse on demand	Yes	No
Frequent snacking	Yes	No
Other	Yes	No
DIAGNOSIS - ASSESSMENT	AT RISK	LOW RISK
3 PRESCRIPTION RECOMMENDATION		
<input type="checkbox"/> Fluoride Varnish	<input type="checkbox"/> Xylitol	<input type="checkbox"/> pH
<input type="checkbox"/> Homecare	<input type="checkbox"/> Nutritional Counseling	<input type="checkbox"/> Caregiver Exam

*Based on Featherstone's clinically proven Caries Risk Assessment.
*Caries risk criteria as defined by the American Dental Association Council on Scientific Affairs, ADA August 2006.

Fig 1: Child's Caries Risk Assessment Form

Caries Risk Assessment Form
Adults/Children over 6 Years

Name: _____ Date: _____
Instructions: Circle all answers that apply.
If 1 or more DISEASE INDICATORS or 2 or more RISK FACTORS are identified, then the patient is AT RISK and preventative therapeutic intervention is recommended to reduce the risk of dental caries (tooth decay).

	AT RISK	LOW RISK
1 SCREENING		
Biometric/Bacterial Test	Yes	No
2 DISEASE INDICATORS		
Visible cavitations	Yes	No
Radiographic lesions	Yes	No
White spot lesions	Yes	No
Cavities in last 3 years	Yes	No
RISK FACTORS		
Visible plaque	Yes	No
Inadequate saliva flow	Yes	No
Hyposalivary Medications	Yes	No
Acidic Beverages	Yes	No
Frequent Snacking (1-3 times daily)	Yes	No
Orthodontic or other appliances present	Yes	No
Deep pits and fissures	Yes	No
Exposed roots	Yes	No
Other	Yes	No
DIAGNOSIS - ASSESSMENT	AT RISK	LOW RISK
3 PRESCRIPTION RECOMMENDATION		
<input type="checkbox"/> Fluoride	<input type="checkbox"/> Antimicrobial	<input type="checkbox"/> pH
<input type="checkbox"/> Xylitol	<input type="checkbox"/> Homecare	<input type="checkbox"/> Diet

*Based on Featherstone's clinically proven Caries Risk Assessment.
*Caries risk criteria as defined by the American Dental Association Council on Scientific Affairs, ADA August 2006.

Fig 2: Adult's Caries Risk Assessment Form.

CLINICAL IMPLICATIONS

The new biofilm science and disease model has significant clinical implications for the practitioner treating patients with dental caries, or with caries risk. Currently there has been a lot of information and research performed on laser caries fluorescence as a diagnostic tool.³⁶ In light of the new disease model, it is important to distinguish between lesion detection or identification and caries diagnosis. Instruments like the DIAGNodent (Kavo Corporation, Lake Zurich, IL) are invaluable clinical instruments that have dramatically improved the precision in lesion detection,³⁷ and bring new objective technology to replace the mirror and explorer.^{38,39} They also play a role in the diagnosis phase of dental caries. However, the diagnosis of the disease process is not limited to the presence and extent of lesions. Diagnosis of dental caries requires all diagnostic data to be gathered and a clinical decision to be made in the best sound judgment of the practitioner. This should include: an oral examination looking for disease indicators previously discussed, a radiographic exam, an evaluation of risk factors, use of a validated caries risk assessment form (Figure 1 and Figure 2), a microbial metric, and, if one exists, the patient's previous history with the practitioner.⁴⁰ Previous microbial metrics involved culturing saliva samples for *Mutans streptococci*.⁴¹ Based on the biofilm disease

model, a better metric might be ATP bioluminescence.⁴² The survival of acidogenic/ aciduric bacteria depends on their ability to produce enough ATP to effectively transport the H⁺ ions out of the cell, thereby maintaining intracellular neutrality.⁴³ The concept of ATP bioluminescence has been tested with excellent correlation values in dental caries risk assessment, and fits the non-specific bacterial biofilm model.⁴² A diagnosis is then made based on weighing all of the evidence. The caries risk assessment form and risk factors further identifies specific contributing conditions to the disease process for each individual patient, and provides a starting point in the design of the evidence based therapy.⁴⁴

The caries risk assessment for

children age 5 and under includes disease indicators: caries activity status of the mother/ primary caregiver, socioeconomic status, visible cavitations, cavity history in the previous 2 years and obvious white-spot lesions (Figure 3).⁴⁵ Caries risk factors for children include: obvious plaque on teeth, the gingiva bleeds easily, inadequate saliva flow, appliances present, no dental home with episodic regular care, developmental problems/special needs, medications inducing xerostomia, continuous availability of a bottle containing anything but water, opportunity to nurse on demand, and frequent snacking.⁴⁵ For children over the age of 6, adolescents, and adults, the caries risk assessment form should include disease factors (Figure 4): visible cavitations, radiographic interproximal lesions penetrating to the dentin or D1 lesions, obvious white-spot lesions, and a cavity in the previous 3 years.⁴⁶ Risk factors for this group should include: visible plaque on the teeth, inadequate saliva flow, hyposalivary medications, frequent acidic beverages, frequent snacking between meals, appliances present, and deep developmental occlusal pits and fissures.³ Taking these disease indicators and risk factors into account with the rest of the diagnostic data, the practitioner can make the best diagnosis and simultaneously develop the most effective treatment plan. The ADA Council on Scientific Affairs established a guideline and definition for these risk categories,⁴⁷ and now also endorses caries risk assessment and has developed a form for practitioners.⁴⁸

CAMBRA is a risk assessment based medical management model for dental caries.⁴⁹ The CAMBRA model protects, preserves, and remineral-



Fig 3: Visible plaque on child's teeth.



Fig 4: Visible cavitations, white spot lesions and plaque on the adult's teeth

izes dental hard tissues. CAMBRA is based on risk assessment, followed by diagnosis of the disease process and the appropriate evidencebased treatment strategies based on the patient's individual risk factors and restorative needs. These treatment strategies include: reparative strategies (remineralization for non-cavitated and/or whitespot lesions and restorative for cavitated lesions), therapeutic (antimicrobial to reduce total bacterial load, metabolic to inhibit growth of bacteria, and pH-neutralizing strategies to stimulate non-*Mutans streptococci* and *Actinomyces* species) and behavioral (modifiable behavior with nutrition counseling and homecare instruction and non-modifiable risk factors such as hyposalivary medications or special needs individuals).⁵ In the past, the profession has depended on fluoride as an anti-caries strategy, and while it aids in remineralization and may inhibit some cariogenic bacteria, fluoride therapy alone has not been enough to control this disease. Chlorhexidine has been a standard

antimicrobial in dentistry, and while it is an effective agent for periodontal pathogens, and against *Mutans streptococci*, it has little effect against *Lactobacilli* and its effectiveness against the rest of the implicated cariogenic bacteria remains uncertain.⁵⁰ Sodium hypochlorite is an effective broad-spectrum antimicrobial agent with little adverse effects, and is an elevated pH.⁵¹ Based on Takahashi and Nyvads' work, pH neutralization strategies appear to be the best fit for the present understanding of the disease model and are a promising although not yet well-tested strategy.⁵²

The pH strategy serves two functions; it elevates the pH to induce remineralization, and it also stimulates or trains the biofilm to behave correctly. Xylitol is an effective metabolic anti-caries agent that provides many benefits and also potentiates the effect of even small amounts of fluoride.⁵³ There are many different strategies available to help practitioners and patients effectively deal with this disease. But the first step is to recognize

the need to diagnose the disease and not just identify cavities.⁵⁴

CONCLUSION

Biofilm research is rapidly changing the dental profession's understanding of this very old disease, dental caries. The biofilm science is creating a new model of this complex disease, from which new diagnosis and treatment strategies are developing. CAMBRA represents an effective medical model for caries management in dental practice. While other dental therapies may be controversial, there is no debate that the standard of care requires CAMBRA evaluation and then treatment for susceptible patients. CAMBRA is not controversial because there is no controversy between scientific right and customary ignorance.⁵⁵

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Dr Kutsch is a founder, fellow, diplomate and first president of the World Congress of Minimally Invasive Dentistry. He has served on the board of directors of the American Academy of Cosmetic Dentistry and is a past president of the International Academy of Laser Dentistry, as well as the Academy of Laser Dentistry.

CPD Questionnaire

1. What is the most current definition of caries?
2. What drives the caries disease process?
3. What was the relevance of Marsh's research?
4. DNA biofilm evidence has shown that a high caries individual has _____ bacterial species present?
5. The concept of dental caries being a disease of *Mutans streptococci* and *Lactobacilli* needs to be permanently put to rest because?
6. Under prolonged periods of low biofilm pH many bacteria adapt and become _____?
7. The research of Takahashi and Nyvad suggests a logic treatment strategy?
8. The survival of acidogenic/aciduric bacteria depends on their ability to do what?
9. Previous microbial metrics involved culturing saliva samples for *Mutans streptococci*. What other methods have been tested to show excellent correlation to the non-specific bacterial biofilm disease model?
10. What is CAMBRA?
11. Why is Sodium hypochlorite an effective treatment agent?