Caries

The Caries Balance: Contributing Factors and Early Detection

John D.B. Featherstone, MSc, PhD

John D.B. Featherstone, MSc, PhD, is professor and chair of the Department of Preventive and Restorative Dental Sciences at the University of California at San Francisco.


Abstract

The aim of this article is to present a brief overview of the dental caries process, in particular, the management of dental caries and the role of early detection methods in the clinical management of caries.

Dental caries is simply tooth decay. There is a small number of key contributing factors. First, specific bacteria are involved that can metabolize fermentable carbohydrates and generate acids as waste products of their metabolism. The two principal groups of bacteria that have been implicated in dental caries are the mutans streptococci and the Lactobacilli species. The principal species in the mutans streptococci group are S. mutans and S. sobrinus. These bacteria are all called acidogenic because they produce acids from carbohydrates. When the acids are produced by the bacteria, they diffuse into the tooth enamel or dentin and dissolve or partially dissolve the mineral from crystals down inside the tooth. The tooth enamel and dentin are tissues made up of millions of tiny crystals. The mineral involved is termed a carbonated hydroxyapatite. This is a calcium phosphate with numerous impurity inclusions, the most important of which is the carbonate ion, which makes the mineral more acid-soluble than pure hydroxyapatite. If the dissolving of the mineral is not halted or reversed, the early subsurface lesion becomes a cavity. Last, dental caries is a transmissible bacterial infection.

Dental caries occurs in deciduous teeth and permanent teeth, regardless of the age of the individual. Lesions vary from the very early lesion of enamel, which is manifested as a "white spot," to frank open cavities. Early childhood caries is a similar process to later childhood and adult dental caries of enamel. Root caries is dental decay of the
tooth root, which occurs in adults after the gingiva recede.\textsuperscript{8}

What We Know About Caries

The process of dental caries is well-understood.\textsuperscript{1} We know how demineralization (loss of mineral) and remineralization (regaining of mineral) occur, and we have a very good understanding of how fluoride works to inhibit or reverse dental caries. Much is known about the multiple roles of saliva and salivary components.\textsuperscript{9} We know that acidogenic bacteria (described above) cause demineralization and, therefore, caries. These bacteria are also termed cariogenic. The one major remaining area where much is still to be learned is in the complex microbiology that occurs in the dental plaque or so-called biofilm on the surface of the tooth. The role of fermentable carbohydrates in foods and beverages is well-understood; and it is known that sucrose, glucose, fructose, and cooked starch all contribute to the caries process.\textsuperscript{1}

Demineralization, i.e., loss of mineral from the tooth, is initiated by the cariogenic (acidogenic) bacteria that produce organic acids during their metabolism. These acids include lactic, acetic, propionic, and formic. All of these acids can readily diffuse into the tooth and dissolve the susceptible mineral. The dental mineral dissolves to produce calcium and phosphate into the aqueous solution between the crystals; and these ions diffuse out of the tooth leading to the formation of an initial carious lesion, which eventually can become a cavity if the process continues without reversal.\textsuperscript{10} The reversal of the process is remineralization (replacement of mineral), which occurs when the acid in the plaque is buffered by saliva allowing calcium and phosphate, primarily from the saliva, to flow back into the tooth and form new mineral on the partially dissolved subsurface crystal remnants.\textsuperscript{11} The new "veneer" on the surface of the crystal is much more resistant to subsequent acid attack, especially if it is formed in the presence of sufficient fluoride.\textsuperscript{1,12}

Fluoride Mechanism of Action

It is now known that the primary mechanisms of action of fluoride are topical, i.e. they work by fluoride being available at the surface of the tooth. These aspects were thoroughly reviewed and agreement reached at an international consensus conference in 1989 and have been summarized extensively in other review papers.\textsuperscript{13-15} In summary:

- Fluoride inhibits demineralization;
- Fluoride enhances remineralization; and
- Fluoride can inhibit plaque bacteria.

The Caries Balance

Based upon the above summary of our in-depth knowledge of the caries process, it is very useful and constructive clinically to consider caries in its progression or reversal as an ongoing and often changing balance between pathological factors and protective factors.\textsuperscript{1,16} As illustrated in \textbf{Figure 1}, if the pathological factors outweigh the
protective factors, then caries progresses. In the reverse situation, caries is arrested or even reversed. The pathological factors include the acidogenic bacteria, reduced salivary function, and the frequency of ingestion of fermentable carbohydrates. The protective factors include saliva and its numerous caries-protective components: the saliva flow; antibacterials, both intrinsic from saliva and extrinsic from other sources; and other factors that can enhance remineralization.

In most individuals, there are numerous acid challenges daily as fermentable carbohydrates are ingested and the struggle between the pathological factors and the protective factors takes place. First, as the acid is produced by the bacteria, mineral dissolves; and subsequently, as the saliva neutralizes the acid, mineral is replaced. Fluoride enhances this remineralization process when it comes from topical sources such as drinking water, food and beverages, toothpastes/dentifrices in general, mouth rinses, office-applied fluoride preparations, or from higher-concentration products including fluoride varnish (Figure 1).

The next question that we must ask is if fluoride is so effective why do millions of people still require treatment annually in the United States for dental decay. Simply, if the bacterial challenge is too high, the beneficial effects of fluoride can be overcome by the acid attack. In the case of high-caries-risk individuals, although fluoride helps to reduce the amount and severity of the decay, it cannot overcome the high bacterial challenge. Figure 2 illustrates the mean decayed, missing, and filled surfaces in three national surveys in the United States. A dramatic reduction in decay levels was observed between 1970 and 1990. Prior to 1970, dramatic reductions in decay were also observed due to the fluoridation of public water supplies. From 1970 onward, several factors were involved including the almost universal utilization of fluoride dentifrices.20

Unfortunately, since 1990 there has been a plateau in the reduction of caries. This means that to further reduce the prevalence of dental caries and to address those who continue to have high levels of dental caries, we must use other measures. A recently published survey on the dental health of California’s children from data that was accumulated in 1993 and 199421 illustrated that:

- 27 percent of preschoolers have untreated decay;
- 53 percent of 6- through 8-year-olds have untreated tooth decay;
- 50 percent to 75 percent of minority high school students need dental care;
- California’s children on average have twice the national average of untreated tooth decay.

In California, early childhood caries is a major issue.7 Essentially the process and etiology are the same as caries in older children and adults and involves the same bacteria. In the case of early childhood caries, the decay is rampant and requires aggressive and multiple intervention strategies to control it.
Caries Is a Transmissible Bacterial Infection

It has been known for many years that caries is a bacterial infection. Studies during the past 25 years clearly indicate that the bacteria involved are transmissible, and the transmission, especially of *S. mutans*, is reviewed thoroughly by Berkowitz. In practical dentistry in the United States, we pay little attention to this basic fact about caries. We treat the manifestations of the disease rather than treating the disease itself. It is obvious that the next steps that must be taken to control, if not eradicate, dental caries must focus on the bacteria. One startling fact, which is not considered in practical dentistry, is that placing restorations to fill the cavities has no measurable effect on the cariogenic bacterial loading in the remainder of the mouth. These earlier findings have been borne out by an ongoing study in which a group of adult subjects with initial frank cavitation has been receiving conventional dental care according to the current "standard of care." The levels of mutans streptococci and lactobacilli were measured at the beginning of the study and after restorations were completed, with the net result that mutans streptococci levels were not statistically significantly different after the completion of restorations unless antibacterial therapy by chlorhexidine was used. This means that for high-risk individuals with high levels of cariogenic bacteria, we must take steps to reduce the bacterial loading along with restorative work. There are several guiding principals that can be followed:

- Fluoride is effective only up to a point;
- A high bacterial challenge cannot be completely overcome by even high-concentration fluoride therapy;
- Placing restorations and conducting restorative work does not reduce the overall cariogenic bacterial loading in the mouth; and
- We need to break the chain of infection if caries is to be controlled in these individuals.

As described briefly above, caries involves multiple acidogenic species of bacteria, which means that a vaccine for one particular species will not necessarily have a beneficial effect on the overall caries status of the individual. We need to utilize antibacterial therapy to reduce the bacterial challenge and allow the protective factors in the above-described caries balance to take over. We need to find ways to break the chain of infection, for example, from mother to child. It is now well-established that mutans streptococci can be readily transferred from mother to child or caregiver to child or indeed from child to child or adult to adult. Antibacterial therapy with chlorhexidine is one immediate alternative, but this is effective for mutans streptococci and not for lactobacilli. New antibacterial therapy is needed. As reviewed by Den Besten and Berkowitz, it is possible that iodine therapy may be more effective than chlorhexidine. Recent studies have shown that xylitol has properties that inhibit the establishment of cariogenic bacteria, and this appears to be an excellent method of breaking the chain of infection.

Conservative Caries Management
Conservative caries management has the idealized outcome that more tooth structure is preserved and fewer teeth become affected by dental decay. There are a few guiding principles:

- Detect caries lesions early enough (see below) so that the early, noncavitated lesion can be reversed or at least arrested from progressing by chemical means rather than by "restoration" (placing fillings);

- Assess the individual risk of caries progression (see below);

- Use fluoride to enhance remineralization and/or reduce the bacterial challenge by the use of antibacterial therapy; and

- Use minimally invasive restorative procedures to conserve tooth structure.\textsuperscript{28,30}

The longevity of the tooth is much greater in the scenario of conservative caries management; many more teeth are preserved as caries free, and those that do require restorative work have much smaller restorations leading to much less fracture in the long term.

Early Caries Detection

Until recently, caries detection methods have been visual, tactile (with an explorer), and radiographic. Visual inspection can be quite effective when done by an experienced dentist, and new classifications are in use in Europe that could well be useful in the United States.\textsuperscript{31} However, this is obviously limited where the surface of the tooth is obscured and in occlusal surfaces, where "hidden lesions" may be missed. Radiographs as done with "bitewings" have long been useful for detecting interproximal lesions. The current standard of care is that if an enamel lesion, as detected by the radiograph, is not past the dentino-enamel junction, then it can be arrested or reversed by remineralization, whereas an opacity into the dentin requires clinical physical intervention (drilling and filling). This method is quite reliable for these lesions. However occlusal surfaces are very different. In this case, there is a large amount of surrounding sound enamel that absorbs the X-rays; and only an advanced lesion can be detected in this fashion with conventional bitewing radiographs. This is illustrated in Figure 3, where the lesion under the occlusal surface was a "hidden lesion" extending histologically to the pulp. It showed only as a faint line at the DEJ in the radiograph. We need methods that can detect occlusal lesions while they are still in the enamel and can be reversed or arrested by fluoride therapy and remineralization.

Recently, the FDA approved a device called the "Diagnodent," (KaVo, Ill.) which shines a red laser into the tooth via a specially designed handpiece and tip. The tip is applied to the occlusal pits and fissures individually. The red light readily penetrates the tooth; and if it interacts with a subsurface lesion that contains certain bacterial byproducts, fluorescence is produced. The fluorescent light comes back from the lesion into the handpiece, interacts with the detector, and is read out as a number and an audible signal if there is a lesion. This instrument is a good first step in providing the practitioner with a tool that can indicate whether there is a hidden lesion under the occlusal surface. Even better devices are expected to become available to detect early
enamel lesions in occlusal surfaces.

The future will see improvements in these and other techniques, and the tools for early detection will be available. The bottom line is that we must use early detection for the purposes of intervention, not to justify more drilling and filling.32

Caries Risk Assessment

Practical caries risk assessment and the consequent clinical actions were the basis for the conference that generated this issue of the Journal. The importance to the pediatric dentist is that such procedures will change the way care is delivered.33 Similarly, in a public health setting, it will change the way interventions are done and how money is spent on programs to help lower-income/high-risk populations. The basis of caries risk assessment as we are currently introducing it into the predoctoral dental teaching clinics at the University of California at San Francisco is the "caries balance" described above (Figure 1). Each of the pathological and protective factors must be assessed in coming to a judgment as to whether the patient is at risk of progression or initiation of dental caries. We have developed a form for the clinician with instructions on the back to guide in risk assessment. When the risk assessment is complete, this influences the treatment plan. If the patient is required to conduct home care, a second form with appropriate instructions highlighted is given to the patient with a prescription, if needed. The reverse side of this form has a lay-person one-page description of dental decay.

The guiding principle (risk factor) questions for this risk assessment tool were as follows:

- Is there existing or has there been new untreated cavities in the past two years?
- Has there been orthodontic appliances or removable partial dentures?
- Is there reduced salivary function as measured by stimulated saliva flow less than 0.7 ml/minute?
- Is there use of hyposalivatory medications?
- Is there frequent ingestion of fermentable carbohydrates (by questioning the patient)?
- Is current use of fluoride products inadequate? and
- Is there high caries bacterial challenge as measured by testing mutans streptococci and lactobacilli?

The number of yes answers to the above questions places the patient into one of three risk categories. If the answers to the first five questions are mostly no, then bacterial testing is not needed. If bacterial testing is needed, the Ivoclar (Amherst, NY) caries resistance test is used; and results are known in 48 hours. This forms the basis for future monitoring of the effectiveness of antibacterial therapy. If the person is at high
risk, this initiates:

- Bacterial testing;
- Fluoride therapy (fluoride office topical followed by higher concentration home use fluoride is used);
- Chlorhexidine therapy (0.12 percent chlorhexidine gluconate for two weeks daily every three months); and
- Regular recall to monitor lesion progress or arrestment and antibacterial therapy success.

In the future, we anticipate that improved chairside bacterial testing and improved antibacterials will be available. For example, it is theoretically possible to design antibacterials to target receptors on the cell wall of the cariogenic bacteria involved. With new advances, these will enhance the success of the principles proposed in this presentation toward the eradication of dental caries.

Acknowledgments

The contributions of many individuals to the work and ideas reviewed here are acknowledged with thanks. They are too numerous to mention specifically. The support of NIH grant RO1-DE 12455 for the ongoing study titled "caries management by risk assessment" is acknowledged. The Delta Dental Plan of California is thanked for support of pilot studies relevant to this work.

References


7. Den Besten PK, Berkowitz RJ, Early childhood caries: An overview with reference


To request a printed copy of this article, please contact: John D.B. Featherstone, MSc, PhD, Department of Preventive and Restorative Dental Sciences, University of California, San Francisco, 707 Parnassus Ave., Box 0758, San Francisco, CA 94143 or jdbf@itsa.ucsf.edu.

**Legends**
Figure 1. Schematic illustration of the "caries balance." Adapted from Featherstone.  

Figure 2. Mean decayed missing and filled surfaces (DMFS) in permanent teeth of U.S. children (age ranges as shown) in three national surveys.

Figure 3. The left panel shows a conventional bitewing radiograph with an interproximal lesion on the left and a shadow at the DEJ under the occlusal surface (arrows). The right panel shows a hemi-section of the same tooth illustrating the hidden lesion extending to the pulp under the occlusal surface (courtesy of
D.A. Young).