THE SCIENCE AND PRACTICE OF CARIES PREVENTION

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A B S T R A C T

Background and Overview. Dental caries is a bacterially based disease. When it progresses, acid produced by bacterial action on dietary fermentable carbohydrates diffuses into the tooth and dissolves the carbonated hydroxyapatite mineral—a process called demineralization. Pathological factors including acidogenic bacteria (mutans streptococci and lactobacilli), salivary dysfunction, and dietary carbohydrates are related to caries progression. Protective factors—which include salivary calcium, phosphate and proteins, salivary flow, fluoride in saliva, and antibacterial components or agents—can balance, prevent or reverse dental caries.

Conclusions. Caries progression or reversal is determined by the balance between protective and pathological factors. Fluoride, the key agent in battling caries, works primarily via topical mechanisms: inhibition of demineralization, enhancement of remineralization and inhibition of bacterial enzymes.

Clinical Implications. Fluoride in drinking water and in fluoride-containing products reduces caries via these topical mechanisms. Antibacterial therapy must be used to combat a high bacterial challenge. For practical caries management and prevention or reversal of dental caries, the sum of the preventive factors must outweigh the pathological factors.

Although the prevalence of dental caries in children has declined markedly over the last 20 years in most countries in the Western world, the disease continues to be a major problem for both adults and children everywhere.

The trends in caries in U.S. children during the last 30 years were recently summarized[1] on the basis of results of four national surveys.[2-5] By the late 1980s, although approximately 75 percent of children aged 5 to 11 years were caries-free, about 70 percent of the 12- to 17-year-olds still had caries. Approximately 25 percent of children and adolescents in the 5- to 17-year age range accounted for 80 percent of the caries in permanent teeth. By age 17 years, however, 40 percent of the population accounted for 80 percent of the caries.[1-6] These findings illustrate the need for management of caries by individual risk assessment and for measures more specifically directed to high-risk people and populations.

Although these prevalence rates still leave much to be desired, the overall caries prevalence in children has indeed declined in the United States. Smaller epidemiologic studies in recent years indicate, however, that the decline in caries has not continued during the 1990s and that it may have plateaued.[6]

The reasons for the reductions in caries prevalence during the last 20 years are difficult to pinpoint. Strong evidence exists, however, that the near universal use of fluoride-containing products such as dentifrice, mouthrinses and topical gels applied in the dental office have been major contributors.[7,8] Earlier caries reductions of 40 to 70 percent (before the 1970s) had resulted from the fluoridation of public water supplies in many communities.[9-12]

Dental caries in adults also continues to be a major problem, as illustrated by a recent U.S. survey.[13] The survey reported that 94 percent of all dentate adults (aged 18 years or older) had evidence of treated or untreated coronal caries.

Caries obviously still is a major problem in adults, as well as children, and we need an improved approach to prevention and therapy. This article reviews and summarizes the scientific basis for and practice of successful intervention in the caries process.

THE CARIES PROCESS

Bacterial plaque and acid production. The caries process is now well-understood; much of it has been described extensively in the dental literature. Some details of the caries process remain to be unraveled, but, in general, we understand the process well enough to initiate better-targeted methods of caries prevention and intervention.

The mechanism of dental caries formation is essentially straightforward.[1] Plaque on the surface of the tooth consists of a bacterial film that produces acids as a byproduct of its metabolism.[14,15] To be specific, certain bacteria within the

plaque are acidogenic—that is, they produce acids when they metabolize fermentable carbohydrates.[12,14,15] These acids can dissolve the calcium phosphate mineral of the tooth enamel or dentin in a process known as demineralization.[16-18] If this process is not halted or reversed via remineralization—the redeposition of mineral via saliva—it eventually becomes a frank cavity.

Dental caries of the enamel typically is first observed clinically as a so-called "white-spot lesion." This is a small area of subsurface demineralization beneath the dental plaque. The

> **The mutans streptococci and the lactobacilli, either separately or together, are the primary causative agents of dental caries.**

body of the subsurface lesion may have lost as much as 50 percent of its original mineral content and often is covered by an "apparently intact surface layer."[19] The surface layer forms by remineralization. The process of demineralization continues each time there is carbohydrate taken into the mouth that is metabolized by the bacteria. The saliva has numerous roles, including buffering (neutralizing) the acid and remineralization by providing minerals that can replace those dissolved from the tooth during demineralization.[1,20,21]

Any fermentable carbohydrate (such as glucose, sucrose, fructose or cooked starch) can be metabolized by the acidogenic bacteria and create the aforementioned organic acids as byproducts.[22] The acids diffuse through the plaque and into the porous subsurface enamel (or dentin, if exposed), dissociating to produce hydrogen ions as they travel.[17,23] The hydrogen ions readily dissolve the mineral, freeing calcium and phosphate into solution, which can diffuse out of the tooth. Most importantly, lactic acid dissociates more readily than the other acids, producing hydrogen ions that rapidly lower the pH in the plaque.[17] As the pH is lowered, acids diffuse rapidly into the underlying enamel or dentin.

The two most important groups of bacteria that predominantly produce lactic acid are the mutans streptococci and the lactobacilli.[14] Each group contains several species, each of which is cariogenic. Mutans streptococci include *Streptococcus mutans* and *S. sobrinus*. The lactobacilli species also are prolific producers of lactic acid and appear in plaque before caries is clinically observed.[24,25] These two groups of bacteria, either separately or together, are the primary causative agents of dental caries.

HOW FLUORIDE COMBATS THE CARIES PROCESS

The ability of fluoride to prevent and arrest caries has been researched extensively. Fluoride has three principal topical mechanisms of action:
■ inhibiting bacterial metabolism after diffusing into the

bacteria as the hydrogen fluoride, or HF, molecule when the plaque is acidified;

▬ inhibiting demineralization when fluoride is present at the crystal surfaces during an acid challenge;

▬ enhancing remineralization and thereby forming a low-solubility veneer similar to the acid-resistant mineral fluorapatite, or FAP, on the remineralized crystals.

Inhibiting bacterial metabolism. Several investigators have studied the possible effects of fluoride on oral bacteria.[26-28] The most significant finding reported is that the ionized form of fluoride, or F^-, cannot cross the cell wall and membrane but can rapidly travel into the cariogenic bacterial cells in the unchanged form as HF.[26-28]

When the pH in the plaque falls as the bacteria produce acids, a portion of the fluoride present in the plaque fluid then combines with hydrogen ions to form HF and rapidly diffuses into the cell, effectively drawing more HF from the outside.[1,26-28] Once inside the cell, the HF dissociates, acidifying the cell and releasing fluoride ions that interfere with enzyme activity in the bacterium. For example, fluoride inhibits enolase, an enzyme necessary for the bacteria to metabolize carbohydrates. As fluoride is trapped in the cell, the process becomes cumulative.

In summary, fluoride from topical sources is converted partially to HF by the acid that the bacteria produce and diffuses into the cell, thereby inhibiting essential enzyme activity.

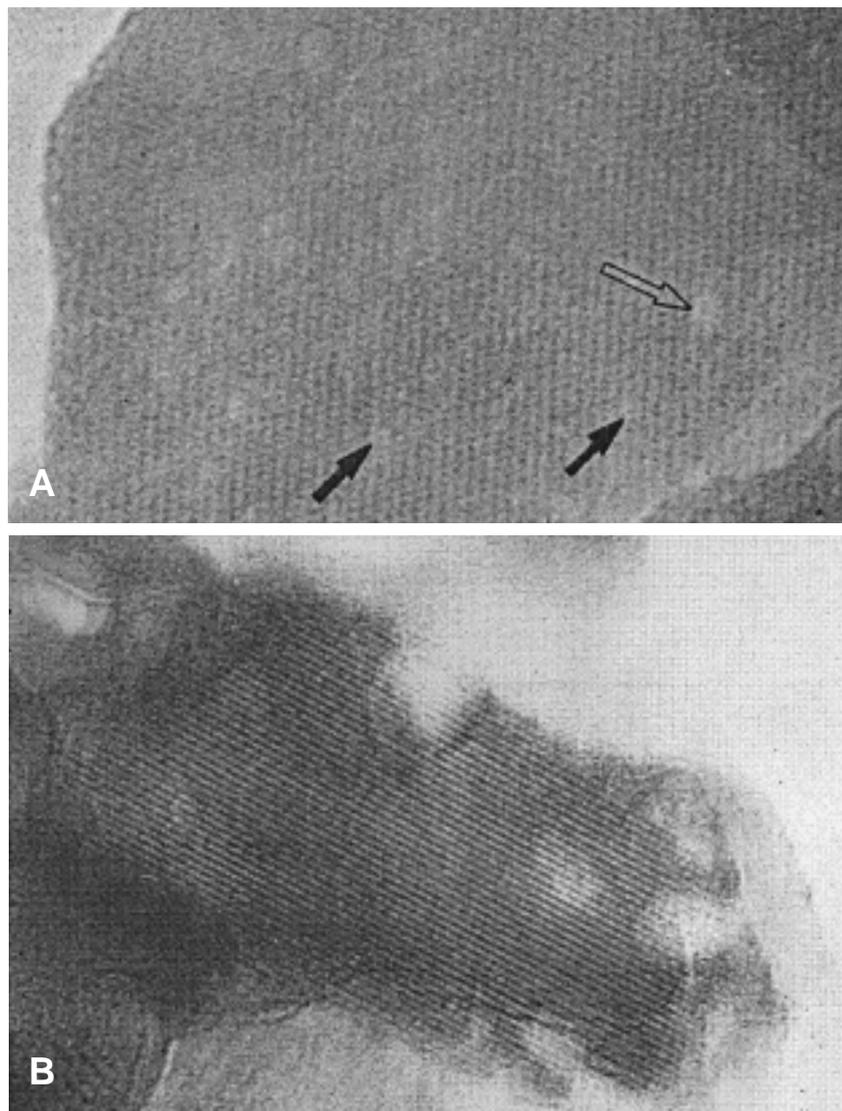**Inhibiting demineralization.** The mineral of our teeth (enamel, cementum, dentin)

Figure 1. High-resolution electron microscope images (magnification approximately ¥2,000,000) of individual enamel crystals. The black lines are rows of calcium atoms, which are visualized by this technique. A. Normal enamel crystal showing white patches (arrows), which are calcium-deficient and carbonate-rich defect regions. B. Demineralized crystal from the body of a natural caries lesion showing "large" hexagonal holes coinciding with the "small" defect regions seen in normal enamel. (Adapted from Featherstone and colleagues[30,31] with permission from Karger, Basel.)

and bones is a carbonated hydroxyapatite[29] that can be approximately represented by this simplified formula:

$$Ca_{10-x}(Na)_x(PO_4)_{6-y}(CO_3)_z(OH)_{2-u}(F)_u$$
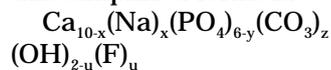
The substitutions in the hydroxyapatite crystal lattice (the arrangement of atoms and ions in the crystal) occur as the mineral is first laid down during tooth development, with the carbonate (CO_3) ion in particular causing major disturbances in the regular array of ions in the crystal lattice.[30,31] During demineralization, the carbonate is lost, and during remineralization it is excluded from the newly formed mineral. The calcium-deficient, carbonate-rich regions of the crystal are espe-
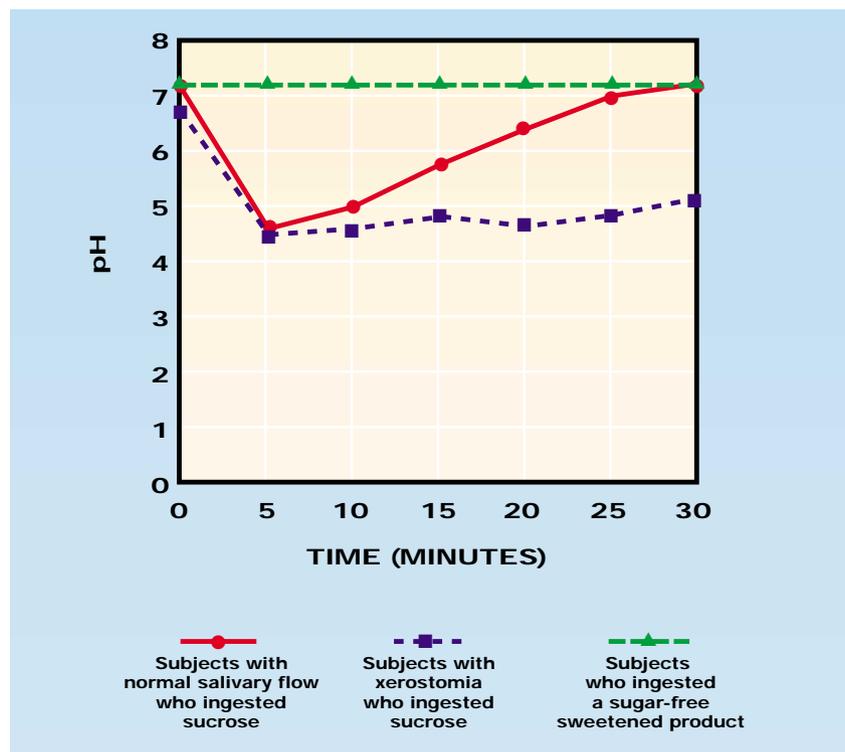
Figure 2. Typical pH curves for normal subjects with normal salivary flow and for subjects with xerostomia (mean for each group) after ingestion of sucrose. A curve for ingestion of a sugar-free sweetened product is shown for comparison. (Reproduced from Featherstone[1] with permission of the publisher. Copyright ©1999, Munksgaard.)

cially susceptible to attack by the acid hydrogen ions during demineralization, as has been shown by several investigators.[21,29-33] High-resolution lattice imaging, which images crystals almost to atomic resolution (viewed at about ×2,000,000 magnification), was used to illustrate the appearance of hexagonal holes in the early stages of enamel crystal dissolution in dental caries (Figure 1), which coincided with the calcium-deficient, carbonate-substituted regions of the crystal.[30-33]

The carbonated hydroxyapatite, or CAP, of our teeth is much more soluble in acid than hydroxyapatite, or HAP (HAP = $Ca_{10}(PO_4)_6(OH)_2$), and that in turn is much more soluble than fluorapatite, or FAP (FAP = $Ca_{10}(PO_4)_6F_2$),[21] in which

the OH^- ion in pure hydroxyapatite is completely replaced by an F^- ion. The resulting mineral FAP is highly resistant to dissolution by acid.

Fluoride inhibits demineralization. Sound enamel, except in its outer few micrometers, generally contains fluoride at levels of about 20 to 100 parts per million, or ppm, depending on the fluoride ingestion during tooth development.[34] Teeth in children who lived in areas with fluoridated drinking water during tooth development have fluoride content toward the higher end of this range. The outer few micrometers of enamel can have fluoride levels of 1,000 to 2,000 ppm.[34]

Fluoride in the solution surrounding CAP crystals has been shown to be much more effective in inhibiting demineraliza-

tion than fluoride incorporated into the crystals at the levels found in enamel.[21,35] Ten Cate,[21] Nelson and colleagues[35] and Featherstone and colleagues[36,37] found no measurable reduction in the acid solubility of synthetic CAP (3 percent CO_3 by weight, comparable to that of dental enamel mineral) with about 1,000 ppm fluoride incorporated. Importantly, this means that fluoride incorporated during tooth mineral development at normal levels of 20 to 100 ppm (even in areas that have fluoridated drinking water or with the use of fluoride supplements) does not measurably alter the acid solubility of the mineral. Even when the outer enamel has higher fluoride levels, such as 1,000 ppm, it does not measurably withstand acid-induced dissolution any better than enamel with lower levels of fluoride. Only when fluoride is concentrated into a new crystal surface during remineralization is it sufficient to beneficially alter enamel solubility. The fluoride incorporated developmentally—that is, systemically into the normal tooth mineral—is insufficient to have a measurable effect on acid solubility.[21,38]

In contrast to the lack of effect of fluoride incorporated into the CAP crystals of tooth mineral developmentally, as little as 1 ppm in the acid solution reduced the dissolution rate of CAP to a rate equivalent to that of HAP.[36] Further increases in fluoride in the acid solution in contact with the CAP mineral surface decreased the solubility rate logarithmically. These results indicate that if fluoride is present in the aqueous solution surrounding the crystals, it is adsorbed strongly to the surface of CAP carbonat-

ed apatite (enamel mineral) crystals and thus acts as a potent protection mechanism against acid dissolution of the crystal surface in the tooth's subsurface region. If fluoride is in the plaque fluid at the time that the bacteria generate acid, it will travel with the acid into the subsurface of the tooth and, therefore, adsorb to the crystal surface and protect it against being dissolved.

In summary, fluoride present in the water phase at low levels among the enamel or dentin crystals adsorbs to these crystal surfaces and can markedly inhibit dissolution of tooth mineral by acid.[21,36] Fluoride that acts in this way comes from the plaque fluid via topical sources such as drinking water and fluoride products. Fluoride incorporated during tooth development is insufficient to play a significant role in caries protection. Fluoride is needed regularly throughout life to protect teeth against caries.

Enhancing remineralization. As the saliva flows over the plaque and its components neutralize the acid, raising the pH (Figure 2), demineralization is stopped and reversed. The saliva is supersaturated with calcium and phosphate, which can drive mineral back into the tooth.[21,39] The partially demineralized crystal surfaces within the lesion act as "nucleators," and new surfaces grow on the crystals (Figure 3). These processes constitute remineralization—the replacement of mineral in the partially demineralized regions of the carious lesion of enamel or dentin (including the tooth root).[20,21] Fluoride enhances remineralization by adsorbing to the crystal surface and attracting calci-
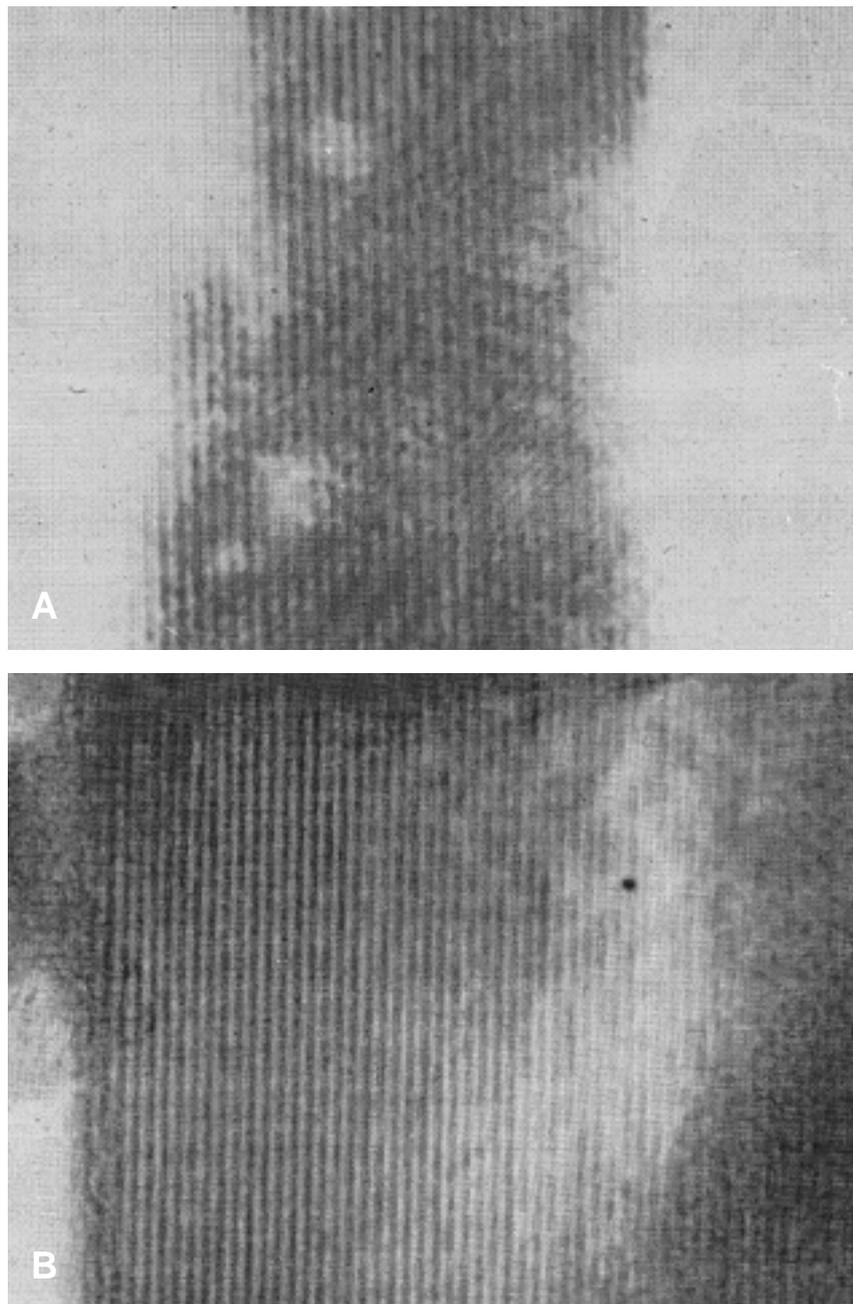

Figure 3. High-resolution electron microscope images (magnification approximately ×2,000,000) of individual enamel crystals that visualize remineralization at the atomic level. The black lines are rows of calcium atoms, which are visualized by this technique. A. Normal enamel crystal dissected from the inner region of enamel, showing "small" white patches of calcium-deficient, carbonate-rich regions. B. Crystal on which a "remineralized" surface veneer has been grown after treatment with fluoride, calcium and phosphate. (Adapted from Featherstone and colleagues, 1981[30] with permission from Karger, Basel.)

um ions, followed by phosphate ions, leading to new mineral formation. The newly formed "veneer" excludes carbonate and has a composition some-

where between HAP and FAP as described above (Figure 4). FAP contains approximately 30,000 ppm F and has a very low solubility in acid. The new
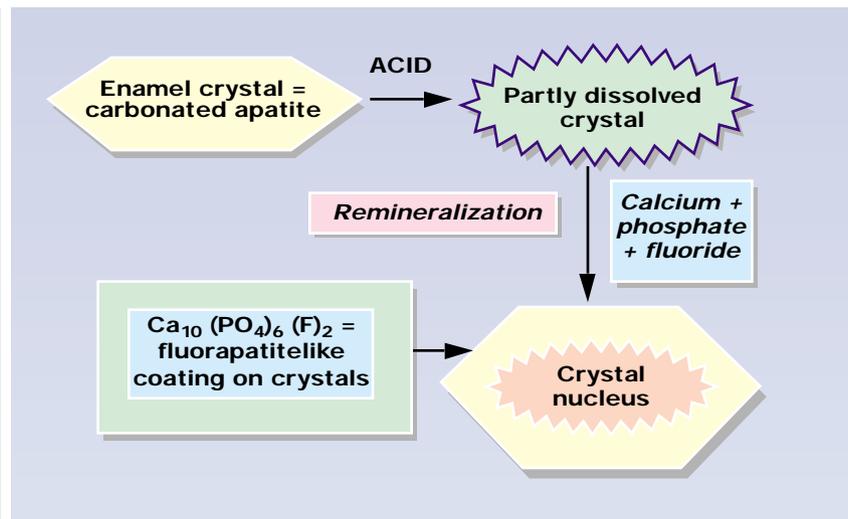
Figure 4. Schematic representation of demineralization followed by remineralization in the caries process. If remineralization is successful, the final result is a crystal with a surface veneer of "fluorapatitelike" mineral of low solubility. (Reproduced from Featherstone[1] with permission of the publisher. Copyright ©1999, Munksgaard.)
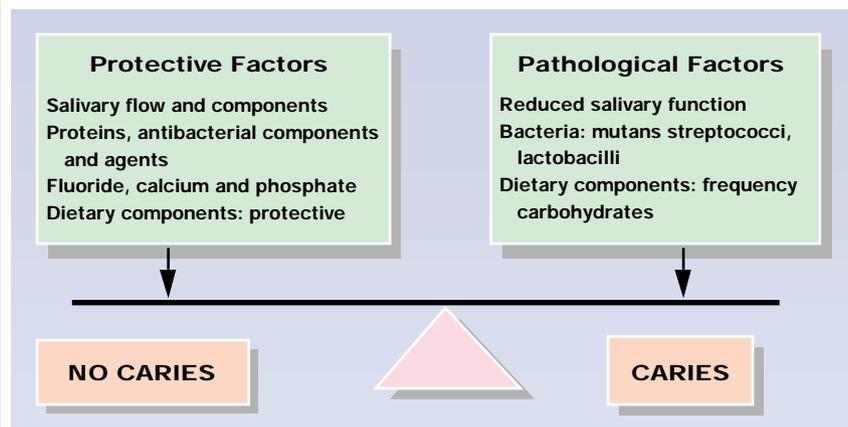

Figure 5. The caries balance: a schematic diagram of the balance between pathological and protective factors in the caries process. (Reproduced from Featherstone[1] with permission of the publisher. Copyright ©1999, Munksgaard.)

remineralized crystal now will behave like low-solubility FAP rather than the highly soluble CAP of the original crystal surface.[36]

In summary, fluoride in solution from topical sources enhances remineralization by speeding up the growth of a new surface on the partially demineralized subsurface crystals in the caries lesion. The new crystal surface veneer is FAP-like, with much lower sol-ubility than the original CAP tooth mineral. Subsequent acid challenges must be quite strong and prolonged to dissolve the remineralized enamel.

Saliva and caries. Saliva has a critical role in the prevention or reversal of the caries process; it provides calcium, phosphate, proteins that maintain super-saturation of calcium in the plaque fluid, proteins and lipids that form a protective pellicle on the surface of the tooth, anti-bacterial substances and buffers.[40] The saliva components neutralize the acids produced by bacterial metabolism in the plaque, raise the pH and reverse the diffusion gradient for calcium and phosphate. Thereby, they return calcium and phosphate to the subsurface lesion, where these ions can regrow new surfaces on the crystal remnants that were produced by demineralization. These so-called "remineralized" crystals have a veneer of much less soluble mineral. Saliva also clears carbohydrates and acids from the plaque.

In the case of salivary dysfunction,[41] all of the above benefits of saliva are reduced or eliminated (as is illustrated partially in Figure 2 by the pH profile of the subjects with xerostomia).

THE CARIES BALANCE

Fluoride's three extensively studied and documented principal mechanisms of action rely on the presence of fluoride in saliva, in the plaque at the tooth surface and in the fluid among the crystals in the subsurface of the enamel or dentin. The clinical effects of fluoride, therefore, can be optimized by using delivery methods that bring fluoride to the surface of the tooth and into the plaque rather than incorporating fluoride into the tooth mineral crystals during tooth development. These topical delivery methods are equally applicable to adults and children and include fluoride in beverages and foods, dental products and drinking water. The benefits of continually providing low levels of fluoride in the saliva and plaque from the aforementioned topical sources are described more fully

in a recent review article.[1]

Pathological and protective factors in the caries balance. Caries progression, as opposed to reversal, consists of a delicate balance between the aforementioned factors—namely, a bacterially generated acid challenge and a combination of demineralization inhibition and reversal by remineralization.[1,42] The balance between pathological factors (such as bacteria and carbohydrates) and protective factors (such as saliva, calcium, phosphate and fluoride) is a delicate one that swings either way several times daily in most people (Figure 5).

Protective factors. Saliva is essential for the protection of the tooth against dental caries and provides many natural protective factors summarized earlier,[40,41] including calcium, phosphate, antibacterial components and other proteins with various functions. Extrinsic antibacterial agents such as chlorhexidine also can be considered as protective factors in this balance, as can fluoride from external sources. The mechanisms of action of fluoride described in this article apply primarily to fluoride from topical sources; systemically incorporated fluoride has only a minor role in protecting against dental caries. This conclusion is supported not only by laboratory data as described previously, but also by epidemiologic studies. For example, a four-year study in England found a 27 percent lower caries incidence among children who were 12 years old when water fluoridation began in their communities, relative to the incidence in control subjects of the same age in nonfluoridated areas.[43] This was a well-conducted study, and it clearly

showed the posteruptive (topical) effects of fluoride in the drinking water. Other studies have illustrated the weak pre-eruptive effects of fluoride. For example, in two groups of Okinawa nursing students aged 18 to 22 years, there was no difference in caries status between those who had received fluoridated water only until about 5 to 8 years of age (and none thereafter) and those who had never received fluoridated drinking water.[44]

The cariostatic effects of fluoride are, in part, related to the sustained presence of low concentrations of ionic fluoride in the oral environment.[1,21,38]

> There is the mistaken belief that drilling out a caries lesion and placing a restoration eliminates the bacteria and thereby stops caries progression.

derived from foods and beverages, drinking water and fluoride-containing dental products such as toothpaste. Prolonged and slightly elevated low concentrations of fluoride in the saliva and plaque fluid decrease the rate of enamel demineralization and enhance the rate of remineralization.[21,36,38,45-48] For example, fluoride at 0.04 ppm in saliva can enhance remineralization. Remineralization of early lesions also requires calci-

um and phosphate, which are derived primarily from saliva and plaque fluid.

Pathological factors. Pathological factors obviously include cariogenic bacteria and the frequency of ingestion of fermentable carbohydrates that sustain these bacteria. The importance of mutans streptococci (which includes *S. mutans* and *S. sobrinus*) in the development of dental caries has been reviewed extensively.[12,14,15,49,50] Numerous cross-sectional studies in humans have shown that greater numbers of mutans streptococci and lactobacilli in saliva or plaque are associated with high caries rates.[15,25,49,51-54] Longitudinal studies have shown that an increase over time in numbers of both of these bacterial groups is associated with caries onset and progression.[24,55,56]

CARIES INTERVENTION

The methods of caries intervention can be summarized by joining the principal components of the caries process with the interventional possibilities (Table).

Cariogenic bacteria and high bacterial challenge. Dental caries is a transmissible, bacterially generated disease. There is the mistaken belief that drilling out a caries lesion and placing a restoration eliminates the bacteria and thereby stops caries progression. Although traditional restorative work may eliminate the bacteria at the site of the restoration, the remainder of the mouth is left untouched, caries continues unchecked in the remainder of the mouth and recolonization commences rapidly at the margins.[57]

It is logical, therefore, to use

TABLE

SUMMARY: THE CARIES PROCESS AND METHODS OF CARIES INTERVENTION.

CARIES COMPONENT	INTERVENTION METHOD
Bacteria	Antibacterial therapy such as treatment with chlorhexidine gluconate (see text)
Carbonated Hydroxyapatite	Make the mineral less soluble by transforming it to other crystalline forms such as hydroxyapatite without carbonate (future caries-preventive treatments by specific laser irradiation will enable this to be done[69,70])
Fermentable Carbohydrates	Reduce the frequency of ingestion; substitute with noncariogenic sweeteners (this method is well-accepted and used in patient education) Recommend use of sugar-free chewing gum, which reduces frequency of fermentable carbohydrate ingestion and also enhances remineralization
Organic Acids Produced by Oral Bacteria	Neutralize the acid by providing extra buffering or enhancing saliva; sugar-free gum assists in this as well
Saliva	Enhance the saliva flow and function
Fluoride	Exploit its known effects on bacteria, inhibition of demineralization and enhancement of remineralization by using "topical" fluoride delivery by means of dental products, drinking water, beverages and foods

antibacterial therapy—such as treatment with chlorhexidine gluconate rinse—as a caries-preventive measure. Although this has been proposed for many years[58-60] and used in several European countries, an antibacterial approach almost never is used in the United States for the prevention of the progression of dental caries.

One of the difficulties in persuading clinicians to use the antibacterial approach is that there have not been rapid and accurate methods of determining the levels of cariogenic bacteria in the mouth. Furthermore, although numerous studies have indicated that mutans streptococci and lactobacilli definitely are risk factors for dental caries, there is no one-to-one direct correlation between levels of these bacteria and caries progression.[24,49] However, it now is well-established that high levels of mutans streptococci, high levels of lactobacilli or both constitute a "high bacterial challenge."[24] This bacterial challenge can be balanced by the protective factors described earlier, which include salivary components—especially calcium, phosphate and fluoride—and the amount of saliva present.[42]

Figure 5 illustrates the balance between pathological factors (including cariogenic bacteria, reduced salivary function and frequency of use of fermentable carbohydrates) and protective factors. If these pathological and protective factors are in balance, caries does not progress. If they are out of balance, caries either progresses or reverses.

Antibacterial therapy for caries control. Currently, the most successful antibacterial therapy against cariogenic bacteria is treatment by chlorhexidine gluconate rinse or gel.[47,61] Chlorhexidine is available by prescription in the United States. Studies have shown that a daily dose of chlorhexidine rinse for two weeks can markedly reduce the cariogenic bacteria in the mouth and that, as a result, recolonization takes place in three to six months rather than immediately.[58] In patients with high levels of bacteria, therefore, chlorhexidine treatments at three-month

intervals are indicated.

The problem faced by clinicians is how to determine, in a timely fashion, whether the bacterial challenge is high, medium or low. For many years, commercial "dip slides" have been available in Europe, and they recently became available in the United States.[58] A saliva sample is taken from the patient and incubated on the dip slide; two days later, a result is provided of the levels of *S. mutans* and lactobacilli bacteria in the mouth.[58] Although these slides are a major advance in convenience and are the best tools available at the time of this writing, it has been shown that this technology is not well-correlated with traditional bacterial plating. It is anticipated that methods of rapid chairside assessment of bacterial challenge, based on molecular biology, will be available in the future.

Several investigators have explored the possibility of using modern molecular biology for better and more rapid methods of bacterial assessment,[62] but they were unable to overcome a number of complications. An exciting development is work by Shi and colleagues,[63] who recently published a method using species-specific monoclonal antibodies that recognize the surface of cariogenic bacteria. With this technology, it is not necessary to split open the bacterial cells to assess the internal DNA or RNA. These probes can be tagged either with a fluorescent molecule or with a marker that can be measured quantitatively in a simple spectrophotometer.

It is anticipated that these probes will be available commercially in the near future, and that clinicians will be able to use them chairside and obtain results within a few minutes. This will enable clinicians to determine the quantitative levels of bacteria in a patient's mouth while he or she is in the operatory and to factor these numbers into an overall risk assessment of caries for that patient. It is envisaged that computer programs will be available that will include the assay numbers, as well as other data. The practitioner will receive guidance as to the level of

> **Methods of rapid chairside assessment of bacterial challenge, based on molecular biology, will be available in the future.**

caries risk and what regimen or regimens to use to prevent further caries and to reduce the bacterial challenge. With the new monoclonal antibody probes, the levels of bacteria and success of the intervention could readily be followed over time. This is an exciting, innovative tool that may become widely used and accepted within a few years.

CARIES RISK ASSESSMENT

Several studies have attempted to determine risk factors that can be reliably used to assess the level of risk of caries progression in individual patients. Studies still are under way, and there is no definitive formula yet available. The status of risk assessment was summarized, however, by the authors of a special supplement to The Journal of the American Dental Association in 1995; this publication can be used as a guide until more definitive information is available.[64] Figure 5 represents a basis for determining caries risk with the information currently available.

It has been established that high-risk patients include those who have a high bacterial challenge, which may consist of a combination of high numbers of mutans streptococci, lactobacilli or both. Although fluoride has excellent properties in terms of balancing caries challenge, if the challenge is too high, then fluoride—even at increased concentrations, with increased use or both—cannot balance that challenge. Therefore, in the case of high bacterial challenge, the bacterial infection must be dealt with, typically with a chlorhexidine rinse, as well as the enhancement of salivary action by topical delivery of fluoride. These principles apply equally well to adults and children. Accurate detection of early caries can increase the reliability of caries risk assessment, particularly if those measurements are made at three- or six-month intervals and caries progression can be measured. In the case of caries progression, obviously, intervention is needed either antibacterially, with fluoride or
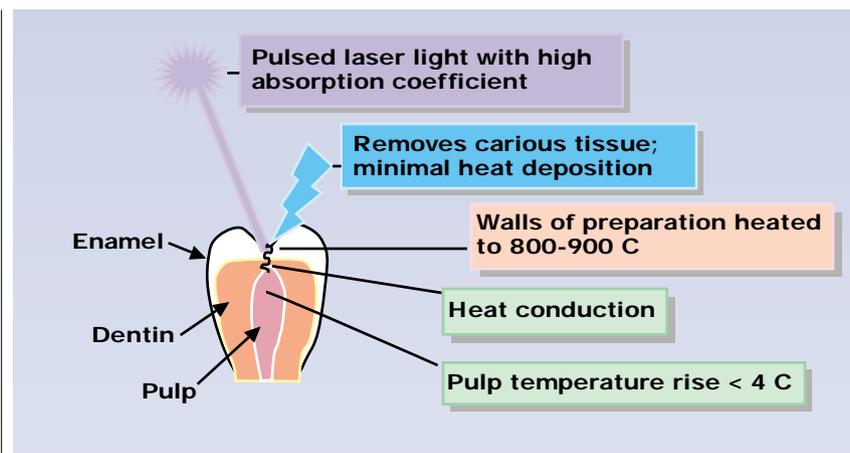
Figure 6. Schematic diagram showing the potential use of specific lasers for precise removal of carious enamel and modification of the surrounding enamel for prevention of further caries progression after restoration. The laser would be set first to remove a minimum of carious tissue. Then the walls and base of the cavity preparation would be treated with the laser to inhibit subsequent caries progression. (Reproduced from Featherstone[71] with the permission of the publisher. Copyright © 2000 Indiana University School of Dentistry.)

with other techniques, some of which are described in the following material.

Caries management by risk assessment. As the caries risk assessment methodologies are refined, we will have more definitive biological and chemical risk assessment measures to guide clinical decision making. These measures form the basis for assessing the direction in which the caries balance is likely to move for a particular patient. Early caries detection, especially in occlusal surfaces, is an essential part of caries management by risk assessment.

Caries management by risk assessment now is receiving considerable attention, and software programs are being developed that will aid practitioners in assessing risk and lead them to the use of current and new technologies by specifying treatments recommended for the various risk categories.[59,60] As we move into the future, tooth restorations will become less and less desirable as a treatment and will be used only as a final resort when new intervention measures have failed or when people have not participated in caries intervention programs such as those indicated previously.

CARIES MANAGEMENT TOOLS FOR THE FUTURE

Several technological advancements are currently close to clinical reality and will be embraced if they are proven successful.

Assessment of bacterial challenge by chairside molecular probes. The use of chairside bacterial probes for assessing a patient's cariogenic bacterial challenge will be an essential component of caries management by risk assessment.

Caries immunization. In a program of caries management by risk assessment, it is logical that all available tools should be used. One such tool that has been investigated for many years is an immunization against caries. There are many obstacles to the success of immunization, as caries is not a systemic infection that can be dealt with simply by administering a specific antibiotic. The infection must be dealt with in the mouth, where the internal body fluids do not pass and, therefore, the normal immune response is not relevant. However, IgA that is produced by the saliva naturally can interfere with the colonization of the surface of the tooth by specific bacteria.

Recent studies by Ma and colleagues[65,66] have illustrated the effectiveness of specific IgA in the inhibition of recolonization of mutans streptococci. The next logical step is to use this technology as one of the tools for caries intervention. It is possible to use genetically engineered plants, such as tobacco or alfalfa, to produce immunoglobulins.[66,67] A study is in progress at the University of California, San Francisco, to test IgA that has been produced using genetically engineered tobacco plants. At press time, the results were not known, but if the trial is successful, this IgA can be applied to the teeth after chlorhexidine treatment has removed the cariogenic bacteria, with the aim of inhibiting future recolonization by mutans streptococci.

Early caries detection and intervention. Successful use of the innovative methods described here for caries intervention will require accurate methods for the early detection of dental caries in enamel and dentin. Early-detection methods such as fluorescence, optical coherence tomography, electrical impedance and

ultrasonography are likely to become available for use by clinicians in the near future.[68] It will be possible to detect lesions in the occlusal surface and to determine whether they have progressed into the dentin and, if so, how far. This is not possible with current radiographic technology.

Once new methods are introduced for the early detection of caries, they can be used in two opposing fashions. Clinicians with traditional training are likely to use these methods to intervene physically at an earlier stage with carious lesions—drilling, filling and placing restorations. This outcome is of concern, as many more restorations would be placed than may be necessary, which weakens the tooth structure. Early detection and intervention by placing a restoration also does not take advantage of the body's natural protective mechanisms of inhibition of demineralization and enhancement of remineralization via saliva.

Alternatively, early detection of caries can be used as an opportunity to promote remineralization via salivary enhancement, use of topical fluoride and chlorhexidine and meticulous oral hygiene. In addition, as innovative methods for early caries intervention are introduced, the need for restorations may be eliminated for many patients, thereby preserving the tooth structure and halting or reversing progression of dental caries.

Caries prevention by laser treatment. In May 1997, the U.S. Food and Drug Administration approved the use of an erbium:yttrium-aluminum-garnet, or Er:YAG,

laser for use on teeth. This was the first approval for laser use on dental hard tissues. This approval by the FDA was for this particular laser to be used for the removal of dental caries and the cutting of sound tissue before the placement of restorations. This event has ushered in a new era for lasers in dentistry. Since then, other lasers have been approved for the same purpose, and additional hard-tissue uses are likely to be approved in the future, including the use of lasers for the inhibition of progression of dental caries by altering the composition of surface enamel

> **As innovative methods for early caries intervention are introduced, the need for restorations may be eliminated for many patients.**

or dentin mineral.

Kantorowitz and colleagues[69] and Featherstone and colleagues[70] have studied the effects of lasers on hard tissues for almost 20 years. The overall objective of these studies is to establish the scientific basis for the choice of laser parameters that can be used clinically for the prevention, removal or treatment of caries lesions. Their studies have demonstrated that specific pulsed carbon dioxide, or CO_2, laser treatment of dental enamel can inhibit subsequent carieslike progression in a severe de-

mineralization-remineralization model in the laboratory by up to 85 percent. They have demonstrated that carbonate is lost from the CAP mineral of the tooth during specific laser irradiation, making the mineral highly resistant to dissolution by acid. Although they have demonstrated in the laboratory, using pH cycling models, that as little as 20 pulses of 100 microseconds each can produce a preventive effect similar to daily use of fluoride dentifrice, these promising and exciting results have not yet been tested in human mouths.[70]

For practical purposes, it would be desirable to develop a laser that can remove carious tissue and subsequently be used to treat the walls of the area from which carious tissue is removed to make them resistant to subsequent caries challenge[71] (Figure 6). Fried and colleagues[72] recently published a report on a new CO_2 laser that efficiently removes carious tissue. After caries and a minimal amount of surrounding tissue are removed, it will be possible to change the laser parameters to perform caries-preventive treatment on the same area. This would be followed by placement of a resin-based composite restoration, thereby inhibiting subsequent caries around that restoration. For example, if an early occlusal lesion was detected (by the new methods described previously) that was deemed to be beyond hope of remineralization, this lesion could be conservatively removed with an appropriate laser. Then the surrounding cavity preparation walls could be treated for caries prevention by the laser and a small conservative restoration

placed. The cavity walls will be highly resistant to acid attack and therefore resistant to secondary caries. Providing bacterial intervention via chlorhexidine rinse was also part of the treatment in the same patient, future caries would be unlikely.

SUMMARY AND CONCLUSIONS

The mechanism of dental caries is well-established to the point where new approaches are being made for caries prevention based on a scientific understanding of the processes involved. Several existing methodologies are available to enable successful management of dental caries by risk assessment. Understanding the balance between pathological factors and protective factors is the key. Beyond the well-established and currently used methods, some innovative and exciting techniques have shown early research successes that most likely will be used for early caries intervention in the future. These methods include fluoride therapy for inhibition of demineralization and enhancement of remineralization, molecular probes for the quantitative detection of cariogenic bacteria at chairside, computerized caries risk assessment programs, genetically engineered IgA for inhibition of recolonization of cariogenic bacteria, specific lasers for conservative removal of carious tissue and specific lasers for the prevention of caries progression.

The use of these technologies will require extensive retraining of clinical dentists. But it will dramatically alter the way in which dentists diagnose, intervene, treat and manage caries, with major benefits to the oral health of their patients. ■

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