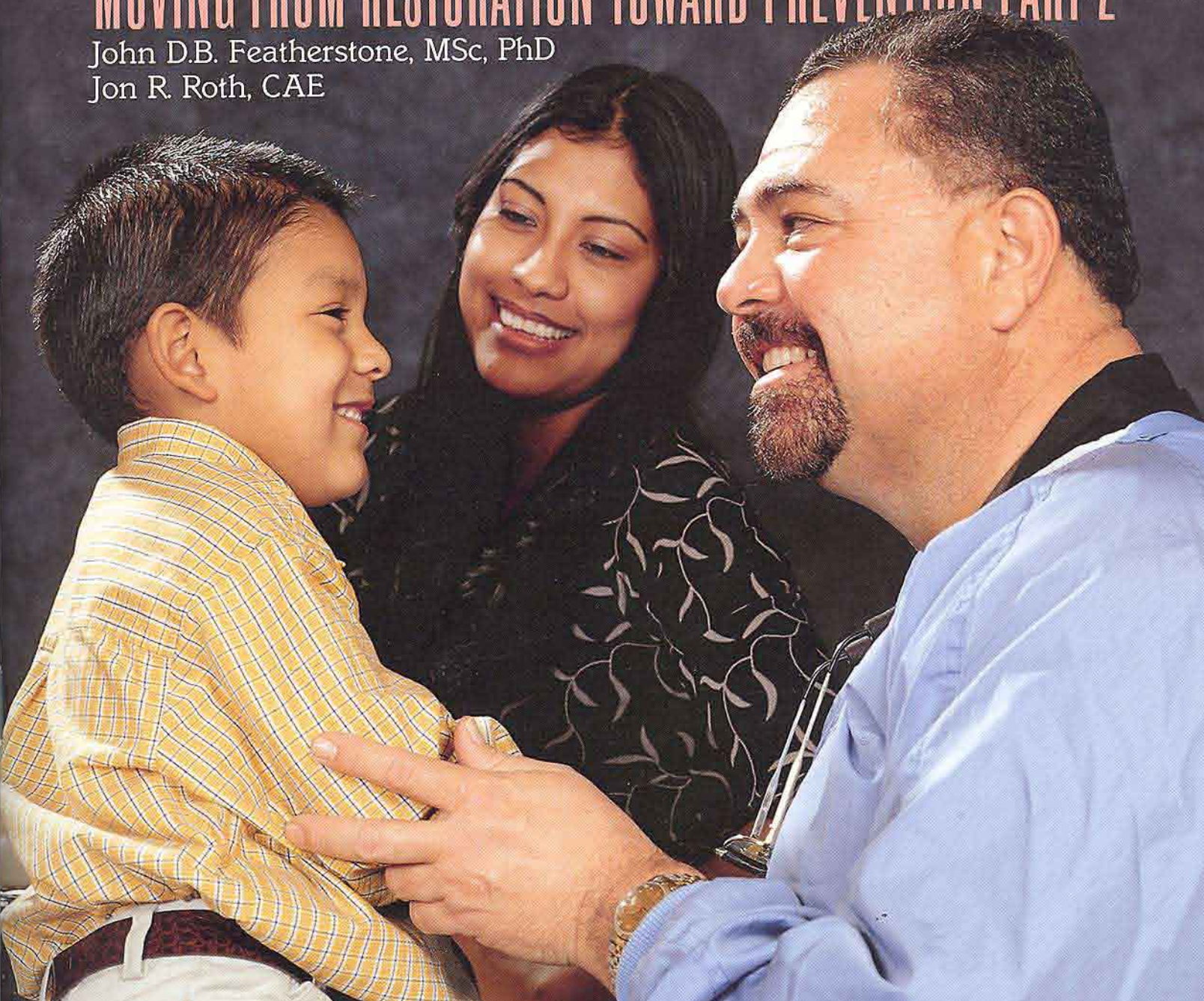
CDA
JOURNAL OF THE CALIFORNIA DENTAL ASSOCIATION VOL.31 NO.3
March 2003

Fluoride Varnishes
Risk Assessment
Xylitol

# CARIES
## MOVING FROM RESTORATION TOWARD PREVENTION PART 2

John D.B. Featherstone, MSc, PhD
Jon R. Roth, CAE



OF THE CALIFORNIA DENTAL ASSOCIATION
Journal
CDA Journal
Volume 31, Number 3
MARCH 2003

## DEPARTMENTS



## FEATURES

CDA JOURNAL, VOL 31, Nº3

# It Is a New Day, 2003

Jack F. Conley, DDS

Opinions and concerns about the California Dental Association and its direction and effectiveness, combined with copious amounts of inaccurate information and misinformation, recently graced the pages of the newsletter of one of CDA's component dental societies. It was one of the most ill-advised and divisive activities that we have observed in organized dentistry for quite some time.

In our opinion, there was nothing discussed in that extremely negative presentation for which the association or its leadership should be criticized. Our purpose in this space is not to defend the association either on a general or specific issue-by-issue basis, although we will comment on some association matters that are related to some of the concerns of the "critics" of our profession. The recent event was another illustration of a communication shortcoming that we have seen our profession suffer through from time to time, although it was typical of a communication difficulty that was actually more prevalent 10 to 20 years ago. It has been our impression that CDA staff and volunteer leadership, in recent years in particular, have made real progress in improving the communication of information to the membership at large. The ever-present communication problem tends to be lateral within the community of colleagues.

In discussing ill-advised criticism, let's start with our basic premise. It is a new day! Most critics seem to overlook the fact that the forces outside of the profession that seek or bring changes that affect the profession have increased significantly in recent years. Access-to-care issues and regulatory matters that directly influence work patterns in the dental office are taking aim at the profession at an increasing pace. Association critics seem to expect, if not demand, that the association win every legal, legislative, or political battle that it faces. If the resolution of the issue does not allow these colleagues to have it their way (i.e., have a regulatory matter decided in dentistry's favor the way it was five, 10, or 25 years ago), their attitude and criticism seems to suggests that the association has failed them and is unworthy of their continuing support.

As a good example of the world in the 21st century, let's take the Proposition 65 matter. Despite the fact that the positions of the opposition often seemed unreasonable to the dental community, the matter consumed many hours and resources of CDA in achieving a settlement that served to resolve the legal actions that were initiated against many of our members. In the perfect world of the CDA critics, I imagine that there was an expectation that CDA should have had these suits against our colleagues thrown out, and the need for dentists to conform to Proposition 65 regulations waived. But that is not the real world in 2003. After some two years of legal challenge, that matter has been finally settled. There were significant costs in time and resources, and there is a requirement that dental offices must meet. Critics and noncritics within our membership may not be happy or satisfied, but our organization achieved the best possible resolution of the matter. Critics need to consider what might have happened to each of us if we had to individually face the legal assaults and costs to meet these challenges if there were no

CDA to represent us, instead of criticizing the efforts or the result.

What about leadership? From our experience, any blanket criticism of staff or volunteer leadership is unfair. For 25 years, it has been my observation that commitment and the best interests of the dental practitioner have always been central to their efforts. In any group-generated discussion or decision, there is always the opportunity for dissent. But that is where leadership becomes even more important. A leader or colleague with access to the component leadership loop who holds a minority view and disagrees with a decision of the trustees or staff, should not violate the rules of fair play and responsibilities of leadership by directly communicating to others his or her view that CDA volunteers or staff made the wrong or an ineffective decision. Instead, he or she must learn to work with elected or appointed leadership to bring about a mutually acceptable resolution. Failure to do this usually results in the word-of-mouth communication of misinformation. Words laced with discontent often have become interpreted as words of fact. It is not long before a segment of the membership that does not have all of the real facts on an issue adopts the view that the association staff, elected officers, or Board of Trustees has failed to represent them in an appropriate manner.

As for top executive leadership, it is a new day, as Peter DuBois this month is commencing his tenure as executive director. The Executive Committee and Board of Trustees welcome him and anticipate growth and progress toward achievement of the association's strategic plan during his administration. Contrary to a belief of some critics, CDA, under the interim leadership of Robert Witt and the elected officers, has maintained a steady course in the past 10 months. Important issues facing dentistry in California have been confronted, and the various committees and councils of the association have continued to provide the appropriate program development, decisions, and policies for consideration by the officers and trustees. The result of this activity has been uninterrupted support for the many membership services provided to the membership.

Another misconception is that the CDA membership market share continues to decline, perhaps due to a notion that CDA is not providing the necessary value for the dues dollar. Analysis of membership statistics shows that in 2002, there was a net membership increase of 300 even after taking into account membership losses of several hundred due to deaths and nonrenewals. It demonstrates that there are component members who believe in the value and the benefits received from being a CDA member and are, therefore, recruiting new members.

While the total of the tripartite dues is not an insignificant amount, how many hours does it really take for the average CDA member to produce the revenue to support his or her annual membership in organized dentistry? We suspect that the average member spends considerably more on elective and recreational activities that are considerably less important than the efforts to preserve the privilege to practice dentistry that members receive as a membership benefit through their dues support of organized dentistry. Insurance, education, and other visible membership services aside, where would we be without a CDA or ADA to provide expert testimony before the Legislature or Congress? Would we be able to muster the same clout if all of the critics dropped their membership, leaving CDA with a less-than-robust percentage of active dentists? The answer, of course, is no. Our leaders and representatives need a strong membership base if we are to demonstrate the clout to external policy makers that will enable us to obtain decisions that are in the best interests of the public and the membership.

Those who would criticize and complain must learn to work with their leadership to confirm the accuracy of all of the facts on any issue they have concern with. They must learn to work within the process to bring about positive progress through leadership, rather than stimulate the negativism that divides and fosters resentment within the membership. If they do that, it will go a long way toward ensuring that the new day at CDA will fulfill the expectations of all members.

CDA JOURNAL, VOL 31, Nº3

## Dental Assistants Key to Successful Practice

They help keep you organized and on schedule. They can charm a wiggly 4-year-old or calm an anxious senior citizen. They take radiographs, educate patients, and help build your practice. They are your dental assistants.

As a way to acknowledge them as an essential part of the dental office team, the American Dental Association, American Dental Assistants Association, Canadian Dental Association, and the Canadian Dental Assistants Association have designated March 2-8 as Dental Assistants Recognition Week. An established tradition for several decades, this week-long event is the perfect time to acknowledge the versatility and talents of dental assistants.

"Full utilization of our skills within our scope of practice provides greater access to care for patients," said California Dental Assistants Association President Diane Owens. "As an invaluable member of the dental team, being considered a professional ranks high as we assist in delivering dental care to our patients."

## Retaining Staff

Once a great dental office team is in place, dentists should turn their focus toward retention. While financial compensation is an important factor in employee retention, recognition and achievement are also essential pieces of the puzzle.

Virginia Moore of Insight Solutions -- a team of professional speakers, consultants, and authors -- said that the feedback she receives from dental assistants is that appreciation and respect are more important than salary.

"One of the things that we hear means the most to dental staff is a word of appreciation specific to the situation," Moore said. "Also important is for them to be able to use their knowledge and skills. When a doctor can delegate with confidence to a dental team member, it is a win-win situation for everyone."

In ADA's Recruiting and Retaining Staff: A Guide for the Dental Office, the Council on Dental Practice highlights a number of factors that play a part in the satisfaction, or dissatisfaction of employees. Satisfaction factors include a sense of teamwork or camaraderie, job security, flexibility, appreciation for staff efforts, benefits, delegated autonomy in patient management, and subsidized continuing education. Factors that contribute to an employee's dissatisfaction include lack of career advancement, lack of respect, monotony of daily routine, legal restrictions on procedures, and limited potential for income growth.

According to the ADA's guide, ways of expressing appreciation on a daily basis, and especially during Dental Assistants Recognition Week, include unexpectedly bringing in lunch for everyone on a rainy day; posting an office bulletin board with each staff member's photo; giving out tickets to the circus, zoo or an amusement park; and planning group activities or outings.

As Dental Assistants Recognition Week illustrates, making dental team members feel appreciated greatly contributes to their overall job satisfaction. CDAA President-Elect Cindy Ramirez, CDA, RDA, explained that loyalty to the doctors, staff, and her professional organization is the main reason she has remained in the field for 26 years.

"Respect, responsibility, and recognition are the three big R's, and, in my opinion, what most dental assistants strive for in the perfect office," Ramirez said. "I appreciate the respect that is shown to me, the responsibilities that have been entrusted to me, and the recognition I receive from being a licensed professional."

## CDC Issues New Hand Hygiene Guideline

To improve adherence to hand hygiene in health care settings, the Centers for Disease Control and Prevention has finalized its new Hand Hygiene Guideline for all health care personnel.

In addition, in 2003 the CDC and the American Dental Association will be issuing revised recommendations for infec-

## A Recognized Shortage

Not only are dental assistants a valued part of the dental team, they are also in demand due to a recognized shortage in the allied dental health field. The 1999 ADA Workforce Needs Assessment Survey reflected that 60 percent of California dentists agree there is an inadequate supply of chairside dental assistants.

To address this shortage, CDA is implementing an allied dental health personnel action plan. According to statistical evidence gathered for the plan, the causes of the dental assistant shortage include insufficient number of graduates and a short career length. The plan comprises four elements: a marketing program, component and member-based activity, collaboration with allied dental health personnel organizations, and professional development. Through marketing and greater public awareness, the ultimate goal of the plan is to inspire and encourage additional, qualified individuals to pursue allied dental health careers.

"The plan addresses both recruitment and retention of dental team members," said CDA Allied Dental Health Coordinator Evelyn Ingalls. "The plan is exciting in that it will allow us to actively promote dental careers as viable career choices and hopefully attract more people into the field."

percent, so that the need for hand hygiene is clear.

In addition to regular hand washing with soap and water, the CDC recommends the use of alcohol-based hand rubs. This recommendation was made to address obstacles to frequent hand washing in health care settings.

"Clean hands are the single most important factor in preventing the spread of dangerous germs and antibiotic resistance in health care settings," says Julie Gerberding, MD, director of the CDC.

The guideline is available online at www.cdc.gov/handhygiene/ or by calling (404) 639-3286.

Communicating With People With Disabilities a Matter of Respect

From the Axis Center for Public Awareness of People with Disabilities in Columbus, Ohio, come these suggestions for communicating with people with disabilities. The items were published in the November 2002 ODA Today, a publication of the Ohio Dental Association.

When talking with a person with a disability, speak directly to that person rather than through a companion or sign language interpreter who may be present.

When introduced to a person with a disability, offer to shake hands. People with limited hand use or who wear an artificial limb can usually shake hands. Shaking hands with the left hand is acceptable.

When meeting a person with a visual impairment, always identify yourself and others who may be with you. When conversing in a group, remember to identify the person to whom you are speaking.

If you offer assistance, wait until the offer is accepted. Then listen to or ask for instructions.

Treat adults as adults. Address people who have disabilities by their first names only when extending that same familiarity to all others present. Never patronize people who use wheelchairs by patting them on the head or shoulder.

Leaning or hanging on a person's wheelchair is similar to leaning or hanging on a person and is generally considered annoying. The chair is part of the personal body space of the person who uses it.

Listen attentively when talking with a person who has difficulty speaking. Wait for the person to finish, rather than correcting or speaking for the person. If necessary, ask short questions that require short answers, a nod, or a shake of the head. Never pretend to understand if you are having difficulty doing so. Instead, repeat what you've understood and allow the person to respond.

When speaking with a person in a wheelchair or a person who uses crutches, place yourself at eye level in front of the person to facilitate the conversation.

To get the attention of a person who is hearing-impaired, tap the person on the shoulder or wave your hand. Look directly at the person and speak clearly, slowly, and expressively to establish if the person can read your lips. When talking to people who lip-read, place yourself facing the light source and keep your hands away from your mouth when speaking.

Relax. Don't be embarrassed if you happen to use accepted common expressions that seem to relate to the person's disability, such as "see you later" or "did you hear about this?"

Be Prepared: Selling a Practice After Unexpected Loss

A dental practice is likely the largest asset most dentists have, but not many dentists think about what that practice might be worth if they're not there to run it, wrote Alan A. Clemens in the New York State Dental Journal, November 2002.

Most dental practices are personal service businesses that depend primarily on one person -- the dentist. If the dentist is unable to see patients, the practice will deteriorate rapidly as patients seek other dentists for their treatment, Clemens wrote.

## Evidence-Based Search Engine and Perio Health Center Developed

A unique search engine for evidence-based dentistry and an international center for evidence-based periodontal health are now available to help dentists in their search for relevant research.

The search engine, called EviDents, at http://medinformatics.uthscsa.edu/EviDents, allows patients and clinicians to sort through vast amounts of information to find the best oral health evidence available, according to Richard Niederman, DMD, director of the Forsyth Center for Evidence-Based Dentistry and the originator of the search engine. The search engine addresses all dental areas, including implants, periodontics, orthodontics, endodontics, prosthodontics, and oral surgery.

The International Centre for Evidence-Based Periodontal Health has been established at the Eastman Dental Institute in London with the aim of enhancing the transfer of research into clinical practice and, thus, improving patient well-being, according to Ian Needleman, BDS, the center director. The new center will identify the most effective methods of diagnosing, preventing, and treating periodontal disease; develop a managed database resource of the best available evidence in periodontology; provide training in evidence-based oral health care and research; and provide consulting services for health care providers, researchers, and educators in periodontology.

For more information on the search engine or the International Centre for Evidence-Based Periodontal Health, please contact the Forsyth Institute at (617) 262-5200 or www.Forsyth.org.

He said there is a critical period after a dentist becomes ill or disabled during which the practice must be sold to realize its full value. That period is usually 30 to 45 days, especially in the case of the death of the owner. Clemens said that during the first month, a grief-stricken spouse and heirs understandably do little or nothing regarding the practice, and others involved may not be well-versed in dental practice evaluation and may believe the practice has a wildly unrealistic value.

Clemens said the first step to prepare for the preservation of the value of a practice in the event of an unexpected loss of the dentist's service is to get a professional evaluation while the practice is still active to determine its worth. He advises consulting an experienced and reliable dental practice transition expert to arrange for an appraisal and to arrange to have the appraisal updated regularly.

Part of the practice evaluation and appraisal should include plans for the transition expert to market the practice as quickly as possible while it still has value. Clemens said it is wise to involve key personnel, especially a spouse, in all aspects of the planning process, so that they know exactly where important papers and records are kept and who the advisers are.

Clemens said that everyone hopes plans such as these will never need to be implemented. But, he wrote, that can't always be the case, and it is for the benefit of everyone involved that the dentists make the small effort to be prepared.

Rotational Oscillation Toothbrushes Best at Reducing Plaque

Powered toothbrushes with rotational oscillation action are more effective in removing plaque and reducing gingivitis than are manual toothbrushes or other types of powered brushes, according to an independent, international research team.

The finding was announced by the Cochrane Collaboration, an independent, international, nonprofit organization.

"Rotational oscillation toothbrushes removed up to 11 percent more plaque and reduced gingival bleeding by up to 17 percent more than did manual or other powered toothbrushes," said William Shaw, PhD, MScD, joint co-coordinating editor of the Cochrane Oral Health Group, which analyzed data from clinical trials conducted over 37 years.

Six reviewers independently extracted data from reports on 29 clinical trials involving 2,547 participants in North America, Europe, and Israel. The clinical trials, conducted between 1964 and 2001, compared the effectiveness of all forms of manual and six types of power toothbrushes with mechanically moving heads.

Shaw emphasized that the review results do not indicate that toothbrushing is only worthwhile with a powered toothbrush. "There is overwhelming evidence that toothbrushing reduces gingivitis," he said. "Brushing may prevent periodontitis, and brushing certainly prevents tooth decay if used with fluoride toothpaste. These benefits occur whether the brush is manual or powered."

## Many Children Miss out on Recommended Annual Dental Visits

While most children visit the physician for annual checkups and many see the dentist once a year, nearly half miss the second yearly dental exam recommended by the American Academy of Pediatric Dentistry, a recent study found.

Investigators from the Health Resources and Services Administration's Maternal and Child Health Information Center used the 1999 National Survey of America's Families to examine trends in physician and dental visits for nearly 36,000 children and adolescents age 17 and under.

In all, slightly more than two-thirds of children made the annual physician visit recommended by the American Academy of Pediatrics and saw a dentist at least once a year. However, nearly half did not receive the AAPD-recommended second annual dental exam, the study found.

Twenty-one percent of the children studied received no preventive dental care at all, with the uninsured, those living in or near poverty, and Hispanic and African-American children least likely to get recommended dental care, according to the study. Very young children -- those 3 and 4 years old -- were also among the least likely to receive recommended preventive dental care, the study found.

"A substantial proportion of U.S. children do not receive preventive care according to professionally recommended standards, particularly dental care," the investigators wrote. "While publicly insured children experience higher rates of recommended well-child visits, much improvement is needed among public programs in providing recommended dental care, especially among adolescents and children in poor general health."

The study, "Factors that Influence Receipt of Recommended Preventive Pediatric Health and Dental Care" was published in the December issue of the journal Pediatrics.

CDA JOURNAL, VOL 31, Nº3

# Cariology in the New World Order: Moving From Restoration Toward Prevention, Part II

JOHN D.B. FEATHERSTONE, MSc, PhD, AND JON R. ROTH, CAE

**CONTRIBUTING EDITORS**

**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. HE IS THE LELAND AND GLADYS BARBER DISTINGUISHED PROFESSOR OF DENTISTRY AT UCSF.

**JON R. ROTH, CAE,** IS EXECUTIVE DIRECTOR OF THE CALIFORNIA DENTAL ASSOCIATION FOUNDATION.

Last month, we reviewed the biological mechanisms of dental caries, looked at the current problems in California, and suggested ways to begin dealing with the problems of caries in the home, dental practice, and community setting.

In Part II of this series, we will explore new hands-on applications, review clinical interventions, and provide sample risk assessment forms for use in your practice that incorporate the research described throughout this two-part series.

Heather Lynch, MD, and Peter Milgrom, DDS, will begin by sharing an overview and clinical applications of xylitol, a naturally occurring, low-calorie sugar substitute with anti-cariogenic properties.

Maxwell Anderson, DDS, MS, MEd, will continue by reviewing the efficacy of chlorhexidine on dental caries and the caries infection. Dr. Anderson will review the literature for chlorhexidine's caries reduction potential as well as the microbiologic reduction of the pathogens associated with dental caries.

Kevin Donly, DDS, MS, will look at the increasing use of fluoride varnishes, which have been approved for use as a cavity preparation lining varnish and as a tooth desensitizing agent.

Steven Adair, DDS, MS, explores new findings with another dental caries prevention strategy, pit and fissure sealants. Dr. Adair will explore the latest interventions using sealants that have developed as the epidemiology of caries has become better-understood.

John Hicks, DDS, PhD, MD; Franklin Garcia-Godoy, DDS, MS; Kevin Donly, DDS, MS; and Catherine Flaitz, DDS, MS, review the efficacy of fluoride-releasing restorative materials and secondary caries. Fluoride-releasing dental materials provide for improved resistance against primary and secondary caries in coronal and root surfaces and plaque as well as elevate salivary fluoride levels to a point that facilitates remineralization.

Ray Stewart, DMD, MS, and Kevin Hale, DDS, present an overview of methods in a practice setting where dental professionals can apply new intervention methods.

William Bird, DDS, DrPH, brings the discussion full circle with information on consumer compliance as a measure of success for any caries control or caries protocol program. Dr. Bird will suggest an outline of six key global areas to be considered in compliance.

The final item is a consensus paper prepared as a result of the conference held in April 2002 and the science reviewed at that meeting and published in the February and March issues of this Journal. It presents a caries management by risk assessment tool for dental and medical professionals to put to use in their practices and in community settings. This document can be reproduced from this Journal.

Our overall goal is to provide the basis for a cross-disciplinary approach from among medicine, dentistry, nursing, and other agencies to positively affect dental health and ultimately result in the reduction and eradication of dental caries in children in every county, community and culture in California by the year 2010. It is through this collaborative approach that we will seek to end the suffering of millions of children, their caregivers, and the elderly who live daily with the constant pain resulting from dental caries.

CDA JOURNAL, VOL 31, Nº3

# Xylitol and Dental Caries: An Overview for Clinicians

Heather Lynch, MD, and Peter Milgrom, DDS

**ABSTRACT** An overview of studies about xylitol and dental caries suggests potential clinical dental applications for xylitol. Xylitol is a naturally occurring, low-calorie sugar substitute with anticariogenic properties. Data from recent studies indicate that xylitol can reduce the occurrence of dental caries in young children, schoolchildren, and mothers, and in children via their mothers. Xylitol, a sugar alcohol, is derived mainly from birch and other hardwoods trees. Short-term consumption of xylitol is associated with decreased Streptococcus mutans levels in saliva and plaque. Aside from decreasing dental caries, xylitol may also decrease the transmission of S. mutans from mothers to children. Commercial xylitol-containing products may be used to help control rampant decay in primary dentition. Studies of schoolchildren in Belize and Estonia, along with data from the University of Washington, indicate that xylitol gum, candy, ice pops, cookies, puddings, etc., in combination with other dental therapies, are associated with the arrest of carious lesions. A prospective trial in Finland has demonstrated that children of mothers treated with xylitol had lower levels of S. mutans than children of mothers treated with chlorhexidine or fluoride varnish. Food products containing xylitol are available commercially and through specialized manufacturers, and have the potential to be widely accessible to consumers.

**AUTHORS**

**Heather Lynch, MD,** is a postdoctoral fellow in the Department of Dental Public Health Sciences at the Northwest/Alaska Center to Reduce Oral Health Disparities, University of Washington, Seattle.

**Peter Milgrom, DDS,** is a professor in the Department of Dental Public Health Sciences at the Northwest/Alaska Center to Reduce Oral Health Disparities, University of Washington, Seattle.

The infectious nature of dental caries and its vertical mode of transmission from mother to child have led to interest in interventions that can interrupt the disease process or prevent its initiation. To this end, xylitol, a naturally occurring sugar substitute with anticariogenic properties, has been a focus of scientific inquiry for several decades. The purpose of this paper is to present an overview of what is currently known about xylitol and dental caries, and to list xylitol's potential clinical applications in the prevention of decay. Current application for dental practice and potential community-based public health interventions are discussed.

## Sugar Alcohols

Xylitol is a sugar substitute with sweetness equal to that of table sugar.1 It is a member of the group of compounds known as sugar alcohols, which includes other common dietary sweeteners such as sorbitol and mannitol. Xylitol is produced commercially from birch trees and other hardwoods containing xylan. It can also be found in small quantities in fruits and vegetables. In contrast, sorbitol, commonly found in sugar-free products such as chewing gum, candies, and toothpaste, is less sweet than sucrose and is generally combined with other sweeteners such as saccharine or aspartame as well as xylitol to improve the flavor of the product.

CDA JOURNAL, VOL 31, Nº3

Xylitol contains 40 percent fewer calories than sucrose. Because xylitol is absorbed slowly by the human gastrointestinal tract, the main side effect associated with its consumption is osmotic diarrhea. This usually occurs only when xylitol is consumed in large quantities, four to five times those needed for the prevention of dental caries.2,3 This side effect is common to all sugar alcohols.

## Xylitol and Streptococcus mutans

Microorganisms do not readily metabolize xylitol, and its consumption has minimal effect on plaque pH.4 However, xylitol does accumulate intracellularly in S. mutans. This accumulation inhibits the bacteria's growth. This has been demonstrated in-vitro and may contribute to a reduction of S. mutans levels in the plaque and saliva of those consuming xylitol.5 In addition, xylitol has a number of effects on S. mutans that may account for some of its clinical effects in caries reduction. Short-term consumption of xylitol is associated with decreased S. mutans levels in both saliva and plaque.5 Long-term habitual consumption of xylitol appears to have a selective effect on S. mutans, resulting in selection for populations less adherent to tooth surfaces. These colonies, therefore, are shed more easily from plaque into saliva.6 This effect may not only be important to the individual's decay experience, but may also influence the transmission of S. mutans from mothers who consume xylitol to their children.

## Clinical Applications

Children at High Risk for Caries

There are surprisingly few well-studied strategies available to clinicians to prevent and control high rates of caries in the primary dentition. In the absence of water fluoridation, fluoridated toothpaste and topical fluoride varnish are the mainstays in the United States. For older children, sealants are added to the regimen. Effective strategies to reduce risk by modifying the diet of children are not readily applicable to dental practice, nor are they typically effective without significant effort. As a result, the use of xylitol is particularly attractive because its action is not dependent upon reducing the amount of other sugars in the diet. Thus, a clinician can recommend adding xylitol to the diet without asking patients to make additional alterations to their dietary patterns. Xylitol-containing products have the potential to improve success in controlling the problem of rampant decay in the primary dentition.

A number of studies conducted among schoolchildren of various ages have shown that consumption of gum containing xylitol reduces the rates of dental decay in the treatment groups (relative risks ranging from 0.27 to 0.56). Increasing use and higher doses lead to greater reductions.7 One study conducted among schoolchildren in Belize with very high rates of dentine caries showed that consumption of xylitol gum was associated with arrest of carious lesions and, as expected, that the highest dose of xylitol had the greatest effect. The number of lesions that rehardened ranged from 9 percent to 27 percent in all groups and from 12 percent to 27 percent in the 100 percent xylitol groups.8 This study is important because the children continued to consume very high levels of sucrose in their everyday diet. However, a major limitation in extending these results to the United States is that chewing gum is not considered safe for very small children and is actively discouraged in schools.

Other xylitol-containing products have been studied. A field trial of the use of xylitol-containing candies among 10-year-old schoolchildren in Estonia showed a 33 percent to 59 percent caries reduction in the groups using the candies and a 53.5 percent caries reduction in the group using the gum relative to the control group. This suggests that candy may be as effective as chewing gum as a vehicle for the delivery of xylitol in caries prevention.9 At the University of Washington, researchers have produced and field tested xylitol-containing ice pops, chewy worms, puddings, macaroons, and sorbet.10 They have initiated studies that suggest that children will fairly readily accept such foods when offered as part of the daily diet and that they suffer no side effects from their use.10,11 Food producers are available to develop these foods, but considerable work is needed to produce commercially viable products and have them accepted.

According to available data, there is no vehicle in the United States for using xylitol in toddlers and preschool children too young to chew gum. In older children, four to five pellets or sticks (1 gram of xylitol per pellet or stick) of xylitol gum per day, chewed for five minutes, should reduce dental caries activity. [PETER – do you have the reference for this recommendation?] Xylitol, approved by the Food and Drug Administration, has been used as a sweetener in foods since the 1960s.12 It is safe for use with children.3 The use of fluoridated toothpaste, topical fluorides, and sealants should also be encouraged.

### *Pregnant Women and New Mothers*

Kohler and colleagues demonstrated that the combination of good dental care, instruction to improve oral hygiene, and chlorhexidine gels and toothpastes led to reductions in maternal S. mutans levels and reduction in the extent of transmission to the child.13 More recently, Hildebrandt and colleagues showed that the use of commercially available chlorhexidine

rinses for two weeks followed by the daily use of xylitol gum (two pellets containing 1.7g xylitol) in high-caries-rate adults with recent restorations led to major reductions in S. mutans.14

A clinical trial comparing the effects of strategies to modify the maternal transmission of S. mutans, conducted in Finland, demonstrated that xylitol had the greatest effect.15 The mothers, all of whom had high S. mutans levels at the beginning of the study, were treated with either chlorhexidine varnish or fluoride varnish or 100 percent xylitol gum (65 percent xylitol by weight, chewed at least two to three times per day) for 18 to 21 months. The outcome measures were decay rates among the children and S. mutans levels in both the mothers and the children. The children of mothers treated with xylitol had the lowest levels of S. mutans during the intervention period (treatment continued until the child was 2 years old) and during followup.16 The percentage of colonization with S. mutans in the children in the xylitol group at 2 years old was 9.7 percent. This was statistically different from the other two groups, in which 28.6 percent were colonized in the chlorhexidine group and 48.5 percent in the fluoride group.17 These children were followed up most recently at 6 years old and were found still to have the lowest S. mutans levels (51.6 percent were colonized in the xylitol group vs. 83.9 percent in the fluoride group and 86.4 percent in the chlorhexidine group).16 Children of mothers treated with xylitol also had the lowest rates of decay. Followup at 5 years of age found that dentinal caries among children in the xylitol group was reduced by 70 percent as compared with children in the fluoride or chlorhexidine groups.15

These studies have been conducted only in settings in which child rearing

**Tabla**

**Products Containing Xylitol in the U.S. Market**

| Bars | Confections | Oral Hygiene | Health Care |
|---|---|---|---|
| Atkins – Diet Advantage Bar | Brown & Haley – Zingos Peppermints | Biotene – Oral Balance | B&T – Echina Spray |
| Biochem – LoCarb 2 Energy Bar | Eco-Dent – Between Dental Gum | Crest – Multicare Toothpaste | Bayer – Flintstone Vitamins |
| Country life – Ultimate LoCarb 2 | Ford Gum – Extreme Sugarfree Gums | Gerber – Tooth & Gum Cleanser | Naturas Life – Sugarless Chews |
| Richardson Labs – Carb Solutions | Godiva Chocolate – Holiday Truffles | Rembrandt – Canker Pain Relief Kit | Nicorette – Gum Extension |
| | HealthTech – Ice Chunk Mints | Rembrandt – Dazzling Beathdrops | Shiseido – Gentle Clean Soap |
| | Leaf – Xylifresh 100 Cinnamon | Rembrandt – Natural Oral Care Line | Shiseido – Moisturizing Lipstick |
| | Nabisco – Carefree Koolerz Gum | Tom's Maine – Baking Soda Line | Shiseido – Purifying Mask |
| | Naturesmart – Xylichew Gum | Tom's of Maine – Sensitive Toothpaste | Shiseido – Skincare |
| | Naturesmart – Xylichew Licorice Gum | | |
| | Naturesmart – Xylichew Mints | | |
| | Scanlab – V6 Dental Chewing Gum | | |
| | Schuster Marketing – Blitz Power Mints | | |
| | Simply Lite – Sweet 'N Low Mint Patty | | |
| | Tic Tac – Silvers | | |
| | Warner-Lambert – Trident Advantage | | |
| | Warner-Lambert – Trident for Kids | | |
| | Wrigley – Everest Mint Gum | | |
| | Wrigley – Stay Alert Gum | | |
| | Wrigley – Sugarfree Gum | | |

is done primarily by the mother and in which mother-to-child transmission is presumed. No studies have been completed in communities where child rearing is shared among greater numbers of people. The use of xylitol gum by mothers as well as other family members is currently being investigated in one such community.18

Whether used alone or in combination with other antimicrobial therapies such as chlorhexidine, xylitol has an important role in the prevention of dental decay among children born to mothers with high levels of S. mutans not only because of its effects on S. mutans levels and bacterial properties during the period of consumption, but also because its beneficial effect on decay reduction in these children appears to persist far beyond the period of consumption.16 Both chlorhexidine and xylitol may be used safely by pregnant women and nursing mothers.19,20 Currently available data suggest that twice daily use of chlorhexidine gluconate rinse (0.12 percent) for two weeks, followed by four to five sticks or pellets of xylitol gum per day chewed for five minutes each time, should lead to a major reduction in S. mutans levels in the mother and should benefit the child. In very high-risk individuals, follow-up periods of chlorhexidine use may also be beneficial.

## Xylitol Products Available in the United States

Food products containing xylitol, including chewing gums and mints, are available commercially in retail consumer settings and through specialized manufacturers (See table). Products containing 100 percent xylitol are generally available through specialized manufacturers such as Advantage International, Inc., which makes Clén*Dent chewing gum and Tundra Trading, Inc., which makes Xylichew products, including chewing gum and mint candies. There is no scientific evidence available to establish the value of xylitol-containing nasal sprays or xylitol-sweetened children's vitamins in preventing dental caries.

Despite the limitations of the current literature, there is sufficient evidence for clinicians to consider including xylitol-containing products in their clinical armamentarium for the prevention of dental decay in high-risk populations. Xylitol's favorable side-effect profile, its benefits as a sugar substitute in other areas of health, and its potential to be widely accessible to the general population through retail vendors add to its utility and applicability.

### Acknowledgments

Work cited in this paper was supported in part by Grants No. 1 P50 DE14254 and T32 DE07132 from the National Institute of Dental and Craniofacial Research, National Institutes of Health, Bethesda, Md.

### References

1. Natah SS, Hussien KR, et al, Metabolic response to lactilol and xylitol in healthy men. Am J Clin Nutr 65:947-50, 1997.
2. Forster H, Quadbeck R, Gottstein U, Metabolic tolerance to high doses of oral xylitol in human volunteers not previously adapted to xylitol. Int J Vitam Nutr Res Suppl 22:67-88, 1982.
3. Akerblom HK, Koivukangas T, et al, The tolerance of increasing amounts of dietary xylitol in children. Int J Vitam Nutr Res Suppl 22:53-66, 1982.
4. Edgar WM, Sugar substitutes, chewing gum and dental caries -- a review. Br Dent J 184(1):29-32, 1998.
5. Trahan L, Xylitol: a review of its action on mutans streptococci and dental plaque -- its clinical significance. Int Dent J 45:77-92, 1995.
6. Trahan L, Soderling E, et al, Effect of xylitol consumption on the plaque-saliva distribution of mutans streptococci and the occurrence and long-term survival of xylitol-resistant strains. *J Dent* Res 71:1785-91, 1992.
7. Hayes C, The effect of non-cariogenic sweeteners on the prevention of dental caries: a review of the evidence. *J Dent* Educ 65(10):1106-9, 2001.
8. Makinen KK, Makinen PL, Pape Jr HR, Stabilisation of rampant caries: polyol gums and arrest of dentine caries in two long-term cohorts studies in young subjects. Int Dent J 45(1 [supplement 1]):93-105, 1995.
9. Alanen P, Isokangas P, Gutman K, Xylitol candies in caries prevention: results of a field study in Estonian children. Community Dent Oral Epidemiol 28:218-24, 2000.
10. Lam M, Riedy CA, et al, Children's acceptance of Xylitol-based foods. Community Dent Oral Epidemiol 28(2):97-101, 2000.
11. Milgrom P, Xylitol clinical studies for caries prevention. 9/30/2001 - 7/31/2007, Computer Retrieval of Information on Scientific Projects: National Institute of Dental and Craniofacial Research.
12. Roberts MC, Riedy CA, et al, How xylitol-containing products affect cariogenic bacteria. *J Am Dent Assoc* 133:435-41, 2002.
13. Kohler B, Bratthall D, Krasse B, Preventive measures in mothers influence the establishment of the bacterium Streptococcus mutans in their infants. Arch Oral Biol 28(3):225-31, 1983.
14. Hildebrandt GH, Sparks BS, Maintaining mutans streptococci suppression with xylitol chewing gum. *J Am Dent Assoc* 131(7):909-16, 2000.
15. Isokangas P, Soderling E, et al, Occurrence of dental decay in children after maternal consumption of xylitol chewing gum, a follow-up from 0 to 5 years of age. *J Dent* Res 79(11):1885-9, 2000.
16. Soderling E, Isokangas P, et al, Influence of maternal xylitol consumption on mother-child transmission of mutans streptococci: 6-year follow-up. Caries Res 35:173-7, 2001.
17. Soderling E, Isokangas P, et al, Influence of xylitol consumption of mutans streptococci by infants. *J Dent* Res 79(3):882-7, 2000.
18. Grossman D, Caries prevention in Alaska native infants. 9/30/01-7/31/05, Computer Retrieval of Information on Scientific Projects: National Institute for Dental and Craniofacial Research.
19. Brambilla E, Gagliani M, et al, Caries prevention during pregnancy: Results of a 30 month study. *J Am Dent Assoc* 129(7):871-7, 1998.
20. Wang Y, van Eys J, Nutritional significance of fructose and sugar alcohols. Ann Rev Nutr 1:437-75, 1981.

To request a printed copy of this article, please contact: Peter Milgrom, DDS, Department of Dental Public Health Sciences, Box 357475, University of Washington, Seattle, WA 98195-7475 or dfrc@u.washington.edu.

CDA JOURNAL, VOL 31, Nº3

# A Review of the Efficacy of Chlorhexidine on Dental Caries and the Caries Infection

Maxwell H. Anderson, DDS, MS, MEd

**ABSTRACT** Chlorhexidine has been used for the past 35 years in the treatment of the two primary diseases of dentistry with varying degrees of success. The purpose of this paper is to review the literature for both the caries reduction potential and the microbiologic reduction of the pathogens associated with dental caries. The literature remains mixed on the success of chlorhexidine for the reduction in dental caries. Its performance as an antimicrobial against Streptococcus mutans is more consistent and favorable.

AUTHORS

**Maxwell H. Anderson, DDS, MS, MEd,** is vice president and dental director of the Washington Dental Service.

Chlorhexidine has been used to address the two primary diseases of dentistry since the mid-1970s. This paper examines the uses of chlorhexidine from several perspectives and is limited to chlorhexidine's interaction with dental caries.

Because of the vast literature regarding chlorhexidine's antimicrobial effects against many different microbial forms, this paper is limited to a review of the past 10 years of reviewed literature associated with "human," "clinical trials" and "dental caries." A search of PubMed on these parameters found 38 articles meeting the search criteria that have been published since 1966. Of these, 26 articles were judged to be relevant to this paper. This review is based on these 26 articles. The majority of these articles examine the efficacy of chlorhexidine for its antimicrobial effects or its ability to reduce dental caries.

## Antimicrobial Effects

Eleven papers were reviewed with regard to antimicrobial effects.1-11 The purpose of limiting the review to "human clinical trials" was to avoid the limitations inherent in laboratory research on planktonic cells and chlorhexidine's effects on dental caries infections that are arguably biofilm-mediated infections. Planktonic cells behave and have different characteristics than biofilm, especially with regard to resistance to antimicrobial compounds.

There was a variety of formulations, ages, and specific end-points tested. Chlorhexidine concentrations range from a high of 40 percent to a low of 0.12 percent. The delivery vehicles tested were gels, gum, varnishes and rinses. Ages tested ranged from infants to elders. The bacterial outcomes tested were vertical transmission of S. mutans, antimicrobial effects on pits and fissures, effects on the

microbial population adjacent to orthodontic brackets, interproximal and root flora, and sampling for the development of chlorhexidine-resistant organisms.

Figure 1 graphically sorts the human clinical trial data on the microbial effects of chlorhexidine. In most cases, the outcomes are expressed as being an "effective" or "not effective." Where a study reported other results, these are captured according to the authors' conclusions. For the majority of these antimicrobial studies, chlorhexidine was found to be effective in controlling or reducing the microbial challenge associated with dental caries. In general, this was an assessment of the impact on S. mutans. There is great difficulty in generalizing these studies. The protocols vary widely. While this limits the continuity of these data, it does provide a reasoned perspective of chlorhexidine's effectiveness in controlling S. mutans through a variety of clinical applications.

## Effects on Dental Caries

The data from the antimicrobial effects are generally offered as a surrogate for the effect on dental caries given the infectious nature of the disease. This section reviews the actual effect on the incidence of dental caries in tested populations. The same wide variation in testing methodologies exists in these reviewed papers as was found in the antimicrobial literature. Ten human clinical trials articles were reviewed for this section.12-21

The reviewed literature showed that chlorhexidine is effective in reducing the incidence of dental caries in the populations tested (Figure 2).

To the credit of the investigators involved, testing and analysis in recent trials have become more narrowly focused on the "at risk" surfaces in the populations being examined. In children, the primary surfaces at risk are the occlusal. In elders, the "at risk" areas are root surfaces and surfaces around existing restorations.

As a general conclusion, chlorhexidine has been shown to be effective in reducing both the number of putative dental caries pathogens and, to a lesser degree, the incidence of dental caries in the tested populations.

## Meta-Analysis

Three papers have been published that use a meta-analysis approach to examining the efficacy of chlorhexidine.22-24

VanRijkom22 found the overall caries-inhibiting effect of the chlorhexidine treatment studies to be 46 percent (95 percent CI = 35 percent to 57 percent). Multiple-regression analysis showed no significant influence on the prevented fractions for the variables "application method," "application frequency," "caries risk," "fluoride regime," "caries diagnosis," or "tooth surface."

Using different selection criteria, Bader and colleagues23 found that among the 22 studies addressing the prevention of carious lesions in caries-active or high-risk individuals, the strength of the evidence was judged to be fair for fluoride varnishes and insufficient for all other methods, including chlorhexidine.

Based on his review of the published data, Kanellis24 recommends the use of chlorhexidine in Women, Infants and Children; Early Head Start; and Head Start programs as part of a more comprehensive caries control program.

The mixed results are primarily based on the selection and inclusion/exclusion criteria used by the individual authors.

Tooth Survival

A useful clinical trial was conducted with the outcome of intervention, based on tooth mortality in elders.25 Studies like this one aim at a different end point than caries, as is appropriate for the population being tested. These data are useful in planning for health care benefits and can be expanded to demonstrate the costs associated with replacement of missing teeth or the consequences to overall health incurred when there is a decision not to restore these surfaces.

## General Economic Comments

The literature related to chlorhexidine is limited with regard to the economic effects of its application. The clinical trials on dental caries do not generally report the number needed to treat or other data that would be useful in policy decision-making for payers or public health programs. These are becoming increasingly important as consumerism reaches the health care markets. Purchasers and individual consumers are beginning to demand data on why specific procedures are recommended or not recommended in specific situations. In the future, it will be important for researchers to engage health care economists in the original planning or clinical trials to assist in gathering appropriate data about the economic dimensions of specific diagnostics, preventives, and therapeutics.

## Conclusion

In general, chlorhexidine appears to be moderately effective in reducing the number of putative caries pathogens in specific populations when applied under the regimen tested. Chlorhexidine is also useful in reducing the consequences of these infections in that it appears to generally reduce the incidence of dental caries in the tested populations. In a majority of cases, the materials tested are not available in the United States, and the extrapolation of the positive results cannot be made to the currently available rinses. Little data are presented to justify the cost of using chlorhexidine in specific

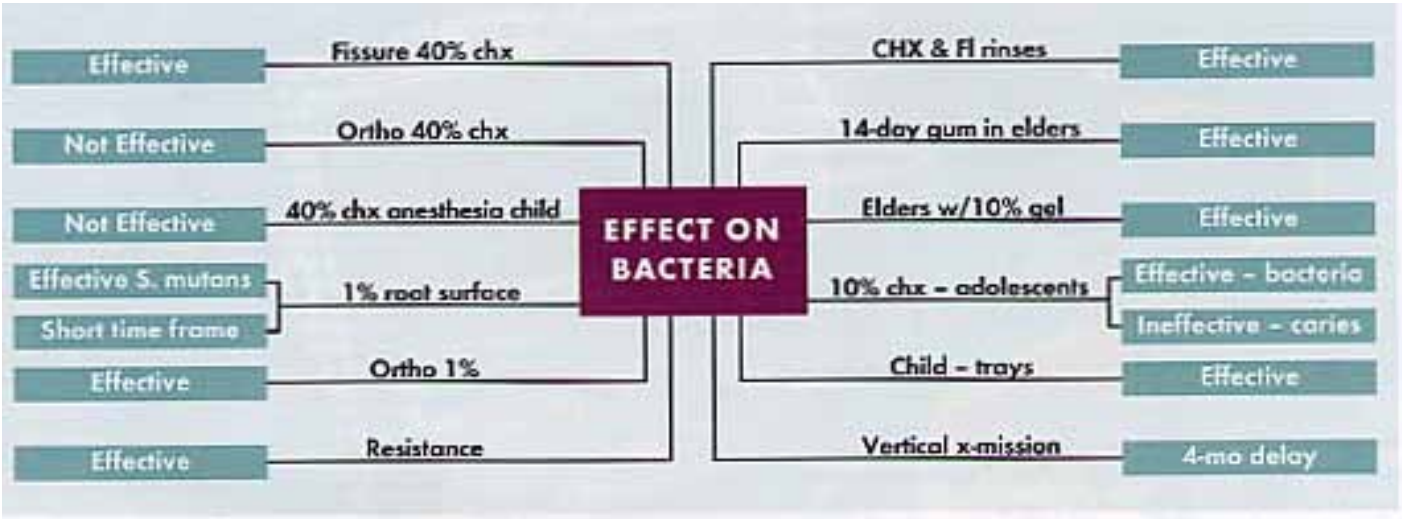



**Figure 1.**

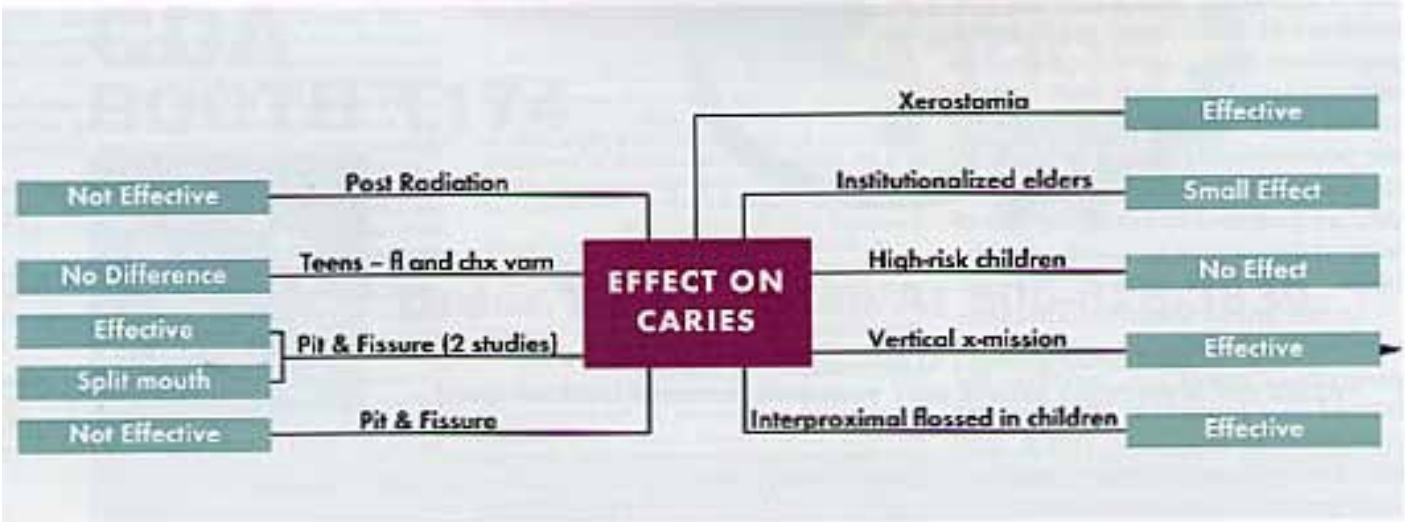



**Figure 2.**

populations.

In the United States, chlorhexidine may be useful in selected caries control programs. Current U.S. products are limited to a formula of 0.12 percent chlorhexidine gluconate. This is suitable for high-risk, highly compliant adult patients who do not exhibit either sensitivity to chlorhexidine or the ethyl alcohol vehicle in which it is usually contained. It is not generally recommended for children because of the diminished likelihood of compliance due to the taste acuity exhibited by children.

The suggested protocol for these selected patients is rinsing for 30 seconds just before bed for one week, repeating the regimen every three months. This protocol limits the amount of extrinsic staining and calculus deposition while systematically suppressing the S. mutans infection.

## References

1. Madlena M, Vitalyos G, et al, Effect of chlorhexidine varnish on bacterial levels in plaque and saliva during orthodontic treatment. J Clin Dent 11(2):42-6, 2000.
2. Jenatschke F, Elsenberger E, et al, Influence of repeated chlorhexidine varnish applications on mutans streptococci counts and caries increment in patients treated with fixed orthodontic appliances. J Orofac Orthop 62(1):36-45, 2001.
3. Forgie AH, Paterson M, et al, A randomised controlled trial of the caries-preventive efficacy of a chlorhexidine-containing varnish in high-caries-risk adolescents. Caries Res 34(5):432-9, 2000.
4. an Lunsen DM, de Soet JJ, et al, Effects of dental treatment and single application of a 40% chlorhexidine varnish on mutans streptococci in young children under intravenous anaesthesia. Caries Res 34(3):268-74, 2000.
5. Achong RA, Briskie DM, et al, Effect of chlorhexidine varnish mouthguards on the levels of selected oral microorganisms in pediatric patients. *Pediatr Dent* 21(3):169-75, 1999.
6. Ekenback SB, Linder LE, and Lonnies H, Effect of four dental varnishes on the colonization of cariogenic bacteria on exposed sound root surfaces. Caries Res 34(1):70-4, 2000.
7. Hase JC, Edwardsson S, et al, Six-month use of 0.2% delmopinol hydrochloride in comparison with 0.2% chlorhexidine digluconate and placebo (II). Effect on plaque and salivary microflora. J Clin Periodontol 25(11 Pt 1):841-9, 1998.
8. Brambilla E, Felloni A, et al, Caries prevention during pregnancy: results of a 30-month study. *J Am Dent Assoc* 129(7):871-7, 1998.
9. Simons D, Kidd EA, et al, The effect of chlorhexidine/xylitol chewing-gum on cariogenic salivary microflora: a clinical trial in elderly patients. Caries Res 31(2):91-6, 1997.
10. Bondestam O, Gahnberg L, et al, Effect of chlorhexidine gel treatment on the prevalence of mutans streptococci and lactobacilli in patients with impaired salivary secretion rate. Spec Care Dentist 16(3):123-7. 1996.
11. Ullsfoss BN, Ogaard B, et al, Effect of a combined chlorhexidine and NaF mouthrinse: an in vivo human caries model study. Scand *J Dent* Res 102(2):109-12, 1994.
12. Banting DW, Papas A, et al, The effectiveness of 10% chlorhexidine varnish treatment on dental caries incidence in adults with dry mouth. Gerodontology 17(2):67-76, 2000.
13. Powell LV, Persson RE, et al, Caries prevention in a community-dwelling older population. Caries Res 33(5):333-9, 1999.
14. Hausen H, Karkkainen S, and Seppa L, Application of the high-risk strategy to control dental caries. Community Dent Oral Epidemiol 28(1):26-34. 2000.
15. Joharji RM and Adenubi JO, Prevention of pit and fissure caries using an antimicrobial varnish: 9 month clinical evaluation. *J Dent* 29(4):247-54, 2001.
16. Gisselsson H, Birkhed D, and Bjorn AL, Effect of a 3-year professional flossing program with chlorhexidine gel on approximal caries and cost of treatment in preschool children. Caries Res 28(5):394-9, 1994.
17. Gunay H, Dmoch-Bockhorn K, et al, Effect on caries experience of a long-term preventive program for mothers and children starting during pregnancy. Clin Oral Investig 2(3):137-42, 1998.
18. Fennis-le YL, Verdonschot EH, et al, Effect of 6-monthly applications of chlorhexidine varnish on incidence of occlusal caries in permanent molars: a 3-year study. *J Dent* 26(3):233-8, 1998.
19. Bratthall D, Serinirach R, et al, A study into the prevention of fissure caries using an antimicrobial varnish. Int Dent J 45(4):245-54. 1995.
20. Petersson LG, Magnusson K, et al, Effect of quarterly treatments with a chlorhexidine and a fluoride varnish on approximal caries in caries-susceptible teenagers: a 3-year clinical study. Caries Res 34(2):140-3, 2000.
21. Giertsen E and Scheie AA, In vivo effects of fluoride, chlorhexidine and zinc ions on acid formation by dental plaque and salivary mutans streptococcus counts in patients with irradiation-induced xerostomia. Eur J Cancer B Oral Oncol 29B(4):307-12, 1993.
22. Bader JD, Shugars DA, and Bonito AJ, A systematic review of selected caries prevention and management methods. Community Dent Oral Epidemiol 29(6):399-411. 2001.
23. van Rijkom HM, Truin GJ, and van't Hof MA, A meta-analysis of clinical studies on the caries-inhibiting effect of chlorhexidine treatment. *J Dent* Res 75(2):790-5, 1996.
24. Kanellis MJ, Caries risk assessment and prevention: strategies for Head Start, Early Head Start, and WIC. J Public Health Dent 60(3):210-7; discussion 218-20, 2000.
25. Hujoel PP, Powell LV, and Kiyak HA, The effects of simple interventions on tooth mortality: findings in one trial and implications for future studies. *J Dent* Res 76(4):867-74, 1997.

To request a printed copy of this article, please contact: Maxwell H. Anderson, DDS, MS, MEd, Washington Dental Service, 9706 Fourth Ave., NE, Seattle, WA 98115 or manderson@ddpwa.com.

CDA JOURNAL, VOL 31, Nº3

# Fluoride Varnishes

Kevin J. Donly, DDS, MS

**ABSTRACT** Fluoride varnishes are available in the United States, and the Federal Drug Administration has approved a fluoride varnish for use as a cavity preparation lining varnish and as a tooth desensitizing agent. The literature, however, supports the use of fluoride varnishes to inhibit tooth demineralization and enhance remineralization. The purpose of this paper is to present an overview of literature and make recommendations according to the available scientific evidence. Findings support the use of fluoride varnishes as a safe and effective topical fluoride agent.

AUTHORS

**Kevin J. Donly, DDS, MS,** is a professor and postdoctoral program director of the Department of Pediatric Dentistry at the University of Texas Health Science Center at San Antonio Dental School.

**Disclosure**

Dr. Kevin J. Donly has received grants or research support from the National Institute of Dental and Craniofacial Research, Health Resources and Services Administration, 3M ESPE, Premier, Bisco, GC, Dentsply, Ivoclar, Kerr, Procter and Gamble, Church and Dwight, Philips, Optiva, Oral-B, Enamelon, Atrix Laboratories, and Guidor Companies.

Although he has received grants or research support from these entities, he has no personal financial interests.

Fluoride varnishes, although available in Europe for more than two decades, have more recently been introduced to the U.S. marketplace. These fluoride varnishes are recognized by the Federal Drug Administration as a device to be used as a desensitizing agent and a cavity lining varnish.1,2 Although fluoridated varnishes are officially recognized as effective cavity varnishes3 and desensitizing agents,4,5 research has demonstrated their caries prevention potential. The purpose of this paper is to provide data associated with the caries inhibition effectiveness associated with fluoride varnishes and to make recommendations for clinical use as a preventive dentistry agent.

## Fluoride Varnishes Available

There are four fluoridated varnishes marketed in the United States:

- Duraphat (Colgate Oral Pharmaceuticals, Inc., Canton, Mass). Duraphat is a 5 percent sodium fluoride varnish provided in tubes containing 10 ml of product.
- Duraflor (Pharmascience, Montreal, Canada). Duraflor is also a 5 percent sodium fluoride varnish, which is provided in 10 ml tubes.
- Fluor Protector (Ivoclar/Vivadent, Amherst, N.Y.). Fluor Protector is a 1 percent difluorsilane varnish provided in 1 ml ampules and 0.4 ml single dose units.
- Cavity Shield (OMNII Oral Pharmaceuticals, West Palm Beach, Fla.). Cavity Shield is a 5 percent sodium fluoride varnish but comes in unit-dose packages with an application brush.

Concern has been directed toward the potential settling of sodium fluoride in product packaging. A recent study indicated that the sodium fluoride contained in cavity varnishes was not equally distributed throughout the varnish tube, resulting in varying doses to be obtained when the

CDA JOURNAL, VOL 31, Nº3

fluoride varnish was extruded.6 Results from the study demonstrated a more uniform fluoride content in Duraphat tubes and Cavity Shield unit-dose packages than in Duraflor tubes. An advertised advantage to Cavity Shield is that the unit dose can be easily mixed and applied to teeth, eliminating the concern of an unknown dose of fluoride.

## Caries Prevention Effectiveness

Numerous studies document the caries-preventive effectiveness of fluoride varnish. Further in vivo and in vitro studies have addressed the use of fluoride varnishes on higher caries-risk patients, such as those receiving orthodontic treatment.7-9 Although data varies in reported effectiveness, a significant reduction in caries is noted.10-31 Several comparative trials have demonstrated equal or superior caries-prevention benefits of 1.23 percent acidulated phosphate fluoride, the standard professionally applied topical fluoride used in the United States.32-34

Although a majority of clinical trials are associated with caries inhibition in the permanent dentition, there are some studies related to the primary dentition.35-42 The average caries reduction in the primary dentition, where fluoride varnishes were typically applied twice per year, appears to be less than the caries reduction seen in the permanent dentition; however more clinical trials are necessary, particularly focusing on very young children.

## Remineralization

There is minimal information regarding the effectiveness of fluoridated varnishes to enhance remineralization. Preliminary in vitro and in vivo studies, however, indicate that fluoride varnish has the potential to aid in the remineralization of incipient caries.43-50

## Occlusal Caries Prevention

Several studies have compared the use of fluoride varnishes and sealants to prevent occlusal caries. Findings from these studies indicate sealants to be the most effective preventive agent for occlusal tooth surfaces.51-54

## Fluoride Varnish Application

The application of fluoride varnish is simple. A prophylaxis is not necessary prior to fluoride varnish application, but brushing with a toothbrush has been recommended.55-56 If there is no evidence of heavy plaque or debris on the teeth, wiping them with cotton gauze is adequate. The teeth can remain moist and the varnish will still adhere to the teeth. A total of 0.3 to 0.6 ml of fluoride varnish is sufficient to cover the dentition. After application, the patient is requested not to brush his or her teeth for the remainder of the day but to return to routine oral hygiene maintenance the following day.

## Safety

A 5 percent sodium fluoride preparation is 50,000 ppm sodium fluoride. Although this is a relatively high-dose fluoride preparation, a minimal amount is applied (0.3 to 0.6 ml).57 This can be converted to a range of approximately 5 to 12 mg fluoride. The fluoride varnish slowly breaks away from the tooth surface, and research has demonstrated that a negligible amount is ingested. Ekstrand and colleagues reported a low plasma fluoride level following placement of a 5 percent fluoride varnish, which was comparable to plasma fluoride levels experienced after toothbrushing with a fluoridated dentifrice.58 This level is significantly lower than plasma fluoride levels seen after a professionally applied 1.23 percent acidulated phosphate fluoride.59 The acidulated phosphate fluoride, even when delivered in trays, dissipates throughout the mouth with subsequent swallowing of the fluoride.

Due to the difficulty of placing topical fluoride delivery trays in children younger than 5, difficulty in obtaining the cooperation of these young children to use a slow-speed suction to remove excess fluoride from the mouth as it dissipates from the delivery tray, and the inability to keep young children from swallowing the acidulated phosphate fluoride in the delivery tray, this young population could benefit from the fluoridated varnishes. Ease of varnish application, safety, and efficacy comparable to 1.23% acidulated phosphate fluoride makes the use of fluoride varnish on young children rational.

## Summary

There is overwhelming evidence that fluoride varnish is effective at inhibiting tooth demineralization. Fluoride varnish is as effective in caries reduction as other professionally applied topical fluoride regimens currently used. The following lists of indications and contraindications summarizes recommendations:

### *Indications*

Biannual professionally applied topical fluoride agent on moderate and high-risk patients, particularly children younger than 5.

- Root desensitizing agent.
- Cavity varnish, in place of a nonfluoridated cavity varnish.
- Institutionalized patients.
- Exposed root surfaces.
- Patients receiving orthodontic therapy.
- Contraindications
- Treatment of cavitated lesions.
- Low risk, caries-free patients, living in a fluoridated community.
- Circumstances where post-fluoride treatment esthetics is a concern.

## References


1. Bawden, JW, Fluoride varnish: A useful new tool for public health dentistry. J Public Health Dent 58:266-69, 1998.
2. Beltran-Aguilar ED, Goldstein JW, Lockwood SA, Fluoride varnishes: A review of their clinical use, cariostatic mechanism, efficacy and safety. *J Am Dent Assoc* 131:589-96, 2000.
3. McComb D, Ben-Amar A, Brown J, Sealing efficacy of therapeutic varnishes used with silver amalgam restorations. Oper Dent 15:122-28, 1990.
4. Arends J, Duschner H, Ruben JL, Penetration of varnishes into demineralized root dentine in vitro. Caries Res 31:201-05, 1997.
5. Tveit AB, Tötdal B, et al, Fluoride uptake by dentin surfaces following topical applications of TiF, NaF and fluoride varnishes in vivo. Caries Res 19:240-47, 1985.
6. Shen C, Autio-Gold J, Assessing fluoride concentration uniformity and fluoride release from three varnishes. *J Am Dent Assoc* 133:176-82, 2002.
7. Seppä L, Tuutti H, Luoma H, Post-treatment effect of fluoride varnishes in children with a high prevalence of dental caries in a community with fluoridated water. *J Dent* Res 63:1221-22, 1984.
8. Todd MA, Staley RN, et al, Effect of a fluoride varnish on demineralization adjacent to orthodontic brackets. Am *J Orthod* Dentofacial Orthop 116:159-67, 1999.
9. Seppä L, Tuutti H, Luoma H, Three-year report on caries prevention using fluoride varnishes for caries risk children in a community with fluoridated water. Scand *J Dent* Res 90:89-94, 1982.
10. Clark D, A review on fluoride varnishes: An alternative topical fluoride treatment. Community Dent Oral Epidemiol 10:117-23, 1982.
11. de Bruyn H, Arends J, Fluoride varnishes -- a review. J Biol Buccale 15:71-82, 1987.
12. Haugejorden O, Nord A, Caries incidence after topical application of varnishes containing different concentrations of sodium fluoride: 3-year results. Scand *J Dent* Res 99:295-300, 1991.
13. Helfenstein U, Steiner J, Fluoride varnishes (Duraphat): A meta-analysis. Community Dent Oral Epidemiol 22:1-5, 1994.
14. Kirkegaard E, Petersen G, et al, Caries-preventive effect of Duraphat varnish applications versus fluoride mouthrinses: 5-year data. Caries Res 1986:20:548-55, 1986.
15. Koch G, Petersson L, Caries preventive effect of a fluoride-containing varnish (Duraphat) after 1 year's study. Community Dent Oral Epidemiol 3:262-66, 1975.
16. Koch G, Petersson L, Rydén H, Effect of fluoride varnish (Duraphat) treatment every six months compared with weekly mouthrinses with 0.2 percent NaF solution on dental caries. Swed Dent J 3:39-44, 1979.
17. Modéer T, Twetman S, Bergstrand F, Three-year study of the effect of fluoride varnish (Duraphat) on proximal caries progression in teenagers. Scand *J Dent* Res 92:400-07, 1984.
18. Petersson L, Arthursson L, et al, Caries-inhibiting effects of different modes of Duraphat varnish reapplication: A 3-year radiographic study. Caries Res 25:70-73, 1991.
19. Primosch R, A report on the efficacy of fluoridated varnishes in dental caries prevention. Clin Prev Dent 7:12-22, 1985.
20. Seppä L, Tuutti H, Luoma H, A 2-year report on caries prevention by fluoride varnishes in a community with fluoridated water. Scand *J Dent* Res 89:143-48, 1981.
21. Seppä L, Pöllänen L, Caries preventive effect of two fluoride varnishes and a fluoride mouthrinse. Caries Res 21:375-79, 1987.
22. Seppä L, Tolonen T, Caries preventive effect of fluoride varnish applications performed two or four times a year. Scand *J Dent* Res 98:102-05, 1990.
23. Seppä L, Pöllänen L, Hausen H, Caries-preventive effect of fluoride varnish with different fluoride concentrations. Caries Res 28:64-67, 1994.
24. Sköld L, Sundquist B, et al, Four-year study of caries inhibition of intensive Duraphat application in 11-15-year-old children. Community Dent Oral Epidemiol 22:8-12, 1994.
25. Wegner H, The clinical effect of application of fluoride varnish. Caries Res 10:318-20, 1976.
26. Petersson LG, Fluoride mouthrinses and fluoride varnishes. Caries Res 27(suppl 1):35-42, 1993.
27. Axelsson P, Paulander J, et al, Effect of fluoride containing dentifrice, mouthrinsing, and varnish on approximal dental caries in a 3-year clinical trial. Community Dent Oral Epidemiol 15:177-80, 1987.
28. Bruun C, Bille J, et al, Three-year caries increments after fluoride rinses or topical applications with a fluoride varnish. Community Dent Oral Epidemiol 13:299-303, 1985.
29. Petersson LG, Westerberg I, Intensive fluoride varnish program in Swedish adolescents: Economic assessment of a 7-year follow-up study on proximal caries incidence. Caries Res 28:59-63, 1994.
30. Seppä L, Luoma H, Hausen H, Fluoride content in enamel after repeated applications of fluoride varnishes in a community with fluoridated water. Caries Res 16:7-11, 1982.
31. Petersson LG, Magnusson K, et al, Effect of quarterly treatments with a chlorhexidine and a fluoride varnish on approximal caries in caries-susceptible teenagers: A 3-year clinical study. Caries Res 34:140-143, 2000.
32. Seppä L, Leppänen T, Hausen H, Fluoride varnish versus acidulated phosphate fluoride gel: A 3-year clinical trial. Caries Res 29:327-30, 1995.
33. Shobha T, Nandlal B, et al, Fluoride varnish versus acidulated phosphate fluoride for school children in Manipal. J Indian Dent Assoc 59:157-60, 1987.
34. Tewari A, Chawla H, Utreja A, Comparative evaluation of the role of NaF, APF & Duraphat topical fluoride applications in the prevention of dental caries -- a 2 1/2 year study. J Indian Soc Pedod Prev Dent 8:28-35, 1990.
35. Frostell G, Birkhed D, et al, Effect of partial substitution of invert sugar for sucrose in combination with Duraphat treatment on caries development in preschool children: The Malmö study. Caries Res 25:304-10, 1991.
36. Grodzka K, Augustyniak L, et al, Caries increment in primary teeth after application of Duraphat fluoride varnish. Community Dent Oral Epidemiol 10:55-59, 1982.
37. Holm A-K, Effect of fluoride varnish (Duraphat) in preschool children. Community Dent Oral Epidemiol 7:241-45, 1979.
38. Kerebel L, LeCabellec M, et al,. Report on caries reduction in French schoolchildren 3 years after the introduction of a preventive program. Community Dent Oral Epidemiol 13:201-04, 1985.
39. Murray J, Winter G, Hurst C, Duraphat fluoride varnish. A 2-year clinical trial in 5-year old children. Br Dent J 143:11-17, 1977.
40. Paunio P, Effect of a fluoride varnish on primary dentition. Dental research in Finland 1982. Proc Finn Dent Soc 78:114, 1982.
41. Twetman S, Petersson LG, Pakhomov GN, Caries incidence in relation to salivary mutans streptococci and fluoride varnish applications in preschool children from low- and optimal-fluoride areas. Caries Res 30:347-53, 1996.
42. Petersson LG, Twetman S, Pakhomov GN, The efficiency of semiannual silane varnish applications: A two-year clinical study in preschool children. J Public Health Dent 58:57-60, 1998.
43. Seppä L, Hausen H, et al, Effect of a sodium fluoride varnish on the progress of initial caries lesions. Scand *J Dent* Res 91:96-98, 1983.
44. Ögaard B, Rölla G, Helgeland K, Fluoride retention in sound and demineralized enamel in vivo after treatment with a fluoride varnish (Duraphat). Scand *J Dent* Res 92:190-97, 1984.
45. Seppä L, Effects of a sodium fluoride solution and a varnish with different fluoride concentrations on enamel remineralization in vitro. Scand *J Dent* Res 96:304-09, 1988.
46. Holmen L, Ögaard B, et al, A polarized light and scanning electron microscope study of the effect of Duraphat treatment on in vivo caries. Scand *J Dent* Res 94:521-29, 1986.
47. Ögaard B, Duschner H, et al, Microradiography and confocal laser scanning microscopy applied to enamel lesions formed in vivo with and without fluoride varnish treatment. Eur J Oral Sci 104:378-83, 1996.
48. Weinstein P, Domoto P, BB Tooth Decay. Results of a promising open trial to prevent baby bottle tooth decay: A fluoride varnish study. *J Dent* Child 61:338-41, 1994.
49. Attin T, Hartman O, et al, Fluoride retention of incipient enamel lesions after treatment with a calcium fluoride varnish in vivo. Arch Oral Biol 40:169-74, 1995.
50. Castellano JB, Donly KJ, In vitro remineralization using different fluoride varnish application techniques. *J Dent* Res 81:A352 (Abst #2816), 2002.
51. Holm G-B, Holst K, Mejàre I, The caries-preventive effect of a fluoride varnish in the fissures of the first permanent molar. ACTA Odontol Scand 42:193-97, 1984.
52. Bravo M, Llodra JC, et al, Effectiveness of visible light fissure sealant (Delton) versus fluoride varnish (Duraphat): 24-month clinical trial. Community Dent Oral Epidemiol 24:42-46, 1996.
53. Bravo M, Baca P, et al, A 24-month study comparing sealant and fluoride varnish in caries reduction on different permanent first molar surfaces. J Public Health Dent 57:184-86, 1997.
54. Bravo M, Garcia-Anllo I, et al, A 48-month survival analysis comparing sealant (Delton) with fluoride varnish (Duraphat) in 6- to 8-year-old children. Community Dent Oral Epidemiol 25:247-50, 1997.
55. Seppä L, Effect of dental plaque on fluoride uptake by enamel from a sodium fluoride varnish in vivo. Caries Res 17:71-75, 1983.
56. Vaikuntam J, Fluoride varnishes: Should we be using them? *Pediatr Dent* 22:513-16, 2000.
57. Roberts JF, Longhurst P, A clinical estimation of the fluoride used during application of a fluoride varnish. Br Dent J 162:463-66, 1987.
58. Ekstrand J, Koch G, Petersson LG. Plasma fluoride concentration and urinary fluoride excretion in children following application of the fluoride-containing varnish Duraphat. Caries Res 14:185-89, 1980.
59. Ekstrand J, Koch G, Petersson LG. Plasma fluoride concentration in pre-school children after ingestion of fluoride tablets and toothpaste. Caries Res 17:379-84, 1983.


To request a printed copy of this article, please contact: Kevin J Donly, DDS, MS, Department of Pediatric Dentistry, Dental School, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, or at donly@uthscsa.edu.

CDA JOURNAL, VOL 31, Nº3

# The Role of Sealants in Caries Prevention Programs

Steven M. Adair, DDS, MS

**ABSTRACT** Pit and fissure sealants have been employed as an element in dental prevention programs for more than 30 years. The technique for sealant placement has evolved over time to become somewhat more invasive today. However, a meticulous technique is still required for success. Practitioners recently estimated that their one- and three-year sealant success rates were 89 percent and 78 percent, respectively. Grand medians for sealant success rates after one year in clinical trials have been reported to be as high as 83 percent for effectiveness and 92 percent for complete retention. Seven-year rates were 55 percent and 66 percent, respectively. Several changes in caries epidemiology have had an impact on the use of sealants. These changes include:

- Declines in overall caries rates in U.S. schoolchildren during the latter decades of the 20th century;
- A relative increase in the percentage of the population DMFS constituted by occlusal caries; and
- A general slowing in the rate of lesion progression.
- Dentists' abilities to diagnose occlusal surface status also affect the decision to seal. Sealant cost-effectiveness can be improved by:
- Targeting at-risk populations;
- Using sealants on incipient lesions and minimally defective restorations; and
- Training more dental auxiliaries to place sealants under a dentists' supervision. Concerns about sealing over decay and the estrogenicity of sealant components are addressed. Sealant guidelines, as promulgated by the Workshop on Guidelines for Sealant Use, are reviewed; and conclusions are presented about the role of sealants in prevention programs.

**AUTHORS**

**Steven M. Adair, DDS,** MS, is chair and director of advanced specialty education in the Department of Pediatric Dentistry at the School of Dentistry, Medical College of Georgia.

CDA JOURNAL, VOL 31, Nº3

The title of Michael Buonocore's 1955 paper in the Journal of Dental Research was unpretentious: "A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces."1 Few but the most perceptive researchers and clinicians foresaw the revolution that his discovery would bring to the fields of restorative dentistry, prevention, and orthodontics. Yet Buonocore's subsequent work led directly to the development of pit and fissure sealants,2 then to enamel bonding, bonded orthodontic brackets, dentin bonding, crown and bridge cements, and other uses. While the techniques and materials have evolved, the sealants we apply today to prevent pit and fissure caries are direct descendants of Buonocore's "simple method."

This paper will provide an overview of current materials and techniques, sealant effectiveness and cost effectiveness, epidemiologic considerations, concerns, and decision making. It will conclude with some considerations of the role of sealants in a caries management plan.

## Technique -- Then and Now

Initially, the sealant technique required prophylaxis of the tooth surfaces to be sealed, a 60-second etch with one of a variety of acids (typically 50 percent liquid phosphoric acid); variable rinse and drying times generally on the order of 30 seconds each; followed by application of an unfilled, UV light cured (later, autopolymerizing) sealant. Longer etch times were advocated for primary teeth. The technique was strictly non-invasive; in fact, that aspect of the technique was touted as one of its attractions. Concerns about inadvertently etching adjacent tooth surfaces were quickly dispelled by studies that demonstrated the remineralization of etched and otherwise untreated enamel over relatively short periods of time. No advantage was seen in applying sealant to a nonrestored portion of an occlusal surface if a restoration had to be placed in another portion of that same surface.

In current practice, etch times are down to 10 to 20 seconds, etchants are less concentrated liquids and gels (typically 37 percent phosphoric acid), and some degree of fissure preparation via rotary instruments or air abrasion is increasingly common. An almost bewildering array of filled and unfilled, tinted and clear, fluoride-releasing and nonreleasing, self-curing and visible light-cured materials is available. Other materials that have been tried as sealants include flowable composites, glass ionomer cements, and even amalgam. Bonding agents have been shown to increase sealant retention, especially on enamel that has become contaminated with saliva after etching.3,4 Still, national surveys indicate that sealants are underutilized in the United States. The Third National Health and Nutrition Examination Survey, conducted from 1988 to 94, found that among U.S. children ages 5 to 17, only 18.5 percent had at least one sealant.5

Primosch and Barr6 recently surveyed 1,210 pediatric dental practitioners and all 55 departments of pediatric dentistry regarding their sealant placement techniques. Responses were received from 70 percent of the practitioners and 90 percent of the departments. Reported use of antisialogogues was rare, with 85 percent of the practitioners and 91 percent of the departments reporting that they never employ them. Cotton roll and/or Dri-Angle isolation, sometimes in conjunction with a saliva ejector, was preferred over a rubber dam. Among practitioners, a range of surface cleansing techniques were evident, from use of an explorer by 54 percent to air abrasion by 2 percent. Techniques taught by departments of pediatric dentistry uniformly included

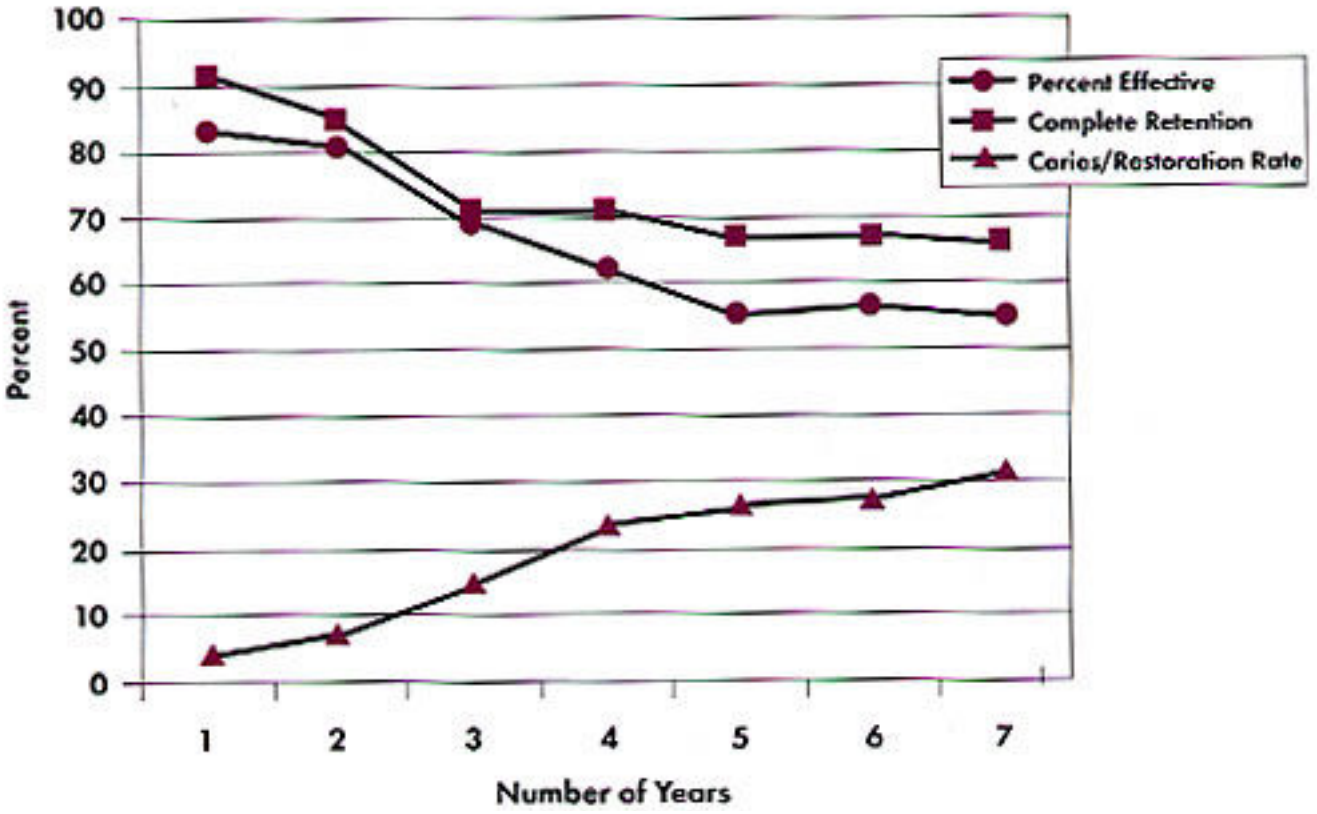



**Figure 1.** Grand medians for percent effectiveness, complete retention, and caries/restoration rates by time since applications.7

some means of surface cleansing, but without use of air abrasion or hydrogen peroxide. 13 percent of practitioners and schools alike did no fissure preparation, while the majority in each group used high- or low-speed rotary instruments for this purpose. Air abrasion for fissure preparation was more commonly employed in practice (18 percent) than in schools (2 percent). Clearly, however, the technique has evolved into a more invasive procedure. A wide range of etching times was reported by both groups for primary and permanent teeth (15 to 120 seconds), with mean etching times for primary and permanent teeth of 29 and 26 seconds, respectively. Practitioners reported using a wider selection of materials than did schools, and some had adopted laser polymerization. Dental schools uniformly employed visible-light-cured materials.

## Sealant Effectiveness

Practitioners in the Primosch and Barr study6 estimated their one-year and three-year sealant retention rates to be 89 percent and 78 percent, respectively. Sealant retention has traditionally been equated in the dental literature with effectiveness under the assumption that pit and fissure caries cannot be initiated or progress under an intact sealant. Unfortunately, there is no uniformity in the literature with regard to reporting the results of sealant studies. Studies have been conducted in optimally fluoridated and fluoride-deficient communities. Results for primary and permanent dentitions or for molars and premolars have been reported separately and combined. Some studies used multiple sites per tooth while others used the tooth as the basis for analysis. Thus, it is difficult for summaries of large numbers of studies to take these methodological differences into account.

The early trials of first-generation (ultraviolet light curing) and second-generation (autopolymerizing) sealants employed half-mouth designs in which a pair of contralateral caries-free molars of the same type were chosen; one was selected at random to be sealed while the other was left unsealed as a negative control. Percent effectiveness was calculated by various formulas that used the sealed and unsealed tooth pair as the unit of analysis. In general, these formulas determined the number of surfaces "saved" by the sealant, divided by the number of pairs with DMF control teeth. A sealant "success" occurred when the sealed tooth remained sound and its matched tooth became carious or was filled. A sealant "failure" occurred when the sealed tooth became carious while its matched tooth remained sound. When "ties" occurred, they were not entered into

the calculation of net gain. As noted by Burt and colleagues8 however, a sealed tooth that decays is still a failure even if the matched tooth becomes carious as well.

Weintraub7 evaluated 20 studies that evaluated the percent effectiveness of first- and second-generation sealants. Studies were conducted in fluoridated and nonfluoridated communities using predominately permanent first molars. The grand medians for effectiveness ranged from 83 percent after one year to 55 percent after seven years (Figure). One small study that included 10-year followups reported 68 percent effectiveness. Most of the studies reported results after only one application of sealant, though sealant effectiveness was increased if they were repaired or replaced as required. There are other factors that can influence percent effectiveness; but, in general, sealants were more effective in optimally fluoridated communities compared to fluoride-deficient communities, and less effective in primary teeth (two studies) than permanent teeth.

Half-mouth designs were employed up until the mid-1970s, when a marketed sealant received full acceptance from the American Dental Association Council on Dental Materials and Devices. At that point, denial of sealant usage was considered unethical. Study designs changed to focus on sealant longevity, or retention, as the benchmark for effectiveness. This approach was based on findings from the earlier studies that effectiveness was directly related to retention.

Weintraub7 reported the grand medians derived from 50 retention studies of second- and third-generation (visible light cured) materials placed on primary and permanent teeth in fluoridated and nonfluoridated communities. Grand medians for the studies ranged from 92 percent complete retention at one year, to 67 percent at five years, to 66 percent at seven years (Figure). Ripa,9 in a 1993 summary, reported similar figures for second-generation materials, ranging from 83 percent at one year to 66 percent at seven years. He reported the average percentage of complete sealant retention for second-generation and third-generation materials to be 77.4 percent and 76.3 percent, respectively. The longest clinical evaluation of a third-generation sealant at the time of Ripa's review was five years. A comparison of the percent effectiveness data with those from retention studies reveals that percent effectiveness parallels percent retention over time but is slightly lower at each time point.

Weintraub7 reviewed 19 studies that evaluated the percent of sealed permanent first molars that became carious and/or restored. The grand medians ranged from 4 percent at one year to 26 percent at five years (Figure). Rates were slightly higher in fluoride-deficient communities than in optimally fluoridated communities. As the percentages of complete retention and effectiveness declined over time, there was a concomitant rise in sealed teeth that became carious or restored.

Finally, Weintraub7 reported on four studies that assessed sealant reapplication rates. These studies revealed that reapplication rates were relatively high after initial sealant placement, possibly reflecting sealant loss from partially or newly erupted teeth. Following those initial replacements, the reapplication rates showed a pattern similar to the rates of caries development or restoration placement in studies where sealants were not reapplied.

One study deserving of mention simply because of the longevity of followup was reported by Wendt and colleagues in 2001.10 That study evaluated 151 permanent first molars and 161 second molars that had been sealed 15 to 20 years earlier. Sealants on first molars had been reapplied if they were missing at the time the second molars were sealed. For first molars, sealants were completely retained on 65 percent and partially retained on 22 percent. Complete and partial retention rates on second molars were 65 percent and 30 percent, respectively. Caries and/or restorations were found in 13 percent of first molars and 5 percent of second molars. Those data included sealants and caries in the buccal pits of lower molars, areas notorious for poor sealant retention and high caries rates. The rate of complete retention was higher than predicted by Weintraub's review, while the caries/restoration rate was lower. While the merits of this study could be debated, it certainly indicated a long-lasting caries protective effect. It is clear from the literature that sealant integrity must be evaluated over time, and sealants must be reapplied as necessary to maintain their benefit.

## Implications of Changes in Caries Epidemiology

Changes in dental caries epidemiology in the latter decades of the 20th century have, in a real sense, enhanced the rationale for sealant usage. Large-scale national surveys11,12 have shown a reduction in the caries experience of children in the United States during the latter part of the 20th century. Of particular interest is the relative increase in the percentage of the DMFS constituted by the occlusal surface -- from 49 percent in 1971 to '74 to 58 percent in 1986 to '87.12 This change was accompanied by concomitant reductions in proximal caries, while buccolingual lesions (also based in pits and fissures) increased slightly. Clearly, smooth surfaces became increasingly less caries-susceptible as a result of increased fluoride exposure, while caries in pits and fissures became relatively more prominent. This pattern was also borne out in a study by Li and colleagues13 comparing surface-specific caries attack rates in U.S. schoolchildren in the 1979 to '80 and 1986 to '87 national surveys. Table 1 shows the permanent tooth surfaces most commonly attacked by caries in each of the two surveys, as well as the percent reduction in the attack rate seen in the second survey. Table 2, from the same study, demonstrates that the reduction in caries attack rates has been disproportionately lower for pit and fissure surfaces compared to other surfaces in permanent teeth. Thus, while the absolute reductions in attack rates were greatest for pit and fissure surfaces, those surfaces had the lowest relative reduction. The caries attack pattern in primary teeth was shown to be more evenly distributed, with the highest proportion on smooth surfaces.

A second epidemiologic change in dental caries is the general slowing of the

CDA JOURNAL, VOL 31, Nº3

rate of lesion progression, a finding that is consistent with the increase in exposure to fluoride.14,15 This slowing of lesion development has extended the period of caries progression into the teenage years. Data from several studies indicate that pit and fissure caries can no longer be dismissed as a possibility within a few years after a tooth has erupted.16,17 Thus we should ignore former recommendations that sealants need not be placed after an apparently caries-free tooth has been in the mouth for four or more years. Teeth deemed to be caries-susceptible because of pronounced pits and fissures should be sealed, regardless of patient age.

The decrease in caries rates and the slowing of lesion progression make caries diagnosis more difficult. Our traditional method of using explorers to probe pits and fissures tends to increase the number of false positive diagnoses. Lussi18 concluded that use of an explorer did not improve the validity of diagnosing occlusal caries compared to a visual inspection alone. Cardoso and colleagues19 determined that clinical experience did not enhance occlusal caries diagnosis via visual inspection supplemented by explorer use. It should be noted that the teeth used in that study were extracted impacted third molars that were assumed to be caries-free, meaning that any diagnosis of caries was assumed to be a false positive. Other studies have confirmed the inefficiencies of the dental explorer for the diagnosis of occlusal caries.20,21 Clearly, dentistry must find ways to improve its diagnostic abilities and thus maximize sealant effectiveness.

**Table 1**

**Pit-and-Fissure Surfaces Attacked per Thousand Surfaces at Risk in Permanent Teeth.**

**Data from Li et al.[13] for U.S. schoolchildren.**

| Tooth | Surface | 1980 | 1987 | Percent reduction |
|---|---|---|---|---|
| Maxillary first molar | Occlusal | 445.12 | 324.40 | 27.12% |
| Mandibular first molar | Occlusal | 432.03 | 312.61 | 27.64% |
| Mandibular second molar | Occlusal | 309.65 | 209.79 | 32.25% |
| Mandibular first molar | Facial | 247.72 | 190.69 | 23.02% |
| Maxillary second molar | Occlusal | 259.65 | 174.09 | 32.95% |
| Maxillary first molar | Lingual | 202.50 | 155.51 | 23.21% |

**Table 2**

**Tooth Surfaces Attacked per Thousand Surfaces at Risk in Permanent Teeth by Type of Surface.**

**Data from Li et al.[13] for U.S. schoolchildren.**

| Type of surface | 1980 | 1987 | Difference | Percent reduction |
|---|---|---|---|---|
| Pit and fissure surfaces | 191.46 | 135.93 | 55.53 | 31.03% |
| Proximal surfaces of posterior teeth | 27.31 | 13.40 | 13.90 | 51.86% |
| All other surfaces | 8.21 | 3.71 | 4.50 | 50.10% |

## Cost-Effectiveness

As caries rates decline, the use of sealants becomes increasingly less cost-effective. Söderholm22 has suggested three approaches to help maintain or improve cost-effectiveness. The first is targeting population risk groups. This option is public-health based and beyond the scope of many dental school programs and private practice. Risk assessment of individuals, however, can be done in these settings, however, and should be a integral part of sealant placement decision-making. A second approach is to encourage dentists to place sealants on incipient lesions and defective restorations. While most dental schools and practitioners would hesitate to adopt this approach, it should be investigated as a means of conserving tooth structure in a program of nonsurgical caries management. The third approach is to increase the prophylactic placement of sealants by dental hygienists and dental assistants. This approach is commonly employed in practices where the auxiliaries are under the supervision of the dentist.

Sealant effectiveness and cost-effectiveness are dependent upon disease levels and the selection of patients and tooth surfaces to be sealed. Thus, another critical way in which sealant usefulness can be increased is by developing and applying evidence-based caries risk assessments to individual patients. The reduction of caries rates in U.S. schoolchildren is not uniform, resulting in a skewed distribution of disease.23 Today, approximately 80 percent of the dental caries burden is found in 20 percent of the population. In general, socioeconomic status is inversely related to caries experience and caries risk. The use of sociodemographic data can be useful in a gross estimation of caries risk, but a myriad of other factors can be applied to refine risk assessment.

## Sealant Concerns

The major concern with sealants has been the inadvertent sealing of dental caries.24 This misapprehension has been put to rest by a number of studies showing that dental caries becomes arrested under intact sealants.25-27 These findings have largely served to comfort those clinicians who were concerned about sealing unrecognized

caries. It is likely that few clinicians employ, and even fewer schools teach, intentional therapeutic use of sealants. However, as noted earlier, the sealant technique is becoming more invasive, so clinicians are more likely to ensure that sealants are not being placed over carious lesions. A second concern has arisen in recent years regarding the estrogenicity of sealant components.28 More recent work29 suggests that these components may not be absorbed, or that they may be present in nondetectable levels in the blood. Dentists can take steps to minimize the patient's exposure to uncured resin components after sealant placement.30

## Sealant Guidelines

The Workshop on Guidelines for Sealant Use, convened in 1994, published its recommendations in 1995.31 The guidelines for sealant use in individual care programs promulgated by that group should form the basis for what is taught in dental schools and residency programs, and for sealant use in practice. The guidelines call first for risk assessment of the individual based on caries experience, dental care utilization pattern, use of preventive services, and pertinent medical history. This is followed by risk assessment of individual teeth based on pit and fissure morphology, level of caries activity, and caries pattern. These assessments are followed by an evaluation of pit and fissure surfaces, with determinations of "caries-free," "questionable," "enamel caries," and "dentin caries." Sealant is recommended for teeth classified as "questionable" or "enamel caries," and for those caries-free teeth deemed to be at risk for caries. At this time, sealants are not recommended for teeth with dentinal caries. Sealants are also not recommended if the tooth cannot be isolated; if a proximal restoration involves a pit and fissure surface; or, in the case of a primary tooth, if the life expectancy of the tooth is short. In addition, the guidelines call for evaluation of previously sealed teeth for sealant integrity, retention, and caries progression.

## The Role of Sealants

Sealants should continue to play a strong role in prevention programs for children, adolescents, and adults. The relative increase in pit and fissure caries enhances that role, even in the face of recent declines in caries prevalence. Schools must teach, and clinicians should apply, caries risk assessment at the levels of the individual and the tooth. Pit and fissure caries diagnosis must be improved by reducing reliance on dental explorers, and through the evaluation and, where appropriate, adoption of new technologies. Schools should teach a meticulous technique, incorporating minimally invasive procedures as warranted. Emphasis should be given to continuous re-evaluation of previously placed sealants, with reapplications when required. Manufacturers should be encouraged to continue to improve their products, and research should continue in the areas of sealant technique, effectiveness, and use of sealants as a therapeutic modality. Sealant use should be promoted as one of the cornerstones of caries prevention programs in community- and practice-based settings.

## Conclusions

- Dental sealants are safe and effective in preventing pit and fissure dental caries on at-risk surfaces when they are properly applied and maintained.
- Decisions regarding sealant placement must be based on caries risk assessments of individual patients and individual teeth.
- Diagnosis of pit and fissure caries must be improved so that clinicians can more reliably distinguish between categories of pit and fissure status: caries-free, questionable, enamel caries, and dentinal caries.
- Guidelines should be developed for the placement by dentists of sealants over incipient caries, over newly placed restorations, and for use in repairing minimally defective restorations.
- Dental auxiliaries should be trained to place prophylactic sealants after diagnosis by a dentist.
- Meticulous sealant placement techniques must be taught in dental schools and auxiliary programs, including the need for periodic re-evaluation and replacement as necessary.
- Manufacturers should be encouraged to continue to develop improved and innovative products.

## References


1. Buonocore MG, A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces. *J Dent* Res 34:849-5, 1955.
2. Cueto EI, Buonocore MG, Sealing of pits and fissures with and adhesive resin: its use in caries prevention. *J Am Dent Assoc* 75:121-8,1967.
3. Feigal RJ, Hitt J, Splieth C, Retaining sealant on salivary contaminated enamel. *J Am Dent Assoc* 124:88-97, 1993.
4. Borem LM, Feigal RJ, Reducing microleakage of sealants under salivary contamination: Digital-image analysis evaluation. Quint Int 25:283-9, 1994.
5. Cherry-Peppers G, Gift HC, et al, Sealant use and dental utilization in U.S. children. *J Dent* Child 62:250-5, 1995.
6. Primosch RE, Barr ES, Sealant use and placement techniques among pediatric dentists. *J Am Dent Assoc* 132:1442-51, 2001.
7. Weintraub JA, The effectiveness of pit and fissure sealants. J Pub Health Dent 49(Spec Iss):317-30, 1989.
8. Burt BA, Berman DS, Silverstone LM, Sealant retention and effects on occlusal caries after 2 years in a public program. Community Dent Oral Epidemiol 5:15-21, 1977.
9. Ripa LW, Sealants revisited: An update of the effectiveness of pit-and-fissure sealants. Caries Res 27(Suppl 1):77-82, 1993.
10. Wendt LK, Koch G, Birkhed D, On the retention and effectiveness of fissure sealant in permanent molars after 15-20 years: a cohort study. Community Dent Oral Epidemiol 29:302-7, 2001.
11. Brunelle JA, Carols JP, Changes in the prevalence of dental caries in US schoolchildren, 1961-1980. *J Dent* Res 62(Spec Iss):1346-51, 1982.
12. Brunelle JA, Oral health of United States children. The national survey of dental caries in US schoolchildren: 1986-87. National and regional findings. NIH pub no 89-2247. Washington, DC: US Department of Health and Human Services, 1989.
13. Li S-H, Kingman A, et al, Comparison of tooth surface-specific dental caries attack patterns in US schoolchildren from two national surveys. *J Dent* Res 1398-1405, 1993.
14. Ripa LW, A critique of topical fluoride methods (dentifrices, mouthrinses, operator- and self-applied gels) in an era of decreased caries and increased fluorosis prevalence. J Public Health Dent 51:23-41, 1991.
15. Newbrun E, Preventing dental caries: current and prospective strategies. *J Am Dent Assoc* 123:68-73, 1992.
16. Ripa LW, Leske GS, Varma AO, Longitudinal study of the caries susceptibility of occlusal and proximal surfaces of first permanent molars. J Public Health Dent 48:8-13, 1988.
17. Ripa LW. Has the decline in caries prevalence reduced the need for fissure sealants in the UK? A review. J PÆdiatr Dent 6:79-84, 1990.
18. Lussi A, Validity of diagnostic and treatment decisions of fissure caries. Caries Res 25:296-303, 1991.
19. Cardoso M, Baratieri LN, Ritter AV, Does clinical experience affect occlusal caries diagnosis and sealant recommendation?

An in vitro study. *J Dent* Child 66:250-4, 2001.
20. Penning C, van Amerongen JP, et al, Validity of probing for fissure caries diagnosis. Caries Res 26:445-9, 1992.
21. Lussi A, Comparison of different methods for the diagnosis of fissure caries without cavitation. Caries Res 27:409-16, 1993.
22. Söderholm K-JM, Reactor paper. The impact of recent changes in the epidemiology of dental caries on guidelines for the use of dental sealants: Clinical perspectives. J Public Health Dent 55(Spec Iss):302-11, 1995.
23. Brown LJ, Selwitz RH, The impact of recent changes in the epidemiology of dental caries on guidelines for the use of dental sealants. J Public Health Dent 55(Spec Iss):274-91, 1995.
24. Chapko MK, A study of the intentional use of pit and fissure sealants over carious lesions. J Public Health Dent 47:139-42, 1987.
25. Handelman SL, Leverett DH, et al, Use of adhesive sealants over occlusal carious lesions: radiographic evaluation. Community Dent Oral Epidemiol 9:256-9, 1981.
26. Handelman SL, Leverett DH, et al, Retention of sealants over carious and sound tooth surfaces. Community Dent Oral Epidemiol 15:1-5, 1987.
27. Mertz-Fairhurst EJ, Schuster GS, Fairhurst CW, Arresting caries by sealants: results of a clinical study. *J Am Dent Assoc* 112:194-7, 1986.
28. Olea N, Pulgar R, et al, Estrogenicity of resin-based composites and sealants used in dentistry. Environ Health Perspect 104:298-305, 1996.
29. Fung EYK, Ewoldsen NW, et al, Pharmacokinetics of bisphenol A released from a dental sealant. *J Am Dent Assoc* 131:51-8, 2000.
30. Rueggeberg FA, Dlugokinski M, Ergle JW, Minimizing patients' exposure to uncured components in a dental sealant. *J Am Dent Assoc* 130:1751-7, 1999.
31. Workshop on Guidelines for Sealant Use. Recommendations. J Public Health Dent 55(Spec Iss):263-73, 1995.

To request a printed copy of this article, please contact: Steven M. Adair, DDS, MS, Department of Pediatric Dentistry, School of Dentistry, Medical College of Georgia, Augusta, GA 30912-1210, or at sadair@mail.mcg.edu.

## Legend

Figure 1. Grand medians for percent effectiveness, complete retention, and caries/restoration rates by time since applications.7

CDA JOURNAL, VOL 31, Nº3

# Fluoride-Releasing Restorative Materials and Secondary Caries

John Hicks, DDS, MS, PhD, MD; Franklin Garcia-Godoy, DDS, MS; Kevin Donly, DDS, MS; and Catherine Flaitz, DDS, MS

**ABSTRACT** Secondary caries is responsible for 60 percent of all replacement restorations in the typical dental practice. Risk factors for secondary caries are similar to those for primary caries development. Unfortunately, it is not possible to accurately predict which patients are at risk for restoration failure. During the past several decades, fluoride-releasing dental materials have become a part of the dentist's armamentarium. Considerable fluoride is released during the setting reaction and for periods up to eight years following restoration placement. This released fluoride is readily taken up by the cavosurface tooth structure, as well as the enamel and root surfaces adjacent to the restoration. Resistance against caries along the cavosurface and the adjacent smooth surface has been show in both in vitro and in vivo studies. Fluoride-releasing dental materials provides for improved resistance against primary and secondary caries in coronal and root surfaces. Plaque and salivary fluoride levels are elevated to a level that facilitates remineralization. In addition, the fluoride released to dental plaque adversely affects the growth of lactobacilli and mutans streptococci by interference with bacterial enzyme systems. Fluoride recharging of these dental materials is readily achieved with fluoridated toothpastes, fluoride mouthrinses, and other sources of topical fluoride. This allows fluoride-releasing dental materials to act as intraoral fluoride reservoirs. The improvement in the properties of dental materials with the ability to release fluoride has improved dramatically in the past decade, and it is anticipated that in the near future the vast majority of restorative procedures will employ fluoride-releasing dental materials as bonding agents, cavity liners, luting agents, adhesives for orthodontic brackets, and definitive restoratives.

CDA JOURNAL, VOL 31, Nº3

AUTHORS

**John Hicks, DDS, MS, PhD, MD,** is in the Department of Pathology, Texas Children's Hospital and Baylor College of Medicine, and Department of Pediatric Dentistry, University of Texas-Houston Health Science Center, Dental Branch, Houston.

**Franklin Garcia-Godoy, DDS, MS,** is in the Division of Biomaterials, Department of Restorative Dentistry, and Clinical Research Center, Tufts University, School of Dental Medicine, Boston.

**Kevin Donly, DDS, MS,** is in the Department of Pediatric Dentistry, University of Texas-San Antonio Health Science Center, Dental School, San Antonio, Texas.

**Catherine Flaitz, DDS, MS,** is in the Division of Oral and Maxillofacial Pathology, and Department of Pediatric Dentistry, University of Texas-Houston Health Science Center, Dental Branch, Houston.

Dental caries is one of the most common diseases occurring in man and is prevalent in developed, developing, and underdeveloped countries.[7,33,35,36,51,60,69,106,111,113,117] Within the United States and Western Europe, there has been an overemphasis on national surveys that indicate that more than 50 percent of children and adolescents are caries-free. In reality, a small percentage of late adolescents and young adults are caries-free. Only about one in six 17-year-olds are caries-free. In fact, 94 percent of all dentate adults in the United States have experienced dental caries. Caries affects some children, adolescents, and adults to a much greater degree than others (Figure 1). One-fourth of 5- to 17-year-olds account for 80 percent of the caries experience. At age 17 years, 80 percent of caries occurs in 40 percent of these late adolescents. A similar trend is noted with older adults.[69,111] In an ambulatory New England population, 11 percent of elders older than 70 accounted for 70 percent of caries. It was noted that these New England elders had a higher caries prevalence rate than New England children.[69] The continuing caries experience throughout adulthood and into the elderly period points out that dental caries is not a disease restricted to children and adolescents. The demand for ongoing preventive and restorative care in adulthood is emphasized in a national survey that found 40 percent of adults between 18 and 74 years of age were in need of immediate dental care.[113,117]

Once a cavity forms, there are several options for restoration of the carious tooth surface. A U.S. Navy Dental Corps study120 reported the dental materials placed in 4,633 restorations in 17- to 84-year-olds. The following restorations were placed during a two-week period:

- Amalgams (78 percent);
- Composite resins (16 percent);
- Sealants (5 percent);
- Glass ionomers (1 percent); and
- Gold restorations (0.2 percent).

It was noted that 67 percent of amalgams and 50 percent of composite resins were placed because of primary caries. The remaining restorations were replacements of existing restorations. A comprehensive survey[8] of more than 9,000 restorations performed in the United Kingdom found the following types of restoration placement:

- Amalgams (54 percent);
- Composite resins (30 percent); and
- Glass ionomers (16 percent).

Initial restoration placements (49 percent) and replacements (51 percent) were quite evenly divided. More recent studies have indicated that approximately 60 percent of adult restorative care is dedicated to replacing restorations.[7,8,33,35,36,38,57,59-62,69-75,81,84-86, 90,106,11-113,115-117,120] With Swedish children and adolescents in 1995,[112] the choice of restorative material was quite different from adult populations. In 3- to 8-year-olds, the restorative materials chosen for primary teeth were:


**Figure 1A.** Variability in caries experience with mixed dentition.


**Figure 1B.** Variability in caries experience in adult. Secondary caries and restoration failure are common experiences in most individuals.

- Compomers (32 percent);
- Glass ionomer (26 percent);
- Zinc oxide-eugenol (23 percent);
- Amalgams (4 percent); and
- Composite resins (3 percent).

With 6 to 19 year-olds, caries in permanent teeth were restored with:

- Composite resins (56 percent);
- Amalgams (31 percent);
- Glass ionomers (9 percent); and
- Compomers (4 percent).

The choice of materials in this Swedish study may reflect the reluctancy toward amalgam usage. Similar studies have not been completed in the United States.

## Reasons for Restoration Placement and Replacement

The principal reason for restoration failure is secondary caries in both the permanent and primary dentitions (Table 1).[7,8,38,57,59-62,70-75,81,84,85,106,112-113,115-117,120] Secondary caries accounts for approximately 60 percent of all reasons for restoration replacement, regardless of restorative material type.[8,70-75,120] Other reasons include material failure, tooth fracture or defect, endodontic involvement, prosthetic abutment utilization, technical errors, and deterioration of esthetic quality with tooth-colored restoratives.[8,120] With pediatric patients, secondary caries is responsible for replacement of restorations in 70 percent of cases. Fracture of either the restoration or the tooth is a less frequent occurrence in children and adolescents.

The longevity of failed restorations is variable and dependent upon the restorative material (Table 1).[7,8,120] Amalgams tend to have the greatest median and mean survival times when compared with composite resins and glass ionomers. It must be realized that amalgam restorative materials have been available for well more than 100 years; and these materials have been refined for posterior tooth restoration. In contrast, the terms "composite resin" and "glass ionomer" in most clinical studies encompass many different formulations with variable strengths and weaknesses. In such studies of restoration failure and longevity, subtypes of composite resins and glass ionomers were not taken into account.

A sequela of secondary caries is the effect on the tooth requiring restoration replacement. With removal and replacement, the size of the restoration changes considerably.[38,56,59-62,74] When secondary caries is present, the original cavity margin is extended by 0.52 mm. When no caries is present, the margin is extended by 0.25 mm. This implies that the replaced restoration width will be larger by 0.5 to 1.04 mm. No doubt after several replacements, the affected tooth will become weakened and may require full coverage.

## Clinical and Histopathologic Features of Secondary Caries

Although secondary caries is the etiology of failure in 50 to 60 percent of restorations (Table 1), there is confusion regarding the definition of secondary or recurrent caries.[7,8,38,57,59-62,70-75,81,84,85,106,111-113,115-117,120] Often, marginal gaps and ditching around restorations may be ascribed to secondary caries. Only when marginal gaps are ≥ 250 µm can secondary caries be identified consistently by clinical and microscopic criteria. Some clinicians equate a marginal defect of ≥ 50 µm with an increased prevalence of secondary caries. With occlusal amalgams, macroscopic caries has been detected in only 20 percent of ditched margins and 4 percent of nonditched margins. Microscopic examination of these restorations showed histologic caries in 47 percent of nonditched margins and 59 percent of ditched margins. Margin defects and staining are not sufficient to predict the presence or absence of secondary caries and do not allow for treatment decisions.

Secondary (recurrent) caries may be defined most simply as caries detected at the margins of an existing restoration. Similar to primary caries, the enamel or root surface adjacent to the restorative material may possess an inactive arrested lesion, an active incipient lesion, or a frankly cavitated lesion (Figure 2).

Clinically, secondary caries has certain features.[59-62,70-75] A high proportion of secondary caries is located along the cervical and interproximal margins (>90 percent of failed amalgams, >60 percent of failed composite resins). With enamel surfaces, recurrent caries may be seen as a white spot (active), or a brown spot lesion (inactive). The surface may undergo a certain degree of softening prior to frank cavitation. The enamel lesion color varies depending upon the adjacent restorative material. When the cavosurface is involved and undermined by caries, the adjacent enamel surface takes on a brown to gray to blue hue; however, amalgam restorations impart such color changes due to corrosion. Transillumination may be helpful with tooth-colored restorative materials. Radiographs can detect interproximal caries, especially along gingival margins. The interface between the tooth and restoration needs to be evaluated with an explorer; however, care should be taken to avoid creating an iatrogenic defect along the cavosurface margin or cavitating the lesion's surface.

Active root surface secondary caries appears as a yellow discoloration and frequently has undergone surface softening.[31,55,81,87-89,91,105,117] In contrast, inactive secondary caries in a root surface may become sclerotic and ebonized, with a hardness level similar to that for sound enamel. With both enamel and root-surface secondary caries, the responsible microorganisms remain the same as those for primary caries. The diagnosis of secondary caries is dependent upon visual inspection, tactile sensation with judicious explorer usage, and radiographic interpretation.

During the past three decades, naturally occurring and artificially induced secondary caries (Figure 2) around restorative materials have been characterized microscopically as two separate, but interrelated lesions.[1-3,8,12,13,16-24, 26,27, 29-31, 43,44,46,48-50,52-55,59-62,64,67,79,80,82-84,93-96,100,102,103,105,107] The primary (outer) surface lesion develops in the enamel or root surface adjacent

**Table 1**

## Reasons for Restoration Placement and Replacement and Longevity of Failed Restorations: Navy Dental Corps Study

| | Amalgams | Composite resin |
|---|---|---|
| Primary caries | 56% | 47% |
| Primary caries requiring removal of existing restoration | 12% | 5% |
| Secondary caries | 15% | 10% |
| Restoration defect | 5% | 5% |
| Fractures/lost restoration | 7% | 9% |
| Fractured tooth | 2% | 11% |
| Pain/sensitivity | <1% | 1% |
| Endodontic treatment | 2% | 4% |
| Prosthetic abutment | <1% | 0% |
| Esthetics, poor | 0% | 7% |
| Longevity of failed restorations | | |
| Mean | 7.4 years | 6.3 years |
| Median | 6.2 years | 5.7 years |

## United Kingdom Study

| | All | Amalgams | Composite resin | Glass ionomer |
|---|---|---|---|---|
| Primary caries | 41% | 42% | 37% | 45% |
| Secondary caries | 22% | 28% | 15% | 13% |
| Tooth fracture | 6% | 7% | 2% | 5% |
| Margin fracture | 6% | 7% | 4% | 5% |
| Noncarious defect | 6% | 2% | na | 14% |
| Bulk fracture | 5% | 7% | 3% | na |
| Pain/discomfort | 4% | 4% | na | 5% |
| Discoloration | 3% | <1% | 5% | na |
| Other | 5% | 3% | 4% | 6% |
| Longevity of failed restorations | | | | |
| Mean | na | 6.8 years | 4.5 years | 3.8 years |
| Median | na | 6 years | 4 years | 3 years |

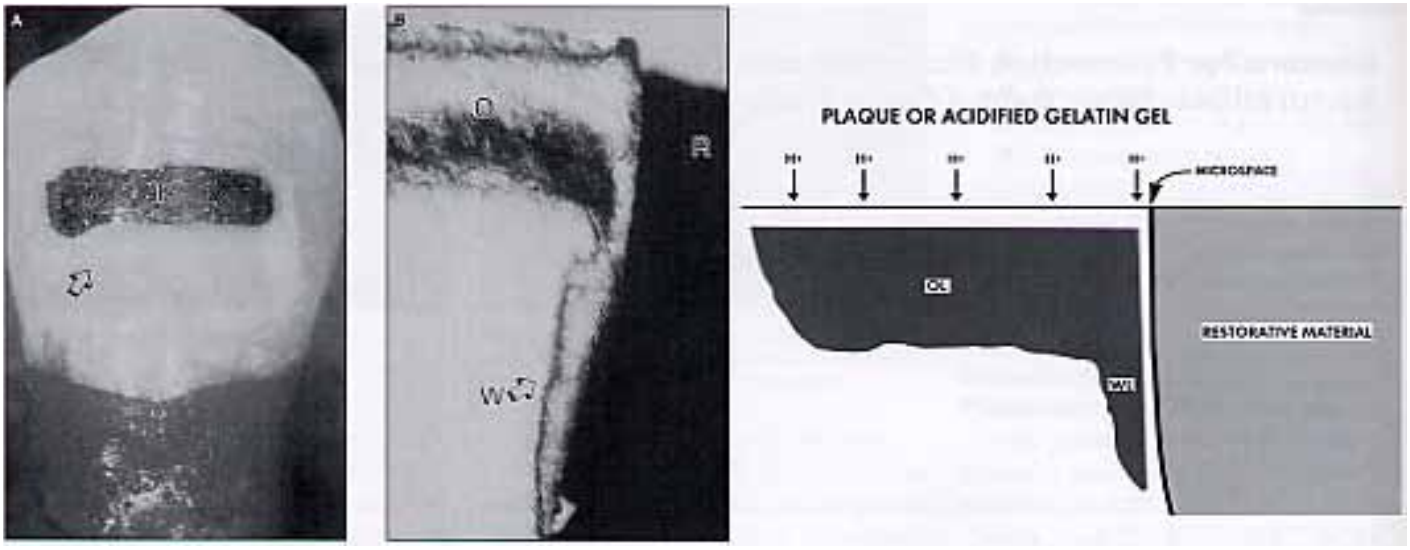
## Permanent Dentition in Children and Adolescents (6 to 19 Years of Age): Swedish Study

| | All restorations |
|---|---|
| Primary caries | 87% |
| Secondary caries | 9% |
| Fracture of restoration or tooth | 2% |
| Other | 2% |

| | All | Amalgams | Composite resins | Glass ionomers | Compomers |
|---|---|---|---|---|---|
| Restoration failure | 13% | 13% | 12% | 25% | 5% |

| | |
|---|---|
| Number of times for restoration replacement | |
| Replaced once | 12% |
| Replaced twice | 1% |
| Replaced more than twice | <1% |

Compiled from References 8, 112, 120




**Figures 2a and b.** Secondary in vitro caries around an amalgam restoration. a. An artificial white spot lesion (arrow) surrounds the amalgam restoration. b and c. Polarized light microscopic examination of this amalgam-restored tooth demonstrates the two components of secondary caries - the primary outer surface lesion (O, OL) and the wall lesion (arrow, WL). The wall lesion is formed due to percolation of acidic byproducts, lytic enzymes and colonization of plaque along the microspace present between the restorative material (R) and the cavosurface tooth structure. Secondary caries formation adjacent to restorations in coronal and root surfaces appears similar.

to the restoration; while the wall lesion forms in the cavosurface tooth structure along the restorative interface. The outer surface lesion may be readily visualized in the enamel or root surface adjacent to the restoration. The wall lesion occurs due to microleakage of oral fluids, percolation of hydrogen ions and lytic enzymes from plaque, and bacterial colonization along the cavosurface wall. Whenever a restorative material is placed, there is a possibility for a microspace (gap) to be formed between the restorative material and the cavosurface enamel, dentin, and cementum. The ability of a material to resist secondary caries development along the cavosurface is dependent upon complete removal of carious tissue leaving no residual caries, formation of an intimate cavosurface-restorative interface with minimal to no microspace, and release of caries-protective agents (fluoride, metal ions, antimicrobials, acidic ions) to the adjacent cavosurface and outer tooth surface.

Risk factors for development of secondary caries are identical to those for primary caries (Table 2).[31,35,36,51,59-62,70-75,81,90,101,104,106] The most reliable predictor of caries risk is the prior caries experience of the patient. One factor that may result in a considerable increase in root surface caries is the larger proportion of dentate elderly in the population with more retained teeth than in the past.[87-89,91,113,117] The increased number of retained teeth leads to a greater risk for periodontal disease development. With periodontal disease onset, gingival recession occurs; and this leads to exposure of caries prone root surfaces.[87-89] It has been shown that four years following root surface exposure, due to periodontal therapy, almost two-thirds of patients develop root caries. These patients had an average of 6.9 new root caries lesions. Twelve years after periodontal treatment, 80 to 90 percent have root caries with an average of 17.2 lesions per person.

Prevention of secondary caries[31,35,36,59-62,70-75] begins at the time of restoration placement with patient education in proper dental hygiene; fluoride regimen implementation (rinses, gels, fluoridated toothpastes); antimicrobials (chlorhexidine); fluoride-releasing restorative material; salivary cariogenic microorganism assessment; salivary flow rate determination; current medication inventory; dietary review; and medical evaluation if necessary. More frequent dental examinations with topical fluoride application may be indicated for especially caries-prone patients.

Various laboratory methods have been developed to investigate microleakage and caries formation along the restorative-cavosurface interface.[1,59-62] Techniques employed include: 1) artificial secondary caries systems evaluated by polarized light microscopy, scanning electron microscopy, and microradiography; and 2) microleakage determination with organic and fluorescent dyes, radioisotopes, bacterial cultures, pigmented chemical tracers, air pressure, neutron activation analysis, and electrical conductivity. Each of these methods has certain advantages and disadvantages. The most often used laboratory techniques are artificial caries systems and microleakage assessment with organic dyes. Studies using these techniques have allowed for rapid evaluation of the effects of restorative materials, bonding agents, remineralizing agents,

CDA JOURNAL, VOL 31, Nº3

**Table 2**

## Risk Factors for Secondary Caries Development

### Saliva factors

High mutans streptococcus and lactobacillus counts

Low flow rate

Low buffering capacity

Low salivary pH

Low fluoride levels

Low IgA levels

Low lysozyme, lactoferrin, and lactoperoxidase concentrations

Primary and secondary xerostomia (medications, Sjogren's syndrome, HIV/AIDS infection, radiation therapy, chemotherapy)

### Caries experience

High caries prevalence in primary and permanent dentitions (DMFS/dmfs; DMFT/dmft)

History of anterior caries

Extractions for caries

Multisurface restorations

Prior secondary caries

Active coronal and/or root-surface lesions

Malocclusion, malposition, and malformed teeth

Enamel and root-surface defects

Deep pits and fissures

Orthodontic appliances

Exposed root surfaces (gingival recession, periodontal disease, periodontal surgery)

Partial dentures with plaque retentive surfaces

### Dental plaque factors

Acidic plaque (acidogenic bacteria, fermentable carbohydrates)

Decreased buffering capacity

Intracellular and extracellular polysaccharides increased

Elevated plaque and gingival indices

Low fluoride and calcium levels

### Dietary factors

Frequent ingestion of high sucrose content and adherent foods

Low topical fluoride, strontium, molybdenum, aluminum, lithium, and boron intake

High selenium intake

Restricted or special diets: (cystic fibrosis, phenylketonuria, metabolic disease, chronic diseases)

### Access to dental care restricted or low dental knowledge

Lack of access to topical sources of fluoride (drinking water, toothpastes, rinses, gels, varnishes)

Lack of preventive and routine dental care

Lack of fluoride-releasing restorative and preventive materials

Low socioeconomic status

Low education status

### Medical conditions

Physically and mentally challenged

Medically compromised (autoimmune disease, immune compromise/deficiency, bulimia, frequent regurgitation/reflux, HIV/AIDS, cancer, radiotherapy, chemotherapy, Sjogren's syndrome)

Compiled from references 3,35,36,51,59-62,70-75,81,90,101,104,106

innovative fluoride delivery systems and fluoride-releasing products on microleakage and secondary caries formation.

## Fluoride-Releasing Dental Materials

There are numerous dental materials from many different manufacturers that have the ability to release fluoride to adjacent tooth structure and into the oral environment. A brief review of the major categories of fluoride-releasing dental materials is in order.[21,30,34,66,76,78] Several decades ago, silicate cements composed of a basic glass and phosphoric acid solution were used as tooth-colored restorative materials. Although these materials were not retained well, it was noted that secondary caries was reduced significantly. This was due to the substantial fluoride release generated by this restorative material. Glass ionomers were developed from aluminosilicate glass with calcium and a fluoride flux. The material requires an acidic polymer to induce an acid-base setting reaction. Considerable quantities of fluoride are released initially with the setting reaction, and continuous release of lower levels of fluoride is detected for long periods. Silver particles have been added to some glass ionomers to increase their physical strength, and these materials are known as glass ionomer cermets. Resin-modified glass ionomer (polyalkenoate) represents a hybrid material with a greater amount of glass ionomer than conventional resin in its makeup. This material uses an acid-base reaction, light- and/or chemical-activated polymerization, and self-curing for its setting reaction (triple-cure). Fluoride is released from this material but to a lesser extent than conventional glass ionomers. Compomer (polyacid modified composite resin) contains a higher content of composite resin with a lessened amount of ionomer material and polymerizable acidified monomer. This material is light-activated for its setting reaction. Fluoride is released primarily during the setting reaction and to a lesser extent over time. Fluoride-releasing composite resins are also available, and these contain some filler particles with releasable fluoride. Long-term fluoride release is quite low. Conventional composite resins lack fluoride-releasing abilities. In summary, there is a continuum of tooth-colored restorative products that range from high fluoride release (glass ionomer) to intermediate fluoride release (resin-

**Table 3**

**Fluoride Release From Restorative Materials**

**Fluoride release (μg Fl/cm²/day)**

| | 3 days | 7 days | 14 days | 50 days | 100 days |
|---|---|---|---|---|---|
| Glass ionomer | 15.5 | 7.5 | 4.5 | 2.2 | 1.8 |
| Resin-modified glass ionomer | 4.0 | 2.6 | 2.0 | 1.1 | 0.9 |
| Compomer | 5.0 | 3.2 | 2.1 | 1.0 | 0.8 |
| Composite resin with 30% TBATFB | 3.3 | 1.9 | 1.9 | 1.9 | 1.9 |

**Fluoride release (ppm Fl/day)**

| | 1 day | 84 days | 253 days |
|---|---|---|---|
| Resin-modified glass ionomer | 14 | 3 | 2 |
| Compomer | 1 | 1 | 1 |

**Varying Fluoride Loading Dose (ppm Fl/day)**

| | 2 days | 10 days | 20 days | 30 days |
|---|---|---|---|---|
| Control resin-modified glass ionomer | 4.2 | 1.0 | 0.9 | 0.5 |
| 1% Fl resin-modified glass ionomer | 8.0 | 1.9 | 1.1 | 0.7 |
| 2% Fl resin-modified glass ionomer | 20.3 | 4.1 | 2.4 | 1.5 |
| 3% Fl resin-modified glass ionomer | 27.0 | 5.8 | 3.6 | 2.4 |

**Cumulative Fluoride Release (ppm Fl)**

| | 1 day | 7 days | 14 days | 28 days | 70 days |
|---|---|---|---|---|---|
| Glass ionomer | 16 | 37 | 52 | 59 | 156 |
| Resin-modified glass ionomer | 15 | 20 | 32 | 41 | 92 |
| Non-fluoride resin | <0.1 | <0.1 | <0.1 | <0.1 | <0.1 |

**Fluoride Release Into Various Media (μg Fl/cm²)**

| | Water | Artificial saliva | Demineralizing-remineralizing fluid |
|---|---|---|---|
| Glass ionomer | 7.6 | 1.3 | 8.3 |
| Resin-modified glass ionomer | 5.9 | 3.1 | 12.3 |
| Compomer | 2.1 | 1.0 | 7.0 |
| Non-fluoride composite resin | 0.1 | 0.1 | 0.3 |

**Fluoride Release with Caries Challenge (μg Fl/cm²)**

| | Remineralizing fluid | Remineralizing fluid |
|---|---|---|
| Glass ionomer | 11.4 | 2.6 |
| Resin-modified glass ionomer | 12.1 | 3.7 |
| Compomer | 6.6 | 1.7 |
| Non-fluoride composite resin | 0.8 | 0.3 |

**Fluoride Uptake by Tooth Structure (ppm Fl)**

| | 30 days | 60 days | 90 days |
|---|---|---|---|
| Glass ionomer | | | |
| Enamel surface | 2,755 | 2,720 | 2,745 |
| Root surface | 16,692 | 7,454 | 6,465 |

| | Enamel Cavosurface | Dentin Cavosurface | Axial Wall |
|---|---|---|---|
| Glass ionomer | 3,900 | 6,700 | 5,700 |
| Resin-modified glass ionomer | 3,800 | 6,300 | 5,600 |

Compiled from references 11, 26, 46, 58, 77, 92, 108, 119



modified glass ionomer) to low fluoride release (compomer and fluoride-releasing composite resin) to no fluoride release (conventional composite resin). Physical properties vary with the degree of glass ionomer and composite resin content. In general, decreased physical properties are associated with increased fluoride release.

Continuing research into the develop-

ment of fluoride-releasing composite resins is ongoing in the hope of maintaining the physical properties of these materials and providing long-term fluoride release.[46] Incorporation of inorganic fluoride (NaF) has resulted in increased fluoride release but with creation of voids in the matrix as the inorganic fluoride leaches out of the material. Dispersion of leachable glass or soluble fluoride salts (YbF3) into the polymer matrix allows for a water-soluble diffusion of fluoride from the composite resin into the local environment. Most of the fluoride is released during the setting reaction, with a smaller amount of long-term fluoride release. The addition of organic fluorides to the polymer matrix has been attempted to increase fluoride release. These organic fluorides include MF-MMA (methacryloyl fluoride-methyl methacrylate), acrylic amine-HF salt, t-BAEM/HF (t-butylamino ethyl methacrylate hydrogen fluoride), MEM/HF (morpholinoethyl methacrylate hydrofluoride) and most recently TBATFB (tetrabutylammonium tetrafluoroborate). These agents hold promise for increasing fluoride delivery to the adjacent tooth structure while maintaining the physicochemical properties of composite resins.

In addition to the "more traditional" fluoride-releasing restorative materials, other methods for fluoride release are available.[4,19,20,23,24,26,29,32,42-44,47,79,93] Glass ionomer, resin-modified glass ionomers, and compomers have been formulated as luting agents and cavity liners. Many bonding agents, total-etch dentin adhesives, and one-step adhesives for various dental materials contain releasable fluoride and protect against secondary caries. Several fluoride-releasing bonding agents for amalgams, as well as fluoridated amalgams, have become available.[95,96,107] Other clinical investigators have proposed exposing the prepared cavity to topical fluoride agents to allow rapid fluoride uptake by dentin, enamel, and cementum prior to restoration.[29,42,43,67]

Fluoride content in a dental material varies considerably ranging from 7 percent to 26 percent in glass ionomers, resin-modified glass ionomers, and compomers.[2,3,5,6,11,14-17,21,30,32,34,39-42,58,66,67,77,78,86,97,108,109,114,119] However, the amount of fluoride made available to the oral cavity is not related to the fluoride content of the material, but to the ability for fluoride to leach from the material or to be exchanged for other ions in the oral environment. With all fluoridated dental materials, there is a burst of fluoride release during the setting reaction and this is followed by a gradual

**Table 4**

## Secondary Caries and Fluoride Releasing Dental Materials

| Secondary caries reduction (% reduction) | Outer lesion Depth | Wall lesion length | Wall lesion depth | Wall lesion frequency |
|---|---|---|---|---|
| Glass ionomer | 74% | 70% | 74% | 79% |
| Glass ionomer-silver (cermet) | 53% | 56% | 50% | na |
| Resin-modified glass ionomer | 46% | na | na | 44% |
| Compomer | 40% | na | na | 31% |
| Fluoridated amalgam | 48% | 35% | 41% | 53% |
| Fluoridated composite resin | 8% | 10% | 25% | na |
| Glass ionomer liner | | | | |
| Amalgam | 18% | 30% | 52% | na |
| Composite resin | 20% | 27% | 28% | na |
| Fluoridated desensitizer with amalgam | 13% | na | na | 42% |
| Fluoridated sealant | 35% | na | na | 50% |
| Glass ionomer sealant | 46% | na | na | 50% |
| APF application and amalgam | 94% | na | na | na |

| | Resin-modified glass ionomer | Compomer | Glass ionomer liner |
|---|---|---|---|
| Demineralization inhibition At dentinal margins (% reduction) | 38% | 39% | 54% |

| | Glass ionomer | Non-fluoridated composite resin |
|---|---|---|
| Microhardness after demineralization (% reduction) | 7% | 44% |

Compiled from references 2-4, 12, 16-23, 26-28, 41, 43, 44, 48, 50, 52-55, 64, 67, 79, 80, 82-84, 93-96, 102, 103, 105, 107

decline in the amount of fluoride leached into the oral environment. The dental material provides a low level of fluoride for a considerable period (Table 3). Several studies have shown well-documented fluoride availability for 2.7 to eight years from glass ionomer-based materials and up to five years from composite resins.[2,3,17,30,32,39,40] The ability to continue to release fluoride in vitro over extended periods is remarkable considering the fact that the materials are constantly exposed to an aqueous environment. The quantity of fluoride available for uptake is dependent upon the media into which the fluoride-containing restorative is placed.[11,58,108,114] Many laboratory studies report the daily or accumulated fluoride released into water. Artificial saliva tends to decrease the release of fluoride, most likely due to precipitation of calcium, phosphate and fluoride complexes on the surface of the restorative material. Exposure of the restorative material to demineralizing solutions increases the available fluoride; while remineralizing fluids decrease the amount of fluoride released.[11,58,108,114] This tends to hold true for all glass ionomer-based materials. It is well-known that during acid challenges glass ionomers mobilize and release increased amounts of fluoride into the environment.[76] This is an important feature for facilitating reprecipitation of mineral into demineralized enamel and root surfaces; thereby enhancing remineralization.

Glass ionomers and other fluoride-releasing restorative materials increase the fluoride composition of adjacent tooth structure (Table 3).[4,6,30,32,37,42,45,92,94,114,118,119] The amount of acquired fluoride in sound enamel and root surfaces adjacent to glass ionomer restorations is substantial and may be appreciated for long periods. In addition, a dramatic increase in fluoride content in enamel and dentinal cavosurfaces, as well as the dentinal axial wall, has been shown in a recent electron probe analysis.118 Both wavelength and energy dispersive spectrometry studies also found that fluoride is incorporated into the hybrid layer formed by fluoride-containing dentin adhesives.[37] Fourier transform infrared spectroscopy of the intermediate layer between glass ionomer and dentin indicates that the primary component of this layer is fluoridated carbonatoapatite.[45] This substance is known to have a low solubility and increased acid resistance. No doubt readily available fluoride from glass ionomers will enhance the in vivo caries resistance of the tooth structure composing the cavosurfaces and tooth surfaces adjacent to such restorations.

## In Vitro Secondary Caries and Fluoride-Releasing Dental Materials

The ability of a fluoride-releasing material to affect in vitro secondary caries formation is illustrated by the reduction in the occurrence of wall lesions along the tooth-restorative material interface (Table 4, Figures 3 and 4).[2-4,12,16-23,26-28,41,43,44,48,50,52-55,67,79,80,82-84,93-96,102,103,105,107] Many different fluoride-releasing materials reduce wall lesion frequency considerably (40 percent to almost 80 percent). Typically, restorative materials with a higher fluoride content tend to provide the greatest degree of protection along cavosurfaces. Not only is wall lesion development affected, but also wall lesion depth and length. Reductions in cavity wall depth and length range from 10 percent to 25 percent for fluoride-releasing composite resins to 35 percent to 41 percent for fluoridated amalgams to 70 percent to 74 percent for conventional glass ionomers. In addition, the higher the fluoride release from the dental material, the greater the chance that wall lesion formation will be inhibited and create caries inhibition zones in the cavosurface tooth structure. Typically, glass ionomers and resin-modified glass ionomers produce inhibition zones in the cavosurface, while compomers and fluoride-releasing composite resins rarely develop such inhibition zones. Outer (primary) surface lesions that form in enamel and root surfaces next to the restorations are also affected by fluoride-releasing dental materials. Reductions in outer lesion depths range from less than 10 percent for fluoridated composite resins to almost 75 percent for conventional glass ionomers. Cavity liners, desensitizers and topical fluoride application decrease substantially both outer and wall lesion depths.

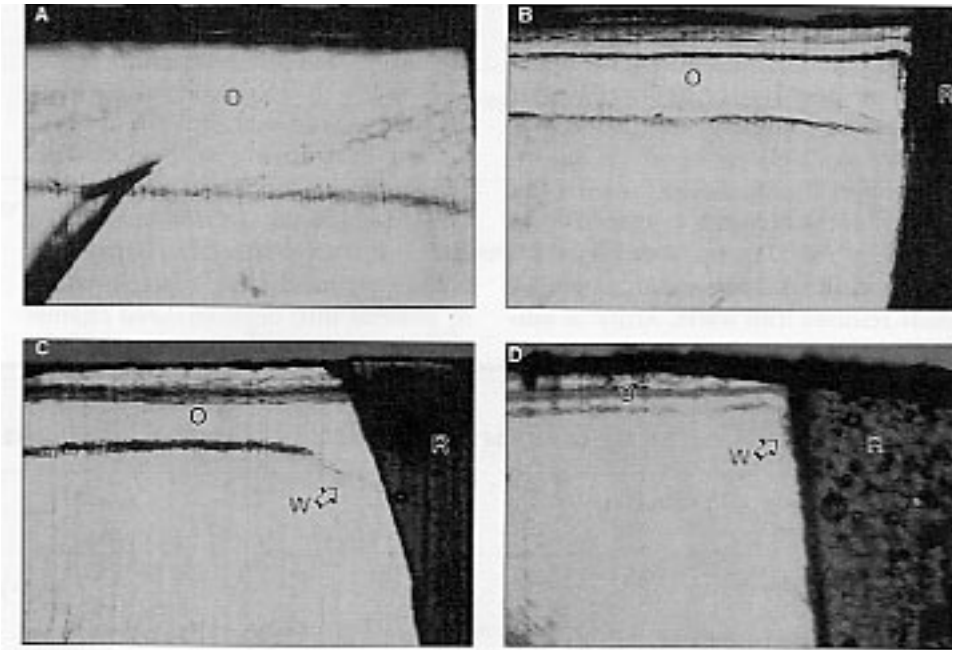
The retention of greater amounts of mineral in secondary caries lesions is also apparent from microhardness studies.[4,64,94] These studies have found that the outer lesion adjacent to a glass ionomer had only a 7 percent reduction in microhardness compared with sound enamel; however, a nonfluoride-releasing composite resin resulted in a 44 percent reduction in microhardness in the outer lesion compared with sound enamel. The availability of fluoride from the adjacent restoration results in a reduction in mineral loss from the outer lesion.

Both lesion depth and mineral loss are related in a linear fashion to the amount of fluoride released over time.[94,105] In fact, under plaque conditions, complete inhibition of secondary caries may be realized if 200 to 300 µg of fluoride are released per cm2 of the dental material during a one-month period.[2,3,17]

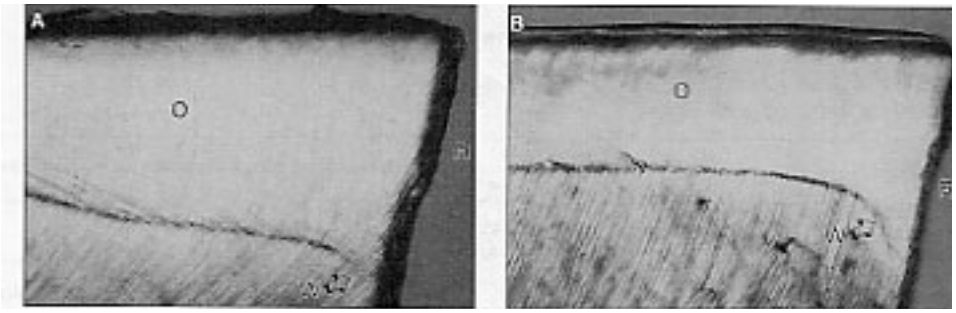
## Remote Effect of Fluoride-Releasing Dental Materials

The local environment for fluoride release is relatively extensive and is not limited to the immediately adjacent cavosurface or surface enamel (Table 5).[17,48,49,92,100] Fluoride uptake in vitro by enamel and root surfaces from conventional glass ionomers is substantial and maintained for at least six months.92 Enamel located 1.5 to 7.5 mm from glass ionomers increases its fluoride content by more than 2,000 ppm. Root surfaces up to 7.5 mm from glass ionomers have a greater ability to absorb fluoride than enamel (more than 5,000 ppm). Perhaps even more remarkable is that the glass ionomer-restored teeth were stored in artificial saliva, which is known to reduce the amount of fluoride available for uptake.

The amount of mineral loss from




**Figures 3a through d.** In vitro secondary caries formation in root surfaces adjacent to restorations (R) filled with nonfluoride releasing (a) and fluoride-releasing dental materials (b-d). Dramatic reductions in the primary outer root surface lesion (O) depth occur when nonfluoride releasing composite resin (a) restorations are compared with fluoride-releasing composite resin (b) restorations, and compomer restorations (c), and resin-modified glass ionomer restorations (d). (arrow = wall lesion).




**Figures 4a and b.** Fluoride-releasing amalgam restorative material and in vitro secondary caries formation in root surfaces. Caries formation in the root surface (O) adjacent to a conventional amalgam restoration (a) is quite extensive and considerably greater than that for the primary outer root surface lesion (O) adjacent to a fluoride-releasing amalgam (b). (R = restored cavity where material was lost during the sectioning procedure; arrow = wall lesion).

in vitro lesions adjacent to and up to 7 mm from glass ionomer restorations is reduced significantly (Table 5).[100] When compared with nonfluoride-releasing restorations, the placement of a glass ionomer reduces mineral loss by almost 80 percent at 0.2 mm from the restoration to 37 percent at 7 mm from the restoration margin. Similarly in polarized light microscopic studies, mean lesion depth for primary tooth enamel and permanent tooth root surfaces increased gradually the farther the lesion was located from the glass ionomer restoration.[48,49] In contrast, in vitro microradiographic studies found a lessened effect for fluoride-releasing composite resins.[17] The lesion depth and mineral loss was reduced in close proximity to the fluoridated resin; whereas tooth surfaces positioned 3 to 4 mm from the fluoridated restoration did not receive the benefits of fluoride release.

Tooth surfaces opposing adjacent teeth restored with glass ionomers receive a certain degree of protection against caries formation in vivo (Figure 5).[4,12,25,28,68,86,98,99,110] In a three-year longitudinal study,[98,99] about 25 percent of interproximal tooth surfaces adjacent to teeth restored with glass ionomer tunnel restorations had developed caries. In marked contrast, slightly more than 80 percent of interproximal surfaces in teeth adjacent to amalgam-restored teeth succumbed to caries. A similar three-year clinical study[86] with primary teeth found an almost twofold increase in interproximal caries in teeth adjacent to amalgams when compared with those next to glass ionomers. Another study found that the use of a resin-modified glass ionomer base under a resin decreased the risk of caries development in the opposing interproximal surface to a similar degree.110

Remineralization of existing lesions may also occur when these lesions are in close proximity to a fluoride-releasing dental material.[10,28] Placement of amalgam, nonfluoride-releasing composite resin and conventional glass ionomer Class II restorations in extracted teeth in contact with other teeth possessing well-defined proximal lesions provided insight into the effects of these restorative materials on adjacent interproximal surfaces. Following a two-week exposure to a cyclic demineralizing-remineralizing artificial caries system, differences were identified among the lesions adjacent to glass ionomer restorations compared with those in contact with fluoride-releasing composite resins and nonfluoridated amalgams. The lesions next to glass ionomers had regressed slightly in area (-2 percent); while the lesions adjacent to amalgams and fluoride-releasing resins had increased by 64 percent and 28 percent, respectively. Fluoride release into the local environment by the glass ionomer restorations resulted in no caries progression with the lesion in the opposing tooth surface.

Similarly, resin-modified glass ionomer has been shown to provide protection against in vitro lesion progression and to induce remineralization to a similar extent as that found with fluoride dentifrices, but less than that for a low-concentration (0.05 percent) sodium fluoride rinse.[68] Lesional areas of artificial caries have been noted to be reduced by 2.45-fold when placed adjacent to resin-modified glass ionomers, by 2.23-fold when exposed to a fluoridated dentifrice, and by 3.74-fold when exposed to a 0.05 percent sodium fluoride mouthrinse.

The ability of glass ionomers to release fluoride to adjacent tooth surfaces accounts for the hypermineralization of enamel and dentinal lesions seen in microradiographic investigations and inhibition zones with polarized light microscopy.[102,103] With an in vitro pH-cycling demineralizing-remineralizing system and an in vivo intraoral partial denture model, it has been noted that enamel and dentinal lesions in contact with glass ionomers possess increased calcium content and mineral volume percentage compared with enamel and dentinal lesions adjacent to nonfluoridated amalgams and composite resins. The mineral content of the glass ionomer-associated hypermineralized layer was reported to be more than threefold greater than those for the lesions adjacent to amalgams and resins. In addition, the hypermineralized area extended up to 300 µm into the underlying tooth structure. In contrast, the carious lesions adjacent to the amalgams and resins progressed and increased in depth by fourfold and threefold, respectively. The glass ionomer material induced remineralization and hypermineralization of adjacent enamel and dentinal lesions, while the nonfluoridated amalgam and resin restorations were associated with progressive demineralization.

**Table 6**

## Remote Effect of Fluoride-Releasing Dental Materials

**Fluoride uptake from glass ionomer (6 months after placement)**

| Distance from ionomer | Enamel surface | Root surface |
|---|---|---|
| 1.5 mm | 2,745 ppm | 6,465 ppm |
| 3.5 mm | 2,283 ppm | 6,061 ppm |
| 5.5 mm | 2,106 ppm | 5,952 ppm |
| 7.5 mm | 2,102 ppm | 5,862 ppm |

**Mineral loss (Volume %, microradiography)**

| Distance from restoration | Composite resin | Glass ionomer | Reduction |
|---|---|---|---|
| 0.2 mm | 8,365 | 1,875 | 78% |
| 0.5 mm | 7,731 | 2,344 | 70% |
| 1.0 mm | 7,691 | 3,280 | 57% |
| 2.0 mm | 6,268 | 2,809 | 55% |
| 4.0 mm | 6,446 | 3,317 | 49% |
| 7.0 mm | 6,951 | 4,360 | 37% |

**Primary (Outer) Lesion Depth and Mineral Loss (% reduction, microradiography)**

| Fluoride-releasing resin | Lesion depth | Mineral loss |
|---|---|---|
| Adjacent to restoration | 35% | 24% |
| 3 to 4 mm from restoration | 0% | <1% |

**Primary (Outer) Outer Lesion Depth (polarized light microscopy)**

| Distance from glass ionomer restoration | Primary tooth enamel | Root surface |
|---|---|---|
| 0.5 mm | 128 µm | 139µm |
| 1.0 mm | 148 µm | 173 µm |
| 2.0 mm | 228 µm | 232µm |
| 4.0 mm | 256 µm | 292µm |

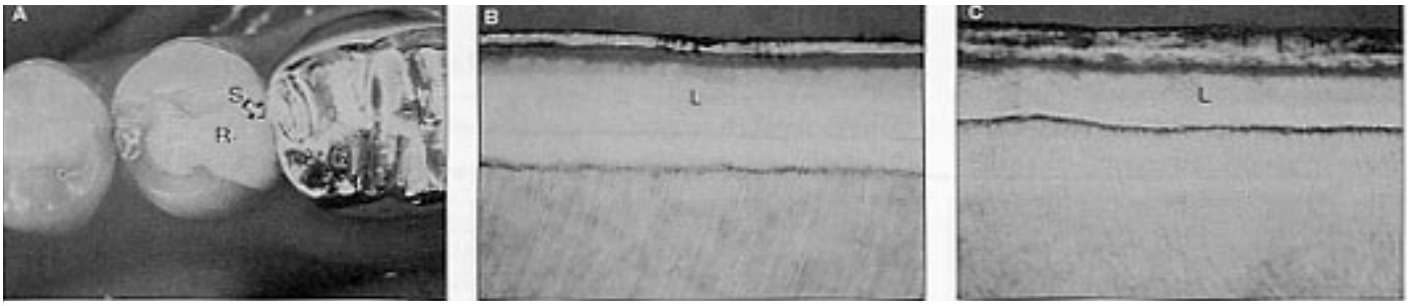
**Caries Development in Proximal Surfaces of Teeth Adjacent to Restored Teeth (clinical studies)**

| | Year 1 | Year 2 | Year 3 | Total |
|---|---|---|---|---|
| Amalgam restoration | 36% | 18% | 27% | 82% |
| Glass ionomer tunnel restoration | 0% | 18% | 9% | 27% |

| | Caries at 2 years |
|---|---|
| Resin-modified glass ionomer-resin restoration | 6% |
| Resin restoration | 11% |

| | Caries at 3 years |
|---|---|
| Primary dentition | |
| Amalgam | 21% |
| Glass ionomer | 12% |

Compiled from References: 17, 48, 49, 86, 92, 98, 99, 100




**Figures 5a through c.** An intraoral method for testing the effect of a fluoride releasing restorative material (R) employs the temporary placement of a gold crown (G) with a mesial slot (S) for retaining sections of human tooth enamel or root surfaces. Development of the intraoral caries in the tooth sections (b, c) is influenced by the release of fluoride from the adjacent restoration into the oral environment. A nonfluoride-releasing dental material allows for more extensive caries formation (L = body of lesion) in a previously sound root surface (b), compared with a lessened degree of caries formation (L = body of lesion) in a previously sound root surface (c) in close proximity to a fluoride-releasing dental material.

## Plaque and Fluoride Releasing Dental Materials

While dental plaque is intimately involved in caries development, this organic film may act as a fluoride reservoir and provide a means to affect the demineralization-remineralization process. Glass ionomer materials release fluoride into the oral environment and are in direct contact with the overlying dental plaque. Clinical studies have shown that plaque adjacent to glass ionomers has increased fluoride concentrations compared with nonfluoridated composite resins.[4,47,63,64,99] The plaque fluoride content ranges from 15.0 to 21.2 µg/g for glass ionomers compared with 0.4 to 3.5 µg/g for nonfluoridated resins. Although these levels seem to be relatively low, only small concentrations of fluoride in plaque, saliva, or calcifying fluids are necessary to shift the equilibrium from demineralization to remineralization. In fact, remineralization of enamel lesions begins with only 0.03 ppm fluoride in artificial saliva, plaque, and calcifying fluids.[35,36,101,104] Remarkably, optimal remineralization requires a fluoride concentration of only 0.08 ppm. Children in communities either with or without water fluoridation have similar baseline salivary fluoride levels of between 0.02 to 0.04 ppm, which is less than optimal for remineralization. In a four-year longitudinal study,[35,36] it has been reported that children with high salivary fluoride levels (≥0.075 ppm) are more frequently caries-free than those with lower salivary fluoride concentrations. A child's salivary fluoride level, regardless of whether fluoridated drinking water is available, is associated with the child's caries status. It is apparent that water fluoridation has less of an effect on caries than the availability of other sources of fluoride, such as dietary fluoride, fluoridated dentifrices, and fluoride mouthrinses.

The importance of relatively frequent exposure to low-dose fluoride sources is emphasized by clinical studies that have shown that caries around orthodontic brackets and in xerostomic patients may be eliminated or greatly reduced with fluoridated dentifrice usage and/or daily sodium fluoride (0.05 percent) rinsing.[24,35,36,101,104] Such preventive agents increase the fluoride content of saliva and plaque above the level necessary to facilitate remineralization for at least two to six hours. The levels may be prolonged and at higher levels if the individual does not rinse following toothbrushing or fluoride mouthrinsing.

As noted previously, glass ionomers may induce remineralization of carious lesions and also induce hypermineralization in enamel and dentin adjacent to the restorative material. These materials continually release small amounts of fluoride into the local environment. This fluoride is then taken up by plaque and saliva.[35,36,76,101,104] In many ways, these materials may be looked at as slow-release fluoride devices. Not only is fluoride available to inhibit demineralization of sound tooth structure and facilitate remineralization of hypomineralized and carious tooth structure, but the released fluoride also affects bacteria within dental plaque. Several clinical studies have shown substantial reductions (46 to 77 percent) in cariogenic bacteria (mutans streptococci, lactobacillus) within plaque adjacent to glass ionomers. This effect has been observed up to six months after restoration placement. Dental plaque fluoride, even in small concentrations, inhibits bacterial metabolism by diffusion of hydrogen fluoride from the plaque into the bacteria. Once inside the bacteria, the hydrogen fluoride acidifies the bacterial cytoplasm and leads to release of fluoride ions. These ions interfere with enzymes essential for bacterial metabolism (enolase, acid phosphatase, pyrophosphatase, pyrophosphorylase, peroxidase, catalase, proton-extruding ATPase). In addition, increased plaque fluoride decreases adherence of bacteria to hydroxyapatite. This results in reduced plaque formation.

## Recharging of Fluoride-Releasing Dental Materials

Most of the fluoride-release studies performed with fluoridated dental materials have evaluated the amount of

**Table 6**

**Caries Prevention Mechanism With Fluoride-Releasing Dental Materials**

- Demineralization inhibited by release of fluoride into local environment
- Fluoride absorption to hydroxyapatite crystal surface enhanced (fluoride veneer)
- Remineralization of lesions and hypamineralized tooth structure facilitated
- Dental plaque bacterial enzyme systems affected
- Salivary and plaque fluoride concentrations elevated
- Continuous low level fluoride delivery system with "recharging" by exogenous fluoride sources
- Physicochemical bonding (glass ionomer-based materials)
- Mechanical bonding (composite resins)

Compiled from references 34-36,50,53,55,66,76,101,104

fluoride released during varying lengths of times without the material being exposed to exogenous sources of fluoride. From the information presented previously, it is obvious that certain materials release fluoride for long periods (up to eight years). This is not equivalent to what happens in the oral environment. The vast majority of individuals in developed and developing countries have access to fluoridated toothpastes, over-the-counter low-dose fluoride rinses, and prescription high-dose fluoride rinses and toothpastes. Exposure of fluoride-containing dental materials to exogenous fluoride sources replenishes the fluoride within the dental material and provides a continuing, renewable source of fluoride for the oral environment.[5,10,25,28,29,39,40,42,65,68,97,108] This is particularly true for all glass ionomer-based restorative materials and less so for composite resin-based materials. Professionally applied acidulated phosphate fluoride treatment provides a 2.5 to 4.0 increase in fluoride release from fluoride-releasing dental materials. Even with commercially available fluoridated toothpastes, the fluoride uptake and release by fluoride-containing materials is substantial and adequate to increase plaque and saliva fluoride to levels sufficient to inhibit demineralization and facilitate remineralization. Significant increases in fluoride release (twofold) may be achieved when conventional and resin-modified glass ionomers are exposed for short periods to only a 50 ppm fluoride solution.

The benefit of recharging glass ionomers and fluoride-releasing composite resins has been demonstrated in vivo using well-defined artificial lesions placed in the interproximal aspects of crowns and opposing fluoridated restorative materials.[28] Changes in the lesional areas due to the fluoride-releasing materials were determined in the absence and presence of fluoridated toothpaste. In a relatively short period in the oral cavity, twice daily exposure to the fluoridated toothpaste for one minute resulted in a reduction in lesional area of about 10 percent for a fluoride-releasing composite resin and about 5 percent for a glass ionomer. In another laboratory study,10 fluoridated toothpaste used in concert with a fluoride-releasing resin and glass ionomer reduced the lesional areas by 18 and 14 percent, respectively. The ability to recharge fluoride-containing restorative materials with fluoridated dentifrices provides continuous low-level fluoride release that may prevent secondary caries in the restored tooth and primary caries in both the restored tooth and neighboring tooth, and remineralize existing caries and hypomineralized tooth structure.

## Caries Preventive Mechanisms of Fluoride-Releasing Materials

Fluoride-releasing dental materials prevent secondary caries by several different mechanisms (Table 6).[34-36,50,53,55,66,76,101,104] The release of fluoride into the local environment may inhibit or slow the process of demineralization. As little as 1 ppm fluoride in demineralizing, acidic and plaque fluids reverses the demineralization process. Fluoride released from restorative materials may coat hydroxyapatite crystals that form the mineral substance of enamel, dentin, and cementum. Although fluorapatite has the greatest degree of acid resistance, the acid solubility of hydroxyapatite with a fluoride coating or veneer approaches that for fluorapatite. Such fluoridated hydroxyapatite may be formed in the presence of fluoride-releasing dental materials and provide even greater caries resistance than native tooth structure. Remineralization of lesions and hypomineralized tooth structure is facilitated by very low levels of fluoride (≥0.03 ppm) in saliva and plaque fluid. As noted previously, bacteria in dental plaque have several enzyme systems that are dysregulated by hydrogen fluoride derived from plaque. These enzyme systems are necessary for glycolysis and energy production by the bacteria. Fluoride-releasing materials provide a source for continuous fluoride that elevates salivary and plaque levels and adversely affects plaque bacteria. Fluoride releasing materials may act as continuous low-level fluoride delivery systems, especially when "recharged" by readily available exogenous fluoride sources. Finally, an intimate interface with a minimal to no microspace between the restorative material and cavosurface may be enhanced by physicochemical bonding with glass ionomer-based materials and by mechanical bonding with composite resins.

### *Appropriate Utilization of Polyacid-Modified Resin-Based Composites*

1. Class III restorations in primary and permanent dentition in mild- to moderate-risk patients.
2. Class V restorations in primary and permanent dentition in mild- to moderate-risk patients.
3. Class I restorations in primary teeth in mild- to moderate-risk patients.
4. Class II restorations in primary teeth where preparation remains within the proximal line angles and/or patient is at mild to moderate risk.

### *Appropriate Utilization of Glass Ionomer or Resin-Modified Glass Ionomer Cements*

1. Root caries restorative material.
2. Base/liner in moderate risk patients.
3. Restorative material in high-risk adult patients.
4. Class III restorations in primary and permanent dentition where tooth isolation is not possible and/or patient is at moderate to high risk.
5. Class V restorations in primary and permanent dentition where tooth isolation is not possible and/or patient is at moderate to high risk.
6. Class I restorations in primary teeth where tooth isolation is not possible and/or patient is at moderate risk.
7. Class II restorations in primary teeth where tooth isolation is not possible and the preparation remains within the proximal line angles and/or patient is at moderate risk.
8. Cementation of orthodontic bands.
9. Cementation of crowns in moderate to high-risk patients.

## References


1. Alani AH, Toh CG, Detection of microleakage around dental restorations: a review. Oper Dent 22:173-85, 1997.
2. Arends J, Dijkman GEHM, Dijkman AG, Review of fluoride release and secondary caries reduction by fluoridating composites. Adv Dent Res 9:367-76, 1995.
3. Arends J, Ruben J, Dijkman AG, The effect of fluoride release from a fluoride-containing composite resin on secondary caries: an in vitro study. Quintessence Int 21:671-4, 1990.
4. Benelli EM, Serra MC, et al, In situ anticariogenic potential of glass ionomer cement. Caries Res 27:280-4, 1993.
5. Bennington PCM, Gillgrass TH, et al, Daily exposure to fluoride mouthrinse produces sustained fluoride release from orthodontic adhesives in vitro. *J Dent* 29:23-9, 2001.
6. Bertacchini SM, Abate PF, et al, Solubility and fluoride release in ionomers and compomers. Quintessence Int 20:193-7, 1999.
7. Brown LJ, Winn DM, White BA, Dental caries, restorations and tooth conditions in U.S. adults, 1988-1991. *J Am Dent Assoc* 127:1315-25, 1996.
8. Burke FJT, Cheung SW, et al, Restoration longevity and analysis of reasons for the placement and replacement of restorations provided by vocational dental practitioners and their trainees in the United Kingdom. Quintessence Int 30:234-42, 1999.
9. Buyukyilmaz T, Ogaard B, Caries-preventive effects of fluoride-releasing materials. Adv Dent Res 9:377-83, 1995.
10. Bynum AM, Donly KJ, Enamel de/remineralization on teeth adjacent to fluoride releasing materials without dentifrice exposure. ASDC *J Dent* Child 66:89-92, 1999.
11. Carvalho AS, Cury JA, Fluoride release from some dental materials in different solutions. Oper Dent 24:14-9, 1999.
12. Creanor SL, Awawdeh LA, et al, The effect of a resin-modified glass ionomer restorative material on artificially demineralised dentine caries in vitro. *J Dent* 26:527-31, 1998.
13. Croll TP, Bar-Zion et al, Clinical performance of resin-modified glass ionomer cement restorations in primary teeth: a retrospective evaluation. *J Am Dent Assoc* 132:1110-1116, 2001.
14. DeMaeyer EAP, Verbeeck RMH, Vercruysse CWJ, Reactivity of fluoride-containing calcium aluminosilicate glasses used in dental glass-ionomer cements. *J Dent* Res 77:2005-11, 1998.
15. DeMaeyer EAP, Verbeeck RMH, Vercruysse CWJ, Stoichiometry of the leaching process of fluoride-containing aluminosilicate glass-ionomer glasses. *J Dent* Res 78:1312-8, 1999.
16. Dijkman GEHM, Arends J, Secondary caries in situ around fluoride-releasing light-curing composites: a quantitative model investigation on four materials with a fluoride content between 0 and 26 vol%. Caries Res 26:351-7, 1992.
17. Dijkman GEHM, de Vries J, et al, Long-term fluoride release of visible light-activated composites in vitro: a correlation with in situ demineralisation data. Caries Res 27:117-23, 1993.
18. Dionysopoulos P, Kotsanos N, et al, Secondary caries formation in vitro around fluoride-releasing restorations. Oper Dent 19:183-8, 1994.
19. Dionysopoulos P, Kotsanos N, Papagadiannis Y, Secondary caries formation in vitro around glass ionomer-lined amalgam and composite restorations. J Oral Rehab 23:511-9, 1996.
20. Dionysopoulos P, Kotsanos N, et al, Artificial secondary caries around two new F-containing restoratives. Oper Dent 23:81-6, 1998.
21. Donly KJ, Enamel and dentin demineralization inhibition of fluoride-releasing materials. Am *J Dent* 7:275-8, 1994.
22. Donly KJ, Grandgenett C, Dentin demineralization inhibition at restoration margins of Vitremer, Dyract and Compoglass. Am *J Dent* 11:245-8, 1998.
23. Donly KJ, Ingram C, An in vitro caries inhibition of photopolymerized glass ionomer liners. ASDC *J Dent* Child 64:128-30, 1997.
24. Donly KJ, Istre S, Istre T, In vitro enamel remineralization at orthodontic band margins cemented with glass ionomer cement. Am *J Orthod* Dentofacial Orthop 107:461-4, 1995.
25. Donly KJ, Nelson JJ, Fluoride release of restorative materials exposed to a fluoridated dentifrice. ASDC *J Dent* Child 64:249-50, 1997.
26. Donly KJ, Segura A, Fluoride release and caries inhibition associated with a resin-modified glass ionomer cement at varying fluoride loading doses. Am *J Dent* 15:8-10, 2002.
27. Donly KJ, Segura A, et al, Clinical performance and caries inhibition of resin-modified glass ionomer cement and amalgam restorations. *J Am Dent Assoc* 130:1459-66, 1999.
28. Donly KJ, Segura A, et al, Evaluating the effects of fluoride-releasing dental materials on adjacent interproximal caries. *J Am Dent Assoc* 130:817-25, 1999.
29. Donly KJ, Stufflebeam M, Garcia-Godoy F, Effect of topical fluoride application before and after amalgam restoration placement on recurrent caries inhibition. Am *J Dent* 11:151-3, 1998.
30. Eichmiller FC, Marjenhoff WA, Fluoride-releasing dental restorative materials. Oper Dent 23:218-28, 1998.
31. Erickson RL, Root surface treatment with glass ionomers and resin composites. Am *J Dent* 7:270-85, 1994.
32. Eronat N, Kocatas N, Alpoz AR, A comparative study of fluoride uptake from dentin bonding agents and glass ionomer cements in permanent and primary tooth enamel. Quintessence Int 30:496-500, 1999.
33. Evans CA, Kleinman DV, The surgeon general's report on America's oral health: opportunities for the dental profession. *J Am Dent Assoc* 131:1721-8, 2000.
34. Ewoldsen N, Herwig L, Decay-inhibiting restorative materials: past and present. Compendium 19:981-90, 1998.
35. Featherstone JDB, The science and practice of caries prevention. *J Am Dent Assoc* 131:887-99, 2000.
36. Featherstone JDB, Prevention and reversal of dental caries: role of low level fluoride. Community Dent Oral Epidemiol 27:31-40, 1999.
37. Ferracane JL, Mitchem JC, Adey JD, Fluoride penetration into the hybrid layer from a dentin adhesive. Am *J Dent* 11:23-8, 1998.
38. Fontana M, Secondary caries: relation with current criteria used to replace restorations. General Dent 43:143-54, 1995.
39. Forss H, Seppa L, Studies on the effect of fluoride released by glass ionomers in the oral cavity. Adv Dent Res 9:389-93, 1995.
40. Forsten L, Fluoride release and uptake by glass-ionomers and related materials and its clinical effect. Biomaterials 9:503-8, 1998.
41. Francci C, Deaton TG, et al, Fluoride release from restorative materials and its effects on dentin demineralization. *J Dent* Res 78:1647-54, 1999.
42. Gao W, Smales RJ, Gale MS, Fluoride release/uptake from newer glass-ionomer cements used with the ART approach. Am *J Dent* 13:201-4, 2000.
43. Garcia-Godoy F, Flaitz CM, Hicks MJ, Secondary caries adjacent to amalgam restorations lined with a fluoridated dentin desensitizer. Am *J Dent* 11:254-8, 1998.
44. Garcia-Godoy F, Jensen ME, Artificial recurrent caries in glass ionomer-lined amalgam restorations. Am *J Dent* 3:89-93, 1990.
45. Geiger SB, Weiner S, Fluoridated carbonatoapatite in the intermediate layer between glass ionomer and dentin. Dent Mater 9:33-6, 1993.
46. Glasspoole EA, Erickson RL, Davidson CL, A fluoride-releasing composite for dental applications. Dent Mater 17:127-33, 2001.
47. Hallgren A, Oliveby A, Twetman S, Fluoride concentrations in plaque adjacent to orthodontic appliances retained with glass ionomer cement. Caries Res 27:51-4, 1993.
48. Hicks J, Flaitz C, Caries development in vitro in primary tooth enamel adjacent to glass ionomer. *Pediatr Dent* 22:248, 2000.
49. Hicks J, Flaitz C, Root surface caries development in vitro adjacent to glass ionomer restorative material. *J Dent* Res 80(AADR Abstracts):37, 2001.
50. Hicks MJ, Flaitz CM, Caries-like lesion formation around fluoride-releasing sealant and glass ionomer. Am *J Dent* 5:329-34, 1992.
51. Hicks MJ, Flaitz CM, Epidemiology of dental caries in the pediatric and adolescent population: a review of past and current trends. J Clin *Pediatr Dent* 18:43-9, 1993.

52. Hicks MJ, Flaitz CM, Caries formation in vitro around a fluoride-releasing pit and fissure sealant in primary teeth. ASDC *J Dent* Child 65:161-8, 1998.
53. Hicks MJ, Flaitz CM, Resin-modified glass-ionomer restorations and in vitro secondary caries formation in coronal enamel. Quintessence Int 31:570-8, 2000.
54. Hicks MJ, Flaitz CM, Garcia-Godoy F, Fluoride-releasing sealant and caries-like enamel lesion formation in vitro. J Clin *Pediatr Dent* 24:215-9, 2000.
55. Hicks J, Garcia-Godoy F, Compomer materials and secondary caries formation. Am *J Dent* 13:231-4, 2000.
56. Hodges DJ, Mangum FI, Ward MT, Relationship between gap width and recurrent dental caries beneath occlusal margins of amalgam restorations. Community Dent Oral Epidemiol 23:200-4, 1995.
57. Jokstad A, Mjor IA, Qvist V, The age of restorations in situ. Acta Odontol Scand 52:234-42, 1994.
58. Karantakis P, Helvatjoglou-Antoniades M et al, Fluoride release from three glass ionomers, a compomer, and a composite resin in water, artificial saliva, and lactic acid. Oper Dent 25:20-5, 2000.
59. Kidd EAM, Caries diagnosis within restored teeth. Adv Dent Res 4:10-3, 1990.
60. Kidd EAM, Caries management. Dental Clinics North America 43:743-63, 1999.
61. Kidd EAM, Joyston-Bechal S, Beighton D, Diagnosis of secondary caries: a laboratory study. Br Dent J 176:135-9, 1994.
62. Kidd EAM, Toffenetti F, Mjor IA, Secondary caries. Int *J Dent* 42:127-38, 1992.
63. Kozai, K, Suzuki J, et al, In vitro study of antibacterial and antiadhesive activities of fluoride-containing light-cured fissure sealants and a glass ionomer liner/base against oral bacteria. ASDC *J Dent* Child 67:117-22, 2000.
64. Kreulen CM, de Soet JJ, et al, In vivo cariostatic effect of resin modified glass ionomer cement and amalgam on dentine. Caries Res 31:384-9, 1997.
65. Kupietzky A, Houpt M et al, Fluoride exchange from glass ionomer preventive resin restorations. *Pediatr Dent* 16:340-5, 1994.
66. Leinfelder KF, Dentin adhesives for the twenty-first century. Dent Clin N Am 45:1-27, 2001.
67. Marchiori S, Baratieri LN, et al, The use of liners under amalgam restorations: an in vitro study of marginal leakage. Quintessence Int 29:637-42, 1998.
68. Marinelli CB, Donly KJ, et al, An in vitro comparison of three fluoride regimens on enamel remineralization. Caries Res 31:418-22, 1997.
69. McGuire SM, Fox CH, Douglass CW, Beneath the surface of coronal caries: primary decay, recurrent decay, and failed restorations in a population-based survey of New England elders. J Public Health Dent 53:26-82, 1993.
70. Mjor IA, The location of clinically diagnosed secondary caries. Quintessence Int 29:313-7, 1998.
71. Mjor IA, The reasons for replacement and the age of failed restorations in general dental practice. Acta Odontol Scand 55:58-63, 1997.
72. Mjor IA, Moorhead JE, Selection of restorative materials, reasons for replacements, and longevity of restorations in Florida. J Am Coll Dentists 65:27-33, 1998.
73. Mjor IA, Qvist V, Marginal failures of amalgam and composite restorations. *J Dent* 25:25-30, 1997.
74. Mjor IA, Reep RL, et al, Change in size of replaced amalgam restorations: a methodological study. Oper Dent 23:272-7, 1998.
75. Mjor IA, Toffenetti F, Secondary caries: a literature review with case reports. Quintessence Int 31:165-79, 2000.
76. Mount GJ, Glass ionomer cements: past, present and future. Oper Dent 19:82-90, 1994.
77. Muller U, Kielbassa AM, et al, Fluoride release from light-curing restorative materials. Am *J Dent* 13;301-4, 2000.
78. Naasan MA, Watson TF, Conventional glass ionomers as posterior restorations. Am *J Dent* 11:36-45, 1998.
79. Nagamine M, Alim NN, et al, Inhibition of carious lesions in vitro around gallium alloy restorations by fluoride releasing resin-ionomer cement. Dental Materials J 18:42-53, 1998.
80. Nagamine M, Itota T, et al, Effect of resin-modified glass ionomer cements on secondary caries. Am *J Dent* 10:173-8, 1997.
81. Ozer L, Thylstrup A, What is known about caries in relation to restorations as a reason for replacement? A review. Adv Dent Res 9:394-402, 1995.
82. Pereira PNR, Inokoshi S, Tagami J, In vitro secondary caries inhibition around fluoride releasing materials. *J Dent* 26:505-10, 1998.
83. Pimenta LAF, Fontana UF, et al, Inhibition of demineralization in vitro around amalgam restorations. Quintessence Int 29:363-7, 1998.
84. Pimenta LAF, Navarro MFL, Consolaro A, Secondary caries around amalgam restorations. J Prosthet Dent 74:219-22, 1995.
85. Pink FE, Minden NJ, Simmonds S, Decisions of practitioners regarding placement of amalgam and composite restorations in general practice settings. Oper Dent 19:127-32, 1994.
86. Qvist V, Lauberg I, et al, Longevity and cariostatic effect of everyday conventional glass-ionomer and amalgam restorations in primary teeth: three-year results. *J Dent* Res 76:1387-96, 1997.
87. Ravald N, Birkhed D, Hamp S-E, Root caries susceptibility in periodontally treated patients: results after 12 years. J Clin Periodontol 20:124-9, 1993.
88. Ravald N, Hamp S-E, Prediction of root surface caries in patients treated for advanced periodontal disease. J Clin Periodontol 8:400-14, 1981.
89. Ravald N, Hamp S-E, Birkhed D, Long-term evaluation of root surface caries in periodontally treated patients. J Clin Periodontol 13:758-67, 1986.
90. Reich E, Lussi A, Newbrun E, Caries-risk assessment. Int Dent J 49:15-26, 1999.
91. Reiker J, van der Velden U, et al, A cross-sectional study into prevalence of root caries in periodontal maintenance patients. J Clin Periodontol 26:26-32, 1999.
92. Retief DH, Bradley EL, et al, Enamel and cementum fluoride uptake from a glass ionomer cement. Caries Res 18:250-7, 1984.
93. Segura A, Donly KJ, Quackenbush B, In vitro dentin demineralization inhibition effect of an experimental fluoridated HEMA and water wetting agent. J Oral Rehab 27:532-7, 2000.
94. Serra MC, Cury JA, The in vitro effect of glass-ionomer cement restoration on enamel subjected to a demineralization and remineralization model. Quintessence Int 23:143-7, 1992.
95. Skartveit L, Riordan PJ, Dallal E, Effect of fluoride in amalgam on secondary caries incidence. Community Dent Oral Epidemiol 22:122-5, 1994.
96. Skartveit L, Wefel JS, Ekstrand J, Effect of fluoride amalgams on artificial recurrent enamel and root caries. Scand *J Dent* Res 99:287-94, 1991.
97. Strother JM, Kohn DH, et al, Fluoride release and re-uptake in direct tooth colored restorative materials. Dent Mater 14:129-36, 1998.
98. Svanberg M, Class II amalgam restorations, glass-ionomer tunnel restorations, and caries development on adjacent tooth surfaces: a 3-year clinical study. Caries Res 26:315-8, 1992.
99. Svanberg M, Krasse B, Ornerfeldt H-O, Mutans streptococci in interproximal plaque from amalgam and glass ionomer restorations. Caries Res 24:133-6, 1990.
100. Tantbirojn D, Douglas WH, Versluis A, Inhibitive effect of a resin-modified glass ionomer cement on remote enamel artificial caries. Caries Res 31:275-80, 1997.
101. ten Cate JM, Current concepts on the theories of the mechanism of action of fluoride. Acta Odontol Scanc 57:325-9, 1999.
102. ten Cate JM, Buijs MJ, Damen JJM, The effects of GIC restorations on enamel and dentin demineralization and remineralization. Adv Dent Res 9:384-8, 1995.
103. ten Cate JM, van Duinen RNB, Hypermineralization of dentinal lesions adjacent to glass-ionomer cement restorations. *J Dent* Res 74:1266-71, 1995.
104. ten Cate JM, van Loveren C, Fluoride mechanisms. Dental Clinics North America 43:713-42, 1999.
105. Torii Y, Itota T et al, Inhibition of artificial secondary caries in root by fluoride-releasing restorative materials. Oper Dent 26:36-43, 2001.
106. Thylstrup A, When is caries caries, and what should we do about it? Quintessence Int 29:594-8, 1998.
107. Valenzuela VS, Barca AM, In vitro inhibition of marginal caries-like lesions with fluoride-containing amalgam. Oper Dent 19:91-6, 1994.
108. Veira AR, De Souza IPR, Modesto A, Fluoride uptake and release by composites and glass ionomers in a high caries challenge situation. Am *J Dent* 12:14-8, 1999.
109. Vermeersch G, Leloup G, Vreven J, Fluoride release from glass-ionomer cements, compomers and resin composites. J Oral Rehab 28:26-32, 2001.
110. Vilkinis V, Horsed-Bindslev P, Baelum B, Two-year evaluation of Class II resin-modified glass ionomer cement/composite open sandwich and composite restoration. Clin Oral Invest 4:133-9, 2000.
111. Warren JJ, Cowen HJ et al, Dental caries prevalence and dental care utilization among the very old. *J Am Dent Assoc* 131:1571-9, 2000.
112. Wendt L-K, Koch G, Birkhed D, Replacement of restorations in the primary and young permanent dentition. Swed Dent 22:149-55, 1998.
113. White BA, Albertini TF, et al, Selected restoration and tooth conditions: United States, 1988-1991. *J Dent* Res 75(Spec Iss):661-7, 1996.
114. Williams JA, Billington RW, Pearson GJ, A long term study of fluoride release from metal-containing conventional and resin-modified glass ionomer cements. J Oral Rehab 28:41-7, 2001.
115. Wilson NHF, Burke FJT, When should we restore lesions of secondary caries and with what materials? Quintessence Int 29:598-600, 1998.
116. Wilson NHF, Burke FJT, Mjor IA, Reasons for placement and replacement of restorations of direct restorative materials by a selected group of practitioners in the United Kingdom. Quintessence Int 28:245-8, 1997.
117. Winn DM, Brunelle JA, et al, Coronal and root caries in the dentition of adults in the United States, 1988-1991. *J Dent* Res 75(Spec Iss):642-51, 1996.
118. Yamamoto H, Iwami Y, et al, Fluoride uptake around cavity walls: two-dimensional mapping by electron probe microanalysis. Oper Dent 25:104-12, 2000.
119. Yip H-K, Smales RJ, Fluoride release from a polyacid-modified resin composite and 3 resin-modified glass-ionomer materials. Quintessence Int 31:261-6, 2000.
120. York AK, Arthur JS, Reasons for placement and replacement of dental restorations in the United States Navy Dental Corps. Oper Dent 18:203-8, 1993.

To request a printed copy of this article, please contact: John Hicks, DDS, MS, PhD, MD, Department of Pathology, MC1-2261, Texas Children's Hospital, 6621 Fannin St., Houston, TX 77030-2399 or mjhicks@texaschildrenshospital.org.

CDA JOURNAL, VOL 31, Nº3

# The Paradigm Shift in the Etiology, Prevention, and Management of Dental Caries: Its Effect on the Practice of Clinical Dentistry

Ray E. Stewart, DMD, MS, and Kevin J. Hale, DDSCatherine Flaitz, DDS, MS

**ABSTRACT** Treatment of dental caries as an infectious disease will require a paradigm shift in the way dentists and other health care professionals approach prevention and management of the disease. Prevention of dental caries has relied upon patient cooperation and often requires significant lifestyle changes that are at best difficult to implement and maintain or at worst ignored. This paradigm shift in the etiology, prevention, and treatment of dental caries demands that clinicians redirect their energies and emphasis from the "surgical" approach to dental caries to a "medical" strategy that focuses on early (prenatal if possible) risk assessment of the mother and implementation of appropriate therapeutic intervention including use of antimicrobials, early risk assessment of infants at 6 months of age, and a reduction in the levels of caries-producing bacteria. This revolution in how dentists practice and think will require that they develop strategies and curriculum to "retrain" practicing dentists and to train current and future dental students and residents as well as other medical colleagues on the essentials of the paradigm shift.

CDA JOURNAL, VOL 31, Nº3

AUTHORS

**Ray E. Stewart, DMD, MS,** is in private practice of pediatric dentistry in Salinas, Calif. He is a past president of the California Society of Pediatric Dentistry and currently serves the trustee of the Western Society of Pediatric Dentistry for the 10 western states in the American Academy. He is an associate professor of pediatric dentistry at the University of California at San Francisco School of Dentistry.

**Kevin J. Hale, DDS,** is in private practice of pediatric dentistry in Brighton, Mich. He is also a faculty member in the Department of Pediatric Dentistry at the University of Michigan.

The fact that dental caries is an infectious and transmissible disease has been known but not widely acknowledged for more than four decades.1 Dental caries arises from an overgrowth of specific organisms that are part of normally occurring human dental flora.2 The mutans streptococci group, which includes Streptococcus mutans and Streptococcus sobrinus, as well as several lactobacilli species are considered to be the principal groups of bacteria that are acidogenic (acid producing) as well as aciduric (acid tolerant) and are responsible for dental caries. Human dental flora is site-specific; and an infant is most readily colonized with normal dental flora after the eruption of the primary dentition, which usually occurs from 6 to 30 months of age.3,4 However, recent studies have shown that some colonization occurs even before teeth erupt. The vertical transmission of S. mutans from mother to infant is well-documented.5,6 In one study, it was shown that genotypes of S. mutans in infants matched those present in mothers in approximately 71 percent of mother-infant pairs.7 Furthermore, another study has suggested that there may be a window of infectivity between about 18 and 36 months of age during which much of the S. mutans colonization occurs.8 Later studies have shown transmission before and after this age range, and also from child to child as well as from adult to adult.

The significance of this information becomes apparent when considering three points. First, high caries index patterns run in families9 and are passed vertically from mother to child from generation to generation. The children of high-caries-index mothers are at a higher risk of decay.10 Secondly, approximately 70 percent of caries is found in 30 percent of our nation's children.11 Lastly, the modification of the mother's dental flora at the time of inoculation of the child can significantly affect the child's caries index.12-14

Treatment of dental caries as an infectious disease will require a paradigm shift in the way dentists and other health care professionals approach prevention and management of the disease. Prevention of dental caries has relied upon patient cooperation and often requires significant lifestyle changes (e.g., rigorous oral hygiene, attention of dietary habits, attention to infant feeding practices) that are at best difficult to implement and maintain or at worst ignored.

The historical approach to the management of dental caries, once demineralization and cavitations have occurred, consists of the removal of the diseased tissue (enamel and dentin) and replacing it with a restorative material such as amalgam, precious metal, or one of the newer composite materials. In extreme cases where bacterial necrosis of the dental pulp has occurred, the method of treatment becomes surgical removal of the tooth or endodontic therapy followed by prosthetic restoration.

In all of these classical restorative approaches to caries management, the basic cause of the disease -- the specific acidogenic bacteria in the plaque biofilm on the remainder of the teeth in the mouth -- remains intact and capable of creating new areas of decalcification and eventual cavitation should the enabling environmental modifiers be present.

Dentists are justifiably proud of and proficient in the art and science of restorative dentistry and of their ability to deliver these services in a relatively comfortable and safe fashion to the very young or apprehensive patient.

This paradigm shift in the etiology, prevention, and treatment of dental caries demands that clinicians redirect their energies and emphasis from the "surgical" approach to dental caries to a "medical" strategy that focuses on:

- Early (prenatal if possible) risk assessment of the mother and implementation of appropriate therapeutic intervention including use of antimicrobials;
- Early risk assessment of infants at 6 months of age; and
- A reduction in the levels of caries-producing bacteria.

This revolution in how dentists practice and think will require that they develop strategies and curriculum to "retrain" practicing dentists and to train current and future dental students and residents as well as other medical colleagues on the essentials of the paradigm shift.

The nature of early childhood caries with its early onset and rapid progression should make it intuitive to health care practitioners that this disease is not exclusively a "dental" problem. Indeed, if dentists are to have any measurable effect on the reduction in the numbers of early childhood caries cases, it is essential that they enlist the assistance and collaboration of their colleagues in medicine. Because pediatricians, pediatric nurse practitioners, and other pediatric health care professionals are far more likely to encounter new mothers and infants than are dentists, it is essential that they be aware of the infectious pathophysiology and associated risk factors of early childhood caries to make appropriate decisions regarding timely and effective intervention. Dentists must educate them as to the scientific and rationale validity of the paradigm shift and encourage them to acquire the knowledge that will enable them to administer an oral health risk assessment and perform an infant oral exam beginning at 6 months of age for those patients who are at moderate to high risk for caries and to direct the parent to establish a dental home for the infant by 1 year of age.

## Primary Prevention and Management of Dental Caries

Once the infectious disease paradigm for the etiology of dental caries is accepted, the medical approach to the management of the caries process, which focuses on the prevention, becomes obvious. The medical strategy for preventing dental decay consists of the systematic application of the following practices:

- Assessing the caries risk of the intimate care provider (usually the mother) and the infant;
- Conducting an infant oral screening examination; and
- Delaying or reducing bacterial colonization of the infant by lowering levels of cariogenic/acidogenic bacteria in the colonization source (mother or caregiver) as well as in the infant.

## Caries Risk Assessment

All health care professionals who treat infants and children must be capable of conducting a caries risk assessment of the infant and mother or other intimate caregiver.

The primary thrust of caries risk assessment is to determine the virulence and/or caries expression of the dental bacterial clone set of the mother/infant pair. For any assessment tool to be widely adopted and used, it must be user-friendly, quick, and easy to interpret. Furthermore, it must meet these criteria in a variety of settings, e.g., dental offices; school screenings; pediatricians' offices; Women, Infants, and Children Program centers; community health centers; etc. Several caries risk assessment tools have been published. This issue of the Journal of the California Dental Association includes a caries risk consensus statement and a series of forms and instructions developed by a group brought together in April 2002 specifically to update and review the literature and develop these practical tools. These forms and instructions provide a template for use in clinical practice. A simple assessment of the mother's caries history can be performed by obtaining a dental history and examining her dentition. Mothers with active decay and/or multiple fillings in multiple quadrants of the mouth are at higher risk than those who have never experienced decay or have but one or two restorations.

Regardless of the parent's caries activity, it is generally agreed that infants who fall into one of the following categories should be considered high risk and referred to a dental home by age 1.

- Children who are medically compromised;
- Children of mothers with a high caries rate;
- Children with demonstrable caries, plaque, demineralization, and/or staining;
- Children who sleep with a bottle or at the breast all night;
- Later -- older siblings of a parent with mildly to moderately high caries rate; and
- Children in families of low socioeconomic status.

## Infant Oral Screening Exam

It is essential that both dental and nondental health professionals who see infants and children be trained and capable of doing oral health risk assessment and dental screening examinations as part of the infant's pediatric health care visit. This oral screening examination should be a matter of routine, starting at about 6 months of age or after the maxillary incisors have erupted.

Prior to the eruption of the primary

CDA JOURNAL, VOL 31, Nº3

incisors, the usual information discussed in neonatal anticipatory guidance -- such as teething, fluoride topical sources/fluoride therapy, and non-nutritive sucking -- should be reviewed.

Once the maxillary incisors have erupted, it is important to include examinations of these structures as part of the routine periodic physical examination. The examination can conveniently be done on the examining table or in the "knee to knee" position with the parent and practitioner sitting facing one another with knees touching. The parent holds the infant facing her and slowly and gently lowers the child's head onto the examiner's lap, while restraining the baby's legs under his or her arms and holding the baby's hands. In this position, the infant will be well-stabilized and firmly supported with his or her face and mouth clearly visible to the examiner. To properly examine the infant's mouth and teeth, there should be a good light source, a disposable mouth mirror, and a soft-bristled toothbrush. The infant's reaction to the examination can vary and it is not unusual for them to cry and resist the procedure, so parents should be assured beforehand that this is a normal response.

The practitioner should lift the upper lip and examine the teeth, checking for the presence of plaque; "white lesions" or cavitations; and abnormal tooth structure (enamel hypoplasia, fusion/germination), tooth eruption, or soft tissues.

Using a soft-bristled toothbrush, the practitioner should brush the teeth using a circular scrubbing motion to remove any plaque. Following plaque removal, any white spots, stains, decay, or pitted enamel should be noted. Using a mouth mirror to see, the practitioner should similarly clean and inspect the posterior surface.

If significant plaque is present on any of the teeth or they are noted to have white lesions or areas of decalcification, the infant should be considered at high risk for caries. As such, the infant should be referred to a dental home where he or she will be seen by a dentist who treats infants and toddlers.

If no abnormalities are present and the teeth are free of plaque or white lesions, the infant is at lower risk of the development of caries. The parents should be instructed to brush the child's teeth daily. They should be encouraged to have the child initiate regular visits with the family's dentist or a pediatric dentist by about 1 year of age so the responsibility for periodic risk assessment and oral screening can be assumed by dental personnel.

If no dentist can be identified who will see the child before 3 or 4 years of age, the pediatrician, family physician, or appropriately trained personnel should assume the responsibility for the risk assessment and screenings as part of the periodic preventive health care visits. In these circumstances, these nondental personnel must be trained to implement intervention strategies such as the application of antimicrobials, fluoride therapy, etc.

## Delay Bacterial Colonization

The goal of the third tier of caries prevention is to delay colonization of the infant by the cariogenic bacteria while lowering the levels of these organisms in the colonization source (mother or caregiver). Delay of colonization can be accomplished by cautioning the parent to avoid bacterial inoculation practices (e.g., sharing toothbrushes, sharing eating utensils, cleaning pacifier with saliva, etc.).15 Limiting the levels of acidogenic/cariogenic flora in the mother prior to and during the colonization process in the infant can have a significant positive impact on the child's caries rate.12,13 Strategies include:

- Removal of all active caries and reducing the levels of cariogenic flora in the remainder of the mouth through the use of appropriate therapeutic measures including restorative procedures and antimicrobials;
- Administration of meticulous oral hygiene;
- Judicious administration of fluorides;
- Avoidance of simple sugars between meals in a frequent and/or protracted fashion; and
- Utilization of xylitol chewing gum, four times a day.14

## Restorative Dentistry

Only carious lesions that are active, frank, and cavitated require the surgical intervention of operative dentistry. Carious lesions that are not active, frank, and cavitated -- such as "white spot lesions" and/or incipient lesions -- are best addressed using the medical approach. This medical management of caries is similar to current medical management of other diseases. The clinician may rely on visual and microbiological diagnosis such as CRT (Caries Resistance Test) by Vivadent (Amherst, N.Y.) to assess the level of bacterial challenge. In concert with the diagnosis, remineralization can be achieved through pharmacotherapeutic interventions such as professionally applied high-concentration fluoride products (foam, varnish, or gel) and home-use topical fluoride products (toothpaste, rinses). In the case of high bacterial challenge, chlorhexidine can be used to control the infection.

## Summary and Recommendations

There are five recommendations as to how dentists might speed acknowledgment and acceptance of the paradigm shift among their peers:

- The paradigm shift -- the concept of early childhood caries as an infectious and preventable disease that is contagious and transmitted to the child from the mother or other close caregiver who harbors high levels of cariogenic flora -- must be aggressively

promoted and taught to all medical and dental providers who have contact with mothers (pre- and postnatal) and infants.

- Educational and teaching tools in various media formats appropriate for a variety of health care professionals must be developed. These materials must focus on the infectious and transmissible nature of early childhood caries, the methods and rationale of oral health risk assessment, infant oral examination, anticipation guidance, and early intervention including the use of antimicrobials and agents that enable the remineralization of early white lesions.
- Every child should receive an oral health risk assessment and oral examination by age 6 months from a qualified, appropriately trained health care professional (dentist, pediatrician, family practice physician, physician's assistant). Infants who fall into one of the following categories should automatically be placed in the high-risk group.
- Children who are medically compromised or have developmental disabilities;
- Children who have mothers or close caregivers at high risk for caries (active decay);
- Children with demonstrable plaque, caries;
- Children who sleep with a nursing bottle or at the breast;
- Children with older siblings with history of early childhood caries; and
- Children from families of low socioeconomic status.
- The American Academy of Pediatrics should be encouraged to support the concept of the a first dental visit by age 1 year and the establishment of a dental home as an independent and specialized primary care provider similar to the medical home concept promoted by the Academy.
- Last but not least, there needs to be a coincidental paradigm shift within the insurance industry and among the governmental agencies, which are responsible for establishing health care policy and regulations. Practitioners should be compensated for the provision of preventive services such as early caries risk assessment and anticipatory guidance to increase these practices.

## References

1. Keyes PH, The infectious and transmissible nature of experimental dental caries. Arch Oral Biol 1:304-20, 1960.
2. Loesch WJ, Clinical and microbiological aspects of chemotherapeutic agents used according to the specific plaque hypothesis. *J Dent* Res 58:2404, 1979.
3. Berkowitz RJ, Jordan HV, and White G, The early establishment of Streptococcus mutans in the mouths of infants. Arch Oral Biol 20:171-4, 1975.
4. Stiles HM, Meyers R, et al, Occurrence of Streptococcus mutans and Streptococcus sanguis in the oral cavity and feces of young children. In, Stiles M, Loesch WJ, O'Brien T, eds, Microbial aspects of dental caries. Information Retrieval, Washington DC, p 187, 1976.
5. Davey AL, Rogers AH, Multiple types of the bacterium Streptococcus mutans in the human mouth and their intra-family transmission. Arch Oral Biol 29:453, 1984.
6. Berkowitz RJ, Jones P, Mouth-to-mouth transmission of the bacterium Streptococcus mutans between mother and child. Arch Oral Biol 30:377, 1985.
7. Li Y, Caufield PW, The fidelity of initial acquisition of mutans streptococcus by infants from their mothers. *J Dent* Res 74:681, 1995.
8. Caufield PW, Cutter GR, Dasanayake AP, Initial acquisition of Streptococcus mutans by infants: Evidence of a discrete window of infectivity. *J Dent* Res 72(1):37-45, 1993.
9. Klein H, Palmer CE, Studies on dental caries V. Familial resemblance in caries experience of siblings. Pub Health Rep 53:1353, 1938.
10. Klein H, The family and dental disease IV: Dental disease (DMF) experience in parents and offspring. *J Am Dent Assoc* 33:735-43, 1946.
11. Kasta LM, Selxitz RJ, et al, Coronal caries in the primary and permanent dentition of children and adolescents 1-17 years of age: United States, 1988-1989. *J Dent* Res 75(spec iss):631-41, 1996.
12. Kohler B, Andreen I, Jonsson B, The effect of caries preventive measures in mothers on dental caries and the oral presence of bacteria Streptococcus mutans and lactobacilli in their children. Arch Oral Biol 11: 879-883, 1984.
13. Brambilla E, Felloni A, et al, Caries prevention during pregnancy: results of a 30 month study. *J Am Dent Assoc* 129: 871-877, 1998.
14. Isokangas P, Soderling K, et al, Occurrence of dental decay in children after maternal consumption of xylitol chewing gum, a follow-up from 0-5 years of age. *J Dent* Res 79 (11): 1885, 2000.
15. Kohler B, Bratthall D, Intrafamilial levels of streptococcus mutans and some aspects of the bacterial transmission. Scand *J Dent* Res 86:35-42, 1978.

To request a printed copy of this article, please contact: Ray E. Stewart, DMD, MS, 631 E. Alvin Drive, Salinas, CA 93906 and drrstewart@aol.com.

CDA JOURNAL, VOL 31, Nº3

# Caries Protocol Compliance Issues

William F. Bird DDS, DrPH

**ABSTRACT** Any caries control or caries protocol program must consider compliance as a measure of success. Lower bacterial counts on saliva tests and lower defs and DMFS scores suggest that some change has occurred. However, compliance with a caries risk protocol is about more than simple and convenient clinical outcomes measures. We tend to think of compliance as an individual activity, but all influences on an individual need to be considered. Change may be from external influences rather than from the individual or even from providers interacting with an individual or community. Few studies have directly addressed caries risk protocol. The paradigm change described in this manuscript suggests six key global areas -- beneficiary education, health provider network education, community and state agencies, legislative commitment, access to care, and research -- as significant factors to be considered in compliance. An outline of the major areas and subheadings for a global caries protocol compliance paradigm are presented.

When considering caries control compliance, it is clear that most issues of behavior change and patient, provider, and payer compliance have not been adequately studied. Many people seem to put the blame on the patient and caregiver for the lack of compliance with and follow through on treatment and prevention recommendations, but the true cause lies in a complex set of interacting and underlying factors. Only a few studies have discussed compliance with caries treatment/prevention regimens.1-5 This lack of research demonstrates the complexity of behavioral modification and compliance with dental caries reduction regimens. Since behavior change and compliance are so complex, it is not an area of study in dentistry that has received much funding support or, therefore, has a strong scientific basis. More studies will be necessary to validate the efficacy of any protocols that are developed.6 In addition, by looking outside of dentistry, it is possible only to generalize compliance outcomes based on other behavioral science/disease and pharmacological compliance studies.7,8 In studies of compliance with recommended medication therapy, it has been estimated that roughly one-half of medications are taken incorrectly and that nearly 20 percent of prescriptions are not even filled. This has strong implications for the type of caries preventive protocol that is recommended. To prescribe only self-applied processes or

medications will not be effective. Other interventions and follow-up measures are needed. As reported by Haynes and colleagues,8 almost all of the interventions that had some effect on long-term care were complex and included combinations of more convenient care, information, counseling, reminders, self-monitoring, reinforcement, family therapy, and other forms of supervision. Even these did not lead to large improvements in adherence and treatment outcomes.

It is not reasonable to expect a layperson to consider dental health as important as dental professionals do. Therefore, basic dental health education needs to be directed to all education levels. School curricula need to be revised to incorporate the paradigm shift, and community health education and mass media messages need to be expanded. Brochures in the dental offices will not get the message to the needy. Using radio and television ads and technology such as videos and DVDs is necessary to get the message out to the masses. Drug companies are using this approach to promote their prescription medicines.

An example of the difficulties of establishing a caries prevention program is shown in the school-based Head Start xylitol gum pilot program tried in Florida. While children accepted the three times per day chewing of the xylitol gum, teachers indicated that it interfered with the school day; and four out of five indicated that they were not willing to participate in another such program.2 Collaboration of the statewide curriculum committees and education of teachers is necessary to gain acceptance of a supervised school-based caries prevention protocol. Attempts at getting compliance for children through parental consultations and group education programs have not shown much success either.3 Children with a high caries rate who were treated by dentists did not change their behavior as a result of more treatment visits. Those that were less cooperative as judged by the dentist had higher rates of re-treatment needs than did those who were judged more cooperative.1 It is unclear whether this indicates that compliance is worse in the noncooperative child or that there is less effort to integrate preventive interventions into the treatment of such children.

Legislators need to make dental disease as much a priority as other major acute and chronic diseases. Most legislators do not see the thousands of hours of schooling and employment lost to dental disease as a significant problem. In the surgeon general's report, it was estimated that more than 50 million school hours are lost each year due to dental-related illness.9 Frequently, budget cuts for health funds are taken from dental programs. An example of this shortsighted thinking is exhibited in California budget deliberations. Adult Denti-Cal funding is frequently considered for drastic reduction or elimination as a way to save money. Most legislators are not aware of the problems created by transmission of dental caries disease to children from untreated caregivers, usually mothers. It is easier to cut or reduce dental funds because it causes less public outcry than medical program cuts. Even though dental disease is less of a life-threatening condition for the most part, it has a significant impact on overall health. The message that dental decay is a contagious disease needs to be promoted to the insurance industry, funding agencies, and legislators. The decision to reduce allocation of resources for a high-risk disease such as dental decay can have a devastating effect on access to care10 and the development and implementation of programs that can assist in the patient's compliance with the recommended protocols.

Access to care for people with low incomes is abhorrent. Many areas of California have no dentist or so few that it is a major barrier for a patient to receive routine and sometime emergency care, let alone preventive care. Certainly, public health measures such as fluoride can help. However, the disease challenge cannot be eliminated by such measures. External, social, economic, and education barriers contribute to the access to care problem.10 Agencies and insurance providers need to include caries risk testing and preventive treatments as benefits. When medical and dental providers are not compensated for their provision of these services, it translates into a lack of access for the patient. Wider access to preventive risk assessment tests could reduce the severe early childhood caries problem in California.

In addition, there needs to be a continued effort to provide education programs for the physicians, nurses, educators, social services counselors, dentists, and all licensed health care providers concerning the huge dental disease problem and the causes. The paradigm shift information must be transmitted to all. If there is not a concerted educational program, any protocol that is recommended will have only moderate success. Dental, dental hygiene, medical and nursing schools need to prepare their graduates with this information. Most schools have not incorporated these important concepts into their curriculum. This is easily seen by questioning the recent graduates about the paradigm shift and prevention and risk assessment strategies. Many are unaware of them. If this is the case, not only will patient compliance be poor, but provider compliance will also be poor . This needs to be remedied in a strategy to implement the caries risk protocol.

For a caries protocol compliance program to be successful, the following components need to be addressed.

## Beneficiary Education

Individual/family/caregiver -- A positive

reinforcement program should be in place. An example would be a "compliance calendar" that can be checked and followed daily and shared with the oral health provider team for each individual and the family.

Educational material development and distribution -- Material needs to be available in appropriate languages and various media, such as CDs, DVDs, videos, and brochures. These educational materials should be focused on a single concept. The DVD included in the February Journal of the California Dental Association is an excellent resource.

School curricula revisions -- Since early learning is so powerful, it is important for the paradigm shift message about caries risk to be included in all messages about oral health. Dentists and hygienists should take an active role in reviewing what is taught at all levels of education and providing guidance to curriculum committees. At the state level, people responsible for curricular oversight need to be informed and urged to take an active role in ensuring that the health components of the curriculum include these current oral health concepts.

Teacher oral health education workshops -- Early exposure to formal education about health, and in particular oral health, is usually provided by teachers. It is important to give them the most accurate and current information. This can be done by in-service or annual education seminars provided by members of the local dental society. Also, it is important that teachers be provided with curricular materials to assist them in their educational efforts.

## Health Provider Network Education

Medical, dental, nursing, hygiene students -- Although many dental and dental hygiene school curricula are changing to include the caries risk paradigm, it is lacking in the curricula for physicians, nurses, and other heath care providers. To promote patient caries protocol compliance, it is imperative that all health care provider schools have a component of their curriculum that addresses the caries risk paradigm.

Physicians, dentists, hygienists, nurses (all licensed categories) -- The existing provider network needs to be informed of the paradigm shift for caries risk management. This can be done through media such as this journal and continuing education programs at national, state and local professional society meetings.

Social workers and counselors -- Since many caries protocol follow-up activities and treatment visits are coordinated through social workers, they can be an important link in achieving compliance.

## Agencies

Development of local and community action programs -- Public health workers must be involved in the planning of education programs in their communities. School-based or community clinic-based prevention and caries risk programs will have a better chance of succeeding and getting compliance from all concerned if public health workers are part of the team.

Third-party and state indemnity/insurance program changes -- It is important for third-party and state indemnity/medical providers to include payment for caries risk tests and prevention programs. The money spent on preventive services and tests will reduce the severity of dental caries and ultimately the cost for care. Providers can have a great impact on implementing the caries risk assessment and early treatment programs and monitoring the compliance of their patients, but they must be compensated for their time and materials.

## Legislative Commitment

Grant support for community action programs -- It is well-established that money spent through the grant process has a great impact on the ability of a community to initiate programs. Grants aimed at developing community action caries risk assessment and treatment programs should be supported by federal and state agencies. There should be mechanisms for communities to develop unsolicited proposals for grants to fund such initiatives. Once begun, the benefits of such programs will become recognized, and their continuance will become important to the people of the communities.

Recognition of the need to increase funding to support program and treatment efforts -- This is largely dependent upon the various professional societies and individual providers getting the attention of legislators to support various program initiatives by schools and communities. Legislators are usually not health care providers, so they also need to be educated as to the benefits of risk assessment and education programs.

Scholarships for providers to go to underserved areas of the state -- Since a great deal of untreated caries is seen in the areas where there are few if any health care providers, in particular dentists, one way to encourage providers to locate in underserved areas is to provide dental school scholarships and educational loan forgiveness in return for service in the specified areas. Some of these providers might decide to permanently locate in those areas. Patients cannot comply if they have no access to care.

## Access to Care

For physicians and dentists, inclusion of prevention and risk assessment tests and treatment procedures in the reimbursement schedules by state and insurance agencies is paramount.

By having a reimbursement mechanism for the providers to receive compensation for tests and prevention treatments, it is more likely that they will provide these services. This will ensure an increased level

of care and thus compliance by the patient.

Programs to support dentists' acceptance of low-income patients -- The lack of acceptance of low-income patients for routine care, and particularly risk assessment and treatment programs, is tied to the lack of adequate compensation for providing such care. It would be better to fully fund these prevention and early treatment services than to have a large number of tertiary services covered at a low level of compensation.

Programs to encourage dentists to seek practice in underserved population areas -- Scholarships, educational loan forgiveness programs and licensure by credential are examples of means to encourage dentists to locate in underserved areas.

More dental provider education programs to increase comfort in providing care for young pediatric patients -- There is a shortage of pediatric dentists and general dentists to provide care to infants and children age 0 through 6. One way to increase the access to care for children is to provide pediatric dentistry education programs for general dentists. Having more general dentists being comfortable with providing pediatric dental care will increase the access to care for these children.

Such a program could consist of didactic education multimedia seminars, laboratory simulation for various procedures, a mentoring system and consultation network by pediatric dentist mentors, and a certificate course on sedation techniques.

## Research

Products to control the bacterial disease and transmission -- Industry and academia need to continually develop and certify the effectiveness of new products to reduce decay-causing bacteria.

Simple chairside caries risk tests and assessment -- Current caries risk tests must be incubated for 48 hours or mailed to a testing center/laboratory to obtain the results. This delay impedes the success of the utilization of these tests. A test that can be used at chairside for immediate feedback to the patient or that can be used in field screenings is more likely to ensure compliance with an assessment program. This chairside test needs to be specific for the particular strains of bacteria known to cause the tooth decay.

Studies on caries protocol compliance -- There are very few studies in the literature on the compliance with protocols to reduce the risk of caries by any means. Identification of factors that ensure or impede compliance is needed to aid in the development and refinement of such protocols. This is an area for behavioral scientists to explore. Compliance with dental disease prevention protocols can be greatly improved by this area of research. National funding for such research would be a great asset to initiating such research.

Educational programs evaluation -- For educational programs to be successful, they need to be kept current. Also, the effectiveness of the educational programs and methods of delivery are an important aspect of the message being acted upon and complied with. Continuing research in this area for caries risk assessment/treatment and compliance to protocol can be helpful for the developers of education materials.

## Summary

Many factors contribute to a successful compliance outcome for any health improvement program. Achieving compliance with a dental caries risk assessment protocol is no exception. Patients, parents, providers, educators, legislators, and agencies all need to work together for a successful result. For compliance to occur, everyone concerned needs to understand the important role that he or she has in achieving this success. The provision of information on the paradigm shift about dental decay and prevention is one step. The next step, and the measure of success, is compliance.

### References

1. Varsio S, Vehkalahti M, Murtomaa H, Dental care of six-year-old high-caries patients in relation to their cooperation. Commun Dent Health 16:171-5, 1999.
2. Auito JT, Courts FJ, Acceptance of the xylitol chewing gum regimen by preschool children and teachers in a Head Start program: a pilot study. *Pediatr Dent* 23(1):71-4.
3. Primosch RE, Balsewich CM, Thomas CW, Outcomes assessment an intervention strategy to improve parental compliance to follow-up evaluations after treatment of early childhood caries using general anesthesia in a Medicaid population. ASDC *J Dent* Child 68(2):102-8, 2001.
4. Anusavice KJ, Chlorhexidine, fluoride varnish, and xylitol chewing gum: underutilized preventative therapies? Gen Dent 46(1):34-8, 40, 1998.
5. Caries diagnosis and risk assessment. A review of preventive strategies and management. *J Am Dent Assoc* 126Suppl:1S-24S, 1995.
6. Milgrom P, The impact of behavioral technology on dental caries. *J Dent* Educ 65(10):1102-5, 2001.
7. Kehoe WA, Katz RC, Health behaviors and pharmacotherapy. Ann Pharmacother 32(10):1076-86, 1998.
8. Haynes RB, Montague P, et al, Interventions for helping patients to follow prescriptions for medications. Cocrane Database Syst Rev 2000(2):CD000011.
9. US Department of Health and Human Services, *Oral Health in America*: A report of the Surgeon General. US Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health, Rockville, Md, 2000.
10. Axelsson P, External modifying factors involved in dental caries. In, Diagnosis and Risk Prediction of Dental Caries, vol 2. Quintessence Publishing Co, Carol Stream, Ill, 2000, pp 77-90.
To request a printed copy of this issue, please contact/William F. Bird, DDS, DrPH, UCSF School of Dentistry, 707 Parnassus Ave., Box 0758, San Francisco, CA 94143 or birdb@dentistry.ucsf.edu.

CDA JOURNAL, VOL 31, Nº3

# Caries Management by Risk Assessment: Consensus Statement, April 2002

JOHN D.B. FEATHERSTONE, MSc, PhD; STEVEN M. ADAIR, DDS, MS; MAXWELL H. ANDERSON, DDS, MS, MEd; ROBERT J. BERKOWITZ, DDS; WILLIAM F. BIRD, DDS, DrPH; JAMES J. CRALL, DDS, ScD; PAMELA K. DEN BESTEN, DDS; KEVIN J. DONLY, DDS, MS; PAUL GLASSMAN, DDS, MA, MBA; PETER MILGROM, DDS; JON R. ROTH, CAE; REED SNOW, DDS; RAY E. STEWART, DMD, MS

The following statement is the consensus of a meeting of a group of experts in dental caries, in particular the science and practice of caries prevention, risk assessment, and management, held at the California Dental Association, April 26-27, 2002. Twelve reviews were presented at the meeting, and the many references contained in those reviews form the basis for the following consensus document.1-12 The reader is referred to these reviews for studies that support the following statements.

As a result of that meeting, this consensus summary statement is presented with practical risk assessment forms and instructions for use in caries management by risk assessment in clinical and community settings. Statements of special significance are in bold italics.

## Basic Guiding Principles

The recommendations and guidelines produced by this conference are based on the best scientific information available at the time of the conference, April 2002. They are intended to be a work in progress subject to improvement and modification as new information becomes available. These recommendations and guidelines form the basis for practical caries intervention and prevention both by individuals and communities, and were crafted for use with children as well as with adults. By necessity, specific rules for special-needs groups are not addressed directly, and some modifications may be needed in those cases. Special-needs patients will be addressed at a separate conference, summarized by Glassman.13

The recommendations and guidelines that follow should be implemented as soon as possible for the improvement of oral health of children and adults in California.

### *The Need for Caries Risk Assessment, Caries Intervention, and Caries Management by Risk Assessment*

Although dental decay significantly declined in the United States from the 1960s through the 1980s, it is still a major problem in adults and children. The dramatic reductions in levels of

decay observed from 1960 to 1990 were undoubtedly related initially to the introduction of fluoride into the drinking water and subsequently to topical fluoride applications, especially through fluoridated dentifrice use and dental office topical fluoride.1,2 However, these tools are only successful up to a point, and we now must be thinking of more-aggressive ways to deal with dental caries as a bacterially based transmissible infection.1-3,12 To place this into perspective, a recently published survey on the dental health of California's children, from data that was accumulated in 1993 and 1994, reported that:

- 27 percent of preschool children have untreated decay;
- 55 percent of 6- through 8-year-olds have untreated decay;
- Up to 75 percent of minority high school students need dental care; and
- California's children on average have twice the national level of untreated tooth decay. Many millions of dollars are spent in California each year on the physical treatment of dental caries, rather than on prevention and intervention. Millions of hours are lost at school and work each year as a result of dental caries. There is a growing epidemic of early childhood caries in the United States, particularly in California.1,2,8

If we were dealing with any other disease in the human body, we would use measures to eradicate the cause of the disease, such as antibiotics for systemic infections, or introduce public health measures, such as immunization at the community level. We need to think of dental decay in this same fashion and treat the disease rather than just the results of the disease. There is enough information available to do this.

### *Overall Objectives of the Consensus Document*

This document provides a summary of the components of successful caries risk assessment and the basis for minimally invasive caries management by risk assessment. The American Academy of Pediatric Dentistry is developing an outline instrument for caries risk assessment, but no one yet has truly addressed the infectious disease that is the basis of dental caries. The overall objective of this document is to provide the basis for a cross-disciplinary approach among medicine, dentistry, nursing, and other agencies that affect dental health to reduce or eradicate dental caries in children in every county, community, and culture in California by the year 2010.

The Caries Balance Concept as the Basis for Caries Risk Assessment and Management

Dental caries (dental decay) is a continual balance, or imbalance, between pathological factors and protective factors, as illustrated schematically in Figure 1.1

Pathological Factors

The pathological factors include:

A. The so-called cariogenic (acid-producing, caries-promoting) bacteria that produce acid by fermentation of carbohydrates. The two major groups of cariogenic bacteria involved are the mutans streptococci (S. mutans and S. sobrinus) and several of the lactobacillus species.1,6

B. The frequency of ingestion of fermentable carbohydrates, including sucrose, glucose, fructose, and cooked starch. Frequency of ingestion is the most important factor, rather than total quantity, since repeated ingestion leads to renewed acid production by the bacteria. In young children, the prolonged use of a bottle or a "sippy-cup" containing anything but water provides an almost continual acid challenge to the teeth as the oral bacteria are bathed in the carbohydrates. High-fructose corn syrup is the major sweetener in the United States.

C. Salivary dysfunction caused by factors such as medications, radiation therapy for cancer of the head and neck, some systemic diseases, or genetically induced conditions that result in reduction of salivary function. In young children, medications such as anti-asthma therapy may cause hyposalivation, which is a major risk factor. Pediatricians, parents, caregivers, and health care professionals must be aware of the importance of medication-induced saliva flow reduction as a risk factor.

### Protective Factors

The protective factors include:

A. Almost all of the components of saliva, including buffers that neutralize the acids;

B. Saliva flow for clearance purposes;

C. Fluoride from topical (to the surface of the teeth) sources to provide inhibition of demineralization and enhancement of remineralization;

D. Antibacterial agents in saliva and/or from extrinsic sources or products;

E. Salivary proteins and lipids that form pellicle and protect the tooth surface; and

F. Calcium and phosphate derived either from the saliva or from the dietary sources, such as cheese.

At any one time, the direction of the caries balance can be tipped toward caries progression and demineralization of the tooth mineral or toward repair of the tooth mineral by remineralization as a result of one or more protective factors. The eventual outcome of either progression, reversal, or status quo determines whether an individual tooth surface becomes cavitated. This concept forms the basis for risk assessment and for car-

ies management based upon risk assessment.

## Bacterial Challenge

The bacteria that cause caries (cariogenic bacteria) are primarily from two groups, the mutans streptococci and the lactobacilli species. The two species in the mutans streptococci group that appear in humans are Streptococcus mutans and S. sobrinus. These acid-producing bacteria are necessary for the progression of dental caries. The cariogenic bacteria are transmitted from one individual to another and in particular from mother or caregiver to child in the early stages of childhood.6 Child-to-child and adult-to-adult transmission also occurs. Early transmission and growth of these pathogenic bacteria lead to more decay later, as compared to children who are colonized later.6 Placing restorative materials (fillings) in cavitated caries (holes in the teeth) or in early caries that are drilled out by the dentist restores the function of the tooth but does not significantly reduce the infection in the remainder of the mouth.

### *Sampling Bacteria in the Mouth*

Levels of these cariogenic bacteria in the mouth can be assessed by selective media culturing either in a microbiological laboratory or in the dental office (see below). In the future, monoclonal antibody technology is expected to be available routinely for rapid in-office assessments of cariogenic bacterial levels. Saliva that is stimulated by chewing can be used as a sampling method to collect bacteria from the teeth and around the mouth and quantify them as colony forming units, except in very young children (about 3 years or younger). Levels of mutans streptococci of 105 cfu/ml and lactobacilli levels of 103 cfu/ml and above in stimulated saliva are considered high risk. The combination of the two groups of bacteria is particularly damaging.1 Combating the Bacterial Challenge Antibacterials that are naturally in saliva -- such as lysozyme, lactoferrin and immunoglobulins -- help to keep the caries bacterial pathogens under control. However, manufactured antibacterials must be used in the case of high-caries-risk (caries-active) individuals, individuals with existing decay, and individuals with high levels of these pathogens in the mouth. In the United States, 0.12 percent chlorhexidine gluconate is available as a mouth rinse and is effective against the mutans streptococci, but not as effective against the lactobacilli.5 Iodine may also prove to be a useful alternative to chlorhexidine, as described in detail below.8 Future antibacterials that are more effective and easier to use will be of considerable added benefit.

### *A Paradigm Shift Is Needed*

In summary, a paradigm shift that underlines the necessity of treating the bacteria as an essential component of dental caries management, rather than simply drilling and filling cavities, is the fundamental basis for the protocols laid out below.

## *Caries Risk Assessment Diagnostics*

### *Diagnostics for Caregivers and Nondental Health Care Personnel*

For young children, a caregiver or health practitioner simply lifting the lip to look for white spot lesions, stained fissures in the biting (occlusal) surfaces of the teeth, or gross cavities (holes) in the teeth is an excellent start. The first line of defense for young children can be the parent or caregiver.8,12 They can easily do this examination to ensure that caries is not starting or progressing. Nondental health care professionals can also readily use these techniques.

A questionnaire that addresses maternal dental history, number of people in the household, family dynamics, socioeconomic status, and frequency of ingestion of fermentable carbohydrates will also help.

For a quantitative measure of bacterial challenge, bacterial assessments and saliva flow testing must be used, as described below. Nondental health care professionals can administer these tests. All of the above procedures can readily be carried out in a community setting by health professionals or their assistants.

## Diagnostics for Dental Professions

Diagnostics for dental professionals include the same list as above for caregivers and nondental health care personnel.3,8,12 The dental professional will add tactile and visual inspection using instruments, such as the explorer. The disadvantage of the explorer is that it is difficult to differentiate between anatomical defects and incipient caries in the occlusal (biting) surfaces. X-rays (dental radiographs) are appropriate for interproximal lesions (on the abutting surfaces of the teeth) and advanced occlusal caries that are well into the dentin. In the case of an interproximal lesion, if the radiograph indicates that the lesion has not penetrated past the dentinoenamel junction, and the surface integrity has been maintained, then it can be reversed, or at least arrested, by remineralization and fluoride therapy. If caries levels are low, then remineralization may be enough to halt the decay. In the case of caries-active individuals (active cavities and/or high bacterial levels), antibacterial therapy will be needed in conjunction with the fluoride therapy.1

New optical imaging devices are becoming available that can assess hidden lesions, especially in occlusal surfaces. The Diagnodent device (KaVo, Ill.) is approved and marketed in the United States for this purpose. Quantitative light fluorescence and optical coherence tomography are experimental methods that are likely to become available to clinicians in a few years.

*Antibacterial Therapeutics*

Therapeutics that can be used by caregivers and other nonhealth care personnel include:

A. Xylitol, which is relatively new to the United States. It is a sweetener that looks and tastes like sucrose but is not fermented by cariogenic bacteria. Xylitol also inhibits attachment and transmission of the bacteria and can be delivered through chewing gum or lozenges as an effective anticaries therapeutic measure. Xylitol gum chewed by mothers during the first two years of their children's lives led to much lower levels of caries in the children later.11

B. Sodium bicarbonate (baking soda), which has antibacterial properties and neutralizes acids produced by bacterial metabolism. It can be delivered via toothpaste or in a solution in hyposalivatory cases.

C. Chlorhexidine gluconate, which is a broad-spectrum antibacterial that works by opening up the cell membrane of the bacteria. It is administered in the United States via prescription. In the United States, only 0.12 percent chlorhexidine gluconate is available as a mouthrinse, and it is effective against the mutans streptococci. Chlorhexidine is used as a mouthrinse, 10 ml once daily for a two- week period every two to three months.5 Recent data indicates that one week every month is similarly effective. In high-bacterial-challenge individuals, this therapy will need to be continued for approximately one year and monitored by bacterial assessment (see below). One of the problems with this compound is that it must be administered by the individual or home caregiver, it affects taste, and compliance is often poor.7

D. Iodine is an effective antibacterial. As described above, chlorhexidine is effective against mutans streptococci in the mouth but not lactobacilli. A potentially useful antibacterial is povidone iodine (sold as 10 percent povidone iodine, which is equivalent to 1 percent available iodine). It has been shown to reduce the incidence of early childhood caries in high-risk children when applied once every two months but has not been thoroughly proven.8 This therapy has the advantage that it can be applied in a dental office or by a health care provider simply by swabbing the teeth and is effective in reducing levels of lactobacilli as well as mutans streptococci.

E. New antibacterial compounds or antibacterial approaches are in development and are expected to be available soon.

Tools for Inhibition of Demineralization and Enhancement of Remineralization -- Fluoride Delivery Forms

The various delivery methods that provide fluoride to the surfaces of the teeth inhibit demineralization, enhance remineralization, and can also inhibit bacterial activity.1 Sources of fluoride for this purpose are those that can provide fluoride to the mouth (topical) and include drinking water; dentifrices (toothpastes and gels); over-the-counter fluoride rinses (0.05 percent sodium fluoride, such as Fluorigard or ACT); and professionally applied office topical varnishes, foams, gels, acidulated phosphate fluoride, and stannous fluoride. Prescription high-concentration fluoride gels and toothpaste (5,000 ppm fluoride, such as Prevident) are valuable for high-risk subjects for home use, especially in adults for root caries or high-caries-risk patients. High-concentration fluorides should be used with great care in children as they are readily ingested and increase the risk of fluorosis. They should not be used for children younger than 6. As with all therapeutics that are self-administered, compliance is a major problem. Patients must be persuaded as to the need to use these products. Persuasion that parental supervision is critical is a key part of successful therapy for children.

Biomaterials for Minimally Invasive Dentistry and Inhibition of Caries Progression

Biomaterials are now available for restoration of cavities with a minimum removal of sound tissue. This conservative approach protects as much as possible of the tooth's integrity so as to retain tooth function in the later years. Preventive resin restorations or small amalgam restorations are used for early lesions in occlusal surfaces. Restorations restore tooth function but do not fix the bacterial nature of the disease. Sealants are available for use to prevent caries in occlusal surfaces.4 Fluoride-containing restorative materials, including glass ionomer products, help prevent further decay at the site of placement.9,10

Interfering With Vertical Transmission of Cariogenic Bacteria -- Mother to Child

Delaying or preventing primary infection by mutans streptococci reduces the risk for future dental caries. Strategies aimed at reducing the risk of vertical (mother-to-child) transmission of cariogenic bacteria translate into improved oral health outcomes for children. All children are at risk for early colonization in the first two to three years of life. On this basis, it is recommended that pregnant women

should have a dental exam and caries risk assessment during the second or third trimester of pregnancy. This exam should include radiographs only if lead shield precautions are utilized to protect the developing fetus. Prospective mothers who are found to be caries-active, either because they have frank cavities or through the risk assessment tools detailed below, should receive aggressive dental care shortly after delivery of their child. Therapy should eliminate all active caries lesions, provide dietary counseling and use topical antimicrobial agents (e.g., chlorhexidine rinses, self-applied fluoride gels) as described in the protocols below to reduce the cariogenic bacterial levels in the mother's mouth. Further, daily use of xylitol-containing chewing gum or mints by mothers during the first two years of the child's life has been shown to reduce the transmission of bacteria from mother to child and to markedly reduce the caries levels later in the child's life. This approach will reduce the maternal salivary levels and/or significantly alter the genotype/phenotype of cariogenic bacteria, thereby reducing the risk of early vertical transmission. Education of mothers about the transmissibility of caries-causing bacteria, how dental decay occurs, and how it can be prevented should be included both pre- and postnatally.

## Caries Risk Assessment Protocol in Simple Steps

Caries risk assessment forms are provided as templates for use or modification. The one-page forms are designed for use with two age groups. The first is for babies and infants from 0 to 5 years of age. The second is for people age 6 years and older, including adults. Special-needs patients will require additional considerations.13 Following each of the forms is a one-page summary of instructions, which is designed to be printed on the back of the form. In practice, this allows for a one sheet, two-sided form. This is followed by a patient check sheet for recommendations for home caries intervention. The back of this form should display the one-page simplified description of the dental decay process aimed at the patient, parent, or caregiver.

## References

1. Featherstone JDB, The caries balance: Contributing factors and early detection. J Cal Dent Assoc 31(2):129-133, 2003.
2. Crall JJ, California children and oral health: Trends and challenges. J Cal Dent Assoc 31(2):125-128, 2003.
3. Adair SM, The role of caries prevention protocols in pediatric dentistry specialty programs. J Cal Dent Assoc 31(2):145-147, 2003.
4. Adair SM, The role of sealants in caries prevention programs. J Cal Dent Assoc 31(3):221-7, 2003.
5. Anderson MH, Chlorhexidine: How useful is it in combating the bacterial challenge and dental caries? J Cal Dent Assoc 31(3):211-4, 2003.
6. Berkowitz RJ, Acquisition and transmission of mutans streptococci. J Cal Dent Assoc 31(2):135-138, 2003.
7. Bird WF, Caries protocol compliance issues. J Cal Dent Assoc 31(3):253-6, 2003.
8. Den Besten PK, Berkowitz RJ, Early childhood caries: an overview with reference to our experience in California. J Cal Dent Assoc 31(2):139-143, 2003.
9. Donly K, Fluoride varnishes. J Cal Dent Assoc 31(3):217-9, 2003.
10. Hicks J, Garcia-Godoy F, et al, Fluoride-releasing restorative materials and secondary caries. J Cal Dent Assoc 31(3):229-45, 2003.
11. Lynch H, Milgrom P, Xylitol and dental caries: An overview for the clinician. J Cal Dent Assoc 31(3):205-9, 2003.
12. Stewart RE, Hale KJ, The paradigm shift in the etiology, prevention, and management of dental caries: Its effect on the practice of clinical dentistry. J Cal Dent Assoc 31(3):247-51, 2003.
13. Glassman P, Miller C, Dental disease prevention and people with special needs. J Cal Dent Assoc 31(2):149-160, 2003.
14. Featherstone JDB, The science and practice of caries prevention. *J Am Dent Assoc* 131:887-899, 2000.
15. Featherstone JDB, Prevention and reversal of dental caries: role of low-level fluoride. Community Dent Oral Epidemiol 27:31-40, 1999.

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 at San Francisco, P.O. Box 0758, 707 Parnassus Ave, San Francisco, CA 94143 or jdbf@itsa.ucsf.edu.

Caries risk assessment forms (PDF format)

CDA JOURNAL, VOL 31, Nº3

# Crow's Feet and White Teeth

Robert E. Horseman, DDS

From the beginning of time until recently, the only middle-aged people with white teeth were wearing prosthetic replacements for their own naturally colored teeth. As they aged, their hair turned white and their teeth turned dark. It was the natural order of things to gradually exchange the bloom of youth for sags, wrinkles, crow's feet and wattles. Many people accepted this metamorphosis with grace and understanding, chuckling ruefully that although they were now in the "Golden Years," the whole phenomenon sucked.

In the entertainment industry, however, aging was simply not acceptable, and the money was there to thwart it. Movie stars in particular sought the services of teams of plastic surgeons and, inevitably, sympathetic dentists who could put the brightness of an 18-year-old's smile back into a 60-year-old face. Thus, we were treated to the startling and sometimes ludicrous look of actors who, distraught by baggy eyes and similar inroads of senility, insisted on rectifying Nature's perversity by displaying smiles frozen white in the dreadful risus sardonicus. Although the result shouted "phony" clear up to the last row in the balcony, the pursuit of youth was not to be denied. An exception was Walter Brennan, who won all sorts of awards with no teeth at all, but it was a sacrifice that never caught on with SAG members.

As with other trends innocent of logic, the Hollywood white smile caught on immediately, and soon dissatisfied shop girls, housewives and scullery maids began clamoring for whitened teeth. Obeying the basic canon of business, dental supply firms were quick to comply.

The whole advertising arm of dentistry concentrated on the transcendent importance of Everyman's right to really, really white teeth regardless of age, social status or access to a mirror. Dental manufacturers, who initially cultivated a hooded, watchful gaze on "bleaching" as it came to be called, began turning out bleaching agents in a wild variety of strengths and viscosities.

Dentists, suspicious at first of whatever chemical reaction was taking place in their patients' mouths, ultimately reckoned this to be a singularly unexpected bonanza. As expected, insurance companies, with Olympian certainty, declared the process of whitening teeth to be a cosmetic procedure, another human fatuity, and therefore not a covered benefit.

No matter, the cost of tooth whitening -- when compared with a full-on face lift, nose job or breast augmentation -- was a sybaritic bargain hunter's delight. Teenagers, whose teeth were already whiter than any middle-aged person could hope to achieve, were clangorous in

their demand to get in on the process.

Inevitably, companies with no background in dentistry streamed like pilgrims to the Kaaba in Mecca in their haste to get in on a good thing. Over-the-counter tooth whitening kits proliferated, featuring bleaching trays suitable for going 15 rounds with Lennox. The dental profession righteously excoriated this transparent attempt to lure away their clientele with a potentially hazardous treatment and dubious results.

About the same time, the American public's penchant for wanting things done NOW began to be heard. The usual bleaching process had been taking up to a week or more of wearing the bleaching tray for eight hours at night or at periodic intervals during the day for extended periods. Nobody, even the attending dentist, seemed to know exactly when the procedure was finished. If teeth got several shades lighter after five nights, patients wondered, would they get 20 shades whiter if the time were extended for two weeks? Or a month? Who knew? Nobody was prepared to define exactly what constituted a shade change, but the numbers were impressive regardless. Impatient patients bleated, "Why can't we do this faster, Doc, we got other fish to fry."

Not a problem for our compassionate researchers. We will sell the profession a high-powered light suitable for illuminating the Coliseum, they enthused. Couple that with a bleach strong enough to whiten the Black Hills of South Dakota and promise the public that their teeth will lighten at least 12 shades (company definition) in one hour. YES! One hour!

So that's where we are today. Some of us are still futzing along with a week or two goal and 15% carbamide peroxide, some of us with healthier bank accounts are toasting teeth with major arc lights and 30%+ chemicals. The public, tilting at Mother Nature's windmill, is still pursuing the chimera of the perfect smile. Wouldn't you like to run this by Drs. Fouchard and Black?