NIH Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life

March 26–28, 2001
William H. Natcher Conference Center
National Institutes of Health
Bethesda, Maryland

Sponsored by:

♦ National Institute of Dental and Craniofacial Research ♦ Office of Medical Applications of Research ♦

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Introduction

The National Institutes of Health (NIH) is sponsoring a Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life on March 26–28, 2001.

Although great strides have been made in dental health in recent decades, dental caries, or tooth decay, remains common in the United States. Caries result when certain species of bacteria in the mouth establish a sticky colony called a biofilm, or dental plaque, on the teeth. The bacteria generate acids that dissolve minerals in tooth enamel, resulting in the formation of opaque white or brown spots beneath the surface of the enamel.

Nearly 20 percent of children between the ages of 2 and 4 have had tooth decay and almost 80 percent of young people have had a cavity—a late manifestation of tooth decay—by age 17. More than two-thirds of adults aged 35 to 44 years have lost at least one permanent tooth due to decay while one-fourth of those aged 65 to 74 have lost all of their natural teeth.

Water fluoridation, dental sealants, and regular professional dental care are among the safe and effective, though underused, measures currently available for preventing and treating dental caries. Scientific research continues to fuel remarkable progress in our understanding of the best ways to diagnose, treat, and prevent dental caries.

This NIH Consensus Development Conference has been convened to examine the current state of dental caries research so that health care providers and the general public can make informed decisions about this important public health issue.

During the first day-and-a-half of the conference, experts will present the latest dental caries research findings to an independent, non-Federal consensus development panel. After weighing all of the scientific evidence, the panel will draft a statement that will be presented to the conference audience on the third day. The consensus development panel's statement will address the following key questions:

- What are the best methods for detecting early and advanced dental caries (validity and feasibility of traditional methods; validity and feasibility of emerging methods)?
- What are the best indicators for an increased risk of dental caries?
- What are the best methods available for the primary prevention of dental caries initiation throughout life?
- What are the best treatments available for reversing or arresting the progression of early dental caries?

- How should clinical decisions regarding prevention and/or treatment be affected by detection methods and risk assessment?

- What are promising new research directions for the prevention, diagnosis, and treatment of dental caries?

On the final day of the meeting, the panel chairperson, Dr. Michael C. Alfano, will read the draft statement to the conference audience and invite comments and questions. A press conference will follow to allow the panel and chairpersons to respond to questions from media representatives.

General Information

Conference sessions will be held in the Natcher Conference Center, National Institutes of Health, Bethesda, Maryland. Sessions will run from 8:30 a.m. to 5:30 p.m. on Monday, from 8 a.m. to 12:45 p.m. on Tuesday, and from 9 a.m. to 11 a.m. on Wednesday. The telephone number for the message center is (301) 496-9966; the fax number is (301) 480-5982.

Cafeteria

The cafeteria in the Natcher Conference Center is located one floor above the auditorium on the main floor of the building. It is open from 7 a.m. to 2 p.m., serving breakfast and lunch.

Sponsors

The primary sponsors of this meeting are the National Institute of Dental and Craniofacial Research and the NIH Office of Medical Applications of Research. Cosponsors include the National Institute on Aging and the U.S. Food and Drug Administration.

Continuing Education Credit

The National Institute of Dental and Craniofacial Research is an ADA CERP recognized provider of continuing education credit.

The NIDCR designates this continuing education activity for a maximum of 14.75 credit hours. Participants should claim only those hours of credit that he/she actually spent in the educational activity. Original continuing education verification is subject to audit by many state dental boards. This verification should be retained by the licensee.

Statement of Interest

Each speaker presenting at this conference has been asked to submit documentation outlining all outside involvement pertaining to the subject area. Please refer to the chart in your participant packet for details.

Agenda

Monday, March 26, 2001

7:30 a.m. Registration

8:30 a.m. Welcome and Introduction
Dushanka V. Kleinman, D.D.S., M.Sc.D., Deputy Director
National Institute of Dental and Craniofacial Research
National Institutes of Health

Welcome and Charge to Panel
Barnett S. Kramer, M.D., M.P.H., Director
Office of Medical Applications of Research
Office of the Director, National Institutes of Health

Purpose of Conference—Issues
Michael C. Alfano, D.M.D., Ph.D., Panel Chair, Dean
New York University College of Dentistry

9:00 a.m. Dental Caries in the Second Millennium
Amid I. Ismail, B.D.S., M.P.H., Dr.P.H., Professor
Department of Cariology, Restorative Sciences, and Endodontics
University of Michigan School of Dentistry

I. Methods for Reviewing the Evidence

9:15 a.m. Systematic Review of Selected Dental Caries Diagnosis and Management Methods
James Bader, D.D.S., M.P.H., Research Professor
Sheps Center for Health Services Research and School of Dentistry
University of North Carolina at Chapel Hill

9:25 a.m. Methods Employed for Non-RTI/UNC Systematic Reviews
Alice M. Horowitz, Ph.D., Senior Scientist
National Institute of Dental and Craniofacial Research
National Institutes of Health

Monday, March 26, 2001 (continued)

II. Diagnosis and Management of Dental Caries

9:35 a.m. The Sensitivity and Specificity of Methods for Identifying Carious Lesions: The RTI/UNC Review
James Bader, D.D.S., M.P.H., Research Professor
Sheps Center for Health Services Research and School of Dentistry
University of North Carolina at Chapel Hill

10:05 a.m. **Discussant 1:** Clinical Diagnosis of Dental Caries: A European Perspective
Nigel B. Pitts, B.D.S., Ph.D., R.C.S., MFPHM, Director
Dental Health Services Research Unit, Dental Hospital and School
University of Dundee

10:15 a.m. **Discussant 2:** Clinical Diagnosis of Dental Caries: A North American Perspective
Stephen F. Rosenstiel, B.D.S., M.S.D., Chair
Department of Restorative Dentistry, Prosthodontics, and Endodontics
Ohio State University College of Dentistry

10:25 a.m. **Discussant 3:** Radiographic Diagnosis of Dental Caries
S. Brent Dove, D.D.S., M.S., Division Head
Oral Diagnosis/Oral Medicine Division
Department of Dental Diagnostic Science
University of Texas Health Science Center at San Antonio Dental School

10:35 a.m. **Discussant 4:** Diagnosis of Root Caries
David W. Banting, D.D.S., Ph.D., DDPH, M.Sc., FRCD(C), Professor
Faculty of Medicine and Dentistry, School of Dentistry, Division of Community Dentistry
University of Western Ontario

10:45 a.m. **Discussant 5:** Diagnosis of Secondary Caries
Edwina Kidd, B.D.S., Ph.D., F.D.S., R.C.S., Professor of Cariology
Division of Conservative Dentistry, GKT Dental Institute
Guy's Hospital, London

10:55 a.m. New Diagnostic Methods
George K. Stookey, Ph.D., Associate Dean for Research
Indiana University School of Dentistry

11:10 a.m. Discussion

12:00 p.m. Lunch

Monday, March 26, 2001 (continued)

III. Indicators of Risk

1:00 p.m. Definitions of "Risk" and "Risk Factors"
Brian A. Burt, B.D.S., Ph.D., M.P.H., Professor
Department of Epidemiology
School of Public Health, University of Michigan

1:05 p.m. Socioeconomic and Behavioral Determinants as Risk Factors for Dental Caries Throughout the Life Span
Susan T. Reisine, Ph.D., Chairman
Department of Behavioral Sciences and Community Health
University of Connecticut Health Center

1:20 p.m. Is Sugar Consumption Still a Major Determinant of Dental Caries? A Systematic Review
Brian A. Burt, B.D.S., Ph.D., M.P.H., Professor
Department of Epidemiology
School of Public Health, University of Michigan

The Relationship Between Low Birthweight and Subsequent Development of Caries: A Systematic Review
Brian A. Burt, B.D.S., Ph.D., M.P.H.

1:35 p.m. The Microbiology of Primary Dental Caries
Jason M. Tanzer, D.M.D., Ph.D., Professor
Department of Oral Diagnosis
University of Connecticut Health Center

1:50 p.m. Inherited Risks for Susceptibility to Dental Caries
Charles F. Shuler, D.M.D., Ph.D., Director and George and Mary Lou Boone Professor of Craniofacial Molecular Biology
Center for Craniofacial Molecular Biology
University of Southern California

2:05 p.m. Exposure to Metal Ions and Susceptibility to Dental Caries
William H. Bowen, B.D.S., Ph.D., Welcher Professor of Dentistry
Center for Oral Biology
University of Rochester School of Medicine and Dentistry

2:20 p.m. Physical and Chemical Aspects of Saliva as Indicators of Risk for Dental Caries
Cataldo W. Leone, D.M.D., D.Sc., Associate Professor
Department of Periodontology and Oral Biology
Boston University School of Dental Medicine

2:35 p.m. Discussion

Monday, March 26, 2001 (continued)

IV. Primary Prevention of Dental Caries

3:15 p.m. Effectiveness of Methods for the Primary Prevention of Dental Caries: A Review of the Evidence
R. Gary Rozier, D.D.S., M.P.H., Professor
Department of Health Policy and Administration, School of Public Health
University of North Carolina at Chapel Hill

V. Methods of Stopping or Reversing Early Carious Lesions

3:30 p.m. Prevention of Early Carious Lesions and Management of Dental Caries in High-Risk Individuals: RTI/UNC Review
James Bader, D.D.S., M.P.H., Research Professor
Sheps Center for Health Services Research and School of Dentistry
University of North Carolina at Chapel Hill

3:50 p.m. **Discussant 1:** Fluoride: A European Perspective
Elizabeth T. Treasure, B.D.S., Ph.D., FRACDS, FDSRCS, Professor
Department of Dental Health and Development, Dental School
University of Wales College of Medicine

4:00 p.m. **Discussant 2:** Topical Fluorides in Caries Prevention and Management: A North American Perspective
Ernest Newbrun, D.M.D., Ph.D., Professor Emeritus
Department of Stomatology
University of California, San Francisco

4:10 p.m. **Discussant 3:** Pit and Fissure Sealants in High-Risk Individuals
Jane A. Weintraub, D.D.S., M.P.H., Lee Hysan Professor
Chair
Division of Oral Epidemiology and Dental Public Health
Department of Preventive and Restorative Dental Sciences
University of California, San Francisco School of Dentistry

4:25 p.m. Discussion

5:30 p.m. Adjournment

Tuesday, March 27, 2001

V. Methods of Stopping or Reversing Early Carious Lesions (continued)

8:00 a.m. **Discussant 4:** Antimicrobial Approaches for the Prevention or Treatment of Dental Caries
Page W. Caufield, D.D.S., Ph.D., Director
Specialized Caries Research Center, School of Dentistry
University of Alabama at Birmingham

8:15 a.m. **Discussant 5:** Salivary Enhancers
Jane C. Atkinson, D.D.S., Assistant Dean, Clinical Affairs
Professor, Department of Oral Medicine
University of Maryland Dental School

8:30 a.m. **Discussant 6:** Application of Methods To Be Employed by Dental Personnel and Other Methods of Stopping/Reversing Dental Disease: Behavior Modification
Peter Milgrom, D.D.S., Professor and Director
Dental Fears Research Clinic
Dental Public Health Sciences and Health Services
University of Washington

8:45 a.m. **Discussant 7:** Non-Cariogenic Sweeteners
Catherine Hayes, D.M.D., D.M.Sc., Assistant Professor
Department of Oral Health Policy and Epidemiology
Harvard School of Dental Medicine

9:00 a.m. **Discussant 8:** Choosing Appropriate Preventive Approaches
Denis O'Mullane, B.D.S., Ph.D., F.D.S., F.F.D., Professor
Oral Health Services Research Centre
University Dental School and Hospital of Wilton, Cork, Ireland

9:15 a.m. Emerging Methods in Prevention of Dental Caries
Brian H. Clarkson, Ph.D., M.S., L.D.S., Department Chair
Department of Cariology, Restorative Sciences, and Endodontics
University of Michigan School of Dentistry

9:30 a.m. Discussion

Tuesday, March 27, 2001 (continued)

VI. Clinical Decision-Making in Caries Management

10:30 a.m. Clinical Decision-Making for Dental Caries Management
B. Alexander White, D.D.S., Dr.P.H., M.S., Senior Investigator
Kaiser Permanente Center for Health Research

10:45 a.m. Clinical Applications and Outcomes of Using Indicators of Risk in Caries Management
Domenick T. Zero, D.D.S., M.S., Professor and Chair
Department of Preventive and Community Dentistry
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Oral Health Research Institute
Indiana University School of Dentistry

11:00 a.m. Clinical Decision-Making for Caries Management in Primary Teeth
Norman Tinanoff, D.D.S., M.S., Professor and Chair
Department of Pediatric Dentistry
University of Maryland Dental School

11:15 a.m. Clinical Decision-Making for Coronal Caries Management in the Permanent Dentition
Kenneth J. Anusavice, Ph.D., D.M.D., Associate Dean for Research
Professor and Chair
Department of Dental Biomaterials
University of Florida College of Dentistry

11:30 a.m. Clinical Decision-Making for Caries Management in Root Caries
James L. Leake, D.D.S., M.Sc., DDPH, FRCD(C), Professor and Discipline Head
Community Dentistry
University of Toronto

11:45 a.m. The Scientific Basis for the Teaching and Practice of Conservative Operative Dentistry
Dorothy D. McComb, B.D.S., M.Sc.D., FRCD(C), Professor and Head
Department of Restorative Dentistry
University of Toronto

12:00 p.m. Discussion

12:45 p.m. Adjournment

Wednesday, March 28, 2001

VII. Recommendations: Consensus Panel

8:00 a.m.	Registration
9:00 a.m.	Presentation of Consensus Statement
9:30 a.m.	Public Discussion
11:00 a.m.	Panel Meets in Executive Session
1:00 p.m.	Press Conference
2:00 p.m.	Adjournment

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Abstracts

The following are abstracts of presentations to the NIH Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life. They are designed for the use of panelists and participants in the conference and as a reference document for anyone interested in the conference deliberations. We are grateful to the authors for their participation and for supplying these summaries.

Alice M. Horowitz, Ph.D.
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Dental Caries in the Second Millennium

Amid I. Ismail, B.D.S., M.P.H., Dr.P.H., Hana Hasson, D.D.S., M.S., Woosung Sohn, D.D.S., Ph.D., Dr.P.H.

This conference has been called to reach consensus on the diagnosis and management of dental caries throughout life. The mission is to reach conclusions that should define what we can do today in these areas and what we need to know to expand the knowledge base on dental caries. The conference was designed to find answers for six specific questions related to diagnosis of early and advanced carious lesions; indicators of caries risk; methods for primary prevention of dental caries; methods for arresting early carious lesions; clinical decision-making; and what research is needed in diagnosing and managing dental caries. On some of these issues, as the subsequent reviews will show, we have made significant progress in finding answers. But for many of these questions, unfortunately, we still have a long way to go.

This narrative review of dental caries diagnosis and management throughout the millennium is based on information obtained from reports published since 1839 and from 36 textbooks on caries diagnosis and management published since the 19th century. A hand search of the Index of Dental Literature published between 1839 and 1965 was conducted to locate publications on caries diagnosis, etiology, prevention, and management.

The history of dental caries diagnosis and management throughout the second millennium can be divided into two distinct periods. The first, which lasted more than 900 years and may still be going on today, is the "observational" era. The second, which has developed and revolutionized our understanding of the causes and treatments of all diseases, is the "scientific era." During the observational era, healers explained what they saw in their patients using reason, logic, and their current knowledge. They provided treatment without evaluating the outcome through the scientific method.

Many of the issues to be discussed by the presenters at this conference have been observed since the 19th century. For example, dentists reported on the presence of enamel and dentinal caries (early and advanced lesions) as early as the 1880s (Darby, 1884). Hidden caries (defined as "caries in the dentin without an opening through the enamel leading to it") was a phenomenon that was noticed in 1868 (Knapp, 1868). Early childhood caries, or "labial decay of childhood," was described in 1884 (Darby, 1884). "Secondary decay" was discussed as a problem in 1880 (Palmer, 1880). Interestingly, the problem of variation among dentists in caries diagnosis and restorative treatment decisions was reported on in 1869 (Anonymous, 1869). The cause of this ongoing problem was claimed to be the "failure in diagnosis of dental decay, even when one intends to be very thorough." The cause of variation was attributed to "the large size of the excavator used for examination," and the solution proposed was to use "the very smallest...hatchet ...with exceedingly thin blade" (Anonymous, 1869). Later on, Black advocated using a "small, very sharp exploring tine which will penetrate the decay area" (Black, 1910). Since then, the practice of using sharp explorers to find carious lesions has become a standard method without much scientific scrutiny.

During the observational era there were several competing theories on why dental caries develops. However, the one theory that was based on limited "observational and experimental the chemico-parasitic theory (Miller, 1883). Dietary or "constitutional" or nutritional factors also were associated with dental caries (Wallace, 1913; Richardson, 1914).

During the late 19th century, American dentists began reporting on the epidemic of dental caries. The rise in dental caries was most noticeable among affluent, urban, white Americans. This observation led to several theories. Dental caries was considered a curse of "civilization" (Wallace, 1913).

Epidemiologic surveys were first initiated in the United States in the 1930s and 1940s. Oral health emerged as a focus for initiatives sponsored by government agencies during and after World War II as a result of the relatively large number of potential recruits who did not meet the liberal dental requirements for enlisting in military service. At the same time, the link between fluoride, fluorosis, and dental caries was confirmed by a number of cross-sectional and incidence studies (Ast, 1944; Dean, Arnold, Elvove, 1942). This link was the first major breakthrough in caries prevention.

In 1945, the first field trial to test the effectiveness of water fluoridation commenced in the United States (Arnold, Dean, Jay, 1956). Additional water fluoridation studies then led to widespread use of fluoride in caries prevention. Water fluoridation was recently cited by the Centers for Disease Control and Prevention as one of the 10 most important public health achievements of the 20th century.

The scientific era in dentistry started in the early years of the 20th century with attempts to test hypotheses and to collect data to support or refute them. Basic research led to significant advances in understanding of the histopathology of caries in enamel and dentin, microbial risk factors, the physiology and pathology of saliva, and understanding of fluoride mechanisms. Research activities led to the development of new preventive interventions and restorative materials that have had a significant impact on the restoration of decayed teeth and the retention of teeth for life. A second major development in caries prevention was scientific validation of the efficacy and effectiveness of pit-and-fissure sealants.

The etiological model proposed by Miller was expanded to include other risk factors or indicators that are associated with dental caries initiation and progression, and dental caries is now considered to have a multifactorial etiology (Clarkson, 1999). Dental caries is also recognized as a biosocial disease whose burden has shifted from affluent members of society to those who are economically disadvantaged.

During the scientific era the prevalence and severity of dental caries in the United States have declined, especially in children. There has been phenomenal growth in the biological understanding of dental caries. However, the knowledge base for diagnosis, risk assessment, translation of prevention into practice, and decision-making on placement and replacement of restorations has not progressed significantly during the last 5 decades. There has been limited investment in clinical research and in the translation of research and biological knowledge into practice. Moreover, dentists still rely on observation and uncontrolled experimentation with a few patients to make general recommendations for dental practice (Christensen, 2000).

Most of the advances in caries research in the second millennium have relied on observation and inductive reasoning. To resolve the current dilemma in caries diagnosis and management, however, the use of a scientific research model is necessary to define the problems we face and design appropriate research projects to find answers. There is an urgent need to develop new tools that can accurately diagnose the earliest signs of tooth demineralization, the natural history of early carious lesions, the determinants of progression and regression, when to restore a carious tooth, and how to classify with a high degree of sensitivity and specificity the risk status of patients. Research on these issues will not be possible without a major funding initiative to support training of a new cadre of basic and applied researchers in cariology and to develop and implement programs to address the real-life problems in diagnosis, risk assessment, and management. If the current weak trend of caries research in the United States continues, history will be harsh on all of us for our failure to use our knowledge and resources to reduce, if not eliminate, the burden of one of the world's most prevalent diseases.

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Systematic Review of Selected Dental Caries Diagnosis and Management Methods

James Bader, D.D.S., M.P.H.

Dental caries is a widespread, chronic, infectious disease experienced by almost 80 percent of children by the age of 18 and by more than 90 percent of adults. Substantial variation exists in dentists' diagnosis of carious lesions as well as in the methods used by dentists to prevent or manage them. New methods of identifying carious lesions have appeared, and new approaches to the management of carious lesions—and for the management of individuals deemed to be at elevated risk for experiencing carious lesions—are emerging. A systematic review of the literature (Bader, Shugars, Bonito, 2000) was conducted to address three related questions concerning the diagnosis and management of dental caries: (a) the performance (that is, sensitivity and specificity) of available diagnostic methods; (b) the efficacy of approaches to the management of noncavitated, or initial, carious lesions; and (c) the efficacy of preventive methods for individuals who have experienced or are expected to experience elevated incidence of carious lesions.

Search Strategy

We conducted two searches of the relevant English-language literature from 1966 to October 1999, using MEDLINE, EMBASE, and the Cochrane controlled trials register. We also did a hand search of relevant journals published in November and December, 1999. (We did not investigate reports in the gray literature—that is, information not appearing in the periodic scientific literature.) One search focused on studies of six diagnostic methods (visual, visual/tactile, radiography, fiberoptic transillumination, electrical conductance, laser fluorescence) and combinations of these methods. A second search focused on studies of preventive or management methods for carious lesions, including fluoride applications, pit and fissure sealants, health education, dental prophylaxis, instruction in oral hygiene, removal of dental plaque, chlorhexidine application, and use of cariostatic agents.

Selection Criteria

The group of diagnostic studies included studies that involved histological validation of caries status and that either reported the results to show the sensitivity and specificity of the diagnostic method or that reported data from which those measures could be calculated. We excluded studies of diagnostic methods not commercially available.

The group of studies on dental caries management included only those on methods applied or prescribed in a professional setting and that were performed in vivo with a comparison group. In our selection of literature on the management of noncavitated carious lesions we only included studies where the lesion was the unit of analysis. In selecting literature on the management of subjects at elevated risk for dental caries, we only included studies where such

determinations had been made on individual subjects, based on their carious lesion experience and/or bacteriological testing.

Data Collection and Analysis

We selected the studies for our report from among 1,407 diagnostic and 1,478 management reports by reading titles, abstracts, and, where necessary, full papers. We ultimately abstracted data (single abstraction, subsequent independent review) from two types of studies, using different forms of abstracting for the diagnostic and management studies. A quality rating form was completed by the research team for each of the three questions mentioned above, with different criteria employed for the two types of studies.

Diagnostic Review Results

We judged the strength of the evidence on the validity of the diagnostic methods evaluated to be *poor*. The evidence did not support the calculation of point estimates of sensitivity. There were almost no reports on the performance of any diagnostic method applied to primary teeth, anterior teeth, or root surfaces. The number of studies available on posterior occlusal and proximal surfaces of permanent teeth was sufficient for calculation of point estimates for some, but not all, of the methods. Even where the number of studies was sufficient, however, variations among them precluded such estimates. With the exception of electrical conductance, the diagnostic methods used criteria that maximized specificity at the expense of sensitivity: false positive diagnoses were proportionally infrequent, compared to false negative diagnoses. In addition to the limited numbers of studies on certain teeth and methods, the studies displayed a variety of serious limitations, including a predominance of in vitro studies, small numbers of examiners, high prevalence of lesions, and inadequate descriptions of subject selection methods, examiner training and reliability, and criteria for diagnosis.

Management Review Results

The literature examined on the management of noncavitated carious lesions consisted of five studies that described seven experimental interventions. Because these interventions varied extensively in terms of method used as well as other characteristics, no conclusions about the efficacy of these methods were possible. We therefore rated the evidence for the efficacy of management methods of noncavitated lesions as *incomplete*. Standardization in the determination of noncavitated status is needed.

The literature on the management of individuals at elevated risk of carious lesions consisted of 22 studies describing 29 experimental interventions. We rated the evidence on the efficacy of fluoride varnish for prevention of dental caries in high-risk subjects as *fair*, and the evidence for all other methods as *incomplete*. Because the evidence on the efficacy of some methods, including the application of chlorhexidine, use of sucrose-free gum containing xylitol, and combined chlorhexidine-fluoride methods is suggestive but not conclusive, these are fruitful areas for further research.

Conclusions

The evidence available to estimate the validity of diagnostic methods for carious lesions is insufficient. There are too few studies on many of the methods, and even when sufficient numbers of studies are available the substantial variations among them produce problematic results. The literature describing the management of two specific dental caries-related conditions—nonsurgical interventions for noncavitated lesions, and prevention of lesions in persons at elevated risk for new lesions—is inadequate to permit conclusions about the efficacy of most methods. For only two specific applications—fluoride varnishes in caries-active/high-risk individuals, and fluoride-based intervention for individuals receiving radiotherapy—was the evidence rated as *fair*. For all other management methods the evidence was judged to be *incomplete*. But the need for better determination of efficacy is acute, since much of modern preventive dental practice is predicated on the assumed efficacy of these methods.

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Methods Employed for Non-RTI/UNC Systematic Reviews

Alice M. Horowitz, Ph.D., and Patricia F. Anderson, M.I.L.S.

The RTI/UNC review was conducted to address some or most aspects of three of the questions developed by the organizing committee of this Consensus Development Conference. Independent reviewers (non-RTI), however, have addressed the majority of the questions. We prevailed upon numerous independent reviewers to conduct systematic reviews.

Because most of the researchers identified in the particular content areas to be addressed were not experienced in the methods used in conducting a systematic review, the National Institute of Dental and Craniofacial Research (NIDCR) provided two training sessions. Dr. Amid Ismail conducted these training sessions; Dr. Jim Bader provided background on how RTI/UNC conducted their reviews; and during the second session, Ms. Patricia Anderson, a University of Michigan librarian, explained how to develop appropriate search strategies of MEDLINE and EMBASE. Each reviewer submitted a proposal for his or her review that was discussed and revised during the second training session.

Subsequently, Ms. Anderson was contracted to conduct the searches for each non-RTI review. The searching for each team was an iterative, multistage process. The findings of these searches can be found on http://www.lib.umich.edu/dentlib/nihcdc/. Each reviewer received lists of references with abstracts. The reviewers read the abstracts and either included or excluded studies, based upon criteria that were developed independently by each review team. The full reports of the included studies were photocopied and abstracted in evidence tables. The reviewers did not conduct meta-analyses of the evidence. The independent reviewers were provided with guidelines on abstraction and a step-by-step manual on how to conduct the reviews.

In the section on primary prevention of dental caries, the review by Dr. Rozier is based on recent systematic and other reviews conducted on fluorides, dental sealants, antimicrobials, and patient counseling.

In the section on clinical decision-making for dental caries management, Dr. White was asked to provide an overview of clinical decision-making as a framework for the presentations on implications for clinical practice and research. Three of the reviewers in this section (Tinanoff, Anusavice, Leake) were asked to synthesize the evidence obtained to provide directions for clinical decision-making for the management of dental caries in primary and permanent dentitions as well as root surfaces and related research.

The Sensitivity and Specificity of Methods for Identifying Carious Lesions: The RTI/UNC Review

James Bader, D.D.S., M.P.H.

Topic is summarized in Dr. Bader's abstract on page 25.

Clinical Diagnosis of Dental Caries: A European Perspective

Nigel B. Pitts, B.D.S., Ph.D., R.C.S., MFPHM

I applaud the organizers for setting out an important and timely agenda for this conference, which is, in a sense, overdue. The focus of the conference is rightly on clinical practice and using current knowledge to provide the best possible care for individual patients. It is important to realize that much of the research in caries diagnosis has overlapped the applications of the diagnostic process in clinical practice, clinical research, and clinical dental epidemiology. The differing objectives, environments, and priorities of research in these areas often confuse attempts to synthesize the relevant literature, particularly when comparisons are being made across countries and cultures.

Since the aim of the conference is to develop scientifically based recommendations that can be applied by dentists and dental hygienists, it is important that the everyday fundamentals of clinical caries diagnosis are addressed clearly and objectively. Clinical diagnosis is the foundation on which the answers to most of the consensus questions will be based, either by providing information on caries detection or being used in the assessment of both primary and secondary preventive strategies as well as playing a key role in informing clinical decision-making. It is vital to consider the findings of the Research Triangle Institute (RTI) systematic review as well as those from other reviews from a variety of countries, even if some of the findings seem to contradict the dental facts of life taught to many of us and do not fit the "classical" findings of research carried out years ago. We would expect that clinicians in various countries may find different recommendations either easy or difficult to apply, and we should learn from the work done in medicine (SIGN, 1999) that there is also a developing literature on how to disseminate the findings of reviews effectively.

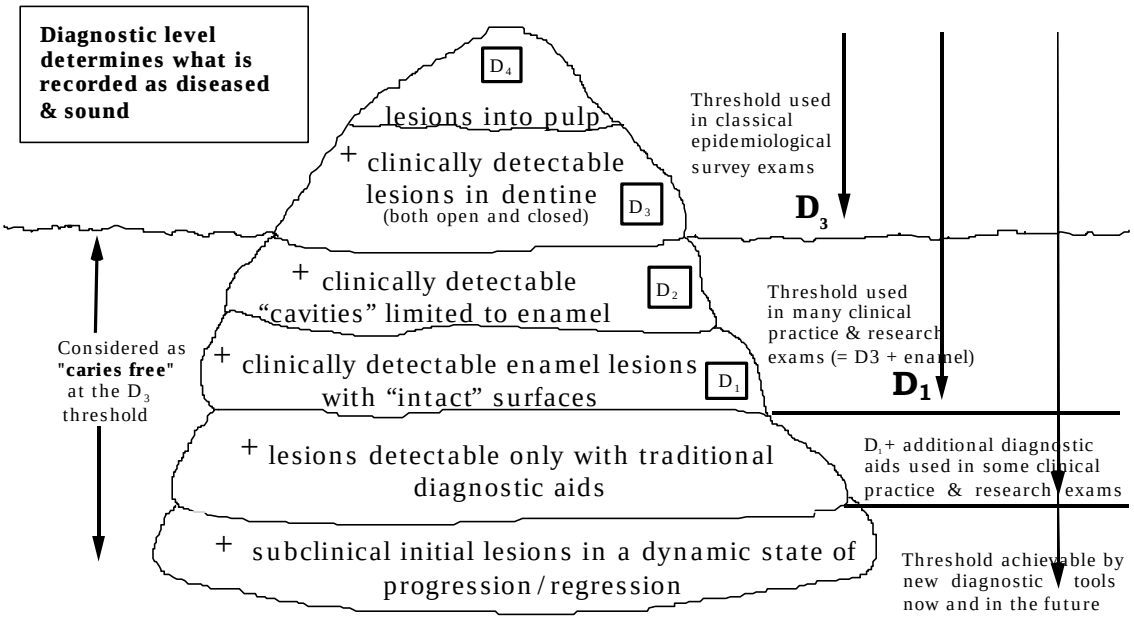
A key area is clarity about definitions and nomenclature. Many apparently similar terms are often used interchangeably in the literature but are taken by different researchers and clinicians to mean very different things. There will have to be clarity with regard to defining the terms "diagnosis" (not just detection), clinical "management" (encompassing preventive care of reversible lesions as well as surgical excision of tooth substance), "dental caries" (the view held for many years in Europe and now increasingly in the United States is that caries is a continuum rather than the macroscopic cavitation that is the late stage of the disease process), "throughout life" (here we need to differentiate early childhood caries from lesions in children, adolescents, adults, and seniors), and to plan minimally invasive care for the long-term benefit of the patient.

A European Perspective on the RTI Review

To make best use of the RTI review, it is important to understand the concepts of the D1 and D3 diagnostic thresholds used in it. Figure 1 shows an updated version of the iceberg analogy (Pitts, 1997a) for conceptualizing dental caries and the impact that a changing diagnostic threshold has on what is considered by dentists and researchers to constitute sound or diseased tooth tissue. The term "caries free" is frequently used when referring to data reported at the D3 (caries into dentin only) diagnostic threshold. This conveys the mistaken impression that there is no disease present, even though large numbers of carious lesions recognized as dental caries in the enamel are present (Pitts, Fyffe, 1988). The diagnosis of so-called "white spot" and "brown spot" caries has been accepted for many years in Europe and monitoring the behavior of these lesions over time is routine (Backer-Dirks, Amerongen, Winkler, 1951). It has been shown that the progression of these enamel lesions with macroscopically intact surfaces is extremely slow, and such lesions on free smooth surfaces do not always progress. They may stop, or even reverse (Backer-Dirks, 1966; Nielson, Pitts, 1991). These enamel lesions are often referred to as D1 lesions, as opposed to the D1 diagnostic threshold which includes both D1 and D3 lesions (see figure 1).

An example of the type of visual diagnostic criteria often used in European studies, which can be reported at either or both the D1 and D3 diagnostic thresholds, is the recently reported Dundee Selectable Threshold Method—(DSTM) (Fyffe, Deery, Nugent, et al., 2000a; Fyffe, Deery, Nugent, et al., 2000b). Traditional diagnostic aids (such as bitewing radiography and fiber-optic transillumination [FOTI]) detect more lesions still. The newer and more sensitive methods of caries diagnosis are now able to detect even more subclinical initial lesions which are in a state of dynamic progression and regression at an early stage of the disease process before they are discernible by conventional clinical methods. This gives the potential for lesions to be detected and the impact of preventive care to be assessed to ensure that cavitation is avoided.

The same iceberg can be used to link the diagnostic divisions of the continuum of dental caries with the type of management option that offers the patient the best long-term benefit. Choice of the most appropriate care option involves balancing the risk of continuing tooth destruction if preventive care fails against restorations placed and then replaced repeatedly over time with the imperfect methods currently available. The approach used in Europe for some years is summarized by the acronyms NAC for "No Active Care" above normal prevention, PCA when stable or noncavitated lesions are diagnosed, and PCA + OCA when both "Preventive and Operative Care Are Advised" for progressive dentinal lesions and lesions with significant cavitation (Pitts, Longbottom, 1995). There is a continuing debate in Europe as to exactly when restorative intervention is indicated, with movement toward recognizing the need to tailor the decision to the needs of individual patients and with a focus on cavitation rather than dentin involvement per se. It should be noted that hidden dentin lesions can sometimes be found in sites that are clinically sound, and that these lesions must be scheduled for operative care (see figure 2). It also must be emphasized that clinical caries diagnosed at the enamel lesion threshold with intact surfaces are not scheduled for restoration but are typically managed preventively in Europe.



The "iceberg" of dental caries

Figure 1. Conceptualizing the caries process.

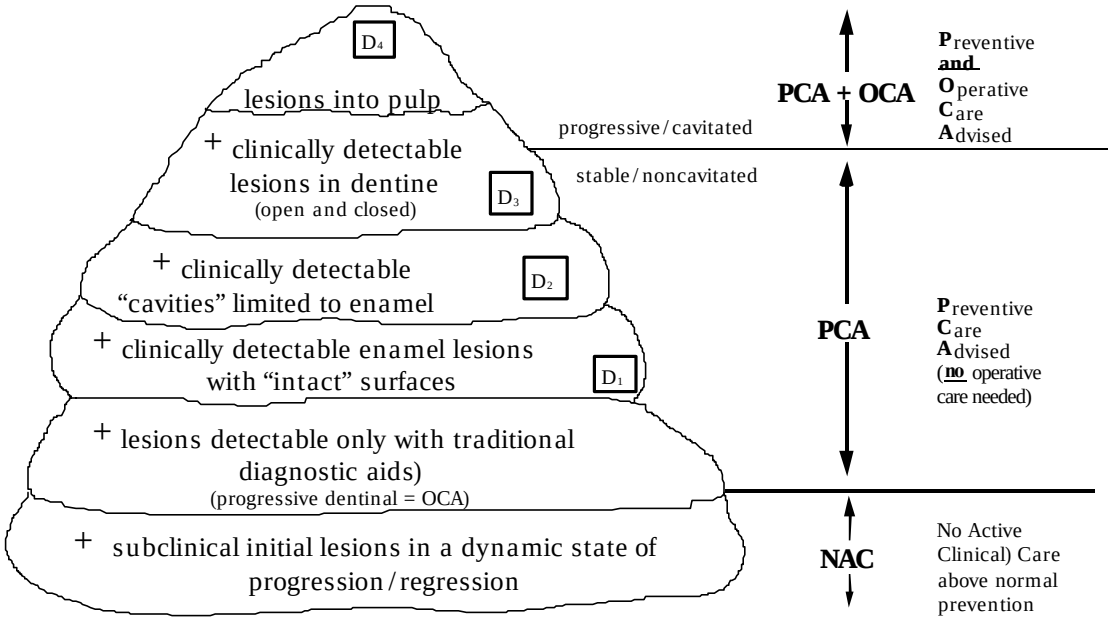


Figure 2. Linking diagnosis to clinical management.

A number of technical aspects of the RTI review are worthy of comment. The key finding that the quality of studies was often found to be poor may be seen as contentious by some in dentistry, and it is frustrating that (when measured against contemporary methodological standards) there are so few usable studies. However, it is important for these findings to be judged in the context of similar reviews in many fields of medical care where similar findings are common. They represent a major challenge to the dental research community.

Some areas of the review might have been improved if more time and resources had been available. A key concern in reviewing diagnostic literature in evidence-based healthcare is that the quality standards imposed in grading the papers are pertinent to the objective(s) of the study. Since data from some papers were employed for a number of different analyses (not always those intended by the authors), it might be argued that some of the quality scores were therefore inappropriate for some evidence tables. The presentation of the data is also complex. Other areas of debate include the possible use of receiver operating characteristic (ROC) analyses, rather than relying solely on sensitivity and specificity. Some argue that this method captures more of the diagnostic information obtained (ten Bosch, Mansson, 2000), while others are less convinced. Differences in the approach to histological validation are a further challenge. On the one hand, in vitro studies are commended as providing a true gold standard; on the other hand, differences between the diagnostic performance achieved in vitro and in vivo casts some doubt on the generalizibility of in vitro findings. The ideal study design (although very demanding in terms of logistics) would be to assess diagnosis in vivo first and then reassess the same surfaces in vitro following extraction of the tooth (for some ethically acceptable reason). A further difficulty occurs when the gold standard classically employed is potentially less sensitive than some of the methods being tested against it.

Studies Not Mentioned in the RTI Review

The papers cited below provide a European perspective on many of the challenges to clinical caries diagnosis raised in the review. The diagnostic challenge should not be underestimated or regarded as a basic or undemanding skill. The presentation of the disease has changed at a time when prevalence and incidence have slowed in some cases but become more polarized between risk groups (Kidd, Ricketts, Pitts, 1993) and as the range of preventive and operative treatment options has expanded (Paterson, Watts, Saunders, et al., 1991). Although clinical examination is the bedrock of daily dental practice, it is clear from many studies that clinical examination used alone will miss many lesions until they become so advanced that preventive intervention to avoid cavitation is compromised. The occlusal surface presents particular difficulties, since gross cavitation seems to occur less frequently and the limitations of the visual method have led to a fear of underdetecting hidden (or occult) lesions involving dentin.

A contentious issue for many clinicians is the lack of evidence supporting the continued use of a sharp explorer as a diagnostic tool. Although its use as part of a visuo-tactile clinical method is widespread and has been widely taught for many years in many countries, many European centers now teach that it is unethical to use an explorer in this way. This is because it was shown many years ago in Sweden (Bergman, Linden, 1969) that iatrogenic damage can readily be produced, particularly on initial caries within occlusal fissures, and favor continued

lesion development. Similar findings were shown by Ekstrand and coworkers nearly 20 years later (Ekstrand, Qvist, Thylstrup, et al., 1987), when it was also shown experimentally that probing with an explorer had a deleterious effect in terms of subsequent enamel demineralization (Van Dorp, Ekterkate, ten Cate, 1988). The potential caries-causing damage was illustrated again by Yassin (1995). Apart from any risk of conveying cariogenic organisms from one fissure system to another, it is argued that a practice likely to cause harm to the patient cannot be justified if it fails to provide a significant balancing benefit. In this case, the absence of any diagnostic benefit from the visual + tactile method over the visual-only method means that the use of the sharp explorer for coronal caries diagnosis should be discontinued. A further complication in interpreting this literature is the difficulty of comparing studies which include open cavities in the assessment of occlusal caries diagnosis (Lussi, 1996).

My paper and presentation will include further elaboration of the content of relevant papers not found or not highlighted in the RTI review and the presentation of some new data. These references are listed below, following the draft recommendations.

1. What are the best methods for detecting early and advanced dental caries (validity and feasibility of traditional methods; validity and feasibility of emerging methods)?

    - Recognize that clinical caries diagnosis (with all its flaws) is the current foundation of lesion detection in clinical practice, clinical research, and clinical epidemiology. Care is needed to distinguish objective methods in each area.

    - Clinical visual methods of caries diagnosis are universally employed and are rapid, economical, and acceptable for detecting early-stage disease (enamel lesions, such as white and brown spot caries on accessible sites), noncavitated dentinal lesions, and late-stage cavitated caries. However, their inherent limitations must be remembered.

    - Although clinical diagnostic methods are highly specific, the low sensitivity achieved (particularly for noncavitated occlusal surfaces in vivo) means that the use of diagnostic aids with superior performance is indicated, and that new methods for caries diagnosis are needed.

    - Although the amount of high quality evidence on new diagnostic methods is less than desirable, the very limited evidence available on the efficacy of traditional diagnostic methods means that clinicians cannot be complacent.

    - Given the potential for caries-inducing and caries-accelerating iatrogenic damage from the use of a sharp explorer, combined with lack of any evidence of additional diagnostic benefit, sharp explorers should no longer be used for coronal caries diagnosis.

    - Educational initiatives will be needed to share the evidence on sharp explorers and persuade those still using them to give them up.

- The long-term benefits to the patient of preventive caries management should be appreciated more readily by practicing dentists and should be the subject of continuing educational initiatives.

- Scientific knowledge regarding caries diagnosis (and related preventive management) has moved ahead of many traditional professional, regulatory, and advisory frameworks.

- The concepts of diagnostic thresholds should be more widely understood, and use of the ambiguous term “caries free” should be avoided.

- It should be recognized that caries diagnosis in clinical practice, clinical research, and clinical dental epidemiology will have to change in light of continuing developments in knowledge. Strategies for systematically sifting, grading, and promoting new diagnostic approaches should be put in place internationally.

- Attempts should be made to harmonize epidemiologic diagnostic methods in order to promote improved comparability and produce more reliable estimates of preventive care and restorative treatment needs.

5. How should clinical decisions regarding prevention and/or treatment be affected by detection methods and risk assessment?

    - There is a need for more reliable diagnostic methods to provide unambiguous indications of the extent, surface status, and activity of lesions.

    - There is a need for diagnostic methods that can reliably assess sealed surfaces.

    - There is a need for better tools for the diagnosis and treatment planning of secondary caries.

    - Before a decision to restore is made, clear evidence of significant cavitation or progressive dentinal involvement is needed.

    - Clinical diagnosis should lead into preventive-biased decision frameworks compatible with a PCA, PCA + OCA style of classification to avoid premature restoration of small noncavitated lesions.

    - There is a need for valid and reliable automated decision-support systems.

6. What are the promising new research directions for the prevention, diagnosis, and treatment of dental caries?

    - There is a need for more effective primary preventive products.

    - There is a need for secondary preventive products that can deliver lesion reversal prior to the cavitation stage.

- There is an urgent need for high quality studies which are well conducted and well reported, using a minimum set of data meeting international standards.
- There is a need for more studies evaluating the same lesions, both in vivo and in vitro.
- There is a need for more studies of caries diagnosis in primary teeth.
- There is a need for more studies evaluating diagnostic performance at the caries into enamel D_1threshold.
- There is a need for more studies on combinations of diagnostic methods with adjunctive and supplemental analyses.
- There is a need for more sensitive, specific, and reliable diagnostic tools for early stage caries.
- There is a need for diagnostic tools for lesions at the size where restorative intervention is indicated.
- There is a need for diagnostic tools tailored for use in epidemiologic settings.
- There is a need for diagnostic tools to detect hidden dentin caries.
- There is a need for better restorative materials with physical properties more closely matching tooth tissue and able to act as a caries preventive agent when presented with a caries challenge.
- There is also a need to develop the evidence base on how to disseminate effectively the findings of systematic reviews in dentistry and, having achieved that, how any changes in clinical practice which might be indicated can best be brought about.

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Clinical Diagnosis of Dental Caries: A North American Perspective

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The most common methods used by U.S. dentists for clinical diagnosis of pit and fissure caries are visual/tactile inspection and visual inspection aided by radiographs (Stookey, Jackson, Zandona, et al., 1999). There is also considerable interest in commercially available and innovative diagnostic systems, such as laser fluorescence (Alfano, Yao, 1981). One commercially available product, known as Diagnodent and produced by KaVo Dental of Germany, is being used by 20 percent of Canadian dentists 2 years after its introduction (Fischman, 2000); this product was introduced to the U.S. market in the spring of 2000.

The Research Triangle Institute (RTI) review concluded that the available evidence on the validity of these innovative methods is *poor*. However, this rating may have been affected by the reviewers' decision to exclude non-English-language publications. That decision understates the body of evidence, since many innovative diagnostic systems have been developed and evaluated by researchers in non-English-speaking countries (Lussi, Hotz, Stich, 1995).

A second limitation of the RTI report is the requirement for histological validation of caries diagnosis. While ensuring a "gold standard," this requirement presents a serious limitation to in vivo studies of permanent teeth. As the report's authors point out, it effectively limits the validity of in vivo studies to those that involve third molars and first premolars, but the fissure patterns and caries presentation of these teeth may not apply to permanent teeth that are clinically more significant. Omitted from the report is mention of the useful work done when investigators "dissect" carious lesions to identify false positives (Miller, Ismail, MacInnis, 1995; Lussi, 2000).

In light of all this, dental educators should emphasize to students and practitioners that current techniques have significant limitations, and test results should be interpreted accordingly (Basting, Serra, 1999). The probability is high that North American dentists have inaccurate beliefs regarding the sensitivity and specificity of their techniques for occlusal caries identification, causing them to overestimate their ability to diagnose caries correctly.

The Clinical Dilemma

Dentists often comment about the difficulty of diagnosing pit and fissure caries in permanent posterior teeth, citing examples of "hidden" lesions (Kidd, Ricketts, Pitts, 1993). They are often uncertain about when to intervene, and can find no unequivocal clinical guidelines as to the management of stained pits and fissures (Clinical Research Associates, 1999). Indeed, some speakers in continuing education programs currently advocate instrumentation of *all* stained fissures.

A recent Web-based study involving more than 400 dentists confirmed the difficulty of diagnosing stained occlusal fissures based on visual appearance alone (Rosenstiel, Rashid, in press). Practicing dentists are aware that they must choose between restorative intervention, with the attendant risk of overtreatment, and "watchful waiting," with the attendant risk of supervised neglect.

Most U.S. dentists also appreciate that the dentist's penalty for overtreatment is considerably less than for undertreatment (see table 1). Financial rewards aside, contemporary restorative techniques, such as air-abrasion and adhesive restorative materials, permit precise removal of only diseased or structurally compromised tissue (Goldstein, Parkins, 1995). These techniques are used to provide minimally sized, tooth-colored, preventive resin restorations (Ripa, Wolff, 1992; Hamilton, 1999).

Dentists and their patients also want to avoid the considerable costs of endodontic treatment and fixed or implant prosthodontics, should nonrestorative management of a "hidden" lesion be unsuccessful. There have been reports that patients prefer restorative intervention to more conservative measures (Clinical Research Associates, 1999). Although some studies of resin restorations show them to have considerable promise (Mertz-Fairhurst, Curtis, Ergle, et al., 1998), practitioners still lack comprehensive information as to their long-term effectiveness.

Clinical Recommendations

Practicing dentists have an advantage over epidemiologists in that they obtain immediate false-positive feedback when they instrument a tooth with no clinical caries, and false-negative feedback when a recall patient exhibits progression of what was an equivocal lesion. Therefore, a rational approach to caries diagnosis in the absence of reliable tests may be to treat the susceptible surfaces as a unit rather than as a series of unrelated clinical observations. A dentist could evaluate the risk factors for a particular patient to identify the most likely fissure to be carious. If the dentist then decides that surgical intervention is justified, he or she can use feedback from that procedure—particularly the extent or absence of caries—to determine if additional intervention is indicated (see figure 1). Support for this approach can be found in studies that identify examiner prediction of future caries activity as a significant predictor of caries risk (Disney, Stamm, Graves, et al., 1990).

Future Research Directions

The recommendations of the RTI review for future research provide useful guidance for researchers seeking to advance knowledge of caries diagnosis. For in vivo work they recommend a standardization of histological validation methods for carious lesions. They also recommend a standard format for the reporting of trials of methods of clinical caries diagnosis. These recommendations, however, do not overcome some of the problems inherent to in vivo studies of permanent teeth, particularly the requirement for extraction subsequent to the test. Information is being obtained on a daily basis by dental practitioners when they determine the extent of suspicious lesions through operative intervention and when they recall patients previously deemed to not require operative intervention. Careful, well-designed sampling of the outcomes of these procedures could be an important source of helpful clinical guidance.

Table 1. Comparison of overtreatment of stained occlusal fissures in permanent teeth with undertreatment

	Overtreatment with preventive resin restoration	Undertreatment with remineralization strategies and watchful waiting
Immediate Advantages	• Increased knowledge of caries extent • Satisfies patient preference • Additional fee to dentist*	• No restorative intervention needed • Lower cost to patient
Immediate Disadvantages	• Additional clinical procedure needed • Additional cost to patient and/or third party	• Uncertainty about caries extent • Patient response is variable • No fee to dentist*
Long-Term Advantages	• Reduced likelihood of extensive carious lesions	• Reduced number of restorations requiring evaluation, maintenance, and replacement • Emphasis on prevention may reduce progress of other lesions
Long-Term Disadvantages	• Average lifetime of restorations is unknown • No well-developed guidelines for the replacement of suspicious preventive resin restorations	• Increased likelihood of extensive carious lesions requiring endodontic treatment • May require more frequent recall

*With most current reimbursement methods.

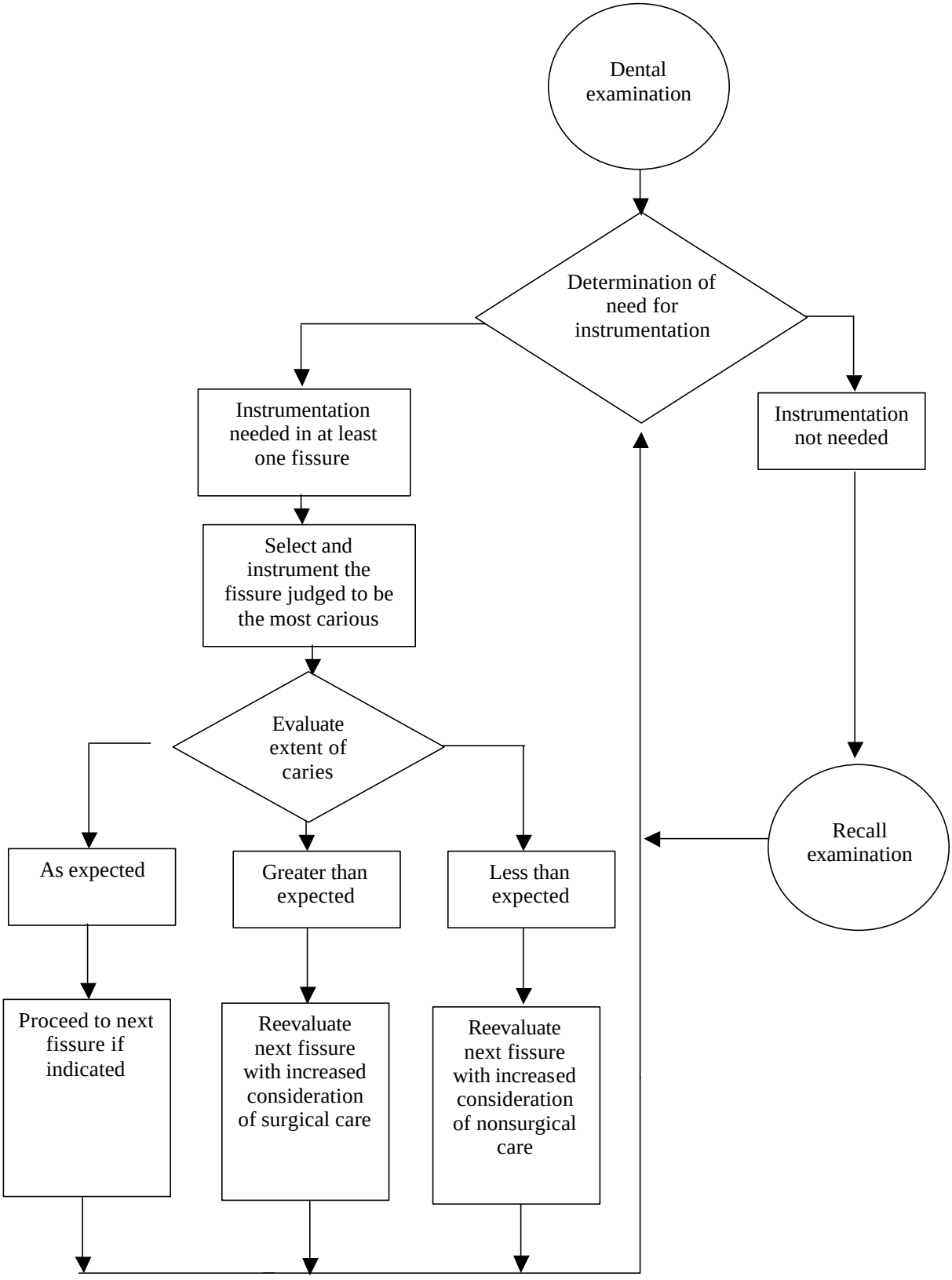

Figure 1. Management of pit and fissure caries.

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Radiographic Diagnosis of Dental Caries

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Almost since the discovery of x-rays by Roentgen in 1895, radiography has been used to detect the effects of dental caries on dental hard tissues. It has been primarily applied for the detection of lesions on the proximal surfaces of teeth that are not clinically visible for inspection. Occlusal caries may also be detected once it has progressed into the dentin.

Radiographic diagnosis of dental caries is based on the fact that as the caries process proceeds, the mineral content of enamel and dentin decreases, with a resultant decrease in the attenuation of the x-ray beam as it passes through the teeth. This process is recorded on the image receptor as an increase in radiographic density that must be detected by the clinician as a sign of a carious lesion. Many different factors can affect accurate detection of these lesions, such as exposure parameters, type of image receptor, image processing, display system, viewing conditions, and ultimately the training and experience of the human observer.

A systematic review of the existing literature was performed to address the question of the validity of six different diagnostic methods for the detection of dental caries in primary and permanent teeth. The diagnostic methods assessed included visual inspection, visual/tactile inspection, radiography, fiber-optic transillumination (FOTI), electrical conductance (EC), laser fluorescence (LF), and combinations of these methods.

Three primary computer indexes were used in searching the literature—MEDLINE, EMBASE, and the Cochrane controlled trials register. The period searched was from 1966 to December, 1999. Inclusion and exclusion criteria were clearly defined prior to performing the search. Studies were limited to those with human subjects and natural carious lesions, publication language in English, histological validation of caries status for each surface studied or visual/tactile validation of intact surface for cavitation only, outcomes expressed as sensitivity and specificity, or data provided from which these outcomes could be derived. While both in vitro and in vivo studies were included in the review, only those methods that are commercially available to the general practitioner were assessed.

Thirty-nine studies were selected from among 1,407 diagnostic reports that satisfied all criteria. These studies reported 126 different assessments of different diagnostic methods. Of these studies, 51 percent evaluated the diagnostic performance of radiographic methods. The studies were critically reviewed and a quality rating scale appraised several elements of internal validity, including study design, duration, sample size, blinding of examiners, baseline assessments, and examiner reliability. The overall strength of evidence supporting the validity of a method was judged in terms of the extent to which it offered unambiguous assessment of a particular method for identifying a specific type of lesion on a specific type of surface.

Systematic review of the dental literature indicates that the strength of evidence for radiographic methods for the detection of dental caries is poor for all types of lesions on posterior and occlusal surfaces. This is primarily due to the large amount of variation in the reported sensitivity and specificity of this method. Little, if any, evidence exists to support the

use of radiographic methods for primary teeth, anterior teeth, or root surfaces. The literature is severely limited by problems associated with both internal and external validity. These include incomplete descriptions of sample selection, diagnostic criteria, and examiner reliability; the use of small numbers of examiners; nonrepresentative teeth samples with high lesion prevalence; and the use of reference standards of questionable reliability.

Although the strength of evidence is considered poor, this does not mean that the accuracy of radiographic methods is of no diagnostic value. It simply means that using the criteria established to evaluate the existing evidence, the evidence is inadequate to validate the method. Better studies designed to address the limitations of the current literature could in fact indicate that the method is valid, but the literature does call into question the relative importance of this method in making treatment decisions.

The evidence suggests that radiographic methods have a higher degree of specificity than sensitivity, which means that false negative diagnoses are proportionally more apt to occur in the presence of disease than are false positive diagnoses in the absence of disease. This outcome may be beneficial if the negative consequences of a false positive diagnosis outweigh those of a false negative diagnosis. If the only type of intervention is surgical removal of the lesion, a false positive results in a perfectly normal tooth being irreversibly damaged. A false negative results in further progression of the lesion and potentially further loss of tooth tissue. This outcome is somewhat abated by the fact that the lesion may be detected at a later time.

Nonsurgical interventions are gaining in popularity as alternatives to mechanical replacement of damaged tooth tissue with artificial materials. These nonsurgical methods are only effective if the lesion is detected prior to cavitation. This means that the lesion must be detected early. To detect the lesion earlier a diagnostic method must provide higher sensitivity, which may result in more false positive diagnoses. If early intervention consists of nonsurgical management that does not result in any permanent damage to the tooth, the negative consequences of a false negative diagnosis outweigh those of a false positive diagnosis.

New digital radiographic techniques which eliminate the use of silver halide emulsion x-ray film by capturing radiographic images on photo-stimulable phosphor imaging plates or charge-coupled devices may improve detection of dental caries. The images acquired with these technologies are digital and can be processed or analyzed to enhance diagnostic performance. The weight of available evidence suggests that the use of some digital methods offers some small gains in sensitivity without reduction in specificity, and that image analysis techniques may offer more substantial gains.

Renewed effort should be made to ensure that future studies address the question of diagnostic validity adequately. Guidelines should be developed for assessing diagnostic methods which assist researchers in developing study designs that will hold up to critical review.

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Diagnosis of Root Caries

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It is not surprising that the Research Triangle Institute (RTI) Evidence Report on the Diagnosis and Management of Dental Caries (2000) was unable to identify any reports on the diagnosis of root caries. There simply are no evaluations of diagnostic methods for root caries that satisfy all of the prerequisites of histological validation, commercial availability, professional application, and comparative clinical study. Nevertheless, there does exist a rather extensive literature on the diagnosis of root caries.

Clinical Root Caries

There is little disagreement regarding the distribution of root caries lesions. Root caries, by definition, occurs on the root of the tooth. It can occur wholly on the root of the tooth or spread from the crown of the tooth to the root. It can occur on its own or around existing restorations.

Root caries occurs most often at or close to the cemento-enamel junction. This has been attributed to the location of the crest of the gingiva at the time conditions were favorable for caries to occur. The location of root caries has been positively associated with age and periodontal disease, which is consistent with the concept that root caries occurs in a location adjacent to the crest of the gingiva where plaque accumulates (i.e., within 2 mm). Most root caries occurs on the proximal (mesial and distal) surfaces, followed by the facial surface. Early root caries tends to be diffuse and tracks along the cemento-enamel junction of the root surface.

Clinical Signs of Root Caries

Clinical diagnosis is the process of recognizing diseases by their characteristic signs and symptoms. It is an imperfect process because there is considerable variation in both the signs and symptoms in individual subjects and in the interpretation of those signs and symptoms by different clinicians. Nevertheless, clinical observations are powerful determinants of diagnosis and prognosis. The most commonly used clinical signs to diagnose root caries utilize visual (contour, surface cavitation, color) and tactile (surface texture) parameters. There are usually no reported clinical symptoms of root caries, although pain may be present in advanced lesions.

Visual-Tactile Diagnosis of Root Caries

Using traditional methods of visual-tactile diagnosis for root caries can produce a correct diagnosis, but not until the lesion is at an advanced stage. Because of the fundamental differences in coronal and root caries, enamel caries is more likely to be confidently diagnosed at an earlier stage than root caries.

Several investigators have therefore advocated expanded classification schemes for visual-tactile root caries diagnosis that incorporate lesion activity and treatment implications. Although additional criteria can generate more information to assist with diagnosis, they can also generate more variability. Despite the subjectivity that is inherent in interpreting the clinical signs used for root caries diagnosis, acceptable interexaminer reliability has been achieved in many clinical studies. Table 1 shows the findings on several measures of examiner reliability as reported in recent studies involving clinical diagnosis of root caries.

Table 1. Reliability of visual-tactile diagnosis of root caries

Investigator(s)	Kappa Statistic (surfaces)	Intraclass Correlation Coefficient (subjects)	Agreement (percent)
Bauer et al., 1988		0.83- 0.96	
Fejerskov et al., 1991	0.88		
Saunders and Handelman, 1991			90
Graves et al., 1992		0.94	
Ravald and Birkhed, 1991	0.71		87
Wallace et al., 1993	0.80		98
Mojon et al., 1995	poor agreement		
Rosen et al., 1996	0.30- 0.51[1]	0.55- 0.75[1]	

[1]Excludes filled surfaces

Intraexaminer reliability has been shown to be slightly, but not dramatically, better than interexaminer reliability in diagnosing root caries.

Clinical diagnosis is an estimate of the probability that a patient has a specific condition after taking into account possible risk factors, clinical findings, and how commonly the disease occurs in the population. The information gained during clinical examination of the patient, together with the clinician's knowledge of the disease and his or her own clinical experience, is (consciously or otherwise) collated, analyzed, and assimilated into a "best guess" of the likelihood of a condition being present. This is the "art" of clinical diagnosis, and clinicians can become highly skilled at it. Although clinical diagnosis uses the concept of probability, it relies on practical knowledge and experience rather than the laws of probability. But because there is a high level of uncertainty associated with the diagnosis of dental caries in general and root caries in particular, clinicians have looked to other diagnostic tests for assistance.

Diagnostic Tests for Root Caries

Two central issues arise in diagnostic tests. The first relates to the validity of the test, the second to whether the test can replace or supplement what is presently being used for diagnosis. Selecting the most appropriate diagnostic test is a complex matter that must take into account test

characteristics, the clinician's "best guess" of the likelihood of the disorder being present, and the purpose of applying the test. Clinicians should be particularly interested in test specificity, since the positive predictive value will always be better with a test that has high specificity. Table 2 presents the characteristics of the diagnostic tests that have been used to diagnose root caries. Guidelines are available to assist the clinician in determining whether or not a particular test is indicated and the steps involved in applying the test and interpreting the result.

Table 2. Characteristics of diagnostic tests for root caries

Test	Investigator	Study Type	Se	Sp	Other
Mutans Streptococci	Banting, 1988	in vivo	0.46	0.93	ppv=0.75
	Ravald and Birkhed, 1991	in vivo	0.36	0.89	
Lactobacilli	Banting, 1988	in vivo	0.38	0.74	
	Ravald and Birkhed, 1991	in vivo	0.59	0.84	
Radiology	Nordenram, 1988	in vivo	0.84	0.67	
Salivary secretion rate	Ravald and Birkhed, 1991	in vivo	0.16	0.95	
Salivary buffer effect	Ravald and Birkhed, 1991	in vivo	0.47	0.78	
Oral sugar clearance time	Ravald and Birkhed, 1991	in vivo	0.26	0.85	
Fluorescent dye	van der Veen and ten Bosch, 1993 van der Veen et al., 1996 van der Veen and ten Bosch, 1996	in vitro			*r*=0.91-0.96
Fluogenic enzyme assay	Collier et al., 1993	in vivo			*r*= 0.87
Electrical conductivity	Baysan et al., submitted	in vivo			*r*= 0.76

Consensus Needs Regarding the Diagnosis of Root Caries

Terminology. The terminology used for root caries diagnosis is not standardized, a situation that gives rise to confusion and even misinterpretation in root caries diagnosis. It therefore needs to be standardized in order to facilitate precision, understanding, and uniformity. Consensus is needed on the following terms:

- Active root caries lesion
- Inactive (arrested) root caries lesion
- Primary root caries lesion
- Secondary (recurrent) root caries lesion
- Severity
- Cavitation
- Probing root lesions

Classification. Once a consensus is reached on terminology, a classification scheme needs to be developed for the determination of appropriate treatment modalities. Consensus is needed regarding the following classifications of root caries:

- Sound (uncertain)/carious
- Active/inactive
- Noncavitated/cavitated
- Observation/chemotherapeutic/debridement/restoration treatment and/or combinations of treatment.

Risk Assessment. Risk assessment methodology can be a useful approach to clinical diagnosis, but it is not widely used in dentistry. A consensus regarding the following aspects of risk assessment as it relates to the diagnosis of root caries is needed:

- The range of pretest probabilities of root caries for different population subgroups
- A "rule of thumb" guideline for test and treatment thresholds for root caries diagnosis.

Diagnostic Tests. Diagnostic tests should be used to supplement/confirm a clinical diagnosis but not as a substitute for clinical decision-making. For root caries diagnosis, a consensus is needed on the following aspects of diagnostic tests:

- When should a diagnostic test be used?
- What existing diagnostic tests are useful?
- How should a diagnostic test be used to supplement/confirm a diagnosis regarding root caries?

Areas for Future Research Pertaining to the Diagnosis of Root Caries

The diagnosis of root caries would benefit from new clinical research designed to:

1. Examine the validity of the clinical signs used to diagnose root caries by comparing them to a histological standard.

2. Determine the characteristics of diagnostic tests for root caries relative to both clinical signs and a histological standard.

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Diagnosis of Secondary Caries

Edwina Kidd, B.D.S, Ph.D., F.D.S., R.C.S.

The specific assignment is to address the findings of the Research Triangle Institute (RTI) report on the diagnosis of secondary caries and translate them into recommendations for research, clinical practice, and education. Since the report did not investigate the diagnosis of secondary caries, there are no findings. This is just as well, since:

- There is minimal literature on the subject
- The definition of secondary caries is in doubt
- There is no appropriate way to validate the diagnosis.

Definitions of Dental Caries and Diagnosis

Before justifying these statements, it is sensible to define what is meant by dental caries and by diagnosis. "Dental caries" is a result of metabolic activities in the microbial deposits covering the tooth surface at any given site. These metabolic processes are a physiological phenomenon, and caries is ubiquitous and natural at the crystal level. Mineral loss and subsequent cavity formation are the result of an imbalance in the dynamic equilibrium between tooth mineral and plaque fluid. The carious lesion reflects the activity of the biofilm, and lesion progression can be controlled (Fejerskov, 1997). "Diagnosis" implies deciding whether a lesion is active, progressing rapidly or slowly, or already arrested. Without this information, a logical decision about treatment is impossible.

The report produced concerns the detection of demineralization (Featherstone, 1996); there is no mention of lesion activity. Perhaps this is inevitable in a report that sees histological validation as an appropriate "gold standard." It is difficult to judge lesion activity histologically and unwise to attempt diagnosis in a laboratory simulation of a clinical setting. Diagnosis requires a warm human being and a clinical nose.

Questions Relevant to Secondary Caries Diagnosis

The following questions are important:

- What is secondary caries?
- Why is it important?
- Where does it occur, and why?
- What does it look like?
- What does it *not* look like?
- What are the problems in validating the diagnosis?

What Is Secondary Caries?

Secondary caries is the lesion at the margin of an existing restoration. Primary caries is the lesion at the margin of an existing filling (Mjör, Toffenetti, 2000). These definitions have been misunderstood for many years by those working only in the laboratory (Kidd, Toffenetti, Mjör, 1992). In that setting, histological examination of artificial and natural lesions around restorations may show lines of demineralized tissue running along the cavity wall. These are called wall lesions, and they are the result of microleakage. They are very commonly seen around amalgam restorations and probably indicate initial leakage prior to sealing of the margin (Kidd, O'Hara, 1990).

It is also important to consider residual caries, which is residual demineralized tissue left in the tooth during cavity preparation. Our thoughts on how much demineralized tissue may be left during cavity preparation should have been profoundly shaken by the careful clinical studies of the Mertz-Fairhurst group (Mertz-Fairhurst, Curtis, Ergle, et al., 1998). This group removed the enamel lid from large occlusal lesions, leaving extensively demineralized dentine. The cavities were then sealed with acid-etch composite restorations. Ten-year results showed that these restorations were satisfactory—provided the patients did not escape to new dentists who took radiographs, noted the demineralization, and replaced the fillings. This work makes sense if it is accepted that dental caries is the tissue destruction caused by bacterial metabolism in the biofilm. If the process can be arrested by simply removing the biofilm, why does the symptom of the process (demineralized dentine) have to be removed at all? Why not just remove the biofilm and seal the hole in the tooth? This argument has profound implications for operative dentistry and for the validation of a diagnosis of secondary caries.

Why Is the Diagnosis of Secondary Caries Important?

This diagnosis is the main reason given by dentists for replacing fillings. Fifty to 60 percent of restorations are replaced because dentists diagnose secondary caries (Mjör, Toffenetti, 2000). Are they correct? This high prevalence is not found in controlled clinical trials, where 1 to 4 percent of secondary caries has been reported. Incidentally, only these latter trials would survive the scrutiny of a systematic review on the causes of the failure of restorations. Why are there huge differences between a general practice setting and a clinical trial? Are general practitioners poorly trained, idiosyncratic, and ignorant about this diagnosis? That explanation seems dangerously facile.

Where Does Secondary Caries Occur and Why?

This is easy to answer. It occurs in areas of plaque stagnation, and therefore the cervical margins of restorations are commonly affected.

What Does It Look Like?

Again, this is easy to answer. If secondary caries is primary caries at the margin of a filling, it looks clinically and radiographically like primary caries (Kidd, 1999).

What Does It *Not* Look Like?

There is some evidence from combined clinical and microbiological studies that ditching and staining around amalgam fillings (Kidd, Joyston-Bechal, Beighton, 1995) and staining around tooth-colored restorations (Kidd, Beighton, 1996) are poor predictors of active secondary caries.

What Are the Problems in Validating the Diagnosis?

Here, there is a major difficulty. There are few reliable validators of the diagnosis. It might be possible to use histology on freshly extracted teeth to relate lesions at the margins of fillings to the overlying plaque (Ozer, 1997). In any laboratory study, however, great care is needed not to confuse active secondary caries with old microleakage or residual caries (Merrett, Elderton, 1984).

Clinical study, where a diagnosis is made and the restoration dissected out to allow examination of the cavity beneath, may be similarly fraught with dangers (Kidd, Joyston-Bechal, Beighton, 1995; Kidd, Beighton, 1996). It would be all too easy to confuse residual caries with secondary caries. Imagine dissecting out a Mertz-Fairhurst type restoration (Mertz-Fairhurst, Curtis, Ergle, et al., 1998). Soft demineralized dentine would be present beneath the filling, but this is residual caries, not primary caries at the margin of the restoration.

Similarly, the clinical and microbiological studies referred to may oversimplify the problem (Kidd, Joyston-Bechal, Beighton, 1995; Kidd, Beighton, 1996). There are now many studies showing that the microbiological load in infected dentine is reduced when it is sealed off from the oral environment (Schouboe, MacDonald, 1962; King, Crawford, Lindahl, 1965; Mertz-Fairhurst, Schuster, Williams et al., 1979; Handelman, 1991; Björndal, Larsen, Thylstrup, 1997; Weerheijm, Kreulen, de Soet, et al., 1999). However, it is not eliminated. The relevance of these residual organisms is not clear. If Mertz-Fairhurst's work is to be believed (Mertz-Fairhurst, Curtis, Ergle, et al., 1998), they have no relevance.

The only valid test is the visual appearance of the lesions in patients. These appearances, however, are open to interpretation, and the authors of the RTI report would dismiss them as poor and insufficient evidence.

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New Diagnostic Methods

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Current diagnostic tools used in dental caries detection are not sensitive enough to diagnose the disease process in its early stages, and once a diagnosis is made restoration is frequently the only effective means of treatment. The purpose of this review is to systematically assess the available literature to determine if emerging diagnostic methods for dental caries are more efficient than traditional methods for detecting and monitoring the progress of caries in permanent and primary teeth. Inclusion and exclusion criteria were established preceding the literature search. Included articles were grouped by type of emerging technology and study design. Types of emerging technologies include laser fluorescence, light fluorescence, digital imaging fiber optic transillumination, and ultrasound. In vitro and preclinical data indicate that some of the reviewed methods show promise for the detection and monitoring of early caries lesions. However, very little clinical data are available to validate these technologies, and none can be recommended at this time as a substitute for traditional diagnostic techniques.

Definitions of "Risk" and "Risk Factors"

Brian A. Burt, B.D.S., Ph.D., M.P.H.

Risk is the possibility that an event will occur. The word, of course, is used in everyday language with more or less that meaning, but it has more specific meanings in the worlds of insurance and epidemiology. In epidemiology it is related to probability and to causality, and it is most often used to express the degree of probability that a particular outcome will occur following a human being's exposure to a particular action or event. There are very few circumstances that constitute a sufficient cause in chronic or infectious disease (a sufficient cause being one where exposure to a specific action or event will probably result in a particular outcome). If there were, it would not be necessary to deal with risk, which essentially deals with varying degrees of necessary cause (a necessary cause being human exposure to an action or event that must always precede a particular outcome). The concept of risk in epidemiological study has also spread to include broader issues, such as risk assessment and risk-benefit analysis. This paper suggests definitions of risk and risk-related terms that can be used by the consensus panel for this conference.

There is general agreement that the term "risk factor" means an action or event that is statistically related in some way to an outcome—smoking, for example, is a risk factor for periodontitis. But beyond that broad generality there is little agreement. There is uncertainty in the literature on whether a risk factor should be truly causal—that is, a necessary link in the etiological chain—or whether it can be only occasionally associated with an outcome.

There is also uncertainty about what strength of association is needed for an action or event to be called a risk factor for a disease, and just how directly it needs to be associated with the outcome. There is also disagreement over whether a risk factor must be immutable, like race or gender, or whether it is something that can be modified—for example, a smoking habit. In the current studies to determine if periodontitis is a risk factor for cardiovascular disease, it is already clear that there is a measure of association between the two factors. However, it is also evident that periodontitis is neither a necessary nor sufficient cause of cardiovascular disease, and it remains to be demonstrated whether periodontitis interacts with other factors in leading to cardiovascular disease, or whether it is causal only in particular circumstances, or whether it is not causal at all but is a marker for other conditions that may be causal—that is, people with periodontitis are likely to exhibit other factors which may be more directly linked with heart disease.

Any branch of science demands specific terminology, where words have precisely the same meaning among researchers who come from a variety of backgrounds, live and work in different parts of the world, and speak different languages. If we think about an enterprise like constructing the orbiting space station, for example, which involves multidisciplinary teams of scientists from different countries, it is clear that the project would quickly degenerate into chaos if there was not total uniformity in the meaning of many complex terms. Even in less demanding scientific projects, a failure to use precise terminology can result in frustration, inefficiency, and ultimately an inability to move our knowledge base forward.

Epidemiology is a relatively new science, and perhaps it is not surprising that there is uncertainty in our use of terms. The literature on measures of risk is replete with terms of uncertain definition, and supposedly standard terms are used in variable ways by different authors. Even the use of a supposedly standard term like "risk factor" is far from uniform. Rarely does an author define how the term is being used, and the evidence that leads to identification of a risk factor is often unclear. The term comes with a cluster of related terms like risk indicator, modifiable risk factor, risk marker, determinant, and demographic risk factor, which are often used more or less interchangeably in the literature. This sort of uncertainty means that the reader has to decide what the author has in mind.

If we turn to the standard dictionaries on epidemiology, we find they are not particularly helpful. In Last's *Dictionary of Epidemiology* (Last,1995), a risk factor (a term only in use since the 1960s) is defined as an aspect of personal behavior or lifestyle, exposure to an environmental event, or an inborn or inherited characteristic which on the basis of epidemiological evidence is known to be associated with health-related condition(s) whose prevention is considered important. That is a broad and rather loose definition that leaves unanswered questions about causal role, strength of association, and modifiability. The definition then goes on to list several different meanings that have been ascribed to the term "risk factor":

- ***Risk marker*:** An attribute or event that is associated with increased probability of disease, but is not necessarily a causal factor.

- ***Determinant:*** An attribute or event that increases the probability of occurrence of disease or other specified outcome.

- ***Modifiable risk factor:*** A determinant that can be modified by intervention, thereby reducing the probability of disease.

Last agrees that the term "risk factor" is rather loosely used, and I think we would agree that these definitions still leave important issues unanswered. In an effort to clarify the matter, Beck (1998) offered a definition that was adopted for the World Workshop on Periodontics in 1996:

> *Risk factor: an environmental, behavioral, or biologic factor confirmed by temporal sequence, usually in longitudinal studies, which if present directly increases the probability of a disease occurring, and if absent or removed reduces the probability. Risk factors are part of the causal chain, or expose the host to the causal chain. Once disease occurs, removal of a risk factor may not result in a cure.*

This definition is longer than the one offered by Last, but in my view it is much clearer. The key contributions of this definition are (a) the emphasis on a temporal sequence of events preceding the outcome; (b) the unequivocal acceptance that a risk factor is part of a causal chain; and (c) the acceptance that risk factors are involved in the onset of disease but not necessarily in its progression or resolution.

Beck argues convincingly that it must be clearly established that the action or event occurred before the outcome, or before conditions exist that make the outcome likely. This in turn means that longitudinal studies are necessary to demonstrate risk factors. However, there are many situations in biomedicine, and certainly in dentistry, where this has not been done, and indeed where it is unlikely that it will ever be done. In these circumstances, exposure to an event that is associated with an outcome only in cross-sectional data is called a "risk indicator." A risk indicator may be a probable, or putative, risk factor, but the cross-sectional evidence upon which it is based is weaker than longitudinal data. This is because a temporal association usually cannot be specified from cross-sectional data.

If these definitions of the terms "risk factor" and "risk indicator" were used consistently, knowledge would most likely progress more quickly.

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Socioeconomic and Behavioral Determinants as Risk Factors for Dental Caries Throughout the Life Span

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The Surgeon General's report (U.S. DHHS, 2000) and other reviews (Burt, Eklund, 1999) conclude that oral health is significantly related to socioeconomic status (SES), with those in the low-income segments of society being at greatest risk for dental caries. This premise is said to hold for caries incidence and prevalence among both children and adults. However, no systematic review of this relationship has been conducted, and the premise is based largely on selective reviews of the literature.

This paper presents the results of a systematic review, based on pre-established criteria, of 299 scientific papers that were deemed relevant to the topic. These 299 were selected from a total of 3,135 initially thought to be relevant. The paper also evaluates the literature on two risk factors that may partly explain SES differences in caries risk, namely, toothbrushing and infant feeding practices.

Improved prevention and management of dental caries among children and adults is the primary objective of this analysis. The results can be used to evaluate how SES serves as a risk factor for caries, and how knowledge of this risk factor can influence management of disease. The results can also be used as the basis for a research agenda on how to intervene to reduce the effects of SES on caries incidence and prevalence. Finally, results on the relationship of toothbrushing and infant feeding practices to caries risk can be integrated into an evidence-based approach to clinical management of caries.

This review focuses on eight questions:

1. Are children under 6 with primary teeth and of lower socioeconomic status at increased risk of dental caries compared with children of the same age and dentition but higher socioeconomic status?

2. Are children ages 6 to 11 with mixed dentition and of lower socioeconomic status at increased risk of dental caries compared with children of the same age and dentition but higher socioeconomic status?

3. Are children ages 12 to 17 with permanent teeth and of lower socioeconomic status at increased risk of dental caries compared with children of the same age and dentition but higher socioeconomic status?

4. Are adults ages 18 to 64 and of lower socioeconomic status at increased risk of dental caries compared with adults of the same age but higher socioeconomic status?

5. Are adults ages 65 or older and of lower socioeconomic status at increased risk of dental caries compared with adults of the same ages but higher socioeconomic status?

6. Are children under 18 who do not brush their teeth one or more times daily at increased risk of dental caries compared with children of the same age who do brush daily?

7. Are adults 18 and older who do not brush their teeth one or more times daily at increased risk of dental caries compared with adults of the same ages who do brush daily?

8. Are children over the age of 12 months who continue to use a baby bottle once or more a day at increased risk of dental caries compared with children of the same age who no longer use a baby bottle?

Search Strategy

A consultant was hired by the Institute of Dental and Clinical Research (NIDCR) to construct search terms and search in two databases, MEDLINE and EMBASE, on the subjects of the study. Because of limitations in resources, we did not conduct hand searches or search unpublished studies. This is a limitation, in that it is possible that only studies showing significant effects for the risk factors of interest have been published. This review may therefore have a bias toward showing more significant relationships than are warranted.

Selection Criteria

The selection of papers on the relation of caries to SES was limited to papers in English published in 1990 or after with 100 subjects or more in more than one SES classification. Investigations of the relation between caries and behavior were limited to studies involving toothbrushing and use of the baby bottle published in 1975 or later with 25 subjects per group. The toothbrushing studies had to include at least one of the following measures of brushing: plaque scores, calculus scores, self-reports of brushing frequency, or use of fluoride toothpaste. The baby bottle studies had to include at least one of the following measures: use of a bottle past the age of 12 months, use of a bottle when the baby was put to bed at night or at nap time, frequency of bottle use during the day, or the contents of the bottle (milk, juice, etc.). Data on breastfeeding was included where reported.

SES and Caries Among Children

The quality of the evidence demonstrating a significant inverse relationship between SES and caries among young children and adolescents was moderate. Relatively few longitudinal studies were found that assessed this relationship, but many cross-sectional studies did so. Bivariate analyses generally found a strong inverse relationship between SES and caries prevalence measured by DMFS/T indices, but few studies made a distinction between occlusal and smooth surface caries. About half of the studies used multivariate analysis to adjust for confounding variables but did not consistently find that SES had a significant effect on caries prevalence. Some of the evidence suggests that the effects of SES on caries risk are attenuated in fluoridated communities.

The evidence on the relationship of SES to caries among adults was weaker, with a smaller number of studies of only moderate quality. The problem of defining caries in adult is more difficult than for children, since the most widely used measures of caries (DMFS/T indices and the root caries index) represent accumulated years of disease. Studies that reported the number of carious lesions present in adults did not provide information on the length of time that individual lesions were present or the severity of the lesions. SES was not consistently related to caries among adults, either in bivariate or multivariate analyses.

Toothbrushing and Caries

Although there were a large number of studies on toothbrushing and caries among children, there were relatively few longitudinal studies and a limited number of multivariate analyses. The results of our review were equivocal: some studies found a strong and consistent relationship between brushing and/or other measures of oral hygiene and caries incidence/prevalence, while others did not. Some studies, in fact, found that more brushing was associated with higher rates of caries. The results of multivariate analyses, where available, also were inconsistent. Other variables significantly related to caries prevalence/incidence included the use of fluoride mouth rinses, regular dental visits, SES, and snacking.

Unlike the literature on the relationship between caries and toothbrushing among children, that on adults was quite small. Only 20 papers met our inclusion criteria. Their quality was poor, and the few longitudinal cohort studies used samples of convenience rather than representative community samples. The indicators of caries were measures of disease over a lifetime. A few included new carious lesions and recurrent decay as caries measures, but those were in the minority. It is therefore not surprising that the data on the association between caries and toothbrushing among adults is equivocal, given the limited evidence.

Baby Bottle Use and Caries

The quality of the 42 papers reviewed on this topic was generally weak; only 23 percent reported multivariate analyses. Most were cross-sectional surveys that relied on retrospective reports of bottle use, making them subject to recall bias. In addition, the majority of the studies used samples of convenience. The studies did not consistently demonstrate that prolonged bottle use, use of the bottle at bed time, or contents of the bottle significantly affect caries risk.

Conclusions

There is considerable evidence that SES may be related to caries risk. The studies in general showed that those in the lower SES groups, particularly young children, demonstrate elevated risk for caries prevalence. But the quality of the data was not strong, and the association between SES and caries risk among adults was inconsistent. Further, the studies did not provide insight into how SES influences caries risk.

Toothbrushing seems to have a protective effect against caries risk, although the quality of the studies (particularly among adults) was poor. Toothbrushing as a strategy for managing caries is not well supported by the literature.

The literature on baby bottle use in relation to caries risk was weak, and no recommendations can be made about either limiting bottle use to prevent caries or altering the current recommendations about prolonged bottle use or putting a child to bed with a bottle.

Recommendations

Longitudinal studies of socioeconomic status in relation to caries risk are needed, particularly among adults. This would require additional discussion of how to define caries as well as how to measure SES in a way that would provide a better understanding of how it contributes to poor health. Likewise, longitudinal studies of toothbrushing and baby bottle use in relation to dental caries are needed to assess the role of these risk factors in caries incidence and prevalence.

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Is Sugar Consumption Still a Major Determinant Of Dental Caries? A Systematic Review

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The recognition that sugars have an etiological role in dental caries has been with us for a long time. This relationship, however, may be changing. Per capita consumption of all sugars in the United States has risen over the last 25 years or so, while the incidence of caries in permanent teeth has declined. This changed relationship may be the result of widespread exposure to fluoride. The specific question to be examined in this review is: In the modern age of extensive fluoride exposure, do individuals with a high level of sugar intake, measured either as total amount or high frequency, experience greater caries severity relative to those with a lower level of intake?

Materials and Methods

Our review began with a search of the MEDLINE and EMBASE databases for papers on sugar and dental caries published between January, 1980, and July, 2000. The year 1980 was chosen as a reasonable starting point for the era of populationwide fluoride exposure in the United States. Only reports in English were considered for inclusion in the review. Other specific inclusion and exclusion criteria were applied, and an extensive search expression was developed with the assistance of an experienced librarian.

The initial search produced 809 reports. This set was divided into two halves alphabetically, and a different reader examined each half. The first assessment was based on each paper's title and abstract, and clearly irrelevant articles were discarded. This reduced the original 809 reports to 134. After those were read, another 65 papers were eliminated because they did not satisfy all inclusion/exclusion criteria. This left 69 papers, including 26 cohort studies, 4 case-control studies, and 39 cross-sectional studies.

Categories for scoring the individual papers were then established. The maximum score was 100, and the scores of the papers ranged from 12 to 79. In order to base the final results on papers of good quality, we included only those that scored 55 or higher, a total of 36. We then rated the risk of sugar-associated caries among the subjects of the papers according to the risk ratio correlation coefficient or beta coefficient given by the authors.

The Results

The two readers were acceptably uniform in their judgments of the papers. The correlations of readers' scores on five randomly-chosen papers was high (Pearson's $r = 0.87$), and there was no significant difference in mean scores ($p = 0.56$).

Table 1 shows the distribution of the reports that found a strong, a moderate, or a weak relation between sugars intake (any measure) and caries experience, and displays these relations by type of study design. By our criteria, only one report showed a strong relation. Nineteen papers found a moderate relationship between sugars intake and caries development, while the remaining 16 found the relationship to be weak-to-none.

Table 1. Distribution of 36 studies showing strong, moderate, and weak relation between sugars intake and dental caries by type of study design.

	Strong	Moderate	Weak	Total
Cohort studies	1	7	4	12
Case-control studies	0	1	0	1
Cross-sectional studies	0	11	12	23
Total	1	19	16	36

Discussion

The predominant design used in the papers was cross-sectional (23 of the 36), even though that was probably the weakest design with which to address the question. A cohort design would be strongest for this question, but such studies are expensive and include a number of inherent problems (e.g., nature of dietary records, definitions of meals and snacks). Of the remaining studies, 12 were cohort studies and only 1 was a case-control study.

Of the 23 cross-sectional studies, 16 studied the permanent dentition, as did 7 of the 12 cohort studies. Eight of those 12 were conducted for periods of 2 years or less, which may hardly be long enough to permit the true relationship to be discerned. Only 2 small-scale studies among the 36 dealt with root caries, and both concluded that a diet which promotes coronal caries also promotes root caries. With an aging population and greater retention of teeth, root caries is likely to grow as a public health issue.

Nearly all of the studies dealt with the relationship between the means of caries status and sugars exposure, rather than distributions. It seems likely that while the reduced risk of sugar consumption in the fluoride age has an overall population benefit, there are still some identifiable subgroups who do not benefit. Further research could focus on these differences.

The findings of our review are relevant to questions 2, 3, and 5 of the six conference questions:

2. What are the best indicators for an increased risk of dental caries?

   Persons with high sugar consumption, whether measured in frequency or amount, usually have higher counts of cariogenic bacteria than people who have low consumption. This relationship is not always linear, however, and what constitutes "high" and "low" consumption is unclear; high bacterial counts do not by themselves always relate to a clinical caries outcome. Sugar consumption, however, is likely to be a more powerful indicator of risk of caries infection in persons who do not have regular exposure to fluoride.

3. What are the best methods available for primary prevention of dental caries initiation throughout life?

   Where there is good exposure to fluoride, sugar consumption is a moderate-to-mild risk factor for caries in most people. Hence, avoiding consumption of excess sugar is a justifiable part of caries prevention, if not the most crucial aspect.

5. How should clinical decisions regarding prevention and/or treatment be affected by detection methods and risk assessment?

   A patient assessed to be at high risk for caries needs to be aware that sugar consumption increases the risk. The clinician can therefore conduct a dietary assessment to identify how sugar consumption can reasonably be curtailed. For a patient assessed to be at low risk of caries, this procedure is probably unnecessary.

In conclusion, our findings are consistent with the view that restriction of sugar consumption still has a role to play in the prevention of caries, but this role is not as strong as it was in the prefluoride era.

Further Research Needs

- Research is needed to determine dietary risk factors for root caries in older people, balanced by the effect of daily fluoride in preventing root caries.

- Research is needed to identify the factors that render some children more susceptible than others to developing caries in the presence of a high-sugar diet. It may be that such individuals are not well-exposed to fluoride, or the explanation may be more complex.

- Studies are needed of how best to bring the benefits of reduced caries enjoyed by the majority of children to high-risk children (the poor, racial/ethnic minorities).

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The Relationship Between Low Birthweight and Subsequent Development of Caries: A Systematic Review

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Low birthweight is a public health issue because it is closely related to infant mortality and a host of infant morbidity conditions. In 1997, 7.5 percent of all live births in the United States were babies of low birthweight (<2500 grams), and 1.4 percent were of very low birthweight (<1500 grams). Risk factors for low birthweight include maternal age (both <17 and >34 years), low socioeconomic status, the mother's being unmarried, and poor obstetric care during pregnancy. One especially depressing fact is that the proportion of low birthweight babies has remained fairly constant over the last 30 years.

The relationship between low birthweight and dental condition has not received much attention, and most of what has been done looks at enamel defects, such as hypoplasia, in low birthweight children. Little is known about whether low birthweight children are more prone to develop caries in later life, so this review addresses the following question: Do low birthweight children (birthweight <2500 grams) subsequently develop more caries than children with normal-to-high birthweight?

Material and Methods

Our study began with a search of the MEDLINE and EMBASE databases for English-language papers published between January, 1966, and July, 2000. Search terms included low birthweight, normal birthweight, premature birth, maternal nutrition, nutrition in pregnancy, enamel hypoplasia, hypomineralization, and hypomineralized enamel. The search terms were drawn up by an experienced librarian, and the full search expression is available from the authors on request.

The initial search produced a total of 198 reports. The first assessment was made by title and abstract, and clearly irrelevant articles were discarded. This reduced the original 198 reports to 37. These 37 were read in full by two readers. Another 33 papers were then eliminated because they did not satisfy all of our inclusion/exclusion criteria; the few differences between the readers at this point were settled by consensus.

Categories for scoring the quality of individual papers were established by the two readers, with a maximum score of 100 for each category. Table 1 shows the categories.

Table 1. Scoring categories for studies of low birthweight relation to caries

Clearly-stated research aims	12
Number of controls	10
Nature of controls	10
Stated inclusion/exclusion criteria for participants	7
Individual birthweights certified	8
Level of caries diagnosed (cavitated, noncavitated)	6
Nature of caries diagnosis (clinical, x-ray, FOTi, etc.)	7
Examiner reliability quantified	8
Confounders accounted for	12
Measure of risk stated	8
Internally valid conclusions	12
Total:	100

The Results

Only four papers qualified under the criteria applied. These were read by both readers, and the few minor differences were settled by consensus. The scores for the four papers were 61, 60, 49, and 31. None of these papers reported any relationship between low birthweight and caries development.

Discussion

One of the reports involved children who were examined soon after eruption of their primary teeth, while the others involved children between 3 and 5 years of age. All four studies assessed the condition of the primary dentition only. (That is, no study was found that related caries in the permanent dentition to low birthweight.) It should be noted, however, that many of the 37 studies found a relationship between developmental defects of enamel and low birthweight, though that issue was not specifically studied. The literature also seems to assume that developmental enamel defects are more prone to become carious than normal enamel. Low birthweight is clearly a health problem to be prevented as far as possible, and seems to be related to conference questions 2 and 5:

2. What are the best indicators for an increased risk of dental caries?

   If low birthweight does turn out to be associated with caries development, the link could either be a directly biological one through hypoplasia and other enamel defects, or it could be because low birthweight is so often a marker for deprived circumstances and all the caries risks that come with it. This review, however, found no evidence that low birthweight in itself is a risk factor for caries.

5. How should clinical decisions regarding prevention and/or treatment be affected by detection methods and risk assessment?

   When clinicians are treating a low birthweight child for caries treatment or prevention, the child should be considered at high risk of caries. Even though a direct link has not been established, low birthweight is a marker of social deprivation that often leaves a child at high risk.

Further research could include documenting any link between developmental enamel defects and subsequent caries development, and the role of birth complications, frequently with the use of ventilators and intubation, in the later development of caries. Studies should also be conducted with older children to assess the effect of low birthweight on the permanent dentition.

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The Microbiology of Primary Dental Caries

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This review was conducted to evaluate the implication of certain microorganisms in the causation of human tooth decay. It examines the evidence concerning bacterial species identified in both early and current literature to be involved in tooth decay, whether originally implicated by wild animal, experimental animal, or human data. It also discusses the source of this putative infection of humans. Attention is focused on the mutans streptococci, the sanguinis streptococci, other streptococci, the enterococci, the lactobacilli, and certain actinomycetes, all of which are resident in the human mouth.

There is an immense literature on this topic. The present review deals with studies of the microbial causes and associations with dental caries in humans, relying on cross-sectional, case-control, longitudinal, and interventional studies. It addresses tooth decay in young children having only deciduous (primary) dentition, older children and adolescents having mixed and permanent (secondary) dentitions, and adults and seniors, whose secondary dentition often presents varying degrees of root exposure. As such, patients and experimental subjects with incipient enamel lesions (white spots) and established cavitations (cavities) of the tooth crowns and root surface lesions are considered. Studies of so-called secondary or recurrent caries have been excluded from this review, as have studies done in vitro, in experimental animals, or with so-called in situ caries models.

Earlier studies have characterized the biological behavior of the implicated microorganisms. The essentials are summarized below.

Mutans streptococci colonize the host only after the first teeth erupt, and their preferential colonization site is the teeth (Carlsson, Grahnen, Jonsson, 1975; Catalanotto, Shklair, Keene, 1975); they are highly localized on the surfaces of the teeth, and their abundance in the plaque is highest over initial lesions (Duchin, van Houte 1978; Babaahmady, Challacombe, Marsh, et al., 1998); their level of colonization within the plaque is increased by sucrose consumption (Folke, Gawronski, Staat, et al., 1972; Staat, Gawronski, Cressey, et al., 1975); they synthesize molecules from sucrose that foster their attachment to the teeth (Freedman, Tanzer, 1974; Tanzer, Freedman, Fitzgerald, et al., 1974); they are rapid producers of acid from simple carbohydrates and are tolerant to low pH (Edwardsson, 1968; Tanzer, 1989); and they are recovered on cultivation of initial and established carious lesion sites (Clarke, 1924; Littleton, Kakehashi, Fitzgerald, 1970; Keene, Shklair, 1974). Interest in them grew after demonstration of their potency in induction and progression of carious lesions in a variety of experimental animals, including mono-infected gnotobiotes (Fitzgerald, Fitzgerald, 1981). Their virulence expression is strongly associated with consumption of carbohydrates, especially sucrose (Tanzer, Freedman, Fitzgerald, 1985; Kuramitsu, 1993).

Lactobacilli do not avidly colonize the teeth and may be transiently found in the mouth before the teeth erupt; they preferentially colonize the dorsum of the tongue and are carried into saliva by sloughing of the tongue's epithelium (van Houte, Gibbons, Pulkkinen, 1972); their numbers in saliva appear to be a reflection of the consumption of simple carbohydrates by the

host (Staat, Gawronski, Cressey, et al., 1975; Holbrook, de Soet, de Graaff, 1993); they too are highly acidogenic from carbohydrates and are acid-tolerant (Wood, 1961). They are often cultured from established carious lesions (Loesche, Syed, 1973). Some lactobacilli are cariogenic in experimental animals, and their cariogenicity is dependent upon consumption of carbohydrate-rich-diets.

Nonmutans streptococci of several types, including the sanguinis group of organisms, and *S. salivarius*, are extremely abundant in the mouth; some are tooth surface colonizers, some mucosal colonizers. Some are quite acidogenic from carbohydrates and are acid-tolerant (Guggenheim, 1968; Edwardsson, 1968; Nyvad, Kilian, 1990). Less evidence exists of their virulence in experimental animals.

Enterococci were the first bacteria shown experimentally to induce caries in gnotobiotic animals (Orland, Blayney, Harrison, et al. 1955). Carbohydrate users, acidogenic, and acid-tolerant, they are seldom abundant in the human oral cavity (Guggenheim, 1968; Edwardsson, 1968; Nyvad, Kilian, 1990).

Actinomycetes are abundant in the human mouth and induce root surface caries in hamsters and gnotobiotic animals (Jordan, Keys, Bellack, 1972). They are also carbohydrate users, but are not powerfully acidogenic or acid-tolerant.

Summary of Current Review

Table 1. Studies on the association of microorganisms and dental caries

Bacterial Group	Total	Interventional	Longitudinal/ Retrospective	Case-Control	Cross-Sectional
Mutans streptococci	189	25	59	20	85
Sanguinis/other streptococci	16	1	2	2	11
Enterococci	3	0	0	0	3
Lactobacilli	144	9	40	20	75
Actinomycetes	27	1	3	3	20

Randomized Clinical Trials on Mutans Streptococci

Twenty-five interventional studies which monitored the putative cariogenic flora and recorded their effects on caries scores were found in the literature. Several of these applied extremely complex strategies (e.g., Gunay, Dmoch-Bockhorn, Gunay, et al., 1998). Some focused on mitigation of the solubility of the teeth with fluorides, some on repair or sealing of

the teeth, some on diet management and/or use of sugar substitutes and thus indirectly on changing the implicated tooth surface flora, and some focused directly on the flora with mechanical plaque control or use of antiseptic agents.

Since the questions for the present review are more straightforward, those multistrategic studies confound interpretations of antibacterial effects with demineralization effects. It is understandable that investigators wish to accept this problem because of the ethical need to offer patients at high risk the best available anticaries strategies. Nonetheless, multistrategy approaches to experimental interventions set a very high threshold for detection of the effects of intervention on the flora and the attribution of anticaries responses to them. Some notable studies have been less confounded, however.

Partial suppression of mutans streptococci by topical chlorhexidine use and dietary counseling in randomized Swedish children (Zickert, Emilson, Krasse, 1983) inhibited mutans streptococcal recoveries and carious lesion development during 3 years, while lactobacillus titers in saliva were not detectably affected.

Treatment of primiparous mothers with 3- to 8-month-old infants in a Swedish community, alternately assigned to treatment or control groups, was aimed at reduction of mutans streptococcal salivary levels by sucrose avoidance counseling, professional toothcleaning (and topical fluoride application), oral hygiene instruction, and excavation of large carious lesions if present, and—if test mothers had salivary mutans streptococcal levels that exceeded a pre-set threshold—by treatment with topical chlorhexidine. This strategy increased the time to colonization by mutans streptococci of young children, time to caries experience of those children, and severity of caries experience of those children (Köhler, Andreen, Jonsson, 1984). There was no significant difference in salivary lactobacilli. Preventive strategies were discontinued when children were detected as colonized. The study ran until children were 36 months old. Four years later, when the children were 7 years old, treated mothers had lower mutans streptococci and lactobacilli than control mothers (Köhler, Andreen, 1994). Far lower percentages of children of treated mothers carried mutans streptococci compared with children of control mothers. The children of test mothers who were carriers also had lower levels of mutans streptococci than control children. Twenty-three percent of the children of test mothers were caries free, compared to 9 percent of the children of control mothers, and total group caries experience for test and control children was 5.2 vs. 8.6 def.

A similar strategy was used to treat 50 to 60-year-old Swedish patients of private dentists (Rask, Emilson, Krasse, et al., 1988). Two randomized groups of high and low risk patients (defined by salivary mutans, salivary flow rate, and buffer capacity) were assigned the test protocol or served as controls who were given standard care as deemed appropriate by their dentists. At year's end, the treated high risk group had lower caries increments and lower mutans and lactobacillus titers than high risk controls, but there was no difference between the two low risk groups. The intervention was discontinued. Four years later there was no difference in microbiological parameters or caries increment between the treated and untreated high risk and low risk groups, and the one-year differential benefits of the test intercession had been lost.

A 3-year study (Gisselsson, Birkhed, Björn, 1988) of 12-year old Swedish children, using an intervention of chlorhexidine-impregnated dental floss treatment of approximal surfaces

compared with placebo-impregnated floss or no floss resulted in about a 50 percent reduction of new DFS in the chlorhexidine-floss compared with the placebo-floss group, and about a 60 percent reduction compared with the no floss group. Chlorhexidine-impregnated floss effects were about 42 percent better than placebo-floss. Salivary monitoring (rather than approximal plaque monitoring) found no differences among the groups, as could be expected.

A 3-year intensive program (Carlsson, Struzycka, Wierzbicka, et al., 1988) focused on personalized education, excavation of cavities, fluoride varnish, professional toothcleaning, and oral hygiene instruction. Study participants were randomized by school class and had group instruction on sugar avoidance, toothbrushing, fluoride toothpaste use, and were provided brushes. The personalized program resulted in about a six-fold decline of new DFS in 10 to 12-year-old Polish children and, after 3 years, significant reductions of mutans and lactobacillus salivary counts.

A 2-year randomized group study of 13-year-old Swedish children (Lindquist, Edward, Torrell, et al., 1989) compared supervised chlorhexidine gel treatment to fluoride varnish, topical FeAlF professional application, or an untreated control group. The antibacterial treatment resulted in about a 50 percent reduction of new DFS when compared with the untreated controls and lesser but still substantial and significant DFS reductions compared with the fluoride groups. There was correlated reduction of salivary mutans streptococci in the chlorhexidine group.

Finnish children 10 to12 years old were randomized to either high content xylitol gum use or not, during the first experimental phase (Isokangas, Tenovuo, Söderling, et al., 1991). Two years later, when the controls were randomly recruited for evaluation, it was found that some had begun to use xylitol gum. Approximal plaque mutans levels were lower in the xylitol users, and continuous users of xylitol gum had lower decay scores 6 years after the beginning of their use than nonusers. Mutans streptococci were lower at approximal sites that were clinically and radiographically sound than at decayed sites.

The use of a xylitol chewing gum by Finnish mothers (Söderling, Isokangas, Pienihäkkinen, et al. 2000; Isokangas, Söderling, Pienihäkkinen, et al., 2000) until their children were 3 years old was recently reported to inhibit the colonization of their children and reduce the caries experience of those children during a 5-year period of observation. Mothers were randomized to either xylitol gum use, chlorhexidine varnish, or fluoride varnish applications. The children did not use the gum or receive varnish treatments. The probability of being caries free was 70 percent for nonmutans colonized children compared to about 25 percent for mutans colonized ones at 5 years of age, and the group mean dmf score for the xylitol intercession cohort was 0.83, while scores for the chlorhexidine and fluoride varnish groups were 3.22 and 2.87, respectively.

Sixty-four longitudinal (prospective and retrospective) and case control studies indicate an important role of mutans streptococci in caries. They examined the relationship between salivary titers or plaque relative abundance of mutans streptococci (and often simultaneously quantified other bacteria, especially lactobacilli, actinomycetes, and sanguis streptococci) as well as inception, prevalence, or incidence of carious lesions. Many studies used randomized subjects, some being dental or medical patients; some subjects were almost totally naive dentally. Some studies have used population samples, and some compared cohorts with high or low caries

experience, fluoridated or nonfluoridated communities, diverse racial/ethnic groups, diverse socioeconomic groups, diverse methods of paying for dental health care, ambulatory and nonambulatory health status, and diverse ages. The longitudinal, case-control, and cross-sectional (not discussed here) studies involved all of the continents except Antarctica. Several of these diverse studies are cited here (deStoppelaar, van Houte, Backer-Dirks, et al., 1969; Edwardsson, Koch, Obriuk, 1972; Loesche, Straffon, 1979; Alaluusua, Renkonen, 1983; Loesche, Eklund, Earnest, et al., 1984; Kristoffersson, Grondahl, Bratthall, 1985; Lang, Holtz, Gusberti, et al., 1987; Kingman, Little, Gomez, et al., 1988; Wilson, Ashley, 1989; Russell, MacFarlane, Aitchison, et al., 1991; Disney, Graves, Stamm, et al., 1992; Bjarnason, Köhler, Wagner, 1993; Schroder, Widenheim, Peyron, et al., 1994; Drake, Hunt, Beck, et al., 1994; Alaluusua, Malmivirta, 1994; Sigurjons, Magnusdottir, Holbrook, 1995; Hallonsten, Wendt, Mejare, et al., 1995; Grindefjord, Dahloff, Nilsson, et al., 1995, 1996; Twetman, Petersson, 1996).

These and other reports, with few exceptions, support a strong positive statistical association of mutans streptococci with inception or incidence of carious lesions. They often report concomitant positive associations with lactobacilli, especially if saliva, rather than discrete plaque samples, were monitored. They sometimes reported negative associations of sanguinis streptococci with mutans streptococci and with lesions. Some suggest that *S. sobrinus* are favored in their ability to colonize by preexisting *S. mutans* colonization. There is also suggestion of an association between *S. sobrinus* and lactobacilli.

These studies often gathered data on other variables of interest – socioeconomic status, sucrose consumption (usually as food types or patterns of consumption), fluoride exposure, oral hygiene status, breast feeding or close personal contact between mothers and their children, and, especially, initial caries status. Some studies asked the clinical examiners to predict the decay experience of study participants.

Some of these studies focused on a related question—the prediction of caries as a function of the sum total of all or many of the variables of interest to cariologists—rather than the microbiological variables targeted in this review. When predictive values were estimated and when multiple regression models included other caries-associated variables (such as candy or soft drink consumption, oral hygiene, SES, and, especially, prior numbers of lesions) and included them in the prediction model, the amount of variance explained by the bacteria of interest became predictably smaller. Prediction of the dependent variable (caries score) by inclusion of the baseline caries score as an independent variable appears inherently tautological in the context of explaining causation of the disease (and is arguably a post hoc, ergo propter hoc problem).

Discernment of microbial etiology from several longitudinal (and cross-sectional) studies was undoubtedly blunted by using salivary (or pooled plaque) monitoring of mutans streptococci as a surrogate for small samples of plaque in areas of high caries risk, as knowledge of the biology of mutans streptococci and expected locations of carious lesions would have seemed to dictate.

Lactobacilli. All of the concerns about confounding and the ambiguity of interpretation in interventional clinical trials stated above for mutans streptococci are applicable to lactobacilli

as well. Several of the random clinical trials that yielded data on mutans streptococci also evaluated changes in lactobacilli. Generally, they resulted in inconsistent evidence that inception of carious lesions in children or decreases of incidence were associated with lactobacillus titer changes in saliva (Köhler, Andreen 1994; Rask, Emilson, Krasse, et al., 1988; Carlsson, Struzycka, Wierzbicka, et al., 1988; Lindquist, Edward, Torell, et al., 1989).

Longitudinal and case-control studies are perhaps more informative. Lactobacilli are late colonizers of the mouth (Hemmens, Blayney, Bradel, 1946; van Houte, Gibbons, Pulkkinen, 1972; Carlsson, Grahnen, Jonsson, 1975; Schroder, Widenheim, Peyron, et al., 1994; Babaahmady, Challacombe, Marsh, et al., 1998). Lactobacilli are recovered from carious lesions, but they are later colonizers of those lesions than mutans streptococci (Loesche, Eklund, Earnest, et al., 1984; Crossner, Claesson, Johansson, et al., 1989; Holbrook, de Soet, de Graaff, et al., 1993). Some data suggest that they are favored in their ability to colonize by preexisting colonization by mutans streptococci, especially *S. sobrinus*. These data thus indicate that lactobacilli are not requisite for the development of lesions. Nonetheless, they may potently contribute to demineralization of the teeth once lesions are established on either crowns or roots (Boyar, Bowden, 1985; Ravald, Hamp, Birkhed, et al., 1986; Fure, Romaniec, Emilson, et al., 1987; Scheinin, Pienihäkkinen, Tiekso, et al., 1994; Grindefjord, Dahllof, Nilsson, et al., 1995; Mazengo, Tenovuo, Hausen, et al., 1996; Fure, 1998). Little information is available concerning the species of lactobacilli that colonizes the human tongue and teeth.

Nonmutans Streptococci. Essentially no data support a causative role for sanguinis streptococci or *S. salivarius* in human caries. In fact, some data suggest an inverse relationship in the abundance of sanguinis streptococci and mutans streptococci, and also that sanguinis streptococci are inversely related to lesion development (deStoppelaar, van Houte, Backer-Dirks, et al., 1969; Loesche, Straffon, 1979; Bowden, Ekstrand, McNaughton, et al., 1990; Emilson, Ravald, Birkhed, et al., 1993).

Enterococci. Essentially no human data support a significant role of enterococci in the development of human carious lesions or in their prevalence in the human mouth.

Actinomycetes. Actinomycetes are prevalent in the human mouth and are frequently found in association with both carious and sound root surfaces, as well as sound crown surfaces. Evidence of their role in root surface carious lesion induction from interventional, longitudinal, case-control, or cross-sectional data is variable and inconclusive. In fact, these data sometimes suggest that actinomycetes are more reflective of noncariogenic than cariogenic status, in contrast with mutans streptococci and lactobacilli.

Just as modern molecular and genetic methods are now used in forensic science, they are also used to trace the spread of infection. They provide perhaps the strongest evidence of the source of transmission of infection. That evidence will be briefly abstracted here. Nonetheless, other evidence of the source of transmission of the bacteria etiologically involved in caries from experimental and longitudinal studies is consistent with even more compelling genetic investigations. Convincing data on the source of infection by cariogenic bacteria almost entirely pertain to mutans streptococci (see table 2).

Table 2. Studies on the transmission of bacterial species implicated in dental caries

Bacterial Group	Total	Molecular and genetic tracing: bacteriocin/ mutacin/phage typing/ endonuclease mapping/ ribotyping	Interventional	Longitudinal/ Case-Control	Cross-Sectional
Mutans streptococci	40	17	8	13	1
Sanguinis/other streptococci	1	0	0	1	0
Enterococci	0	-	-	-	-
Lactobacilli	7	-	4	3	0
Actinomycetes	0	-	-	-	-

Study of mutans streptococci isolated from children and their parents/siblings/caretakers as to bacteriocin typing, phage typing, mutacin typing, endonuclease DNA mapping, and ribotyping establish that these bacteria are transmitted to humans early in their lives, mainly from their mothers (Berkowitz, Jordan, 1975; Berkowitz, Jones, 1985; Caufield, Ratanapridakul, Allen, et al., 1988; Kulkarni, Chan, Sandham, 1989; Caufield, Walker, 1989; Li, Caufield, 1995; Emanuelsson, Li, Bratthall, 1998; Redmo Emanuelsson, Wang, 1998; Gronroos, Saarela, Matto, et al., 1998). Only two reports suggest significant patrilineal transmission. While it is common for children to share more than one genotype or bacteriocin type of mutans with their mothers, failure to detect all of the types among mother/child pairs suggests that some may be lost with time. New genotypes have been reported to colonize children during longitudinal studies, suggesting that extrafamilial transmission also occurs.

Longitudinal study of children led investigators to propose the existence of a "window of infectivity" by mutans streptococci (Caufield, Cutter, Dasanayake, et al., 1993), but that concept does not appear well-supported. Children become colonized both before and after the "window" period (Aaltonen, Tenovuo, 1994; van Loveren, Buijs, Bokhout, et al., 1998; Straetemans, van Loveren, de Soet, et al., 1998; Mohan, Morse, O'Sullivan, et al., 1998). Also, as reported in essentially all of the studies of adults (cited above), virtually all dentate adults appear colonized to some degree by mutans streptococci. There are likely to be other events of transmission or, alternatively, the methods historically used to cultivate mutans streptococci may fail to detect transmission which has in fact occurred.

Interventional studies of transmission are clearly inhibited by the ethical impossibility of exchanging children with mothers shortly after birth. Nonetheless, controlled experiments aimed at altering the probability of transmission of mutans streptococci from mothers to their children support the concept that the mother is the usual source of transmission to her child (Köhler,

Andreen, 1994; Brambilla, Felloni, Gagliani, et al., 1998; Söderling, Isokangas, Pienihäkkinen, et al., 2000).

There are few data on the source of transmission of lactobacilli to children. Despite the use of very specific selective media for the cultivation of lactobacilli, speciation of them is laborious and is usually not done in an epidemiological context. Also, the literature does not yield studies of the genetics of the lactobacilli in the mouth, vagina, and gastro-intestinal tract of mothers and their children. It is clear that while lactobacilli can be found in the mouths of infants, they appear to be transient and are not a common feature of the oral cavity until after teeth erupt or obturators are placed for cleft palate management. There is even less information on the source of colonization of the mouth by sanguinis group streptococci, enterococci, and actinomycetes. *S. salivarius* is long known to colonize the mouth, usually within a day of birth.

Conclusion

Evidence from the current review strongly supports a central role of the mutans group of streptococci in the initiation of caries on the smooth surfaces and fissures of the crowns of the teeth of adults and children, and suggests that they have a potent etiologic role in the induction of root surface caries. Lactobacilli are also implicated as important contributory bacteria in tooth decay, but their role in induction of lesions is not well supported. Evidence that other streptococci, enterococci, or actinomycetes are prominent etiological agents of dental caries in humans is equivocal at best. The mutans streptococci are spread vertically in the population, mostly but not exclusively from mothers to their children.

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Inherited Risks for Susceptibility to Dental Caries

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Dental caries incidence is affected by host factors that may be related to the structure of dental enamel, the immunologic response to cariogenic bacteria, or the composition of saliva. The specificity of these factors is dependent on the genetic makeup of each individual and the expression of specific genes. It is possible that allelic variation related to a host factor may contribute to increased risks for the development of carious lesions. The present review examined the literature to address the question, Is the risk for dental decay related to patterns of genetic inheritance?

The basic sequence of the human genome is now becoming readily available. The information contained in the genome will provide new approaches to understanding the etiology of human disease and provide new opportunities for diagnosis and management. There have been numerous reports that there is a genetic contribution to the development of dental caries, but there has been no evidence-based analysis of those reports. Establishing a basis for a genetic contribution to dental caries will provide a foundation for future studies of the disease process.

The evidence shows that inherited disorders of tooth development that result in altered enamel structure increase the incidence of dental caries. Dental enamel that is insufficiently mineralized and retains organic components is more susceptible to decay. Patients affected with these syndromes can be readily identified and categorized by well-accepted diagnostic criteria. Such patients are often identified prior to the onset of extensive dental caries on the basis of appearance of the teeth.

Thus, the genetic mutations that are associated with these syndromes provide a link between inheritance and increased susceptibility to dental caries. The specific genetic linkage for all of these syndromes of altered tooth development has not yet been determined. Consequently, it has not been possible to complete genetic screens of large populations to determine whether the same genes/mutations are also associated with increased susceptibility to dental caries in nonsyndromic patients.

Alterations in the immune response to cariogenic bacteria may also increase the incidence of caries. There have been reports of a relationship between human histocompatibility antigen types and an increased incidence of dental caries. At this time the association between specific patterns of HLA genetic inheritance is weak and does not provide a predictable basis for predicting future decay rates. Additional research is required to further examine the contribution of specific HLA types and the risk for dental caries.

Salivary function is critical to maintaining dental enamel mineralization and altering the pathogenicity of cariogenic bacteria. The evidence is strong that xerostomia greatly increases dental caries risk. There is only very weak evidence that xerostomia has a defined genetic basis rather than being the result of some acquired effect that reduces the functioning of the salivary glands. Information on saliva constituents and dental caries is insufficient to make a determination of genetic linkages predisposing to dental caries.

The evidence supporting an inherited susceptibility to dental caries is limited, but information generated from the human genome project should provide a resource for further investigation of the genetic contribution to dental caries. Genetic linkage investigations of well-characterized populations with clearly defined dental caries incidence will be required to further analyze the relationship between inheritance and dental caries.

Exposure to Metal Ions and Susceptibility to Dental Caries

William H. Bowen, B.D.S., Ph.D.

There are large unexplained disparities in the prevalence of dental caries from one region of the United States to another. Disparities in the levels of caries that have not been explained by conventional hypotheses are found within states, counties, and cities. The highest prevalence of dental caries in children is found in the northeastern part of the United States and in the inner cities. Coincidentally, those are also the areas where the highest exposures to lead occur.

There are good theoretical reasons for believing that exposure to lead during and possibly after tooth formation may enhance susceptibility to dental caries. Lead in its atomic structure resembles calcium and may replace calcium in the bones and teeth of young people, thus altering their solubility and other properties. Furthermore, lead may combine with fluoride to form lead fluoride, which is virtually insoluble. It is also well recognized that exposure to lead during fetal development may affect the maturation of infants' sympathetic and parasympathetic innervation, which have been shown to affect the development of salivary glands. Reduced salivary flow enhances susceptibility to dental caries.

Lead and Disease

Lead is one of the most toxic and pervasive pollutants in our society. High levels of lead in the blood are the most prevalent environmental threat to the health of children in the United States (Healthy People 2000). The Centers for Disease Control has lowered the acceptable concentration of lead in the blood in young children from $\leq$25 to $\leq$ 10 ug/dL. Despite the documenting of lead's danger to health, however, little information has been obtained on the toxicity of lead to oral health. Nevertheless, the preponderance of existing epidemiological data show an adverse relationship between lead in the environment and the prevalence of dental caries. Furthermore, all the available data show that lead may disrupt the formation of enamel and dentin. The results of studies conducted with rats also illustrate the potential for lead to affect salivary gland function adversely. We have identified seven clinical studies between 1969 and 1999 that showed a positive correlation between elevated levels of lead in soil, drinking water, and tooth enamel, and prevalence of dental caries. One study showed no correlation between levels of lead in enamel and the prevalence of caries. Two studies using rats showed a positive relationship between prenatal and perinatal exposure to lead, levels of lead in enamel, and incidence of dental caries. On the other hand, numerous studies have failed to show a relationship between postnatal exposure to lead and caries experience in rats.

We did not find any literature on studies exploring the effect of lead on salivary gland function in humans. Results from three studies conducted with rats, however, show very clearly that exposure prenatal or postnatally may reduce stimulated salivary flow. The effects on resting flow were not explored.

Although the clinical studies mentioned above may have flaws, the relationship between lead exposure and caries is consistent. Results from humans and animals show that enamel accumulates lead, and that enamel formation can be adversely affected.

Many states now require that the blood levels of lead in infants be determined and recorded. If it is agreed that exposure to lead constitutes a risk for dental caries, the blood lead levels of children should be part of their dental record. This information could form the basis for preventive measures and alert the dental practitioner to behavioral and other problems associated with lead intoxication.

Physical and Chemical Aspects of Saliva as Indicators of Risk for Dental Caries

Cataldo W. Leone, D.M.D., D.M.Sc., and Frank G. Oppenheim, D.M.D., Ph.D.

Dental caries remains a widely prevalent bacterial infection despite tremendous advances in prevention and treatment, and continues to comprise a significant portion of total U.S. expenditures on health care. Why caries continues to be a major public health problem remains an unanswered question, but insight may be gained through assessment of the risk factors associated with the disease. The etiology and pathogenesis of dental caries are known to be multifactorial, but the interplay between intrinsic and extrinsic factors is still not fully understood. As in other host/parasite interactions, there appear to be marked variations in individual susceptibility to the disease. It therefore appears that intrinsic host factors play a key role in modulating the initiation and progression of caries. This report offers a critical evaluation of the role and effects of saliva in caries pathogenesis.

Focused Questions

The general question addressed is: Is there clinical evidence that saliva has a protective effect against caries? Such an evaluation is complicated by the fact that saliva is a complex body fluid whose clinical and physical properties show considerable intra- and intersubject variability. In addition, a number of medical conditions lead to salivary alterations which, in turn, may increase the risk for caries. To develop a comprehensive search strategy, we addressed the following questions:

1. Are individuals with altered salivary physiology at increased risk for dental carious lesions compared with individuals of the same age and dentition with normal salivary physiology?

2. Are individuals with altered electrolyte biochemistry in saliva at increased risk for dental carious lesions compared with individuals of the same age and dentition with normal electrolyte biochemistry?

3. Are individuals with altered macromolecules in saliva at increased risk for dental carious lesions compared with individuals of the same age and dentition with normal salivary macromolecular composition?

4. Are individuals with medical conditions or diseases that affect saliva at increased risk for dental carious lesions compared with individuals of the same age and dentition who do not have such conditions/diseases?

Search Strategy

To deal with these questions, we conducted a broad-based search in the MEDLINE and EMBASE databases to ensure that we found all potentially relevant information in English. Search dates depended on the database, but ranged from 1970 to August, 2000. One broad caries hedge was used with each of four saliva hedges developed for the four questions. This resulted in the retrieval of eight sets of literature and a total of 3,086 articles. In addition, we conducted hand searches of bibliographies and abstracts that were not retrieved initially (IADR/AADR, ICOB, ORCA). We also sought opinions and guidance from experts in the field.

Selection and Exclusion Criteria

Abstracts were then handscreened by one reviewer to identify duplicates and to exclude articles clearly inappropriate to our review (e.g., caries or salivary status not clearly defined). The literature sets were then merged into one new set of about 600 abstracts. Full-length articles were subjected to a second round of screening with additional inclusion criteria, resulting in the final number of articles formally reviewed and included in the evidence table. The additional criteria were English-language articles reporting original in vivo studies with a defined control group between 1986 and August, 2000, with $\geq$30 subjects. All longitudinal studies meeting these criteria were included. Otherwise, only articles satisfying AHRQ level II-3 or above were included. Consequently, purely descriptive studies of large subject populations were excluded from the evidence table, but they are described in the evidence report. Articles or portions of articles which dealt with salivary microbiology, fluoride treatment, or food and nutrition factors were deemed beyond the scope of the present review.

Data Collection and Analysis

We developed an extraction form to ensure complete and consistent collection and abstraction of data. This form was used to facilitate calibration and to produce a preliminary evidence table. Once agreement between the extractors was attained, data from the articles were entered directly into the evidence table. Two persons independently abstracted data from each article. Data were synthesized descriptively according to (1) general description; (2) experimental design; (3) caries status assessments; (4) saliva status assessments; and (5) clinical evidence for the presence or absence of a protective effect of saliva against caries. We focused on both quantitative and qualitative aspects of saliva to evaluate the relationship between caries and salivary status. Salivary parameters deemed important were salivary flow rate, buffer capacity, and the amounts of salivary constituents belonging to the immune and nonimmune defense systems. The data were not further analyzed quantitatively, and no meta-analysis was conducted.

Principal Results

The preponderance of the literature supports the belief that a normal salivary flow rate imparts a strong protective effect against caries. This effect remains consistent, for the most part, regardless of salivary source (whole saliva or glandular secretions) or stimulation status

(stimulated or unstimulated; masticatory or gustatory stimulation). Significantly diminished salivary flow rate, on the other hand, is associated with a number of predisposing medical conditions, reflecting either the predisposing medical condition itself (e.g., Sjogren's syndrome) or treatment of the condition (e.g., head and neck radiation; medications exhibiting xerostomic side effects). The overall result points clearly toward salivary gland hypofunction causing lowered secretion rates; this, in turn, tends to increase the caries risk. There is little evidence to suggest that normal healthy individuals have idiopathic alterations in salivary secretion rates.

There also is reasonably good evidence of protection against caries because of salivary buffering capacity. This parameter is usually measured using a salivary pH endpoint in acid-base titrations. Individuals with a lower (i.e., more acidic pH) value are deemed to have diminished buffer capacity, and they seem to be at increased risk for caries. The literature is somewhat unclear on this characteristic, however, because buffering capacity involves extrinsic factors, such as dietary and oral hygiene habits, as well as intrinsic factors, such as salivary bicarbonate content. As a consequence, buffer capacity appears to be a weak-to-moderate predictor of caries risk when considered as a single independent variable.

Surprisingly, the literature was almost equally divided for or against the protective role of salivary immunoglobulins, especially secretory IgA. Studies evaluating caries risk in subjects with humoral immunodeficiency do not report a consistent pattern. Some immunodeficient individuals appear to have increased susceptibility, while others demonstrate one or more compensatory salivary mechanisms (both immune and nonimmune) which may obviate any increased caries risk.

Finally, there is insufficient evidence on whether other physico-chemical characteristics of saliva provide a protective effect against caries. A small number of articles suggest that certain components of saliva are protective (e.g., salivary peroxidase, lysozyme, lactoferrin, histatins, and other antimicrobial proteins), but these associations have not been well-demonstrated. Large intra- and intersubject variability is a recurring issue, and it is not clear if this reflects human variation or limitations in experimental approaches.

Conclusions

Saliva provides a general protective function for exposed oral hard tissues, such as enamel and dentin, and a clinically significant decrease in salivary flow can be considered an etiologic factor contributing to caries risk. Consequently, clinicians should identify individuals with reduced salivary output and modify their treatment and prevention programs in ways that diminish the risk of caries. To a lesser degree of certainty, it can be concluded that individuals whose salivary buffering capacity is reduced are at higher caries risk. Thus, the general salivary parameters of flow rate and buffer capacity are clinically useful diagnostic indicators.

No convincing evidence is presently available, however, that other biological characteristics of saliva are useful in predicting an increased risk of caries. The role of the salivary immune and nonimmune systems remains uncertain, but it is likely that further research in this area will clarify such issues.

Effectiveness of Methods for the Primary Prevention of Dental Caries: A Review of the Evidence

R. Gary Rozier, D.D.S., M.P.H.

Effective caries-preventive methods for use by dental professionals, by individuals, and by public health practitioners have been developed and refined since the introduction of community water fluoridation in the 1940s (U.S. DHHS, 2000). The literature on these methods is extensive. This paper summarizes the evidence for the effectiveness of the preventive methods available to dental professionals and includes professionally administered fluoride, pit-and-fissure dental sealants, antimicrobial agents, and counseling of patients. Counseling can involve a large number of recommended actions to be performed by the patient outside the dental office, such as use of fluoride products, use of antimicrobial agents, oral hygiene practices, and consumption of food containing sugar substitutes. Those are not included in this review.

Review Method

A systematic search of the literature published in English from 1980 through October, 2000, was undertaken in MEDLINE, using the primary search words "caries," "carious," -analysis," and "review." EMBASE was searched for the period 1988 through June, 2000, using the same search strategy and keywords. Articles that did not focus on the caries-inhibiting effect of preventive methods were excluded. The 821 articles retrieved through MEDLINE and the 206 in EMBASE were examined for specific preventive methods. A search of nonelectronic sources was also conducted to identify reviews not published in peer-reviewed journal literature.

Search Results

Close to 40 reviews were identified that focused on the clinical effectiveness of fluorides, pit-and-fissure sealants, antimicrobial agents, and patient counseling. Four reviews were identified that included multiple preventive methods. The search identified systematic reviews of professionally applied topical fluoride gels (van Rijkom, Truin, van't Hol, 1998), fluoride varnish (Helfenstein, Steiner, 1994), pit-and-fissure sealants (Llodra, Bravo, 1993), antimicrobials (van Rijkom, Truin, van't Hof, 1996) and patient counseling (Kay, Locker, 1996; Kay, Locker, 1998; Sprod, Anderson, Treasure, 1996).

Conclusions

The overall preventive effect of professional fluoride gel treatments on caries increments between children treated and children not treated was between 18 and 25 percent. Clinical investigations of the application of fluoride varnish to permanent teeth of children provided preventive effects of between 25 and 50 percent.

Placebo control studies have been deemed unethical since the 1970s because of the almost universal availability of fluoride dentifrices, so few recent studies of professionally applied fluorides have been conducted. Although fluoride is clearly effective in preventing and controlling dental caries, no randomized control trials of the incremental benefit of in-office fluoride treatment for low-risk patients also exposed to fluoridated toothpaste and other sources of fluoride have been reported. Estimates of the number of patients needed for treatment with gels or varnishes to prevent a cavity (1 DMF) suggest that the additional effect of professional fluoride treatments is low in patients who are at reduced risk for dental caries. Little information is available on the caries-inhibiting effects of professional applied topical fluoride treatments in populations other than children.

The literature offers strong evidence that sealants are effective in preventing pit-and-fissure caries. The overall effectiveness of autopolymerized fissure sealants was between 69 and 72 percent. No studies have reported on the preventive effects of sealant according to caries risk status. However, estimates of the number that would have to be treated suggest that the benefit in populations at low-risk for of pit-and-fissure caries may be low.

Antimicrobial agents have been employed in high-risk patients for short periods to reduce or eliminate decay-causing bacteria. Chlorhexidine gel, the most commonly used agent in office-based care, is effective in the prevention and control of dental caries. The overall caries-inhibiting effect of chlorhexidine is between 35 and 57 percent.

A number of effective preventive methods are available to the public for individual use. The evidence on patient counseling suggests that dental knowledge can be improved with health promotion and counseling activities. However, a causal link between professional counseling in a clinical setting and use of caries-preventive methods at home has not been established.

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Prevention of Early Carious Lesions and Management of Dental Caries in High-Risk Individuals: RTI/UNC Review

James Bader, D.D.S., M.P.H.

Topic is summarized in Dr. Bader's abstract on page 25.

Fluoride: A European Perspective

Elizabeth T. Treasure, B.D.S., Ph.D., FRACDS, FDSRCS

The diversity of Europe is such that it is not possible to present one point of view as the European perspective. The use of fluoride across Europe varies greatly, from countries with fluoridation of public water supplies and household salt to countries where there is considerable use of topical fluorides to still others where the emphasis is on fluoridated toothpaste. The differences in the delivery of dental services also vary from emphasis on independent practitioners to employment of practitioners in salaried agencies. There are also wide variations in the importance given to a population approach to the prevention of disease. In essence, then, the discussion I give you has to be influenced by my background, which is that of a British practitioner of dental public health with considerable experience in undertaking systematic reviews.

The aims of this presentation are:

- To identify if any studies were missed by the RTI
- To discuss the limitations identified
- To make recommendations for future research.

The first task was to identify any studies that were missing from the review. This was undertaken in the following way:

- The searches were repeated, using slightly different key words
- The searches were limited to MEDLINE and excluded languages other than English as well as the grey literature.
- The abstracts were scanned against the inclusion criteria, and when the papers appeared to meet the inclusion criteria, they were read.[1]

Two additional papers were found that, in my opinion, fulfilled the inclusion criteria. The first (Bruun, Bille, Hansen, et al., 1985) compared a 0.2 percent sodium fluoride rinse with a difluorosilane varnish using radiographs on the approximal surfaces of molar and premolar teeth. The progression of initial lesions was slightly less in the varnish group, but statistical tests were not reported for this analysis.

The second paper (Forsman, 1974) reported a comparison of 0.2 percent sodium fluoride with 0.025 percent sodium fluoride, both used as a weekly rinse. The author reports the surprising conclusion that the 0.025 percent solution was more effective at preventing caries than the 0.2 percent solution. The picture becomes more confusing when examination focuses on initial lesions. More initial lesions progressed with the lower concentration, but more also regressed with it. Again, statistical tests were not reported for this analysis.

[1] It is important to note that, unlike the report, these processes were not double-checked.

These two additional studies do not add much to those cited in the main report. The total number of studies only increases from five to seven, and there remain the very varied study design and population characteristics with which to contend. They do not alter the conclusions of the main report.

Several issues were identified for discussion. A conflict in outcome measures was found, but the outcome measures commonly reported in clinical trials were not those that this review was looking for. This has to lead to recommendations for future research. The second problem—the ability to measure initial dental caries—has been reviewed in detail in the first part of this conference. Only if this can be done accurately in a clinical setting is it possible to evaluate accurately the effect of any clinical intervention on initial lesions.

From a European perspective, fluoride toothpaste is seen as the major item in control of caries, both at an individual level and in the public health approach. Most would only wish for a clinical method that produced better results than the use of fluoridated toothpaste by an individual. There are sound practical and ethical reasons for taking this approach. With the exception of Scandinavia, it is not possible to envisage a situation in Europe where professional application of fluoride would be available on a very frequent basis except to specific high-risk groups.

Several areas are suggested for future research. The first is to identify suitable study designs for answering this question. It is necessary that this should be specified in some detail, including the study populations to be used, the data that need to be recorded, and the confounding variables that should be considered. As suggested in the report, radiographic studies need to be reanalysed where possible, using the criteria decided in the earlier part of this conference, although the methodological problems of doing this need examination.

The third research task should be completion of further systematic reviews. These should pose slightly different questions and use different inclusion criteria. The first would look at caries preventive methods using ‘in situ’ methodology. Although this is an unusual suggestion for a systematic review, it would be of benefit here as a way of suggesting which techniques might be most promising to test in a clinical setting.

The second review would look at the effects of fluoridated toothpaste on caries in general and on initial caries in particular, while the third would look at the effects of topical fluorides on caries in general. Protocols for these are currently registered with the Cochrane Collaboration. The problems caused by the great heterogeneity of the existing studies are large, and that is something that needs to be considered in future research.

Once these tasks are finished, it will then be possible to commission appropriate research designed to fill in the lacunae identified by the reviews. These would fulfil the criteria identified in the first piece of research on study design. By planning the research in this way, it would be possible to reduce greatly the heterogeneity between studies and allow studies to be combined.

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Additional papers for evidence table

	Study	Type of Design	Duration	Country and Fluoride Status	Experimental agent	Frequency	Comparison agent	Subject Age	N of Subjects in analysis
1	Bruun, Bille, Hansen, et al, 1985	Non-RCT (double blind)	36 mo	Sweden NR	Difluorosilane varnish	Twice a year	0.2% NaF solution 10mls every 2 weeks	9 to 12 years	251
2	Forsman (1974)	RCT (double blind)	24 mo	Sweden <0.2 ppm	NaF 0.025% solution, 10 Mlles	Weekly	NaF 0.2% solution, 10 Mlles weekly	11 to 12 years	270

	Tooth Type	Surface	Exp. Lesion N	Com. Lesion N	Criteria for Non-Cavitated Lesion at Baseline	Criteria for Progression	Criteria for Reversal
1	Molar and premolars	Approximal surfaces	50	75	Radiographic changes in enamel that have not reached ADJ	Must have reached ADJ	NR
2	Molars and premolars	Approximal surfaces	91	109	Radiographic changes in enamel only	Lesion into dentine	No radiographic evidence of lesion

	No. of Examiners	Inter-Examiner Reliability	Mean Intra-Examiner Reliability	Type of Analysis	Compliance Estimate	Attrition from Baseline
1	1	NR	NR	All at final examination	NR	30%
2	1 (with confirmation when necessary)	NR	NR	All at final examination	NR	6%

	Percent of Lesions Progressing			Percent of Lesions Reversing			
	Exp.	Com.	P-value	Exp.	Com.	P-value	Quality Score
1	50	44	NR	NR	NR	NR	65
2	30	23	NR	9	3	NR	65

Topical Fluorides in Caries Prevention and Management: A North American Perspective

Ernest Newbrun, D.M.D., Ph.D.

A review of the evidence-based literature indicates that there is incomplete evidence for the efficacy of most measures currently used for caries prevention, with the exception of fluoride varnishes and the use of fluoride-based interventions in the management of patients with hyposalivation. Not all fluoride agents and treatments are equal, however. Different fluoride compounds, different vehicles, and vastly different concentrations of fluoride are used, with different frequencies and durations of application.

These variables can influence the clinical outcome with respect to caries prevention and management. The efficacy of topical fluoride in caries prevention depends on (a) the concentration of fluoride used, (b) the frequency and the duration of application, and, to a certain extent, (c) the specific fluoride compound used. The more concentrated the fluoride and the greater the frequency of application, the greater the caries reduction. Factors besides efficacy, such as practicality, cost, and expected compliance influence the clinician's choice of preventive therapy.

For noncavitated smooth surface carious lesions in a moderate caries-risk patient, the appropriate fluoride regimen would be semiannual professional topical application of a fluoride varnish containing 5 percent NaF (22,600 ppm of fluoride). In addition, the patient should use twice or thrice daily for at least 1 minute a fluoridated dentifrice containing NaF, MFP, or SnF_2 (1,000–1,500 ppm of fluoride), and once daily for 1 minute a fluoride mouthrinse containing .05 percent NaF (230 ppm of fluoride). If the noncavitated carious lesion involves a pit or fissure, the application of an occlusal sealant would be the most appropriate preventive therapy.

The management of the high caries-risk patient requires the use of several preventive interventions and behavioral modification, in addition to the use of topical fluorides. For adults and for children over 6 years of age, both office and self-applied topical fluoride treatments are recommended. For office fluoride therapy at the initial visit, a high-concentration agent, either an APF gel with 1.23 percent F (12,300 ppm of fluoride) for 4 minutes in a tray or a 5 percent NaF varnish (22,600 ppm of fluoride) should be applied directly to the teeth four times a year. Self-applied fluoride therapy should consist of the daily 5-minute application of 1.1 percent NaF or APF gel (5,000 ppm of fluoride) in a custom-fitted tray. For those who cannot tolerate tray delivery because of gagging or nausea, a daily 0.05 percent NaF rinse (230 ppm of fluoride) for 1 minute is a less effective alternative. In addition, the patient should use twice or thrice daily for at least 1 minute a fluoridated dentifrice as described above for treatment of noncavitated carious lesions.

To avoid unintentional ingestion and the risk of fluorosis in children under 6 years of age, fluoride rinses and gels should not be used at home. Furthermore, when using a fluoride dentifrice, children in that age group should apply only a pea-size portion on the brush, should be instructed not to eat or swallow the paste, and should expectorate thoroughly after brushing. Toothbrushing should be done under parental supervision. To avoid etching of porcelain crowns

and facings, neutral NaF is indicated in preference to APF gels for patients who have such restorations and are applying the gel daily. The rationale for these recommendations is discussed and important deficiencies in our knowledge that require further research on topical fluoride therapy in populations with specific needs are identified.

Pit and Fissure Sealants in High-Risk Individuals

Jane A. Weintraub, D.D.S., M.P.H.

In 1983 the National Institutes of Health hosted a consensus development conference on dental sealants in the prevention of tooth decay (NIH, 1984). The panel's conclusion was that the "placement of sealants is a highly effective means
panel said that sealants were 100 percent effective in pits and fissures that remained completely sealed, although sealant retention declines over time. Since then, there have been comprehensive reviews (Weintraub, 1989; Ripa, 1985, 1993) and a meta-analysis (Llodra, Bravo, Delgado-Rodriguez, et al., 1993) that confirmed the effectiveness of sealants and a workshop that developed guidelines for their use (Siegal, Kumar, 1995). Sealants are still needed, since 78 percent of 17-year-olds in the United States have experienced dental caries (Surgeon General, 2000), and most of the disease occurs in pits and fissures (Kaste, Selwitz, Oldakowski, et al., 1996). Sealants, however, are far from being universally applied. In 1988-94, only 23 percent of U.S. 8-year-old children and 15 percent of 14-year-old children had received sealants (U.S. DHHS, Healthy People 2010). The current charge is to examine the evidence demonstrating the effectiveness of sealants in high risk children and to discuss the findings of the Research Triangle Institute/University of North Carolina group.

The RTI/UNC group used four initial criteria to select caries management studies: (1) studies of methods applied or prescribed in a professional setting (or professional provision); (2) in vivo studies; (3) studies with a concurrent comparison group; and (4) studies using traditional outcome measures of caries experience. For studies of the management of noncavitated lesions they included studies where the lesion was the unit of analysis. For studies on the management of caries in high-risk individuals, the risk determination was "made on an individual subject level based on carious lesion experience and/or bacteriologic testing." In other words, high-risk status conferred by group membership, such as a school or community with a high caries rate or low socioeconomic status, was not sufficient.

Because of these restrictive criteria, the investigators found only one study (Heller, 1995) that met the criteria and examined sealant use in noncavitated lesions, and only two studies that met the criteria and used sealants alone (Sheykholeslam, Houpt, 1978) or sealants in combination with other preventive agents in high-risk individuals (Zickert, Emilson, Krasse, 1982). Another sealant study was listed in the references but is not found in the tables (Carlsson, Petersson, Twetman, 1997).

This presentation will describe the RTI/UNC criteria, as well as those four studies and their limitations, in more detail. Additional studies are also discussed to better reflect the nature of sealant studies and include the studies that appear in this abstract's tables 1 and 2.

Many of the first trials of sealants used a half-mouth design where children with one or two pairs of sound, homologous molars were included. Sealant was applied to one randomly selected molar while its pair was left unsealed. Most of those trials did not specifically discuss caries risk status, but review indicates that some of them specifically selected children with prior caries experience (Buonocore, 1970, 1971; Brooks, Mertz-Fairhurst, Della-Giustina, et al., 1976; Mertz-Fairhurst, Fairhurst, Williams, et al., 1984; Sheykholeslam, Houpt, 1978; Houpt, Shey, 1983; McCune, Bojannini, Abodeely, et al., 1979), either in general or specifically first permanent molars. In the latter case, studies such as those by Rock, Gordon, and Bradnock (1978) and Rock and Evans (1982) required all four first permanent molars to be erupted and caries-free in 6-7 and 8-year-olds, respectively. Thus, these children might have been at lower caries risk than children who did not have all four molars caries-free (McCune, Horowitz, Heifetz, et al., 1973; Weintraub, Stearns, Burt, et al., 1993.)

Other studies with a half-mouth design included children with one or two pairs of sound, homologous, first permanent molars. The proportion of children contributing only one pair may be indicative of at least one member of the other pair being unerupted or (more likely) carious, depending on the age of the child. The proportions of pairs of caries-free teeth available may have been a surrogate measure of the child's caries status, indirectly correlated with caries experience and caries risk. These studies likely included a mix of low- and high-risk children. The current effectiveness of sealants is underestimated because the first generation of material used, polymerized by ultraviolet light, was less effective than newer materials and is no longer in use (Ripa, 1993). The retention rate in any sealant trial is also dependent on the accuracy with which examiners can identify the presence of sealant. Misclassification occurs more often when a clear resin rather than an opaque resin is used (Rock, Potts, Marchment, et al., 1989).

Caries risk can be considered at the personal level or at the tooth level. Some studies have compared sealants on carious vs. noncarious teeth (Leverett, Brenner, Handelman, et al., 1983), or on sound surfaces vs. surfaces with incipient lesions (Heller, Reed, Bruner, et al., 1995). In 1991, Handelman reviewed radiographic and bacteriologic studies investigating the therapeutic use of sealants and concluded that "caries is inhibited and may in fact regress under intact sealants." Some (Weerheijm, Groenn, Bast, et al., 1992) have expressed concern about occlusal radiolucencies beneath sealed surfaces. In retrospective sealant studies, dentists may or may not have selected high-risk children for sealant placement, but sealed and unsealed teeth can be compared in children, based on their prior caries experience as a measure of their caries risk status (Weintraub, Stearns, Rozier, et al., In press.) Recent attempts to target high-risk children have compared sealant survival rates (Kumar, Cavila, Green, et al., 1997), caries reduction (Carlsson, Petersson, Twetman, et al., 1997), or reduction of *S. mutans* levels (Mass, Eli, Lev-Dor-Samovici, et al., 1999) in teeth sealed in high-risk children compared to unsealed or sealed teeth in low-risk children.

Table 1. Pit and fissure sealants in high risk children: half-mouth study design

First Author	Year	Type of Sealant	N at Start	Age at start	Caries Risk Determination	Follow-up Years	Full Retention (at final exam)	Effectiveness (at final exam) %
Buonocore	1970 1971	UV-light	60	4-15 (mean 9)	Caries-free individuals with well coalesced occlusal surfaces excluded	2	87%	99%—permanent teeth 87%—primary teeth
McCune Horowitz	1973 1976, 1977	UV-light Nuva-Seal	128 301 429	K, 1st, 6th, 7th Total	Sealant placed on paired and unpaired teeth (usually homologue had already decayed)	5	42% (50%, 26% in paired and unpaired teeth after 4 years)	30%—younger group 38%—older group 98% where sealant completely present 50% unpaired sealed teeth dev caries 26% of paired sealed teeth, 41% paired control teeth
Brooks Mertz- Fairhurst	1976 1984	Nuva-Seal Delton	385	6-8	Caries-free children excluded (about 48% of those screened) 79% of possible first perm molar pairs treated	7	31%— NuvaSeal, 66% Delton	12% NuvaSeal, 55% Delton (10% of completely sealed teeth became carious-combined data from both sealant types)
Houpt	1978, 1983	Delton	205	6-10 (mean 7.5)	Evidence of caries and a pair of caries-free homologous first perm molars (21% screened were eligible)	6	58%	56%
Charbeneau	1977, 1979	Kerr, Chem- cured	143	5-8	81% of possible first perm molar pairs included	4	52.4%	53.4%
McCune	1979	Delton	200	6-8	At least one carious tooth	3	87%	85%
Thylstrup	1976, 1978	Concise Chem.- polymer.	217	7	40% one first perm molar pair, 60% two pairs	2	60%	98%—full 50%—partial 10%—lost
Richardson Gibson	1980, 1982	Chem- cure, pink colored	266	2nd grade	80% of eligible molars, teeth sealed if sound or “sticky”	5	67.4%	51.2%
Vrbirc	1983, 1986	Contact Seal	244	6.8	76% of possible first perm molar pairs	5	52%	55%

Table 2. Pit and fissure sealants in high risk children: other study designs

First Author	Date	Study Design	Control/ Comparison	Type of Sealant	N at Start	Age at Start	Follow-Up Years	Caries Risk Determination	Outcome	Conclusions
Leverett	1983	Half-mouth, benefit/cost analysis	Sealants on one side, restorative care on other	Nuva-Seal	292	6-9	4	Caries-active (sealants placed on a carious surface) Caries-inactive (sealant placed on sound surface)	1 year retention—52%, resealed; After 4 years, sealed surfaces 74% less caries increment than unsealed	Benefit cost ratios based on time or costs were more favorable for caries-active. Sealants should not be used unless evidence of past or current caries experience
Weintraub	1993	Retrospective cohort, patient records, Life table analysis, cost-effectiveness	Children with none, any or 4 molars sealants; children with and without prior restorations	Varied	275	7.4	5.8—mean (up to 11 years)	Restorations on first molars prior to sealant placement on remaining molars	8-year survival: sealed teeth with and without prior restorations—85%, 94%; unsealed teeth—23% and 46%	Cost savings from sealants were obtained within 4-6 years for children with prior restorations; after 8 years without prior restorations
Heller	1995	Retrospective cohort study, patient health center records	96 children with and 17 without sealants, sealed and unsealed teeth	Delton	113	1st grade	5	Tooth surfaces rated sound, "incipient", or frank caries	Decay rates for initially sound sealed and non-sealed surfaces were 0.81 and 0.125 (OR=1.63); for initially incipient surfaces, .108 and .518 (OR=8.88)	Initially sound teeth were unlikely to become carious in 5 years; sealants more effective in preventing further caries on surfaces initially with incipient lesions
Kumar	1997	Survival analysis	Sealed high-risk first molars (65% sites) compared to unsealed low-risk first molars (35% sites)	Helioseal, Delton	1,122	7-9	4	Eligibility required prior caries experience. Teeth with shallow anatomy, occlusal or proximal D or F excluded	Retention (with some resealing)—65-82%; Time to restoration or caries similar for both groups. Cumulative survival rate for 4 years: .89-.94	Targeting approach was effective

Table 2. Pit and fissure sealants in high risk children: other study designs (continued)

First Author	Date	Study Design	Control/ Comparison	Type of Sealant	N at Start	Age at Start	Follow-Up Years	Caries Risk Determination	Outcome	Conclusions
Carlsson	1997	Prospective study, tx based on caries risk assessment, radiographs used	High-risk children (121) received sealant, low risk did not (83)	Helioseal-F (fluoride)	204	6-7	2	Risk based on salivary mutans streptococci, lactobacilli, buffer capacity, past caries experience, cariogenic diet	76.6% complete sealant retention, First molar DFS and dfs incidence lower for sealed group, but NS, enamel caries incidence sig diff in both dentitions	Two-year caries incidence was 11-70% lower in high risk sealed group (range based on dentition and outcome measure)
Maas	1998	Prospective study of two groups receiving sealants; sealant delayed 3 months on one side	Group 1 – mean deft =2.40 (low risk), Group 2 – mean deft = 6.60 (high risk)	Helioseal	52	6-8	0.5	Initially, deft "microbial replica" measured occlusal *S. mutans*	For both groups, *S. mutans* was significantly reduced immediately after sealing and lasted up to six months	Sealants reduced bacterial levels for both low-and high-risk groups
Weintraub	In press	Retrospective cohort, Medicaid claims, discrete time hazard model	Sealed and unsealed teeth	Dentists' choice	15,438	4-7	8	Low risk --no prior Caries-Related Service involving Occlusal surface (CRSO) Middle risk– 1 prior CRSO, High risk $\geq$ 2 prior CRSO	Unsealed molars 3x more likely to get CRSO than sealed molars. Low risk –sealants effective up to 4 years, middle risk – lower odds for 6 years; high risk – reductions up to 7 years	Medicaid expenditure savings for high-risk children within 2 years; not for low risk.

Conclusions

1. Sealants are very effective if completely retained on the tooth surface.

2. Most sealant studies have included low-risk children (all four first molars caries-free), high-risk children (prior caries experience), or a mixture of both low- and high-risk children. However, analyses may not have been stratified by caries risk status. Sealants have been effective to varying degrees in all of these studies.

3. There is evidence that sealants are more effective in preventing further caries and providing cost savings in a shorter time span if placed in individuals (or teeth) with high caries risk compared to individuals with low caries risk.

4. Most caries risk assessment methods used in these studies relied on past caries experience or presence of incipient lesions. Caries risk assessment methods are needed to predict high risk prior to clinical caries development so that sealants can be used to prevent caries on all susceptible teeth.

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Antimicrobial Approaches for the Prevention or Treatment of Dental Caries

Page W. Caufield, D.D.S., Ph.D.

Because dental caries is an infectious disease of bacterial origin, antimicrobial agents constitute a reasonable approach toward attenuating not only the bacterial biofilm in situ but also its transmission from host to host. This approach, while based upon certain constraints inherit to the oral cavity, has its roots in early attempts at plaque control and extends from mechanical to chemical approaches.

Although the extension of this approach to present-day chemotherapeutic tactics seems well-reasoned and grounded in the best traditions of the "medical model," several assumptions that underpin the chemotherapeutic approach need re-examination. For example, a global reduction of the plaque biofilm mass may not lead to the desired effect of selectively eliminating or reducing the caries-associated microorganism. The exception to this may be fluoride, since differential suppression of mutans streptococci has been shown in artificial plaque models. Thus, the aim of the antimicrobial approach for the control of caries should not be toward elimination of all plaque organisms but toward effecting an ecological shift from a cariogenic to a noncariogenic biofilm. To date, the antibacterial effects of chemotherapeutic agents have been assessed mainly by monitoring the levels of mutans streptococci. It is likely, however, that other microbes in the plaque biofilm must be affected in order to cause an ecological shift. Monitoring the change in the ratio of mutans streptococci to *S. sanguinis* is one example of using an ecological shift as a surrogate predictor of efficacy.

The last 30 years have been seen a focus on defining and then targeting specific members of the oral microbial flora in the tradition of Koch's tenet of "one bug, one disease, one bullet." On closer inspection, however, we find that most (if not all) chemotherapeutic applications to the oral cavity are nonspecific in terms of their spectrum of antimicrobial activity and methods of application. Broad spectrum antimicrobials, such as chlorhexidine, iodine, and various formulations of fluoride continue to enjoy widespread acceptance as antimicrobials. Careful examination of the published literature, however, shows that these agents, when topically applied, produce only short-term effects on cariogenic bacteria, with marginal or small reductions in caries outcome. Presumably, the plaque biofilm recolonizes tooth surfaces following disinfection. Reservoirs for cariogenic as well as noncariogenic organisms may exist within areas unaffected by disinfection, including the tongue and the subsurface lesions, fissures, and margins of existing restorations. In fact, one study showed that after treatment, cariogenic mutans streptococci appeared in numbers higher than before treatment. It was hypothesized that the antibiotic affected the exposed microbes, while those buried deep in the caries lesion were not affected. Disinfecting or obtunding (e.g., sealants) these potential reservoirs of recolonization should be considered in future antimicrobial approaches to caries prevention.

In addition, antimicrobial suppression of all the microbes in dental plaque may be unrealistic or undesirable for ecological reasons. Because plaque microorganisms are members of the indigenous biota of humans, they constitute a well-organized "multicellular organism" that

has enjoyed a long-term coevolution with its human host. It seems likely that most of the nearly 1,000 different microbes in dental plaque are benign symbionts that confer some selective benefits to their host. One example of this may be the elaboration of peptide antibiotics, such as the mutacins, that may play a role in preventing overt, nonindigenous pathogens from colonizing the oral cavity.

Because chemotherapeutic agents that are safe for oral use are applied to the entire plaque community, all microbes are presumable affected. One possible exception to this may be fluoride compounds that selectively affect homofermentative acid producers via enolase inhibition. Disruption of enolase displays varying effects on different bacterial groups, depending on their primary modes of catabolism and inherent resistance to fluoride action or uptake. Experiments using in vitro artificial plaque models suggest that the proportions of acidogenic bacteria, such as the mutans streptococci, in the oral cavity can be altered by the presence of relatively small amounts of fluoride. Effecting an ecological shift by selectively depressing acid-producing bacteria constitutes a rational approach to caries control, and the translation of these findings to the human has been underexploited. Practical questions, such as the scheduling of applications and the dosage needed for successful clinical trials, have not yet been answered.

Another use of chemotherapeutic agents could be to suppress the transmission of cariogenic organisms from mother to child. Studies by Swedish investigators show that treating mothers with chlorhexidine gels affects both the infectivity of mutans streptococci in their children as well as the latter's caries experience. Efforts to affirm this approach, however, have led to various outcomes, mostly to no effect. Timing of the treatment to the mother at the time of acquisition of cariogenic bacteria may be an ecologically sound approach to suppressing transfer, and knowledge as to when the indigenous biota are transferred will contribute to eventual success. Although colonization of mutans streptococci follows the emergence of primary teeth during what has been termed the "window of infectivity," the initial transfer of indigenous biota may occur at birth, with the tooth-dependent colonizers existing in yet-to-be-discovered reservoirs, such as the tonsils, tongue, or gastro-intestinal tract. Thus, chemotherapeutic applications to the mother around the time of birth may alter the transmission of indigenous biota, including cariogenic bacteria.

In summary, rational use of chemotherapeutic agents to control or prevent dental caries will necessitate a more holistic understanding of the plaque microcommunity. Shotgun suppression of the entire flora without acknowledging the overall effect on ecology is unlikely to succeed. Chemotherapeutic approaches must be better targeted against specific microbes, with the goal of reestablishing an ecologically stable noncariogenic plaque. In addition, chemotherapy will need to be coupled with mechanical measures to reduce or eliminate reservoirs for recolonization.

Salivary Enhancers

Jane C. Atkinson, D.D.S., and Bruce J. Baum

Saliva provides the principal protective milieu for the teeth, and patients with significantly decreased salivary output have an increased prevalence of dental caries.

Therefore, therapies that increase the overall fluid output of these individuals are believed to have the potential of reversing early carious lesions. Although many systemic diseases are associated with alterations in salivary output, the most pronounced salivary dysfunction occurs in patients with Sjögren's syndrome, patients who have received therapeutic radiation to the head and neck, and patients taking medications that interfere with salivary secretory processes.

Salivary hypofunction secondary to medication is by far the most common cause of salivary dysfunction. Medications often inhibit cholinergic signaling pathways in salivary tissues, and thereby decrease the fluid output of the gland. Interference in other peripheral and central signaling pathways can also reduce salivary output and alter salivary composition. While 300 to 400 medications are believed to interfere with salivary secretion, the specific inhibitory mechanisms are defined for only small subsets of drugs. The impact of prolonged anticholinergic medication on salivary tissues still requires definition. The most practical and common method for treatment is to work with the patient's primary care physician to either alter the medication to a less xerogenic type or reduce the dose while maintaining the required therapeutic effect.

Salivary hypofunction after gland irradiation is very difficult to treat because salivary parenchyma within the radiation field are permanently damaged. Similarly, clusters of infiltrating lymphocytes replace the salivary parenchyma of patients with advanced Sjögren's syndrome. Both conditions are reasonably common in the United States. Head and neck cancer affects 30,000 to 40,000 new patients each year, most of whom are treated with therapeutic irradiation. These patients are typically middle-aged males, and often are individuals from economically disadvantaged backgrounds. Sjögren's syndrome affects about 1 million persons in the United States, currently estimated to reflect a 9:1, female:male ratio. In most studies, the mean age at diagnosis is between 40 and 50.

Both irradiation and Sjögren's syndrome lead to the loss of salivary acinar cells, the only cell type in the glands that is capable of fluid movement. Both conditions exhibit considerable heterogeneity. Some patients experience minimal parenchymal cell loss, while others may have no epithelial tissue surviving, with glands entirely replaced by nonsecretory tissue (e.g., connective tissue, inflammatory cells). Patients with remaining functional acinar tissue can be treated pharmacologically, using a parasypathomimetic secretogogue.

The first such drug approved in the United States was pilocarpine, marketed as Salagen. Pilocarpine possesses both modest, relatively nonspecific muscarinic agonist activity as well as weak β-adrenergic agonist activity. Its effectiveness in increasing salivary output has been demonstrated in several clinical studies of patients with radiation-induced salivary hypofunction or Sjögren's syndrome. Recently, a second secretogogue for such patients, Cevimeline, was approved for use by the U.S. Food and Drug Administration. Cevimeline is a more specific drug,

with a preference for activation of the primary muscarinic receptor subtype responsible for fluid flow from salivary glands, the so-called M3 receptor. However, this medication has not been tested in clinical trials as extensively as pilocarpine.

Radiation damage to salivary glands can be limited by preradiation planning (conformal and static multisegmental intensity modulated technique) that spares as much salivary tissue as possible. Use of the oxygen radical scavenger amifostine during radiation treatment may also decrease damage to glands. Other investigators are surgically repositioning submandibular salivary glands to the submental space before radiation to maintain gland function. While several anti-inflammatory medications have been tested for the treatment of Sjögren's syndrome, only alpha interferon treatment has been shown to increase salivary output.

For patients with more extensive gland damage there is currently no conventional therapy to enhance salivary secretion. This circumstance provided the impetus ~10 years ago for the application of gene transfer technology to repair irradiation- or autoimmune-damaged salivary glands. The initial goal of these studies was to re-engineer the function of the surviving nonfluid secreting ductal cells in damaged glands to a secretory phenotype.

The first peer-reviewed publication on gene transfer to salivary glands was published in 1994. Since then, several laboratories have reported that gene transfer to salivary glands can readily be accomplished. Most of these studies have utilized viral vectors to mediate gene transfer. Viral vectors can be extremely efficient in transferring genes, but can pose a safety risk. An alternative means of gene transfer is to use nonviral methods. Perhaps the most successful form of nonviral gene transfer involves the use of cationic liposomes. This method is much less efficient than preferred viral vectors, but poses relatively little safety risk.

In 1997, a study reported by Delporte and colleagues described the "correction" of irradiation-induced salivary hypofunction in rats through transfer of the cDNA encoding aquaporin 1, a mammalian water channel (permeability pathway). Gene transfer was accomplished using a replication-deficient, first generation, recombinant adenovirus. Irradiated rats administered a control adenovirus exhibited salivary flow rates ~65 percent lower than sham-irradiated animals. Conversely, when animals were administered the aquaporin 1-encoding adenovirus 4 months after irradiation, salivary flow rates were indistinguishable from control levels at 3 days postadministration. This approach is currently being tested in large animal studies.

Thus, the specific value of aquaporin 1 gene transfer for irradiated salivary glands must be considered speculative and not ready for clinical testing. It is not known whether insertion of a water channel into the surviving ductal cells will lead to correction of glandular hypofunction. However, gene transfer without question can be readily accomplished in vivo in salivary glands and is potentially of considerable clinical value to enhance salivary secretions. If aquaporin 1 cannot be used as a transgene for repair of damaged glands, physiological studies will doubtless lead to a better choice.

Gene transfer can also be utilized to augment salivary secretions, such as the transfer of a the gene for a secretory protein that will be secreted in an exocrine manner. The proof of concept for this possibility has been shown in animal studies through transfer of the human histatin

3 cDNA in rat submandibular glands. Histatin 3, which normally is not secreted in rodent saliva, was secreted at high levels (up to 1 mg/ml) after gene transfer. DNA vaccination is another potential clinical use for salivary glands as a gene transfer target site to enhance saliva. For example, Kawabata and colleagues (1999) showed that delivery of the cDNA for the *P. gingivalis* fimbrial protein into murine salivary glands led to the production of secretory immunoglobulin A directed at this microbial protein.

Gene transfer to repair damaged glands can only be an option if epithelial tissue survives either irradiation or autoimmune damage. If the gland is fully replaced by fibrotic tissue, gene transfer cannot lead to an enhancement of saliva production, since no system exists to produce and transport fluid into the mouth. To address this circumstance, we recently began to develop an artificial salivary gland using well-established principles of tissue engineering in combination with genetic engineering. The prototype design includes a biodegradable substratum shaped as a blind end tube (i.e., like a test tube) coated with a layer of purified extracellular matrix proteins involved in cellular organization, followed by a monolayer lining of polarized epithelial cells capable of unidirectional fluid secretion. Initial feasibility studies have been reported. Given the success of other groups in developing functional, fluid-secreting bioartificial organs, notably the bladder, it is reasonable to expect that an artificial salivary gland suitable for clinical testing will be developed within the next decade.

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Application of Methods To Be Employed by Dental Personnel and Other Methods of Stopping/Reversing Dental Disease: Behavior Modification

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Models of self-regulated patient adherence to specific health promotion recommendations by professionals are available and have been shown to be effective in changing behavior (Ramsay, 2000). Kay and Locker (1998) recently reviewed the behavioral research literature and found seven randomized trials, mostly involving school children, and a number of quasi-experimental studies on toothbrushing with a fluoridated dentifrice. They concluded that the interventions reduced the incidence of dental caries but ascribed the effect to the fluoride dentifrice and not the toothbrushing.

There have been a few relatively unsophisticated studies that examined similar behavioral techniques in the promotion of oral hygiene. These studies came about during a period when the main focus of dental researchers was periodontal disease, and they found the effects of promotion to be modest and short-term. Moreover, it is unclear whether reduction of plaque would result in caries control because toothbrushing may fail to control plaque on the surfaces at greatest risk.

The major problem found in the toothbrushing and oral hygiene studies, however, is that the desired behavior decreases in frequency when external reinforcement is withdrawn. This is often seen as evidence that the technique is not efficacious, rather than as simply a confirmation of the underlying theory that reinforcement is needed. The reality is that modest, short-term behavioral programs have modest, short-term results.

Ramsay (2000) has argued that technological improvements, such as timers on electric toothbrushes and toothpaste tubes that beep if not opened every day, are based on sound theory and will increase adherence whether the goal is oral hygiene or delivering fluoride. He has argued, similarly, that if a toothpaste tube sends an automatic e-mail to the dentist when it is not opened, the health care provider can be more effective as an external change agent. This could also apply as feedback to a parent to increase the reinforcement of behavior with a child who brushes his/her own teeth. Based on what is known from the generic behavioral literature, interventions of greater effectiveness for tooth care can be designed and investigated.

But it is a fundamental mis-specification of the caries prevention problem to look to techniques that affect the regulation of individual behavior to directly impact dental caries. Behavioral techniques are used to enhance the probability that an individual will initiate, increase, or maintain established caries reduction/control strategies or cease or decrease behavior that increases caries (Weinstein, Getz, Milgrom, 1991). Behavioral techniques can also be used to affect the regulation of parental behavior in a cascade of effects that can eventually lead to healthier children (Milgrom, Weinstein, 1999).

Studies are needed where behaviorally oriented caries prevention actions are thought of as manipulating self-regulatory behavior and the focus of action is either on the individual or on someone else, such as a parent. A third category of studies should center on provider competency. The table at the end of this abstract provides a number of examples.

1. Examples of Self-Regulatory Behavior Where the Burden of Action Is on the Individual

The best understood example of regulation of individual behavior is toothbrushing with a fluoridated dentifrice. This behavior is well accepted by the public, largely because of industry advertising, and there is also little controversy about whether frequent professionally administered toothcleaning with a fluoride vehicle is effective in controlling caries (Hotz, 1998). On the other hand, there is mixed evidence about the effectiveness of the same activity when done by individuals who are not under supervision. Nevertheless, the evidence suggests that the problem with at-home data is toothbrushing skill rather than erratic performance (caries control tends to be more effective in easy-to-brush front teeth). Studies are needed to specify the brushing time/effectiveness relationship relative to caries, even though we know that there is a relationship between brushing time and plaque removal in both children and adults.

Studies to initiate, increase, or maintain toothbrushing with a fluoridated dentifrice will fail to demonstrate effectiveness in caries control if the underlying efficacy of the toothbrushing/fluoride intervention is not clear or if the problem is described as a performance problem (frequency per day or time per brushing episode) rather than a skill problem (quality of brushing) (Weinstein, Getz, Milgrom, 1991).

A second example involves chewing gum. The RTI team failed to report on the extensive literature on xylitol, although it touched on sugarless chewing gum. Much valid controversy exists about the interpretation of xylitol trials and the proposed mechanism of action, and behaviorists will be reluctant to conduct studies to test the effectiveness of xylitol chewing gum if controversy exists about its efficaciousness. Moreover, scientists will be reluctant to develop alternative xylitol vehicles, such as foods that might be used in Department of Agriculture-sponsored meal programs, in the presence of controversy.

2. Self-Regulatory Behavior Where the Burden of Action Is on Another

An example of the problem when the burden of regulation is on someone else is urging parents to brush a preschool child's teeth with or without a fluoridated dentifrice. Studies are needed that focus on the efficacy and effectiveness of this behavior, even though it is now widely accepted and recommended. Studies do not exist that clearly demonstrate a frequency-response relationship or even the optimal time of day for the behavior (assuming that it matters). Public health officials are, in fact, sending the message that overuse of fluoridated dentifrice results in unacceptable levels of fluorosis. A behaviorist can construct a strategy to help a parent regulate his/her behavior, and these strategies can be tested, but the results of such tests are confounded if the underlying efficacy of the caries control strategy is in question.

A second example involves the relation of feeding habits to caries. Professionals are convinced that taking away children's night and naptime bottles and weaning at one year are effective strategies for controlling early childhood caries. Yet the evidence for these convictions is primarily cross-sectional and retrospective. Moreover, efforts to change this behavior are likely to have ramifications for the remainder of children's diets. Prospective studies are needed.

A third example relates to the mother's experience with dental care. We have shown that low-income mothers are less likely to take their child to the dentist if they are afraid of the dentist (Milgrom, Mancl, King, et al., 1998). This behavior is critical, because dentists are the main source of knowledge on oral health that is available to mothers. Moreover, caries is transmissible, and the mother (who may herself be in poor oral health) is both a source of oral bacteria and the regulator of the child's oral habits. Studies are needed to show that mothers with a customary source of dental care are more adherent to professional recommendations and have healthier children (Skaret, Milgrom, Raadal, et al., 2000). Studies are also needed on how to overcome barriers in the Medicaid program, where pregnant women and mothers receive poorer benefits than their children.

3. Examples of Health Promotion Aimed at Professional Competency

A third area of promising research for the prevention and treatment of dental caries relates to the competency of health care workers. Weinstein and colleagues, for example, are conducting a study using motivational interviewing techniques to impact the behavior of pregnant women and new mothers relative to oral health (Weinstein, 2000). This study is using peer counselors and offers mothers alternative strategies to prevent/control early childhood caries. The choices of prevention strategies available to the behavioral scientist, however, are relatively few, and in the context of this conference not well-founded scientifically, but serve as a positive example.

Similarly, Grembowski and colleagues are conducting a study in which a dental prepayment plan offers financial incentives to dentists to use strategies such as fluoride varnish to prevent secondary caries and prolong the life of restorations (Grembowski, 2000). Again, behavioral intervention by dentists may be effective yet not improve health because the efficacy of the action is uncertain.

Lewis and colleagues are studying the role of pediatricians in oral health guidance and fluoride treatments for children (Lewis, Grossman, Domoto, et al., 2000). In a survey of 1,400 pediatricians nationwide, the researchers found that the willingness of pediatricians to apply fluoride varnish to teeth was most strongly related to (i) familiarity with the varnish, (ii) agreement that pediatricians should provide guidance on oral health, and (iii) seeing caries in everyday practice. Studies are needed on the dynamics of physician practice and how best to incorporate and maintain guidance activities.

Behavioral Research Problems Related to Dental Caries

1. Examples of self-regulatory behavior where the burden of action is on the individual
    - Initiate or increase or maintain toothbrushing with a fluoridated dentifrice twice daily
    - Increase or maintain the amount of time an individual brushes with a fluoridated dentifrice
    - Increase or maintain the quality of individual brushing
    - Initiate or increase or maintain use of a chlorhexidine or fluoride rinse twice daily
    - Initiate or increase or maintain use of xylitol or nonsucrose chewing gum 3-5 times daily
    - Decrease sugar intake in the diet or increase the amount of nonrefined carbohydrates
    - Initiate or increase or maintain visits to the dental office for preventive treatments two or more times per year
2. Examples of self-regulatory behavior where the burden of action is on someone else
    - Initiate or increase or maintain a parent's frequency of brushing a child's teeth with a fluoridated dentifrice or initiate brushing twice daily
    - Increase or maintain the quality of a parent's brushing of a child's teeth
    - Reduce the frequency of refined carbohydrate snacks for a child
    - Reduce the frequency of short bottle or breast-feeding episodes, especially before naps or at night
    - Wean a child at one year, either cold turkey or gradually.
    - The relationship between a mother having a usual source of dental care and taking the child to the dentist
3. Examples of health promotion aimed at professional competency
    - Improve the teaching and reinforcement of the skill components of oral hygiene
    - Increase the amount of time devoted to teaching and reinforcement of oral hygiene
    - Learn to offer alternative strategies to individual patients and parents to control disease and estimate their potential effectiveness
    - Reduction of fear/pain-causing behavior of dental personnel that results in reduced compliance with preventive visits
    - Increase anticipatory guidance by public health nurses, family doctors, and pediatricians

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Non-Cariogenic Sweeteners

Catherine Hayes, D.M.D., D.M.Sc.

Dental caries continues to be a significant public health problem, affecting a majority of the world's population. The role of sucrose and other fermentable carbohydrates in the etiology of dental caries has been well established, and the use of sugar substitutes in candy, food, and gum and their effects on dental caries have been investigated in several studies.

It is believed that the benefits of sugar-free gum may be twofold. First, since sugars are not available for fermentation, lactic acid is not produced. Therefore, the pH of the oral cavity is not lowered to a range that would increase the risk for dental caries. Second, the use of chewing gum is believed to stimulate salivary flow, thus providing caries-preventive benefits, such as the buffering of acids in plaque formed from dietary carbohydrates, increased supersaturation of dental tissue with mineral ions leading to enhanced remineralization, and enhanced clearance of sugars from the mouth. Thus, sugar substitution and salivary stimulation could be equally responsible for the noncariogenicity of sugar-free chewing gum (Edgar, 1998).

The majority of sugar-free gums have been sweetened with sorbitol, a sugar alcohol derived from glucose. Xylitol, a sugar alcohol derived from the pentose sugar xylol, is another sweetener and has been the subject of many studies. Xylitol is nonacidogenic and is phosphorylated to an inhibitory compound upon entering cells. Other substitutes include mannitol, saccharin, and aspartame, which enhance shelf life and product taste (Edgar, 1998).

Studies of the relationship of sugar substitutes to dental caries have included both clinical trials and community-based observational studies. Although clinical trials are considered the "gold standard" of clinical research, it is important to consider information from observational studies as well. Information from multiple studies of both types points to the protective effect of xylitol against dental caries.

Clinical Trials

One clinical trial investigated the effect of sugar-free gum on the incidence of dental caries in 2,601 male and female schoolchildren in grades 5-7 in three communities in Puerto Rico. This population had a high prevalence of caries, low levels of professional dental care, and drinking water with negligible amounts of fluoride. Participants were assigned to either a no-gum group or a sugar-free gum group. Subjects in the gum group had a significantly smaller increase in caries rates than those in the no-gum group (Beiswanger BB, Boneta EA, Mau MS, et al., 1998).

Another study involved patients in the VA system who were enrolled in a randomized clinical trial. Patients with exposed root surfaces were randomly assigned to either sorbitol or xylitol chewing gum and were then followed for 1.8 years. Neither subjects nor examiners knew which patients got which type of gum. There were 40 subjects in each of the intervention groups. The relative risk for caries incidence in the xylitol versus sorbitol group was 0.19 (Makinen KK,

Pemberton D, Makinen PL, et al., 1996a). A longitudinal study in Finland also demonstrated a decreased rate of caries among schoolchildren in an xylitol group (Isokongas, 1987).

Observational Studies

In a double-blind cohort study conducted in Belize, 1,277 schoolchildren were randomly assigned (by school) into nine treatment groups: one control group (no gum), four xylitol groups (4.3-9.0 g/day), two xylitol-sorbitol groups (8.0-9.7 g/day), one sucrose group (9 g/day), and one sorbitol group (9 g/day). The largest reduction in caries occurred in the four xylitol groups and was significant in comparison to reductions in the sorbitol and sucrose groups (Makinen KK, Bennett CA, Hujoel PP, et al., 1995a).

A 5-year followup study of Estonian schoolchildren to evaluate the effect of xylitol gum or candy on caries rates was recently reported. In this study, the effects of xylitol consumption by 740 10-year-old children in 12 schools over a 2-year period were evaluated. Children using either xylitol gum or candy experienced a significant reduction in caries incidence (53.5 percent and 59 percent) compared to those in a control group (Alanen P, Isokangas P, Gutmann K, et al., 2000).

Another study in Belize with 6-year-old subjects found a lower rate of caries in xylitol or sorbitol groups as compared to a group of children not assigned to a chewing group, with relative risks reported as 0.35 (.21-.59) and .44 (.30-.63), respectively (Makinen KK, Hujoel PP, Bennett CC, et al., 1996b). Another analysis by Makinen and colleagues (1995b) of arrested or nonprogressed lesions also found a significant improvement in the xylitol group.

Studies of Streptococcus Mutans

Changes in streptococcus mutans levels as a result of sugar-free chewing gum have also been investigated. One study reported significant decreases in streptococcus levels in subjects using xylitol gum for 3 months as compared to subjects in a placebo or no-gum group. All subjects in that study rinsed daily with chlorhexidine for 2 weeks and were later randomized into three treatment groups and evaluated after 3 months. Streptococcus levels were no different in the three groups at baseline or after the chlorhexidine rinse period. The increase in streptococcus levels 3 months after rinsing was fortyfold in the placebo group, twenty-five fold in the control group, and eightfold in the test group (Hildebrand, Sparks, 2000).

A study in Finland examined the influence of maternal xylitol use on streptococcus levels in infants. Mothers participating in a postnatal oral health program were randomly assigned to xylitol chewing gum, chlorhexidine varnish, or fluoride varnish, and evaluated at 6, 12, and 18 months after delivery. Plaque samples were taken from the children, and saliva samples were taken from the mothers. The level of streptococcus did not differ significantly among the three groups at baseline, but the children of the mothers in the xylitol group had significantly lower levels of streptococcus than either of the other two groups after 18 months (Soderling E, Isokangas P, Pienihäkkinen K, et al., 2000). A third study also demonstrated a decrease in strep mutans levels in children in a chewing gum group (Makinen KK, Soderling E, Isokangas P, et al., 1989).

Long-Term Effects

The long-term effects of sugar-free gum have been reported in a single study in which children were reexamined 5 years after a 2-year study ended. Comparisons were made between sorbitol, xylitol, xylital-sorbital, and no gum. The sorbitol group did not show a significant long-term reduction in caries, but the xylitol and xylitol/sorbitol groups demonstrated significant long-term caries reductions, with relative risks of 0.41 (0.23, 0.75) and 0.56 (0.36,0.89) respectively. The protective effect of xylitol depended on when teeth erupted. Children whose teeth erupted after 1 year of gum chewing or after the 2-year period had ended demonstrated the most significant long-term caries reductions (93 percent and 88 percent, respectively).

Summary

The use of xylitol as a sugar substitute in chewing gum has been evaluated in several observational studies as well as clinical trials, with results consistently demonstrating that xylitol had a protective effect against caries incidence. Limitations of the studies included small sample sizes, lack of radiographs for caries diagnosis, high loss of subjects to follow-up, potential confounding, and bias due to the nature of long-term community intervention studies. In order to effectively evaluate the effect of xylitol chewing gum on caries incidence, well-controlled double-blind clinical trials are needed with careful attention to study power, compliance, reliable caries assessments, and retention of participants.

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Choosing Appropriate Preventive Approaches

**Denis O'Mullane, B.D.S., Ph.D., F.D.S., F.F.D.,
and John Clarkson, B.D.S., Ph.D.**

The extent to which practitioners make use of new methods for identifying patients at risk of dental caries and for diagnosing early carious lesions is not known. However, a worldwide increase in sales of new instruments for carrying out these tasks would seem to indicate rising interest in new techniques.

It is likely that dental practitioners choose combinations of appropriate preventive approaches for arresting or reversing early carious lesions. In the systematic review conducted by Research Triangle Institute (Bader, Shugars, Rozier, et al., 2000), it is pointed out that surprisingly few studies have been conducted on the results obtained with combined methods. For example, only four studies were found that had examined the effectiveness of combining chlorhexidine and fluoride (Spets-Happonen, Luoma, Forss, et al., 1991; Luoma, Ronnberg, 1987; Tenovuo, Hakkinen, Paunio, et al., 1992; Petersson, Magnusson, Andersson, et al., 1998) and only one study was found on the combined effect of chlorhexidine and sealants (Zikert, Emilson, Krasse, 1982).

Yet there is considerable theoretical data available to support the idea of using a combination of methods to stop or reverse early carious lesions. For example, it is now well-established that fluoride's primary method of action is a topical one. Fluoride ions, when present at the plaque/enamel interface, reduce demineralization and promote remineralization in the presence of a cariogenic challenge (Margolis, 1993). To ensure that fluoride bestows maximum preventive benefit, it is important to maintain the ambient level of fluoride in saliva and plaque. Clearly, combining fluoride mouth rinses, fluoride toothpastes, fluoride tablets, and fluoride gels and varnishes in patients in either fluoridated or nonfluoridated communities will help maintain fluoride levels (Mainwaring, Naylor, 1978; Blinkhorn, Holloway, Davies, 1983; Murray, Rugg-Gunn, Jenkins, 1991).

Another example of a theoretical basis for a combined preventive approach involves the distribution of coronal caries by tooth surface in many communities, particularly those in which fluoride is widely used. Since the preventive effects of fluoride are concentrated on smooth surfaces, it is not surprising that data from many of these communities show that caries lesions in children and young adults tend to be confined to posterior teeth and occlusal surfaces. Hence, additional benefit is likely to be obtained by the concurrent use of fluorides and fissure sealants (Horowitz 1980). With respect to root caries, epidemiologists have traditionally attempted to distinguish between lesions which are soft and theoretically active and lesions which are hard and theoretically inactive. Thus, measures that promote the transition from soft to hardened status are considered to be beneficial (Baysan, Lynch, Ellwood, et al., 2001).

The preceding discussion forms the basis for our conference presentation. For example, studies by Ripa and colleagues (1987), Goggin and colleagues (1991), Sterritt and colleagues (1994), and Selwitz and colleagues (1995) have measured the benefits of a combined fluoride and fissure sealant approach. Ripa and colleagues found that a combination of pit and fissure

sealants and weekly fluoride mouthrinsing almost completely eliminated the incidence of new carious lesions over a 2-year period. However, these studies also illustrate the difficulties in choosing an appropriate experimental design for studies of combined therapies in which the contribution of each therapy needs to be established. Those difficulties will be highlighted in our presentation, and proposals for future studies will be presented. New technologies aimed at maintaining an effective level of fluoride ions in the oral cavity, such as low-release devices, will also be considered (Toumba, Curzon, 1993).

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Emerging Methods in Prevention of Dental Caries

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The purpose of our review was to appraise and synthesize the relevant literature on several questions pertaining to the prevention of dental caries:

1. Does the partitioning of calcium from phosphate and fluoride in toothpaste increase the remineralization of demineralized enamel or dentin, or increase the resistance to demineralization of these tissues to a greater extent than a nonpartitioned toothpaste containing the same ingredients in similar concentrations?

   This question was broken down into four subquestions by treating enamel, dentin, increasing remineralization, and increasing resistance to demineralization as separate entities. A further breakdown was conducted under the headings human (clinical), animal, and laboratory studies.

2. Is lased enamel or dentin more, or less, susceptible to demineralization, compared to nonlased enamel and dentin?

   For this question only laboratory studies were found, and enamel and dentin were treated as separate questions.

3. Do fluoride-releasing dental materials increase the remineralization of demineralized (carious) human enamel or dentin, or increase the resistance to demineralization (caries) of these tissues?

   Only human clinical trials and human in situ studies were reviewed in addressing this question. Enamel and dentin were treated as different subjects of inquiry, as were remineralization and demineralization. Only studies reporting direct measures of changes in enamel and dentin remineralization and increased resistance to demineralization were considered in the appraisal. Investigators using such indirect measures as, for example, fluoride uptake or plaque accumulation were excluded.

A further question more closely linked to repairing dentinal caries and not as relevant to caries prevention was also considered:

4. Do human, animal, or in vitro studies show that bone morphogenic proteins, in particular BMP-7 (OP-1), can be used to stimulate pulpal cells to produce new dentin?

   Since no human studies have been reported and in vitro studies did not show tubular dentin formation, only animal studies were reviewed.

Methods

A search was made of articles published in peer-reviewed journals, written in English, and indexed in MEDLINE or EMBASE. References in review articles were also used as a source if they were not cross-referenced in MEDLINE or EMBASE. All articles had to be published after 1976. The databases were searched using appropriate key words for each of the questions asked. Use was also made of the caries hedge setup for reviewers involved in literature review for this Consensus Development Conference. References in the reviewed articles were also searched for other relevant reports.

The two investigators independently read all the abstracts from the MEDLINE, EMBASE, and hand searches. Relevant reports were then tagged. Discrepancies between the investigators were resolved by consensus after a further reading of the disputed abstracts. Articles with tagged abstracts were then photocopied and distributed equally between the two investigators, except for articles on bone morphogenic protein, which were read by only one investigator (Clarkson).

All articles were then abstracted and entered into the evidence tables under various headings, and then scored, except for those on studies that had no controls. Purely descriptive studies on BMP activity were included, but descriptive studies in which statistical analysis was deemed appropriate but was not carried out, and articles in which conclusions were drawn from inappropriate statistics, were not.

The scoring system was an all-or-none system based on the evidence table headings. If information was available in the article under the heading, it was given a score of 1; if it was missing, it was given a score of 0. Publication date, author's name, and study type were not included in this scoring system, nor was the information under the heading "findings." The total score differed for each question. The score assigned, and the possible total score for each article, are given in the last column of the evidence table. All articles were scored independently, and disagreements were resolved by consensus.

Results

For question 1 (partitioned toothpastes), only 12 of the 35 abstracts dealt with a toothpaste in which the calcium was separate from the phosphate and fluoride until ions were delivered to the tooth surface. Of these, seven were in vitro investigations, three were animal studies, and two were clinical trials. After the full articles were read, one in vitro study and one clinical study were excluded because of insufficient data.

All of the studies that were included had one author's name in common, but the research was carried out at several different institutions. One of the animal studies and six of the in vitro studies dealt with remineralization of enamel, but none of the studies reported on remineralization of dentin. The other two animal studies tested the partitioned toothpaste's ability to increase the resistance of enamel to demineralization. There were no studies of dentin resistance to demineralization. The one clinical trial tested the partitioned toothpaste's ability to inhibit both coronal and root caries. All these studies showed positive results except for the clinical trial, in which the partitioned toothpaste reduced root caries but not coronal caries. Thus,

in all but the class I (as designated by AHRQ's U.S. preventive service task force grading of the evidence) clinical trial investigating enamel caries, the partitioned toothpaste showed either greater remineralizing enhancement or greater increase in resistance to demineralization of enamel and dentin compared to a nonpartitioned toothpaste containing calcium, phosphate, and fluoride.

Question 2 focused on the demineralization potential of lased versus nonlased enamel or dentin. Of the 84 abstracts initially read, 14 in vitro studies were evaluated. Seven of these were excluded because of no, or inappropriate, statistics. Of the seven remaining, five out of six concluded that lased enamel was less soluble than nonlased, while the one article on lased dentin reached the same conclusion.

Question 3 asked whether fluoride-releasing restorative materials increase the remineralization or the resistance to demineralization of enamel or dentin. Of the eight clinical trials, two were excluded because there were no control groups. Of the remaining six, one was designated a class 11-1 study, four were class 11-2 studies, and one was a class 11-3 study as designated by the U.S. preventive services task force grading. Of the six in situ studies that were also reviewed, two were excluded, one because it used bovine tissue and one for incomplete data. All but one of the clinical and in situ studies were short-term—that is, less than 16.3 months. The other lasted 3 years. They used a variety of methods for measuring remineralization and resistance to demineralization of both enamel and dentin. The study participants (or specimens) were also subjected to several different caries challenges. Eight of the 10 studies did not report on examiner calibration or reliability. Of the six clinical trials, five dealt with enhancing the resistance of enamel to demineralization and one dealt with dentin remineralization. No clinical trials on enamel remineralization or increasing dentin resistance to demineralization are discussed here, either because no studies had been conducted or those that had been conducted did not meet our criteria. Of the four in situ studies, two dealt with increasing the resistance of enamel to demineralization, one looked at both increasing the resistance to enamel demineralization and enhancing enamel remineralization, and one looked at dentin remineralization. No in situ studies on enhancing the resistance of dentin to demineralization were found.

In the five clinical trials investigating the effects of fluoride-releasing materials in enhancing enamel's resistance to demineralization, four recorded increased resistance and one showed no difference between the experimental and control groups. In the one study on increasing the remineralization of dentinal lesions with these materials, no difference was seen between the experimental and control groups. The one study that looked at remineralization of enamel in conjunction with increasing enamel resistance to demineralization failed to state the remineralization results. Of the in situ studies, the one study investigating the remineralization of dentin by fluoride-releasing materials showed increased remineralization, while the three examining enamel resistance to demineralization all recorded increased resistance.

For Question 4 on BMP's ability to stimulate dentin formation, all six articles reviewed were animal studies. Irrespective of the species, all showed that BMP stimulated new dentin formation. The reparative dentin included both tubular and nontubular (osteo) dentin. One study that tested transdental transition of BMP showed that BMP activity did, in fact, cross dentin. Two of these studies used a crude BMP extract, while four used BMP-7 (OP-1).

Conclusions

Question 1: In spite of the fact that all the studies on using the partitioning of the active ingredients of toothpaste had one author in common and that only a few studies had been conducted, there is sufficient evidence from the animal and in vitro studies to suggest that this technology has promise in enamel caries prevention. In humans, however, the sole class I clinical trial did not show a difference in enamel caries reductions between experimental and control groups in a high-risk population. But in the same study the partitioned toothpaste prevented root caries to a greater extent than a conventional toothpaste. Independent, randomized, controlled clinical trials need to be conducted to determine if this therapy's usefulness can be generalized to all population groups. Studies also need to be conducted on its usefulness for preventing dentin caries.

Question 2: In vitro testing of the solubility of lased enamel has demonstrated that it is less susceptible to demineralization than nonlased enamel. The results for dentin were similar, but only two studies met the criteria for inclusion in this review. Further in vitro investigations to determine if lased dentin is indeed less soluble should be undertaken.

The reviewed studies used several different laser types, application times, laser wavelengths, power, demineralization models, and target distances (i.e., distance from laser head to tissue) and made it impossible to recommend a standard procedure. Investigations should be performed to establish the standard protocol for application in clinical trials that must be completed before this therapy can be recommended for caries prevention.

Question 3: The small number of studies using direct measures of caries prevention and the short duration of those studies made it impossible to draw any conclusions about the long-term benefits of these measures. Randomized, controlled clinical trials need to be conducted over a period of at least 2 years to answer the four subquestions reviewed in this paper—whether fluoride-releasing dental materials increase the remineralization of carious enamel and dentin, and whether these materials increase the resistance of enamel and dentin to caries.

Question 4: All of the animal studies reviewed reported that crude BMP extracts and BMP-7 were able to regenerate dentin (tubular and atubular) when placed on vital pulps. One study also showed that the active signaling molecule can cross dentin and stimulate a pulpal response. One anecdotal report of a clinical trial using BMP-7 suggested that the results of the study were equivocal. Nevertheless, the animal studies suggest that this therapy provides positive results. Further investigations should be undertaken, controlling for the drug carrier and studying the effect of inflammation on the BMP-7 activity. After these animal studies are completed, human clinical trials should be conducted.

Clinical Decision-Making for Dental Caries Management

B. Alexander White, D.D.S., Dr.P.H., M.S.,
and Gerardo Maupomé, Ph.D.

Preceding presentations have reviewed the scientific literature on diagnosis and management of dental caries, indicators of risk, primary prevention of dental caries, and methods of stopping or reversing early carious lesions. For the practicing dentist, however, such data may not address specific clinical questions that arise in everyday practice. The purpose of this paper is to describe a framework—clinical decision-making—and its potential application to diagnosis and management of dental caries. Subsequent papers will use this framework to describe clinical decision-making for coronal caries in the primary dentition and coronal and root caries in the permanent dentition.

Clinical information is imperfect, yet dentists are expected to make decisions about patient care every day. Patients vary in clinically important ways, uncertainty abounds in diagnostic and prognostic information, and the effectiveness of many preventive and treatment alternatives has not been formally assessed. Scientific information is not available—and likely will never be available—to answer all important clinical questions. Clinical decisions therefore will continue to be made based (at least in part) on probabilistic, as contrasted with definitive, information.

Clinical decision-making—explicit use of information to quantify probabilities and outcomes under conditions of uncertainty—can provide a framework to analyze the impact of uncertainty in clinical information. Clinical decision-making is not descriptive, in that it does not seek to identify the ways in which clinicians actually make decisions. Rather, it seeks to identify how clinical decisions should be made to achieve optimal outcomes.

Clinical decision-making in dental caries management involves four basic steps. First, the clinical question must be identified and characterized. In this step, the relevant population for study (e.g., children, adolescents, adults, elderly) and alternative diagnostic, preventive, and management options are identified. For clinical decision-making to be useful, the clinical question must involve choosing between two or more clinical strategies with meaningful tradeoffs. Clinical questions may focus on such topics as caries detection, including diagnostic techniques and clinical examination; characterization of caries risk status; primary, secondary, and tertiary prevention of dental caries; and arresting or reversing a carious lesion.

Second, the decision problem is structured to address the relevant clinical problem. A model or decision tree that represents the logical and temporal sequence of caries management is described. The decision tree should be sufficiently complex to reflect important events and outcomes associated with the clinical problem, yet sufficiently simple to be understandable and useable. A well-defined clinical starting point must be specified, including such dimensions as age and sociodemographic characteristics; caries risk status; prior and current caries experience; behavioral factors; diet; fluoride exposure; and general health status, including use of xerostomic medications and diseases that may affect salivary gland function. The relationship of relevant

diagnostic, preventive, and/or treatment strategies should be identified, and important outcomes—biological, clinical, psychosocial, and economic—described.

Third, the information needed to answer the clinical question is characterized. Much of this information comes from systematic reviews of a literature ideally based on randomized clinical trials. An important feature of the information is its probabilistic nature. Here, the probability of different events (e.g., detection of a carious lesion with a particular diagnostic test, reversing a demineralized lesion), the outcomes associated with those events (including patient preferences regarding the outcome), and the degree of associated uncertainty, are quantified.

Finally, a preferred course of action is chosen, based on the decision tree structure and relevant probability and outcome data. Synthesis of this information does not identify a "correct" course of action, but rather a "preferred" course of action that would yield the best outcome, given the information. Since uncertainty is associated with the probability and outcome estimates, a sensitivity analysis must be done to assess the impact of uncertainty on the conclusions. In some instances the preferred course of action will be robust over a wide range of probability and outcomes estimates. In other cases the preferred course of action will change within a narrow—but clinically important—range of probabilities and outcomes, suggesting that additional information is needed to more fully characterize the clinical problem.

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Clinical Applications and Outcomes of Using Indicators of Risk in Caries Management

Domenick T. Zero, D.D.S., M.S., Margherita Fontana, D.D.S., Ph.D., and Áine M. Lennon, B.Dent.Sc., Ph.D.

Other papers at this conference have discussed individual risk indicators of caries. This review focuses on studies of the predictive validity of various combinations of risk indicators. Such indicators may be useful in the clinical management of dental caries by helping dental professionals determine if additional diagnostic procedures are required, identify patients who require caries control measures, assess the impact of caries control measures, make treatment planning decisions, and determine the timing of recall appointments. Although there is a high level of interest in identifying risk indicators, only a few studies have attempted to determine how the application of risk indicators affects dental health outcomes (Brambilla, Gagliani, Felloni, et al., 1999; Hausen, Karkkinena, Seppa, et al., 2000).

Multifactorial modeling has proved its value in longitudinal caries prediction studies by showing the interrelations and interactions of risk factors. Beck and colleagues (1988) indicated that one or more social, behavioral, microbiologic, environmental, and clinical variables should be included in such a model, given the many factors that influence dental caries. Modeling has usually been based on a dichotomized dependent variable, either as "no" versus "some" caries increment (Beck, Weintraub, Disney, et al., 1992) or with specified cut-off points in populations with high caries incidence (Abernathy, Graves, Bohannon, et al., 1987). The accuracy of models has rarely been 80 percent, which is considered to be the minimum level for screening purposes. "To be useful, a working model should produce a sensitivity of 0.75 or higher and specificity level of at least 0.85 or higher" (Stamm, Disney, Graves, et al., 1988). It has therefore been suggested that a risk model should have a combined sensitivity and specificity of at least 160 percent (Kingman, 1990).

Objective

The aim of this review was to systematically assess the clinical evidence to determine the predictive validities of currently available multivariate caries risk-assessment strategies. The intent was to answer "What are the best (combination of) indicators for an increased risk of dental caries?" That, in turn, should help to answer Question 5, "How should clinical decisions regarding prevention and/or treatment be affected by detection methods and risk assessment?"

Search Strategy

A search of relevant publications dating from 1980 was conducted in the MEDLINE and EMBASE databases. Only English language publications concerning humans were included in

the search. To help identify as many papers as possible the following key word headings were used:

- For primary dentition: [(Caries AND Risk hedge) AND Diagnosis hedge/limited to human, English, 1980+] AND (age group limit OR primary dentition hedge).

- For root caries: [(Caries AND Risk hedge) AND Diagnosis hedge/limited to human, English, 1980+] NOT (age group limit OR primary dentition hedge) AND root caries hedge.

- For permanent dentition: [(Caries AND Risk hedge) AND Diagnosis hedge/limited to human, English, 1980+] NOT [(age group limit OR primary dentition hedge) OR root caries hedge].

Due to the large number of references obtained in our electronic search, it was decided that secondary hand searching would not be feasible.

Selection Criteria

Inclusion and exclusion criteria for the papers selected for review included: (1) the use of more than one type of caries risk predictor category used to calculate the predictive outcome, and (2) the presence of a clear outcome prediction. Every included article was listed, as were excluded articles. The following types of articles were excluded: reviews, in vitro studies, research using population approaches rather than individual approaches, and papers not related to dentistry. Except for review papers, these are not listed in the exclusion table.

Data Collection and Analysis

A list of included and excluded articles for each category (primary teeth, permanent teeth, and root caries) was prepared. At the time of preparation of this abstract, 151 papers had been added to either the inclusion or exclusion tables, and 27 were still being sought. Papers that conformed to the selection criteria and reported a predictive outcome for the model were included (N= 24 for primary teeth; N= 37 for permanent teeth; and N= 13 for root caries). The tabulation of excluded articles (N= 77) included the reason for exclusion (e.g., lack of more than one risk factor, no outcome data, etc). Four evidence tables were prepared: primary teeth, permanent teeth in children and/or adolescents, permanent teeth in adults, and root caries. When an article appeared in one data set (e.g., primary teeth) but contained information on another data set, it was transferred to the appropriate inclusion table. Articles reporting information on more than one type of caries were included in more than one table. Included articles were also grouped by study design as longitudinal-prospective, retrospective, or cross-sectional.

Main Results

Of the 24 articles on primary teeth, 17 were prospective studies, 1 was a retrospective study, and 6 were cross-sectional studies. The articles on permanent teeth were separated into

those involving caries in risk prediction in children/ adolescents (< 20 years old) and those used to predict caries in adults. Of the 30 articles on permanent teeth in children/adolescents, 20 were prospective studies, 2 were retrospective studies, and 8 were cross-sectional studies. Of the total of 7 articles on permanent teeth in adults, 2 were prospective studies and 5 were cross-sectional studies. For root caries, 13 articles were found: 9 prospective studies and 4 cross-sectional studies. All models included some aspect of past caries experience as a predictor. The second most frequent predictor was "other variables." The third most frequent predictor was "microflora," followed by "host factors." In the case of root caries the "host factors" category was more frequently used than the "microbiology" category.

References were systematically assessed for their validity. Since valid evidence is considered best obtained from randomized, controlled longitudinal (prospective) studies, those were given the highest scores in our review. Studies were graded as "good," "fair," or "poor," depending on the amount of information they provided to support the methodology used. The main variables assessed for this purpose (other than the inclusion criteria) were: (1) whether the study reported how samples were obtained, (2) whether the examiners were trained/calibrated, (3) whether examiner reliability was reported, and (4) whether examiners were blinded during the study. Tables 1, 2, and 3 include the longitudinal prospective studies considered to be good sources of evidence for predictions in primary teeth, permanent teeth in children and adolescents, and permanent teeth in adults. None of the root caries studies reviewed met these criteria.

Of all the models reviewed, none of those graded as "good" had a combined sensitivity and specificity in excess of 160 percent, although the model reported by Demers and colleagues (1992) comes very close (159 percent). These authors concluded that previous caries experience was the strongest predictor in their model, followed by parents' education. For primary teeth which combined sensitivities and specificities totaled 170 percent (Holst, Martensson, Lavrin, et al., 1997). That study used infants 1 year old, for 2 years, and all categories of risk assessment factors. Visible plaque, deep fissures, and oral hygiene were the strongest predictors.

Table 1. Primary teeth-prospective studies (good level of evidence)

Researcher	N	Age at onset	Study Design	Variables: Past Caries or Disease Experience	Variables: Microflora	Variables: Host	Variables: Other	Outcome = Validation criteria = true disease	Sensitivity	Specificity
[Isokangas et al., 1993]	297 (3-4 year olds)	3-4	Prospective (1 year)	Caries, **Predicted caries**	Not used	Not used	Sociodemographic	≤1 dentinal caries lesion in need of restoration (actual data NR)	45%	92%
[Demers et al., 1992]	302	5 year olds	Prospective (1 year)	**Caries experience: dmfs=0 or dmfs>0 (WHO, no radiographs)**	SM, **LB (Bactotest)**	Buffer capacity	Age, sex, parent's education, family structure, fluoride consumption, oral hygiene (debris index)	≥1 ds (mean dfs increment: 2.1 ± 3.6)	81.8% 78.3% (for caries experience only)	77.4% 77.4% (for caries experience only)

* **Bold**: included in final models or strongest predictors
MS: mutans streptococci
LB: Lactobacilli
LRA: logistic regression analysis
LDA: logistic discriminant analysis
NR: Not reported

Table 1. Primary teeth-prospective studies (good level of evidence) (continued)

Researcher	Baseline Scores (Mean ± SD)	True High Risk Criteria Used	Method of modeling	Country	Sampling method	Training of examiners reported	Reliability of examiners	Blinding of examiners	Blinding of patients	Subject Attrition	Authors conclusion
[Isokangas et al., 1993]	NR	High risk: Any caries increment	Not used	Finland (Ylivieska)	All 3-16 year olds in public dental care were included	15 clinicians participated. No training reported.	NR (dentists examined different children)	Not possible for ethical reasons	NR	NR	Clinicians can predict risk using only caries and socio-demographic variables available at annual examinations
[Demers et al., 1992]	NR	At least one new carious lesion in primary teeth: high risk	(LRA; 9 variables studied)	Canada (Montreal) Non-fluoridated community	Random selection of schools	Calibrated (2 examiners)	For caries: Intraexaminer reliability: intraclass correlation coefficient >0.95. The same true for interexaminer reliability For micro test: Intraexaminer reliability:0.80-1.00; interexaminer reliability: 0.79-0.87.	NR	NR	126	Previous caries experience was the best predictor, followed by parent's education.

Table 2. Permanent teeth-children and adolescents; prospective studies (good level of evidence)

Researcher	N	Age at onset	Study Design	Variables: Past Caries or Disease Experience	Variables: Microflora	Variables: Host	Variables: Other	Outcome=Validation criteria=true disease	Sensitivity	Specificity
[Disney et al., 1992b] North Carolina Study "High Risk Prediction Model"	4158: 2079 (Aiken, GA) 2096 (Portland, ME) Both: fluoride deficient, high caries experience	6 years (1st grade) and 10 years old (5th grade)	Prospective (3 years)	**DMFS (Radike, no radiographs),** dmfs, **predicted caries;** fluorosis, white spot lesions	SM (Cariescreen), LB (Bactotest), mean plaque score	Pit and Fissure Morphology	Sociodemographic (higher in Portland-exclusively white); examiner, age, brushing frequency, between meals snacks	≥4 DMFS ≥ 2 DMFS (At 3 years-DMFS increment: Aiken: 1.9 (grade 1), 3.1 (grade 5) Portland: 0.8 (grade 1), 1.5 (grade 5)	59% (grade 1); 62% grade 5 59% (grade 1); 62% (grade 5)	83% (grade 1); 81% (grade 5) 84% (grade 1); 84% (grade 5)
[Isokangas et al., 1993]	1464 (5–16 year olds)	3–16	Prospective (1 year)	Caries, **Predicted caries**	Not used	Not used	Socio-demographic	≤1 dentinal caries lesion in need of restoration (actual data NR)	5-16 year olds; 58%	5-16 year olds:84%;

Table 2. Permanent teeth-children and adolescents; prospective studies (good level of evidence) (continued)

Researcher	Baseline Scores (Mean ± SD)	True High Risk Criteria Used	Method of modeling	Country	Sampling method	Training of examiners reported	Reliability of examiners	Blinding of examiners	Blinding of patients	Subject Attrition	Authors conclusion
[Disney et al., 1992b] North Carolina Study "High Risk Prediction Model"	Aiken: DMFS:0.3 (grade 1), 3.0 (grade 5) dmfs: 9.3 (grade 1), 4.4 (grade 5) Portland; DMFS:0.2 (grade 1), 1.7 (grade 5) dmfs: 2.9 (grade 1), 2.4 (grade 5)	High risk:25% of the total sample size.	(LRA, stepwise, 38–43 variables studied)	USA	NR	Trained	Examiner reliability; intraclass correlations above 90% for 10/12 comparisons. Reliability for noncaries data showed fair agreement among examiners.	Yes	NR	Lost approx. 20% from baseline (more than N)	Models had high specificity for children at low risk. Clinical predictors were the most important ones, while the other factors contributed little to the prediction.
[Isokangas et al., 1993]	NR	High risk: Any caries increment	Not used	Finland (Ylivieska)	All 3-16 year olds in public dental care were included	15 clinicians participate. No training reported.	NR (dentists examined different children)	Not possible for ethical reasons	NR	NR	Clinicians can predict risk using only caries and sociodemographic variables available at annual examinations

Table 3. Permanent teeth adults-prospective studies (good level of evidence)

Researcher	N (dentate)	Age (t outset	Study Design	Variables: Past Caries or Disease Experience	Variables: Microflora	Variables: Host	Variables: Other	Outcome= Validation criteria= true disease	Sensitivity %	Specificity %
[Hawkins et al., 1997;van Houte, 1993]	699	50+	Prospective 3 years	No calculus removed no radiographs Third molars excluded Mean AL (baseline) No of teeth (baseline) Coronal DF	Not Used	Not Used	**Educational level** **Marital status** Age Total household income Dental visiting pattern Born in Canada Major life event in past 6 months Wearing partial denture	One or more net coronal DFS increments	80.2	46.2

Table 3. Permanent teeth adults-prospective studies (good level of evidence) (continued)

Researcher	Baseline Scores (Mean ± SD)	True High Risk Criteria Used	Method of modeling	Country	Sampling method	Training of examiners reported	Reliability of examiners	Blinding of examiners	Blinding of patients	Subject Attrition	Authors conclusion
[Hawkins et al., 1997;van Houte, 1993]	Caries incidence 57% Mean net increment 1.91±2.60	NR	LRA	Canada, Ontario	Random	Calibration reported	94%kappa 0.76 coefficient of reproducibility 0.97 ($p<0.001$)	NR	NR	206	Non-clinical factors, which showed significant effects were education and marital status, both of these factors may influence attitudes towards oral health. The baseline no. of teeth and mean periodontal AL may measure the number of tooth surfaces at risk of decay.

Conclusions

- The predictive validity of the models reviewed depended strongly on caries prevalence and characteristics of the population on which they were based.
- Many models included similar categories of predictors but provided very different outcomes.
- In many instances the use of a single predictor gave results as good as those of a combination of predictors.
- Previous caries experience was a significant predictor in most models tested for primary, permanent, and root caries.
- The desired combination of sensitivity and specificity (more than 160 percent) was only achieved in a few cases.
- None of the studies rated as "good" reached the desirable combined level of sensitivity + specificity.
- None of the controlled longitudinal studies conducted to predict root caries were rated as "good."
- Most of the research in this area has been done in children. There is, therefore, a need to develop better evidence to support caries risk assessment strategies in adults.

Future Research

Clearly, there is a need for further research to identify and validate caries risk assessment strategies that can be applied in dental practice. More importantly, studies are required to establish whether identification of high-risk individuals can lead to more effective long-term patient management that arrests or reverses the progression of carious lesions.

Another recommendation follows from the consistent finding that past caries experience is a strong predictor of future disease. Most studies have used the DMFS (decayed, missing, filled surfaces) index to determine past caries experience. This approach does not necessarily separate out the D component from the F component. Furthermore, this approach does not establish whether decayed lesions are active (progressing) or inactive (arrested). The presence of caries activity should be a much stronger predictor of future carious lesions (frank cavitations) than the DMFS index. The development of technology to detect early caries lesions and to directly assess caries lesion status may prove to be the best way to identify patients who need aggressive preventive intervention.

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Clinical Decision-Making for Caries Management in Primary Teeth

Norman Tinanoff, D.D.S., M.S., and Joanna Douglass, B.D.S., D.D.S.

Historically, dental management of both primary and permanent teeth has involved clinical or radiographic identification of carious lesions followed by surgical intervention to remove affected enamel and dentin and placement of restorative material to rebuild missing tooth structure. Even with preventive therapies and improved understanding of the dental caries disease process, only modest changes have occurred in this surgical model of treatment.

The dental caries process involves cyclical exposure of tooth enamel and dentin to periods of demineralization and remineralization. An acidic oral environment, primarily due to acid byproducts of bacteria that adhere to teeth, will demineralize teeth, especially if the acidic periods are frequent and prolonged. Remineralizing periods, due to salivary buffering and trace amounts of fluoride, can reverse mineral loss. If demineralization over time exceeds remineralization, however, an initial carious lesion can develop that may progress to a frank cavity.

Dental therapy needs to address this disease process by fostering remineralization as well as restoring teeth. Treatment of a child requires an understanding of the carious process that includes the patient's age, caries risk, prior treatment outcomes, and location and extent of lesions. A child who has been identified as being at low risk for dental caries may need fewer diagnostic procedures and therapy. Conversely, a child who is caries-active may need more frequent examinations and therapy.

Primary Teeth

The vast majority of the literature regarding diagnosis and prevention of caries relates to permanent teeth. Although much of this information can be extrapolated to primary teeth, there are important differences. The pits and fissures of primary teeth are less pronounced than those of permanent teeth, making these surfaces less susceptible to caries. However, primary teeth have thinner enamel and dentin and broader proximal contacts than permanent teeth, making them more caries-susceptible (American Academy, 1999-2000).

Unlike therapy for permanent teeth, therapy for primary teeth only needs to last several years. Yet primary teeth are critical for eating and for aesthetics reasons as well as for maintaining space for succedaneous teeth.

Caries in the Primary Dentition

An understanding of the natural history of caries progression in the primary dentition is necessary to determine where lesions are likely to occur, to assess an individual's caries risk, and

to determine what therapy is best. Those teeth that have been exposed to a cariogenic environment the longest generally will be the first to show signs of disease. Consequently, children may develop lesions on their maxillary anterior teeth soon after eruption. If these children continue to be at high risk they may develop fissure caries of the molars and, later, proximal caries of the molars (Johnsen, Gerstenmaier, DiSantis, et al., 1986; Douglass, O'Sullivan, Tinanoff, 1996). Children with moderate caries risk may develop caries at a later age. These are normally fissure caries on the primary molars and possibly posterior proximal lesions (Johnsen, 1995; Douglass, Tinanoff, Tang, et al., 2000). In general, caries on maxillary anterior primary teeth, on the smooth surfaces of primary molars, or on the mandibular primary anterior teeth all suggest high caries activity.

At the individual lesion level, caries progression and appropriate therapy are dependent on the site of the lesion and risk factors. Buccal-lingual smooth surface lesions, even if cavitated, may be readily amenable to preventive regimens, while cavitated pit and fissure or cavitated proximal lesions may need restorative and preventive therapy. The potential for remineralization and appropriate restorative therapy in primary teeth depends on caries activity. One study found that proximal lesion progression through the enamel among a group of high-risk subjects not receiving fluoride took approximately 1½ years, compared to 3½ years in low-risk children receiving regular topical fluoride therapy (Shwartz, Grondahl, Pliskin, et al., 1984).

Caries Risk Assessment for Primary Teeth

The goal of dental caries therapy is to minimize caries experience while employing the fewest possible interventions consistent with the child's risk. A weakness in current caries risk assessment is the lack of a single predictor with both high positive predictive values (proportion of children predicted to get the disease who actually do so) and high negative predictive values (proportion of children predicted to not get the disease who do not). Since caries has multiple causes, multiple risk factors may have to be assessed to determine risk. Combinations of biological variables (e.g., caries experience, plaque index, streptococcus, lactobacillus, and salivary fluoride levels) (Leverett, Featherstone, Proskin, et al., 1993) and social variables (e.g., race, parents' education) (Demers, Brodeur, Mouton, et al., 1992; Disney, Graves, Stamm, et al., 1992) have shown better assessment results than single factors.

In the child patient, key risk factors are the age at which a child becomes colonized with cariogenic flora (Thibodeau, O'Sullivan, Tinanoff, 1993) and the age at which visual caries is found (O'Sullivan, Tinanoff, 1983). Additional information for caries risk assessment includes exposure to fluoride (both systemically and topically), tooth cleaning ability, and diet. Even though these factors do not provide sufficient evidence for a risk assessment analysis, collection of this data may be valuable in developing a prevention program.

Parent and Practitioner Preferences

A child's parent(s), with the advice of the dental professional, are the people who must make decisions for dental therapy (Rule, Veatch, 1993). In light of their own experience, many parents expect surgical treatment of their children's dental caries. The dental professional should present parents with enough information to enable them to make an informed choice from among

all available therapies. Such decisions should also take into account the effects of various therapies on the prevention of disease in teeth that have not erupted. Because of their training and experience, dental professionals may favor certain therapeutic approaches, and such preferences also need to be considered in treatment decisions.

Preventive Therapy

Daily response to fluoride exposure through water supplies or supplemental tablets should be recommended for all children as a primary preventive measure. Perhaps the next best method is daily use of a fluoridated dentifrice. Other kinds of fluoride use should be based on the child's risk. Professional fluoride treatments have been shown to reduce dental caries in primary teeth and should be administered to children at risk. Fluoride varnishes have been shown to be efficacious and have gained popularity recently because they are easy to use and less fluoride is delivered to the mouth (this conference). Fluoride mouth rinses or brush-on fluoride gels have been advised for patients at high risk, but no studies were found that analyzed whether home fluoride protocols reduce caries in primary teeth.

Evidence has accumulated that certain antimicrobials can reduce cariogenic flora and therefore may affect caries activity (this conference). Further research is needed to determine the efficacy and optimal antimicrobial regimen necessary for preventing caries in high-caries risk children.

Sealants are a conservative way to prevent pit and fissure caries by obliterating the deep fissures in primary and permanent molars (this conference). Numerous studies have shown the efficacy of pit and fissure sealant for both permanent and primary teeth (Ripa, 1979), and such treatment should be considered for children who are likely to develop carious lesions in fissures.

Restraint in sugar consumption is also regarded as an important approach to reducing caries. Numerous epidemiological, laboratory, and clinical studies (this conference) make it clear that restricting consumption of sucrose may reduce dental caries. Unfortunately, there are no reports of studies demonstrating that dietary counseling can be effective in reducing caries activity.

But there is good evidence that chewing gum with xylitol reduces caries in primary teeth. Several trials have shown that children who changed to xylitol gum have fewer caries lesions than children who chewed sugared gum, and remarkably, than children who did not chew gum (this conference).

Poor oral hygiene is widely considered a factor in caries activity. Conversely, toothbrushing, flossing, and professional tooth cleaning have long been considered basic components of caries prevention. Yet clinical studies generally do not demonstrate a relationship between dental plaque scores and dental caries prevalence, or between unmedicated toothcleaning procedures and caries prevalence (Sutcliffe, 1966). Even though there may be no firm scientific connection between oral hygiene and caries, caries reductions have been noted in children who receive frequent professional prophylaxis along with some form of fluoride therapy (Lindhe, Axelsson, Tollskog, 1975) or who brushed frequently with a fluoridated dentifrice (Leske, Ripa, Barenie, 1976). If the specific contribution of toothcleaning remains unknown,

however, there does exist a significant body of research suggesting that regular brushing should at least be encouraged as a delivery system for a fluoride dentifrice (this conference).

Restorative Therapy

Restorative therapy should always be used in conjunction with preventive therapy and should also be based on an understanding of a child's risk factors and age. The principal role of restorative therapy is to eliminate cavitations that make plaque removal difficult and consequently increase the likelihood that a tooth will undergo further demineralization. Restorations are essential where a remineralization environment cannot be maintained, where initial therapy was unsuccessful, or where restoration of tooth integrity and function is necessary. If, for example, a posterior proximal cavitation is not restored, it will most likely progress and threaten the integrity of cusps, cause space loss, and eventually affect the pulp.

The size of the carious lesion, the therapeutic and esthetic requirements of the restorative material, and caries risk factors and age must be considered when restoring a tooth. There is an emerging class of restorative materials that are considered therapeutic because they release fluoride. Although some of these materials may not have the integrity of conventional materials, they can be used in certain situations or for certain age groups. Young children at high risk for future caries should be treated aggressively to minimize the need for additional restorations. There is good evidence that stainless steel crown restorations function better in such children than multisurface intercoronal restorations (Levering, Messer, 1988).

Summary

The information presented in this and other papers at this conference suggests that sufficient evidence exists to transcend traditional surgical management of dental caries. New information on diagnosis, lesion progression, risk assessment, and caries prevention provides insight on tooth management that relies less on surgical techniques and more on monitoring and prevention. Patients and practitioners alike will derive great benefit from treatment decisions based on our emerging understanding of dental caries as a multifaceted disease process that should be approached with broad-ranging, outcomes-based therapy.

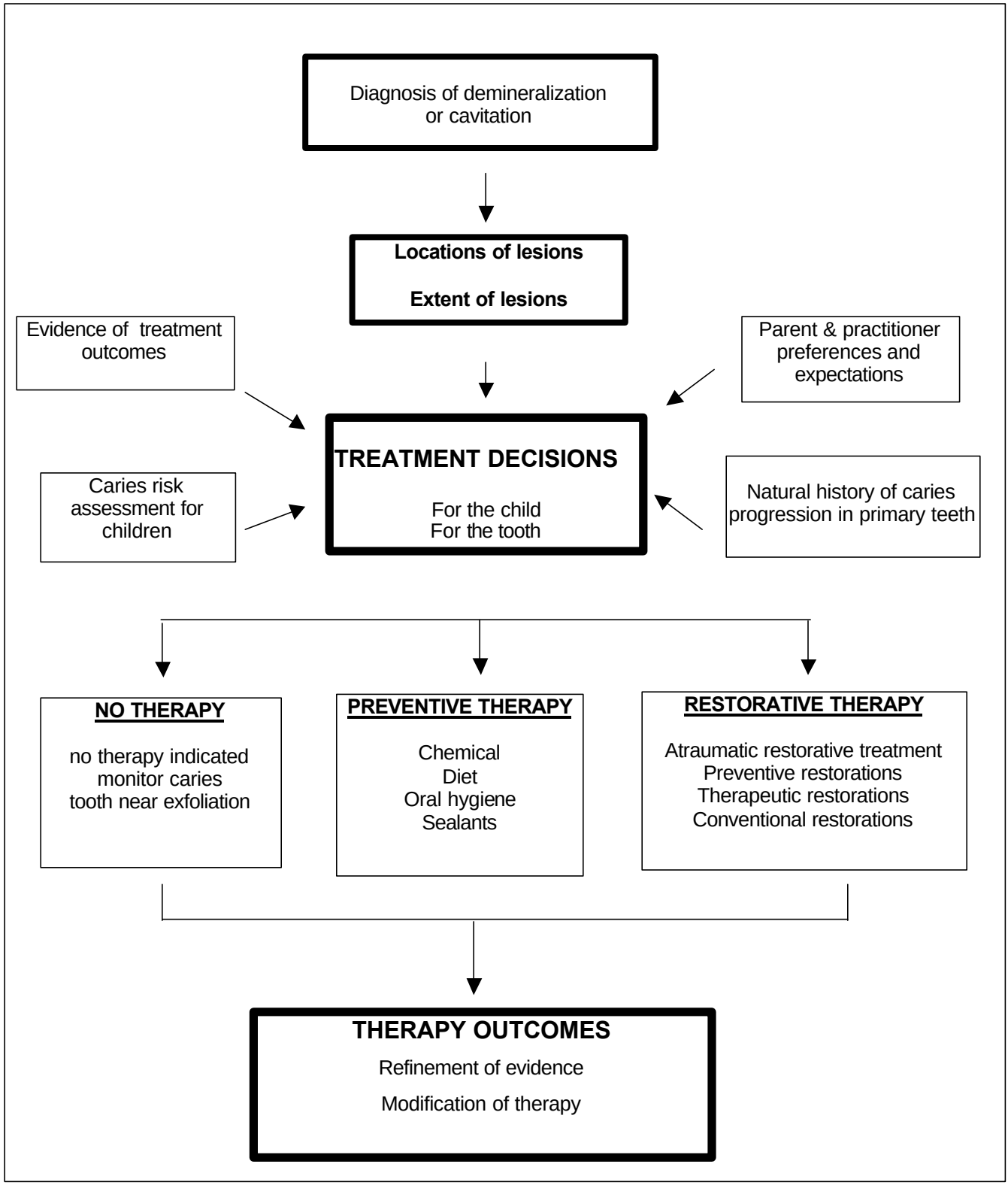

Figure. A concept for primary teeth diagnosis and therapy based on caries risk assessment

Table. Possible diagnostic procedures, preventive therapy, and restorative therapy in primary teeth based on a child's caries risk assessment and age

	Low Risk	Moderate Risk	High Risk
Caries Risk Factors	dmfs < ½ child's age	dmfs >1/2 child's age	dmfs > child's age
	no new lesions in 2 years	1 or more lesion in 2 years	2 or more lesions in 1 year
	no white spot lesions	infrequent white spot lesions	numerous white spot lesions
	low titers of mutans strep high SES	moderate titers of mutans strep	appliances in mouth
		moderate SES	high titers of mutans strep.
			low SES
			high frequency sugar consumption
Diagnostic Procedures	examination interval 12–18 months	examinations interval 6–12 months	examination interval 3–6 months
	radiograph interval 12–24 months	radiograph interval 12 months	radiograph interval 6–12 months
	initial mutans strep evaluation	initial mutans strep evaluation	mutans strep testing to monitor compliance
			diet analysis
Preventive Therapy	fluoridated dentifrice	fluoridated dentifrice	fluoridated dentifrice
	fluoride supplements *	fluoride supplements *	fluoride supplements *
		professional topical fluorides tx	professional topical fluoride tx
		sealants	sealants
			daily home fluoride or antimicrobials
			dietary counseling and adjustments
Restorative Therapy			
• age 2-4	monitoring, therapeutic or conventional restorations	therapeutic or conventional restorations	therapeutic or conventional restorations
• age 4-6	monitoring or conventional restorations	therapeutic or conventional restorations	therapeutic or conventional restorations
• age 6-8	monitoring or conventional restorations	therapeutic or conventional restorations	therapeutic or conventional restorations
• age 8-10	monitoring or conventional restorations	semi-permanent, therapeutic or conventional restorations	semi-permanent, therapeutic or conventional restorations

* depending on age and water supply fluoridation

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Clinical Decision-Making for Coronal Caries Management in the Permanent Dentition

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Clinical decisions on caries diagnosis and appropriate treatment are quite variable. Because of the limitations of diagnostic devices and uncertainty in interpreting images and tactile responses, treatment decisions can lead to both overtreatment and undertreatment. Overtreatment is of major concern because premature or unnecessary restoration eliminates the chance for remineralization and does not necessarily reduce the caries risk of patients. Undertreatment, on the other hand, may lead to undetected progression of caries lesions and result in larger restorations. For low-risk patients, however, more conservative treatment decisions are justifiable, and the consequences of undertreatment should be less significant for them than the consequences of undertreatment for high-risk patients.

In this era of evidence-based dentistry, decisions to place initial restorations or to replace "faulty" ones are being questioned. As the prevalence of caries has declined, we have realized that it is critically important that patients at low risk for caries should not be prescribed the same treatment as high-risk patients. In addition, we now know that noncavitated enamel lesions can be arrested, and that noncavitated tooth enamel can be remineralized and hardened. We have also learned that caries lesions generally progress rather slowly. Thus, questionable or early caries lesions can be monitored for several years before a decision is made to intervene surgically. There is considerable uncertainty in diagnosing early lesions accurately because of the rather low sensitivity of current diagnostic methods. For a successful treatment decision to be made, the presence of a lesion must be determined at a sufficiently high level of certainty. It is not sufficient simply to determine the presence of a lesion, since many noncavitated lesions are arrested and tooth structure can be remineralized. It is important to determine whether a lesion is active prior to making a decision to restore or re-restore.

The first step in the decision-making process is to conduct a thorough analysis of the patient's health and dental history, based on (1) individual, family, and community health levels; (2) a clinical oral exam; and (3) risk factors, including previous dental experience (DMFS, DFS, DMFT, DFT), smoking, general health, manual dexterity, learning ability, sociodemographic data, behavioral factors, diet and nutrition, fluoride exposure, and dental health knowledge. The oral exam may require visual, tactile, radiographic, bacterial, and other diagnostic methods to record plaque levels and potentially high-risk areas of enamel demineralization. The exam should also identify high-risk tooth surfaces for caries initiation and progression, such as existing white spots or areas where plaque accumulation is likely. Caries risk may be defined as the probability that an initial lesion will develop or that an existing lesion will progress over a specified period of time. The exam must be sufficiently accurate to positively diagnose the presence of caries lesions, if present, and questionable lesions, if high sensitivity in diagnosis is not possible.

The second step is to describe the extent of all lesions, if possible, using a classification such as the following: E0 (no enamel lesion); E1 (lesion in the outer half of enamel); E2 (lesion

in the inner half of enamel); D1 (outer third of dentin); D2 (middle third of dentin); and D3 (inner third of dentin). Such a classification will permit lesion activity over time to be determined and the success of caries arresting and remineralizing treatments to be assessed.

The third step is to list possible treatment options as a function of present and predicted risk levels. Treatment options for a nonrestored site (in the most general sense) include (1) no treatment except for oral prophylaxis and monitoring; (2) oral prophylaxis followed by chemotherapeutic management of infection (fluoride only, or chlorhexidine and fluoride) and monitoring; and (3) placement of a sealant, repair or sealing of a restoration, or placement/replacement of a restoration.

Optimizing the Decision-Making Process

The main objective of this review is to answer the following question: What are the appropriate treatment options for coronal caries in permanent teeth for patients at low-, moderate-, or high-risk for primary and secondary caries initiation and progression? One needs to know whether the lesion is slightly or well into enamel, or slightly or well into dentin. Furthermore, one must know whether the caries process is active or arrested. This can best be determined by monitoring the lesion over time. For a high-risk individual, one might choose to restore or monitor a lesion that extends slightly into dentin.

There is some evidence that supports the placement of a restoration when the lesion has progressed 0.5 mm or more into dentin. However, this recommendation may have been based on individuals at a moderate- to high-risk of caries progression. What threshold level is appropriate for low-risk patients is unknown. For the most minimally invasive strategy, actual observation of tooth surface cavitation can be considered the threshold for placement or replacement of a restoration. The long-term goal is to ensure that the best outcome is reached, based on the most reliable scientific evidence and practical experience.

To competently answer the question posed at the beginning of this section about treatment options, the following additional information is required:

1. Probability of lesion progression as a function of caries risk level

2. Probability of tooth surface cavitation over a specified period of time

3. Best treatment methods to arrest active lesions and potentially to remineralize teeth with noncavitated lesions as a function of patient risk level

4. Lesion depth at which a restoration should be placed (threshold for surgical intervention) for a patient’s initial risk level and at recall exams.

Obviously, the optimal outcome for a high-risk patient with a D1 lesion would be based on a treatment decision to not restore the tooth with a D1 lesion but to monitor the lesion over time. For an approximal lesion, tooth separation would be required to ensure that cavitation of the approximal surface has not occurred. This may not be deemed practical by most dentists, and

the next best option would be to use probability data based on the studies of Pitts and Rimmer (1992) and others.

Chemotherapeutic Agents for Reducing Caries Risk

Unfortunately, few randomized, controlled clinical trials have been conducted to answer questions related to management of caries as a chronic infectious disease. Thus, we may need to use data from studies that are based on populations rather than studies in which the caries risk of individual subjects was assessed.

We can justify delaying the restorative treatment of enamel lesions in the inner half of enamel (and even slightly into dentin) on the basis that caries progression through enamel in moderate-risk and high-risk patients is slow (Shwartz, Pliskin, Grondahl, et al., 1984; Berkey, Douglass, Valachovic, et al., 1988). Caries progression has been decreasing over recent decades (Ekanayake, Sheiham, 1987) and is slower in patients who have received regular fluoride treatment or who consume fluoridated water (Pitts, 1983; Shwartz, Pliskin, Grondahl, et al., 1984a; Schwartz, Grondahl, Pliskin, 1984b). Progression time through enamel may take from 6 to 8 years. Since many enamel lesions remain unchanged or progress very slowly over long periods, and because progression rates through dentin may also be comparably slow (Emslie, 1959; Kolehmainen, Rytömaa, 1977), there is adequate time to apply infection control and monitoring procedures to assess caries risk and lesion activity. Furthermore, the percentage of radiographically visible approximal lesions in the outer half of dentin that are cavitated has declined over the past several decades to approximately 41 percent.

Preservative dentistry is based on a refined model of decision-making consisting of accurate caries diagnosis, classification of caries severity using radiographs, assessment of patient's caries risk (high, moderate, or low), placement of restorations in teeth with cavitated lesions, arresting of active lesions, remineralizing of noncavitated arrested lesions, monitoring of noncavitated lesions over time, and assessing of management outcomes (change in DMFS, DFS, D/DMFS, D/DFS, and D/DFS) at predetermined intervals. The bacterial infection which causes the production of demineralizing acids should be controlled to ensure the arrest of demineralization and, potentially, the initiation of remineralization. Once a decision has been made to monitor rather than restore primary or secondary lesions, the next decision is to decide whether caries risk can be reduced through the use of fluoride agents alone or in combination with antimicrobial therapy.

The effectiveness and sustantivity (sustaining power) of chlorhexidine in reducing the levels of S. mutans and potentially to enhance remineralization of demineralized enamel for high-risk patients provide renewed optimism for reducing caries risk and increasing the probability that a restoration decision may never need to be made (Schiøtt, Briner, Löe, 1976; Schiøtt, Briner, Kirkland, et al., 1976; Emilson, 1977; Emilson, Lindquist, Wennerholm, 1987; Katz, 1982; Zickert, Emilson, Ekbloom, et al., 1987; Schaeken, DeHaan, 1989; Schaeken, Keltjens, Van Der Hoeven, 1991; Persson, Truelove, LeResche, et al., 1991; Joyston-Bechal, Hayes, Davenport, et al., 1992; Sorvari, Spets-Happonen, Luoma, 1994; Tenovuo, Hakkinen, Paunio, et al., 1992; Ullsfoss, Ögaard, Arends, et al., 1994; Pienihäkkinen, Soderling, Ostela, et al., 1985; Anusavice, 1998; Petersson, Magnusson, Andersson, et al., 1988). However, only

limited data are available on the optimum strategy for treatment of individual patients. Thus, data obtained in private practice from combined chemotherapeutic and fluoride treatment will be required in addition to published clinical trial data to further develop our ability to manage caries.

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Clinical Decision-Making for Caries Management in Root Caries

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This is a review of studies on diagnosing, predicting, and intervening in the disease known as root caries that may help clinicians communicate information for their decisions on care to patients.

Questions Addressed in This Review

5. What is the natural history of root caries among North American populations? Natural history in this case includes definitions of lesions at different stages; the activity of lesions (active, inactive); rate of progression from stage to stage; reversibility under natural conditions of lesions, by stage; and outcome of untreated root caries.

6. How accurate and reliable are the methods we have to diagnose active and inactive root caries?

7. For persons with root caries, are there differences in outcomes (absolute improvement in number of teeth retained and functional, or relative improvement, or number needing treatment) between subjects randomly assigned to receive therapeutic care and those not receiving such care?

Search Strategy

A search strategy was developed by a consultant to the project, and searches of EMBASE and MEDLINE resulted in a database of 807 annotated references. The annotated references were read independently by at least two people to achieve consensus on 94 that were selected for retrieval. The reference lists in those 94 were then checked, and studies that appeared to be related to our questions were added, producing a final database of 162 references.

Ideally, the evidence should have been selected from high-scoring studies with strong design, as described in criteria of the Agency for Health Care Policy and Research (AHCPR). Many studies, however, were both weak in design and of limited value. Since the evidence on management of root caries is rarely supported by more than a few studies, recommendations on how to do so can only be tentative.

Findings on Prevalence

Prevalence estimates of root decayed and filled surfaces (RDFS) were taken from the NHANES III study. The adjusted prevalence for U.S. adults, as measured by those with one or more lesions, was 25.1 percent. Prevalence increased with age, and by age 75, 55.9 percent had one or more lesions. Severity as measured by the mean number of RDFS was 1.2, of which

58.3 percent were filled. As expected, severity was also age-dependent. Women had lower prevalence (23.3 percent vs. 27.1 percent), lower mean scores (1.0 vs. 1.4) and lower proportions filled (50.0 percent vs. 64.3 percent) than men. Among patients age 34 and older, the prevalence was roughly 20 percent less than a person's age. For example, a person age 50 would have a 30 percent probability of having one or more RDFS.

Findings on Incidence

Eight papers met the inclusion criteria for determining the incidence of root lesions (Hand, Hunt, Beck, 1988a; Hand, Hunt, Beck, 1988b; Leske, Ripa, 1989; Wallace, Retief, Bradley, 1988; Lawrence, Hunt, Beck, 1995; Lawrence, Hunt, Beck, et al., 1996; Locker, 1996; Powell, Leroux, Persson, et al., 1998). These eight discussed five different investigations (two of the studies each discussed two papers). The studies that lasted 16 to 18 months showed much higher incidence estimates that did the studies lasting 3 years or more. Calculation of a duration/sample-size weighted estimate from the results of the four longest studies showed that 8.2 percent of study subjects would be expected to acquire one or more new root caries in 1 year. Those four studies plus one other showed that, on average, dentate people would be expected to acquire 0.19 new RDFS per year.

Clarkson (1995) added a cautionary note when she pointed out that conventional studies of incidence would not pick up restorations of secondary root caries, leading to an understatement of the actual incidence of lesions by as much as two-thirds.

Description of Root Caries Lesions

Diagnosis of a root caries lesion is established through the use of clinical descriptors. These vary, and are subjective. Clinical description is based on color, texture, surface smoothness, depth of the lesion, and distinctiveness of its border, overlayed with a judgment on whether the lesion is active or inactive. Variability in the diagnostic criteria, and the question of whether restored roots are included, strongly affect estimates of the prevalence and severity of root caries lesions (Katz, 1980; DePaola, Soparker, Kent, 1989; Aherne, O'Mullane, Bennett, 1990; Stamm, Banting, Imrey, 1990; Banting, 1993; Fejerskov, Baelum, Östergaard, 1993). The variability in diagnostic criteria limits validity because lesions which apparently "reverse either true reversals or examiner error (Lawrence, Hunt, Beck, et al., 1986; Beck, Lawrence, Koch, 1995).

Katz (1986) defined active and inactive lesions, but that was a statement of consensus.

Severity Index

Billings (1986) developed a staging classification, termed a "severity index," of root caries lesions as follows: Grade I (incipient), Grade II (shallow), Grade III (cavitation), and Grade IV (pulpal). This index, however, was not derived from longitudinal studies of the same teeth in the same individuals.

Diagnostic System

Five articles provided material for the evidence table on diagnostic systems. The evidence indicates that practitioners have little alternative but to use systems for diagnosing root caries lesions that have low reliability and whose accuracy is unknown. While there is little to recommend any one system over the others, the texture (soft/hard) components of the Billings (1985) and the Hellyer (1990) systems have at least been shown to correspond to histopathology findings (Schupbach, Guggenheim, Lutz, 1990) and penetration by micro-organisms (Beighton, Lynch, Heath, 1993).

Therapy for Root Caries

Seven studies that dealt with remineralization of a tooth with a root caries lesion are included in the evidence table (Billings, Brown, Koster, 1985; Wallace, Retiel, Bradley, 1993; DePaola, 1993; Schaehen, Keltjens, Van Der Hoeven, 1991; Emilson, Ravald, Birkhed, 1993; Johansen, Papas, Fong, et al., 1987; Nyvad, Feyerskov, 1986). The available evidence supports remineralizing with fluoride rinses and, somewhat more tentatively, with fluoride gels and varnishes or chlorhexidine varnish. Also offered as a treatment option was recontouring before remineralizing with fluoride. However, the efficacy of recontouring followed by fluoride treatment was only demonstrated in six people with a total of 13 lesions.

Evidence on restoration of lesions is even more tentative. No studies were found that compared methods of restoring root caries over what would be considered a sufficiently long term. Of the four studies in the evidence table (Billings, Brown, Koster, 1985; Levy, Jenson, Doering, et al., 1989; Duke, Robbins, Snyder, 1991; Sheth, Lesen, Wefel, et al., 1988), the longest was 3 years in duration; the only controlled comparison ran for 1 year. The very limited data suggest that dentists may restore root caries with composite resins, although conventional practice may allow glass ionomer or even amalgam restorations (though no studies are listed).

Conclusions

Generally, studies on the management of root caries do not offer strong evidence on how to care for patients. They are few in number, and they are compromised either in design or duration. The literature is so limited that the issue of which approaches might be more appropriate in terms of patient preference, costs, and efficiency cannot be addressed. Research is needed to validate the accuracy of current diagnostic methods, provide evidence on the efficacy of therapeutic measures through more rigorous designs extending over longer periods, and address the issue of patient-based measures of outcomes.

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The Scientific Basis for the Teaching and Practice of Conservative Operative Dentistry

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Once a carious lesion requires operative intervention to halt the caries process and restore lost tooth structure, what form should that intervention take and what factors are involved in providing maximum longevity of the resulting restoration and tooth? This paper looks at the evidence for conservative operative intervention, attempts to assess the relationship between cavity preparation and restoration survival, and documents the major factors involved in restoration failure.

Conservative Cavity Preparation

Traditional operative dentistry involves standardized preparation that utilizes differing degrees of "convenience form" (access to caries) and "extension for prevention" (placing cavity margins in less caries-susceptible locations) and can reduce the structural and biological integrity of teeth. Conservative forms of operative intervention have now been recommended that concentrate more specifically on removal of carious dentine and preservation of as much sound tooth structure as possible. These are discussed below.

The Proximal "Tunnel" Restoration

The "tunnel" concept, which accesses proximal dentinal caries through a sound mesial or distal occlusal pit to preserve the proximal marginal ridge, was described by Hunt (1984). A total of 10 clinical trials in permanent teeth and 2 in primary teeth on this concept had been conducted through the 1990s. Early clinical reports utilized small numbers of glass ionomer restorations and indicated that the technique was promising. but later reports found higher failure rates. Use of a metal cermet glass ionomer gave little evidence of inhibition of recurrent caries, while the most frequent causes of restoration failure were marginal ridge fracture and recurrent decay. A higher proportion of the marginal ridge fractures was associated with more extensive tunnel preparations. The longest clinical study (7 years) reported a 50 percent survival time of 6 years for restorations, (Hasselrot, 1998), while two recent multi-operator trials provided evidence of high rates of associated caries (41-45 percent) as early as 3 years (Nordbo, Leiskar, von der Fehr,1998; Pilebro, van Dijken, Stenberg, 1999). Poor performance in primary teeth has also been documented (Hasselrot, 1993; de Freitas, de Andrada, Baratieri, 1994).

Many studies of the tunnel concept utilizing baseline radiographs have reported evidence of inadequate caries removal (Hasselrot, 1993, 1998; Strand, Nordbo, Tveit, 1996; Pilebro, van Dijken, Stenberg, 1999). This was presumably due to the blind approach provided by limited access. Visibility was only improved by enlarging the occlusal access, thus reducing the conservative nature of the technique (Knight, 1992). Low restoration survival was associated with limited preparation-extension in high caries individuals, especially where demineralized

proximal enamel was left in order to avoid cavitation of the proximal surface (Strand, Nordbo, Tveit, et al., 1996; Pilebro, van Dijken, Stenberg, 1999).

This technique is limited to treatment of early dentinal decay, often prior to enamel cavitation. Since cavitation is becoming accepted as the stage that defines the necessity for operative intervention, the technique has limited use. The low effectiveness reported argues in favor of a more direct approach to proximal caries. It also affirms the difficulty in arresting proximal caries.

The Proximal "Box-Only" Restoration

Traditional Class 2 cavity preparation for the treatment of proximal caries involves both a proximal and occlusal portion of the tooth. Changes in this approach have been recommended where only the proximal tooth structure is carious. Although "box-only" (or "slot") preparations for amalgam were introduced in 1973 (Almquist and colleagues) and "adhesive slot" preparations for resin composite were introduced in 1978 (Simonsen), such conservative restorations are still relatively rare in general dental practice. Our search of the literature turned up only three clinical studies of these kinds of restorations in permanent teeth. One study found no failures in 68 composite box-only restorations over 5 years (Kreulen, Tobi, van Amerongen, 1998). Another found that the 10-year success rate for composite proximal "saucer" preparations was 68.6 percent (Nordbo, Leiskar, von der Fehr, 1998). Half of the failures were due to recurrent decay, and half were considered technique-related. Recurrent caries, when present, occurred only at the gingival margin, not bucco-lingually, justifying the minimal lateral and occlusal extension. Loss of retention did not occur. A third study found no failures in amalgam restorations of this kind over periods of 5 to 7 years (Lumley, Fisher, 1995). All three trials give us good evidence that the proximal slot-only restoration is a viable treatment option, providing similar or better longevity compared to conventional Class 2 composite or amalgam restorations, and greater tooth preservation. In short, the technique was reported to be superior to tunnel restorations, probably because of better operator visibility.

Four studies of modified proximal restorations in primary teeth involved were found, of which three were of only 1-year duration. The fourth and longest (3 years) showed poor performance for a cermet glass ionomer but significant improvement with a resin-modified glass ionomer, with an estimated median survival time exceeding 42 months (Espelid, Tveit, Tornes, et al., 1999).

Gingival Margin Location

Gingival extension of Class 2 restorations, whether traditional or box-only design, is of particular importance. Most recurrent decay occurs in the gingival proximal location (Mjor, 1998; Klausner, Green, Charbeneau, 1987). The "extension for prevention" concept suggests that subgingival margins reduce the risk of secondary caries, but the evidence for this comes from the prefluoride era. The need for appropriate location of the gingival proximal margin was shown to be important in a rare clinical trial that examined the relationship between proximal cavity design and recurrent caries (Otto, Rule, 1988). Restorations with gingival margins that did not clear the contact area had a significantly higher rate of caries at all time intervals over a 2-year period.

Since creating a "self-cleansing" location for the gingival margin of proximal restorations is impossible, good home care by patients is essential. Whether conservative gingival extension increases the risk of recurrent caries in the absence of such home care remains to be determined.

The Preventive Resin Restoration

The preventive resin restoration (PRR) is a conservative occlusal restoration that involves replacement of discrete areas of carious tooth structure with composite, followed by application of an overlying fissure sealant, instead of the traditional "extension for prevention" (Simonsen, 1980).

A total of 18 clinical studies on the PRR were published between 1978 and 1999. Although they report generally favorable outcomes, all 18 also report the loss of all or a portion of the sealant as a major problem. The success rates of the studies are not easily comparable, since definitions of failure were variously reported as presence of actual caries or loss of sealant. Three of the studies involved a direct comparison of PRR with silver amalgam (Azhdari, Sveen, Buonocore, 1979; Welbury, Walls, Murray, et al., 1990; Cloyd, Gilpatrick, Moore, 1997). The PRR was at least as successful as amalgam in two of the trials for a period up to 5 years, with the added advantage of preservation of sound tooth structure, but Cloyd and colleagues found sealant failure to be a significant problem, leading to recurrent caries in 8.1 percent of patients. No amalgam failures were recorded over 3 years. None of the 18 studies found occlusal caries when the sealant remained intact, though many did not utilize radiographs at recall. All cases of occlusal caries (up to 24 percent after 9 years) were associated with sealant failure, but the incidence of sealant failure was significantly higher than the presence of caries (Houpt, Fukus, Eidelman, 1994). Loss of sealant over glass ionomer restorative materials (Gray, Paterson, 1994; Kilpatrick, Murray, McCabe, 1996) and larger areas of composite restoration (Gray, 1999) was high. Another study (Mertz-Fairhurst, Curtis, Ergle, et al., 1998) found that sealed composite restorations were able to halt the radiographically observed progress of frank carious dentin over a period of 10 years. This provides some reassurance in cases of inadvertent sealing of incipient dentinal caries and has implications for the conservative treatment of deep carious dentine in the vicinity of the pulp.

In summary, PRR is a predictable and effective conservative treatment for localized areas of occlusal decay, with longevity dependent on retention of the overlying sealant.

Factors Involved in Restoration Failure

Secondary caries is the most frequently cited reason for restoration failure or replacement, followed by fractured restorations. The reasons for replacement are related to many clinical variables that have been grouped as either patient, operator, or dental material factors. A systematic review of dental restoration longevity (Downer, Azli, Bedi, et al., 1999) found strong indications of both patient (age and caries activity) and operator factors. High caries activity in relation to bacterial assay and salivary flow rates (Bentley, Broderius, Drake, et al., 1990; Köhler, Rasmussen, Odman, 2000), poor oral hygiene and PI scores (Goldberg, Tanzer, Munster, et al., 1981; Eriksen, Biertness, Hansen, 1986) and incidence of new primary or secondary caries (Jokstad, Mjor, 1991a and b) are all common reasons for restoration replacement.

The frequency of restoration replacement is higher in younger populations, and highest in the primary dentition (Wendt, Koch, Birkhed, 1998). Both recurrent caries and failure of materials figure prominently in primary dentition studies. Whereas there is some evidence for caries susceptibility as a factor in primary restoration failure, there is also strong evidence that age at time of treatment and size of the restoration are factors (Wong, Day, 1990). Problems with materials are pronounced, with survival times longest for stainless steel crowns and shortest for conventional glass ionomer restorations in posterior teeth (Papathanasiou, Curzon, Fairpo, 1994; Kilpatrick, 1993).

While materials and operator skill are important factors in recurrent caries, the problems seem to be more closely related to patient management of tooth care.

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