
Oral Health in America: A Report of the Surgeon General

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National Institute of Dental
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Message from Donna E. Shalala

Secretary of Health and Human Services

The intent of this first-ever Surgeon General's Report on Oral Health is to alert Americans to the full meaning of oral health and its importance to general health and well-being. Great progress has been made in reducing the extent and severity of common oral diseases. Successful prevention measures adopted by communities, individuals, and oral health professionals have resulted in marked improvements in the nation's oral and dental health.

The terms oral health and general health should not be interpreted as separate entities. Oral health is integral to general health; this report provides important reminders that oral health means more than healthy teeth and that you cannot be healthy without oral health. Further, the report outlines existing safe and effective disease prevention measures that everyone can adopt to improve oral health and prevent disease.

However, not everyone is experiencing the same degree of improvement. This Surgeon General's report addresses the inequities and disparities that affect those least able to muster the resources to achieve optimal oral health. For whatever the reason, ignoring oral health problems can lead to needless pain and suffering, causing devastating complications to an individual's well-being, with financial and social costs that significantly diminish quality of life and burden American society.

For a third decade, the nation has developed a plan for the prevention of disease and the promotion of health, including oral health, embodied in the U.S. Department of Health and Human Services document, Healthy People 2010. This Surgeon General's Report on Oral Health emphasizes the importance of achieving the Healthy People goals to increase quality of life and eliminate disparities. As a nation, we hope to address the determinants of health—individual and environmental factors—in order to improve access to quality care, and to support policies and programs that make a difference for our health. We hope to prevent oral diseases and disorders, cancer, birth defects, AIDS and other devastating infections, mental illness and suicide, and the chronic diseases of aging.

We trust that this Surgeon General's report will ensure that health promotion and disease prevention programs are enhanced for all Americans. This report proposes solutions that entail partnerships—government agencies and officials, private industry, foundations, consumer groups, health professionals, educators, and researchers—to coordinate and facilitate actions based on a National Oral Health Plan. Together, we can effect the changes we need to maintain and improve oral health for *all* Americans.

Foreword

The growth of biomedical research since World War II has wrought extraordinary advances in the health and well-being of the American people. The story is particularly remarkable in the case of oral health, where we have gone from a nation plagued by the pains of toothache and tooth loss to a nation where most people can smile about their oral health. The impetus for change—to take on the challenge of addressing oral diseases as well as the many other health problems that shorten lives and diminish well-being—led to the postwar growth of the National Institutes of Health. In 1948 the National Institute of Dental Research—now the National Institute of Dental and Craniofacial Research—joined the National Cancer Institute and the National Heart, Lung, and Blood Institute as the third of the National Institutes of Health.

The Institute's research initially focused on dental caries and studies demonstrating the effectiveness of fluoride in preventing dental caries, research that ushered in a new era of health promotion and disease prevention. The discovery of fluoride was soon complemented by research that showed that both dental caries and periodontal diseases were bacterial infections that could be prevented by a combination of individual, community, and professional actions. These and other applications of research discoveries have resulted in continuing improvements in the oral, dental, and craniofacial health of Americans. Today, armed with the high-powered tools, automated equipment, and imaging techniques of genetics and molecular and cell biology, scientists have set their sights on resolving the full array of craniofacial diseases and disorders, from common birth defects such as cleft lip and palate to the debilitating, chronic oral-facial pain conditions and oral cancers that occur later in life.

The National Institute of Dental and Craniofacial Research has served as the lead agency for the development of this Surgeon General's Report on Oral Health. As part of the National Institutes of Health, the Institute has had ready access to ongoing federal research and the good fortune to work collaboratively with many other agencies and individuals, both within and outside government. The establishment of a Federal Coordinating Committee provided a formal mechanism for the exchange of ideas and information from other departments, including the U.S. Department of Agriculture, Department of Education, Department of Justice, Department of Defense, Department of Veterans Affairs, and Department of Energy. Active participation in the preparation and review of the report came from hundreds of individuals who graciously gave of their expertise and time. It has been a pleasure to have had this opportunity to prepare the report, and we thank Surgeon General David Satcher for inviting us to participate.

Despite the advances in oral health that have been made over the last half century, there is still much work to be done. This past year we have seen the release of Healthy People 2010, which emphasizes the broad aims of improving quality of life and eliminating health disparities. The recently released U.S. General Accounting Office report on the oral health of low-income populations further highlights the oral health problems of disadvantaged populations and the effects on their well-being that result from lack of access to care. Agencies and voluntary and professional organizations have already begun to lay the groundwork for research and service programs that directly and comprehensively address health disparities. The National Institutes of Health has joined these efforts and is completing an agencywide action plan for research to reduce health disparities. Getting a healthy start in life is critical in these efforts, and toward that end, a Surgeon

General's Conference on Children and Oral Health, *The Face of a Child*, is scheduled for June 2000. Many other departmental and agency activities are under way.

The report concludes with a framework for action to enable further progress in oral health. It emphasizes the importance of building partnerships to facilitate collaborations to enhance education, service, and research and eliminate barriers to care. By working together, we can truly make a difference in our nation's health—a difference that will benefit the health and well-being of all our citizens.

Ruth L. Kirschstein MD
Acting Director
National Institutes of Health

Harold C. Slavkin DDS
Director
National Institute of Dental
and Craniofacial Research

Preface

from the Surgeon General
U.S. Public Health Service

As we begin the twenty-first century, we can be proud of the strides we have made in improving the oral health of the American people. At the turn of the last century, most Americans could expect to lose their teeth by middle age. That situation began to change with the discovery of the properties of fluoride, and the observation that people who lived in communities with naturally fluoridated drinking water had far less dental caries (tooth decay) than people in comparable communities without fluoride in their water supply. Community water fluoridation remains one of the great achievements of public health in the twentieth century—an inexpensive means of improving oral health that benefits all residents of a community, young and old, rich and poor alike. We are fortunate that additional disease prevention and health promotion measures exist for dental caries and for many other oral diseases and disorders—measures that can be used by individuals, health care providers, and communities.

Yet as we take stock of how far we have come in enhancing oral health, this report makes it abundantly clear that there are profound and consequential disparities in the oral health of our citizens. Indeed, what amounts to a “silent epidemic” of dental and oral diseases is affecting some population groups. This burden of disease restricts activities in school, work, and home, and often significantly diminishes the quality of life. Those who suffer the worst oral health are found among the poor of all ages, with poor children and poor older Americans particularly vulnerable. Members of racial and ethnic minority groups also experience a disproportionate level of oral health problems. Individuals who are medically compromised or who have disabilities are at greater risk for oral diseases, and, in turn, oral diseases further jeopardize their health.

The reasons for disparities in oral health are complex. In many instances, socioeconomic factors are the explanation. In other cases, disparities are exacerbated by the lack of community programs such as fluoridated water supplies. People may lack transportation to a clinic and flexibility in getting time off from work to attend to health needs. Physical disability or other illness may also limit access to services. Lack of resources to pay for care, either out-of-pocket or through private or public dental insurance, is clearly another barrier. Fewer people have dental insurance than have medical insurance, and it is often lost when individuals retire. Public dental insurance programs are often inadequate. Another major barrier to seeking and obtaining professional oral health care relates to a lack of public understanding and awareness of the importance of oral health.

We know that the mouth reflects general health and well-being. This report reiterates that general health risk factors common to many diseases, such as tobacco use and poor dietary practices, also affect oral and craniofacial health. The evidence for an association between tobacco use and oral diseases has been clearly delineated in every Surgeon General's report on tobacco since 1964, and the oral effects of nutrition and diet are presented in the Surgeon General's report on nutrition (1988). Recently, research findings have pointed to possible associations between chronic oral infections and diabetes, heart and lung diseases, stroke, and low-birth-weight, premature births. This report assesses these emerging associations and explores factors that may underlie these oral-systemic disease connections.

To improve quality of life and eliminate health disparities demands the understanding, compassion, and will of the American people. There are opportunities for all health professions, individuals, and communities to work together to improve health. But more needs to be done if we are to make further improvements in America's oral health. We hope that this Surgeon General's report

will inform the American people about the opportunities to improve oral health and provide a platform from which the science base for craniofacial research can be expanded. The report should also serve to strengthen the translation of proven health promotion and disease prevention approaches into policy development, health care practice, and personal lifestyle behaviors. A framework for action that integrates oral health into overall health is critical if we are to see further gains.

David Satcher MD, PhD
Surgeon General

Acknowledgments

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Ruth L. Kirschstein MD

Acting Director, National Institutes of Health, DHHS,
Bethesda, MD

Harold C. Slavkin DDS

Director, National Institute of Dental and Craniofacial
Research, National Institutes of Health, DHHS,
Bethesda, MD

David Satcher MD, PhD

Assistant Secretary for Health and Surgeon General,
Office of Public Health and Science, Office of the
Secretary, Washington, DC

Nicole Lurie MD, MSPH

Principal Deputy Assistant Secretary for Health, Office
of Public Health and Science, Office of the Secretary,
Washington, DC

Beverly L. Malone PhD, RN, FAAN

Deputy Assistant Secretary for Health, Office of Public
Health and Science, Office of the Secretary,
Washington, DC

Arthur Lawrence PhD

Assistant Surgeon General, USPHS, Deputy Assistant
Secretary for Health (Operations), Office of Public
Health and Science, Office of the Secretary,
Washington, DC

Kenneth Moritsugu MD, MPH

Deputy Surgeon General, USPHS, Office of the
Surgeon General, Office of the Secretary,
Washington, DC

Allan S. Noonan MD, MPH

Captain, USPHS, Office of the Surgeon General,
Office of the Secretary, Washington, DC

PROJECT TEAM

Caswell A. Evans DDS, MPH

Project Director and Executive Editor
Assistant Director, Los Angeles County Department
of Health Services, Los Angeles, CA

Dushanka V. Kleinman DDS, MScD

Co-Executive Editor
Assistant Surgeon General, USPHS
Deputy Director, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Bethesda, MD

William R. Maas DDS, MPH, MS

Assistant Surgeon General, Chief Dental Officer,
USPHS
Director, Division of Oral Health, Centers for Disease
Control and Prevention, DHHS, Atlanta, GA

Joan S. Wilentz MA

Science Writer and Editor, Bethesda, MD

Roseanne Price ELS

Editor, Silver Spring, MD

Marla Fogelman

Editor, Silver Spring, MD

CONTRIBUTORS

Margo Adesanya DDS

Staff Scientist, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Bethesda, MD

Alfredo Aguirre DDS, MS

Director and Associate Professor, Advanced Oral and
Maxillofacial Pathology, State University of New York
at Buffalo School of Dental Medicine, Buffalo, NY

*Ronald Andersen PhD, MA

Wasserman Professor of Health Services and Professor
of Sociology, University of California Los Angeles
School of Public Health, Los Angeles, CA

*Indicates Coordinating Author

Acknowledgments

- Jay R. Anderson DMD, MHSA
Chief Dental Officer, Division of Community and Migrant Health, Bureau of Primary Health Care, Health Resources and Services Administration, DHHS, Rockville, MD
- Kenneth J. Anusavice PhD, DMD
Associate Dean for Research, Chair, Department of Dental Biomaterials, University of Florida College of Dentistry, Gainesville, FL
- *Kathryn A. Atchison DDS, MPH
Professor and Associate Dean for Research, University of California Los Angeles School of Dentistry, Los Angeles, CA
- James Bader DDS, MPH
Professor, University of North Carolina School of Dentistry, Chapel Hill, NC
- Charles Bertolami DDS, D.Med.Sc.
Dean, University of California San Francisco School of Dentistry, San Francisco, CA
- Aljernon Bolden DMD, MPH
Director, Community Health Programs and Co-Director, Division of Dental Public Health, Boston University Goldman School of Dental Medicine, Boston, MA
- L. Jackson Brown DDS, PhD
Associate Executive Director, American Dental Association, Chicago, IL
- Janet A. Brunelle MS
Statistician (retired), National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Brian A. Burt BDS, MPH, PhD
Professor of Dental Public Health, Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor, MI
- Nita Chainani-Wu DMD, MPH
University of California San Francisco School of Dentistry, San Francisco, CA
- David W. Chen MD, MPH
Commander, USPHS, Deputy Director, Division of Associated, Dental, and Public Health Professions, Bureau of Health Professions, Health Resources and Services Administration, DHHS, Rockville, MD
- Joseph Ciardi PhD
Consultant, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Betty DeBerry-Summer DDS, MPH
Captain, USPHS, Chief Dental Officer, Bureau of Primary Health Care, Health Resources and Services Administration, DHHS, Bethesda, MD
- Teresa A. Dolan DDS, MPH
Professor and Associate Dean, University of Florida College of Dentistry, Gainesville, FL
- *Chester W. Douglass DMD, PhD
Professor and Chair, Department of Oral Health Policy and Epidemiology, Harvard School of Dental Medicine, Boston, MA
- M. Ann Drum DDS, MPH
Captain, USPHS, Acting Director, Division of Research, Education and Training, Maternal and Child Health Bureau, Health Resources and Services Administration, DHHS, Rockville, MD
- Thomas F. Drury PhD
Senior Scientist, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Burton L. Edelstein DDS, MPH
Director, Children's Dental Health Project, American Academy of Pediatrics, Washington, DC
- Frederick C. Eichmiller DDS
Director, Paffenbarger Research Center, American Dental Association Health Foundation, National Institute of Standards and Technology, Gaithersburg, MD
- Mary P. Faine MS, RD, CD
Associate Professor and Director of Nutrition Education, University of Washington School of Dentistry, Seattle, WA
- Denise J. Fedele DMD, MS
Chief Professional Development and Research in Dental Care Clinical Research, Veterans Administration Medical Health Care System, Department of Veterans Affairs, Perrypoint, MD
- Christopher H. Fox DMD, DMSc
Director of Professional Relations, Europe, Colgate-Palmolive Co., New Jersey
- Philip C. Fox DDS
Director, Research and Development Affiliation, Amarillo Biosciences, Inc., Amarillo, TX
- Lawrence J. Furman DDS, MPH
Captain, USPHS, Head, Department of Health Services, U.S. Merchant Marine Academy, Kings Point, NY
- Isabel Garcia DDS, MPH
Captain, USPHS, Special Assistant for Science Transfer, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- *Robert J. Genco DDS, PhD
Distinguished Professor and Chair, Department of Oral Biology, State University of New York at Buffalo School of Dental Medicine, Buffalo, NY
- Gretchen Gibson DDS, MPH
Director, Special Care Dental Clinics, Veterans Administration North Texas Healthcare Systems, Department of Veterans Affairs, Dallas, TX

- Sharon M. Gordon DDS, MPH
 Director, Office of Education, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Catherine Hayes DMD, DMSc
 Assistant Professor, Department of Oral Health Policy and Epidemiology, Harvard School of Dental Medicine, Boston, MA
- Alice M. Horowitz PhD
 Senior Scientist, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Herschel Horowitz DDS, MPH
 Consultant, Dental Research and Public Health, Bethesda, MD
- Jeffrey Hyman DDS, PhD
 Epidemiologist, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Amid I. Ismail BDS, MPH, DrPH
 Professor, University of Michigan School of Dentistry, Ann Arbor, MI
- *Marjorie Jeffcoat DMD
 Rosen Professor and Chair, Department of Periodontics, University of Alabama School of Dentistry, Birmingham, AL
- Candace Jones RDH, MPH
 Captain, USPHS, Director, Dental Diseases Prevention Program, Indian Health Service, DHHS, Rockville, MD
- David Jones DDS, MPH
 Captain, USPHS (retired), Rockville, MD
- Judith A. Jones DDS, MPH
 Senior Research Associate, Veterans Administration Center for Health Quality, Outcomes and Economic Research, Department of Veterans Affairs and, Associate Professor, Boston University Goldman School of Dental Medicine, Bedford, MA
- Kaumudi J. Joshipura BDS, DSc
 Assistant Professor, Harvard School of Dental Medicine, Boston, MA
- Linda M. Kaste DDS, PhD
 Associate Professor and Division Director, Division of Dental Public Health and Oral Epidemiology, Medical University of South Carolina College of Dental Medicine, Charleston, SC
- William Kohn DDS, MS
 Captain, USPHS, Associate Director for Science, Division of Oral Health, Centers for Disease Control and Prevention, DHHS, Atlanta, GA
- Mary Sue Lancaster RN, MA
 Health Education Specialist, Centers for Disease Control and Prevention, DHHS, Atlanta, GA
- Linda LeResche ScD
 Research Professor, University of Washington School of Dentistry, Seattle, WA
- James Lipton DDS, PhD
 Captain, USPHS, Assistant Director for Training and Career Development, National Institute of Dental and Craniofacial Research, DHHS, Bethesda, MD
- Reginald Louie DDS, MPH
 Captain, USPHS, Regional Dental Consultant, Health Resources and Services Administration, DHHS, San Francisco, CA
- *Irwin D. Mandel DDS
 Professor Emeritus, Columbia University School of Dental and Oral Surgery, New York, NY
- Stephen E. Marcus PhD
 Epidemiologist, National Cancer Institute, National Institutes of Health, DHHS, Bethesda, MD
- Frank Martin DDS, MPH
 Captain, USPHS, Assistant Chief, Dental Services Branch (retired), Indian Health Service, DHHS, Rockville, MD
- Wendy E. Mouradian MD
 Director, Children's Hospital and Regional Medical Center, Departments of Pediatrics, Pediatric Dentistry, Medical History and Ethics, University of Washington, Seattle, WA
- *Linda Niessen DMD, MPH, MPP
 Professor, Baylor University College of Dentistry and Vice President, DENTSPLY International, York, PA
- Ruth E. Nowjack-Raymer RDH, MPH
 Public Health Researcher, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD
- Steven Offenbacher DDS, PhD, MMSc
 Professor and Director, Center for Oral and Systemic Diseases, University of North Carolina, Chapel Hill, NC
- H. Whitney Payne, Jr. DDS, MPH
 Captain, USPHS, Chief of Dental Services, Federal Correctional Institution, Federal Bureau of Prisons, Department of Justice, Seogoville, TX
- Edward Peters DMD
 Instructor in Oral Health and Epidemiology, Department of Oral Health Policy and Epidemiology, Harvard School of Dental Medicine, Boston, MA
- Douglas E. Peterson DMD, PhD
 Professor and Head, Department of Oral Diagnosis, University of Connecticut School of Dental Medicine, Farmington, CT
- Maryann Redford DDS, MPH
 Health Scientist Administrator, National Institute of Dental and Craniofacial Research, National Institutes of Health, DHHS, Bethesda, MD

Acknowledgments

- *Susan Reisine PhD
Professor, University of Connecticut School of Dental
Medicine, Hartford, CT
- John Rossetti DDS, MPH
Captain, USPHS, Chief Dental Officer, Health
Resources and Services Administration, DHHS,
Rockville, MD
- *R. Gary Rozier DDS, MPH
Professor, Department of Health Policy
Administration, University of North Carolina School
of Public Health, Chapel Hill, NC
- Susan Runner DDS, MA
Captain, USPHS, Branch Chief Dental Devices, Center
for Devices and Radiological Health, Food and Drug
Administration, Rockville, MD
- John S. Rutkauskas DDS, MS
Executive Director, American Association of Pediatric
Dentistry, Chicago, IL
- Margaret Scarlett DMD
Captain, USPHS, Office of the Secretary, Office of
Public Health and Science, DHHS, Washington, DC
- Don Schneider DDS, MPH
Captain, USPHS, Chief Dental Officer, Health Care
Financing Administration, Center for Medicaid and
State Operations, DHHS, Baltimore, MD
- Stephen T. Schultz DDS, MS, MPH
Lieutenant Commander, U.S. Navy, National Institute
of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Robert H. Selwitz DDS, MPH
Captain, USPHS, Senior Dental Epidemiologist,
National Institute of Dental and Craniofacial Research,
National Institutes of Health, DHHS, Bethesda, MD
- Gerald Shklar DDS, MS
Charles A. Brackett Professor of Oral Pathology,
Harvard School of Dental Medicine, Boston, MA
- Charles Shuler DMD, PhD
Director, Center for Craniofacial Molecular Biology,
George and Mary Lou Boone Professor of Craniofacial
Molecular Biology, University of Southern California
School of Dentistry, Los Angeles, CA
- Gary Slade BDS, DDPH, PhD
Assistant Professor, University of North Carolina
School of Dentistry, Chapel Hill, NC
- Ronald P. Strauss DMD, PhD
Professor, School of Dentistry and School of Medicine,
University of North Carolina, Chapel Hill, NC
- Lawrence Tabak DDS, PhD
Senior Associate Dean for Research, University of
Rochester School of Medicine and Dentistry,
Rochester, NY
- George W. Taylor DMD, DrPH
Assistant Professor of Dentistry, University of
Michigan School of Dentistry, Ann Arbor, MI
- Amardeep Singh Thind MD, PhD
Assistant Professor, University of California Los
Angeles School of Public Health, Los Angeles, CA
- Scott L. Tomar DMD, DrPH
Epidemiologist/Dental Officer, National Center for
Chronic Disease Prevention and Health Promotion,
Centers for Disease Control and Prevention, DHHS,
Atlanta, GA
- Benedict I. Truman MD, MPH
Senior Scientist and Epidemiology Program Officer,
Centers for Disease Control and Prevention, DHHS,
Atlanta, GA
- William Wathen DMD
Associate Dean, Center for Professional Development,
Baylor College of Dentistry, Dallas, TX
- *Jane A. Weintraub DDS, MPH
Lee Hysan Professor and Chair, Division of Oral
Epidemiology and Dental Public Health, University of
California San Francisco School of Dentistry, San
Francisco, CA
- *B. Alex White DDS, DrPH
Assistant Program Director, Economic, Social, and
Health Services Research, Kaiser Permanente Center
for Health Research, Portland, OR
- Deborah Winn PhD
Senior Investigator, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Bethesda, MD
- Athanasios I. Zavras DMD, MS, DMSc
Assistant Professor, Department of Health Policy and
Epidemiology, Harvard School of Dental Medicine,
Boston, MA

SENIOR REVIEWERS

- Clifton Dummett Sr. DDS
Distinguished Professor Emeritus, University
of Southern California School of Dentistry,
Los Angeles, CA
- Helen C. Rodriguez-Trias MD, FAAP
Past-President, American Public Health Association,
Brookdale, CA
- John Stamm DDS, DDPH, MScD
Dean, University of North Carolina School of
Dentistry, Chapel Hill, NC

REVIEWERS

- Michael C. Alfano DMD, PhD
Dean, New York University College of Dentistry,
New York, NY
- Myron Allukian Jr. DDS, MPH
Director, Oral Health, Boston Public Health
Commission, Boston, MA

- Alexia Antczak-Bouckoms DMD, ScD, MPH, MS
Assistant Professor of Medicine, Tufts University
School of Medicine, Medford, MA
- Howard L. Bailit DMD, PhD
Health Policy and Primary Care Research Center,
University of Connecticut Health Center,
Farmington, CT
- David Barmes BSc, DSc, MPH
Special Expert for International Health, National
Institute of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Bruce J. Baum DMD, PhD
Chief of Gene Therapy and Therapeutic Branch,
National Institute of Dental and Craniofacial Research,
National Institutes of Health, DHHS, Bethesda, MD
- Stephen C. Bayne MS, PhD
Professor, University of North Carolina School of
Dentistry, Chapel Hill, NC
- James D. Beck PhD
Kenan Professor, University of North Carolina School
of Dentistry, Chapel Hill, NC
- Eugenio Beltran DMD, MPH, MS, BrPH
Senior Research Fellow, Division of Oral Health,
Centers for Disease Control and Prevention, DHHS,
Atlanta, GA
- Henning Birkedal-Hansen DDS, PhD
Scientific Director, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Bethesda, MD
- Karina Boehm MPH
Chief, Health Promotion Branch, National Institute of
Dental and Craniofacial Research, National Institutes
of Health, DHHS, Bethesda, MD
- William H. Bowen BDS, PhD
Welscher Professor of Dentistry, School of Dentistry,
University of Rochester Medical Center, Rochester, NY
- Norman S. Braveman PhD
Associate Director for Clinical, Behavioral and Health
Promotion Research, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Bethesda, MD
- Bruce B. Brehm DMD, MPH
Lieutenant Colonel, U.S. Army, Tri-Service Center
for Oral Health Studies, Uniformed Services University
of the Health Sciences, Department of Defense,
Bethesda, MD
- Patricia S. Bryant PhD
Director, Behavioral and Health Promotion Research,
National Institute of Dental and Craniofacial Research,
National Institutes of Health, DHHS, Bethesda, MD
- Maria Teresa Canto MS, DDS, MPH
Public Health Research Specialist, National Institute of
Dental and Craniofacial Research, National Institutes
of Health, DHHS, Bethesda, MD
- Eli Capilouto DMD, MPH, ScD
Dean, School of Public Health, University of Alabama
at Birmingham, Birmingham, AL
- Victoria A. Cargill MD, MSCE
Medical Officer, Office of AIDS Research, National
Institutes of Health, DHHS, Bethesda, MD
- D. Walter Cohen DDS
Chancellor Emeritus, Medical College of Pennsylvania,
Hahneman University, Philadelphia, PA
- Lois K. Cohen PhD
Director, Office of International Health, National
Institute of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Robert J. Collins DMD, MPH
Deputy Executive Director, International and
American Associations for Dental Research,
Alexandria, VA
- Stephen B. Corbin DDS, MPH
Director of Health and Research Initiatives, Special
Olympics, Inc., Washington, DC
- James G. Corrigan PhD
Evaluation Officer, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Bethesda, MD
- James J. Crall DDS, ScD
Chairman, Department of Pediatric Dentistry,
University of Connecticut School of Dental Medicine,
Farmington, CT
- Ananda P. Dasanayake BDS, MPH, PhD
Associate Professor, Department of Oral Biology
University of Alabama School of Dentistry,
Birmingham, AL
- Dominick P. DePaola DDS, PhD
President and CEO, The Forsyth Institute, Boston, MA
- Richard D'Eustachio DDS
Private Practice, Cherry Hill, NJ
- Ray Dionne DDS, PhD
Captain, USPHS, Clinical Director, National Institute
of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Marylin Dodd RN, PhD
Professor, University of California San Francisco
School of Nursing, San Francisco, CA
- Scott Dubowsky DMD, FAGD
Private Practice, Bayonne, NJ
- Robert Dumbaugh DDS, MPH
Dental Executive Director, Palm Beach County Health
Department, Palm Beach, FL
- Joel Epstein DMD, MSD
Research Associate Professor, Department of Oral
Medicine, University of Washington School of
Dentistry, Seattle, WA

Acknowledgments

- Ronald Ettinger BDS, MDS, DDS
Professor, Department of Prosthodontics and Dows
Institute for Dental Research, University of Iowa,
Iowa City, IA
- Raymond Fonseca DMD
Dean, University of Pennsylvania School of Dental
Medicine, Philadelphia, PA
- Allan J. Formicola DDS
Dean, School of Dental and Oral Surgery, Columbia
University, New York, NY
- Jane L. Forrest Ed.D., RDH
Assistant Dean, Dental Hygiene Research and
Instructional Technology, Director, National Center for
Dental Hygiene Research, University of Southern
California School of Dentistry, Los Angeles, CA
- Robert T. Frame DMD, MS, CHE
Assistant Under Secretary for Health for Dentistry,
Department of Veterans Affairs, Washington, DC
- Raul I. Garcia DMD
Professor and Chair, Department of Health Policy,
Boston University Goldman School of Dental
Medicine, Boston, MA
- Jay Alan Gershen DDS, PhD
Executive Vice Chancellor, University of Colorado
Health Sciences Center, Denver, CO
- Michael Glick DMD
Professor of Oral Medicine, Director, Programs
for Medically Complex Patients, University of
Pennsylvania School of Dental Medicine,
Philadelphia, PA
- Barbara F. Gooch DMD, MPH
Dental Officer, Division of Oral Health, Centers for
Disease Control and Prevention, DHHS, Atlanta, GA
- John Greene DMD, MPH
Dean Emeritus, University of California San Francisco
School of Dentistry, San Rafael, CA
- Deborah Greenspan BDS, DSc, ScD (hc),
FDSRCS Ed (hon)
Professor of Clinical Oral Medicine, University of
California San Francisco School of Dentistry, San
Francisco, CA
- John S. Greenspan BSc, BDS, PhD, FRCPath
Professor and Chair, Department of Stomatology,
University of California San Francisco School of
Dentistry, San Francisco, CA
- Robert O. Greer DDS, ScD
Professor of Pathology and Medicine, University of
Colorado Health Sciences Center, Schools of Medicine
and Dentistry, Denver, CO
- David Grembowski PhD
Professor of Health Services and Dental Public Health
Sciences, University of Washington, Seattle, WA
- Ellen R. Gritz PhD
Professor and Chair, Department of Behavioral
Science, M.D. Anderson Cancer Center, University of
Texas, Houston, TX
- Kenneth A. Gruber PhD
Chief, Chronic Diseases Branch, National Institute of
Dental and Craniofacial Research, National Institutes
of Health, DHHS, Bethesda, MD
- Kevin Hardwick DDS, MPH
Captain, USPHS, International Health Officer, National
Institute of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Hazel J. Harper DDS, MPH
Private Practice, Washington, DC
- John Hauth MD
Professor, Interim Chairman and Director, Center for
Research in Women's Health, University of Alabama at
Birmingham, Birmingham, AL
- Maxine Hayes MD, MPH
Assistant Secretary, Community and Family Health,
Washington State Department of Health, Olympia, WA
- Marc W. Heft DMD, PhD
Professor and Director of the Claude Pepper
Center, University of Florida College of Dentistry,
Gainesville, FL
- Joseph Henry DDS, PhD, ScD
Dean Emeritus, Howard University College of
Dentistry and Professor, Harvard School of Dental
Medicine, Boston, MA
- Mark C. Herzberg DDS, PhD
Professor, University of Minnesota School of Dentistry,
Minneapolis, MN
- Alan R. Hinman MD, MPH
Senior Consultant for Public Health Programs,
Taskforce for Child Survival and Development,
Decatur, GA
- Cynthia Hodge DMD, MPH
Private Practice, Nashville, TN
- Dorthe Holst DDS, MPH
Professor, Department of Community Dentistry,
University of Oslo, Oslo, Norway
- John P. Howe III MD
President, University of Texas Health Science Center at
San Antonio, San Antonio, TX
- David Johnsen DDS, MS
Dean and Professor of Pediatric Dentistry, University
of Iowa College of Dentistry, Iowa City, IA
- Ralph Katz DMD, PhD
Professor, Department of Behavioral Science and
Community Health, University of Connecticut School
of Dental Medicine, Farmington, CT
- H. Asuman Kiyak MA, PhD
Director, Institute on Aging and Professor, University
of Washington School of Dentistry, Seattle, WA

- Kenneth S. Kornman DDS, PhD
Chief Scientific Officer, Interleukin Genetics, Inc.,
San Antonio, TX
- Eleni Kousvelari DDS, DSc
Health Scientist Administrator, National Institute of
Dental and Craniofacial Research, National Institutes
of Health, DHHS, Bethesda, MD
- Javanth V. Kumar DDS, MPH
Assistant Director, Bureau of Dental Health, New York
State Department of Health, Albany, NY
- Raymond Kuthy DDS, MPH
Professor and Chair, Preventive and Community
Dentistry, University of Iowa College of Dentistry,
Iowa City, IA
- Ira Lamster DDS, MMSc
Professor and Vice Dean, Columbia University School
of Dental and Oral Surgery, New York, NY
- Philip R. Lee MA, MD
Professor Emeritus of Social Medicine and Senior
Advisor, Institute for Health Policy Studies,
University of California San Francisco School of
Medicine, San Francisco, CA
- Racquel Z. LeGeros BS, MS, PhD
Professor, Department of Dental Materials Science
and Director, Research Center for Minority Oral
Health, New York University College of Dentistry,
New York, NY
- David Locker BDS, PhD
Professor, Faculty of Dentistry, University of Toronto,
Toronto, Canada
- Stuart Lockwood DDS, MPH
Dental Officer/Epidemiologist, Division of Oral Health,
Centers for Disease Control and Prevention, DHHS,
Atlanta, GA
- Harold Loe DDS
Director Emeritus, National Institute of Dental and
Craniofacial Research, National Institutes of Health,
DHHS, Florida and Norway
- G.M. Nana Lopez DDS, MPH
Dental Program Manager, City of Austin, Austin, TX
- Mark D. Macek DDS, PhD, MPH
Assistant Professor, Department of Oral Health Care
and Delivery, University of Maryland School of
Dentistry, Baltimore, MD
- Dolores M. Malvitz DrPH
Chief, Surveillance Investigations and Research
Branch, Division of Oral Health, Centers for Disease
Control and Prevention, DHHS, Atlanta, GA
- Dennis F. Mangan PhD
Chief, Infectious Disease and Immunity Branch,
National Institute of Dental and Craniofacial Research,
National Institutes of Health, DHHS, Bethesda, MD
- Kathleen Mangskau RDH, MPA
Director, North Dakota Oral Health Program, North
Dakota Department of Health, Bismarck, ND
- Georgetta Manning-Cox DDS, MPH
Chairman, Community Dentistry and Associate
Professor, Howard University College of Dentistry,
Washington, DC
- Don Marianos DDS, MPH
Consultant in Public Health, Pinetop, AZ
- Gary C. Martin DDS, MPH
Lieutenant Colonel, U.S. Air Force, Tri-Service Center
for Oral Health Studies, Uniformed Services University
of the Health Sciences, Department of Defense,
Bethesda, MD
- Ricardo J. Martinez MD, MPH
Director, Division of Extramural Research, National
Institute of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Richard Mascola DDS
Private Practice, Jericho, NY
- Carolyn Beth Mazzella RN
Assistant Surgeon General, USPHS, Office of the PHS
Chief Nurse, Health Resources and Services
Administration, DHHS, Rockville, MD
- Kim McFarland DDS, MS
Dental Health Director, State of Nebraska, Nebraska
Department of Health, Lincoln, NE
- J. Michael McGinnis MD
Senior Vice President and Director, Health Group, The
Robert Wood Johnson Foundation, Princeton, NJ
- Robert Mecklenburg DDS, MPH
Coordinator, Tobacco and Oral Health Initiatives for
the Tobacco Control Research Branch, National Cancer
Institute, DHHS, Bethesda, MD
- Roseann Mulligan DDS, MS
Professor and Chairman, Department of Dental
Medicine and Public Health, University of Southern
California School of Dentistry, Los Angeles, CA
- Juan M. Navia PhD
Professor Emeritus, University of Alabama at
Birmingham, Birmingham, AL
- Edward O'Neil PhD, MPA
Professor, Dental Public Health and Hygiene, Director,
Center for Health Professions, University of California,
San Francisco, San Francisco, CA
- Roy C. Page DDS, PhD
Professor, Departments of Periodontics and Pathology,
Schools of Dentistry and Medicine, University of
Washington, Seattle, WA
- No-Hee Park DMD, PhD
Dean, School of Dentistry, University of California
Los Angeles, Los Angeles, CA

Acknowledgments

- Steven Perlman DDS, MScD
Associate Professor of Pediatric Dentistry, Boston
University Goldman School of Dental Medicine,
Boston, MA
- Poul Erik Petersen DDS, Dr Odont Sci, BA, MSc
Professor, University of Copenhagen, School of
Dentistry, Copenhagen N, Denmark
- Kathy Phipps RDH, DrPH
Associate Professor, Oregon Health Sciences
University, Newport, OR
- Scott Presson DDS, MPH
Captain, USPHS, Chief, Program Services Branch,
Division of Oral Health, Centers for Disease Control
and Prevention, DHHS, Atlanta, GA
- Francisco Ramos-Gomez DDS, MS, MPH
Associate Professor, Department of Growth and
Development, University of California San Francisco,
San Francisco, CA
- E. Diane Rekow DDS, PhD
Professor and Chair, Department of Orthodontics,
University of Medicine and Dentistry of New Jersey,
Newark, NJ
- Michael Rethman DDS, MS
Colonel, U.S. Army, Chief of Periodontics, Tripler
Dental Clinic, Honolulu, HI
- S. Timothy Rose DDS, MS
Private Practice, Appleton, WI
- Bruce Rothwell DMD, MSD
Associate Professor and Chairman, University
of Washington, Restorative/Hospital Dentistry,
Seattle, WA
- Shirley B. Russell PhD
Professor and Chairperson, Department of
Microbiology, Associate Dean for Research, Meharry
Medical College, Nashville, TN
- Ann L Sandberg PhD
Chief, Neoplastic Diseases Branch, National Institute
of Dental and Craniofacial Research, National
Institutes of Health, DHHS, Bethesda, MD
- Charles Sanders DDS
Dean, Howard University College of Dentistry,
Washington, DC
- Michèle J. Saunders DMD, MS, MPH
Endowed Professor of Clinical Dentistry, University of
Texas Health Science Center, San Antonio, TX
- Crispian Scully MD, PhD, MDS
Dean, Director of Studies and Research, International
Centers for Excellence in Dentistry and Eastman
Dental Institute for Oral Health Care Sciences,
University College of London, London, England
- Leslie W. Seldin DDS, PC
Private Practice, New York, NY
- Aubrey Sheiham BDS, PhD, DHC
Professor, Department of Epidemiology and Public
Health, University College London Medical School,
London, England
- Cynthia Sherwood DDS
Private Practice, Independence, KS
- Mark D. Siegal DDS, MPH
Chief, Bureau of Oral Health Services, Ohio
Department of Health, Columbus, OH
- Sol Silverman Jr. MA, DDS
Professor of Oral Medicine, University of California
San Francisco School of Dentistry, San Francisco, CA
- Susan F. Silverton MD, PhD
Assistant Professor and Enid Neidle Scholar,
University of Pennsylvania School of Dental Medicine,
Philadelphia, PA
- Jeanne Sinkford DDS, MS, PhD
Associate Executive Director, American Dental
Education Association, Washington, DC
- Judy A. Small PhD
Chief, Craniofacial Anomalies and Injuries Branch,
National Institute of Dental and Craniofacial Research,
National Institutes of Health, DHHS, Bethesda, MD
- Christian Stohler DMD, Dr.Med.Dent
William R. Mann Professor and Chair, Department of
Biologic and Materials Sciences, University of
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- George Stookey PhD
Executive Associate Dean, Indiana University School
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Minneapolis, MN
- Richard Valachovic DMD, MPH
Executive Director, American Dental Education
Association, Washington, DC
- Clemencia M. Vargas DDS, MPH, PhD
Assistant Professor, University of Maryland School of
Dentistry, Baltimore, MD
- Rueben Warren DDS, MPH, DrPH
Associate Administrator for Urban Affairs, The Agency
for Toxic Substances and Disease Registry, DHHS,
Atlanta, GA
- Reginald Wells PhD
Deputy Commissioner, Administration for Children
and Families, Administration on Developmental
Disabilities, DHHS, Washington, DC
- Terrie Wetle PhD
Deputy Director, National Institute on Aging, National
Institutes of Health, DHHS, Bethesda, MD
- David A. Whiston DDS
Private Practice, Falls Church, VA

Ardell Wilson DDS, MPH
 Bureau Chief, State of Connecticut Department of
 Public Health, Avon, CT
 Kenneth M. Yamada MD, PhD
 Branch Chief, Craniofacial Development Biology and
 Regeneration, National Institute of Dental and
 Craniofacial Research, National Institutes of Health,
 DHHS, Bethesda, MD

**EDITORIAL AND PRODUCTION
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 William Kohn DDS
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 Division of Oral Health, Centers for Disease Control
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 Commander, U.S. Navy, Assistant Professor, Tri-Service
 Center for Oral Health Studies, Uniformed Services
 University of the Health Sciences, Department of
 Defense, Bethesda, MD
 Lawrence McKinley DDS
 Captain, U.S. Navy, Senior Consultant for Dentistry,
 Tricare Management Activity, Department of Defense,
 Falls Church, VA
 Edward Sondik PhD
 Director, National Center for Health Statistics,
 Centers for Disease Control and Prevention, DHHS,
 Hyattsville, MD

Acknowledgments

Sue Swenson

Commissioner, Administration on Developmental
Disabilities, Administration for Children and Families,
DHHS, Washington, DC

Jeanette Takamura

Assistant Secretary for Aging, Administration on
Aging, DHHS, Washington, DC

Ron J. Vogel

Acting Deputy Administrator of the Food and
Nutrition Programs, Food and Nutrition Service,
Department of Agriculture, Alexandria, VA

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Executive Summary

Publication of this first Surgeon General's Report on Oral Health marks a milestone in the history of oral health in America. The report elaborates on the meaning of oral health and explains why oral health is essential to general health and well-being. In the course of the past 50 years, great progress has been made in understanding the common oral diseases—dental caries (tooth decay) and periodontal (gum) diseases—resulting in marked improvements in the nation's oral health. Most middle-aged and younger Americans expect to retain their natural teeth over their lifetime and do not expect to have any serious oral health problems.

The major message of this Surgeon General's report is that oral health is essential to the general health and well-being of all Americans *and* can be achieved by all Americans. However, *not all* Americans are achieving the same degree of oral health. In spite of the safe and effective means of maintaining oral health that have benefited the majority of Americans over the past half century, many among us still experience needless pain and suffering, complications that devastate overall health and well-being, and financial and social costs that diminish the quality of life and burden American society. What amounts to “a silent epidemic” of oral diseases is affecting our most vulnerable citizens—poor children, the elderly, and many members of racial and ethnic minority groups (GAO 2000). (See box entitled “The Burden of Oral Diseases and Disorders.”)

The word *oral* refers to the mouth. The mouth includes not only the teeth and the gums (gingiva) and their supporting tissues, but also the hard and soft palate, the mucosal lining of the mouth and throat, the tongue, the lips, the salivary glands, the chewing muscles, and the upper and lower jaws. Equally important are the branches of the nervous, immune, and vascular systems that animate, protect,

and nourish the oral tissues, as well as provide connections to the brain and the rest of the body. The genetic patterning of development in utero further reveals the intimate relationship of the oral tissues to the developing brain and to the tissues of the face and head that surround the mouth, structures whose location is captured in the word *craniofacial*.

A major theme of this report is that **oral health means much more than healthy teeth**. It means being free of chronic oral-facial pain conditions, oral and pharyngeal (throat) cancers, oral soft tissue lesions, birth defects such as cleft lip and palate, and scores of other diseases and disorders that affect the oral, dental, and craniofacial tissues, collectively known as the *craniofacial complex*. These are tissues whose functions we often take for granted, yet they represent the very essence of our humanity. They allow us to speak and smile; sigh and kiss; smell, taste, touch, chew, and swallow; cry out in pain; and convey a world of feelings and emotions through facial expressions. They also provide protection against microbial infections and environmental insults.

The craniofacial tissues also provide a useful means to understanding organs and systems in less accessible parts of the body. The salivary glands are a model of other exocrine glands, and an analysis of saliva can provide telltale clues of overall health or disease. The jawbones and their joints function like other musculoskeletal parts. The nervous system apparatus underlying facial pain has its counterpart in nerves elsewhere in the body. A thorough oral examination can detect signs of nutritional deficiencies as well as a number of systemic diseases, including microbial infections, immune disorders, injuries, and some cancers. Indeed, the phrase *the mouth is a mirror* has been used to illustrate the wealth of information that can be derived from examining oral tissues.

New research is pointing to associations between chronic oral infections and heart and lung diseases, stroke, and low-birth-weight, premature births. Associations between periodontal disease and diabetes have long been noted. This report assesses these associations and explores mechanisms that might explain the oral-systemic disease connections.

The broadened meaning of *oral health* parallels the broadened meaning of *health*. In 1948 the World Health Organization expanded the definition of health to mean “a complete state of physical, mental, and social well-being, and not just the absence of infirmity.” It follows that oral health must also include well-being. Just as we now understand that nature and nurture are inextricably linked, and mind and body are both expressions of our human biology, so, too, we must recognize that oral health and general health are inseparable. We ignore signs and symptoms of oral disease and dysfunction to our

detriment. Consequently, a second theme of the report is that **oral health is integral to general health**. You cannot be healthy without oral health. Oral health and general health should not be interpreted as separate entities. Oral health is a critical component of health and must be included in the provision of health care and the design of community programs.

The wider meanings of *oral* and *health* in no way diminish the relevance and importance of the two leading dental diseases, caries and the periodontal diseases. They remain common and widespread, affecting nearly everyone at some point in the life span. What has changed is what we can do about them.

Researchers in the 1930s discovered that people living in communities with naturally fluoridated water supplies had less dental caries than people drinking unfluoridated water. But not until the end

The Burden of Oral Diseases and Disorders

Oral diseases are progressive and cumulative and become more complex over time. They can affect our ability to eat, the foods we choose, how we look, and the way we communicate. These diseases can affect economic productivity and compromise our ability to work at home, at school, or on the job. Health disparities exist across population groups at all ages. Over one third of the U.S. population (100 million people) has no access to community water fluoridation. Over 108 million children and adults lack dental insurance, which is over 2.5 times the number who lack medical insurance. The following are highlights of oral health data for children, adults, and the elderly. (Refer to the full report for details of these data and their sources.)

Children

- Cleft lip/palate, one of the most common birth defects, is estimated to affect 1 out of 600 live births for whites and 1 out of 1,850 live births for African Americans.
- Other birth defects such as hereditary ectodermal dysplasias, where all or most teeth are missing or misshapen, cause lifetime problems that can be devastating to children and adults.
- Dental caries (tooth decay) is the single most common chronic childhood disease—5 times more common than asthma and 7 times more common than hay fever.
- Over 50 percent of 5- to 9-year-old children have at least one cavity or filling, and that proportion increases to 78 percent among 17-year-olds. Nevertheless, these figures represent improvements in the oral health of children compared to a generation ago.
- There are striking disparities in dental disease by income. Poor children suffer twice as much dental caries as their more affluent peers, and their disease is more likely to be untreated. These poor-nonpoor

differences continue into adolescence. One out of four children in America is born into poverty, and children living below the poverty line (annual income of \$17,000 for a family of four) have more severe and untreated decay.

- Unintentional injuries, many of which include head, mouth, and neck injuries, are common in children.
- Intentional injuries commonly affect the craniofacial tissues.
- Tobacco-related oral lesions are prevalent in adolescents who currently use smokeless (spit) tobacco.
- Professional care is necessary for maintaining oral health, yet 25 percent of poor children have not seen a dentist before entering kindergarten.
- Medical insurance is a strong predictor of access to dental care. Uninsured children are 2.5 times less likely than insured children to receive dental care. Children from families without dental insurance are 3 times more likely to have dental needs than children with either public or private insurance. For each child without medical insurance, there are at least 2.6 children without dental insurance.
- Medicaid has not been able to fill the gap in providing dental care to poor children. Fewer than one in five Medicaid-covered children received a single dental visit in a recent year-long study period. Although new programs such as the State Children’s Health Insurance Program (SCHIP) may increase the number of insured children, many will still be left without effective dental coverage.
- The social impact of oral diseases in children is substantial. More than 51 million school hours are lost each year to dental-related illness. Poor children suffer nearly 12 times more restricted-activity days than children from higher-income families. Pain and suffering due to untreated diseases can lead to problems in eating, speaking, and attending to learning.

of World War II were the investigators able to design and implement the community clinical trials that confirmed their observations and launched a better approach to the problem of dental caries: prevention. Soon after, adjusting the fluoride content of community water supplies was pursued as an important public health measure to prevent dental caries.

Although this measure has not been fully implemented, the results have been dramatic. Dental caries began to decline in the 1950s among children who grew up in fluoridated cities, and by the late 1970s, decline in decay was evident for many Americans. The application of science to improve diagnostic, treatment, and prevention strategies has saved billions of dollars per year in the nation's annual health bill. Even more significant, the result is that far fewer people are edentulous (toothless) today than a generation ago.

Adults

- Most adults show signs of periodontal or gingival diseases. Severe periodontal disease (measured as 6 millimeters of periodontal attachment loss) affects about 14 percent of adults aged 45 to 54.
- Clinical symptoms of viral infections, such as herpes labialis (cold sores), and oral ulcers (canker sores) are common in adulthood, affecting about 19 percent of adults 25 to 44 years of age.
- Chronic disabling diseases such as temporomandibular disorders, Sjögren's syndrome, diabetes, and osteoporosis affect millions of Americans and compromise oral health and functioning.
- Pain is a common symptom of craniofacial disorders and is accompanied by interference with vital functions such as eating, swallowing, and speech. Twenty-two percent of adults reported some form of oral-facial pain in the past 6 months. Pain is a major component of trigeminal neuralgia, facial shingles (post-herpetic neuralgia), temporomandibular disorders, fibromyalgia, and Bell's palsy.
- Population growth as well as diagnostics that are enabling earlier detection of cancer means that more patients than ever before are undergoing cancer treatments. More than 400,000 of these patients will develop oral complications annually.
- Immunocompromised patients, such as those with HIV infection and those undergoing organ transplantation, are at higher risk for oral problems such as candidiasis.
- Employed adults lose more than 164 million hours of work each year due to dental disease or dental visits.
- For every adult 19 years or older without medical insurance, there are three without dental insurance.
- A little less than two thirds of adults report having visited a dentist in the past 12 months. Those with incomes at or above the poverty level are twice as likely to report a dental visit in the past 12 months as those who are below the poverty level.

The theme of prevention gained momentum as pioneering investigators and practitioners in the 1950s and 1960s showed that not only dental caries but also periodontal diseases are bacterial infections. The researchers demonstrated that the infections could be prevented by increasing host resistance to disease and reducing or eliminating the suspected microbial pathogens in the oral cavity. The applications of research discoveries have resulted in continuing improvements in the oral health of Americans, new approaches to the prevention and treatment of dental diseases, and the growth of the science.

The significant role that scientists, dentists, dental hygienists, and other health professionals have played in the prevention of oral disease and disability leads to a third theme of this report: **safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease.** These measures include daily oral hygiene

Older Adults

- Twenty-three percent of 65- to 74-year-olds have severe periodontal disease (measured as 6 millimeters of periodontal attachment loss). (Also, at all ages men are more likely than women to have more severe disease, and at all ages people at the lowest socioeconomic levels have more severe periodontal disease.)
- About 30 percent of adults 65 years and older are edentulous, compared to 46 percent 20 years ago. These figures are higher for those living in poverty.
- Oral and pharyngeal cancers are diagnosed in about 30,000 Americans annually; 8,000 die from these diseases each year. These cancers are primarily diagnosed in the elderly. Prognosis is poor. The 5-year survival rate for white patients is 56 percent; for blacks, it is only 34 percent.
- Most older Americans take both prescription and over-the-counter drugs. In all probability, at least one of the medications used will have an oral side effect—usually dry mouth. The inhibition of salivary flow increases the risk for oral disease because saliva contains antimicrobial components as well as minerals that can help rebuild tooth enamel after attack by acid-producing, decay-causing bacteria. Individuals in long-term care facilities are prescribed an average of eight drugs.
- At any given time, 5 percent of Americans aged 65 and older (currently some 1.65 million people) are living in a long-term care facility where dental care is problematic.
- Many elderly individuals lose their dental insurance when they retire. The situation may be worse for older women, who generally have lower incomes and may never have had dental insurance. Medicaid funds dental care for the low-income and disabled elderly in some states, but reimbursements are low. Medicare is not designed to reimburse for routine dental care.

procedures and other lifestyle behaviors, community programs such as community water fluoridation and tobacco cessation programs, and provider-based interventions such as the placement of dental sealants and examinations for common oral and pharyngeal cancers. It is hoped that this Surgeon General's report will facilitate the maturing of the broad field of craniofacial research so that gains in the prevention of craniofacial diseases and disorders can be realized that are as impressive as those achieved for common dental diseases.

At the same time, more needs to be done to ensure that messages of health promotion and disease prevention are brought home to all Americans. In this regard, a fourth theme of the report is that **general health risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health.** The evidence for an association between tobacco use and oral diseases has been clearly delineated in almost every Surgeon General's report on tobacco since 1964, and the oral effects of nutrition and diet are presented in the Surgeon General's report on nutrition (1988). All the health professions can play a role in reducing the burden of disease in America by calling attention to these and other risk factors and suggesting appropriate actions.

Clearly, promoting health and preventing diseases are concepts the American people have taken to heart. For the third decade the nation has developed a plan for the prevention of disease and the promotion of health, embodied in the U.S. Department of Health and Human Services (2000) document, *Healthy People 2010*. As a nation, we hope to eliminate disparities in health and prevent oral diseases, cancer, birth defects, AIDS and other devastating infections, mental illness and suicide, and the chronic diseases of aging. To live well into old age free of pain and infirmity, and with a high quality of life, is the American dream.

Scientists today take that dream seriously in researching the intricacies of the craniofacial complex. They are using an ever-growing array of sophisticated analytic tools and imaging systems to study normal function and diagnose disease. They are completing the mapping and sequencing of human, animal, microbial, and plant genomes, the better to understand the complexities of human development, aging, and pathological processes. They are growing cell lines, synthesizing molecules, and using a new generation of biomaterials to revolutionize tissue repair and regeneration. More than ever before, they are working in multidisciplinary teams to bring new knowledge and expertise to the goal of understanding complex human diseases and disorders.

THE CHALLENGE

This Surgeon General's report has much to say about the inequities and disparities that affect those least able to muster the resources to achieve optimal oral health. The barriers to oral health include lack of access to care, whether because of limited income or lack of insurance, transportation, or the flexibility to take time off from work to attend to personal or family needs for care. Individuals with disabilities and those with complex health problems may face additional barriers to care. Sometimes, too, the public, policymakers, and providers may consider oral health and the need for care to be less important than other health needs, pointing to the need to raise awareness and improve health literacy.

Even more costly to the individual and to society are the expenses associated with oral health problems that go beyond dental diseases. The nation's yearly dental bill is expected to exceed \$60 billion in 2000 (Health Care Financing Administration 2000). However, add to that expense the tens of billions of dollars in direct medical care and indirect costs of chronic craniofacial pain conditions such as temporomandibular disorders, trigeminal neuralgia, shingles, or burning mouth syndrome; the \$100,000 minimum individual lifetime costs of treating craniofacial birth defects such as cleft lip and palate; the costs of oral and pharyngeal cancers; the costs of autoimmune diseases; and the costs associated with the unintentional and intentional injuries that so often affect the head and face. Then add the social and psychological consequences and costs. Damage to the craniofacial complex, whether from disease, disorder, or injury, strikes at our very identity. We see ourselves, and others see us, in terms of the face we present to the world. Diminish that image in any way and we risk the loss of self-esteem and well-being.

Many unanswered questions remain for scientists, practitioners, educators, policymakers, and the public. This report highlights the research challenges as well as pointing to emerging technologies that may facilitate finding solutions. Along with the quest for answers comes the challenge of applying what is already known in a society where there are social, political, economic, behavioral, and environmental barriers to health and well-being.

THE CHARGE

The realization that oral health can have a significant impact on the overall health and well-being of the nation's population led the Office of the Surgeon General, with the approval of the Secretary of Health and Human Services, to commission this report.

Recognizing the gains that have been made in disease prevention while acknowledging that there are populations that suffer disproportionately from oral health problems, the Secretary asked that the report “define, describe, and evaluate the interaction between oral health and health and well-being [quality of life], through the life span in the context of changes in society.” Key elements to be addressed were the determinants of health and disease, with a primary focus on prevention and “producing health” rather than “restoring health”; a description of the burden of oral diseases and disorders in the nation; and the evidence for actions to improve oral health to be taken across the life span. The report also was to feature an orientation to the future, highlighting leading-edge technologies and research findings that can be brought to bear in improving the oral health of individuals and communities.

THE SCIENCE BASE FOR THE REPORT

This report is based on a review of the published scientific literature. Standards established to determine the quality of the evidence, based on the study design and its rigor, were used where appropriate. In addition, the strength of the recommendations, where they are made, is based on evidence of effectiveness for the population of interest. The scope of the review encompassed the international English literature. Recent systematic reviews of the literature are referenced, as are selected review articles. A few referenced articles are in press, and there are occasional references to recent abstracts and personal communications.

The science base in oral health has been evolving over the past half century. Initial research in this area was primarily in the basic sciences, investigating mechanisms of normal development and pathology in relation to dental caries and periodontal diseases. Prevention research has included controlled clinical studies, with and without randomization, as well as community trials and demonstration research. More recent research has broadened the science base to include studies of the range of craniofacial diseases and disorders and is moving from basic science to translational, clinical, and health services research.

The clinical literature includes the full range of studies, from randomized controlled studies to case studies. Most of the literature includes cross-sectional and cohort studies, with some case-control studies. General reviews of the literature have been used for Chapters 2 through 10. Chapter 4 includes both published and new analyses of national and state databases that have been carefully designed and for which

quality assurance has been maintained by the Centers for Disease Control and Prevention. Studies of smaller populations are also included where relevant. In Chapter 5, tables present information on the association of oral infections and systemic conditions, and in Chapter 7, tables exhibit oral disease prevention and health promotion measures. The published literature related to the development of new technologies, their potential impact, and the need for further research are described in the course of addressing the requested futures orientation.

The report was generated with the advice and support of a Federal Coordinating Committee composed of representatives of agencies with oral health components and interests. The chapters were based on papers submitted by experts working under the guidance of a coordinating author for each chapter. Independent peer review was conducted for all sections of the report at various stages in the process, and the full manuscript was reviewed by a number of senior reviewers as well as the relevant federal agencies. All who contributed are listed in the Acknowledgments section of the full report.

ORGANIZATION OF THE REPORT

The report centers on five major questions, which have been used to structure the report into five parts.

Part One: What Is Oral Health?

The meaning of oral health is explored in Chapter 1, and the interdependence of oral health with general health and well-being is a recurrent theme throughout the volume.

Chapter 2 provides an overview of the craniofacial complex in development and aging, how the tissues and organs function in essential life processes, and their role in determining our uniquely human abilities. Our craniofacial complex has evolved to have remarkable functions and abilities to adapt, enabling us to meet the challenges of an ever-changing environment. An examination of the various tissues reveals elaborate designs that serve complex needs and functions, including the uniquely human function of speech. The rich distribution of nerves, muscles, and blood vessels in the region as well as extensive endocrine and immune system connections are indicators of the vital role of the craniofacial complex in adaptation and survival over a long life span. In particular, the following findings are noted:

- Genes controlling the basic patterning and segmental organization of human development, and specifically the craniofacial complex, are highly

conserved in nature. Mutated genes affecting human development have counterparts in many simpler organisms.

- There is considerable reserve capacity or redundancy in the cells and tissues of the craniofacial complex, so that if they are properly cared for, the structures should function well over a lifetime.

- The salivary glands and saliva subserve tasting and digestive functions and also participate in the mucosal immune system, a main line of defense against pathogens, irritants, and toxins.

- Salivary components protect and maintain oral tissues through antimicrobial components, buffering agents, and a process by which dental enamel can be remineralized.

Part Two: What Is the Status of Oral Health in America?

Chapter 3 is a primer describing the major diseases and disorders that affect the craniofacial complex. The findings include:

- Microbial infections, including those caused by bacteria, viruses, and fungi, are the primary cause of the most prevalent oral diseases. Examples include dental caries, periodontal diseases, herpes labialis, and candidiasis.

- The etiology and pathogenesis of diseases and disorders affecting the craniofacial structures are multifactorial and complex, involving an interplay among genetic, environmental, and behavioral factors.

- Many inherited and congenital conditions affect the craniofacial complex, often resulting in disfigurement and impairments that may involve many body organs and systems and affect millions of children worldwide.

- Tobacco use, excessive alcohol use, and inappropriate dietary practices contribute to many diseases and disorders. In particular, tobacco use is a risk factor for oral cavity and pharyngeal cancers, periodontal diseases, candidiasis, and dental caries, among other diseases.

- Some chronic diseases, such as Sjögren's syndrome, present with primary oral symptoms.

- Oral-facial pain conditions are common and often have complex etiologies.

Chapter 4 constitutes an oral health status report card for the United States, describing the magnitude of the problem. Where data permit, the chapter also describes the oral health of selected population

groups, as well as their dental visit behavior. The findings include:

- Over the past five decades, major improvements in oral health have been seen nationally for most Americans.

- Despite improvements in oral health status, profound disparities remain in some population groups as classified by sex, income, age, and race/ethnicity. For some diseases and conditions, the magnitude of the differences in oral health status among population groups is striking.

- Oral diseases and conditions affect people throughout their life span. Nearly every American has experienced the most common oral disease, dental caries.

- Conditions that severely affect the face and facial expression, such as birth defects, craniofacial injuries, and neoplastic diseases, are more common in the very young and in the elderly.

- Oral-facial pain can greatly reduce quality of life and restrict major functions. Pain is a common symptom for many of the conditions affecting oral-facial structures.

- National and state data for many oral and craniofacial diseases and conditions and for population groups are limited or nonexistent. Available state data reveal variations within and among states in patterns of health and disease among population groups.

- Research is needed to develop better measures of disease and health, to explain the differences among population groups, and to develop interventions targeted at eliminating disparities.

Part Three: What Is the Relationship Between Oral Health and General Health and Well-being?

Chapters 5 and 6 address key issues in the report's charge—the relationship of oral health to general health and well-being. Chapter 5 explores the theme of the mouth as reflecting general health or disease status. Examples are given of how oral tissues may signal the presence of disease, disease progression, or exposure to risk factors, and how oral cells and fluids are increasingly being used as diagnostic tools. This is followed by a discussion of the mouth as a portal of entry for infections that can affect local tissues and may spread to other parts of the body. The final sections review the literature regarding emerging associations between oral diseases and diabetes, heart disease and stroke, and adverse pregnancy outcomes.

The findings include:

- Many systemic diseases and conditions have oral manifestations. These manifestations may be the initial sign of clinical disease and as such serve to inform clinicians and individuals of the need for further assessment.

- The oral cavity is a portal of entry as well as the site of disease for microbial infections that affect general health status.

- The oral cavity and its functions can be adversely affected by many pharmaceuticals and other therapies commonly used in treating systemic conditions. The oral complications of these therapies can compromise patient compliance with treatment.

- Individuals such as immunocompromised and hospitalized patients are at greater risk for general morbidity due to oral infections.

- Individuals with diabetes are at greater risk for periodontal diseases.

- Animal and population-based studies have demonstrated an association between periodontal diseases and diabetes, cardiovascular disease, stroke, and adverse pregnancy outcomes. Further research is needed to determine the extent to which these associations are causal or coincidental.

Chapter 6 demonstrates the relationship between oral health and quality of life, presenting data on the consequences of poor oral health and altered appearance on speech, eating, and other functions, as well as on self-esteem, social interaction, education, career achievement, and emotional state. The chapter introduces anthropological and ethnographic literature to underscore the cultural values and symbolism attached to facial appearance and teeth. An examination of efforts to characterize the functional and social implications of oral and craniofacial diseases reveals the following findings:

- Oral health is related to well-being and quality of life as measured along functional, psychosocial, and economic dimensions. Diet, nutrition, sleep, psychological status, social interaction, school, and work are affected by impaired oral and craniofacial health.

- Cultural values influence oral and craniofacial health and well-being and can play an important role in care utilization practices and in perpetuating acceptable oral health and facial norms.

- Oral and craniofacial diseases and their treatment place a burden on society in the form of lost days and years of productive work. Acute dental conditions contribute to a range of problems for employed adults, including restricted activity, bed days, and work loss, and school loss for children. In addition, conditions such as oral and pharyngeal

cancers contribute to premature death and can be measured by years of life lost.

- Oral and craniofacial diseases and conditions contribute to compromised ability to bite, chew, and swallow foods; limitations in food selection; and poor nutrition. These conditions include tooth loss, diminished salivary functions, oral-facial pain conditions such as temporomandibular disorders, alterations in taste, and functional limitations of prosthetic replacements.

- Oral-facial pain, as a symptom of untreated dental and oral problems and as a condition in and of itself, is a major source of diminished quality of life. It is associated with sleep deprivation, depression, and multiple adverse psychosocial outcomes.

- Self-reported impacts of oral conditions on social function include limitations in verbal and non-verbal communication, social interaction, and intimacy. Individuals with facial disfigurements due to craniofacial diseases and conditions and their treatments may experience loss of self-image and self-esteem, anxiety, depression, and social stigma; these in turn may limit educational, career, and marital opportunities and affect other social relations.

- Reduced oral-health-related quality of life is associated with poor clinical status and reduced access to care.

Part Four: How Is Oral Health Promoted and Maintained and How Are Oral Diseases Prevented?

The next three chapters review how individuals, health care practitioners, communities, and the nation as a whole contribute to oral health. Chapter 7 reviews the evidence for the efficacy and effectiveness of health promotion and disease prevention measures with a focus on community efforts in preventing oral disease. It continues with a discussion of the knowledge and practices of the public and health care providers and indicates opportunities for broad-based and targeted health promotion. The findings include:

- Community water fluoridation, an effective, safe, and ideal public health measure, benefits individuals of all ages and socioeconomic strata. Unfortunately, over one third of the U.S. population (100 million people) are without this critical public health measure.

- Effective disease prevention measures exist for use by individuals, practitioners, and communities. Most of these focus on dental caries prevention, such as fluorides and dental sealants, where a combination of services is required to

achieve optimal disease prevention. Daily oral hygiene practices such as brushing and flossing can prevent gingivitis.

- Community-based approaches for the prevention of other oral diseases and conditions, such as oral and pharyngeal cancers and oral-facial trauma, require intensified developmental efforts.

- Community-based preventive programs are unavailable to substantial portions of the underserved population.

- There is a gap between research findings and the oral disease prevention and health promotion practices and knowledge of the public and the health professions.

- Disease prevention and health promotion approaches, such as tobacco control, appropriate use of fluorides for caries prevention, and folate supplementation for neural tube defect prevention, highlight opportunities for partnerships between community-based programs and practitioners, as well as collaborations among health professionals.

- Many community-based programs require a combined effort among social service, health care, and education services at the local or state level.

Chapter 8 explores the role of the individual and the health care provider in promoting and maintaining oral health and well-being. For the individual, this means exercising appropriate self-care and adopting healthy behaviors. For the provider, it means incorporating the knowledge emerging from the science base in a timely manner for prevention and diagnosis, risk assessment and risk management, and treatment of oral diseases and disorders. The chapter focuses largely on the oral health care provider. The management of oral and craniofacial health and disease necessitates collaborations among a team of care providers to achieve optimal oral and general health. The findings include:

- Achieving and maintaining oral health require individual action, complemented by professional care as well as community-based activities.

- Individuals can take actions, for themselves and for persons under their care, to prevent disease and maintain health. Primary prevention of many oral, dental, and craniofacial diseases and conditions is possible with appropriate diet, nutrition, oral hygiene, and health-promoting behaviors, including the appropriate use of professional services. Individuals should use a fluoride dentifrice daily to help prevent dental caries and should brush and floss daily to prevent gingivitis.

- All primary care providers can contribute to improved oral and craniofacial health. Inter-

disciplinary care is needed to manage the oral health–general health interface. Dentists, as primary care providers, are uniquely positioned to play an expanded role in the detection, early recognition, and management of a wide range of complex oral and general diseases and conditions.

- Nonsurgical interventions are available to reverse disease progression and to manage oral diseases as infections.

- New knowledge and the development of molecular and genetically based tests will facilitate risk assessment and management and improve the ability of health care providers to customize treatment.

- Health care providers can successfully deliver tobacco cessation and other health promotion programs in their offices, contributing to both overall health and oral health.

- Biocompatible rehabilitative materials and biologically engineered tissues are being developed and will greatly enhance the treatment options available to providers and their patients.

Chapter 9 describes the roles of dental practitioners and their teams, the medical community, and public health agencies at local, state, and national levels in administering care or reimbursing for the costs of care. These activities are viewed against the changing organization of U.S. health care and trends regarding the workforce in research, education, and practice.

- Dental, medical, and public health delivery systems each provide services that affect oral and craniofacial health in the U.S. population. Clinical oral health care is predominantly provided by a private practice dental workforce.

- Expenditures for dental services alone made up 4.7 percent of the nation's health expenditures in 1998—\$53.8 billion out of \$1.1 trillion. These expenditures underestimate the true costs to the nation, however, because data are unavailable to determine the extent of expenditures and services provided for craniofacial health care by other health providers and institutions.

- The public health infrastructure for oral health is insufficient to address the needs of disadvantaged groups, and the integration of oral and general health programs is lacking.

- Expansion of community-based disease prevention and lowering of barriers to personal oral health care are needed to meet the needs of the population.

- Insurance coverage for dental care is increasing but still lags behind medical insurance.

For every child under 18 years old without medical insurance, there are at least two children without dental insurance; for every adult 18 years or older without medical insurance, there are three without dental insurance.

- Eligibility for Medicaid does not ensure enrollment, and enrollment does not ensure that individuals obtain needed care. Barriers include patient and caregiver understanding of the value and importance of oral health to general health, low reimbursement rates, and administrative burdens for both patient and provider.

- A narrow definition of “medically necessary dental care” currently limits oral health services for many insured persons, particularly the elderly.

- The dentist-to-population ratio is declining, creating concern as to the capability of the dental workforce to meet the emerging demands of society and provide required services efficiently.

- An estimated 25 million individuals reside in areas lacking adequate dental care services, as defined by Health Professional Shortage Area (HPSA) criteria.

- Educational debt has increased, affecting both career choices and practice location.

- Disparities exist in the oral health profession workforce and career paths. The number of under-represented minorities in the oral health professions is disproportionate to their distribution in the population at large.

- Current and projected demand for dental school faculty positions and research scientists is not being met. A crisis in the number of faculty and researchers threatens the quality of dental education; oral, dental, and craniofacial research; and, ultimately, the health of the public.

- Reliable and valid measures of oral health outcomes do not exist and need to be developed, validated, and incorporated into practice and programs.

Part Five: What Are the Needs and Opportunities to Enhance Oral Health?

Chapter 10 looks at determinants of oral health in the context of society and across various life stages. Although theorists have proposed a variety of models of health determinants, there is general consensus that individual biology, the physical and socioeconomic environment, personal behaviors and lifestyle, and the organization of health care are key factors whose interplay determines the level of oral health achieved by an individual. The chapter provides examples of these factors with an emphasis

on barriers and ways to raise the level of oral health for children and older Americans. The findings include:

- The major factors that determine oral and general health and well-being are individual biology and genetics; the environment, including its physical and socioeconomic aspects; personal behaviors and lifestyle; access to care; and the organization of health care. These factors interact over the life span and determine the health of individuals, population groups, and communities—from neighborhoods to nations.

- The burden of oral diseases and conditions is disproportionately borne by individuals with low socioeconomic status at each life stage and by those who are vulnerable because of poor general health.

- Access to care makes a difference. A complex set of factors underlies access to care and includes the need to have an informed public and policymakers, integrated and culturally competent programs, and resources to pay and reimburse for the care. Among other factors, the availability of insurance increases access to care.

- Preventive interventions, such as protective head and mouth gear and dental sealants, exist but are not uniformly used or reinforced.

- Nursing homes and other long-term care institutions have limited capacity to deliver needed oral health services to their residents, most of whom are at increased risk for oral diseases.

- Anticipatory guidance and risk assessment and management facilitate care for children and for the elderly.

- Federal and state assistance programs for selected oral health services exist; however, the scope of services is severely limited, and their reimbursement level for oral health services is low compared to the usual fee for care.

Chapter 11 spells out in greater detail the promise of the life sciences in improving oral health in the coming years in the context of changes in American—and global—society. The critical role of genetics and molecular biology is emphasized.

Chapter 12, the final chapter, iterates the themes of the report and groups the findings from the earlier chapters into eight major categories. These findings, as well as a suggested framework for action to guide the next steps in enhancing the oral health of the nation, are presented below.

MAJOR FINDINGS

Oral diseases and disorders in and of themselves affect health and well-being throughout life. The burden of oral problems is extensive and may be particularly severe in vulnerable populations. It includes the common dental diseases and other oral infections such as cold sores and candidiasis that can occur at any stage of life, as well as birth defects in infancy and the chronic facial pain conditions and oral cancers seen in later years. Many of these conditions and their treatments may undermine self-image and self-esteem, discourage normal social interaction, cause other health problems, and lead to chronic stress and depression as well as incur great financial cost. They may also interfere with vital functions such as breathing, food selection, eating, swallowing, and speaking and with activities of daily living such as work, school, and family interactions.

Safe and effective measures exist to prevent the most common dental diseases—dental caries and periodontal diseases. Community water fluoridation is safe and effective in preventing dental caries in both children and adults. Water fluoridation benefits all residents served by community water supplies regardless of their social or economic status. Professional and individual measures, including the use of fluoride mouthrinses, gels, dentifrices, and dietary supplements and the application of dental sealants, are additional means of preventing dental caries. Gingivitis can be prevented by good personal oral hygiene practices, including brushing and flossing.

Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use, and poor dietary choices affect oral and craniofacial health as well. These individual behaviors are associated with increased risk for craniofacial birth defects, oral and pharyngeal cancers, periodontal disease, dental caries, and candidiasis, among other oral health problems. Opportunities exist to expand the oral disease prevention and health promotion knowledge and practices of the public through community programs and in health care settings. All health care providers can play a role in promoting healthy lifestyles by incorporating tobacco cessation programs, nutritional counseling, and other health promotion efforts into their practices.

There are profound and consequential oral health disparities within the U.S. population. Disparities for various oral conditions may relate to income, age, sex, race or ethnicity, or medical status. Although common dental diseases are preventable, not all members of society are informed about or able to avail themselves of appropriate oral-health-

promoting measures. Similarly, not all health providers may be aware of the services needed to improve oral health. In addition, oral health care is not fully integrated into many care programs. Social, economic, and cultural factors and changing population demographics affect how health services are delivered and used, and how people care for themselves. Reducing disparities requires wide-ranging approaches that target populations at highest risk for specific oral diseases and involves improving access to existing care. One approach includes making dental insurance more available to Americans. Public coverage for dental care is minimal for adults, and programs for children have not reached the many eligible beneficiaries.

More information is needed to improve America's oral health and eliminate health disparities. We do not have adequate data on health, disease, and health practices and care use for the U.S. population as a whole and its diverse segments, including racial and ethnic minorities, rural populations, individuals with disabilities, the homeless, immigrants, migrant workers, the very young, and the frail elderly. Nor are there sufficient data that explore health issues in relation to sex or sexual orientation. Data on state and local populations, essential for program planning and evaluation, are rare or unavailable and reflect the limited capacity of the U.S. health infrastructure for oral health. Health services research, which could provide much needed information on the cost, cost-effectiveness, and outcomes of treatment, is also sorely lacking. Finally, measurement of disease and health outcomes is needed. Although progress has been made in measuring oral-health-related quality of life, more needs to be done, and measures of oral health per se do not exist.

The mouth reflects general health and well-being. The mouth is a readily accessible and visible part of the body and provides health care providers and individuals with a window on their general health status. As the gateway of the body, the mouth senses and responds to the external world and at the same time reflects what is happening deep inside the body. The mouth may show signs of nutritional deficiencies and serve as an early warning system for diseases such as HIV infection and other immune system problems. The mouth can also show signs of general infection and stress. As the number of substances that can be reliably measured in saliva increases, it may well become the diagnostic fluid of choice, enabling the diagnosis of specific disease as well as the measurement of the concentration of a variety of drugs, hormones, and other molecules of interest. Cells and fluids in the mouth may also be

used for genetic analysis to help uncover risks for disease and predict outcomes of medical treatments.

Oral diseases and conditions are associated with other health problems. Oral infections can be the source of systemic infections in people with weakened immune systems, and oral signs and symptoms often are part of a general health condition. Associations between chronic oral infections and other health problems, including diabetes, heart disease, and adverse pregnancy outcomes, have also been reported. Ongoing research may uncover mechanisms that strengthen the current findings and explain these relationships.

Scientific research is key to further reduction in the burden of diseases and disorders that affect the face, mouth, and teeth. The science base for dental diseases is broad and provides a strong foundation for further improvements in prevention; for other craniofacial and oral health conditions the base has not yet reached the same level of maturity. Scientific research has led to a variety of approaches to improve oral health through prevention, early diagnosis, and treatment. We are well positioned to take these prevention measures further by investigating how to develop more targeted and effective interventions and devising ways to enhance their appropriate adoption by the public and the health professions. The application of powerful new tools and techniques is important. Their employment in research in genetics and genomics, neuroscience, and cancer has allowed rapid progress in these fields. An intensified effort to understand the relationships between oral infections and their management, and other illnesses and conditions is warranted, along with the development of oral-based diagnostics. These developments hold great promise for the health of the American people.

A FRAMEWORK FOR ACTION

All Americans can benefit from the development of a National Oral Health Plan to improve quality of life and eliminate health disparities by facilitating collaborations among individuals, health care providers, communities, and policymakers at all levels of society and by taking advantage of existing initiatives. Everyone has a role in improving and promoting oral health. Together we can work to broaden public understanding of the importance of oral health and its relevance to general health and well-being, and to ensure that existing and future preventive, diagnostic, and treatment measures for oral diseases and disorders are made available to all Americans. The following are the principal components of the plan:

Change perceptions regarding oral health and disease so that oral health becomes an accepted component of general health.

- *Change public perceptions.* Many people consider oral signs and symptoms to be less important than indications of general illness. As a result, they may avoid or postpone needed care, thus exacerbating the problem. If we are to increase the nation's capacity to improve oral health and reduce health disparities, we need to enhance the public's understanding of the meaning of oral health and the relationship of the mouth to the rest of the body. These messages should take into account the multiple languages and cultural traditions that characterize America's diversity.

- *Change policymakers' perceptions.* Informed policymakers at the local, state, and federal levels are critical in ensuring the inclusion of oral health services in health promotion and disease prevention programs, care delivery systems, and reimbursement schedules. Raising awareness of oral health among legislators and public officials at all levels of government is essential to creating effective public policy to improve America's oral health. Every conceivable avenue should be used to inform policymakers—informally through their organizations and affiliations and formally through their governmental offices—if rational oral health policy is to be formulated and effective programs implemented.

- *Change health providers' perceptions.* Too little time is devoted to oral health and disease topics in the education of nondental health professionals. Yet all care providers can and should contribute to enhancing oral health. This can be accomplished in several ways, such as including an oral examination as part of a general medical examination, advising patients in matters of tobacco cessation and diet, and referring patients to oral health practitioners for care prior to medical or surgical treatments that can damage oral tissues, such as cancer chemotherapy or radiation to the head and neck. Health care providers should be ready, willing, and able to work in collaboration to provide optimal health care for their patients. Having informed health care professionals will ensure that the public using the health care system will benefit from interdisciplinary services and comprehensive care. To prepare providers for such a role will involve, among other factors, curriculum changes and multidisciplinary training.

Accelerate the building of the science and evidence base and apply science effectively to improve oral health. Basic behavioral and biomedical research, clinical trials, and population-based research have

been at the heart of scientific advances over the past decades. The nation's continued investment in research is critical for the provision of new knowledge about oral and general health and disease for years to come and needs to be accelerated if further improvements are to be made. Equally important is the effective transfer of research findings to the public and health professions. However, the next steps are more complicated. The challenge is to understand complex diseases caused by the interaction of multiple genes with environmental and behavioral variables—a description that applies to most oral diseases and disorders—and translate research findings into health care practice and healthy lifestyles.

This report highlights many areas of research opportunities and needs in each chapter. At present, there is an overall need for behavioral and clinical research, clinical trials, health services research, and community-based demonstration research. Also, development of risk assessment procedures for individuals and communities and of diagnostic markers to indicate whether an individual is more or less susceptible to a given disease can provide the basis for formulating risk profiles and tailoring treatment and program options accordingly.

Vital to progress in this area is a better understanding of the etiology and distribution of disease. But as this report makes clear, epidemiologic and surveillance databases for oral health and disease, health services, utilization of care, and expenditures are limited or lacking at the national, state, and local levels. Such data are essential in conducting health services research, generating research hypotheses, planning and evaluating programs, and identifying emerging public health problems. Future data collection must address differences among the subpopulations making up racial and ethnic groups. More attention must also be paid to demographic variables such as age, sex, sexual orientation, and socioeconomic factors in determining health status. Clearly, the more detailed information that is available, the better can program planners establish priorities and targeted interventions.

Progress in elucidating the relationships between chronic oral inflammatory infections, such as periodontitis, and diabetes and glycemic control as well as other systemic conditions will require a similar intensified commitment to research. Rapid progress can also occur with efforts in the area of the natural repair and regeneration of oral tissues and organs. Improvements in oral health depend on multidisciplinary and interdisciplinary approaches to biomedical and behavioral research, including partnerships among researchers in the life and physical sciences,

and on the ability of practitioners and the public to apply research findings effectively.

Build an effective health infrastructure that meets the oral health needs of all Americans and integrates oral health effectively into overall health. The public health capacity for addressing oral health is dilute and not integrated with other public health programs. Although the Healthy People 2010 objectives provide a blueprint for outcome measures, a national public health plan for oral health does not exist. Furthermore, local, state, and federal resources are limited in the personnel, equipment, and facilities available to support oral health programs. There is also a lack of available trained public health practitioners knowledgeable about oral health. As a result, existing disease prevention programs are not being implemented in many communities, creating gaps in prevention and care that affect the nation's neediest populations. Indeed, cutbacks in many state budgets have reduced staffing of state and territorial dental programs and curtailed oral health promotion and disease prevention efforts. An enhanced public health infrastructure would facilitate the development of strengthened partnerships with private practitioners, other public programs, and voluntary groups.

There is a lack of racial and ethnic diversity in the oral health workforce. Efforts to recruit members of minority groups to positions in health education, research, and practice in numbers that at least match their representation in the general population not only would enrich the talent pool, but also might result in a more equitable geographic distribution of care providers. The effect of that change could well enhance access and utilization of oral health care by racial and ethnic minorities.

A closer look at trends in the workforce discloses a worrisome shortfall in the numbers of men and women choosing careers in oral health education and research. Government and private sector leaders are aware of the problem and are discussing ways to increase and diversify the talent pool, including easing the financial burden of professional education, but additional incentives may be necessary.

Remove known barriers between people and oral health services. This report presents data on access, utilization, financing, and reimbursement of oral health care; provides additional data on the extent of the barriers; and points to the need for public-private partnerships in seeking solutions. The data indicate that lack of dental insurance, private or public, is one of several impediments to obtaining oral health care

and accounts in part for the generally poorer oral health of those who live at or near the poverty line, lack health insurance, or lose their insurance upon retirement. The level of reimbursement for services also has been reported to be a problem and a disincentive to the participation of providers in certain public programs. Professional organizations and government agencies are cognizant of these problems and are exploring solutions that merit evaluation. Particular concern has been expressed about the nation's children, and initiatives such as the State Children's Health Insurance Program, while not mandating coverage for oral health services, are a positive step. In addition, individuals whose health is physically, mentally, and emotionally compromised need comprehensive integrated care.

Use public-private partnerships to improve the oral health of those who still suffer disproportionately from oral diseases. The collective and complementary talents of public health agencies, private industry, social services organizations, educators, health care providers, researchers, the media, community leaders, voluntary health organizations and consumer groups, and concerned citizens are vital if America is not just to reduce, but to eliminate, health disparities. This report highlights variations in oral and general health within and across all population groups. Increased public-private partnerships are needed to educate the public, to educate health professionals, to conduct research, and to provide health care services and programs. These partnerships can build and strengthen cross-disciplinary, culturally competent, community-based, and community-wide efforts and demonstration programs to expand initiatives for health promotion and disease prevention. Examples of such efforts include programs to prevent tobacco use, promote better dietary choices, and encourage the use of protective gear to prevent sports injuries. In this way, partnerships uniting sports organizations, schools, the faith community, and other groups and leaders, working in concert with the health community, can contribute to improved oral and general health.

CONCLUSION

The past half century has seen the meaning of oral health evolve from a narrow focus on teeth and gingiva to the recognition that the mouth is the center of

vital tissues and functions that are critical to total health and well-being across the life span. The mouth as a mirror of health or disease, as a sentinel or early warning system, as an accessible model for the study of other tissues and organs, and as a potential source of pathology affecting other systems and organs has been described in earlier chapters and provides the impetus for extensive future research. Past discoveries have enabled Americans today to enjoy far better oral health than their forebears a century ago. But the evidence that not all Americans have achieved the same level of oral health and well-being stands as a major challenge, one that demands the best efforts of public and private agencies and individuals.

REFERENCES

- Health Care Financing Administration (HCFA). National Health Expenditures projections: 1998-2008. Office of the Actuary. <http://www.hcfa.gov/stats/NHE-Proj/>. 2000 Apr 25.
- U.S. Department of Health and Human Services (USDHHS). Healthy People 2010 (Conference Edition, in two volumes). Washington; 2000 Jan.
- U.S. General Accounting Office (GAO). Oral health in low-income populations. GAO/HEHS-00-72. 2000 Apr.

PROJECT TEAM

- Caswell A. Evans DDS, MPH
Project Director and Executive Editor
Assistant Director, Los Angeles County Department of Health Services
- Dushanka V. Kleinman DDS, MScD
Co-Executive Editor
Deputy Director, National Institute of Dental and Craniofacial Research, National Institutes of Health
- William R. Maas DDS, MPH, MS
Chief Dental Officer, U.S. Public Health Service
Director, Division of Oral Health,
Centers for Disease Control and Prevention
- Harold C. Slavkin DDS
Director, National Institute of Dental and Craniofacial Research, National Institutes of Health
- Joan S. Wilentz MA
Science Writer and Editor
- Roseanne Price ELS
Editor
- Marla Fogelman
Editor

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What Is Oral Health?

The two chapters that follow explore the answer to this question, an answer that continues to evolve throughout the report. Oral health means more than healthy teeth and the absence of disease. It involves the ability of individuals to carry out essential functions such as eating and speaking as well as to contribute fully to society. Chapter 1, the introduction, explains how the meaning of oral health has developed in tandem with progress in understanding the two chief dental diseases—dental caries and periodontal diseases—which historically have been the major preoccupation of patients, providers, and research investigators alike. There is a marvelous success story here regarding the role of fluoride in preventing dental caries and the research that proved that dental caries and periodontal diseases are infections and can be prevented. These investigations were complemented by studies of the tissues of the mouth and adjacent areas—the craniofacial complex. Chapter 2 describes the tissues and organs of the craniofacial complex, providing a primer and guide to their essential features that emphasizes the ways they contribute to the richness of human experience, and at the same time protect and nurture the human body.

The Meaning of Oral Health

The intent of this first-ever Surgeon General's report on oral health is to alert Americans to the full meaning of oral health and its importance in relation to general health and well-being. Great progress has been made in reducing the extent and severity of common oral diseases, and recent history has seen marked improvements in the nation's oral and dental health, thanks to successful prevention measures adopted by communities, individuals, and oral health professionals. However, not everyone is experiencing the same degree of improvement. What amounts to a "a silent epidemic" of dental and oral diseases is affecting some population groups—a burden of disease that restricts activities in school, work, and home, and often significantly diminishes the quality of life.

The word *oral*, both in its Latin root and in common usage, refers to the mouth. The mouth includes not only the teeth and the gums (gingiva) and their supporting connective tissues, ligaments, and bone, but also the hard and soft palate, the soft mucosal tissue lining of the mouth and throat, the tongue, the lips, the salivary glands, the chewing muscles, and the upper and lower jaws, which are connected to the skull by the temporomandibular joints. Equally important are the branches of the nervous, immune, and vascular systems that animate, protect, and nourish the oral tissues, as well as provide the connections to the brain and the rest of the body. The genetic patterning of development in utero further reveals the intimate relationship of the oral tissues to the developing brain and to the tissues of the face and head that surround the mouth, structures whose location is captured in the word *craniofacial*.

A major theme of this report is that **oral health means much more than healthy teeth**. It means being free of chronic oral-facial pain conditions, oral and pharyngeal (throat) cancers, oral soft tissue lesions, birth defects such as cleft lip and palate, and

scores of other diseases and disorders that affect the oral, dental, and craniofacial tissues, collectively known as the *craniofacial complex*. These are tissues whose functions we often take for granted, yet they represent the very essence of our humanity. They allow us to speak and smile; sigh and kiss; smell, taste, touch, chew, and swallow; cry out in pain; and convey a world of feelings and emotions through facial expressions. They also provide protection against microbial infections and environmental insults.

The craniofacial tissues also provide a useful means to understanding organs and systems in less accessible parts of the body. The salivary glands are a model of other exocrine glands, and an analysis of saliva can provide telltale clues of overall health or disease. The jawbones are examples of other skeletal parts. The nervous system apparatus underlying facial pain has its counterpart in nerves elsewhere in the body.

A thorough oral examination can detect signs of nutritional deficiencies as well as a number of systemic diseases, including microbial infections, immune disorders, injuries, and some cancers. Indeed, the phrase *the mouth is a mirror* has been used to illustrate the wealth of information that can be derived from examining oral tissues.

New research is pointing to associations between chronic oral infections and heart and lung diseases, stroke, and low-birth-weight, premature births. Associations between periodontal disease and diabetes have long been noted. This report assesses these associations and explores mechanisms that might explain these oral-systemic disease connections.

In parallel with the broadened meaning of *oral health*, the meaning of *health* has evolved. The standard definition of health, "freedom from disease, defect, or pain," defines what health is not, rather

than what it is. A more positive definition, one that the World Health Organization established in 1948, states that health is a complete state of physical, mental, and social well-being, and not just the absence of infirmity.

The broadened meaning of *oral health* parallels the broadened meaning of *health*. In 1948 the World Health Organization expanded the definition of health to mean "a complete state of physical, mental, and social well-being, and not just the absence of infirmity." It follows that oral health must also include well-being. Just as we now understand that nature and nurture are inextricably linked, and mind and body are both expressions of our human biology, so, too, we must recognize that oral health and general health are inseparable. We ignore signs and symptoms of oral disease and dysfunction to our detriment. Consequently, a second theme of the report is that **oral health is integral to general health**. You cannot be healthy without oral health. Oral health and general health should not be interpreted as separate entities. Oral health is a critical component of health and must be included in the provision of health care and the design of community programs.

The wider meanings of *oral* and *health* in no way diminish the relevance and importance of the two leading dental diseases, caries (tooth decay) and the periodontal diseases. They remain common and widespread, affecting nearly everyone at some point in the life span. What has changed is what we can do about them.

At the start of the twentieth century, most Americans expected to be toothless by age 45, and most were. Expectations have changed, and most people now assume that they will maintain their teeth over their lifetime, and take active measures to do so. Researchers in the 1930s discovered that people living in communities with naturally fluoridated water supplies had less dental caries than people drinking unfluoridated water. But not until the end of World War II were the investigators able to design and implement the community clinical trials that confirmed their observations and launched a better approach to the problem of dental caries: prevention. Soon after, adjusting the fluoride content of community water supplies was pursued as an important public health measure to prevent dental caries.

Although this measure has not been fully implemented, the results have been dramatic. Dental caries began to decline in the 1950s among children who grew up in fluoridated cities, and by the late 1970s, declines in decay were evident for many Americans. The application of oral science to improved diagnos-

tic, treatment, and prevention strategies has saved billions of dollars per year in the nation's annual health bill. Even more significant, the result is that far fewer people are edentulous (toothless) today than a generation ago.

The theme of prevention gained momentum as pioneering investigators and practitioners in the 1950s and 1960s showed that not only dental caries but also periodontal diseases are bacterial infections. The researchers demonstrated that the infections could be prevented by increasing host resistance to disease and reducing or eliminating the suspected microbial pathogens in the oral cavity. The applications of research discoveries have resulted in continuing improvements in the oral health of Americans, new approaches to the prevention and treatment of dental diseases, and the growth of the science.

The significant role that scientists, dentists, dental hygienists, and other health professionals have played in the prevention of oral disease and disability leads to a third theme of this report: **safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease**. These measures include daily oral hygiene procedures and other lifestyle behaviors, community programs such as community water fluoridation and tobacco cessation programs, and provider-based interventions such as the placement of dental sealants and examinations for common oral and pharyngeal cancers. It is hoped that this Surgeon General's report will facilitate the maturing of the broad field of craniofacial research so that gains in the prevention of craniofacial diseases and disorders can be realized that are as impressive as those achieved for common dental diseases.

At the same time, more needs to be done to ensure that messages of health promotion and disease prevention are brought home to all Americans. In this regard, a fourth theme of the report is that **general health risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health**. The evidence for an association between tobacco use and oral diseases has been clearly delineated in almost every Surgeon General's report on tobacco since 1964, and the oral effects of nutrition and diet are presented in the Surgeon General's report on nutrition (1988). All the health professions can play a role in reducing the burden of disease in America by calling attention to these and other risk factors and suggesting appropriate actions.

Clearly, promoting health and preventing disease are concepts the American people have taken to heart. For the third decade the nation has developed a plan for the prevention of disease and the promo-

tion of health, embodied in the U.S. Department of Health and Human Services (2000) document, Healthy People 2010. As a nation, we hope to eliminate disparities in health and prevent oral diseases, cancer, birth defects, AIDS and other devastating infections, mental illness and suicide, and the chronic diseases of aging. To live well into old age free of pain and infirmity, and with a high quality of life, is the American dream.

Scientists today take that dream seriously in pursuing the intricacies of the craniofacial complex. They are using an ever-growing array of sophisticated analytic tools and imaging systems to study normal function and diagnose disease. They are completing the mapping and sequencing of human, animal, microbial, and plant genomes, the better to understand the complexities of human development, aging, and pathological processes. They are growing cell lines, synthesizing molecules, and using a new generation of biomaterials to revolutionize tissue repair and regeneration. More than ever before, they are working in multidisciplinary teams to bring new knowledge and expertise to the goal of understanding complex human diseases and disorders.

THE CHALLENGE

This Surgeon General's report has much to say about the inequities and disparities that affect those least able to muster the resources to achieve optimal oral health. The barriers to oral health include lack of access to care, whether because of limited income or lack of insurance, transportation, or the flexibility to take time off from work to attend to personal or family needs for care. Individuals with disabilities and those with complex health problems may face additional barriers to care. Sometimes, too, the public, policymakers, and providers may consider oral health and the need for care to be less important than other health needs, pointing to the need to raise awareness and improve health literacy.

Even more costly to the individual and to society are the expenses associated with oral health problems that go beyond dental diseases. The nation's yearly dental bill is expected to exceed \$60 billion in 2000 (Health Care Financing Administration 2000). However, add to that expense the tens of billions of dollars in direct medical care and indirect costs of chronic craniofacial pain conditions such as temporomandibular disorders, trigeminal neuralgia, shingles, or burning mouth syndrome; the \$100,000 minimum individual lifetime costs of treating craniofacial birth defects such as cleft lip and palate; the costs of oral and pharyngeal cancers; the costs of

autoimmune diseases; and the costs associated with the unintentional and intentional injuries that so often affect the head and face. Then add the social and psychological consequences and costs. Damage to the craniofacial complex, whether from disease, disorder, or injury, strikes at our very identity. We see ourselves, and others see us, in terms of the face we present to the world. Diminish that image in any way and we risk the loss of self-esteem and well-being.

Many unanswered questions remain for scientists, practitioners, educators, policymakers, and the public. This report highlights the research challenges as well as pointing to emerging technologies that may facilitate finding solutions. Along with the quest for answers comes the challenge of applying what is already known in a society where there are social, political, economic, behavioral, and environmental barriers to health and well-being.

THE CHARGE

The realization that oral health can have a significant impact on the overall health and well-being of the nation's population led the Office of the Surgeon General, with the approval of the Secretary of Health and Human Services, to commission this report. Recognizing the gains that have been made in disease prevention while acknowledging that there are populations that suffer disproportionately from oral health problems, the Secretary asked that the report "define, describe, and evaluate the interaction between oral health and health and well-being [quality of life], through the life span in the context of changes in society." Key elements to be addressed were the determinants of health and disease, with a primary focus on prevention and "producing health" rather than "restoring health"; a description of the burden of oral diseases and disorders in the nation; and the evidence for actions to improve oral health to be taken across the life span. The report also was to feature an orientation to the future, highlighting leading-edge technologies and research findings that can be brought to bear in improving the oral health of individuals and communities.

THE SCIENCE BASE FOR THE REPORT

This report is based on a review of the published scientific literature. Where appropriate, standards established to determine the quality of the evidence, based on the study design and its rigor, were used. In addition, the strength of the recommendations, where they are made, is based on evidence of effectiveness for the population of interest. The scope

of the review encompassed the international English literature. Recent systematic reviews of the literature are referenced, as are selected review articles. A few referenced articles are in press, and there are occasional references to recent abstracts and personal communications.

The science base in oral health has been evolving over the past half century. Initial research in this area was primarily in the basic sciences, investigating mechanisms of normal development and pathology in relation to dental caries and periodontal diseases. Prevention research has included controlled clinical studies, with and without randomization, as well as community trials and demonstration research. More recent research has broadened the science base to include studies of the range of craniofacial diseases and disorders and is moving from basic science to translational, clinical, and health services research.

The clinical literature in the oral health sciences includes the full range of studies, from randomized controlled studies to case studies. Most of the literature includes cross-sectional and cohort studies, with some case-control studies. General reviews of the literature have been used for Chapters 2 through 10. Chapter 4 includes both published and new analyses of national and state databases that have been carefully designed and for which quality assurance has been maintained by the Centers for Disease Control and Prevention. Studies of smaller populations are also included where relevant. In Chapters 5 and 7, evidence tables are presented for the discussion of the association of oral infections and systemic conditions and for oral disease prevention and health promotion measures, respectively. Experts in the respective fields contributed to the report, and independent expert peer review was conducted for all sections of the report. The published literature related to the development of new technologies, their potential impact, and the need for further research are described in the course of addressing the requested futures orientation.

ORGANIZATION OF THE REPORT

The report centers on five major questions, which have been used to structure the report into five parts.

What Is Oral Health?

The meaning of oral health is discussed in the opening pages of this chapter, and the interdependence of oral health with general health and well-being is, as noted, a recurrent theme throughout the volume. Chapter 2 provides an overview of the craniofacial

complex in development and aging, how the tissues and organs function in essential life processes, and their role in determining our uniquely human abilities. The later chapters elaborate further on the meaning of oral health. Of particular importance is the discussion of oral health in relation to well-being and quality of life described in Chapter 6.

What Is the Status of Oral Health in America?

Chapter 3 is a primer describing the major diseases and disorders that affect the craniofacial complex. Chapter 4 constitutes an oral health status report card on the noninstitutionalized civilian population of the United States, describing the magnitude of the problem. It is based on the most recent national and state data available for a range of craniofacial diseases, disorders, and conditions. In general, the national data provide information categorized by sex, age, income (poor versus nonpoor), and broad racial and ethnic categories. In addition, the chapter includes a profile of the oral and general health of selected population groups. These include racial and ethnic groups such as African Americans, Hispanics, Asians, Native Hawaiians and Other Pacific Islanders, and American Indians/Alaska Natives. The health status of women and individuals with disabilities is highlighted. Although it is clearly desirable to describe the health status of additional populations, data are insufficient or lacking for groups defined by sexual orientation or rural residency or categorized as homeless, migrant workers, or incarcerated. As an initial step toward understanding the burden of disease in relation to the provision of care, available data on the number of dental visits are provided.

What Is the Relationship Between Oral Health and General Health and Well-being?

Chapters 5 and 6 address key issues in the charge—the relationship of oral health to general health and well-being. Chapter 5 explores the theme of the mouth as a mirror that in some measure can reflect general health or disease status. Examples are given of how oral tissues may signal the presence of disease, disease progression, or risk factor exposure levels, and how oral cells and fluids are increasingly being used as diagnostic tools. This is followed by a discussion of the mouth as a portal of entry for infections that can affect local tissues and may spread to other parts of the body. The next section reviews the

literature regarding emerging associations between oral diseases and disorders and diabetes, heart disease and stroke, and adverse pregnancy outcomes.

Chapter 6 demonstrates the relationship between oral health and quality of life, presenting data on the consequences of poor oral health and altered appearance on speech, eating, and other functions, as well as on self-esteem, social interaction, education, career achievement, and emotional state. Anthropological and ethnographic literature is introduced to underscore the cultural values and symbolism attached to facial appearance and teeth.

How Is Oral Health Promoted and Maintained and How Are Oral Diseases Prevented?

The next three chapters review how individuals, health care practitioners, communities, and the nation as a whole contribute to oral health. Chapter 7 reviews the evidence for the efficacy and effectiveness of oral health promotion and disease prevention measures with a focus on community efforts in preventing dental disease. It continues with a discussion of the need to expand efforts in such areas as oral cancer prevention.

Chapter 8 explores the role of the individual and the health care provider in promoting and maintaining oral health and well-being. For the individual, this means exercising appropriate self-care and adopting healthy behaviors. For the provider, it means incorporating the knowledge emerging from the science base in a timely manner for prevention and diagnosis, risk assessment and risk management, and treatment of oral diseases and disorders. The chapter focuses largely on the oral health care provider. The management of oral and craniofacial health and disease necessitates collaborations among a team of care providers to achieve optimal oral and general health.

Chapter 9 describes the roles of dental practitioners and their teams, the medical community, and public health agencies at local, state, and national levels in administering oral health care or reimbursing for the costs of care. These activities are viewed against the changing organization of U.S. health care

and trends regarding the workforce in research, education, and practice.

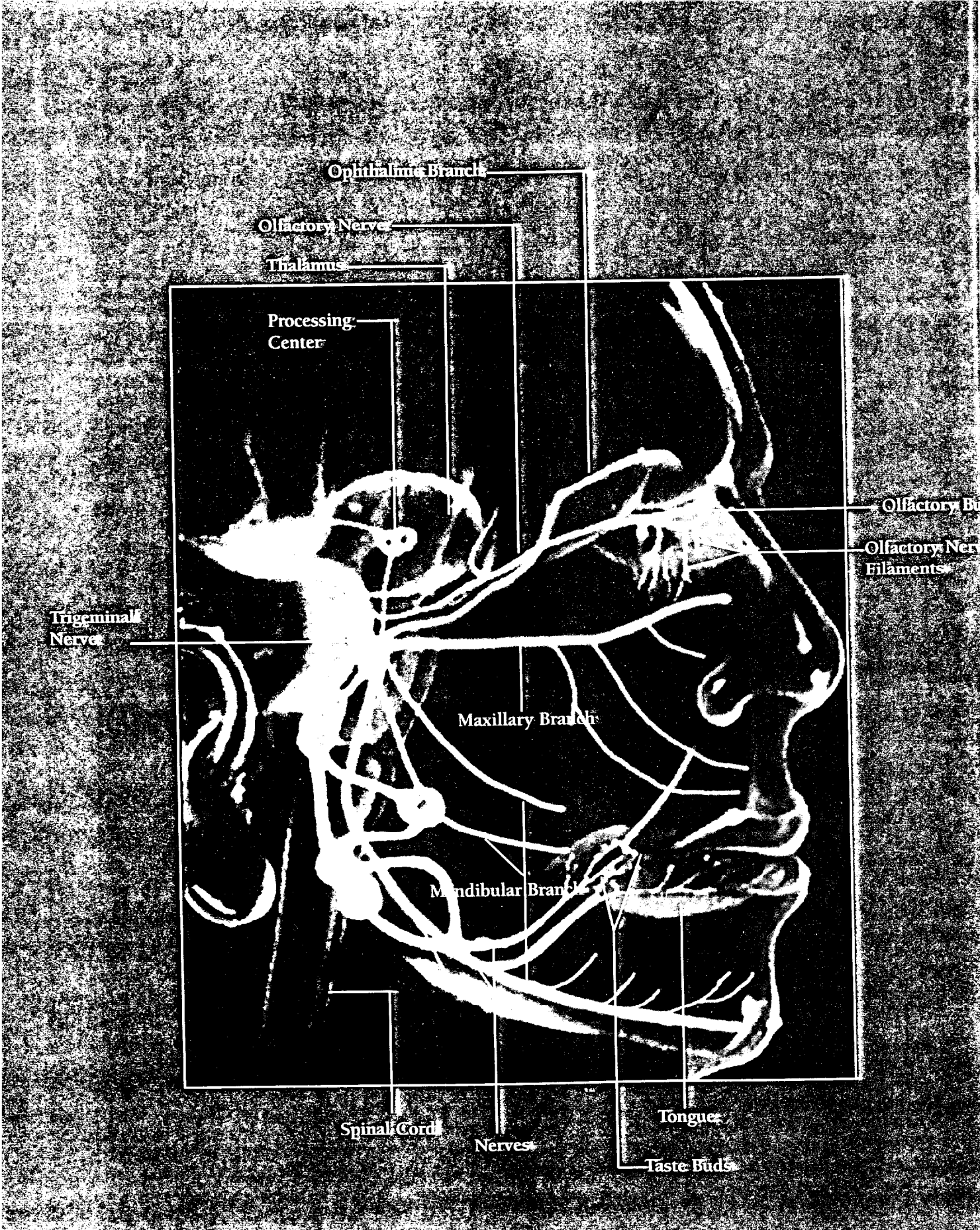
What Are the Needs and Opportunities to Enhance Oral Health?

Chapter 10 looks at determinants of oral health in the context of society and across various life stages. Although theorists have proposed a variety of models of health determinants, there is general consensus that individual biology, the physical and socioeconomic environment, personal behaviors and lifestyle, and the organization of health care are key factors whose interplay determines the level of oral health achieved by an individual. The chapter provides examples of these factors and, in the latter half, illustrates their varying effects at different stages of the life span, with an emphasis on children and older Americans. Barriers and ways to raise the level of oral health that can be achieved at each life stage are presented.

Chapter 11 spells out in greater detail the promise of the life sciences in improving oral health in the coming years in the context of changes in American—and global—society. The critical role of genetics and molecular biology is emphasized.

A Call to Action

Chapter 12, the final chapter, summarizes the major findings of the report and suggests actions to guide the next steps in enhancing the oral health of the nation. The need for partnerships between public and private sectors in carrying out a proposed National Oral Health Plan is emphasized. To ensure progress, these partnerships need to include individual patients and the general public and to reflect all population groups in the nation. All the health care disciplines need to be involved, along with industry, academia, and government, as well as health care organizations, health professional associations, health insurers, and patient groups. United by the evidence that oral health is essential to general health and well-being, the combined forces and collective wisdom of all interested parties and stakeholders can make optimal oral health a reality for all people.



Ophthalmic Branch

Olfactory Nerve

Thalamus

Processing Center

Trigeminal Nerve

Maxillary Branch

Mandibular Branch

Spinal Cord

Nerves

Tongue

Taste Buds

Olfactory Bulb

Olfactory Nerve Filaments

The Craniofacial Complex

The first line a child draws on a face is usually the mouth. The mouth is the center of communication and contact. Along with the eyes, ears, and nose, it is positioned near the brain, ensuring close integration and coordination. We use the craniofacial complex—the oral, dental, and the other craniofacial tissues that house the organs of taste, vision, hearing, and smell—to experience and interact with the world around us. These sense organs have evolved to serve as superb information processors and aids to survival. At the most elemental level, they enable us to sense our environment—from alerting us to predators or poisons to recognizing family, friends, or prospective mates.

Our ability to act on the nerve signals from these organs results from the abundant supply of paired cranial nerves that innervate the craniofacial tissues. No place in the body is as rich in both the number of sensory nerves and the ratio of motor nerves to muscle fibers as the face, and, within it, the mouth and teeth. The nerve circuits not only trigger protective reflexes to make us blink, gape, and start in surprise, but also enable us to perform countless functions we take for granted. We speak and taste and chew and swallow. We express our feelings through smiles and scowls; we grimace and cry out in pain; we murmur endearments and kiss our loved ones.

Beyond the special senses of vision, hearing, taste, and smell, the craniofacial complex includes nerve endings sensitive to the body's position in space and to touch, pressure, temperature, and pain. In sum, if the brain is the command-and-control center of the body, evolution has ensured that the staff reporting from the field and carrying out orders are stationed a strategic few inches away, in the craniofacial complex.

CONTACT AND COMMUNICATION

Taste and Smell

The developmental process by which vital nerve centers came to be concentrated in the head, mouth, and teeth began early in evolution. Even paramecia have mouths, as do worms, bugs, and all more complex organisms. The mouth appears in the human embryo during the third week of development. By the thirteenth or fourteenth week, the fetal mouth can open in response to stimulation of the lower lip. During the next 2 months, the fetus will practice protruding and then pursing the lips, eventually achieving the ability to suck vigorously at 29 weeks. In the meantime, inspiratory gasps, tongue movements, lip curling, and swallowing responses will have been established (Wilentz 1968).

At birth, the taste buds are found on the roof of the mouth and in the throat, as well as on the tip, sides, and back of the tongue. Taste buds, such as those on the tip of the tongue, are particularly subject to wear and tear, and may be replaced every 2 weeks. The total number of buds diminishes over time, but there appears to be considerable reserve capacity, so there is normally little loss in the sense of taste as we age.

It has been said that taste is 90 percent smell, and to some extent that is true. The taste cells that line the buds respond to only five known qualities: sweet, sour, salty, bitter, and glutamate. The experience of taste is a complex mixture of smell, temperature, taste, and texture.

The day-old newborn responds to sweet stimuli. Suckling, the first oral function following the cry, is enhanced by the presence of lactose, the sugar in breast milk. Liquids sweetened with sugar or honey have been used widely after birth to stimulate appetite, and later to wean children from breast-

feeding. The ability to recognize a sweet taste clearly has survival value, enabling the recognition and selection of carbohydrates—a vital source of energy.

The other taste modalities are also physiologically important. A sour taste, for example, can signal an unripe fruit. However, sour substances may be involved in more subtle biochemistry, such as maintaining the body's ion balance. They also satisfy thirst and promote digestion by stimulating the secretion of saliva. A bitter taste is sufficiently unpleasant to evoke aversion, a useful response given that many bitter-tasting plant alkaloids are toxic. A salty taste indicates the presence of sodium, which is essential to maintaining the fluid balance between cells and the extracellular compartment.

Humans probably began as herbivores before the advent of hunting and gathering and retained taste-discriminating ability as an evolutionary advantage. The combination of taste and smell remains important not only for the maintenance of an optimal diet (Young 1977), but also clearly for the pleasures of eating.

Unlike taste cells, olfactory cells can respond to many thousands of odorants, including ones that have been newly synthesized. Millions of olfactory receptors line a postage-stamp-sized area in the upper part of the nose, the olfactory epithelium. Although some smells may be judged universally as unpleasant or foul, others are subject to learning and cultural conditioning. For example, a ripe Roquefort cheese may delight some people, but repel others.

The primary receptor cells of the olfactory system are neurons with fine hairs at one end that project into the olfactory epithelium to pick up the olfactory stimulus. The cells translate the stimulus to nerve impulses transmitted to the brain along the two olfactory nerves—one from each nostril. Olfactory neurons have the unusual ability to regenerate (although recent studies suggest that neurons in other parts of the adult brain may also have this ability (Johansson et al. 1999)). Olfactory pathways are widely distributed in the brain, relaying to emotional and cognitive centers in the cortex. These connections undoubtedly account for the ability of odors to stir old memories as well as stimulate a range of feelings, from fear to sexual arousal.

Touch, Temperature, and Pain

The mouth also contains large numbers of nerve endings, similar to those found elsewhere in the body, that are sensitive to touch (mechanoreceptors), hot and cold temperatures (thermoreceptors), and pain (nociceptors). The dense concentration of these

receptors in the facial skin, joints, muscle, and oral soft tissues, relayed to an image of the body mapped onto the sensory cortex of the brain, accounts for the finesse with which we can discriminate the qualities and precise location of these sensations. In particular, the periodontal ligament, which anchors the teeth in the jaws, is a tactilely sensitive tissue providing important feedback with regard to mastication (chewing) and dental occlusion (bite). As a test of this sensibility, a human hair placed between the tips of the fingers will rarely be sufficient to stimulate the nerve endings, but the same hair placed between the lips or incisors will instantly be felt.

Pain and thermal sensitivity in the teeth are transmitted through nerve endings in the pulp. Because the pulp is in a narrow canal composed of connective tissue, blood vessels, and nerves and surrounded by hard tissue, any infection or inflammation that would normally cause tissue to swell creates pressure on the pulpal nerves. That pressure, along with bacterial or immune system products that stimulate the nerve endings, produces the severe pain of pulpal infections.

Neuroscientists have long studied oral-facial pain, not only because of its importance in oral disease, but also because it provides an accessible model of pain elsewhere in the body. These investigations have greatly enriched our understanding of the basic mechanisms of pain perception and modulation. They have helped delineate the complex pathways and multiple transmitters that convey pain signals to the brain and spinal cord, as well as the mechanisms and molecules that can modulate and inhibit nociceptive input. These studies have also exploited new brain-imaging techniques to confirm the wide distribution of pain pathways and relay centers in the cerebral hemispheres and cerebellum.

This research has generated new approaches to the control of acute and chronic pain. These approaches include the use of nonsteroidal, anti-inflammatory drugs and long-acting local anesthetics for acute oral and dental pain, and the use of more potent drugs, drug combinations, and other kinds of therapies to treat chronic pain. Researchers have emphasized the importance of adequate pain control in patients with chronic pain conditions. Otherwise, the constant barrage of signals can effect long-term changes in the brain that actually worsen the pain (producing hyperalgesia) and cause normally non-painful stimuli to be perceived as painful (a condition called allodynia). Unrelieved chronic pain may also suppress the immune system.

Recently, investigators discovered a link between certain taste sensations, pain, and temperature. Their

findings indicate that capsaicin, the ingredient that makes hot peppers taste hot, binds to a receptor on the surface of nociceptors that also responds to noxious heat. The researchers have cloned the gene for the capsaicin receptor (called vanilloid receptor 1); they believe it is involved in several chronic pain conditions, especially those where inflammation plays a role, such as viral and diabetic neuropathy, rheumatoid arthritis, and oral mucositis pain caused by cancer chemotherapy or radiation (Caterina et al. 1997).

There is evidence that the prevalence of a number of pain conditions varies by gender and that men and women respond differently to different analgesic drugs. These findings have prompted studies aimed at determining whether there are sex differences in pain anatomy and neurochemistry and whether (and how) nociception is affected by sex hormones.

Speech

Human speech and language are the faculties that most distinguish us from other higher primates; they are also the links that bind people together in diverse social groups and cultures.

Central to speech are laryngeal mechanisms involving the vocal cords. Equally critical are the respiratory system, the pharynx, and the nasal and oral cavities. The tongue is the most important structure of the peripheral speech mechanisms, working in conjunction with the lips, teeth, and palate to produce a rich repertoire of sounds. Abnormalities in oral structures, from missing or malformed teeth and malocclusion to cleft lip and palate, can seriously affect articulation. The movements of speech are orchestrated by brain centers that coordinate the muscles of mastication, facial expression, and jaw movements.

Hearing impairments can also affect speech. To learn to speak, children must be able to hear others and monitor the feedback from their own voices. Congenital deafness and the serious hearing defects associated with some craniofacial syndromes (see Chapter 3) can severely compromise speech acquisition.

THE ORAL CAVITY

The mouth is the gateway to the body, performing dozens of functions that place high demands on its unique hard and soft tissues. The point of entry is the lips, which open into the oral cavity. The cheeks form the sides of the cavity, and the roof is formed by the palate, which separates the mouth from the nose above and the pharynx (throat) behind. The anterior

palate is hard, formed by underlying bone, and serves as a shield against trauma to the face and head. The posterior palate is soft, composed of muscles and connective tissue that blend into the walls of the pharynx. Hanging from the rear of the soft palate is the uvula, a mass of muscle and connective tissue. Under the tongue is the floor of the mouth, composed primarily of muscle and salivary glands. The paired tonsils and adenoids, important components of the immune system, lie at the sides of the palate and within the nasopharynx, respectively.

The pharynx opens into channels leading either to the lungs for respiration or the esophagus for further digestion and passage to the stomach. This is a point of vulnerability: should food or some other obstruction lodge in the airway, it could lead to death by asphyxiation.

Externally, the oral cavity is bounded by the maxilla (the upper jaw bone), attached to the cranium, and the mandible (the lower jaw), attached to the temporal bone of the skull by the temporomandibular joint.

The Oral Mucosa

Except for the teeth, the oral tissues are covered by a mucous membrane called the oral mucosa, which varies in color from pink to brownish purple, depending on an individual's skin color. Like skin, the oral mucosa acts as a major barrier against chemical irritants and mechanical forces; it can even withstand temperatures that would be painful to the skin. In areas subject to chewing forces and food movements, the surface layer is relatively hard, composed of epithelial cells filled with insoluble keratin, the fibrous protein found in skin, nails, hair, and animal horn. Elsewhere—in the mucosal lining of the cheeks, for example—the surface layers are softer and more flexible, enabling the mobility we need to speak, chew, and make facial expressions. To aid in their barrier function, surface mucosal cells are square-shaped and closely juxtaposed, with specialized organelles and cell products that promote cell-cell adherence. The cells can also secrete sticky molecules to plug gaps between them and further impede penetration by damaging chemicals or microorganisms. Still another type of oral mucosa forms the pebbly surface of the back and sides of the tongue. Lining the depths of these surface "papillae" are the taste buds.

Interestingly, the epithelium that lines the gingival surface completely lacks a keratin layer, yet this "naked" epithelium lies next to one of the most dense concentrations of bacteria to be found in the body.

Thus there is an opportunity for infectious agents or their byproducts to penetrate the naked epithelial barrier and initiate an inflammatory response, as happens in gingival disease.

Special cells in the basal layer of the oral mucosa generate replacements for surface cells as they wear out. The painful oral ulcers and oral mucositis that may develop in patients undergoing radiation or chemotherapy for head and neck cancer occur because these cancer-killing agents attack all cells undergoing rapid turnover, whether healthy or cancerous.

The Teeth

The most prominent features of the oral cavity are the teeth. The 20 primary, or deciduous, teeth erupt generally between 6 months and 2 to 3 years of age and are succeeded by the permanent teeth beginning at about age 6. The primary teeth enable infants to eat solid foods, aid speech development, and serve as placeholders for the permanent dentition. Keeping primary teeth healthy is important, not only in sparing an infant pain and disease, but also in preserving the dimensions of the dental arches and lessening the risk of dental caries in the permanent teeth. A period of mixed primary and permanent dentition occurs from about ages 6 to 13. There are 28 to 32 permanent teeth, depending on whether the 4 wisdom teeth (third molars), which are last to erupt, are present. Teeth are anchored in the jaws by the periodontal ligament. This ligament connects the cervix (neck) of the tooth, at the junction between the crown and root, to the gingiva. Below that, the ligament connects the outer layer of the tooth root, the cementum, to the adjacent alveolar bone (the part of the jaw bone that supports the tooth roots).

The evolutionary forces that shaped the human mouth designed an apparatus for optimal food intake. The front four upper and lower incisor teeth are chisel-shaped for biting, cutting, and tearing and exert forces of 30 to 50 pounds. The canines, or cuspids, are larger and stronger and have deeper roots than the incisors; their conical cusps are effective for ripping and tearing. The premolars, or bicuspid, and the molars are designed for heavy grinding and chewing, exerting forces as high as 200-plus pounds. The temporomandibular joint, the most complex synovial joint in the body, equips the human jaw with extraordinary mobility, enabling movements in three dimensions. Its range of motion is controlled by three sets of muscles of mastication—the masseter, temporalis, and pterygoid muscles (Oberger 1994). Chewing reduces food to

small particles and mixes it with saliva to form a bolus for swallowing.

The Salivary Glands

Saliva is the mixed product of multiple salivary glands that lie under the mucosa. The three major glands are the paired parotid, submandibular, and sublingual glands. The parotids, near the ears, secrete a watery saliva into the mouth via ducts in the cheeks. The walnut-sized submandibular glands lie in the floor of the mouth and secrete a mucous fluid. The secretions of the almond-shaped sublingual glands, also in the floor of the mouth but near the front, usually join with those of the submandibular glands. Tiny minor salivary glands are scattered within the inner surfaces of the lips, cheeks, and soft and hard palates; these secrete a mucinous saliva directly onto the soft tissue surfaces.

Saliva moistens food and provides mucinous proteins to lubricate the bolus for ease of swallowing. The combined movements of the tongue and cheeks move the bolus to the back of the mouth. Saliva also contains the enzyme amylase, which initiates the digestion of starch. By solubilizing food components and facilitating their interaction with the taste buds on the tongue and palate, saliva also contributes to taste enhancement.

Tissue Protection

The main function of saliva is not—as is commonly believed—to aid digestion, but to protect the integrity of the oral tissues. The moment a baby passes through the birth canal and takes its first breath, microbes begin to take up residence in its mouth. Later, as the teeth erupt, additional bacteria establish colonies on tooth surfaces (Mandel 1989). Nearly 500 species of microbes in all, most of which are not harmful, will colonize the oral cavity (Kroes et al. 1999). The microbes form a biofilm, in which their numbers greatly exceed the number of human inhabitants on Earth.

Millions of years before there were toothbrushes, dental floss, and water irrigators, evolutionary forces generated protective mechanisms to combat potentially harmful microbes. The physical flow of saliva helps to dislodge pathogens (viruses, bacteria, and yeast) from teeth and mucosal surfaces, just as tearing and blinking, sneezing, and coughing and expectorating clear the eye, nose, and throat. Saliva can also cause microbes to clump together so that they can be swallowed before they become firmly attached. Saliva can destroy orally shed infected

white blood cells by virtue of its low salt content: the infected cells—of higher salt content—swell and burst when exposed to fluids of lower salinity (Baron et al. 1999).

Salivary secretions, like tears and other exocrine gland secretions, are rich in antimicrobial components. Certain molecules in saliva, such as lysozyme, lactoferrin, peroxidase, and histatins, can directly kill or inhibit a variety of microbes; the histatins are particularly potent antifungal agents (Xu et al. 1991). Several salivary proteins exhibit antiviral properties, including secretory leukocyte protease inhibitor (SLPI), recently discovered to have the ability to inhibit HIV from invading cells (Shugars and Wahl 1998).

The ability of saliva to limit the growth of pathogens, in some instances even preventing them from establishing a niche in the biofilm community in the first place, is a major determinant of general as well as of oral health. When salivary flow is compromised, the gateway to the body can open wide to local as well as to systemic pathogens.

Barrier and Buffering Properties

Salivary components protect oral tissues in other ways as well. Mucins have unique properties that enable them to concentrate on mucosal surfaces and provide an effective barrier against drying and physical and chemical irritants (Tabak 1995). They act as natural waterproofing, control the permeability of the tissue surfaces, and help limit penetration of potential irritants and toxins in foods and beverages, as well as toxic chemicals and potential carcinogens in tobacco and tobacco smoke and from other sources. This barrier function complements the barrier formed by the oral mucosa itself. The mucosa has a specific permeability coefficient that can change under various conditions of stress, nutritional status, and other challenges.

Saliva contains several effective buffering systems that can help maintain a normal pH when acidic foods and beverages are introduced, thereby protecting oral tissues against acidic attack. When swallowed, these buffers protect the esophagus, helping neutralize the reflux acids of conditions such as heartburn and hiatal hernia (Sarosiek et al. 1996).

Wound Healing

Saliva also contains molecules that nurture and preserve the oral tissues, even helping them to repair and regenerate (Mandel 1989, Tabak 1995, Zelles et al. 1995). Experimental studies have shown that wound healing is significantly enhanced by saliva, in

part because of the presence of a potent molecule, epidermal growth factor (EGF) (Zelles et al. 1995). When swallowed, EGF can also protect the tissue surfaces of the esophagus (Sarosiek et al. 1996). Vascular endothelial growth factor (VEGF) has also been identified in saliva. VEGF stimulates blood vessels and may contribute to the remarkable healing capacity of oral tissues (Taichman et al. 1998, Zelles et al. 1995).

Caries Protection

Saliva also guards against dental caries (tooth decay), the disease that has been the greatest threat to teeth. Caries is caused by bacteria that generate acids that attack tooth mineral (see Chapter 3). The buffering systems in saliva, augmented by the neutralizing components urea and ammonium, counter the acid formation. The physical flow of saliva also helps flush out sugars and food particles that are the bacterial food source. Mineral salts in saliva—calcium and phosphate—can remineralize tooth enamel, effectively reversing the decay process. This regenerative function is greatly enhanced by the presence of fluoride in saliva. Finally, saliva forms a film on teeth made up of selectively adsorbed proteins that have a high affinity for tooth mineral. This acquired pellicle is insoluble and limits the diffusion of acids into the teeth and the dissolution of tooth mineral (Lamkin and Oppenheim 1993).

The Immune System

The salivary glands and the oral mucosa, along with the body's other mucosal linings and the lymphatic circulation, constitute a major component of the body's defense system—the mucosal immune system (Mestecky 1987). When the area of the oral mucosa is combined with the areas of the mucosal linings and passageways of the respiratory, gastrointestinal, urinary, and genital tracts, the total represents the largest surface area of the body—nearly 400 square meters, or 200 times larger than the total skin area.

The great majority of infectious diseases affect, or are acquired through, mucosal surfaces. Immune cells that line the mucous membranes throughout the body secrete antibodies targeted to specific disease-causing microbes (Mandel and Ellison 1985). The mucosal immune system works in concert with the bloodborne immune system to detect and dispose of foreign substances and invading microbes.

The two components of the immune system consist of molecules and cells that provide both broad and specific defense mechanisms. In the broad group

are some circulating white blood cells (monocytes and granulocytes) associated with the inflammatory response. These cells migrate to a site of injury or infection and move into damaged tissues manifesting the four signs of inflammation: swelling, heat, redness, and pain. The cells promote an increase in blood flow to begin the healing process, and they recruit other cells able to engulf and dispose of the offending organism.

The specific immune system is associated with two major classes of immune cells: T cells and B cells. T cells react to antigens (proteins associated with microbes or irritants) and can stimulate B cells to make antigen-specific antibodies. These are the Y-shaped molecules called immunoglobulins.

T cells are the instruments of cell-mediated immunity; they are able to detect telltale surface markers on diseased or foreign cells that distinguish them from normal body cells. Some T cells can kill infected cells and cancer cells directly. T cells are also involved in the rejection of organ transplants.

Certain T cells are memory cells, preserving the information from earlier encounters with specific pathogens. Thus they are able to initiate more rapid and effective responses in the event of a repeat encounter with the pathogen. Helper T cells assist in activating killer T and B cells. It is the loss of helper T cells that leads to the many infections that cause illness and death in HIV disease. Still another group of T cells, suppressor T cells, moderates the activities of both B and T lymphocytes.

Activated T cells generate and release cytokines—potent families of proteins, such as the interleukins, that can stimulate immune cells to divide, migrate, attack, and engulf invaders or participate in the inflammatory response. Other cytokines include varieties of tumor necrosis factor and adhesins (proteins that facilitate the binding of immune cells to each other or to blood vessel linings). Feedback mechanisms provide a system of checks and balances to regulate cytokine production.

The immune system interacts with the nervous and endocrine systems. For example, immune cytokines secreted into the brain can induce the fever associated with infection: the high temperature may help destroy the infectious agent. The brain's response to stress also has repercussions for the immune system. The hypothalamus-pituitary-adrenal axis is a major pathway activated in response to stress, which results in the secretion of cortisol, the stress hormone, from the adrenal glands. Cortisol promotes the body's fight-or-flight mechanisms, but via feedback loops, cortisol acts to depress immune reactions.

Much of what we know about the immune system has come from studies of serum factors, but research in the last two decades has generated much new information about mucosal immunity (McGhee and Kiyono 1999). The mucosal immune system can be divided into inductive and effector compartments. The nasal-associated lymphoreticular tissues (NALT) in the nasopharyngeal area (which includes the tonsils) and the gut-associated lymphoreticular tissues (GALT) in gut tissue are inductive regions, where foreign invaders are encountered. If, for example, infectious bacteria are swallowed, they can stimulate immune cells in GALT to circulate T and B cells through the lymph system to various effector sites in the gastrointestinal and upper respiratory tracts and in the salivary and other exocrine glands. The B cells in the gland produce antibodies, designated S-IgA, to which is attached a secretory component (Mestecky and Russell 1997). These antibodies are the dominant type found in saliva, tears, breast milk, and colostrum and in the gastrointestinal and genitourinary tracts.

The uses of the mucosal immune system extend beyond its normal surveillance and defense functions. The tissues can be used as routes for delivery of oral (swallowed) or nasal (inhaled) vaccines, as sites for gene transfer to augment host defenses, and as a means of invoking oral tolerance—the suppression of overactive or inappropriate immune responses that occur in chronic inflammatory and autoimmune diseases (Baum and O'Connell 1995, Hajishengallis and Michalek 1999).

CRANIOFACIAL ORIGINS

The extraordinary successes of research in molecular genetics over the past decade, coupled with the National Institutes of Health's project to map and sequence the human genome, have proved to be a boon in understanding craniofacial development. The use of automated gene-sequencing equipment, the Internet availability of genome databases, and the ability to transfer genes or create "knockout" animals—in which a gene of interest has been eliminated—have greatly facilitated progress. The events that govern the transformation of a fertilized human egg cell into a healthy newborn with all organs and systems in place are being unfolded at the molecular level. Families of master and regulatory genes have been identified, and their role in controlling how the body's general shape and specialized tissues and organs are formed is coming to light.

Early Development

The Three Germ Cell Layers

By the time the face and the mouth are ready to form, the human embryo is in the third week of development. The embryo has evolved from a sphere to an oval two-layered disk with a head-to-tail orientation. The outer layer is the epiblast and will become the ectodermal germ layer. A narrow groove, called the primitive streak, extends from the tail toward the center of the disk, where it ends in a spot surrounding a small depression called the primitive pit. Epiblast cells migrate toward the streak and pit, detach from the surface, and slip downward to form the two additional germ cell layers, the mesoderm and, below that, the endoderm.

The ectodermal layer gives rise to tissues that relate the body to the outside world: the nervous system; the sensory epithelium of the ears, nose, and eyes; skin, hair, nails, salivary glands, tonsils, and tooth enamel; and the pituitary, mammary, and sweat glands. At the head end, the mesodermal layer gives rise to a primitive connective tissue, called mesenchyme, which will interact with the ectoderm to form parts of the head and mouth. The remaining mesoderm develops into the muscle, cartilage, bone, and subcutaneous skin tissue of the rest of the body. The mesoderm is also the origin of the vascular and urogenital systems (except for the bladder), the spleen, and the adrenal cortex. The innermost, endodermal layer provides the linings of the gut, the respiratory system, bladder, liver, pancreas, thyroid and parathyroid glands, and parts of the middle ear.

Neural Tube and Neural Crest

Further migrations and descending movements of cells result in the formation of the notochord, a solid cord of cells along the midline that will become the backbone. The ectoderm above the notochord next thickens to form a neural plate. The sides of the plate curve up and inward to form a neural tube, beginning at the head, with fusion completed by the end of the fourth week. The tail end of the tube will form the spinal cord; the head end differentiates into the three parts of the primitive brain: the forebrain, midbrain, and hindbrain.

What happens next is of central importance to the craniofacial complex: cells that were at the edges of the neural plate break away to form neural crest cells, which migrate to the forebrain area and to the nearby branchial arches, a series of swellings on either side of the embryo, adjacent to the hindbrain. The hindbrain becomes organized into eight rhom-

bomeres, segments of future nerve tissue arranged in an orderly fashion so that the first two rhombomeres innervate branchial arch 1, and so on.

During the formation of the midbrain and hindbrain, cranial neural crest cells migrate into the developing facial areas and differentiate into neuronal and nonneuronal tissues. The neuronal tissues include the clusters of nerve cells (ganglia) that lie adjacent to the spinal cord, parts of the ganglia of four cranial nerves, and two of the meningeal layers of the brain. The nonneuronal tissues include major bones, cartilage, the dentin and cementum of teeth, and the various types of connective tissues of the craniofacial complex, as well as the muscles of the eye. The branchial arches give rise to the bones, cartilage, nerves, muscles, and blood supply of successive segments of the head and neck.

The Face and Mouth

The branchial arches play a key role in the formation of the facial structures. Toward the end of the fourth week of gestation, a primitive mouth appears. This "stomadeum" is flanked by a series of swellings, or prominences, derived from the first pair of branchial arches. A single frontonasal prominence forms the upper border of the stomadeum. On either side of this prominence are two thickened regions of ectoderm—the nasal placodes. At the sides of the stomadeum and below it are pairs of maxillary and mandibular prominences.

In the course of the next 3 weeks, differential growth and movements of the various prominences and fusions of tissues that come together at the midline will sculpt the bridge, crest, sides, and tip of the nose, the upper and lower lips, and the upper and lower jaws (Bhaskar and Orban 1990, Sadler and Langman 1995).

The external merger at the midline of a pair of prominences that helps to form the nose occurs inside the mouth as well, resulting in an intermaxillary segment that will contribute to the formation of the four upper incisors and parts of a small triangular-shaped primary palate and the upper jaw. The bulk of the palate, the secondary palate, forms from shelflike outgrowths of the maxillary prominences. These growths appear in the sixth week, and in the following week fuse along the midline above the tongue. (The tongue appears at approximately 4 weeks, the front two-thirds forming from the first branchial arch, and the posterior third from parts of the second, third, and fourth branchial arches.) The palatal shelves also fuse with the primary palate along a triangular border called the incisive foramen.

This border is considered the line of division among clefting abnormalities. Lateral cleft lip, cleft upper jaw, and clefts between the primary and secondary palates are associated with defects anterior to the incisive foramen. Cleft palate and cleft uvula occur because of defects affecting closure of the palatal shelves posterior to the foramen (Bhaskar and Orban 1990, Sadler and Langman 1995).

The Teeth

Tooth development begins in the sixth week with the appearance of an epithelial band lining the upper and lower jaws. A part of the band develops into a dental lamina, which forms a series of projections into the jaw. These are the tooth buds and correspond to the sites of deciduous teeth. The epithelial tissue of the bud develops into an enamel organ that forms a cap over tissue that is differentiating in the jaw to become the dental papilla. The two structures—the enamel organ, derived from the epithelium, and the dental papilla, derived from neural crest mesenchyme—constitute the tooth germ.

With further development, the tooth germ assumes a bell shape and separates from the oral epithelium. At the same time, the internal epithelial layer of the enamel organ undergoes a series of infoldings that will shape the future crown of the tooth.

Mineralization of the tooth begins at the late bell stage. The first mineralized tissue to form is dentin, which provides the foundation for the deposition of enamel. The differentiation of the odontoblasts (the dentin-producing cells) depends on organizing influences from enamel organ cells. Thus the development of these two different hard tissues is a mutually dependent process.

As dentin is laid down, the odontoblasts move toward the center of the papilla, trailing thin cellular processes, which become embedded in the mineralized matrix. When dentin formation is completed, dentin completely surrounds the pulp, protecting it from injury. The enamel layer of the tooth starts to form soon after the first dentin appears, synthesized by special enamel-forming cells, or ameloblasts, which develop from the enamel organ. The tooth root, and its outer layer of cementum, form only after the crown erupts.

Genetic Controls

Only in the last decade have scientists begun to understand how certain genes and gene families control embryonic development. Their findings have

come from detailed studies of species ranging from fruit flies, nematodes, and zebrafish to frog, chick, mouse, and human embryos. In many cases, the simpler organism has been the source of discoveries of genes or developmental processes that are highly conserved in the course of evolution (Alberts et al. 1994).

Research on the fruit fly, for example, has revealed that particular families of genes are responsible for the fundamental head to thorax to tail patterning of the fly's body. Another set of genes determines the back-to-front positioning of organs, and a third set subdivides this general body plan into a series of discrete segments. With further development, yet another family of genes confers a positional memory on the cells within a segment. These "homeotic selector" genes ensure that cells in one part of a particular segment "know" that they are destined to be wings and not legs, or to be eyes and not antennae. In flies the homeotic genes are known as *Hom* genes. Their arrangement on the fly chromosome is ordered with genes at one end of the chromosome specifying the developmental destiny of cells in the most anterior segments of the fly's body and genes at the other end specifying the fate of cells in the most posterior segments.

In the course of evolution, mammals have developed four overlapping sets of positional memory gene clusters homologous to the fly's single *Hom* complex. The four mammalian *Hox* gene families are ordered in a similar anterior-posterior fashion along four different chromosomes. The mammalian genes appear to operate like the *Hom* genes: they code for DNA-binding proteins that control gene expression. The similarity from fly to human is particularly evident when maps of the expression domains of *Hom* genes in anterior segments of the fly embryo are compared to maps of *Hox* gene expression as seen in the rhombomeres and branchial arches of mammals.

Molecular genetic studies of flies and other non-mammalian species show some variation in how and when the basic body patterns and repeating segments are formed. Sometimes the head-to-tail pattern is laid down in the egg cell before fertilization—dictated by egg polarity genes. Although egg polarity genes do not operate in humans, mutations have been found in a human gene homologous to the fly egg polarity gene and account for serious syndromes in which there are defects in anterior organs, such as the pituitary gland and heart.

None of these developmental controls work in isolation. Much remains to be understood about the genetic clock that determines when and where developmental genes act, how they interact, and what

mechanisms are used to sustain as well as terminate their function. The systems that govern programmed cell death are also important: normal development depends as much on the elimination of cells as it does on the orderly movement, proliferation, and differentiation of cells.

When it comes to processes that control the development of particular tissues or organs—bones, skin, or heart—developmental biologists observe that there is often an “organizer,” that is, a cell or set of cells that initiates the process. The organizer induces changes in the behavior of neighboring cells through cell-cell interactions, so that these cells develop into the specified type—bone or skin or heart muscle. The interaction with the neighboring cell is often in the form of a signaling molecule, such as a growth factor (e.g., transforming growth factor β , epidermal growth factor, fibroblast growth factor) that attaches to a receptor on the surface membrane of the recipient cell. This interaction is translated to the interior of the cell, where a chain of molecular interactions eventually reaches the cell nucleus to effect gene expression. One of the more startling discoveries of the past decade has been the finding that a series of mutations, each associated with a change in only one nucleotide of the gene for the fibroblast growth factor receptor—a so-called point mutation—accounts for a range of organ defects seen in at least a half dozen craniofacial syndromes. Interestingly, all these syndromes include craniosynostosis, a premature closure of the bones that form the skull.

THE AGING OF CRANIOFACIAL TISSUES

Normal aging describes the developmental processes that begin at conception, continue in childhood, and merge gradually into maturation and senescence. The milestones of development such as the age when children teethe, begin to walk, talk, enter puberty, attain their full height, and so on, are under genetic and hormonal controls, subject to important environmental factors such as nutrition and exercise. Despite the complexity and interrelationships of the variables involved, a reasonably accurate picture of normal age-related changes in the craniofacial complex is emerging (Ship 1999).

Barring major illness or injury, destructive behaviors, or severe or unusual environmental circumstances, the cells, tissues, and fluids of the face and mouth are hardy survivors, eminently durable and functional over a long life span. For any given individual the combination of life experience and

lifestyle (including medical and dental history) creates a unique craniofacial portrait, one that inspired George Orwell to remark, “By the age of fifty, a man gets the face he deserves.”

The Teeth

One of the more dramatic discoveries in biomedical science in the twentieth century has been the realization that tooth loss is not an inevitable consequence of aging, but the result of disease or injury. Aging does produce a number of other dental changes, however. Teeth change in form and color with age. Wear and attrition alter the biting and chewing surfaces, as do food choices and oral habits. The altered surface structure produces a different pattern of light reflection in older teeth, resulting in some yellowing and a general loss of translucency (Mjor 1986). Fully formed enamel is acellular, hence there is no metabolic activity or turnover as occurs in skin, for example. Dentin and cementum have limited cellular activity. In contrast, tooth pulp and periodontal ligament undergo relatively high levels of tissue turnover.

Tooth surfaces can be eroded by chemical dissolution from fruit acids and from acids from sugars in foods such as soft drinks and candies. This destructive process can occur at any age, resulting in loss of translucency as well as some tissue loss from demineralization (Zero 1996). Countering the erosive forces are the natural components in saliva that help remineralize the enamel surface, a process that is enhanced when fluoride is present (see “Caries Protection” above).

The cementum increases in thickness with age. Gingival recession caused by normal aging exposes the cementum to the oral environment (and is the origin of the expression “long in the tooth”). The exposed cementum can often be worn away mechanically, exposing the underlying dentin, which can then become hypersensitive. Dentin responds through a series of protective changes that work to close off the connections between dentin and nerves in the pulp, reducing transmission of painful stimuli.

The Jaws

The bones of the maxilla and mandible that support the teeth, called the alveolar processes, are, like bone elsewhere in the body, subject to cellular turnover in a coordinated process of bone resorption and

formation. Alveolar bone is well adapted to mechanical stresses, and changes continuously during facial growth, tooth eruption, tooth wear, and tooth loss. This lifelong adaptation makes orthodontic treatments to reposition teeth in adults possible.

Because the primary function of alveolar bone is to support the teeth, the loss of teeth will lead to bone atrophy, making prosthetic replacements difficult. The rate of bone loss is affected by both local disease such as periodontal disease and systemic conditions such as osteoporosis (Bhaskar 1991).

The Oral Mucosa

The oral mucosa appears to age in much the same way as skin does. The oral epithelium thins and becomes less hydrated, increasing vulnerability to injury. The rate of cell division is slower, but the basic cell architecture and patterning of cell types throughout the oral cavity are maintained. It is not certain to what extent these changes are a natural consequence of aging; they may be due to altered protein synthesis or lowered responsiveness to regulatory molecules. They may also be an effect of diminished vascularity, which could limit cellular access to oxygen and nutrients (Mjor 1986).

Overall immune system function deteriorates with age, and it is likely that mucosal immunity does as well. Such a decline could result in an increased risk of transmission of infectious agents across the mucosa and probably contributes to delayed wound healing in oral tissues with aging.

Sensory and Motor Functioning

The high density of sensory nerve endings in the craniofacial tissues and their functional abilities are well-preserved in aging. There may be minor increases in threshold detection levels or in judgments of intensity, but, for the most part, sensory cells can turn over or have a built-in reserve capacity that allows for near-optimal functioning in aging. The exception is olfaction, which declines in both men and women with age. This decrement in smell may lead to some dissatisfaction with how foods taste and increased use of flavor enhancers to compensate. But for most people, the ability to enjoy food is not appreciably diminished as time goes by. Any dramatic change in sensory function—complaints of a continued unpleasant taste or smell or the sudden complete loss of a sensory modality—should be taken seriously as a sign of possible oral or systemic disease

or a side effect of medication and not dismissed as a natural by-product of aging.

The distribution of motor fibers in the craniofacial tissues is also abundant and sufficiently fine-tuned to allow for a full range of movement of the tongue, jaws, and oral-facial muscles. There is some loss of muscle tone in aging, along with changes in tongue shape and function in articulating specific speech sounds. Subtle changes may also occur in preparing food for swallowing. As with sensory changes, these developments do not seriously interfere with motor function in healthy older adults.

The Salivary Glands

Studies of normative aging indicate that individuals vary in the quantity of “whole” saliva they produce. Whole saliva consists of the secretions of the various salivary glands plus other oral contents, such as cells shed from the mucosa. These individual patterns are consistent across the life span. In healthy adults, there is no diminution in the production of saliva from the major salivary glands in the course of aging.

This constancy may seem surprising given the morphological changes documented in aging salivary glands. Both the parotid and the submandibular glands lose between 20 and 30 percent of their essential tissue volume in the course of aging. The loss primarily affects the acinar components, the cells that secrete saliva. Increases in the number of ductal cells and in fat, vascular, and connective tissues compensate for this loss, however—evidence of the remarkable functional reserve capacity of the glands, which enables them to maintain a stable salivary output across the life span (Baum 1986).

In contrast, studies of age-related changes in the chemistry of salivary secretions suggest that there are significant reductions in the concentration of mucins from the submandibular gland (Navazesh et al. 1992), which could result in reduced lubrication and contribute to a sensation of mouth dryness. There are also subtle changes in the protective ability of salivary secretory IgA antibody (Smith et al. 1987).

FINDINGS

Natural selection has served *Homo sapiens* well in evolving a craniofacial complex with remarkable functions and abilities to adapt, enabling the organism to meet the challenges of an ever-changing environment. An examination of the various tissues reveals elaborate designs that have evolved to serve the basic needs and functions of a complex mammal as well as those that are uniquely human, such as

speech. The rich distribution of nerves, muscles, and blood vessels in the region as well as extensive endocrine and immune system connections is an indication of the vital role of the craniofacial complex in adaptation and survival over a long life span. In particular,

- Genes controlling the basic patterning and segmental organization of human development, and specifically the craniofacial complex, are highly conserved in nature. Mutated genes affecting human development have counterparts in many simpler organisms.

- There is considerable reserve capacity or redundancy in the cells and tissues of the craniofacial complex, so that if they are properly cared for, the structures should function well over a lifetime.

- The salivary glands and saliva subserve tasting and digestive functions and also participate in the mucosal immune system, a main line of defense against pathogens, irritants, and toxins.

- Salivary components protect and maintain oral tissues through antimicrobial components, buffering agents, and a process by which dental enamel can be remineralized.

REFERENCES

- Alberts B, Bray D, Lewis J, Raff M, Roberts K, Watson JD. Cellular mechanisms of development. In: Molecular biology of the cell. 3rd ed. New York: Garland; 1994. p. 1036-137.
- Baron S, Poast J, Cloyd MW. Why is HIV rarely transmitted by oral secretions? Saliva can disrupt orally shed, infected leukocytes. *Arch Intern Med* 1999 Feb 8;159(3):303-10.
- Bhaskar SN. Maxilla and mandible (alveolar process). In: Bhaskar SN, editor. *Orban's oral histology and embryology*. St. Louis (MO): Mosby Year Book; 1991. p. 239-59.
- Bhaskar SN, Orban BJ. *Orban's oral histology and embryology*. 11th ed. St. Louis (MO): Mosby Year Book; 1990. Chapter 1, Development of the face and oral cavity.
- Baum BJ. Age changes in salivary glands and salivary secretions. In Holm-Pederson P, Løe H, editors. *Geriatric dentistry*. Copenhagen: Munksgaard; 1986. p. 114-22.
- Baum BJ and O'Connell BC. The impact of gene therapy on dentistry. *J Am Dent Assoc* 1995;126:179-89.
- Caterina MJ, Schumacher MA, Tominaga M, Rosen TA, Levine JD, Julius D. The capsaicin receptor: a heat-activated ion channel in the pain pathway. *Nature* 1997;389:816-24.
- Hajishengallis G, and Michalek SM. Current status of a mucosal vaccine against dental caries. *Oral Microbiol Immunol* 1999;14:1-20.
- Johansson CB, Momma S, Clarke DL, Risling M, Lendahl U, Frisen J. Identification of a neural stem cell in the adult mammalian central nervous system. *Cell* 1999 Jan 8;96(1):25-34.
- Kroes I, Lepp PW, Relman DA. Bacterial diversity within the human subgingival crevice. *Proc Natl Acad Sci USA* 1999 Dec 7;96(25):14547-52.
- Lamkin MS, Oppenheim FG. Structural features of salivary function. *Crit Rev Oral Biol Med* 1993;4(3-4):251-9.
- Mandel ID. The role of saliva in maintaining oral homeostasis. *J Am Dent Assoc* 1989 Aug;119:298-304.
- Mandel ID, Ellison SA. The biological significance of the nonimmunoglobulin defense factors. In: Pruitt K, Tenovuo J, editors. *The lactoperoxidase system*. New York: Marcel Dekker; 1985. p. 1-14.
- McGhee JR, Kiyono H. The mucosal immune system. In: Paul WE, editor. *Fundamental immunology*. 4th ed. New York: Lippincott-Raven; 1999. p. 909-45.
- Mestecky J. The common mucosal immune system and current strategies for induction of immune responses in external secretions. *J Clin Immunol* 1987 Jul;7(4):265-76.
- Mestecky J, Russell MW. Mucosal immunoglobulins and their contribution to defense mechanisms: an overview. *Biochem Soc Trans* 1997 May;25(2):457-62.
- Mjor IA. Age changes in the teeth. In: Holm-Pedersen P, Løe H, editors. *Geriatric dentistry*. Copenhagen: Munksgaard; 1986. p. 94-101.
- Navazesh M, Mulligan RA, Kipnis V, Denny PA, Denny PC. Comparison of whole saliva flow rates and mucin concentrations in healthy Caucasian young and aged adults. *J Dent Res* 1992 Jun;71(6):1275-8.
- Oberg S. *Dissector for Netter's atlas of human anatomy: discussions*. Vol 2. Summit (NJ): Ciba-Geigy; 1994.
- Sadler TW, Langman J. *Langman's medical embryology*. Baltimore: Williams & Wilkins; 1995.
- Sarosiek J, Scheurich CJ, Marcinkiewicz M, McCallum RW. Enhancement of salivary esophagoprotection: rationale for a physiological approach to gastroesophageal reflux disease. *Gastroenterology* 1996;110:675-81.
- Ship JA. The oral cavity. In: Hazzard WR et al., editors. *Principles of geriatric medicine and gerontology*. New York: McGraw-Hill; 1999.
- Shugars DC, Wahl SM. The role of the oral environment in HIV-1 transmission. *J Am Dent Assoc* 1998 Jul;129(7):851-8.
- Smith DJ, Taubman MA, Ebersole JL. Ontogeny and senescence of salivary immunity. *J Dent Res* 1987 Feb(2);66:451-6.
- Tabak L. In defense of the oral cavity: structure, biosynthesis, and functions of salivary mucins. *Annu Rev Physiol* 1995;57:547-64.

- Taichman NS, Cruchley AT, Fletcher LM, Hagi-Pavli EP, Paleolog EM, Abrams WR, Booth V, Edwards RM, Malamud D. Vascular endothelial growth factor in normal salivary glands and saliva: a possible role in the maintenance of mucosal homeostasis. *Lab Invest* 1998 Jul;78(7):869-75.
- Wilentz J. *The senses of man*. New York: Thomas Y. Crowell; 1968.
- Xu T, Levitz SM, Diamond RD, Oppenheim FG. Anticandidal activity of major human salivary histatins. *Infect Immun* 1991 Aug;59(8):2549-54.
- Young PT. Role of hedonic processes in development of sweet taste. In: Weiffenbach, JM, editor. *Taste and development—the genesis of sweet preference*. Bethesda (MD): National Institutes of Health, Public Health Service, U.S. Department of Health, Education and Welfare Report no. NIH 77-1068; 1977. p. 399-417.
- Zelles T, Purushotham KR, Macauley SP, Oxford GE, Humphreys-Beher MG. Saliva and growth factors: the fountain of youth resides in us all. *J Dent Res* 1995 Dec;74(12):1826-32.
- Zero DT. Etiology of dental erosion—extrinsic factors. *Eur J Oral Sci* 1996 Apr;104(2 Pt 2):162-77.

PART TWO

What Is the Status of Oral Health in America?

To begin to answer this question, Chapter 3 guides the reader through a discussion of oral diseases and disorders in such categories as infections, inherited disorders, and neoplasms. Whether or not an individual succumbs to the disease or disorder in question depends on subtle interactions of genetic, environmental, and behavioral variables. Risk factors common to systemic diseases and disorders, such as tobacco use, excessive alcohol use, and inappropriate dietary practices, also contribute to many oral diseases and disorders. As more details on the causes of diseases unfold, specific strategies for disease prevention can be developed.

Chapter 4 describes the magnitude of the problem facing the nation due to oral diseases and disorders. These conditions are prevalent and complex, and they affect individuals across the life span. Although major improvements have been seen nationally for most Americans, disparities exist in some population groups as classified by age, sex, income, and race/ethnicity. National and state-based epidemiologic data presented against the backdrop of demographic and socioeconomic variables provide some information on racial and ethnic minorities, but serious shortcomings exist. The paucity of data at national, state, and local levels extends to other populations, including individuals with disabilities, those with alternate sexual orientation, migrant populations, and the homeless, and limits the capacity to fully document the magnitude of the problem and develop needed programs. The chapter provides a basis for understanding disparities in oral health by presenting available data on dental visits. More work is needed to understand the dimensions of oral health problems in the United States and the reasons for differences among populations.

Diseases and Disorders

As the gateway to the body, the mouth is challenged by a constant barrage of invaders—bacteria, viruses, parasites, fungi. Thus infectious diseases, notably dental caries and periodontal diseases, predominate among the ills that can compromise oral health. Injuries take their toll as well, with the face and head particularly vulnerable to sports injuries, motor vehicle crashes, violence, and abuse. Less common but very serious are oral and pharyngeal cancers, with a 5-year survival rate of hardly better than 50 percent (Kosary et al. 1995). Birth defects and developmental disorders frequently affect the craniofacial complex. These appear most commonly as isolated cases of cleft lip or palate, but clefting or other craniofacial defects can also be part of complex hereditary diseases or syndromes. Additionally, acute and chronic pain can affect the oral-facial region, particularly in and around the temporomandibular (jaw) joint, and accounts for a disproportionate amount of all types of pain that drive individuals to seek health care.

Many systemic diseases such as diabetes, arthritis, osteoporosis, and AIDS, as well as therapies for systemic diseases, can directly or indirectly compromise oral tissues. The World Health Organization's *International Classification of Diseases and Stomatology* currently lists more than 120 specific diseases, distributed in 10 or more classes, that have manifestations in the oral cavity (WHO 1992).

This chapter concentrates on six major oral disease categories: dental and periodontal infections; mucosal disorders; oral and pharyngeal cancers; developmental disorders; injuries; and a sampling of chronic and disabling conditions, including Sjögren's syndrome and oral-facial pain.

DENTAL AND PERIODONTAL INFECTIONS

The most common oral diseases are dental caries and the periodontal diseases. Individuals are vulnerable to dental caries throughout life, with 85 percent of adults aged 18 and older affected. Periodontal diseases are most often seen in maturity, with the majority of adults experiencing some signs and symptoms by the mid-30s. Certain rare forms of periodontal disease affect young people. The major oral health success story of the past half century is that both caries and periodontal diseases can be prevented by a combination of individual, professional, and community measures.

Dental Caries

The word *caries* derives from the Latin for rotten, and many cultures early on posited a tooth worm as the cause of this rotteness. By the twentieth century, caries came to describe the condition of having holes in the teeth—cavities. This description, although not incorrect, is misleading. In actuality, a cavity is a late manifestation of a bacterial infection.

The bacteria colonizing the mouth are known as the *oral flora*. They form a complex community that adheres to tooth surfaces in a gelatinous mat, or *biofilm*, commonly called dental plaque. A cariogenic biofilm at a single tooth site may contain one-half-billion bacteria, of which species of mutans streptococci are critical components. These bacteria are able to ferment sugars and other carbohydrates to form lactic and other acids. Repeated cycles of acid generation can result in the microscopic dissolution of minerals in tooth enamel and the formation of an opaque white or brown spot under the enamel surface (Mandel 1979). Frequency of carbohydrate consumption (Gustafsson et al. 1954), physical

characteristics of food (e.g., stickiness), and timing of food intake (Burt and Ismail 1986) also play a role.

The essential role of bacteria in caries initiation was established in landmark experiments in the 1950s. Investigators observed that germ-free animals fed high-sugar diets remained caries-free until the introduction of mutans streptococci (a particular group of bacterial strains having a number of common characteristics, and which adhere tightly to the tooth). Later experiments demonstrated the transmissibility of the bacteria from mother to litter and from caries-infected to uninfected cage-mates (Fitzgerald and Keyes 1960). Species of *Lactobacillus*, *Actinomyces*, and other acid-producing streptococci within the plaque may also contribute to the process (Bowden 1990).

If the caries infection in enamel goes unchecked, the acid dissolution can advance to form a cavity that can extend through the dentin (the component of the tooth located under the enamel) to the pulp tissue, which is rich in nerves and blood vessels. The resulting toothache can be severe and often is accompanied by sensitivity to temperature and sweets. Treatment requires endodontic (root canal) therapy. If untreated, the pulp infection can lead to abscess, destruction of bone, and spread of the infection via the bloodstream.

Dental caries can occur at any age after teeth erupt. Particularly damaging forms can begin early, when developing primary teeth are especially vulnerable. This type of dental caries is called *early childhood caries* (ECC). Some 6 out of 10 children in the United States have one or more decayed or filled primary teeth by age 5 (U.S. Department of Health and Human Services, National Center for Health Statistics 1997). ECC may occur in children who are given pacifying bottles of juice, milk, or formula to drink during the day or overnight. The sugar contents pool around the upper front teeth, mix with cariogenic bacteria, and give rise to rapidly progressing destruction (Ripa 1988). Other risk factors for ECC include arrested development of tooth enamel, chronic illness, altered salivary composition and volume (resulting from the use of certain medications or malnourishment), mouth breathing, and blockage of saliva flow in a bottle-fed infant (Bowen 1998, Seow 1998).

Although there have been continuing reductions in dental caries in permanent teeth among children and adolescents over the past few decades, caries prevalence in the primary dentition may have stabilized or increased slightly in some population groups (Petersson and Bratthall 1996, Rozier 1995). Reductions in caries in permanent teeth also have

been proportionately greater on the smooth surfaces rather than on the pit-and-fissure surfaces characteristic of chewing surfaces. The gingival tissues tend to recede over time, exposing the tooth root to cariogenic bacteria that can cause root caries. An important risk factor for root caries in older people is the use of medications that inhibit salivary flow, leading to dry mouth (xerostomia).

Saliva contains components that can directly attack cariogenic bacteria, and it is also rich in calcium and phosphates that help to remineralize tooth enamel. Demineralization of enamel occurs when pH levels fall as a result of acid production by bacteria. It can be reversed at early stages if the local environment can counteract acid production, restoring pH to neutral levels. Remineralization can occur through the replacement of lost mineral (calcium and phosphates) from the stores in saliva. Fluoride in saliva and dental plaque and the buffering capacity of saliva also contribute to this process. Indeed, it is now believed that fluoride exerts its chief caries-preventive effect by facilitating remineralization. Several studies have demonstrated that remineralization results in an increase in tooth hardness and mineral content, rendering the tooth surface more resistant to subsequent acid attack (Larsen 1987, Linton 1996, Retief 1983, Shannon 1978, Vissink et al. 1985, White 1988).

Overt caries lesions develop when there is insufficient time for remineralization between periods of acidogenesis, or when the saliva production is compromised. Over 400 medications list dry mouth as a side effect, notably some antidepressants, antipsychotics, antihistamines, decongestants, antihypertensives, diuretics, and antiparkinsonian drugs (Sreebny et al. 1992). The effects of xerostomia may be particularly severe in cancer patients receiving radiation to the head or neck because the rays can destroy salivary gland tissue rather than simply inhibiting salivary secretion.

The professional application of dental sealants (plastic films coated onto the chewing surfaces of teeth) is an important caries-preventive measure that complements the use of fluorides. The films prevent decay from developing in the pits and fissures of teeth, channels that are often inaccessible to brushing and where fluoride may be less effective.

The rate of caries progression through enamel is relatively slow (Berkey 1988, Ekanayake 1987, Schwartz et al. 1984) and may be slower in patients who have received regular fluoride treatment or who consume fluoridated water (Pitts 1983, Schwartz et al. 1984). Because a large percentage of enamel lesions remain unchanged over periods of 3 to 4 years, and

because progression rates through dentin are comparably slow (Craig et al. 1981, Emslie 1959, Kolehmainen and Rytömaa 1977), the application of infection control and monitoring procedures to assess caries risk status, lesion activity status, evidence of lesion arrest, and evidence of lesion remineralization over extended periods of time is recommended.

Experts believe that the earlier mutans streptococci are acquired in infancy, the higher the caries risk. Most studies indicate that infants are infected before their first birthday, around the time the first incisors emerge. However, one study found the median age of acquisition to be 26 months, coinciding with the emergence of the primary molars (Bowen 1998, Caufield et al. 1993, Seow 1998). DNA fingerprinting has demonstrated that the source of transmission is usually the mother (Caufield et al. 1993).

It is not clear why some individuals are more susceptible and others more resistant to caries. Genetic differences in the structure and biochemistry of enamel proteins and crystals (Slavkin 1988), as well as variations in the quality and quantity of saliva and in immune defense mechanisms are among the factors under study. Analysis of mutans streptococci genomes may also shed light, indicating which species are particularly virulent and which genes contribute to that virulence.

Even the most protective genetic endowment and developmental milieu are unlikely to confer resistance to decay in the absence of positive personal behaviors. These include sound dietary habits and good oral hygiene, including the use of fluorides, and seeking professional care. There are indications, however, that some destructive oral habits are on the rise, such as the use of smokeless (spit) tobacco products by teenage boys. Although the chief concern here lies in the long-term risk for oral cancers, spit tobacco that contains high levels of sugar is also associated with increased levels of decay of both crown and root surfaces (Tomar and Winn 1998).

Periodontal Diseases

Like dental caries, the periodontal diseases are infections caused by bacteria in the biofilm (dental plaque) that forms on oral surfaces. The basic division in the periodontal diseases is between gingivitis, which affects the gums, and periodontitis, which may involve all of the soft tissue and bone supporting the teeth. Gingivitis and milder forms of periodontitis are common in adults. The percentage of individuals with moderate to severe periodontitis, in which the

destruction of supporting tissue can cause the tooth to loosen and fall out, increases with age.

Gingivitis

Gingivitis is an inflammation of the gums characterized by a change in color from normal pink to red, with swelling, bleeding, and often sensitivity and tenderness. These changes result from an accumulation of biofilm along the gingival margins and the immune system's inflammatory response to the release of destructive bacterial products. The early changes of gingivitis are reversible with thorough toothbrushing and flossing to reduce plaque. Without adequate oral hygiene, however, these early changes can become more severe, with infiltration of inflammatory cells and establishment of a chronic infection. Biofilm on tooth surfaces opposite the openings of the salivary glands often mineralizes to form calculus or tartar, which is covered by unmineralized biofilm—a combination that can exacerbate local inflammatory responses (Mandel 1995). A gingival infection may persist for months or years, yet never progress to periodontitis.

Gingival inflammation does not appear until the biofilm changes from one composed largely of gram-positive streptococci (which can live with or without oxygen) to one containing gram-negative anaerobes (which cannot live in the presence of oxygen). Numerous attempts have been made to pinpoint which microorganisms in the supragingival (above the gum line) plaque are the culprits in gingivitis. Frequently mentioned organisms include *Fusobacterium nucleatum*, *Veillonella parvula*, and species of *Campylobacter* and *Treponema*. But as Ranney (1989) notes, "The complexity of the results defies any attempt to define a discrete group clearly and consistently associated with gingivitis."

Gingival inflammation may be influenced by steroid hormones, occurring as puberty gingivitis, pregnancy gingivitis, and gingivitis associated with birth control medication or steroid therapy. The presence of steroid hormones in tissues adjacent to biofilm apparently encourages the growth of certain bacteria and triggers an exaggerated response to biofilm accumulation (Caton 1989). Again, thorough oral hygiene can control this response.

Certain prescription drugs can also lead to gingival overgrowth and inflammation. These include the antiepileptic drug phenytoin (Dilantin); cyclosporin, used for immunosuppressive therapy in transplant patients; and various calcium channel blockers used in heart disease. Treatment often requires surgical removal of the excess tissue followed by appropriate personal and professional oral health care.

A form of gingivitis common 50 years ago but relatively rare today is acute necrotizing ulcerative gingivitis, also known as Vincent's infection or trench mouth. This aggressive infection is characterized by destruction of the gingiva between the teeth, spontaneous bleeding, pain, and oral odor. People under extreme stress have an increased susceptibility. Spirochetes and other bacteria have been found in the connective tissue of those affected. An association between smoking and this type of gingivitis is well recognized and was demonstrated as early as 1946 (Pindborg 1947, 1949). This condition has been seen in some HIV-positive patients (Murray 1994). Treatment requires a combination of professional periodontal treatment and antibacterial therapy along with professional smoking cessation assistance as appropriate.

Adult Periodontitis

The most common form of adult periodontitis is described as general and moderately progressing; a second form is described as rapidly progressing and severe, and is often resistant to treatment. The moderately progressive adult form is characterized by a gradual loss of attachment of the periodontal ligament to the gingiva and bone along with loss of the supporting bone. It is most often accompanied by gingivitis (Genco 1990). It is not necessarily preceded by gingivitis, but the gingivitis-related biofilm often seeds the subgingival plaque. The destruction of periodontal ligament and bone results in the formation of a pocket between the tooth and adjacent tissues, which harbors subgingival plaque. The calculus formed in the pocket by inflammatory fluids and minerals in adjacent tissues is especially damaging (Mandel and Gaffar 1986).

The severity of periodontal disease is determined through a series of measurements, including the extent of gingival inflammation and bleeding, the probing depth of the pocket to the point of resistance, the clinical attachment loss of the periodontal ligament measured from a fixed point on the tooth (usually the cemento-enamel junction), and the loss of adjacent alveolar bone as measured by x-ray (Genco 1996). Severity is determined by the rate of disease progression over time and the response of the tissues to treatment.

Adult periodontitis often begins in adolescence but is usually not clinically significant until the mid-30s. Prevalence and severity increase but do not accelerate with age (Beck 1996). One view proposes that destruction occurs at a specific site during a defined period, after which the disease goes into remission (Socransky et al. 1984). The current view

is that the disease process may not be continuous but rather progresses in random bursts in which short periods of breakdown of periodontal ligament and bone alternate with periods of quiescence. These episodes occur randomly over time and at random sites in the mouth. Part of the difficulty in determining the pattern of progression reflects variation in the sensitivity of the instruments used to measure the loss of soft tissue and bone. The latest generation of probes finds evidence of both continuous and multiple-burst patterns of loss in different patients and at different times (Jeffcoat and Ready 1991).

Most researchers agree that periodontitis results from a mixed infection but that a particular group of gram-negative bacteria are key to the process and markedly increase in the subgingival plaque. The bacteria most frequently cited are *Porphyromonas gingivalis*, *Prevotella intermedia*, *Bacteroides forsythus*, *Treponema denticola*, and *Actinobacillus actinomycetemcomitans* (Genco 1996). Their role in disease initiation and progression is determined in part by their "virulence factors." These include the ability to colonize subgingival plaque, generate products that can directly injure tissues, and elicit an inflammatory or immune response. The potentially noxious bacterial products include hydrogen sulfide, polyamines, the fatty acids butyrate and propionate, lipopolysaccharide (also known as endotoxin), and a number of destructive enzymes. The interaction of this arsenal with the host response is at the core of periodontal pathology (Genco 1992, Socransky and Haffajee 1991, 1992). Sequencing of the genomes of several key periodontal pathogens is under way and should provide further insight into these pathogens as well as catalyze new treatment approaches.

Delicate Balances. Neutrophils (a type of white blood cell) and antibodies are the major immune defenses against bacterial attack. Neutrophils move to the site of infection, where they engulf bacteria and elaborate antibacterial agents and enzymes to destroy bacteria. Although stimulation of the immune system to attack the offending bacteria is generally protective, immune hyperresponsiveness and hypersensitivity can be counterproductive, leading to the destruction of healthy tissue. Nevertheless, the neutrophil/antibody axis is critical for full protection against periodontal diseases (Genco 1992).

Also important is the release of certain potent molecules called cytokines and prostaglandins, especially prostaglandin E₂ (PGE₂) which can contribute to tissue destruction. Cytokines are proteins secreted by immune cells that help regulate immune responses and also affect bone, epithelial,

and connective tissues. Most prominent in periodontal diseases are interleukin 1 (IL-1), tumor necrosis factor α (TNF- α), and interferon γ (IFN- γ). These cytokines mediate the processes of bone resorption and connective tissue destruction.

Susceptibility and Resistance. PGE₂ may play a central role in the tissue destruction that occurs in periodontal diseases. Levels of PGE₂ in periodontal tissue are low or undetectable in health, increase in gingivitis, and rise significantly in periodontitis. Now there is increasing evidence that the level of PGE₂ produced in response to bacterial challenge (especially by endotoxin) can be used as a measure of susceptibility (Offenbacher et al. 1993).

Presumably, the level of PGE₂ production is subject to genetic influence. Studies of identical and fraternal twins, either reared together or apart, provide evidence that genetic factors may indeed influence susceptibility or resistance to the common adult form of periodontitis (Michalowicz 1994). Recently, a commercial test for a genetic marker of susceptibility has been introduced. The marker is associated with increased production of a particular form of interleukin 1 β (IL-1 β) when stimulated by periodontopathic bacteria (Kornman et al. 1997). Newman (1996) found that nonsmoking adults who are positive for the marker are 6 to 19 times more likely to develop severe periodontitis.

Susceptibility to adult periodontitis has also been explored in relation to a variety of behavioral and demographic variables as well as to the presence of other diseases. One of the strongest behavioral associations is with tobacco use. The risk of alveolar bone loss for heavy smokers is 7 times greater than for those who have not smoked (Grossi et al. 1995). Cigarette smoking also may impair the normal host response in neutralizing infection (Seymour 1991), resulting in the destruction of healthy periodontal tissues adjacent to the site of infection (Lamster 1992). Smokers also have decreased levels of salivary and serum immunoglobulins to *Prevotella intermedia* and *Fusobacterium nucleatum* (Bennet and Reade 1982, Haber 1994) and depressed numbers of helper T cells as well (Costabel et al. 1986). Finally, smoking alters the cells that engulf and dispose of bacteria—neutrophils and other phagocytes—affecting their ability to clear pathogens (Barbour et al. 1997).

Epidemiologic studies have found that such additional factors as increasing age, infrequent dental visits, low education level, low income, co-morbidities, and inclusion in certain racial or ethnic populations are associated with increased prevalence of

periodontitis (Page 1995). It is important that epidemiologic studies also take into consideration the fact that tobacco use, oral hygiene, professional prophylaxis, and routine dental care are correlated to socioeconomic status, as are race and ethnicity. Sex is another factor. Males tend to have higher levels of periodontal diseases, presumably because of a history of greater tobacco use and differences in personal care and frequency of dental visits. However, female hormones may play a protective role (as they do in protecting against osteoporosis) (Genco 1996).

Certain systemic diseases heighten susceptibility. Epidemiological studies have confirmed that patients with diabetes mellitus, both type 1 and type 2, are more susceptible to periodontal diseases (Genco 1996). Measures such as the gingival index, pocket depth, and loss of attachment are more severe if the diabetic patients are smokers (Bridges et al. 1996). The likelihood of periodontal disease increases markedly when diabetes is poorly controlled. In contrast, periodontal diseases respond well to therapy and can be managed successfully in patients with well-controlled diabetes. Such therapy can result in improvements in the diabetic condition itself (Mealey 1996) (see Chapter 5).

There is some evidence that osteoporosis may be a risk factor for periodontal disease. More clinical attachment loss and edentulousness have been reported in osteoporotic than in nonosteoporotic women (Jeffcoat and Chestnut 1993). Two studies in 1996 showed that estrogen replacement therapy in postmenopausal women not only gives protection against osteoporosis, but also results in fewer teeth lost to periodontal disease (Grodstein et al. 1996, Jacobs et al. 1996).

The less common rapidly progressive form of adult periodontitis typically affects people in their early 20s and 30s. It is characterized by severe gingival inflammation and rapid loss of connective tissue and bone. Many patients have an inherent defect in neutrophil response to infection. Several systemic diseases have been associated with this form of periodontal disease, including type 1 diabetes, Down syndrome, Papillon-Lefevre syndrome, Chediak-Higashi syndrome, and HIV infection (Caton 1989). Specific bacteria associated with rapidly progressive disease include *Porphyromonas gingivalis*, *Prevotella intermedia*, *Eikenella corrodens*, and *Wolinella recta* (Scheutz et al. 1997). Most recently, mutations in the cathepsin C gene have been associated with the Papillon-Lefevre syndrome (Hart et al. 1999) and how the defect can result in periodontal disease (Toomers et al. 1999).

Refractory Periodontitis. Refractory periodontitis is not a specific form of disease, but refers to cases in which patients continue to exhibit progressive disease at multiple sites despite aggressive mechanical therapy to remove biofilm and calculus, along with the use of antibiotics. Refractory sites exhibit elevated levels of a number of different bacteria, with the dominant species different in different subjects. It is not known whether variations in pathogenicity of the bacteria, defects in the subject's defense systems, or combinations of these factors are responsible for the refractory nature of the disease (Haffajee et al. 1988). The adoption of new diagnostic technology to detect predominant bacterial species, followed by selective antibiotic treatment, may help resolve infection and disease in these patients.

Early-onset Periodontitis

The forms of periodontitis occurring in adolescents and young adults generally involve defects in neutrophil function (Van Dyke et al. 1980). Localized juvenile periodontitis (LJP) mainly affects the first molar and incisor teeth of teenagers and young adults, with rapid destruction of bone but almost no telltale signs of inflammation and very little supragingival plaque or calculus. *Actinobacillus actinomycetemcomitans* has been isolated at 90 to 100 percent of diseased sites in these patients, but is absent or appears in very low frequency in healthy or minimally diseased sites (Socransky and Haffajee 1992). It is possible that the bacteria are transmitted among family members through oral contacts such as kissing or sharing utensils, because the same bacterial strain appears in affected family members. However, evidence of a neutrophil defect argues for a genetic component. Another organism frequently associated with LJP is *Capnocytophaga ochracea*. Neither of these bacteria dominate in the generalized adult form of the disease, where *Porphyromonas gingivalis* is considered of greatest significance (Schenkein and Van Dyke 1994).

Prepubertal periodontitis is rare and can be either general or localized. The generalized form begins with the eruption of the primary teeth and proceeds to involve the permanent teeth. There is severe inflammation, rapid bone loss, tooth mobility, and tooth loss. The localized form of the disease is less aggressive, affecting only some primary teeth. The infection involves many of the organisms associated with periodontitis, but the mix may differ somewhat, with *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, *Eikenella corrodens*, and several species of *Capnocytophaga* implicated (Caton 1989).

Defects in neutrophil function noted in both forms of the disease (Schenkein and Van Dyke 1994) may explain why patients are highly susceptible to other infections as well (Suzuki 1988).

SELECTED MUCOSAL INFECTIONS AND CONDITIONS

Like the skin, the mucosal lining of the mouth serves to protect the body from injury. This lining is itself subject to a variety of infections and conditions, ranging from benign canker sores to often fatal cancers.

Oral Candidiasis

Chronic hyperplastic candidiasis is a red or white lesion that may be flat or slightly elevated and may adhere to soft or hard tissue surfaces, including dental appliances. It is caused by species of *Candida*, especially *Candida albicans*, the most common fungal pathogen isolated from the oral cavity. Normally, the fungi are present in relatively low numbers in up to 65 percent of healthy children and adults and cause no harm (McCullough et al. 1996). Problems arise when there is a change in oral homeostasis—the normal balance of protective mechanisms and resident oral flora that maintain the health of the oral cavity—so that defense mechanisms are compromised (Scully et al. 1994). Under these circumstances the fungal organisms can overgrow to cause disease. A primary disruption in homeostasis occurs with the use of antibiotics and corticosteroids, which can markedly change the composition of the oral flora. Deficiencies in the immune and endocrine systems are also important. Indeed, the diagnosis of candidiasis in an otherwise seemingly healthy young adult may be the first sign of HIV infection. Other causes of candidiasis include cancer chemotherapy or radiotherapy to the head and neck, xerostomia resulting from radiation to the head and neck, medications, chronic mucosal irritation, certain blood diseases, and other systemic conditions. Also, tobacco use has been identified as a cofactor.

Candidiasis often causes symptoms of burning and soreness as well as sensitivity to acidic and spicy foods. Patients may complain of a foul taste in the mouth. However, it can also be asymptomatic. Genomic analysis of the *Candida albicans* genome is helping investigators identify numerous genes that code for virulence factors, including enzymes that can facilitate adhesion to and penetration of mucous membranes. At the same time, researchers are exploring novel gene technologies to increase production of

a family of native salivary proteins, the histatins, that have known anticandidal and other antimicrobial effects.

The most common form of oral candidiasis is denture stomatitis. It occurs when tissues are traumatized by continued wearing of ill-fitting or inadequately cleaned dental appliances and is described as chronic erythematous candidiasis. Another form, candidal angular cheilosis, occurs in the folds at the angles of the mouth and is closely associated with denture sore mouth (Tyldesley and Field 1995). Other common forms of *Candida* infection are pseudomembranous candidiasis (thrush), which may affect any of the mucosal surfaces, and acute erythematous candidiasis, a red and markedly painful variant commonly seen in AIDS patients.

In most cases, *Candida* infection can be controlled with antifungal medications used locally or systemically. Control is difficult, however, in patients with immune dysfunction, as in AIDS, or other chronic debilitating diseases. Often the organisms become resistant to standard therapy, and aggressive approaches are necessary (Tyldesley and Field 1995). The spread of oral candidiasis to the esophagus or lungs can be life-threatening and is one of the criteria used to define frank AIDS (Samaranayake and Holmstrup 1989).

Herpes Simplex Virus Infections

In any given year, about one-half-million Americans will experience their first encounter with the herpes simplex virus type 1 (HSV-1), the cause of cold sores. That first encounter usually occurs in the oral region and may be so mild as to go unnoticed. But in some people, particularly young children and young adults, infection may take the form of primary herpetic stomatitis, with symptoms of malaise, muscle aches, sore throat, and enlarged and tender lymph nodes, prior to the appearance of the familiar cold sore blisters. These blisters usually show up on the lips, but any of the mucosal surfaces can be affected. Bright-red ulcerated areas and marked gingivitis may also be seen (Tyldesley and Field 1995).

Herpes viruses also cause genital infections, which are transmitted sexually. Both HSV-1 and HSV-2 have been found in oral and genital infections, with HSV-1 predominating in oral areas and HSV-2 in genital areas (Wheeler 1988). Herpes viruses have also been implicated as cofactors in the development of oral cancers. Crowded living conditions can result in greater contact with infected individuals, which aids in transmission of HSV (Whitley 1992).

Normally, the immune system mounts a successful attack on the viruses, with symptoms abating by the time neutralizing antibodies appear in the bloodstream, in about 10 days. However, herpes viruses are notorious for their ability to avoid immune detection by taking refuge in the nervous system, where they can remain latent for years. In oral herpes the virus commonly migrates to the nearby trigeminal ganglion, the cluster of nerve cells whose fibers branch out to the face and mouth. In about 20 to 40 percent of people who are virus-positive, the virus may reactivate, with infectious virus particles moving to the oral cavity to cause recurrent disease (Scott et al. 1997).

The usual site of a recurrent lesion is on or near the lips. Recurrences are rarely severe, and lesions heal in 7 to 10 days without scarring (Higgins et al. 1993). The recurrences may be provoked by a wide range of stimuli, including sunlight, mechanical trauma, and mild fevers such as occur with a cold. Emotional factors may play a role as well.

Oral Human Papillomavirus Infections

There are more than 100 recognized strains of oral human papillomavirus (HPV), a member of the papovavirus family, implicated in a variety of oral lesions (Regezi and Sciubba 1993). Most common are papillomas (warts) found on or around the lips and in the mouth. HPV is found in 80 percent of these oral squamous papillomas (de Villiers 1989). The virus has also been identified in 30 to 40 percent of oral squamous cell carcinomas (Chang et al. 1990) and has been implicated in cervical cancer as well. Whether a cancer or nonmalignant wart develops may depend on which virus is present or on which viral genes are activated.

Oral warts are most often found in children, probably as a result of chewing warts on the hands. In adults, sexual transmission from the anogenital region can occur (Franchesi et al. 1996).

In general, viral warts spontaneously regress after 1 or 2 years. The immune system normally keeps HPV infections under control, as evidenced by the increased prevalence of HPV-associated lesions in HIV-infected patients and others with immunodeficiency.

Recurrent Aphthous Ulcers

Recurrent aphthous ulcers (RAU), also referred to as recurrent aphthous stomatitis, is the technical term for canker sores, the most common and generally

mild oral mucosal disease. Between 5 and 25 percent of the general population is affected, with higher numbers in selected groups, such as health professional students (Axéll et al. 1976, Embil et al. 1975, Ferguson et al. 1984, Kleinman et al. 1991, Ship 1972, Ship et al. 1967).

The disease takes three clinical forms: RAU minor, RAU major, and herpetiform RAU. The minor form accounts for 70 to 87 percent of cases. The sores are small, discrete, shallow ulcers surrounded by a red halo appearing at the front of the mouth or the tongue. The ulcers, which usually last up to 2 weeks, are painful and may make eating or speaking difficult. About half of RAU patients experience recurrences every 1 to 3 months; as many as 30 percent report continuous recurrences (Bagan et al. 1991).

RAU major accounts for 7 to 20 percent of cases and usually appears as 1 to 10 larger coalescent ulcers at a time, which can persist for weeks or months (Bagan et al. 1991). Herpetiform RAU has been reported as occurring in 7 to 10 percent of RAU cases. The ulcers appear in crops of 10 to 100 at a time, concentrating in the back of the mouth and lasting for 7 to 14 days (Bagan et al. 1991, Rennie et al. 1985).

RAU can begin in childhood, but the peak period for onset is the second decade (Lehner 1968). About 50 percent of close relatives of patients with RAU also have the condition (Ship 1965), and a high correlation of RAU has been noted in identical but not fraternal twins. Associations have been found between RAU and specific genetic markers (Scully and Porter 1989).

RAU has also been associated with hypersensitivities to some foods, food dyes, and food preservatives (Woo and Sonis 1996). Nutritional deficiencies—especially in iron, folic acid, various B vitamins, or combinations thereof—have also been reported, and improvements noted with suitable dietary supplements (Nolan et al. 1991).

The two factors that have been found to have the strongest association with RAU are immunologic abnormality, possibly involving autoimmunity, and trauma (Lehner 1968, Ship 1996, Woo and Sonis 1996).

Volunteers with and without a history of RAU were studied for their reaction to the trauma of a needle prick to the inner cheek tissue. No ulcers developed in non-RAU subjects, but nearly half of those prone to canker sores had a recurrence (Wray et al. 1981).

RAU also can occur in a number of systemic diseases, including HIV infection, ulcerative colitis, Crohn's disease, and Behçet's disease (Ship 1996). In

general, people who are immunocompromised are more susceptible to RAU, as are people with a variety of blood diseases.

RAU itself does not give rise to other illnesses but is uncomfortable. Symptomatic treatment includes topical analgesics, antibacterial rinses, topical corticosteroids, and a new prescription medication that reduces pain and healing time (Khandwala et al. 1997, Ship 1996).

ORAL AND PHARYNGEAL CANCERS AND PRECANCEROUS LESIONS

In 2000, oral or pharyngeal cancer will be diagnosed in an estimated 30,200 Americans and will cause more than 7,800 deaths (Greenlee et al. 2000). Over 90 percent of these cancers are squamous cell carcinomas—cancers of the epithelial cells. The most common oral sites are on the tongue, the lips, and the floor of the mouth. Oral cancer is the sixth most common cancer in U.S. males and takes a disproportionate toll on minorities; it now ranks as the fourth most common cancer among African American men (Kosary et al. 1995). The prominent role of tobacco use, especially in combination with alcohol, in causing these cancers is a major incentive to develop effective health promotion and disease prevention efforts.

Heightening the Risk

Oral cancer develops as a clone from a single genetically altered cell (Solt 1981). It generally has a long latency period and invariably develops from a precancerous lesion on the oral mucosa, such as a white leukoplakia, or more commonly, a reddish erythroplakia (Mashberg 1978, Shklar 1986). Both kinds of lesions are usually induced by tobacco use alone or in combination with heavy use of alcohol. The development of squamous cell carcinoma from initial erythroplakia lesions has been well demonstrated experimentally. Silverman (1998) reported rates of malignant transformation for leukoplakias of between 0.13 and 17.5 percent. However, there is considerable debate as to the actual malignant potential of the leukoplakia lesion associated with the use of smokeless (spit) tobacco. Meaningful data for determining a specific malignant transformation rate or relative risk of oral cancer due to smokeless tobacco use are difficult to obtain because of the confounding effects of other habits such as concurrent smoking and alcohol consumption and because of the variations in smokeless (spit) products and how they are used.

Another oral precancerous lesion that has received attention is submucous fibrosis. It is commonly seen in India and Southeast Asia and is related to betel nut use (Canniff et al. 1986).

Early epidemiologic studies identified behaviors such as smoking and environmental factors such as exposure to solar radiation and x-rays as causes of intraoral and lip cancers (Pindborg 1977). Researchers then sought experimentally to explain the mechanisms of initiation. In the 1980s and 1990s, investigators exploited the techniques of molecular biology and genetics to probe what was going on deep inside the cell. These studies revealed an abundance of systemic and local factors, including viral and fungal infections, that affect cell behavior. Some factors stimulate cell division and others inhibit it—even to the point of initiating a program of cell “suicide,” called apoptosis. How a given cell behaves at any given time in its life cycle is the net result of the signals it receives from neighboring cells and molecules, from circulating factors in the blood or immune system, and from its own internal controls. The following sections provide a brief description of these factors and how they may participate in enhancing the risk for the development of oral cancers.

Tobacco and Alcohol

Tobacco and alcohol are the major risk factors for oral cancers, and their effects have been studied for many years (Rothman and Keller 1972, Decker and Goldstein 1982). Tobacco contains substances that are frankly carcinogenic or act as initiators or promoters of carcinogenesis. Among these are *N*-nitrosonornicotine, 4-nitroquinoline-*N*-oxide, and benzpyrene. The most damaging carcinogens are found in the tars of tobacco smoke, but many forms of smokeless (spit) tobacco, including snuff, have been implicated in the development of mouth cancer (Advisory Committee to the Surgeon General 1986, International Agency for Research on Cancer 1985, Winn 1984). Other habits that have been related to oral cancer include chewing betel nut in the presence of tobacco, as is done primarily in Southeast Asia (Hirayama 1966, Mehta et al. 1981), and, more recently, using marijuana (Donald 1986).

The role of alcohol in oral carcinogenesis has been demonstrated experimentally (Wight and Ogden 1998) and appears to be related to its damaging effect on the liver. Major metabolites of alcohol, such as acetaldehyde—a known carcinogen in animals—may also be important. Alcohol is also thought to act as a solvent that facilitates the pene-

tration of tobacco carcinogens into oral tissues. That observation may partly explain why the combined use of tobacco and alcohol produces a greater risk for oral cancer than use of either substance alone. Indeed, tobacco and alcohol, working in tandem, are thought to account for 75 to 90 percent of all oral and pharyngeal cancers in the United States (Blot et al. 1988).

Viruses

The role of viruses in causing cancer in animals was established early in the century when Rous showed that a virus, later named the Rous sarcoma virus (RSV), caused tumors in chickens. The issue of whether viruses could cause cancer in humans remained unexplored until the mid-1970s, when Varmus and Bishop showed that RSV had a special gene, which they called *src* (for sarcoma), that could transform the cell it infected into a malignant cell (Bishop et al. 1978). It was an oncogene, or cancer-causing gene. The researchers subsequently, and surprisingly, discovered that *src* was not native to the virus, but had been picked up by some ancestor virus from a chicken cell's own genome, where *src* had presumably played a role in the chicken cell's normal growth and development. Somehow RSV was able to subvert *src* when it infected a chicken cell to cause the cell to divide uncontrollably. Varmus and Bishop called the normal cellular *src* gene a *proto-oncogene*, meaning that it had the potential to be converted to an oncogene. Subsequent research led to the discovery of other viruses that could cause tumors in animals and revealed the presence of proto-oncogenes in birds and mammals. These genes could also be converted to oncogenes, behaving exactly like those carried by cancer viruses. In 1982 an oncogene isolated from a human bladder cancer turned out to be virtually identical to *ras*, the oncogene found in a rat sarcoma virus (Parada et al. 1982).

Viruses that have been implicated in oral cancer include herpes simplex type 1 and human papillomavirus. Epstein-Barr virus, also a herpes virus, is now accepted as an oncogenic virus responsible for Burkitt's lymphoma, occurring primarily in Africa, and nasopharyngeal carcinoma, occurring primarily in China. HPV is a major etiologic agent in cervical cancer (Howley 1991), and has been found in association with oral cancer as well (Sugerman and Shillitoe 1997). HPV DNA sequences have been found in oral precancerous lesions as well as in squamous cell carcinomas (Adler-Storthz et al. 1986, Syrjanen et al. 1988), and experimental evidence has shown that HPV-16 can be an important cofactor in

oral carcinogenesis (Park et al. 1991, 1995). Herpes simplex type 1 antibodies were demonstrated in patients with oral cancer, and herpes was found to induce dysplasia (abnormal cellular changes) in the lips of hamsters when combined with the application of tobacco tar condensate (Park et al. 1986).

More recently, human herpes virus 8, a newly identified member of the herpes virus family, has been found in Kaposi's sarcoma, an otherwise rare cancer occurring in patients with AIDS. These tumors often appear initially within the oral cavity (Epstein and Scully 1992). Other uncommon oral malignant tumors, such as Hodgkin's lymphoma and non-Hodgkin's lymphoma, can also occur in the mouths of AIDS patients.

In addition to viruses, infection with strains of the fungus *Candida albicans* has been linked to the development of oral cancers via the fungal production of nitrosamines, which are known carcinogens.

Genetic Derangements

Of the more than 50 known oncogenes, many have been reported to be present in oral cancer, and multiple oncogenes have been reported in oral and pharyngeal cancer (Spandidos et al. 1985). Some of these are *Bcl-1*, *c-erb-B2*, *c-myc*, *ins-2*, and members of the *ras* family (Berenson et al. 1989, Bos 1989, Riviere et al. 1990, Somers et al. 1990, 1992).

The genetic derangements that can give rise to oral cancer, including many mutations associated with the transformation of proto-oncogenes, have received notable attention (Sidransky 1995, Wong et al. 1996). In some instances a change in a single nucleotide base—a point mutation—in a gene encoding a proto-oncogene is enough to transform it into an oncogene. Cancerous changes may also involve alterations, deletions, and break points in chromosomes that affect the position of genes.

Mutations that disarm the cell's DNA repair mechanisms, as well as mutations in tumor suppressor genes, which inhibit abnormal cell growth, play a major role in cancer development. If an individual inherits or acquires a mutation in one or more tumor-suppressor genes, for example, the loss of this protective mechanism reduces the number of other deleterious changes needed for cancer to develop.

Tumor suppressor genes suspected to be mutated in oral and pharyngeal cancers include those for Rb, p16 (*MTS1*, *CDKN2*, or *IN4a*), and p53. Todd et al. (1995) recently reported a novel oral tumor suppressor gene, named "*deleted in oral cancer-1 (doc-1)*." Of the group of tumor suppressor genes, that coding for p53 is considered of major importance, with muta-

tions in the p53 gene detected in many types of cancer (Greenblatt et al. 1994, Hollstein et al. 1991), including oral and pharyngeal cancer (Langdon and Partridge 1992, Somers et al. 1992). The p53 gene has been called the "guardian of the genome" (Lane 1992) because of its ability to recognize damage to a cell's DNA and stop the process of growth and division until the damage is repaired. If repair is not possible, p53 can trigger apoptosis. Mutations in the p53 gene in oral cancer have been linked to smoking and alcohol use (Brennan et al. 1995).

Loss of Immunosurveillance and Control

The immune system can, as first noted by Paul Ehrlich in 1909, seek and destroy initial clones of transformed cancer cells (Ehrlich 1957). Ehrlich called this process *immunosurveillance*, and it has been confirmed in experimental animals (Burnet 1970, Shklar et al. 1990) and in humans with induced immunosuppression (Penn 1975).

One mechanism of immunosurveillance involves stimulating cytotoxic macrophages and lymphocytes to migrate to the tumor site and release tumor necrosis factors α and β (Shklar and Schwartz 1988). Another mechanism operative in oral cancer appears to be stimulation of Langerhans cells, a special group of immune cells, in the oral mucosa (Schwartz et al. 1985). Other immune cells implicated in tumor rejection are natural killer cells and lymphokine-activated killer cells (Reif 1997).

There is an increased incidence of cancer in patients with AIDS or other immunodeficient conditions or with induced immunosuppression prior to organ transplantation.

In addition, there is evidence that smoking depresses the immune system (Chretien 1978), and this may be one of the ways in which smoking acts as a major risk factor in oral cancer.

Growth Factors

Immune cells are potent generators of growth factors and other molecules that can stimulate other cells to migrate and proliferate. This capacity is important in normal cell growth and turnover, in wound healing, and in coping with infection. Unfortunately, the release of growth factors can contribute to oral cancer by stimulating keratinocyte (oral epithelial cell) proliferation (Aaronson 1991, Issing et al. 1993, Wong 1993). Increased levels of transforming growth factor α (TGF- α) and epidermal growth factor have been found in oral and pharyngeal cancers and therefore could serve as markers for malignancy (Grandis and Twardy 1993). Nicotine at high doses stimulates

the release of growth hormones, among other endocrine effects (Pomerleau and Pomerleau 1984, Seyler et al. 1984, 1986).

Prevention and Management

Studies of experimental carcinogenesis are elucidating the role of micronutrients in tumor development and progression. Alpha tocopherol (vitamin E) also has been studied as an antioxidant in nutritional approaches to the prevention or control of oral cancer. Antioxidants can trap free radicals, the highly reactive molecules that build up in cells and can damage DNA. The control of oral cancer and precancerous lesions has been demonstrated experimentally using a variety of antioxidant micronutrients, such as retinoids, carotenoids, and glutathione, as well as alpha tocopherol. For example, it was found that alpha tocopherol inhibited tumor development and tumor angiogenesis (blood vessel formation) in hamsters, as well as the expression of TGF- α , a potent angiogenesis stimulator (Shklar and Schwartz 1996). Animal research and tissue culture studies using animal- and human-derived cancer cell lines have shown combinations of micronutrients to be more effective than single micronutrients and to work synergistically. The nutrients not only were able to inhibit experimental carcinogenesis, but also could completely prevent tumor development and cause established squamous cell carcinomas to regress (Shklar and Schwartz 1993). The mechanisms of cancer control by micronutrients are gradually becoming clarified and involve common pathways of activity at the molecular level (Shklar and Schwartz 1994). Clinical studies in humans have shown an inhibitory effect on oral leukoplakia (Benner et al. 1993, Blot et al. 1993, Garewal 1993, Garewal et al. 1990, Hong et al. 1986), suggesting a potential role for nutrients in the overall prevention and management of early oral cancer and precancerous leukoplakia. However, a recommendation to employ such approaches clinically at this time is premature.

DEVELOPMENTAL DISORDERS

The importance of the face as the bearer of identity, character, intelligence, and beauty is universal. Craniofacial birth defects, which can include such manifestations as cleft lip or palate, eyes too closely or widely spaced, deformed ears, eyes mismatched in color, and facial asymmetries, can be devastating to the parents and child affected. Surgery, dental care, psychological counseling, and rehabilitation may

help to ameliorate the problems but often at great cost and over many years.

Although each developmental craniofacial disease or syndrome is relatively rare, the number of children affected worldwide is in the millions. In addition, craniofacial defects form a substantial component of many other developmental birth defects, largely because they occur very early in gestation, when many of the same genes that orchestrate the development of the brain, head, face, and mouth are also directing the development of the limbs and many vital internal organs, such as the heart, lungs, and liver.

By about the third week after fertilization, the three germ layers of the embryo—the ectoderm, endoderm, and mesoderm—have formed, as well as the first of four sets of paired swellings—the branchial arches—that appear at the sides of the head end of the embryo. (See Chapter 2 for more details on this process.) Some craniofacial defects result from failure of the arches to complete their morphogenetic development. Other craniofacial defects are the result of the abnormal differentiation of cells derived from the ectoderm and endoderm or from ectomesenchyme cells, which originate in a part of the ectoderm (the neural crest), in interaction with future connective tissue (the mesenchyme).

Craniofacial Anomalies Caused by Altered Branchial Arch Morphogenesis

Cleft Lip/Palate and Cleft Palate

The most common of all craniofacial anomalies—and among the most common of all birth defects—are clefts of the lip with or without cleft palate and cleft palate alone; these occur at a rate of 1 to 2 out of 1,000 births, resulting in over 8,000 affected newborns every year. Cleft lip/palate and cleft palate are distinct conditions with different patterns of inheritance and embryological origins (Lidral et al. 1997, Murray et al. 1997). The male to female ratio of cleft lip/palate is 2:1; the ratio for cleft palate alone is just the reverse, 1:2.

These anomalies result from the failure of the first branchial arches to complete fusion processes (Murray 1995, Robert et al. 1996). Clefting can occur independently or as part of a larger syndrome that may include mental retardation and defects of the heart and other organs. Not all cases of clefting are inherited; a number of teratogens (environmental agents that can cause birth defects) have been implicated, as well as defects in essential nutrients such as

folic acid. Smoking by the mother during pregnancy also increases the risk. It is becoming increasingly evident that most diseases and disorders, not just craniofacial anomalies, result from interactions involving multiple genes and environmental factors.

Infants with clefts have difficulty with vital oral functions such as feeding, breathing, speaking, and swallowing. They are also susceptible to repeated respiratory infections. As these children grow, they must cope with the social consequences of a facial deformity, delayed and altered speech, frequent illness, and repeated surgeries that may persist through late adolescence.

Current molecular epidemiology investigations have examined both syndromic and nonsyndromic (isolated) cleft lip/palate and cleft palate. Linkage studies have identified a number of candidate genes (Lewanda and Jabs 1994), including *MSX1*, *RAR*, an X-linked locus, and the genes for *TGF- β 3* and *TGF- α* . The pattern of inheritance in cleft lip/palate and cleft palate suggests that between 2 and 20 genes may be involved, with one gene representing a major component in the development of the cleft. One of the common syndromic forms of cleft lip/palate, the Van der Woude syndrome, is caused by an autosomal dominant form of inheritance at a locus on chromosome 1 (Sander et al. 1995). Future molecular genetic studies will be needed to provide the information necessary for prenatal diagnosis, calculation of risk, and potential gene therapy.

The Treacher Collins Syndrome— Mandibulofacial Dysostosis

Children with the Treacher Collins syndrome have downward-sloping eyelids; depressed cheekbones; a large fishlike mouth; deformed ears with conductive deafness; a small, receding chin and lower jaw; a highly arched or cleft palate; and severe dental malocclusion (Dixon 1996). These defects result from defective cranial neural crest cell differentiation, migration, and proliferation (see Chapter 2). Consequently, the first branchial arch structures are deficient, and all derivative craniofacial components are affected.

The underdeveloped facial structures can contribute to airway blockage and repeated upper respiratory infections, either of which can be fatal. The faulty development of the ears leads to a conductive deafness. The severe facial deformities exacerbate the psychological difficulties these youngsters face.

Investigators have identified the gene involved in an autosomal dominant form of the syndrome (Wise

et al. 1997). The function of the gene is not yet known, but its identity will provide opportunities for prenatal diagnosis, gene therapy, and further understanding of craniofacial development.

The Pierre Robin Syndrome

Deficient development of the first-branchial-arch-derived mandibular portion results in the lower jaw's being set far back in relation to the forehead. As a result, the tongue is set back and may obstruct the posterior airway, compromising respiration (Elliott et al. 1995, Tomaski et al. 1995) and, in severe cases, leading to inadequate aeration and failure to thrive. The infant is also at risk for the development of cor pulmonale, an enlargement of the right ventricle of the heart that occurs secondarily to a chronic lung condition. Cleft palate may be another consequence.

The DiGeorge/Velocardiofacial Syndrome

The primary defect in the DiGeorge syndrome results from altered development of the fourth branchial arch and the third and fourth pharyngeal pouches (Goldmuntz and Emanuel 1997). Deficiencies affecting the thymus, parathyroid glands, and the great vessels that derive from these structures result. The facial features are subtle and include a squared-off nasal tip, small mouth, and widely spaced eyes. Similar facial features, along with heart defects, are seen in the velocardiofacial syndrome. Both syndromes are associated with deletions on the long arm of chromosome 22 (22q11) (Gong et al. 1997, Gottlieb et al. 1997). Further characterization of this chromosomal deletion region will provide information on the specific gene(s) affected and its function in craniofacial development.

The thymus defects severely compromise cellular immunity, depriving the body of thymus-derived T cells and paving the way for severe infectious disease. Inadequate or missing parathyroid glands cause severe hypocalcemia (low blood calcium levels) and seizures. The great vessel abnormalities alter cardiac output and lead to compromised circulation to heart tissues.

Cranial Bone and Dental Anomalies

Defects in the timing of developmental events can cause premature fusion of cranial bones. Impairments of tooth development can result from interruptions of the developmental sequence at several different stages.

Craniosynostoses

Some craniofacial anomalies are associated with so-called master genes that orchestrate a program by which the embryo assumes its basic shape. Craniosynostosis, which occurs in approximately 1 out of 3,000 births, is one such anomaly. It results in the premature fusion of the cranial sutures, a dangerous condition that puts pressure on the developing brain. A number of diseases and syndromes, including Crouzon's, Apert's, Boston-type craniosynostosis, Pfeiffer's, and Saethre-Chotzen, share this anomaly, but differ in other features, which can include structural defects such as webbing of the hands and feet as well as mental retardation. Boston-type craniosynostosis has been linked to *MSX2*, one of the master genes. Several of the other syndromes involve point mutations at one or another locus in genes that code for fibroblast growth factor receptors (*FGFR 1, 2, and 3*) (Howard et al. 1997, Meyers et al. 1996). Collectively, these genes are associated with cell regulation, either through mediating growth factor effects or by serving as transcription factors (Cohen 1997).

Hereditary Hypodontia or Anodontia

Conditions of underdeveloped teeth (hypodontia) or their complete absence (anodontia) have been correlated with specific genes, such as *MSX1* and *LEF1*. The complete absence of teeth alters the bony development of the mandible and maxilla.

Amelogenesis Imperfecta and Dentinogenesis Imperfecta

Amelogenesis imperfecta and dentinogenesis imperfecta are linked to defects in structural genes that code for proteins essential to the development of tooth enamel (amelogenesis imperfecta) or dentin (dentinogenesis imperfecta). The teeth are weak and extremely sensitive to temperature and pressure. The ordinary forces of chewing are painful and can lead to further wear and pain.

The enamel matrix genes include tuftelin, ameloblastin, and amelogenin; researchers have begun to link mutations in these genes with amelogenesis imperfecta. Similarly, genes labeled *DSP* and *DPP* have been characterized for dentin matrix and are associated with the inheritance of dentinogenesis imperfecta.

Craniofacial Defects Secondary to Other Developmental Disorders

A number of genetic diseases occur in which craniofacial defects are secondary to a more generalized structural or biochemical defect.

Osteogenesis Imperfecta

Inherited mutations of collagen genes lead to a number of "brittle bone" diseases characterized by defects in mineralized tissues that form from a collagen-rich matrix. Osteogenesis imperfecta presents a spectrum of deficiencies that includes fragile bones, clear or blue sclera, deafness, loose ligaments, and painful dentinogenesis imperfecta-like changes in the teeth.

Epidermolysis Bullosa—Recessive Dystrophic Type

The gene defect in epidermolysis bullosa—recessive dystrophic type—manifests as blisters or bullae that appear shortly after birth on skin areas following minor trauma. Mutations in keratin genes that contribute to the epithelial cell cytoskeleton have been correlated with this condition.

The oral manifestations include both mucosal bullae and altered teeth. Altered teeth with hypoplastic enamel develop and exhibit an increased susceptibility to caries. Oral bullae develop from even the slightest mucosal trauma. The condition is painful and dangerous because of the constant risk that the bullae will become infected.

Craniofacial Manifestations of Single-Gene Defects

In many craniofacial defects, mutations within a single gene manifest as complex syndromes with varied organ and limb defects as well as facial anomalies.

Ectodermal Dysplasias

The ectodermal dysplasias (EDs) are a family of hereditary diseases first observed by Charles Darwin over a century ago. They involve defects in two or more tissues derived from the ectoderm—skin, hair, teeth, nails, and sweat glands. The ectodermal structures fail to differentiate properly owing to altered epithelial-mesenchymal signaling. A gene, *EDA*, at an X-linked locus has been linked to the syndrome, and ongoing research is aimed at determining the function of the gene and the molecular mechanism of the syndromes (Kere et al. 1996, Zonana et al. 1994).

More recently, investigators have discovered genes linked to autosomal (i.e., non-sex-linked) forms of ED, displaying both dominant and recessive inheritance (Monreal et al. 1999).

Oral manifestations of the ectodermal dysplasias are associated with the teeth. Alterations in tooth development can include hypodontia, anodontia, and conically shaped teeth.

The Waardenburg Syndrome

The Waardenburg syndrome has been subdivided into several types. All involve a variety of abnormalities in the position and appearance of the nose and eyes, with pigment changes that may cause one eye to differ in color from the other. Other signs include deafness, a mildly protruding jaw, cleft lip and palate, and skeletal deformities (Reynolds et al. 1995). The syndrome is inherited in an autosomal dominant manner with complete penetrance and variable expression. Specific genes associated with this syndrome are members of the homeobox family that regulate the transcription of other genes: Waardenburg type 1 with *PAX3*; Waardenburg type 2 with *MITF*, 3q14.1; and Waardenburg type 3 with *PAX3*, 2q35 (Asher et al. 1996).

Cleidocranial Dysplasia

The inheritance of a regulatory gene defect in cleidocranial dysplasia leads to features that include delayed tooth eruption, supernumerary teeth, altered or missing collarbones, short stature, and possible failure of cranial suture closure. The exact mechanism of the associated gene, *CBFA1*, located on chromosome 6, has not been determined but appears to be essential for bone development.

INJURY

The common perception is that injuries are random occurrences that are unpredictable and hence unpreventable. In actuality, experts in the field make the point that there are no basic scientific distinctions between injury and disease (Haddon and Baker 1981). Injuries have been categorized as "intentional" and "unintentional." People identified as being at risk for certain injuries, as well as the causes of those injuries, can be targeted for appropriate prevention strategies. Such an approach is broadly applicable to sports, falls, and motor vehicle injuries (unintentional) as well as to injuries caused by abusive and violent behaviors (intentional).

Injuries are a major public health problem, outranking cancer and heart disease as a leading cause of

death in some age groups of the population (Kraus and Robertson 1992). Cranial injuries in particular are a leading cause of mortality. Oral-facial injuries can bring disfigurement and dysfunction, greatly diminishing the quality of life and contributing to social and economic burdens (Reisine et al. 1989).

The leading causes of oral and craniofacial injuries are sports, violence, falls, and motor vehicle collisions (Kraus and Robertson 1992). Oral cavity injuries may also be caused by foreign objects in food (Hyman et al. 1993).

Sports

Craniofacial sports injuries occur not only in contact sports, but also in individual activities such as bicycling, skating, and gymnastics, especially on trampolines. Each sport predisposes its participants to a specific array of extrinsic risk factors (Pinkham and Kohn 1991). These include physical contact, projectiles such as balls and pucks, and the quality of the playing field and equipment. In contact sports the absence of protective equipment such as headguards and mouthguards is a major risk factor. In a recent survey of school-aged children in organized sports, football was the only sport in which the majority of participants used mouthguards and headgear (Nowjack-Raymer and Gift 1996).

There are intrinsic risk factors as well, relating to characteristics of the individual participant. These include age, sex, injury history, body size, aerobic fitness and muscle strength, central motor control, and general mental ability (Taimela et al. 1990).

Falls

Falls are a major cause of trauma to teeth, primarily to incisors. Unlike bone fractures, fractures of the crowns of the teeth do not heal or repair, and affected teeth often have an uncertain prognosis. Problems may develop later due to damage to the pulp.

Motor Vehicle Collisions

The effects of motor vehicle collisions may range from minor and reversible effects to long-term medical, surgical, and rehabilitative consequences. Post-traumatic headaches and chronic oral-facial pain can occur. Neuromuscular and glandular damage may cause short- or long-term problems with chewing, swallowing, and tearing or result in facial tics or paralysis.

Violence

The family is the single most frequent locus of violence in Western society. Domestic violence includes child abuse, spousal and elder abuse, and abuse of the disabled. Child abuse is of particular concern to the oral health community because 65 percent of cases involve head and oral-facial trauma (Mathewson 1993, Needleman 1986) and dentists are required to report suspected cases of child abuse. In the young child, head injury is the most common cause of death. Psychological trauma from abuse can result in sleep disturbances, eating disorders, developmental growth failure in young children, and nervous habits such as lip and fingernail biting and thumb sucking. Effects may also include chronic underachievement in school and poor peer relationships (Mathewson 1993). In abusive families, physical neglect is commonplace, with inadequate provision of basic needs, including medical and oral health care (Mathewson 1993).

SELECTED CHRONIC AND DISABLING CONDITIONS

Oral, dental, or craniofacial signs and symptoms play a critical role in autoimmune disorders such as Sjögren's syndrome and in a number of chronic and disabling pain conditions.

Sjögren's Syndrome

Sjögren's syndrome is one of several autoimmune disorders in which the body's own cells and tissues are mistakenly targeted for destruction by the immune system. Like other autoimmune conditions, Sjögren's syndrome is more prevalent among women. The ratio of females to males affected is 9:1, with symptoms usually developing in middle age. There are an estimated 1 to 2 million individuals in the United States with Sjögren's syndrome (Talal 1992).

The disease occurs in two forms. Primary Sjögren's involves the salivary and lacrimal (tear) glands. In secondary Sjögren's the glandular involvement is accompanied by the development of a connective tissue or collagen disease, most often rheumatoid arthritis, lupus erythematosus, scleroderma, or biliary cirrhosis.

The glandular involvement causes a marked reduction in fluid secretion, resulting in xerostomia and xerophthalmia (dry eyes). The constant oral dryness causes difficulty in speaking, chewing, and swallowing; the dry eyes often itch and feel gritty. There

is no cure for Sjögren's, and patients often carry eye-drops and water bottles or saliva substitutes in an attempt to provide symptomatic relief. Clinically, the reduction in salivary flow changes the bacterial flora, which, in addition to the reduction in salivary protective components, increases the risk of caries and candidiasis (Daniels and Fox 1992). Recent studies have indicated that there is a reduction in masticatory function (Dusek et al. 1996) and an increased prevalence of periodontal disease (Najera et al. 1997). In advanced stages the salivary glands may swell because of obstruction and infection or lymphatic infiltration. In both forms of the disease, other systems may eventually become affected. Nasal, laryngeal, and vaginal dryness may occur, as well as abnormalities in internal organs (Oxholm and Asmussen 1996).

Diagnosis is difficult in the early stages, and women often report that it took many years and consultations with many specialists before they received the correct diagnosis. Diagnosis involves demonstration of specific antibodies in the blood characteristic of an autoimmune disorder, a labial (minor) salivary gland biopsy, and a series of eye tests to measure flow rate and tissue characteristics. Confirmatory tests include an evaluation of salivary flow and chemistry.

Patients with Sjögren's syndrome are at some risk of developing diseases such as non-Hodgkin's lymphoma; clinical data indicate that such lymphomas develop in 5 percent of patients with Sjögren's syndrome (Moutsopoulos et al. 1978).

Histological examination shows that immune cells infiltrate the glands and cluster around the secretory elements, resulting in a breakdown of the normal structure of the gland. The mechanisms by which this occurs involve immune-cell-mediated inflammation and stimulation of the salivary gland cells themselves to produce tissue-destructive molecules such as cytokines. Another hypothesis is that a viral infection of the glands may trigger the immune response that leads to autoimmunity, whereas genetic or regulatory alterations might lead to abnormalities in apoptosis (Fox and Speight 1996).

In addition to saliva substitutes and artificial tears, some medications, such as pilocarpine and cevimeline, are prescribed to increase salivary flow from the residual healthy gland tissue, again providing symptomatic relief only. The problems that develop in the other organ systems are also treated symptomatically. At advanced stages, steroids are employed intermittently to alleviate problems.

Acute and Chronic Oral-Facial Pain

Since the nineteenth century, when two dentists, Horace Wells and Frederick Morton, demonstrated the analgesic powers of nitrous oxide and ether, oral health investigators have been recognized leaders in the field of pain management worldwide. Their analyses of the cells, pathways, and molecules involved in the transmission and modulation of pain have given rise to a growing variety of medications, often combined with other approaches, that can control acute and chronic pain. Pain researchers today stress that chronic pain can become a disease in itself, causing long-term detrimental changes in the nervous system. These changes may affect resistance to other diseases as well as effectively destroy quality of life. Most people have experienced acute pain involving teeth and the oral tissues at one time or another.

Atypical Facial Pain

Atypical facial pain is characterized by a continuous dull ache on one or both sides, most frequently in the region of the maxilla (the upper jaw). The pain tends to be episodic and is aggravated by fatigue, worry, or emotional upset. It is often accompanied by pain elsewhere in the body and depression. Once a dental cause can be ruled out, pain resolution depends on the successful use of antidepressants, psychotherapy, or both (Tyllesley and Field 1995).

Tic Douloureux

The oral-facial region is also subject to pain that can be paroxysmal or continuous along a distinct nerve distribution. The most frequently encountered of these oral facial neuralgias is tic douloureux, or trigeminal neuralgia, a disease of unknown etiology affecting one, two, or all three branches of the trigeminal nerve. The pain is highly intense and of a stabbing nature, and lasts for a few seconds. This transient attack may be repeated every few minutes or several hours. There may be no precipitating factor, or it may occur in response to a gentle touch or a breeze wafting across the face—a condition experts call *allodynia*, the feeling of pain in response to a normally nonpainful stimulus. On other occasions, there may be a specific trigger zone. Although spontaneous remission for weeks or months may occur, it is rarely permanent. Given the unknown, unpredictable nature of tic douloureux, it is not surprising that fear of pain comes to dominate these patients' lives, as they avoid doing anything that might trigger an attack.

Trigeminal neuralgia generally occurs in later life, but also occurs in younger individuals affected

by multiple sclerosis, where it is assumed to be associated with lesions (multiple sclerosis "plaques") in the brain stem. Medical treatment depends largely on the use of a drug that has become a virtual specific, the antiepileptic drug carbamazepine. For those patients with no consequential adverse effects, it can control the disease. An alternative for chronic sufferers is the surgical removal of a small vein or artery that may be exerting pressure on the nerve root or the selective destruction of the nerve fibers themselves using chemical or electrical methods. In many cases, these procedures can produce complete relief from pain (Tyllesley and Field, 1995).

Temporomandibular Disorders

Various etiological factors, including trauma, can give rise to pain and dysfunction in the temporomandibular joint and surrounding muscles, conditions collectively called temporomandibular disorders (TMDs). The pain may be localized or radiate to the teeth, head, ears, neck, and shoulders. Abnormal grating, clicking, or crackling sounds, known as crepitus, in the joint often accompany localized pain. Pain is also found in response to clinical palpation of the affected structures. TMDs are common, occurring in as many as 10 million Americans. Although surveys indicate that both sexes are affected, the majority of individuals seeking treatment are women of childbearing age, a phenomenon suggesting that hormonal influences should be investigated.

Several factors can contribute to the onset or exacerbation of TMD symptoms. These factors include certain developmental anomalies; injury to the jaw from accidents or abuse; oral habits that greatly stress the joint and musculature, such as tooth grinding (bruxism); jaw manifestations of systemic diseases such as fibromyalgia and arthritic diseases; and some irreversible treatments for initial signs and symptoms.

The multiplicity of factors that may cause or contribute to TMDs has unfortunately led to a multiplicity of treatments. Most of these treatments have not been tested in randomized controlled clinical trials. During the 1970s and 1980s, many individuals underwent surgery, which proved unsuccessful in many cases.

Leading investigators have proposed standardized research diagnostic criteria to clarify the kinds of pathology that can give rise to TMDs and to classify the most common forms of TMDs. Such criteria could be used in designing clinical trials and could ultimately lead to better diagnostics, treatments, and prevention. The criteria use two dimensions or axes:

axis I delineates various forms of joint or muscle pathology; axis II explores pain-related disability and psychological status. The approach requires detailed clinical examinations and patient histories (Dworkin and LeResche 1992).

A MIRROR, A MODEL, AND A BETTER UNDERSTANDING OF DISEASES AND DISORDERS

Studying the diseases and disorders that affect craniofacial tissues can provide scientists with models of systemic pathology. Because some craniofacial tissues, such as bones, mucosa, muscles, joints, and nerve endings, have counterparts in other parts of the body and these tissues are often more accessible to research analysis than deeper-lying tissues, researchers studying craniofacial tissues can gain valuable insights into how cancer develops, the role of inflammation in infection and pain, the effects of diet and smoking, the consequences of depressed immunity, and the changes that can arise from a mutated gene.

Other craniofacial tissues—teeth, gingiva, tongue, salivary glands, and the organs of taste and smell—are unique to the craniofacial complex. Study of the diseases affecting these tissues has revealed a wealth of information about their special nature as well as the molecules and mechanisms that normally operate for the protection, maintenance, and repair of all the oral, dental, and craniofacial tissues. When factors perturb these nurturing elements, the oral health scale can tip toward disease. When those factors stem from systemic diseases or disorders, the mouth can sometimes mirror the body's ill health. Similarly underscoring the connection between oral and general health are studies suggesting that poor dental health, mainly due to chronic dental infections, may heighten the risk for both cardiovascular disease and stroke independently of factors such as social class and established cardiovascular risk factors (Grau et al. 1997). The interplay between craniofacial and systemic health and disease has become a lively focus of interest and research, as discussed in Chapter 5.

Current research on developmental disorders and diseases affecting the craniofacial complex is facilitating and complementing the intense effort of the Human Genome Project to map and sequence all 100,000 genes. This goal should be accomplished early in the twenty-first century and should begin to yield information on the genetic program that governs morphogenesis, organ development, and disease etiology and pathogenesis, with the potential for

interventions that can correct errors in the program. The continued sequencing of the genomes of microbial pathogens involved in oral diseases also should lead to new diagnostic and preventive approaches.

FINDINGS

- Microbial infections, including those caused by bacteria, viruses, and fungi, are the primary cause of the most prevalent oral diseases. Examples include dental caries, periodontal diseases, herpes labialis, and candidiasis.
- The etiology and pathogenesis of diseases and disorders affecting the craniofacial structures are multifactorial and complex, involving an interplay among genetic, environmental, and behavioral factors.
- Many inherited and congenital conditions affect the craniofacial complex, often resulting in disfigurement and impairments that may involve many body organs and systems and affect millions of children worldwide.
- Tobacco use, excessive alcohol use, and inappropriate dietary practices contribute to many diseases and disorders. In particular, tobacco use is a risk factor for oral cavity and pharyngeal cancers, periodontal diseases, candidiasis, and dental caries, among other diseases.
- Some chronic diseases, such as Sjögren's syndrome, present with primary oral symptoms.
- Oral-facial pain conditions are common and often have complex etiologies.

REFERENCES

- Aaronson SA. Growth factors and cancer. *Science* 1991 Nov 22;254(5035):1146-53.
- Adler-Storthz K, Newland JR, Tessin BA, Yeudall WA, Shillitoe EJ. Human papillomavirus type 2 DNA in oral verrucous carcinoma. *J Oral Pathol* 1986 Oct;15(9):472-5.
- Advisory Committee to the Surgeon General. The health consequences of using smokeless tobacco. Chapter 2, Carcinogenesis associated with smokeless tobacco use. Bethesda (MD): Public Health Service; 1986. NIH Pub. no. 86-2874.
- Asher JH Jr, Harrison RW, Morell R, Carey ML, Friedman TB. Effects of PAX3 modifier genes on craniofacial morphology, pigmentation, and viability: a murine model of Waardenburg syndrome variation. *Genomics* 1996 Jun;34(3):285-98.
- Axéll T, Mörnstad H, Sundström B. The relation of the clinical picture to the histopathology of snuff dipper's lesions in a Swedish population. *J Oral Pathol* 1976;5(4):229-36.

- Bagan JV, Sanchis JM, Milian MA, Penarrocha M, Silverstein FJ. Recurrent aphthous stomatitis. A study of the clinical characteristics of lesions in 93 cases. *J Oral Pathol Med* 1991;20:395-7.
- Barbour SE, Nakashima K, Zhang JB, Tangada S, Hahn CL, Schenkein HA, Tew JG. Tobacco and smoking: environmental factors that modify the host response (immune system) and have an impact on periodontal health. *Crit Rev Oral Biol Med* 1997;8(4):437-60.
- Beck JD. Periodontal implications: older adults. *Ann Periodontol* 1996;7(1):322-57.
- Benner SE, Winn RJ, Lippman SM, Poland J, Hansen KS, Luna MA, Hong WK. Regression of oral leukoplakia with alpha-tocopherol: a community clinical oncology program chemopreventative study. *J Natl Cancer Inst* 1993 Jan 6;85(1):44-7.
- Bennet KR, Reade PC. Salivary immunoglobulin A levels in normal subjects tobacco smokers, and patients with minor aphthous ulceration. *Oral Surg Oral Med Oral Pathol* 1982 May;53(5):461-5.
- Bishop JM, Baker B, Fujita D, McCombe P, Sheiness D, Smith K, Spector DH, Stehelin D, Varmus HE. Genesis of a virus-transforming gene. *Natl Cancer Inst Monogr* 1978 May;(48):219-23.
- Berenson JR, Yang J, Mickel RA. Frequent amplification of the bcl-1 locus in head and neck squamous cell carcinomas. *Oncogene* 1989 Sep;4(9):1111-6.
- Berkey CS, Douglass CW, Valachovic RW, Chauncey HH. Longitudinal radiographic analysis of carious lesion progression. *Community Dent Oral Epidemiol* 1988;16:83-90.
- Blot WJ, McLaughlin JK, Winn DM, Austin DF, Greenberg RS, Preston-Martin S, Bernstein L, Schoenberg JB, Stemhagen A, Fraumeni JF Jr. Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Res* 1988;48:3282-7.
- Blot WJ, Li JY, Taylor PR, Guo W, Dawsey S, Wang GQ, Yang CS, Zheng SF, Gail M, Li GY, et al. Nutrition intervention trials in Linxian, China: supplementation with specific vitamin/mineral combinations, cancer incidence, and disease-specific mortality in the general population. *J Natl Cancer Inst* 1993 Sep;85(18):1483-91.
- Bos JL. *ras* oncogenes in human cancer: a review. *Cancer Res* 1989 Sep;49(17):4682-9.
- Bowden GH. Microbiology of root surface caries in humans. *J Dent Res* 1990;69:1205-10.
- Bowen WH. Response to Seow: biological mechanisms of early childhood caries. *Community Dent Oral Epidemiol* 1998;26(Suppl 1):28-31.
- Brennan JA, Boyle JO, Koch WM, Goodman SN, Hruban RH, Eby YJ, Couch MJ, Forastiere AA, Sidransky D. Association between cigarette smoking and mutation of the p53 gene in squamous-cell carcinoma of the head and neck. *N Engl J Med* 1995 Mar;332(11):712-7.
- Bridges RB, Anderson JW, Saxe SR, Gregory K, Bridges SR. Periodontal status of diabetic and non-diabetic men: effects of smoking, glycemic control, and socioeconomic factors. *J Periodontol* 1996;67:1185-92.
- Burnet F. The concept of immunologic surveillance. *Prog Exper Tumor Res* 1970;13:1-20.
- Burt BA, Ismail AI. Diet, nutrition and food cariogenicity. *J Dent Res* 1986;65(SI):1475-84.
- Canniff JP, Harvey W, Harris M. Oral submucous fibrosis: its pathogenesis and management. *Br Dent J* 1986 Jun;160(12):429-34.
- Caton J. Periodontal diagnosis and diagnostic aids. In: *Proceedings of the World Workshop in Clinical Periodontics*; 1989 July 23-27. Princeton (NJ): American Academy of Periodontology; 1989. p. 1-1-22.
- Caufield PW, Cutter GR, Dasanayake AP. Initial acquisition of mutans streptococci by infants: evidence for a discrete window of infectivity. *J Dent Res* 1993;72:37-45.
- Chang F, Syrjanen S, Nuutinen J, Karja J, Syrjanen K. Detection of human papilloma virus (HPV) in oral squamous cell carcinomas by in situ hybridization and polymerase chain reaction. *Arch Dermatol Res* 1990;282(8):493-7.
- Chretien PB. The effects of smoking on immunocompetence. *Laryngoscope* 1978 Jan;88(1 Pt 2 Suppl 8):11-3.
- Cohen MM Jr. Transforming growth factor beta s and fibroblast growth factors and their receptors: role in structural biology and craniosynostosis. *J Bone Miner Res* 1997 Mar;12(3):322-31.
- Costabel U, Bross KJ, Reuter C, Ruhle KH, Mathys H. Alterations in immunoregulatory T-cell subsets in cigarette smokers. A phenotypic analysis of bronchoalveolar and blood lymphocytes. *Chest* 1986;90:39-44.
- Craig GG, Powell CR, Cooper MH. Caries progression in deciduous molar teeth: 24-month results from a minimal treatment program. *Community Dent Oral Epidemiol* 1981;9:260-5.
- Daniels TE, Fox PC. Salivary and oral components of Sjögren's syndrome. *Rheum Dis Clin North Am* 1992;18:571-89.
- Decker J, Goldstein JC. Risk factors in head and neck cancer. *N Engl J Med* 1982 May;306(19):1151-5.
- de Villiers EM. Papilloma viruses in cancers and papillomas of the aerodigestive tract. *Biomed Pharmacother* 1989;43:31-6.
- Dixon MJ. Treacher Collins syndrome. *Hum Mol Genet* 1996;5(Spec No):1391-6.
- Donald PJ. Marijuana smoking—possible cause of head and neck carcinoma in young patients. *Otolaryngol Head Neck Surg* 1986 Apr;94(4):517-21.
- Dusek M, Simmons J, Buschang PH, Al-Hashimi I. Masticatory function in patients with xerostomia. *Gerodontology* 1996;13(1):3-6.
- Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria,

- examinations, and specifications: critique. *J Craniomandib Disord* 1992;6(4).
- Ehrlich P. Collected papers. In: Himmelweit F, editor. Vol. 2. New York: Pergamon Press; 1957. p. S50.
- Ekanayake LS, Sheiham A. Reducing rates of progression of dental caries in British schoolchildren. *Br Dent J* 1987;163:265-9.
- Elliott MA, Studen-Pavlovich DA, Ranalli DN. Prevalence of selected pediatric conditions in children with Pierre Robin sequence. *Pediatr Dent* 1995 Mar-Apr;17(2):106-11.
- Embil JA, Stephens RG, Manuel FR. Prevalence of recurrent herpes labialis and aphthous ulcers among young adults on six continents. *Can Med Assoc J* 1975 Oct 4;113(7):627-30.
- Emslie RD. Radiographic assessment of approximal caries. *J Dent Res* 1959;38:1225-6.
- Epstein JB, Scully C. Neoplastic diseases in the head and neck of patients with AIDS. *Int J Oral Maxillofac Surg* 1992 Aug;21(4):219-26.
- Ferguson MM, Carter J, Boyle P. An epidemiological study of factors associated with recurrent aphthae in women. *J Oral Med* 1984 Oct-Dec;39(4):212-7.
- Fitzgerald RJ, Keyes PH. Demonstration of the etiologic role of streptococci in experimental caries in the hamster. *J Am Dent Assoc* 1960;61:9-19.
- Fox PC, Speight PM. Current concepts of autoimmune exocrinopathy: immunologic mechanisms in the salivary pathology of Sjögren's syndrome. *Crit Rev Oral Biol Med* 1996;7(2):144-58.
- Franchesi S, Munoz N, Bosch XF, Suijers PJ, Walboomers JM. Human papillomavirus and cancers of the upper aerodigestive tract: a review of epidemiological and experimental evidence. *Cancer Epidemiol Biomarkers Prev* 1996;5:567-75.
- Garewal HS. Beta-carotene and vitamin E in oral cancer prevention. *J Cell Biochem* 1993;17F(Suppl):262-9.
- Garewal HS, Meyskens FL Jr, Killen D, Reeves D, Kiersch TA, Elletson H, Strosberg A, King D, Steinbronn K. Response of oral leukoplakia to beta-carotene. *J Clin Oncol* 1990 Oct;8(10):1715-20.
- Genco R. Classification of clinical and radiographic features of periodontal diseases. In: Genco R, Goldman H, Cohen W, editors. *Contemporary periodontics*. St. Louis: CV Mosby; 1990. p. 63-81.
- Genco RJ. Host responses in periodontal disease: current concepts. *J Periodontol* 1992 Apr;63(4 Suppl):338-55.
- Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996 Oct;67(10 Suppl):1041-9.
- Goldmuntz E, Emanuel BS. Genetic disorders of cardiac morphogenesis. The DiGeorge and velocardiofacial syndromes. *Circ Res* 1997 Apr;80(4):437-43.
- Gong W, Emanuel BS, Galili N, Kim DH, Roe B, Driscoll DA, Budarf ML. Structural and mutational analysis of a conserved gene (DGSI) from the minimal DiGeorge syndrome critical region. *Hum Mol Genet* 1997 Feb;6(2):267-76.
- Gottlieb S, Emanuel BS, Driscoll DA, Sellinger B, Wang Z, Roe B, Budarf ML. The DiGeorge syndrome minimal critical region contains a goosecoid-like (GSCL) homeobox gene that is expressed early in human development. *Am J Hum Genet* 1997 May;60(5):1194-201.
- Grandis JR, Tweardy DJ. Elevated levels of transforming growth factor alpha and epidermal growth factor receptor messenger RNA are early markers of carcinogenesis in head and neck cancer. *Cancer Res* 1993 Aug;53(15):3579-84.
- Grau AJ, Buggle F, Ziegler C, Schwarz W, Meuser J, Tasman AJ, Buhler A, Benesch C, Becher H, Hacke W. Association between acute cerebrovascular ischemia and chronic and recurrent infection. *Stroke* 1997;28(9):1724-9.
- Greenblatt MS, Bennett WP, Hollstein M, Harris CC. Mutations in the p53 tumor suppressor gene: clues to cancer etiology and molecular pathogenesis. *Cancer Res* 1994 Sep;54(18):4855-78.
- Greenlee RT, Murray T, Bolden S, Wingo PA. Cancer statistics, 2000. *CA Cancer J Clin* 2000;50:7-33.
- Grodstein F, Colditz GA, Stampfer MJ. Post-menopausal hormone use and tooth loss. A prospective study. *J Am Dent Assoc* 1996;127:370-7.
- Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, Dunford R, Zambon JJ, Hausmann E. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol* 1995;66(1):23-9.
- Gustafsson BE, Quensel CE, Lanke US, Lundquist F, Grahnen H, Bonow BE, Krasse B. The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. *Acta Odontol Scand* 1954;11:232-364.
- Haber J. Cigarette smoking: a major risk factor for periodontitis. *Compendium Cont Educ Dent* 1994;15:1002-14.
- Haddon W Jr, Baker SP. Injury control. In: Clark DW, MacMahon B, editors. *Preventive and community medicine*. 2nd ed. Boston: Little, Brown; 1981. p. 109-40.
- Haffajee AD, Socransky SS, Dzink JL, Taubman MA, Ebersole JL. Clinical, microbiological and immunological features of subjects with refractory periodontal diseases. *J Clin Periodontol* 1988;15(6):390-8.
- Hart TC, Hart PS, Bowden DW, Michalec MD, Callison SA, Walker SJ, Zhang Y, Firatli E. Mutations of the cathepsin C gene are responsible for Papillon-Lefevre syndrome. *J Med Genet* 1999 Dec;36(12):881-7.
- Higgins CR, Schofield JK, Tatnall FM, Leigh IM. Natural history, management and complications of herpes labialis. *J Med Virol* 1993;1(Suppl):22-6.
- Hirayama T. An epidemiological study of oral and pharyngeal cancer in Central and South-East Asia. *Bull World Health Organ* 1966;34(1):41-69.
- Hollstein M, Sidransky D, Vogelstein B, Harris CC. p53 mutations in human cancers. *Science* 1991 Jul;253(5015):49-53.

- Hong WK, Endicott J, Itri LM, Doos W, Batsakis JG, Bell R, Folonoff S, Byers R, Atkinson EN, Vaughan C, et al. 13-*cis*-retinoic acid in the treatment of oral leukoplakia. *N Engl J Med* 1986 Dec;315(24):1501-5.
- Howard TD, Paznekas WA, Green ED, Chiang LC, Ma N, Ortiz de Luna RI, Garcia Delgado C, Gonzalez-Ramos M, Kline AD, Jabs EW. Mutations in TWIST, a basic helix-loop-helix transcription factor, in Saethre-Chotzen syndrome. *Nat Genet* 1997 Jan;15(1):36-41.
- Howley PM. Role of the human papillomaviruses in human cancer. *Cancer Res* 1991 Sep;51(18 Suppl):5019s-22s.
- Hyman FN, Klontz KC, Tollefson L. Eating as a hazard to health: preventing, treating dental injuries caused by foreign objects in food. *J Am Dent Assoc* 1993;124(11):65-9.
- International Agency for Research on Cancer. IARC monograph on the evaluation of the carcinogenic risk of chemicals to humans: tobacco habits other than smoking; betel-quid and areca-nut chewing; and some related nitrosamines. Vol. 37. Lyons, France: International Agency for Research on Cancer; 1985.
- Issing WJ, Wustrow TP, Heptt WJ. Oncogenes related to head and neck cancer. *Anticancer Res* 1993 Nov-Dec; 13(6B):2541-51.
- Jacobs R, Ghyselen J, Koninckx P, van Steenberghe D. Long-term bone mass evaluation of mandible and lumbar spine in a group of women receiving hormone replacement therapy. *Eur J Oral Sci* 1996; 104:10-6.
- Jeffcoat MK, Chestnut C. Systemic osteoporosis and oral bone loss: evidence shows increased risk factors. *J Am Dent Assoc* 1993;124:49-56.
- Jeffcoat MK, Ready MS. Progression of probing attachment loss in adult periodontitis. *J Periodontol* 1991;62:185-91.
- Kere J, Srivastava AK, Montonen O, Zonana J, Thomas N, Ferguson B, Munoz F, Morgan D, Clarke A, Baybayan P, et al. X-linked anhidrotic (hypohidrotic) ectodermal dysplasia is caused by mutation in a novel transmembrane protein. *Nat Genet* 1996 Aug;13(4):409-16.
- Khandwala A, Van Inwegen RG, Alfano MC. 5% amlexanox oral paste, a new treatment for recurrent minor aphthous ulcers. I. Clinical demonstration of acceleration of healing and resolution of pain. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997;83(2):222-30.
- Kleinman DV, Swango PA, Niessen LC. Epidemiologic studies of oral mucosal conditions—methodologic issues. *Community Dent Oral Epidemiol* 1991 Jun;19(3):129-40.
- Kolehmainen L, Rytömaa I. Increment of dental caries among Finnish dental students over a period of 2 years. *Community Dent Oral Epidemiol* 1997;5: 140-4.
- Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, Wilson TG Jr, Higginbottom FL, Duff GW. The interleukin-1 genotype as a severity factor in adult periodontal disease. *J Clin Periodontol* 1997;24:72-7.
- Kosary CL, Ries LA, Miller BA, Hankey BF, Hurray A, Edwards BK, editors. SEER cancer statistics review. 1973-1992 tables and graphs. Bethesda (MD): National Cancer Institute; 1995 Dec. p.17, 34, 542, 355, 361. NIH Pub. no. 96-2789.
- Kraus JF, Robertson LS. Injuries and the public health. In: Last JM, Wallace RB, editors. Public health and preventive medicine. 13th ed. East Norwalk (CT): Appleton and Lange; 1992. p. 1021-34.
- Lamster IB. The host response in gingival crevicular fluid. Potential applications in periodontitis clinical trials. *J Periodontol* 1992;63:1117-23.
- Lane DP. Cancer. p53, guardian of the genome. *Nature* 1992 Jul;358(6381):15-6.
- Langdon JD, Partridge M. Expression of the tumour suppressor gene p53 in oral cancer. *Br J Oral Maxillofac Surg* 1992 Aug;30:214-20.
- Larsen MJ, Fejerskov O. Chemical and structural challenges in remineralization of dental enamel lesions. *Scand J Dent Res* 1987;97:285-96.
- Lehner T. Autoimmunity in oral diseases with special reference to recurrent oral ulcerations. *Proc R Soc Med* 1968;61:515-24.
- Lewanda AF, Jabs EW. Genetics of craniofacial disorders. *Curr Opin Pediatr* 1994 Dec;6(6):690-7.
- Lidral AC, Murray JC, Buetow KH, Basart AM, Schearer H, Schiang R, Naval A, Layda E, Magee K, Magee W. Studies of the candidate genes *TGFB2*, *MSX1*, *TGFA*, and *TGFB3* in the etiology of cleft lip and palate in the Philippines. *Cleft Palate Craniofac J* 1997 Jan;34(1):1-6.
- Linton JL. Quantitative measurements of remineralization of incipient caries. *Am J Orthod Dentofacial Orthop* 1996 Dec;110(6):590-7.
- Mandel ID. Dental caries. *Am Sci* 1979;67:680-8.
- Mandel ID. Calculus update: prevalence, pathogenicity and prevention. *J Am Dent Assoc* 1995 May;126: 573-80.
- Mandel ID, Gaffar A. Calculus revisited. *J Clin Periodontol* 1986;13:249-57.
- Mashberg A. Erythroplasia: the earliest sign of asymptomatic oral cancer. *J Am Dent Assoc* 1978 Apr;96(4):615-20.
- Mathewson RJ. Child abuse and neglect: the dental profession's responsibility. *Compendium* 1993 May;14(5):658-62.
- McCullough MJ, Ross BC, Reade PC. *Candida albicans*: a review of its history, taxonomy, epidemiology, virulence attributes, and methods of strain differentiation. *Int J Oral Maxillofac Surg* 1996;25:136-44.
- Mealey BL. Periodontal implications: medically compromised patients. *Ann Periodontol* 1996 Nov;1(1):256-321.

- Mehta FS, Gupta PC, Pindborg JJ. Chewing and smoking habits in relation to precancer and oral cancer. *J Cancer Res Clin Oncol* 1981;99(1-2):35-9.
- Meyers GA, Day D, Goldberg R, Daentl DL, Przylepa KA, Abrams LJ, Graham JM Jr, Feingold M, Moeschler JB, Rawnsley E, et al. FGFR2 exon IIIa and IIIc mutations in Crouzon, Jackson-Weiss, and Pfeiffer syndromes: evidence for missense changes, insertions and a deletion due to alternative RNA splicing. *Am J Hum Genet* 1996 Mar;58(3):491-8.
- Michalowicz BS. Genetic and heritable risk factors in periodontal disease. *J Periodontol* 1994;65(5 Suppl):479-88.
- Monreal AW, Ferguson BM, Headon DJ, Street SL, Overbeek PA, Zonana J. Mutations in the human homologue of mouse dl cause autosomal recessive and dominant hypohidrotic ectodermal dysplasia. *Nat Genet* 1999 Aug;22(4):366-9.
- Moutsopoulos HM, Kassan SS, Gardy M. Sjögren's syndrome: an update and overview. *Am J Med* 1978;64(5):732-41.
- Murray JC. Face facts: genes, environment and clefts. *Am J Hum Genet* 1995 Aug;57(2):227-32.
- Murray JC, Daack-Hirsch S, Buetow KH, Munger R, Espina L, Paglinawan N, Villanueva E, Rary J, Magee K, Magee W. Clinical and epidemiologic studies of cleft lip and palate in the Philippines. *Cleft Palate Craniofac J* 1997 Jan;34(1):7-10.
- Murray PA. Periodontal diseases in patients infected by human immunodeficiency virus. *Periodontol* 2000 1994;6:50-67.
- Najera MP, Al-Hashimi I, Plemmons JM, Rivera-Hidalgo F, Rees TD, Haghghat N, Wright JM. Prevalence of periodontal disease in patients with Sjögren's syndrome. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997 Apr;83(4):453-7.
- Needleman HL. Orofacial trauma in child abuse: types, prevalence, management and the dental profession's involvement. *Pediatr Dent* 1986 May;8(1 Spec No):71-80.
- Newman HN. Focal infection. *J Dent Res* 1996 Dec;75(12):1912-9.
- Nolan A, McIntosh WB, Allan BF, Lamey PJ. Recurrent aphthous ulceration: Vitamin B1, B2, B6 status and response to replacement therapy. *J Oral Pathol Med* 1991;20:389-91.
- Nowjack-Raymer RE, Gift HC. Use of mouthguards and headgear in organized sports by school-aged children. *Public Health Rep* 1996;30(1):82-6.
- Offenbacher S, Heasman PA, Collins JG. Modulation of host PGE₂ secretion as a determinant of periodontal disease expression. *J Periodontol* 1993;64:432-44.
- Oxholm P, Asmussen K. Primary Sjögren's syndrome: the challenge for classification of disease manifestations. *J Intern Med* 1996;239:467-74.
- Page RC. Critical issues in periodontal research. *J Dent Res* 1995;74:1118-28.
- Parada LF, Tabin CJ, Shih C, Weinberg RA. Human EJ bladder carcinoma oncogene is homologue of Harvey sarcoma virus *ras* gene. *Nature* 1982 Jun 10;297(5866):474-8.
- Park NH, Sapp JR, Herbosa EG. Oral cancer induced in hamsters with herpes simplex infection and simulated snuff dipping. *Oral Surg Oral Med Oral Pathol* 1986 Aug;62(2):164-8.
- Park NH, Min BM, Li SL, Huang MZ, Cherick HM, Doniger J. Immortalization of normal human oral keratinocytes with type 16 human papillomavirus. *Carcinogenesis* 1991 Sep;12(9):1627-31.
- Park NH, Gujuluva CN, Back JH, Cherrick HM, Shin KH, Min BM. Combined oral carcinogenicity of HPV-16 and benzo(a)pyrene: an in vitro multistep carcinogenesis model. *Oncogene* 1995 Jun;10(11):2145-53.
- Penn I. Immunosuppression and cancer. Importance in head and neck surgery. *Arch Otolaryngol* 1975 Nov;101(11):667-70.
- Petersson HG, Bratthall D. The caries decline: a review of reviews. *Eur J Oral Sci* 1996;104:436-43.
- Pindborg JJ. Tobacco and gingivitis. I. Statistical examination of the significance of tobacco in the development of ulceromembranous gingivitis and in the formation of calculus. *J Dent Res* 1947;26:261-4.
- Pindborg JJ. Tobacco and gingivitis. II. Correlation between consumption of tobacco, ulceromembranous gingivitis and calculus. *J Dent Res* 1949;28:460-3.
- Pindborg JJ. Epidemiological studies of oral cancer. *Int Dent J* 1977 Jun 2;27(2):172-8.
- Pinkham JR, Kohn DW. Epidemiology and prediction of sports-related traumatic injuries. *Dent Clin North Am* 1991;35(4):609-26.
- Pitts NB. Monitoring of caries progression in permanent and primary posterior approximal enamel by bite-wing radiography. *Community Dent Oral Epidemiol* 1983;11:228-35.
- Pomerleau OF, Pomerleau CS. Neuroregulators and the reinforcement of smoking: towards a biobehavioral explanation. *Neurosci Biobehav Rev* 1984 Winter;8(4):503-13.
- Ranney RC. Criteria for efficacy of plaque control agents for periodontal disease: microbiology. *J Dent Res* 1989 June;68(Spec No):848-1052.
- Regezi JA, Sciubba J. Oral pathology. Clinical-pathologic correlations. 2nd ed. Philadelphia: Saunders; 1993.
- Reif AE. Cancer immunology. In: *Encyclopedia of human biology*. 2nd ed. San Diego: Academic Press; 1997.
- Reisine ST, Fertig J, Weber J, Leder S. Impact of dental conditions on patients' quality of life. *Community Dent Oral Epidemiol* 1989;17:7-10.
- Rennie JS, Reade PC, Hay KD, Scully C. Recurrent aphthous stomatitis. *Br Dent J* 1985 Dec 7;159(11):361-7.
- Retief DH, Bradley EL, Holbrook M, Switzer P. Enamel fluoride uptake, distribution and retention from topical fluoride agents. *Caries Res* 1983;17:44-51.

- Reynolds JE, Meyer JM, Landa B, Stevens CA, Arnos KS, Israel J, Marazita ML, Bodurtha J, Nance WE, Diehl SR. Analysis of variability of clinical manifestations in Waardenburg syndrome. *Am J Med Genet* 1995 Jul;57(4):540-7.
- Ripa LW. Nursing caries: a comprehensive review. *Pediatr Dent* 1988;10:268-82.
- Riviere A, Wilckens C, Loning T. Expression of c-erbB-2 and c-myc in squamous epithelia and squamous cell carcinomas of the head and neck and the lower female genital tract. *J Oral Pathol Med* 1990 Oct;19(9):408-13.
- Robert E, Kallen B, Harris J. The epidemiology of orofacial clefts. I. Some general epidemiological characteristics. *J Craniofac Genet Dev Biol* 1996 Oct-Dec;16(4):234-41.
- Rothman K, Keller A. The effect of joint exposure to alcohol and tobacco on risk of cancer of the mouth and pharynx. *J Chronic Dis* 1972 Dec;25(12):711-6.
- Rozier RG. The impact of recent changes in the epidemiology of dental caries on guidelines for the use of dental sealants: epidemiologic perspectives. *J Public Health Dent* 1995;55(SI):292-301.
- Samaranayake LP, Holmstrup P. Oral candidiasis and human immunodeficiency virus infection. *J Oral Pathol Med* 1989;18:554-64.
- Sander A, Murray JC, Scherpbier-Heddema T, Buetow KH, Weissenbach J, Zingg M, Ludwig K, Schmelzle R. Microsatellite-based fine mapping of the Van der Woude syndrome locus to an interval of 4.1 cM between D1S245 and D1S414. *Am J Hum Genet* 1995 Jan;56(1):310-8.
- Schenkein HA, Van Dyke TE. Early onset periodontitis: systemic aspects of etiology and pathogenesis. *Periodontol* 2000 1994;6:7-25.
- Scheutz F, Matee MI, Andsager L, Holm AM, Moshi J, Kagoma C, Mpemba N. Is there an association between periodontal condition and HIV infection? *J Clin Periodontol* 1997 Aug;24(8):580-7.
- Schwartz JL, Frim Sr, Shklar G. RA can alter the distribution of ATPase-positive Langerhans cells in the hamster cheek pouch in association with DMBA application. *Nutr Cancer* 1985;7(1-2):77-84.
- Scott DA, Couter WA, Lamey PJ. Oral shedding of herpes simplex virus type I: a review. *J Oral Pathol Med* 1997;26(10):441-7.
- Scully C, Porter CR. Recurrent aphthous stomatitis: current concepts of etiology, pathogenesis and management. *J Oral Pathol Med* 1989;18:21-7.
- Scully C, el-Kabir M, Samaranayake LP. *Candida* and oral candidosis: a review. *Crit Rev Oral Biol Med* 1994;5(2):125-57.
- Seow WK. Biological mechanisms of early childhood caries. *J Public Health Dent* 1998;26(Suppl 1):8-27.
- Seyler LE Jr, Fertig J, Pomerleau O, Hunt D, Parker K. The effects of smoking on ACTH and cortisol secretion. *Life Sci* 1984 Jan 2;34(1):57-65.
- Seyler LE Jr, Pomerleau OF, Fertig JB, Hunt D, Parker K. Pituitary hormone response to cigarette smoking. *Pharmacol Biochem Behav* 1986 Jan;24(1):159-62.
- Seymour GJ. Importance of the host response in the periodontium. *J Clin Periodontol* 1991;18:421-6.
- Shannon IL, Edmonds EL. Effect of fluoride concentration on rehardening of enamel by a saliva substitute. *Int Dent J* 1978;28:421-6.
- Ship II. Inheritance of aphthous ulcers of the mouth. *J Dent Res* 1965;44:837-44.
- Ship II. Epidemiologic aspects of recurrent aphthous ulceration. *Oral Surg Oral Med Oral Pathol* 1972;33(3):400-6.
- Ship II, Brightman VJ, Laster LL. The patient with recurrent aphthous ulcers and the patient with recurrent herpes labialis: a study of two population samples. *J Am Dent Assoc* 1967 Sep;75(3):645-54.
- Ship JA. Recurrent aphthous stomatitis: an update. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996 Feb;81(2):141-7.
- Shklar G. Oral leukoplakia. *N Engl J Med* 1986;315(24):1544-6.
- Shklar G, Schwartz J. Tumor necrosis factor in experimental cancer regression with alphatocopherol, beta-carotene, canthaxanthin, and algae extract. *Eur J Cancer Clin Oncol* 1988 May;24(5):839-50.
- Shklar G, Schwartz J. Oral cancer inhibition by micronutrients. The experimental basis for clinical trials. *Eur J Cancer B Oral Oncol* 1993 Jan;29B(1):9-16.
- Shklar G, Schwartz JL. A common pathway for the destruction of cancer cells: experimental evidence and clinical implications. *Int J Oncol* 1994;4:215-24.
- Shklar G, Schwartz JL. Vitamin E inhibits experimental carcinogenesis and tumour angiogenesis. *Eur J Cancer B Oral Oncol* 1996 Mar;32B(3):114-9.
- Shklar G, Schwartz JL, Trickler DP, Reid S. Prevention of experimental cancer and immunostimulation by vitamin E (immunosurveillance). *J Oral Pathol Med* 1990 Feb;19(2):60-4.
- Shwartz M, Gröndahl H-G, Pliskin JS, Boffa J. A longitudinal analysis from bite-wing radiographs of the rate of progression of approximal carious lesions through human dental enamel. *Arch Oral Biol* 1984;29(7):529-36.
- Sidransky D. Molecular genetics of head and neck cancer. *Curr Opin Oncol* 1995 May;7(3):229-33.
- Silverman S. *Oral cancer*. 4th ed. Hamilton, Ontario: B.C. Decker; 1998.
- Slavkin H. Gene regulation in the development of the oral tissues. *J Dent Res* 1988;67:1142-9.
- Socransky SS, Haffajee AD. Microbial mechanisms in the pathogenesis of destructive periodontal diseases: a critical assessment. *J Periodontol Res* 1991 Apr;26:195-212.
- Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: current concepts. *J Periodontol* 1992 Apr;63(4 Suppl):322-31.

- Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. *J Clin Periodontol* 1984 Jan;11(1):21-32.
- Solt DB. Localization of gamma-glutamyl transpeptidase in hamster buccal pouch epithelium treated with 7, 12-dimethylbenz[a]anthracene. *J Natl Cancer Inst* 1981 Jul;67(1):193-200.
- Somers KD, Cartwright SL, Schechter GL. Amplification of the int-2 gene in human head and neck squamous cell carcinomas. *Oncogene* 1990 Jun;5(6):915-20.
- Somers KD, Merrick MA, Lopez ME, Incognito LS, Schechter GL, Casey G. Frequent p53 mutations in head and neck cancer. *Cancer Res* 1992;52(21):5997-6000.
- Spandidos DA, Lamothe A, Field JK. Multiple transcriptional activation of cellular oncogenes in human head and neck solid tumors. *Anticancer Res* 1985;5(2):221-4.
- Sreebny LM, Yu A, Green A, Valdin A. Xerostomia in diabetes mellitus. *Diabetes Care* 1992 Jul;15(7):900-4.
- Sugerman PB, Shillitoe EJ. The high risk of human papillomaviruses and oral cancer: evidence for and against a causal relationship. *Oral Dis* 1997;3:130-47.
- Suzuki JB. Diagnosis and classification of the periodontal diseases. *Dent Clin North Am* 1988 Apr;32(2):195-216.
- Syrjanen SM, Syrjanen KJ, Happonen RP. Human papillomavirus (HPV) DNA sequences in oral precancerous lesions and squamous cell carcinoma demonstrated by in situ hybridization. *J Oral Pathol* 1988 Jul;17(6):273-8.
- Taimela S, Kujala UM, Osterman K. Intrinsic risk factors and athletic injuries. *Sports Med* 1990 Apr;9(4):205-15.
- Talal N. Sjögren's syndrome: historical overview and clinical spectrum of disease. *Rheum Dis Clin North Am* 1992 Aug;18(3):507-15.
- Todd R, McBride J, Tsuji T, Donoff RB, Nagai M, Chou MY, Chiang T, Wong DT. Deleted in oral cancer-1 (doc-1), a novel oral tumor suppressor gene. *FASEB J* 1995 Oct;9(13):1362-70.
- Tomar SL, Winn DM. Coronal and root caries among U.S. adult users of chewing tobacco [abstract]. *J Dent Res* 1998;77(S1):256.
- Tomaski SM, Zalzal GH, Saal HM. Airway obstruction in the Pierre Robin sequence. *Laryngoscope* 1995 Feb;105(2):111-4.
- Toomers C, James J, Wood AJ, et al. Loss of function mutations in the cathepsin C gene result in periodontal disease and palmoplantar keratosis. *Nat Genet* 1999 Dec;22:421-4.
- Tyldesley WR, Field AE. *Oral medicine*. 4th ed. Oxford: Oxford University Press; 1995.
- U.S. Department of Health and Human Services, National Center for Health Statistics. National Health and Nutrition Examination Survey III, 1988-1994. CD-ROM series 11, no.1. Hyattsville (MD): National Center for Health Statistics, Centers for Disease Control and Prevention; 1997. Available from National Technical Information Service (NTIS), Springfield, VA.
- Van Dyke TE, Horoszewicz HU, Cianciola LJ, Genco RJ. Neutrophil chemotaxis dysfunction in human periodontitis. *Infect Immun* 1980 Jan;27(1):124-32.
- Vissink A, 's-Gravenmade EJ, Gelhard TB, Panders AK, Franken MH. Rehardening properties of mucin- or CMC-containing substitutes on softened human enamel. Effects of sorbitol, xylitol and increasing viscosity. *Caries Res* 1985;19(3):212-8.
- Wheeler CE. The herpes simplex problem. *J Am Acad Dermatol* 1988;18(1 Pt 2):163-8.
- White DJ. Reactivity of fluoride dentifrices with artificial caries; II. Effects on subsurface lesions, F uptake, surface hardening, and remineralization. *Caries Res* 1988;22:27-36.
- Whitley RJ. Neonatal herpes simplex virus infections: pathogenesis and therapy. *Pathol Biol (Paris)* 1992 Sep;40(7):729-34.
- Wight AJ, Ogden GR. Possible mechanisms by which alcohol may influence the development of oral cancer—a review. *Oral Oncol* 1998;34:441-7.
- Winn DM. Tobacco chewing and snuff dipping: an association with human cancer. *IARC Sci Publ* 1984;(57):837-49.
- Wise CA, Chiang LC, Paznekas WA, Sharma M, Musy MM, Ashley JA, Lovett M, Jabs EW. *TCOF1* gene encodes a putative nucleolar phosphoprotein that exhibits mutations in Treacher Collins syndrome throughout its coding region. *Proc Natl Acad Sci USA* 1997 Apr 1;94(7):3110-5.
- Wong DT. TGF-alpha and oral carcinogenesis. *Eur J Cancer B Oral Oncol* 1993 Jan;30B(4):3-7.
- Wong DT, Todd R, Tsuji T, Donoff RB. Molecular biology of human oral cancer. *Crit Rev Oral Biol Med* 1996;7(4):319-28.
- Woo SB, Sonis ST. Recurrent aphthous ulcers: a review of diagnosis and treatment. *J Am Dent Assoc* 1996 Aug;127(8):1202-13.
- World Health Organization (WHO). *World Health Organization's international classification of diseases and stomatology, IDC-DA*. 3rd ed. Geneva: WHO; 1992.
- Wray D, Graykowski EA, Notkins AL. Role of mucosal injury in initiating recurrent aphthous stomatitis. *Br Med J (Clin Res Ed)* 1981 Dec 12;283(6306):1569-70.
- Zonana J, Jones M, Clarke A, Gault J, Muller B, Thomas NS. Detection of de novo mutations and analysis of their origin in families with X linked hypohidrotic ectodermal dysplasia. *J Med Genet* 1994 Apr;31(4):287-92.

The Magnitude of the Problem

The range of oral, dental, and craniofacial diseases and conditions that take a toll on the U.S. population is extensive. This chapter provides highlights of diseases and conditions affecting the U.S. population using available national and state data to describe the burden of disease in the United States. To capture the dimensions and extent of these diseases and conditions, the data are presented where possible by demographic measures such as race/ethnicity, sex, age, education, and economic status. Statistics and trends are presented for each of the six categories of oral diseases and disorders whose etiology and pathogenesis are described in Chapter 3: dental and periodontal infections, oral and pharyngeal cancers, mucosal infections and conditions, developmental disorders, intentional and unintentional injuries, and chronic and disabling conditions. Included are conditions as common as dental caries and periodontal diseases as well as relatively rare clefting syndromes. Also mentioned are conditions that are more common in certain demographic subpopulations—for example, Sjögren's syndrome and temporomandibular disorders, which are more common in women, and injuries, which are more common in men. (See Box 4.1 for a glossary of terms used in this chapter.)

There is no single measure of oral health or the burden of oral diseases and conditions, just as there is no single measure of overall health or overall disease. As a result, this chapter assembles clinical and epidemiologic measures for specific conditions affecting the craniofacial structures. Note too that the chapter presents an incomplete picture. State-specific data on oral diseases are extremely limited. There is a paucity of national data on rare conditions as well as on the health of selected populations and their subgroups. Some characteristics and unique needs of these populations are highlighted, and a number of questions raised. More extensive analyses of the differences among racial/ethnic, sex, and

income groups are warranted. The relationship of oral health to the use of dental services is described. However, the effects of health care visits and of specific services rendered need further study.

Most of the data in the figures and tables presented in this chapter are derived from large, nationally representative surveys of the U.S. civilian, noninstitutionalized population. These include complex sample surveys, such as the National Health and Nutrition Examination Survey (NHANES), which use a sample of individuals selected with known probability to estimate the prevalence of particular characteristics and conditions in the nation as a whole. The multipurpose NHANES provides data on the frequency of the most common oral diseases and conditions. The most recent survey, NHANES III, was conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control between 1988 and 1994. Trained interviewers gathered demographic, health, and related data from eligible households. Selected persons in these households were invited to a mobile examination center, where they underwent multiple health assessments, including an oral examination by a trained dentist (NCHS 1996). Related surveys such as the National Health Interview Survey (NHIS) also use complex survey sampling and household interviews to obtain health information about the U.S. population (Kovar 1989). The NHIS conducted in 1989 included data on oral-facial pain conditions; several of these surveys have captured data on dental visits. The most extensive dental utilization survey that provides demographic and socioeconomic data and data on reasons for not visiting a dentist was conducted in 1989.

Surveys conducted by the National Institute of Dental and Craniofacial Research of a probability sample of U.S. schoolchildren in 1979-80 and 1986-87 (Snowden and Miller-Chisholm 1992) used

BOX 4.1 Glossary

Complex sample survey. A survey of individuals selected with known probability to estimate the prevalence and/or extent of particular characteristics and conditions.

dfs. The count (number) of decayed (untreated) or filled primary tooth surfaces per person.

dft. The count (number) of decayed (untreated) or filled primary teeth per person.

DMFS. The count (number) of decayed (untreated), missing (due to caries), or filled permanent tooth surfaces per person.

DMFT. The count (number) of decayed, missing (due to caries),¹ or filled permanent teeth per person.

Epidemiology. The study of the distribution and determinants of disease frequency in human populations.

Incidence. The number of cases of a disease, or condition, that occurs during a specified period of time.

Incidence rate. The number of cases of a disease, or condition, that occurs during a specified period of time, per specified unit of population.

Loss of periodontal attachment.² The distance from the cementoenamel junction to the bottom of the gingival sulcus.

Mortality rate. The number of deaths during a stated period of time divided by the total number of persons in the population.

Prevalence. The number of existing cases of a disease, or condition, at a designated point in time.

Prevalence rate. The number of existing cases of a disease, or condition, at a designated point in time, per the number of persons in the population.

Relative survival rate. Survival rates for persons with a particular disease or condition corrected for the expected occurrence of death in persons in the age group.

Survival rate. The number of persons surviving over a specified time period divided by the number of persons alive at the start of the time period.

¹In the Third National Health and Nutrition Examination Survey, the M (missing) component of the DMFT (S) index reflects teeth (or tooth surfaces) missing because of dental caries or periodontal disease. Teeth missing because of trauma, orthodontic treatment, or other non-disease-related reasons were not scored.

²Periodontal disease status is assessed by measuring the distance from the gingival margin (FGM) to depth of the gingival sulcus, or pocket depth, and the distance from the gingival margin to the cementoenamel junction (FGM-CEJ). A third measure, loss of attachment, is determined by calculating the difference between pocket depth and the FGM-CEJ distance. Loss of attachment is important because it serves as a measure of how much support from the tissues surrounding the tooth has been lost.

similar oral examination procedures as in NHANES. Because school attendance is high in the United States, these surveys are considered representative of noninstitutionalized children throughout the United States and are used in this chapter.

Record-based surveys are another approach to obtaining health data for the nation as a whole or for selected broad areas. Mortality statistics are obtained by determining the number of deaths in the United States and dividing that figure by the total U.S. population as determined from U.S. Census data (Kovar 1989). Cancer statistics are derived from population-based cancer registries in selected large geographic areas of the United States using reports of cancer occurrences from hospitals, doctors, and laboratories. This data system, maintained by the National Cancer Institute, is called the Surveillance, Epidemiology, and End Results program (Ries et al. 1999). Birth certificate registries in geographic areas and surveys of health care facilities provide valuable information from record-based systems about other aspects of oral and craniofacial health such as birth defects (Schulman et al. 1993). The Centers for Disease Control and Prevention's Behavioral Risk and State Surveillance System provides essential state data on edentulousness (Tomar 1997). In selected cases, survey findings other than from national probability surveys are used. State-specific data are provided for those conditions for which there are data from most states—that is, cancer mortality statistics and self-reports of edentulism.

Economic status is derived from annual income data. Unless otherwise stated, "poor" is defined in this chapter as an annual income below the U.S. poverty level. For both national and other surveys, the race and ethnicity terms used in this report are consistent with the terms used in the supporting documentation as referenced in the text and cited in the reference list. Available national data for most conditions are limited primarily to Hispanic, non-Hispanic black, and non-Hispanic white populations, due to the sampling design of the national surveys. The NHANES III oversampled Mexican Americans, so the data from that survey are available for this subpopulation of Hispanics.

WHO HAS WHAT DISEASES AND CONDITIONS?

Dental Caries, Periodontal Diseases, and Tooth Loss

Dental Caries

Dental caries is one of the most common childhood diseases. In this section, *decayed* refers to teeth with caries that have not been treated. The term *filled* refers to treated caries. *Dental caries* refers to both decayed and filled teeth. Among 5- to 17-year-olds, dental caries is more than 5 times as common as a reported history of asthma and 7 times as common as hay fever (Figure 4.1). Prevalence increases with age. The majority (51.6 percent) of children aged 5 to 9 years had at least one carious lesion or filling in the coronal portion of either a primary or a permanent tooth. This proportion increased to 77.9 percent for 17-year-olds and 84.7 percent for adults 18 or older. Additionally, 49.7 percent of people 75 years or older had root caries affecting at least one tooth (NCHS 1996, NHANES III).

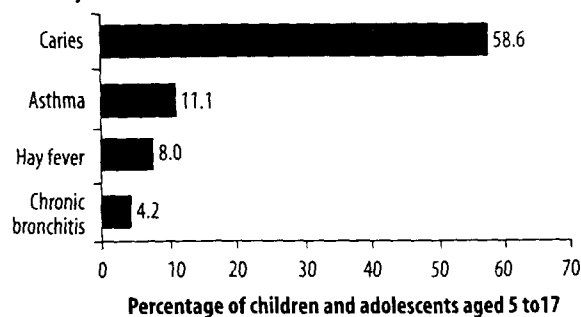
Despite progress in reducing dental caries, individuals in families living below the poverty level experience more dental decay than those who are economically better off. Furthermore, the caries seen in these individuals is more likely to be untreated than caries in those living above the poverty level (Figure 4.2); more than one third (36.8 percent) of poor children aged 2 to 9 have one or more untreated decayed primary teeth, compared to 17.3 percent of nonpoor children.

In addition to poverty level, the proportion of teeth affected by dental caries also varies by age and race/ethnicity. Poor Mexican American children aged

2 to 9 have the highest number of primary teeth affected by dental caries (a mean of 2.4 decayed or filled teeth) compared to poor non-Hispanic blacks (mean 1.5) and non-Hispanic whites (mean 1.9). Among the nonpoor, Mexican American 2- to 9-year-olds have the highest number of affected teeth (mean 1.8), followed by non-Hispanic blacks (1.3) and non-Hispanic whites (1.0).

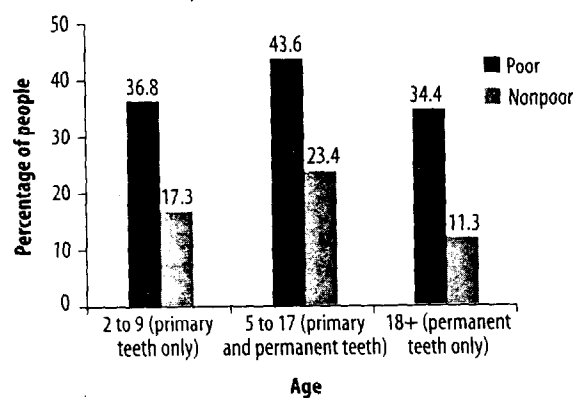
There are also differences by race/ethnicity and poverty level in the proportion of untreated decayed teeth for all age groups. Poor Mexican American children aged 2 to 9 have the highest proportion of untreated decayed teeth (70.5 percent), followed by poor non-Hispanic black children (67.4 percent) (Figure 4.3). Nonpoor children have lower propor-

FIGURE 4.1
Dental caries is one of the most common diseases among 5- to 17-year-olds



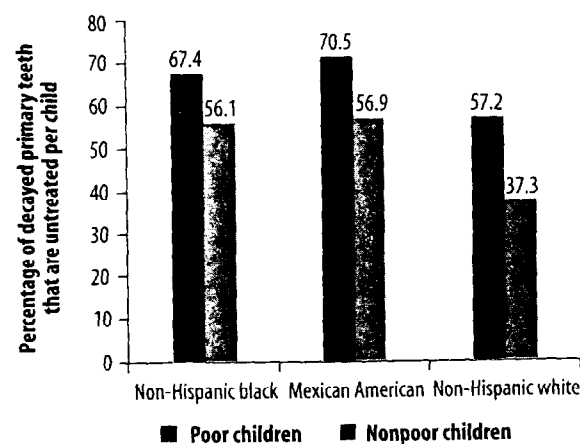
Note: Data include decayed or filled primary and/or decayed, filled, or missing permanent teeth. Asthma, chronic bronchitis, and hay fever based on report of household respondent about the sampled 5- to 17-year-olds.
Source: NCHS 1996.

FIGURE 4.2
A higher percentage of poor people than nonpoor have at least one untreated decayed tooth



Source: NCHS 1996.

FIGURE 4.3
Poor children aged 2 to 9 in each racial/ethnic group have a higher percentage of untreated decayed primary teeth than nonpoor children

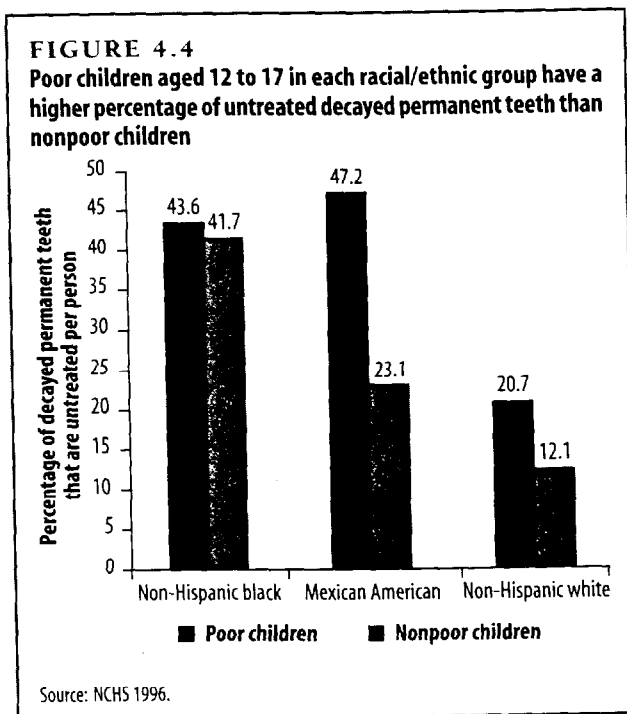


Source: NCHS 1996.

tions of untreated decayed teeth, although the group with the lowest proportion (non-Hispanic whites) still has an average of 37.3 percent of decayed teeth untreated.

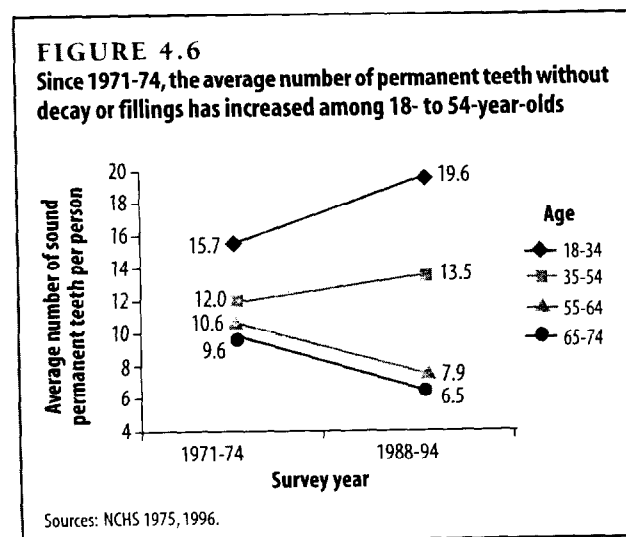
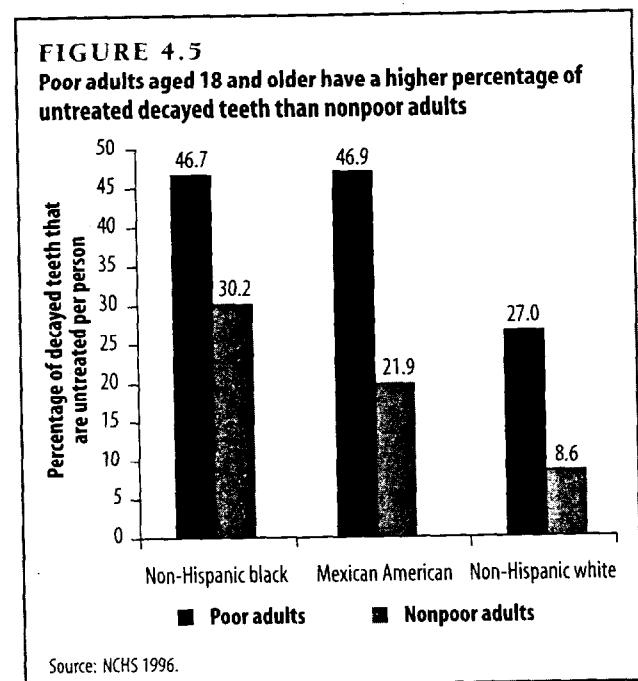
Poor adolescents aged 12 to 17 in each racial/ethnic group have a higher percentage of untreated decayed permanent teeth than the corresponding nonpoor adolescent group (Figure 4.4). Poor Mexican American (47.2 percent) and poor non-Hispanic black adolescents (43.6 percent) have more than twice the proportion of untreated decayed teeth than poor non-Hispanic white adolescents (20.7 percent). For nonpoor adolescents the proportion of untreated decayed permanent teeth is highest in non-Hispanic black adolescents (41.7 percent)—a proportion only slightly lower than for this group's poor counterparts (43.6 percent). The mean number of permanent teeth affected by dental caries (decayed or filled) for this age group is similar among Mexican Americans (2.7), non-Hispanic whites (2.5), and non-Hispanic blacks (2.3). As income level increases, the percentage of adolescents with decayed teeth decreases and the proportion of decayed teeth that have been filled increases (Vargas et al. 1998).

Adult populations (aged 18 and older) show a similar pattern, with the proportion of untreated decayed teeth higher among the poor than the nonpoor (Figure 4.5). Regardless of poverty level status, adult non-Hispanic blacks and Mexican Americans have higher proportions of untreated decayed teeth than their non-Hispanic white counterparts.



Improvements have been noted over the past 25 to 30 years with regard to dental caries. Among most age groups, the average number of teeth per person affected by dental caries has decreased, and the average number of teeth per person that show no signs of infection, as well as the proportion of the population that is caries-free, has increased.

Since 1971-74, major increases have been noted in the percentage of children and adolescents aged 5 to 17 who have never experienced dental caries in their permanent teeth. Younger adults have experienced a decline in dental caries during this time period, as measured by the average number of teeth without decay or fillings (Figure 4.6). These trends are not found among those 55 to 74 years of age.



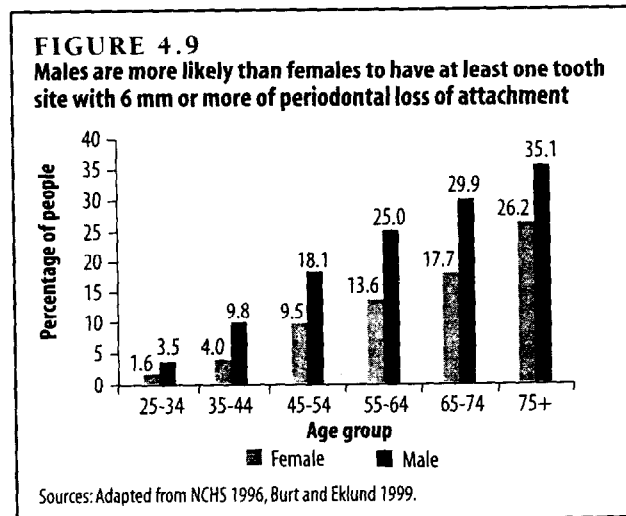
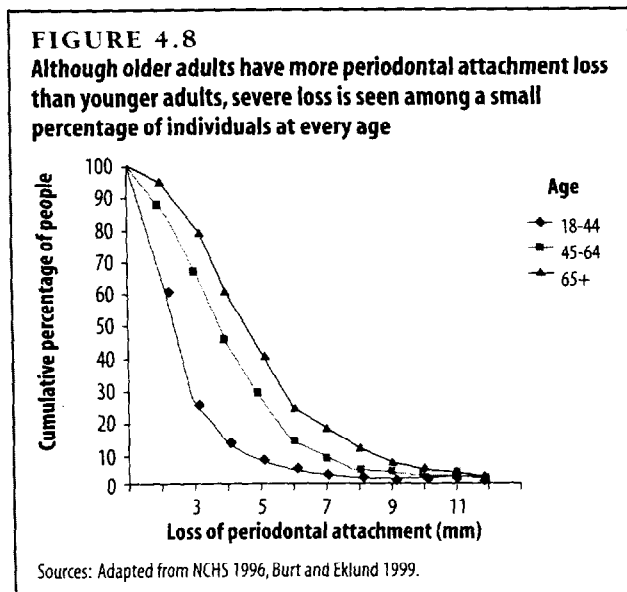
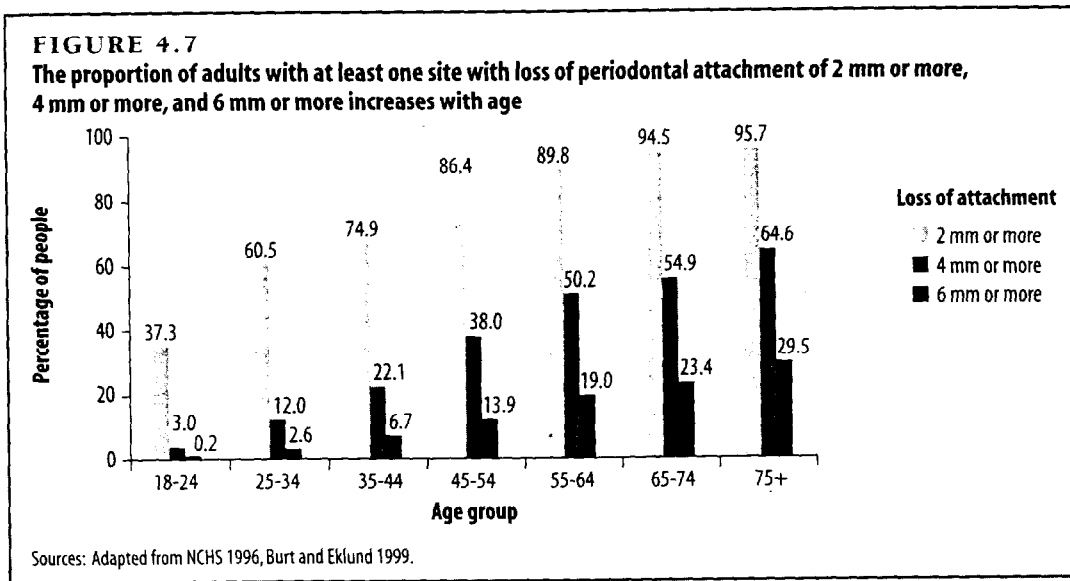
The number of untreated decayed teeth per person across all age groups has also declined.

Periodontal Diseases

The presence of periodontal disease is measured clinically in several ways, one of which is by calculating the loss of periodontal attachment. Figure 4.7 shows that most adults 25 years and older have at least 2 mm or more loss of attachment. The disease is more serious as the amount of attachment loss and number of tooth sites affected increase. More severe disease can be defined as having 4 mm or more loss of attachment in at least one site. The percentage of adults with 6 mm or more loss of attachment at one or more sites increases at older age groups, with 19.0 percent of 55- to 64-year-olds and 23.4 percent of 65- to 74-

year-olds having this amount of loss or more. Figure 4.8 displays these data in a different format and shows that a small but increasing percentage of the population at each older age group has severe disease.

At all ages, men are more likely than women to have at least one tooth site with a 6 mm or more loss of attachment (Figure 4.9). In addition to age and sex, the prevalence of periodontal loss of attachment also varies by racial/ethnic group (Figure 4.10). A higher percentage of non-Hispanic black persons at each age group have at least one tooth site with 6 mm or more of periodontal attachment loss as compared to other groups. Within each racial/ethnic group, the highest percentage affected is found among individuals 70 years and older. At every age, a higher proportion of those at the lowest socioeconomic status



(SES) level have at least one site with attachment loss of 6 mm or more, compared to those at higher SES levels (Figure 4.11) (Burt and Eklund 1999).¹

Gingivitis as measured by gingival bleeding, a sign of inflammation, is more evident among Mexican Americans (63.6 percent) than among non-Hispanic blacks (55.7 percent) and non-Hispanic whites (48.6 percent) (Albandar et al. 1999).

Early-onset periodontitis, a severe, rapidly progressive disease occurring in individuals under age

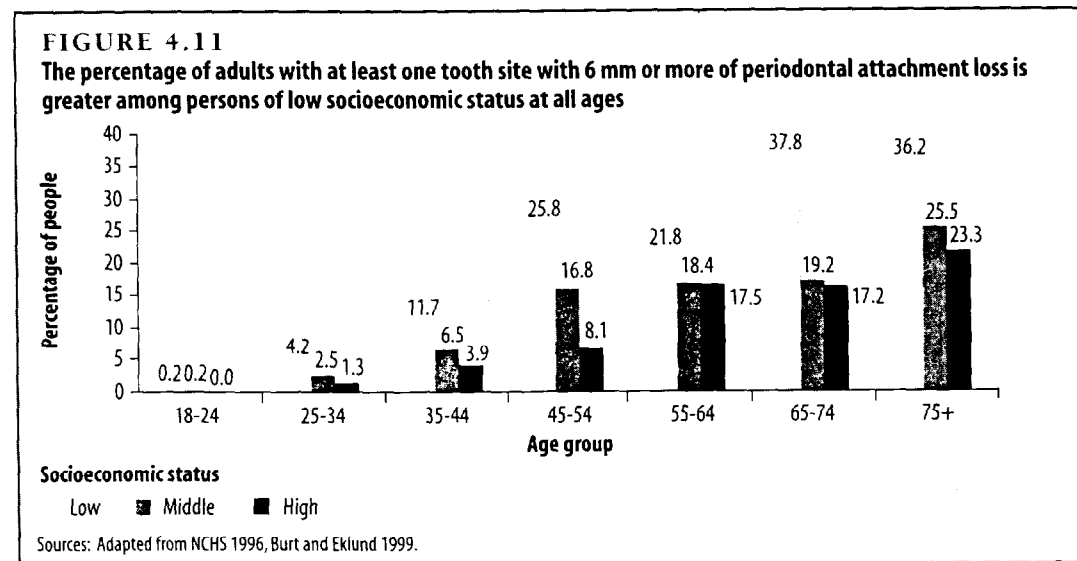
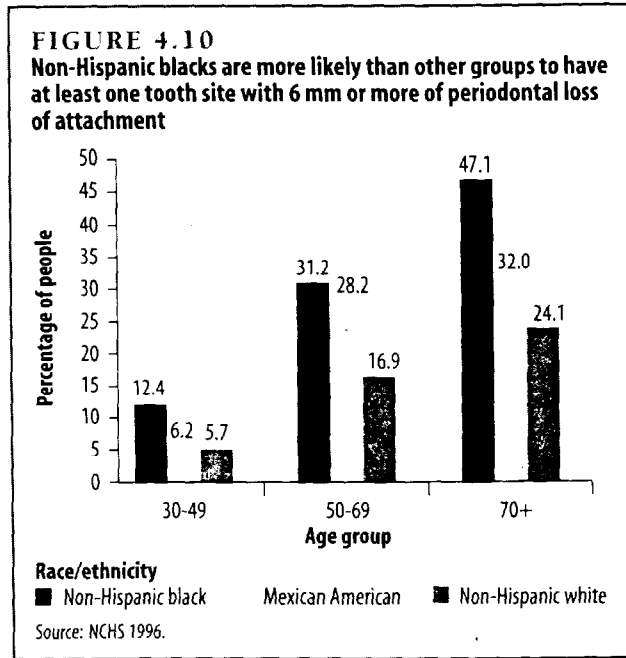
35, has been reported to be 4 times more common in males than in females (Løe and Brown 1991); among 13- to 17-year-olds, it has been found to be highest among African Americans (10.0 percent), as compared with Hispanics (5.0 percent) and whites (1.3 percent) (Albandar et al. 1997).

Tooth Loss and Edentulism

Although teeth are lost for a number of reasons, including trauma, orthodontic treatment, and removal of third molars (wisdom teeth), most teeth are lost because of periodontal disease or dental caries (Phipps and Stevens 1995, Neissen and Weyant 1989). By age 17, more than 7.3 percent of U.S. children have lost at least one permanent tooth because of caries; by age 50, Americans have lost an average of 12.1 teeth, including the third molars.

Men and women are nearly equally likely to be edentulous. Overall, a higher percentage of individuals living below the poverty level are edentulous than are those living above (Figure 4.12). Individuals with incomes equal to or above twice the poverty level have a rate of edentulism of 6.9 percent. This rate is less than half the rate for those with incomes below twice the poverty level (14.3 percent).

Although the overall rate of edentulism for adults 18 and older is approximately 10 percent (9.7 percent), the rate increases with age, so that about a third (33.1 percent) of those 65 and older are edentulous. Comparisons across race/ethnicity for the population 18 years and older indicate that the edentulous rate



¹In this section, income levels are defined as low (less than 185 percent of the U.S. poverty level or below), middle (185.1 percent to 350 percent of the poverty level), and high (350.1 percent of the poverty level or higher).

for non-Hispanic whites is 10.9 percent; for non-Hispanic blacks, it is 8.0 percent; and for Mexican Americans, 2.4 percent. Non-Hispanic whites, both poor and nonpoor, have the highest rates of edentulism compared to non-Hispanic blacks and Mexican Americans. Of the three population groups, Mexican Americans are the least likely to lose all of their teeth, and the proportion of Mexican Americans who are edentulous varies only slightly by economic status. A lower proportion of U.S. adults have lost all their natural teeth now than was the case two decades ago (Figure 4.13). The decline is most pronounced at older ages.

Edentulism is one of a few conditions for which state-specific data exist. These data reveal a wide variation in the percentages of the population aged 65 and older who have no teeth, from a low of 13.9 percent in Hawaii to a high of 47.9 percent in West Virginia; this is more than a threefold difference (Table 4.1) (Tomar 1997). Reasons for these differences are unknown at this time.

Oral and Pharyngeal Cancers and Precancerous Lesions

Oral and Pharyngeal Cancers

Every year, about 1.2 million people develop cancer in the United States (based on 2000 estimates). Sites

in the oral cavity and pharynx (throat) account for about 30,200 cases, or 2.4 percent of all cancers, and about 7,800 Americans die from these cancers each year (ACS 1999). The life of each person with these cancers is shortened by an average of 16.5 years. The median age at diagnosis of oral and pharyngeal cancer is 64, and the rate of occurrence increases with age. More than 95 percent of oral cancers occur in individuals aged 35 and older (Ries et al. 1999).

The overall 5-year survival rate for people with oral and pharyngeal cancers is 52 percent, which is worse than that for—among others—cancers of the prostate, corpus and uterus, breast, bladder, larynx, cervix, colon, and rectum in both blacks and whites (Ries et al. 1999). People with oral cancers detected at an early stage have a 5-year survival rate of 81.3 percent; however, only 35 percent of individuals with oral and pharyngeal cancers are diagnosed at an early stage. The 5-year survival rate drops to 21.6 percent

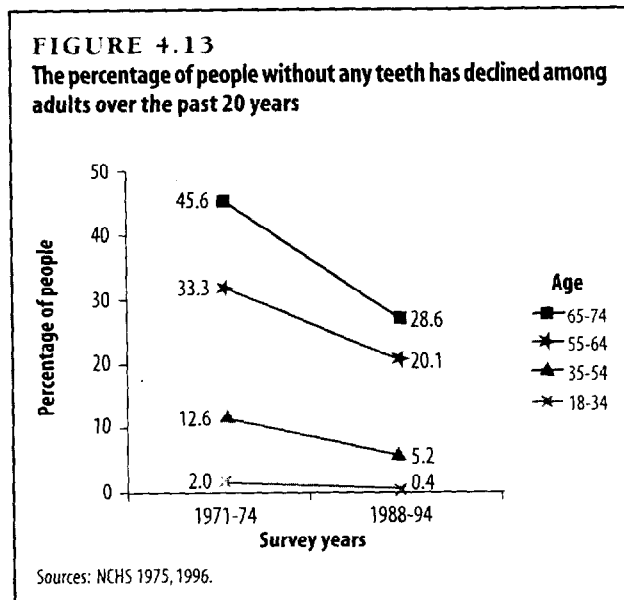
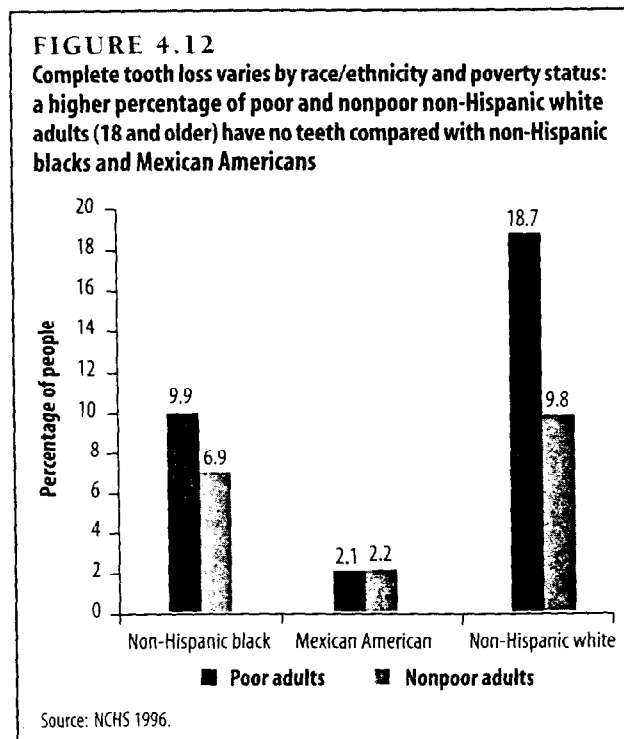


TABLE 4.1
Five states with highest and lowest percentages of edentulous persons aged 65 and older

States with Highest Percentage		States with Lowest Percentage	
State	Percentage Edentulous	State	Percentage Edentulous
West Virginia	47.9	Hawaii	13.9
Kentucky	44.0	California	16.2
Louisiana	43.0	Oregon	16.5
Arkansas	39.2	Arizona	18.5
Maine	37.8	Wisconsin	19.4

Source: Tomar 1997.

The Magnitude of the Problem

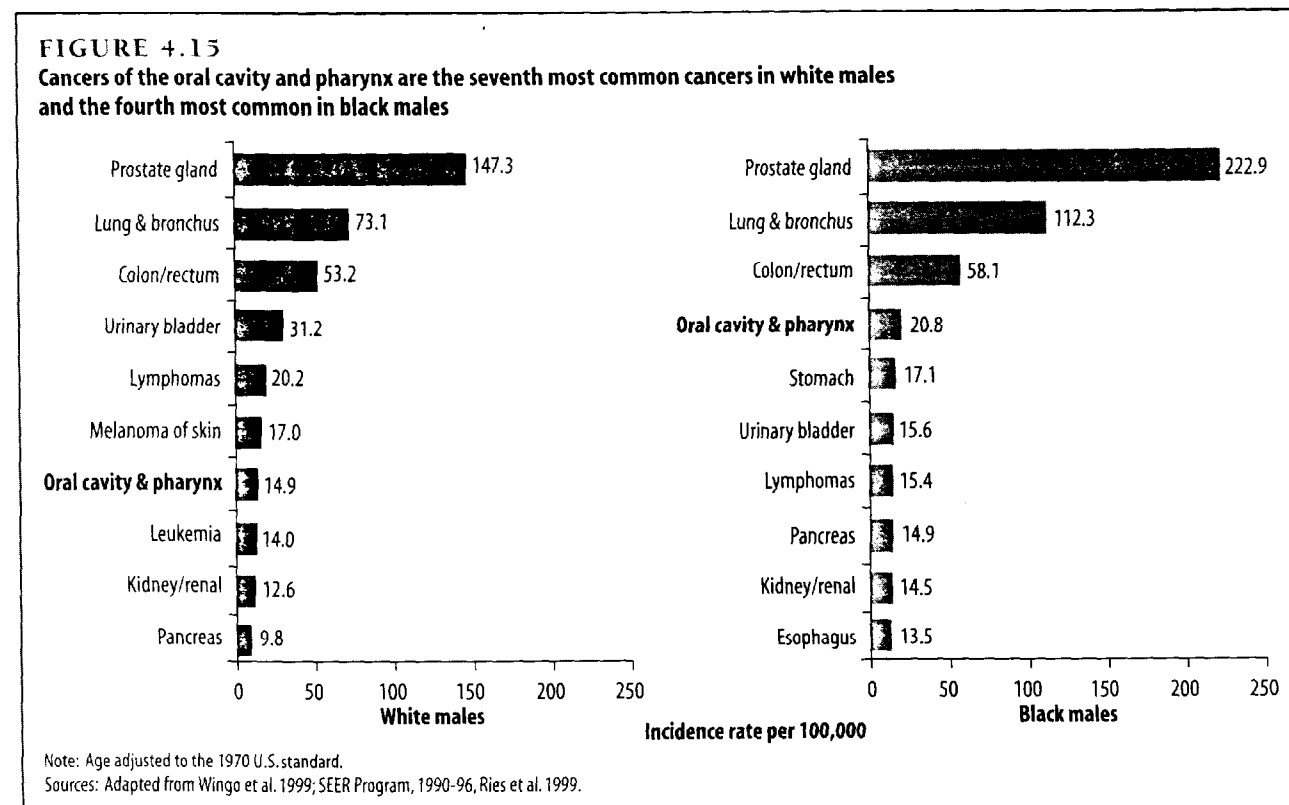
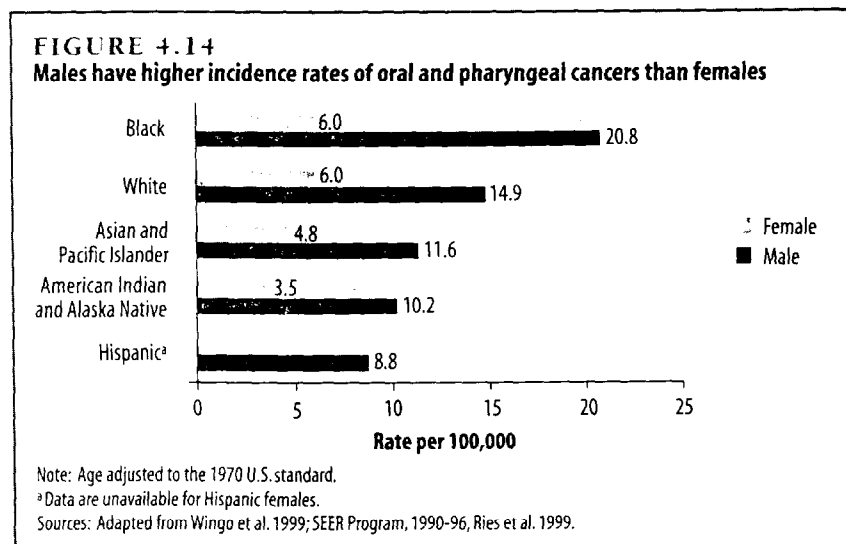
among people diagnosed with advanced-stage cancers (Ries et al. 1999). Compared to patients with other types of cancer, oral and pharyngeal cancer patients who survive have the highest rate of development of new cancers in the mouth or in other parts of the body (Winn and Blot 1985).

Incidence rates for oral and pharyngeal cancers are higher for black individuals than for whites: 12.5 cases versus 10.0, respectively, per 100,000 people each year. In the United States, Asians and Pacific

Islanders (7.9 per 100,000), American Indians and Alaska Natives (6.4 per 100,000), and Hispanics (5.8 per 100,000) have lower incidence rates than whites and blacks (Wingo et al. 1999).

Figure 4.14 provides incidence rates for selected racial/ethnic groups by sex. Males have higher incidence rates than females; specifically, they are 2.6 times more likely to develop oral and pharyngeal cancers than women (Ries et al. 1999). The incidence rates of oral and pharyngeal cancers for black males are 39.6 percent higher than for white males (20.8 versus 14.9, respectively, per 100,000 males per year). Rates for black and white females are the same (6.0 per 100,000 females per year) (Ries et al. 1999). Oral and pharyngeal cancers are the seventh most common cancer among white males and the fourth most frequently diagnosed cancer among black males (Figure 4.15) (Wingo et al. 1999).

As for many other cancer sites, the overall 5-year survival rate for oral and pharyngeal cancers is lower for blacks than for whites: 34 versus 56 percent

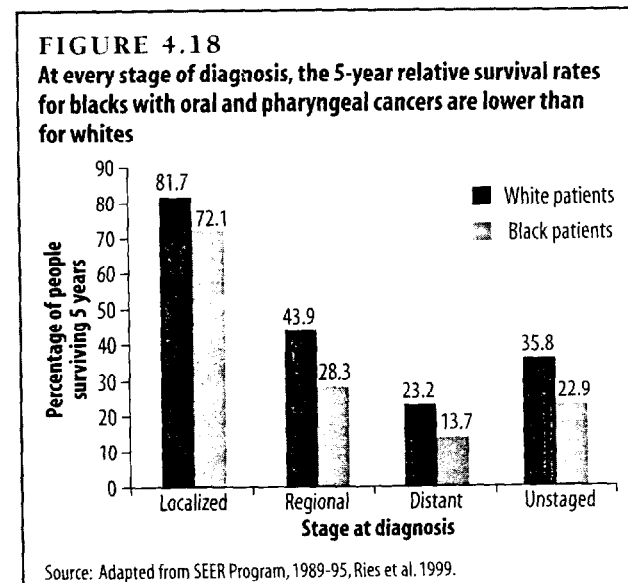
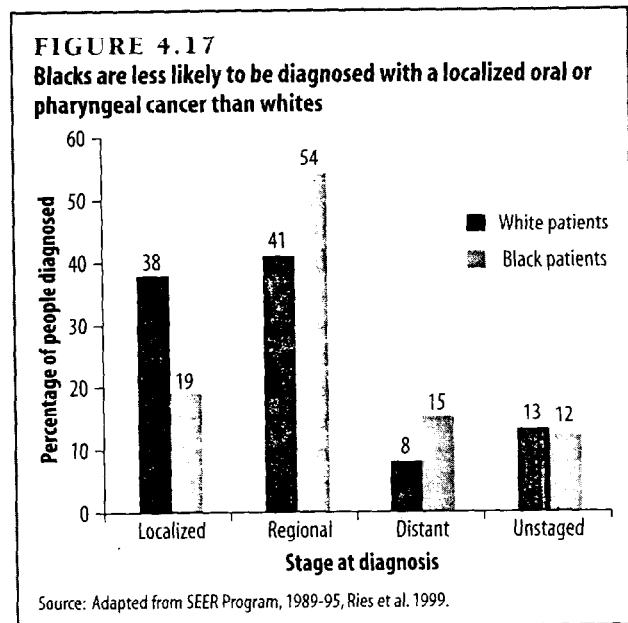
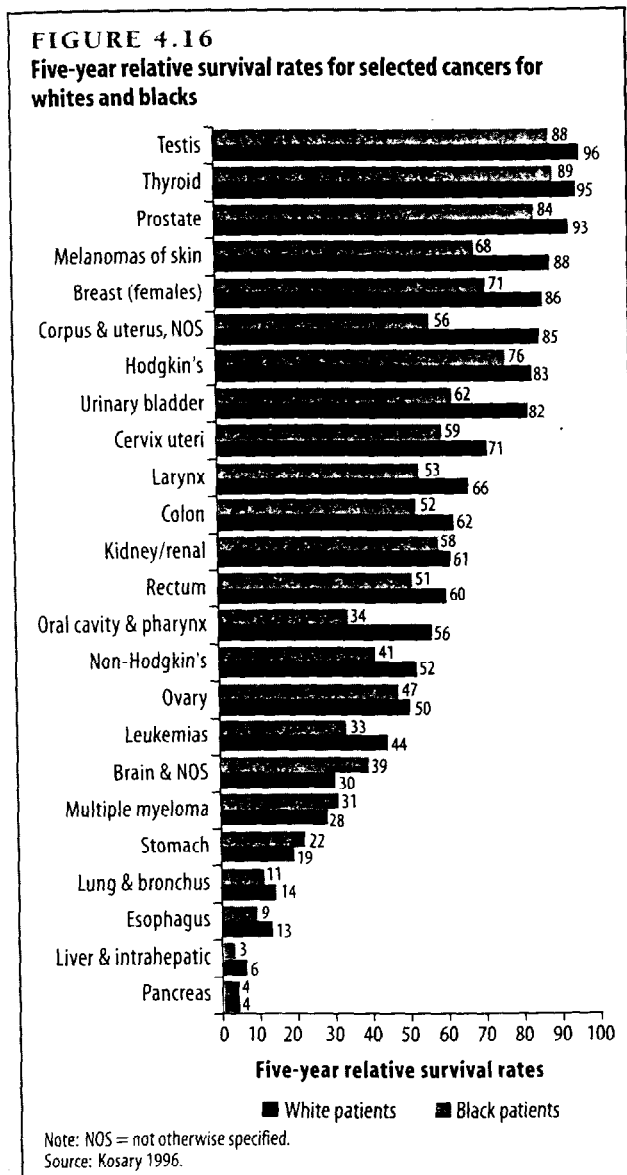


(Ries et al. 1999) (Figure 4.16). However, only 19 percent of blacks with oral and pharyngeal cancers are diagnosed when the cancer is at the local, and more easily treatable, stage, compared to 38 percent for whites (Figure 4.17). At every stage of diagnosis, the survival rate for blacks is lower than for whites (Figure 4.18).

The occurrence of cancers in specific sites within the oral cavity and pharynx varies by sex and race/ethnicity. A relatively rare subtype of pharyngeal cancer, nasopharyngeal cancer, occurs more often in American males and females of Chinese descent than among other racial/ethnic groups (Miller et al. 1996). Blacks are twice as likely as whites to develop cancers of the pharynx: 6.0 and 2.9 per 100,000 per year, respectively (Ries et al. 1999). Individuals with can-

cers of the pharynx generally have a worse survival rate than those with cancers in oral cavity sites: 5-year pharyngeal cancer survival rates range from 53.3 to 29.5 percent, depending on the subsite, whereas oral cavity cancer survival rates range from 94.3 to 48.3 percent. The incidence of lip cancer, a highly treatable cancer, is more common in whites than among blacks (1.2 per 100,000 persons per year compared to 0.1).

Overall, the incidence rate for oral cavity and pharyngeal cancers is decreasing, with an estimated annual percentage decrease of 0.5 percent per year between 1973 and 1996. There are wide variations in the incidence of site-specific cancers. The largest



annual declines in incidence were noted for lip cancer (-3.4 percent per year between 1973 and 1996) (Ries et al. 1999). In contrast, the incidence of tongue cancer, the most common form of oral and pharyngeal cancer, may be increasing among young men (Day et al. 1994).

Although overall mortality rates for oral and pharyngeal cancers declined by 1.6 percent per year between 1973 and 1996, the 5-year survival rate for individuals with oral and pharyngeal cancers has shown no improvement for the past 25 years (Ries et al. 1999).

Mortality statistics by state allow for analysis of deaths due to oral cavity and pharyngeal cancers. Table 4.2 highlights the wide variation in mortality found in the country. The highest rate is in the District of Columbia—6.7 per 100,000 population; this is nearly 5 times the lowest rate, 1.4 in Utah. Again, reasons for this variation need to be studied (Ries et al. 1999).

Tobacco-related Lesions

Tobacco use has been estimated to account for over 90 percent of cancers of the oral cavity and pharynx (Peto et al. 1995) and thus represents the greatest single preventable risk factor for oral cancer. Both smoking and spit (smokeless) tobacco (moist snuff and chewing tobacco) are associated with a number of other oral conditions, including oral mucosal lesions, that may progress to oral cancer (Silverman 1998).

One type of tobacco-related lesion is seen in people who use spit tobacco. A national survey of U.S. schoolchildren in 1985-86 showed that 6.1 percent of males and 0.1 percent of females used spit tobacco. The survey also showed that 34.9 percent of current snuff users aged 12 to 17 and 19.6 percent of current adolescent chewing tobacco users had a spit tobacco lesion (Figure 4.19) (Tomar et al. 1997). The preva-

lence of tobacco-related lesions increased with increasing duration and frequency of spit tobacco use.

In some American Indian tribes, both adolescent males and females commonly use spit tobacco and have an especially high frequency of spit tobacco lesions. On a Sioux reservation, 37.0 percent of students in grades 7 through 12 used spit tobacco. Spit tobacco lesions occurred in over one third of those tobacco-using adolescents (CDC 1988). In another study of Navajo adolescents, three fourths of male adolescents (75.4 percent) and one half of female adolescents (49.0 percent) used spit tobacco. Of Navajo adolescents who used spit tobacco, 25.5 percent had spit tobacco lesions—29.6 percent of males and 17.0 percent of females (Wolfe and Carlos 1987).

Selected Mucosal Infections and Diseases

Oral Herpes Simplex Virus Infections

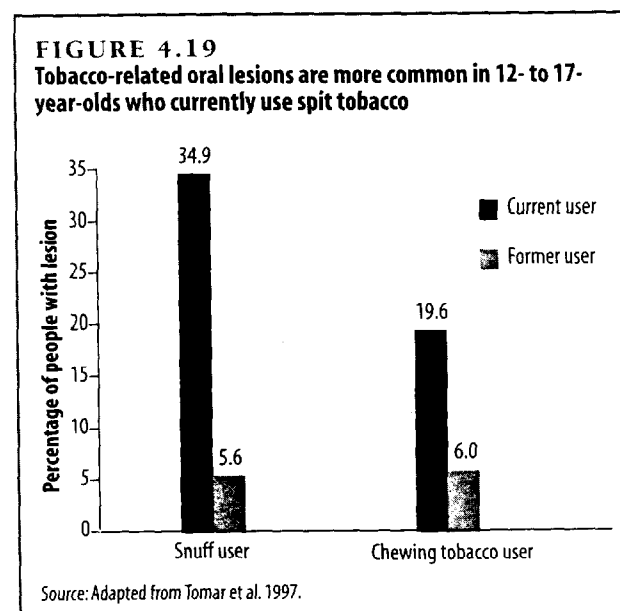
The prevalence of recurrent herpes lesions is estimated to be between 15 and 40 percent (Scully 1989). The proportion of the U.S. population with herpes simplex virus type 1 (HSV-1) antibodies is 68.2 percent (as evidenced by positive antibody titer). The proportion reporting a history of herpes lesions in the past 12 months is 17.7 percent. The presence of antibodies and occurrence of herpes lesions vary by age (Figure 4.20). The frequency of recurrence also varies greatly, ranging from once to several times per year.

Infection with the oral herpes simplex virus has been related to socioeconomic factors, with 75 to 90 percent of individuals from lower socioeconomic

TABLE 4.2
Five states with highest and lowest oral and pharyngeal cancer mortality rates

States with Highest Rates		States with Lowest Rates	
State	Mortality Rate per 100,000 Population	State	Mortality Rate per 100,000 Population
District of Columbia	6.7	Minnesota	2.0
Delaware	4.0	Wyoming	2.0
South Carolina	3.9	Colorado	2.0
Louisiana	3.5	North Dakota	1.8
Florida	3.4	Utah	1.4

Note: Ages adjusted to the 1970 standard population.
Source: Ries et al. 1999.



populations developing antibodies by the end of the first decade of life (Whitley 1993a,b). In comparison, 30 to 40 percent of individuals from middle and upper socioeconomic groups evidence antibodies by the middle of the second decade of life.

The prevalence of one or more herpes labialis lesions within the past 12 months is 8.4 percent for non-Hispanic blacks, 16.2 percent for Mexican Americans, and 19.7 percent for non-Hispanic whites (NHANES III).

Recurrent Aphthous Ulcers

Various epidemiologic studies of recurrent aphthous ulcers have indicated that the prevalence in the gen-

eral population can vary from 5 to 25 percent (Axell et al. 1976, Embil et al. 1975, Ferguson et al. 1984, Ship 1972, Ship et al. 1967). In NHANES III, 17.2 percent of persons reported having a recurrent aphthous ulcer within the past 12 months, and occurrences were most common among young adults (18 to 24 years old) (Figure 4.21). In selected population groups, the prevalence of recurrent aphthous ulcers can be as high as 50 to 60 percent (Miller and Ship 1977, Ship et al. 1961, 1977).

Other Mucosal Lesions

Other mucosal conditions contribute to the burden of oral diseases. The following are among the most common:

- *Oral candidiasis* (commonly called thrush) is a particular problem for individuals with impaired immune function. A prevalence of 9.4 percent has been reported in renal transplant patients (King et al. 1994); Samaranyake (1992) reports prevalences between 43 and 93 percent among HIV-infected patients. It is estimated that 3.6 percent of full denture wearers have candidiasis.

- *Denture stomatitis*, a condition in which the mucosa underneath a denture becomes inflamed and sometimes painful, affects 25.6 percent of people aged 18 and older who have two full dentures. Additionally, 32.2 percent of those with one full denture are affected,

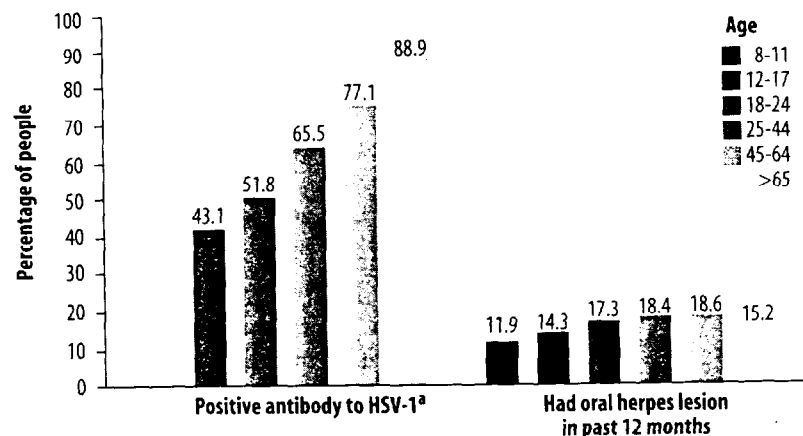
26.7 percent of those with one or more partial dentures, and 0.87 percent of those who do not have full or partial dentures.

- *Oral human papillomavirus infections*, oral and genital papillomas (or condyloma acuminata, also called venereal warts), are especially common among HIV-positive patients. Human papillomaviruses may be associated with some oral leukoplasias with a high risk for malignant transformation (Palefsky et al. 1995).

Developmental Disorders

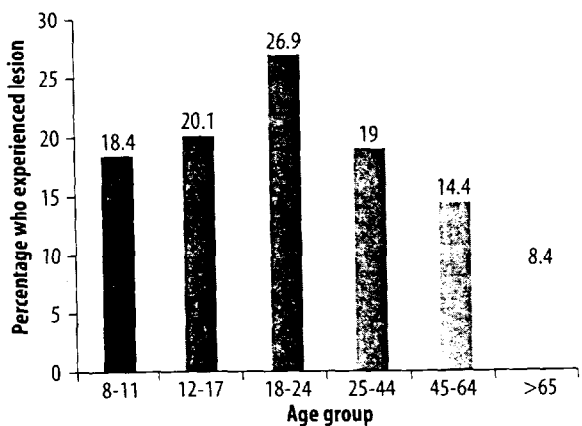
Numerous developmental disorders affect the oral, dental, and craniofacial complex. These include congenitally missing teeth (all or specific tooth types); congenital problems involving tooth enamel, pulp, or dentin; and craniofacial birth defects or syndromes.

FIGURE 4.20
Herpes simplex type 1 virus infection is widespread, and oral herpes lesions (cold sores/fever blisters) are common



^a Data not available for the 8- to 11-year-old age group.
Source: NCHS 1996.

FIGURE 4.21
A substantial percentage of the population, particularly among young adults, has experienced recurrent aphthous lesions (canker sores) in the past 12 months



Source: Adapted from NCHS 1996.

Cleft lip and palate are the most common congenital anomalies and may occur as isolated defects or as part of other syndromes. Other craniofacial defects and syndromes that have been the focus of recent genetics research include ectodermal dysplasia, Treacher Collins syndrome, Apert's syndrome, and Waardenburg syndrome. Craniofacial defects and syndromes have many serious consequences including unusual facial features; severe functional problems; and the need for extensive surgical, medical, and rehabilitative interventions and prosthetic devices.

Cleft Lip/Palate

Oral clefts are one of the most common classes of congenital malformations in the United States, with prevalence rates in the general population of 1.2 per 1,000 births for cleft lip with or without cleft palate and 0.56 per 1,000 births for cleft palate alone (Schulman et al. 1993). These conditions affect facial appearance throughout life.

The rate of oral clefts for whites is more than 3 times that for blacks (1.7 versus 0.5 per 1,000 live births) (Figure 4.22). Oral clefts are more common among North American Indians (3.7 per 1,000 births) (Lowry et al. 1989). Cleft palate occurs more frequently in females, whereas cleft lip or cleft lip/palate

is more common in males (Burman 1985, Fraser and Calnan 1961, Habib 1978, Owens et al. 1985).

Malocclusions

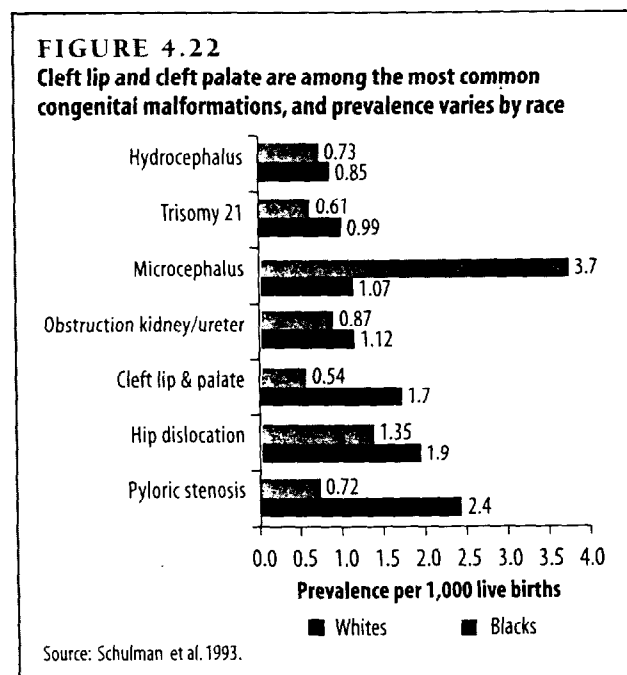
Malocclusions can occur due to congenital or acquired misalignments (crowding) of the teeth or jaws. In a national study of individuals between 8 and 50 years old, 25 percent were found to have no crowding of the incisors (front teeth), whereas 11 percent were found to have severe crowding (Brunelle et al. 1996). About 9 percent had a posterior crossbite, where there is poor contact of the back upper and lower chewing surfaces. This crossbite was most common in non-Hispanic whites. Severe overjet—where the upper front teeth project far forward—was found in approximately 8 percent of this population, with a similar percentage demonstrating a severe overbite—where the front top and bottom teeth greatly overlap when the mouth is closed. Less than 5 percent of non-Hispanic whites had an open bite, an inability to bring the upper and lower front teeth together.

Injury

Injuries to the head, face, and teeth are very common. They can range in severity from the very mild to those that cause death. Although injuries have a major impact on oral health, data on the number and severity of head and face injuries in the United States are very limited.² Most of our knowledge about the number of injuries that occurs comes from more severe injuries that involve a visit to the emergency room.

In 1993 and 1994, there were 20 million visits per year to emergency departments for craniofacial injuries. Less serious injuries can be treated on an outpatient basis. More than 5.9 million injuries in 1991 were treated by dentists in private offices (Gift and Bhat 1993). Overall, 25 percent of all persons aged 6 to 50 have had an injury that resulted in damage to one or more anterior teeth (Kaste et al. 1996a). An estimated 2.9 million emergency room visits for all age groups related to tooth or mouth injuries between 1997 and 1998. Twenty-five percent of these injuries were seen in children under the age of 4 (NCHS 1997b).

The leading causes of head and face injuries that result in emergency room visits include falls, assaults,



²This section reports national data that should provide some estimation of the scope of craniofacial injuries in the population. However, the findings may not be directly comparable because they are from different sources at different times, and because those at risk for each type of injury are not quantified in most cases.

sports injuries, and motor vehicle collisions (De Wet 1981, Pinkham and Kohn 1991, Sane 1988). In the National Health Care Survey of emergency rooms, assaults and falls each accounted for 31 percent of visits related to head and face injury. Other studies have reported that up to 19 percent of head and face injuries are sports-related (McDonald 1994), and 5 percent of head and 19 percent of face injuries result from riding bicycles and tricycles (U.S. Consumer Product Safety Commission 1987).

There are differences in rates of emergency room visits for head and face injuries among demographic groups. Males had higher rates than females, except among older adults. The rates of injury were higher for younger and older adults than for those in the middle years.

Chronic and Disabling Conditions

Oral-Facial Pain

Oral-facial pain can greatly reduce quality of life. These types of pain may be due to tooth-related infections, mucosal sores, and irritations, and may include burning sensations, pain in the jaw joint area, and aching pain across the face or cheek. Over 39 million people, or 22 percent of adults 18 years of age and older in the civilian U.S. population, experienced at least one of five types of oral-facial pain during a recent 6-month period (Lipton et al. 1993). Based on the results of a national study of the prevalence and distribution of oral-facial pain, it is estimated that during a 6-month period, 1 American adult in 8 (12.2 percent) suffers from toothache, 1 in 12 (8.4 percent) from painful oral sores, 1 in 19 (5.3 percent) from jaw joint pain, and 1 in 71 (1.4 percent) from face or cheek pain (Lipton et al. 1993).

The prevalence of toothache and pain due to oral sores decreases with age, whereas the prevalence of burning mouth pain increases with age. Women are twice as likely as men to report two specific types of oral-facial pain: jaw joint pain and face/cheek pain (Figure 4.23). Non-Hispanic blacks and Hispanics were slightly more likely to report toothache than non-Hispanic whites (Lipton et al. 1993). Adults living in poverty were more likely to report toothaches than adults living above the poverty level (Vargas et al. 2000).

Temporomandibular Disorders

Symptoms of temporomandibular disorders (TMDs) vary but may include severe pain in the jaw musculature, severe pain or difficulty when opening the

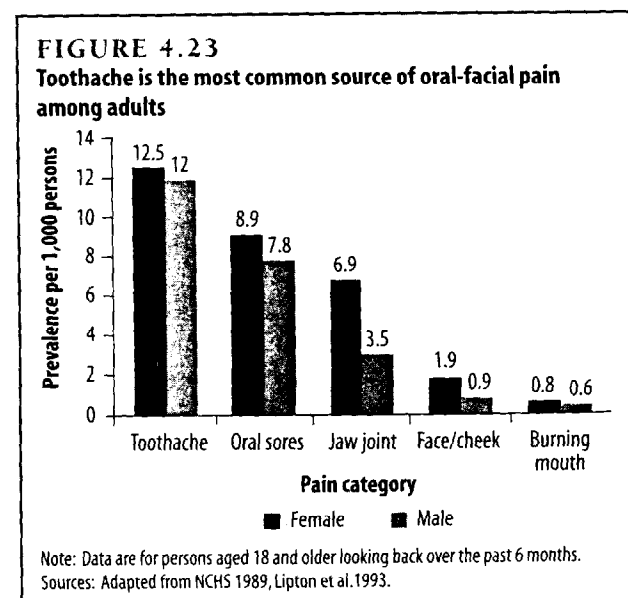
mouth and chewing, headaches, and ear pain. Based upon assessments of pain in or around the jaw joint, these disorders are estimated to affect 10 million Americans (Lipton et al. 1993).

Data from the few available population-based epidemiologic studies indicate that the prevalence of self-reported pain symptoms and clinical signs of TMD pain is between 5 and 15 percent, with peak prevalence in young and middle-aged adults (20 to 40 years of age) (Von Korff 1995).

Although physical signs associated with TMDs have been shown to occur with nearly equal frequency among men and women, clinical studies have found that women in the third and fourth decades of life were much more likely than men of the same age to have sought care for reported facial pain in the temporomandibular region (Carlsson and LeResche 1995).

Sjögren's Syndrome

Sjögren's syndrome, an autoimmune disorder that causes xerostomia (dry mouth), difficulty in swallowing, and xerophthalmia (dry eyes), is estimated to affect 1 to 2 million people in the United States (Talal 1992). The diagnosis is most often made in women in middle age. One estimate of the average annual incidence rate for Sjögren's syndrome, based on the Olmsted County, Minnesota, medical database, is about 3 to 5 cases per 100,000 population; this may be low, however (Pillemer et al. 1995). As with most other autoimmune conditions (e.g., rheumatoid arthritis, systemic lupus erythematosus), Sjögren's syndrome affects more women than men. The female-to-male ratio depends on the study, but may be as high as 9:1 (Fox 1996).



WHAT IS THE BURDEN OF DISEASE IN SELECTED POPULATIONS? CHALLENGES AND OPPORTUNITIES

The national data provide a broad-brush picture of America's oral health. For selected populations, however, oral health and disease status has a different profile. By 2050 about 50 percent of the U.S. population is expected to be Asian, non-Hispanic black, Hispanic, and American Indian (Council of Economic Advisers 1998). Currently available data for these groups present a picture of disease that is generally poorer than that for non-Hispanic whites. These subgroups present a unique cluster of health, socioeconomic, and cultural issues. At the same time, data for the subgroups within each of these categories are lacking. In addition to racial/ethnic groups, other groups such as individuals with disabilities, the homeless, incarcerated individuals, and migrant workers have unique needs and challenges. Cutting across all subgroups are gender-specific health issues. For improvements to be made in America's overall health, a better understanding of the full dimension of the problems faced by these populations and development of specific solutions are needed. This part of the chapter examines each subgroup in greater depth.

Racial and Ethnic Minorities

Although there have been gains in oral health status for the population as a whole, they have not been evenly distributed across subpopulations. Non-Hispanic blacks, Hispanics, and American Indians and Alaska Natives generally have the poorest oral health of any of the racial and ethnic groups in the U.S. population. Other health statistics, such as life expectancy and infant mortality, indicate that the general health of these groups is varied and also poor compared to other population groups (Council of Economic Advisers 1998). To address the elimination of these disparities in health—and also in housing, education, and other indicators of social and economic well-being—the administration has launched “The President's Initiative on Race: One America in the 21st Century.” Recommendations for improving the oral and general health status of racial and ethnic minorities are also a prominent feature of Healthy People 2010, the goal-setting health agenda developed for the decade by the USDHHS (2000).

African Americans

Numerous studies over the decades have compared the health status of blacks and whites in American

society, but relatively little systematic attention has been focused on the oral health of blacks. Although the overall oral health status of Americans has been improving, many oral diseases and conditions among blacks remain a serious problem—despite the fact that for almost three decades these disparities have been highlighted and recommendations made for addressing health issues including research, education, human resources, and delivery systems (National Dental Association 1972). These recommendations still represent opportunities for improvement in the oral health status of African Americans.

Baseline data for the Healthy People 2010 objectives establish that, for children aged 2 to 4 years, 24.0 percent of non-Hispanic blacks have experienced dental caries in their primary teeth, compared to 15.0 percent for their non-Hispanic white counterparts. For children aged 6 to 8, there were no differences among the races; but for 15-year-olds, a higher percentage of non-Hispanic blacks were affected than of whites. In addition the percentage of people of all ages who had untreated caries was substantially higher for blacks than for whites—about twice as many. Higher levels of gingivitis and periodontal loss of attachment were also seen in non-Hispanic blacks as compared to non-Hispanic whites.

A greater percentage of non-Hispanic blacks 18 years and older have missing teeth when compared to non-Hispanic whites. Relative to non-Hispanic whites, however, non-Hispanic blacks aged 18 and older are less likely to have lost all their teeth (edentulism) regardless of whether they are poor.

African American males have the highest incidence rate of oral cavity and pharyngeal cancers in the United States compared with women and other racial/ethnic groups (Wingo et al. 1999). The distribution of oral cancer cases reveals that blacks also have a higher proportion of pharyngeal cancer than oral cavity cancer compared to whites. Also, the 5-year relative survival rate (1989-95) for oral cancer was much lower among blacks than whites: 34 versus 56 percent (American Cancer Society (ACS) 1999). This latter finding may be related to the fact that a high percentage of these cancers are diagnosed in later stages of disease in blacks as compared to whites (ACS 1999).

On the other hand, for several conditions, African Americans have a lower disease burden than do whites. The incidence rate for cleft lip and cleft palate in African Americans is 0.54 per 1,000, about a third the rate for whites (1.70 per 1,000). Also the prevalence of having one or more herpes labialis lesion within the past 12 months is 50 percent less

than that for Mexican Americans and non-Hispanic whites.

Hispanics

Disparities in oral and general health status between Hispanic and non-Hispanic populations in the United States have been long recognized. Yet the health profile of Hispanics is incomplete due to insufficient sampling of subgroups in national surveys, inconsistent or inadequate assessment of ethnicity, or ambiguities in reporting of ethnic identity (Hahn 1992). Recent efforts to improve data collection, identify subgroups, and provide more baseline data for Hispanics have addressed the situation somewhat, but much work remains to ensure accurate data for health planning and research (Delgado and Estrada 1993).

Among preschool Hispanic children early childhood caries is a particular concern. Two reports have documented early childhood caries among 12.9 percent of Hispanic children examined in San Antonio and 37 percent of predominantly Hispanic children in San Francisco (Garcia-Godoy et al. 1994, Ramos-Gomez 1999). Most recently, national survey data suggest that a higher proportion of Mexican American children ages 12 to 23 months may experience dental caries than other race/ethnicity groups (Kaste et al. 1996b).

Preliminary data from NHANES III indicate that young Mexican American children aged 2 to 4 are more likely to have experienced dental caries in their primary teeth, have on average more decayed and filled tooth surfaces, and have more untreated disease than either non-Hispanic white or non-Hispanic black children (Kaste et al. 1996b). Mexican American children aged 2 to 5 years—especially those from lower-income households—were more likely than their African American and non-Hispanic white counterparts to have one or more decayed primary teeth (Vargas et al. 1998).

Dental caries continues to affect large numbers of school-age children and youth, as only 30 percent of Mexican Americans, 32 percent of non-Hispanic whites, and 41 percent of non-Hispanic blacks 12 to 17 years of age were free of caries in their permanent teeth (USDHHS 1996). However, most of the dental caries in the permanent teeth of non-Hispanic white children aged 12 to 17 had been treated or filled (87 percent), compared to 63 percent for Mexican Americans and 60 percent for non-Hispanic blacks.

The only large-scale survey that permits comparison among Hispanic subgroups is the Hispanic Health and Nutrition Examination Survey (HHANES 1982-84). After controlling for age, sex, income, and

education, HHANES results show that Cuban American and Puerto Rican adults had about twice as many missing teeth as Mexican Americans. Puerto Ricans and Cuban Americans also had on average more filled teeth than Mexican Americans. Puerto Rican children and adults under 45 years old had more gingivitis than Cuban Americans and Mexican Americans; the highest prevalence of periodontal disease was reported among Puerto Ricans compared to the two other Hispanic groups (Ismail and Szpunar 1990).

A national survey found that employed Hispanic adults were twice as likely to have untreated dental caries as non-Hispanic whites. In this study, gingivitis and periodontal problems (attachment loss and pockets) were also among the more common problems among the Hispanic adults studied (Watson and Brown 1995).

Analysis of a more recent survey (NHANES III) that sampled Mexican Americans is particularly revealing. After adjusting for age, sex, educational attainment, and annual family income, Mexican American adults are similar to their white non-Hispanic counterparts on most oral health indicators. However, among Mexican Americans, individuals in families with less than \$20,000 annual income were 1.6 times less likely to have an intact dentition, 3.1 times more likely to have any untreated decay on the crowns of their teeth, and 4.2 times more likely to have severely decayed teeth (very large cavities or only the roots of teeth remaining) than non-Hispanic whites (Garcia and Drury 1999). Also, Mexican Americans were less likely to be edentulous regardless of poverty status than either non-Hispanic whites or non-Hispanic blacks (Drury et al. 1999).

These findings confirm the importance of controlling for sociodemographic factors in reporting on oral health status as well as the need to assess other factors related to health status. As a group, Hispanics have lower median incomes, higher poverty rates, more unemployment, and less education than non-Hispanic whites (Ramirez 1999). However, sociodemographic factors are just one aspect of the questions raised when attempting to understand differences in oral health. The effect of financial barriers and nonfinancial factors such as language, culture, dietary patterns, and behaviors on access, care seeking, and health outcomes must also be examined. Variations in conditions such as diabetes also may contribute to differences in oral health.

It is estimated that Hispanics will surpass African Americans as the country's largest minority group by 2020 (U.S. Bureau of the Census 2000). Aggregate statistics obscure substantial variations within

Hispanic subgroups. More than 20 different countries of varied cultural, socioeconomic, and political backgrounds are currently included in this category of the U.S. population. Narrowing the gap in oral health between Hispanic and non-Hispanic groups will require improved data on health status, barriers to access, and disease factors underlying differences in oral health in these populations.

Asians, Native Hawaiians, and Other Pacific Islanders

National data for the oral health of Asian, Native Hawaiian, and other Pacific Islander (ANHPI) groups that can be generalized to the U.S. population are not available. Instead the profile of disease and health in this category is only available through studies of specific states and locales. Among all ethnic groups in California in 1993 and 1994, Asian and Pacific Islander American (APIA) children in Head Start had the highest prevalence of early childhood caries—20 percent compared to 14 percent for all Head Start children (Pollick et al. 1997). These data are comparable to other survey findings of 16 to 20 percent and 29 percent early childhood caries among APIA children in Hawaii and California, respectively (Greer unpublished, Louie et al. 1990).

A California study of 6- to 8-year-olds found disparities in the oral health status of APIA children in the state when compared to all children nationally. Among the California APIA children, 71 percent had untreated dental caries, with a significant portion of this group requiring urgent dental treatment. By comparison, NHANES III data indicate that in 1988-94, 29 percent of children in the United States aged 6 to 8 years had untreated dental decay.

There is variation in oral health status among subgroups of ANHPI children. In a recent survey in Hawaii, the prevalence of early childhood caries among APIA children was 16 percent, ranging from a low of 8 percent among Japanese children to a high of 25 percent among Filipino children. The prevalence of untreated dental caries in 6- to 8-year-old APIA children was 39 percent, which ranged from a low of 16 percent among Japanese children to 40 percent among Native Hawaiians, 48 percent among Southeast Asians, and 62 percent among non-Native Hawaiian Pacific Islanders (Greer 1999).

Oral cancer incidence and mortality rates for APIAs are lower than those for white non-Hispanics and African Americans. However, nasopharyngeal cancer incidence and mortality rates among Chinese and Vietnamese populations are many times higher than other groups (Miller et al. 1996), and therefore pose a unique health problem for these subgroups.

Until recent years, vital statistics and other health-related data were virtually nonexistent for the APIA population. Data for this group generally appeared in the “other” category of national surveys, and thus were not helpful in determining specific population-based oral or general health needs. Little national focus has been given to defining and measuring the oral health problems and related health care needs of the APIA population. These needs are now highlighted in the 2010 Healthy People Oral Health Objectives. A few statewide oral health data exist for some APIA child populations, but no ethnic subgroupings can be assessed. Again, this category of the U.S. population is extremely heterogeneous. It is estimated that 76 percent is from one of five ethnic origins and that 74 percent in 1990 were foreign born. More than 63 percent live in four states: California, New York, Hawaii, and Texas. Consequently, determining the reasons for variations in oral health will require additional data.

American Indian/Alaska Native Populations

Data on the oral health of American Indians and Alaska Natives (AI/AN) are available through studies conducted by the Indian Health Service (IHS) (Niendorfs 1994). The AI/AN people constitute about 1 percent of the U.S. population, or an estimated 2.5 million people in 2000. Little is known or can be easily determined about the general or oral health status of the 1 million AI/AN people not served by the IHS system. For this reason, with the exception of overall death rates obtained from census data, the statistics described in this section will be limited to the 1.5 million AI/AN served by the IHS. By and large, this group represents AI/AN people living on or near reservations.

Preliminary analyses of the IHS-wide Oral Health Status Survey of over 13,000 dental patients in 1999 revealed that some conditions have worsened and some improved since an earlier survey conducted in 1991 (IHS 1994, 2000). Across the IHS service population there was a statistically significant increase in caries among adults over 55 as measured by the decayed, missing, and filled teeth index. The decayed and filled tooth rate increased from 7.5 to 8.8 teeth, with no change in the average number of missing teeth for this age group.

Among AI/AN children across the IHS, there was a significant decline in caries in the permanent dentition and a significant increase in caries in the primary dentition. Among children aged 2 to 5 years, the increase in decayed and filled primary teeth surfaces went from 8.6 to 11.4. In general, AI/AN populations have much greater rates of dental caries and

periodontal disease in all age groups than the general U.S. population. AI/AN children aged 2 to 4 years have 5 times the rate of dental decay compared to all children, and 6- to 8-year-old AI/AN children have about twice the rate of dental caries experience. Rates for untreated decay in these age groups are 2 to 3 times higher than in the same age groups in the general U.S. population. Periodontal disease in AI/AN adults is 2.5 times greater than in the general U.S. population. High prevalence rates of diabetes among AI/AN populations are a significant contributing factor to this periodontal disease (IHS 2000).

Substantial unmet dental needs and quality of life issues have also been identified in IHS surveys, which included studies of representative AI/AN communities with regard to the effect of oral conditions on well-being and quality of life (Chen et al. 1997). (See Chapter 6 for a general discussion.) One third of schoolchildren report missing school because of dental pain. Twenty-five percent of schoolchildren avoid laughing or smiling, and 20 percent avoid meeting other people because of the way their teeth look. As a consequence of dental pain, almost a quarter of the adults are unable to chew hard foods, almost 20 percent report difficulty sleeping, and 15 percent limit their work and leisure activities. Three quarters of the elderly experience dental symptoms, and half perceive their dental health as poor or very poor and are unable to chew hard food. Almost half the adults avoid laughing, smiling, and conversation with others because of the way their teeth look.

Again, the available data allow for obtaining a picture only of the AI/AN population residing on reservations where services, including dental services, have been provided by the IHS or contracted to tribes or urban AI/AN organizations. In 1989, American Indians, residing in the current reservation states had a median household income of \$19,897. Almost one third (31.6 percent) of AI/ANs lived below the poverty level. For some groups, diabetes and high rates of tobacco and alcohol use are prevalent and contribute to poor oral health.

Women's Health

Analysis of data from NHANES III indicates that women have benefited from the trend in general improvements in oral health that has been enjoyed by the U.S. population overall. Many, but not all, statistical indicators show women to have improved their oral health status as compared to men (NHANES III, Redford 1993). Adult females are less likely than males at each age group to have severe periodontal disease as measured by periodontal loss

of attachment of 6 mm or more for any tooth. Both black and white females (6.0 and 6.0 per 100,000) have a substantially lower incidence rate of oral and pharyngeal cancers compared to black and white males, respectively (20.8 and 14.9 per 100,000). A higher prevalence of females than males have orofacial pain, including pain from oral sores, jaw joints, face/cheek, and burning mouth syndrome. However, there are large areas for which information for either sex, even at the descriptive level, is only partial or nonexistent. Data gaps regarding craniofacial injuries, soft tissue pathologies, and salivary gland dysfunctions are notable examples.

Most oral diseases and conditions are complex and represent the product of interactions between genetic, socioeconomic, behavioral, environmental, and general health influences (Chapters 3 and 5). Multiple factors may act synergistically to place subgroups of women at higher levels of risk for oral diseases. For example, the comparative longevity of women, compromised physical status over time, and the combined effects of multiple chronic conditions often with multiple medications, can result in increased risk of oral disease (Redford 1993). Many women live in poverty, are not insured, and are the sole head of their household. For these women, obtaining needed oral health care may be difficult. In addition, gender-role expectations of women may also affect their interaction with dental care providers and could affect treatment recommendations as well (Redford 1993).

During the past decade, women's health has emerged as a significant issue in the nation's health agenda. The scientific community is beginning to respond to this concern by studying and reporting the effects of sex and gender differences on health and disease management. Although most of the effort has focused upon women, comparisons with men's health have begun to elucidate sex- and gender-specific differences.

Research has demonstrated sex and gender differences in the response to kappa opioid analgesics for the control of postoperative pain (Gear et al. 1996). These findings have heightened conjecture about differences in the female and male nervous systems in response to pain stimuli. There are studies in mice that suggest that there are sex-specific responses to pain and analgesics (Mogil et al. 1996, 1997). Taken together, these findings could help explain why women report certain painful conditions more than men; for example, temporomandibular joint disorders, trigeminal neuralgia, migraine headaches, and burning mouth syndrome (USDHHS 1999).

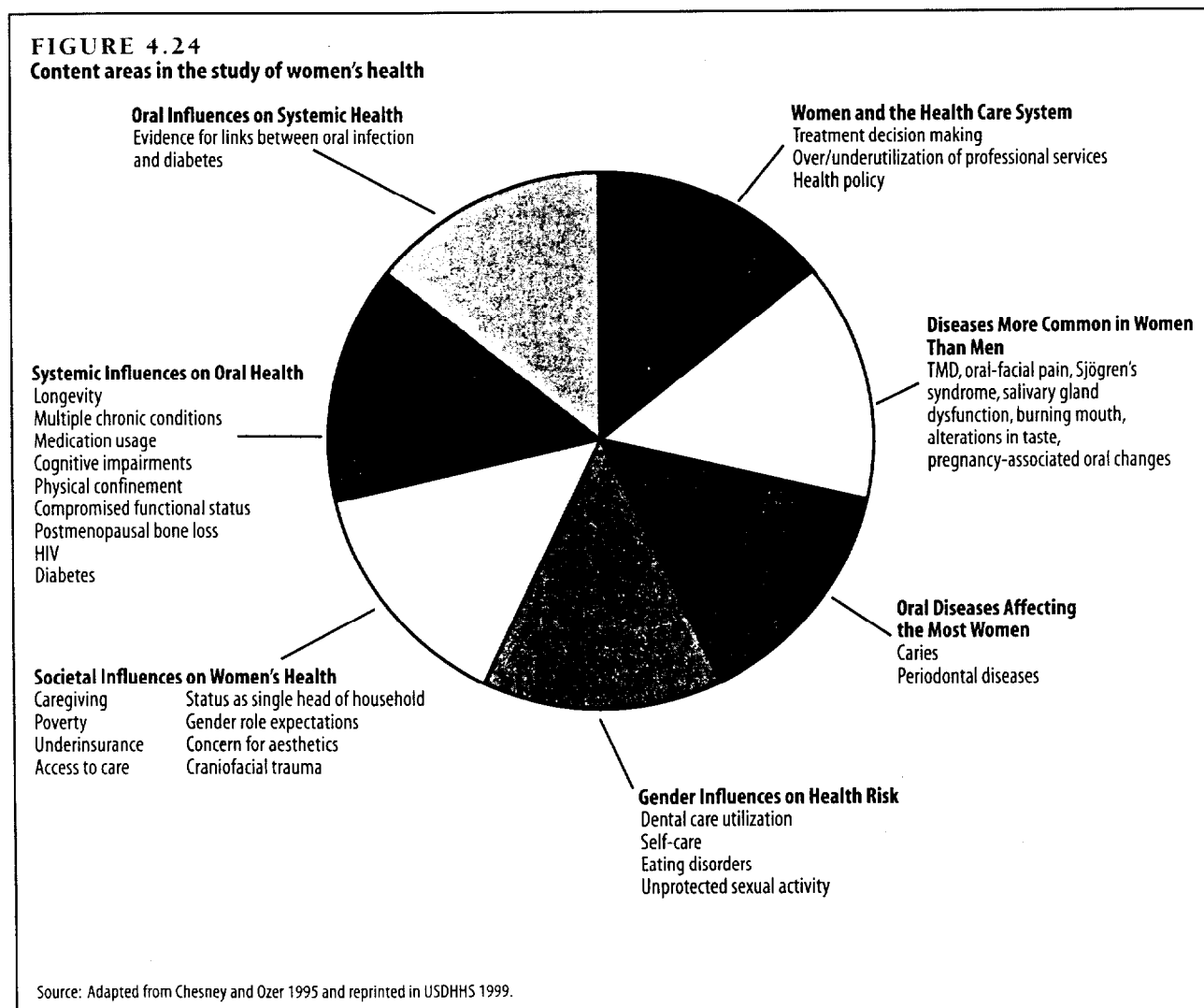
Recent research has also demonstrated sex and gender differences in taste perception. Women are more likely than men to be “supertasters” of a bitter compound known as 6-n-propylthiouracil (PROP) (Bartoshuk et al. 1994). PROP supertasters experience more intense tastes (particularly for bitter and sweet), a greater sensation of oral burning in response to alcohol, and more intense sensations from fats in food (Bartoshuk et al. 1994, 1996, Tepper and Nurse 1997). PROP supertasters also have more fungiform papillae on their tongues than medium PROP tasters or those who cannot taste PROP at all.

The Agenda for Research on Women’s Health for the 21st Century noted that the ability to interpret oral health in the context of sex and gender was limited by large gaps in knowledge. For example, pertinent oral health data, even at the descriptive level, are partial or nonexistent for many conditions and diseases for either sex. In addition, limited knowledge of etiologic factors, natural history of diseases, behav-

ioral and environmental differences—to name a few—decreases the utility of those data that are available. For example, women are reported to be more inclined to self-care, to visit the dentist more often, and to be more likely to report symptoms such as pain. However, the effects of these behaviors on their oral health status cannot be determined fully. Figure 4.24 suggests content areas in the study of women’s oral health.

Individuals with Disabilities

No national studies have been conducted to determine the prevalence of oral and craniofacial diseases among the various populations with disabilities. Several local and regional reports, however, provide some relevant data in this regard. For example, some smaller-scale studies show that the population with mental retardation or other developmental disabilities has significantly higher rates of poor oral hygiene



and needs for periodontal disease treatment than the general population, due, in part, to limitations in individual understanding of and physical ability to perform personal prevention practices or to obtain needed services. There is a wide range of caries rates among people with disabilities, but overall their rates are higher than those of people without disabilities. Much of the variation stems from where people reside (e.g., in large institutions where services are available versus in the community where services must be secured from community practitioners). Almost two thirds of community-based residential facilities report that inadequate access to dental care is a significant issue (Beck and Hunter 1985, White et al. 1995, Waldman et al. 1998, Dwyer, Northern Wisconsin Center for the Developmentally Disabled unpublished data, 1996). Parents consistently report dental care as one of the top needed services for their children with disabilities regardless of age (Haveman et al. 1997). Local studies of independent living centers reported that 24 to 30 percent of adults with cerebral palsy, 14 percent with spinal cord injuries, 30 percent with head injuries, and 17 percent who were deaf had dental problems (Arnett 1994). Results from 1999 oral assessments of U.S. Special Olympics athletes (all ages), based on an extremely conservative assessment protocol (without the use of x-rays, mirrors, or explorers), and carried out by the Special Olympics Special Smiles Program in 20 states, indicate that 12.9 percent of the athletes reported some form of oral pain, 39 percent demonstrated signs of gingival infection, and nearly 25 percent had untreated decay (Special Olympics, Inc., unpublished data). Note that this is a population that tends to be from higher-income families.

The oral health problems of individuals with disabilities are complex. These problems may be due to underlying congenital anomalies as well as to inability to receive the personal and professional health care needed to maintain oral health. There are more than 54 million individuals defined as disabled under the Americans with Disabilities Act, including almost a million children under age 6 and 4.5 million children between 6 and 16 years of age. A greater percentage of males than females and of African Americans than Hispanics and whites have disabilities (Federal Interagency Forum 1997, Waldman et al. 1999). Children with disabilities have chronic physical, developmental, behavioral, and emotional limitations, including mental retardation, autism, attention deficit hyperactivity disorders, and cerebral palsy. Also, children from families with incomes below the poverty level are about one third more likely than children in nonpoor families to have an exist-

ing special health care need. Similarly, children from less educated households exhibit a higher likelihood of a special health care need. Children in single-parent families are about 40 percent more likely than children from two-parent households to have special health care needs (Newacheck et al. 1998). Deinstitutionalization has resulted in highlighting the problem these individuals have regarding access to dental care as they move from childhood to adulthood. Availability of dental providers trained to serve special needs populations and limited third-party support for the delivery of complex services (see Chapter 9) further complicate the issues entailed in addressing the needs of this population.

Given the wide variability among groups with disabilities, this review of oral health status and needs is quite limited. More in-depth assessment and analysis of the determinants of oral health status, access to care, and the role of oral health in the overall quality of life and life expectancy of individuals with disabilities are needed (see Chapter 10).

UTILIZATION OF PROFESSIONAL CARE: WHAT DO WE KNOW ABOUT THE RELATIONSHIP OF ORAL HEALTH AND USE OF DENTAL SERVICES?

With few exceptions, maintenance of oral health through a lifetime requires timely receipt of advice for self-care, preventive therapies, early detection and treatment of problems, and restoration of function. Chapter 7 describes community-based and professional interventions that have played a significant role in the improvement of oral health achieved over the past 50 years; their full promise has not, however, been realized. Chapter 8 describes current and emerging strategies for personal and provider approaches to maintain and restore oral health, with tooth-conserving approaches being employed more and more frequently. As noted earlier, almost everyone experiences oral diseases and conditions over the course of a lifetime, and, unlike the common cold, most diseases do not resolve over time. Consequently, receipt of dental services complements self-care as a critical factor in achieving and maintaining good oral health.

Although certain counseling and screening services provided by physicians are recommended (U.S. Preventive Services Task Force 1996), data to indicate how many persons receive such services or oral health-related recommendations from their physician are very limited. There are also no data on physician-based services for oral and craniofacial conditions. The data that *are* available describe utilization

of dental visits. Unfortunately, most of these data are cross-sectional, describing the experience of the population in any given year, but providing little detail about how patterns of care over time contribute to oral health. Nevertheless, utilization of care is used as a surrogate measure of an individual's or a population's capacity to maintain or improve health status. An understanding of utilization of dental visits and differences in such visits among age, racial/ethnic, sex, and income groups is important in identifying opportunities for improvement in oral health that would follow from timely receipt of professional care.

Characteristics of groups with different levels of dental care utilization suggest barriers to care as well as factors that predispose or enable access to dental care. Explanations for variation in utilization are alluded to in the following section, and are discussed in further detail in Chapter 10. More studies are needed to understand the dimensions of disease and the role of professional care and use of services. Also, for oral health in particular, the contributions of all health professions and the interdisciplinary nature of care need to be emphasized.

Dental Care Utilization

Visiting a health care provider at least once per year and the number of visits made within the past year are used as indicators of an individual's ability to access professional services. Dental care utilization statistics are traditionally based on an individual's reporting "at least one dental visit in the past year," although there are variations with shorter recall intervals and different forms of the question. Depending on the question and survey method, annual dental care use estimates vary. The 1996 Medical Expenditures Panel Survey (MEPS) estimates that 43 percent of the U.S. population 2 years and older had at least one dental visit that year (MEPS 2000). Responding to a variation of a question that had been asked in many previous surveys, some 65.1 percent of the U.S. population 2 years and older reported in 1997 that they had visited a dentist in the preceding year (NCHS 1997b), up from 55.0 percent in 1983 (Bloom et al. 1992). The average number of visits per person remains at about two per year. Further research is needed to understand reasons for variations in estimates from different survey approaches, but differences among persons with different characteristics are quite similar regardless of survey method.

Data from the 1997 National Health Interview Survey, reprinted in Healthy People 2010, indicate that the highest percentage reporting at least one

TABLE 4.3
Percentage of persons 25 years of age and older with a dental visit within the preceding year, by selected patient characteristics, selected years

	1983 ^a	1989 ^a	1990	1991	1993
Total ^{b,c}	53.9	58.9	62.3	58.2	60.8
Age					
25 to 34 years	59.0	60.9	65.1	59.1	60.3
35 to 44 years	60.3	65.9	69.1	64.8	66.9
45 to 64 years	54.1	59.9	62.8	59.2	62.0
65 years and older	39.3	45.8	49.6	47.2	51.7
65 to 74 years	43.8	50.0	53.5	51.1	56.3
75 years and older	31.8	39.0	43.4	41.3	44.9
Sex^c					
Male	51.7	56.2	58.8	55.5	58.2
Female	55.9	61.4	65.6	60.8	63.4
Poverty status^{c,d}					
Below poverty	30.4	33.3	38.2	33.0	35.9
At or above poverty	55.8	62.1	65.4	61.9	64.3
Race and Hispanic origin^c					
White, non-Hispanic	56.6	61.8	64.9	61.5	64.0
Black, non-Hispanic	39.1	43.3	49.1	44.3	47.3
Hispanic ^e	42.1	48.9	53.8	43.1	46.2
Education^c					
Less than 12 years	35.1	36.9	41.2	35.2	38.0
12 years	54.8	58.2	61.3	56.7	58.7
13 years or more	70.9	73.9	75.7	72.2	73.8
Education, race, and Hispanic origin^c					
Fewer than 12 years					
White, non-Hispanic	36.1	39.1	41.8	38.1	41.2
Black, non-Hispanic	31.7	32.0	37.9	33.0	33.1
Hispanic ^e	33.8	36.5	42.7	28.9	33.0
12 years					
White, non-Hispanic	56.6	59.8	62.8	58.8	60.4
Black, non-Hispanic	40.5	44.8	51.1	43.1	48.2
Hispanic ^e	48.7	56.5	59.9	49.5	54.6
13 years or more					
White, non-Hispanic	72.6	75.8	77.3	74.2	75.8
Black, non-Hispanic	54.4	57.2	64.4	61.7	61.3
Hispanic ^e	58.4	66.2	67.9	61.2	61.8

^a Data for 1983 and 1989 are not strictly comparable with data for later years. Data for 1983 and 1989 are based on responses to the question "About how long has it been since you last went to a dentist?" Starting in 1990, data are based on the question "During the past 12 months, how many visits did you make to a dentist?"

^b Includes all other races not shown separately and unknown poverty status and education level.

^c Age adjusted.

^d Poverty status is based on family income and family size using Bureau of the Census poverty thresholds.

^e Persons of Hispanic origin may be of any race.

Notes: Data are based on household interviews of a sample of the civilian noninstitutionalized population. Denominators exclude persons with unknown dental data. Estimates for 1983 and 1989 are based on data for all members of the sample household. Beginning in 1990, estimates are based on one adult member per sample household. Estimates for 1993 are based on responses during the last half of the year only.

Source: Data from NCHS 1989.

dental visit was third-grade children (82 percent). Those aged 25 years and older with less than a high school education had the lowest rates (41 percent) for annual dental visits as compared to those with at least some college education (74 percent) (USDHHS 2000).

Variation by Sex, Race/Ethnicity, Income, and Insurance

Dental care utilization varies with sex and race/ethnicity for individuals 25 and older (NCHS 1997a). Females had slightly higher rates of utilization (67

percent) than males (63 percent). Hispanic individuals had the lowest utilization (53 percent), and non-Hispanic whites had the highest rates (68 percent). Table 4.3 provides an overview of utilization from 1983 through 1993. A higher percentage of females reported a dental visit than males in each survey year. Fewer non-Hispanic blacks and Hispanics reported a dental visit than non-Hispanic whites in each survey year. Income and education are also key variables in utilization. In 1993, almost twice as many individuals 25 and older living at or above the poverty line had a dental visit than did those living below the poverty line in 1993 (64.3 versus 35.9 percent). Similarly, almost twice as many individuals with 13 years or more of education had a visit than did those with fewer than 12 years of education (73.8 versus 38.0 percent) in that same year.

Data from the 1989 National Health Interview Survey showed that the overall age-adjusted number of visits for blacks was 1.2 visits compared to 2.2 visits for whites (Bloom et al. 1992).

Table 4.4 shows the percentage distributions of the interval since their most recent dental visit for people aged 2 and older in selected demographic and socioeconomic categories. Individuals who have never visited a dentist ranged from a high of 13.1 percent of Mexican Americans to 5.8 percent of blacks and 4.4 percent of whites. Eleven percent of the population had not had a dental visit in 5 years or more. Individuals with fewer than 9 years of education represented the highest proportion, 30.6 percent, of those reporting no dental visit in 5 years

TABLE 4.4
Age-adjusted percentage distribution of persons 2 years and older by interval since last dental visit, by selected characteristics, 1989

	Interval Since Last Dental Visit					
	All Intervals	Less Than 1 Year	1 Year to Less Than 2 Years	2 Years to Less Than 5 Years	5 Years or More	Never
All ages	100.0	57.3	9.5	12.3	11.0	4.6
Sex						
Male	100.0	54.7	9.6	13.4	12.1	4.9
Female	100.0	59.9	9.4	11.2	10.1	4.4
Race						
White	100.0	59.5	9.1	11.6	10.5	4.4
Black	100.0	43.2	12.3	16.9	15.1	5.8
Other	100.0	51.6	9.7	14.0	10.8	6.7
Hispanic origin						
Non-Hispanic	100.0	58.5	9.4	12.0	10.8	4.1
Hispanic	100.0	46.0	10.5	14.6	13.0	9.7
Mexican American	100.0	40.5	8.9	15.3	15.8	13.1
Other Hispanic	100.0	53.2	12.3	13.7	9.9	5.1
Place of residence						
MSA ^a	100.0	58.4	9.4	11.9	10.1	4.5
Central city	100.0	54.9	10.1	12.9	10.9	5.1
Not central city	100.0	60.6	9.0	11.3	9.6	4.2
Not MSA ^a	100.0	53.6	9.7	13.6	14.1	5.1
Geographic region						
Northeast	100.0	60.7	10.4	10.7	9.0	3.5
Midwest	100.0	61.5	8.3	11.3	10.7	3.5
South	100.0	52.2	10.3	13.9	13.6	5.9
West	100.0	57.8	8.6	12.3	9.1	4.9
Education level						
Less than 9 years	100.0	30.6	9.9	18.4	30.6	5.9
9 to 11 years	100.0	39.0	10.7	20.3	23.5	1.3
12 years	100.0	54.6	10.6	15.0	14.4	0.5
13 years or more	100.0	70.2	8.5	10.3	6.9	0.2
Family income						
Less than \$10,000	100.0	42.2	10.9	16.3	20.1	7.0
\$10,000 to \$19,999	100.0	43.9	11.8	17.4	16.1	6.6
\$20,000 to \$34,999	100.0	58.2	10.5	3.2	10.4	4.6
\$35,000 or more	100.0	72.5	7.8	8.5	5.5	2.9
Dental insurance coverage						
With private dental insurance	100.0	70.4	8.7	9.2	6.6	3.3
Without private dental insurance	100.0	50.8	10.7	15.4	14.2	6.0

^aMSA = metropolitan statistical area.

Source: Bloom et al. 1992.

or more, compared with 6.9 percent of those with 13 years or more of education. A larger proportion of individuals without private dental insurance had not had a dental visit in 5 years or more compared with those with private dental insurance (14.2 versus 6.6 percent). Hispanic individuals have the lowest rate of dental insurance coverage—29.0 percent, compared with 32.4 percent for non-Hispanic blacks and 41.8 percent for non-Hispanic whites (U.S. Bureau of the Census 1997).

Professional care is necessary for several critical dental disease prevention measures, such as the application of dental sealants. Unfortunately, dental sealants are 3 times less likely to be found on the teeth of Mexican American and African American children than among white children aged 5 to 17 (Selwitz et al. 1996). Asian and Pacific Islander American children in California also demonstrated a low rate of sealant use (Pollick et al. 1997).

Variation by Oral Health Status

Utilization of dental care is associated with self-reported health status, as shown in Table 4.5. Of those who reported “excellent” or “very good” health, 61.4 percent had had a dental visit within the past year, compared with about 45.1 percent of those reporting “fair” or “poor” health. Functional limita-

tions are also related to dental service utilization. Of those who reported no physical limitations in activities, 58.5 percent reported a dental visit within the past year, compared to 46.6 percent of those who were unable to carry out their usual activities (Table 4.5) (Bloom et al. 1992).

Whether a person had natural teeth was strongly associated with dental care utilization (Table 4.5). Dentate persons were more than 4 times more likely to report a dental visit within the past year than edentulous people: 65.5 versus 14.3 percent. Over half (55.2 percent) of those who were edentulous reported that they had not had a dental visit in 5 years or more.

Recent analyses of data from NHANES III show that adults 18 and older who reported a dental visit in the past 12 months were nearly 9 times more likely to be dentate and 4.4 times more likely to have a complete dentition than adults who did not report visiting a dentist within the preceding 12 months. Dentate adults who reported a dental visit in the past 12 months were 3.1 times less likely to have untreated coronal decay and 1.5 times less likely to have gingivitis than dentate adults who did not report a recent dental visit (T. Drury, NIDCR, personal communication, 1999).

A study comparing individuals who had had a dental visit in the past 12 months with those who had not reported that dentate adults who had a recent visit were less likely to have untreated coronal and root caries, pulpal pathology, and retained tooth roots. They also were more likely to rate the general condition of their teeth and gums as excellent or very good (Drury and Redford 2000).

Examination of NHANES III data by low socioeconomic status (SES) provides an additional perspective. In a recent analysis, SES was measured by a composite index based on educational attainment and the ratio of annual family income to the poverty threshold. Among all adults, people with lower SES scores were nearly 9 times more likely to be edentulous than those with higher SES scores. Among the dentate, those with lower SES scores were 6 times more likely to have coronal decay and nearly 4 times less likely to have visited a dentist in the past 12 months (Drury et al. 1999).

TABLE 4.5
Age-adjusted percentage distribution of persons 2 years and older by interval since last dental visit, by selected health characteristics, 1989

	Interval Since Last Dental Visit					
	All Intervals	Less Than 1 Year	1 Year to 2 Years	2 Years to Less Than 5 Years	5 Years or More	Never
Assessed health status						
Excellent or very good	100.0	61.4	9.3	11.2	9.0	4.3
Good	100.0	51.9	10.1	13.9	12.6	5.8
Fair or poor	100.0	45.1	10.0	16.6	17.4	5.9
Limitation of activity						
Unable to carry on usual activity	100.0	46.6	9.8	15.6	16.6	5.1
Limited in amount or kind of major activity	100.0	52.3	9.8	14.0	14.4	4.7
Limited, but not in major activity	100.0	59.1	8.3	12.8	11.7	4.2
Not limited in activity	100.0	58.5	9.5	12.0	10.1	4.6
Dentition status						
Dentate	100.0	65.5	9.6	12.8	10.1	0.5
Edentulous	100.0	14.3	6.4	19.8	55.2	0.4

Source: Bloom et al. 1992.

Reasons for Nonutilization

Reasons for nonutilization of dental services are complex. Principal reasons cited by respondents of all ages (Bloom et al. 1992) are given in Table 4.6. Slightly less than half of those reporting no dental visit in the past year (46.8 percent) said that they perceived having no dental problem. This perception was the predominant response of individuals in all demographic categories, except for those 65 and older, who gave having no teeth as the predominant reason. Younger individuals were more likely than older to cite "no dental problem." Blacks were more likely to report "no problems" (58.5 percent) as a reason for no dental visit, compared to 44.3 percent of whites (Bloom et al. 1992).

Having no teeth (14.3 percent) was the next most frequently reported reason for no dental visit. About half of the people 65 and older in the 1989 survey gave this as their reason for no dental visit—39.2 percent of blacks compared to 51.2 percent of whites.

The third most frequently cited reason was the cost of care, mentioned by 13.7 percent of respondents. Whites (14.3 percent) were more likely than blacks (11.4 percent) to cite cost. Other surveys have reported substantially higher percentages of individuals indicating cost as a barrier, particularly those in underserved or low-income areas (Bloom et al. 1992). The age group most sensitive to the cost of care was 18- to 34-year-olds, 19.1 percent of whom gave cost as the reason for no dental visit. Finally, a small proportion of respondents (4.3 percent) reported fear as a personal barrier to receipt of care.

Unmet Needs

Unmet health needs can be assessed in many ways. Because oral diseases are common and do not resolve over time in the absence of intervention, the lack of dental visits is used as an indicator of unmet health needs. In addition, the National Access to Care Survey documented the extent of dental care that individuals wanted but could not obtain ("wants") in the total population and among various population subgroups (Mueller et al. 1998). About 8.5 percent of the U.S. population wanted, but did not obtain, dental care in 1994 (Table 4.7). In contrast, only 5.6 percent reported unmet medical or surgical care wants. Adult women aged 19 to 64 reported the greatest level of dental care wants; elderly people 65 and older had the lowest level. Blacks, people in fair or poor health or with one or more chronic conditions, and people living in the South reported higher levels of dental care wants than comparable groups. About

16.4 percent of those in households whose family income was less than 150 percent of the poverty level reported dental care wants. More than 22 percent of the uninsured reported dental care wants. Insured children with special health care needs were 4 times more likely to report unmet need for dental care (23.9 percent versus 6.1 percent) if they were uninsured than if they were insured, according to a recent analysis of data from the National Health Interview Survey (Newacheck et al. 2000).

Outcomes of Appropriate Levels of Access and Utilization: An Example

The effects on health of a system of care with assured access and positive expectations of care-seeking and utilization behavior have been demonstrated by the U.S. Department of Defense. There are currently over 1.4 million men and women on active duty in the U.S. military. The population is predominantly male (86 percent). The racial distribution is 68 percent white, 20 percent black, 7 percent Hispanic, 3 percent Asian, and 2 percent other groups. Slightly over 30 percent of active duty personnel are between the ages of 20 and 24, and 91 percent are younger than 40. In 1997, 59 percent were married. Seventy-six percent had a high school degree, and 19 percent were college graduates.

Free dental care, one of the benefits provided to active duty military personnel, eliminates one of the significant barriers that has been identified as limiting access to care for many in the civilian population. In addition, military personnel are required to receive a dental examination annually, even if the individual perceives that he or she has "no problem." Dental care is available to most military personnel at their duty station, eliminating the need to travel long distances. A comparison of the oral health and utilization of dental care of the military and civilian populations illustrates the impact of elimination of these barriers to care on oral health, even for persons from demographic groups that are traditionally underserved.

In 1994 the Tri-Service Comprehensive Oral Health Survey examined and administered questionnaires to 13,050 active duty military personnel using a complex, weighted survey design to examine the oral health status, dental treatment needs, dental utilization, and perceived need for care in this population (York et al. 1995). The study found that nearly all (99.2 percent) active duty military personnel had seen a dentist within the past 2 years. Eighty percent of active duty personnel received a dental examination within the past year, 60 percent had a dental

TABLE 4.6
Percentage of persons with no dental visit in past year by reason reported, by selected characteristics, 1989

	All with No Visits in Past Year	Fear	Cost	Access Problem	No Dental Problem	No Teeth	Not Important	Other Reason
Age								
All ages	100.0	4.3	13.7	1.7	46.8	14.3	2.3	8.7
2 to 17 years	100.0	1.3	15.0	1.5	56.8	0.2	1.9	11.9
18 to 34 years	100.0	5.9	19.1	2.4	52.4	0.7	3.2	9.5
35 to 64 years	100.0	5.8	12.8	1.5	43.3	17.8	2.2	8.4
65 years and older	100.0	2.2	4.1	1.1	31.2	49.7	1.1	3.9
Sex								
Male								
All ages	100.0	4.0	13.0	1.5	49.1	12.1	2.6	9.3
2 to 17 years	100.0	1.2	14.9	1.3	56.2	*0.2	2.0	12.1
18 to 34 years	100.0	5.5	17.5	2.0	54.8	0.6	3.4	9.7
35 to 64 years	100.0	5.4	11.2	1.5	45.4	16.1	2.8	9.5
65 years and older	100.0	1.7	4.0	1.0	33.6	48.6	1.3	3.5
Female								
All ages	100.0	4.6	14.3	1.8	44.4	16.6	1.9	8.1
2 to 17 years	100.0	1.5	15.2	1.6	57.4	*0.2	1.8	11.6
18 to 34 years	100.0	6.5	21.0	2.9	49.5	0.8	3.0	9.3
35 to 64 years	100.0	6.2	14.4	1.5	41.1	19.5	1.6	7.3
65 years and older	100.0	2.5	4.2	1.2	22.5	50.6	0.9	4.1
Race								
White								
All ages	100.0	4.4	14.3	1.8	44.3	15.7	2.4	9.4
2 to 17 years	100.0	1.3	16.4	1.7	54.0	0.2	2.0	13.3
18 to 34 years	100.0	6.2	20.7	2.6	49.6	0.7	3.4	10.6
35 to 64 years	100.0	5.8	13.0	1.6	41.3	19.0	2.4	9.1
65 years and older	100.0	2.1	3.7	1.1	30.5	51.2	1.1	3.9
Black								
All ages	100.0	4.0	11.4	1.0	58.5	8.8	1.5	5.1
2 to 17 years	100.0	1.3	10.7	*0.7	68.3	*0.2	1.2	6.6
18 to 34 years	100.0	4.9	13.3	1.5	63.8	*0.7	2.5	4.6
35 to 64 years	100.0	6.0	11.7	0.9	52.8	13.0	1.1	4.9
65 years and older	100.0	3.0	7.0	*1.0	36.6	39.2	*0.9	3.4
Other								
All ages	100.0	3.7	10.8	1.6	52.1	6.1	2.2	9.2
2 to 17 years	100.0	*1.7	11.4	*0.3	49.8	*0.0	*2.4	12.6
18 to 34 years	100.0	4.6	11.7	*2.5	59.4	*0.3	*2.2	8.8
35 to 64 years	100.0	4.6	10.8	*1.9	51.0	8.2	*2.6	7.7
65 years and older	100.0	*2.8	*4.2	*0.7	31.4	44.9	*0.7	*5.9
Hispanic origin								
Non-Hispanic								
All ages	100.0	4.3	13.0	1.7	45.7	15.6	2.2	9.1
2 to 17 years	100.0	1.3	14.4	1.3	56.2	0.2	1.9	12.8
18 to 34 years	100.0	6.0	18.9	2.5	51.6	0.7	3.2	10.1
35 to 64 years	100.0	5.8	12.0	1.5	42.5	18.8	2.2	8.7
65 years and older	100.0	2.1	4.0	1.1	30.9	50.4	1.1	3.9
Hispanic, total								
All ages	100.0	4.0	19.1	1.8	56.1	3.5	2.6	5.9
2 to 17 years	100.0	1.6	18.4	2.4	59.5	*0.1	2.2	7.3
18 to 34 years	100.0	5.2	20.1	1.5	57.9	*0.2	3.3	5.6
35 to 64 years	100.0	5.3	20.7	1.6	52.2	6.5	2.3	5.2
65 years and older	100.0	*4.6	8.2	*1.4	40.7	31.9	*1.6	*3.4

	All with No Visits in Past Year	Fear	Cost	Access Problem	No Dental Problem	No Teeth	Not Important	Other Reason
Hispanic, Mexican American								
All ages	100.0	3.6	20.7	1.7	56.2	2.4	2.3	5.3
2 to 17 years	100.0	*1.4	19.4	2.6	60.7	*0.1	2.2	5.5
18 to 34 years	100.0	4.7	21.0	*0.9	57.5	*0.1	2.8	4.8
35 to 64 years	100.0	5.2	24.3	*1.4	50.1	4.5	*1.7	6.4
65 years and older	100.0	*4.3	*11.1	*1.4	38.7	30.5	*1.8	*1.8
Hispanic, other								
All ages	100.0	4.7	16.3	2.0	55.9	5.3	3.1	7.0
2 to 17 years	100.0	*1.8	16.3	*1.9	56.9	*0.2	*2.0	11.3
18 to 34 years	100.0	6.2	18.5	*2.4	58.7	*0.4	4.4	7.0
35 to 64 years	100.0	5.5	16.3	*1.8	54.9	8.9	*3.1	3.8
65 years and older	100.0	*4.9	*5.7	*1.4	42.8	33.2	*1.4	*4.9
Place of residence								
MSA, total^a								
All ages	100.0	4.4	13.4	1.8	46.6	12.8	2.4	9.0
2 to 17 years	100.0	1.3	14.1	1.4	55.7	0.3	2.0	12.1
18 to 34 years	100.0	5.7	18.5	2.6	51.6	0.5	3.3	9.6
35 to 64 years	100.0	6.1	12.6	1.6	43.1	16.0	2.4	8.7
65 years and older	100.0	2.3	4.2	1.1	31.4	47.6	1.2	4.0
MSA, central city^a								
All ages	100.0	4.4	14.0	1.7	48.0	11.9	2.6	7.8
2 to 17 years	100.0	1.6	14.6	1.4	56.5	*0.2	2.0	10.0
18 to 34 years	100.0	5.3	17.9	2.5	54.2	0.4	3.6	8.4
35 to 64 years	100.0	6.4	14.1	1.3	43.9	14.8	2.5	7.3
65 years and older	100.0	2.5	4.7	1.3	31.5	46.3	1.7	4.2
MSA, not central city^a								
All ages	100.0	4.3	13.0	1.8	45.5	13.5	2.3	9.9
2 to 17 years	100.0	1.2	13.7	1.4	55.1	*0.4	2.0	13.7
18 to 34 years	100.0	6.0	19.0	2.6	49.5	0.6	3.1	10.6
35 to 64 years	100.0	5.8	11.6	1.7	42.6	16.8	2.4	9.6
65 years and older	100.0	2.1	3.9	1.0	31.3	48.5	0.8	4.0
Not MSA^a								
All ages	100.0	4.1	14.4	1.4	47.7	18.8	1.7	8.0
2 to 17 years	100.0	1.3	18.0	1.6	60.0	*0.1	1.5	11.2
18 to 34 years	100.0	6.9	21.1	1.7	55.4	1.2	2.7	9.1
35 to 64 years	100.0	4.9	13.1	1.2	43.8	23.1	1.6	7.7
65 years and older	100.0	2.0	3.9	1.0	30.7	55.2	0.9	3.5
Family income^b								
Less than \$10,000								
All ages	100.0	3.8	19.7	1.7	42.8	22.5	1.4	6.4
2 to 17 years	100.0	*1.1	19.4	2.6	60.0	*0.3	1.9	9.6
18 to 34 years	100.0	5.7	28.8	2.4	51.7	*0.9	1.9	7.2
35 to 64 years	100.0	6.3	25.2	*1.1	35.5	25.1	1.4	5.4
65 years and older	100.0	2.0	6.6	*0.9	27.4	57.4	*0.7	3.9
\$10,000 to \$19,999								
All ages	100.0	4.0	18.8	1.5	47.0	17.4	1.7	6.5
2 to 17 years	100.0	1.4	21.9	1.4	58.8	*0.1	1.3	7.9
18 to 34 years	100.0	6.1	27.8	1.8	53.2	*0.5	2.4	7.9
35 to 64 years	100.0	4.7	19.2	1.5	43.6	21.7	1.6	5.7
65 years and older	100.0	3.0	3.4	1.2	31.4	51.9	1.3	4.3
\$20,000 to \$34,999								
All ages	100.0	4.8	13.7	1.7	51.3	11.5	2.3	11.1
2 to 17 years	100.0	1.6	14.4	0.9	59.7	*0.1	1.7	13.8
18 to 34 years	100.0	6.4	18.1	2.8	54.7	0.8	3.3	12.3
35 to 64 years	100.0	6.4	12.5	1.4	46.2	18.5	2.0	10.2
65 years and older	100.0	1.8	2.4	*1.3	38.5	47.7	*1.5	4.7

(continues)

The Magnitude of the Problem

TABLE 4.6 continued

	All with No Visits in Past Year	Fear	Cost	Access Problem	No Dental Problem	No Teeth	Not Important	Other Reason
\$35,000 or more								
All ages	100.0	5.9	6.8	2.6	52.3	8.1	4.1	14.1
2 to 17 years	100.0	1.1	5.8	2.0	56.8	*0.6	3.1	20.2
18 to 34 years	100.0	7.0	9.2	3.6	55.4	*0.6	5.4	13.4
35 to 64 years	100.0	7.8	6.0	2.3	49.7	13.2	4.0	12.9
65 years and older	100.0	*2.8	3.8	*1.6	37.9	41.6	*2.0	4.4
Dental insurance coverage								
With dental insurance								
All ages	100.0	6.2	7.2	2.5	53.2	10.1	3.4	15.2
2 to 17 years	100.0	1.2	7.8	1.2	61.4	*0.3	2.4	18.8
18 to 34 years	100.0	8.5	9.5	4.1	55.5	0.8	5.1	16.3
35 to 64 years	100.0	8.0	6.0	2.2	48.8	17.4	3.1	13.7
65 years and older	100.0	2.5	*1.6	*1.1	39.3	44.7	*1.0	5.7
Without dental insurance								
All ages	100.0	4.0	18.5	1.5	48.7	17.2	2.0	7.0
2 to 17 years	100.0	1.6	20.5	1.7	60.1	*0.2	1.9	9.9
18 to 34 years	100.0	5.5	26.7	1.9	56.3	0.7	2.7	7.6
35 to 64 years	100.0	5.4	18.9	1.3	45.1	19.9	2.1	6.8
65 years and older	100.0	2.3	4.9	1.2	31.9	52.5	1.2	3.9
Insurance status unknown								
All ages	100.0	1.6	3.9	0.7	23.8	9.3	0.8	2.9
2 to 17 years	100.0	*0.6	4.7	*1.0	25.7	*0.1	*0.6	3.6
18 to 34 years	100.0	2.3	5.1	*0.9	28.2	*0.3	1.1	3.5
35 to 64 years	100.0	1.5	3.4	*0.3	20.8	9.2	*0.7	2.0
65 years and older	100.0	*1.4	*1.6	*0.6	19.8	36.8	*0.6	2.6
Limitation of activity								
Unable to carry on usual activity								
All ages	100.0	4.4	15.4	1.7	33.2	31.9	1.1	6.2
2 to 17 years	100.0	*6.0	*27.6	*0.0	36.6	*0.0	*1.5	*20.1
18 to 34 years	100.0	7.4	25.3	*1.9	47.1	*1.9	*1.9	6.1
35 to 64 years	100.0	5.4	18.3	*1.2	34.5	28.9	*1.1	6.6
65 years and older	100.0	*1.7	6.6	2.4	26.0	49.9	*0.7	4.6
Limited in amount or kind of major activity								
All ages	100.0	4.4	15.2	1.9	34.5	29.7	1.7	7.5
2 to 17 years	100.0	*1.7	18.9	*2.5	58.0	*0.3	*2.0	9.1
18 to 34 years	100.0	6.9	30.2	*3.9	44.3	*0.8	*1.8	10.4
35 to 64 years	100.0	6.1	17.6	1.7	31.2	27.1	2.2	9.3
65 years and older	100.0	2.4	5.1	*1.2	26.3	54.3	*1.1	3.9
Limited, but not in major activity								
All ages	100.0	4.4	12.4	0.9	34.3	35.8	1.5	6.3
2 to 17 years	100.0	*1.7	28.0	*2.2	53.0	*0.0	*1.7	*10.8
18 to 34 years	100.0	8.0	29.2	*1.9	50.5	*1.4	*1.7	10.3
35 to 64 years	100.0	6.3	17.5	*0.8	35.3	24.0	*2.0	8.0
65 years and older	100.0	2.6	3.9	*0.6	28.2	54.4	*1.2	3.9
Not limited in activity								
All ages	100.0	4.3	13.5	1.7	49.6	10.4	2.4	9.2
2 to 17 years	100.0	1.3	14.7	1.4	56.9	0.2	1.9	11.9
18 to 34 years	100.0	5.8	18.2	2.4	52.9	0.6	3.3	9.6
35 to 64 years	100.0	5.8	11.3	1.5	46.1	15.1	2.4	8.6
65 years and older	100.0	2.1	3.4	0.9	34.3	47.3	1.2	3.7

^aMSA = metropolitan statistical area.

^bPersons with unknown income not shown separately.

Note: Data are based on household interviews of the civilian noninstitutionalized population.

* = Figure does not meet the standard of reliability or precision (more than 30 percent relative standard error and numerator of percent or rate).

Source: Bloom et al. 1992.

TABLE 4.7
Estimated number and percentage of people with unmet health care wants,
by selected characteristics, 1994

	Number of People (in millions)	Dental Care (percentage)	Medical or Surgical Care (percentage)
All people	259.2	8.5	5.6
Age and sex			
Children, 1 to 18 years	73.5	5.9 ^a	2.9 ^a
Adult men, 19 to 64 years	75.3	9.5	5.8
Adult women, 19 to 64 years	79.3	12.1 ^a	9.3 ^a
Elderly people, 65 years and older	31.1	3.6 ^a	2.4 ^a
Race/ethnicity			
White	191.4	7.4	4.6
Black	32.2	15.0 ^a	10.2 ^a
Hispanic	23.9	8.2	6.2
Other	11.7	9.9	8.6
Health status			
Fair or poor	24.6	16.1 ^a	11.2 ^a
Good or excellent	233.5	7.7	5.0
Number of chronic conditions			
None	158.6	7.3	4.6
One or more	100.6	10.4	7.1
Geographical region			
Northeast	47.8	6.9	5.8
Midwest	65.8	6.9	4.5
South	92.6	11.2 ^a	6.1
West	53.1	7.4	5.9
Rural/urban status			
Metropolitan statistical areas	208.2	8.6	5.6
Nonmetropolitan statistical areas	50.5	8.1	5.9
Education level of head of household			
High school or less	117.5	9.4	6.8
Some post-high school	141.2	7.9	4.7
Family income status			
Less than 150 percent of the poverty level	55.7	16.4 ^a	9.1 ^a
150 percent of the poverty level or more	174.3	6.3 ^a	4.5
Health insurance status			
Private	166.6	5.9 ^a	4.1
Medicare	36.0	5.6 ^a	3.1 ^a
Medicaid	22.2	12.2	8.0
Uninsured	32.5	22.6 ^a	14.9 ^a
Type of private health insurance			
Health maintenance organization/ independent practice association	45.1	5.5 ^a	5.0
Preferred provider organization	30.7	4.6 ^a	4.1
Fee-for-service	73.5	5.3 ^a	3.1 ^a

^a The estimate differs from the percentage for the "all people" demographic at the 1 percent confidence level based on a two-tailed t-test of the difference in weighted estimates.

Note: The standard error of each percentage is less than 30 percent of the percentage estimate.

Source: Mueller et al. 1998. Access to dental care in the United States. JADA 1998 April; 129(4):429-37. Copyright 1998 by Journal of the American Dental Association. Reprinted by permission of ADA Publishing Co. Inc. (2000).

prophylaxis, and 29 percent had at least one tooth filled.

Edentulism is virtually nonexistent in the active duty military population. Also, active duty military personnel have a significantly lower proportion of their decayed, missing, and filled surfaces that are untreated; this is primarily due to dramatic improvements in the oral health of active duty blacks when compared to their civilian counterparts. Active duty whites also have somewhat better oral health than white civilians of a similar age.

The relative proportion of unfilled surfaces as a component of decayed and filled tooth surfaces in the military and civilian populations is illustrated in Figure 4.25.

ORAL HEALTH STATUS IN CHANGING TIMES

The burden of oral diseases and conditions in the United States is extensive and affects persons throughout their life span. Birth defects such as cleft lip/palate, dental caries, and facial trauma are common in the young. Periodontal diseases, autoimmune disorders, and other chronic disabling conditions are seen in adults, while complete tooth loss and oral cavity and pharyngeal cancers are seen more often in older Americans. Because the most common oral disease, dental caries, is so widespread in the population, nearly every American has experienced oral disease.

The effects of oral diseases and conditions on quality of life and well-being are discussed in Chapter 6. In sum, conditions such as cleft lip and palate and oral cancer not only involve costly and difficult surgeries and treatments, they also alter facial appearance and impair oral functioning. Pain disorders and pain as a consequence of dental disease are prevalent in certain groups and can affect daily living.

The available trend data reveal improvements in dental health for most Americans; however, despite improvements in dental status, disparities remain. Diseases disproportionately affect some sex, income, and racial/ethnic groups, and the magnitude of the differences is striking. All the reasons for these disparities are not clear. Some of the most common dental diseases, such as dental caries, are preventable (prevention of oral diseases and conditions is presented in Chapters 8 and 9). It appears, however, that not all individuals are benefiting from interventions that involve professional care, as represented by the data on dental visits. At the same time, as presented in Chapter 7, about 40 percent of the public does not receive the benefits of community water fluoridation. The emerging data on the effects of socioeconomic status on oral health are beginning to explain some, but not all, racial/ethnic differences. For other diseases, health disparities appear not to be related to professional services; a better understanding of the reasons for these differences is needed.

This review of available data on oral diseases and conditions also reveals the lack or limitation of national or state data on oral diseases for many population subgroups and for many conditions that affect the craniofacial structures. Information on the variables needed to explain health status differences, such as detailed utilization and expenditure data and data on services rendered, is limited as well. Data on specific services—self-care, services provided by professionals, and services that are community-based—are needed to understand the dimensions of oral health. (Some of these services are described in Chapters 7 and 8.) Although some data on expenditures for care and health care personnel are available

to (Chapter 9) complement the statistics needed to assess oral health in the United States, almost all these data come from cross-sectional surveys that do not allow for analysis of the outcomes of disease and related care.

Available state data reveal variations within and among states in patterns of oral health and disease among population groups. Having state-specific and local data that augment national data is critical in identifying high-risk populations and areas and in addressing health disparities. These data also are vital in program evaluation, planning, and policy decisions. Yet state and local data are almost nonexistent. In recent years, the need for state and local data has intensified as more programs are funded by local authorities and responsibilities are shifted from national to state-based levels.

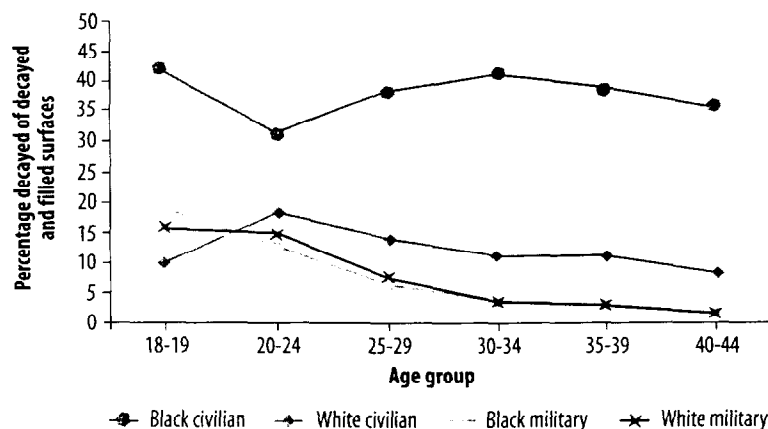
The nation's health information system is undergoing constant change to meet the current and future needs for health information. Consequently, many factors influence how and what data are collected and analyzed. These factors include emerging technologies, legislation about how data are to be collected, and confidentiality and privacy concerns.

The need for epidemiologic and surveillance data change as the understanding of specific diseases and conditions evolves and as society's goals and priorities change. The increasing focus on the long-term benefits of disease prevention and health promotion and the need to close the gap on disparities also affects how and what data are collected. For example, major initiatives such as the Department of Health and Human Services's Healthy People 2010 have provided a framework for data collection and analysis tied to specific objectives and have helped identify needs for new health data systems.

The Healthy People initiative now includes objectives for the nation's health status as well as for preventive interventions and objectives that would improve infrastructure and capacity building to provide the necessary services and monitoring.

This overview of the magnitude of oral diseases and conditions in America raises many questions still to be researched. If certain oral diseases are preventable, why do we have populations with extensive and untreated disease? Once socioeconomic factors are controlled, why do we see differences in services received? Why

FIGURE 4.25
The percentage of unfilled decayed surfaces is higher for civilian males than for males in the U.S. military



Source: York et al. 1995.

are some conditions more prevalent in certain populations than in others? How will the rapidly changing and projected demographics of America contribute to future trends in oral and craniofacial health and disease? These and many other questions require more research, new databases, and an active and trained group of researchers.

FINDINGS

- Over the past five decades, major improvements in oral health have been seen nationally for most Americans.

- Despite improvements in oral health status, profound disparities remain in some population groups as classified by sex, income, age, and race/ethnicity. For some diseases and conditions, the magnitude of the differences in oral health status among population groups is striking.

- Oral diseases and conditions affect people throughout their life span. Nearly every American has experienced the most common oral disease, dental caries.

- Conditions that severely affect the face and facial expression, such as birth defects, craniofacial injuries, and neoplastic diseases, are more common in the very young and in the elderly.

- Oral-facial pain can greatly reduce quality of life and restrict major functions. Pain is a common symptom for many of the conditions affecting oral-facial structures.

- National and state data for many oral and craniofacial diseases and conditions and for population groups are limited or nonexistent. Available state data reveal variations within and among states in patterns of health and disease among population groups.

- Research is needed to develop better measures of disease and health, to explain the differences among population groups, and to develop interventions targeted at eliminating disparities.

REFERENCES

- Albandar JM, Brown LJ, Loe H. Clinical features of early-onset periodontitis. *J Am Dent Assoc* 1997 Oct;128(10):1393-9.
- Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999 Jan;70(1):13-29.
- American Cancer Society (ACS). *Cancer facts and figures*. Atlanta: American Cancer Society; 1999.
- Arnett H. First round results of the access to health survey for selected disabilities and secondary conditions. Boston: Independent Living Centers; 1994.
- Axell T, Mornstad H, Sundstrom B. The relation of the clinical picture to the histopathology of snuff dipper's lesions in a Swedish population. *J Oral Pathol* 1976 Jul;5(4):229-36.
- Bartoshuk LM, Duffy VB, Miller IJ. PTC/PROP tasting: anatomy, psychophysics, and sex effects. *Physiol Behav* 1994 Dec;56(6):1165-71.
- Bartoshuk LM, Duffy VB, Reed D, Williams A. Supertasting earaches and head injury: genetics and pathology alter our taste worlds. *Neurosci Biobehav Rev* 1996;20(1):79-87.
- Beck JD, Hunter RJ. Oral health status in the United States: problems of special patients. *J Dent Educ* 1985;149:407-25.
- Bloom B, Gift HC, Jack SS. Dental services and oral health: United States, 1989. *Vital Health Stat* 10 1992 Dec;(183):1-95.
- Brunelle JA, Bhat M, Lipton JA. Prevalence and distributions of selected occlusal characteristics in the US population, 1988-1991. *J Dent Res* 1996 Feb;75(Spec No):706-13.
- Burman NT. A case: control study of oro-facial clefts in Western Australia. *Aust Dent J* 1985 Dec;30(6):423-9.
- Burt BA, Eklund SA. *Dentistry, dental practice, and the community*. Philadelphia: W.B. Saunders Co.; 1999.
- Carlsson GE, LeResche L. Epidemiology of temporomandibular disorders. In: Sessle BJ, Bryant PS, Dionne RA, editors. *Temporomandibular disorders and related pain conditions*. Seattle: IASP Press; 1995. p. 211-26.
- Centers for Disease Control (CDC). Prevalence of oral lesions and smokeless tobacco use in Northern Plains Indians. *MMWR Morb Mortal Wkly Rep* 1988 Oct 7;37(39):608-11.
- Chen MS, Andersen RM, Barmes DE, Leclercq MH, Lyttle CS. *Comparing oral health care systems: A second international collaborative study*. Geneva: World Health Organization; 1997.
- Chesney MA, Ozer EM. *Women and health: in search of a paradigm. Women's health: research on gender, behavior, and policy*. Hillsdale (NJ): Lawrence Erlbaum Associates; 1995. p. 3-26.
- Council of Economic Advisers. *Changing America: indicators of social and economic well-being by race and Hispanic origin*. Washington: Council of Economic Advisers, Executive Office of the President; 1998 1 Sep. Available from: US GPO, Superintendent of Documents, Washington, DC.
- Day GL, Blot WJ, Shore RE, Schoenberg JB, Kohler BA, Greenberg RS, Liff JM, Preston-Martin S, Austin DF, McLaughlin JK, et al. Second cancers following oral and pharyngeal cancer: patients' characteristics and survival patterns. *Eur J Cancer B Oral Oncol* 1994 Nov;30B(6):381-6.
- De Wet FA. The prevention of orofacial sports injuries in the adolescent. *Int Dent J* 1981 Dec;31(4):313-9.
- Delgado JL, Estrada L. Improving data collection strategies. *Public Health Rep* 1993;108:540-5.

- Drury TF, Redford M. Completing the clinical picture of selected aspects of America's adult oral health: a first description. *J Dent Res* 2000;79s:503.
- Drury TF, Garcia I, Adesanya M. Socioeconomic disparities in adult oral health in the United States. In: Adler NE, Marmot M, McEwen BS, Stewart J, editors. *Socioeconomic status and health in industrial nations. Social, psychological, and biological pathways.* Ann NY Acad of Sci 1999;896:322-4.
- Embil JA, Stephens RG, Manuel FR. Prevalence of recurrent herpes labialis and aphthous ulcers among young adults on six continents. *Can Med Assoc J* 1975 Oct 4;113(7):627-30.
- Federal Interagency Forum on Children and Family Status. *America's children: key national indicators of well being.* Washington: Federal Interagency Forum on Children and Family Status; 1997.
- Ferguson MM, Carter J, Boyle P. An epidemiological study of factors associated with recurrent aphthae in women. *J Oral Med* 1984 Oct-Dec;39(4):212-7.
- Fox RI. Clinical features, pathogenesis, and treatment of Sjögren's syndrome. *Curr Opin Rheumatol* 1996 Sep;8(5):438-45.
- Fraser GR, Calnan JS. Cleft lip and palate: seasonal incidence, birth weight, birth rank, sex, site, associated malformations and parental age. A statistical survey. *Arch Dis Childhood* 1961 Aug;36:420-3.
- Garcia I, Drury TF. Mexican-American/White non-hispanic disparities in adult oral health. *J Dent Res* 1999;78:Abstract no. 2079.
- Garcia-Godoy F, Mobley CC, Jones DL. Prevalence of dental caries in San Antonio pre-school children. *J Dent Res* 1994;73:144. Abstract no. 342.
- Gear RW, Miaskowski C, Gordon NC, Pawl SM, Heller PH, Levine JD. Kappa-opioids produce significantly greater analgesia in women than in men. *Nat Med* 1996 Nov;2(11):1184-5.
- Gift HC, Bhat M. Dental visits for orofacial injury: defining the dentist's role. *J Am Dent Assoc* 1993 Nov;124(11):92-6,98.
- Greer MH. Statewide oral health assessment of public school children in Hawaii: 1993-94 [unpublished data]. Honolulu: Hawaii Department of Health, Division of Dental Health.
- Greer MH. Hawaii public school oral health assessment. Honolulu: Hawaii Department of Health, Division of Dental Health; 1999.
- Habib Z. Factors determining occurrence of cleft lip and cleft palate. *Surg Gynecol Obstet* 1978 Jan;146(1):105-10.
- Hahn RA. The state of federal health statistics on racial and ethnic groups. *JAMA* 1992;267:268-71.
- Haveman M, Van Berkum G, Reijnders R, et al. Differences in services needs, time demands, and care-giving burden among parents of persons with mental retardation across the life cycle. *Fam Relations* 1997;46:417-25.
- Indian Health Service (IHS). *The oral health of Native Americans: a chart book of recent findings, trends and regional differences.* Rockville (MD): Indian Health Service, U.S. Department of Health and Human Services; 1994.
- Indian Health Service (IHS). *Oral health status survey, 1999.* Rockville (MD): Unpublished analysis by the Office of Health Programs, Indian Health Service, U.S. Department of Health and Human Services; 2000.
- Ismail AI, Szpunar SM. The prevalence of total tooth loss, dental caries, and periodontal disease among Mexican Americans, Cuban Americans, and Puerto Ricans: findings from HHANES 1982-1984. *Am J Public Health* 1990;80:66-70.
- Kaste LM, Gift HC, Bhat M, Swango PA. Prevalence of incisor trauma in persons 6-50 years of age: United States, 1988-1991. *J Dent Res* 1996a Feb;75(Spec No):696-705.
- Kaste LM, Selwitz RH, Oldakowski RJ, Brunelle JA, Winn DM, Brown LJ. Coronal caries in the primary and permanent dentition of children and adolescents 1-17 years of age: United States, 1988-1991. *J Dent Res* 1996b Feb;75(Spec No):631-41.
- King GN, Healy CM, Glover MT, Kwan JT, Williams DM, Leigh IM, Thornhill MH. Prevalence and risk factors associated with leukoplakia, hairy leukoplakia, erythematous candidiasis, and gingival hyperplasia in renal transplant recipients. *Oral Surg Oral Med Oral Pathol* 1994 Dec;78(6):718-26.
- Kosary CL, Ries LA, Miller BA, Hankey BF, HARRAS A, Edwards BK, editors. *SEER cancer statistics review, 1973-1992. Tables and graphs.* Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute; 1995 Dec. p. 17, 34, 52, 355, 361. NIH Pub. no. 96-2789.
- Kovar MG. Data systems of the National Center for Health Statistics. *Vital Health Stat* 1 1989 Mar;(23):1-21.
- Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. *J Am Dent Assoc* 1993 Oct;124(10):115-21.
- Löe H, Brown LJ. Early onset periodontitis in the United States of America. *J Periodontol* 1991 Oct;62(10):608-16.
- Louie R, Brunelle JA, Maggiore ED, Beck R. Caries prevalence in Head Start children, 1986-87. *J Public Health Dent* 1990;50(2):299-305.
- Lowry RB, Thunem NY, Uh SH. Birth prevalence of cleft lip and palate in British Columbia between 1952 and 1986; stability of rates. *Can Med Assoc J* 1989 May 15;140(10):1167-70.
- McDonald AK. *The National Electronic Injury Surveillance System: a tool for researchers.* Washington: U.S. Consumer Product Safety Commission; 1994 Oct.

- Medical Expenditure Panel Survey 1996. Analysis by Center for Cost and Financing Studies. Rockville (MD): Agency for Healthcare Research and Quality; 2000.
- Miller BA, Kolonel LN, Bernstein L, Young JL Jr, West D, Key CR, Liff JM, Glover CS, Alexander GA, et al., editors. Racial/ethnic patterns of cancer in the United States 1988-92. Bethesda (MD): National Cancer Institute; 1996. NIH Pub. no. 96-4104.
- Miller MF, Ship II. A retrospective study of the prevalence and incidence of recurrent aphthous ulcers in a professional population, 1958-1971. *Oral Surg Oral Med Oral Pathol* 1977 Apr;43(4):532-7.
- Mogil JS, Sternberg WF, Marek P, Sadowski B, Belknap JK, Liebeskind JC. The genetics of pain and pain inhibition. *Proc Natl Acad Sci USA* 1996 Apr;93(7):3048-55.
- Mogil JS, Richards SP, O'Toole LA, Helms ML, Mitchell SR, Kest B, Belknap JK. Identification of a sex-specific quantitative trait locus mediating nonopioid stress-induced analgesia in female mice. *J Neurosci* 1997 Oct 15;17(20):7995-8002.
- Mueller CD, Schur CL, Paramore LC. Access to dental care in the United States. *J Am Dent Assoc* 1998 Apr;129(4):429-37.
- National Center for Health Statistics (NCHS). Design and estimation for the National Health Interview Survey, 1985-94. *Vital Health Stat* 1989 Aug;2(110).
- National Center for Health Statistics (NCHS). First National Health and Nutrition Examination Survey (NHANES I). Hyattsville (MD): NCHS, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control; 1975.
- National Center for Health Statistics (NCHS). Third National Health and Nutrition Examination Survey (NHANES III) reference manuals and reports [CD-ROM]. Hyattsville (MD): NCHS, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention; 1996.
- National Center for Health Statistics (NCHS). Preliminary data from the Centers for Disease Control and Prevention. *Mon Vital Stat Rep* 1997a;46(1 Suppl 2).
- National Center for Health Statistics (NCHS). Prevalence of selected chronic conditions: United States, 1990-92. Series 10: data from the National Health Survey no. 194. Hyattsville (MD): U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 1997b Jan. DHHS Pub. no. PH-S97-1522.
- National Dental Association (NDA). "Charet" status of dental health in the black community. In: *Proceedings of the National Dental Association recommendations for program planning*, 1972 Jul; New Orleans, Louisiana.
- Neissen LC, Weyant RJ. Causes of tooth loss in a veteran population. *J Public Health Dent* 1989;49:19-23.
- Newacheck PW, Strickland B, Shonkoff JP, Perrin JM, McPherson M, McManus M, Lauver C, Fox H, Arango P. An epidemiologic profile of children with special health care needs. *Pediatrics* 1998;102:117-23.
- Newacheck PW et al. Access to health care for children with special needs. *Pediatrics* 2000;105(4):760-6.
- Niendorfs W. The oral health of Native Americans: a chart book of recent findings, trends and regional differences. U.S. Department of Health and Human Services, Indian Services; 1994 Aug.
- Owens JR, Jones JW, Harris F. Epidemiology of facial clefting. *Arch Dis Child* 1985 Jun;60(6):521-4.
- Palefsky JM, Silverman S Jr, Abdel-Salaam M, Daniels TE, Greenspan JS. Association between proliferative verrucous leukoplakia and infection with human papillomavirus type 16. *J Oral Pathol Med* 1995;24:193-7.
- Peto R, Lopez A, Boreham J, Thun M, Heath C. In: Slama K, editor. *Tobacco and health. Proceedings of the 9th World Conference on Tobacco and Health. Health effects of tobacco use: global estimates and projections*. New York: Plenum Press; 1995.
- Phipps KR, Stevens VJ. Relative contribution of caries and periodontal disease in adult tooth loss for an HMO dental population. *J Public Health Dent* 1995;55:250-2.
- Pillemer SR, Matteson EL, Jacobsson LT, Martens PB, Fox PC. Incidence of Sjögren's syndrome in Olmsted County, Minnesota [abstract]. *Arthritis Rheum* 1995;38(Suppl):S376.
- Pinkham JR, Kohn DW. Epidemiology and prediction of sports-related traumatic injuries. *Dent Clin North Am* 1991 Oct;35(4):609-26.
- Pollick HF, Isman R, Fine JL, Wellman J, Kipnis P, Ellison J. Report of the California oral health needs assessment of children, 1993-94. San Rafael (CA): The Dental Health Foundation; 1997.
- Ramirez RB. Hispanic population in the United States: 1999 Mar CPS. U.S. Bureau of the Census. Available at: <http://www.census.gov/population/www/socdemo/hispanic/ho99.html>.
- Ramos-Gomez FJ. Risk factors for early childhood caries [abstract]. *International Association for Dental Research*. Vancouver; 1999.
- Redford M. Beyond pregnancy gingivitis: bringing a new focus to woman's oral health. *J Dent Educ* 1993 Oct;57(10):742-8.
- Ries LA, Kosary CL, Hankey BF, Miller BA, Clegg L, Edwards BK, editors. *SEER cancer statistics review, 1973-1996*. Bethesda (MD): National Cancer Institute; 1999.
- Samaranayake LP. Oral mycoses in HIV infection. *Oral Surg Oral Med Oral Pathol* 1992 Feb;73(2):171-80.
- Sane J. Comparison of maxillofacial and dental injuries in four contact team sports: American football, bandy, basketball, and handball. *Am J Sports Med* 1988 Nov-Dec;16(6):647-51.

- Schulman J, Edmonds LD, McClearn AB, Jensvold N, Shaw GM. Surveillance for and comparison of birth defect prevalences in two geographic areas—United States, 1983-88. *MMWR Morb Mortal Wkly Rep* 1993 Mar 19;42(1):1-7.
- Scully C. Herpes simplex virus (HSV). In: Millard HD, Mason DK, editors. *World Workshop on Oral Medicine*, 1988 Jun 19-25. Chicago: Year Book Medical Publishers; 1989. p. 160.
- Selwitz RH, Winn DM, Kingman A, Zion GR. The prevalence of dental sealants in the U.S. population: findings from NHANES III, 1988-1991. *J Dent Res* 1996;75(SI):652-60.
- Ship II. Epidemiologic aspects of recurrent aphthous ulcerations. *Oral Surg Oral Med Oral Pathol* 1972 Mar;33(3):400-6.
- Ship II, Ashe WK, Scherp HW. Recurrent "fever blister" and "canker sore." Tests for herpes simplex and other viruses with mammalian cell cultures. *Arch Oral Biol* 1961 Feb;3:117-24.
- Ship II, Brightman VJ, Laster LL. The patient with recurrent aphthous ulcers and the patient with recurrent herpes labialis: a study of two population samples. *J Am Dent Assoc* 1967 Sep;75(3):645-54.
- Ship II, Miller MF, Ram C. A retrospective study of recurrent herpes labialis (RHL) in a professional population, 1958-1971. *Oral Surg Oral Med Oral Pathol* 1977 Nov;44(5):723-30.
- Silverman S Jr. Leukoplakia and erythroplasia. In: Silverman S Jr, editor. *Oral cancer*. 4th ed. Hamilton (Ontario): B.C. Decker; St. Louis: Mosby-Year Book; 1998. p. 25-40.
- Snowden CB, Miller-Chisholm AJ. Oral health of United States children: the National Dental Caries Prevalence Survey: 1979-1980 [public use file documentation and survey methodology]. Bethesda (MD): National Institutes of Health, National Institute of Dental Research; 1992.
- Talal N. Sjögren's syndrome: historical overview and clinical spectrum of disease. *Rheum Dis Clin North Am* 1992 Aug;18(3):507-15.
- Tepper BJ, Nurse RJ. Fat perception is related to PROP taster status. *Physiol Behav* 1997 Jun;61(6):949-54.
- Tomar S. Total tooth loss among persons aged greater than or equal to 65 years—selected states, 1995-1997. *MMWR Morb Mortal Wkly Rep* 1997;48:206-10.
- Tomar SL, Winn DM, Swango PA, Giovino GA, Kleinman DV. Oral mucosal smokeless tobacco lesions among adolescents in the United States. *J Dent Res* 1997 Jun;76(6):1277-86.
- U.S. Bureau of the Census. Health insurance coverage: 1996. Current population reports, P60-199. Washington: U.S. Department of Commerce; 1997 Sept.
- U.S. Bureau of the Census. National population projections I. Summary files. Available at: <http://www.census.gov/population/projections/nation/summary/np-t5-e.pdf>. 2000 Jan.
- U.S. Consumer Product Safety Commission (CPSC). Tricycles. Reporting hospitals and estimates reports, 1982-1986. Washington: National Electronic Injury Surveillance System, U.S. Consumer Product Safety Commission; 1987.
- U.S. Department of Health and Human Services (USDHHS). Healthy People 2000 Review 1995-96. Washington: U.S. Department of Health and Human Services; 1996. Pub. no. 96-1256.
- U.S. Department of Health and Human Services (USDHHS), Public Health Service, National Institutes of Health. Agenda for research on women's health for the 21st century. A report of the Task Force on the NIH Women's Health Research Agenda for the 21st century. Vol. 2. Bethesda (MD): National Institutes of Health; 1999. p. 136.
- U.S. Department of Health and Human Services (USDHHS). Healthy People 2010: understanding and improving health. Washington: U.S. Department of Health and Human Services; 2000. Available from: US GPO.
- U.S. Preventive Services Task Force. Guide to clinical preventive services. 2nd ed. Baltimore: Williams and Wilkins; 1996.
- Vargas CM, Crall JJ, Schneider DA. Sociodemographic distribution of dental caries: NHANES III, 1988-1994. *J Am Dent Assoc* 1998;129:1229-38.
- Vargas CM, Macek MD, Marcus SE. Sociodemographic correlates of tooth pain among adults: United States, 1989. *Pain* 2000 Mar;85(1-2):87-92.
- Von Korff M. Health services research and temporomandibular pain. In: Sessle BJ, Bryant PS, Dionne RA, editors. *Temporomandibular disorders and related pain conditions*. Seattle: IASP Press; 1995. p. 227-36.
- Waldman HB, Perlman SP, Swerdloff M. What if dentists did not treat people with disabilities? *J Dent Child* 1998;65:96-101.
- Waldman HB, Swerdloff M, Perlman SP. Children with disabilities: more than just numbers. *J Dent Child* 1999;66:192-6.
- Watson MR, Brown LJ. The oral health of U.S. Hispanics: evaluating their needs and their use of dental services. *J Am Dent Assoc* 1995;126:789-95.
- White BA, Caplan DJ, Weintraub JA. A quarter century of changes in oral health in the United States. *J Dent Educ* 1995 Jan;59(1):19-60.
- Whitley RJ. Prospects for vaccination against herpes simplex virus. *Pediatr Ann* 1993a;22:726,729-32.
- Whitley RJ. Neonatal herpes simplex virus infections. *J Med Virol* 1993b;(Suppl 1):13-21.
- Wingo PA, Ries LAG, Giovino GA, Miller DS, Rosenberg HM, Shopland DR, Thun MJ, Edwards BK. Annual report to the nation on the status of cancer, 1973-1996. With a special section on lung cancer.

- cer and tobacco smoking. *J Natl Cancer Inst* 1999;91:675-90.
- Winn DM, Blot WJ. Second cancer following cancer of the buccal cavity and pharynx in Connecticut, 1935-82. In: Boice JD Jr, Curtis RE, Kleinerman RA, Storm HH, Jensen OM, Jensen HS, Flannery JT, Fraumeni JF Jr, editors. *Multiple primary cancers in Connecticut and Denmark*. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute; 1985.
- Wolfe MD, Carlos JP. Oral health effects of smokeless tobacco use in Navajo Indian adolescents. *Community Dent Oral Epidemiol* 1987 Aug;15(4): 230-5.
- York AK, Poindexter FR, Chisick MC. 1994 Tri-Service comprehensive oral health survey; active duty report. 1995 Jun. NDRI Report no. PR-9503.

What Is the Relationship Between Oral Health and General Health and Well-being?

The next two chapters establish that oral health is essential to general health and well-being. Chapter 5 examines multiple linkages between oral and general health. The mouth and the face reflect signs and symptoms of health and disease that can serve as an adjunct for diagnosis for some conditions. Diagnostic tests using oral cells and fluids—especially saliva—are available to detect drug abuse, hormonal changes, and specific diseases; and more are being developed. The mouth is also a portal of entry for pathogens and toxins, which can affect the mouth and, if not cleared by the many defense mechanisms that have evolved to protect the oral cavity, may spread to the rest of the body. Recent epidemiologic and experimental animal research provides evidence of possible associations between oral infections—particularly periodontal disease—and diabetes, cardiovascular disease, and adverse pregnancy outcomes, and this evidence is reviewed. The review highlights the need for an aggressive research agenda to better delineate the specific nature of these associations and the underlying mechanisms of action.

Chapter 6 looks at the impact of oral health problems on the quality of life and includes examples of the kinds of questionnaires used to measure oral-health-related quality of life. Oral health is highly valued by society and individuals, and the chapter begins with a brief description of the reflections of those values in myth and folklore concerning facial appearance and the meaning of teeth. It then explores dimensions beyond the biological and the physical to examine how oral diseases and disorders can interfere with the functions of daily living, including participation in work or school, and what is known about their psychosocial impacts and economic costs. The deleterious effects of facial disfigurement and tooth loss may be magnified in a society such as ours that celebrates youth and beauty. Self-reported impacts of oral conditions on social functions include limitations in communication, social interactions, and intimacy. Research on the oral-health-related quality of life is needed to permit further exploration of the dimensions of oral health and well-being.

Linkages with General Health

The mouth and face are highly accessible parts of the body, sensitive to and able to reflect changes occurring internally. The mouth is the major portal of entry to the body and is equipped with formidable mechanisms for sensing the environment and defending against toxins or invading pathogens. In the event that the integrity of the oral tissues is compromised, the mouth can become a source of disease or pathological processes affecting other parts of the body. It can also become a source of contagion by means of contaminated fluids or materials passed to others. This chapter explores what the mouth and face can reveal about general health, describes the role the mouth plays as a portal of entry for infection, and concludes with studies that are associating oral infections with serious systemic diseases and conditions.

THE MOUTH AND FACE AS A MIRROR OF HEALTH AND DISEASE

A physical examination of the mouth and face can reveal signs of disease, drug use, domestic physical abuse, harmful habits or addictions such as smoking, and general health status. Imaging (e.g., x-ray, MRI, SPECT) of the oral and craniofacial structures may provide early signs of skeletal changes such as those occurring with osteoporosis and musculoskeletal disorders, and may also reveal salivary, congenital, neoplastic, and developmental disorders. Oral cells and fluids, especially saliva, can be tested for a wide range of substances, and oral-based diagnostics are increasingly being developed and used as a means to assess health and disease without the limitations and difficulties of obtaining blood and urine.

Physical Signs and Symptoms of Disease and Risk Factors

A number of signs and symptoms of disease, lifestyle behaviors, and exposure to toxins can be detected in or around the craniofacial complex. Pathogens entering the mouth may proliferate locally with oral and pharyngeal signs and symptoms; other pathogens may enter the bloodstream directly or through lymphatic channels and cause generalized disease. Oral signs suspected to be indications of systemic illness may be confirmed by the presence of rash, fever, headache, malaise, enlarged lymph nodes, or lesions elsewhere on the body.

Swollen parotid glands are a cardinal sign of infection with the mumps virus and can also be seen in individuals with Sjögren's syndrome and HIV. The salivary glands are also frequently involved in tuberculosis and histoplasmosis infections. Oral signs of infectious mononucleosis, caused by Epstein-Barr virus, include sore throat, gingival bleeding, and multiple pinpoint-sized hemorrhagic spots (petechiae) on the oral mucosa. The oral signs and symptoms associated with some viral, bacterial, and fungal infections are listed in Table 5.1. There can be a large overlap in the clinical appearance of oral manifestations of various diseases with different etiologies, and the clinical diagnosis often involves ancillary procedures, which may include laboratory tests, diagnostic imaging, and biopsy.

Oral tissues may also reflect immune deficiency. For example, nearly all HIV-infected individuals develop oral lesions at some time during their illness (Greenberg 1996, Greenspan and Greenspan 1996, Phelan 1997). Other immunosuppressed individuals may have the same lesions (Glick and Garfunkel 1992). However, the presentation and the extent, severity, and management of some of these lesions may reflect nuances due to variation in the underlying

TABLE 5.1
Diseases and conditions causing lesions of the oral mucosa

Condition	Usual Location	Clinical Features	Course
Viral Diseases			
Primary acute herpetic gingivostomatitis (herpes simplex virus type 1, rarely type 2)	Lip and oral mucosa	Labial vesicles that rupture and crust, and intraoral vesicles that quickly ulcerate; extremely painful; acute gingivitis, fever, malaise, foul odor, and cervical lymphadenopathy; occurs primarily in infants, children, and young adults	Heals spontaneously in 10 to 14 days unless secondarily infected
Recurrent herpes labialis	Mucocutaneous junction of lip, perioral skin	Eruption of groups of vesicles that may coalesce, then rupture and crust; painful to pressure or spicy foods	Lasts about 1 week, but condition may be prolonged if secondary infection occurs
Recurrent intraoral herpes simplex	Palate and gingiva	Small vesicles that rupture and coalesce; painful	Heal spontaneously in about 1 week
Chickenpox (varicella-zoster virus)	Gingiva and oral mucosa	Skin lesions may be accompanied by small vesicles on oral mucosa that rupture to form shallow ulcers; may coalesce to form large bullous lesions that ulcerate; mucosa may have generalized erythema	Lesions heal spontaneously within 2 weeks
Herpes zoster (reactivation of varicella-zoster virus)	Cheek, tongue, gingiva, or palate	Unilateral vesicular eruption and ulceration in linear pattern following sensory distribution of trigeminal nerve or one of its branches	Gradual healing without scarring; postherpetic neuralgia is common
Infectious mononucleosis (Epstein-Barr virus)	Oral mucosa	Fatigue, sore throat, malaise, low-grade fever, and enlarged cervical lymph nodes; numerous small ulcers usually appear several days before lymphadenopathy; gingival bleeding and multiple petechiae at junction of hard and soft palates	Oral lesions disappear during convalescence
Warts (papillomavirus)	Anywhere on skin and oral mucosa	Single or multiple papillary lesions, with thick, white keratinized surfaces containing many pointed projections; cauliflower lesions covered with normal-colored mucosa or multiple pink or pale bumps (focal epithelial hyperplasia)	Lesions grow rapidly and spread
Herpangina (coxsackievirus A; also possibly coxsackievirus B and echovirus)	Oral mucosa, pharynx, tongue	Sudden onset of fever, sore throat, and oropharyngeal vesicles, usually in children under 4 years, during summer months; diffuse pharyngeal congestion and vesicles (1 to 2 mm), grayish-white surrounded by red areola; vesicles enlarge and ulcerate	Incubation period 2 to 9 days; fever for 1 to 4 days; recovery uneventful
Hand, foot, and mouth disease (type A coxsackieviruses)	Oral mucosa, pharynx, palms, and soles	Fever, malaise, headache with oropharyngeal vesicles that become painful, shallow ulcers	Incubation period 2 to 18 days; lesions heal spontaneously in 2 to 4 weeks
Primary HIV infection	Gingiva, palate, and pharynx	Acute gingivitis and oropharyngeal ulceration, associated with febrile illness resembling mononucleosis and including lymphadenopathy	Followed by HIV seroconversion, asymptomatic HIV infection, and usually ultimately by HIV disease

Condition	Usual Location	Clinical Features	Course
Bacterial or fungal diseases			
Acute necrotizing ulcerative gingivitis ("trench mouth," Vincent's infection)	Gingiva	Painful, bleeding gingiva characterized by necrosis and ulceration of gingival papillae and margins plus lymphadenopathy and foul odor	Continued destruction of tissue followed by remission, but may recur
Prenatal (congenital) syphilis	Palate, jaws, tongue, and teeth	Gummatous involvement of palate, jaws, and facial bones; Hutchinson's incisors, mulberry molars, glossitis, mucous patches, and fissures of corners of mouth	Tooth deformities in permanent dentition irreversible
Primary syphilis (chancre)	Lesion appears where organism enters body; may occur on lips, tongue, or tonsillar area	Small papule developing rapidly into a large, painless ulcer with indurated border; unilateral lymphadenopathy; chancre and lymph nodes containing spirochetes; serologic tests positive by third to fourth week	Healing of chancre in 1 to 2 months, followed by secondary syphilis in 6 to 8 weeks
Secondary syphilis	Oral mucosa frequently involved with mucous patches, primarily on palate, also at commissures of mouth	Maculopapular lesions of oral mucosa, 5 to 10 mm in diameter with central ulceration covered by grayish membrane; eruptions occurring on various mucosal surfaces and skin accompanied by fever, malaise, and sore throat	Lesions may persist from several weeks to 1 year
Tertiary syphilis	Palate and tongue	Gummatous infiltration of palate or tongue followed by ulceration and fibrosis; atrophy of tongue papillae produces characteristic bald tongue and glossitis	Gumma may destroy palate, causing complete perforation
Gonorrhea	Lesions may occur in mouth at site of inoculation or secondarily by hematogenous spread from a primary focus elsewhere	Earliest symptoms are burning or itching sensation, dryness, or heat in mouth followed by acute pain on eating or speaking; tonsils and oropharynx most frequently involved; oral tissues may be diffusely inflamed or ulcerated; saliva develops increased viscosity and fetid odor; submaxillary lymphadenopathy with fever in severe cases	Lesions may resolve with appropriate antibiotic therapy
Tuberculosis	Tongue, tonsillar area, soft palate	A solitary, irregular ulcer covered by a persistent exudate; ulcer has an undermined, firm border	Lesions may persist
Cervicofacial actinomycosis	Swellings in region of face, neck, and floor of mouth	Infection may be associated with an extraction, jaw fracture, or eruption of molar tooth; in acute form resembles an acute pyogenic abscess, but contains yellow "sulfur granules" (gram-positive mycelia and their hyphae)	Acute form may last a few weeks; chronic form lasts months or years; prognosis excellent; actinomycetes respond to antibiotics (tetracyclines or penicillin) but not antifungal drugs
Histoplasmosis	Any area in mouth, particularly tongue, gingiva, or palate	Numerous small nodules may ulcerate; hoarseness and dysphagia may occur because of lesions in larynx usually associated with fever and malaise	May be fatal
Candidiasis	Any area of oral mucosa	Pseudomembranous form has white patches that are easily wiped off leaving red, bleeding, sore surface; erythematous form is flat and red; rarely, candidal leukoplakia appears as white patch in tongue that does not rub off; angular cheilitis due to <i>Candida</i> involves sore cracks and redness at angle of mouth; <i>Candida</i> seen on KOH preparation in all forms	Responds to antifungals

(continues)

TABLE 5.1 continued

Condition	Usual Location	Clinical Features	Course
Dermatologic diseases			
Mucous membrane pemphigoid	Primarily mucous membranes of the oral cavity, but may also involve the eyes, urethra, vagina, and rectum	Painful, grayish-white collapsed vesicles or bullae with peripheral erythematous zone; gingival lesions desquamate, leaving ulcerated area	Protracted course with remissions and exacerbations; involvement of different sites occurs slowly; glucocorticoids may temporarily reduce symptoms but do not control the disease
Erythema multiforme (Stevens-Johnson syndrome)	Primarily the oral mucosa and skin of hands and feet	Intraoral ruptured bullae surrounded by an inflammatory area; lips may show hemorrhagic crusts; the "iris," or "target" lesion, on the skin is pathognomonic; patient may have severe signs of toxicity	Onset very rapid; condition may last 1 to 2 weeks; may be fatal; acute episodes respond to steroids
Pemphigus vulgaris	Oral mucosa and skin	Ruptured bullae and ulcerated oral areas; mostly in older adults	With repeated recurrence of bullae, toxicity may lead to cachexia, infection, and death within 2 years; often controllable with steroids
Lichen planus	Oral mucosa and skin	White striae in mouth; purplish nodules on skin at sites of friction; occasionally causes oral mucosal ulcers and erosive gingivitis	Protracted course, may respond to topical steroids
Other conditions			
Recurrent aphthous ulcers	Anywhere on nonkeratinized oral mucosa (lips, tongue, buccal mucosa, floor of mouth, soft palate, oropharynx)	Single or clusters of painful ulcers with surrounding erythematous border; lesions may be 1 to 2 mm in diameter in crops (herpetiform), 1 to 5 mm (minor), or 5 to 15 mm (major)	Lesions heal in 1 to 2 weeks but may recur monthly or several times a year; topical steroids give symptomatic relief; systemic glucocorticoids may be needed in severe cases; a tetracycline oral suspension may decrease severity of herpetiform ulcers
Behçet's syndrome	Oral mucosa, eyes, genitalia, gut, and central nervous system	Multiple aphthous ulcers in mouth; inflammatory ocular changes; ulcerative lesions on genitalia; inflammatory bowel disease and CNS disease	Ulcers may persist for several weeks and heal without scarring
Traumatic ulcers	Anywhere on oral mucosa; dentures frequently responsible for ulcers in vestibule	Localized, discrete ulcerated lesion with red border; produced by accidental biting of mucosa, penetration by a foreign object, or chronic irritation by a denture	Lesions usually heal in 7 to 10 days when irritant is removed, unless secondarily infected

Source: Greenspan, in Fauci et al. 1998. Harrison's principles of internal medicine. Reprinted by permission from McGraw-Hill (2000). Copyright 2000 by McGraw-Hill.

systemic condition. For example, the linear gingival erythema and necrotizing ulcerative periodontitis sometimes seen in HIV infection have been difficult to resolve with routine dental curettage and prophylaxis (Glick et al. 1994b).

The appearance of soft or hard tissue pigmentation is associated with a number of diseases and treatments. Malignant melanoma can appear in the mouth as brown or black flat or raised spots. Kaposi's sarcoma can appear as a flat or raised pig-

mented lesion. Addison's disease causes blotches or spots of bluish-black or dark brown pigmentation to occur early in the disease. Congenital discrete brown or black patches (nevi) can appear in any part of the mouth. Pigmentation of the tooth crowns may be seen in children with cystic fibrosis and porphyria and those exposed to tetracycline during tooth development.

The oral tissues can also reflect nutritional status and exposure to risk factors such as tobacco. The

tongue appears smooth in pernicious anemia. Group B vitamin deficiency is associated with oral mucositis and ulcers, glossitis, and burning sensations of the tongue. Scurvy, caused by severe vitamin C deficiency, is associated with gingival swelling, bleeding, ulceration, and tooth loosening. Lack of vitamin D in utero or infancy impairs tooth development. Enamel hypoplasia may result from high levels of fluoride or from disturbances in calcium and phosphate metabolism, which can occur in hypoparathyroidism, gastroenteritis, and celiac disease. The mouth also can reflect the effects of tobacco use, perhaps providing the only visible evidence of its adverse effects.

Oral Manifestations of HIV Infection and of Osteoporosis

The mouth can serve as an early warning system, diagnostic of systemic infectious disease and predictive of its progression, such as with HIV infection. In the case where oral cells and tissues have counterparts in other parts of the body, oral changes may indicate a common pathological process. During routine oral examinations and perhaps in future screening tests, radiographic or magnetic resonance imaging of oral bone may be diagnostic of early osteoporotic changes in the skeleton. The following sections provide details.

HIV Infection

The progressive destruction of the body's immune system by HIV leads to a number of oral lesions, such as oral candidiasis and oral hairy leukoplakia, that have been used not only in diagnosis but also in determining specific stages of HIV infection (CDC 1992, 1994, Montaner et al. 1992, Redfield et al. 1986, Royce et al. 1991, Seage et al. 1997). Oral candidiasis is rarely seen in previously healthy young adults who have not received prior medical therapy such as cancer chemotherapy or treatment with other immunosuppressive drugs (Klein et al. 1984). It was associated with AIDS as early as 1981 in the first report of the syndrome (CDC 1981a) and was frequently noted among otherwise asymptomatic HIV-positive populations (Duffy et al. 1992, Feigal et al. 1991). Oral candidiasis may be the first sign of HIV infection and often occurs as part of the initial phase of infection—the acute HIV syndrome (Tindall et al. 1995). It tends to increase in prevalence with progression of HIV infection when CD4 lymphocyte counts fall (Glick et al. 1994a, Lifson et al. 1994). It also appears to be the most common oral manifestation in pediatric HIV infection (Kline 1996, Leggott

1995, Ramos-Gomez et al. 1996) and has been demonstrated to proceed to esophageal candidiasis, a sign of overt AIDS (Saah et al. 1992). Both the pseudomembranous and the erythematous forms of candidiasis appear to be important predictors of progression of HIV infection (Dodd et al. 1991, Klein et al. 1984, 1992).

Like oral candidiasis, oral hairy leukoplakia in HIV-positive persons heralds more rapid progression to AIDS (Glick et al. 1994a, Greenspan et al. 1987, Lifson et al. 1994, Morfeldt-Manson et al. 1989). Oral hairy leukoplakia is an oral lesion first reported in the early days of the AIDS epidemic (Greenspan D et al. 1984, Greenspan JS et al. 1985). Since its discovery, hairy leukoplakia has been found in HIV-negative persons with other forms of immunosuppression, such as organ or bone marrow recipients and those on long-term steroid therapy (Epstein et al. 1988, Greenspan et al. 1989, Itin et al. 1988, King et al. 1994, Zakrzewska et al. 1995), and less frequently among immunocompetent persons (Eisenberg et al. 1992, Felix et al. 1992).

In a comprehensive review of periodontal conditions, Mealey (1996) noted that linear gingival erythema and necrotizing ulcerative periodontitis may be predictive of progression of HIV infection. Necrotizing ulcerative periodontitis, a more serious periodontal condition observed in HIV-infected persons, is a good predictor of CD4+ cell counts of under 200 per cubic millimeter, and in one study was a strong predictor of rapid progression to death (Glick et al. 1994a,b, Winkler and Robertson 1992, Winkler et al. 1988). In addition, the numerous ulcerative and nonulcerative conditions that affect the oral cavity (Aldous and Aldous 1991, Coates et al. 1996, Cruz et al. 1996, Gandolfo et al. 1991, Itin et al. 1993, Mealey 1996) may affect the biologic activity of HIV and are affected by its treatments.

Other oral conditions, unexpected in the oral cavity, have been noted in the early stages of HIV infection. The increased incidence of Kaposi's sarcoma among young men in New York and California was one of the earliest signs of the AIDS epidemic (CDC 1981b). In addition, some conditions create a problem for differential diagnosis. For example, because involvement of the gingiva is common, early non-Hodgkin's lymphoma lesions are frequently mistaken for common periodontal or dental infections (Epstein and Silverman 1992).

The changing face of the HIV epidemic and changes in the therapies used to manage complications are reflected in changes in the oral manifestations, which warrant continued surveillance and research. The increasing resistance of microorganisms

to antibiotics and antifungals is challenging. On the other hand, the completion of the *Candida albicans* genome may yield better treatments for this opportunistic infection.

Osteoporosis and Oral Bone Loss

With growing numbers of Americans living longer, there have been concomitant increases in the numbers affected by age-related chronic degenerative diseases. Prominent among these conditions are bone and joint diseases. It is likely, for example, that some temporomandibular joint disorders are manifestations of osteoarthritis, rheumatoid arthritis, or myofascial pain. Paget's disease, characterized by enlarged and deformed bone, can be particularly painful and debilitating when it affects the cranial and jaw bones.

Osteoporosis, a degenerative disease characterized by the loss of bone mineral and associated structural changes, has long been suspected as a risk factor for oral bone loss. In addition, measures of oral bone loss have been proposed as potential screening tests for osteoporosis (Jeffcoat 1998). Osteoporosis affects over 20 million people in the United States, most of whom are women, and results in nearly 2 million fractures per year (National Institute of Arthritis, Musculoskeletal and Skin Diseases 2000). The disease is more prevalent in white and Asian American women than in black women.

Oral bone loss has been reported to be more prevalent in women than in men. Studies by Ortman et al. (1989) found a higher percentage of women than men with severe alveolar ridge resorption. This finding parallels the findings of Humphries et al. (1989), who showed that loss of bone mineral density with age in edentulous adult mandibles was more significant in women than in men. Also, the association between estrogen status, alveolar bone density, and history of periodontitis in postmenopausal women has been studied (Payne et al. 1997).

Most of the studies in this area have examined bone loss in women, and most investigators have reported a correlation between oral and skeletal bone loss measured in a variety of ways. Studies of non-osteoporotic women by Kribbs et al. (1990) showed that mandibular bone mass is significantly correlated with skeletal bone mass. Dual photon absorptiometry measurements of jawbone volume in women with osteoporosis have shown that reduction in mandibular bone mass is directly related to the reduction in total skeletal mass density (Kribbs and Chesnut 1984, Kribbs et al. 1983, von Wowerm 1985, 1988, von Wowerm et al. 1994). Kribbs et al. (1989) further showed that mandibular mass is correlated with all

skeletal measures in osteoporotic women and that the height of the edentulous ridge is correlated with total body calcium and mandibular bone mineral density. Hirai et al. (1993) found that the presence of skeletal osteoporosis strongly affects the reduction of the residual ridge in edentulous patients. A small case-control study comparing older female patients with osteoporotic fractures and non-osteoporotic women without fractures found greater periodontal attachment loss in the osteoporotic women than in the controls (von Wowerm et al. 1994).

Studies that have controlled for confounding factors also have found correlations between oral bone loss and skeletal bone density. Controlling for pack-years of smoking, education, body mass, and years since menopause, Krall et al. (1994, 1996) found a significant positive relationship between number of teeth and bone mineral density of the spine and the radius. In a cohort of 70 postmenopausal women, Wactawski-Wende et al. (1996) measured skeletal bone mineral density at the Ward's triangle area of the femur and compared it with periodontal disease assessed by attachment loss and the height of alveolar bone measured by radiographs. After adjusting for age, years since menopause, estrogen use, body mass index, and smoking, the investigators concluded that osteopenia (low bone mass) is related to alveolar crestal height and tooth loss in postmenopausal women.

Methods used to measure oral and skeletal bone loss have varied among investigators and have shown different outcomes. Kribbs (1990) found that patients in an osteoporotic group had lost more teeth, had less mandibular bone, and had a thinner bone measured at a part of the jaw (cortex at the gonion) than a comparable non-osteoporotic group. However, using periodontal attachment loss as an indicator of mandibular bone loss, they found no differences between the osteoporotic and the non-osteoporotic group. Mohajery and Brooks (1992) compared non-osteoporotic postmenopausal women with women with mild to moderate osteoporosis and found no correlation between mandibular and skeletal bone mineral density. This study raises questions about the quantification of mild and moderate osteoporosis. Defining healthy periodontal tissues as having no periodontal pockets deeper than 5 millimeters, Hildebolt et al. (1997) studied postmenopausal women with healthy periodontal tissues and found no relationship between periodontal attachment loss and postcranial bone mineral density. However, preliminary studies from the oral ancillary study of the NIH Women's Health Initiative report significant correlations between mandibular basal bone mineral density and hip bone mineral density

($r = 0.74$, $P < .001$) (Jeffcoat et al. in press). In this study, digital subtraction radiography methods were used for mandibular bone measurements, and dual-energy X-ray absorptiometry (DXA) scans were used for the hip bone measurements. The authors of this study propose the possibility that high-quality intra-oral radiographs may be used in the future for screening osteopenia.

Larger cross-sectional studies, as well as longitudinal and mechanism studies, are needed to better define the relationship between osteoporosis, osteopenia, and oral bone loss, periodontal disease, and tooth loss. The role of factors involved in the reg-

ulation of bone mineral density in men as well as in postmenopausal women needs to be evaluated further with reference to oral bone loss, tooth loss, and periodontal disease. Variables such as sex, race, dietary calcium and phosphorus, vitamin D intake, exercise, body mass index, smoking, genetics, medication use, reproductive history, and psychosocial factors need to be assessed in depth. In addition, reliable and valid criteria and imaging technologies for assessing osteoporosis and oral bone loss are needed to better elucidate the full relationship between skeletal and mandibular bone mineral density, periodontal disease, alveolar ridge resorption, and tooth loss.

TABLE 5.2
Saliva/oral fluids: sampled analytes and current FDA-approved tests

Category	Analytes	FDA-Approved Tests
Drugs of abuse ^a	Alcohol	Cannabinoids
	Amphetamines	Cocaine
	Barbiturates	Cotinine
	Benzodiazepines	Methamphetamine
	Cocaine	Opiates
	LSD	PCP
	Marijuana	Ethanol
	Nicotine	
	Opiates	
	PCP	
	Antibodies ^b	HIV
HPV		
HHV-8/KSH		
<i>C. parvum</i>		
<i>Helicobacter pylori</i>		
Hormones ^c	Cortisol	Estriol
	Progesterone	
	Testosterone	
	Substance P	
	Met-enkephalin	
Environmental toxins ^d	Cadmium	
	Lead	
	Mercury	
Therapeutics ^e	Antipyrene	
	Carbamazepine	
	Ciprofloxacin	
	Irinotecan	
	Lithium	
	Methotrexate	
	Phenytoin	
	Phenobarbital	
	Theophylline	

^aCone et al. 1993, 1997.

^bConstantine et al. 1997.

^cDabbs 1993, Ellison 1993.

^dGonzalez et al. 1997, Joselow et al. 1968.

^eWilson 1993.

Source: Constantine et al. 1997.

Oral-fluid-based Diagnostics: The Example of Saliva

The diagnostic value of salivary secretions to detect systemic diseases has long been recognized (Mandel 1990), and oral fluids and tissues (buccal cells) are increasingly being used to diagnose a wide range of conditions. Saliva- and oral-based diagnostics use readily available samples and do not require invasive procedures. Researchers have detected antibodies in saliva that are directed against viral pathogens such as human immunodeficiency virus (Malamud 1997) and hepatitis A virus (O'Farrell et al. 1997) or B virus (Richards et al. 1996). Saliva is being used to detect antibodies, drugs, hormones, and environmental toxins (Malamud and Tabak 1993) (Table 5.2). The simplest tests are those that detect the presence or absence of a substance in the saliva, such as various drugs. Greater technical challenges are presented for tests that will be used for therapeutic monitoring since accurate levels of a substance and/or its metabolites are needed. In these instances the saliva/plasma concentration ratio must be determined experimentally (Haeckel 1993). Tests beyond those listed in Table 5.2 are currently on the market, but do not yet have FDA approval. Saliva is also the fluid of choice to assess the integrity of the mucosal immune system (Mandel 1990).

Most recently, oral fluids have been used as a source of microbial or host DNA. With the advent of polymerase chain reaction methods, the DNA contained within a single cell is sufficient for detection of viruses (e.g., Kaposi's sarcoma-associated herpes virus, Koelle et al. 1997; Epstein-Barr virus, Falk et al. 1997; mumps virus, Afzal et al. 1997) or bacteria (e.g., *Helicobacter pylori*, Reilly et al. 1997). Similarly, DNA extracted from sloughed buccal epithelial cells can be used to genotype persons. This has found application in forensics (Roy et al. 1997) and may be

used for diagnostic purposes in the future (van Schie and Wilson 1997).

Saliva has the potential of replacing blood, the current standard for testing many diseases and conditions (e.g., diabetes, infectious disease, Parkinson's disease, alcoholic cirrhosis, Sjögren's syndrome, and cystic fibrosis sarcoidosis). Important goals for the future are the development of new diagnostic tests for early disease detection, defining individual patient risk of adverse response to drugs, monitoring therapeutic progress, and determining outcomes of treatment. Key issues in the development of a new generation of saliva diagnostics include their selectivity, sensitivity, response time, dynamic range (values of interest), representative sampling, and, perhaps most important, their reliability or stability as well their ability to assess multiple substances simultaneously.

Conclusion

For the clinician the mouth and face provide ready access to physical signs and symptoms of local and generalized disease and risk factor exposure. These signs and symptoms augment other clinical features of underlying conditions. Comprehensive care of the patient requires knowledge of these signs and symptoms, their role in the clinical spectrum of general diseases and conditions, and their appropriate management. Oral biomarkers and surrogate measures are also being explored as means of early diagnosis. With further development and refinement, oral-based diagnostics such as salivary tests can become widely used and acceptable tools for individuals, health care professionals, researchers, and community programs. The continued refinement of imaging techniques also has the potential of using oral imaging to identify early signs of skeletal bone degeneration.

THE MOUTH AS A PORTAL OF ENTRY FOR INFECTION

Chapter 3 provides an overview of the effects of oral microbial infections with viruses, bacteria, and fungi. More than 500 bacterial strains have been identified in dental biofilm, and more than 150 bacterial strains have been isolated from dental pulp infections. More recently, 37 unique and previously unknown strains of bacteria were identified in dental plaque (biofilm) (Kroes et al. 1999). Most oral lesions are opportunistic infections, that is, they are caused by microorganisms commonly found in the mouth, but normally kept in check by the body's defense mechanisms.

These microorganisms can induce extensive localized infections that compromise general well-being in and of themselves. However, they also may spread to other parts of the body if normal barriers are breached. The oral mucosa is one such barrier that provides critical defense against pathogens and other challenges (Schubert et al. 1999). Salivary secretions are a second major line of defense. Damage to the oral mucosa from mechanical trauma, infection, or salivary dysfunction with resulting derangements in lubricatory and antimicrobial functions of saliva, as a result of chemotherapy, radiation, and medications causing hyposalivation, allows a portal of entry for invading pathogens.

Oral Infections and Bacteremia

Oral microorganisms and cytotoxic by-products associated with local infections can enter the bloodstream or lymphatic system and cause damage or potentiate an inappropriate immune response elsewhere in the body. Dissemination of oral bacteria into the bloodstream (bacteremia) can occur after most invasive dental procedures, including tooth extractions, endodontic therapy, periodontal surgery, and scaling and root planing. Even routine oral hygiene procedures such as daily toothbrushing, subgingival irrigation, and flossing may cause bacteremia. However, these distant infections have been seen more often in high-risk patients such as those who are immunocompromised.

Oral bacteria have several mechanisms by which they invade mucosal tissues, perhaps contributing to their ability to cause bacteremias. For example, oral bacteria and their products may invade the periodontal tissues directly. *Actinobacillus actinomycetemcomitans* has been found in gingival connective tissue in patients with localized juvenile periodontitis (Christersson et al. 1987a,b, Meyer et al. 1991, Riviere et al. 1991). Invasion of tissue by *Porphyromonas gingivalis* has also been described in vivo (Saglie et al. 1988) and in vitro (Njoroge et al. 1997, Sandros et al. 1993, 1994, Weinberg et al. 1997). Although oral bacteria can enter the blood through injured or ulcerated tissue, bacterial invasion of periodontal tissues represents another possible mechanism.

In the immunocompetent individual, bacteremia originating from the oral cavity is usually transient and harmless. However, if the individual's immune system is compromised, the normally harmless oral bacteria may pose a significant risk. The morbidity and mortality associated with oral foci of infections are hard to assess. This is due to the formidable task

of tracking the source of an infection unless the responsible pathogen is indigenous to a specific anatomic location.

Viridans group streptococci (VGS) have a low degree of virulence but can be associated with morbidity and mortality under certain circumstances. Increased pathogenicity of *Streptococcus viridans* is most prominent in individuals with neutropenia (low blood counts of circulating white blood cells called neutrophils) and has been associated with a toxic-shock-like syndrome (TSLs) or viridans streptococcal shock syndrome (VSSS), as well as with adult respiratory distress syndrome (ARDS) (Bochud et al. 1994).

Although a high degree of morbidity is associated with viridans streptococcal bacteremia, a low incidence of mortality has been reported (Heimdahl et al. 1989). Several studies have shown that under adverse circumstances oral flora and oral infections are associated with increased incidence of morbidity and even mortality (Engelhard et al. 1995, Lucas et al. 1998, Martino et al. 1995, Ruescher et al. 1998, Sparrelid et al. 1998, Sriskandan et al. 1995). Reduction of oral foci of infection decreases systemic complications, specifically in severely neutropenic patients undergoing chemotherapy (Heimdahl et al. 1984). In addition, hospital stays for patients with oral mucositis undergoing autologous bone marrow transplants were longer than for those without oral mucositis (Ruescher et al. 1998).

Other cohorts identified at increased risk for systemic complications due to oral bacteria include hospitalized patients unable to perform adequate oral hygiene, those receiving saliva-reducing medications, and those taking antibiotics that alter the oral flora. A positive dental plaque culture for aerobic pathogens was significantly associated with the development of hospital-acquired pneumonia and bacteremia in a study of individuals in an intensive care unit (ICU) (Fourrier et al. 1998).

In addition, several case reports have been published implicating indigenous oral flora in the development of brain abscesses (Andersen and Horton 1990, Andrews and Farnham 1990, Baker et al. 1999, Gallagher et al. 1981, Goteiner et al. 1982, Saal et al. 1988). This serious condition is associated with a mortality rate of almost 20 percent and full recovery in only slightly more than 50 percent of all patients (Goteiner et al. 1982). These data are based on single case reports and most probably represent rare events. However, they provide additional examples that point to the potential pathogenicity of the normal oral flora during special adverse circumstances.

Oral Infections as a Result of Therapy

Chemotherapy

Oral mucositis can be a major dose-limiting problem during chemotherapy with some anticancer drugs, such as 5-fluorouracil, methotrexate, and doxorubicin. It is estimated that approximately 400,000 patients undergoing cancer therapy each year will develop oral complications (NIH 1990). Infection of ulcerated mucous membranes often occurs after chemotherapy, especially since patients are usually immunocompromised. Bacterial, fungal, and viral causes of mucositis have been identified (Feld 1997). The mechanism by which cancer-chemotherapy-induced mucositis occurs is likely associated with the rapid rate of turnover of oral epithelial cells. In addition, other components likely include upregulation of pro-inflammatory cytokines and metabolic by-products of colonizing oral microflora (Sonis 1998). Chemotherapy alters the integrity of the mucosa and contributes to acute and chronic changes in oral tissue and physiologic processes (Carl 1995). The ulcerated mucosa is susceptible to infection by microbial flora that normally inhabit the oral cavity, as well as by exogenous organisms, and exacerbates the existing mucositis. Further, these microflora can disseminate systemically (Pizzo et al. 1993, Rolston and Bodey 1993). Compromised salivary function can further elevate risk for systemic infection of oral origin.

Both indigenous oral flora and hospital-acquired pathogens have been associated with bacteremias and systemic infection (Schubert et al. 1999). Changes in infection profiles in myelosuppressed (immunosuppressed) cancer patients tend to occur in cyclic fashion over many years. This evolving epidemiology is caused by multiple factors including use of antibiotics. Gram-positive organisms including viridans streptococci and enterococci are currently associated with systemic infection of oral origin in myelosuppressed cancer patients. In addition, gram-negative pathogens including *P. aeruginosa*, *Neisseria* spp., and *Escherichia coli* remain of concern.

Cancer patients undergoing bone marrow radiation who have chronic periodontal disease may also develop acute periodontal infections with systemic complications (Peterson et al. 1987). The extensive ulceration of gingival sulcular epithelium associated with periodontal disease is not directly observable clinically, yet may represent a source for disseminated infection by an extensive array of organisms. Inflammatory signs may be masked due to the underlying bone marrow suppression.

Viruses are also associated with clinically important oral disease in patients receiving chemotherapy (Rolston and Bodey 1993, Pizzo et al. 1993). Infections caused by herpes simplex virus, varicella-zoster virus, and Epstein-Barr virus typically result from reactivation of a latent virus, whereas cytomegalovirus infections can result via reactivation of a latent virus or a newly acquired virus. The severity of the infection, including fatal outcome, depends on the degree of immunocompromise.

Many agents and protocols have been investigated to manage or prevent mucositis (Peterson 1999, Schubert et al. 1998). For example, various biologic response modifiers, including transforming growth factor β 3 or keratinocyte growth factor, have been under recent study in randomized clinical trials. Allopurinol mouthwash and vitamin E have been cited as agents that can decrease the severity of mucositis, although more extensive testing is necessary. Prostaglandin E2 was not shown to be effective in prophylaxis of oral mucositis following bone marrow transplant; however, more recent studies indicate possible efficacy when administered via a different dosing protocol. Oral cryotherapy appears to be efficacious in reducing severity of oral mucositis caused by 5-fluorouracil and related compounds (Rocke et al. 1993).

Local application of capsaicin preparations may be effective in controlling oral mucositis pain as distinguished from tissue injury itself (Berger et al. 1995). Capsaicin and its analogs are the active ingredients in chili peppers. Capsaicin's clinical potential derives from the fact that it elevates the threshold for pain in areas to which it is applied.

Radiation Therapy

Radiation therapy disrupts cell division in healthy tissue as well as in tumors and also affects the normal structure and function of craniofacial tissues, including the oral mucosa, salivary glands, and bone. Oral-facial complications are common after radiation therapy to the head and neck. The most frequent, and often the most distressing, complication is mucositis, but adverse reactions can affect all oral-facial tissues (Scully and Epstein 1996).

Radiation can cause irreversible damage to the salivary glands, resulting in dramatic increases in dental caries. Oral mucosal alterations may become portals for invasion by pathogens, which may be life-threatening to immunosuppressed or bone-marrow-suppressed patients. A less common but very serious adverse consequence is destruction of bone cells and bone death, called osteoradionecrosis (ORN). ORN can result in infection of the bone and soft tissue and

can require surgery to excise the dead tissue, which can in turn leave the face badly disfigured as well as functionally impaired (Field et al. 2000). The likelihood of ORN is increased with trauma to the bone, including that caused by tooth extraction (Murray et al. 1980a, b). The risk is especially marked when the trauma occurs near the time of radiation (Epstein et al. 1987). Management includes elimination of acute or potential dental and periodontal foci of disease, increased patient participation in oral hygiene, use of oral topical fluorides for caries prevention, and use of antiviral, antifungal, or antimicrobial therapy for management of infections associated with mucositis.

Combined Cancer Therapies

Rapid developments have occurred in the use of blood cell growth factors for treatment of various conditions, including the anemia of end-stage renal disease, the neutropenia occurring with cancer care, and the bone marrow toxicity and mucositis that can follow aggressive chemotherapy or radiation therapy (Sonis et al. 1997, Williams and Quesenberry 1992). Sonis et al. (1997) found that topical application of transforming growth factor beta (TGF- β) in the hamster model of oral mucositis significantly reduced basal cell proliferation and reduced the severity of mucositis associated with 5-fluorouracil treatment.

Other growth factors considered for use in reducing mucositis include granulocyte-monocyte colony-stimulating factor and granulocyte colony-stimulating factor. Bone morphogenetic proteins are also in development for alleviating the toxicity and mucositis that follow chemotherapy and radiation therapy. Other approaches to reducing mucositis and adverse oral effects of chemotherapy and radiation therapy include fractionating the dose of radiation, and combining chemotherapy with growth factors or with less toxic oncostatic agents. Although the oral mucositis occurring in chemotherapy and in head and neck radiation patients shares many characteristics, distinct differences also exist (NIH 1990, Schubert et al. 1998, Wilkes 1998). For example, in contrast to chemotherapy-associated lesions, radiation damage is anatomically site-specific; toxicity is localized to irradiated tissue volumes. The degree of damage depends on treatment-regimen-related factors, including the type of radiation used, the total dose administered, the fractionation, and field size. Thus, research involving both cohorts of cancer patients remains essential to enhancing patient management.

Development of new technologies to prevent cancer-therapy-induced oral mucositis could

substantially reduce the risk for oral and systemic infections, oral pain, and the number of hospital days. Improvement in quality of life and reduction in health costs are also likely and desirable outcomes.

The new technologies could also provide a setting in which novel classes of chemotherapeutic drugs, utilized at increased doses, could be implemented. These advances in turn could lead to enhanced cancer patient survival and lengthen the duration of disease remission.

Pharmaceuticals

A number of medications used to treat systemic diseases can cause oral complications, ranging from xerostomic effects to alterations in the surface structure of the enamel or mucosa. More than 400 over-the-counter and prescription drugs have xerostomic side effects (Sreebny and Schwartz 1997). These include tricyclic antidepressants, antihistamines, and diuretics. The dimensions and impact of these side effects vary depending on the response of the individual patient and the duration of medication use.

Staining of the teeth or mucosa is associated with a variety of drugs, including tranquilizers, oral contraceptives, and antimalarials. The antibiotic tetracycline can cause enamel hypoplasia when taken by the mother during pregnancy and by children during tooth development. The antimicrobial mouthrinse agent chlorhexidine also can stain the teeth, but this staining is external and can be removed by dental prophylaxis.

Other drugs have been associated with gingival overgrowth, including cyclosporin, which has been used as an immunosuppressant in the United States since 1984 to prevent rejection of transplanted organs and bone marrow. This drug has also been used in other countries for treatment of type 2 diabetes, rheumatoid arthritis, psoriasis, multiple sclerosis, malaria, sarcoidosis, and several other diseases with an immunological basis (Adams and Davies 1984). Other drugs that cause gingival overgrowth include calcium ion channel blocking agents used in the treatment of angina pectoris and postmyocardial syndrome, such as nifedipine and verapamil (Lucas et al. 1985), and phenytoin (sodium 5,5-phenylhydantoin), used in the treatment of epilepsy and also for management of other neurological disorders. Treatment often consists of using an alternate drug, although this is not always possible. Conservative periodontal therapy can reduce the inflammatory component of enlargement; however, surgery is often required. Oral candidiasis is typically caused by opportunistic overgrowth of *Candida albicans*. Drugs that cause systemic bone marrow suppression, oral

mucosal injury, or salivary compromise collectively promote the risk for clinical infection. In addition, antibiotics and concurrent steroid therapy often alter oral flora, thereby creating an environment for fungal overgrowth. In high-risk cancer patients, fungal infection can cause severe morbidity and even death.

Infective Endocarditis

The purported connection between oral infection and a specific heart disease, infective endocarditis, has a long history. Endocarditis is caused by bacteria that adhere to damaged or otherwise receptive surfaces of the tissue that lines heart valves (the endocardium) (Weinstein and Schlesinger 1974). Dental and other surgical procedures may predispose susceptible patients to infective endocarditis by inducing bacteremias (Lacassin et al. 1995). However, bacteremias from oral infections that occur frequently during normal daily activities, coincidental even with chewing food, toothbrushing, and flossing, contribute more substantially to the risk of infective endocarditis (Bayliss et al. 1983, Dajani et al. 1997, Strom et al. 1998). Oral organisms are common etiologic agents of infective endocarditis (Bayliss et al. 1983). For example, strains of *S. sanguis*, as well as gram-negative oral bacteria including *Haemophilus aphrophilus*, *A. actinomycetemcomitans*, *E. corrodens*, *Capnocytophaga* spp., and *Fusobacterium nucleatum*, have been associated with bacterial endocarditis (Barco 1991, Geraci and Wilson 1982, Kaye 1994, Mouldsdale et al. 1980).

Infective endocarditis occurs with different incubation periods, which differ in causative bacteria and signs and symptoms. For example, *Staphylococcus aureus* endocarditis may have a rapid onset and fatal course if it affects the left side of the heart. With a more indolent course, patients may often be unaware of infection and may experience fever, night chills, myalgia, and arthralgia for a considerable period of time before diagnosis. The infection is often curable if diagnosed and treated early.

The classic risk factors for endocarditis include cardiac valve disorders (valvulopathies) that include rheumatic and congenital heart disease, complex cyanotic heart disease in children, and mitral valve prolapse with regurgitation. Recent studies indicate that the use of certain diet drugs (fenfluramine and dexfenfluramine) has induced cardiac valvulopathy, which may in some cases be transient. Among at-risk persons, bacteremias are more likely to occur in those with periodontal disease (Silver et al. 1977). However, the oral pathogens causing periodontitis have only rarely been shown to cause endocarditis.

Prevention of infective endocarditis from oral bacteria depends on limiting the entry and dissemination of bacteria through the bloodstream and lymphatic circulation. Antibiotic prophylaxis for dental procedures that are likely to provoke bacteremia has historically been recommended (Dajani et al. 1997, Durack 1995). A recent study, however, suggests that receiving dental treatment does not significantly increase the risk of infective endocarditis, even in patients with valvular abnormalities (Strom et al. 1998). Further research is necessary to determine whether some heart or valvular conditions or certain dental procedures, such as surgery or scaling, would require coverage with pre-procedural antibiotics and others would be precluded.

Oral Infections and Respiratory Disease

Pathogens in the oral cavity can also gain access to the airway, sometimes with serious consequences. In adults, bacterial pneumonias are strongly associated with aspiration of bacteria into the lower respiratory tract, which is normally sterile. Common respiratory pathogens such as *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Mycoplasma pneumoniae*, and *Haemophilus influenzae* can colonize the oropharynx and the lower airway. In addition, oral bacteria including *A. actinomycetemcomitans* (Yuan et al. 1992), *Actinomyces israelii* (Morris and Sewell 1994, Zijlstra et al. 1992), *Capnocytophaga* spp. (Lorenz and Weiss 1994), *Eikenella corrodens* (Joshi et al. 1991), *Prevotella intermedia*, and *Streptococcus constellatus* (Shinzato and Saito 1994) can be aspirated into the lower airways (Scannapieco 1998, 1999).

Chronic obstructive pulmonary disease, characterized by obstruction of airflow due to chronic bronchitis or emphysema and by recurrent episodes of respiratory infection, has been associated with poor oral health status (Hayes et al. 1998, Scannapieco et al. 1998). A positive relationship between periodontal disease and bacterial pneumonia has been shown by Scannapieco and Mylotte (1996).

Although oral bacteria, including periodontal pathogens, have the potential for causing respiratory infections, the frequency and nature of such infections are not known and merit further study.

Oral Transmission of Infections

Besides being a portal of entry for infections, the mouth is an important source of potentially pathogenic organisms and is often the vehicle by which infection is delivered to the bodies of others. Microorganisms were not discovered in the mouth

until the seventeenth century, when van Leewenhoek examined dental plaque using a microscope he had constructed. In 1884, Koch demonstrated that tuberculosis could be transmitted by airborne droplets from the mouth and respiratory tract. Since that time, we have learned that many common respiratory infections, such as influenza, the common cold, pneumonia, and tuberculosis, can be transmitted from oral secretions. Before the development of effective vaccines, orally transmitted diseases such as chickenpox, measles, mumps, polio, and diphtheria were a major source of morbidity and mortality in childhood. Viral diseases such as hepatitis B, herpes labialis, acute herpetic gingivostomatitis, cytomegalovirus, and infectious mononucleosis may also originate from oral contact.

Disease-causing microorganisms can be spread by direct contact (with saliva or blood from the mouth) or indirect contact (with saliva- or blood-contaminated surfaces, including hands or lips), droplet infection (from coughing, sneezing, or even normal speech), or by aerosolized organisms. These organisms can be inhaled, ingested, or taken in through mucous membranes in the eyes, nose, or mouth or through breaks in the skin. A number of diseases can be spread via oral sexual contact, including gonorrhea, syphilis, trichomoniasis, chlamydia, and mononucleosis.

As mentioned earlier, the oral mucosa and saliva provide significant defense against disease transmission. Epidemiological and animal studies are providing evidence, however, that the oral cavity may be the site for transmission of serious systemic infections despite the protective factors in saliva (see Chapter 2). Infection with HIV provides a case in point (Baba et al. 1996, Dillon et al. 2000, Pope et al. 1997, Ruprecht et al. 1999, Stahl-Hennig et al. 1999, Baron et al. 2000).

Early in the 1980s, when AIDS was first identified in the United States, concern was expressed about casual (i.e., nonsexual) transmission of HIV (CDC 1983, 1985). Detailed household studies did not demonstrate transmission of HIV, even when family members shared eating utensils and toothbrushes with an HIV-affected member (Fischl et al. 1987, Rogers et al. 1990, Sande 1986). Similarly, surveillance data collected over time showed no evidence of casual transmission (Ward and Duchin 1997).

Only one nonoccupational episode of HIV transmission has been attributed to blood-contaminated saliva (CDC 1997); this incident involved intimate kissing between sexual partners. There have been a few cases of HIV transmission from performing oral

sex on a person infected with HIV, and it is also possible to become infected with HIV by receiving oral sex. In the San Francisco Options Study of men who have sex with men identified within 12 months of HIV seroconversion, oral transmission represented 7.8 percent of primary HIV infections (Dillon et al. 2000). Rothenberg et al. (1998) reviewed epidemiologic studies and reports of 38 cases of oral transmission of HIV in the literature. They concluded that although oral-genital contact may be less efficient than needle-sharing or anal intercourse for the transmission of HIV, its increased use by men who have sex with men (Ostrow and DiFranceisco 1996, Schwarcz et al. 1995) and in crack cocaine smokers (Faruque et al. 1996a,b) may increase its contribution to HIV transmission over time. Several studies provide evidence that when the oral environment is compromised, the mouth can be a potential site of transmission of infectious microbes. Data from Faruque et al. (1996a,b) and Wallace et al. (1996) suggest that there is a positive association between the presence of oral lesions resulting from crack cocaine use, receptive oral intercourse, and HIV transmission. A case report has documented the passage of HIV from a partner who is HIV-positive to one who is HIV-negative in the presence of periodontal disease but in the absence of other risk factors (Padian and Glass 1997). Because the type, duration, and frequency of oral contact in past studies may not have been specified, the risk could be somewhat higher for oral transmission of HIV than previously reported. The risk might also vary depending on factors such as viral load, infectious dose, area of exposure, and presence or absence of oral lesions. Additional studies are needed to evaluate the risk of oral-genital transmission of HIV; some are under way (J. Greenspan, K. Page-Schafer, personal communication, 1999).

Other sexually transmitted diseases (STDs) can occur through oral contact. For example, pharyngeal infection with *Chlamydia trachomatis* has been found in 3 to 6 percent of men and women attending STD clinics. Most infections are asymptomatic (Holmes et al. 1999). Another common sexually transmitted infection, herpes simplex virus, commonly infects the pharynx and is seen in 20 percent of patients with primary genital herpes. The painless chancre of primary syphilis can be found in the oral cavity; however, there are no data on the prevalence of this site of infection for *Treponema pallidum*. Among persons with gonorrhea, pharyngeal infection occurs in 3 to 7 percent of heterosexual men, 10 to 20 percent of heterosexual women, and 10 to 25 percent of men who have sex with men (Holmes et al. 1999). Gonococcal

infection can cause acute pharyngitis, but is usually asymptomatic. The transmission of pharyngeal gonorrhea to sex partners had been thought to be rare. However, in one study, 17 of 66 men who had sex with men who had urethral gonorrhea reported insertive oral sex as their only risk factor in the past 2 months (Lafferty et al. 1997).

Conclusion

The role of the mouth as a portal of entry for infection presents ever-new challenges for study. Although oral tissues and fluids normally provide significant barriers and protection against microbial infections, at times these infections can not only cause local disease but, under certain circumstances, can disseminate to cause infections in other parts of the body. The control of existing oral infections is clearly of intrinsic importance and a necessary precaution to prevent systemic complications.

ASSOCIATIONS AMONG ORAL INFECTIONS AND DIABETES, HEART DISEASE/STROKE, AND ADVERSE PREGNANCY OUTCOMES

Recent studies have reported associations between oral infections—primarily periodontal infections—and diabetes, heart disease and stroke, and adverse pregnancy outcomes, but sufficient evidence does not yet exist to conclude that one leads to the other. This section characterizes the nature of these associations by describing the quality of the evidence supporting the reports. Both observational and experimental studies were accepted as admissible evidence. Table 5.3 presents the hierarchy of evidence used to interpret these associations. Where there are operative mechanisms proposed that support an association between oral infectious agents and the systemic conditions in question, they are introduced at the outset. These are followed by animal studies and then by epidemiologic or population-based studies. The evidence for each association is presented in the table in rank order according to the rigor of the study design.

The Periodontal Disease–Diabetes Connection

There is growing acceptance that diabetes is associated with increased occurrence and progression of periodontitis—so much so that periodontitis has been called the “sixth complication of diabetes” (Løe 1993).

The risk is independent of whether the diabetes is type 1 or type 2. Type 1 diabetes is the condition in which the pancreas produces little or no insulin. It usually begins in childhood or adolescence. In type 2 diabetes, secretion and utilization of insulin are impaired; onset is typically after age 30. Together, these two types of diabetes affect an estimated 15.7 million people in the United States and represent the seventh leading cause of death (NIDDK 1999). The goal of diabetic care is to lower blood glucose levels to recommended levels. Some investigators have reported a two-way connection between diabetes and periodontal disease, proposing that not only are diabetic patients more susceptible to periodontal disease, but the presence of periodontal disease affects glycemic control. This section explores the bidirectional relationship, beginning with the effects of diabetes on periodontal disease.

Effects of Diabetes on Periodontitis Prevalence and Severity

Several reviews have described candidate mechanisms to explain why individuals with diabetes may be more susceptible to periodontitis (Grossi and Genco 1998, Manouchehr-Pour and Bissada 1983, Murrah 1985, Oliver and Tervonen 1994, Salvi et al. 1997, Wilton et al. 1988). These include vascular changes, alterations in gingival crevicular fluid, alterations in connective tissue metabolism, altered host immunological and inflammatory response, altered subgingival microflora, and hereditary patterns. Studies were classified by type of diabetes and age of study population (see Table 5.4).

Type 1 Diabetes. Ten reports focused principally on children and adolescents with type 1 diabetes, comparing them with groups of similar ages without diabetes (Cianciola et al. 1982, de Pommereau et al. 1992, Faulconbridge et al. 1981, Firatli 1997, Firatli et al. 1996, Goteiner et al. 1986, Harrison and Bowen 1987, Novaes et al. 1991, Pinson et al. 1995, Ringelberg et al. 1977). All but one of the studies (Goteiner et al. 1986) reported greater prevalence, extent, or severity of at least one measure or index of periodontal disease (e.g., gingival inflammation, probing pocket depth, loss of periodontal attachment, or radiographic evidence of alveolar bone loss) among subjects with diabetes, even though these investigations were conducted in a variety of countries across several continents.

Another set of studies on the relationship between type 1 diabetes and periodontal disease included subjects with and without diabetes between the ages of 15 and 35 (Cohen et al. 1970, Galea et al. 1986, Guven et al. 1996, Kjellman et al. 1970, Rylander et al. 1987, Sznajder et al. 1978). All six studies reported greater prevalence, extent, or severity of at least one measure or index of periodontal disease.

A third set of studies conducted in Scandinavia looked at the relationship between periodontal disease and type 1 diabetes (or diabetes reported as requiring insulin therapy without specification of diabetes type) in adults between 20 and 70 years old. Three of the four studies were cross-sectional (Glavind et al. 1968, Hugoson et al. 1989, Thorstensson and Hugoson 1993), and one was a treatment follow-up study (Tervonen and

TABLE 5.3
Hierarchy of evidence used in analyzing and interpreting results

Quality of Evidence

- I: Evidence obtained from at least one properly randomized controlled trial.
- II-1: Evidence obtained from well-designed controlled trials without randomization.
- II-2: Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group.
- II-3: Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments (such as the results of the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence.
- III: Opinions of respected authorities, based on clinical experience; descriptive studies and case reports; or reports of expert committees.

Strength of Recommendation

- A: There is good evidence to support the recommendation.
- B: There is fair evidence to support the recommendation.
- C: There is insufficient evidence to recommend for or against, but recommendations may be made on other grounds.
- D: There is fair evidence to support the recommendation that the intervention be excluded.
- E: There is good evidence to support the recommendation that the intervention be excluded.

Source: Adapted from U.S. Preventive Services Task Force 1996.

TABLE 5.4

Summary of studies of the association between diabetes and periodontal diseases, classified by strength of evidence, diabetes type, and age group

	Country	Study Design	Diabetes Type ^a	Number of Subjects a. Diabetes b. Control	Ages ^b a. Diabetes b. Control	Measure of Periodontal Disease Status: Diabetes Effect ^c	Other Diabetes-Related Variables Considered	Evidence Level ^d
Firatli 1997	Turkey	Prospective	1	a. 44 b. 20	a. 12.2 (mean) b. 12.3 (mean)	Ging: 0s Ppd: 0s Lpa: 1s	Glycemic control Duration of diabetes	II-2
Cohen et al. 1970	USA	Prospective	1*	a. 21 b. 18	a. 18, 35 b. 18, 35	Ging: 1s Lpa: 1r, 1s	None	II-2
Tervonen and Karjalainen 1997	Finland	Prospective	1	a. 36 b. 10	a. 24, 36 b. 24, 36	Ging: 0e Ppd: 1r Lpa: 1e	Glycemic control Duration of diabetes Diabetes complications	II-2
Novaes et al. 1996	Brazil	Prospective	2	a. 30 b. 30	a. 30, 77 b. 30, 67	Ppd: 1s, 1r Lpa: 1s, 1r	Glycemic control	II-2
Nelson et al. 1990	USA	Prospective	2	a. 720 b. 1,553	a. 15, 55+ b. 15, 55+	XRBL: 1i, 1p	None	II-2
Taylor et al. 1998a	USA	Prospective	2	a. 24 b. 338	a. 15, 57 b. 15, 57	XRBL: 1i, 1r	None	II-2
Taylor et al. 1998b	USA	Prospective	2	a. 21 b. 338	a. 15, 49 b. 15, 49	XRBL: 1i, 1r	Glycemic control	II-2
Goteiner et al. 1986	USA	Cross-sectional	1	a. 169 b. 80	a. school ages b. 5, 18	Ging: 0s Lpa: 0p, 0s PDI: 0s	None	III
Harrison and Bowen 1987	USA	Cross-sectional	1	a. 30 b. 30	a. 4, 19 b. 4, 19	Ging: 1s Lpa: 1p	Glycemic control	III
Novaes et al. 1991	Brazil	Cross-sectional	1	a. 30 b. 30	a. 5, 18 b. 5, 18	Ging: 1s Ppd: 0s XRBL: 1s	None	III
Cianciola et al. 1982	USA	Cross-sectional	1	a. 263 b. 208	a. <10, >19 b. <10, >19	Ging: 1p Lpa: 1p XRBL: 1p, 1s JPS: 1p, 1s	Duration of diabetes	III
de Pommereau et al. 1992	France	Cross-sectional	1	a. 85 b. 38	a. 12, 18 b. 12, 18	Ging: 1e Lpa: 0e, 0p, 0s XRBL: 0e, 0p, 0s	Glycemic control Duration of diabetes	III
Ringelberg et al. 1977	USA	Cross-sectional	1	a. 56 b. 41	a. 10, 16 b. 10, 12	Ging: 1s MGI: 1s	None	III
Firatli et al. 1996	Turkey	Cross-sectional	1	a. 77 b. 77	a. 12.5 (mean) b. 12.6 (mean)	Ging: 0s Ppd: 1s Lpa: 1s	Duration of diabetes	III
Pinson et al. 1995	USA	Cross-sectional	1	a. 26 b. 24	a. 7-18 b. 7-18	Ging: 1s Ppd: 0s Lpa: 0s	Glycemic control Duration of diabetes	III
Faulconbridge et al. 1981	England	Cross-sectional	1	a. 94 b. 94	a. 5, 17 b. 5, 17	Ging: 1s	Duration of diabetes	III
Kjellman et al. 1970	Sweden	Cross-sectional	1*	a. 105 b. 52	a. 15, 24 b. 15, 24	Ging: 1e Ppd: 0s XRBL: 0s	Glycemic control Diabetes complications	III
Guyen et al. 1996	Turkey	Cross-sectional	1	a. 10	a. 18, 27	Ging: 1e	None	III

(continues)

TABLE 5.4 continued

	Country	Study Design	Diabetes Type ^a	Number of Subjects a. Diabetes b. Control	Ages ^b a. Diabetes b. Control	Measure of Periodontal Disease Status: Diabetes Effect ^c	Other Diabetes-Related Variables Considered	Evidence Level ^d
Rylander et al. 1987	Sweden	Cross-sectional	1	a. 46 b. 41	a. 18, 26 b. 19, 25	Ging: 1e, 1p Ppd: 0e Lpa: 1e, 1p XRBL: 0p	Diabetes complications	III
Sznajder et al. 1978	Argentina	Cross-sectional	1*	a. 20 b. 26	a. 9, 29 b. 9, 29	Ging: 1s Lpa: 0s	None	III
Galea et al. 1986	Malta	Cross-sectional	1*	a. 82 b. unknown	a. 5, 29 b. 5, 29	Ppd: 1p	Glycemic control Duration of diabetes Diabetes complications	III
Hugoson et al. 1989	Sweden	Cross-sectional	1	a. 154 b. 77	a. 20, 70 b. 20, 70	Ging: 1e Ppd: 1e, 1p, 1s XRBL: 1s	Duration of diabetes	III
Glavind et al. 1968	Denmark	Cross-sectional	1*	a. 51 b. 51	a. 20, 40 b. 20, 40	Ging: 0s Ppd: 0s Lpa: 1s XRBL: 1s	Duration of diabetes Diabetes complications	III
Thorstensson and Hugoson 1993	Sweden	Cross-sectional	1	a. 117 b. 99	a. 40, 70 b. 40, 70	Ging: 0e Ppd: 1e, 1s XRBL: 1s	Duration of diabetes Onset age	III
Morton et al. 1995	Mauritius	Cross-sectional	2	a. 24 b. 24	a. 26, 76 b. 25, 73	Ging: 1p Ppd: 1s Lpa: 1s	None	III
Shlossman et al. 1990	USA	Cross-sectional	2	a. 736 b. 2,483	a. 5, 45+ b. 5, 45+	Lpa: 1p XRBL: 1p	None	III
Emrich et al. 1991	USA	Cross-sectional	2	a. 254 b. 1,088	a. 15, 55+ b. 15, 55+	Lpa: 1p, 1s XRBL: 1p, 1s	None	III
Wolf 1977	Finland	Cross-sectional	1,2	a. 186 b. 156	a. 16, 60 b. 16, 60	Ging: 1s Lpa: 1s XRBL: 1s	Glycemic control Duration of diabetes Diabetes complications	III
Benveniste et al. 1967	USA	Cross-sectional	1,2*	a. 53 b. 71	a. 5, 72 b. 5, 72	Ging: 0s Ppd: 0p, 0s	None	III
Finestone and Boorjy 1967	USA	Cross-sectional	1,2*	a. 189 b. 64	a. 20, 79 b. 20, 79	PI: 1s	Glycemic control Duration of diabetes Diabetes complications	III
Belting et al. 1964	USA	Cross-sectional	1,2*	a. 78 b. 79	a. 20, 79 b. 20, 79	PI: 1s	Diabetes severity	III
Oliver and Tervonen 1993	USA	Cross-sectional	1,2	a. 114 b. 15,132	a. 20, 64 b. 20, 64	Ppd: 1e, 1p Lpa: 1e, 0p, 0s	None	III
Yavuzilmaz et al. 1996	Turkey	Cross-sectional	1,2	a. 17 b. 17	a. 25, 74 b. 19, 29	Ppd: 1s	None	III
Bridges et al. 1996	USA	Cross-sectional	1,2	a. 118 b. 115	a. 24, 78 b. 24, 78	Ging: 0s Ppd: 0s Lpa: 1s	Glycemic control Duration of diabetes	III
Sandler and Stahl 1960	USA	Cross-sectional	1,2*	a. 100 b. 3,894	a. 20, 69 b. 20, 69	PDR: 1e	None	III
Bacic et al. 1988	Yugoslavia	Cross-sectional	1,2	a. 222 b. 189	a. <20, 60+ b. <20, 60+	Ppd: 1e, 1p, 1s	Glycemic control Duration of diabetes Diabetes complications	III
Hove and Stallard 1970	USA	Cross-sectional	1,2*	a. 28 b. 16	a. 20, 40+ b. 20, 40+	Ging: 0s Ppd: 0s XRBL: 0s	Duration of diabetes Diabetes severity	III

	Country	Study Design	Diabetes Type ^a	Number of Subjects a. Diabetes b. Control	Ages ^b a. Diabetes b. Control	Measure of Periodontal Disease Status: Diabetes Effect ^c	Other Diabetes-Related Variables Considered	Evidence Level ^d
Mackenzie and Millard 1963	USA	Cross-sectional	9	a. 124 b. 92	a. 32, 78 b. 32, 78	XRBL: 0s	None	III
Sznajder et al. 1978	Argentina	Cross-sectional	9	a. 63 b. 39	a. 30, 49 b. 30, 50	Ging: 1s Lpa: 1s	None	III
Dolan et al. 1997	USA	Cross-sectional	9	Weighted a. 107 b. 554	a. 45, 75+ b. 45, 75+	Lpa: 1e, 1p, 1s	None	III
Grossi et al. 1994	USA	Cross-sectional	9	a. 1,426 b. 69	All: 25, 74; unknown for diabetes	Lpa: 1s, 1p	None	III
Tervonen and Knuutila 1986	Finland	Cross-sectional	9	a. 50 b. 53	a. <30, 40+ b. <30, 40+	Ging: 1e Ppd: 1e, 1p XRBL: 0s	Glycemic control	III
Campbell 1972	Australia	Cross-sectional	9	a. 70 b. 102	a. 17, 39 b. 17, 39	PI: 1p, 1s	None	III
Albrecht et al. 1988	Hungary	Cross-sectional	9	a. 1,360 b. 625	a. 15, 65+ b. 15, 65+	Ging: 1s PI: 0s	None	III
Szpunar et al. 1989 (NHANES I)	USA	Cross-sectional	9	a. 474 b. 15,174	a. 6, 65+ b. 6, 65+	PI: 1s	None	III
Szpunar et al. 1989 (HHANES)	USA	Cross-sectional	9	a. 322 b. 8,040	a. 15, 65+ b. 12, 65+	PI: 1s	None	III

^aDiabetes type: 1 = type 1 diabetes mellitus; 2 = type 2 diabetes mellitus; 1,2 = both type 1 and type 2 diabetes mellitus; 9 = diabetes type not specified and not clearly ascertainable from other information in the report; * = diabetes type not specified but ascertained by reviewer from other information in the report.

^bAges: subjects' ages presented as minimum, maximum reported for those with diabetes and controls unless otherwise specified.

^cMeasure of periodontal disease status. Measures used include Ging = gingivitis or gingival bleeding; Ppd = probing pocket depth; Lpa = loss of periodontal attachment; XRBL = radiographic bone loss; JPS = juvenile periodontal score; MGI = modified gingival index; PI = Russell's periodontal index; PDR = periodontal disease rate (proportion of teeth affected by periodontal disease). The number following the measure corresponds to greater disease in those with diabetes (1) or no difference between those with diabetes and controls (0). The letter following the number corresponds to the parameter(s) assessed in the study: e = extent; i = incidence; p = prevalence; s = severity; r = progression.

^dLevels of evidence are delineated in Table 5.3.

Karjalainen 1997). All four studies reported greater prevalence, extent, or severity of at least one measure of periodontal disease.

Type 2 Diabetes. There are fewer reports on the relationship between type 2 diabetes and periodontal disease, particularly where type 2 diabetes is explicitly identified or discernible from the ages of subjects. Seven studies limited to subjects with type 2 diabetes included a comparison group without diabetes. Two of these studies included only adult subjects (Morton et al. 1995, Novaes et al. 1996); the remaining five were large population-based studies of diabetes and periodontal disease in Pima Indians, a group with the highest known prevalence of type 2 diabetes in the world. The Pima Indian studies included subjects aged 5 years and older (Shlossman et al. 1990) or 15 and older (Emrich et al. 1991, Nelson et al. 1990,

Taylor et al. 1998a,b). All seven studies reported greater prevalence, extent, or severity of periodontal disease among subjects with diabetes for at least one measure of periodontal disease. Three of these studies were longitudinal (Nelson et al. 1990, Taylor et al. 1998a,b) and showed that the progression of periodontal disease was greater in diabetes patients than in individuals without diabetes.

In addition to finding significant differences in various measures of periodontal status between subjects with and without type 2 diabetes, a number of these reports also provide estimates of association and risk. Using periodontal attachment loss as the measure, Emrich et al. (1991) estimated that people with type 2 diabetes were 2.8 times more likely to have destructive periodontal disease (odds ratio, 2.8; 95 percent CI, 1.9 to 4.1). When they used radiographic bone loss as the measure and controlled for

other important covariates, the estimate rose to 3.4 (odds ratio, 3.4; 95 percent CI, 2.3 to 5.2). Nelson et al. (1990) quantified the increased risk of advanced periodontal disease in Pima Indians with and without type 2 diabetes, finding the prevalence of periodontal disease in subjects with diabetes to be 2.6 times greater (95 percent CI, 1.0 to 6.6) than that of subjects without diabetes. Taylor et al. (1996), in another analysis of data from the Pima Indians, reported that type 2 diabetes was a significant risk factor for more severe alveolar bone loss progression (odds ratio, 4.2; 95 percent CI, 1.8 to 9.9), in addition to being a significant risk factor for the prevalence of alveolar bone loss.

Studies of Individuals with Type 1 or Type 2 Diabetes. Twelve reports included analyses in which subjects with type 1 and type 2 diabetes were not separated. All of these studies were cross-sectional and included adults; two studies included children or adolescents as well (Benveniste et al. 1967, Wolf 1977). Nine of the 12 studies reported greater prevalence, extent, or severity of periodontal disease among the diabetic subjects for at least one measure or index of periodontal disease (Bacic et al. 1988, Belting et al. 1964, Bridges et al. 1996, Finestone and Boorujy 1967, Hove and Stallard 1970, Oliver and Tervonen 1993, Sandler and Stahl 1960, Yavuzylmaz et al. 1996, Wolf 1977).

Hove and Stallard (1970) and Benveniste et al. (1967) did not find significant differences in periodontal disease between subjects with and without diabetes. The Hove and Stallard report included 28 subjects with diabetes and 16 without diabetes and may not have had enough statistical power to detect clinical differences, although they were able to detect a significantly higher prevalence of gingival vascular changes in subjects with diabetes. Benveniste et al. (1967) commented that their results may have been influenced by use of relatives without diabetes as the comparison group and that the subjects with diabetes were all under reasonably good control with either insulin or dietary regulation. Both factors may have minimized differences between the groups.

Diabetes Type Not Specified. The final set of reports on the association between diabetes and periodontal diseases consists of seven cross-sectional studies in which the type of diabetes was not specified and was not easily determined from other information provided. Four of the seven studies included only adults (Dolan et al. 1997, Grossi et al. 1994, Mackenzie and Millard 1963, Tervonen and Knuuttila 1986). In the other three studies, subjects ranged in age from

childhood to older adulthood (Albrecht et al. 1988, Campbell 1972, Szpunar et al. 1989). Szpunar et al. (1989) presented analyses of two separate national surveys (the National Health and Nutrition Examination Survey, NHANES I, conducted between 1971 and 1974, and the Hispanic Health and Nutrition Examination Survey, HHANES, conducted between 1982 and 1984).

All seven studies found subjects with diabetes to have increased prevalence, extent, and severity of periodontal disease. The statistical significance of the diabetes effect was markedly diminished in the final linear regression model used by Szpunar et al. (1989) in analysis of the NHANES I data. Two of the population-based surveys, Grossi et al. (1994) and Dolan et al. (1997), provided epidemiologic estimates of the association of diabetes and attachment loss severity with odds ratios of 2.3 (95 percent CI, 1.2 to 4.6) and 1.9 (95 percent CI, 1.3 to 3.0), respectively, while controlling for other covariates.

Conclusion. Diabetes is a risk factor for the occurrence and prevalence of periodontal diseases. Although there is insufficient evidence of a causal association, the findings of greater prevalence, severity, or extent of at least one manifestation of periodontal disease in individuals with diabetes is remarkably consistent in the overwhelming majority of studies. Furthermore, there are no studies with superior design features in the literature to refute this assessment. The studies were conducted in distinctly different settings, with subjects from different ethnic populations and of different ages, and with a variety of measures of periodontal status. This inevitable variation in methodology and study populations limits the possibility that the same biases apply in all the studies. There is a need for further research using stronger designs that also control for confounding variables such as socioeconomic status.

Glycemic Control

Several lines of evidence support the plausibility that periodontal infections contribute to problems with glycemic control, thus compromising the health of diabetic patients. It has been reported that the chronic release of tumor necrosis factor alpha (TNF- α) and other cytokines such as those associated with periodontitis interferes with the action of insulin and leads to metabolic alterations (Hotamisligil et al. 1993, Flier 1993). Other studies have noted relationships between insulin resistance and active inflammatory connective tissue diseases (Hallgren and Lundquist 1983, Svenson et al. 1987), other clinical diseases (Beck-Nielsen 1992, Beisel 1975), acute

infection (Drobny et al. 1984, Sammalkorpi 1989), and periodontal disease (Grossi et al. 1999). Grossi and Genco (1998) have proposed a model whereby periodontal infection contributes to hyperglycemia and complicates metabolic control in diabetes.

Clinical Studies. The effects of periodontal infection on glycemic control have been investigated in a small number of clinical studies that looked at metabolic control at baseline and following various periodontal treatments (see Table 5.5; Aldridge et al. 1995, Christgau et al. 1998, Grossi et al. 1996, 1997, Miller et al. 1992, Seppala and Ainamo 1994, Seppala et al. 1993, Smith et al. 1996, Westfelt et al. 1996, Williams and Mahan 1960, Wolf 1977). One report is based on an epidemiological cohort study (Taylor et al. 1996).

The randomized controlled trials of Grossi et al. (1997) involving populations with type 2 diabetes found that use of the systemic antibiotic doxycycline to treat periodontitis patients with diabetes resulted in a transient (3 to 6 months) improvement in glycemic control. On the other hand, the two controlled trials conducted in London by Aldridge et al. (1995) involving patients with type 1 diabetes found no effect. Taken together, these three studies provide inconsistent results and are limited in how well they generalize to broader populations. A small uncontrolled study of 10 patients by Miller et al. (1992) also reported an improvement in glycemic control of diabetic patients whose periodontal disease was treated with mechanical therapy and systemic doxycycline. Five of the above-mentioned studies did not include control groups (Miller et al. 1992, Seppala et al. 1993, Smith et al. 1996, Williams and Mahan 1960, Wolf 1977), and four were not specifically designed to address the relationship between periodontal therapy and glycemic control (Grossi et al. 1996, Seppala et al. 1993, Smith et al. 1996, Westfelt et al. 1996), although the data collected allowed the investigators to address the issue. One nonrandomized but controlled clinical trial of nonsurgical periodontal therapy found no significant influence on medical data for the diabetic patients (Christgau et al. 1998). A clear relationship between improvement in periodontal health and glycemic control has not been shown. The studies seem to suggest that antibiotic treatments may help in glycemic control. A recent longitudinal study indicates inflammation may be a precursor to the onset of type 2 diabetes (Schmidt et al. 1999). Thus periodontal infection may contribute to that inflammation.

Conclusion. The body of literature concerning the relationship between periodontal infection and

impaired glycemic control is varied in the strength, quantity, breadth, and consistency of evidence presented. The preliminary evidence, while encouraging, does not support a clear-cut conclusion that treating periodontal infection can contribute to management of glycemic control in type 1 or type 2 diabetes. As the table indicates, only studies using systemic antibiotic treatment affected glycemic control favorably. The results suggest that infections other than periodontitis may be implicated or that intensive treatment of periodontal infections with systemic antibiotics is necessary to affect glycemic control favorably. Further rigorous controlled studies in diverse populations are warranted.

The Oral Infection–Heart Disease and Stroke Connection

During the past decade, infectious agents have become recognized as causes of systemic diseases, without fever or other traditional signs of infection. *Helicobacter pylori* is associated with peptic ulcers and, along with *Chlamydia pneumoniae* and cytomegalovirus, is now thought to be associated with increased risk for cardiovascular disease as well as malignancies (Wu et al. 2000). Studies investigating the relationship between oral and dental infections and the risk for cardiovascular disease suggest that there is potential for oral microorganisms, such as periodontopathic bacteria, and their effects to be linked with heart disease.

Mechanisms of Action

Infectious agents are thought to affect the risk of heart disease through several possible mechanisms. Bacteria or viruses originating in tissues such as the lungs or oral mucosa may directly infect blood vessel walls. Such infection may be largely asymptomatic, but may cause local vascular inflammation and injury, which would contribute to the development of lipid-rich plaques and atherosclerosis. Bacteria or viruses may also interact with white blood cells or platelets, both of which integrate into the developing atherosclerotic plaque. Cells of the blood vessel wall and white blood cells and platelets can release prostaglandins (especially PGE₂), interleukins (especially IL-1), thromboxane B₂ (TBX₂), and tumor necrosis factor alpha (TNF- α). Bacterial products in the blood may also stimulate liver production of other pro-inflammatory or pro-coagulant molecules such as C-reactive protein and fibrinogen. Microbes may also stimulate expression of tissue factor, which would activate coagulation. During the process of

TABLE 5.5
Effects of periodontal disease and its treatment on glycemic control: clinical and epidemiological evidence

Study Design ^a	Diabetes Type	Number of subjects a. Treatment (ages) b. Control (ages)	Follow-up Time	Periodontal Therapy	Metabolic Control Outcome	Effects on Metabolic Control	Evidence Level ^b
Aldridge et al. 1995, Study 1	RCT	Type 1 a. 16 (16-40) b. 15 (16-40)	2 months	Experimental group: oral hygiene instruction, scaling, adjustment of restoration margins, and reinforcement after 1 month; control group: no treatment	Glycated hemoglobin, fructosamine	Periodontal treatment had no effect on change in glycated hemoglobin.	I
Aldridge et al. 1995, Study 2	RCT	Type 1 a. 12 (20-60) b. 10 (20-60)	2 months	Experimental group: oral hygiene instruction, scaling and root planing, extractions, root canal therapy; control group: no treatment	Glycated hemoglobin	Periodontal treatment had no effect on change in glycated hemoglobin.	I
Grossi et al. 1996, 1997	RCT	Type 2 a. 89 (25-65) b. 24 (25-65)	12 months	Experimental groups received either systemic doxycycline or placebo and ultrasonic bactericidal curettage with irrigation using either water, chlorhexidine, or povidone-iodine	Glycated hemoglobin	The three groups receiving doxycycline and ultrasonic bacterial curettage showed significant reductions ($P < 0.04$) in mean glycated hemoglobin at 3 months.	I
Christgau et al. 1998	Treatment study, non-RCT	Type 1 and type 2 a. 20 (30-66) b. 20 (30-66)	2 months	Scaling/root planing; subgingival irrigation with chlorhexidine; oral hygiene instruction; and extractions	Glycated hemoglobin	No effect on glycated hemoglobin.	II-1
Westfelt et al. 1996	Treatment study, non-RCT	Type 1 and type 2 a. 20 (45-65) b. 20 (45-65)	5 years	Baseline oral hygiene instruction, scaling and root planing followed by periodic prophylaxis, oral hygiene instruction, localized subgingival plaque removal, and surgery at sites with bleeding on probing and a periodontal probing depth of >5 mm	Glycated hemoglobin	"The mean value of HbA1c between baseline and 24 months was not significantly different from that between 24 and 60 months."	II-1
Smith et al. 1996	Treatment study, non-RCT	Type 1 a. 18 (26-57) b. 0	2 months	Scaling and root planing with ultrasonics and cures; oral hygiene instruction	Glycated hemoglobin	Found no statistically or clinically significant change in glycated hemoglobin.	II-1
Taylor et al. 1996	Historical prospective cohort	Type 2 No treatment or control subjects 49 (severe periodontitis) 56 (less severe periodontitis)	2-4 years	Not applicable	Glycated hemoglobin	Those with severe periodontitis were ~6 times more likely to have poor glycemic control at follow-up.	II-2
Miller et al. 1992	Treatment study, non-RCT	Type 1 a. 10 (not given) b. 0	8 weeks	Scaling and root planing, systemic doxycycline	Glycated hemoglobin, glycated albumin	Found decrease in glycated hemoglobin and glycated albumin in patients with improvement in gingival inflammation ($P < 0.01$). Patients with no improvement in gingival inflammation had either no change or increase in glycated hemoglobin post treatment.	III

coagulation, platelets would become trapped in the growing clot or thrombus. Microthrombus formation is one of the key factors in the development of atherosclerotic plaques. As atherosclerotic plaques enlarge,

the lumen of the coronary blood vessels narrows and the blood supply to the heart muscle becomes reduced. A frank heart attack or myocardial infarction results when a larger part of the coronary artery

Study Design ^a	Diabetes Type	Number of subjects a. Treatment (ages) b. Control (ages)	Follow-up Time	Periodontal Therapy	Metabolic Control Outcome	Effects on Metabolic Control	Evidence Level ^b
Seppala et al. 1993, Seppala and Ainamo 1994	Type 1	a. 38 for 1 year; 22 for 2 years; 26 PIDD-1y (48 ± 6) 12 CIDD-1y (43 ± 5) 16 PIDD-2y 6 CIDD-2y b. 0	2 years	Scaling and root planing, periodontal surgery, and extractions	Medical history for baseline control status; glycosylated hemoglobin A1 and blood glucose for assessing response to treatment	Reported an improvement of the HBA1 levels of the PIDD and CIDD subjects ($P < 0.068$, <i>t</i> -test).	III
Williams and Mahan 1960	Not specified	a. 9 (20-32) b. 0	3-7 months	Extractions, scaling and curettage, gingivectomy, systemic antibiotics	Insulin requirement; diabetes control (not operationally defined)	7 of 9 subjects had "significant" reduction in insulin requirements.	III
Wolf 1977	Type 1 and type 2	a. 117 (16-60) b. 0	8-12 months	Scaling and home care instruction, periodontal surgery, extractions, endodontic treatment, restorations, denture replacement or repair	Blood glucose, 24-h urinary glucose, insulin dose	Compared 23 subjects with improved oral infections with 23 who had no improvement after treatment for oral infection and inflammation. The subjects with improved oral inflammation and infection tended to demonstrate diabetes control improvement ($P < 0.1$). However, Wolf states in discussion, "treatment of periodontal inflammation and periapical lesions . . . does little to improve the control of diabetes."	III

^a RCT = randomized controlled trial.
^b Levels of evidence are delineated in Table 5.3. Note that because this body of literature is small, this review does not distinguish between "well-designed" studies and otherwise in assigning the evidence levels.
^c PIDD = poorly controlled insulin-dependent diabetes; CIDD = controlled insulin-dependent diabetes.

lumen becomes occluded. Failing to receive enough blood, the heart muscle dies, resulting in an infarct.

Animal Studies

Bacteria originating in the oral cavity may also contribute to platelet clotting or thrombosis, as proposed by Herzberg et al. (1983, 1994). These investigators have suggested that the association between periodontal disease and cardiovascular disease may be due in part to the potential for oral bacteria such as *S. sanguis* and *P. gingivalis* to induce platelet aggregation. Platelets aggregate in response to these bacteria as a result of mistaken identity: a protein structure on the surface of certain common strains of *S. sanguis* and *P. gingivalis* mimics the platelet-interactive regions of collagen molecules (Erickson and Herzberg 1993, Herzberg et al. 1994). Exposure of flowing blood to collagen triggers clotting, the cen-

tral event in stopping blood flow. When an experimental bacteremia was created with a strain of *S. sanguis* that carried the collagen-like protein, rabbits showed changes in blood pressure, electrocardiogram readings, heart rate, and cardiac contractility (Herzberg and Meyer 1996, 1998). Platelets also aggregated in the circulation, resulting in significant declines in platelet counts. From the electrocardiographic tracings, rabbit heart muscle also appeared to have suffered ischemic damage. The investigators concluded that oral bacteria carrying the collagen-like protein induced platelet aggregation or clotting in the bloodstream. These clots were of sufficient size to obstruct coronary arteries and produce ischemic heart damage, an early warning sign of a heart attack or an infarction. Because *S. sanguis* is present in large numbers in dental plaque and is a causative agent in infective endocarditis, it is likely that these bacteria

have an opportunity to induce platelet clotting during human bacteremias from oral sources. Bacteria-induced platelet clotting could contribute to microthrombosis during the development of atherosclerotic plaques and occlusive thrombus formation with occasional myocardial infarction.

Population-based Studies

Any study investigating the possibility of a unique role for oral pathogens as risk factors for cardiovascular disease, including atherosclerosis and the formation of a blood clot in a coronary artery of the heart, which typically precedes myocardial infarction, must take into consideration such known risk factors as smoking, hypertension, obesity, diabetes, genetic susceptibility, and elevated cholesterol. Genco (1998) and Beck et al. (1998) have recently reviewed studies examining the associations between oral conditions (including periodontitis) and atherosclerosis and coronary heart disease, the latter of which affects 12 million people in the United States and is the leading cause of death. These are summarized in Table 5.6.

Of the ten studies cited in the table, six are prospective cohort studies, in which oral health status was established at the outset (baseline) of the study period and the subjects were followed at periodic intervals to a previously defined endpoint, for example, diagnosis of coronary heart disease or an acute myocardial infarction, or death. Beck et al. (1996) combined data from the Veterans Administration Dental Longitudinal Study and its parent longitudinal study, the Normative Aging Study, for a total of 203 cases and 891 noncases, to determine whether periodontal disease, judged by measuring alveolar crestal bone, is a risk factor for cardiovascular disease. After adjusting for age, blood pressure, cholesterol, and body mass index, the investigators found that subjects with periodontal disease were 1.5 times more likely to develop coronary heart disease over a 25-year period than controls (odds ratio of 1.5). Similarly, after adjusting for age, smoking, and blood pressure, the investigators found that veterans with periodontal disease were 1.9 times more likely to develop fatal coronary heart disease (odds ratio of 1.9).

In a longitudinal study to eliminate the potential confounding effects of smoking, Genco et al. (1997) measured the incidence of periodontal disease and cardiovascular disease in 1,372 American Indians of the Gila River Indian Reservation. Although diabetes is prevalent in this population, cigarette smoking is rare in these individuals (a fact confirmed in this study), so it was considered not to be a risk factor for either cardiovascular disease or periodontal disease

in this study. Periodontal disease was measured at baseline, and the incidence of cardiovascular disease was followed over the next 10 years. When the analysis was restricted to individuals under age 60, the risk of cardiovascular disease was 2.7 times higher in subjects with periodontal disease than in those with little or no periodontal infection. This association was seen even after adjusting for other risk factors for cardiovascular disease or periodontal disease such as age, sex, cholesterol, weight, high blood pressure, diabetes, and insulin use. The investigators concluded that periodontal disease is an important risk factor for cardiovascular disease for individuals under 60 in this group, second only to the presence of long-term diabetes. Further analysis of death due to cardiovascular disease is needed in this population to complete the study.

Mattila and coworkers have conducted both prospective and retrospective studies. A prospective study (Mattila et al. 1995) showed that new episodes of myocardial infarction occurred more frequently in subjects with more extensive "dental" disease. The authors used a measure of dental disease that included a composite index that assessed caries, periodontitis, pericoronitis, and periapical lesions. The composite index estimates the combined infectious load that contributes to many possible oral infections. After combining the prospective study data with data from an earlier retrospective study (Mattila et al. 1989) and adjusting for age, triglyceride levels, cholesterol, C-reactive protein, smoking, social class, diabetes, and hypertension, the investigators found a significant association between dental infections and acute myocardial infarction in men under age 50 ($P < 0.01$). A more recent study (Mattila et al. 2000) compared 85 patients with proven coronary heart disease and 53 matched controls. This case-control study showed that dental indices were higher among coronary heart disease patients than controls, but the differences were not statistically significant. Participants in the study were older, which the authors believed was the most likely reason for the results. In the first National Health and Nutrition Examination Survey, 9,670 subjects were followed for over 14 years. DeStefano et al. (1993) found that there was a 25 percent increased risk of cardiovascular disease in individuals with periodontitis compared with those with minimal periodontal disease. The strongest association was seen in men under 50 (relative risk, 1.7). A limitation of this study, which the authors acknowledge, was the lack of baseline data on smoking, a major risk factor for both periodontal and cardiovascular disease. Morrison et al. (1999) evaluated a retrospective cohort study using participants in the

TABLE 5.6
Summary of studies assessing the association between oral conditions, atherosclerosis, and coronary heart disease

	Study Design ^a	Subjects (cases/controls)	Oral Condition	Cardiovascular Outcome	Adjustments ^b	Association (odds ratio or relative risk)	Evidence Level ^c
DeStefano et al. 1993	Prospective	1,786/7,974	Russell's periodontal index	Coronary heart disease (admission to hospital or death)	Age, sex, race, education, poverty, marital status, cholesterol, BMI, diabetes, smoking	1.72 (1.1-2.68) (only for men under age 50)	II-2
Mattila et al. 1995	Prospective	52/162	Dental index (caries, periodontal disease, pulpal infection)	New acute myocardial infarction or death	Smoking, hypertension, age, sex, triglycerides, socioeconomic status, diabetes, lipids, BMI, previous MI	Yes, $P < 0.01$	II-2
Joshi-pura et al. 1996	Prospective	757/44,119	Periodontal disease (self-reported), tooth loss due to self-reported periodontal disease	New coronary heart disease	Age, BMI, exercise, smoking, alcohol, vitamin C, family history, MI	1.67 (1.03-2.71)	II-2
Beck et al. 1996	Prospective	203/891	Alveolar crestal bone loss	New coronary heart disease, fatal coronary heart disease, stroke	Age, BMI, total cholesterol, socioeconomic status, DBP, LDL, smoking, cholesterol	1.5 (1.04-2.14) 1.9 (1.0-3.43) 2.8 (1.45-5.48)	II-2
Genco et al. 1997	Prospective	68/1,304	Alveolar crestal bone loss	New coronary heart disease	Age, sex, smoking, BMI, diabetes, cholesterol, hypertension	2.68 (1.35-5.60)	II-2
Morrison et al. 1999	Retrospective	10,000	Severe gingivitis; periodontitis; edentulousness	New coronary heart disease; cerebrovascular deaths	Age, sex, serum total cholesterol, smoking, diabetes, hypertension	1.37 (0.80-2.35) for periodontitis 1.90 (1.17-3.10) for edentulousness for fatal CHD	II-2
Joshi-pura et al. 1999	Prospective	42,151	Number of teeth lost	Ischemic stroke	Age, smoking, obesity, alcohol, exercise, aspirin, family history, profession, hypertension, hypercholesterolemia	≤10 teeth 1.75 (1.03-2.99) 11-15 teeth 1.95 (1.07-3.64) 17-24 teeth 1.48 (1.02-2.13)	II-2
Mattila et al. 1989	Case-Control	100/102	Dental index (caries, periodontal disease, pulpal infections)	Acute myocardial infarctions	HDL, smoking, C-reactive protein, hypertension, age, cholesterol, diabetes, social class	Yes, $P < 0.01$	II-2
Grau et al. 1997	Case-Control	166/166	Dental index (caries, periodontal disease, periapical infections)	Stroke	Diabetes, preexisting vascular disease, socioeconomic status, smoking	Odds ratio 2.6 (1.18-5.70)	II-2
Mattila et al. 2000	Case-control	85/53	Dental index (caries, periodontal disease, pulpal infections)	New coronary heart disease	Age, sex, smoking, socioeconomic class, hypertension, number of teeth, serum lipid levels	—	II-2

^aFor the prospective studies, the total number of subjects in the cohort is the sum of the two numbers given, the first number of which represents the subjects followed to the endpoint. For the case-control studies, the first number represents the cases, and the second the controls.

^bBMI = body mass index; MI = myocardial infarction; LDL = low-density lipoproteins; HDL = high-density lipoproteins.

^cLevels of evidence are delineated in Table 5.3.

1970-72 National Canada Survey. In the younger cohort, those under age 69, they found that gingivitis, periodontitis, and edentulousness were related to fatal coronary heart disease in a statistically significant manner. However, in analyzing those over age 70, none of these dental conditions was associated with fatal heart disease. These results were adjusted for age, sex, serum total cholesterol, smoking status, diabetes status, hypertension status, and province of resi-

dence. This pattern of higher risk observed among younger subjects may, to some extent, reflect the relative instability of risk estimates. However, it is also possible that periodontal disease, like other co-morbid relative risks for coronary heart disease, generally declines with age (Semenciw et al. 1988).

Wu et al. (1999) found periodontal disease to be a potential factor for coronary heart disease and stroke based on an analysis of the first National

Health and Nutrition Examination Survey and its 21-year follow-up. In this analysis, periodontitis was found to be a significant risk factor for cerebrovascular disease, in particular nonhemorrhagic stroke. Compared with no periodontal disease, relative risk (95 percent CI) for incident nonhemorrhagic stroke was 2.11 (1.30 to 3.42) for periodontitis. There was no significant relationship for gingivitis or edentulousness, which were 1.24 (0.74 to 2.08) and 1.41 (0.96 to 2.06), respectively. The increased relative risk for total cerebrovascular disease and nonhemorrhagic stroke was not seen for hemorrhagic stroke. Similar relative risks for total cerebrovascular disease and nonhemorrhagic stroke associated with periodontitis were seen in white men and women and African Americans. A conclusive statement about a cause-and-effect relationship between periodontitis and the risk of developing cerebrovascular disease, in particular nonhemorrhagic stroke, cannot be made at this time. The consistency of the findings in different racial groups and the strength of the association warrant further examination of the potentially important association between these two clinical conditions, which are highly prevalent in the adult population.

In the largest cohort studied, Joshipura et al. (1996) found that among a group of male health professionals who were relatively homogeneous socioeconomically and who self-reported preexisting periodontal disease, those with 10 or fewer teeth were at increased risk of new coronary heart disease, compared with those with 25 or more teeth (relative risk, 1.67). These results were adjusted for standard cardiovascular disease risk factors.

In a case-control study of 166 patients with acute cerebrovascular disease and 166 age- and sex-matched controls, Grau et al. (1997) found that "poor dental status" was independently associated with cerebrovascular ischemia. These results were based on a subgroup of patients and controls who completed the dental examination. A modified form of the Total Dental Index was used to measure dental status. In an 8-year follow-up of 42,151 male health professionals who were free of cardiovascular disease at baseline, Joshipura et al. (1999) reported that edentulousness was associated with an increased risk of ischemic stroke after adjusting for age, smoking, obesity, alcohol, exercise, aspirin, family history of cardiovascular disease, profession, hypertension, and hypercholesterolemia.

Conclusion

None of the studies reviewed to date achieves the level of rigor that can unequivocally establish periodontitis as an independent risk factor for cardiovas-

cular disease or stroke. The methods used to measure or identify periodontal disease ranged widely from self-report, to composite indices that included dental caries experience, to precise measures of periodontitis severity. Nevertheless, there were consistent findings of increased odds ratios and significant probability (*P*) values pointing to an association of periodontal and other oral infections with an increased risk for cardiovascular disease. Further studies are needed to determine whether periodontal disease alone or in the presence of other oral infections is an independent risk factor for cardiovascular or cerebrovascular disease. Research to elucidate the underlying pathological mechanisms is also essential. Studies must also clarify the potentially confounding effects of sex, age, socioeconomic level, and race/ethnicity.

Periodontal Disease and Adverse Pregnancy Outcomes

Preterm birth and low birth weight are considered the leading perinatal problems in the United States (Gibbs et al. 1992). Although infant mortality rates have decreased substantially over the past generation, the incidence of low birth weight (just under 300,000 cases in 1995) has not shown a comparable decline (Institute of Medicine 1985, USDHHS 1984). Over 60 percent of the mortality of infants without structural or chromosomal congenital defects can be attributed to low birth weight (Shapiro et al. 1980).

Mechanisms of Action

Periodontal disease may contribute to adverse outcomes of pregnancy as a consequence of a chronic oral inflammatory bacterial infection. Toxins or other products generated by periodontal bacteria in the mother may reach the general circulation, cross the placenta, and harm the fetus. In addition, the response of the maternal immune system to the infection elicits the continued release of inflammatory mediators, growth factors, and other potent cytokines, which may directly or indirectly interfere with fetal growth and delivery.

Evidence of increased rates of amniotic fluid infection, chorioamnion infection, and histologic chorioamnionitis supports an association between preterm birth, low birth weight, and general infection during pregnancy. It is noteworthy that the largest proportion of such infections occurred during the pregnancies of the most premature births (Hillier et al. 1988, 1995). The biological mechanisms involve bacteria-induced activation of cell-mediated immunity leading to cytokine production and the synthesis

and release of prostaglandins, which may trigger preterm labor (Hillier et al. 1988). Elevated levels of prostaglandin as well as cytokines (interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF- α)) have been found in the amniotic fluid of patients in preterm labor with amniotic fluid infection (Romero et al. 1993), compared with levels in patients with preterm labor without infection.

Animal Models

A variety of studies have used the pregnant hamster model. Some investigators have examined the effects of lipopolysaccharide, produced by oral gram-negative pathogens, on cytokine production (Collins et al. 1994a). In other studies, hamsters have been infected with *P. gingivalis*, with or without prior immunization. Collins et al. (1994b) challenged the animals with nondisseminating, low levels of *P. gingivalis*, introduced in a subcutaneous chamber at a dorsolumbar site. Although the doses were insufficient to induce fever or wasting, the hamster litters of the infected animals showed a significant reduction in fetal weight (24 percent) in comparison with control animals ($P < 0.0001$). This suppressive effect on fetal weight was accompanied by a proportional intrachamber rise in tumor necrosis factor alpha (TNF- α) and prostaglandin E₂ (PGE₂) ($P < 0.0001$). Immunization prior to mating did not provide protection from a challenge during pregnancy, but rather potentiated the effects, indicating the potential strength of a chronic infection.

In another series of hamster studies, researchers observed the effects of experimental periodontitis on pregnancy outcomes and amniotic fluid mediators (Offenbacher et al. 1998). The investigators noted a 20 percent decrease in fetal weight ($P = 0.002$). Periodontal infection in the pregnant hamster also was associated with a significant rise in intra-amniotic PGE₂ from 3.31 ± 1.1 to 13.5 ± 4.1 micrograms per milliliter ($P = 0.03$). These data suggest a link between oral infection and changes in the fetal environment.

Epidemiologic Studies

Human case-control studies have demonstrated that mothers of low-birth-weight infants born as a result of either preterm labor or premature rupture of membranes tend to have more severe periodontal disease than mothers with normal-birth-weight infants (Offenbacher et al. 1996, 1998). A case was defined as a mother whose baby weighed less than 2,500 grams and who had one or more of the following factors: gestational age of infant of less than 37 weeks;

preterm labor; or preterm, premature rupture of membranes. Controls were all normal-birth-weight, full-term infants. In a case-control study of 124 subjects, the mean clinical periodontal attachment level for the mothers of low-birth-weight babies was $3.10 + 0.74$ (SD) millimeters (mm) per site (93 subjects) versus $2.80 + 0.61$ (SD) mm per site (31 subjects) for mothers of normal-weight infants ($P = 0.038$ for all cases and controls). For a subset of mothers for whom this was the first child, the mean clinical periodontal attachment level for those with low-birth-weight babies was $2.98 + 0.84$ (SD) mm per site (46 subjects) versus $2.56 + 0.54$ (SD) mm per site for controls (20 subjects) at $P = 0.041$ (Offenbacher et al. 1996). This subset was analyzed separately to avoid the confounding effects of mothers with periodontal disease who had previously given birth to low-birth-weight infants but who later had normal-weight infants.

Logistic regression models demonstrated that severe periodontal disease was associated with a sevenfold increase in risk of low birth weight, after controlling for known risk factors such as smoking, race, alcohol use, age, nutrition, and genitourinary tract infection. This study suggests an association between periodontal disease and prematurity.

In a subsequent case-control study of 44 subjects, additional biochemical and microbial parameters of periodontal disease status were studied to assess the relationship of current periodontal status to current pregnancy outcome (Offenbacher et al. 1998). Results indicate that PGE₂ levels in gingival crevicular fluid were significantly higher in mothers of low-birth-weight infants than in controls ($131.4 + 21.8$ (SE) versus $62.6 + 10.3$ (SE) nanograms per milliliter), respectively (at $P = 0.02$). Furthermore, within the group of mothers of low-birth-weight infants there was a significant inverse association between birth weight, gestational age, and gingival crevicular fluid PGE₂ levels (at $P = 0.023$ for current births). These data suggest that the level of PGE₂ in gingival crevicular fluid, serving as a marker of current periodontal disease activity, varies inversely with birth weight; that is, the higher the PGE₂ level, the lower the birth weights. In this study the periodontal disease was more severe in mothers with adverse pregnancy outcomes, as determined by biochemical and microbial biomarkers, but the difference in clinical attachment levels did not reach statistical significance ($P = 0.11$).

Studies also have been reported in other countries. In the United Kingdom a preliminary analysis of 167 cases and 323 controls did not show an association between periodontal disease and pregnancy

outcomes (Davenport et al. 1998); however, the investigators did not control for confounding factors. Dasayanake (1998) conducted a matched case-control study with 55 cases in Thailand. Gingivitis was associated with a higher risk of having a growth-restricted infant (odds ratio = 0.3; 95 percent CI, 0.12 to 0.72), controlling for mother's height, prenatal care, dental caries status, and the infant's gender. Smoking was not controlled for in this study.

Conclusion

As a remote gram-negative infection, periodontal disease may have the potential to affect pregnancy outcome. Not all the obstetric risk factors that result in babies being born too soon and too small have been fully identified (Gibbs et al. 1992, McCormick 1985). Oral infections have been investigated as a potential risk factor for preterm labor or premature rupture of membranes, which are major obstetric antecedents to spontaneous preterm births. Although the findings from animal research and case-control studies are promising, additional work, including longitudinal studies, research on mechanisms, and intervention trials, is needed to determine whether periodontitis is a risk factor and what the mechanisms of action may be for adverse pregnancy outcomes. In the United States, longitudinal and intervention studies are under way.

Conclusion

This critical look at the emerging associations among oral infections and specific conditions establishes the need for an aggressive research agenda to better understand the specific aspects of these associations and the underlying mechanisms. Prospective and intervention studies are under way and should provide additional and stronger evidence of the presence and direction of an association. It is essential for such studies to include populations at known risk for the underlying conditions as well as the general population. Of the conditions reviewed, the relationship of periodontal disease and diabetes has the strongest evidence, demonstrating that the risk of periodontitis is higher in individuals with diabetes. However, the effect of periodontitis on glycemic control is less clear, a reflection of the difficulty of controlling for the effect of systemic antibiotic treatments used to manage periodontal disease in diabetic patients in clinical trials.

IMPLICATIONS OF THE LINKAGES

This review of oral health linkages with general health reveals implications for the clinical practice of both medicine and dentistry. The recognition of well-known and established signs and symptoms of oral diseases may assist in the early diagnosis and prompt treatment of some systemic diseases and disorders. The presence of these signs also may lead to the institution of enhanced disease prevention and health promotion procedures. All health professionals, and the public, should be aware of these signs and symptoms. Individuals, practitioners, and community programs may also benefit from the accelerated development and testing of readily accessible, acceptable, and simple oral-based diagnostics.

A better understanding of the role of the oral cavity and its components in protecting against infection is needed. This information should permit the development of interventions to enhance these components. For example, research is under way investigating how to augment some of the natural antimicrobial molecules that are present in saliva and how to use oral and nasal vaccination routes to enhance immunity. Also, host susceptibility factors contributing to the dissemination of oral infections to other parts of the body should be investigated, especially in populations at high risk for disease and infection. In addition, further studies are needed to elucidate the role of the mouth as a means of transmitting infectious microbes. This in turn will allow the development of interventions to prevent transmission and curb the progression of infections once established.

The associations between oral infections and diabetes, heart disease and stroke, and adverse pregnancy outcomes warrant a comprehensive and targeted research effort. If any of these associations prove to be causal, major changes in care delivery and in the training of health professionals will be needed.

Awareness of the oral complications of medications and other therapies for disease management and for health promotion needs to be enhanced among health care professionals, the public, drug manufacturers, and the research community. For some of these therapies, known interventions exist and should be followed before initiating the therapy to minimize or modulate its side effects. To prevent the oral complications of other therapies, new approaches are needed. Ultimately, and ideally, the side effects of therapies should be considered in the development of new drugs and biologics.

FINDINGS

- Many systemic diseases and conditions have oral manifestations. These manifestations may be the initial sign of clinical disease and as such serve to inform clinicians and individuals of the need for further assessment.

- The oral cavity is a portal of entry as well as the site of disease for microbial infections that affect general health status.

- The oral cavity and its functions can be adversely affected by many pharmaceuticals and other therapies commonly used in treating systemic conditions. The oral complications of these therapies can compromise patient compliance with treatment.

- Individuals such as immunocompromised and hospitalized patients are at greater risk for general morbidity due to oral infections.

- Individuals with diabetes are at greater risk for periodontal diseases.

- Animal and population-based studies have demonstrated an association between periodontal diseases and diabetes, cardiovascular disease, stroke, and adverse pregnancy outcomes. Further research is needed to determine the extent to which these associations are causal or coincidental.

REFERENCES

- Adams D, Davies G. Gingival hyperplasia induced by cyclosporine A. A report of two cases. *Br Dent J* 1984 Aug;157(3):89-90.
- Afzal MA, Buchanan J, Dias JA, Cordeiro M, Bentley ML, Shorrocks CA, Minor PD. RT-PCR based diagnosis and molecular characterisation of mumps viruses derived from clinical specimens collected during the 1996 mumps outbreak in Portugal. *J Med Virol* 1997 Aug;52(4):349-53.
- Albrecht M, Banoczy J, Tamas G Jr. Dental and oral symptoms of diabetes mellitus. *Community Dent Oral Epidemiol* 1988;16(6):378-80.
- Aldous JA, Aldous SG. Management of oral health for the HIV-infected patient. *J Dent Hyg* 1991;65:143-5.
- Aldridge JP, Lester V, Watts TL, Collins A, Viberti G, Wilson RF. Single-blind studies of the effects of improved periodontal health on metabolic control in type 1 diabetes mellitus. *J Clin Periodontol* 1995;22(4):271-5.
- Andersen WC, Horton HL. Parietal lobe abscess after routine periodontal recall therapy: report of a case. *J Periodontol* 1990;61:243-7.
- Andrews M, Farnham S. Brain abscess secondary to dental infection. *Gen Dent* 1990;38:224-5.
- Baba TW, Trichel AM, An L, et al. Infection and AIDS in adult macaques after nontraumatic oral exposure to cell-free SIV. *Science* 1996;272:1486 (abstract).
- Bacic M, Plancak D, Granic M. CPITN assessment of periodontal disease in diabetic patients. *J Periodontol* 1988 Dec;59(12):816-22.
- Baker SB, Weinzwieg J, Bartlett SP, Whitaker LA. Brain abscess as a complication of orthognathic surgery: diagnosis, management, and pathophysiology. *Plast Reconstruct Surg* 1999;104:480-2.
- Barco CT. Prevention of infective endocarditis: a review of the medical and dental literature. *J Periodontol* 1991 Aug;62(8):510-23.
- Baron S, Poast J, Richardson CJ, Nguyen D, Cloyd M. Oral transmission of human immunodeficiency virus by infected seminal fluid and milk: a novel mechanism. *J Infect Dis* 2000;181:498-504.
- Bayliss R, Clarke C, Oakley CM, Somerville W, Whitfield AG, Young SE. The microbiology and pathogenesis of infective endocarditis. *Br Heart J* 1983 Dec;50(6):513-9.
- Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol* 1996 Oct;67(10 Suppl):1123-37.
- Beck JD, Offenbacher S, Williams R, Gibbs P, Garcia R. Periodontics: a risk factor for coronary heart disease? *Ann Periodontol* 1998 Jul;3(1):127-41.
- Beck-Nielsen H. Clinical disorders of insulin resistance. In: Alberti KG, Defronzo RA, Keen H, Zimmet P, editors. *International textbook of diabetes mellitus*. New York: Wiley; 1992. p. 531-68.
- Beisel WR. Metabolic response to infection. *Annu Rev Med* 1975;26:9-20.
- Belting CM, Hiniker JJ, Dummett CO. Influence of diabetes mellitus on the severity of periodontal disease. *J Periodontol* 1964;35:476-80.
- Benveniste R, Bixler D, Conneally PM. Periodontal disease in diabetics. *J Periodontol* 1967 Jul-Aug;38(4):271-9.
- Berger A, Henderson M, Nadoolman W, et al. Oral capsaicin provides temporary relief for oral mucositis pain secondary to chemotherapy/radiation therapy. *J Pain Symptom Manage* 1995;10(3):243-8.
- Bochud PY, Calandra T, Francioli P. Bacteremia due to viridans streptococci in neutropenic patients: a review. *Am J Med* 1994;97:256-64.
- Bridges RB, Anderson JW, Saxe SR, Gregory K, Bridges SR. Periodontal status of diabetic and non-diabetic men: effects of smoking, glycemic control, and socioeconomic factors. *J Periodontol* 1996 Nov;67(11):1185-92.
- Campbell MJ. Epidemiology of periodontal disease in the diabetic and the non-diabetic. *Aust Dent J* 1972 Aug;17(4):274-8.
- Carl W. Oral complications of local and systemic cancer treatment. *Curr Opin Oncol* 1995 Jul;7(4):320-4.
- Centers for Disease Control (CDC). *Pneumocystis pneumonia—Los Angeles*. *MMWR Morb Mortal Wkly Rep* 1981a Jun 5;30(21):250-2.

- Centers for Disease Control (CDC). Kaposi's sarcoma and *Pneumocystis pneumonia* among homosexual men—New York City and California. *MMWR Morb Mortal Wkly Rep* 1981b Jul 3;30(25):305-8.
- Centers for Disease Control (CDC). Acquired immunodeficiency syndrome (AIDS): precautions for health care workers and allied professionals. *MMWR Morb Mortal Wkly Rep* 1983;32:450-2.
- Centers for Disease Control (CDC). Update: acquired immunodeficiency syndrome—United States. *MMWR Morb Mortal Wkly Rep* 1985;34:245-8.
- Centers for Disease Control (CDC). 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *MMWR Morb Mortal Wkly Rep* 1992 Dec 18;41(No. RR-17):1-19.
- Centers for Disease Control (CDC). 1994 revised classification system for human immunodeficiency virus infection in children less than 13 years of age. *MMWR Morb Mortal Wkly Rep* 1994;43(No. RR-12).
- Centers for Disease Control and Prevention (CDC). Transmission of HIV possibly associated with exposure of mucous membrane to contaminated blood. *MMWR Morb Mortal Wkly Rep* 1997;46:620-3.
- Christersson LA, Albin B, Zambon JJ, Wikesjö UM, Genco RJ. Tissue localization of *Actinobacillus actinomycetemcomitans* in human periodontitis. I. Light, immunofluorescence and electron microscopic studies. *J Periodontol* 1987a Aug;58(8):529-39.
- Christersson LA, Wikesjö UM, Albin B, Zambon JJ, Genco RJ. Tissue localization of *Actinobacillus actinomycetemcomitans* in human periodontitis. II. Correlation between immunofluorescence and culture techniques. *J Periodontol* 1987b Aug;58(8):540-5.
- Christgau M, Palitzsch KD, Schmalz G, Kreiner U, Frenzel S. Healing response to non-surgical periodontal therapy in patients with diabetes mellitus: clinical, microbiological, and immunologic results. *J Clin Periodontol* 1998 Feb;25(2):112-24.
- Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ. Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes). *J Am Dent Assoc* 1982 May;104(5):653-60.
- Coates E, Slade GD, Goss AN, et al. Oral conditions and their social impact among HIV dental patients. *Aust Dent J* 1996;41:33-6.
- Cohen DW, Friedman LA, Shapiro J, Kyle GC, Franklin S. Diabetes mellitus and periodontal disease: two-year longitudinal observations. I. *J Periodontol* 1970 Dec;41(12):709-12.
- Collins JG, Smith MA, Arnold RR, Offenbacher S. Effects of *Escherichia coli* and *Porphyromonas gingivalis* lipopolysaccharide on pregnancy outcome in the golden hamster. *Infect Immun* 1994a;62(10):4652-5.
- Collins JG, Windley HW 3rd, Arnold RR, Offenbacher S. Effects of a *Porphyromonas gingivalis* infection on inflammatory mediator response and pregnancy outcome in hamsters. *Infect Immun* 1994b Oct;62(10):4356-61.
- Cone EJ. Saliva testing for drugs of abuse. *Ann NY Acad Sci* 1993 Sep 20;694:91-127.
- Cone EJ, Oyler J, Darwin WD. Cocaine disposition in saliva following intravenous, intranasal, and smoked administration. *J Anal Toxicol* 1997 Oct;21(6):465-75.
- Constantine NT, et al. Diagnostic challenges for rapid human immunodeficiency virus assays: performance using HIV-1 Group O, HIV-1 Group M, and HIV-2 samples. *J Hum Virol* 1997:145-51.
- Cruz GD, Lamster IB, Begg MD, et al. The accurate diagnosis of oral lesions in human immunodeficiency virus infection. Impact on medical staging. *Arch Otolaryngol Head Neck Surg* 1996;122:68-73.
- Dabbs JM Jr. Salivary testosterone measurements in behavioral studies. *Ann NY Acad Sci* 1993 Sep 20;694:177-83.
- Dajani AS, Taubert KA, Wilson W, Bolger AF, Bayer A, Ferrieri P, Gewitz MH, Shulman ST, Nouri S, Zuccaro G Jr., et al. Prevention of bacterial endocarditis: recommendations by the American Heart Association. *Clin Infect Dis* 1997 Dec;25(6):1448-58.
- Dasanayake AP. Poor periodontal health of the pregnant woman as a risk factor for low birth weight. *Ann Periodontol* 1998;3:206-12.
- Davenport ES, et al. The East London study of maternal chronic periodontal disease and preterm low birth weight infants: study design and prevalence data. *Ann Periodontol* 1998;3:213-21.
- de Pommereau V, Dargent-Pare C, Robert JJ, Brion M. Periodontal status in insulin-dependent diabetic adolescents. *J Clin Periodontol* 1992 Oct;19(9 Pt.1):628-32.
- DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM. Dental disease and risk of coronary heart disease and mortality. *BMJ* 1993 Mar;306(6879):688-91.
- Dillon B, Hecht FM, Swanson M, Goupil-Sormany I, Grant RM, Chesney MA, Kahn JO. Primary HIV infections associated with oral transmission (abstract 473). 7th Conference on Retroviruses and Opportunistic Infections; 2000 Jan 30-Feb 2. Chicago, IL.
- Dodd CL, Greenspan D, Katz MH, Westenhouse JL, Feigal DW, Greenspan JS. Oral candidiasis in HIV infection: pseudomembranous and erythematous candidiasis show similar rates of progression to AIDS. *AIDS* 1991 Nov;5(11):1339-43.
- Dolan TA, Gilbert GH, Ringelberg ML, Legler DW, Antonson DE, Foerster U, Heft MW. Behavioral risk indicators of attachment loss in adult Floridians. *J Clin Periodontol* 1997 Apr;24(4):223-32.
- Drobny EC, Abramson EC, Baumann G. Insulin receptors in acute infection: a study of factors conferring insulin resistance. *J Clin Endocrinol Metab* 1984 Apr;58(4):710-6.

- Duffy RE, Adelson R, Niessen LC, Wescott WB, Watkins K, Rhyne RR. VA oral HIV surveillance program: understanding the disease. *J Am Dent Assoc* 1992 Oct;123(10):57-62.
- Durack DT. Prevention of infective endocarditis. *N Engl J Med* 1995 Jan 5;332(1):38-44.
- Eisenberg E, Krutchkoff D, Yamase H. Incidental oral hairy leukoplakia in immunocompetent persons. A report of two cases. *Oral Surg Oral Med Oral Pathol* 1992 Sep;74(3):332-3.
- Ellison PT. Measurements of salivary progesterone. *Ann NY Acad Sci* 1993 Sep 20;694:161-76.
- Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. *J Periodontol* 1991 Feb;62(2):123-31.
- Engelhard D, Elishoov H, Or R, et al. Cytosine arabinoside as a major risk factor for *Streptococcus viridans* septicemia following bone marrow transplantation: a 5-year prospective study. *Bone Marrow Transpl* 1995;16:565-70.
- Epstein JB, Silverman S Jr. Head and neck malignancies associated with HIV infection. *Oral Surg Oral Med Oral Pathol* 1992 Feb;73(2):193-200.
- Epstein JB, Rea G, Wong FL, Spinelli J, Stevenson-Moore P. Osteonecrosis: study of the relationship of dental extractions in patients receiving radiotherapy. *Head Neck Surg* 1987;10:48-54.
- Epstein JB, Priddy RW, Sherlock CH. Hairy leukoplakia-like lesions in immunosuppressed patients following bone marrow transplantation. *Transplantation* 1988 Sep;46(3):462-4.
- Erickson PR, Herzberg MC. Evidence for the covalent linkage of carbohydrate polymers to a glycoprotein from *Streptococcus sanguis*. *J Biol Chem* 1993 Nov 15;268(32):23780-3.
- Falk KI, Zou JZ, Lucht E, Linde A, Ernberg I. Direct identification by PCR of EBV types and variants in clinical samples. *J Med Virol* 1997 Apr;51(4):355-63.
- Faruque S, Edlin B, McCoy CB, et al. Crack cocaine smoking and oral sores in three inner-city neighborhoods. *J Acquir Immun Defic Syndr Hum Retrovirol* 1996a;7-92.
- Faruque S, Edlin BR, McCoy CB, et al. Oral lesions among crack smokers in three inner-city neighborhoods: implications for HIV transmission through oral sex. *3rd Conf Retro Opportun Infect* 1996b;73.
- Faulconbridge AR, Bradshaw WC, Jenkins PA, Baum JD. The dental status of a group of diabetic children. *Br Dent J* 1981 Oct 20;151(8):253-5.
- Feigal DW, Katz MH, Greenspan D, Westenhause J, Winkelstein W Jr, Lang W, Samuel M, Buchbinder SP, Hessel NA, Lifson AR, et al. The prevalence of oral lesions in HIV-infected homosexual and bisexual men: three San Francisco epidemiological cohorts. *AIDS* 1991 May;5(5):519-25.
- Feld R. The role of surveillance cultures in patients likely to develop chemotherapy-induced mucositis. *Support Care Cancer* 1997 Sep;5(5):371-5.
- Felix DH, Watret K, Wray D, Southam JC. Hairy leukoplakia in an HIV-negative, nonimmunosuppressed patient. *Oral Surg Oral Med Oral Pathol* 1992 Nov;74(5):563-6.
- Field MJ, Lawrence RL, Zwanziger L, editors. Extending Medicare coverage for preventive and other services. Washington: Institute of Medicine, National Academy Press; 2000.
- Finestone AJ, Boorujy SR. Diabetes mellitus and periodontal disease. *Diabetes* 1967 May;16(5):336-40.
- Firatli E. The relationship between clinical periodontal status and insulin-dependent diabetes mellitus. Results after 5 years. *J Periodontol* 1997 Feb;68(2):136-40.
- Firatli E, Yilmaz O, Onan U. The relationship between clinical attachment loss and the duration of insulin-dependent diabetes mellitus (IDDM) in children and adolescents. *J Clin Periodontol* 1996 Apr;23(4):362-6.
- Fischl MA, Dickinson GM, Scott GB, et al. Evaluation of heterosexual partners, children, and household contacts of adults with AIDS. *JAMA* 1987;257:640-4.
- Flier JS. An overview of insulin resistance. In: Moller DE, editor. *Insulin resistance*. New York: Wiley; 1993. p. 1-8.
- Fourrier F, Duvivier B, Boutigny H, Roussel-Devallez M, Chopin C. Colonization of dental plaque: a source of nosocomial infections in intensive care unit patients. *Crit Care Med* 1998;26:301-8.
- Galea H, Aganovic I, Aganovic M. The dental caries and periodontal disease experience of patients with early onset insulin dependent diabetes. *Int Dent J* 1986 Dec;36(4):219-24.
- Gallagher DM, Erickson K, Hollin SA. Fatal brain abscess following periodontal therapy: a case report. *Mt Sinai J Med* 1981;48:158-60.
- Gandolfo AM, De LP, Modugno G, et al. The oral status of HIV+ patients. The clinico-statistical results with reference to 2 examination check-ups. [Ital]. *Minerva Stomatologica* 1991;40:651-6.
- Genco RJ. Periodontal disease and risk for myocardial infarction and cardiovascular disease. *Cardiovasc Rev Rep* 1998;19(3):34-40.
- Genco R, Chadda S, Grossi S, Dunford R, Taylor G, Knowler W, Pettitt D. Periodontal disease is a predictor of cardiovascular disease in a Native American population. *J Dent Res* 1997 Mar;76(Spec No):14-519 (abstract 3158).
- Geraci JE, Wilson JR. Symposium on infective endocarditis. III. Endocarditis due to gram-negative bacteria. Report of 56 cases. *Mayo Clin Proc* 1982 Mar;57(3):145-8.
- Gibbs RS, Romero R, Hillier SL, Eschenbach DA, Sweet RL. A review of premature birth and subclinical infection. *Am J Obstet Gynecol* 1992 May;166(5):1515-28.

- Glavind L, Lund B, Løe H. The relationship between periodontal state and diabetes duration, insulin dosage and retinal changes. *J Periodontol* 1968 Nov;39(6):341-7.
- Glick M, Garfunkel AA. Common oral findings in two different diseases—leukemia and AIDS. Part 1. *Compendium* 1992 Jun;13(6):432,434,436 passim.
- Glick M, Muzyka BC, Lurie D, Salkin LM. Oral manifestations associated with HIV-related disease as markers for immune suppression and AIDS. *Oral Surg Oral Med Oral Pathol* 1994a Apr;77(4):344-9.
- Glick M, Muzyka BC, Salkin LM, Lurie D. Necrotizing ulcerative periodontitis: a marker for immune deterioration and a predictor for the diagnosis of AIDS. *J Periodontol* 1994b May;65(5):393-7.
- Gonzalez M, Banderas JA, Baez A, Belmont R. Salivary lead and cadmium in a young population residing in Mexico City. *Toxicol Lett* 1997 Sep 19;93(1):55-64.
- Goteiner D, Sonis ST, Fasciano R. Cavernous sinus thrombosis and brain abscess initiated and maintained by periodontally involved teeth. *J Oral Med* 1982;37:80-3.
- Goteiner D, Vogel R, Deasy M, Goteiner C. Periodontal and caries experience in children with insulin-dependent diabetes mellitus. *J Am Dent Assoc* 1986 Aug;113(2):277-9.
- Grau AJ, Buggle F, Ziegler C, Schwarz W, Meuser J, Tasman AJ, Buhler A, Benesch C, Becher H, Hacke W. Association between acute cerebrovascular ischemia and chronic and recurrent infection. *Stroke* 1997 Sep;28(9):1724-9.
- Greenberg MS. HIV-associated lesions. *Dermatol Clin* 1996 Apr;14(2):319-26.
- Greenspan D, Greenspan JS. HIV-related oral disease. *Lancet* 1996 Sep;348(9029):729-33.
- Greenspan D, Greenspan JS, Conant M, Petersen V, Silverman S Jr, De Souza Y. Oral "hairy" leukoplakia in male homosexuals: evidence of association with both papillomavirus and a herpes-group virus. *Lancet* 1984 Oct 13;2(8407):831-4.
- Greenspan D, Greenspan JS, Hearst NG, Pan LZ, Conant MA, Abrams DI, Hollander H, Levy JA. Relation of oral hairy leukoplakia to infection with the human immunodeficiency virus and the risk of developing AIDS. *J Infect Dis* 1987 Mar;155(3):475-81.
- Greenspan D, Greenspan JS, Souza YG, Levy JA, Ungar AM. Oral hairy leukoplakia in an HIV-negative renal transplant recipient. *J Oral Pathol Med* 1989 Jan;18(1):32-4.
- Greenspan JS. In: Fauci AS, Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Kasper DI, editors. *Harrison's principles of internal medicine*. 14th ed. New York: McGraw-Hill; 1998.
- Greenspan JS, Greenspan D, Lennette ET, Abrams DI, Conant MA, Petersen VH. Oral viral leukoplakia—a new AIDS-associated condition. *Adv Exp Med Biol* 1985;187:123-8.
- Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. *Ann Periodontol* 1998 Jul;3(1):51-61.
- Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, Norderyd OM, Genco RJ. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* 1994 Mar;65(3):260-7.
- Grossi SG, Skrepcinski FB, DeCaro T, Zambon JJ, Cummins D, Genco RJ. Response to periodontal therapy in diabetics and smokers. *J Periodontol* 1996 Oct;67(10 Suppl):1094-102.
- Grossi SG, Skrepcinski FB, DeCaro T, Robertson DC, Ho AW, Dunford RG, Genco RJ. Treatment of periodontal disease in diabetes reduces glycated hemoglobin. *J Periodontol* 1997 Aug;68(8):713-9.
- Grossi SG, Donahue R, Quattrin T, Trevisan M, Ho A, Genco RJ. Periodontal disease increases the risk for insulin resistance. *J Dent Res* 1999;78(IADR abstract no. 3486).
- Guyen Y, Satman I, Dinccag N, Alptekin S. Salivary peroxidase activity in whole saliva of patients with insulin-dependent (type-1) diabetes mellitus. *J Clin Periodontol* 1996 Sep;23(9):879-81.
- Haeckel R. Factors influencing the saliva/plasma ratio of drugs. In: Malamud D, Tabak L, editors. *Saliva as a diagnostic fluid*. *Ann NY Acad Sci* 1993;694:128-42.
- Hallgren R, Lundquist G. Elevated serum levels of pancreatic polypeptide are related to impaired glucose handling in inflammatory states. *Scand J Gastroenterol* 1983 May;18(4):561-4.
- Harrison R, Bowen WH. Periodontal health, dental caries, and metabolic control in insulin-dependent diabetic children and adolescents. *Pediatr Dent* 1987 Dec;9(4):283-6.
- Hayes C, Sparrow D, Cohen M, Vokonas PS, Garcia RI. The association between alveolar bone loss and pulmonary function: the VA Dental Longitudinal Study. *Ann Periodontol* 1998 Jul;3(1):257-61.
- Heimdahl A, Gahrton G, Groth G-C, et al. Selective decontamination of the alimentary tract microbial flora in patients treated with bone marrow transplantation. *Scand J Infect Dis* 1984;16(1):51-60.
- Heimdahl A, Mattson T, Dahllöf F, Lonnquist B, Ringden O. The oral cavity as a port of entry for early infections in patients treated with bone marrow transplantation. *Oral Surg Oral Med Pathol* 1989;68:711-6.
- Herzberg MC, Meyer MW. Effects of oral flora on platelets: possible consequences in cardiovascular disease. *J Periodontol* 1996 Oct;67(10 Suppl):1138-42.
- Herzberg MC, Meyer MW. Dental plaque, platelets, and cardiovascular diseases. *Ann Periodontol* 1998 Jul;3(1):151-60.
- Herzberg MC, Brintzenhofe KL, Clawson CC. Aggregation of human platelets and adhesion of *Streptococcus sanguis*. *Infect Immun* 1983 Mar;39(3):1457-69.

- Herzberg MC, MacFarlane GD, Liu P, Erickson PR. The platelet as an inflammatory cell in periodontal diseases: interactions with *Porphyromonas gingivalis*. In: Genco R, Hamada S, Lehner T, McGhee J, Mergenhagen S, editors. Molecular pathogenesis of periodontal disease. Washington: American Society for Microbiology; 1994. p. 247-55.
- Hildebolt CF, Pilgram TK, Dotson M, Yokoyama-Crothers N, Muckerman J, Hauser J, Cohen S, Kardaris E, Vannier MW, Hanes P, Shrout MK, Civitelli R. Attachment loss with postmenopausal age and smoking. *J Periodontal Res* 1997 Oct;32(7):619-25.
- Hillier SL, Martius J, Krohn MJ, Kiviat N, Holmes KK, Eschenbach DA. A case-control study of chorioamnionic infection and histologic chorioamnionitis in prematurity. *N Engl J Med* 1988 Oct;319(15):972-8.
- Hillier SL, Nugent RP, Eschenbach DA, Krohn MA, Gibbs RS, Martin DH, Cotch MF, Edelman R, Pastorek JG 2nd, Rao AV, et al. Association between bacterial vaginosis and preterm delivery of a low-birth-weight infant. The vaginal infections and prematurity study group. *N Engl J Med* 1995 Dec;333(26):1737-42.
- Hirai T, Ishijima T, Hashikawa Y, Yajima T. Osteoporosis and reduction of residual ridge in edentulous patients. *J Prosthet Dent* 1993 Jan;69(1):49-56.
- Holmes K, Sparling PF, Mardh P, Lemon SM, Stamm W, Piot P, Wasserheit J, editors. Sexually transmitted diseases. 3rd ed. New York: McGraw-Hill; 1999.
- Hotamisligil GS, Shargill NS, Spiegelman BM. Adipose expression of tumor necrosis factor- α : direct role in obesity-linked insulin resistance. *Science* 1993 Jan;259(5091):87-91.
- Hove KA, Stallard RE. Diabetes and the periodontal patient. *J Periodontol* 1970 Dec;41(12):713-8.
- Hugoson A, Thorstensson H, Falk H, Kuylenstierna J. Periodontal conditions in insulin-dependent diabetics. *J Clin Periodontol* 1989 Apr;16(4):215-23.
- Humphries S, Devlin H, Worthington H. A radiographic investigation into bone resorption of mandibular alveolar bone in elderly edentulous adults. *J Dent* 1989 Apr;17(2):94-6.
- Institute of Medicine (IOM), Committee to Study the Prevention of Low Birth Weight, Division of Health. Promotion and disease progression. Preventing low birth weight. Washington: National Academy Press; 1985.
- Itin P, Ruffli T, Rudlinger R, Cathomas G, Huser B, Podvinec M, Gudat F. Oral hairy leukoplakia in a HIV-negative renal transplant patient: a marker for immunosuppression? *Dermatologica* 1988;177(2):126-8.
- Itin PH, Lautenschlager S, Fluckiger R, et al. Oral manifestations in HIV-infected patients: diagnosis and management. *J Am Acad Dermatol* 1993;29:749-60.
- Jeffcoat MK. Osteoporosis: a possible modifying factor in oral bone loss. *Ann Periodontol* 1998;3:312-21.
- Jeffcoat MK, Lewis CE, Reddy MS, Wang CY, Reford M. Post-menopausal bone loss and its relationships to oral bone loss. *Periodontology* 2000. (in press).
- Joselow MM, Ruiz R, Goldwater LJ. Absorption and excretion of mercury in man. XIV. Salivary excretion of mercury and its relationship to blood and urine mercury. *Arch Environ Health* 1968 Jul;17(1):39-43.
- Joshi N, O'Bryan T, Appelbaum PC. Pleuropulmonary infections caused by *Eikenella corrodens*. *Rev Infect Dis* 1991 Nov-Dec;13(6):1207-12.
- Joshi KJ, Rimm EB, Douglass CW, Trichopoulos D, Asherio A, Willett WC. Poor oral health and coronary heart disease. *J Dent Res* 1996 Sep;75(9):1631-6.
- Joshi KJ, Ascherio A, Rimm E, Douglass CW, Willett WC. The relation between tooth loss and incidence of ischemic stroke. *Circulation* 1999;99:1121.
- Kaye D. Infective endocarditis. In: Fauci AS, Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Kasper DL, editors. *Harrison's principles of internal medicine*. 13th ed. New York: McGraw-Hill; 1994. p. 520-6.
- King GN, Healy CM, Glover MT, Kwan JT, Williams DM, Leigh IM, Thornhill MH. Prevalence and risk factors associated with leukoplakia, hairy leukoplakia, erythematous candidiasis, and gingival hyperplasia in renal transplant recipients. *Oral Surg Oral Med Oral Pathol* 1994 Dec;78(6):718-26.
- Kjellman O, Henriksson CO, Berghagen N, Andersson B. Oral conditions in 105 subjects with insulin-treated diabetes mellitus. *Sven Tandlak Tidsskr* 1970 Feb;63(2):99-110.
- Klein A, Bruser B, Bast M, Rachlis A. Progress of HIV infection and changes in the lipid membrane structure of CD4+ cells. *AIDS* 1992 Mar;6(3):332-3.
- Klein RS, Harris CA, Small CB, Moll B, Lesser M, Friedland GH. Oral candidiasis in high-risk patients as the initial manifestation of the acquired immunodeficiency syndrome. *N Engl J Med* 1984 Aug 9;311(6):354-8.
- Kline MW. Oral manifestations of pediatric human immunodeficiency virus infection: a review of the literature. *Pediatrics* 1996 Mar;97(3):380-8.
- Koelle DM, Huang ML, Chandran B, Vieira J, Piepkorn M, Corey L. Frequent detection of Kaposi's sarcoma-associated herpesvirus (human herpesvirus 8) DNA in saliva of human immunodeficiency virus-infected men: clinical and immunologic correlates. *J Infect Dis* 1997;176(1):94-102.
- Krall EA, Dawson-Hughes B, Papas A, Garcia RI. Tooth loss and skeletal bone density in healthy postmenopausal women. *Osteoporos Int* 1994 Mar;4(2):104-9.
- Krall EA, Garcia RI, Dawson-Hughes B. Increased risk of tooth loss is related to bone loss at the whole body, hip, and spine. *Calcif Tissue Int* 1996 Dec;59(6):433-7.

- Kribbs PJ. Comparison of mandibular bone in normal and osteoporotic women. *J Prosthet Dent* 1990 Feb; 63(2):218-22.
- Kribbs PJ, Chesnut CH 3rd. Osteoporosis and dental osteopenia in the elderly. *Gerodontology* 1984 Summer;3(2):101-6.
- Kribbs PJ, Smith DE, Chesnut CH 3rd. Oral findings in osteoporosis. Part II: Relationship between residual ridge and alveolar bone resorption in generalized skeletal osteopenia. *J Prosthet Dent* 1983 Nov;50(5): 719-24.
- Kribbs PJ, Chesnut CH 3rd, Ott SM, Kilcoyne RF. Relationships between mandibular and skeletal bone in an osteoporotic population. *J Prosthet Dent* 1989 Dec;62(6):703-7.
- Kribbs PJ, Chesnut CH 3rd, Ott SM, Kilcoyne RF. Relationships between mandibular and skeletal bone in a population of normal women. *J Prosthet Dent* 1990 Jan;63(1):86-9.
- Kroes I, Lepp PW, Relman DA. Bacterial diversity within the human subgingival crevice. *Proc Natl Acad Sci USA* 1999 Dec 7;96(25):14547-52.
- Lacassin F, Hoen B, Leport C, Selton-Suty C, Delahaye F, Goulet V, Etienne J, Briancon S. Procedures associated with infective endocarditis in adults. A case control study. *Eur Heart J* 1995 Dec;16(12):1968-74.
- Lafferty WE, Hughes JP, Handsfield HH. Sexually transmitted diseases in men who have sex with men. Acquisition of gonorrhea and nongonococcal urethritis by fellatio and implications for STD/HIV prevention. *Sex Transm Dis* 1997 May;24(5):272-8.
- Leggott PL. Oral manifestations in pediatric HIV infection. In: Greenspan JS, Greenspan D, editors. *Oral manifestations of HIV infection*. Chicago: Quintessence; 1995. p. 234-9.
- Lifson AR, Hilton JF, Westenhouse JL, Canchola AJ, Samuel MC, Katz MH, Buchbinder SP, Hessel NA, Osmond DH, Shiboski S, et al. Time from HIV seroconversion to oral candidiasis or hairy leukoplakia among homosexual and bisexual men enrolled in three prospective cohorts. *AIDS* 1994 Jan;8(1):73-9.
- Löe H. Periodontal disease—the sixth complication of diabetes mellitus. *Diabetes Care* 1993 Jan;16(1): 329-34.
- Lorenz KA, Weiss PJ. Capnocytophagal pneumonia in a healthy man. *West J Med* 1994 Jan;160(1):79-80.
- Lucas RM, Howell LP, Wall BA. Nifedipine-induced gingival hyperplasia. A histochemical and ultrastructural study. *J Periodontol* 1985 Apr;56(4):211-5.
- Lucas VS, Roberts GJ, Beighton D. Oral health of children undergoing allogeneic bone marrow transplantation. *Bone Marrow Transpl* 1998;22:801-8.
- Mackenzie RS, Millard HD. Interrelated effects of diabetes, arteriosclerosis and calculus on alveolar bone loss. *J Am Dent Assoc* 1963;66:191-8.
- Malamud D. Oral diagnostic testing for detecting human immunodeficiency virus-1 antibodies: a technology whose time has come. *Am J Med* 1997; 102(4A):9-14.
- Malamud D, Tabak L, editors. *Saliva as a diagnostic fluid*. *Ann NY Acad Sci* 1993;694:128-42.
- Mandel ID. The diagnostic uses of saliva. *J Oral Pathol Med* 1990;19:119-25.
- Manouchehr-Pour M, Bissada NF. Periodontal disease in juvenile and adult diabetic patients: a review of the literature. *J Am Dent Assoc* 1983 Nov;107(5): 766-70.
- Martino R, Subira M, Manteiga R, Badell I, Argiles B, Sureda A, Brunet S. Viridans streptococcal bacteremia and viridans streptococcal shock syndrome in neutropenic patients: comparison between children and adults receiving chemotherapy or undergoing bone marrow transplantation. *Clin Infect Dis* 1995;20:476-7.
- Mattila KJ, Nieminen MS, Valtonen VV, Rasi VP, Kesaniemi YA, Syrjala SL, Jungell PS, Isoluoma M, Hietaniemi K, Jokinen MJ. Association between dental health and acute myocardial infarction. *BMJ* 1989 Mar 25;298(6676):779-81.
- Mattila KJ, Valtonen VV, Nieminen M, Huttunen JK. Dental infection and the risk of new coronary events: prospective study of patients with documented coronary artery disease. *Clin Infect Dis* 1995 Mar;20(3): 588-92.
- Mattila KJ, Asikainen S, Wolf J, Jousimies-Somer H, Valtonen VV, Nieminen M. Age, dental infections, and coronary heart disease. *J Dent Res* 2000;79: 756-60.
- McCormick MC. The contribution of low birth weight to infant mortality and childhood morbidity. *N Engl J Med* 1985 Jan;312(2):82-90.
- Mealey BL. Periodontal implications: medically compromised patients. *Ann Periodontol* 1996;1:256-321.
- Meyer DH, Sreenivasan PK, Fives-Taylor PM. Evidence for invasion of a human oral cell line by *Actinobacillus actinomycetemcomitans*. *Infect Immun* 1991; 59:2719-26.
- Miller LS, Manwell MA, Newbold D, Reding ME, Rasheed A, Blodgett J, Kornman KS. The relationship between reduction in periodontal inflammation and diabetes control: a report of 9 cases. *J Periodontol* 1992 Oct;63(10):843-8.
- Mohajery M, Brooks SL. Oral radiographs in the detection of early signs of osteoporosis. *Oral Surg Oral Med Oral Pathol* 1992 Jan;73(1):112-7.
- Montaner JS, Le TN, Le N, Craib KJ, Schechter MT. Application of the World Health Organization system for HIV infection in a cohort of homosexual men in developing a prognostically meaningful staging system. *AIDS* 1992 Jul;6(7):719-24.
- Morfeldt-Manson L, Julander I, Nilsson B. Dermatitis of the face, yellow toe nail changes, hairy leukoplakia and oral candidiasis are clinical indicators of progression to AIDS/opportunistic infection in patients with HIV infection. *Scand J Infect Dis* 1989;21(5):497-505.
- Morris JF, Sewell DL. Necrotizing pneumonia caused by mixed infection with *Actinobacillus actinomycetem-*

- comitans* and *Actinomyces israelii*: case report and review. *Clin Infect Dis* 1994 Mar;18(3):450-2.
- Morrison HI, Ellison LF, Taylor GW. Periodontal disease and risk of fatal coronary heart and cerebrovascular diseases. *J Cardiovasc Risk* 1999;6:7-11.
- Morton AA, Williams RW, Watts TL. Initial study of periodontal status in non-insulin-dependent diabetics in Mauritius. *J Dent* 1995 Dec;23(6):343-5.
- Mouldsdale MT, Eykyn SJ, Phillips I. Infective endocarditis, 1970-1979. A study of culture-positive cases in St. Thomas' Hospital. *Q J Med* 1980;49(195):315-28.
- Murrah VA. Diabetes mellitus and associated oral manifestations: a review. *J Oral Pathol* 1985 Apr;14(4):271-81.
- Murray CG, Herson J, Daly TE, Zimmerman S. Radiation necrosis of the mandible: a 10-year study. Part I. Factors influencing the onset of necrosis. *Int J Radiat Oncol Biol Phys* 1980a;6:543-8.
- Murray CG, Herson J, Daly TE, Zimmerman S. Radiation necrosis of the mandible: a 10-year study. Part II. Dental factors; onset, duration and management of necrosis. *Int J Radiat Oncol Biol Phys* 1980b;6:549-53.
- National Institute of Arthritis, Musculoskeletal and Skin Diseases. Available from: <http://www.nih.gov/niams/healthinfo/opbkg.htm> [2000].
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). Diabetes statistics. Bethesda (MD): National Institutes of Health; 1999 Mar. NIH Pub. no. 99-3892.
- National Institutes of Health (NIH). Consensus Development Conference on Oral Complication of Cancer Therapy, Diagnosis, Prevention and Treatment. NIH monograph no. 9. Bethesda (MD): National Institutes of Health; 1990.
- Nelson RG, Shlossman M, Budding LM, Pettitt DJ, Saad MF, Genco RJ, Knowler WC. Periodontal disease and NIDDM in Pima Indians. *Diabetes Care* 1990 Aug;13(8):836-40.
- Njoroge T, Genco RJ, Sojar HT, Hamada N, Genco CA. A role for fimbriae in *Porphyromonas gingivalis* invasion of oral epithelial cells. *Infect Immun* 1997 May; 65(5):1980-4.
- Novaes Junior AB, Pereira AL, de Moraes N, Novaes AB. Manifestations of insulin-dependent diabetes mellitus in the periodontium of young Brazilian patients. *J Periodontol* 1991 Feb;62(2):116-22.
- Novaes Junior AB, Gutierrez FG, Novaes AB. Periodontal disease progression in type II non-insulin-dependent diabetes mellitus patients (NIDDM). Part I—Probing pocket depth and clinical attachment. *Braz Dent J* 1996;7(2):65-73.
- O'Farrell BJ, Rajan E, Albloushi SS, Courtney MG, Fielding J, Shattock AG. The reliability of saliva as a sample for the detection of hepatitis A immunoglobulins under various sampling conditions. *Clin Diagn Virol* 1997;7(3):153-7.
- Offenbacher S, Katz V, Fertik G, Collins J, Boyd D, Maynor G, McKaig R, Beck J. Periodontal infection as a possible risk factor for preterm low birth weight. *J Periodontol* 1996 Oct;67(10 Suppl):1103-13.
- Offenbacher S, Jared HL, O'Reilly PG, Wells SR, Salvi GE, Lawrence HP, Socransky SS, Beck JD. Potential pathogenic mechanisms of periodontitis associated pregnancy complications. *Ann Periodontol* 1998 Jul;3(1):233-50.
- Oliver RC, Tervonen T. Periodontitis and tooth loss: comparing diabetics with the general population. *J Am Dent Assoc* 1993 Dec;124(12):71-6.
- Oliver RC, Tervonen T. Diabetes—a risk factor for periodontitis in adults? *J Periodontol* 1994 May;65(5 Suppl):530-8.
- Ortman LF, Hausmann E, Dunford RG. Skeletal osteopenia and residual ridge resorption. *J Prosthet Dent* 1989 Mar;61(3):321-5.
- Ostrow DG, DiFrancisco W. Why some gay men choose condoms and others switch to "safer" sex. *Int Conf AIDS* 1996;11:260.
- Padian N, Glass S. Transmission of HIV possibly associated with exposure of mucous membrane to contaminated blood. *MMWR Morb Mortal Wkly Rep* 1997;46:620-3.
- Payne B, Zachs NR, Reinhardt RA, et al. The association between estrogen status and alveolar bone density changes in post menopausal women with a history of periodontitis. *J Periodontol* 1997;68:24-31.
- Peterson DE. Research advances in mucositis. *Curr Opin Oncol* 1999;11:261-6.
- Peterson DE, Minah GE, Overholser CD, et al. Microbiology of acute periodontal infection in myelosuppressed cancer patients. *J Clin Oncol* 1987; 5:1461-8.
- Phelan JA. Oral manifestations of human immunodeficiency virus infection. *Med Clin North Am* 1997 Mar;81(2):511-31.
- Pinson M, Hoffman WH, Garnick JJ, Litaker MS. Periodontal disease and type 1 diabetes mellitus in children and adolescents. *J Clin Periodontol* 1995 Feb;22(2):118-23.
- Pizzo PA, Meyers J, Freifeld AG, Walsh T. Infections in the cancer patient. In: DeVita VT, Hellman S, Rosenberg SA, editors. *Cancer: principles and practice of oncology*. 4th ed. Philadelphia: J.B. Lippincott Co.; 1993. p. 2292-337.
- Pope M, Frankel S, Steinman R, Elmore D, Ho D, Marx P. Cutaneous dendritic cells promote replication of immunodeficiency viruses. *Adv Exp Med Biol* 1997; 417:395-9.
- Ramos-Gomez FJ, Hilton JF, Canchola AJ, Greenspan D, Greenspan JS, Maldonado YA. Risk factors for HIV-related oral-facial soft-tissue manifestations in children. *Pediatr Dent* 1996 Mar-Apr; 18(2):121-6.
- Redfield RR, Wright DC, Tramont EC. The Walter Reed staging classification for HTLV-III/LAV infection. *N Engl J Med* 1986 Jan;314(2):131-2.

- Reilly TG, Poxon V, Sanders DS, Elliot TS, Walt RP. Comparison of serum, salivary, and rapid whole blood diagnostic tests for *Helicobacter pylori* and their validation against endoscopy based tests. *Gut* 1997;40(4):454-8.
- Richards AL, Rerrault JG, Garingal LT, Manaloto CR, Sie A, Graham R, Ramos RM, Leonardo JB, Hyams KC. A non-invasive assessment of hepatitis B virus carrier status using saliva samples. *Southeast Asian J Trop Med Public Health* 1996;27(1):80-4.
- Ringelberg ML, Dixon DO, Francis AO, Plummer RW. Comparison of gingival health and gingival crevicular fluid flow in children with and without diabetes. *J Dent Res* 1977 Feb;56(2):108-11.
- Riviere GR, Weisz KS, Adams DF, Thomas DD. Pathogen-related oral spirochetes from dental plaque are invasive. *Infect Immun* 1991 Oct;59(10):3377-80.
- Rocke LK, Loprinzi CL, Lee JK, et al. A randomized clinical trial of two different durations of oral cryotherapy for prevention of 5-fluorouracil-related stomatitis. *Cancer* 1993; 72:2234-8.
- Rogers MF, White CR, Sanders R. Lack of transmission of HIV from infected children to their household contacts. *Pediatrics* 1990;85:210-4.
- Rolston KVI, Bodey GP. Infections in patients with cancer. In: Holland JF, Frei E, Bast RC, Kufe DW, Morton DL, Weichselbaum RR, editors. *Cancer medicine*. 3rd ed. Philadelphia: Lea & Febiger; 1993. p. 2416-41.
- Romero R, Baumann P, Gomez R, Salafia C, Rittenhouse L, Barberio D, Behnke E, Cotton DB, Mitchell MD. The relationship between spontaneous rupture of membranes, labor, and microbial invasion of the amniotic cavity and amniotic fluid concentrations of prostaglandins and thromboxane B2 in term pregnancy. *Am J Obstet Gynecol* 1993 Jun;168(6 Pt 1):1654-64; discussion 1664-8.
- Rothenberg RR, Scarlett MI, delRio C, Reznik D, Daniels C. Oral transmission of HIV. *AIDS* 1998;12:2095-105.
- Roy R, Burghoff R, Steffens DL. Detection of STR, D1S80, and gender alleles from body fluids collected on isocode (TM) foam collectors and paper-based devices. *Crime Lab Digest* 1997;24(1):4-9.
- Royce RA, Luckmann RS, Fusaro RE, Winkelstein W Jr. The natural history of HIV-1 infection: staging classifications of disease. *AIDS* 1991 Apr;5(4):355-64.
- Ruescher TJ, Sodeifi A, Scrivan SJ, Kaban LB, Sonis ST. The impact on alpha-hemolytic streptococcal infection in patients undergoing autologous bone marrow transplantation for hematologic malignancies. *Cancer* 1998;82:2275-81.
- Ruprecht RM, Baba TW, Liska V, Ray NB, Martin LN, Murphey-Corb M, Rizvi TA, Bernacky BJ, Keeling ME, McClure HM, Andersen J. Oral transmission of primate lentiviruses. *J Infect Dis* 1999 May;179 (Suppl 3):S408-12.
- Rylander H, Ramberg P, Blohme G, Lindhe J. Prevalence of periodontal disease in young diabetics. *J Clin Periodontol* 1987 Jan;14(1):38-43.
- Saah AJ, Munoz A, Kuo V, Fox R, Kaslow RA, Phair JP, Rinaldo CR Jr, Detels R, Polk BF. Predictors of the risk of development of acquired immunodeficiency syndrome within 24 months among gay men seropositive for human immunodeficiency virus type 1: a report from the Multicenter AIDS Cohort Study. *Am J Epidemiol* 1992;135:1147-55.
- Saal CJ, Mason JC, Cheuk SI, Hill MK. Brain abscess from chronic odontogenic cause: report of case. *J Am Dent Assoc* 1988;117:453-5.
- Saglie FR, Marfany A, Camargo P. Intra-gingival occurrence of *Actinobacillus actinomycetemcomitans* and *Bacteroides gingivalis* in active destructive periodontal lesions. *J Periodontol* 1988 Apr;59(4):259-65.
- Salvi GE, Lawrence HP, Offenbacher S, Beck JD. Influence of risk factors on the pathogenesis of periodontitis. *Periodontol* 2000 1997 Jun;14:173-201.
- Sammalkorpi K. Glucose intolerance in acute infections. *J Intern Med* 1989 Jan;225(1):15-9.
- Sande MA. Transmission of AIDS. The case against casual contagion. *N Engl J Med* 1986;314:380-2.
- Sandler HC, Stahl SS. Prevalence of periodontal disease in a hospitalized population. *J Dent Res* 1960 May-Jun;39(3):439-49.
- Sandros J, Papapanou PN, Dahlen G. *Porphyromonas gingivalis* invades oral epithelial cells in vitro. *J Periodont Res* 1993 May;28(3):219-26.
- Sandros J, Papapanou PN, Nannmark U, Dahlen G. *Porphyromonas gingivalis* invades pocket epithelium in vitro. *J Periodont Res* 1994 Jan;29(1):62-9.
- Scannapieco FA. Position paper of the American Academy of Periodontology: periodontal disease as a potential risk factor for systemic diseases. *J Periodontol* 1998 Jul;69(7):841-50.
- Scannapieco FA. Role of oral bacteria in respiratory infection. *J Periodontol* 1999;70:793-802.
- Scannapieco FA, Mylotte JM. Relationships between periodontal disease and bacterial pneumonia. *J Periodontol* 1996 Oct;67(10 Suppl):1114-22.
- Scannapieco FA, Papandonatos GD, Dunford RG. Associations between oral conditions and respiratory disease in a national sample survey population. *Ann Periodontol* 1998 Jul;3(1):251-6.
- Schmidt MI, Duncan BB, Sharrett AR, Lindberg G, Savage PJ, Offenbacher S, Azambuja MI, Tracy RP, Heiss G. Markers of inflammation and prediction of diabetes mellitus in adults (Atherosclerosis Risk in Communities study): a cohort study. *Lancet* 1999 May 15;353:1649-52.
- Schubert MM, Epstein JB, Peterson DE. Oral complications of cancer therapy. In: Yagelia JA, Neidle EA, Dowd FJ, editors. *Pharmacology and therapeutics in dentistry*. St. Louis: Mosby-Year Book Inc.; 1998. p. 644-55.
- Schubert MM, Peterson DE, Lloid M. Oral complications. In: Thomas E, Blume KG, Forman SJ, editors.

- Hematopoietic cell transplantation. 2nd ed. Malden (MA): Blackwell Science, Inc.; 1999. p. 751-63.
- Schwarzc SK, Kellogg TA, Kohn RP, et al. Temporal trends in human immunodeficiency virus seroprevalence and sexual behavior at the San Francisco municipal sexually transmitted disease clinic, 1989-1992. *Am J Epidemiol* 1995;142:314-22.
- Scully C, Epstein JB. Oral health care for the cancer patient. *Eur J Cancer B Oral Oncol* 1996 Sep;32B(5):281-92.
- Seage GR 3rd, Gatsonis C, Weissman JS, Haas JS, Craven DE, Makadon H, Goldberg J, Coltin K, Lewin KS, Epstein AM, et al. The Boston AIDS Survival Score (BASS): a multidimensional AIDS severity instrument. *Am J Public Health* 1997 Apr;87(4):567-73.
- Semenciw R, Morrison H, Mao Y, Johansen H, Davies JW, Wigle D. Major risk factors for cardiovascular disease mortality in adults: results from the Nutrition Canada Survey. *Int J Epidemiol* 1988;17:317-24.
- Seppala B, Ainamo J. A site-by-site follow-up study on the effect of controlled versus poorly controlled insulin-dependent diabetes mellitus. *J Clin Periodontol* 1994 Mar;21(3):161-5.
- Seppala B, Seppala M, Ainamo J. A longitudinal study on insulin-dependent diabetes mellitus and periodontal disease. *J Clin Periodontol* 1993 Mar;20(3):161-5.
- Shapiro S, McCormick MC, Starfield BH, Krischer JP, Bross D. Relevance of correlates of infant deaths for significant morbidity at 1 year of age. *Am J Obstet Gynecol* 1980 Feb 1;136(3):363-73.
- Shinzato T, Saito A. A mechanism of pathogenicity of "Streptococcus milleri group" in pulmonary infection: synergy with an anaerobe. *J Med Microbiol* 1994 Feb;40(2):118-23.
- Shlossman M, Knowler WC, Pettitt DJ, Genco RJ. Type 2 diabetes mellitus and periodontal disease. *J Am Dent Assoc* 1990 Oct;121(4):532-6.
- Silver JG, Martin AW, McBride BC. Experimental transient bacteraemias in human subjects with varying degrees of plaque accumulation and gingival inflammation. *J Clin Periodontol* 1977 May;4(2):92-9.
- Smith GT, Greenbaum CJ, Johnson BD, Persson GR. Short-term responses to periodontal therapy in insulin-dependent diabetic patients. *J Periodontol* 1996 Aug;67(8):794-802.
- Sonis ST. Oral mucositis as a biological process: a new hypothesis for the development of chemotherapy induced stomatotoxicity. *Oral Oncol* 1998;34:39-43.
- Sonis ST, Van Vugt AG, Brien JP, Muska AD, Bruskin AM, Rose A, Haley JD. Transforming growth factor-beta 3 mediated modulation of cell cycling and attenuation of 5-fluorouracil-induced oral mucositis. *Oral Oncol* 1997 Jan;33(1):47-54.
- Sparrelid E, Häggglund H, Remberger M, Ringden O, Lonnquist B, Ljungman P, Andersson J. Bacteraemia during the aplastic phase after allogeneic bone marrow transplantation is associated with early death from invasive fungal infection. *Bone Marrow Transpl* 1998;22:795-800.
- Sreebny LM, Schwartz SS. A reference guide to drugs and dry mouth. 2nd ed. *Gerodontology* 1997;14:33-47.
- Sriskandan S, Soto A, Evans TJ, Cohen J. Viridans streptococcal bacteraemia: a clinical study. *QJM* 1995;88:415-20.
- Stahl-Hennig C, Steinman RM, Tenner-Racz K, Pope M, Stolte N, Matz-Rensing K, Grobschupff G, Raschdorff B, Hunsmann G, Racz P. Rapid infection of oral mucosal-associated lymphoid tissue with simian immunodeficiency virus. *Science* 1999 Aug 20;285(5431):1261-5.
- Strom BL, Abrutyn E, Berlin JA, et al. Dental and cardiac risk factors for infective endocarditis. *Arch Intern Med* 1998 Nov 15;129(10):761-9.
- Svenson KL, Lundquist G, Wide L, Hallgren R. Impaired glucose handling in active rheumatoid arthritis: relationship to the secretion of insulin and counter-regulatory hormones. *Metabolism* 1987 Oct;36(10):940-3.
- Sznajder N, Carraro JJ, Rugna S, Sereday M. Periodontal findings in diabetic and nondiabetic patients. *J Periodontol* 1978 Sep;49(9):445-8.
- Szpunar SM, Ismail AI, Eklund SA. Diabetes and periodontal disease: analyses of NHANES I and HHANES. *J Dent Res* 1989;68(SI):164-438 (abstract 1605).
- Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M, Knowler WC, Pettitt DJ. Severe periodontitis and risk for poor glycemic control in patients with non-insulin-dependent diabetes mellitus. *J Periodontol* 1996 Oct;67(10 Suppl):1085-93.
- Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M, Knowler WC, Pettitt DJ. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. *J Periodontol* 1998a Jan;69(1):76-83.
- Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in type 2 diabetes. *Ann Periodontol* 1998b Jul;3(1):30-9.
- Tervonen T, Karjalainen K. Periodontal disease related to diabetic status. A pilot study of the response to periodontal therapy in type 1 diabetes. *J Clin Periodontol* 1997 Jul;24(7):505-10.
- Tervonen T, Knuuttila M. Relation of diabetes control to periodontal pocketing and alveolar bone level. *Oral Surg Oral Med Oral Pathol* 1986 Apr;61(4):346-9.
- Thorstensson H, Hugoson A. Periodontal disease experience in adult long-duration insulin-dependent diabetics. *J Clin Periodontol* 1993 May;20(5):352-8.
- Tindall B, Carr A, Cooper DA. Primary HIV infection: clinical, immunologic, and serologic aspects. In: Sande MA, Volberding PA, editors. *The medical management of AIDS*. Philadelphia: W.B. Saunders; 1995. p. 105-29.

- U.S. Department of Health and Human Services (USDHHS), Center for Research for Mothers and Children. Public Health Service/National Institutes of Health Progress Report. Washington: U.S. Department of Health and Human Services; 1984.
- U.S. Preventive Services Task Force. Guide to clinical preventive services. 2nd ed. Washington: US GPO; 1996.
- van Schie RC, Wilson ME. Saliva: a convenient source of DNA for analysis of bi-allelic polymorphisms of Fcg receptor IIA (CD32) and Fcg receptor IIIB (CD16). *J Immunol Methods* 1997;208:91-101.
- von Wowern N. Dual-photon absorptiometry of mandibles: in vitro test of a new method. *Scand J Dent Res* 1985 Apr;93(2):169-77.
- von Wowern N. Bone mineral content of mandibles: normal reference values—rate of age-related bone loss. *Calcif Tissue Int* 1988 Oct;43(4):193-8.
- von Wowern N, Klausen B, Kollerup G. Osteoporosis: a risk factor in periodontal disease. *J Periodontol* 1994 Dec;65(12):1134-8.
- Wactawski-Wende J, Grossi SG, Trevisan M, Genco RJ, Tezal M, Dunford RG, Ho AW, Hausmann E, Hreshchyshyn MM. The role of osteopenia in oral bone loss and periodontal disease. *J Periodontol* 1996;67(10 Suppl):1076-84.
- Wallace JI, Bloch D, Whitmore R, et al. Fellatio is a significant risk activity for acquiring AIDS in New York City streetwalking sex workers. *Int Conf AIDS* 1996;11:381.
- Ward JW, Duchin JS. The epidemiology of HIV and AIDS in the United States. *AIDS Clin Rev* 1997;45.
- Weinberg A, Belton CM, Park Y, Lamont RJ. Role of *Porphyromonas gingivalis* fimbriae invasion of gingival epithelial cells. *Infect Immun* 1997 Jan;65(1):313-6.
- Weinstein L, Schlesinger JJ. Pathoanatomic, pathophysiologic and clinical correlations in endocarditis (second of two parts). *New Engl J Med* 1974;291:832-6, 1122-6.
- Westfelt E, Rylander H, Blohme G, Jonasson P, Lindhe J. The effect of periodontal therapy in diabetics. Results after 5 years. *J Clin Periodontol* 1996 Feb;23(2):92-100.
- Wilkes JD. Prevention and treatment of oral mucositis following cancer chemotherapy. *Semin Oncol* 1998;25:538-51.
- Williams ME, Quesenberry PJ. Hematopoietic growth factors. *Hematol Pathol* 1992;6(3):105-24.
- Williams RC Jr, Mahan CJ. Periodontal disease and diabetes in young adults. *JAMA* 1960 Feb 20;172:776-8.
- Wilson JT. Clinical correlates of drugs in saliva. *Ann NY Acad Sci* 1993 Sep 30;694:48-61.
- Wilton JM, Griffiths GS, Curtis MA, Maiden MFJ, Gillet IR, Wilson DT, Sterne JA, Johnson NW. Detection of high-risk groups and individuals for periodontal diseases. Systemic predisposition and markers of general health. *J Clin Periodontol* 1988 Jul;15(6):339-46.
- Winkler JR, Robertson PB. Periodontal disease associated with HIV infection. *Oral Surg Oral Med Oral Pathol* 1992 Feb;73(2):145-50.
- Winkler JR, Grassi M, Murray PA. Clinical description and etiology of HIV-associated periodontal diseases. In: Robertson PB, Greenspan JS, editors. Perspectives on oral manifestations of AIDS: diagnosis and management of HIV-associated infections. Littleton (MA): PSG Publishing; 1988.
- Wolf J. Dental and periodontal conditions in diabetes mellitus. A clinical and radiographic study. *Proc Finn Dent Soc* 1977;73 (4-6 Suppl):1-56.
- Wu T, Trevisan M, Genco RJ, Dorn JP, Falkner KL, Sempos CT. Periodontal disease and risk of cerebrovascular disease: a prospective study of a representative sample of U.S. adults. *Am J Epidemiol* 1999;149:(11)290, Suppl S Jun 1.
- Wu TJ, Trevisan M, Genco RJ, Dorn JP, Falkner KL, Sempos CT. Examination of the relation between periodontal health status and cardiovascular risk factors: serum total and high density lipoprotein, cholesterol, C-reactive protein, and plasma fibrinogen. *Am J Epidemiol* 2000;151:273-82.
- Yavuzylmaz E, Yumak O, Akdoganli T, Yamalik N, Ozer N, Ersoy F, Yenijay I. The alterations of whole saliva constituents in patients with diabetes mellitus. *Aust Dent J* 1996 Jun;41(3):193-7.
- Yuan A, Yang PC, Lee LN, Chang DB, Kuo SH, Luh KT. *Actinobacillus actinomycetemcomitans* pneumonia with chest wall involvement and rib destruction. *Chest* 1992 May;101(5):1450-2.
- Zakrzewska JM, Aly Z, Speight PM. Oral hairy leukoplakia in a HIV-negative asthmatic patient on systemic steroids. *J Oral Pathol Med* 1995 Jul;24(6):282-4.
- Zijlstra EE, Swart GR, Godfroy FJ, Degener JE. Pericarditis, pneumonia and brain abscess due to a combined *Actinomyces-Actinobacillus actinomycetemcomitans* infection. *J Infect* 1992 Jul;25(1):83-7.

Effects on Well-being and Quality of Life

Fifty years ago the World Health Organization (WHO) defined health as the “complete state of physical, mental, and social well-being and not merely the absence of infirmity” (WHO 1948). In its definition the WHO acknowledged that an individual who is technically “cured” of disease may not necessarily be “well” and went on to indicate three dimensions of well-being. *Physical well-being* assumes the ability to function normally in activities such as bathing, dressing, eating, and moving around. *Mental well-being* implies that cognitive faculties are intact and that there is no burden of fear, anxiety, stress, depression, or other negative emotions. *Social well-being* relates to one’s ability to participate in society, fulfilling roles as family member, friend, worker, or citizen or in other ways engaging in interactions with others.

The WHO declaration resonated with ongoing developments in the social sciences as theoreticians recognized the need for multiple indicators in assessing health and treatment outcomes (Bergner et al. 1981, Fries et al. 1982, Hunt et al. 1985, Meenan et al. 1980). These efforts led to definitions of “health-related quality of life” (Guyatt et al. 1993) as well as explanatory models. The model proposed by Wilson and Cleary (1995), for example, posits five dimensions by which to measure treatment outcomes: biological and physiological variables, symptom status, functional status, general health perceptions, and overall quality of life. These factors are not independent but may be reciprocally connected. For example, a diabetic patient with symptoms of depression may experience a rise in serum glucose as a result of less vigilant glucose monitoring; the depression may then lead to a deterioration in physical and social activities. Most importantly, measures of biological and physiological factors are often inconsistent with patients’ own reports of symptoms, ability to function, general health perceptions, and overall

quality of life. In the wake of these developments in general medicine, researchers began to elaborate multidimensional models of “oral-health-related” quality of life.

The efforts to understand these relationships are particularly relevant given the aging of the population. As Gift and Atchison (1995) stated, measuring health-related quality of life allows assessment of “the trade-off between how long and how well people live.” Diseases and disorders that result in dental and craniofacial defects can thwart that goal, disturbing self-image, self-esteem, and well-being. Orofacial pain and loss of sensorimotor functions limit food choices and the pleasures of eating, restrict social contact, and inhibit intimacy.

Oral complications of many systemic diseases also compromise the quality of life. Problems with speaking, chewing, taste, smell, and swallowing are common in neurodegenerative conditions such as Parkinson’s disease; oral complications of AIDS include pain, dry mouth, mucosal infections, and Kaposi’s sarcoma; cancer therapy can result in painful ulcers, mucositis, and rampant dental caries; and periodontal disease is a complication of diabetes and osteoporosis. Prescription and nonprescription drugs often have the side effect of dry mouth.

The ability to measure the quality of life has the practical value of guiding policymakers, health service researchers, epidemiologists, program evaluators, and clinicians interested in the effects of interventions. The measures can also provide useful information to patients and family members, third-party payers, and employers. For example, measures of the ability to perform activities of daily living may indicate areas where the patient is able and competent, as well as areas where further therapy may be helpful.

This chapter reviews oral-health-related quality of life findings along functional, psychosocial, and economic dimensions, taking into consideration the

influence of cultural and spiritual values. The results of studies in which investigators asked adults how they value their oral health and whether they are satisfied with their oral health care are included. The study of the association between oral health and quality of life is a relatively new but rapidly growing field. A variety of questionnaires have been designed to assess oral-health-related quality of life, and the chapter concludes with a discussion of their use in surveys and analytic studies, and their potential importance in outcome research.

THE CULTURAL CONTEXT

The determination of the health-related quality of life of an individual is implicitly made against a cultural background that includes a set of values, standards, customs, and traditions associated with a particular society.

Decisions about whether to seek care from a dentist, a physician, or other care provider may be influenced by cultural or ethnic perspectives and understanding (Aday and Forthofer 1992, Andersen and Davidson 1997, Davidson and Andersen 1997, Diehnelt et al. 1990, Kiyak 1993, Lee and Kiyak 1992). Different population groups differ in the way they think about health, and in how they define a health problem, determine its seriousness, and decide whether to seek care. In one cultural setting a painful tooth may be enough to motivate care seeking. In another, bleeding, swelling, or fever may be necessary before care is sought. Similarly, decisions about whether to comply with a suggested treatment regimen, whether to engage in self-care, and whether to return for a follow-up appointment are also culturally influenced.

The anthropology and ethnography literature is rich in references to the ways in which different cultures at different times and places have regarded the human body (Hufford 1992, Kleinman 1979). Cultural beliefs regarding the body, health, and disease are often embedded in religious or spiritual traditions, which in turn may govern how diseases and disorders are regarded and treated. A brief description of Western and non-Western perspectives follows.

Cultural Models

In the medical model typical of Western society the body is partitioned into organs and systems, each with identifiable functions. The body is seen as functioning well unless disease disrupts it. Diseases in themselves are understood to be invariable across

cultures. The medical model has traditionally dichotomized body and mind/soul/spirit—science and magic. Such a perspective sees the body as relatively objective and value-free, immune to nonsomatic influences.

That perspective began to change with the pioneering work of Hans Selye in the 1930s on the importance of stress in health and disease (McEwen 1999). Research in the intervening half century has confirmed the reciprocal connections of the nervous, endocrine, and immune systems, not only in relation to stress, but also in terms of the effects of emotions and cognitive processes on health status.

The model that has emerged as a new paradigm in the study of health and disease incorporates biological with psychological and social factors. This biopsychosocial model is defining agendas for research in such fields as behavioral medicine and psychoneuroimmunology. Social and psychological factors are routinely incorporated into health assessments, the better to describe the quality of life. Other societies hold views of the body strikingly different from the medical model. In some cultures, individuals and their care providers conceive of the body as the union of soul and soma. Illness may occur as a result of a “failure in harmony” or “an imbalance of forces.” Schools of medicine in China, India, and other non-Western societies incorporate such principles into their teaching and practice (Hufford 1992).

Combining Perspectives

All Americans hold culturally influenced perspectives on healing and illness (Henderson et al. 1997), some of which come from more traditional beliefs. Some people accept pain as an inevitable part of illness, a necessary evil, or even punishment for past iniquities or shortcomings and may shun pain-relieving drugs (Zborowski 1952). Many pragmatically combine cultural, folk, complementary, and alternative healing practices with participation in conventional care delivery systems. A recent survey indicates that over 50 percent of Americans sought non-traditional therapies for a number of ailments (Eisenberg et al. 1998).

Traditional beliefs are often comforting and satisfying to individuals (Selikowitz 1994). Certainly, Western culture and science have not always improved the quality of people's lives (Harris et al. 1993). Dietary changes to refined foods have been associated with dental caries (Godson and Williams 1996, Navia 1994), obesity, and other deleterious health changes (Selikowitz 1994). The marketing of tobacco products has added to the burden of cancer

and heart and lung problems worldwide. Migrations from traditional community rural life to urban centers have been associated with family disruption and violence, drug abuse, sexually transmitted diseases, and hypertension in developing countries.

On the other hand, Western science may inform some cultural groups that certain traditional child-rearing practices can be detrimental to oral health (Kolasa 1978, Scheper-Hughes 1990). Early childhood caries is a form of tooth decay with complex etiologies. Researchers studying high rates of infant caries among some cultural groups are exploring the extent to which traditional means of soothing crying babies or handling bedtime routines play a role, as well as investigating prenatal nutrition and transmission of infection from caregiver to child (Febres et al. 1997, Kelly and Bruerd 1987, Ripa 1988, Tinanoff and O'Sullivan 1997).

People who hold different cultural perspectives may distance themselves from Western, scientific worldviews (Lee et al. 1993), a behavior that must be addressed in any program of health promotion and disease prevention. Health professionals who understand indigenous or local healing practices and concepts are better able to motivate patients and thereby enable them to integrate elements from various healing systems (Kleinman 1979).

America is undergoing major demographic changes, with the expectation that at some point before 2050 the white population will no longer represent the majority (Henderson et al. 1997). As these changes occur, cultural elements that now reflect minority groups may become more accepted and dominant. However, cultural values are neither static nor omnipotent in shaping people's lives. Furthermore, individuals within a culture manifest their cultural identity in different ways. Therefore, both the direction of these changes and their effects may be hard to predict.

ORAL-HEALTH-RELATED QUALITY OF LIFE DIMENSIONS

Multiple factors act and interact in determining one's quality of life, as Wilson and Cleary (1995) and others have observed. Thus the idea of assessing quality of life along multiple "dimensions" implies a departure from a simple linear scale with excellent quality of life at one end and greatly diminished quality of life at the other. The following sections explore several dimensions, beginning with effects along functional and psychosocial dimensions and concluding with a discussion of economic effects on quality of life.

Functional Dimensions

Investigators have reported on the effects of dental and craniofacial diseases on the ability to eat and enjoy the full range of dietary choices. The impact of less-than-optimal oral health also has been studied in relation to sleep problems, primarily in relation to oral-facial pain.

Eating

Both dental and systemic diseases can profoundly affect appetite and the ability to eat, and hence can compromise overall health and well-being. Because chronic illness and medications increase in aging populations, these effects may be particularly evident among the frail elderly (Ship et al. 1996). Undernutrition was observed in 50 percent of geriatric residents in a U.S. long-term care facility; in many cases, it was linked to eating and swallowing problems (Keller 1993).

Less severe oral disorders have more subtle effects on functions relating to eating, although the high prevalence of those disorders elevates their relative importance among health problems. For example, data from the National Health and Nutrition Examination Survey III indicate that 33.1 percent of people aged 65 and older have no teeth (Marcus et al. 1996). Furthermore, clinical studies indicate that the masticatory efficiency of replacement teeth is at least 30 to 40 percent lower than that of natural teeth (Idowu et al. 1986). Consistent with these findings, surveys of elderly populations in the United States indicate that self-reported chewing problems affect significant proportions of people. For example, in California 1 percent of Medicare enrollees were unable to swallow comfortably, whereas 37 percent of senior center residents reported trouble biting or chewing foods (Table 6.1).

A number of studies have indicated that having missing teeth is linked to a qualitatively poorer diet. For example, in studies of U.S. veterans (Chauncey et al. 1984), Canadians (Brodeur et al. 1993), and Finns (Ranta et al. 1987), people with impaired dentitions preferred soft, easily chewed foods that were lower in fiber and had lower nutrient density than foods eaten by people with intact dentitions. Quality of life clearly suffers when individuals are forced to limit food choices and the foods chosen do not provide optimal nutrition. For example, they would be hard put to comply with the healthful diet recommendation of "five-a-day" helpings of fiber-rich fruits and vegetables. In the elderly, edentulousness and poor oral health may contribute to significant weight loss,

TABLE 6.1
Prevalence of self-reported eating dysfunction in surveys of elderly Americans

Dysfunction Attributed to Oral Condition(s)	Percentage of Population Group Reporting Dysfunction	
	Elderly Persons in California ^a	
	Senior Center Residents	Medicare Enrollees
Had trouble biting or chewing	37	13
Limited the kinds of foods eaten	23	10
Unable to swallow comfortably	10	1
	Elderly Persons in Florida ^b	
Mouth sometimes dry	39	
Noticed an unpleasant taste in mouth	23	
Unable to chew hard things	19	
Experienced change in sense of taste	9	
Difficulty tasting some foods	6	
Noticed change in sense of smell	5	
	Elderly Persons in North Carolina ^c	
	African Americans	Whites
Difficulty chewing any foods	18	6
Felt sense of taste had worsened	13	3
Uncomfortable eating foods	13	6
Had to avoid eating some foods	10	4
Felt digestion had worsened	8	<1
Had to interrupt meals	6	<1

^a n = 1,842 elderly persons (Atchison and Dolan 1990).

^b n = 390 dentate persons aged 65 and older living in retirement communities (Gilbert et al. 1993).

^c n = 440 persons aged 70 and older in five North Carolina counties (Hunt et al. 1995).

which may affect overall health (Blaum et al. 1995, Ritchie et al. 2000, Sullivan et al. 1993).

Clinical research has demonstrated a general reduction in chewing function as the number of missing teeth increases, even when dentures are worn (Carlsson 1984, Feldman et al. 1980, Helkimo et al. 1978). However, clinical studies in Scandinavia have observed relatively good chewing performance when no more than 12 teeth are missing and the remaining 20 teeth are distributed fairly equally, providing good contact between the upper and the lower jaw (Agerberg and Carlsson 1975). This finding has led some researchers to advocate the concept of a "shortened dental arch" as a treatment goal for older adults, that is, retention of at least 20 well-distributed teeth (Kayser 1981). A consistent finding from research into treatment of tooth loss is that removable dentures produce only partial improvement in chewing performance (Garrett et al. 1996, van der Bilt et al. 1994, Witter et al. 1989). Dentures anchored by implants result in significantly better chewing performance than conventional, removable dentures (Geertman et al. 1996).

Self-reported measures of eating ability, satisfaction with eating, and avoidance of foods are widely

used to evaluate the effects of tooth loss and replacement. These measures capture aspects of eating that are not necessarily reflected in the clinical measures of chewing performance with specific foods (Demers et al. 1996). Such studies of self-reported chewing ability confirm several relationships already noted, including the findings that chewing ability declines as the extent of tooth loss increases and that removable dentures do not fully compensate for the reduction (Hildebrandt et al. 1997, Leake 1990, Slade et al. 1996, Wayler et al. 1984). In a study of male veterans with varying levels of tooth loss, Wayler et al. (1982) reported that levels of taste, smell, texture, perceived ease of chewing, and frequency of ingestion were adversely affected only among people with severely compromised dentitions. However, in some studies of prosthodontic treatment, self-reported chewing ability improved following treatment, even

in the absence of significant change in clinical measures of chewing performance (van der Bilt et al. 1994).

Impaired eating due to conditions other than tooth loss has been evaluated less frequently. In a study of dental patients with intact dentitions, Ernest (1993) correlated reduced salivary flow with decreased intake of 18 of 22 nutrients. Locker and Slade (1988) found that 6 percent of the Canadians surveyed who reported symptoms of temporomandibular disorders also reported problems with eating, talking, or swallowing. These conditions are often associated with limited mouth opening and severe pain, which may be constant or present whenever jaw movements are made. The constant dry mouth of patients with Sjögren's syndrome, a disease in which the salivary glands are progressively destroyed, is a major source of discomfort that affects speaking, chewing, and swallowing.

As part of a Performance Status Scale study of oral and pharyngeal cancer patients, List et al. (1990) reported that only one third of patients achieved a perfect score on a measure of normalcy of diet and only 60 percent on a measurement of eating in public. Communication skills also suffered; only 55 percent scored perfectly on understandability of speech.

Sleeping

Sleep problems associated with oral conditions appear to be most closely related to chronic pain, either directly or indirectly in cases where pain and insomnia are exacerbated by depression. In a survey of elderly Floridians, Gilbert et al. (1993) found that 3 percent of the population reported trouble sleeping because of pain or discomfort from dental problems, whereas in a Canadian study Locker and Grushka (1987) reported that 14.2 percent of those with acute or chronic oral-facial pain (or 5.5 percent of the total population) experienced sleep disturbance related to pain. Goulet et al. (1995) assessed sleep problems not specifically attributed to pain among adults in Quebec. The prevalence of sleep problems was 13 percent for persons with no temporomandibular (jaw) pain. The proportion with sleep disturbance among persons with jaw pain increased with the severity of the pain, rising from 20 percent for those with mild pain, to 32 percent for moderate pain, and 59 percent for severe pain.

Psychosocial Dimensions

The social and psychological dimensions of well-being and quality of life are deeply intertwined in everyday life and so are considered together here. Findings are reported for those oral conditions where the most research has been conducted: dental diseases (primarily those that affect appearance or involve extensive tooth loss), pain conditions, craniofacial deformities (primarily clefting syndromes), and oral and pharyngeal cancers. Psychological distress associated with oral health problems has been measured through individual questions (e.g., items assessing worry) and through standardized psychological instruments such as the Beck Depression Inventory (Beck 1967) or the Symptom Checklist 90 (SCL-90) (Derogatis 1983). Overall, the results point to a poorer quality of life and a tendency to avoid social contact as a result of concerns over facial appearance. Persistent pain has similar isolating and depressing effects.

Cultural Significance of Teeth

Cultural beliefs and customs are a major influence on the psychosocial effects of dental disease on individuals. In a historical essay on the art of pulling teeth, Kunzle (1989) noted that "the tooth has always been accorded a special, even magical, role among all peoples and at all times, and has stood for power, and its loss for loss of power." The practice of hiding or burying a lost tooth was based on the fear that its

recovery by an enemy could be used magically to inflict harm on the tooth owner. Remnants of that belief persist in the ritual of the tooth fairy, who rescues the hidden baby tooth and leaves a reward. The potency of teeth is well recorded in the metaphors of language. As Ziolkowski (1976) observed, "if something sets our teeth on edge or if someone casts an insult in our teeth, we can gnash our teeth in anger, show our teeth belligerently, grit our teeth resolutely, take the bit in our teeth, arm ourselves to the teeth, and fight tooth and nail in the teeth of great danger and, with luck, escape by the skin of our teeth."

The perception of healthy teeth contrasts sharply with that of diseased teeth. The sixteenth-century French surgeon Ambroise Paré remarked that "toothache was the greatest and most eternal of all pains . . . the fiery torture of the damned in hell" (Kunzle 1989); and a preacher of that time saw the root of original sin in a rotting tooth, as quoted in Kunzle (1989): "We unfortunate humans: We all have toothache and suffer ever and always from the teeth with which Adam bit the forbidden apple." In the nineteenth century, Sir James Frazer reported that African tribal kings could not be crowned if they were symbolically emasculated by having a broken tooth, a theme Freud echoed in declaring that dreams of pulling teeth were symbols of castration (Ziolkowski 1976).

The link between diseased teeth and weakness, impotence, and even moral turpitude and sin has been analyzed by scholars exploring the vast legacy of dental themes in art and literature. These authors offer a variety of political, social, psychological, and economic interpretations of diseased versus healthy teeth, attesting to the seriousness with which humanity has invested dental pain and tooth loss.

Cultures have sanctioned a variety of alterations to teeth by shaping and filing, embedding with jewels, bleaching, capping, or providing orthodontic treatments to improve occlusion and aesthetics. These procedures have been variously designed to enhance the status, power, and attractiveness of the owner. Cultures have also dictated specific practices to indicate social position, such as the former Japanese custom of dyeing a married woman's teeth black to denote her marital status.

Decisions about aesthetic surgery (Kaw 1993), the definition of what constitutes a severe malocclusion, and the need for dental aesthetics (Cons et al. 1986) and surgical intervention (Strauss 1985) depend on social norms. Recently, researchers have begun to examine the perceived attractiveness of the human smile in terms of tooth length, shape, and color, lip line and plumpness, and tooth exposure

(Dunn et al. 1996). Although there are no reliable studies to document the proportion of people who would seek treatment for perceived defects, the growth of practitioner groups and dental supply companies that specialize in aesthetic dentistry attests to a substantial increase in cosmetic dental treatments. Follow-up studies of patients who have undergone tooth bleaching indicate high levels of satisfaction with the extent and stability of the color change (Haywood et al. 1994).

Dental Problems and Social Function

Given the importance of the mouth and teeth in verbal and nonverbal communication, diseases that disrupt their functions are likely to damage self-image and alter the ability to sustain and build social relationships. The social functions of individuals encompass a variety of roles, from the most intimate in dating and mating behaviors, to other interpersonal contacts, to participation in social or community activities. Dental diseases and disorders can interfere with these social roles at any or all levels (Patrick and Bergner 1990). Whether because of social embarrassment or functional problems, people with oral conditions may avoid conversation or laughing, smiling, or other nonverbal expressions that show their mouth and teeth.

Two surveys of different segments of the adult population in Ontario, Canada, addressed the issue of self-consciousness or embarrassment related to oral health problems. Both inquired into the frequency of such problems in the prior year. Among persons over 18 years old in North York, Ontario, 7 percent reported limiting conversation with others because of oral problems in the prior year, 15 percent reported that they had avoided laughing or smiling because of oral problems, and 19 percent reported being embarrassed at least sometimes by the appearance or health of their teeth or mouth (Locker and Miller 1994). Among persons 50 years of age and older throughout the province, 24 percent reported being self-conscious about their appearance at least occasionally because of problems with their teeth, mouth, or dentures, 22 percent reported feeling uncomfortable at least occasionally, and 13 percent reported that they avoided smiling (Locker and Slade 1993).

Recently, investigators have begun to develop quantitative tools to assess patterns of facial animation, including normal facial expressions, in order to assess functional impairment in these movements (Trotman et al. 1998)—and ultimately to address implications for quality of life. The effects of facial

expressions, particularly smiling, on social relationships warrant further research.

The more subtle aspects of social function may also be affected by relatively common oral conditions such as tooth loss. In a study of elderly people with tooth loss in the United Kingdom, 30 percent reported difficulty chewing. Although only 5 percent had changed their diet, 9 percent felt uncomfortable chewing in front of others, and 13 percent reported embarrassment during social interactions (Smith and Sheiham 1979). Bergendal (1989) acknowledged tooth loss in adults to be a serious life event with a more difficult period of readjustment than retirement. In a study by Fiske et al. (1998), 25 percent of people without teeth reported that they had avoided close relationships because of fear of rejection when their toothlessness was discovered. The authors reported that reactions to tooth loss by the elderly include lowered self-confidence and altered self-image, bereavement, dislike of appearance, and the perception of being more advanced in age. A national study surveying 3,000 homes in Great Britain regarding the effect of oral health on the quality of life was performed in 1997-98. Seventy percent of respondents reported that their oral health affected their quality of life—either positively or negatively. Older people in higher socioeconomic groups, especially those who had seen a dentist within the last year, were more likely to report an enhancing effect (McGrath and Bedi 1998). In a study of older Californians, people who viewed themselves as more attractive than other people their age were more likely to have a greater number of natural teeth. They were also more likely to report less emotional anxiety, to rate their health as excellent or very good, and to be less likely to wear full or partial removable dentures (Matthias et al. 1993).

Less severe oral conditions may also have adverse effects on social function. Table 6.2 summarizes self-reported levels of impact on personal contact and social integration from surveys of numerous population groups within the United States. Generally, the prevalence of adverse effects ranges from 1 to 10 percent, although 20 percent of senior center residents in California reported feeling uncomfortable when eating with others. Avoidance of smiling or conversation tended to be more frequent than limitation of social interaction. However, the largest amount of variation appeared to be among population subgroups, even within studies. For example, within the Boston area, Veterans Affairs ambulatory care patients in the Veterans Health Study had substantially higher prevalence rates of

adverse effects than community-dwelling, healthy men enrolled in the Normative Aging Study.

The effects on people's perceived ability to perform their usual social roles appear to be more subtle (Table 6.2). Depending on the phrasing of questions and the population subgroup surveyed, the prevalence of limitation in social roles varied from 0 to 5 percent, although as many as 10 percent of Veterans Affairs patients reported that their daily activities were affected because of oral conditions.

There are very few studies of the effects of dental treatment on specific forms of social function. One study of handicapped adults in the United Kingdom did, however, report large improvements in five aspects of social functioning following provision of

general dental care, including prosthodontic care (Fiske et al. 1990).

Impacts of Oral-Facial Pain

Acute pain from dental caries can usually be treated effectively or, as in the case of recurrent aphthous ulcers (canker sores), will resolve over time. Chronic pain in the oral-facial region presents a different situation in that the cause may not be removable and treatment focuses on pain management. Researchers have compared chronic oral-facial pain to other types of pain in terms of severity and emotional impact. In a study of adult enrollees of a large health maintenance organization, Von Korff et al. (1988)

TABLE 6.2
Reported impacts of oral conditions on social function among U.S. population groups

Social Function Dimension(s) Assessed	Percentage of Population Group Reporting Effect		
	Good Effect	Bad Effect	No Effect
Intimacy			
Elderly Persons in North Carolina ^a			
Perceived effect of mouth on:			
Sex appeal	75	21	5
Kissing	78	18	4
Romantic relationships	81	17	3
Personal contact/social integration			
Persons Aged 18 to 61 in Five U.S. Sites ^b			
Avoid conversation		4	
Personal contact/social integration			
Elderly Persons in California ^c			
Reported impacts due to oral conditions:			
Senior Center Residents		Medicare Enrollees	
Limiting contact with people	10		1
Feeling uncomfortable eating with others	20		4
Personal contact/social integration			
Elderly Persons in Florida ^d			
Avoid laughing or smiling		6	
Avoid conversation		3	
Social roles			
Limit activities			
		5	
Selected Populations in Boston Area^e			
Personal contact/social integration			
Normative Aging Study		Veterans Health Study	
Women's Health Project			
Avoidance of conversation	2	10	7
Social interaction affected	1	9	7
Social roles			
Daily activities affected	1	10	10
Elderly Persons in North Carolina^f			
Personal contact/social integration			
African Americans		Whites	
Avoided smiling	10		3
Unable to enjoy people's company	5		1
Avoided going out with others	3		0
Less tolerant of others	3		0
Social roles			
Difficulty doing jobs	1		0
Unable to work	3		0

^a Strauss and Hunt 1993.

^d Gilbert et al. 1993.

^b Gooch et al. 1989.

^e Kressin 1997.

^c Atchison and Dolan 1990.

^f Hunt et al. 1995.

compared pain in the temporomandibular region to back pain, headache, chest pain, and abdominal pain on a number of dimensions. The usual intensity of temporomandibular disorder (TMD) pain (4.3 on a 10-point scale) was similar to that of chest pain (4.3) and back pain (4.7); abdominal pain and headache were rated as more intense (5.1 and 5.9, respectively). TMD pain and back pain were more persistent than other pains, with 28 percent of persons with TMD pain (and 29 percent of those with back pain) reporting pain on more than half of the days in the prior 6 months. The daily duration of pain was also relatively high for TMD pain, with 27 percent reporting pain for 9 or more hours per day; this proportion was roughly the same as for headache and back pain. In this setting, persons with TMD pain sought care for their pain at about the same rate as persons with other pain, with about one quarter having sought care in the past 6 months and about 60 percent having sought care at some time in the past.

Locker and Grushka (1987) reported that more than 70 percent of persons with all types of acute and chronic pain worried about their dental health. Using scales of the Symptom Checklist 90 (SCL-90), age- and sex-standardized to the population under study, persons in the general population who reported TMD pain had substantially higher levels of anxiety and depression than those without a current pain condition (Von Korff et al. 1988). In the same study, items from the SCL-90 were used to assign an algorithm for diagnosis of depression. The percentage of persons with TMD who met the criteria for a possible diagnosis of major depression was 11 percent, compared with 2 percent among those without a current pain condition and 3 to 5 percent in the general population. Recent studies suggest that depression in TMD patients follows the onset of symptoms and reflects uncertainty about the cause(s) and the lack of effective treatments.

Social Responses to Facial Appearance

Just as cultural considerations color an individual's response to dental disease and tooth loss, so, too, does culture play a major role in the psychosocial impact of craniofacial deformities. More than any other body part, the face bears the stamp of individual identity, a fact neurobiology confirms in identifying an area of the brain that is dedicated to the recognition of faces.

In ancient Greece the face was seen as the mortal reflection of the gods, and those faces deemed most beautiful were also judged to be morally superior. Although cultures differ in the details, there appear

to be some invariant factors in the judgment of facial beauty and deformity (Stafford et al. 1989), and they are learned early in life. One U.S. study concluded that perceptions of what does and does not constitute an attractive face are established by age 7 (Cross and Cross 1971).

Aristotle also thought that the face, because of its position in the uppermost part of the body, pointed to the highest, most spiritual parts of the cosmos. Something of this mystical belief is preserved in linguistic traditions that regard what is "higher" and "ascending" as nobler than what is "lower" and "descending," whether in reference to body parts, human feelings, or classes and castes in society.

But if facial beauty is the mark of truth, moral superiority, nobility, and the soul, what of the opposite? Faces judged ugly or disharmonious, or those marred or scarred by birth defects or injury, have been associated with defects in character, intelligence, and morals. There is a long tradition of reading character on the basis of facial and head shapes according to one or another school of "physiognomics." Some schools likened human faces to animal species, such as cats, monkeys, or horses, endowing the human with the traits of the animal in question. Others constructed facial geometries that purported to show which races were nobler, which closer to animal ancestors. During the nineteenth century, Cesare Lombroso, an Italian physician and criminologist, developed a measuring scheme linking certain facial types to criminality (Magli 1989).

Given such a tradition, it is not surprising that people continue to "judge a book by its cover." A large body of research indicates that attractiveness has an important effect on psychological development and social relationships (Berscheid 1980). Noteworthy among early social psychological studies was the demonstration that physical attractiveness plays a key role in social expectations (Clifford and Walster 1973). Schoolteachers who were asked to evaluate the educational potential of students from school records and facial photographs judged attractive students to have higher educational potential and social ability than unattractive students. This finding has been replicated in a variety of school settings and grade levels.

The process of attributing positive characteristics to physically attractive persons is called the "beauty is good hypothesis." The positive impact of attractive appearance has been demonstrated to influence a wide range of social activities, from legal proceedings (Sigall and Ostrove 1975) to psychotherapeutic prognosis. This body of research indicates that appearance affects social expectations not only in educa-

tional performance, but also in vocational, marital, legal, and health care endeavors. However, the concept may not apply to all personal attributes. Bennett and Stanton (1993) found that appearance may have little effect on perceptions of intelligence or honesty.

Discrimination and stigmatization have historically characterized social responses to deformities. People whose appearance is significantly different from what society considers "normal" continue to be stigmatized. Examples in literature abound, in fable and myth, novels, movies, and plays (e.g., *Beauty and the Beast*, *The Elephant Man*, *The Hunchback of Notre Dame*, *The Phantom of the Opera*, and *Scarface*). Goffman (1963) provided a theory of stigmatization useful in understanding social responses to human differences and health conditions (Ablon 1981). Persons seen as deviant experience social devaluation associated with prejudices about the causes and consequences of congenital and acquired deformities (Macgregor 1974).

Ethnographic studies of facial deformities demonstrate marked variation in how birth defects are explained and treated (Cheng 1990, Meyerson 1990, Schepher-Hughes 1990, Toliver-Weddington 1990). Many cultures have regarded birth defects and the appearance of multiple births as ill omens, with the infants fated to be abandoned or killed. Even today, when it is understood that birth defects may result from an inherited genetic disorder, decisions about what that means and what if anything should be done about it are affected by religious or spiritual beliefs (Strauss 1988).

Progress in dentistry, medicine, and surgery has raised expectations that deformed or injured persons can be treated to enhance their appearance. Although critics have objected to the high value that U.S. society places on attractiveness and youthful appearance, and on how this emphasis influences opportunity, advocates of remediation counter that social values are hard to change and that individuals must adapt to a society's norms and stereotypes. Corrective surgery does produce positive assessments from others (Berscheid and Gangestad 1982). People in postoperative photographs following reconstructive and plastic surgery were seen as kinder, more sexually appealing, more likely to be better marriage partners, and more employable and successful than the same individuals in preoperative views.

Cleft Lip/Palate and Malocclusion

Persons with cleft lip/palate have a variety of problems related to appearance, eating, and speech. Studies of self-concept (Broder and Strauss 1989, Kapp-Simon 1986), psychosocial development (Richman and Eliason 1982, Richman et al. 1988), and social perception by peers (Schneiderman and Harding 1984, Tobiasen 1987), parents and teachers (Mitchell et al. 1984, Schneiderman and Auer 1984), and the public (Middleton et al. 1986) highlight the psychosocial problems of children with cleft lip/palate (Broder and Richman 1987).

These problems often continue into adolescence, with appearance and speech remaining problematic (Richman et al. 1985), even when the individual has had comprehensive care (Strauss et al. 1988). Studies show an association between cleft lip/palate and the increased reporting of conduct problems at home (Tobiasen and Hiebert 1984) and of behavioral and learning problems at school (Tobiasen et al. 1987). Although children with cleft lip/palate have not been shown to suffer from a negative self-concept (Kapp 1979, Richman 1983) or psychological disorders, they often become socially inhibited and self-conscious (Richman and Eliason 1982).

Adolescent girls with cleft lip/palate have higher rates of social adjustment problems, particularly related to appearance (Leonard et al. 1991). Using psychological projection methods, one study (Pillemer and Cook 1989) also concluded that children with facial deformities may have inhibited personality styles as well as reduced expectations for success in social interactions. Kapp-Simon (1986) found that social relationships negatively influence overall self-esteem and that adjustment of adolescents is associated with the degree of inhibition (Kapp-Simon et al. 1992).

Many of the earliest studies of the consequences of oral disease examined the impact of developmental disorders such as facial clefts and malocclusion on personal contact and social integration. There are few outcome studies that demonstrate how social, marital, and occupational status may be affected in adults with facial deformities or cleft lip/palate. In studies dating from the 1970s, persons with cleft lip/palate achieved greater educational levels than did their fathers, but did not exceed their fathers' occupational status (McWilliams and Paradise 1973). Persons with cleft lip/palate were less upwardly mobile than their siblings and achieved lower adult incomes (Peter et al. 1975). Although persons with cleft

lip/palate had higher occupational desires, they had lower income expectations than did controls; adults felt socially inept and had a tendency to shy away from group activities (Van Demark and Van Demark 1970). Subtle forms of discrimination and stigmatization were experienced among adults with cleft lip/palate (McWilliams 1970).

Studies of marital status have indicated that persons with cleft lip/palate are less likely than nonaffected peers to date (Peter et al. 1975) or marry (McWilliams and Paradise 1973), and that when they marry, they marry later (Heller et al. 1981, Peter and Chinsky 1974). Persons with cleft lip/palate were also more likely to remain childless or have fewer children (Peter and Chinsky 1974).

The evidence concerning anatomical misalignments of the jaws (e.g., overbite, open bite, crossbite) is less consistent, with some early studies showing no clear relationship between the malocclusion and job opportunities or social discrimination (Shaw et al. 1980a). However, people with severe malocclusions are likely to be teased and to have difficulty interacting socially. These problems improve following orthodontic and orthognathic treatment (Cunningham et al. 1996, Helm et al. 1985, Shaw et al. 1980b).

Oral and Pharyngeal Cancers

Surgical treatment for oral and pharyngeal cancers can result in functional impairment as well as permanent disfigurement. Problems may include the loss of part of the tongue, loss of taste, loss of chewing ability, difficulty in speaking, and pain. Furthermore, in addition to concerns about their function and their future, oral and pharyngeal cancer patients must cope with an altered appearance. In a study of patients who were disease-free from 6 months to 8 years following surgical tumor removal, Gamba et al. (1992) reported that those with more pronounced disfigurement had greater changes in self-image, a worsened relationship with their partner, reduced sexuality, and increased social isolation. A study by Bjordal and Kaasa (1995) also noted that 30 percent of oral and pharyngeal cancer patients were still experiencing psychological distress 7 to 11 years after treatment. Depression, too, is frequent in cancer patients. Patients with oral and pharyngeal cancers are at an even greater risk for depression than other cancer patients, due to surgeries that alter their appearance (Gritz et al. 1999). Because oral and pharyngeal cancers are also frequently associated with chronic alcohol and tobacco use, depression may be related to withdrawal from these substances or to

preexisting psychopathology. Persistent pain, as noted earlier, may also be a contributing factor to depression.

Gritz et al. (1999) conducted a prospective analysis of changes in quality of life over time with the aim of identifying which factors might be predictive of future improvements or declines. Participants were 186 oral and pharyngeal cancer patients, all smokers or recent former smokers, diagnosed with primary carcinomas of the oral cavity, pharynx, or larynx. The patients were tested at baseline, at 1 month after radiation and/or surgery, and 1 year later (for a subset of 105 patients available for follow-up). Measures used included the Karnofsky Performance Scale, which uses expert judgments of functional performance scored from 0 to 100; the Cancer Rehabilitation Evaluation System Short Form, in which patients rate their quality of life along physical, psychosocial, marital, sexual, and medical interaction scales; the previously mentioned Performance Status Scale for Head and Neck Cancer Patients (which includes scales for eating and speaking); and the Profile of Mood States, in which patients rate their feelings over the previous week, yielding analyses that enable scaling along six mood states: tension-anxiety, depression-dejection, anger-hostility, confusion-bewilderment, and vigor-activity. Results indicated that in spite of functional improvement on some scales over time, there was continued dysfunction in speech and eating. Patients also reported declines in marital and sexual functioning, as well as an increase in alcohol use. Interestingly, the best predictor of quality of life 1 year after treatment was the scores obtained after initial smoking cessation advice was given, while the patients were undergoing treatment and in recovery. Other predictors were treatment type (quality of life was generally poorer for radiation patients) and score on the vigor subscale of the Profile of Mood States. The investigators concluded that medical follow-up must integrate tailored psychological and behavioral interventions to achieve better quality of life for oral and pharyngeal cancer patients.

Indirect Economic Costs

The financial impact of oral disease on quality of life is easiest to quantify in terms of direct per capita costs of oral disease and treatment as well as the costs of publicly supported dental care programs. The Health Care Financing Administration includes these costs in its annual total health care expenditure reports. These expenditures are presented in Chapter 9, and the related data on utilization of care are given

in Chapter 4. Estimating the indirect costs associated with oral health conditions and treatments, such as disability days or lost productivity, is more difficult. Several methodologies are used to estimate these costs, although relatively few studies have actually estimated the indirect costs associated with mortality or lost productivity due to dental conditions or treatments (Reisine and Locker 1995).

The annual National Health Interview Survey routinely collects data on disability days associated with selected conditions. As shown in Table 6.3, there were 3.7 days of restricted activity per 100 employed persons 18 years and older reported in the United States in 1996 associated with an acute dental condition, as defined by a dental symptom or treatment visit. Restricted activity days were most prevalent among adults aged 18 to 24 years, women, blacks, and individuals with annual incomes of less than \$10,000. Compared to the 624.0 restricted activity days per 100 persons per year for all acute conditions, the 3.7 restricted activity days for dental conditions represent a relatively small loss on an individual basis. They do, however, add up to a sizable number of days lost from work or school every year for the population as a whole.

In addition, Table 6.3 presents the number of bed days and work-loss days per 100 employed persons aged 18 and older. Also, for youths 5 to 17 years of age, 3.1 days of school were lost per year.

An important aspect of identifying the specific health-related quality of life burden of oral disease will be to acquire more data on how the overall health-related quality of life of persons with oral disease differs from that of healthy persons, those with other diseases and activity limitations, and those with co-morbid oral and general health problems. The CDC's "Healthy Days" measures and population

data—used by all states since 1993 on the Behavioral Risk Factor Surveillance System (BRFSS) and recently added to the National Health and Nutrition Examination Survey (NHANES)—offer the potential of examining quality of life outcomes in relation to oral health and other disease measures used in these surveys (Gift 1996). These productivity-related measures ask about the number of recent days when physical health was not good, mental health was not good, and activities were limited. These measures were found to be valid in use with general populations and among older persons and adults with disabilities (Moriarty and Zack 1999). Related measures on activity limitations and recent days of pain, depression, anxiety, sleeplessness, and vitality are also asked by about half of all BRFSS states and—when used with oral disease measures—could help to identify the impact of oral disease prevention programs at the state and local levels.

In the 1996 National Health Interview Survey, the percentage of all acute conditions that are medically attended for all ages is 67.9 percent. "Medically attended" is defined as having contacted a physician (or other provider) or having a condition that causes a person to cut back on activities for at least half a day. For acute dental conditions, 59.6 percent are medically attended for all ages (NCHS 1996).

The most dramatic oral diseases by virtue of their high mortality rates are oral and pharyngeal cancers. The Centers for Disease Control and Prevention estimated in 1988 that 16.2 years of life were lost per person dying of cancer of the oral cavity and pharynx (CDC 1991). This exceeds the average for all cancer sites, which was 15.4 years lost.

Researchers are beginning to assess costs associated with chronic craniofacial conditions such as periodontal diseases, pain syndromes, and congenital anomalies. These estimates may also include the costs to caregivers who take time off from work to attend to these needs or to take children to the dentist. Recent estimates put the lifetime costs of multiple surgeries and the other medical, dental, and rehabilitation therapies typical of the team approach to the habilitation of individuals with cleft lip or palate at a minimum of \$101,000 (Waitzman et al. 1996). Overall costs of chronic pain conditions in America are estimated to be \$79 billion (Bonica 1990). Given the prevalence of headaches and

TABLE 6.3
Disability days due to all acute conditions and acute dental conditions,
United States, 1996

	All Acute Conditions		Acute Dental Conditions	
	Total Days (in thousands)	Days per 100 Persons	Total Days (in thousands)	Days per 100 Persons
School-loss days ^a	152,305	296.9	1,611	3.1 ^b
Work-loss days ^c	358,377	284.0	2,442	1.9 ^b
Bed days ^d	717,868	271.7	4,602	1.7 ^b
Restricted activity ^d	1,648,932	624.0	9,705	3.7 ^b

^a Youths 5 to 17 years of age.

^b Figure does not meet NCHS standard of reliability or precision.

^c Currently employed persons 18 years and older.

^d Persons of all ages.

Source: NCHS 1996.

temporomandibular disorders, the amount representing chronic oral-facial pain would certainly be in the billions.

RATINGS OF ORAL HEALTH

Researchers use two ways to assess how individuals rate their oral health: global ratings and satisfaction ratings. Global ratings employ a ranking scale, with excellent health at one end and poor health at the other. Satisfaction ratings are more akin to oral-health-related quality of life measures insofar as they ask individuals how satisfied (or dissatisfied) they are with their oral health status in relation to symptoms, physical function, appearance, social function, and psychological status.

Global Ratings

The dental survey conducted in connection with the International Collaborative Survey II (ICS II) asked younger (35 to 44 years) and older adults (65 to 74 years) of various ethnic groups at three sites in the United States to rate their oral health from 1 (excellent) to 6 (poor). The vast majority of younger and older white adults rated their oral health as good to excellent (82 and 80 percent, respectively, in Baltimore, and 71 and 68 percent, respectively, in San Antonio). In contrast, American Indians, Hispanics, and African Americans were more likely to rate their oral health as fair or poor. More detailed analysis found a significant relationship between oral health ratings and perceived general health, dentate status, importance of oral health, income, oral pain, symptoms, and dental visits (Chen et al. 1997).

Studies in the United States (Bloom et al. 1992, Chen et al. 1997, Gift et al. 1997, Matthias et al. 1995) have related global ratings of oral health to demographic, clinical, and psychosocial factors. A special supplement on oral health in the National Health Interview Survey in 1989 asked participants to rate their oral health as excellent, very good, good, fair, or poor (Bloom et al. 1992). The majority of people (67 percent) rated their oral health as excellent or very good, 23 percent rated it good, and only 10 percent rated it fair or poor. Men, younger people, and more frequent users of dental services tended to rate their oral health better than women, older adults, and less frequent users. African Americans, Hispanics, and American Indians were less likely to rate their oral health positively than were whites in similar geographic locations (Atchison and Gift 1997).

A study of Medicare participants in California (Matthias et al. 1995) also found that when asked to

rate oral health as excellent, very good, good, fair, or poor, most people rated it as good or above (74 percent). Interestingly, the relationship between self-rated global oral health and clinical measures used to rate the severity of dental caries or periodontal disease was weak. The finding of such inconsistency has been confirmed in other studies (Gooch et al. 1989, Reisine and Bailit 1980, Rosenberg et al. 1988) and was noted by Wilson and Cleary (1995). The most important correlates of self-rated oral health were worry about teeth and appearance, race, education, general health status, and depression scores.

Satisfaction Ratings

According to Jokovic and Locker (1997), "expressions of satisfaction and dissatisfaction are important oral health status indicators since they synthesize objective health states, subjective responses, and culturally based values and expectations." ICS I represented an early effort at assessing satisfaction with dental status (Arnlijot and WHO 1985). Specifically, it looked at satisfaction with teeth and gingiva among adults and adolescents in metropolitan and non-metropolitan areas in 10 countries. With the exception of Japanese participants, satisfaction with teeth and gingiva among adolescents was fairly high, but the percentage of adults satisfied with teeth among participants from all countries was relatively low. A decade later, ICS II (Chen et al. 1997) again assessed whether younger and older adults were satisfied with the way their teeth looked. American Indians were the least satisfied with the appearance of their teeth. Other community surveys of satisfaction (Barenthin 1977, Gilbert et al. 1994, Jokovic and Locker 1997, Murtomaa and Laine 1985, Rosenoer and Sheiham 1995, Van Waas et al. 1994) show that most people are satisfied with the performance and appearance of their teeth.

ORAL-HEALTH-RELATED QUALITY OF LIFE MEASURES

Much of the research on oral health and quality of life focuses on the negative impact of craniofacial diseases and disorders, diverting attention from the positive effects of good oral health. Although it could be argued that the benefits are self-evident, few studies have investigated how people value oral health. Strauss and Hunt (1993) found that older adults in North Carolina felt that the presence of teeth enhanced their appearance, ability to eat, and enjoyment of food, and that teeth also had a positive effect on comfort, confidence, speech, enjoyment, and

longevity. More generally, a large multisite study in the United States found that most adults across ethnic groups believe in the seriousness of oral disease and the importance of oral health for general health (Davidson et al. 1996, Nakazono et al. 1997).

During the last decade, researchers have identified and described specific effects of oral disease on quality of life, and there are now at least 11 questionnaires designed to measure oral-health-related quality of life (Slade et al. 1998). These range from the three-item Rand Dental Health Index (pain, worry, conversation) to the 49-item Oral Health Impact Profile, which includes items relating to function, pain, physical disability, psychological disability, social disability, and handicap.

Other researchers have used generic quality of life questionnaires (Reisine and Weber 1989) or analyzed oral health survey data, statistically summarizing clinical measures with self-reported symptoms, perceptions, and behaviors to create scales of oral-health-related quality of life (Gift et al. 1997, Gilbert et al. 1997). An essential requirement for these analyses is the establishment of theoretical models that describe and distinguish among quality of life dimensions.

Although the questionnaires have been used in population studies, there is increased emphasis on the need to incorporate concepts of quality of life into outcome research, using the questionnaires in longitudinal or intervention studies to examine changes in quality of life following provision of dental care. Individual items from the questionnaires can be used in describing the impact of oral health status on specific functions. For example, the questions concerning eating dysfunction presented in Table 6.1 are derived from such questionnaires. Multidimensional indicators also can be used to produce quantitative scores that indicate the severity of the impact caused by oral health problems. Because there are seldom any reference values or population norms to indicate thresholds at which such scores represent "abnormal" or "severe" impacts on quality of life, the results are more valuable for analytic studies examining trends and associations than for describing impacts of oral health within populations.

Surveys in the United States and elsewhere have used multidimensional questionnaires and have revealed consistent correlations between reduced quality of life and poorer clinical oral status and reduced access to dental care. Table 6.4 provides an overview of these studies, their populations, and descriptions of the assessment tools used. In the studies using varied tools, there are consistent relationships between quality of life and standard epi-

demiological indices of missing teeth, decayed teeth, and periodontal disease—conditions that are linked to a lack of dental care. In several studies, infrequent dental visits and problem-motivated dental visits were found to be independently associated with reduced quality of life in multivariate models (Gilbert et al. 1997, Slade and Spencer 1994a). Lower socioeconomic status was an additional explanatory factor in five studies (Atchison and Dolan 1990, Gilbert et al. 1997, Gooch et al. 1989, Leao and Sheiham 1995, Locker and Slade 1994).

All of the studies in Table 6.4 used cross-sectional designs to analyze associations with quality of life; it is thus not possible to infer a causal link between quality of life and the factors under study. Information about change in quality of life from those questionnaires is available from only two population-based longitudinal studies. In one, a study of elderly Floridians, small and statistically nonsignificant changes in the Geriatric Oral Health Assessment Index were observed following an oral health promotion program (Dolan 1997). In the second, a 2-year longitudinal study of elderly South Australians, change in quality of life was measured using the Oral Health Impact Profile. The study did not find that regular dental attendees had consistently better patterns of change than episodic dental attendees (Slade and Spencer 1994b).

In contrast, some information from experimental studies of carefully selected clinical samples indicates effects of specific dental treatments on quality of life. Using the Sickness Impact Profile in a U.S. study of 30 patients treated for TMDs, Reisine et al. (1989) found significant improvements for rest/sleep, home tasks, work, and leisure following treatment. A randomized clinical trial of 63 Canadians treated for TMDs compared changes in quality of life, measured using visual analog scales. The investigators found equivalent improvements in quality of life, which mirrored improvements in pain ratings, in all three treatment groups (Dao et al. 1994). A German study by Schliephake et al. (1996) reported improvements in quality of life, measured using a 22-item scale, among 85 patients following tumor removal and reconstructive surgery.

Two multidimensional scales have been specifically designed to assess global disability (e.g., disability days, pain interference with daily activities) related to recurrent or chronic pain. Both the Graded Chronic Pain Scale (Von Korff et al. 1992) and the Multidimensional Pain Inventory (Kerns et al. 1985) have been used to assess disability related to TMDs, as well as back pain and headache. Although TMD patients are generally found to have lower mean levels

of disability than patients with other types of pain using these scales, both scales can discriminate the full range of disability in TMDs. For example, the Graded Chronic Pain Scale is an ordinal scale that assigns persons with pain to hierarchical grades indicating low-intensity, nondisabling pain (grade I); high-intensity, nondisabling pain (grade II); high-disability, moderately limiting pain (grade III); and high-disability, severely limiting pain (grade IV). In a sample of HMO patients with TMDs, 41 percent were grade I, 43 percent grade II, 11 percent grade III, and 5 percent grade IV (Von Korff et al. 1992). That is, about 16 percent of patients seeking care for TMDs were moderately or severely limited by their pain.

HEIGHTENED EXPECTATIONS

Advances in public health and biomedical research in the late twentieth century have transformed our lives remarkably, adding years to the life span, providing cures for many diseases and disorders, and promising a new era of genetic medicine and bioengineering. The new field of science aimed at formulating con-

cepts and methods for determining health-related quality of life is a reflection of these advances. People living in societies that enjoy the benefits of this progress are responding with heightened expectations: They want to live long and live well, free of infirmity, impairment, disability, and handicap; they want an optimal quality of life and well-being. In terms of oral health, new social norms and cultural values now dictate that teeth should be retained over the lifetime, and oral pain and dysfunction forever banished. Quality of life measures applied to oral health outcomes will be used to further those goals.

FINDINGS

Examination of efforts to characterize the functional and social implications of oral and craniofacial diseases reveals the following findings:

- Oral health is related to well-being and quality of life as measured along functional, psychosocial, and economic dimensions. Diet, nutrition, sleep, psychological status, social interaction, school, and work are affected by impaired oral and craniofacial health.

TABLE 6.4
Studies of multidimensional quality of life measures

	Population Studied	Numbers of Individuals	Assessment Tool	Description of Assessment Tool
Gooch et al. 1989	U.S. insured adults aged 18-61 years	902 female 756 male	Rand Dental Health Index	Three dental questions written to represent factors contributing to adverse effects of dental disease
Atchison and Dolan 1990	California Medicare recipients aged 65+	1,000 female 755 male	Geriatric Oral Health Assessment Index (GOHAI)	A series of 12 questions measuring patient-reported oral functional problems
Hunt et al. 1995	N.C. elderly aged 70+	440	Oral Health Impact Profile (OHIP)	A comprehensive measure of self-reported dysfunction consisting of 49 questions
Kressin et al. 1996	Male veterans aged 47+	1,242 male	Oral Health-related Quality Of Life (OHQOL)	A brief global assessment of the impact of oral conditions consisting of three items
Gift et al. 1997	U.S. aged 18+	760 female 555 male	1981 Health Resources and Services Administration study	Multidimensional concept using data from a large national sample
Gilbert et al. 1997	Floridians aged 45+	491 female 383 male	Oral Disadvantage Assessment	Eight self-reported measures of avoidance in daily activities due to decrements in oral health
Locker and Miller 1994	Canadians aged 18+	299 female 244 male	Subjective Oral Health Status Indicators	Five oral health status indicators based on WHO's International Classification of Impairments, Disabilities and Handicaps
Locker and Slade 1994	Canadians aged 50+	168 female 144 male	Oral Health Impact Profile (OHIP)	A comprehensive measure of self-reported dysfunction consisting of 49 questions
Slade and Spencer 1994b	Australians aged 60+	660 female 557 male	Oral Health Impact Profile (OHIP)	A comprehensive measure of self-reported dysfunction consisting of 49 questions
Leao and Sheiham 1995	Brazilians aged 35-44	303 female 359 male	Dental Impact on Daily Living (DIDL)	Thirty-six questions that assess the oral health impacts on daily living
Coates et al. 1996	Australian dental patients	635 (+795 previously surveyed)	Oral Health Impact Profile (OHIP)	A comprehensive measure of self-reported dysfunction consisting of 49 questions

- Cultural values influence oral and craniofacial health and well-being and can play an important role in care utilization practices and in perpetuating acceptable oral health and facial norms.

- Oral and craniofacial diseases and their treatment place a burden on society in the form of lost days and years of productive work. Acute dental conditions contribute to a range of problems for employed adults, including restricted activity, bed days, and work loss, and school loss for children. In addition, conditions such as oral and pharyngeal cancers contribute to premature death and can be measured by years of life lost.

- Oral and craniofacial diseases and conditions contribute to compromised ability to bite, chew, and swallow foods; limitations in food selection; and poor nutrition. These conditions include tooth loss, diminished salivary functions, oral-facial pain conditions such as temporomandibular disorders, alterations in taste, and functional limitations of prosthetic replacements.

- Oral-facial pain, as a symptom of untreated dental and oral problems and as a condition in and of itself, is a major source of diminished quality of life. It is associated with sleep deprivation, depression, and multiple adverse psychosocial outcomes.

- Self-reported impacts of oral conditions on social function include limitations in verbal and non-verbal communication, social interaction, and intimacy. Individuals with facial disfigurements due to craniofacial diseases and conditions and their treatments may experience loss of self-image and self-esteem, anxiety, depression, and social stigma; these in turn may limit educational, career, and marital opportunities and affect other social relations.

- Reduced oral-health-related quality of life is associated with poor clinical status and reduced access to care.

REFERENCES

- Ablon J. Stigmatized health conditions. *Soc Sci Med [Med Anthropol]* 1981 Jan;15B(1):5-9.
- Aday LA, Forthofer RN. A profile of black and Hispanic subgroups' access to dental care: findings from the National Health Interview Survey. *J Public Health Dent* 1992;52(4):210-5.
- Agerberg G, Carlsson GE. Symptoms of functional disturbances of the masticatory system. A comparison of frequencies in a population sample and in a group of patients. *Acta Odontol Scand* 1975;33(4):183-90.
- Andersen RM, Davidson PL. Ethnicity, aging and oral health outcomes: a conceptual framework. *Adv Dent Res* 1997 May;11(2):203-9.
- Arnlijot HA, World Health Organization (WHO), editors. *Oral health care systems: an international collaborative study*. London: Quintessence; 1985.
- Atchison KA, Dolan TA. Development of the Geriatric Oral Health Assessment Index. *J Dent Educ* 1990 Nov;54(11):680-7.
- Atchison KA, Gift HC. Perceived oral health in a diverse sample. *Adv Dent Res* 1997 May;11(2):272-80.
- Barenthin I. Dental health status and dental satisfaction. *Int J Epidemiol* 1977 Mar;6(1):73-9.
- Beck AT. *Depression: clinical, experimental, and theoretical aspects*. New York: Hoeber; 1967.
- Bennett ME, Stanton ML. Psychotherapy for persons with craniofacial deformities: can we treat without theory? *Cleft Palate Craniofac J* 1993 Jul;30(4):406-10.
- Bergendal B. The relative importance of tooth loss and denture wearing in Swedish adults. *Community Dent Health* 1989;6:103-11.
- Bergner M, Bobbitt RA, Carter WB, Gilson BS. The Sickness Impact Profile: development and final revision of a health status measure. *Med Care* 1981 Aug;19(8):787-805.
- Berscheid E. Overview of the psychological effects of physical attractiveness. In: Lucker GW, Ribbens KA, McNamara JA, editors. *Psychological aspects of facial form*. Proceedings of a sponsored symposium honoring Professor Robert E. Moyers; 1980 Feb and Mar; Ann Arbor: Center for Human Growth and Development, The University of Michigan; 1980, 1981. Craniofacial growth series; monograph no. 11. p. 1-23.
- Berscheid E, Gangestad S. The social psychological implications of facial physical attractiveness. *Clin Plast Surg* 1982 Jul;9(3):289-96.
- Bjordan K, Kaasa S. Psychological distress in head and neck cancer patients 7-11 years after curative treatment. *Br J Cancer* 1995 Mar;71(3):592-7.
- Blaum CS, Fries BE, Fiatorone MA. Factors associated with low body mass index and weight loss in nursing home residents. *J Gerontol Med Sci* 1995;50:M162-8.
- Bloom B, Gift HC, Jack SS. Dental services and oral health. *Vital Health Stat* 10 1992 Dec;(183):1-95.
- Bonica JJ. General considerations of chronic pain. In: Bonica JJ, Loeser JD, Chapman CR, Fordyce WE, editors. *The management of pain*. Vol. 1. Philadelphia: Lea and Febiger; 1990. p. 180-96.
- Broder HL, Richman LC. An examination of mental health services offered by cleft/craniofacial teams. *Cleft Palate J* 1987 Apr;24(2):158-62.
- Broder HL, Strauss RP. Self-concept of early primary school age children with visible or invisible defects. *Cleft Palate J* 1989 Apr;26(2):114-7.
- Brodeur JM, Laurin D, Vallee R, Lachapelle D. Nutrient intake and gastrointestinal disorders related to masticatory performance in the edentulous elderly. *J Prosthet Dent* 1993 Nov;70(5):468-73.

- Carlsson GE. Masticatory efficiency: the effect of age, the loss of teeth and prosthetic rehabilitation. *Int Dent J* 1984 Jun;34(2):93-7.
- Centers for Disease Control (CDC). Cigarette smoking among adults—United States, 1988. *JAMA* 1991 Dec 11;266(22):3113-4.
- Chauncey HH, Muench ME, Kapur KK, Wayler AH. The effect of the loss of teeth on diet and nutrition. *Int Dent J* 1984;34(2):98-104.
- Chen M, Andersen RM, Barmes DE, Leclercq MH, Lyttle CS. Comparing oral health care systems—a second international collaborative study. Geneva: World Health Organization; 1997.
- Cheng LR. Asian-American cultural perspectives on birth defects: focus on cleft palate. *Cleft Palate J* 1990 Jul;27(3):294-300.
- Clifford M, Walster E. The effect of physical attractiveness on teacher expectations. *Soc Educ* 1973;46:248-58.
- Coates E, Slade GD, Goss AN, Gorkic E. Oral conditions and their social impact among HIV dental patients. *Aust Dent J* 1996 Feb;41(1):33-6.
- Cons NC, Jenny J, Kohout FJ. DAI: the dental aesthetic index. Iowa City: University of Iowa College of Dentistry; 1986.
- Cross JF, Cross J. Age, sex, race and the perception of facial beauty. *Dev Psychol* 1971;5:433-9.
- Cunningham SJ, Crean SJ, Hunt NP, Harris M. Preparation, perceptions, and problems: a long-term follow-up study of orthognathic surgery. *Int J Adult Orthodon Ortognath Surg* 1996;11(1):41-7.
- Dao TT, Lavigne GJ, Charbonneau A, Feine JS, Lund JP. The efficacy of oral splints in the treatment of myofascial pain of the jaw muscles: a controlled clinical trial. *Pain* 1994 Jan;56(1):85-94.
- Davidson PL, Andersen RM. Determinants of dental care utilization for diverse ethnic and age groups. *Adv Dent Res* 1997 May;11(2):254-62.
- Davidson PL, Andersen RM, Marcus M, Atchison KA, Reifel N, Nakazono T, Rana H. Indicators of oral health in diverse ethnic and age groups: findings from the International Collaborative Study of Oral Health Outcomes (ICS-II) USA research locations. *J Med Syst* 1996 Oct;20(5):295-316.
- Demers M, Bourdages J, Brodeur JM, Benigeri M. Indicators of masticatory performance among elderly complete denture wearers. *J Prosthet Dent* 1996 Feb;75(2):188-93.
- Derogatis LR. Symptom Checklist-90. Administration, scoring and procedures manual for the revised version. Baltimore: Clinical Psychometric Research; 1983.
- Diehnelt D, Kiyak HA, Beach BH. Predictors of oral health behaviors among elderly Japanese-Americans. *Spec Care Dentist* 1990;10:112-8.
- Dolan T. The sensitivity of the Geriatric Oral Health Assessment Index to dental care. *J Dent Educ* 1997;61(1):37-46.
- Dunn WJ, Murchison DF, Broome JC. Esthetics: patients' perceptions of dental attractiveness. *J Prosthodont* 1996 Sep;5(3):166-71.
- Eisenberg DM, Davis RB, Ettner SL, Appel S, Wilkey S, Van Rompay M, Kessler RC. Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. *JAMA* 1998 Nov 11;280(18):1569-75.
- Ernest SL. Dietary intake, food preferences, simulated salivary flow rate, and masticatory ability in older adults with complete dentitions. *Spec Care Dentist* 1993 May-Jun;13(3):102-6.
- Febres C, Echeverri EA, Keene HJ. Parental awareness, habits, and social factors and their relationship to baby bottle tooth decay. *Pediatr Dent* 1997 Jan-Feb;19(1):22-7.
- Feldman RS, Kapur KK, Alman JE, Chauncey HH. Aging and mastication: changes in performance and in the swallowing threshold with natural dentition. *J Am Geriatr Soc* 1980 Mar;28(3):97-103.
- Fiske J, Gelbier S, Watson RM. The benefit of dental care to an elderly population assessed using a socio-dental measure of oral handicap. *Br Dent J* 1990;168(4):153-6.
- Fiske J, Davis DM, Frances C, Gelbier S. The emotional effects of tooth loss in edentulous people. *Brit Dent J* 1998;184(2):90-3.
- Fries JF, Spitz PW, Young DY. The dimensions of health outcomes: the health assessment questionnaire, disability and pain scales. *J Rheumatol* 1982 Sep-Oct;9(5):789-93.
- Gamba A, Romano M, Grosso IM, Tamburini M, Cantu G, Molinari R, Ventafridda V. Psychosocial adjustment of patients surgically treated for head and neck cancer. *Head Neck* 1992 May Jun;14(3):218-23.
- Garrett NR, Kapur KK, Perez P. Effects of improvements of poorly fitting dentures and new dentures on patient satisfaction. *J Prosthet Dent* 1996 Oct;76(4):403-13.
- Geertman ME, Boerrigter EM, Van't Hof MA, Van Waas MA, van Oort RP, Boering G, Kalk W. Two-center clinical trial of implant-retained mandibular overdentures versus complete dentures—chewing ability. *Community Dent Oral Epidemiol* 1996 Feb;24(1):79-84.
- Gift HC. Quality of life—an outcome of oral health care? *Public Health Dent* 1996;56(2):67-8.
- Gift HC, Atchison KA. Oral health, health, and health-related quality of life. *Med Care* 1995 Nov;33(11 Suppl):NS57-77.
- Gift HC, Atchison KA, Dayton CM. Conceptualizing oral health and oral health-related quality of life. *Soc Sci Med* 1997 Mar;44(5):601-8.
- Gilbert GH, Heft MW, Duncan RP. Oral signs, symptoms, and behaviors in older Floridians. *J Public Health Dent* 1993 Summer;53(3):151-7.
- Gilbert GH, Heft MW, Duncan RP, Ringelberg ML. Perceived need for dental care in dentate older adults. *Int Dent J* 1994 Apr;44(2):145-52.

- Gilbert GH, Duncan RP, Heft MW, Dolan TA, Vogel WB. Oral disadvantage among dentate adults. *Community Dent Oral Epidemiol* 1997 Aug;25(4):301-13.
- Godson JH, Williams SA. Oral health and health related behaviours among three-year-old children born to first and second generation Pakistani mothers in Bradford, UK. *Community Dent Health* 1996 Mar;13(1):27-33.
- Goffman E. *Stigma: notes on the management of spoiled identity*. Englewood Cliffs (NJ): Prentice-Hall; 1963. p. 2-3.
- Gooch BF, Dolan TA, Bourque LB. Correlates of self-reported dental health status upon enrollment in the Rand Health Insurance Experiment. *J Dent Educ* 1989 Nov;53(11):629-37.
- Goulet JP, Lavigne GJ, Lund JP. Jaw pain prevalence among French-speaking Canadians and related symptoms of temporomandibular disorders. *J Dent Res* 1995 Nov;74(11):1738-44.
- Gritz ER, Carmack CL, de Moor C, Coscarelli A, Schacherer CW, Meyers EG, Abemayor E. First year after head and neck cancer: quality of life. *J Clin Oncol* 1999;17(1):352-60.
- Guyatt AH, Feeny DH, Patrick DL. Measuring health-related quality of life. *Ann Intern Med* 1993 Apr 15;118(8):622-9.
- Harris EF, Woods MA, Robinson QC. Dental health patterns in an urban Midsouth population: race, sex and age changes. *Quintessence Int* 1993 Jan;24(1):45-52.
- Haywood VB, Leonard RH, Nelson CF, Brunson WD. Effectiveness, side effects and long-term status of nightguard vital bleaching. *J Am Dent Assoc* 1994 Sep;125(9):1219-26.
- Helkimo E, Carlsson GE, Helkimo M. Chewing efficiency and state of dentition. A methodologic study. *Acta Odontol Scand* 1978;36(1):33-41.
- Heller A, Tidmarsh W, Pless IB. The psychosocial functioning of young adults born with cleft lip or palate. A follow-up study. *Clin Pediatr* 1981;20:459-65.
- Helm S, Kreiborg S, Solow B. Psychosocial implications of malocclusion: a 15-year follow-up study in 30-year-old Danes. *Am J Orthod* 1985 Feb;85(2):110-8.
- Henderson GE, et al., editors. *The social medicine reader*. Durham (NC): Duke University Press; 1997. p. 6-11.
- Hildebrandt GH, Dominguez BL, Schork MA, Loesche WJ. Functional units, chewing, swallowing, and food avoidance among the elderly. *J Prosthet Dent* 1997 Jun;77(6):588-95.
- Hufford DJ. Folk medicine in contemporary America. In: Kirkland J, et al., editors. *Herbal and magical medicine*. Durham: Duke University Press; 1992. p. 14-31.
- Hunt RJ, Slade GD, Strauss RP. Differences between racial groups in the impact of oral disorders among older adults in North Carolina. *J Public Health Dent* 1995 Fall;55(4):205-9.
- Hunt SM, McEwen J, McKenna SP. Measuring health status: a new tool for clinicians and epidemiologists. *J R Coll Gen Pract* 1985 Apr;35(273):185-8.
- Idowu AT, Graser GN, Handelman SL. The effect of age and dentition status on masticatory function in older adults. *Spec Care Dentist* 1986 Mar-Apr;6(2):80-3.
- Jokovic A, Locker D. Dissatisfaction with oral health status in an older adult population. *J Public Health Dent* 1997 Winter;57(1):40-7.
- Kapp K. Self-concept of the cleft lip and/or palate child. *Cleft Palate J* 1979 Apr;16(2):171-6.
- Kapp-Simon K. Self-concept of primary-school-age children with cleft lip, cleft palate, or both. *Cleft Palate J* 1986 Jan;23(1):24-7.
- Kapp-Simon KA, Simon DJ, Kristovich S. Self-perception, social skills, adjustment, and inhibition in young adolescents with craniofacial anomalies. *Cleft Palate Craniofac J* 1992 Jul;29(4):352-6.
- Kaw E. Medicalization of racial features: Asian-American women and cosmetic surgery. *Med Anthropol Q* 1993;7(1):74-89.
- Kayser AF. Shortened dental arches and oral function. *J Oral Rehabil* 1981 Sep;8(5):457-62.
- Keller HH. Malnutrition in institutionalized elderly: how and why? *J Am Geriatr Soc* 1993 Nov;41(11):1212-8.
- Kelly M, Bruerd B. The prevalence of baby bottle tooth decay among two Native American populations. *J Public Health Dent* 1987 Spring;47(2):94-7.
- Kerns RD, Turk DC, Rudy TE. The West Haven-Yale Multidimensional Pain Inventory (WHYMPI). *Pain* 1985 Dec;23(4):345-56.
- Kiyak HA. Age and culture: influences on oral health behavior. *Int Dent J* 1993 Feb;43(1):9-16.
- Kleinman A. *Sickness as cultural semantics: issues for an anthropological medicine and psychiatry*. In: Ahmed P, Coelho G, editors. *Toward a new definition of health: psychosocial dimensions*. New York: Plenum; 1979. p. 53-65.
- Kolasa K. I won't cook turnip greens if you won't cook kielbasa: food behavior or Polonia and its health implications. In: Bauwens E, editor. *The anthropology of health*. St. Louis: Mosby; 1978. p. 130.
- Kressin NR. Oral health-related quality of life measure (OHQOL). In: Slade GD, editor. *Measuring oral health and quality of life*. Chapel Hill: University of North Carolina, School of Dental Ecology; 1997.
- Kressin N, Spiro A 3rd, Bosse R, Garcia R, Kazis L. Assessing oral health-related quality of life: findings from the normative aging study. *Med Care* 1996 May;34(5):416-27.
- Kunzle D. The art of pulling teeth in the seventeenth and nineteenth centuries: from public martyrdom to private nightmare and political struggle? Part 3. In: Feher M, with Nadaff R, Razi N, editors. *Cambridge (MA): distributed by MIT Press; 1989. p. 28-89*.
- Leake JL. An index of chewing ability. *J Public Health Dent* 1990 Summer;50(4):262-7.

- Leao A, Sheiham A. Relation between clinical dental status and subjective impacts on daily living. *J Dent Res* 1995 Jul;74(7):1408-13.
- Lee J, Kiyak HA. Oral disease beliefs, behaviors, and health status of Korean-Americans. *J Public Health Dent* 1992 Spring;52(3):131-6.
- Lee KL, Schwarz E, Mak KY. Improving oral health through understanding the meaning of health and disease in a Chinese culture. *Int Dent J* 1993 Feb;43(1):2-8.
- Leonard BJ, Brust JD, Abrahams G, Sielaff B. Self-concept of children and adolescents with cleft lip and/or palate. *Cleft Palate Craniofac J* 1991 Oct;28(4):347-53.
- List MA, Ritter-Sterr C, Lansky SB. A performance status scale for head and neck cancer patients. *Cancer* 1990;66:564-9.
- Locker D, Grushka M. The impact of dental and facial pain. *J Dent Res* 1987 Sep;66(9):1414-7.
- Locker D, Miller Y. Subjectively reported oral health status in an adult population. *Community Dent Oral Epidemiol* 1994 Dec;22(6):425-30.
- Locker D, Slade G. Prevalence of symptoms associated with temporomandibular disorders in a Canadian population. *Community Dent Oral Epidemiol* 1988 Oct;16(5):310-3.
- Locker D, Slade G. Oral health and the quality of life among older adults: the Oral Health Impact Profile. *J Can Dent Assoc* 1993 Oct;59(10):830-3,837-8,844.
- Locker D, Slade G. Association between clinical and subjective indicators of oral health status in an older adult population. *Gerodontology* 1994 Dec;11(2):108-14.
- Macgregor FC. Transformation and identity, the face and plastic surgery. New York: Quadrangle/New York Times; 1974. p. 119.
- Magli P. The face and the soul. In: Feher M, with Nadaff R, Razi N, editors. *Fragments for a history of the human body. Part 2.* Cambridge (MA): distributed by MIT Press; 1989. p. 87-127.
- Marcus SE, Drury TF, Brown LJ, Zion GR. Tooth retention and tooth loss in the permanent dentition of adults: United States, 1988-1991. *J Dent Res* 1996 Feb;75(Spec No):684-95.
- Matthias RE, Atchison KA, Schweitzer SO, Lubben JE, Mayer-Oakes A, De Jong F. Comparisons between dentist ratings and self-ratings of dental appearance in an elderly population. *Spec Care Dentist* 1993 Mar-Apr;13(2):53-60.
- Matthias RE, Atchison KA, Lubben JE, De Jong F, Schweitzer SO. Factors affecting self-ratings of oral health. *J Public Health Dent* 1995;55(4):197-204.
- McEwen B. Stress and the brain. In: Conlan R, editor. *States of mind.* New York: John Wiley; 1999. p. 81-102.
- McGrath C, Bedi R. A study of the impact of oral health on the quality of life of older people in the UK—findings from a national survey. *Gerodontology* 1998;15(2):93-8.
- McWilliams BJ. Psychosocial development and modification. Washington: American Speech and Hearing Association; 1970. ASHA Reports No. 5.
- McWilliams BJ, Paradise LP. Educational, occupational, and marital status of cleft palate adults. *Cleft Palate J* 1973 Jul;10:223-9.
- Meenan RF, Gertman PM, Mason JH. Measuring health status in arthritis. The arthritis impact measurement scales. *Arthritis Rheum* 1980 Feb;23(2):146-52.
- Meyerson MD. Cultural considerations in the treatment of Latinos with craniofacial malformations. *Cleft Palate J* 1990 Jul;27(3):279-88.
- Middleton GF, Lass NJ, Starr P, Pannbacker M. Survey of public awareness and knowledge of cleft palate. *Cleft Palate J* 1986 Jan;23(1):58-62.
- Mitchell CK, Lott R, Pannbacker M. Perceptions about cleft palate held by school personnel: suggestions for in-service training development. *Cleft Palate J* 1984 Oct;21(4):308-12.
- Moriarty D, Zack M. Validation of the Centers for Disease Control and Prevention's Healthy Days measures [abstract]. *Qual Life Res* 1999;8(7):617.
- Murtomaa H, Laine P. Differentiating dental satisfaction in Finns by means of discriminant analyses. *Community Dent Oral Epidemiol* 1985 Feb;13(1):7-10.
- Nakazono TT, Davidson PL, Andersen RM. Oral health beliefs in diverse populations. *Adv Dent Res* 1997 May;11(2):235-44.
- National Center for Health Statistics (NCHS). Current estimates from the National Health Interview Survey, 1996. Series 10, no. 200. Hyattsville (MD): Public Health Service; 1996.
- Navia JM. Carbohydrates and dental health. *Am J Clin Nutr* 1994 Mar;59(Suppl 3):719S-275.
- Patrick DL, Bergner M. Measurement of oral health status in the 1990s. *Annu Rev Public Health* 1990;11:165-83.
- Peter JP, Chinsky RR. Sociological aspects of cleft palate adults: I. Marriage. *Cleft Palate J* 1974 Jul;11(0):295-309.
- Peter JP, Chinsky RR, Fisher MJ. Sociological aspects of cleft palate adults: III. Vocational and economic aspects. *Cleft Palate J* 1975 Apr;12:193-9.
- Pillemer FG, Cook KV. The psychosocial adjustment of pediatric craniofacial patients after surgery. *Cleft Palate J* 1989 Jul;26(3):201-7, Discussion 207-8.
- Ranta K, Tuominen R, Paunio I. Perceived oral health status and ability to chew among an adult Finnish population. *Gerodontology* 1987 Jun;3(3):136-9.
- Reisine ST, Bailit HL. Clinical oral health status and adult perceptions of oral health. *Soc Sci Med [Med Psychol Med Sociol]* 1980 Dec;14A(6):597-605.
- Reisine S, Locker D. Social, psychological and economic impact of oral conditions and treatments. In: Cohen LK, Gift HC, editors. *Disease prevention and oral health promotion: socio-dental sciences in action.* Copenhagen: Munksgaard; 1995. p. 33-72.

- Reisine ST, Weber J. The effects of temporomandibular joint disorders on patients' quality of life. *Community Dent Health* 1989 Sep;6(3):257-70.
- Reisine ST, Fertig J, Weber J, Leder S. Impact of dental conditions on patients' quality of life. *Community Dent Oral Epidemiol* 1989 Feb;17(1):7-10.
- Richman LC. Self-reported social, speech, and facial concerns and personality adjustment of adolescents with cleft lip and palate. *Cleft Palate J* 1983 Apr;20(2):108-12.
- Richman LC, Eliason M. Psychological characteristics of children with cleft lip and palate: intellectual, achievement, behavioral and personality variables. *Cleft Palate J* 1982 Oct;19(5):249-57.
- Richman LC, Holmes CS, Eliason MJ. Adolescents with cleft lip and palate: self-perceptions of appearance and behavior related to personality adjustment. *Cleft Palate J* 1985 Apr;22(2):93-6.
- Richman LC, Eliason MJ, Lindren SD. Reading disability in children with clefts. *Cleft Palate J* 1988 Jan;25(1):21-5.
- Ripa LW. Nursing caries: a comprehensive review. *Pediatr Dent* 1988 Dec;10(4):268-82.
- Ritchie CS, Silliman RA, Joshipura KJ, Millen B, Douglass CW. Oral health predictors of significant weight loss among community-dwelling older adults. In press. *J Gerontol Med Sci*; 2000.
- Rosenberg D, Kaplan S, Senie R, Badner V. Relationships among dental functional status, clinical dental measures, and generic health measures. *J Dent Educ* 1988 Nov;52(11):653-7.
- Rosenoer LM, Sheiham A. Dental impacts on daily life and satisfaction with teeth in relation to dental status in adults. *J Oral Rehabil* 1995 Jul;22(7):469-80.
- Scheper-Hughes N. Difference and danger: the cultural dynamics of childhood stigma, rejection, and rescue. *Cleft Palate J* 1990 Jul;27(3):301-7;discussion 307-10.
- Schliephake H, Ruffert K, Schneller T. Prospective study of the quality of life of cancer patients after intraoral tumor surgery. *J Oral Maxillofac Surg* 1996 Dec;54(6):664-9;discussion 669-70.
- Schneiderman CR, Auer KE. The behavior of the child with cleft lip and palate as perceived by parents and teachers. *Cleft Palate J* 1984 Jul;21(3):224-8.
- Schneiderman CR, Harding JB. Social ratings of children with cleft lip by school peers. *Cleft Palate J* 1984 Jul;21(3):219-23.
- Selikowitz HS. Acknowledging cultural differences in the care of refugees and immigrants. *Int Dent J* 1994 Feb;44(1):59-61.
- Shaw WC, Addy M, Ray C. Dental and social effects of malocclusion and effectiveness of orthodontic treatment: a review. *Community Dent Oral Epidemiol* 1980a Feb;8(1):36-45.
- Shaw WC, Meek SC, Jones DS. Nicknames, teasing, harassment and the salience of dental features among school children. *Br J Orthod* 1980b Apr;7(2):75-80.
- Ship JA, Duffy V, Jones JA, Langmore S. Geriatric oral health and its impact on eating. *J Am Geriatr Soc* 1996 Apr;44(4):456-64.
- Sigall H, Ostrove N. Beautiful but dangerous: effects of offender attractiveness and nature of the crime in juristic judgement. *J Pers Soc Psychol* 1975;31:410-4.
- Slade GD, Spencer AJ. Development and evaluation of the Oral Health Impact Profile. *Community Dent Health* 1994a Mar;11(1):3-11.
- Slade GD, Spencer AJ. Social impact of oral conditions among older adults. *Aust Dent J* 1994b Dec;39(b):358-64.
- Slade GD, Spencer AJ, Davies MJ, Burrow D. Intra-oral distribution and impact of caries experience among South Australian school children. *Aust Dent J* 1996 Oct;41(5):343-50.
- Slade GD, et al. Conference: assessing oral health outcomes: measuring health status and quality of life: final report. 1998. Rockville (MD): U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, Center for Research Dissemination and Liaison.
- Smith JM, Sheiham A. How dental conditions handicap the elderly. *Community Dent Oral Epidemiol* 1979;7(6):305-10.
- Stafford BM, La Puma J, Schiedermaier DL. One face of beauty, one picture of health: the hidden aesthetic of medical practice. *J Med Philos* 1989 Apr;14(2):213-30.
- Strauss RP. Culture, rehabilitation, and facial birth defects: international case studies. *Cleft Palate J* 1985 Jan;22(1):56-62.
- Strauss R. Genetic counseling in the cross-cultural context: the case of highly observant Judaism. *Patient Educ Couns* 1988;11:43-52.
- Strauss RP, Hunt RJ. Understanding the value of teeth to older adults: influences on the quality of life. *J Am Dent Assoc* 1993 Jan;124(1):105-10.
- Strauss RP, Broder H, Helms RW. Perceptions of appearance and speech by adolescent patients with cleft lip and palate and by their parents. *Cleft Palate J* 1988 Oct;25(4):335-42.
- Sullivan DH, Martin W, Flaxman N, Hagen J. Oral health problems and involuntary weight loss in a population of frail elderly. *J Am Geriatr Soc* 1993;41:725-31.
- Tinanoff N, O'Sullivan DM. Early childhood caries: overview and recent findings. *Pediatr Dent* 1997 Jan-Feb;19(1):12-6.
- Tobiasen JM. Social judgments of facial deformity. *Cleft Palate J* 1987 Oct;24(4):323-7.
- Tobiasen JM, Hiebert JM. Parent's tolerance for the conduct problems of the child with cleft lip and palate. *Cleft Palate J* 1984 Apr;21(2):82-5.
- Tobiasen JM, Levy J, Carpenter MA, Hiebert JM. Type of facial cleft, associated congenital malformations, and parents' ratings of school and conduct problems. *Cleft Palate J* 1987 Jul;24(3):209-15.

- Toliver-Weddington G. Cultural considerations in the treatment of craniofacial malformations in African Americans. *Cleft Palate J* 1990 Jul;27(3):289-93.
- Trotman CA, Stohler CS, Johnston LE Jr. Measurement of facial soft tissue mobility in man. *Cleft Palate Craniofac J* 1998 Jan;35(1):16-25.
- Van Demark DR, Van Demark AA. Speech and socio-vocational aspects of individuals with cleft palate. *Cleft Palate J* 1970 Jan;7:284-99.
- van der Bilt A, Olthoff LW, Bosman F, Oosterhaven SP. Chewing performance before and after rehabilitation of post-canine teeth in man. *J Dent Res* 1994 Nov;73(11):1677-83.
- Van Waas M, Meeuwissen J, Meeuwissen R, Kayser A, Kalk W, Van't Hof M. Relationship between wearing a removable partial denture and satisfaction in the elderly. *Community Dent Oral Epidemiol* 1994 Oct;22(5 Pt 1):315-8.
- Von Korff M, Dworkin SF, Le Resche L, Kruger A. An epidemiologic comparison of pain complaints. *Pain* 1988 Feb;32(2):173-83.
- Von Korff M, Ormel J, Keefe FJ, Dworkin SF. Grading the severity of chronic pain. *Pain* 1992 Aug;50(2):133-49.
- Waitzman NJ, Scheffler RM, Romano PS. The cost of birth defects: estimates of the value of prevention. Lanham (MD): University Press of America; 1996.
- Wayler AH, Kapur KK, Feldman RS, Chauncey HH. Effects of age and dentition status on measures of food acceptability. *J Gerontol* 1982 May;37(3):294-9.
- Wayler AH, Muench ME, Kapur KK, Chauncey HH. Masticatory performance and food acceptability in persons with removable partial dentures, full dentures and intact natural dentition. *J Gerontol* 1984 May;39(3):284-9.
- Wilson IB, Cleary PD. Linking clinical variables with health-related quality of life. A conceptual model of patient outcomes. *JAMA* 1995 Jan 4;273(1):59-65.
- Witter DJ, van Elteren P, Kayser AF, van Rossum MJ. The effect of removable partial dentures on the oral function in shortened dental arches. *J Oral Rehabil* 1989 Jan;16(1):27-33.
- World Health Organization (WHO). Constitution of the World Health Organization. Geneva: WHO Basic Documents; 1948.
- Zborowski M. Cultural components in responses to pain. *J Health Soc Behav* 1952;8:16-30.
- Ziolkowski T. The telltale teeth: psychodontia to sociodontia. *PMLA* 1976 Jan;91:9-22.

PAR ■ FOUR

How Is Oral Health Promoted and Maintained and How Are Oral Diseases Prevented?

Safe and effective disease prevention measures for the common dental diseases exist and allow individuals, health care providers, and the community each to play a role, one that is enhanced by active partnerships among these groups. Unfortunately, not everyone has access to these measures. For example, some 40 percent of the U.S. population resides in communities that do not have optimal fluoride levels in their water supply.

Chapter 7 reviews the evidence for current prevention measures. Community water fluoridation remains an ideal public health measure, which benefits individuals of all ages and all socioeconomic strata. Other methods to deliver fluoride are reviewed, as is the use of dental sealants in caries prevention. The prevention of periodontal diseases and conditions such as oral and pharyngeal cancers and craniofacial injuries is at an early stage. Surveys of the knowledge and practices of the public and care providers reveal opportunities for enhanced education.

Attaining and maintaining oral health require a commitment to self-care and professional care. Chapter 8 highlights both individual responsibilities and emerging roles for health care providers. With greater understanding of the pathophysiology of oral diseases, providers can incorporate new preventive, diagnostic, and treatment strategies. These include developing risk assessment approaches for individual patients and adopting new strategies for the control of infections. Care providers are well positioned to instruct patients on tobacco cessation, appropriate dietary practices during pregnancy, and other healthful behaviors.

The professional provision of oral health care in America involves contributions from the dental, medical, and public health components. These are reviewed in Chapter 9, which focuses primarily on the dental component. A number of factors limit the capacity to improve the nation's oral health. Public assistance programs as currently designed are not meeting the oral health needs of eligible populations. A troubling lack of diversity exists in the oral health workforce, along with continued shortfalls in the number of men and women attracted to positions in oral health education and research. Correcting these limitations would contribute to increased access to care for underserved populations, enhanced preparation of future practitioners, and an expanded ability to pursue the many research questions generated in this report.

Community and Other Approaches to Promote Oral Health and Prevent Oral Disease

The remarkable improvements in oral health over the past half century reflect the strong science base for prevention of oral diseases that has been developed and applied in the community, in clinical practice, and in the home. This chapter presents the evidence for key preventive measures for those oral conditions that pose the greatest burden to U.S. society. Because the emphasis given to each condition discussed here reflects the extent of the evidence for the associated preventive measures, the chapter is heavily weighted toward the prevention and control of dental caries, for which multiple effective preventive modalities have been developed.

The dental profession has long championed disease prevention and health promotion approaches to oral health. The initial observations in the 1930s that people living in communities served by naturally fluoridated water had lower dental caries inspired the trailblazing clinical prevention studies of the 1940s and 1950s. Researchers compared whole cities agreeing to fluoridate their water supplies to control cities whose drinking water contained only trace amounts of fluoride. Five years into the studies, follow-up with schoolchildren who had been examined at baseline revealed dramatic reductions in dental caries in the children drinking fluoridated water, as compared to controls. The overwhelming success of the studies led to a widespread adoption of community water fluoridation in the United States as a high-benefit, low-cost preventive method that benefited old and young, rich and poor alike. It also provided momentum for health practitioners, researchers, industry, and public health directors to consider other kinds of community-wide, provider-based, and individual strategies aimed at improving oral and general health.

Most common oral diseases can be prevented through a combination of community, professional, and individual strategies. The strategies selected here

include disease prevention and health promotion interventions directed toward the public, practitioners, and policymakers to create a healthy environment, reduce risk factors, inform target groups, and improve knowledge and behaviors. They were selected on the basis of the significance of the health problem they were designed to prevent, whether in terms of prevalence, incidence, severity, cost, or impact on quality of life (see Chapters 4 and 6). Table 7.1 summarizes the strategies for the primary prevention of caries, periodontal diseases, oral and pharyngeal cancers, inherited disorders, and trauma, distinguishing among those that can be implemented community-wide, through health professionals, or through the exercise of individual responsibility. Some strategies can be applied at multiple levels. Box 7.1 provides a glossary of terms related to community health programs.

This chapter also includes a discussion of knowledge and practices of the public and health care providers regarding the three oral conditions about which we have the most knowledge. The purpose of this discussion is not to outline specific health promotion strategies to enhance knowledge and practices but to indicate the opportunities and needs for both broad-based and targeted health promotion programs and activities.

WEIGHING THE EVIDENCE THAT INTERVENTIONS WORK

Researchers, policymakers, and practitioners make judgments about whether a health intervention works based on estimates of its efficacy or effectiveness. Estimates of an intervention's efficacy are best based on randomized controlled trials, which may be conducted under ideal circumstances. Evidence for whether an intervention works when applied in the community at large is referred to as its effectiveness

(O'Mullane 1976). The distinction between efficacy and effectiveness is often blurred in dental public health programs because the studies and their settings can be very similar. Nevertheless, the major difference between the two lies in the degree of control exerted over factors that can affect results. Effectiveness studies more accurately reflect results that may be expected from the implementation of interventions.

The current trend in health care and public health is to base recommendations on evidence derived from systematic reviews of the literature and an assessment of the quality of evidence. The U.S. Preventive Services Task Force (1996) and the Canadian Task Force on the Periodic Health Examination (Ismail and Lewis 1993, Lewis and Ismail 1995) are examples of groups that have used systematic reviews to establish the evidence of efficacy or

TABLE 7.1
Community, provider, and individual strategies for primary prevention of key oral diseases and conditions

Community Strategies	Professional Strategies	Individual Strategies
Dental caries		
Community-wide health promotion interventions ^a	Counseling to follow measures to reduce risk of disease	Being informed about strategies to prevent disease
Fluoride use	Fluoride use	Fluoride use
Community water fluoridation	Prescriptions for fluorides (supplements or rinses)	Dentifrice
School-based dietary fluoride tablets	Gels and other high-fluoride topicals	Mouthrinse, over the counter
School-based fluoride mouthrinse	Topical remineralization solutions	
	Fluoride-containing restorative materials	
School-based and school-linked sealant programs	Provision of sealants Prescriptions for antimicrobial agents	Asking about sealants Use of antimicrobial agents
School-linked screening and referral	Individualized recall schedule	Self-initiated use of dental services
Periodontal diseases		
Community-wide health promotion interventions ^a	Counseling to follow measures to reduce risk of disease	Being informed about strategies to prevent disease
School-based personal hygiene, reinforcement of personal oral hygiene habits in Headstart or primary school classrooms	Control of plaque bacteria by mechanical means (prophylaxis or scaling) Chemical plaque control Chemotherapeutic agents	Oral hygiene measures Toothbrushing and flossing Toothbrushing with dentifrices Plaque control
School-linked screening and referral	Monitoring and early detection of disease	Self-initiated use of dental services
Oral and pharyngeal cancers		
Community-wide health promotion interventions ^a	Professional education and patient counseling on risk factors	Being informed about strategies to prevent disease Avoidance of tobacco use Reduction of alcohol use Use of sunscreen and lip protector
Cancer screening programs (such as health fairs)	Routine soft-tissue oral examination for early detection of precancerous lesions	Self-initiated use of dental services Request for cancer screening
Inherited disorders		
Early detection programs	Interdisciplinary early detection programs	
Trauma		
Community-wide health promotion interventions ^a	Professional education and patient counseling on risk factors	Being informed about strategies to prevent trauma
Mouth protector fittings for entire team	Fabrication of mouth protectors	Use of mouth protectors and helmets
^a Community-wide health promotion interventions (education, political, regulatory, and organizational) are directed toward the public, practitioners, and policymakers to create a healthy environment, reduce risk factors, inform target groups, and improve knowledge and behaviors.		

BOX 7.1

Glossary: The Nature of Community Health Programs

Community health programs are defined as health promotion and disease prevention activities that address health problems in populations. Community health programs often provide a level of organization and resources beyond those available to an individual. The programs thus complement personal care and professional services. Many programs target populations with limited access to professional services or limited resources to pay for services. Government agencies, religious organizations, charities, schools, foundations, and other private and public groups may spearhead such programs, tapping into the expertise, enthusiasm, and knowledge of community values of staff and volunteers. Some programs are sponsored by national, state, and local dental societies and their members.

Five terms related to community health programs—community, health promotion, health literacy, health education, and disease prevention—have been further articulated by experts in the field.

Community. According to Last (1995), a community is “a group of individuals organized into a unit, or manifesting some unifying trait or common interest.” The unit can be a town, a geographic area, the state, nation, or body politic (Last 1995). The unit may also be a selected subgroup, such as disadvantaged children living in a large city or women urged to have mammograms according to specified schedules.

In designing and implementing community programs, planners must take into consideration that no two communities are identical. In a classic expression of this concept, McGavran (1979) wrote that a community is “an entity different from every other community as an individual is different from his neighbor: different in its physical makeup, its geographic and demographic limitation, different in its social structure, its power structure, its governmental and legal structure, different in mental and emotional patterns, in its ethnic groups, its mores, its religious and nutritional patterns, and different in its educational procedure, its institutions, and its community organization.” On the other hand, communities may have similar risk factors for poor oral health, allowing common solutions to similar problems. Lessons learned in one community may be applicable to those with similar characteristics.

In recent years, investigators have begun to examine characteristics of communities, noting that some communities provide an environment that contributes to the overall health and well-being of the members, whereas others appear to be detrimental. All communities, however, have both positive and negative influences on health and well-being—the challenge is to minimize the negative factors and maximize the positive in each community. Healthy communities have been characterized as having a degree of openness and cooperation—neighbors helping neighbors. Healthy communities also are ones in which there are less extreme separations of individuals by social class (Wilkinson 1996).

Health Promotion. Health promotion is “any planned combination of education, political, regulatory, and organizational supports for action and conditions of living conducive to the health of individuals, groups, or communities” (Green and Kreuter 1999). Examples of broad-based health promotion activities include programs encouraging people of all ages to stop using tobacco, regulations requiring the use of mouthguards in contact sports, laws to prohibit tobacco sales to minors, and labels that indicate the amount of sugar in a product.

Health Literacy. Health literacy is “the capacity of individuals to obtain, interpret, and understand basic health information and services and the competence to use such information and services in ways which enhance health” (Joint Commission on National Health Education Standards 1995). Health literacy is correlated with general literacy, and both vary by educational achievement, socioeconomic status, race, and ethnicity. This is an important concern in a society that is becoming more diverse in terms of language, religion, culture, race, and ethnicity. Programs intending to serve, immigrants, for example, must attend to ensuring that information, programs, and systems are accessible, understandable, and culturally sensitive, particularly if the target audience for health information and services does not speak English, if there are unique cultural and religious beliefs at variance with those of the dominant culture, or if living arrangements are such that individuals lack access to sources of health information and care.

Health Education. Health education is an important part of health promotion. It is defined as “any planned combination of learning experiences designed to predispose, enable, and reinforce voluntary behavior conducive to health in individuals, groups, or communities” (Green and Kreuter 1999). Examples include the multiple campaigns to prevent tobacco use among youth. An example at the statewide level is Arizona’s promotion of the use of dental sealants with an educational campaign that says “Sealants Are in the Groove.”

Disease Prevention. The term *prevention* embodies the goal of promoting and preserving health and minimizing suffering and distress. Community health programs generally focus on either *primary prevention*—removing or reducing risks or providing protection from disease before it occurs—or *secondary prevention*—screening and early detection and intervention to arrest the progress of disease after it occurs. *Tertiary prevention*—rehabilitating and restoring structure and function—is provided in some community-based programs, such as clinical dental care organized and delivered under conditions determined by the community.

effectiveness of clinical preventive services for the purpose of making recommendations. Similar reviews of the evidence of effectiveness for community preventive services are currently under way by the Task Force on Community Preventive Services (2000). These reports provide clear statements about the evidence and recommendations for or against a given strategy.

The discussion in this chapter is more illustrative than comprehensive. Readers are encouraged to seek specific guidance from the reports of the U.S. Preventive Services Task Force where available. Furthermore, because of the interest in community preventive services, "expert opinion" about the merits of community interventions is included, even though the work of the Task Force on Community Preventive Services has not been completed. Expert opinion is formed by less systematic reviews of the literature or addresses interventions to be applied in settings other than those previously studied.

In particular, suggestions are offered for several interventions intended to reduce oral disease and promote oral health that reflect the opinion of experts who contributed to this report. Until findings from additional research are available, expert opinion remains the best guidance for community interventions where only efficacy studies have been done or where they were applied to populations with different attributes or risk factors than those of current interest. Also, expert opinion has been used where there is an interest in criteria that were not considered in previous efficacy studies, such as cost-effectiveness and practicality.

Readers interested in more detailed information about interventions in areas such as control of tobacco use or motor vehicle safety are directed to the upcoming report of the Task Force on Community Preventive Services (2000).

Interventions included in this chapter (and highlighted in Table 7.1) are those that have been shown to be effective in certain settings, but which can be applied in other settings. The anticipated benefits may be difficult to determine. In general, the per capita cost of an intervention is lower for community interventions and is usually a function of the number of people reached for a given level of professional effort. Effectiveness, however, is often a function of the risk characteristics of a given individual in the group receiving the intervention. Such risk factors are often easier to target by individual practitioners than by community programs. In the absence of contemporary data, the promotion of strategies deemed to be more cost-effective than others relies on the opinion of experts. Individual decision making

regarding self- or provider care further reflects the subjective value placed on the outcome of care. Therefore, it is not possible to make general statements about the superiority of any given approach.

PREVENTION AND CONTROL OF DENTAL CARIES

Although many caries prevention strategies, notably community water fluoridation and use of a fluoride-containing dentifrice, benefit adults and children alike, most of our understanding of the effectiveness of these strategies comes from the study of children, during a life stage when caries incidence is high. Caries prevention programs have been designed and evaluated for children and have used a variety of fluoride and dental sealant strategies applied separately and together. Because these strategies are complementary, their use in combination has the potential of virtually eliminating dental caries in all children. However, dental caries is a problem for all ages. Although direct evidence of caries preventive strategies in adults is limited, the evidence that does exist is consistent with expected effects based on experience with children. The Centers for Disease Control and Prevention (CDC) recently convened an expert work group to develop recommendations for modalities to prevent and control dental caries based on a review of publications selected by the work group and other experts. The resulting recommendations are summarized in Table 7.2, where they are organized according to quality of evidence, strength of recommendation, and target population in accordance with criteria adapted from the U.S. Preventive Services Task Force (CDC in press).

Fluoride

Fluoride reduces the incidence of dental caries and slows or reverses the progression of existing lesions (i.e., helps prevent cavities). Today, all Americans are exposed to fluoride to some degree, and there is little doubt that widespread use of fluoride has been a major factor in the overall decline in recent decades in the prevalence and severity of dental caries in the United States and other economically developed countries (Bratthall et al. 1996).

Fluoride is the ionic form of the element fluorine, the thirteenth most abundant element in the crust of the Earth. Because of its high affinity for calcium, fluoride is mainly associated with calcified tissues (i.e., bones and teeth). The ability of fluoride to inhibit, and even reverse, the initiation and progression of dental caries is well known. Fluoride's mech-

mechanisms of action include incorporation of fluoride into enamel preeruptively, inhibition of demineralization, enhancement of remineralization, and inhibition of bacterial activity in dental plaque.

A variety of theories regarding fluoride's mechanisms of action account for the range of fluoride products available (Burt and Eklund 1999, Stookey and Beiswanger 1995). The initial theory of action was based on the belief that incorporation of fluoride into the hydroxyapatite of developing tooth enamel in the preeruptive phase reduced the mineral's solubility, thereby increasing enamel resistance. Because of the length of time the tooth is at risk of caries during the posteruptive phase, however, the topical effects of fluoride are considered to predominate (Clarkson et al. 1996). These effects are based on fluoride's role in the aqueous phase around the tooth, both in saliva and in dental biofilm (plaque). Fluoride in plaque contributes to the remineralization of demineralized enamel when bound fluoride is released during an acid challenge, resulting in a more

acid-resistant enamel surface structure. Fluoride also has been shown to inhibit the process of glycolysis by which fermentable carbohydrates are metabolized by cariogenic bacteria to produce acid. All these effects occur after the tooth erupts, while it is functioning in the mouth, enabling fluoride to prevent caries over a lifetime in both children and adults.

The first use of fluoride for caries prevention was in 1945 in the United States and Canada, when the fluoride concentration was adjusted in the drinking water supplying four communities (Arnold et al. 1962, Ast and Fitzgerald 1962, Blayney and Hill 1967, Hutton et al. 1956). This public health approach followed a long period of epidemiologic studies of the effects of naturally occurring fluoride in drinking water (Burt and Eklund 1999).

The success of the community water fluoridation trials in reducing dental caries led to the development of other important fluoride-containing products, such as dietary supplements and, most notably, fluoride-containing dentifrices, in the early 1960s.

Fluoride-containing gels, solutions, pastes, and varnishes were also developed for topical use, either applied by professionals or self-applied at home or in other settings. All of these products were tested for safety and effectiveness in reducing caries. Products designed for professional use generally have higher concentrations and are used at less frequent intervals than those designed for self-application.

Controlled clinical trials from the 1940s through the 1970s documented the benefits of professionally applied fluoride in reducing dental caries, and several excellent reviews are available (Horowitz and Ismail 1996, Johnston 1994, Ripa 1990, Stookey and Beiswanger 1995). Professional application of fluoride is inherently more expensive than self-applied methods, so the use of such an approach for groups and individuals at low risk of dental caries is unlikely to be cost-effective. For patients at high risk of dental caries, however, professionally applied fluoride is still considered cost-effective. It is not clear whether fluoride varnishes and

TABLE 7.2
Quality of evidence, strength of recommendation, and target population of recommendation for each modality to prevent and control dental caries

Modality ^a	Quality of Evidence (grade)	Strength of Recommendation (code)	Target Population ^b
Community water fluoridation	II-1	A	All areas
School water fluoridation	II-3	C	Rural, nonfluoridated areas
Fluoridated dentifrices	I	A	All persons
Fluoride mouthrinses	I	A	High risk ^c
Fluoride supplements			
Pregnant women	I	E	None
Children aged <6 years	II-3	C	High risk
Children aged 6 to 16 years	I	A	High risk
Persons aged >16 years	N.A.	C	High risk
Fluoride gels	I	A	High risk
Fluoride varnishes	I	A	High risk
Dental sealants	I	A	High risk ^d

Notes: Criteria for quality of evidence and strength of recommendation designations are adapted from USPSTF as Table 5.3.

N.A. = no published studies of effectiveness of fluoride supplements in controlling dental caries among persons aged >16 years.

^a Assume that the modalities are used as directed in terms of dosage and age of user.

^b The quality of evidence for targeting some modalities to persons at high risk is grade III, representing the opinion of respected experts, and is based on considerations of cost-effectiveness that were not included in the studies establishing efficacy or effectiveness.

^c Groups believed to be at high risk for caries are members of families of low socioeconomic status (SES) or with low levels of parental education; those seeking dental care on an irregular basis; and persons without dental insurance or access to dental service. Individual factors contributing to increased risk are currently active dental caries; a history of high caries experience in older siblings or caregivers; exposed root surfaces; high levels of infection with cariogenic bacteria; impaired ability to maintain oral hygiene; reduced salivary flow due to medications, radiation treatment, or disease; and the wearing of orthodontic appliances or prostheses.

^d Assessment of risk is based on both patient and tooth-specific factors.

Source: Modified from CDC in press, and ASTDD 1995.

gels would be most efficiently used in clinical programs targeting groups at high risk of dental caries or whether they should be reserved for individual high-risk patients.

The U.S. Preventive Services Task Force (Greene et al. 1989, USPSTF 1996) and the Canadian Task Force on Periodic Health Examination (Lewis and Ismail 1995) affirm that there is strong evidence to support the major methods for providing fluoride to prevent dental caries.

The safety of fluoride is well documented and has been reviewed comprehensively by several scientific and public health organizations (Institute of Medicine (IOM) 1997, National Research Council (NRC) 1993, Newbrun 1996, U.S. Department of Health and Human Services (USDHHS) 1991, World Health Organization (WHO) 1984). When used appropriately, fluoride has been demonstrated to be both safe and effective in preventing and controlling dental caries. The IOM (1997) classified fluoride as a micronutrient, citing it, along with calcium, phosphorus, magnesium, and vitamin D, as an important constituent in maintaining health.

Appropriate use of fluoride products can minimize the potential for enamel fluorosis, a broad term applied to certain visually detectable changes in the opacity of tooth enamel associated with areas of fluoride-related developmental hypomineralization. There are also many developmental changes in enamel that are not fluoride-related (Fejerskov et al. 1990). Most enamel fluorosis seen today is of the mildest form, which affects neither aesthetics nor dental function. Cosmetically objectionable enamel fluorosis can occur when young children ingest higher than optimal amounts of fluoride, from any source, while tooth enamel is forming (up to age 6). Its occurrence appears to be most strongly associated with the total cumulative fluoride intake during the period of enamel development, but the condition's severity depends on the dose, duration, and timing of fluoride intake. Specific recommendations have been made to control fluoride intake by children during the years of tooth development (USDHHS 1991).

Fluoridation of Drinking Water

For more than half a century, community water fluoridation has been the cornerstone of caries prevention in the United States; indeed, CDC has recognized water fluoridation as one of the great public health achievements of the twentieth century (CDC 1999). All water contains at least trace amounts of fluoride.

Water fluoridation is the controlled addition of a fluoride compound to a public water supply to achieve a concentration optimal for dental caries prevention. In the 1940s, Dean et al. (1941) concluded that 1 ppm (part per million) fluoride was the optimal concentration for climates similar to that of the Chicago area; this concentration would significantly reduce the prevalence of dental caries with an acceptably low prevalence of enamel fluorosis. Current U.S. Public Health Service (USPHS) recommendations for fluoride use include an optimally adjusted concentration of fluoride in drinking water ranging from 0.7 to 1.2 ppm, depending on the mean maximum daily air temperature of the area (Galagan and Vermillion 1957, USDHEW 1962). A lower fluoride concentration is recommended for communities in warmer climates than cooler climates, because it is assumed that persons living in warmer climates drink more tap water.

Effectiveness

Numerous studies in naturally fluoridated areas preceded the field trials. There are no randomized, double-blind, controlled trials of water fluoridation because its community-wide nature does not permit randomization of people to study and control groups. Similar results have been derived from numerous well-conducted field studies by various investigators on thousands of subjects in different parts of the world. Conducting a study in which individuals are randomized to receive or not receive fluoridated water is unnecessary and is not feasible.

In 1945, Grand Rapids, Michigan, became the first city in the United States to fluoridate its water supply; the oral health of its schoolchildren was periodically compared with that of schoolchildren in the control city, Muskegon, Michigan. Dramatic declines in dental caries among children in Grand Rapids and three other cities conducting studies shortly thereafter led to fluoridation in many other cities. In an extensive review of 95 studies conducted between 1945 and 1978, Murray et al. (1991) reported the modal caries reduction following water fluoridation to be between 40 and 50 percent for primary teeth and 50 and 60 percent for permanent teeth. Newbrun (1989) reported on more than 60 studies conducted during the 1970s and early 1980s, limiting his review to those with concurrent control groups because of the continuing decline in dental caries in both fluoridated and nonfluoridated areas. Comparisons of fluoride-deficient and fluoridated communities in the United States, Australia, Britain, Canada, Ireland, and New Zealand have consistently demonstrated the

continued effectiveness of water fluoridation. Caries reductions ranged between 15 and 40 percent in fluoridated, as compared with fluoride-deficient, communities (USDHHS 1991).

Fluoridation also benefits middle-aged and older adults. Benefits to adults include reductions in both coronal and root caries. These benefits are important because older people typically experience gingival recession, which results in exposed root surfaces, which are susceptible to caries. In addition, tooth retention in older U.S. cohorts has increased in recent decades, so that the number of teeth at risk for caries in older age groups is also increasing. Finally, many medications used to treat chronic diseases common in aging have the side effect of diminished salivary flow, depriving teeth of the many protective factors in saliva.

Other evidence of the benefits of fluoridation comes from studies of populations where fluoridation has ceased. Examples in the United States, Germany, and Scotland have shown that when fluoridation is withdrawn and there are few other fluoride exposures, the prevalence of caries increases. In Wick, Scotland, which began water fluoridation in 1969 but stopped it in 1979, the caries prevalence in 5- to 6-year-olds with limited exposure to other sources of fluoride increased by 27 percent between 1979 and 1984. This was despite a national decline in caries and increased availability of fluoride-containing dentifrices (Kugel and Fischer 1997, Seppä et al. 1998, Stephen et al. 1987).

Costs and Cost-effectiveness

The increase in other fluoride exposures since water fluoridation was first introduced in 1945—particularly from fluoride-containing dentifrices, mouth-rinses, and foods and beverages processed using fluoridated water—has led to smaller differences in the prevalence of dental caries between people in fluoridated and those in nonfluoridated communities than in the past. Most public health experts believe that water fluoridation continues to be a highly cost-effective strategy, even in areas where the overall caries level has declined and the cost of implementing water fluoridation has increased (Burt 1989, CDC 1999).

Compared to the cost of restorative treatment, water fluoridation actually provides cost savings, a rare characteristic for community-based disease prevention strategies (Garcia 1989). The mean annual per capita cost of fluoridation ranges from \$0.68 for systems serving populations greater than 50,000 (large systems) and \$0.98 for systems serving

between 10,000 and 50,000 (medium systems), to \$3.00 for systems serving less than 10,000 (small systems) (reported in 1999 dollars) (Ringelberg et al. 1992). In 1992, approximately 60 percent of the U.S. population receiving fluoridated water was served by large systems, 31 percent by medium systems, and 9 percent by small systems (USDHHS 1993).

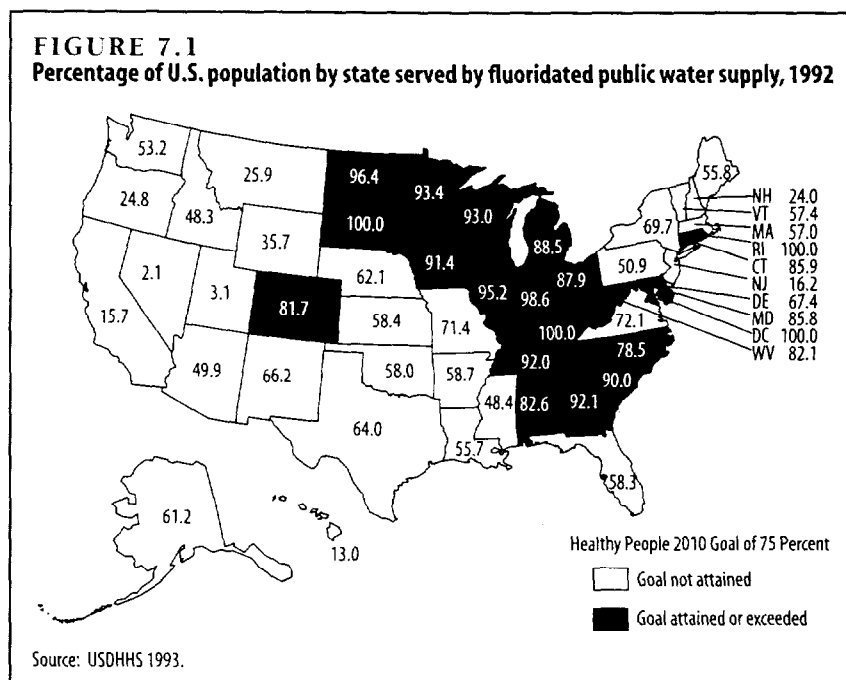
Access to Optimally Fluoridated Water in the United States

The most recent national data on the extent of community water fluoridation reflect the status of fluoridation in 1992 (see Figure 7.1 and Table 7.3). About 145 million people, or 62 percent of the population served by public water supplies, consume water with optimal fluoride levels. Of the 50 largest cities in the United States, 43 are fluoridated (Table 7.4). Residents of the seven unfluoridated cities in the group are among the almost 100 million persons in the United States who lack this method of caries prevention.

Although many states and large cities had been quick to implement fluoridation programs in the 1950s and 1960s, the trend then began to level off. In the absence of legislative mandates in most states and categorical federal funding, fluoridation decisions are left to the states, and frequently to local governments and city councils. Thus expansion of fluoridation in the United States is not simple and requires decisions at many levels. The national health promotion and disease prevention objectives in Healthy People 2010 (USDHHS 2000) call for increasing the percentage of Americans on public water supplies drinking fluoridated water from 62 to 75 percent—a 21 percent improvement (see Figure 7.1). This would mean adding 30 million people served by well over 1,000 community water systems to those who currently have access to fluoridated public water systems (USDHHS 1993).

Summary: Community Water Fluoridation

Epidemiological studies carried out during the last five decades provide strong evidence supporting the effectiveness of water fluoridation in preventing coronal and root caries in children and adults. Further support of effectiveness comes from studies that indicate that caries experience increases in communities that no longer fluoridate the water supply (and where there are few other exposures to fluorides). Given the modest cost of less than 1 dollar per person per year to fluoridate water systems serving most people, community water fluoridation is recommended as a very effective and cost-effective



the children were present for only portions of the day and year.

Although the strategy shares some of the advantages of community water fluoridation—serving rich and poor alike and requiring no action on the part of the children (other than drinking the water)—a number of disadvantages were evident from the outset. These included the limitations inherent in beginning exposure to fluoride only when children were of school age and then providing only intermittent exposure. Also, the possibility that the exposure would not confer benefits after the children left school was a concern. Practical considerations

TABLE 7.3
Population served by fluoride-adjusted and naturally fluoridated water, United States, 1992

Type of Fluoridation	Population	Number of Systems	Number of Communities
Adjusted	134.6 million	10,567	8,572
Natural	10.0 million	3,784	1,924
Both	144.6 million	14,351	10,496

Source: USDHHS 1993.

included the cost of operations, personnel, logistical difficulties, and mandatory water testing (CDC 1995). Moreover, the intervening decades have seen increased school consolidations, increased coverage of schools by community-wide water systems, declining numbers of children who could benefit from such programs, and a continuing general decline in dental caries in children. Another concern is that schools increasingly enroll preschoolers into daycare programs for which school water fluoridation at higher levels than for community water systems is not appropriate. Only four intervention studies evaluating the effectiveness of school water fluoridation have been published.

method of preventing coronal and root caries in children and adults. Moreover, water fluoridation benefits all residents served by community water supplies regardless of socioeconomic status. Few barriers to its implementation exist, with the important exception of the political opposition that the measure often engenders and certain technical difficulties and costs involved in fluoridating very small water systems.

Summary: School Water Fluoridation

Given the limitations of the evidence for effectiveness, as well as the difficulties of implementation and operation, school water fluoridation has limited application. Decisions to initiate or continue school fluoridation programs should be based on an assessment of present caries risk in the target school(s), alternative preventive modalities that may be available, and periodic evaluation of program effectiveness.

School Water Fluoridation

During the 1960s, 1970s, and 1980s, programs were initiated to bring the benefits of fluoride in drinking water to children living in homes supplied by well water and whose schools had independent water supplies. The idea was to adjust the fluoride content of the water supplies of the schools these children attended, especially consolidated rural schools, to levels higher than those that would be used for community water fluoridation, taking into account that

Dietary Fluoride Supplements

Dietary fluoride supplements are available as tablets that are swallowed or chewed, drops that are swallowed, and lozenges that dissolve slowly in the

mouth. They can provide topical and systemic fluoride for children in the absence of optimally fluoridated drinking water. In the United States, supplements are available by prescription only, to be used once a day beginning at 6 months and ending at age 16. According to a 1986 National Health Interview Survey (NHIS), slightly more than 16 percent of children younger than 2 years used fluoride dietary supplements (Nourjah et al. 1994).

The fluoride supplement dosage schedule in use in the United States was last revised by the American Dental Association (ADA) in 1994 (Table 7.5) (ADA 1995). This schedule, based on the level of fluoride in the community water supply and on the age of the child, has also been endorsed by the American Academy of Pediatric Dentistry and the American Academy of Pediatrics. Fluoride supplements should not be prescribed for individuals living in optimally fluoridated communities.

Effectiveness of Home Use

The current fluoride supplement dosage schedule does not recommend prescribing fluoride for infants younger than 6 months. A double-blind study of fluoride supplements conducted to ascertain the effects of fluoride administered to the mother during the last 6 months of pregnancy followed by 5 years of supplements to the child after birth found no additional benefits from prenatal fluoride use (Leverett et al. 1997). In a randomized, double-blind, controlled trial in which supplements were administered from birth, Hennon et al. (1967) had found statistically significant 4-year reductions in caries in primary and permanent teeth of 65 and 41 percent, respectively. Beyond this study, which was conducted when other sources

TABLE 7.4
Water fluoridation status of top 50 U.S. cities

	Population Estimate (7/1/96)	Size Rank 1996	Fluoride/ No Fluoride ^a
New York, New York	7,380,906	1	F
Los Angeles, California	3,553,638	2	F
Chicago, Illinois	2,721,547	3	F
Houston, Texas	1,744,058	4	F
Philadelphia, Pennsylvania	1,478,002	5	F
San Diego, California ^b	1,171,121	6	NF
Phoenix, Arizona	1,159,014	7	F
San Antonio, Texas	1,067,816	8	NF
Dallas, Texas	1,053,292	9	F
Detroit, Michigan	1,000,272	10	F
San Jose, California	838,744	11	NF
Indianapolis, Indiana	746,737	12	F
San Francisco, California	735,315	13	F
Jacksonville, Florida	679,792	14	F-nat
Baltimore, Maryland	675,401	15	F
Columbus, Ohio	657,053	16	F
El Paso, Texas	599,865	17	F-nat
Memphis, Tennessee	596,725	18	F
Milwaukee, Wisconsin	590,503	19	F
Boston, Massachusetts	558,394	20	F
Washington, D.C.	543,213	21	F
Austin, Texas	541,278	22	F
Seattle, Washington	524,704	23	F
Nashville-Davidson (remainder), Tennessee	511,263	24	F
Cleveland, Ohio	498,246	25	F
Denver, Colorado	497,840	26	F
Portland, Oregon	480,824	27	NF
Fort Worth, Texas	479,716	28	F
New Orleans, Louisiana	476,625	29	F
Oklahoma City, Oklahoma	469,852	30	F
Tucson, Arizona ^b	449,002	31	NF
Charlotte, North Carolina	441,297	32	F
Kansas City, Missouri	441,259	33	F
Virginia Beach, Virginia	430,385	34	F
Honolulu, Hawaii	423,475	35	NF
Long Beach, California	421,904	36	F
Albuquerque, New Mexico	419,681	37	F
Atlanta, Georgia	401,907	38	F
Fresno, California	396,011	39	NF
Tulsa, Oklahoma	378,491	40	F
Las Vegas, Nevada	376,906	41	F
Sacramento, California	376,243	42	F
Oakland, California	367,230	43	F
Miami, Florida	365,127	44	F
Omaha, Nebraska	364,253	45	F
Minneapolis, Minnesota	358,785	46	F
St. Louis, Missouri	351,565	47	F
Pittsburgh, Pennsylvania	350,363	48	F
Cincinnati, Ohio	345,818	49	F
Colorado Springs, Colorado	345,127	50	F

^a F = fluoride, NF = no fluoride, and F-nat = natural, nonadjusted fluoride in the water supply.

^b Voted but not yet started.

Source: T. Reeves, CDC Division of Oral Health, personal communication, April 18, 2000.

of fluoride were not as widespread as today, there are no well-designed clinical trials of home-based administration of postnatal supplements. As Murray and Naylor (1996) noted, many studies are difficult to interpret, either because of small size, short experimental period, or inadequate reporting. The studies are further complicated by problems in self-selection bias, in choosing comparable control groups, and in compliance to the daily regimen.

Notwithstanding the paucity of true randomized controlled clinical trials to demonstrate efficacy of supplement use in children, at least 60 studies have reported on the effectiveness of fluoride tablets or drops in home- or school-based programs (Driscoll 1974, Murray and Naylor 1996, Stephen 1993). However, none used the current prescribing schedule. Altogether, the evidence for using fluoride supplements to prevent and control dental caries is mixed. Although many studies have reported that the use of fluoride supplements by infants and children before their permanent teeth erupt reduces caries in permanent teeth, many other studies have reported that it does not (CDC in press). For children aged 6 to 16 who take supplements after most teeth have erupted, the evidence is much clearer that fluoride reduces caries experience (DePaola and Lax 1968, Driscoll et al. 1978, Stephen and Campbell 1978).

Most of the supplements taken at home are prescribed by physicians and dentists in private practice, with physicians prescribing the larger share. Two difficulties are associated with home use. First, the provider may prescribe incorrectly; second, compliance with home-based tablet programs can be very poor. More public and professional education is needed to overcome the difficulties inherent in following recommended regimens for home use of fluoride supplements, which require motivation and adherence on the part of children, parents, and prescribers.

Effectiveness of School-based Programs

Most community fluoride supplement programs are school-based. Each school day, participating students receive a tablet, which they chew under supervision, swishing the resultant solution between the teeth for 30 seconds before swallowing.

Supplement programs in schools have been shown to be effective in preventing caries in permanent teeth when administration is tightly controlled and children are instructed to let the tablet dissolve slowly, to ensure as much topical fluoride exposure as possible. Under these conditions, randomized controlled trials in the United States reported caries

reductions of 20 to 28 percent over periods of 3 to 6 years (DePaola and Lax 1968, Driscoll et al. 1978). In a randomized, double-blind, 3-year study of Scottish schoolchildren who were 5.5 years of age at the start of the study, a much higher percentage reduction in caries in permanent teeth was observed (Stephen and Campbell 1978). In this study, teachers were specifically requested to encourage children each school day to let the sodium fluoride tablet dissolve slowly. These children were from lower socioeconomic groups and may not have had access to fluoride-containing dentifrices and other sources of fluoride, factors that most likely put them at high risk for caries.

Costs of School-based Programs

The costs of a school-based tablet program are low because equipment is not necessary, the procedure does not take long, and an entire classroom of children can participate at once. A 1988 survey of five programs ranging from 7 to 49 schools and 657 to 10,751 children found an average direct cost of approximately \$2.53 per child per school year (Garcia 1989). The costs ranged from \$0.81 to \$5.40, depending on whether paid personnel or volunteers supervised the procedure. The economic benefits of a fluoride supplement program were assessed in randomized controlled clinical trials in Manchester, England, and results showed overall health and cost benefits for the experimental group (O'Rourke et al. 1988).

Summary: Dietary Fluoride Supplements

For children not exposed to optimal fluoride concentration in their water supply, the evidence from studies conducted prior to the 1980s supporting the effectiveness of home use of daily dietary fluoride supplements in preventing dental caries in school-aged children is weak. However, the evidence of the effectiveness of school-based fluoride supplement programs is strong. Such programs require highly

TABLE 7.5
Dietary fluoride supplement dosage schedule

Age of Child	Fluoride Dosage (milligrams per day) at Fluoride in Water Concentration of		
	<0.3 ppm	0.3 to 0.6 ppm	>0.6 ppm
Birth to 6 months	None	None	None
6 months to 3 years	0.25	None	None
3 years to 6 years	0.50	0.25	None
6 years to 16 years	1.00	0.50	None

Source: ADA 1995.

motivated teachers and students, a requirement that likely has limited their widespread adoption. Experts recommend that school-based dietary fluoride supplement programs are likely to be effective in providing topical fluoride protection for children at high risk for dental caries in settings where supervising personnel are highly motivated (CDC in press, Clarkson 1992, Ismail 1994, WHO 1994). Under these conditions, such programs may also be cost-effective.

Fluoride Mouthrinses

Several different formulations of fluoride mouthrinses are available, differing in the amount of fluoride and suggested frequency of use. Rinses with low fluoride concentrations (0.05 percent neutral sodium fluoride or 0.1 percent stannous fluoride) are designed for daily use and are available over-the-counter. Higher-concentration rinses (0.2 percent sodium fluoride) are designed for weekly use and are available only by prescription or in public programs.

School-based Programs

Fluoride mouthrinses were developed in the 1960s as a public health measure for use primarily in schools. They were conceived as a way of avoiding the high costs associated with professional applications of gels and other topical fluoride products in school settings and the poor acceptance by children of brush-on fluoride pastes.

For children in the first grade and up, the procedure consists of vigorously rinsing with 10 milliliters (ml) of solution for 60 seconds. After the rinsing, the fluoride solution is expectorated into a cup, a napkin is inserted to absorb the solution, and both are disposed. Kindergarten children rinse with only 5 ml of solution.

Effectiveness

School-based fluoride mouthrinse programs have been evaluated extensively during the past three decades and have been the subject of numerous reviews (Adair 1998, Birkland and Torell 1978, Bohannon et al. 1985, Petersson 1993, Ripa 1991, Stamm et al. 1984, Torell and Ericsson 1974). Of the many studies during the 1970s and 1980s, 13 satisfied the strict criteria of randomized controlled clinical trials. Caries reductions ranging from 20 to 50 percent were observed, firmly establishing their efficacy. No recent controlled trials have been done. After the efficacy of fluoride mouthrinses was established, a 17-site national school-based demonstration

program showed that a protocol involving weekly rinsing with 0.2 percent sodium fluoride was eminently practical. Most studies done after efficacy was established used a before-and-after design with no concurrent comparison group. This design might overestimate the caries reduction effects. On the whole, however, the programs appear to have been effective.

A survey conducted in 1984 found fluoride mouthrinsing programs in 48 states, with 3.2 million children participating (Bednarsh and Connolly 1984). A later study by CDC reported that 3.25 million schoolchildren were participating in mouthrinsing programs at 11,683 sites in 1988 (Burt 1989), although there are reports that some states have recently curtailed use of these programs (R. Kuthy, personal communication, 2000).

Cost-effectiveness

The cost of the procedure in 1988 ranged between \$0.52 and \$1.78 per child per school year, depending on whether paid or volunteer adult supervisors were used (Garcia 1989). An extensive study during the late 1970s, when downward trends of caries rates were noted, questioned the cost-effectiveness of rinse programs (Klein et al. 1985). Fluoride mouthrinses may be more cost-effective when targeted to schoolchildren with high caries activity (Bawden et al. 1980, Leverett 1989, Torell and Ericsson 1965).

Summary: School-based Fluoride Mouthrinse Programs

Sufficient evidence exists from studies conducted before 1985 to support the effectiveness of 0.2 percent sodium fluoride mouthrinses in preventing coronal caries in school populations. There is evidence that with a declining prevalence of dental caries, the cost-effectiveness of these procedures is reduced. Experts recommend that school-based rinsing once a week with 0.2 percent sodium fluoride is likely to be effective if used in schools and classrooms where students are at high risk for caries and if applied consistently over time (CDC in press). Fluoride mouthrinse programs are not recommended for preschool children in the United States, and programs for kindergarten children should use only 5 ml of solution.

Fluoride Varnishes

Fluoride varnishes have not been approved for use in the United States with an anticaries indication. However, the U.S. public health community has begun to investigate the use of fluoride varnishes,

which became available in this country in 1994. The varnishes are viscous, resinous lacquers painted onto teeth. Because the varnish adheres to enamel surfaces for up to 12 hours or more, fluoride retention in the mouth is greater than with solutions or gels. Varnishes have been used in Europe for 30 years.

No data are available on the use of varnishes in children under 3 years, and, although the results were positive, only two randomized clinical trials have been conducted abroad using preschoolers (Holm 1979, Peyron et al. 1992). Many fluoride rinsing programs in Finland have been replaced with fluoride varnish application programs (Seppä 1991, Sundberg et al. 1996). Studies conducted in Canada (Clark et al. 1987) and Europe (de Bruyn and Arends 1987, Helfenstein and Steiner 1994, Twetman et al. 1996) have found that fluoride varnish is efficacious in preventing dental caries. Applied semiannually, this modality is as effective as professionally applied fluoride gel (Seppä et al. 1995). Some researchers advocate application of fluoride varnish up to 4 times per year to achieve maximum effect, but the evidence of benefits from more than two applications per year remains inconclusive (Mandel 1994, Seppä 1991, Seppä and Tolonen 1990). Other studies have shown that three applications in 1 week, once a year, may be more effective than the more conventional biannual regimen (Petersson et al. 1991, Skold et al. 1994). European studies have shown that fluoride varnishes prevent decalcification (a very early stage of dental caries) beneath orthodontic bands (Adriaens et al. 1990) and slow the progression of existing enamel lesions (Peyron et al. 1992). Findings on cost-effectiveness are mixed (Kirkegaard et al. 1986, Koch et al. 1979, Seppä and Pollanen 1987, Vehmanen 1993).

Dental Sealants

The pits and fissures that characterize the biting surfaces of posterior teeth provide a haven for food debris and decay-causing bacteria. Not surprisingly, these sites are often the first and most frequent to be affected by decay in children and adolescents. The width of most pits and fissures is narrower than a single toothbrush bristle, making cleaning of their deepest recesses almost impossible. According to national estimates, as much as 90 percent of all dental caries in schoolchildren occurs in pits and fissures (Kaste et al. 1996). The teeth at highest risk by far are permanent first and second molars.

Enamel bonding, a technology introduced in the mid-1950s, led to the development of sealants. These are clear or opaque plastic resinous materials designed for professional application to the pit-and-

fissure surfaces of teeth. The material hardens within 60 seconds or so into a thin, hard, protective coating. Sealants were introduced in the late 1960s and received the American Dental Association Seal of Approval in 1976 (ADA 1976). Most of the dozen products approved by the ADA do not contain a therapeutic agent, but work by providing a physical barrier that prevents microorganisms and food particles from collecting in the pits and fissures (ADA 1997). First-generation sealants used ultraviolet light to harden or "cure" the material; improved second- and third-generation sealants cure by chemical or visible light activation, respectively.

Sealant placement requires meticulous attention to technique, but they can be successfully provided in "field" settings using portable dental equipment. To be most effective, sealants should be placed on teeth soon after they erupt, but they can be applied across a wide age range. Not only does the risk for caries continue across the life span, but an individual's risk can increase for any number of reasons. Sealants are particularly helpful for persons with medical conditions associated with higher caries rates, children who have experienced extensive caries in their primary teeth, and children who already have incipient caries in a permanent molar tooth.

Efficacy

Initial clinical trials using a random half-mouth design and first- or second-generation sealant materials established their efficacy. Several comprehensive reviews and a meta-analysis of the amount of caries prevented testify to the utility of these materials (Llodra et al. 1993, Ripa 1993, Weintraub 1989). Llodra et al. (1993) used a systematic process to select and review studies of one-time sealant placement on permanent teeth in subjects unexposed to other preventive measures. Pooled results from 17 studies meeting their selection criteria found that second-generation sealants reduced caries over 70 percent.

These early trials firmly established retention as essential to preventing caries; a sealant is virtually 100 percent effective if it is fully retained on the tooth (NIH 1984). Mertz-Fairhurst (1984) reported 92 to 96 percent retention rates in second-generation sealants after 1 year, with 67 to 82 percent retention after 5 years. A review of studies of long-term retention of second-generation sealants showed 41 to 57 percent intact after 10 years (Ripa 1993). The longest-running study of a one-time application of a first-generation sealant indicated a reduction in pit-and-fissure caries by 52 percent after 15 years

(Simonsen 1991). Retention results for third-generation sealants are similar to those for second-generation systems (Ripa 1993).

Effectiveness

Administrators of school-linked sealant programs (Collins et al. 1985, Sterritt and Frew 1988) and of school-based programs with either fixed clinics (Ismail et al. 1989, Messer et al. 1997, Whyte et al. 1987) or portable equipment (Bravo et al. 1996, Calderone and Davis 1987, Calderone and Mueller 1983, Hardison 1983, Kumar et al. 1997, Morgan et al. 1998) have reported on their experiences with these programs. These studies, using second-generation sealants, have shown effectiveness results comparable to those of clinical trials, regardless of the physical delivery site or personnel used for sealant application. Complete retention after approximately 1 year varied from 83 to 94 percent (Calderone and Mueller 1983, Hardison 1983, Ismail et al. 1989, Sterritt and Frew 1988, Whyte et al. 1987).

A Consensus Development Conference sponsored by the National Institutes of Health concluded that "an extensive body of knowledge has firmly established the scientific basis for the use of sealants" (NIH 1984). The panel urged the development of educational materials to enhance public and professional acceptance as well as third-party reimbursement. Consensus on the value of sealants is reflected by the inclusion of sealant objectives in Healthy People 2000 and Healthy People 2010 (see Table 7.6). In addition, sealant placement is supported in federally funded programs for women and children, and sealants are covered services in all state Medicaid programs. A Workshop on Guidelines for Sealant Use has made recommendations for sealant use in both community and individual care programs (ASTDD 1995).

Community Dental Sealant Programs

Several community-based public health initiatives have arisen to promote sealant use among private practitioners and through community-based programs. These activities include reaching dentists through continuing education courses (Bader et al. 1987, Callanen et al. 1986, Siegal et al. 1996); directing large-scale promotional activities to consumers, community leaders, and third-party payers (Siegal et al. 1997a); and providing sealants directly to children in school programs.

Community programs that provide sealants directly to schoolchildren generally target vulnerable populations less likely to receive private dental care,

such as children eligible for free or reduced-cost lunch programs. School-based programs are usually conducted entirely on site. School-linked programs conduct some portion of the program in schools, such as patient selection and parental permission, but generally provide the sealants at an off-site private practice or clinic. Nationally, 88 community-based sealant placement programs were in operation in the 1992-93 school year. These programs served children in 1,636 schools (Siegal et al. 1997b).

Combining Sealants with a Fluoride Program

Dramatic evidence of the impact of a combined fluoride and sealant program is provided by a program in Guam (Sterritt et al. 1990). For many years the children on this island had experienced dental caries rates more than double those of their U.S. mainland counterparts. In 1984 a school-linked pit-and-fissure sealant program was added to an existing school-based fluoride mouthrinse program. More than 15,000 children participated annually in the sealant program. After 8 years of fluoride mouthrinsing (from 1976 to 1984), mean decayed, missing, and filled surface (DMFS) scores declined by 1.79 surfaces per child. Only 7 percent of that decline was due to prevention of caries on surfaces that can benefit from sealants. With the addition of the sealant program to mouthrinsing, overall DMFS scores decreased an additional 2.34 surfaces per child in only 2 years. Most of this decline took place on pit-and-fissure surfaces. For the 10-year period a reduction of 4.13 DMFS per child was seen—a decline from 7.06 DMFS per child at baseline to 2.93 DMFS in 1986. At the end of the 10 years, participating children on Guam had caries rates close to those of mainland schoolchildren.

The National Preventive Dentistry Demonstration Program, a large project conducted in 10 U.S. cities between 1976 and 1981 to compare the costs and effectiveness of combinations of caries prevention procedures, found that the inclusion of sealants was critical to the cost-effectiveness of prevention strategies (Disney et al. 1989, Klein et al. 1985). In another combined program, Morgan et al. (1998) found that a 3-year sealant program and a fluoride mouthrinse program for secondary schoolchildren incurred a low cost for each tooth surface saved from caries. The incremental cost-effectiveness ratios comparing the intervention to the control group varied from a cost of \$35.60 per tooth surface spared to a net savings of \$7.00, depending on the assumptions used in the analysis.

Sealing Incipient Caries

Heller et al. (1995) evaluated the effect of sealants placed as part of a school-based program on permanent first molar teeth after 5 years. Sealants were applied to both sound teeth and those with incipient carious lesions (where the fissure is stained but not yet cavitated). For the initially incipient carious surfaces, the 5-year decay rate was 10.8 percent for sealed surfaces and 51.8 percent for unsealed surfaces. Initially sound surfaces had a decay rate of 8.1 percent for sealed surfaces and 12.5 percent for unsealed surfaces. Initially sound tooth surfaces were unlikely to become decayed in 5 years and did not benefit greatly from the application of sealants. The study showed potential efficiencies in targeting teeth with incipient caries for sealants.

Cost-effectiveness of Sealant Programs

Studies suggest that sealants are an efficient use of resources when used in populations with higher-than-average disease incidence rates and when selection methods limit sealants to teeth at highest risk of disease. Weintraub et al. (1993) demonstrated cost savings or improving cost-effectiveness with time in a sealant study at a children's dental clinic for low-income families. A strategy of identifying children with prior molar restorations (an indicator of high risk) and sealing the remaining molars showed cost savings within 4 to 6 years.

Summary: Dental Sealant Programs

Studies carried out during the last 20 years provide strong evidence to support the effectiveness of sealants in preventing the development of caries in tooth pits and fissures. Economic analyses suggest that community sealant programs are cost-effective and may even provide cost savings when used in high-risk populations. Experts recommend that programs should be limited to high-risk children and high-risk teeth.

TABLE 7.6
Baseline data for Healthy People 2010 objective 21-8a & b: increase the proportion of children who have received dental sealants on their molar teeth

	Percentage of Children Who Have Received Sealants ^a	
	Aged 8 Years	Aged 14 Years
2010 target	50	50
Total, 1988–94 baseline	23	15
Race and ethnicity		
American Indian or Alaska Native (1999) ^b	37	26
Asian or Pacific Islander	DSU	DSU
Asian	DNC	DNC
Native Hawaiian and other Pacific Islander	DNC	DNC
Black or African American	11	5
White	26	19
Hispanic or Latino	DSU	DSU
Mexican American	10	7
Not Hispanic or Latino	25	DNA
Black or African American	11	5
White	29	18
Gender		
Female	24	14
Male	22	16
Education level (head of household)		
Less than high school	17	4
High school graduate	12	6
At least some college	35	28
Disability status		
Persons with disabilities	DNC	DNC
Persons without disabilities	DNC	DNC
Selected populations		
Third-grade students	26	NA

^a DNA = data have not been analyzed. DNC = data are not collected. DSU = data are statistically unreliable. NA = not applicable.

^b Data are for IHS service areas.

Data sources: National Health and Nutrition Examination Survey (NHANES), NCHS, CDC; Oral Health Survey of Native Americans, 1999, IHS.

Source: USDHHS 2000.

PREVENTION AND CONTROL OF PERIODONTAL DISEASES

Periodontal diseases, caused by specific bacteria in dental plaque, affect most adults at some point in their lives. The mildest and most common form of periodontal disease is gingivitis. Over time, periodontitis, the more severe form of periodontal disease, can lead to the destruction of the soft tissue and bone that anchor the teeth into the jaw. Lacking support, teeth can loosen and be lost.

Periodontal diseases can be prevented and controlled through an array of mechanical and chemical means (Ismail and Lewis 1993, AAP 1996). Conscientious oral hygiene and professional oral cleanings to reduce plaque can reverse gingivitis (Löe et al. 1965).

Methods for personal oral hygiene include tooth-brushing and flossing, which may be augmented by over-the-counter and prescription mouthrinses with antimicrobial action.

Community Programs to Prevent Gingivitis

With the confirmation of specific bacteria in dental plaque as the cause of gingivitis, public health officials began to seek ways to educate the public about plaque control in community settings, primarily in schools. These efforts have had equivocal results. Although knowledge and attitudes were enhanced in demonstration programs, improvements in plaque levels and gingivitis were short-lived in clinical trials (Horowitz et al. 1980).

Prevention of Periodontitis

Tobacco use is a major risk factor for the development and progression of periodontal diseases, and proven strategies aimed at reducing tobacco use should aid in the prevention of periodontitis. The following section on oral and pharyngeal cancers includes a discussion of such intervention strategies. Until recently, most interest in controlling tobacco use reflected concerns about oral cancers. As appreciation of the role of tobacco in the progression of periodontal diseases and tooth loss increases, attention to these oral health effects may increase attention to tobacco cessation in primary oral health care. Periodontitis can also be a complication of poorly controlled diabetes. (See Chapters 3 and 5 for a discussion of other periodontal risk factors; Chapter 5 discusses the connection between periodontal disease and diabetes.)

Some efforts have been directed at alerting dental practitioners to the need to educate patients about diseases affecting the periodontal tissues (Bader et al. 1990, Brown and Spencer 1989). These efforts have met with some success, but they tend to reach only those people who already use dental services. Currently, there are no broad community-based intervention programs that address periodontal diseases.

Summary

Gingivitis can be controlled with available methods, and its control is the principal way to prevent periodontitis. However, the currently available methods are individually or professionally based and require conscientious oral hygiene practices and regular dental visits. Although some schools instruct children in

proper methods of oral hygiene, no community methods, other than programs designed to discourage tobacco use, are available for preventing gingivitis or periodontitis in the general population.

PREVENTION AND CONTROL OF ORAL AND PHARYNGEAL CANCERS

The term *oral and pharyngeal cancers* refers to a diverse group of tumors affecting the oral cavity and pharynx, the majority of which are squamous cell carcinomas. Usually included are cancers of the lips, tongue, pharynx, and oral cavity. These malignancies are among the most debilitating and disfiguring of all cancers. More than 30,000 new cases of oral and pharyngeal cancers are diagnosed each year, and more than 8,000 people die annually from these diseases. The overall 5-year survival rate (52 percent) has not changed in the past four decades (Murphy et al. 1995, Silverman 1998).

Primary risk factors for oral cancers in the United States are the use of tobacco and alcohol products and, for lip cancer, exposure to sun. Tobacco and alcohol independently increase the risk of oral and pharyngeal cancers and also act synergistically, so that persons who use both are at much higher risk than those who use only one. Other risk factors include insufficient fruits and vegetables in the diet, failure to use ultraviolet protection, and infection with certain viruses (Winn et al. 1998).

In 1996 CDC convened the National Oral Cancer Strategic Planning Conference to develop strategies for preventing and controlling oral and pharyngeal cancers in the United States. The conference, which was co-sponsored by the National Institute of Dental and Craniofacial Research and the ADA, included over 125 experts in oral and pharyngeal cancer prevention and control, treatment, and research (CDC 1998). These experts developed recommendations concerning public advocacy, collaboration, and coalition building; public education; professional education and practice; and data collection, evaluation, and research. An ongoing multidisciplinary subgroup from that conference, the Oral Cancer Working Group, met in 1997 and again in 1999 to share information on progress made and to discuss steps to implement a national plan. This group's work will augment existing interventions directed at the reduction of tobacco use, for which several community-based interventions have already been shown to be effective. The group is also developing several statewide models for the prevention and early detection of oral and pharyngeal cancers.

Many recommendations from the 1996 Strategic Planning Conference relate to the inclusion of primary prevention (i.e., reducing risk factors) and early detection. These include a recommendation that because people at high risk for oral cancers are more likely to visit a physician than a dentist, and because physicians may be less likely than dentists to perform an oral cancer examination on such patients, all primary care providers should assume more responsibility for counseling patients about behaviors that put them at risk for developing these cancers; should perform oral cancer examinations on all patients who are at high risk for developing the disease because of tobacco use or excessive alcohol consumption; and should refer patients to the appropriate specialist for management of suspicious oral lesions (CDC 1994c, Elwood and Gallagher 1985, Lynch and Prout 1986, Prout et al. 1990, Yellowitz and Goodman 1995). Further research is needed to better define screening parameters. Comprehensive education of medical and dental practitioners in diagnosing and promptly managing early lesions was recommended to facilitate the multidisciplinary collaboration needed to detect oral cancers in their earliest stages. Furthermore, because of the public's lack of knowledge about the risk factors for oral cancers and because these diseases can often be detected in the early stages, it is also recommended that programs to raise the public's awareness of oral cancers (including their risk factors, signs, and symptoms) be increased.

Community-based Interventions

Community-based interventions for oral and pharyngeal cancer prevention have depended on tobacco control programs.

School-based Prevention Programs

On average, more than 3,000 children and teenagers become regular smokers each day (USDHHS 1994). Prevention efforts aimed at young people are extremely important because nearly all initiation of tobacco use in the United States occurs by age 18. Moreover, the finding that the earlier that smoking begins the more likely it is to lead to heavy use in adulthood makes preventing tobacco use among school-age youth all the more critical (CDC 1994a).

Programs identifying the social influences that foster tobacco use in schoolchildren and teaching skills to resist such influences have yielded consistent and significant results. Reductions or delays in adolescent smoking have been documented, ranging

from 25 to 60 percent and persisting from 1 to 4 years (CDC 1994b). The interventions were based on a CDC review of published research, including the conclusions of the National Cancer Institute's (NCI) Expert Advisory Panel on School-based Smoking Prevention Programs and findings from the 1994 Surgeon General's report, *Preventing Tobacco Use Among Young People* (CDC 1994b). The *Guidelines for School Health Programs to Prevent Tobacco Use and Addiction* cites seven recommendations (CDC 1994a,b):

1. Develop and enforce a school policy on tobacco use.
2. Provide instruction about the short- and long-term negative physiologic and social consequences of tobacco use, social influences on tobacco use, peer norms regarding tobacco use, and refusal skills.
3. Provide tobacco-use-prevention education in kindergarten through 12th grade; this instruction should be especially intensive in junior high or middle school and reinforced in high school.
4. Provide program-specific training for teachers.
5. Involve parents or families in support of school-based programs to prevent tobacco use.
6. Support cessation efforts among students and all school staff who use tobacco.
7. Assess the tobacco-use-prevention program at regular intervals.

A major part of most successful interventions has been the decrease of illegal sales to minors. This strategy has been accomplished by increasing merchant education and enforcement of laws prohibiting tobacco sales to minors under 18 and increasing the cost of cigarettes (CDC 1994a,b, Lewit et al. 1997, Lynch and Bonnie 1994). All 50 states and the District of Columbia have laws prohibiting the sale of tobacco, including smokeless (spit) tobacco, to minors.

In recent years, attempts to prevent and reduce the use of spit tobacco have increased. These informational and educational efforts have largely targeted baseball clubs, Little League baseball teams, and 4-H Club members. A major new initiative, the National Spit Tobacco Education Program, has been launched by Oral Health America, with support from NIH and CDC and funding largely from the Robert Wood Johnson Foundation in collaboration with the Major League Baseball Players Association, to help break the link between spit tobacco and Major League Baseball.

Other Program Models

The majority of community programs designed to prevent or reduce the use of tobacco products have focused on cigarette smoking. Initially, NCI funded randomized trials of interventions to prevent smoking in adolescents and promote cessation in adults. The value of multiple interventions delivered through multiple channels was confirmed in NCI's Community Intervention Trial for Smoking Cessation (COMMIT 1995a,b).

Findings from more than 100 intervention trials continue to provide important information about how to reach smokers and potential smokers. A major conclusion from these studies is that large-scale reductions in smoking prevalence are unlikely when interventions focus on the individual, but that interventions can be effective when community-based. Further, researchers found a statistically significant difference in the proportion of light-to-moderate (but not heavy) smokers who quit in the intervention communities compared with control communities (COMMIT 1995a,b, Klausner 1997, NCI 1995).

Findings from COMMIT and other studies in the United States and abroad led to planning for ASSIST (American Stop Smoking Intervention Study for Cancer Prevention). In 1990, California adopted the ASSIST model, and early success in the California Statewide Tobacco Control Program clearly showed an impact on per capita cigarette consumption in that state compared with consumption in the United States as a whole (Manley et al. 1997a,b, Shopland 1993). The ASSIST model uses surveillance systems that allow for time-series analysis designs comparing intervention and control communities. Media-led tobacco control campaigns, as well as efforts to increase state excise taxes on cigarettes and thereby discourage teenagers from smoking, are included in the model.

There are now dedicated tobacco-control coalitions in all 50 states, and the Agency for Healthcare Research and Quality (formerly Agency for Healthcare Policy and Research) has developed clinical practice guidelines on smoking cessation to aid health professionals in interventions with patients (Fiore 1997).

Although the major focus in reducing the risk for oral and pharyngeal cancers has been on tobacco cessation programs, reduction in alcohol use is clearly indicated. Currently, alcoholic beverages carry the Surgeon General's warning label stating that pregnant women should not drink because of the risk of birth defects and admonishing that alcohol impairs the

ability to drive and operate machinery and may cause other health problems. Many communities have programs that stress responsible drinking by adults related to the use of motor vehicles and completely discourage drinking among young drivers. Community approaches have also been developed to discourage drinking among young people. Targets are youth and adults who are at risk for alcohol-related problems, such as college students who may need to develop skills to avoid binge drinking, or women attending women's clinics who might not know the risk of fetal alcohol syndrome. Because alcohol use, like tobacco use, usually begins in adolescence, development and testing of community- and school-based programs that provide youth with the skills to avoid alcohol use are warranted.

Early Diagnosis of Oral and Pharyngeal Cancers

Primary care providers can counsel patients about lifestyle behaviors that increase the risk for oral cancers. Dental as well as medical personnel have provided successful tobacco control programs in their offices (see Chapter 8). Generally, Americans are ill-informed about the risk factors as well as the signs and symptoms of oral cancers (Horowitz and Nourjah 1996, Horowitz et al. 1995). The mass media have paid little attention to the topic, and health education textbooks are nearly devoid of discussion (Canto et al. 1998b, Chung et al. 2000, Gold and Horowitz 1993, Horowitz et al. 1998). The scant attention that has been paid to oral cancers has focused on the role of spit tobacco.

At present, the principal test for oral and pharyngeal cancers is a comprehensive clinical examination that includes a visual/tactile examination of the mouth, full protrusion of the tongue with the aid of a gauze wipe, and palpation of the tongue, floor of the mouth, and lymph nodes in the neck. The U.S. Preventive Services Task Force concluded that there was insufficient evidence to recommend for or against routine screening for oral cancers, but noted that clinicians should remain vigilant for signs and symptoms of oral cancers and premalignancy in people who use tobacco or regularly use alcohol (USPSTF 1996). The Canadian Task Force on Periodic Health Examination (1997) states that although there is insufficient evidence to include or exclude screening for oral cancers from the periodic health examination for the general public, those at high risk (smokers and heavy drinkers) over 60 warrant an annual oral cancer exam by a physician or dentist (Lewis and

Ismail 1995). The American Cancer Society recommends annual examinations for individuals 40 and older and for individuals who are exposed to known risks. Nevertheless, a 1992 national survey showed that only 15 percent of U.S. adults reported ever having had an oral cancer examination (Horowitz and Nourjah 1996).

There are large gaps in knowledge of the efficacy of oral cancer examinations and the effectiveness and cost-effectiveness of community approaches to early detection of oral cancers. Methodologies and settings differ across studies. Moreover, these studies do not provide definitive evidence supporting the oral cancer exam, and there have been no controlled clinical trials for defining the effectiveness of screening programs. Further research is thus needed.

Summary

Although no school- or community-based interventions specifically designed for the prevention or early detection of oral and pharyngeal cancers are now in place, scientists representing the agencies in the newly formed oral cancer consortium have begun to develop statewide model protocols, beginning with the state of Maryland. In the meantime, any program that aims at eliminating tobacco use will reduce the primary risk factor for oral and pharyngeal cancers, along with other tobacco-related diseases. The evidence on the effectiveness of school-based programs to prevent tobacco use and addiction among children and adolescents provides strong support for their use as part of the school health education curriculum. Further, other community-based interventions such as COMMIT and ASSIST are recommended because they have demonstrated effectiveness in getting light-to-moderate smokers to quit. After reviewing the evidence, an expert panel convened by AHCPR (now the Agency for Healthcare Research and Quality) recommended that all primary care clinicians be trained to provide smoking cessation activities (see Chapter 8). In addition, providers should perform oral cancer examinations on high-risk persons regularly. The recommendation to use all of these interventions to prevent or cease tobacco use in communities is based on expert opinion.

Oral cancers occur in sites that lend themselves to early detection by most primary health care providers and, to a lesser extent, by self-examination. Heightened awareness in the general population could help with early detection and could stimulate dialogue between patients and their primary health care providers about behaviors that may increase their risk. Recent advances in understanding the

molecular events involved in developing cancer might provide the tools needed to design novel preventive, diagnostic, prognostic, and therapeutic regimens to combat oral cancers. Acquiring greater knowledge of the biology, immunology, and pathology of the oral mucosa may also help reduce the morbidity and mortality from these cancers.

PREVENTION AND CONTROL OF CRANIOFACIAL BIRTH DEFECTS

The causes of craniofacial birth defects are often complex and multifactorial—the result of gene-environment interactions occurring from the time of conception to birth. Even when a mutation in a single gene has been discovered as the cause of a particular syndrome, there can be considerable variation in susceptibility, with some infants showing little or no sign of a problem and others experiencing multiple organ defects.

The work to complete the mapping and sequencing of the human genome will undoubtedly shed light on the hundreds of genes involved in craniofacial development and provide details on when and how they function in development. This knowledge may in turn lead to gene therapies that restore or “rescue” the function of a defective gene and thus prevent the anomaly.

Craniofacial defects also may occur because the susceptible embryo or fetus was exposed to an environmental teratogen, a diminished oxygen supply, or a deficit in an essential nutrient. Chapter 5 reports an association between low-birth-weight, premature babies who may show other subtle craniofacial anomalies and mothers with chronic oral infectious disease. In addition, diets poor in folic acid increase the risk of spina bifida and possibly clefting syndromes. Clinical trials using vitamin supplementation with varying levels of folic acid are under development to determine if they can lower the risk of clefts in high-risk pregnancies. Outcomes of clinical trials of nutrient supplementation in pregnancy may lead to new nutritional guidelines and the development of enriched food products, which can form the basis for community-wide health promotion and disease prevention programs.

Given the array of variables affecting prenatal growth and development, the key to public health programs aimed at preventing birth defects lies primarily in health promotion and education campaigns. Individuals need to be made aware of known risk and protective factors in pregnancy. Such programs should emphasize the importance of good nutrition, avoidance of tobacco and alcohol use, and

prenatal care. Education includes knowledge about the teratogenic effects of prescription drugs, such as the antiepileptic drug phenytoin and the retinoic acid drugs used to treat cystic acne.

Summary

As information from developmental biology, genetics, and epidemiologic and clinical studies accrues, dental care providers are better positioned to provide counseling. The public is best served by health promotion and disease prevention campaigns that communicate findings about risk and protective factors in pregnancy.

PREVENTION AND CONTROL OF INTENTIONAL AND UNINTENTIONAL INJURY

Intentional and unintentional injuries are related to behaviors and are thus amenable to prevention. As studies of motor vehicle and sports injuries have demonstrated, injuries are frequently due to a sequence of predictable events, and a public health approach can be successful in injury prevention and control.

The interventions that have proved to be most effective in controlling injuries have been passive; that is, they do not require the individual to participate. Examples include the use of environmental controls such as vehicle and roadway design, speed limits, passenger restraints, and airbags to prevent injuries from motor vehicle collisions (Karlson 1992, Smith and Falk 1987). Passive measures such as these are more easily implemented at the state or federal level. However, many preventive measures for oral-facial injuries have been directed at the individual and professional health service levels, rather than at the population at large (see Table 7.7).

Craniofacial Injuries

The principal causes of craniofacial injuries are motor vehicle collisions, falls, assaults, and sporting activities. Except in relation to sports, injuries to the craniofacial region have received little attention. These injuries are hardly insignificant, however, and efforts to prevent them are gaining acceptance. For example, to increase public awareness of the importance of facial protection, the inaugural National Facial Protection Month was celebrated in April 2000. This national campaign, providing information to the media and the public, was sponsored by the American Association of Oral and Maxillofacial Surgeons (AAOMS 2000).

Motor vehicle collisions are the leading cause of death during the first three decades of life in the United States and the leading cause of death from injury over most of the life span (Baker et al. 1992). Data from multiple sources indicate that craniofacial injuries account for a substantial subset of these injuries annually (USDOT 1998). Even though it is likely that passive measures enacted to reduce fatalities have reduced nonfatal craniofacial injuries, no supporting data exist.

Various sources report the number of motorcycle- and pedal-cycle-related craniofacial injuries. Data from the National Electronic Injury Surveillance System indicate that head injuries account for 50 percent of all pedal-cycle-related injuries; of those, bicycle-related events accounted for 19 percent of all facial injuries within the study period (McDonald 1994). In similar studies, tricycle-related incidents were found to be responsible for up to 61 percent of injuries to the head, face, or mouth (CDC 1987, USCPSC 1986). Motorcycle injuries are a major source of fatal and nonfatal head trauma in the United States (Rivara et al. 1988).

Helmet use reduces head and facial injuries among bicyclists (Acton et al. 1995, Grimard et al. 1995, Rivara et al. 1997) and motorcyclists (Bachulis et al. 1988, Johnson et al. 1995, Lee et al. 1995) by up to 50 percent. Health promotion efforts have increased acceptance at the community level for helmet use by bicyclists; however, helmet use regulations vary by state (Sacks et al. 1996) and with the public whim (Sosin et al. 1990). Over a dozen states currently have bicycle helmet laws, and half of the states have motorcycle helmet laws (NCHS 1992).

Many authors have described craniofacial injuries related to sports. Information is usually obtained from community or regional surveys of injuries or mouthguard use and effectiveness. Craniofacial injuries sustained during sporting activities are a major source of nonfatal injury and disability (Baker et al. 1992), possibly accounting for up to one third of all sports injuries (Cathcart 1982, Meadow et al. 1984). The increasing participation of women in competitive sports means that young women should be alerted to the risks and advised of the need for additional protective gear as appropriate. The most comprehensive data on the effectiveness of protective equipment have been collected by agencies such as the National Alliance of Football Rules Committee, the National Collegiate Athletic Association, and the U.S. Consumer Product Safety Commission. Data on craniofacial injuries from participation in football before and after the enactment of mandatory mouthguard regulations indicate a

TABLE 7.7
Community-based interventions for the prevention and control of craniofacial injuries

	Restraints and Airbags	Helmets	Mouthguards
Guidelines for use	Combination of manual lap and automatic shoulder restraints plus airbag; emphasis on passive systems to overcome noncompliance	Cyclists, both motorized and nonmotorized, wear approved protective helmets, preferably with a full face mask for motorcyclists	Football: Wear helmet with face mask and use mouthguard Hockey: Wear helmet with face mask and use mouthguard because face shields do not prevent injury to lower face
Public policies	Restraints: Mandatory use required by law in 48 states Airbags: All late model vehicles required to have driver-side airbags, and future models to add passenger-side airbags; National Traffic and Motor Vehicle Safety Act legislates policies	<ul style="list-style-type: none"> • 13 states have bicycle helmet laws; 25 have motorcycle helmet laws • Post-law bicycle helmet use rates increase by up to 50 percent among children (National Center for Injury Prevention and Control 1995) • States with helmet use laws have higher rates of helmet use 	Football: Requirement since 1962 for mandatory mouthguard use in football accompanied by significant decline in incidence of oral-facial injuries Hockey: Mandatory requirement for full facial protection in Canada has reduced facial injuries among youth (Rampton et al. 1997)
Utilization rates	Restraints: Compliance with seatbelt laws ranges from 29 to 74 percent (Reinfurt et al. 1991); current use may be as high as 67 percent (NCHS 1992) Airbags: Utilization may become an issue if on-off switch is implemented	Bicycle: Approximately half of bicyclists own a helmet, and half of those consistently wear it (Sacks et al. 1996); 62 percent of motorcyclists and 17.6 percent of bicyclists wear helmets (NCHS 1992)	Football: 72 percent of children wear headgear and mouthguards Baseball/softball: 35 percent of children wear headgear; 7 percent consistently wear mouthguards Soccer: 4 percent of children wear headgear; 7 percent of children wear mouthguards (Nowjack-Raymer and Gift 1996) Basketball: 4 percent of respondents reported wearing mouthguards (Maestrello-deMoya and Primosch 1989)
Type of evidence for effectiveness and PHS ratings	<ul style="list-style-type: none"> • Hospital and trauma registry studies (Sutyak et al. 1997, Orsay et al. 1990) • Modeling from insurance studies (Sorenson 1993) • Case-control study (Marine et al. 1994) 	<ul style="list-style-type: none"> • Hospital and trauma registry studies (Bachulis et al. 1988, Johnson et al. 1995) • National survey (Sacks et al. 1996) • Case-control study (Thompson et al. 1996) 	<ul style="list-style-type: none"> • Hospital and trauma registry studies • Questionnaire (baseball and basketball) • Before and after NFA rule in 1962 • Descriptive survey (Maestrello-deMoya and Primosch 1989)
Evidence for effectiveness	Restraints: Use reduces facial injuries by 30 percent (Orsay et al. 1990) Airbags: Projected facial injury harm reduction of up to 90 percent for airbag added to restraint (Sorenson 1993); report of facial injuries may increase with airbags due to a decrease in fatalities and more severe injuries (Blacksin 1993)	Motorcycle: A twofold decrease in incidence of maxillofacial trauma in helmeted versus nonhelmeted motorcyclists (Bachulis et al. 1988); nonhelmeted 3 times more likely to have facial fractures than those with helmets (Johnson et al. 1995) Bicycle: Helmet wearers have a 65 percent reduction in upper- and mid-face injuries (Thompson et al. 1996); head injuries decreased 67 percent in children concomitant with rate of helmet use increase of 35 percent following educational campaign (Rivara et al. 1994); helmets with face protection decrease facial injuries by a factor of 3 (Vaughan 1977); helmet wearing alone is not sufficient to prevent serious injury and fatality (Rivara et al. 1997)	Football: Face mask reduces oral-facial injury by 50 percent; addition of a mouthguard reduces risk to less than 1 percent (AAHPER 1960) Hockey: Full-face protection reduced chance of upper facial injury; half visor same as no face protection (Rampton et al. 1997) Baseball and basketball: 60 times more likely to sustain oral injury without mouthguard (McNutt et al. 1989); 30 percent reduced risk of oral-facial injury for those wearing mouthguards (Powers et al. 1984)

	Restraints and Airbags	Helmets	Mouthguards
Risks	<p>Restraints:</p> <ul style="list-style-type: none"> • Improper car seat design and use for infants <p>Airbags:</p> <ul style="list-style-type: none"> • Concern regarding higher risk for death in children and small women • Case report of facial desquamation from ruptured airbag 	<ul style="list-style-type: none"> • No injuries have been documented secondary to the helmet itself; helmets do not decrease risk of injury to lower face, mandible, and mouth 	<p>Costs are high because mouthguards should be fabricated for each individual by a health professional</p>
Costs and effectiveness	<p>Airbags and restraints: U.S. costs are calculated for all injuries or fatalities, not craniofacial injuries; Australian report estimated savings of 108 million (Australian) dollars per year due to facial injury reduction (Sorenson 1993)</p>	<p>Bicycle: Hospital treatment costs for bicyclists estimated at \$1 billion annually—includes mortality and morbidity (Sacks et al. 1996)</p>	<p>Hockey: Direct costs: 3 million (Canadian) dollars annually (Rampton et al. 1997)</p>
Recommendations (abstracted from CDC 1987, USDHHS 2000)	<ul style="list-style-type: none"> • Extend safety belt laws to all 50 states • Increase airbag efficacy and safety research for craniofacial injuries 	<ul style="list-style-type: none"> • Helmets should meet recommended industry manufacturing standards • Implement national mandatory helmet requirement law for motorcyclists in all states • Implement national mandatory helmet requirement law for child bicyclists in all states—strongly recommend helmet use for adult bicyclists • Combine helmet use with education and health promotion and environmental controls (e.g., separation of cyclists and motor vehicles, features to make cyclist more visible) • State and local health departments should engage in health promotion for helmet use, develop and work for legislation for mandatory helmet use, and evaluate programs 	<ul style="list-style-type: none"> • Extend mandatory mouthguard use for all team sports sponsored by organizations, agencies, and institutions • Utilize health promotion and education of trainers, athletes, and parents to increase use of protective sporting equipment

significant decline in craniofacial injuries (Sane 1988). Further, the U.S. Consumer Product Safety Commission's review of National Electronic Injury Surveillance System data showed that mouth injuries were more frequent in baseball than in any other sport monitored (USCPSC 1981). These combined reports were instrumental in implementing policies for protective equipment use in these two sports. (See Box 7.2, Sports Injuries and Oral-Facial Trauma.)

Research on elderly and disabled individuals has led to the development of safety measures to prevent

unintentional injuries from falls in the home. These include installing adequate lighting and handrails, using nonskid materials on floors and in bathrooms, and positioning furniture to reduce the risk of tripping. Wider distribution and adoption of such safety measures should lower the risk of oral and craniofacial injuries due to falls for the general population as well, not only in the home but also in the workplace and other settings.

Summary: Prevention of Craniofacial Injuries

Health education and injury prevention campaigns addressing the need for protective gear in sports and cycling activities can increase awareness and use. More rapid adoption can occur through legislation or regulation. Greater dissemination of safety measures for home and workplace can similarly lower the risk of falls and other unintentional injuries. With regard to reducing unintentional injuries in the United States, current and ongoing policy discussions, legislative proposals, and research efforts are necessary first steps toward appropriate programs.

ORAL HEALTH PROMOTION AND DISEASE PREVENTION KNOWLEDGE AND PRACTICES

To take full advantage of emerging science-based health and health care practices, individuals, health care providers, and policymakers need to be sufficiently informed that they can take appropriate actions for themselves, for those for whom they have responsibility, and for the community at large. For the individual, these actions include brushing with a fluoride-containing dentifrice for caries prevention, brushing and flossing to prevent gingivitis and periodontal diseases, and avoiding tobacco and other substances that are detrimental to health.

Lack of knowledge can affect care. If parents are not familiar with the importance and care of their child's primary teeth or if they do not know that dental sealants exist, they are unlikely to take appropriate action or seek professional services. If the public is not aware of the benefits of community water fluoridation, public referenda and funding for such interventions are not likely to be supported. Similarly, if individuals do not know that an oral cancer examination exists, they may not ask about the need for one. However, it is well established that

BOX 7.2

Sports Injuries and Oral-Facial Trauma

The national concern regarding oral-facial injury is addressed in the Healthy People 2010 objective 15-31, which is to increase the proportion of public and private schools that require use of appropriate head, face, eye, and mouth protection for students participating in school-sponsored physical activities. The National Youth Sports Safety Foundation estimates that more than 3 million teeth will be knocked out in youth sporting activities this year, an injury almost completely preventable by wearing a mouthguard. Even more significant, oral-facial trauma from sports injuries will result in facial bone fractures, concussion, permanent brain injury, temporomandibular dysfunction, blinding eye injuries, and even death.

Currently, no systematic monitoring for oral-facial injuries exists in the United States. Progress toward a more broadly targeted Healthy People 2000 objective proved to be difficult to track because of the data requirements of monitoring all organizations, agencies, and institutions sponsoring sporting and recreational events that pose risk of injury. By focusing on schools, not only should the monitoring of progress be feasible, but healthy habits will be formed early. The hope is that by the time the athletes reach young adulthood they will recognize the hazards posed by their athletic interests and, perhaps, be more comfortable using protective devices than they would be without them.

It is estimated that as many as one third of all dental injuries are sports-related. A particularly high proportion of all baseball injuries (41 percent) is estimated to occur to the head, face, mouth, or eyes. Nowjack-Raymer and Gift (1996) reported that in 1991 more than 14 million U.S. school-aged youngsters participated in at least one sport that was listed on the 1991 National Health Interview Survey questionnaire, with more than 9 million of these children in organized baseball or softball.

Baseball and softball are the most popular organized sports, with nearly one quarter of the school-aged population playing. Unlike football, not all baseball/softball leagues or teams require the use of safety equipment. In many cases, only selected positions such as catchers and batters are covered by rules. Thus only 35 percent of players reported that they wore headgear all or most of the time, and only 7 percent wore mouthguards all or most of the time.

Further analysis of the interview data revealed a variety of socioenvironmental differences in the wearing of headgear and mouthguards. Forty percent of males who played baseball or softball reported wearing protective headgear "all or most of the time," compared with only 25 percent of females. Differences were also found by poverty level, with 36 percent of those at or above poverty level wearing headgear, compared with 24 percent of those below. Better educated parents were somewhat more likely than less educated parents to have responded that their child wore headgear "sometimes" (45 percent versus 38 percent) and non-Hispanics reported occasional use more than Hispanics (43 percent versus 30 percent). Parents of a greater percentage of baseball or softball players of high school age (12 percent) than elementary school-aged players (6 percent) reported that their child wore a mouthguard "all or most of the time." Also, more black (17 percent) than white (6 percent) children reported the use of mouthguards.

These socioeconomic differences might be greater were it not for the safety efforts of school athletic programs. Still, many parents and coaches are not as proactive as they could be and are not aware that facial injuries also occur in sports that are not considered high contact. For example, basketball players typically do not wear mouthguards. Yet approximately 34 percent of all injuries to basketball players involve teeth and/or the oral cavity.

Examples of community-based interventions to prevent sports-related, oral-facial trauma include the development of rules and regulations for the use of headgear and mouthguards in sports where craniofacial injury is a risk; efforts to alert players, parents, sports officials, and organizers to the potential for injury; better product design; and the creation of supportive environments for sports-related equipment and recreation areas.

knowledge alone will not necessarily lead to appropriate practices. For example, even if individuals know that tobacco use is unhealthful and that it contributes to multiple life-threatening illnesses, some continue to smoke. The majority of people who need such information most—those in low-income groups and those with lower levels of education—also are the ones who lack the information and skills (oral health literacy) to ask for and obtain specific preventive services or treatment options. Health professionals are in an ideal position to provide up-to-date health information and care to their patients. They also have an opportunity to enhance their knowledge and practices as well as increase their communication to patients about the procedures they provide and the reasons for these procedures.

Few national studies of public and professional knowledge, attitudes, and practices exist. Highlights from these as well as from state and local studies that evaluated the prevention of dental caries, periodontal diseases, and oral cancers are provided below. Generally, the public is unable to discriminate between methods that prevent dental caries and those that prevent periodontal diseases (Corbin et al. 1985, Gift et al. 1994). This confusion has been attributed to the prevailing marketing message that refers to them as “plaque diseases” preventable by thorough tooth cleaning with a toothbrush and floss. In addition, the general public and health care providers are not fully informed about the relative value of fluoride and the appropriate recommended applications of regimens to prevent dental caries (Corbin et al. 1985, Gallup 1992, Gift et al. 1994, O’Neil 1984). More work is needed to improve knowledge and practices related to oral cancer prevention as well. As with other areas of investigation, additional survey research is needed to better understand findings to date and to develop tailored interventions. Research is ongoing to improve the design of survey instruments and the wording of questions to address cultural and ethnic differences and interpretations.

Dental Caries Prevention

The Public

Most members of the general public, regardless of socioeconomic level, tend to believe that the best way to prevent dental caries is by brushing their teeth (Corbin 1985, Gift et al. 1994, O’Neil 1984). In the 1990 National Health Interview Survey (NHIS), respondents were asked the purpose of adding

fluoride to public drinking water. About two thirds of the respondents 25 to 65 years of age knew that water fluoridation helps prevent caries, compared with only 51 percent and 49 percent of those 65 and older and 18 to 24 years of age, respectively. Blacks and Hispanics were less likely to know the value of this preventive procedure than whites. In the same survey, when asked to indicate the one best way to prevent tooth decay from five answers (limiting sugary snacks, using fluorides, chewing sugarless gum, brushing and flossing the teeth, and visiting the dentist every 6 months), only 7 percent of the respondents answered correctly that fluoride was the most effective (Gift et al. 1994). More than two thirds said tooth brushing and flossing were the most effective. These results paralleled those of earlier studies (Gift et al. 1994, O’Neil 1984). A lower perceived value of fluorides by the public in preventing dental caries also was seen in the 1985 NHIS (Corbin et al. 1985). In a survey of knowledge and beliefs of the public, dentists, and dental researchers about the best way to prevent dental caries, the public and the dentists identified tooth brushing, whereas dental researchers unanimously ranked fluorides, as most important (O’Neil 1984). A small study among Latina mothers showed that they believed that brushing with baking soda is a good way to prevent dental caries; they knew little about brushing with a fluoride-containing dentifrice (Watson et al. 1999).

Dental sealants and appropriate use of fluoride are critical for caries prevention. In the 1990 NHIS, about 32 percent of the public had heard of dental sealants, but among those only three fourths knew the purpose of this preventive measure (Gift et al. 1994). In 1991 the Gallup Organization conducted a poll for the American Academy of Pediatric Dentistry among a national sample of 1,200 parents of children 16 years and younger. The results indicated that only 58 percent believed fluoride to be very important to a child’s oral health; another 36 percent considered it to be somewhat important. Eight of 10 parents did not know when a child should be prescribed fluoride supplements, and virtually no one knew when supplements should be stopped. Only 25 percent of parents in nonfluoridated communities reportedly give their children fluoride supplements (Gallup 1992).

Health Care Providers

In a national survey of U.S. dental hygienists’ knowledge, opinions, and practices regarding dental caries etiology and prevention, over 90 percent agreed that “adults benefit from the use of fluorides” and that “root surface caries is an emerging problem.” A little

less than one third did not provide fluoride treatments to adults. This same survey found that only 57 percent of the respondents recognized remineralization as fluoride's most important mechanism of action; rather, flossing was selected as the most effective procedure for preventing caries in adults. Also, only 18 percent reported providing the recommended time for acidulated phosphate fluoride (APF) gel treatment (Forrest 1998). A city-based survey of dentists and dental hygienists found that nearly 70 percent of the offices used lower than recommended topical fluoride application times and that some of the fluoride products reportedly used had not been clinically tested (Warren et al. 1996).

Periodontal Disease Prevention

The Public

In the 1990 NHIS the majority of household respondents (79 percent) could identify one common sign of "gum" disease. Level of education was directly related to knowledge of gum disease. Eighty-nine percent of those with more than a high school level of education were able to name a common sign of gum disease, compared with 79 percent of those with a high school education and 60 percent of those with less than a high school education (Gift et al. 1994).

A Roper report on oral health surveyed more than 1,000 adults 18 and older. Eighty percent reported that they did not believe they have had periodontal disease. However, 70 percent reported having experienced at least one symptom of gum disease—bleeding gums; swollen, painful, or receding gums; a change in bite; or loose teeth. Although 41 percent of the respondents said that losing their teeth was their greatest fear regarding oral health, only 38 percent who had bleeding gums said they told their dentists about the problem. Further, only 30 percent of the respondents who had experienced warning signs of gum disease were worried about developing periodontal problems in the future. Fifty-eight percent knew that plaque is the main cause of gum disease and that flossing alone will not prevent gum disease, whereas 77 percent knew that brushing alone would not prevent gum disease. The majority (90 percent) knew that gum disease could strike anyone at any age (Roper Report 1994).

In a recently reported study on the oral hygiene practices of a convenience sample of 34,897 users and nonusers of tobacco products who obtained dental care in 75 dental practices, 74 percent reported brushing twice a day and 36 percent reported flossing once daily (Andrews 1998). Tobacco users brushed

and flossed much less frequently than nonusers. Patients with more than a high school education were less likely to use tobacco products and more likely to brush at least 2 times a day and floss daily than were those with less education.

A 1996 study of 1,000 U.S. adults showed that nearly one third (29 percent) of respondents were extremely or very concerned about getting gum disease. Concern was highest among younger respondents 18 to 49 years of age and those who very or somewhat frequently experienced bleeding gums. Only 6 percent said they frequently suffered from bleeding gums (2 percent very frequently and 4 percent somewhat frequently). Only 13 percent said a dental professional had diagnosed them with any kind of periodontal disease (gingivitis, pyorrhea, and periodontitis). Older respondents were somewhat more likely than younger ones to have been diagnosed with gum disease, and 17 percent reported experiencing gingival bleeding occasionally (Andrews 1998).

Health Care Providers

Studies of dental professionals regarding periodontal disease prevention practices are limited. In 1989, Dental Products Report launched a study to determine the involvement of general practitioners in periodontal care. Overall, general dentists and their hygienists have become more involved in the periodontal exam phase of patient treatment. This positive trend suggests that periodontal diagnosis and treatment are well integrated into general practice. For example, when asked "what phases of periodontal treatment are you providing at present?" 100 percent reported gingival exam and evaluation, 97 percent reported pocket probing, and 88 percent reported providing patient education. The majority of dentists (67 percent) used as many as six measurement sites per tooth. Nearly all (93 percent) reported having a referral relationship with a periodontist (Dental Products Report 1996).

Oral Cancer Prevention and Early Detection

The Public

U.S. adults generally are ill-informed regarding risk factors for and signs and symptoms of oral cancers. Further, a 1990 national survey found that only 14 percent of adults 40 and older reported that they had ever had an oral cancer examination. Of those, only 7 percent had had an exam within the last year

Horowitz et al. 1995). In a statewide survey in Maryland, 85 percent of the adults claimed to have heard of oral or mouth cancer, but only 28 percent reported ever having an oral cancer examination (Horowitz et al. 1996). A state-based study of veterans—a population at high risk for oral cancers—found that they were ill-informed and misinformed about these cancers (Canto et al. 1998a). Finally, a study among Latino youths who reported use of tobacco and alcohol found that they, too, were not knowledgeable regarding risk factors for oral cancers (Canto et al. 1998b).

Health Care Providers

A recent national pilot survey of U.S. dentists found that the respondents' knowledge regarding risk factors for and signs and symptoms of oral cancers and their reported practice of examination procedures were limited (Yellowitz et al. 1998). Most respondents believed they were adequately trained to provide oral cancer examinations, and 70 percent provided annual oral cancer exams to patients 40 and older. Seventy-four percent reported their knowledge of oral cancers to be current, yet only 30 percent correctly identified the age cohort most frequently diagnosed with oral cancers. Further, less than 50 percent correctly identified the stage at which most oral cancer lesions are diagnosed, and nearly one third of respondents could not identify the two most common sites of these lesions. Although 86 percent claimed to assess their patients' current tobacco use, only 50 percent assessed current alcohol use; relatively few dentists assessed past alcohol or tobacco use. There was a modest amount of misinformation as well. For example, 65 percent believed, incorrectly, that ill-fitting dentures and partials were a risk factor for oral cancers, and 47 percent believed, also incorrectly, that poor oral hygiene was a risk factor. Further, although the majority of dentists claimed to provide oral cancer examinations to the majority of their patients, a large proportion did not palpate the lymph nodes—part of a comprehensive oral cancer examination. These results confirm an earlier study conducted among a convenience sample of Maryland dentists and physicians in that both groups believed their knowledge and skills related to oral cancer prevention and early detection to be wanting (Yellowitz and Goodman 1995).

A recent national survey among U.S. dental hygienists found that although 98 percent agreed that oral cancer examinations should be provided annually for adults 40 and older, only 64 percent reported performing such an exam 100 percent of the time,

and nearly 17 percent reported not performing an exam at any time (Forrest 1998). Further inconsistencies were found between knowledge of risk factors and performance. For example, although 94 percent correctly identified alcohol use as a risk factor for oral cancer, only 49 percent asked about alcohol use. Less than a majority (45 percent) reported their knowledge of oral cancers to be current. A majority (61 percent) believed they were adequately trained to palpate lymph nodes; still, only 24 percent reported routine palpating of lymph nodes, while 51 percent indicated they did not do so at any time.

Summary

Findings from national surveys, together with those from local studies, suggest that there are opportunities for enhanced educational efforts for both the public and health professionals to improve oral health. These studies focus on the public and the dental profession for selected diseases. New research is needed to assess knowledge, attitudes, and practices of all health professionals and for other conditions and risk factors related to oral health as well.

BUILDING UPON SUCCESS

As research and technology advance our understanding of the causes of major craniofacial diseases and disorders and lead to improved methods of diagnosis, treatment, and prevention, opportunities for new community-based prevention programs will grow. Ultimately, the application of any preventive intervention is driven by a combination of individual behaviors, community interventions, and professional practice. Only a few studies have taken into account all three spheres of action in determining health outcomes in a community (Arnljot et al. 1985, Chen et al. 1997). Our knowledge of the effects of multiple interventions is limited because most interventions were developed and tested singly.

In the past half century, however, advances in our understanding of oral diseases and the application of multiple preventive measures have resulted in continuing declines in the prevalence and severity of both dental caries and periodontal diseases for a sizeable majority of Americans. For dental caries, for example, experts now believe that most people can maintain a low risk of the disease by a combination of drinking fluoridated water and brushing daily with a fluoride dentifrice. They recommend that additional provider- and community-based dental prevention programs be targeted to high-risk individuals and groups.

Many of the studies reviewed in this chapter were conducted when higher rates of caries prevailed, community water fluoridation was less widespread, and use of fluoride dentifrices and supplements was not as common as today. These facts must be taken into consideration in contemporary decision making by public health professionals and policymakers. The validity and reliability of recommendations will benefit from the systematic reviews of the scientific evidence by the Task Force on Community Preventive Services (2000) to be included in a *Guide to Community Preventive Services*. Oral health promotion strategies are among those currently being evaluated.

Future innovations include implementing programs in new settings, such as workplaces, senior centers, and nursing homes, where individuals at high risk can be reached. Even if these programs are more expensive, the yield may be worth it if they

reach those at high risk for disease. Similarly, focusing community-based interventions on populations at greatest risk will make optimal use of available resources. However, continued research to understand risk and improve ways to measure it is equally important for the success of these ventures.

A review of progress in reaching the Healthy People 2000 oral health objectives reveals relatively little gain across many of the objectives (Table 7.8). Progress in the next decade will require diligent efforts to identify public health problems, mobilize resources, and ensure that the necessary conditions are in place and crucial services received. Public health agencies will be instrumental in carrying out these functions, and state and local dental directors can perform a leadership role. Box 7.3 describes the public health services that are essential if a community is to realize fully the benefits of available disease prevention and health promotion interventions.

TABLE 7.8
Progress in meeting Healthy People 2000 oral health objectives

		Age	Baseline Data	HP 2000 Goal	Final Data	Summary
13.1	Reduce dental caries in children	6-8	54%	35%	52%	Prog
	Reduce dental caries in adolescents	15	78%	60%	61%	Prog +++
13.2	Reduce untreated dental decay in children	6-8	28%	20%	29%	Reversed
	Reduce untreated dental decay in adolescents	15	24%	15%	20%	Prog ++
13.3	Increase adults who have never lost a permanent tooth	35-44	31%	45%	31%	No Change
13.4	Reduce adults who have lost all their teeth	65+	36%	20%	30%	Prog ++
13.5	Reduce gingivitis among adults	35-44	41%	30%	48%	Reversed
13.6	Reduce destructive periodontal disease	35-44	25%	15%	22%	Prog +
13.7	Reduce oral and pharyngeal deaths in males	45-74	13.6%	10.5%	10.3%	Met
	Reduce oral and pharyngeal deaths in females	45-74	4.8%	4.1%	3.5%	Met
13.8	Increase sealants in children	8	11%	50%	23%	Prog ++
	Increase sealants in adolescents	14	8%	50%	24%	Prog ++
13.9	Increase persons on public water receiving fluoridated water		61%	75%	62%	Prog
13.10	Increase topical/systemic fluorides among nonfluoridated		50%	85%	No data	No data
13.11	Increase caregivers using feeding practices that prevent early childhood caries		55%	75%	No data	No data
13.12	Increase oral health screening, referral, follow-up, first time school attendee		66%	90%	75%	Prog ++
13.13	For long-term care, oral exam and services provided within 90 days		No data	100%	No data	No data
13.14	Increase use of oral health care system (adults)	35+	54%	70%	63%	Prog ++
13.15	Increase states with system for recording and referring orofacial clefts		11 states	40 states	23 states	Prog ++
13.16	Extend use of protective head, face, eye, and mouth equipment		No data	No data	No data	No data
13.17	Reduce smokeless tobacco use among males	12-17	6.6%	4%	3.7%	Met
		18-24	8.9%	4%	6.9%	Prog ++

Source: Adapted from NCHS 1999.

BOX 7.3 Essential Public Health Services for Oral Health

The Association of State and Territorial Dental Directors' *Guidelines for State and Territorial Oral Health Programs* (ASTDD 1997) identifies the following essential public health services to improve oral health:

- I. Assessment
 - A. Assess *oral health status and needs* so that problems can be identified and addressed.
 - B. Analyze *determinants* of identified oral health needs, including resources.
 - C. Assess the *fluoridation status* of water systems, and other sources of fluoride.
 - D. Implement an *oral health surveillance system* to identify, investigate, and monitor oral health problems and health hazards.
- II. Policy Development
 - A. Develop *plans and policies* through a collaborative process that support individual and community oral health efforts to address oral health needs.
 - B. Provide *leadership* to address oral health problems by maintaining a strong oral health unit within the health agency.
 - C. Mobilize *community partnerships* between and among policymakers, professionals, organizations, groups, the public, and others to identify and implement solutions to oral health problems.
- III. Assurance
 - A. Inform, educate, and empower *the public* regarding oral health problems and solutions.
 - B. Promote and enforce *laws and regulations* that protect and improve oral health, ensure safety, and assure accountability for the public's well-being.
 - C. *Link people* to needed population-based oral health services, personal oral health services, and support services and assure the availability, access, and acceptability of these services by enhancing system capacity, including directly supporting or providing services when necessary.
 - D. Support services and implementation of programs that focus on *primary and secondary prevention*.
 - E. Assure that the *public health and personal health workforce* has the capacity and expertise to effectively address oral health needs.
 - F. Evaluate *effectiveness, accessibility, and quality* of population-based and personal oral health services.
 - G. Conduct *research and support demonstration projects* to gain new insights and applications of innovative solutions to oral health problems.

FINDINGS

- Community water fluoridation, an effective, safe, and ideal public health measure, benefits individuals of all ages and socioeconomic strata. Unfortunately, over one third of the U.S. population (100 million persons) are without this critical public health measure.
- Effective disease prevention measures exist for use by individuals, practitioners, and communities. Most of these focus on dental caries prevention, such as fluorides and dental sealants, where a combination of services is required to achieve optimal disease prevention. Daily oral hygiene practices such as brushing and flossing can prevent gingivitis.
- Community-based approaches for the prevention of other oral diseases and conditions, such as oral and pharyngeal cancers and oral-facial trauma, require intensified developmental efforts.
- Community-based preventive programs are unavailable to substantial portions of the underserved population.
- There is a gap between research findings and the oral disease prevention and health promotion practices and knowledge of the public and the health professions.
- Disease prevention and health promotion approaches, such as tobacco control, appropriate use of fluorides for caries prevention, and folate supplementation for neural tube defect prevention, highlight opportunities for partnerships between community-based programs and practitioners, as well as collaborations among health professionals.
- Many community-based programs require a combined effort among social service, health care, and education services at the local or state level.

REFERENCES

- Acton CH, Thomas S, Nixon JW, Clark R, Pitt WR, Battistutta D. Children and bicycles: what is really happening? *Studies of fatal and non-fatal bicycle injury.* *Inj Prev* 1995 Jun;1:86-91.
- Adair SM. The role of fluoride mouthrinses in the control of dental caries: a brief review. *Pediatr Dent* 1998 Mar-Apr;20(2):101-14.
- Adriaens ML, Dermaut LR, Verbeeck RM. The use of 'fluor protector', a fluoride varnish, as a caries prevention method under orthodontic molar bands. *Eur J Orthod* 1990;12:316-9.
- American Academy of Periodontology (AAP). Proceedings of the 1996 World Workshop in Periodontics. *Ann Periodontol* 1996;1(1):223-55.
- American Association for Health, Physical Education and Recreation, and American Dental Association (ADA). Report of the Joint Commission on Mouth Protectors. 1960.

- American Association of Oral and Maxillofacial Surgeons (AAOMS). National Facial Protection Month. Available from: <http://www.aaoms.org/facpromonth.htm>. [accessed 2000 Apr 6].
- American Dental Association (ADA). Caries diagnosis and risk assessment. A review of preventive strategies and management. *J Am Dent Assoc* 1995 Jun;126(Suppl):1S-4S.
- American Dental Association (ADA) Council on Access, Prevention and Interprofessional Relations and Council on Scientific Affairs. Dental sealants. *J Am Dent Assoc* 1997 Apr;128(4):485-8.
- American Dental Association (ADA) Council on Dental Materials and Devices. Pit and fissure sealants. *J Am Dent Assoc* 1976 Jul;93(1):134.
- Andrews JA, Severson HH, Lichtenstein E, Gordon JS. Relationship between tobacco use and self-reported oral hygiene habits. *J Am Dent Assoc* 1998;129:313-20.
- Arnljot H, et al., editors. Oral health care systems: an international collaborative study coordinated by the World Health Organization. London: Quintessence; 1985.
- Arnold FA Jr, Likins RC, Russell AL, Scott DB. Fifteenth year of the Grand Rapids fluoridation study. *J Am Dent Assoc* 1962;65:780-5.
- Association of State and Territorial Dental Directors (ASTDD), the New York State Health Department, the Ohio Department of Health and the School of Public Health, University of Albany, State University of New York. Workshop on guidelines for sealant use: recommendations. *J Public Health Dent* 1995;55(5 Spec No):263-73.
- Association of State and Territorial Dental Directors (ASTDD). Guidelines for state and territorial oral health programs. 1997 Jul.
- Ast DB, Fitzgerald B. Effectiveness of water fluoridation. *J Am Dent Assoc* 1962;65:581-7.
- Bachulis BL, Sangster W, Gorrell GW, Long WB. Patterns of injury in helmeted and nonhelmeted motorcyclists. *Am J Surg* 1988 May;155(5):708-11.
- Bader JD, Sams DH, O'Neil EH. Estimates of the effects of a statewide sealant initiative on dentists' knowledge and attitudes. *J Public Health Dent* 1987 Fall;47(4):186-92.
- Bader JD, Rozier RG, McFall WT, Sams DH, Graves RC, Slome BA, Ramsey DL. Evaluating and influencing periodontal diagnostic and treatment behaviors in general practice. *J Am Dent Assoc* 1990 Dec;121(6):720-4.
- Baker SP, O'Neill B, Ginsburg MJ, Guohua L, editors. The injury fact book. 2nd ed. New York: Oxford University Press; 1992. p. 211-2.
- Bawden JW, Granath L, Holst K, Koch G, Krasse P, Rootzen H. Effect of mouthrinsing with a sodium fluoride solution in children with different caries experience. *Swed Dent J* 1980;4(3):111-7.
- Bednarsh H, Connolly GN. A report on fluoride mouthrinsing programs among states. Presented at the 112th annual meeting of the American Public Health Association; 1984; Anaheim, CA.
- Birkland JM, Torell P. Caries-preventive fluoride mouthrinses. *Caries Res* 1978;12(Suppl):38-51.
- Blacksin MF. Patterns of fracture after air bag deployment. *J Trauma* 1993 Dec;35(6):840-3.
- Blayne JR, Hill IN. Fluorine and dental caries. *J Am Dent Assoc* 1967;74(SI):233-302.
- Bohannon HM, Stamm JW, Graves R, Disney JA, Bader JD. Fluoride mouthrinse programs in fluoridated communities. *J Am Dent Assoc* 1985 Nov;111(5):783-9.
- Bratthall D, Hansel Petersson G, Sundberg H. Reasons for the caries decline: what do the experts believe? *Eur J Oral Sci* 1996;104:416-22.
- Bravo M, Llodra JC, Baca P, Osorio E. Effectiveness of visible light fissure sealant (Delton) versus fluoride varnish (Duraphat): 24-month clinical trial. *Community Dent Oral Epidemiol* 1996 Feb;24(1):42-6.
- Brown LF, Spencer AJ. Special report—continuing education in periodontology—the Adelaide study. *Periodontol* 1989;10:12-13.
- Burt BA, editor. Proceedings for the workshop: cost-effectiveness of caries prevention in dental public health. *J Public Health Dent* 1989;49(SI)352:251-352.
- Burt BA, Eklund SA. Dentistry, dental practice, and the community, 5th ed. Philadelphia: W.B. Saunders; 1999.
- Calderone JJ, Davis JM. The New Mexico sealant program: a progress report. *J Public Health Dent* 1987 Summer;47(3):145-9.
- Calderone JJ, Mueller LA. The cost of sealant application in a state dental disease prevention program. *J Public Health Dent* 1983 Summer;43(3):249-54.
- Callanen VA, Weintraub JA, French DP, Connolly GN. Developing a sealant program: the Massachusetts approach. *J Public Health Dent* 1986 Summer;46(3):141-6.
- Canadian Task Force on the Periodic Health Examination. The periodic health examination. *Can Med Assoc J* 1979 Nov 3;121(9):1193-254.
- Canto MT, Horowitz AM, Goodman HS, Watson MR, Cohen LA, Fedele DJ. Maryland veterans' knowledge of risk factors for and signs of oral cancers and their use of dental services. *Gerodontology* 1998a;15(2):79-86.
- Canto MT, Kawaguchi Y, Horowitz AM. Coverage and quality of oral cancer information in the popular press: 1987-98. *J Public Health Dent* 1998b Summer;58(3):241-7.
- Cathcart JF. Mouth protectors for contact sports. *Dent Digest* 1982;57:348.
- Centers for Disease Control (CDC). Bicycle-related injuries: data from the National Electronic Injury Surveillance System. *MMWR Morb Mortal Wkly Rep* 1987 May;36(17):269-71.

- Centers for Disease Control (CDC). Guidelines for school health programs to prevent tobacco use and addiction. *MMWR Morb Mortal Wkly Rep* 1994a Feb 25;43(RR-2):1-18.
- Centers for Disease Control (CDC). Preventing tobacco use among young people: a report of the Surgeon General. Executive summary. *MMWR Morb Mortal Wkly Rep* 1994b Mar 11;(RR-4):1-10.
- Centers for Disease Control and Prevention (CDC). Examinations for oral cancer—United States, 1992. *MMWR Morb Mortal Wkly Rep* 1994c;43:198-200.
- Centers for Disease Control and Prevention (CDC). Engineering and administrative recommendations for water fluoridation, 1995. *MMWR Morb Mortal Wkly Rep* 1995 Sep 29;44(RR-13):1-40.
- Centers for Disease Control and Prevention (CDC). Preventing and controlling oral and pharyngeal cancer. Recommendations from a National Strategic Planning Conference. *MMWR Morb Mortal Wkly Rep* 1998 Aug 28;47(RR-14):1-12.
- Centers for Disease Control and Prevention (CDC). Fluoridation of drinking water to prevent dental caries. *MMWR Morb Mortal Wkly Rep* 1999;48:933-940.
- Centers for Disease Control and Prevention (CDC). Recommendations for using fluoride to prevent and control dental caries in the United States. *MMWR Morb Mortal Wkly Rep* (in press).
- Chen MS, Andersen RM, Barmes DE, Leclercq MH, Lytle CS. Comparing oral health care systems: A second international collaborative study. Geneva: World Health Organization; 1997.
- Chung V, Horowitz AM, Canto MT, Siriphant P. Oral cancer educational materials for the general public: 1998. *J Public Health Dent* 2000;60:49-52.
- Clark DC, Stamm JW, Tessier C, Robert G. The final results of the Sherbrooke-Lac Megantic fluoride varnish study. *J Can Dent Assoc* 1987;53:919-22.
- Clarkson J. A European view of fluoride supplementation. *Br Dent J* 1992 May 9;172(9):357.
- Clarkson HB, Fejerskov O, Ekstrand J, Burt BA. Rational use of fluorides in caries control. In: Fejerskov O, Ekstrand J, Burt BA, editors. *Fluorides in dentistry*. 2nd ed. Copenhagen: Munksgaard; 1996. p. 347-57.
- Collins WJ, McCall DR, Strang R, Main C, Campbell D, Stephen KW, McKenzie R. Experience with a mobile fissure sealing unit in the Greater Glasgow area: results after three years. *Community Dent Health* 1985 Sep;2(3):195-202.
- Community Intervention Trial for Smoking Cessation (COMMIT): I. Cohort results from a four-year community intervention. *Am J Public Health* 1995a; 85(2):183-92.
- Community Intervention Trial for Smoking Cessation (COMMIT): II. Changes in adult cigarette smoking prevalence. *Am J Public Health* 1995b;85(2):193-200.
- Corbin SB, Maas WR, Kleinman DV, Backinger CL. NHIS findings on public knowledge and attitudes about oral diseases and preventive measures. *Public Health Rep* 1985;102:53-60.
- Dean HT, Jay P, Arnold FA Jr, Elvove E. Domestic water and dental caries II. A study of 2,832 white children aged 12-14 years, of eight suburban Chicago communities, including *L. acidophilus* studies of 1,761 children. *Public Health Rep* 1941;56:761-92.
- Dean HT, Arnold FA Jr, Jay P, Knutson JW. Studies on mass control of dental caries through fluoridation of the public water supply. *Public Health Rep* 1950;65:1403-8.
- de Bruyn H, Arends J. Fluoride varnishes—a review. *J Biol Buccale* 1987;15:71-82.
- Dental Products Report. Periodontal care report. Periodontal care in general practice: overview & update. 1996 Aug; p. 46-51.
- DePaola PF, Lax M. The caries-inhibiting effect of acidulated phosphate-fluoride chewable tablets: a two-year double-blind study. *J Am Dent Assoc* 1968 Mar;76(3):554-7.
- Disney JA, Graves RJ, Stamm JW, Bohannon HM, Abernathy JR. Comparative effects of a 4-year fluoride mouthrinse program on high and low caries forming grade 1 children. *Community Dent Oral Epidemiol* 1989;17:139-43.
- Driscoll WS. The use of fluoride tablets for the prevention of dental caries. In: Forrester DJ, Schulz EM, editors. *International workshop for the prevention of dental caries*. Baltimore: University of Maryland; 1974. p. 25-111.
- Driscoll WS, Heifetz SB, Korts DC. Effect of chewable fluoride tablets on dental caries in schoolchildren: results after six years of use. *J Am Dent Assoc* 1978 Nov;97(5):820-4.
- Elwood JM, Gallagher RP. Factors influencing early diagnosis of cancer of the oral cavity. *Can Med Assoc J* 1985;133:651-6.
- Fejerskov O, Manji F, Baelum V. The nature and mechanisms of dental fluorosis in man. *J Dent Res* 1990;69(SI):692-700.
- Fiore MC. Smoking cessation: a systems approach. Clinical practice guideline no. 18. Rockville (MD): Agency for Health Care Policy and Research; 1997. AHCPR no. 97-0698. Available from: <http://www.ahcpr.gov/clinic/smoksys.htm>.
- Forrest JL, Horowitz AM, Shmueli Y. Dental hygienists' caries prevention knowledge and practices. *J Dent Res* 1998 [abstract].
- Galagan DJ, Vermillion JR. Determining optimum fluoride concentrations. *Public Health Rep* 1957;72: 491-3.
- Gallup Organization, Inc. A Gallup study of parents' behavior, knowledge and attitudes toward fluoride. Dent Office 1992:9-10.
- Garcia AI. Caries incidence and costs of prevention programs. *J Public Health Dent* 1989;49(5 Spec No): 259-71.

- Gift HC, Corbin SB, Nowjack-Raymer RE. Public knowledge about prevention of dental disease—1990 NHIS. *Public Health Rep* 1994;109:397-404.
- Gold RS, Horowitz AM. Oral health information in textbooks. Paper presented at: 121st Scientific Session of the American Public Health Association; 1993 Oct 26; San Francisco.
- Green LW, Kreuter MW. Health promotion planning: an educational and environmental approach. 3rd ed. Mountain View (CA): Mayfield Publishing Company; 1999.
- Greene JC, Louie R, Wycoff SJ. Preventive dentistry. I. Dental caries. *JAMA* 1989 Dec 22;262(24):3459-63.
- Grimard G, Nolan T, Carlin JB. Head injuries in helmeted child bicyclists. *Inj Prev* 1995 Mar;1(1):21-5.
- Hardison JR. The use of pit-and-fissure sealants in community public health programs in Tennessee. *J Public Health Dent* 1983 Summer;43(3):233-9.
- Helfenstein U, Steiner M. Fluoride varnishes (Duraphat): a meta-analysis. *Community Dent Oral Epidemiol* 1994 Feb;22(1):1-5.
- Heller KE, Reed SG, Bruner FW, Eklund SA, Burt BA. Longitudinal evaluation of sealing molars with and without incipient dental caries in a public health program. *J Public Health Dent* 1995 Summer;55:148-53.
- Hennon DK, Stookey GK, Muhler JC. The clinical anticariogenic effectiveness of supplementary fluoride-vitamin preparations—results at the end of four years. *J Dent Child* 1967 Nov;34(6):439-43.
- Holm AK. Effect of fluoride varnish (Duraphat) in pre-school children. *Community Dent Oral Epidemiol* 1979 Oct;7(5):241-5.
- Horowitz AM, Nourjah PA. Patterns of screening for oral cancer among U.S. adults. *J Public Health Dent* 1996;56:331-5.
- Horowitz AM, Suomi JD, Peterson JK, Mathews BL, Voglesong RH, Lyman BA. Effects of supervised daily dental plaque removal by children after 3 years. *Community Dent Oral Epidemiol* 1980;8:171-6.
- Horowitz AM, Nourjah P, Gift HG. U.S. adult knowledge of risk factors for and signs of oral cancers: 1990. *J Am Dent Assoc* 1995;126:39-45.
- Horowitz AM, Goodman HS, Yellowitz JA, Nourjah PA. The need for health promotion in oral cancer prevention and early detection. *J Public Health Dent* 1996;56:319-30.
- Horowitz AM, Moon HS, Goodman HS, Yellowitz JA. Maryland adults' knowledge of oral cancer and having oral cancer examinations. *J Public Health Dent* 1998;58:281-7.
- Horowitz HS, Ismail AI. Topical fluorides in caries prevention. In: Fejerskov O, Ekstrand J, Burt BA, editors. *Fluoride in dentistry*. 2nd ed. Copenhagen: Munksgaard; 1996. Chapter 17.
- Hutton WL, Linscott BW, Williams DB. Final report of local studies on water fluoridation in Brantford. *Can J Public Health* 1956;47:89-92.
- Institute of Medicine (IOM), Food and Nutrition Board. Dietary reference intakes: calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington: National Academy Press; 1997.
- Ismail AI. Fluoride supplements: current effectiveness, side effects and recommendations. *Community Dent Oral Epidemiol* 1994 Jun;22(3):164-72.
- Ismail AI, Lewis DW. Periodic health examination, 1993 update: 3. Periodontal diseases: classification, diagnosis, risk factors and prevention. Canadian Task Force on the Periodic Health Examination. *Can Med Assoc J* 1993 Nov 15;149(10):1409-22.
- Ismail AI, King W, Clark DC. An evaluation of the Saskatchewan pit and fissure sealant program: a longitudinal followup. *J Public Health Dent* 1989;49(4):206-11.
- Johnson RM, McCarthy MC, Miller SF, Peoples JB. Craniofacial trauma in injured motorcyclists: the impact of helmet usage. *J Trauma* 1995 Jun;38(6):876-8.
- Johnston DW. Current status of professionally applied topical fluorides. *Community Dent Oral Epidemiol* 1994 Jun;22(3):159-63.
- Joint Commission on National Health Education Standards. National health education standards: achieving health literacy. Atlanta: American Cancer Society; 1995.
- Karlson TA. Injury control and public policy. *Crit Rev in Environ Control* 1992;22:195-241.
- Kaste LM, Selwitz RH, Oldakowski RJ, Brunelle, JA, Winn DM, Brown LJ. Coronal caries in the primary and permanent dentition of children and adolescents 1-17 years of age: United States, 1988-1991. *J Dent Res* 1996 Feb;75(Spec No):631-41.
- Kirkegaard E, Petersen G, Poulsen S, Holm SA. Caries-preventive effect of Duraphat varnish application versus fluoride mouthrinses: 5-year data. *Caries Res* 1986;20:548-55.
- Klausner R. Evolution of tobacco control studies at the National Cancer Institute. *Tob Control* 1997;6(Suppl):2:S1-2.
- Klein SP, Bohannon HM, Bell RM, Disney JA, Foch CB, Graves RC. The cost and effectiveness of school-based preventive dental care. *Am J Public Health* 1985 Apr;75(4):382-91.
- Koch G, Petersson LG, Ryden H. Effect of fluoride varnish (Duraphat) treatment every six months compared with weekly mouthrinses with 0.2 percent NaF solution on dental caries. *Swed Dent J* 1979;3(2):39-44.
- Kugel W, Fischer T. Rise and fall of caries prevalence in German towns with different F concentrations in drinking water. *Caries Res* 1997;31:166-73.
- Kumar JV, Davila ME, Green EL, Lininger LL. Evaluation of a school-based sealant program in New York State. *J Public Health Manag Pract* 1997 May;3(3):43-51.
- Last JM. A dictionary of epidemiology. 3rd ed. New York: Oxford University Press; 1995.

- Lee MC, Chiu WT, Chang LT, Liu SC, Lin SH. Craniofacial injuries in unhelmeted riders of motorbikes. *Injury* 1995 Sep;26(7):467-70.
- Leverett DH. Effectiveness of mouthrinsing with fluoride solutions in preventing coronal and root caries. *J Public Health Dent* 1989;49(5 Spec No):310-6.
- Leverett DH, Adair SM, Vaughn BW, Proskin HM, Moss ME. Randomized clinical trial of the effect of prenatal fluoride supplements in preventing dental caries. *Caries Res* 1997;31(3):174-9.
- Lewis DW, Ismail AI. Periodic health examination, 1995 update: 2. Prevention of dental caries. Canadian Task Force on the Periodic Health Examination. *Can Med Assoc J* 1995 Mar 5;152(6):836-46.
- Lewit EM, Hyland A, Kerrebrock N, Cummings KM. Price, public policy, and smoking in young people. *Tob Control* 1997;6(Suppl):2:S17-24.
- Llodra JC, Bravo M, Delgado-Rodriguez M, Baca P, Galvey R. Factors influencing the effectiveness of sealants—a meta-analysis. *Community Dent Oral Epidemiol* 1993 Oct;21(5):261-8.
- Loe H, Theilade E, Jensen SB. Experimental gingivitis in man. *J Periodontol* 1965 May-Jun;36:177-87.
- Lynch BS, Bonnie RJ. Institute of Medicine. Growing up tobacco free: preventing nicotine addiction in children and youths. Washington: National Academy Press; 1994.
- Lynch GR, Prout MN. Screening for cancer by residents in an internal medicine program. *J Med Educ* 1986;61:387-93.
- Maestrello-deMoya MG, Primosch RE. Orofacial trauma and mouth-protector wear among high school varsity basketball players. *ASDC J Dent Child* 1989 Jan-Feb;56(1):36-9.
- Mandel ID. Fluoride varnishes—a welcome addition [editorial]. *J Public Health Dent* 1994;54:67.
- Manley MW, Lynn W, Epps R, Grande D, Glynn T, Shopland D. The American Stop Smoking Intervention Study for cancer prevention: an overview. *Tob Control* 1997a;6(Suppl 2):S5-11.
- Manley MW, Pierce JP, Gilpin EA, Rosbrook B, Berry C, Wun LM. Impact of the American Stop Smoking Intervention Study on cigarette consumption. *Tob Control* 1997b;6(Suppl 2):S12-6.
- Marine WM, Kerwin EM, Moore EE, Lezotte DC, Baron AE, Grosso MA. Mandatory seatbelts: epidemiologic, financial, and medical rationale from the Colorado matched pairs study. *J Trauma* 1994 Jan;36(1):96-100.
- McDonald AK. The National Electronic Injury Surveillance System: a tool for researchers. U.S. Consumer Product Safety Commission, October 1994.
- McGavran EG. What is public health? In: Barr HH, Barrie FH, editors. *Edward G. McGavran: guardian of the body politic*. Chapel Hill: School of Public Health, The University of North Carolina; 1979.
- McNutt T, Shannon SW Jr, Wright JT, Feinstein RA. Oral trauma in adolescent athletes: a study of mouth protectors. *Pediatr Dent* 1989 Sep;11(3):209-13.
- Meadow D, Lindner G, Needleman H. Oral trauma in children. *Pediatr Dent* 1984 Dec;6(4):248-51.
- Mertz-Fairhurst EJ. Current status of sealant retention and caries prevention. *J Dent Educ* 1984 Feb;48(2 Suppl):18-26.
- Messer LB, Calache H, Morgan MV. The retention of pit and fissure sealants placed in primary school children by Dental Health Services, Victoria. *Aust Dent J* 1997 Aug;42(4):233-9.
- Morgan MV, Crowley SJ, Wright C. Economic evaluation of a pit and fissure dental sealant and fluoride mouthrinsing program in two nonfluoridated regions of Victoria, Australia. *J Public Health Dent* 1998 Winter;58(1):19-27.
- Murphy GP, Lawrence W, Lenhard RE, editors. *American Cancer Society textbook of clinical oncology*. 2nd ed. Atlanta: American Cancer Society; 1995.
- Murray JJ, Naylor MN. Fluorides and dental caries. In: Murray JJ, editor. *The prevention of oral disease*. 3rd ed. Oxford; New York: Oxford University Press; 1996.
- Murray JJ, Rugg-Gunn AJ, Jenkins GN. Fluorides in caries prevention. 3rd ed. Oxford; Boston: Wright; 1991.
- National Cancer Institute (NCI). Community-based interventions for smokers: the COMMIT field experience. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute, 1995. Smoking and tobacco control monograph 6. NIH Pub. no. 95-4028.
- National Center for Health Statistics (NCHS). Healthy People 2000 review. Hyattsville (MD): U.S. Department of Health and Human Services Public Health Service, Centers for Disease Control and Prevention, National Center for Health Statistics; 1992. p. 90-95. Available from US GPO, Washington. PHS Pub. no. 93-1232-1.
- National Center for Health Statistics (NCHS). Healthy People 2000 review, 1998-99. Hyattsville (MD): U.S. Public Health Service; 1999.
- National Center for Injury Prevention and Control, Centers for Disease Control and Prevention. Injury-control recommendations: bicycle helmets. *MMWR Morb Mortal Wkly Rep* 1995 Feb 17;44(RR-1):1-17.
- National Institutes of Health (NIH). Consensus Development Conference Statement. Dental sealants in the prevention of tooth decay. *J Dent Educ* 1984 Feb;48(2 Suppl):126-31.
- National Research Council (NRC). *Health effects of ingested fluoride*. Washington: National Academy Press; 1993.
- Newbrun E. Effectiveness of water fluoridation. *J Public Health Dent* 1989;49(5 Spec No):279-89.

- Newbrun E. The fluoridation war: a scientific dispute or a religious argument? *J Public Health Dent* 1996;56(5 Spec No):246-52.
- Nourjah P, Horowitz AM, Wagener DK. Factors associated with the use of fluoride supplements and fluoride dentifrice by infants and toddlers. *J Public Health Dent* 1994;54(1):47-54.
- Nowjack-Raymer RE, Gift HC. Use of mouthguards and headgear in organized sports by school-aged children. *Public Health Rep* 1996 Jan-Feb;111(1):82-6.
- O'Mullane DM. Efficiency in clinical trials of caries preventive agents and methods. *Community Dent Oral Epidemiol* 1976 Sep;4(5):190-4.
- O'Neil HW. Opinion study comparing attitudes about dental health. *J Am Dent Assoc* 1984;109:910-5.
- O'Rourke CA, Attrill M, Holloway PJ. Cost appraisal of a fluoride tablet programme to Manchester primary school children. *Community Dent Oral Epidemiol* 1988 Dec;16(6):341-4.
- Orsay EM, Dunne M, Turnbull TL, Barrett JA, Langenberg P, Orsay CP. Prospective study of the effect of safety belts in motor vehicle crashes. *Ann Emerg Med* 1990 Mar;19(3):258-61.
- Petersson LG. Fluoride mouthrinses and fluoride varnishes. *Caries Res* 1993;27(Suppl 1):35-42.
- Petersson LG, Arthursson L, Ostberg C, Jonson P, Gleerup A. Caries-inhibiting effects of different modes of Duraphat varnish reapplication: a 3-year radiographic study. *Caries Res* 1991;25:70-3.
- Peyron M, Matsson L, Birkhed D. Progression of approximal caries in primary molars and the effect of Duraphat treatment. *Scand J Dent Res* 1992 Dec;100(6):314-8.
- Powers JM, Godwin WC, Heintz WD. Mouth protectors and sports team dentists. Bureau of Health Education and Audiovisual Services, Council on Dental Materials, Instruments, and Equipment. *J Am Dent Assoc* 1984 Jul;109(1):84-7.
- Prout MN, Heeren TC, Barber CE, et al. Use of health services before diagnosis of head and neck cancer among Boston residents. *Am J Prev Med* 1990;6:77-83.
- Rampton J, Leach T, Therrien SA, Bota GW, Rowe BH. Head, neck, and facial injuries in ice hockey: the effect of protective equipment. *Clin J Sports Med* 1997 Jul;7(3):162-7.
- Reeves T. (CDC Division of Oral Health) Personal communication. 1999 May 2.
- Reinfurt DW, St Cyr CL, Hunter WW. Usage patterns and misuse rates of automatic seat belts by system type. *Accid Anal Prev* 1991 Dec;23(6):521-30.
- Ringelberg ML, Allen SJ, Brown LJ. Cost of fluoridation: 44 Florida communities. *J Public Health Dent* 1992;52(2):75-80.
- Ripa LW. An evaluation of the use of professional (operator-applied) topical fluorides. *J Dent Res* 1990 Feb;69(Spec No):786-96.
- Ripa LW. A critique of topical fluoride methods (dentifrices, mouthrinses, operator and self-applied gels) in an era of decreased caries and increased fluorosis prevalence. *J Public Health Dent* 1991 Winter;51(1):23-41.
- Ripa LW. Sealants revisited: an update of the effectiveness of pit and fissure sealants. *Caries Res* 1993;27(Suppl 1):77-82.
- Rivara FP, Dicker BG, Bergman AB, Dacey R, Herman C. The public cost of motorcycle trauma. *JAMA* 1988 Jul 8;260(2):221-3.
- Rivara FP, et al. The Seattle children's bicycle helmet campaign: changes in helmet use and head injury admissions. *Pediatrics* 1994 Apr;93(4):567-9.
- Rivara FP, Thompson DC, Thompson RS. Epidemiology of bicycle injuries and risk factors for serious injury. *Inj Prev* 1997 Jun;3(2):110-4.
- Roper Report on Oral Health. Study reveals conflicting ideas about periodontal disease. *Dent Today* 1994 Feb.
- Sacks JJ, Kresnow M, Houston B, Russell J. Bicycle helmet use among American children, 1994. *Inj Prev* 1996 Dec;2(4):258-62.
- Sane J. Comparison of maxillofacial and dental injuries in four contact team sports: American football, bandy, basketball, and handball. *Am J Sports Med* 1988 Nov-Dec;16(6):647-51.
- Seppä L. Studies of fluoride varnishes in Finland. *Proc Finn Dent Soc* 1991;87(4):541-7.
- Seppä L, Pollanen L. Caries preventive effect of two fluoride varnishes and a fluoride mouthrinse. *Caries Res* 1987;21(4):375-9.
- Seppä L, Tolonen T. Caries preventive effect of fluoride varnish applications performed two or four times a year. *Scand J Dent Res* 1990;98:102-5.
- Seppä L, Leppanen T, Hausen H. Fluoride varnish versus acidulated phosphate fluoride gel: a 3-year clinical trial. *Caries Res* 1995;29:327-30.
- Seppä L, Karkkainen S, Hausen H. Caries frequency in permanent teeth before and after discontinuation of water fluoridation in Kuopio, Finland. *Community Dent Oral Epidemiol* 1998;26:256-62.
- Shopland DR. Smoking control in the 1990s: a National Cancer Institute model for change. *Am J Public Health* 1993 Sep;83:1208-10.
- Siegel MD, Garcia AI, Kandray DP, Giljahn LK. The use of dental sealants by Ohio dentists. *J Public Health Dent* 1996 Winter;56(1):12-21.
- Siegel MD, Farquhar CL, Bouchard JM. Dental sealants. Who needs them? *Public Health Rep* 1997a Mar-Apr;112(2):98-106.
- Siegel MD, Lalumandier JA, Farquhar CL, Bouchard JM. School-based and school-linked public health sealant programs in the United States, 1992-93. Columbus (OH): Association of State and Territorial Dental Directors; 1997b.
- Silverman S. American Cancer Society. Oral cancer. 4th ed. St. Louis: Mosby-Year Book; 1998.
- Simonsen RJ. Retention and effectiveness of dental sealant after 15 years. *J Am Dent Assoc* 1991 Oct;122(11):34-42.

- Skold L, Sundquist B, Eriksson B, Edeland C. Four-year study of caries inhibition of intensive Duraphat application in 11-15-year-old children. *Community Dent Oral Epidemiol* 1994;22:8-12.
- Smith GS, Falk H. Unintentional injuries. *Am J Prev Med* 1987;3:143-63.
- Sorenson WW. A look at air bag effectiveness using U.S. insurance company data. *Proceedings of the International Conference on Air Bags and Seat Belts: evaluation and implications for public policy*; 1993.
- Sosin DM, Sacks JJ, Holmgren P. Head injury—associated deaths from motorcycle crashes. Relationship to helmet-use laws. *JAMA* 1990 Nov 14; 264(18): 2395-9.
- Stamm JW, Bohannon HM, Graves RC, Disney JA. The efficiency of caries prevention with weekly fluoride mouthrinses. *J Dent Educ* 1984 Nov;48(11):617-26.
- Stephen KW. Systemic fluorides: drops and tablets. *Caries Res* 1993;27(Suppl 1):9-15.
- Stephen KW, Campbell D. Caries reduction and cost benefit after 3 years of sucking fluoride tablets daily at school. A double blind trial. *Br Dent J* 1978 Apr 4;144(7):202-6.
- Stephen KW, McCall DR, Tullis JL. Caries prevalence in northern Scotland before, and 5 years after, water defluoridation. *Br Dent J* 1987 Nov 21;163(10): 324-6.
- Sterritt GR, Frew RA. Evaluation of a clinic-based sealant program. *J Public Health Dent* 1988 Fall;48(4): 220-4.
- Sterritt GR, Frew RA, Rozier RG, Brunelle JA. Evaluation of a school-based fluoride mouthrinsing and clinic-based sealant program on a non-fluoridated island. *Community Dent Oral Epidemiol* 1990 Dec;18(6):288-93.
- Stokey GK, Beiswanger BB. Topical fluoride therapy. In: Harris NO, Christen AG, editors. *Primary preventive dentistry*. 4th ed. Norwalk (CT): Appleton and Lange; 1995. Chapter 9. p. 193-233.
- Sundberg H, Bjerner B, Sjogren K. Estimation of the prophylactic measures in Swedish public dental health care. Results from a questionnaire. *Eur J Oral Sci* 1996 Aug;104(4 Pt 2):477-9.
- Sutyak JP, Passi V, Hammond JS. Air bags alone compared with the combination of mechanical restraints and air bags: implications for the emergency evaluation of crash victims. *South Med J* 1997 Sep;90(9): 915-9.
- Task Force on Community Preventive Services. *Introducing the guide to community preventive services: methods, first recommendations and expert commentary*. *Am J Prev Med* 2000 Jan;18(1S).
- Thompson DC, Nunn ME, Thompson RS, Rivara FP. Effectiveness of bicycle safety helmets in preventing serious facial injury. *JAMA* 1996;276(24):1974-5.
- Torell P, Ericsson Y. Two-year clinical tests with different methods of local caries-preventive fluorine application in Swedish school-children. *Acta Odontol Scand* 1965 Jun;23:287-322.
- Torell P, Ericsson Y. The potential benefits to be derived from fluoride mouthrinses. In: Forrester DJ, Schulz EM, editors. *International workshop on fluorides and dental caries reductions*. Baltimore: University of Maryland; 1974. p. 113-76.
- Twetman S, Petersson LG, Pakhomov GN. Caries incidence in relation to salivary mutans streptococci and fluoride varnish applications in preschool children from low- and optimal-fluoride areas. *Caries Res* 1996;30(5):347-53.
- U.S. Consumer Product Safety Commission (USCPC). *Overview of sports related injuries to persons 5-14 years of age*. Washington: U.S. Consumer Product Safety Commission, 1981.
- U.S. Consumer Product Safety Commission (USCPC). *Tricycles: reporting hospitals and estimates reports 1982-1986*. Washington: National Electronic Injury Surveillance System, U.S. Consumer Product Safety Commission; 1986.
- U.S. Department of Health, Education, and Welfare (USDHEW). *Public Health Service drinking water standards, revised 1962*. Washington: U.S. Department of Health, Education, and Welfare. Public Health Service; 1962. PHS Pub. no. 956.
- U.S. Department of Health and Human Services (USDHHS). *Review of fluoride benefits and risks: report of the Ad Hoc Subcommittee on Fluoride of the Committee to Coordinate Environmental Health and Related Programs*. Washington: U.S. Department of Health and Human Services, Public Health Service; 1991.
- U.S. Department of Health and Human Services (USDHHS). *Fluoridation census, 1992*. Atlanta: U.S. Public Health Service, Centers for Disease Control and Prevention, Division of Oral Health; 1993.
- U.S. Department of Health and Human Services (USDHHS). *Preventing tobacco use among young people: a report of the Surgeon General*. Atlanta: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1994.
- U.S. Department of Health and Human Services (USDHHS). *Healthy People 2010*. (conference edition in two volumes). Washington: U.S. Department of Health and Human Services; 2000.
- U.S. Department of Transportation (USDOT). *Fatal accident reporting system: a review of the information fatal traffic crashes in the United States annual reports, 1975-1993*. Washington: U.S. Department of Transportation, National Highway Traffic Safety Administration, National Center for Statistics and Analysis; 1998.
- U.S. Preventive Services Task Force (USPSTF). *Guide to clinical preventive services*. 2nd ed. Baltimore: Williams and Wilkins; 1996.
- Vaughan RG. Motorcycle helmets and facial injuries. *Med J Aust* 1977 Jan 29;1(5):125-7.

- Vehmanen R. An economic evaluation of two caries preventive methods [dissertation]. Turku, Finland: University of Turku; 1993 Nov.
- Warren DP, Henson HA, Chan JT. A survey of in-office use of fluorides in the Houston area. *J Dent Hyg* 1996;70:166-71.
- Watson MR, Horowitz AM, Garcia I, Canto MT. Caries conditions among 2-5 year old immigrant Latino children related to parents' oral health knowledge, opinions and practices. *Community Dent Oral Epidemiol* 1999;27:8-15.
- Weintraub JA. The effectiveness of pit and fissure sealants. *J Public Health Dent* 1989;49(5 Spec No):317-30.
- Weintraub JA, Stearns SC, Burt BA, Beltran E, Eklund SA. A retrospective analysis of the cost-effectiveness of dental sealants in a children's health center. *Soc Sci Med* 1993 Jun;36(11):1483-93.
- Whyte RJ, Leake JL, Hawkey TP. Two-year follow-up of 11,000 dental sealants in first permanent molars in the Saskatchewan Health Dental Plan. *J Public Health Dent* 1987 Fall;47(4):177-81.
- Wilkinson R. *Unhealthy societies: the afflictions of inequality*. London: Routledge; 1996.
- Winn DM, Sandberg AL, Horowitz AM, Diehl SR, Gutkind S, Kleinman DV. Reducing the burden of oral and pharyngeal cancers. *J Calif Dent Assoc* 1998 Jun;26(6):445-51,454.
- World Health Organization (WHO). *Fluorine and fluorides: environmental health criteria 36*. Geneva: World Health Organization; 1984.
- World Health Organization (WHO). *Fluorides and oral health*. Geneva: The World Health Organization; 1994.
- Yellowitz JA, Goodman HS. Assessing physicians' and dentists' oral cancer knowledge, opinions and practices. *J Am Dent Assoc* 1995 Jan;126(1):53-60.
- Yellowitz JA, Horowitz AM, Goodman HS, Canto MT, Farooq NS. Knowledge, opinions and practices of general dentists regarding oral cancer: a pilot survey. *J Am Dent Assoc* 1998 May;129(5):579-83.

Personal and Provider Approaches to Oral Health

Oral health is not a given. It takes conscious and repeated efforts on the part of the individual, caregivers, health care providers, and the community. For the individual, daily hygiene routines and healthy lifestyle behaviors provide a frontline defense in disease prevention and health promotion. Equally important are periodic professional assessments of the individual's oral health status, which may include diagnostic, preventive, and therapeutic services and counseling. Community activities complement personal and provider approaches to oral health. As discussed in the previous chapter, these include water fluoridation, dental sealant applications for children, tobacco cessation campaigns, the use of mouthguards in sports, and a variety of other school- and community-based oral health promotion and disease prevention activities. The interaction of these components is critical to oral health, as it is to overall health. In particular, there is now a better understanding of the relationship of individual health to the health of the community in which the individual lives, and the importance of this relationship is one of the underlying premises of Healthy People 2010. This chapter discusses actions individuals can take to maintain their oral health and prevent disease, and reviews emerging approaches taken by dentists and other health care providers to promote oral health, assess risks, and prevent disease.

INDIVIDUAL RESPONSIBILITY: PERSONAL APPROACHES TO ORAL HEALTH

Sound personal hygiene practices and adherence to a healthy lifestyle are the mainstays of personal approaches to oral health. Long before the germ theory of disease, the need for tooth cleaning was recognized—if only to rid the mouth of food debris, eliminate odor, and improve appearance. Tools developed

for this purpose have ranged from primitive tooth sticks and picks, still used in parts of the world, to the water irrigators and electronic toothbrushes available in industrialized societies. An impressive array of oral care products greets the shopper in supermarkets and pharmacies today. Beyond the dozens of toothbrush shapes and sizes, there are flavored and textured dental flosses, floss holders, rubber tips, toothpicks, small brushes for cleaning between teeth, scores of dentifrices, and a range of fluoride-containing, antitartar, and antiseptic mouthrinses.

Daily oral hygiene efforts contribute to the prevention of dental caries and periodontal diseases. The biofilm on tooth and root surfaces (dental plaque) can be disrupted to a large extent by the mechanical action of brushing and flossing. Daily efforts are necessary, not only because of food intake, but also because dental plaque is never completely removed. It starts to build up even after the most assiduous cleaning (or prophylaxis) in the dental office and even after the application of a potent antimicrobial mouthrinse. The oral and dental tissues and structures thus require more intensive daily care than do other body areas exposed to the environment.

Daily Hygiene and Dental Caries Prevention

The use of a fluoride-containing dentifrice is critical for dental caries prevention. Even more beneficial than the physical removal of plaque in toothbrushing is the delivery of a small amount of fluoride to the tooth surfaces. Investigators have conducted numerous clinical trials on fluoride dentifrices using rigorous designs and including randomized groups, double-blind designs, and placebo controls. All together, these studies provide strong evidence that using a

fluoride dentifrice is effective (Clarkson et al. 1993, Lewis and Ismail 1995, Stookey et al. 1993). Fluoride dentifrices account for more than 90 percent of the market in the United States, Canada, and other developed countries (Levy 1994).

A fluoride dentifrice is an effective means of reducing the prevalence of dental caries for all persons. Although children's teeth should be cleaned daily from the time they erupt, parents and caregivers should consult a dentist or other health care provider about the use of a fluoride dentifrice for children under the age of 2. For children under 6, fluoride dentifrices should be used in small amounts to minimize swallowing of the product. Mild enamel fluorosis can result from excessive dentifrice use, because children under 6 do not have adequate control of the swallowing reflex or may intentionally swallow a flavored dentifrice. Experts recommend that for children under 6, the parent or caregiver should supervise toothbrushing, apply a pea-sized amount (0.25 gram) of dentifrice to the toothbrush, and encourage the child to spit out the excess (Bawden 1992).

Because the topical benefits of fluoride have been shown to be highly effective, and daily exposure to small amounts of fluoride can reduce the risk of dental caries in all age groups, experts recommend that all persons drink water with an optimal fluoride concentration in addition to brushing daily with a fluoride dentifrice (Bawden 1992, CDC in press). This combination provides a cost-effective and easy way to prevent dental caries and is an excellent example of the individual-community partnership. For persons at low risk of dental caries, these two exposures to fluoride may be the only ones necessary. For persons at moderate or high risk of dental caries, additional fluoride may be helpful and can come from daily use of another fluoride product. These can include mouthrinses, prescribed supplements, and professionally applied topical fluoride products (CDC in press).

Daily Hygiene and the Prevention of Periodontal Diseases

Toothbrushing and flossing also play a critical role in the prevention of periodontal diseases. Unlike dental caries prevention, prevention and control of gingivitis and periodontitis are achieved directly through the mechanical removal and disruption of dental plaque (Genco and Newman 1996). Some dentifrices also contain chemical therapeutics to control the formation of tartar (calculus) (Mandel 1995) and to reduce plaque formation and gingival inflammation (Hancock 1996). Both manual and electric tooth-

brushing are effective at removing plaque and preventing gingivitis (Walsh et al. 1989, Axelsson et al. 1991). Interproximal (between the teeth) cleaning is also important in maintaining gingival health (Lang et al. 1994). In one short-term evaluation of adults, the addition of flossing to the daily regimen of brushing resulted in an almost twofold reduction in gingival inflammation (Graves et al. 1989). Because preventive measures in periodontics rely mainly on the removal of bacterial plaque and calculus, methods typically include personal oral hygiene measures combined with professional diagnostic and prophylactic measures (i.e., regular exam and cleaning). Periodic professional care for removal of plaque and calculus deposits has also been demonstrated to improve the periodontal health of participants (Cutress et al. 1991, Ronis et al. 1993).

Healthy Lifestyles

There is more to the individual's role in promoting oral health and hygiene than brushing and flossing. Other behaviors that have an influence on oral health include use of tobacco and/or alcohol products, diet, oral habits such as bruxing and clenching the teeth, and use of helmets, mouthguards, or other protective devices. Table 8.1 summarizes selected behaviors that have an effect on oral, dental, and craniofacial health status. These are described more fully in Chapters 3, 7, and 10.

Individuals can obtain credible information regarding oral health from various sources, including health care providers, professional organizations, government agencies, and patient advocacy groups. Increasingly, the World Wide Web is a source for health care information. For example, the National Oral Health Information Clearinghouse offers information on oral health, with an emphasis on special care patients and their health care providers.

Care Seeking

In addition to self-care, individuals also need to seek professional health care—both dental and medical—on a regular basis and whenever a problem manifests. The recall interval is based on the provider's assessment of the individual's dental and medical history and lifestyle behaviors, among other factors. In the case of children and dependent adults, parents and caregivers are responsible for teaching and encouraging healthy behaviors and seeking timely and appropriate care. As noted at the outset, it is only through the combination of individual and professional care, reinforced by community-based health promotion

and disease prevention programs, that optimal oral and general health can be achieved. The remainder of this chapter focuses on the role of the professional in oral health care.

PROVIDER-BASED CARE

The range of conditions and diseases that affect the craniofacial complex is extensive and can provide clinicians with important indications about the patient's general as well as oral health status. Management of the oral health-general health interface calls for interdisciplinary and coordinated care and an enhanced role for primary care providers. Dentists, oncologists, dermatologists, infectious dis-

ease specialists, hematologists, endocrinologists, plastic surgeons, and rheumatologists are just a few of the specialists who may be involved in the diagnosis and management of conditions affecting the craniofacial complex.

Dentists, their allied staff, and medical and nursing personnel are in a unique position to incorporate new approaches for prevention,¹ diagnostic, and treatment strategies in their practices. Advances in oral science are providing the basis for a shift in emphasis from the repair and restoration of damaged tissues to earlier diagnoses, control of infections, and remineralization and regeneration of lost tissues. The application of risk assessment strategies and interventions tailored to individuals and groups is

TABLE 8.1
Selected individual behaviors affecting oral, dental, and craniofacial health

Behavior	Effect
Diet and nutrition	Nutrition and diet contribute to oral and craniofacial development and to the maintenance of these tissues throughout life. Nutritional deficiencies during pregnancy can affect tooth size, enamel solubility, time of tooth eruption, salivary gland function, saliva composition, epithelial tissue, and susceptibility to dental caries. Deleterious effects specific to the dentition include protein-calorie malnutrition, deficiencies of vitamin A, ascorbic acid, vitamin D, calcium, phosphorus, iron, and iodine, and excessive fluoride. Linear enamel hypoplasia and hypomineralization during the first year of life increase susceptibility to dental caries in both deciduous and permanent teeth. The physical consistency, sequence, and frequency of carbohydrate intake (primarily refined sugars) have been linked to the development of caries. Reduced calcium intake is associated with greater levels of periodontal disease. Early clinical signs of nutritional deficiencies and eating disorders are often seen in and around the mouth. Oral lesions may also affect systemic nutrition. (See Chapter 10, Box 10.1.)
Oral hygiene and home care practices	Regular toothbrushing with a fluoride-containing dentifrice prevents dental caries. Rinsing with fluoride mouthrinse can aid in reducing caries and in the remineralization of tooth structure. Regular toothbrushing and proper flossing can prevent gingivitis.
Care seeking	Seeking health care—both dental and medical—on a regular basis and whenever a problem manifests is important. In the case of children or dependent adults, it calls for the caregivers to teach and encourage healthy behaviors and to seek appropriate care from a variety of care providers. Prenatal care, as well as oral health care prior to major treatments such as chemotherapy, is critical to overall oral and craniofacial health.
Parafunctional habits	Habitual grinding (bruxism) and/or clenching teeth are forms of abnormal motor behavior. These habits often occur during sleep. As with clenching and other oral habits such as frequent gum-chewing, bruxism can cause tooth wear and affect muscles of mastication. Treatment may begin with making the individual aware of the problem, providing an occlusal splint to prevent tooth wear, and using behavioral strategies.
Tobacco use	The use of tobacco in all forms increases the risk for oral and pharyngeal cancers, and smoking is a leading risk factor for periodontal diseases. Increased risk for dental caries has been associated with spit tobacco use. In HIV-infected individuals, tobacco use is a risk factor contributing to increased risk of the development of oral candidiasis. (See Chapter 10, Box 10.2.)
Alcohol use	Alcohol alone, as well as acting synergistically with tobacco, greatly increases the risk for oral and pharyngeal cancers. Independently, alcohol in excess is associated with circulatory and neurological problems, liver disease, and other organ-specific diseases and disorders. Alcohol use in pregnancy can lead to birth defects, such as fetal alcohol syndrome and its associated craniofacial defects and mental retardation.
Injury control practices	Proper use of helmets, mouthguards, safety belts, and other protective devices helps prevent injuries to the head, neck, and mouth.

¹The term *prevention*, as used in this chapter, includes interventions aimed at reducing the incidence of disease in relatively healthy patients. It includes both health promotion and specific protection to control one or more risk factors. Some strategies, such as the prevention of tobacco use, are applicable to many oral diseases, including oral cancer, oral candidiasis, and periodontal diseases, whereas other strategies are specific, such as the use of dental sealants and fluorides for caries prevention.

expanding with the increased understanding of risk factors and the development of biomarkers that signal host resistance, susceptibility, and the presence and progression of disease.

The changing demographics of the U.S. population and a greater understanding of the relationship between oral health and general health are presenting new challenges. Making clinical decisions for patients requires integrating a range of interacting biological, psychological, social, cultural, and environmental factors. In order for disease to manifest, the etiologic agent(s) must be present, the host must be susceptible, the environment conducive, and sufficient time available for the factors to interact (Figure 8.1). Early diagnosis and prompt treatment require an understanding of the pathology and of the diagnostic, prevention, and treatment modalities available. As genetic information accumulates, clinical judgments will increasingly be informed by knowledge of an individual's genetic susceptibility or resistance to particular diseases and disorders. The development of tailored treatment plans will require incorporating all these factors together with input from the patient's health providers, taking into consideration the patient's interests and needs. The following sections provide an overview of emerging approaches to clinical management and highlight selected diseases as examples.

Risk Assessment

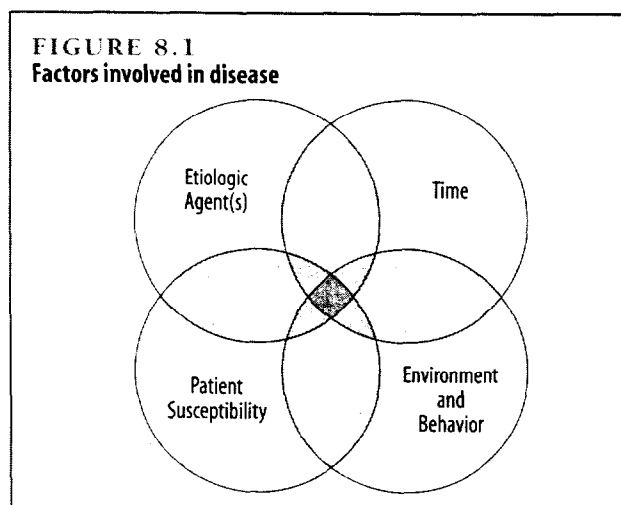
Given the greater understanding of disease etiology, epidemiology, patient characteristics, and genetic information, it is becoming increasingly possible to determine an individual's risk of disease and tailor treatments accordingly. Risk assessment for dentistry has been defined as "the use of knowledge of factors

that are associated with dental disease to determine which patients are more or less likely to prevent or control their dental disease" (Douglass 1998). The factors can include co-morbidities, medications used, and patient characteristics such as sex, age, and lifestyle behaviors, among others. By compiling such factors and sorting them by risk category, patients can be classified into high- or low-risk groups, enabling providers to make more comprehensive diagnoses and identify patients who would benefit from more aggressive prevention strategies. Such analyses conducted during the early stages of disease can result in treatments that reverse or contain the disease process (Douglass 1998). Knowledge of risk factors for oral and craniofacial diseases and disorders allows other health care providers to screen for these risk factors and contribute to improving oral health.

Risk assessment and disease prediction studies have focused primarily on dental caries and periodontal diseases (Genco 1996, Page and Beck 1997, Pitts 1998, Powell 1998). In addition, risk factors for oral and pharyngeal cancers have been explored (Johnson 1991). The evidence base for risk assessment is developing from population-based studies. It involves a research process in which a suspected risk factor is related in a multivariate model to other factors and confounders (Beck 1990). The resulting model is tested in a second group of subjects, and a targeted intervention study is conducted to confirm the predictive validity of the risk factor.

Although the application of research findings of risk assessment has begun in some practices, the prediction of future disease at the individual patient level has not yet been extensively studied. Douglass (1998) has posed six clinically oriented questions that need to be addressed if risk assessment is to be adopted into routine clinical practice:

1. Does the scientific theory or biologic logic of the risk factor fit with our current body of knowledge about the disease in question?
2. Has the technical merit of identifying the risk factor (such as imaging technologies and bacterial assays) been evaluated?
3. Has the efficacy of the risk factor in predicting disease been evaluated in terms of sensitivity, specificity, and positive and negative predictive values?²
4. Has the potential effect of the risk factor on the disease management decision been explored? Can knowledge of the presence of a particular risk factor or pattern of risk factors alter the treatment plan?



5. Has the influence of the risk factor on oral health outcomes been assessed?
6. Has the cost-effectiveness of collecting risk factor data from each patient been evaluated? Is the added expense justified either by increased effectiveness or by avoiding other expenses?

Diagnostic Tests

Whereas risk assessment aims to predict future disease and disease progression, diagnostic tests evaluate a patient's current status with regard to a specific disease or disorder. They enable the provider to formulate, in cooperation with the patient, a treatment plan. In relation to dental caries and periodontal diseases, the diagnosis ideally should not only detect the presence of disease, but also distinguish between active and arrested disease.

Today, most diagnostic tests for oral conditions are based primarily on anatomic clinical evidence. However, microbiological, pathological, immunological, genetic, and tissue metabolite tests are increasingly available and valuable. Table 8.2 cross-references diseases with categories of diagnostic tests available. The following sections describe elements of a general health assessment and highlight risk assessment, diagnosis, and prevention of selected diseases and conditions.

Oral Health Assessment

An oral health assessment involves an evaluation of an individual's overall health status, including any risk factors and personal needs that can affect health and treatment options. For the majority of craniofa-

cial conditions, this assessment and subsequent care are coordinated with a range of health care providers, with the intent of enhancing the patient's overall health and well-being.

The information gathered for the assessment is derived from patient information, extraoral and intraoral clinical examinations, and imaging and other diagnostic tests as needed. The patient provides demographic and lifestyle behavior information and a medical and dental history, including current complaints, if any. Symptom analysis entails an additional series of questions that explore symptom onset, characteristics, and course. Figure 8.2 provides an example of a medical history form used in dental practice.

The clinician will take into consideration the patient's general appearance and ability to function, as noted by characteristics of facial symmetry or asymmetry and speech. In addition, the patient's vital signs may be assessed, and a thorough examination of the head, neck, temporomandibular joints, and other structures will be conducted. The intraoral portion of the examination involves an extensive assessment of the tissues: the lips and labial mucosa, buccal mucosa and mucobuccal fold, the floor of the mouth, tongue, hard and soft palate, oropharynx, muscles of mastication, salivary glands and saliva, gingiva, periodontium, and teeth.

Depending on the needs of the patient, the initial physical examination is usually augmented by supplementary data from radiographs and sometimes by other diagnostic tests, including tissue biopsies and samples of oral cells and fluids. Such samples can be used to type specific bacteria, viruses, or fungi or to detect elevated levels of tissue metabolites or

TABLE 8.2
Categories of diagnostic methods for selected oral, dental, and craniofacial diseases and disorders

Diagnostic Procedure	Caries	Periodontal Diseases	Oral Infections	Mucosal Diseases	Temporomandibular Disorders	Craniofacial Defects	Oral Cancers
Interview							
Patient history	◆	◆	◆	◆	◆	◆	◆
Physical							
Clinical examination	◆	◆	◆	◆	◆	◆	◆
Probing/caries	◆						
Probing/periodontal		◆					
Imaging	◆	◆			◆	◆	◆
Biologic							
Histology/cytology			◆	◆			◆
Microbiology	◆	◆	◆	◆			◆
Genetics/DNA		◆		◆		◆	◆

²Sensitivity is a measure of how often the test is positive when applied to patients known to have a particular disease or condition; specificity is a measure of how successful the test is in judging the absence of a disease or condition.

FIGURE 8.2
Medical history form for use in dental practice

Medical History Form _____ Date _____

Name _____ Home Phone (____) _____

Address _____ Business Phone (____) _____

City _____ State _____ Zip Code _____

Occupation _____ Social Security No. _____

Date of Birth ____/____/____ Sex M F Height _____ Weight _____ Single _____ Married _____

Name of Spouse _____ Closest Relative _____ Phone (____) _____

if you are completing this form for another person, what is your relationship to that person? _____

Referred by _____

For the following questions, *circle yes or no*, whichever applies. Your answers are for our records only and will be considered confidential. Please note that during your initial visit you will be asked some questions about your responses to this questionnaire and there may be additional questions concerning your health.

- | | | |
|--|-----|----|
| 1. Are you in good health? | Yes | No |
| 2. Has there been any change in your general health within the past year? | Yes | No |
| 3. My last physical examination was on _____ | | |
| 4. Are you now under the care of a physician? | Yes | No |
| If so, what is the condition being treated? _____ | | |
| 5. The name and address of my physician(s) is _____ | | |
| 6. Have you had any serious illness, operation, or been hospitalized in the past 5 years? | Yes | No |
| If so, what was the illness or problem? _____ | | |
| 7. Are you taking any medicine(s) including non-prescription medicine? | Yes | No |
| If so, what medicine(s) are you taking? _____ | | |
| 8. Do you have or have you had any of the following diseases or problems? | | |
| a. Damaged heart valves or artificial heart valves, including heart murmur or rheumatic heart disease | Yes | No |
| b. Cardiovascular disease (heart trouble, heart attack, angina, coronary insufficiency, coronary occlusion, high blood pressure, arteriosclerosis, stroke) | Yes | No |
| 1. Do you have chest pain upon exertion? | Yes | No |
| 2. Are you ever short of breath after mild exercise or when lying down? | Yes | No |
| 3. Do your ankles swell? | Yes | No |
| 4. Do you have inborn heart defects? | Yes | No |
| 5. Do you have a cardiac pacemaker? | Yes | No |
| c. Allergy | Yes | No |
| d. Sinus trouble | Yes | No |
| e. Asthma or hay fever | Yes | No |
| f. Fainting spells or seizures | Yes | No |
| g. Persistent diarrhea or recent weight loss | Yes | No |
| h. Diabetes | Yes | No |
| i. Hepatitis, jaundice, or liver disease | Yes | No |
| j. AIDS or HIV infection | Yes | No |
| k. Thyroid problems | Yes | No |
| l. Respiratory problems, emphysema, bronchitis, etc. | Yes | No |
| m. Arthritis or painful swollen joints | Yes | No |
| n. Stomach ulcer or hyperacidity | Yes | No |
| o. Kidney trouble | Yes | No |
| p. Tuberculosis | Yes | No |
| q. Persistent cough or cough that produces blood | Yes | No |
| r. Persistent swollen glands in neck | Yes | No |
| s. Low blood pressure | Yes | No |
| t. Sexually transmitted disease | Yes | No |
| u. Epilepsy or other neurological disease | Yes | No |
| v. Problems with mental health | Yes | No |
| w. Cancer | Yes | No |
| x. Problems of the immune system | Yes | No |
| 9. Have you had abnormal bleeding? | Yes | No |
| a. Have you ever required a blood transfusion? | Yes | No |

10. Do you have any blood disorder such as anemia?	Yes	No
11. Have you ever had any treatment for a tumor or growth?	Yes	No
12. Are you allergic or have you had a reaction to:		
a. Local anesthetics	Yes	No
b. Penicillin or other antibiotics	Yes	No
c. Sulfa drugs	Yes	No
d. Barbiturates, sedatives, or sleeping pills	Yes	No
e. Aspirin	Yes	No
f. Iodine	Yes	No
g. Codeine or other narcotics	Yes	No
h. Other		
13. Have you had any serious trouble associated with any previous dental treatment?	Yes	No
If so, explain		
14. Do you have any disease, condition, or problem not listed above that you think I should know about?	Yes	No
If so, explain		
15. Are you wearing contact lenses?	Yes	No
16. Are you wearing removable dental appliances?	Yes	No
17. Do you currently use tobacco of any type?	Yes	No
If so, which type?		
18. Are you a former tobacco user?	Yes	No
If so, which type of tobacco?		
19. How many years have/did you use tobacco?		
20. How much tobacco do/did you use a day?		
21. If you have stopped using tobacco products, how long ago did you stop?		
22. Have you ever used alcoholic beverages?	Yes	No
23. How long ago did you stop using alcoholic beverages?		
24. Do you currently use alcoholic beverages?	Yes	No
If so, which type?		
25. How many times a week do you use alcoholic beverages?		
Women		
26. Are you pregnant?	Yes	No
27. Do you have any problems associated with your menstrual period?	Yes	No
28. Are you nursing?	Yes	No
29. Are you taking birth control pills?	Yes	No
Chief Dental Complaint		
<p>I certify that I have read and understand the above. I acknowledge that my questions, if any, about the inquiries set forth above have been answered to my satisfaction. I will not hold my dentist, or any other member of his/her staff, responsible for any errors or omissions that I may have made in the completion of this form.</p>		
Signature of Patient		
For completion by the dentist.		
Comments on patient interview concerning medical history:		
Significant findings from questionnaire or oral interview:		
Dental management considerations:		
Date	Signature of Dentist	
Medical history update:		
Date	Comments	Signature
.....
.....

Source: Adapted from American Dental Association, as reproduced in Rose and Steinberg 2000.

immune system factors associated with disease. The number of such tests is increasing and will be supplemented by genetic tests to indicate an individual's susceptibility to specific diseases.

Currently, the assessment of oral and craniofacial health and disease involves intraoral radiographs as well as radiographic imaging, including arthrography, motion-based tomography, and computed tomography (Jeffcoat 1992). Intraoral radiographs permit detection of lesions between teeth and are the only widely available clinical test that can assess periodontal bone support *in situ* (Jeffcoat et al. 1995). Radiographs are an essential tool for treatment planning of complex prosthetic reconstructions as well as a diagnostic method to assess periodontal progression. However, the mere presence of bone loss on a radiograph does not imply progressive osseous destruction, although it does increase the patient's risk of future bone loss (Armitage 1996, Genco 2000). Radiographs have high specificity for disease progression, and low sensitivity. Because all radiographic examinations expose the patient to some, albeit small, level of ionizing radiation, current guidelines indicate that radiographs should not be taken routinely (FDA 1987), but should be prescribed after an initial assessment by the dentist.

Image processing techniques, such as digital radiography, enhance the clinician's ability to detect small intraoral osseous changes over time and aid in the detection of small changes in skeletal tissues between examinations. Direct digital radiography uses an intraoral detector to capture a radiographic image of the diagnostic area of interest (Ellwood et al. 1997, Matsuda et al. 1997). Several proposed methods for quantitative estimation of lesion mass or volumetry using digital subtraction radiography exist (Armitage 1996). A recent multicenter validation study has indicated that simulated lesions as small as 1 mg in mass may be detected with better than 90 percent sensitivity and specificity (Jeffcoat et al. 1996). These techniques are currently in use in clinical trials.

New diagnostic methods are also becoming available as adjuncts to existing methods for caries diagnosis. Comparing data between bite-wing radiographs of potential occlusal fissure lesions, Lussi et al. (1995) found that electrical resistance measurement may provide a substantial improvement in caries diagnosis.

Other imaging approaches are used to assess craniofacial anatomy, temporomandibular joints, maxillary sinuses, and other associated tissues, and in the assessment of the size and quality of bone to receive dental implants. Magnetic resonance imaging

(MRI) is also receiving increased attention for craniofacial applications, such as for the assessment of the temporomandibular joints. Finally, light-based imaging of teeth and associated structures, using a small intraoral camera, gives both the patient and the provider a wide-screen view of the oral cavity, aiding in patient education.

In the course of conducting a general assessment, the clinician notes disease-specific signs and symptoms. While examining the teeth, the clinician may detect signs of relatively rare hereditary diseases such as ectodermal dysplasias, or more common destructive habits such as bruxism, where the enamel and at times the dentin may be abraded. Examinations of the face and oral cavity may reveal the effects of intentional and unintentional injuries. With the results of the general assessment in hand, the clinician will classify the patient's general and oral health status and make treatment and/or referral recommendations.

A classification system adopted in 1962 by the American Society of Anesthesiologists, used to categorize a patient's risk on the basis of physical status, also has been applied, along with the patient's general and oral health risk assessment, to determine the need for coordinated multidisciplinary referral and whether care in a hospital is indicated rather than in the dental office (Bricker et al. 1994) (Table 8.3).

Changing Approaches to Selected Diseases and Conditions

The science and technology base is providing new approaches to risk assessment, diagnosis, prevention, and treatment. Highlights of selected diseases and conditions follow.

Dental Caries

Dental caries is caused by a transmissible microbial infection that affects tooth mineral. A number of factors play a role in the initiation and progression of the disease, including bacterial biofilm, specifically the presence of mutans streptococci and species of lactobacilli; the frequency of simple sugars in the diet; the flow and composition of saliva; the availability of fluoride; the structure of tooth mineral in a given individual; and oral hygiene behaviors. Sound caries management takes all these factors into account (Figure 8.3) (Burt and Ismail 1986). Today there is the prospect that clinicians will be able to balance protective and pathologic factors and work with the patient to control disease (Anderson et al. 1993, Edelstein 1994, Featherstone 1996).

Risk Assessment. Reviews of caries risk prediction models conclude that clinical variables, especially past caries experience, are the best predictors of new caries experience (Newbrun and Leverett 1990, Powell 1998). Table 8.4 shows a timeline summarizing the strongest predictors of caries incidence based

on a review of the modeling literature. At the time of initial tooth eruption, the presence of mutans streptococci appears to be the primary predictor of future caries. With continued tooth eruption, this variable disappears as a primary predictor and is replaced by the status of the most recently exposed or erupted tooth surface. For example, the presence of carious lesions in the primary incisors has been found to be the best predictor of caries in the later-erupting primary molars (Powell 1998).

Despite recent declines, dental caries is a prevalent disease, with some age and population groups particularly vulnerable (see Chapter 4). A guide for the identification of vulnerable patients and the treatment of caries as an infectious disease has been developed (ADA 1995). Figure 8.4, from that guide, proposes questions to be considered at an initial examination. These questions, together with information gathered at recall examinations, allow classification of child and adult patients into high-, moderate-, and low-risk disease categories (Table 8.5). This approach has been incorporated in a variety of caries risk assessment forms adopted by some dental

schools and managed care programs (C.W. Douglass, personal communication, 1999). Studies are needed to determine the validity and reliability of such approaches for different patient populations and practice settings.

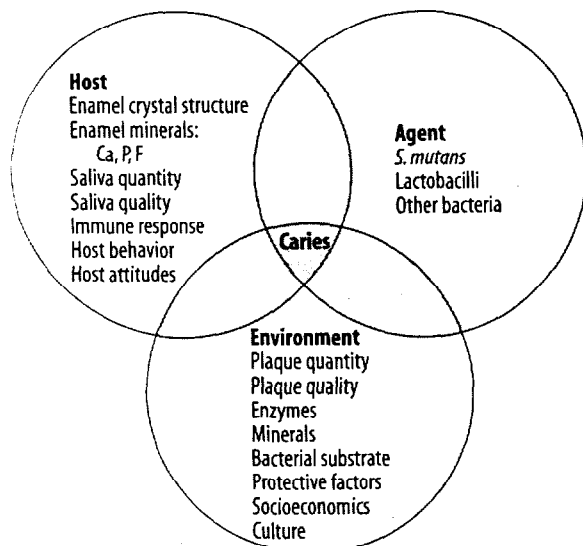
The use of tests to assess caries risk to determine the activity status of preclinical disease is becoming more widespread. A range of diagnostic aids for caries activity testing are available. Microbial tests can detect the presence and quantify the levels of lactobacilli and mutans streptococci. The development and use of these tests are based on studies that have associated these microbes individually and together with different types of carious lesion development. Measurements of plaque and salivary pH have been used to evaluate the oral environment overall and to note the changes in pH that occur after eating various foods. Salivary flow and composition analyses add another dimension. Decreased flow has been related to caries susceptibility, as have increases in viscosity. These factors warrant further study to determine their sensitivity and specificity.

TABLE 8.3
American Society of Anesthesiologists—medical risk categories and associated dental considerations

ASA Classification	Dental Consideration
Physical status 1 A patient without systemic disease; a normal, healthy patient	Routine dental therapy without modification
Physical status 2 A patient with mild systemic disease	Routine dental therapy with possible treatment limitations or special considerations (e.g., duration of therapy, stress of therapy, prophylactic consideration, possible sedation, and medical consultation)
Physical status 3 A patient with severe systemic disease that limits activity but is not incapacitating	Dental therapy with possible strict limitations or special considerations
Physical status 4 A patient with incapacitating systemic disease that is a constant threat to life	Emergency dental therapy only, with severe limitations or special considerations

Source: Genco 2000. Risk factors for periodontal disease. In Rose LF, Genco RJ, Cohen DW, Mealey BL. Periodontal medicine. Hamilton: B.C. Decker Inc. 2000:35-43. Copyright 2000 by B.C. Decker Inc. Reprinted by permission of B.C. Decker Inc. (2000).

FIGURE 8.3
Multifactorial model of dental caries



Source: Burt and Ismail 1986. Copyright 1986 by Journal of Dental Research. Reprinted by permission of Journal of Dental Research (2000).

Diagnosis. Clinical signs, patient-derived history, and radiographic images remain the primary means of dental caries diagnosis. Tooth surface pitting and cavitation, white and/or brown spots, areas soft to tactile probing, and radiolucencies are used to detect the effects of this disease. The most common diagnostic approaches include visual inspection, the use of an explorer (a probelike instrument) to determine the integrity of the tooth surface, the use of a light source to detect difference in reflectance across tooth structure (transillumination), and radiographs. Table 8.6 compares the reported sensitivity and specificity of selected methods. No single method stands out as superior with regard to both sensitivity and specificity for all tooth surfaces.

The most basic diagnostic methods—visual alone and visual examination with an explorer—have limited sensitivity, but excellent specificity. The visual examination may be combined with a radiographic series for the initial assessment. Bite-wing radiographs are frequently used to diagnose interproximal caries (between teeth) and for these surfaces provide excellent sensitivity and specificity. Radiographic examination allows examination of otherwise inaccessible areas. Specifically, the depth of a lesion and its relationship to the pulp chamber can be evaluated for interproximal lesions. However, radiographs are of little value in detecting caries on the occlusal surfaces of the teeth. For these surfaces, a negative radiographic diagnosis does not imply lack of a carious lesion in enamel.

Precavitated carious lesions and caries in restored teeth pose an additional diagnostic challenge. A review of the literature on the clinical diagnosis of precavitated carious lesions concluded that

visual detection of these lesions has low sensitivity and moderate specificity (Ismail 1997). It is difficult with these lesions to determine whether there is no caries or whether only the enamel or outer layer of dentin is involved. Carious lesions forming around restorations are seen more frequently at the approximal and cervical margins of these restorations (Mjör 1985). Distinctive color changes around a restoration alone are not diagnostic of active caries (Kidd 1990).

Currently, the progression of carious lesions is the most definitive diagnostic parameter for disease activity. Progression can be determined over specific time intervals only by professional assessment.

Prevention. The primary prevention of dental caries starts with adequate prenatal and perinatal nutrition to ensure normal development of the teeth and supporting structures. It continues with interventions aimed at preventing transmission of cariogenic microbes from caregivers to infants, and proceeds with specific strategies employed across the life span. These approaches include the provision of sufficient fluoride, the use of dental sealants, the adoption of healthy behaviors, including avoiding unhealthy dietary practices and practicing appropriate oral hygiene, and the timely use of care services. Although many factors are brought to bear on the primary prevention of dental caries, the combination of fluoride in its multiple forms and dental sealants is the foundation (as described in Chapter 7).

Fluoride is available in a variety of products that can be used by health professionals, individuals, and public programs. Topical solutions and gels, mouthrinses, and dentifrices are available for daily, weekly, or as-prescribed frequency. In addition,

TABLE 8.4
Timeline of strongest clinical predictors of caries incidence

Age (years)	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14-21	22-45	>45															
Dentition	Primary					Mixed					Early permanent		Mature permanent																			
Event	Eruption primary molars					Eruption first permanent molar					Eruption second permanent molar		Progression of gingival recession																			
Predictor	Mutans streptococci		dmfs, especially primary incisors			Mutans streptococci and lactobacilli			dmfs, especially primary molars		First molar occlusal morphology		DMFS		DMFS, especially first permanent molars		First molar occlusal morphology		Incipient smooth surface lesions		Incipient smooth surface lesions		DMFS		Not studied		Coronal and root DMFS		Number of teeth		Periodontal disease	

Note: dmfs = decayed, missing, or filled primary tooth surfaces; DMFS = decayed, missing, or filled permanent tooth surfaces.

Source: Powell 1998. Caries prediction: a review of the literature. *Community Dentistry and Oral Epidemiology* 1998;26:361-71. Copyright 1998 by Munksgaard International Publishers Ltd., Copenhagen, Denmark. Reprinted by permission of Munksgaard International Publishers Ltd., Copenhagen, Denmark (2000).

FIGURE 8.4**Caries risk questions for initial examination****INITIAL VISIT—QUESTIONS TO CONSIDER**

Is there current caries activity?

Are there indications that yield potential for development of caries within the next year?

- Prior DMFS (decayed, missing, or filled surfaces)
- Tooth morphology
- Medications that decrease saliva flow and/or affect viscosity of saliva
- Medical condition or treatment(s)

What is the individual's caries risk?

- Low
- Moderate
- High

What are the modifiable risk factors that may be responsible for or may contribute to this caries activity?

- Insufficient systemic and topical fluoride
- Medications
- Poor oral hygiene habits or skills
- Deep pits and fissures without sealants
- Poor dietary habits

What can be done to prevent new caries or caries progression within the next year?

- Sealants
- Increase fluoride use
- Oral hygiene instruction/education
- Dietary counseling
- Monitor bacterial count
- Antimicrobial agents
- Conservative restorative techniques—to minimize removal of tooth structure

What is the prognosis for successful intervention?

- Patient compliance
- Clinician skill (diagnosis, intervention counseling)
- Prevention modalities are accepted/applied
- Severity at onset

Are there other considerations that may affect the decision process that cannot be changed? (effect modifiers, confounders)

- Age
- Socioeconomic considerations
- Medically and/or physically compromising conditions

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fluoride-containing prophylactic pastes are available for professional application (see Chapter 7). Clinical judgment of risk factors determines the type and frequency of interventions needed.

Although there is general agreement on the overall value of topical fluorides in reducing dental caries (ADA 1986, 1994, Moss 1976, Stookey et al. 1993),

comparative clinical trials are needed to determine which of the existing fluoride formulations (acidulated phosphate fluoride, stannous fluoride, amino-fluoride, or sodium fluoride) and which delivery system (gel, varnish, dentifrice, or solution) are most efficacious.

A second line of defense is through control of the etiologic agent. Chemotherapeutic agents (including the antimicrobial mouthrinse agent chlorhexidine and fluoride) can be used to reduce plaque. Dietary measures aimed at reducing the frequency and quantity of sugars and the substitution of sugars by sugar-free sweeteners may effectively starve the bacteria.

The process of tooth demineralization and remineralization has received significant attention over the past four decades (Geiger et al. 1992, Koulourides et al. 1961, Larsen and Fejerskov 1987, Linton 1996, Silverstone et al. 1981, White 1988), although the concept was documented in the early 1900s (Head 1912) (see Chapter 3). Investigators are studying the effectiveness of therapeutic agents for arresting carious lesions and remineralizing enamel in populations at high risk for dental caries. For example, a combined chlorhexidine-fluoride solution can enhance remineralization of incipient lesions and arrest caries in patients who suffer from radiation-induced caries (Katz 1982). The use of a twice-daily rinse with 0.05 percent sodium fluoride to prevent demineralization and induce remineralization in subjects with radiation-induced hyposalivation has also been found to be effective (Meyerowitz et al. 1991). This study also addressed the effects of chlorhexidine use alone, which has been associated with tooth staining, alterations in taste, and potential hypersensitivity reactions (Ohtoshi et al. 1986, Okano et al. 1989). Schaecken et al. (1991) showed that the application of 40 percent by weight chlorhexidine varnish every 3 months enhanced remineralization of root caries more than fluoride varnish, although both treatments were associated with fewer filled root surfaces than the control group after 1 year. A chlorhexidine varnish has not yet been approved in the United States, and large-scale, double-blind, placebo-controlled clinical trials are not yet available to test the effects of specific regimens in relation to caries risk.

Studies also are evaluating interventions to prevent mutans streptococci transmission. Findings from cross-sectional studies indicate that infants are initially infected by their parents, specifically mothers, around the time the teeth erupt (Berkowitz et al. 1975, Caufield et al. 1993, Kohler and Bratthall 1978). A longitudinal study using DNA fingerprinting demonstrated that mothers were the source of the

TABLE 8.5
Caries risk classification guidelines

Risk Category	Age Category for Recall Patients ^a	
	Child/Adolescent	Adult
Low	No carious lesions in last year Coalesced or sealed pits and fissures Good oral hygiene Appropriate fluoride use Regular dental visits	No carious lesions in last 3 years Adequately restored surfaces Good oral hygiene Regular dental visits
Moderate	One carious lesion in last year Deep pits and fissures Fair oral hygiene Inadequate fluoride White spots and/or interproximal radiolucencies Irregular dental visits Orthodontic treatment	One carious lesion in last 3 years Exposed roots Fair oral hygiene White spots and/or interproximal radiolucencies Irregular dental visits Orthodontic treatment
High	≥2 carious lesions in last year Past smooth surface caries Elevated mutans streptococci count Deep pits and fissures No/little systemic and topical fluoride exposure Poor oral hygiene Frequent sugar intake Irregular dental visits Inadequate saliva flow Inappropriate bottle feeding or nursing (infants)	≥2 carious lesions in last 3 years Past root caries; or large number of exposed roots Elevated mutans streptococci count Deep pits and fissures Poor oral hygiene Frequent sugar intake Inadequate use of topical fluoride Irregular dental visits Inadequate saliva flow

^aAt initial visit for new patient, if time of last caries experience cannot be determined, a person with no decayed, missing, or filled surfaces (DMFS = 0) would be classified as low risk. A person with past caries experience (DMFS > 0) and/or one active lesion would be classified as moderate risk. A person with past caries experience and/or two active caries or one smooth surface lesion would be classified as high risk.

Parents of young children and expectant parents need additional counseling on inappropriate nursing or bottle feeding practices that can lead to the development of early childhood caries. Parents and caregivers should be advised to introduce children to a cup in an effort to discontinue use of the bottle by the age of 1 year. Also, parents and caregivers should be advised never to place anything other than plain water in a naptime or nighttime bottle. Children should not be allowed to bottle feed at will and should be weaned from the bottle by the age of 1 year.

Many medically compromised individuals are likely to be assessed in the higher risk categories because of their use of certain medications and possible xerostomia.

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TABLE 8.6
Sensitivity and specificity of selected dental caries diagnostic procedures

	Sensitivity (percentage)	Specificity (percentage)	References
Visual examination of noncavitated fissures	12-31	70-99	Lussi 1993, Ketley and Holt 1993
Examination using explorer	14-24	70-99	Penning et al. 1992, Lussi 1993
Radiographs of approximal lesions	50-90	85+	Gröndahl 1989, Benn and Watson 1989

bacteria in their infants and the degree of matching to maternal strains was higher for female infants than for males (Li and Caufield 1995). Based on a study of child-mother pairs (with the child initially at 1 year of age), the application of a 1.0 percent chlorhexidine rinse alternated with a 0.2 percent sodium fluoride gel to the mother's teeth (3 times per day on 2 consecutive days, twice per year for 3 years) delayed, and in some cases prevented, the colonization of their children's teeth by mutans streptococci (Tenovou et al. 1992). Timing of colonization has been shown to be correlated with caries prevalence. In a longitudinal study that followed children in 4-month intervals from 15 months to 4 years of age, children who were infected earlier had a higher caries prevalence than those in whom the infection was detected at later ages. Studies also have been aimed at reducing the levels of cariogenic bacteria in the infants themselves.

Work continues on the development of a caries vaccine. One approach focuses on the production and release of antibodies against cariogenic bacteria antigens (Russell et al. 1995). Specific antigens have been purified and synthesized. Another approach involves biological replacement therapy, where nonpathogenic bacteria, instilled in the mouth, prevent pathogenic bacteria from colonizing (Hillman et al. 2000). Yet another approach employs passive immunization in which antibodies, produced outside the body (in cultures, animals, eggs, or plants), are applied to the teeth and oral tissues to protect against disease. A recent study indicated that "plantibodies" painted on the teeth could prevent mutans streptococci colonization for 120 days, the period of the experiment (Ma et al. 1998).

Treatment. Prompt treatment of early carious lesions permits the preservation of tooth structure through conservative approaches. A 10-year study reported that caries did not progress under a dental sealant placed over cavitated lesions where the lesions were no more than halfway through the dentin (Mertz-Fairhurst et al. 1998).

Materials that can bond to enamel and to dentin continue to be refined and improved. Glass ionomer cements have contributed to materials that can bond to enamel and dentin, release fluoride, and increase remineralization in adjacent teeth (Mount and Hume 1998, Qvist et al. 1997). These cements, together with polymeric resin composites and hybrids of these two materials, are now available for tooth restoration with other materials. Based on the available materials and emerging techniques, such as air abrasion and laser ablation (Featherstone et al. 1998, Kantorowitz et al. 1998), restoration procedures are more conservative than ever before (Mount and Hume 1997).

A proposed categorization of carious lesions for the purpose of conservative management places lesions into three categories: lesions where no treatment is advised, lesions where preventive care is advised, and lesions where restorative treatment is advised (Pitts and Longbottom 1995). This approach, using caries as an infectious disease paradigm, resulted in a marked reduction of operative procedures in Danish schoolchildren (Thylstrup et al. 1995) and has been proposed as a means to preserve tooth structure and maximize appropriate care in the United States (Ismail 1997).

New imaging and laser technologies are emerging as tools for early diagnosis and prompt treatment of dental caries. For example, quantitative light-induced fluorescence is showing promise (de Josselin de Jong et al. 1996) for dental caries diagnosis. Two different methods, the quantitative infrared laser fluorescence method and electrical conductance measurements, are currently commercially available. At present, these methods are being used to augment conventional diagnostic tools but are not yet part of routine practice. However, they could potentially be used for close monitoring of the lesions and for patient motivation (Angmar-Månsson et al. 1996). Laser treatments for soft tissue surgery have been used in dentistry in recent years. Currently, *in vitro* studies are under way for the application of lasers for hard tissues, specifically to prevent dental caries by altering tooth mineral and inhibiting progression of artificial caries-like lesions (Featherstone et al. 1998, Kantorowitz et al. 1998).

Despite the best efforts of the individual and health care provider, caries may progress. Advances

in materials science over the last two decades have fortunately led to major improvements in dental restorative materials, resulting in a wide range of aesthetically pleasing, longer-lasting restorations that can be placed with less trauma. Traditional materials such as amalgam fillings and gold crowns are now augmented by aesthetic materials, including bonded composite resins, porcelain fused to metal crowns, and facings.

When teeth have been lost, the options for rehabilitation include a range of prosthetic devices. Removable full and partial dentures and fixed bridges provide aesthetic and serviceable restorations for many patients. Still another option is the use of dental implants. These are used not only in patients who have lost teeth due to caries and periodontal diseases, but also to restore form and function in patients treated for trauma, craniofacial cancers, hereditary tooth defects, and other abnormalities.

The evidence base for the survival of the endosseous dental implants, an implant that is placed directly into a tooth socket, is extensive and has been recently reviewed (Cochran 1996, Fritz 1996). The predictability of endosseous dental implants in fully and partially edentulous patients has been clearly demonstrated in longitudinal studies (Albrektsson 1988, Albrektsson et al. 1988, Buser et al. 1991, Spiekermann et al. 1995). Many implant designs and surfaces have shown high success rates (often exceeding 95 percent in good-quality bone and 85 percent in poorer-quality bone, such as the posterior maxilla) (Buser et al. 1988, Cochran 1996, Fritz 1996).

Rehabilitation of lost tooth structure or even the whole tooth itself may be revolutionized in the next century, based on discoveries of the natural repair and regeneration mechanisms the body uses. The new sciences of biomimetics and tissue engineering combine engineering principles and materials science with rapidly growing knowledge of the progenitor cells and molecules that give rise to specific tissues such as skin, bone, teeth, and cartilage. Already it is possible to generate new cartilage and bone of a prescribed shape to replace tissue lost from injury or disease (Reddi 1995). Eventually, it may be possible to use a patient's own oral cells and cell products to generate new tooth enamel, dentin, and cementum for the natural repair of carious lesions.

Periodontal Diseases

Periodontal diseases are caused by microbial infections, and are plaque-related complex diseases like dental caries, presenting as several clinical variants

(see Chapter 3). The mildest form is gingivitis, characterized by inflammation of the gingiva with a marked loss of gingival collagenous material (Page and Schroeder 1976, Schroeder et al. 1973). In a more advanced disease, periodontitis, there is involvement of the soft tissue and bone that support the teeth. If untreated, periodontitis may progress and result in abscesses, mobile teeth, and tooth loss. Periodontitis also may be associated with certain systemic diseases and conditions (see Chapter 5).

Gram-negative anaerobic bacteria in plaque are implicated as causative agents in periodontitis. However, host immune system factors, specifically, a chronic inflammatory response, are now considered to be the primary determinants of disease progression and outcome (Page 1998). The disease process is very similar across the different types of periodontal disease and involves interactions between infectious agents and their virulence factors and host defense mechanisms, operating within a context of environmental, acquired, and genetic risk factors specific to a given individual. Figure 8.5 illustrates the pathogenesis of these diseases (Page and Beck 1997).

Risk Assessment. Sufficient knowledge of demographic and systemic risk factors and indicators has been acquired to guide clinical decisions in the management of periodontal diseases (Genco 1996, 2000, Page and Beck 1997, Papapanou 1998). Table 8.7 provides an overview of the strength of the associations of local and systemic factors with destructive periodontal diseases (Genco 1996, 2000). Table 8.8 presents the odds ratios derived from studies that investigated the likelihood of developing periodontal disease given a specific risk factor, indicator, or marker/predictor (Jeffcoat et al. 1997, Page and Beck 1997). The presence of pathogenic bacteria, poor oral hygiene, tobacco smoking, diabetes mellitus, and preexisting periodontal disease are some of the factors that contribute to the likelihood of disease presence, progression, and treatment outcomes.

A systematic identification of risk factors, indicators, and predictors has been proposed as the first step in diagnosing and managing periodontal diseases (Genco 1996, Page and Beck 1997, Papapanou 1998). Clinicians can weigh the known risks for individual patients and devise treatment plans appropri-

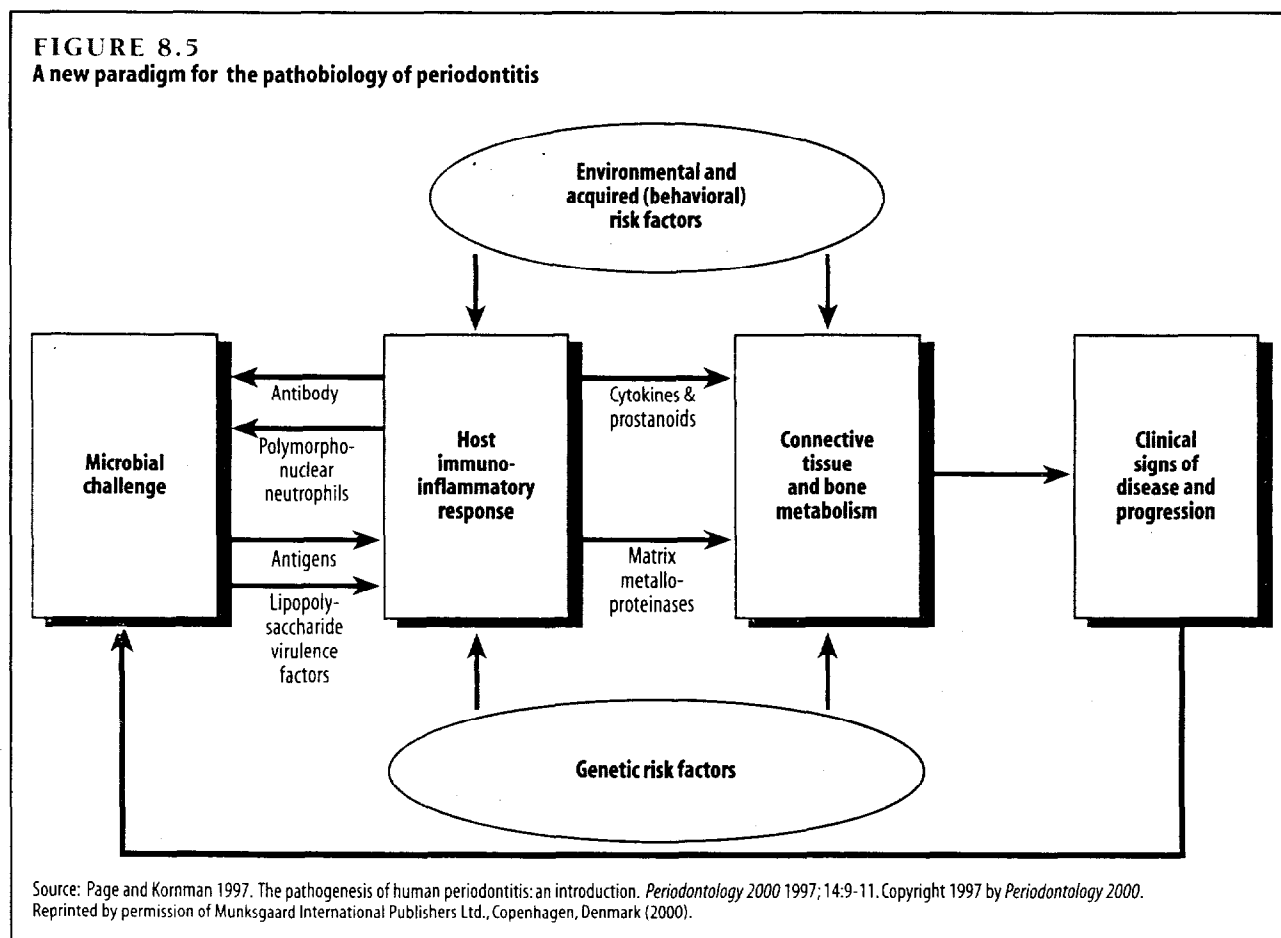


TABLE 8.7
The strength of association of local and systemic factors with destructive periodontal disease

Factor	Case Report Studies	Case-Control Studies	Cross-sectional Studies	Longitudinal Studies	Intervention Studies
Specific bacteria					
<i>P. gingivalis</i>	Yes	Yes	Yes	Yes	Yes
<i>B. forsythus</i>	Yes	Yes	Yes	Yes	Yes
<i>P. intermedia</i>	Yes	Yes	Yes	Yes	Yes
Sex					
Male	Yes	NR	Yes	NR	NR
Age	Yes	Yes	Yes	No (to 7th decade)	NR
Diabetes mellitus					
Type 2	Yes	Yes	Yes	Yes	Yes (treatment reduces glycosylated hemoglobin)
Type 1	Yes	Yes	Yes	NR	NR
Smoking	NR	Yes	Yes	Yes	Yes (smokers heal poorly)
Osteoporosis	Yes	Yes	Yes	NR	NR
Stress, distress, coping	Yes	Yes	Yes	NR	NR
Polymorphonuclear disorders	Yes	Yes	NR	Yes (case series)	NR
Genetic factors (IL-1 polymorphisms)	NR	Yes	NR	NR	NR
Dietary calcium	NR	Yes	Yes	NR	NR
Preexisting periodontal disease	Yes	Yes	Yes	Yes	Yes

Note: NR = not reported, or not relevant.

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TABLE 8.8
Risk of periodontal disease

	Strength of Association With	Odds Ratio
Demographic characteristics		
Age, 35-44 years	alveolar bone loss	2.60
Age, 65-74 years	alveolar bone loss	24.08
Risk factors		
Smoking, light	periodontal disease	2.05
	alveolar bone loss	1.48
Smoking, heavy	periodontal disease	4.75
	alveolar bone loss	7.28
Bacterial risk factors		
Poor oral hygiene	periodontal disease	20.52
<i>P. gingivalis</i>	periodontal disease	3.60
<i>A. actinomycetemcomitans</i>	periodontal disease	2.50
Clinical measurement		
Bleeding on probing ^a	progression of periodontitis	2.7

^aMeta-analysis (Armitage 1996).

Sources: Jeffcoat et al. 1997, Page and Beck 1997.

ate to their risk category. These same factors and the outcomes of treatment can also be used to assess prognosis upon completion of therapy. Studies are under way to determine the feasibility and validity of assessing a complex of risk factors to predict states of

periodontal health and disease (Genco 1996, 2000, Genco et al. 1999, Papapanou 1998).

Most recently, putative genetic markers for susceptibility for oral disease have been studied. In particular, a specific genotype of the polymorphic IL-1 gene cluster has been shown to be associated with severe periodontitis in nonsmokers (Kornman et al. 1997). IL-1 β is of interest because the proinflammatory cytokines are key regulators of the host immune response to microbial infection and extracellular matrix catabolism and bone resorption. Functionally, this polymorphism is associated with high levels of IL-1 production, and high levels of IL-1 have been associated with progressive periodontal breakdown (Cavanaugh et al. 1998).

A consensus has been reached by a specialty organization that all patients in general and specialty care should be screened for periodontal disease (AAP 1996). The recommended approach is to apply the Periodontal Screening and Recording examination (PSR). Related screening tests include the Community Periodontal Index of Treatment Needs (CPITN) (Ainamo et al. 1982) and the Basic Periodontal Examination.

Diagnosis. The strengths and weaknesses of the range of tests and methods used to diagnose periodontal

diseases are presented in Table 8.9. Most diagnostic tests for periodontal diseases rely on a physical examination to note any swelling, redness, gingival bleeding, or tooth mobility. Periodontal probing, radiographs, and microbiologic and histological examinations of biopsied tissue provide important additional information. These tests indicate the presence, extent, and severity of gingival and periodontal tissue destruction; they do not indicate the cause of disease or whether it is quiescent or actively progressing.

Gingival inflammation may be assessed using a variety of methods, including bleeding on probing and the use of indices such as the gingival index (Løe and Silness 1963) to grade redness and bleeding. In adult periodontitis, the absence of inflammation is associated with a lack of disease progression, but the presence of inflammation does not indicate inevitable progression to destruction (Armitage 1996, Halazonetis et al. 1989, Okamoto et al. 1988). Longitudinal studies have also been conducted in patients who participate in maintenance programs. The

absence of gingival bleeding, especially at recall visits, has been shown to be a valid indicator of gingival health in these patients (Lang et al. 1986).

Measurement of probing depths (also termed pocket depths) is an integral part of the periodontal examination. Longitudinal studies have shown that shallow probing depths and minimal loss of attachment are associated with lack of disease progression. The mere presence of a pocket does not herald progressive periodontitis at that site. Although teeth with moderate to deep probing depths are at higher risk for additional destruction, a single examination cannot determine the fate of the tooth with certainty (Armitage 1996, Haffajee et al. 1983, Halazonetis et al. 1989, Okamoto et al. 1988).

Radiographs are used to obtain a visual image of the bony support around a tooth or dental implant. They are an essential tool in planning complex prosthetic reconstructions, as well as a necessary diagnostic aid in assessing periodontal progression.

At least 15 different organisms have been associated with adult periodontitis. The 3 species most

TABLE 8.9
Strengths and weaknesses of tests and methods used to diagnose periodontal diseases

	Application	Strengths	Weaknesses	Type of Evidence
Periodontal screening and recording (PSR)	All patients in every practice	Cost-effective, quick, easy; detects patients with periodontal disease.	Does not provide a tooth-by-tooth assessment for later comparison during maintenance. A full periodontal examination is needed for this purpose.	Epidemiologic studies
Probing pocket depths	All patients	Shallow probing depths are associated with lack of future disease progression.	Moderate to deep pockets in single probing depth examination will not distinguish with certainty which teeth will undergo progressive periodontal destruction.	Longitudinal studies
Gingival inflammation	Assessed in all patients	Absence of inflammation is associated with a lack of future progression. In treated patients, bleeding on probing is associated with an increased risk for progressive loss of attachment.	Presence of inflammation will not distinguish with certainty which teeth will undergo progressive periodontal destruction.	Longitudinal studies
Radiographic evidence of bone loss	At-risk patients as determined by PSR screening or periodontal examination	Absence of bone loss is associated with a lower risk of future progression.	Presence of bone loss on a single radiograph will not distinguish with certainty which teeth will undergo progressive periodontal destruction.	Longitudinal studies
Microbial/plaque tests	High-risk or refractory patients	Absence of supragingival plaque is associated with lack of disease progression. In compromised or refractory patients, may be useful in determining the presence of pathogens.	At this time, routine testing offers limited benefit in adult periodontitis.	Cross-sectional and Longitudinal studies Case reports
Biochemical profiles in gingival crevicular fluid	Not yet determined	A number of biochemical markers may identify individuals at risk.	At present, there are no specific biochemical profiles that characterize specific periodontal diseases.	Cross-sectional and Longitudinal studies

strongly linked are *Porphyromonas gingivalis*, *Bacteroides forsythus*, and *Treponema denticola*. *Actinobacillus actinomycetemcomitans* is most strongly linked to early-onset periodontitis (Haffajee and Socransky 1994). No single bacterial species has been shown to satisfy Koch's postulates (Moore 1987, Socransky and Haffajee 1992), leading some investigators to suspect that periodontitis is a mixed infection (Ranney 1993). As a result, diagnostic tests for periodontal diseases have included assessments of the presence and amount of several putative microbes in the subgingival plaque.

Routine bacterial testing of patients with adult periodontitis is not usually necessary and indeed is not supported by the preponderance of the evidence (Armitage 1996, AAP 1996). In formulating treatment programs for special patient populations and as a research tool, however, the tests can be very helpful. Such patients include those refractory to previous therapy, patients with rapidly progressive or early-onset periodontitis, and certain medically compromised patients.

The traditional method for assessing the subgingival flora is by culturing samples extracted from the site of infection. Culturing allows the clinician to determine the antibiotic sensitivity of the organisms, but it is technique-sensitive: scrupulous care is required when sampling the periodontal pocket. This is especially true for microbes that are strict anaerobes, because they are killed by even brief exposure to air. The requirement that bacteria have time to grow also precludes chairside testing.

With the advent of molecular biology, bacterial species can be identified by their DNA (Moncla et al. 1988, Savitt et al. 1988, 1990) or by unique antigenic components (Zambon et al. 1986). Either method will detect putative periodontopathic bacteria quickly and with a high degree of sensitivity and specificity, usually above 90 percent. The tests do not indicate whether there is actual disease, however. Nor do the tests reveal anything about the antibiotic sensitivity of the detected bacteria. Because DNA is very stable, the tests can be applied to nonliving plaque samples, simplifying the collection process. Kits are available that allow DNA testing to be performed in the dental office; otherwise the samples are sent to a reference laboratory.

Other tests are available for the detection of groups of putative periodontopathic bacteria (Loesche 1986). The BANA test detects a trypsin-like enzyme that is present in *P. gingivalis*, *T. denticola*, and *B. forsythus* (Loesche et al. 1990). Somewhat less accurate than the tests described above, the BANA

test is 92 percent sensitive and 70 percent specific in detecting these groups of bacteria.

Once a periodontal infection is established, tell-tale metabolic changes occur in the body as a result of inflammation, injury, or death of tissue. A sample of fluid exudate from the gingiva (gingival crevicular fluid) in an affected pocket can be analyzed for these changes. They include elevated levels of prostaglandin E₂ (Cavanaugh et al. 1998, Offenbacher et al. 1986), interleukin 1 and interleukin 6 (Cavanaugh et al. 1998, Geivelis et al. 1993, Masada et al. 1990, Tsai et al. 1995), tumor necrosis factor (Rossomando et al. 1990), β -glucuronidase (Lamster et al. 1994, 1995), aspartate aminotransferase (Chambers et al. 1991, Persson and Page 1992), elastase (Armitage et al. 1994, Palcanis et al. 1992), and collagenase (Lee et al. 1995). Most of these analyses are based on inserting a filter paper strip into the isolated pocket to collect the fluid and testing for the metabolite of interest. A positive result usually indicates that inflammatory or destructive pathways have been triggered, but provides no clues concerning the etiologic factor or factors. Because of differences in experimental designs in the clinical studies, it is difficult to compare the sensitivity and specificity of each metabolite in detecting disease.

Prevention. Because periodontal diseases are plaque-associated infections, prevention and management of the early signs of these diseases depend on effective plaque control. This can be accomplished using both mechanical and chemotherapeutic approaches (Table 8.10). The prophylaxis performed in the dental office on periodontally healthy patients reduces plaque and removes stains and calculus. How often patients should be recalled for such preventive procedures is based on an assessment of risk factors such as the patient's age, oral hygiene, personal habits (e.g., smoking and diet), and a medical history indicating a heightened risk of infection (such as noted with diabetes or HIV infection) (Hancock 1996, Mealey 1996).

Chemical plaque control has become an important part of the clinician's armamentarium and may be prescribed for patient care at home (Table 8.10). Reviews of the literature by Hancock (1996) and Drisko (1996) provide detailed supporting evidence. Significant reductions in gingival inflammation have been demonstrated for chlorhexidine, triclosan copolymer when used in conjunction with a fixed combination of essential oils, and stannous fluoride. The magnitude of gingival inflammation reduction was greatest for chlorhexidine. The evidence supporting

these effects includes multiple randomized, double-blind, controlled clinical trials.

Treatment. Once periodontal disease is established, the resultant bone and connective tissue loss may be quiescent or actively progressing. The goal of treatment is to determine whether the disease is active in order to prevent further tissue loss. This entails professional plaque removal and careful instruction of the patient on scrupulous self-care.

The concept of management of a patient's risk factors as part of treatment is reasonably well documented for individuals who smoke and those who are diabetic and may be important for other risk factors such as stress (Genco et al. 1999) and low dietary calcium (Nishida et al. in press). Several studies have shown that treatment of periodontal disease in smokers is not as successful as in nonsmokers (Grossi et al. 1996). Thus, the management of smoking as a risk factor will contribute to the success of periodontal therapy. Furthermore, it appears that treatment of diabetic patients with periodontal dis-

ease may require more intense therapy since several studies have shown that antibiotic therapy is successful not only in reducing periodontal disease, but also in reducing glycated hemoglobin (Grossi and Genco 1998).

Professional plaque removal typically employs scaling and root planing, in which hardened deposits of plaque and other debris are removed from the periodontal pocket and the tooth root surface is smoothed over. The effectiveness of scaling and root planing has been demonstrated repeatedly in longitudinal, cohort, and randomized clinical trials and was reviewed by Cobb (1996). Demonstrated benefits include decreased gingival inflammation, decreased probing depth, and facilitation of maintenance of clinical attachment level. The evidence indicates that similar results may be obtained with ultrasonic and sonic instruments as with manual instruments. Regardless of the methods used, meticulous attention to detail is required to achieve optimal results (Cobb 1996).

TABLE 8.10
Periodontal diseases: mechanical therapy and chemotherapeutics

Category of Treatment	Treatment	Strengths	Weaknesses	Type of Evidence
Professional mechanical therapy—used in the treatment of gingivitis and periodontitis	Scaling and root planing with manual instrument	Decreases gingival inflammation by 40 to 60 percent Decreases probing depth Facilitates gain in clinical attachment level	Requires attention to detail	Numerous longitudinal, cohort, and randomized clinical trials
	Ultrasonic and sonic scaling and root planing	Results are similar to manual scaling and root planing		Longitudinal, cohort, and randomized clinical trials
Chemical plaque control with mouthrinses and dentifrices	Chlorhexidine Triclosan co-polymer or triclosan zinc-citrate Essential oils Stannous fluoride	Significant reductions in gingival inflammation	No clear evidence that there is a substantial long-term benefit for periodontitis except to control co-existing inflammation	Randomized double-blind clinical trials
Irrigation	Supra- and subgingival irrigation used as an adjunct to brushing	Aids in the reduction of gingivitis	No clear evidence that there is a substantial long-term benefit for periodontitis	Randomized double-blind clinical trials
Sustained release antimicrobials	Intrapocket resorbable or non-resorbable delivery systems containing a tetracycline antibiotic	When used as an adjunct to scaling and root planing, gains in clinical attachment level and decreases in probing depth and bleeding	Few reported side effects include transient discomfort, erythema, recession, allergy, and rarely candidiasis	Randomized double-blind clinical trials
Systemic antibiotics	Tetracyclines, metronidazole, spiramycin, clindamycin, and combinations such as metronidazole and amoxicillin	May be useful to treat aggressive destructive periodontitis	Not indicated for gingivitis Not indicated for most adult periodontitis patients	Assessment of risk-benefit ratio Randomized double-blind clinical trials Randomized double-blind clinical trials, longitudinal assessment of patients

Topical administration of antimicrobial agents contributes to the control of gingival inflammation (Table 8.10). Supragingival irrigation (e.g., applying a jet of water under pressure) may be used as an adjunct to toothbrushing and has been shown to aid in the reduction of gingival inflammation. However, no clear substantial long-term benefits for the treatment of periodontitis have been shown if irrigation is applied subgingivally.

Surgical therapy is employed to provide access to root surfaces and bony defects for debridement and root planing. Surgery can facilitate regeneration, augment the gingiva, and promote root coverage (Table 8.11). It is also necessary in placing dental implants.

Palcanis (1996) reviewed the evidence regarding surgical therapy. The overall goal is to make plaque control easier for the patient, thereby reducing disease progression. Many surgical techniques are available. Extensive randomized clinical trials and longitudinal studies form the basis of the evidence for the efficacy of these procedures (Kaldhal et al. 1996, Knowles et al. 1979, Pihlstrom et al. 1983, Ramfjord et al. 1987). All procedures decrease pocket depth, and, with the exception of gingivectomy, all increase clinical attachment level. A caveat to be noted, however, is that procedures designed to reduce probing depth may increase gum recession, exposing the root and possibly compromising aesthetics. Thus, selection of a particular surgical procedure must always be based on the individual needs of the patient. Regardless of the approach selected, maintenance is important to long-term success.

Systemic administration of antibiotics, including the tetracyclines,³ metronidazole, spiramycin, and clindamycin, has been extensively studied and reviewed (Drisko 1996). The risk of generating antibiotic resistance in bacteria precludes the use of systemic agents in treating simple gingivitis (AAP 1996). Similarly, systemic antibiotics should not be used for the routine first-line treatment of common forms of adult periodontitis (AAP 1996, Drisko 1996). The preponderance of evidence from well-controlled, randomized, blinded clinical trials indicates that the agents do not offer sufficient benefit to overcome risks of either drug sensitivity or the emergence of antibiotic-resistant pathogens.

The situation is different in cases of aggressive forms of periodontitis, such as early-onset, rapidly progressive, or refractory periodontitis, which affect less than 10 percent of periodontitis patients. Randomized, double-blind clinical trials, as well as longitudinal assessments, indicate that the use of systemic antibiotics can slow disease progression in these patients (AAP 1996, Drisko 1996).

To circumvent the problems of systemic therapy, investigators have applied antimicrobial agents directly into the pocket. Antimicrobials incorporated into either resorbable and nonresorbable inter-pocket delivery systems have been studied in randomized, double-blind, controlled clinical trials and are now FDA approved and on the market (Goodson et al. 1991, Jeffcoat et al. 1998). When used as an adjunct to scaling and root planing, gains in clinical attachment level and decreases in probing depth and gingival bleeding were demonstrated. Because

TABLE 8.11
Periodontal disease: selected surgical procedures

Category and Goal	Procedures	Strengths	Weaknesses	Type of Evidence
Pocket therapy— provides access to root surfaces and bony defects, reduces probing depths, facilitates plaque control, and enhances restorative and cosmetic dentistry	Modified Widman Flap to provide access to roots and bony defects for debridement	All procedures decrease pocket depth. With the exception of gingivectomy, all increase clinical attachment level.	Procedures designed to reduce probing depths may increase recession.	Randomized clinical trials Longitudinal studies
	Apically repositioned flap with or without bony recontouring	After 5 years, greatest reduction in probing depth with osseous recontouring.	Lack of professional maintenance and patient compliance can be detrimental to long-term success.	
	Gingivectomy	Apically repositioned flap with or without bony recontouring used in crown-lengthening procedures to provide biologic width.		

³Certain systemic tetracyclines, notably doxycycline, are safe and effective in low doses for prevention of bone loss associated with periodontitis. Doxycycline hyclate (20-mg capsule) is approved for twice-a-day use for up to 9 months for this indication. At these low doses, the doxycycline appears to reduce the elevated collagenase activity, rather than function as an antimicrobial.

these delivery systems are relatively new, there is a paucity of evidence addressing their long-term effectiveness.

For patients who have lost significant bone and/or connective tissue, there are a number of regeneration procedures to facilitate the growth of new periodontal ligament, cementum, and alveolar bone over previously diseased root surfaces. The evidence base for bone-grafting techniques using either natural or synthetic bone materials has been reviewed by Garrett (1996). Natural bone grafts may use autografts, in which bone is transferred from one site to another in the same patient; allografts, which use bone grafts from a human donor; and xenografts, which use tissues from other species. Limited case report evidence shows that extraoral autogenous bone, such as hip grafts, has high potential for bone growth (Garrett 1996). Extraoral sites require a second surgical site, and in some cases fresh grafts may be associated with root resorption. Case report evidence indicates bone fill exceeding 50 percent of the osseous defect may be achieved (Garrett 1996). Controlled studies comparing grafted to nongrafted sites report significant improvements in clinical attachment levels and bone gain, but the magnitude of gain is less than that indicated in case reports.

Freeze-dried demineralized bone represents one of the most frequently used and well-studied bone graft materials in periodontics. Freeze-dried demineralized bone is an allograft material, harvested, prepared, and demineralized prior to grafting. The demineralization step is important because it retains the activity of bone morphogenetic proteins—compounds in the graft material found to be essential for new bone formation (Urist 1965, Urist and Iwata 1973). Case reports and controlled clinical trials have demonstrated the bone-

forming potential of such material, with some variability in the amount of bone fill achieved (Garrett 1996). Because allografts are derived from donor tissues, proper collection, handling, and storage are

TABLE 8.12 a
Odds ratios for risk factors for oral and pharyngeal cancers

	Both Sexes	Males	Females
Cigarettes^a			
Never		1.0	1.0
Ever		1.9	3.0
Smoking status, adjusted for alcohol drinking^a			
None		1.0	1.0
Short duration/former		1.1	1.0
1 to 19 per day for 20+ years		1.6	3.0
20 to 39 per day for 20+ years		2.8	4.4
40+ per day for 20+ years		4.4	10.2
Cigars^b			
Never		1.0	—
Ever		2.8	—
Pipes^b			
Never		1.0	—
Ever		1.8	—
Smokeless tobacco among nonsmokers^a			
Never		—	1.0
Ever		—	6.2
Number of drinks of beer per week, adjusting for smoking^a			
<1		1.0	1.0
1 to 4		1.2	2.2
5 to 14		1.7	2.9
15 to 29		3.4	2.3
30+		4.7	18.0
Number of drinks of hard liquor per week, adjusting for smoking^a			
<1		1.0	1.0
1 to 4		1.0	1.3
5 to 14		1.3	1.5
15 to 29		2.6	4.9
30+		5.5	7.8
Alcohol and tobacco combined^a			
No alcohol or cigarettes		1.0	—
Both 30+ drinks per week and 40+ cigarettes per day for 20+ years		23.0	—
Lip cancer: lifetime quartiles of lifetime sun exposure^c			
Lowest	1.0		
Highest	13.5		
Marijuana use^d			
Never	1.0		
Ever	2.6		
Human papillomavirus, Type 16^e			
Present	2.3		
Absent	1.0		

Note: First row for each factor is the referent group. Dashes indicate not studied or too few to analyze.

^aBlot et al. 1988.

^bSpitz et al. 1988.

^cPogoda and Preston-Martin 1996.

^dZheng et al. 1999.

^eSchwartz et al. 1998.

TABLE 8.12b

Odds ratios for protective factors for oral and pharyngeal cancers

	Both Sexes	Males	Females
Diet high in fruits, by quartiles of intake^a			
First (lowest intake)	1.0		
Second	0.6		
Third	0.4		
Fourth (highest intake)	0.4		
Years since quit smoking cigarettes^a			
Never smoked		1.0	1.0
0 (never)		3.4	4.7
1 to 9		1.1	1.8
10 to 19		1.1	0.8
20+		0.7	0.4
Lip cancer: daily use of lip protection (mostly colored lipstick)^b			
None		—	1.0
≤1 per day		—	3.2
≥2 per day		—	7.3

Note: First row for each factor is the referent group. Dashes indicate not studied or too few to analyze.

^aBlot et al. 1988.

^bPogoda and Preston-Martin 1996.

essential to ensure viability and prevent contamination with viruses or other pathogens (Mellonig 1995).

Alloplasts represent a class of synthetic resorbable or non-resorbable graft materials. When evaluated in controlled clinical trials, they demonstrated improvements in probing depth and attachment level (Garrett 1996). Histology, however, indicates that, in general, synthetic grafts act primarily as space fillers, with little, if any, regeneration.

Beginning in the 1980s a number of investigators explored a procedure called *guided tissue regeneration*. The idea was to employ either a resorbable or non-resorbable membrane at the diseased site that would selectively allow passage of cells able to regenerate periodontal attachment apparatus and bone, while prohibiting migration of non-regenerative cells such as fibroblasts. The evidence for the efficacy of guided tissue regeneration ranged from randomized controlled clinical trials to case reports (Garrett 1996). Although less evidence is available for resorbable membranes than for non-resorbable membranes, significant improvements in clinical attachment levels have been shown compared to debridement alone. Most favorable results are reported for bone loss between the roots of mandibular tooth defects (Class II furcations). Less favorable results were reported in maxillary molar and Class III (through and through) furcation defects (Garrett 1996).

Oral and Pharyngeal Cancers

Oral and pharyngeal cancers, like other neoplastic diseases, are caused by mutations in cell regulatory genes. The mutations contribute to carcinogenesis by promoting uncontrolled cell growth, suppressing the function of tumor suppressor genes, promoting the growth of new blood vessels (angiogenesis) to nourish the growing tumor, or facilitating metastasis. A model depicting the genetic progression for oral and pharyngeal cancers has been proposed (Califano et al. 1996).

Potentially malignant lesions can present in a variety of ways and can include erythroplakia or leukoplakia (red or white flat lesions, respectively), ulceration, failure of a wound to heal, lym-

phadenopathy, induration, dysphagia, and tissue growth. Erythroplakia lesions are considered to have a higher rate of malignancy than leukoplakia (Silverman 1990). *Candida albicans* infection of a leukoplakia lesion appears to increase the risk of malignant transformation (Field et al. 1989, Scully 1995). In addition, infections with strains of human papillomavirus (HPV) and herpes simplex virus (HSV) have been implicated in the etiology of oral cancers. Other potentially predisposing factors include chronic iron deficiency anemia, erosive lichen planus, oral submucous fibrosis, and actinic keratosis.

Risk Assessment. Risk assessment for oral and pharyngeal cancers (Table 8.12a) includes an evaluation of the patient's exposure to tobacco and alcohol and an examination to identify suspicious lesions and conditions thought to predispose to cancer. All tobacco products (see Chapters 3 and 10) have been associated with oral and pharyngeal cancers. The risk of oral cancer is increased 6 to 28 times in current smokers. Alcohol is also a risk factor for oral cancer, and combined with tobacco use accounts for 75 to 90 percent of oral cancer in the United States (Blot et al. 1988, Vokes et al. 1993). A dose-response relationship has been demonstrated for cigar smokers, and the overall risk of cancer is 7 to 10 times higher among users than for those who never smoked (NCI 1998). In addition, individuals who have had oral cancer are at increased risk for a second primary

cancer, and this risk is higher than that for other cancers (Boice et al. 1985, Winn and Blot 1985).

Other factors, such as infection with HPV or HSV, as noted earlier, and use of high-alcohol-content mouthwashes, also have been associated with oral cancers (Flaitz and Hicks 1998, Sugerman and Shillitoe 1997, Winn et al. 1991). Lip cancer is associated with exposure to the sun (Pogoda and Preston-Martin 1996).

Biomarkers—measurable alterations in molecules derived from human tissues or fluids—are being developed to identify those at risk for oral cancer and to identify which patients may benefit from specific treatments (Lippman et al. 1993, Patterson et al. 1996, Sidransky 1997). These markers may also be able to predict oral cancer recurrence or the occurrence of new primaries (Shin et al. 1996).

Diagnosis. At present, the diagnosis of oral and pharyngeal cancers involves a systematic extra- and intraoral physical examination to identify lesions and conditions that may be precancerous or indicate a predisposition to cancer (USDHHS 1998). Biopsies are essential to confirm the clinical observations. The sensitivity and specificity of a brush biopsy have been established in a multisite clinical trial (Sciubba 1999). Imaging technology may also be employed to determine the extent of the lesion. Although the sensitivity and specificity of the physical examination have not been established in clinical studies, it is clear that persons with localized oral and pharyngeal cancers have a better prognosis than persons whose cancers were detected with regional or distant spread (Ries et al. 1999). A thorough examination for oral and pharyngeal cancers is recommended by the American Cancer Society annually for individuals over 40 and for individuals who are exposed to known risks (Murphy et al. 1995).

Prevention. Primary prevention of oral and pharyngeal cancers involves avoiding known carcinogenic agents (Blot et al. 1988, Vokes et al. 1993), primarily tobacco in any form and excessive use of alcohol. In addition, the use of lip balms with ultraviolet radiation blockers is recommended (see Table 8.12b). A high dietary intake of fruits and vegetables may reduce oral and pharyngeal cancer risk by as much as 30 to 50 percent (McLaughlin et al. 1988). Dentists, physicians, and nurse practitioners among others are in a critical position to counsel patients on tobacco and alcohol use, pointing out that tobacco cessation lowers the risk for oral and pharyngeal cancers (Blot et al. 1988). Physicians, dentists, and other health care professionals have been shown to be effective in

increasing tobacco cessation rates (Cohen et al. 1989, Ockene et al. 1991, Stevens et al. 1995, Wilson et al. 1988). A prime reference for use in national and international antismoking efforts is the *Clinical Practice Guideline on Smoking Cessation* (Number 180) developed by the Agency for Health Care Policy and Research (Fiore 1997).

Treatment. Diagnosis of cancer at an early stage can be followed by prompt and conservative treatment of the affected tissues. Some early lesions may be successfully treated with excisional biopsy; more advanced cases will require additional surgery, radiation, and/or chemotherapy (Shah and Lydiatt 1995, Vokes and Athanasiadis 1996). Preservation of function and appearance is emphasized. Advanced cancers require follow-up reconstruction and rehabilitation to improve function and aesthetics.

Birth Defects

There are hundreds of genetic diseases and syndromes as well as congenital anomalies that affect the craniofacial, oral, and dental tissues. However, some craniofacial anomalies may be spontaneous and manifest only at the time of birth. Chapter 3 describes a number of these disorders. Rapidly advancing knowledge of the genetics of development and of mutations associated with specific birth defects is aiding in the development of screening tests for genetic disorders and identifying high-risk individuals and families.

A complete diagnosis of the craniofacial disorder may involve a multidisciplinary team of experts in imaging, genetics, and other areas. Similarly, long-term management of the disorder, often extending to adulthood, generally calls for a team of specialists, including physicians and dentists, surgeons, nurses, rehabilitation experts, speech pathologists, psychologists, and social workers. Quality of life considerations, including social and psychological effects of birth defects such as cleft lip and palate, are taken into account (see Chapters 3 and 6).

Prevention. Primary prevention involves minimizing exposure to known teratogens, and genetic counseling as appropriate. The importance of educating parents or potential parents on behavioral risk factors, especially tobacco and alcohol use, the teratogenic potential of certain prescription drugs, and the need for adequate nutrition in the perinatal period is emphasized. In a study by Tolarova and Harris (1995), supplementation of the diet by multivitamins and folic acid during the periconceptional period (i.e., before, during, and after conception) markedly

diminished the occurrence of cleft lip and palate in a high-risk group. Unfortunately, only about 29 percent of women of childbearing age consume recommended amounts of these essential nutrients (Werler et al. 1999). The evidence associating moderate to severe periodontal disease in pregnant women with low-weight preterm births warrants attention to the importance of maintaining optimal oral health in pregnancy. The oral care clinician can contribute to birth defect prevention not only by treating oral disease, but also by providing educational messages to patients to promote the birth of healthy, full-term babies.

Treatment. A number of birth defects may not be apparent at birth because they are not manifested until later in development. One example is the ectodermal dysplasias (EDs), disorders characterized by abnormalities of skin, hair, sweat glands, and teeth. Dentists are essential in the management of care for children with these disorders, who must be repeatedly fitted with dentures throughout childhood. More recently, clinical studies have demonstrated that fitting ectodermal dysplasia patients as young as 12 years old with dental implants not only is effective, but also provides greater functional utility and satisfaction (Guckes et al. 1998, Kearns et al. 1999). As with other complex craniofacial anomalies, management by a multidisciplinary team is the best approach, with experts able to advise on the various oral, skin, and sweat gland complications.

Mutations have recently been identified for several forms of ED, including the anhydrotic form (absence of sweat glands). Ultimately, the development of genetic diagnostic tests can confirm the diagnosis in the child and permit counseling of parents.

Chronic Craniofacial Pain and Sensorimotor Conditions

A variety of problems involving pain and other sensorimotor abnormalities affect the craniofacial complex. These conditions can include burning mouth syndrome, trigeminal neuralgia, various facial palsies, postherpetic neuralgia affecting branches of the trigeminal nerve, temporomandibular disorders, fibromyalgia, and disorders of taste or olfaction. Some of these are infectious in origin (e.g., postherpetic neuralgia and some taste disorders); some are traumatic (e.g., some cases of temporomandibular disorder); and for others, the cause or causes are unknown (see Chapter 3). Patients with facial palsies and trigeminal neuralgia are generally referred to neurologists for treatment. Disorders of taste and

smell also require neurological consultation as well as brain imaging because they can be symptomatic of brain tumors.

Pain relief may also improve function and can be combined with adjunctive measures such as the use of hot or cold compresses and behavioral treatments such as relaxation and imaging therapy to reduce muscle tension. The variety of pain medications has greatly increased in recent years. They include aspirin and other nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, new antiepileptic drugs, the selective serotonin re-uptake inhibitors, and the more potent opiate family of drugs.

If the pain problem has recently developed, providers take steps to prevent the pain from becoming chronic. This will entail a general health assessment to determine whether there are co-morbidities, including other pain problems, as well as patient questionnaires to provide information on how the pain problem is affecting overall health and well-being. The data collected will record the extent to which the problem interferes with work, social interaction, and sleep, whether the patient is experiencing mood changes and symptoms of depression, and what coping skills are manifest. Such patient profiles allow for more selective treatment tailored to the needs of the individual patient.

Patients in whom pain has become chronic and intractable may be referred to an established pain clinic for multidisciplinary treatment and may also be alerted to patient organizations where individuals with similar pain problems can find information and support.

Temporomandibular Disorders

Among the common types of craniofacial pain likely to be seen by oral care providers are temporomandibular disorders, characterized by symptoms of pain and dysfunction in and around the temporomandibular joints or the masticatory muscles.

Temporomandibular disorders may occur as a result of injury, arthritis, or fibromyalgia or for unknown reasons. Approaches used to obtain a differential diagnosis of these conditions can range from a physical examination that may include palpation and measuring the mouth opening, to the use of complex imaging and instrumentation, including procedures such as arthroscopy (Table 8.13) (Clark et al. 1993, NIH 1996, Rao 1995, Rao et al. 1990).

Diagnosis of temporomandibular disorders is based on the physical examination and a complete medical and dental history, including information about hearing, speech, and swallowing problems,

as well as pain and dysfunction. This information can be complemented by data from imaging and other diagnostic tests (Clark et al. 1993, NIH 1996). Evaluation encompasses examination of oral-facial tissues, musculature, and neurological function.

Particular attention is paid to measures of the range of motion, mouth opening, existence of any parafunctional conditions (e.g., clenching, grinding), and the presence of joint or muscle tenderness and cutaneous hyperalgesia. Features of the reliability studies on the examination methods have been reviewed (Clark et al. 1993, Mohl et al. 1990). Psychosocial assessments using validated instruments can determine the extent to which pain and dysfunction diminish the patient's quality of life (Dworkin 1994) and can suggest appropriate treatments (NIH 1996, Travell and Simons 1983, Zarb 1994).

The evidence base for the efficacy of treatment modalities is severely limited and has resulted in a wide range of diagnostics and therapies. Treatments range from conservative and reversible approaches to joint surgical procedures. At present the evidence is insufficient to warrant prophylactic intervention for management of these disorders (NIH 1996).

Currently available epidemiological evidence suggests that temporomandibular disorders can frequently resolve over time and that conservative, reversible approaches are the treatments of choice. Ideally, the practitioner and the patient should work together to develop a treatment plan that is evidence-based and patient-centered, taking into consideration all etiologic factors, the level of pain and dysfunction the patient is experiencing, and their impact on the patient's quality of life (see Chapter 3).

Mucosal and Autoimmune Diseases

Microbial infections and autoimmune disorders contribute to a range of mucosal and gingival conditions. The physical examination may be sufficient to identify the lesions associated with herpes virus infections (cold sores), papillomaviruses (oral warts), or fungal infection (commonly, infection by *Candida albicans*), with definitive diagnosis confirmed by cytology, biopsy, culture, or in situ hybridization. The patient's history and immune status can supply additional information indicating risk factors, including the presence of systemic diseases. Selected major mucosal diseases and associated clinical findings are described in Chapter's 3 and 5. Tissue biopsy is critical for the diagnosis of many mucosal diseases. In addition, oral sites may be convenient biopsy sites for autoimmune diseases such as Sjögren's syndrome, which have both a systemic and an oral-facial component. Although the evidence base in terms of randomized, double-blind, controlled clinical trials for the treatment of oral manifestations of mucocutaneous disorders is limited, treatment options generally depend on the severity and extent of the disease. Because many of the available drugs may have significant side effects, evaluation of the risk-benefit ratio for the patient is always of great importance. Coordination of care with other health care providers is warranted.

FACTORS AFFECTING FUTURE HEALTH CARE PRACTICES

The last decades of the twentieth century were witness to major improvements in the prevention, diagnosis, and treatment of oral diseases—a trend that will continue to accelerate the paradigm shift in the management of oral diseases from repair of damaged tissues to the control of infections. In addition, modification of risk factor exposures will result in improvements in health and in the management of disease. A closer look into factors that will affect the future of oral health care requires an overview of the current state of guidelines for oral care and the status of evidence-based practice. The approaches used to determine the evidence for practice and the development of guidelines for care are an emerging field of activity. Education in the health professions is already emphasizing the

TABLE 8.13
Diagnosis of temporomandibular disorders

Test	Application	Strengths	Weaknesses	Type of Evidence
Clinical exam	All patients	Important to rule out contributing factors/other disease states	Does not provide information on etiology	Epidemiologic studies
Radiographic survey	All patients	Rules out structural problems	Depending on type of survey, certain information is lost	Epidemiologic studies
Specialized imaging (arthrography, CT, MRI, etc.)	Patients with history and exams indicative of anatomic alterations	May locate the disk, rule out or indicate anatomic change	Depending on type of survey, information may not be obtained	Epidemiologic studies; case reports

importance of relying on randomized, controlled clinical trials, the gold standard for judging the merits of proposed interventions, wherever possible.

Evidence-based Practice

During the 1990s, "evidence-based medicine" emerged as both popular phraseology and practice philosophy. The origins of evidence-based medicine go back to mid-nineteenth-century Paris and earlier, yet the approach is still a relatively young discipline that is now rapidly evolving (Sackett et al. 1996). Evidence-based medicine has been defined as the integration of "individual clinical expertise with the best available external clinical evidence from systematic research" and with patients' choices (Sackett et al. 1996). The skills required include defining a clinical problem, critically appraising the relevant literature, and deciding whether and how to integrate this information into practice (Evidence-Based Medicine Working Group 1992). Evidence-based medicine is neither a "cookbook" nor an ivory tower approach (Sackett et al. 1996).

The philosophy is being adopted across a range of disciplines, leading to the terms "evidence-based dentistry" and "evidence-based nursing," among others. The practice of evidence-based dentistry "incorporates the judicious use of the best evidence available from systematic reviews, when possible, with knowledge of patients' preferences and clinicians' experiences to make recommendations for the provision of the right care, for the right patient, and at the right time" (Ismail et al. 1999).

The reliance on evidence using systematic reviews of the literature has led to initiatives in the United States, Canada, and Europe to enhance the conduct and use of systematic reviews. The Agency for Healthcare Research and Quality (AHRQ) created 12 evidence-based practice centers in 1997 to conduct systematic reviews and develop evidence reports. The Cochrane Collaboration and the Centre for Reviews and Dissemination at the University of York are examples of prominent activities in the United Kingdom to support systematic reviews. The Cochrane Oral Health Review Group, one of 50 specialty review groups within the Cochrane Collaboration, has a number of systematic reviews completed or under way of interest to oral health practitioners (see Table 8.14) (Tavender 1999). In Canada, considerable contributions to the field have been made by McMaster University and the Canadian Coordinating Office for Health Technology Assessment.

In the United States, the National Institute of Dental and Craniofacial Research joined efforts with AHRQ in 1999 to designate one of AHRQ's Evidence-based Practice Centers to conduct reviews on oral, dental, and craniofacial diseases and disorders. The work of this center should significantly strengthen the scientific base of knowledge related to the diagnosis and management of oral, dental, and craniofacial conditions. Examples of topics that will be reviewed include the management of dental caries, and dental care of medically compromised patients, including patients with HIV disease.

Clinical Practice Guidelines

The development of clinical practice guidelines is one of the intended outcomes of evidence-based reviews. The classic definition for clinical practice guidelines describes them as "systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances" (Field and Lohr 1992). A 1995 review of the status of clinical practice guidelines in dentistry (Shugars and Bader 1995) found that a limited number of guidelines were available and that few extant guidelines met all of the desirable attributes for guidelines identified by the Institute of Medicine (Field and Lohr 1992). Most notably, a majority of guidelines were based on consensus among selected professionals, with little evidence of support from the scientific literature. Since 1995, the number of dental practice guidelines has grown slowly but steadily. Table 8.15 lists selected sets of guidelines that, taken together, represent an estimated 50 percent of all current published dental guidelines intended for national distribution. When 36 national dental organizations representing clinical aspects of dentistry and dental practice were surveyed in early 1999, 12 of 22 responding organizations indicated that they had developed guidelines (J.D. Bader, personal communication, 1999), 8 of which are listed in the table. Some of these guidelines have not been widely distributed, are not published in the scientific literature, nor are available on the sponsoring organization's Web site. Not shown are guidelines developed by care delivery organizations for use in their clinical practices. These are generally not available for public or external professional scrutiny.

The table provides information on two important characteristics of clinical practice guidelines: the extent to which they are evidence-based and their level of specificity, which will determine their clinical applicability. Clinical applicability is a key feature of

practice guidelines, and it is heightened as the amount of specific information in a guideline is increased to identify patient and condition characteristics to which the guideline applies. As the "evidence-based" concept gains popularity, there is a growing expectation that clinical practice guidelines will reflect systematic evaluation of the relevant literature and will present an evaluation of the strength of the evidence for each recommendation (Ismail et al. 1999). Such information, which ensures the content validity of the guidelines, not only helps practitioners and patients understand exactly what is and is not known about the effectiveness of proposed treatments, but also identifies research needed to evaluate current practice. A less comprehensive but still useful approach to identifying the scientific support for clinical interventions is the traditional approach of citing specific studies and reviews when discussing specific treatments. Practice guidelines without explicit linkages to the literature, such as those supported only by selected citations not linked to specific statements and those without any citations, can still be useful, but users have less assurance that the content is valid.

As the expectations for the content and use of clinical practice guidelines mature, "perhaps the main task of guideline development [will be] to summarize the strength of the evidence for the effectiveness of a given clinical practice in relation to risks and costs" (Fletcher and Fletcher 1998). Thus, prac-

tice guidelines will need to be revised considerably to incorporate the literature on treatment outcomes as they become available. Currently, information on both the effectiveness of specific dental treatments and the range of outcomes examined is extremely limited (Bader and Shugars 1995). For example, none of the guidelines in the table address patient preference or patient utility issues. Dental organizations have the opportunity to address these information gaps as practice guidelines are developed. Professional dental organizations are the most likely developers, but other organizations can also be vigorous participants. Finally, organizations developing guidelines should also develop a plan for their dissemination, evaluation, and revision. The existence of guidelines does not ensure that appropriate treatment decisions will be made. Passive distribution of clinical practice guidelines is generally ineffective in altering practice, whereas more active, multifaceted interventions can achieve some measure of desired change (Bero et al. 1998).

Science and Technology Contributions

During the past several decades, there have been major improvements in the prevention, diagnosis, and treatment of oral diseases. Enhanced disease prevention and health promotion will require the participation of all health professions, especially in addressing common risk factors such as tobacco, alcohol, and inappropriate dietary practices. The field of diagnostic tests for oral diseases should continue to expand, enabling clinicians to analyze the risk of disease and disease progression for individual patients. Full assessment of the strengths and weaknesses of new diagnostic tests and evaluation of when they are best used will be key to proper interpretation of the results, permitting tailored referrals and treatments. Treatment options for individual patients are increasing, including the regeneration of lost bone and connective tissue. Restorative materials are continuously improving, resulting in safe, effective, and aesthetic restorations. The growing field of biomimetics should continue to revolutionize oral health and oral health care. Development of bio-compatible restorative and implant

TABLE 8.14
Systematic reviews on oral health topics conducted by the Cochrane Collaboration Oral Health Review Group

	Status ^a
Interventions for treating oral leukoplakia	Protocol
Prevention of oral mucositis or oral candidiasis for patients with cancer receiving chemotherapy (excluding oral and pharyngeal cancers)	Full review
Treatment of oral candidiasis for patients receiving chemotherapy or radiotherapy	Protocol
Treatment of oral mucositis or its associated pain for patients receiving chemotherapy or radiotherapy	Protocol
Topical fluoride for preventing dental caries in children and adolescents	Protocol
Pit-and-fissure sealants for preventing decay in the permanent teeth of children and adolescents	Protocol
Guided tissue regeneration for periodontal intrabone defects	Protocol
Orthodontic treatments for posterior cross-bites	Full review
Interventions for treating oral lichen planus	Full review
Potassium-nitrate-containing dentifrice for dentin hypersensitivity	Protocol

^aProtocols are the introduction, objectives, materials, and methods for reviews currently being prepared.
 Source: Tavender 1999.

materials will continue, as well as development of new biologically engineered substitutes for lost bone, connective tissue, and diseased articular disks, to name several possibilities. Harnessing other basic science knowledge will enable the development of new therapies such as genetically engineered growth factors. Improved understanding of the genetic risk factors, limitation of exposures to teratogens, and attention to diet may markedly diminish the occurrence of congenital anomalies such as cleft lip and palate.

Science is continuing to reveal the intricacies and complexities of disease etiology and pathogenesis. In turn, the classification and diagnosis of diseases and conditions will improve and lead to tailored treatment options. The recent efforts to understand and define early childhood caries are an example of this evolutionary process (Ismail et al. 1999). This example further demonstrates that disease definitions are important for population-based research (Drury et al. 1999, Kaste et al. 1999).

TABLE 8.15
Characteristics of selected dental clinical practice guidelines

	Use of the Scientific Literature				Level of Specificity/Applicability		
	Strength Evaluated	Text Linked to Citations	Selected Citations	None Cited	Algorithm or Protocol	Recommendations	Lists of Considerations
U.S. professional dental organizations							
American Dental Association dental practice parameters (1996) ^a				◆			◆
American Association of Endodontists appropriateness of care guidelines (1994)			◆				◆
American Association of Orthodontists clinical practice guidelines (1996)			◆				◆
American Association of Oral and Maxillofacial Surgeons parameters of care (1995)			◆	◆			◆
American Academy of Pediatric Dentistry Guidelines (1998)			◆	◆			◆
American Academy of Periodontology parameters of care (1996)			◆				◆
American College of Prosthodontists parameters of care (1996)			◆				◆
American Cleft Palate-Craniofacial Association parameters (1993)			◆	◆			
U.S. government agencies							
U.S. Preventive Services Task Force counseling to prevent dental and periodontal disease (1996)	◆	◆	◆			◆	
Office of Medical Applications of Research management of temporomandibular disorders (1996)	◆					◆	
Food and Drug Administration selection of patients for x-ray examinations (1987)		◆	◆		◆	◆	
Other organizations							
American Heart Association prevention of bacterial endocarditis (1997)	◆	◆			◆	◆	
Workshop on Guidelines for Sealant Use Proceedings (1995)		◆					
Canadian Task Force on Preventive Health Care prevention of periodontal disease (1993)	◆	◆				◆	
Canadian Paediatric Society the use of fluorides in infants and children (1996)		◆			◆	◆	
Task Force on Periodontal Regeneration of Intra-bony Pockets ^b periodontal regeneration of intra-bony defects (1995)		◆			◆	◆	

^aRefers to publication date of collected set of guidelines.

^bCorporate sponsorship.

One area critical to the ability of dentists to adopt new treatment modalities or diagnostic techniques is the development of diagnostic codes. In contrast to medicine, these diagnostic codes currently have no impact on reimbursement. However, the development and introduction of such codes are essential for the conduct of needed outcomes research, and their widespread use is necessary for practice-based research. Such codes permit the documentation of preexisting conditions, monitoring of disease progression, and provision of surveillance data in public health programs. A pilot study in a Canadian public health program has proposed and implemented a set of diagnostic codes (Leake et al. 1999). The American Dental Association has undertaken the development of a comprehensive set of diagnostic codes, expected to be released in the near future.

Broadening the Base for the Provision of Oral Health Care

Further biologic, scientific, and technological advances and changes in the organization of health care delivery will continue to alter future professional and individual health care practices (see Chapters 9 and 11). The increased knowledge of risk factors, the importance of monitoring disease progression and treatment effects, and the ability to diagnose conditions and intervene earlier will necessitate increased involvement of all health professionals in oral health care and may reflect changes in care provision and referral patterns. Management of conditions such as oral and pharyngeal cancers, cleft lip/palate, and chronic pain requires multidisciplinary teams. The promotion of oral health and the prevention of oral disease are at a turning point. A systematic approach to integrate the scientific findings into evidence-based assessments will provide clearer guidance to all health care professions and the public. To capitalize on the rapidly emerging science base, the active participation of a full range of health care providers and individuals and the community is needed.

FINDINGS

- Achieving and maintaining oral health require individual action, complemented by professional care as well as community-based activities.
- Individuals can take actions, for themselves and for persons under their care, to prevent disease and maintain health. Primary prevention of many oral, dental, and craniofacial diseases and conditions is possible with appropriate diet, nutrition, oral

hygiene, and health-promoting behaviors, including the appropriate use of professional services. Individuals should use a fluoride dentrifice daily to help prevent dental caries and should brush and floss daily to prevent gingivitis.

- All primary care providers can contribute to improved oral and craniofacial health. Interdisciplinary care is needed to manage the oral health-general health interface. Dentists, as primary care providers, are uniquely positioned to play an expanded role in the detection, early recognition, and management of a wide range of complex oral and general diseases and conditions.
- Nonsurgical interventions are available to reverse disease progression and to manage oral diseases as infections.
- New knowledge and the development of molecular and genetically based tests will facilitate risk assessment and management and improve the ability of health care providers to customize treatment.
- Health care providers can successfully deliver tobacco cessation and other health promotion programs in their offices, contributing to both overall health and oral health.
- Biocompatible rehabilitative materials and biologically engineered tissues are being developed and will greatly enhance the treatment options available to providers and their patients.

REFERENCES

- Ainamo J, Barmes D, Beagrie G, Cutress T, Martin J, Sardo-Infirri J. Development of the World Health Organization (WHO) community periodontal index of treatment needs (CPITN). *Int Dent J* 1982;32(3): 281-91.
- Albrektsson T. A multicenter report on osseointegrated oral implants. *J Prosthet Dent* 1988;60:75-84.
- Albrektsson T, Dahl E, Enbom L, Engevall S, Engquist B, Eriksson AR, Feldmann G, Freiberg N, Glantz PO, Kjellman O, et al. Osseointegrated oral implants: a Swedish multicenter study of 8139 consecutively inserted Nobelpharma implants. *J Periodontol* 1988; 59:287-96.
- American Academy of Periodontology (AAP). World Workshop on Clinical Periodontitis Consensus Report. *Ann Periodontol* 1996;1:926-32.
- American Dental Association (ADA). A guide to the use of fluorides for the prevention of dental caries. *J Am Dent Assoc* 1986;113:505-65.
- American Dental Association (ADA). New fluoridation guidelines proposed. *J Am Dent Assoc* 1994;125:136.
- American Dental Association (ADA), Council on Access, Prevention and Interprofessional Relations. Caries diagnosis and risk assessment. A review of preventive strategies and management. *J Am Dent Assoc* 1995;126:15-245.

- Anderson MH, Bales DJ, Omnell KA. Modern management of dental caries: the cutting edge is not the dental bur. *J Am Dent Assoc* 1993;124:37-44.
- Angmar-Månsson B, Al-Khateeb S, Tranaeus S. Intraoral use of quantitative light-induced fluorescence for caries detection. In: Stookey GK, editor. Early detection of dental caries. Indianapolis: Indiana University School of Dentistry; 1996. p. 105-18.
- Armitage GC. Periodontal diseases: diagnosis. *Ann Periodontol* 1996;1:37-215.
- Armitage GC, Jeffcoat MK, Chadwick DE, Taggart EJ Jr, Numabe Y, Landis JR, Weaver SL, Sharp TJ. Longitudinal evaluation of elastase as a marker for the progression of periodontitis. *J Periodontol* 1994; 65:120-8.
- Axelsson P, Lindhe J, Nystrom B. On the prevention of caries and periodontal disease. Results of a 15-year longitudinal study in adults. *J Clin Periodontol* 1991;18(3):182-9.
- Bader JD, Shugars D. Variation in dentists' clinical decisions. *J Public Health Dent* 1995;55:181-8.
- Bawden JW, editor. Changing patterns of fluoride intake. Proceedings of the workshop. *J Dent Res* 1992;71:1212-27.
- Beck J. Identification of risk factors. In: Bader JD, editor. Risk assessment in dentistry: proceedings of a conference, 1989 June 2-39: Chapel Hill, NC. Chapel Hill: University of North Carolina School of Dentistry; 1990.
- Benn DK, Watson TF. Correlation between film position, bitewing shadows, clinical pitfalls and the histologic size of approximal lesions. *Quintessence Int* 1989;20:131-41.
- Berkowitz RJ, Jordan HV, White G. The early establishment of *Streptococcus mutans* in the mouths of infants. *Arch Oral Biol* 1975;30:171-4.
- Bero LA, Grilli R, Grimshaw JM, Harvey E, Oxman AD, Thomson MA. Closing the gap between research and practice: an overview of systematic reviews of interventions to promote the implementation of research findings. The Cochrane effective practice and organization of care review group. *BMJ* 1998 Aug 15; 317(7156):465-8.
- Blot WJ, McLaughlin JK, Winn DM, Austin DF, Greenberg RS, Preston-Martin S, Bernstein L, Schoenberg JB, Stemhagen A, Fraumeni JF. Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Res* 1988;48:3282-7.
- Boice JD, et al. Multiple primary cancers in Connecticut and Denmark. Washington: US GPO; 1985.
- Bricker SL, Langlais RP, Miller CS. Oral diagnosis, oral medicine, and treatment planning. Philadelphia: Lea and Febiger; 1994.
- Burt BA, Ismail AI. Diet, nutrition, and food cariogenicity. *J Dent Res* 1986;65:1475-84.
- Buser D, Schroeder A, Sutter F, Lang N. The new concept of ITI hollow-cylinder and hollow-screw implants: part 2. Clinical aspects, indications, and early clinical results. *Int J Oral Maxillofac Implants* 1988;3:173-81.
- Buser D, Weber HP, Bragger U, Balsiger C. Tissue integration of one-stage ITI implants: 3-year results of a longitudinal study with hollow-cylinder and hollow-screw implants. *Int J Oral Maxillofac Implants* 1991; 6:405-12.
- Califano J, van der Riet P, Westra W, Nawroz H, Clayman G, Piantadosi S, Corio R, Lee D, Greenberg B, Sidransky D, et al. Genetic progression model for head and neck cancer: implications for field cancerization. *Cancer Res* 1996;56(11):2488-92.
- Caulfield PW, Cutter GR, Dasanayake AP. Initial acquisition of mutans streptococci infections in infants: evidence for a discrete window of infectivity. *J Dent Res* 1993;72:37-45.
- Cavanaugh PF, Meredith MP, Buchanan W, Doyle MJ, Reddy MS, Jeffcoat MK. Coordinate production of PGE₂ and IL-1 in the gingival crevicular fluid of adults with periodontitis: its relationship to alveolar bone loss and disruption by twice daily treatment with ketorolac tromethamine oral rinse. *J Periodontal Res* 1998;33:75-82.
- Centers for Disease Control and Prevention (CDC). Recommendations for using fluoride to prevent and control dental caries in the United States. In press.
- Chambers DA, Imrey PB, Cohen RL, Crawford JM, Alves ME, McSwiggin TA. A longitudinal study of aspartate aminotransferase in human gingival crevicular fluid. *J Periodontal Res* 1991;26:65-74.
- Clark GT, Delcanho RE, Goulet JP. The utility and validity of current diagnostic procedures for defining temporomandibular disorder patients. *Adv Dent Res* 1993;7:97-112.
- Clarkson JE, Ellwood RP, Chandler RE. A comprehensive summary of fluoride dentifrice caries clinical trials. *Am J Dent* 1993 Sep;6(Spec No):S59-106.
- Cobb CM. Non-surgical pocket therapy: mechanical. *Ann Periodontol* 1996;1:443-90.
- Cochran D. Implant therapy I. *Ann Periodontol* 1996;1: 707-90.
- Cohen SJ, Stookey GK, Katz BP, Drook CA, Christen AG. Helping smokers quit: a randomized controlled trial with private practice dentists. *J Am Dent Assoc* 1989;118:41-5.
- Cutress TW, Powell RN, Kilisimasi S, Tomiki S, Holborow D. A 3-year community-based periodontal disease prevention programme for adults in a developing nation. *Int Dent J* 1991;41(6):323-34.
- de Josselin de Jong E, Hall AF, van der Veen MH. Quantitative light-induced fluorescence for caries detection. In: Stookey GK, editor. Early detection of dental caries. Indianapolis: Indiana University School of Dentistry; 1996. p. 91-103.
- Douglass CW. Risk assessment in dentistry. *J Dent Educ* 1998;62:756-61.
- Drisko CH. Non-surgical pocket therapy: pharmacotherapeutics. *Ann Periodontol* 1996;1:491-566.

- Drury TF, Horowitz AM, Ismail AI, Maertens MP, Rozier RG, Selwitz RH. Diagnosing and reporting early childhood caries for research purposes. A report of a workshop sponsored by the National Institute of Dental and Craniofacial Research, the Health Resources and Services Administration, and the Health Care Financing Administration. *J Public Health Dent* 1999 Summer;59(3):192-7.
- Dworkin SF. Perspectives on the interaction of biological, psychological and social factors in TMD. *J Am Dent Assoc* 1994;125:856-63.
- Edelstein BL. The medical management of dental caries. *J Am Dent Assoc* 1994;125:31s-39s.
- Ellwood RP, Davies R, Worthington HV. Evaluation of a dental subtraction radiography system. *J Periodontol Res* 1997;32(2):241-8.
- Evidence-Based Medicine Working Group. Evidence-based medicine: a new approach to teaching the practice of medicine. *JAMA* 1992;268:2420-5.
- Featherstone JD. Clinical implications of early caries detection: new strategies for caries prevention. In: Stookey GK, editor. *Early detection of dental caries*. Indianapolis: Indiana University School of Dentistry; 1996. p. 285-93.
- Featherstone JD, Barrett-Vespone NA, Fried D, Kantorowitz Z, Seka W. CO₂ laser inhibition of artificial caries-like lesion progression in dental enamel. *J Dent Res* 1998;77:1397-403.
- Field EA, Field JK, Martin MV. Does *Candida* have a role in oral epithelial neoplasia? *J Med Vet Mycol* 1989;27:277-94.
- Field M, Lohr K, editors. *Guidelines for clinical practice: from development to use*. Washington: National Academy Press; 1992.
- Fiore MC. Smoking cessation: a systems approach. Clinical practice guideline, no. 18. Rockville (MD): Agency for Health Care Policy and Research; 1997. AHCPR Pub. no. 97-0698. Available from: <http://www.ahcpr.gov/clinic/smoksys.htm>.
- Flaitz CM, Hicks MJ. Molecular piracy: the viral link to carcinogenesis. *Oral Oncol* 1998;34:448-53.
- Fletcher SW, Fletcher RH. Development of clinical guidelines. *Lancet* 1998;12;352(9144):1876.
- Food and Drug Administration (FDA). The selection of patients for x-ray examination: dental radiographic examinations. Rockville (MD): Food and Drug Administration; 1987. DHHS Pub. no. 88-8273.
- Fritz ME. Implant therapy II. *Ann Periodontol* 1996;1:796-815.
- Garrett S. Periodontal regeneration around natural teeth. *Ann Periodontol* 1996;1:621-66.
- Geiger AM, Gorelick L, Gwinnett AJ, Benson BJ. Reducing white spot lesions in orthodontic populations with fluoride rinsing. *Am J Orthod Dentofac Orthop* 1992;101(5):403-7.
- Geivellis M, Turner DW, Pederson ED, Lamberts BL. Measurements of interleukin-6 in gingival crevicular fluid from adults with destructive periodontal disease. *J Periodontol* 1993;64:980-3.
- Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996 Oct;67(10 Suppl):1041-9.
- Genco RJ. Risk factors for periodontal disease. In: Rose LF, Genco RJ, Cohen DW, Mealey BL, editors. *Periodontal medicine*. Hamilton (Ontario): B.C. Decker Inc.; 2000.
- Genco RJ, Newman MG, editors. Consensus report on prevention. Proceedings of the 1996 World Workshop in Periodontics. *Ann Periodontol* 1996;1:250-5.
- Genco RJ, Ho AW, Grossi SG, Dunford RG, Tedesco LA. Relationship of stress, distress, and inadequate coping behaviors to periodontal disease. *J Periodontol* 1999;70:711-23.
- Goodson JM, Cugini MA, Kent RL, Armitage GA, Cobb CM, Fine D, Fritz ME, Green E, Imoberdorf MJ, Killoy WJ, et al. Multicenter evaluation of tetracycline fiber therapy: II. Clinical response. *J Periodontol Res* 1991;26:371-9.
- Graves RC, Disney JA, Stamm JW. Comparative effectiveness of flossing and brushing in reducing interproximal bleeding. *J Periodontol* 1989;60(5):243-7.
- Gröndahl HG. Radiographic assessment of caries and caries progression. In: Anusavice KJ, editor. *Quality evaluation of dental restoration: criteria for placement and replacement*. Chicago: Quintessence Publishing Co.; 1989. p. 151-67.
- Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. *Ann Periodontol* 1998 Jul;3(1):51-61.
- Grossi SG, Skrepcinski FB, DeCoro T, Zambon JJ, Cummin SD, Genco RJ. Response to periodontal therapy in diabetics and smokers. *J Periodontol* 1996;67:1094-102.
- Guckes AD, Roberts MW, McCarthy GR. Pattern of permanent teeth present in individuals with ectodermal dysplasia and severe hypodontia suggests treatment with dental implants. *Pediatr Dent* 1998;20:278-80.
- Haffajee AD, Socransky SS. Microbial etiological agents of destructive periodontal diseases. *Periodontol* 2000 1994;5:78-111.
- Haffajee AD, Socransky SS, Goodson JM. Clinical parameters as predictors of destructive periodontal disease activity. *J Clin Periodontol* 1983;10:257-65.
- Halazonetis TD, Haffajee AD, Socransky SS. Relationship of clinical parameters to attachment loss in subsets of subjects with destructive periodontal diseases. *J Clin Periodontol* 1989;16:563-8.
- Hancock EB. Prevention. *Ann Periodontol* 1996;1:223-49.
- Head JA. A study of saliva and its action on tooth enamel in reference to its hardening and softening. *JAMA* 1912;59:2118-22.
- Hillman JD, Brooks TA, Michalek SM, Harmon CC, Snoep JL, van Der Weijden CC. Construction and characterization of an effector strain of *Streptococcus*

- mutans* for replacement therapy of dental caries. *Infect Immun* 2000;68:543-9.
- Ismail AI. Clinical diagnosis of precavitated carious lesions. *Community Dent Oral Epidemiol* 1997;25:13-23.
- Ismail AI, Bader JD, Kamerow DB. Systematic reviews and the practice of evidence-based dentistry: professional and policy implications. *J Am Coll Dent* 1999;66:5-12.
- Jeffcoat MK. Imaging techniques for the periodontium. In: Wilson TG, Kornman K, Newman MG, editors. *Advances in periodontics*. Chicago: Quintessence; 1992.
- Jeffcoat MK, Wang IC, Reddy MS. Radiographic diagnosis in periodontitis. *Periodontol* 2000 1995;7:54-68.
- Jeffcoat MK, Reddy MS, Magnusson I, Johnson B, Meredith MP, Cavanaugh PF Jr, Gerlach RW. Efficacy of quantitative digital subtraction radiography using radiographs exposed in a multicenter trial. *J Periodontol Res* 1996;31(3):157-60.
- Jeffcoat MK, McGuire M, Newman MG. Evidence-based periodontal treatment: highlights from the 1996 World Workshop in Periodontics. *J Am Dent Assoc* 1997;128:713-24.
- Jeffcoat MK, Bray K, Ciancio SG, Dentino AR, Fine DH, Gordon JM, Gunsolley JC, Killoy WJ, Lowenguth RA, Magnusson NI, et al. Adjunctive use of a subgingival controlled-release chlorhexidine chip reduces probing pocket depth and improves attachment level compared with scaling and root planing alone. *J Periodontol* 1998 Sep;69(9):989-97.
- Johnson NW. Orofacial neoplasms: global epidemiology, risk factors and recommendations for research. *Int Dent J* 1991;41(6):365-75.
- Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP, Dyer JK. Long-term evaluation of periodontal therapy: I. Response to 4 therapeutic modalities. *J Periodontol* 1996;67:93-102.
- Kantorowitz Z, Featherstone JD, Fried D. Caries prevention by CO₂ laser treatment: dependency on the number of pulses used. *J Am Dent Assoc* 1998;129:585-91.
- Kaste LM, Drury TF, Horowitz AM, Beltran E. An evaluation of NHANES III estimates of early childhood caries. *J Public Health Dent* 1999;59:198-200.
- Katz S. The use of fluoride and chlorhexidine for the prevention of radiation caries. *J Am Dent Assoc* 1982;104:164-70.
- Kearns G, Sharma A, Perrott D, Schmidt B, Kaban L, Vargarik K. Placement of endosseous implants in children and adolescents with hereditary ectodermal dysplasia. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;88(1):5-10.
- Ketley CE, Holt RD. Visual and radiographic diagnosis of occlusal caries in first permanent molars and in second primary molars. *Br Dent J* 1993;174:364-70.
- Kidd EA. Caries diagnosis within restored teeth. *Adv Den Res* 1990 Jun;4:10-3.
- Knowles JW, Burgett FG, Nissle RK, Shick RA, Morrison EC, Ramfjord SP. Results of periodontal treatment related to pocket depth and attachment level. Eight years. *J Periodontol* 1979;50:225-33.
- Kohler B, Bratthall D. Intra-familial levels of *Streptococcus mutans* and some aspects of the bacterial transmission. *Scand J Dent Res* 1978;86:35-42.
- Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, Wilson TJ Jr, Higginbottom FL, Duff GW. The interleukin-1 genotype as a severity factor in adult periodontal disease. *J Clin Periodontol* 1997;24(1):72-7.
- Koulourides T, Ceuto H, Pigman W. Rehardening of softened enamel surfaces of human teeth by solutions of calcium phosphates. *Nature* 1961;189:226-7.
- Lamster IB, Smith QT, Celenti RS, Singer RE, Grbic JT. Development of a risk profile for periodontal disease: microbial and host response factors. *J Periodontol* 1994;65:511-20.
- Lamster IB, Holmes LG, Gross KB, Oshrain RL, Cohen DW, Rose LF, Peters LM, Pope MR. The relationship of β -glucuronidase activity in crevicular fluid to probing attachment loss in patients with adult periodontitis. Findings from a multicenter study. *J Clin Periodontol* 1995;22(1):36-44.
- Lang NP, Joss A, Orsanic T, Gusberti FA, Siegrist BE. Bleeding on probing. A predictor for the progression of periodontal disease? *J Clin Periodontol* 1986;13:590-6.
- Lang WP, Farghaly MM, Ronis DL. The relation of preventive dental behaviors to periodontal health status. *J Clin Periodontol* 1994;21(3):194-8.
- Larsen MJ, Fejerskov O. Chemical and structural challenges in remineralization of dental enamel lesions. *Scand J Dent Res* 1987;97:285-96.
- Leake JL, Main PA, Sabbah W. A system of diagnostic codes for dental health care. *J Public Health Dent* 1999;59(3):162-70.
- Lee W, Aitken S, Sodek J, McCulloch CA. Evidence of a direct relationship between neutrophil collagenase activity and periodontal tissue destruction in vivo: role of active enzyme in human periodontitis. *J Periodontol Res* 1995 Jan;30(1):23-33.
- Levy SM. Review of the fluoride exposures and ingestion. *Community Dent Oral Epidemiol* 1994 Jun;22(3):173-80.
- Lewis DW, Ismail AI. Periodic health examination, 1995 update: 2. Prevention of dental caries. Canadian Task Force on the Periodic Health Examination. *Can Med Assoc J* 1995;152:836-46.
- Li Y, Caufield PW. The fidelity of initial acquisition of *mutans streptococci* by infants from their mothers. *J Dent Res* 1995 Feb;74(2):681-5.
- Linton JL. Quantitative measurements of remineralization of incipient caries. *Am J Orthod Dentofac Orthop* 1996;110:590-7.
- Lippman SM, Benner SE, Hong WK. Chemoprevention. *Cancer* 1993;984-90.

- Løe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand* 1963; 21:533-51.
- Loesche WJ. The identification of bacteria associated with periodontal disease and dental caries by enzymatic methods. *Oral Microbiol Immunol* 1986;1: 65-70.
- Loesche WJ, Giordona J, Hujoeel PP. The utility of the BANA test for monitoring anaerobic infections due to spirochetes (*Treponema denticola*) in periodontal disease. *J Dent Res* 1990 Oct;69(10):1696-702.
- Lussi A. Comparison of different methods for the diagnosis of fissure caries without cavitation. *Caries Res* 1993;27:409-16.
- Lussi A, Firestone A, Schoenberg V, Hotz P, Stich H. In vivo diagnosis of fissure caries using a new electrical resistance monitor. *Caries Res* 1995;29:81-7.
- Ma JK, Hikmat BY, Wycoff K, Vine ND, Chargelegue D, Yu L, Hein MB, Lehner T. Characterization of a recombinant plant monoclonal secretory antibody and preventive immunotherapy in humans. *Nat Med* 1998 May;4(5):601-6.
- Mandel ID. Calculus update: prevalence, pathogenicity and prevention. *J Am Dent Assoc* 1995 May;126(5): 573-80.
- Masada MP, Persson R, Kenney JS, Lee SW, Page RC, Allison AC. Measurement of interleukin-1 alpha and -1 beta in gingival crevicular fluid: implications for the pathogenesis of periodontal disease. *J Periodontol Res* 1990;25:156-63.
- Matsuda Y, Byrd V, et al. A comparison of the accuracy of a direct digital radiography and conventional film based subtraction system in detecting bony change. In: Farman AG, editor. *Advances in maxillofacial imaging: IADMFRCMI'97 selected proceedings of the 11th Congress of the International Association of Dentomaxillofacial Radiology and the 3rd International Congress and Exposition on Computed Maxillofacial Imaging*, Louisville, KY, 1977 Jun 21-27. New York: Elsevier; 1997. p. 56-66.
- McLaughlin JK, Gridley G, Block G, Winn DM, Preston-Martin S, Schoenberg JB, Greenberg RS, Stemhagen A, Austin DF, Ershow AG, et al. Dietary factors in oral and pharyngeal cancer. *J Natl Cancer Inst* 1988;80(15):1237-43.
- Mealey BL. Periodontal implications: medically compromised patients. *Ann Periodontol* 1996;1:256-321.
- Mellonig JT. Donor selection, testing, and inactivation of the HIV virus in freeze-dried bone allografts. *Pract Periodontics Aesthetic Dent* 1995;7(6):13-22.
- Mertz-Fairhurst EJ, Curtis JS, Ergle JW, Rueggeberg FA, Adair SM. Ultraconservative and cariostatic sealed restorations: results at year 10. *J Am Dent Assoc* 1998;129:55-66.
- Meyerowitz C, Featherstone JD, Billings RJ. Use of an intra-oral model to evaluate 0.05% sodium fluoride mouthrinse in radiation-induced hyposalivation. *J Dent Res* 1991;70(5):894-8.
- Mjör IA. Frequency of secondary caries at various anatomical locations. *Oper Dent* 1985;10:88-92.
- Mohl ND, Lund JP, Widmer CG, McCall WD Jr. Devices for the diagnosis and treatment of temporomandibular disorders. *J Prosthet Dent* 1990;63: 332-6.
- Moncla BJ, Strockbine L, Braham P, Karlinsey J, Roberts MC. The use of whole-cell DNA probes for the identification of *Bacteroides intermedius* isolates in a dot blot assay. *J Dent Res* 1988;67:1267-70.
- Moore WE. Microbiology of periodontal disease. *J Periodontol Res* 1987;22:335-41.
- Moss SJ, editor. *Fluorides: an update for dental practice*. The American Academy of Pedodontics. New York: Medcom; 1976.
- Mount GJ, Hume WR. A revised classification of carious lesions by site and size. *Quintessence Int* 1997;28: 301-3.
- Mount GJ, Hume WR. *Preservation and restoration of tooth structure*. London: Mosby; 1998.
- Murphy GP, Lawrence W Jr, Lenhard RE Jr, editors. *American Cancer Society textbook of clinical oncology*. Atlanta: American Cancer Society; 1995. p. 180.
- National Cancer Institute (NCI). *Cigars. Health effects and trends*. Bethesda (MD): National Institutes of Health; 1998. p. 232. NIH Pub no. 98-4302.
- National Institutes of Health (NIH). *Technology Assessment Conference Statement, 1996 Apr 29-May 1. Management of temporomandibular disorders*. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, Office of Medical Applications of Research; 1996. p. 1-31.
- Newbrun E, Leverett D. Risk assessment dental caries working group summary statement. In: Bader JD, editor. *Risk assessment in dentistry: proceedings of a conference, June 2-3, 1989, Chapel Hill, North Carolina*. Chapel Hill: University of North Carolina School of Dentistry; 1990. p. 304.
- Nishida M, Grossi SG, Dunford RG, Ho AW, Trevisan M, Genco RJ. Calcium and risk for periodontal disease. *Periodontol* 2000;70(7):in press.
- Ockene JK, Kristellar J, Goldberg R. Increasing the efficacy of physician-delivered smoking interventions: a randomized clinical trial. *J Gen Intern Med* 1991;6: 1-8.
- Offenbacher S, Odle BM, Van Dyke TE. The use of crevicular fluid prostaglandin E₂ levels as a predictor of periodontal attachment loss. *J Periodontol Res* 1986;21:101-12.
- Ohtoshi T, Yamauchi K, Tadokoro K, Miyachi S, Suzuki S, Miyamoto T, Muranaka M. IgE antibody-mediated shock reaction caused by topical application of chlorhexidine. *Clin Allergy* 1986;16:155-61.
- Okamoto H, Yoneyama T, Lindhe J, Haffajee A, Socransky S. Methods of evaluating periodontal disease data in epidemiological research. *J Clin Periodontol* 1988;15:430-9.
- Okano M, Nomura M, Hata S, Okada N, Sato K, Kitano Y, Tashiro M, Yoshimoto Y, Hama R, Aoki T. Anaphylactic symptoms due to chlorhexidine gluconate. *Arch Dermatol* 1989;125(1):50-2.

- Page RC. Periodontal diseases: a new paradigm. *J Dent Educ* 1998;62:812-21.
- Page RC, Beck JD. Risk assessment for periodontal diseases. *Int Dent J* 1997;47:61-87.
- Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. *Periodontol* 2000 1997;14:9-11.
- Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest* 1976 Mar;34(3):235-49.
- Palcanis KG. Surgical pocket therapy. *Ann Periodontol* 1996;1:589-617.
- Palcanis KG, Larjava IK, Wells BR, Suggs KA, Landis JR, Chadwick DE, Jeffcoat MK. Elastase as an indicator of periodontal disease progression. *J Periodontol* 1992;63:237-42.
- Papapanou PN. Risk assessments in the diagnosis and treatment of periodontal diseases. *J Dent Educ* 1998;62:822-39.
- Patterson IC, Eveson JW, Prime SS. Molecular changes in oral cancer may reflect aetiology and ethnic origin. *Eur J Cancer* 1996;32B:150-3.
- Penning C, van Amerongen JP, Seef RE, ten Cate JM. Validity of probing for fissure caries diagnosis. *Caries Res* 1992;26:445-9.
- Persson GR, Page RC. Diagnostic characteristics of crevicular fluid aspartate aminotransferase (AST) levels associated with periodontal disease activity. *J Clin Periodontol* 1992;19:43-8.
- Pihlstrom BL, McHugh RB, Oliphant TH, Ortiz-Campos C. Comparison of surgical and non-surgical treatment of periodontal disease. *J Clin Periodontol* 1983;10:524-41.
- Pitts NB. Risk assessment and caries prediction. *J Dent Educ* 1998;62:762-70.
- Pitts NB, Longbottom C. Preventive care advised (PCA)/Operative care advised (OCA)—categorising caries by the management option. *Community Dent Oral Epidemiol* 1995;23:55-9.
- Pogoda JM, Preston-Martin S. Solar radiation, lip protection, and lip cancer in Los Angeles County women (California, United States). *Cancer Causes Control* 1996;7:458-63.
- Powell LV. Caries prediction: a review of the literature. *Community Dent Oral Epidemiol* 1998;26:361-71.
- Qvist V, Laurberg L, Poulsen A, Teglers PT. Longevity and cariostatic effects of everyday conventional glass-ionomer and amalgam restorations in primary teeth: three year results. *J Dent Res* 1997;76(7):1387-96.
- Ramfjord SP, Caffesse RG, Morrison EC, Hill RW, Kerry GJ, Appleberry EA, Nissle RP, Stults DL. Four modalities of periodontal treatment compared over 5 years. *J Clin Periodontol* 1987;14(8):445-52.
- Ranney RR. Classification of periodontal disease. *Periodontol* 2000 1993;2:13-25.
- Rao VM. Imaging of the temporomandibular joint. *Semin Ultrasound CT MR* 1995;16(6):513-26.
- Rao VM, Farole A, Karasick D. Temporomandibular joint dysfunction; correlation of MR imaging, arthrography and arthroscopy. *Radiology* 1990;174:663-7.
- Reddi H. Bone morphogenetic proteins. *Adv Dent Res* 1995;9(3 Suppl):13.
- Ries LAG, Kosary CL, Hankey BF, Miller BA, Edwards BK, editors. *SEER Cancer Statistics Review, 1973-1996*. Bethesda (MD): National Cancer Institute; 1999.
- Ronis DL, Lang WP, Farghaly MM, Passow E. Tooth brushing, flossing and preventive dental visits by Detroit-area residents in relation to demographic and socioeconomic factors. *J Public Health Dent* 1993 Summer;53(3):138-45.
- Rose LF, Steinberg BJ. Clinical history and laboratory tests. In: Rose LF, Genco RJ, Cohen DW, Mealey BL, editors. *Periodontal medicine*. Hamilton (Ontario): B.C. Decker Inc.; 2000.
- Rossomando EF, Kennedy JE, Hadjimichael J. Tumour necrosis factor alpha in gingival crevicular fluid as a possible indicator of periodontal disease in humans. *Arch Oral Biol* 1990;35:431-4.
- Russell MW, Hajishengallis G, Childers NK, Michalek SM. Secretory immunity in defense against cariogenic mutans streptococci. *Caries Res* 1995;33:4-15.
- Sackett DL, Rosenberg WM, Muir Gray JA, Haynes RB, Richardson WS. Evidence based medicine: what it is and what it isn't. *BMJ* 1996;312:71-2.
- Savitt ED, Strzempko MN, Vaccaro KK, Peros WJ, French CK. Comparison of cultural methods and DNA probe analyses for detection of *Actinobacillus actinomycetemcomitans*, *Bacteroides gingivalis*, and *Bacteroides intermedius* in subgingival plaque samples. *J Periodontol* 1988;59:431-8.
- Savitt ED, Keville MW, Peros WJ. DNA probes in the diagnosis of periodontal microorganisms. *Arch Oral Biol* 1990;35(Suppl):153S-9S.
- Schaeken MJ, Keltjens HM, van der Hoeven JS. Effects of fluoride and chlorhexidine on the microflora of dental root surfaces and progression of root-surface caries. *J Dent Res* 1991;70(2):150-3.
- Schroeder HE, Munzel-Pedrazzoli S, Page R. Correlated morphometric and biochemical analysis of gingival tissue in early chronic gingivitis in man. *Arch Oral Biol* 1973;18:899-923.
- Schwartz SM, Daling JR, Doody DR, Wipf GC, Carter JJ, Madeleine MM, Mao EJ, Fitzgibbons ED, Huang S, Beckmann AM, et al. Oral cancer risk in relation to sexual history and evidence of human papillomavirus infection. *J Natl Cancer Inst* 1998;90:1626-36.
- Sciubba JJ. Improving detection of precancerous and cancerous oral lesions: computer-assisted analysis of the oral brush biopsy. *J Am Dent Assoc* 1999;130:1445-58.
- Scully C. Oral precancer: preventive and medical approaches to management. *Oral Oncol Euro J Cancer* 1995;31B:16-26.
- Shah JP, Lydiatt W. Treatment of cancer of the head and neck. *CA Cancer J Clin* 1995;45:352-68.

- Shin DM, Lee JS, Lippman SM, Lee JJ, Tu ZN, Choi G, Heyne K, Shin HJ, Ro JY, Goepfert H, et al. P53 expressions: predicting recurrence and second primary tumors in head and neck squamous cell carcinoma. *J Natl Cancer Inst* 1996;88(8):519-29.
- Shugars D, Bader J. Practice parameters in dentistry: where do we stand? *J Am Dent Assoc* 1995;126:1134-43.
- Sidransky D. Nucleic acid-based methods for the detection of cancer. *Science* 1997;278:1054-9.
- Silverman S Jr. Oral cancer. Atlanta: American Cancer Society; 1990.
- Silverstone LM, Wefel JS, Zimmerman BF, Clarkson BH, Featherstone MJ. Remineralization of natural and artificial lesions in human dental enamel in vitro. *Caries Res* 1981;15:138-57.
- Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: current concepts. *J Periodontol* 1992;63:322-31.
- Spiekermann H, Jansen V, Richter EJ. A 10-year follow-up study of IMZ and TPS implants in the edentulous mandible using bar-retained overdentures. *Int J Oral Maxillofac Implants* 1995;10:231-43.
- Spitz MR, Fueger JJ, Goepfert H, Hong WK, Newell GR. Squamous cell carcinoma of the upper digestive tract. A case comparison analysis. *Cancer* 1988;61:203-8.
- Stevens VJ, Severson H, Lichtenstein E, Little SJ, Leben J. Making the most of a teachable moment; a smokeless-tobacco cessation intervention in the dental office. *Am J Public Health* 1995;85(2):231-5.
- Stookey GK, DePaola PF, Featherstone JD, Fejerskov O, Moller IJ, Rotberg S, Stephen KW, Wefel JS. A critical review of the relative anticaries efficacy of sodium fluoride and sodium monofluorophosphate dentifrices. *Caries Res* 1993;27(4):337-60.
- Sugerman PB, Shillitoe EJ. The high risk human papillomaviruses and oral cancer: evidence for and against a causal relationship. *Oral Dis* 1997;3:130-47.
- Tavender E. The Cochrane collaboration and the oral health review group. Evidence-based dentistry; 1999 Jun.
- Tenovuo J, Häkkinen P, Paunio P, Emilson CG. Effects of chlorhexidine-fluoride gel treatments in mothers on the establishment of mutans streptococci in primary teeth and development of dental caries in children. *Caries Res* 1992;26:275-80.
- Thylstrup A, Vinther D, Christiansen J. Treatment time studies during 1 year in a Danish public child dental health service. *Caries Res* 1995;29:292.
- Tolarova MM, Harris J. Reduced recurrence of orofacial clefts after peri-conceptional supplementation with high dose folic acid and multivitamins. *Teratology* 1995;51:71-8.
- Travell J, Simons D. Myofascial pain and dysfunction: the trigger point manual. The upper extremities. Baltimore: Williams and Wilkins; 1983.
- Tsai CC, Ho YP, Chen CC. Levels of interleukin-1 and interleukin-8 in gingival crevicular fluids in adult periodontitis. *J Periodontol* 1995;66:852-9.
- Urist MR. Bone formation by autoinduction. *Science* 1965;150:893-9.
- Urist MR, Iwata H. Preservation and biodegradation of the morphogenetic property of bone matrix. *J Theor Biol* 1973;38:155-67.
- U.S. Department of Health and Human Services (USDHHS). Detecting oral cancer: a guide for health care professionals. Washington: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institute of Dental Research; 1998 Jan.
- Vokes EE, Athanasiadis I. Chemotherapy for squamous cell carcinoma of head and neck: the future is now. *Ann Oncol* 1996;7:15-29.
- Vokes EE, Weichselbaum RR, Lipman SM. Head and neck cancer. *N Engl J Med* 1993;328:184-94.
- Walsh M, Heckman B, Leggott P, Armitage G, Robertson PB. Comparison of manual and power toothbrushing with and without adjunctive oral irrigation, for controlling plaque and gingivitis. *J Clin Periodontol* 1989;16(7):419-27.
- Werler MM, Louik C, Mitchell AA. Achieving a public health recommendation for preventing neural tube defects with folic acid. *Am J Public Health* 1999;89:1637-40.
- White DJ. Reactivity of fluoride dentifrices with artificial caries: II. Effects on subsurface lesions, F uptake, surface hardening, and remineralization. *Caries Res* 1988;22:27-36.
- Wilson DM, Taylor DW, Gilbert JR, Best JA, Lindsay EA, Willms DG, Singer J. A randomized trial of a family physician intervention for smoking cessation. *JAMA* 1988;260(11):1570-4.
- Winn DM, Blot WJ. Second cancer following cancer of the buccal cavity and pharynx in Connecticut, 1935-82. In: Boice JD, et al. Multiple primary cancers in Connecticut and Denmark. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute; 1985.
- Winn DM, Blot WJ, McLaughlin JK, Austin DF, Greenberg RS, Preston-Martin S, Schoenberg JB, Fraumeni JF Jr. Mouthwash use and oral conditions in the risk of oral and pharyngeal cancer. *Cancer Res* 1991;51(11):3044-7.
- Zambon JJ, Bochacki V, Genco RJ. Immunological assays for putative periodontal pathogens. *Oral Microbiol Immunol* 1986;1:39-44.
- Zarb GA. Temporomandibular joint and masticatory muscle disorders. 2nd ed. Copenhagen: Munksgaard; 1994.
- Zheng Z-F, et al. Marijuana use and increased risk of squamous cell carcinoma of the head and neck. *Cancer Epidemiol Biomark Prev* 1999;8:1071-8.

Provision of Oral Health Care

Achieving optimal oral, dental, and craniofacial health requires a commitment to self-care and preventive behaviors as well as the receipt of appropriate professional care. Community-wide approaches to support oral health and the role of professional services are covered in Chapters 7 and 8, respectively. Although the services provided by dental practitioners are the first that come to mind when people consider the nation's resources to address the diseases and disorders that affect the craniofacial complex, the dental component is augmented by two other components—medicine and public health. These three do not constitute a single system of care, but serve as individual components variously involved in the promotion of health and the provision of services to individuals and families, communities, and the population at large. The linkages and overlaps among the components mirror those between oral and general health described elsewhere in this report (e.g., Chapters 2, 3, 5, and 6), and may also play a role in the disparities noted in Chapter 4.

As has been noted in previous chapters, data regarding the contributions to oral health care made by the medical and public health components are not nearly as available as those that describe the contributions made by dental practitioners. Most of this care is provided by dentists in private practice. Expenditures for their services represented over 96 percent of the estimated \$53.8 billion spent on dental care in 1998, or 4.7 percent of the \$1.1 trillion spent on all health care in the United States that year (HCFA 2000b). Although they surely undercount the contributions of the medical and public health components, these expenditures indicate the burden that oral diseases and conditions place on the American people, as well as their willingness to invest in the prevention, treatment, and rehabilitation of oral conditions—a reflection of the value they place on oral health.

There have been notable achievements in oral health over the years, among them the dramatic and continuing reduction in the prevalence of dental caries in sizable population groups (see Chapter 4). This has led to an impressive decline in tooth loss, with the result that the majority of Americans can now expect to retain their natural teeth over their lifetimes. At the same time, all three components have participated in the revolutions in biomedical and behavioral sciences and technology that have deepened our understanding of the biological, environmental, behavioral, and genetic origins of many oral, dental, and craniofacial diseases. Americans today can benefit from oral health services that are among the best in the world. Moreover, as new and improved preventive, diagnostic, and treatment measures emerge (see Chapter 8), they create further opportunities for improving the nation's oral health.

COMPONENTS OF PROFESSIONAL CARE

The dental, medical, and public health contributions to oral health differ dramatically in their size, focus, financing, and resources. Following is a brief description of each component and their areas of overlap.

The Dental Component

Comprehensive oral health care in America is largely supplied by a private dental care system composed of dentists, dental hygienists, dental assistants, laboratory technicians, and other professional staff in independent dental offices. The estimated numbers of active dental personnel are presented in Table 9.1. Of the 156,500 professionally active dentists in the United States in 1997, 91.7 percent were in private practice (ADA 1998a). Women constitute 14.4 percent of the total, and minorities 11.1 percent (ADA

1998a). Other professionals, who are educators, biomedical and behavioral science researchers, technicians, manufacturers of dental products, and administrators, complement this workforce.

In contrast to medicine, where only 40 percent of physicians were in primary care practices in 1990, approximately 80 percent of dentists are general practitioners (ADA 1998a). The remainder qualify as specialists in one or more of the nine disciplines formally recognized by the American Dental Association: orthodontics, oral and maxillofacial surgery, oral and maxillofacial radiology, periodontics, pediatric dentistry, endodontics, prosthodontics, dental public health, and oral and maxillofacial pathology. More than half of these specialists are orthodontists or oral surgeons (ADA 1998a). A small number of dental practitioners focus on special interest areas such as anesthesiology or oral medicine. There has been little change in the approximately

4 to 1 ratio of general dentists to specialists in the past 10 years.

New technologies and changing patterns of disease are broadening the scope of dental practice. The average general practitioner and staff now engage in more preventive services than in years past (ADA 1990). A reduction in the number of amalgam and resin restorations per patient per year from 1980 to 1995 has also been noted (Eklund et al. 1997). Although dentists perform fewer extractions and restorations, preserving the teeth of an aging population has increased the need for crowns and periodontal treatment.

Since oral health is an integral part of total health, most dentists provide primary care services to their patients. In addition to educating patients on oral health care, dentists and their staff may counsel patients on tobacco and other substance use and cessation, nutrition, and dietary practices. In addition, information that dentists obtain from a patient's history and from screening and diagnostic tests may suggest the presence of systemic disease, warranting a referral of the patient to other health care professionals.

Dental services are provided in a practice model that is different from that used by the medical profession. Most private dental practices consist of one dentist (68.7 percent) or two dentists (19.6 percent). The remaining practices (11.7 percent) are group practices of three or more dentists who share expenses and revenues. This distribution of dentists by practice size, along with the number of hours worked per week, has remained remarkably constant over the

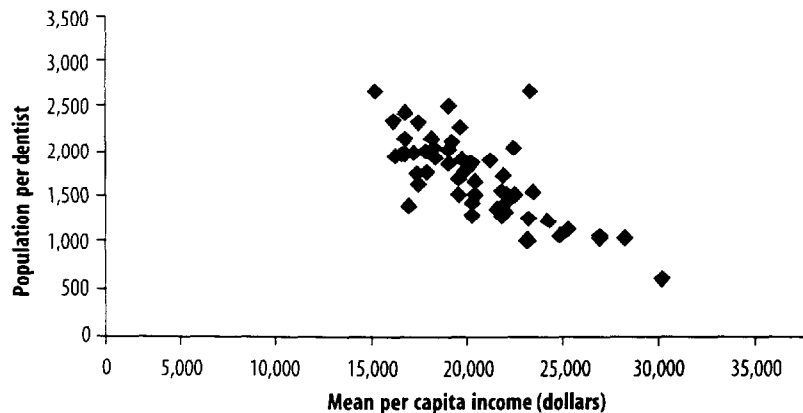
years (ADA 1998a). The size, number, and location of dental practices are important determinants of availability of care and accessibility to services, as well as of the unit cost of care. Figure 9.1 shows the association between the availability of dentists and state mean per capita income (Burt and Eklund 1999). Dental care is also provided in dental schools and public health clinics, hospitals, nursing homes, and other institutional settings. These are sometimes the principal source of oral health care for communities and special population groups with limited access to health care.

TABLE 9.1
Estimated numbers of active oral health personnel, United States, selected years

	1980	1990	1996	1997
Dentists	121,900	147,500	154,900	156,500
Dental hygienists	54,000	81,000	94,000	NA
Dental assistants	156,000	201,000	212,000	NA
Dental laboratory technicians	43,000	50,000	53,000	NA

Note: NA = not available.
Source: HRSA 1999.

FIGURE 9.1
The association between state mean per capita income and the population-to-dentist ratio, by individual state, United States, 1995



Source: ADA 1997a. Reprinted by permission of American Dental Association, Survey Center. Distribution of dentists by region and state, 1995. Copyright 1997 by American Dental Association, Survey Center.

The Medical Component

In the context of oral, dental, and craniofacial health care, the medical component includes dentists, physicians, nurses, and allied health professionals whose services are provided through hospitals, nursing homes, ambulatory care facilities, and health professional offices. Data on the nature and extent of such oral health services, as well as on the number of nondental professionals who supply them, are limited. For some conditions—particularly developmental anomalies, injuries, infectious diseases, pain syndromes, and oral and pharyngeal cancers—the medical component provides comprehensive care, often working in tandem with dental specialists and general practitioners. For example, physicians and oral and maxillofacial surgeons may plan treatments together and operate on individuals born with cleft lip/palate to repair the clefts as the children age. Orthodontists, pediatric dentists, prosthodontists, and other dental specialists, speech and hearing therapists, plastic surgeons, neurologists, radiologists, nutritionists, psychologists, other health professionals, and social workers are also part of the craniofacial team.

Collaboration and coordination between physicians and dentists are needed to provide integrated medical and oral health care for cardiac patients and those undergoing chemo- and radiation therapy or implant and organ transplant procedures. Nondental health care personnel in long-term and geriatric care facilities may be the principal sources of oral health care given to residents.

Although most hospitals have dental personnel on staff to handle emergency situations, emergency room physicians and other hospital personnel are often called on to initiate treatment of acute oral-facial injury or pain of dental origin, with referrals to dentists for follow-up. Also, patients with chronic oral-facial pain conditions are sometimes treated by family practice, internal medicine, or neurology physicians, sometimes with referral to dental or other medical specialists.

The Public Health Component

Federal, state, and local government agencies support a range of oral health activities and programs benefiting individuals and communities. This component includes health professionals and administrators who participate in publicly funded care delivery programs, research, disease surveillance, policy development, and implementation of programs aimed at preventing disease and promoting health.

Federal agencies under the jurisdiction of the U.S. Department of Health and Human Services (USDHHS) and the U.S. Departments of Defense, Veterans Affairs, Agriculture, Education, Transportation, and Justice, among others, serve public health needs in diverse ways. These agencies may include units or programs specifically dedicated to oral health, as well as components that collect, organize, or make available information or services related to oral health as part of general health programs. For example, the National Institutes of Health (NIH) is the primary federal agency supporting biomedical and behavioral research and research training. Assurance of the safety of foods, cosmetics, drugs, and devices is provided through the regulatory authorities of the Food and Drug Administration (FDA). The Centers for Disease Control and Prevention (CDC) focuses on state-based programs for monitoring and preventing disease and, through the National Center for Health Statistics, orchestrates the collection of nationally representative health information and population data. The Agency for Healthcare Research and Quality (AHRQ) uses evidence-based practice centers to evaluate literature relevant to the management of diseases and conditions, conducts national expenditure and care utilization surveys, and supports research directed at understanding health care systems. The Medicaid, Medicare, and newly enacted State Children's Health Insurance Program (SCHIP) programs are directed by the Health Care Financing Administration (HCFA), which funds a variety of care services prescribed by law or regulation.

Several federal agencies provide direct services to specific, often disadvantaged populations or to military personnel and their dependents. The U.S. Departments of Defense, Transportation, and Veterans Affairs, the U.S. Department of Justice's Bureau of Prisons, and the USDHHS's Indian Health Service (IHS) and Health Resources and Services Administration (HRSA) provide oral health care directly to selected populations. Oral health education also is provided through the U.S. Department of Agriculture's Women, Infants and Children (WIC) program. In addition, HRSA provides funds for health professional education and administers the Ryan White Comprehensive AIDS Resources Emergency Act. States, counties, and cities also support dental programs for disadvantaged populations under federally mandated and funded Maternal and Children's Health Programs or as part of Medicaid or the State Children's Health Insurance Program. They may also provide direct support through tax

revenues. The Head Start programs of the Administration for Children and Families (ACF) provide health education services and in some cases may pay for oral care services for enrolled low-income children 5 years old and younger.

Many organizations have activities that could be used to promote oral health, given appropriate collaboration. In this regard, studies of the federal oral health infrastructure have emphasized the need for federal programs to maximize partnerships within and beyond the federal government. A 1989 report recommended increasing the capacity of USDHHS agencies to direct dental expertise to programs that can affect oral health or dental care, and establishing a mechanism for coordinating programs and sharing expertise across agencies. The report recommended that a national advisory committee be established to assess opportunities, accomplishments, and needs (USDHHS 1989). A 1994 report determined that the collection and analysis of data related to oral health and dental care had not been maintained at a level consistent with analogous general health and health care data used for public program planning, development, and evaluation (SysteMetrics, Inc. 1994). Despite some progress, many of the recommendations of these reports have not been implemented; thus many of the deficiencies noted have not been fully addressed.

Table 9.2 presents the scope of services and activities supported by USDHHS agencies. It identifies each agency's principal activities in terms of the "essential public health services" conducted at federal, state, and local levels. The approximate fiscal year 2000 funding levels provide a sense of how the proportion of oral health programs varies across agencies.

Local public health departments, community health centers, nongovernmental organizations, dental schools, and volunteer groups are examples of entities that implement oral health programs in association with government agencies and the private sector. These collaborations are enhanced by state oral health programs as they direct and integrate public health services. Not every state health agency has an oral health program, however. Further, not all state oral health programs have sufficient resources to address oral health needs. For example, although 31 states and five territories currently have full-time state dental directors, in 20 states (including the District of Columbia), the state dental director positions are part time or vacant. Additionally, 21 states, with 67 million people, have two or fewer full-time equivalents staffing a state oral health program. In 25 states, fewer than 10 percent of the counties have oral health programs in their local health departments (ASTDD 1999).

The Association of State and Territorial Dental Directors (ASTDD) recently assessed the resources needed to achieve the objectives in Healthy People 2010. The study focused on the gaps in infrastructure and capacity of state oral health programs. Infrastructure consists of the systems, people, relationships, and resources that would enable state oral health programs to perform public health functions. Capacity describes the expertise and competence needed to enable the implementation of strategies. Box 7.3 describes the essential public health services for oral health in the areas of assessment, policy development, and assurance as noted in ASTDD's Guidelines for State and Territorial Oral Health Programs. In particular, states have high needs for oral health surveillance systems and staff with epi-

TABLE 9.2
Scope of essential public health services supported by agencies of the U.S. Department of Health and Human Services

	AHRQ	CDC	FDA	HRSA	IHS	NIH	HCFA	ACF
Monitor health status to identify and solve community health problems	X	X			X	X		
Diagnose and investigate community health problems		X		X	X			
Educate and empower people about health issues	X	X	X	X	X	X	X	X
Develop policies and plans that support individual and community efforts		X		X	X			X
Enforce laws and regulations that protect health and ensure safety			X				X	
Link people to personal health services; ensure provision of care when otherwise unavailable				X	X		X	X
Ensure a competent public health and personal health care workforce	X	X		X	X	X		
Evaluate effectiveness, accessibility, and quality of personal and population-based services	X	X		X	X		X	X
Conduct research for new insights and innovative solutions to health problems	X	X	X	X		X		
Oral health component (FY 2000) (\$ millions)	<1	<10	<3	<150	<80	<250	2000	<10
Total agency budget (FY 2000) (\$ billions)	>0.2	3.1	1.4	4.2	2.8	16	343	38
Oral health as proportion of agency budget	<0.5%	0.3%	0.2%	0.3%	3%	1.5%	<0.2%	<0.1%

Sources: Data from PHS Oral Health Coordinating Committee, personal communication, 2000, Public Health Functions Steering Committee 2000.

demographic and other public health expertise (ASTDD 2000). Similar gaps occur in many local public health departments that lack adequate oral health programs or appropriately trained personnel (USDHHS 2000).

Public health agencies at all levels have identified disparities in oral health and access to care, in terms of both population subgroups and geographical areas. In 1998, there were 1,036 dental Health Professional Shortage Areas (HPSAs), which required 3,984 dentists. Of 686 consolidated Community Health Center grantees, 385 (or 56 percent) provide dental services (J. Anderson, HRSA, personal communication, 1999).

Community Health Centers provide preventive and basic dental care to 1.2 million patients nationwide (HRSA 1998). Health Centers are located in medically underserved urban and rural areas and target low-income, migrant, homeless, and other disadvantaged populations. Individuals pay for dental services on a sliding fee scale adjusted by their ability to pay. Health Centers are a primary source of care for 2.84 million Medicaid-eligible individuals, who make up 33 percent of Health Center clients. An additional 3.55 million uninsured patients represent 41 percent of their clients (HRSA 1998). Health Centers that provide oral health care include it as part of an integrated primary care system. In addition, federal programs such as the National Health Service Corps offer scholarships and loan repayment opportunities to encourage newly licensed dentists to locate in underserved areas.

Areas of Overlap

The various components of oral health care work together in diagnosis, prevention, and treatment services. As mentioned above, dental and medical specialists work on teams treating patients with craniofacial birth defects. Oncologists, radiologists, otolaryngologists, plastic surgeons, and surgeons specializing in head and neck surgery similarly may partner with oral and maxillofacial surgeons and prosthodontists in treating oral and pharyngeal cancers and other tumors of the oral cavity and pharynx. Dentists also are active members of general oncology teams. They participate in the examination of patients about to undergo chemotherapy, radiation, or bone marrow transplantation, for example, to ensure that proven preventive measures are taken before treatment to minimize the effects of the therapy on the oral mucosa, salivary glands, and dentition.

Private medical and public health professionals often collaborate in implementing immunization

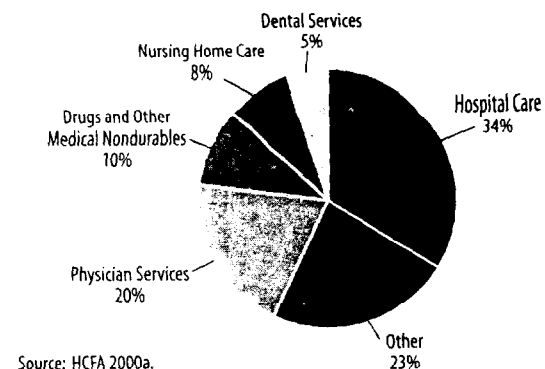
programs and other preventive strategies to reduce a specific disease or to change risk behaviors. Similarly, dental personnel in the private and public sectors cooperate in the implementation of mouthguard programs for sports injury prevention, statewide programs to apply sealants to the teeth of low-income children, and the promotion of oral health self-care behaviors. Private practitioners can deliver care that is paid for by public programs or can work as contractors to Migrant and Community Health Centers and local health departments, among others. Finally, all three components can work together to promote programs that address cross-cutting issues such as tobacco cessation and the prevention and control of HIV disease, oral and pharyngeal cancers, and early childhood caries.

EXPENDITURES FOR ORAL HEALTH CARE

The \$1.1 trillion spent in the United States on health care services in 1998 includes the cost of hospital care, physician and dental services, home health care, nursing home care, prescription drugs, medical equipment, private health insurance, public health activities, and research and represents an increase of 5.6 percent from 1997 (HCFA 2000b). Analysts project that this amount will double by 2007 to total more than \$2.1 trillion (HCFA 2000a).

Expenditures for dental services in the United States in 1998 were \$53.8 billion, a 5.3 percent increase from 1997 and 4.7 percent of the total spent on health care that year (Table 9.3 and Figure 9.2). This figure is an undercount, however, because it represents only those costs associated with care delivered by dentists in practice settings. A generation earlier, in 1960, \$2 billion was spent on dental care, which represented 7.3 percent of that year's total

FIGURE 9.2
Dental services as a percentage of total U.S. health care expenditures by type of service, 1997



health care expenditures. During the 1970s, dental expenditures grew at approximately the same rate as personal health care expenditures, with both exceeding the growth of the economy overall. But starting in 1978, dental expenditures began to flatten and, until 1994, increased more slowly than expenditures for personal health care. Since 1994, dental expenditures have increased at a higher rate than personal health care expenditures (Levit et al. 1998).

Real per capita dental care expenditures (1995 dollars) are currently at about the level they were in the early 1980s, and in some years have declined (Figure 9.3) (ADA 1997b, Beazoglou et al. 1993, Beazoglou 1998). The American Dental Association estimated that \$174.12 was spent per capita in 1995 for dental services (ADA 1997b); HCFA estimated the same year's per capita consumer expenditures for dental services at \$164 (U.S. Bureau of the Census 1998).

The annual percentage change in fees for medical, physician, and dental services as measured by the Consumer Price Index (CPI) has generally exceeded that for the index as a whole (U.S. Bureau of Labor Statistics 1999) (Table 9.4 and Figure 9.4). Percentage changes in the dental CPI have generally

followed those for other medical services; since 1983, however, prices for dental services have increased at a rate faster than those for physician and all medical services. These trends signal different market forces for dental care services as compared to other health services.

In addition to dental care expenditures for services provided by dentists in practice settings, the full cost of oral health care in the United States must take into consideration the breadth of oral, dental, and craniofacial conditions for which services are provided in hospital and other institutional settings, often by nondentists. For example, the Healthcare Cost and Utilization Project (2000) estimated inpatient hospital charges for diseases of the mouth and disorders of the teeth and jaw to be \$451 million in 1996. Estimates for the management of severe early childhood caries range from \$1,500 to \$2,000, depending on whether hospitalization is necessary (Griffin et al. 2000, Kanellis et al. 2000). In Iowa the average cost of treating this condition in a hospital operating room was estimated to be \$2,578 (Damiano et al. 1996). In California, the lifetime cost per case for cleft lip/palate repair is estimated at \$101,000 (Waitzman et al. 1996).

TABLE 9.3
U.S. national health expenditures by source of funds and type of expenditure, 1998 (\$ billions)

	Total	Private					Government		
		All Private Funds	Consumer				Total	Federal	State and Local
			Total	Out of Pocket	Private Insurance	Other			
National health expenditures	1,149.1	626.4	574.6	199.5	375.0	51.8	522.7	376.9	145.8
Health services and supplies	1,113.7	613.4	574.6	199.5	375.0	38.8	500.4	360.4	140.0
Personal health care	1,019.3	574.5	536.5	199.5	337.0	37.9	444.9	343.6	101.3
Hospital care	382.8	149.9	130.9	12.8	118.0	19.1	232.9	187.4	45.5
Physician services	229.5	156.2	151.7	35.7	116.0	4.5	73.3	60.8	12.4
Dental services	53.8	51.5	51.3	25.8	25.5	0.2	2.3	1.3	1.0
Other professional services	66.6	52.4	47.4	27.2	20.2	5.0	14.2	11.2	3.0
Home health care	29.3	13.7	10.0	6.0	4.0	3.7	15.5	13.1	2.4
Drugs and other medical nondurables	121.9	103.1	103.1	55.4	47.8	—	18.8	10.7	8.1
Vision products and other medical durables	15.5	9.0	9.0	8.2	0.8	—	6.5	6.4	0.1
Nursing home care	87.8	34.8	33.2	28.5	4.7	1.6	53.0	35.4	17.7
Other personal health care	32.1	3.8	—	—	—	3.8	28.3	17.1	11.2
Program administration and net cost of private health insurance	57.7	38.9	38.0	—	38.0	0.9	18.8	12.6	6.2
Government public health activities	36.6	—	—	—	—	—	36.6	4.2	32.4
Research and construction	35.3	13.0	—	—	—	13.0	22.3	16.5	5.8
Research	19.9	1.6	—	—	—	1.6	18.3	15.5	2.8
Construction	15.5	11.5	—	—	—	11.5	4.0	1.0	3.0

Note: Research and development expenditures of drug companies and other manufacturers and providers of medical equipment and supplies are excluded from research expenditures, but are included in the expenditure class in which the product falls. Numbers may not add to totals because of rounding.
Source: HCFA 2000b.

FINANCING AND REIMBURSEMENT

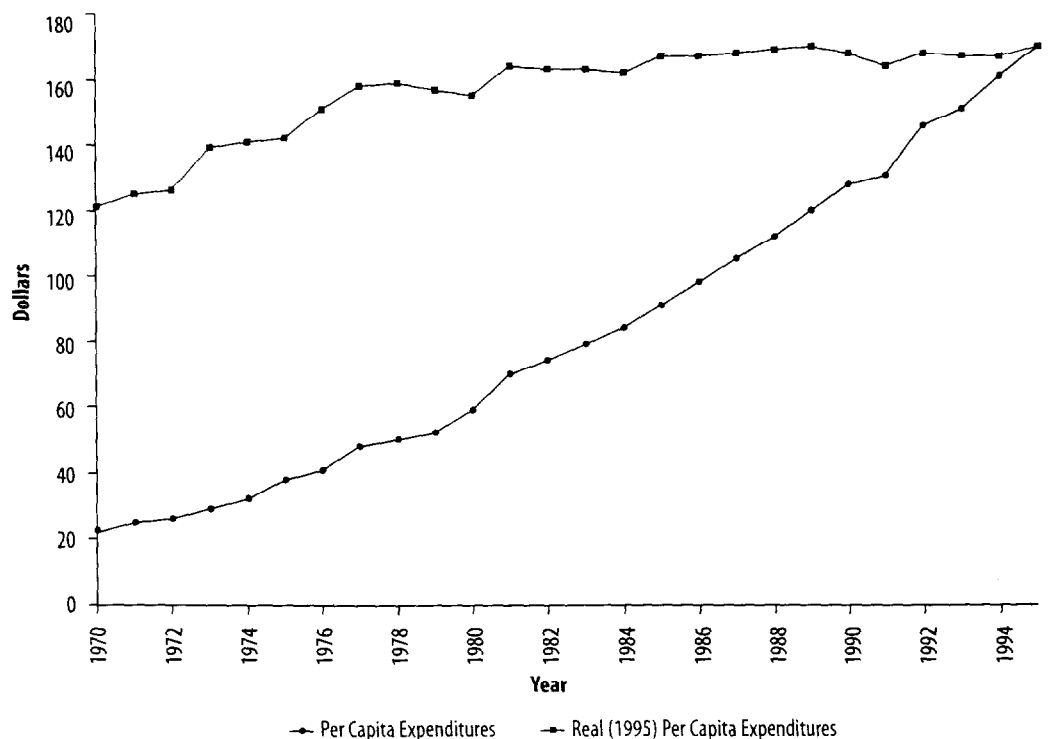
Dental care is financed principally through private sources, either as out-of-pocket payments made directly to the dentist or through employment-based dental insurance benefits. Since 1960, these two sources have financed over 93 percent of all dental expenditures (Figure 9.5). Table 9.5 shows the change in contributions for dental expenditures from 1970 to 1996. The proportion of dental expenditures that private dental insurance covers has increased over the past two decades. Dental insurance now contributes about 48 percent of dental expenditures, as compared to 50.1 percent contributed by medical insurance for physician services. In contrast, the percentage of out-of-pocket payments for dental services is over 3 times that for physician services (Figure 9.6). Sharp differences are also evident in terms of federal, state, and local government contributions to the cost of dental care as compared to physician services. Only 4.0 percent of dental care costs, or \$2.3 billion in 1998, is financed publicly (largely through federal-state Medicaid programs), compared to 32.2 percent for medical care. Few hospital dental services are reimbursed by Medicare, and state Medicaid programs may provide low reimbursement for dental

services. In contrast, public sources pay a large part of hospital costs for medical care.

Insurance

Insurance is a major determinant of dental utilization: 70.4 percent of individuals with private dental insurance reported seeing a dentist in the past year, compared to 50.8 percent of those without dental insurance (Bloom et al. 1992). Private dental care benefits are available to most full-time employees (59 percent) in medium-sized and large businesses. Fewer small businesses offer dental benefits. For the 22.6 million employees with employer-provided dental benefits, dental care may be offered as part of a comprehensive medical and dental plan or as a separate plan. Firms often offer employees a choice of medical plans as well as a separate dental plan that can accompany any medical plan. For employees with employer-provided dental benefits, 81 percent received care through fee-for-service plans, 11 percent from preferred provider organizations, and 8 percent from health maintenance organizations (HMOs) in 1998 (EBRI 1998).

FIGURE 9.3
Per capita dental expenditures in current and real (1995) dollars, United States, 1970 to 1995



Sources: U.S. Bureau of the Census 1998, U.S. Bureau of Labor Statistics 1999.

Provision of Oral Health Care

Most participants in employer dental plans receive benefits through a fee-for-service plan, which reimburses patients or providers after services are received. Such plans are commonly obtained through a commercial insurer, or are self-insured (the firm sets aside funds to meet expected charges), or are a combination of the two. Among self-insured plans is a type of dental plan called direct reimbursement, which enables patients to pay the dentist directly based on what they have been charged. The patients

are reimbursed by the plan based on their expenditures, up to a predetermined limit for total expenditures, but not according to the type of service they receive.

Dental insurance plans that reimburse dentists by type of service performed typically cover technical procedures but not counseling services, treatment planning, or disease management. Diagnostic and preventive care usually includes dental examinations, prophylaxes, sealants, and radiographs. Restorative

procedures may be limited to fillings, but may include crowns. Other services that may be covered include periodontal care, endodontic care, prosthetics, and oral surgery. Orthodontic care is covered less often by dental plans than are other procedures. In addition, most plans limit orthodontic coverage to dependent children and set maximum allowable payments. Dental implants, cosmetic procedures, and some preexisting conditions typically are not covered.

Dental insurance plans are similar to medical plans in defining the terms of payment on a fee-for-service basis. Typically, they may pay a percentage of the fee; they may pay up to a specified dollar amount using a table of allowances; they may require the patient to pay initial costs up to a fixed amount (a deductible); or they may pay a varying percentage of dental charges, based on a patient's past use of dental services. In all cases, the patient pays the difference. Copayments are a larger percentage of the total cost of dental care than is the case for medical care.

Dental coverage varies by race/ethnicity, income, and educational levels. Whites (41.8 percent), people with 13 years or more of education (51.4 percent), and families with annual incomes of \$35,000 or more (60.8 percent) have the highest percentage of insurance coverage in their demographic categories (Figure 9.7). Hispanic individuals have the lowest percentage of coverage

TABLE 9.4
Consumer Price Index for dental services, physician services, all medical care, and all items, United States, 1960 to 1997

	Dental	Physician	All Medical	All Items	Dental/ Physician	Dental/ All Medical	Dental/ All Items
1960	27.0	21.9	22.3	29.6	1.23	1.21	0.91
1961	27.1	22.4	22.9	29.9	1.21	1.18	0.91
1962	27.8	23.1	23.5	30.2	1.20	1.18	0.92
1963	28.6	23.6	24.1	30.6	1.21	1.19	0.93
1964	29.4	24.2	24.6	31.0	1.21	1.20	0.95
1965	30.3	25.1	25.2	31.5	1.21	1.20	0.96
1966	31.3	26.5	26.3	32.4	1.18	1.19	0.97
1967	32.8	28.4	28.2	33.4	1.15	1.16	0.98
1968	34.6	30.0	29.9	34.8	1.15	1.16	0.99
1969	37.1	32.1	31.9	36.7	1.16	1.16	1.01
1970	39.2	34.5	34.0	38.8	1.14	1.15	1.01
1971	41.7	36.9	36.1	40.5	1.13	1.16	1.03
1972	43.4	38.0	37.3	41.8	1.14	1.16	1.04
1973	44.8	39.3	38.8	44.4	1.14	1.15	1.01
1974	48.2	42.9	42.4	49.3	1.12	1.14	0.98
1975	53.2	48.1	47.5	53.8	1.11	1.12	0.99
1976	56.5	53.5	52.0	56.9	1.06	1.09	0.99
1977	60.8	58.5	57.0	60.6	1.04	1.07	1.00
1978	65.1	63.4	61.8	65.2	1.03	1.05	1.00
1979	70.5	69.2	67.5	72.6	1.02	1.04	0.97
1980	78.9	76.5	74.9	82.4	1.03	1.05	0.96
1981	86.5	84.9	82.9	90.9	1.02	1.04	0.95
1982	93.1	92.9	92.5	96.5	1.00	1.01	0.96
1983	99.4	100.1	100.6	99.6	0.99	0.99	1.00
1984	107.5	107.0	106.8	103.9	1.00	1.01	1.03
1985	114.2	113.3	113.5	107.6	1.01	1.01	1.06
1986	120.6	121.5	122.0	109.6	0.99	0.99	1.10
1987	128.8	130.4	130.1	113.6	0.99	0.99	1.13
1988	137.5	139.8	138.6	118.3	0.98	0.99	1.16
1989	146.0	150.1	149.2	124.0	0.97	0.98	1.18
1990	155.8	160.8	162.8	130.7	0.97	0.96	1.19
1991	167.4	170.5	177.0	136.2	0.98	0.95	1.23
1992	178.7	181.2	190.1	140.3	0.99	0.94	1.27
1993	188.1	191.3	201.4	144.5	0.98	0.93	1.30
1994	197.1	199.8	211.0	148.2	0.99	0.93	1.33
1995	206.8	208.8	220.5	152.4	0.99	0.94	1.36
1996	216.5	216.4	228.2	156.9	1.00	0.95	1.38
1997	226.6	222.9	234.6	160.5	1.02	0.97	1.41

Source: U.S. Bureau of Labor Statistics 1999.

TABLE 9.5
U.S. national expenditures on dental services, 1970 to 1996 (\$ millions)

	Total Dental Expenditures	Out-of-Pocket Payments	Private Health Insurance	Other Private Funds	Public Funds (Medicaid, etc.)
1970	4,669	4,240	212	0	217
1971	5,181	4,672	248	0	261
1972	5,516	4,934	292	0	290
1973	6,323	5,605	378	4	336
1974	7,076	6,130	550	7	389
1975	7,956	6,530	939	11	475
1976	8,972	6,965	1,486	15	506
1977	10,055	7,582	1,908	19	547
1978	10,957	7,918	2,504	22	512
1979	11,893	8,237	3,072	27	557
1980	13,323	8,833	3,811	27	652
1981	15,698	10,082	4,839	39	738
1982	16,953	10,547	5,737	43	626
1983	18,271	11,010	6,578	44	639
1984	19,833	11,578	7,613	51	591
1985	21,650	12,243	8,682	73	653
1986	23,108	12,658	9,677	81	693
1987	25,343	13,118	11,409	86	730
1988	27,460	13,845	12,758	94	763
1989	29,496	14,485	14,115	110	786
1990	31,566	15,389	15,134	124	919
1991	33,348	16,139	15,948	134	1,127
1992	37,013	17,782	17,686	143	1,402
1993	39,099	18,647	18,398	154	1,900
1994	41,665	19,727	19,798	167	1,974
1995	44,695	21,007	21,477	173	2,038
1996	47,551	22,116	23,166	183	2,086

Source: HCFA 2000b.

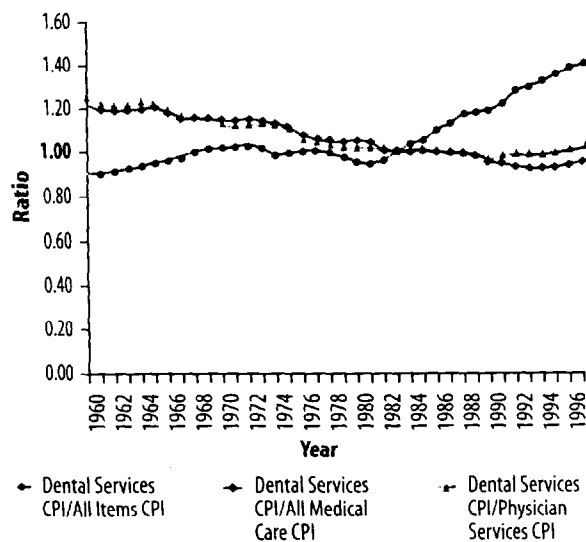
(29.7 percent), followed by blacks (32.4 percent), a pattern seen in medical insurance as well. Because private dental insurance is typically an employment-related benefit, some individuals lose their dental coverage when they retire. As a consequence, people 65 and older reported the lowest levels of coverage (NCHS 1992).

Although over 14 percent of children under 18 have no form of private or public medical insurance, more than twice that many, 23 million children, have no dental insurance (Vargas et al. 2000). Over 15 percent of persons 18 and older have no form of medical insurance, but 3 times as many, over 85 million persons, have no form of dental insurance (NCHS 2000).

The Changing Market

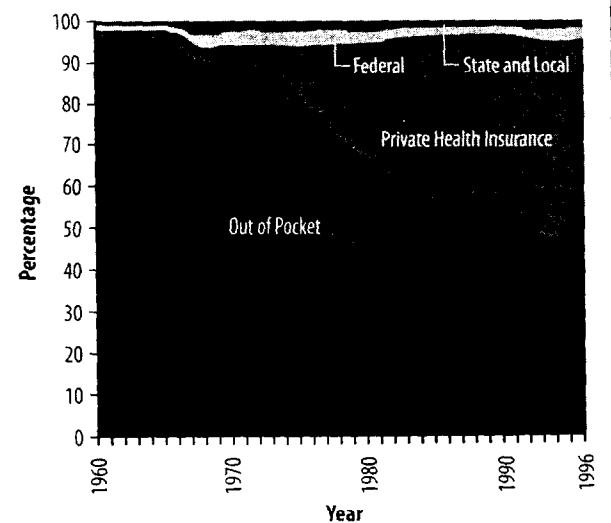
Increasingly, private dental insurance plans are entering into contractual agreements with dentists. The purpose of these agreements is to shift some or all of the financial risk to the clinician, the beneficiary, or both. These alternative

FIGURE 9.4
Ratio of dental services CPI to physician services CPI, all medical care CPI, and all items CPI, 1960 to 1997



Source: U.S. Bureau of Labor Statistics 1999.

FIGURE 9.5
Percentage of total U.S. dental expenditures by source and year, 1960 to 1996



Sources: HCFA 2000b, U.S. Bureau of the Census 1998, U.S. Bureau of Labor Statistics 1999.

reimbursement systems have been labeled “managed dental care.” As defined by the Physician Payment Review Commission (1997), managed care is “any system of health services payment or delivery arrangements where the health plan attempts to control or coordinate use of health services by its enrolled members in order to contain health expenditures, improve quality, or both.”

In dentistry the primary alternative reimbursement systems in place are the dental health mainte-

nance organization, dental preferred provider organization (PPO), and dental referral network. Between 1995 and 1996, dental HMO enrollment grew 17.7 percent; it grew another 8.6 percent between 1996 and 1997, for a total enrollment of approximately 26.5 million people. Dental PPO enrollment grew 30.9 percent in 1996 and 32.6 percent in 1997 to a total of about 24.5 million people (Table 9.6). Dental indemnity increased by 10.1 percent in 1996 and 2.6 percent in 1997 to about 90.6 million persons (NADP 1998). By comparison, the Health Care Financing Administration reported an increased shift of employers and employees from indemnity to managed care health plans, in the past several years. According to HCFA, 86 percent of all insured workers were covered by managed care health plans in 1998, an increase of 54 percent over 1993 (HCFA 2000b).

The rapid changes in the health care environment have emphasized the development of performance measures that can be used by both public and private purchasers of care, consumers, and health care professionals. Specifically, health care quality oversight has focused on the collection and use of data that provide the basis for assessing and monitoring care delivery performance. These performance

FIGURE 9.6
Source of funds for dental and physician services, United States, 1997

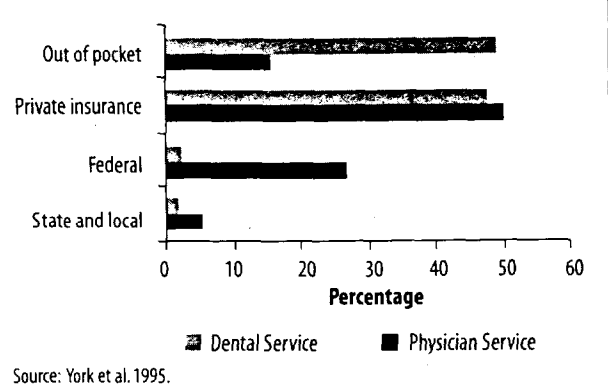
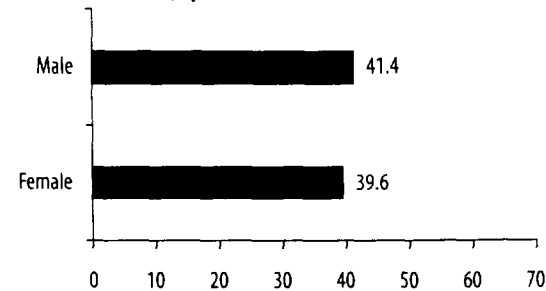
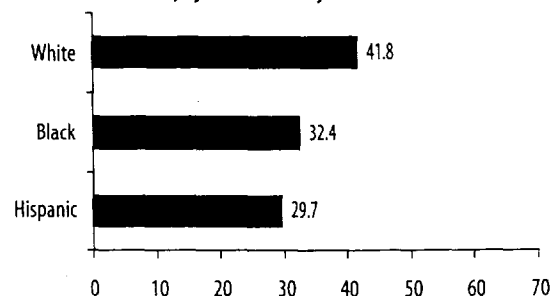


FIGURE 9.7
Dental insurance coverage, United States, 1989

Percentage of persons aged 2 and older who are covered by private dental insurance, by sex

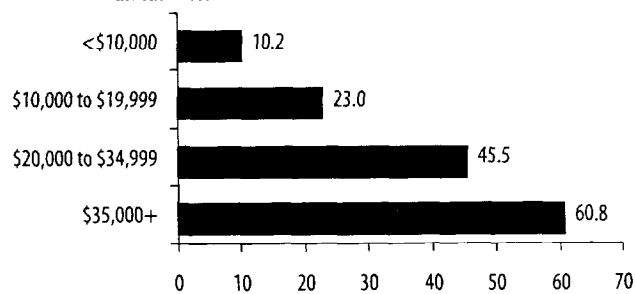


Percentage of persons aged 2 and older who are covered by private dental insurance, by race/ethnicity

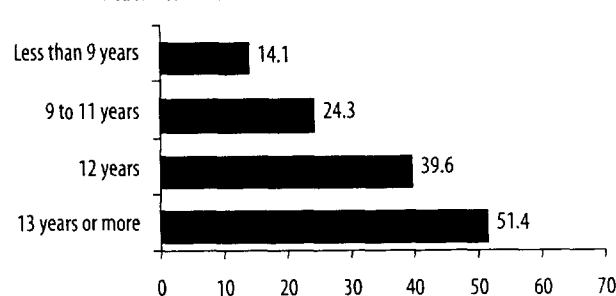


Sources: ADA 1997b, NCHS 1992.

Percentage of families with private dental insurance with an annual income of



Percentage of persons with private dental insurance with an education level of



or outcome measures require development and testing to determine their reliability and validity, and depend on enhanced data collection and information systems for their application. An example of performance measures is the Health Plan Employer Data and Information Set (HEDIS), a set of standardized measures developed by the National Committee for Quality Assurance. Recently, pediatric oral health measures have been reviewed and additional measures proposed for HEDIS consideration (Crall et al. 1999). A framework for the development of outcome measures for oral health care has been proposed along four dimensions: biologic, clinical, psychosocial, and economic. This schema is designed for potential use by patients, health care providers, purchasers of care, and the public (Bader and Ismail 1999). Efforts are needed to proceed with the development and testing of reliable and valid outcome measures in all four dimensions for oral health care and their incorporation into practice and programs.

Federal and State Programs

Medicaid

The Medicaid program, established as Title XIX of the Social Security Amendments of 1965, was designed to provide health care for all indigent and medically indigent persons, with funding shared between federal and state governments. Although states differ in eligibility rules and expenditures for services provided, amendments to the Medicaid program instituted in 1968 required all states to include dental care for individuals under 21 years of age as part of the Early and Periodic Screening, Diagnostic, and Treatment (EPSDT) service. In addition, the Omnibus Budget Reconciliation Act of 1989 required the provision of all medically needed dental services for EPSDT beneficiaries beyond what is covered under the state's Medicaid plan. Medicaid funds

dental care for low-income individuals and persons with disabilities at usual and customary fees, or the Medicaid fee schedule rate, whichever is lower. Although some states have increased their medical reimbursement to 80 percent of usual and customary fees, the norm is 47 percent (Colby 1994).

In 1998, total governmental outlays for dental services were \$2.3 billion (\$1.3 billion federal, \$1.0 billion state and local). Of this total, \$2.0 billion represented dental Medicaid expenditures, which is approximately 1.25 percent of the \$159.6 billion designated for all Medicaid personal health care expenditures, a proportion that is much lower than it was in the early years of the Medicaid program (HCFA 2000b). Some states have tightened their eligibility requirements and have reduced the range of covered dental services for adults. States have not been able to meet the mandatory components of the EPSDT dental program, partly because of low levels of reimbursement to providers and difficulties regarding access to care for eligible enrollees.

Eligibility for Medicaid, as with any form of insurance coverage, does not ensure receipt of adequate dental care. A 1996 report by the USDHHS Inspector General estimated that 80.3 percent of eligible infants, children, and youth up to 20 years of age, for whom disease levels are known to be high (see Chapter 4), did not receive preventive dental services (USDHHS 1996). The report stated that the reasons were complex and included the following factors: few dentists see Medicaid patients, Medicaid families give dental services low priority, and the youngest patients are the least likely to obtain care.

The State Children's Health Insurance Program

Legislation passed by Congress in 1997 created the State Children's Health Insurance Program, which provides billions of dollars to states (supplemented by required state contributions) to extend coverage for health care to uninsured children. For a child to be eligible, family income may be as high as twice the federal poverty level, exceeding eligibility for Medicaid. The states must cover immunizations and well-child care within specified program requirements, but are otherwise free to decide how the money is spent. By midsummer 1999, only 1.3 million of the 10 million uninsured children had been enrolled in SCHIP, with some states using the funds to expand Medicaid coverage and

TABLE 9.6
Estimated number of dental plan members and annual percentage change by market sector and year, United States, 1995 to 1997

	Number of Members			Percentage Change 1995 to 1996	Percentage Change 1996 to 1997
	Year End 1995	Year End 1996	Year End 1997		
Dental HMO	20,697,452	24,359,434	26,457,650	17.7	8.6
Dental PPO	14,085,181	18,442,216	24,460,062	30.9	32.6
Dental referral	1,920,330	5,033,866	5,453,264	162.1	8.3
Dental indemnity	80,255,346	88,323,803	90,640,826	10.1	2.6
Total/average	116,958,309	136,159,319	147,011,802	16.4	8.0

Source: Data from NADP 1998.

others designing new programs that may or may not include dental care (see Chapter 10 for more details).

Medicare

Dental services covered under the Medicare program are limited. Unlike Medicaid, Medicare (Title XVIII of the Social Security Amendments of 1965) is financed totally by the federal government; it was originally designed to provide physician and hospital services for all persons 65 and older, rich and poor alike. Medicare is split into hospital insurance (Part A) and physicians' services (Part B), the latter being a voluntary supplemental insurance program paid for by the individual.

Medicare was not designed to insure routine dental care. Rather, as an exception to the statutory exclusion from Medicare of dental services, it covers dental services needed by hospitalized patients with specific conditions. These include dental services in connection with jaw fractures or with preparation of patients for radiation in cases of oral and pharyngeal cancers or as part of a comprehensive workup prior to renal transplant surgery (Table 9.7). Total Medicare payments for dental services in 1998 were \$0.1 billion (HCFA 2000b).

Recently, the Institute of Medicine (IOM) was asked to study the short- and long-term benefits and costs to the Medicare program of extending coverage to include "medically necessary dental care" to beneficiaries for a limited number of conditions. In the Medicare program, the term "medically necessary dental services" is used narrowly to mean care that occurs as the direct result of an underlying medical condition or its treatment or that has a direct effect

on such a condition. Under this definition, care for serious periodontal disease would not be "medically necessary" unless, for example, it threatened the health of someone with leukemia or was caused by the disease or its treatment (and could otherwise be health threatening if untreated). The IOM report noted that such a restrictive definition may suggest that "periodontal or other tooth-related infections are somehow different from infections elsewhere" and "imply that the mouth can be isolated from the rest of the body, notions neither scientifically based nor constructive for individual or public health."

The IOM committee concluded that it is reasonable for Medicare to cover both tooth-preserving care and extractions for patients undergoing radiation for oral and pharyngeal cancers, and a dental examination, cleaning of teeth, and treatment of acute infections of the teeth or gingiva for a leukemia patient prior to chemotherapy. The report suggested that further study would enable recommendations to be made—on a condition-by-condition basis—for coverage of effective dental services needed in conjunction with surgery, chemotherapy, and radiation for other conditions (Field et al. 1999).

FACTORS AFFECTING THE CAPACITY TO MEET ORAL HEALTH NEEDS

The nation's capacity to provide care that is accessible and acceptable to address the oral health needs and wants of Americans in the next century is challenged by numerous factors. Among them are concerns about a declining dentist-to-population ratio, an inequitable distribution of oral health care providers, a low number of underrepresented minorities

TABLE 9.7
Current Medicare coverage for dental services

Clinical Condition	Covered Service	Part A (hospital)	Part B (physician)
Underlying medical condition and clinical status require hospitalization for dental care	Inpatient hospital services only	X	
Severity of dental procedure requires hospitalization for dental care	Inpatient hospital services only	X	
Any oral condition for which nondental services are covered	All dental services if incident to and an integral part of covered procedure or service		X
Neoplastic jaw disease	Extractions prior to radiation		X
Renal transplant surgery	Oral/dental examination on an inpatient basis	X (if dentist is on staff at hospital where service is provided)	X (outpatient payment for physicians only)

Source: HCFA 2000b.

applying to dental schools, the effects of the cost of dental education and graduation debt on decisions to pursue a career in dentistry, the type and location of practice upon graduation, current and expected shortages in personnel for dental school faculties and oral health research, and an evolving curriculum with an ever-expanding knowledge base.

Numbers of Dental Personnel

The ratio of dentists to the total population is declining: in 1996, there were approximately 58.4 professionally active dentists per 100,000 people in the United States, down from 59.1 in 1990. The current ratio equates to one dentist for every 1,700 people (HRSA 1999). The dentist-to-population ratio is a very crude measure of dental care capacity, because it does not consider dentist productivity (affected by hours worked, use of auxiliary personnel, and mix of services provided) or location of practices relative to underserved populations; there is no agreement on the number that is optimal. Nevertheless, this ratio does indicate trends. By 2020 the dentist-to-population ratio is expected to drop to 53.7 per 100,000 (Figure 9.8). Moreover, it appears that the absolute number of active dentists will decline after 2000. In part, this drop reflects the retirement of older dentists (estimated to range from 2,500 to over 4,300 per year between 1996 and 2021 (HRSA 1999) with insufficient numbers of new graduates (estimated at about 4,000 per year) replacing them (ADA 1999). In comparison, the ratio of active physicians to population has been increasing; it was 251.6 per 100,000 in 1997, up from 226.1 in 1990 (HRSA 1999). The trend in the reduction of the dentist-to-population ratio and the absolute number of dentists

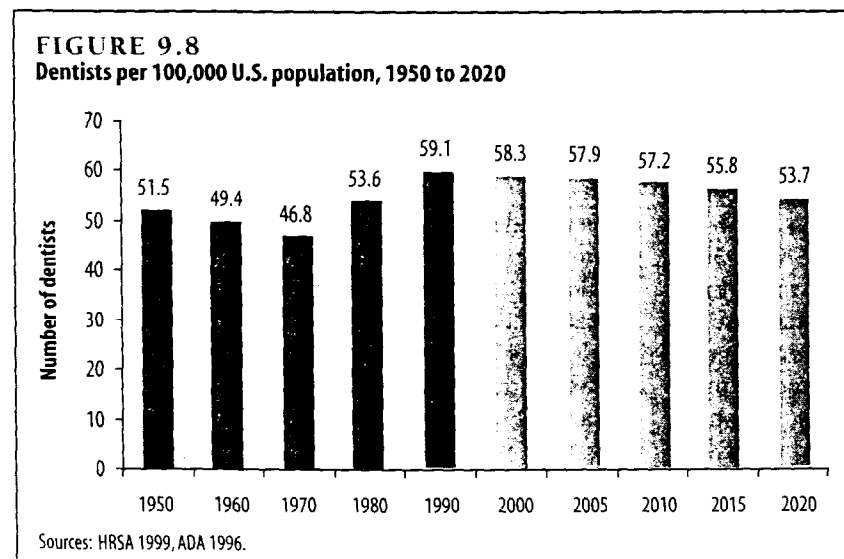
implies a shortage of dentists in the future. This trend may, however, be offset by innovation in dental practice. However, if the impact of future technology changes is similar to that produced by changes over the past 20 to 30 years, it will not substantially affect the projections.

The entering supply of dentists and dental hygienists depends on the number of graduates from dental and dental hygiene schools. The number of applicants to dental schools almost doubled between 1989 and 1997. However, the number of applicants declined by 4 percent in 1998, with further declines of 8 to 10 percent expected for 1999 and 2000. Based on the sharp decline that has occurred in the number of individuals taking the Dental Admissions Test, similar declines may continue into the early 2000s. During the 1989-97 time period, dental school first-year enrollment increased only about 9 percent. Little further growth in enrollment is anticipated because the current infrastructure in dental education has limited ability to expand, coupled with the declines occurring in dental school applicants. Along with concerns about a possible shortage of dentists, there is concern that the pool of qualified applicants may not be sufficient to supply a dental workforce that meets the needs of society, as well as the needs of dental education and research.

In contrast, the number of dental hygiene programs and students has increased almost 18 percent since 1990. The number of first-year dental hygiene students currently stands at 6,000, more than recovering from the 15 percent decline that occurred in these programs during the late 1970s through the mid-1980s. The last 4 years has seen a steady 11 percent growth in dental hygiene positions.

The numbers of dentists and sites of health pro-

profession education programs have been influenced by government policies and social factors. During the late 1950s an emerging shortage of health care providers (including dentists) was expected arising from the "baby boom" that began in the late 1940s. Beginning in the early 1960s, the federal government supported an expansion in the number of medical and dental schools and in class sizes. By the mid-1970s, the number of dental schools had grown from 47 to 60. First-year enrollments grew from 3,612 to 6,301. By the mid-1970s, a possible oversupply of physicians and



dentists became a concern. Government support for all health profession education was substantially reduced. Through the 1980s, dental schools reduced their enrollment by 37 percent. By 1993, six dental schools, all affiliated with private universities, had closed.

Following the growth in dental school enrollments that has occurred since 1989, the 1998-99 first-year enrollment stood at 4,268 in 55 dental schools located in 33 states, the Commonwealth of Puerto Rico, and the District of Columbia. One more dental school (at Northwestern University) is scheduled to close in 2001. One is scheduled to open in 2001, at the University of Nevada at Las Vegas (ADA 1996). Total dental school enrollment in 1998-99 was 17,033 students, down from a peak of 22,842 in 1980-81.

Sex and Racial/Ethnic Composition of Dental Personnel

The number and percentage of women in the dental and medical professions have continued to increase. Thirty years ago, women represented 1.3 percent of first-year enrollment in dental schools. By 1988, that proportion had risen to 35.7 percent, a level that has been relatively constant over the past 10 years. Recent trends indicate that the proportion of women in dentistry will continue to increase: by 2000, more than 26,000 women will be active practitioners; this is almost twice the number in 1990 (HRSA 1993, 1999). However, data from 1990 show that the proportion of dentists who were women (9.5 percent) was smaller than the proportion of female physicians (17.0 percent), female pharmacists (28.9 percent), and female optometrists (14.6 percent). The percentage change in the numbers of first professional degrees conferred to women by health field of study and race/ethnicity from 1981 through 1990 shows that although dentistry is second lowest (next to allopathic medicine), the percentage changes for Hispanic women and American Indian/Alaska Native (AIAN) women in dentistry were among the highest (46.7 percent and 500.0 percent, respectively), even though the actual numbers were low (HRSA 1993).

The participation of racial and ethnic minorities in dentistry does not mirror the dramatic increase in the entrance of women into the profession in the course of a single generation. The demographic profile of the U.S. population is changing rapidly, and is likely to continue to do so, with continued increases in racial and ethnic minority groups in comparison to whites. However, these trends and projections are not reflected in the dental or medical workforce.

The overall percentage of minority students has increased significantly, to the point that, in 1998, a little over 34 percent of the first-year students were members of a minority group. This overall percentage is up from 13 percent in 1980. However, the primary increase has come among Asian/Pacific Islander students, increasing from about 5 percent of enrollment in 1980 to almost 25 percent in 1998. At the same time, the proportion of black/African American, Hispanic/Latino, and American Indian students, together, has shown only a small percentage point increase since 1980, from about 7.5 percent to nearly 10 percent. The percentage of first-year enrollment in 1998 for black/African American students was 4.4 percent. It was 4.9 percent for Hispanic/Latino students and 0.4 percent for American Indians. These percentages for black/African American, Hispanic/Latino, and American Indian students are far less than their percentages in the U.S. population. In addition, a specific look at black dental school graduates during the 1980s and 1990s showed that although the number of black female graduates had increased, the increase was insufficient to offset the decline in black male graduates (HRSA 1993).

In 1996, African Americans made up 12.0 percent of the general population, but only 2.2 percent of active dentists (Brown and Lazar 1999). Similarly underrepresented were Hispanics, who accounted for 10.7 percent of the population, but only 2.8 percent of active dentists. The Hispanic population is the fastest-growing segment of the population and by 2002 will exceed the number of blacks (U.S. Bureau of the Census 2000). American Indians, 0.7 percent of the population, represented only 0.2 percent of active dentists. Table 9.8 shows the 1996 dentist-to-population ratios by race/ethnicity of the dentist.

As has been shown in Chapter 4 and elsewhere in this report, oral health problems disproportionately affect disadvantaged populations among underrepresented minority groups. This disparity will not be ameliorated through technology improvements or increases in clinical productivity. Moreover, recent data show that underrepresented racial and ethnic minority dentists are more likely to provide care to minority populations. In 1996, black dentists reported that 61.8 percent of their patients were black, and Hispanic dentists reported that Hispanic patients made up 45.4 percent of their practice; 76.6 percent of white dentists' patients were white (Brown and Lazar 1999). A recent study of the role of black and Hispanic physicians in the provision of care for underserved populations demonstrated that these physicians practiced in communities with a higher percentage of their racial or ethnic group (Komaromy

et al. 1996). Also, black physicians saw more Medicaid patients, and Hispanic physicians more uninsured patients, than other physicians. If this pattern of treatment of Medicaid patients and the uninsured is similar for dentists, the underrepresentation of minority dentists may also contribute to the unmet needs of minority patients. This issue warrants further research.

Regarding the importance of reaching parity in the dental profession, the American Association of Dental Schools comments, "The production of underrepresented minority [URM] dentists is totally out of synch with projected U.S. demographics. The U.S. population is expected to increase by 60 percent, reaching 394 million by 2050. At that time, nearly half (48 percent) of the population will be constituted from racial and ethnic minority groups. Strategic measures are needed to increase the number of URM dental graduates that will improve access to care for minorities throughout the nation" (AADS 1999).

Recruitment and retention of underrepresented minorities and women into the health professions will continue to be a challenge in the coming years. Activities such as enrichment programs in science and mathematics for grades K-12 and precollege are designed to increase the interest and capacity of all students, including women and underrepresented minorities, in health professions and science careers. These efforts will require careful design, implementation, and evaluation.

Student Indebtedness and Its Effects

The American Association of Dental Schools reports that in 1998 graduates of dental schools had incurred, on average, over \$84,000 in educational debt (G. Luke, AADS, personal communication, 1999). Average debt ranged from \$71,000 for graduates of public schools to \$98,000 for private/state-related schools and \$108,000 for private schools. This was over 14 percent more than the educational debt of graduating medical students. Specialty education

may result in additional debt. Setting up an office involves additional costs. In the end the burden of debts to be repaid is a driving force in decision making for many new graduates regarding career direction and practice site.

Fewer dentists establish practices in low-income communities. The National Health Service Corps (NHSC) was created in 1970 as a program of the U.S. Public Health Service to provide financial assistance to health professionals who agree to locate in a Health Professional Shortage Area (HPSA). The NHSC offers programs for both students and clinicians, including scholarships, loan repayment programs, and rotations in Community Health Centers. Currently, there are approximately 2,526 clinicians, including 306 dental care providers, delivering care to more than 4.6 million people through these programs. Only about 6 percent of the dental need is currently being met in the approved 1,198 dental HPSAs with a population of 25.9 million. It is estimated that an additional 4,873 dental care providers are needed to meet the current demand. In fiscal year (FY) 1999 the NHSC provided 139 new and continuing dental loan repayment awards, valued at \$9 million. In FY 1998, there were 308 dental NHSC scholars, a 40 percent increase since 1994. Outreach and program development are critical to foster growth and create opportunities for placing dentists in underserved areas, where the needs are great.

In addition to the NHSC, the Indian Health Service operates a loan repayment program to identify health professionals who will practice full-time at an IHS facility or approved tribally managed site in exchange for repayment of their eligible health professions educational loans. Funding for this program has remained level for the past 8 years, in spite of the fact that student debt has nearly doubled during that time.

Primary care dental residency programs supported by Health Professions Training Funds also play a role in meeting the oral health care needs of the nation. An evaluation performed for the USDHHS

TABLE 9.8
U.S. dentist-to-population ratios by race/ethnicity of the dentist, 1996

	Total	Black	Hispanic	Asian/Pacific Islander	American Indian	White
U.S. population	265,189,000	31,933,000	28,092,000	9,181,000	1,954,000	194,029,000
Active dentists	154,900	5,201	5,178	10,693	194	133,634
Number of dentists per 100,000 population	58.4	16.3	18.4	116.5	9.9	68.9
Number of people per dentist	1:1,712	1:6,140	1:5,425	1:859	1:10,072	1:1,452

Source: HRSA 1999.

found that 87 percent of General Dentistry trainees remain in primary care practice and over 30 percent of General Dentistry program graduates receiving federal support over the last 4 years entered practice in underserved communities.

The issue of indebtedness not only is an important consideration for the graduate in deciding where to practice, but also has become an obstacle to college students contemplating a career in dentistry and other health professions. Moreover, it can affect the choices graduates make about whether they will pursue careers in academia or research. The National Institutes of Health created three loan repayment programs to attract health care professionals to research in its facilities. In addition, innovative loan repayment incentives, such as awarding "extramural" loan repayment to researchers working in dental education institutes, have been proposed to overcome the current critical shortage of dental faculty/researchers.

Personnel Needs for Faculty and Clinical Research

The education and training of dentists and allied dental health personnel are essential to the country's capacity to meet its oral health needs. Dental education institutions and their allied academic health centers play a critical role in providing the infrastructure for oral, dental, and craniofacial research and continuing education for dental professionals. A task force report on the future of dental school faculty shows that the number of faculty vacancies in the clinical sciences has more than doubled in recent years, rising from 139 in 1992-93 to more than 300 in 1999 (AADS 1999). The task force projects that retirements will rapidly increase in the coming decade given the average age of the faculty (47 percent of all faculty members are aged 50 and older, and 19 percent are 61 and older). Kennedy (1990) estimated that dental institutions need at least 208 to 218 new faculty members each year, based on a faculty turnover rate of approximately 33 percent every 5 years.

Curriculum Needs

New technologies such as telehealth, bioinformatics, and virtual reality, as well as databases specifying human, animal, and microbial genomes, are altering public awareness, attitudes, and behavior regarding health issues. The new knowledge and tools available are also changing dramatically how health care pro-

fessionals are taught, how they learn, how they practice, and how they retain clinical practices.

These developments, along with new paradigms for the treatment of oral, dental, and craniofacial diseases and disorders, have led to several recent studies of oral health professional education and curricula (Field 1995). A 1995 Institute of Medicine study on the future of dental education called for greater integration and collaboration of dental schools with the parent university and academic health center, a commitment to research programs and the building of research capacity, and an enrichment of the curriculum to incorporate new scientific knowledge and its transformation into clinical applications. The report's first strategic policy principle affirmed that "oral health is an integral part of total health, and oral health care is an integral part of comprehensive health care, including primary care." Ideally, curricula for all health professional schools should reflect this principle by integrating knowledge and management of oral and medical health and disease.

Work is beginning on revisions to educational materials necessitated by these advances in research and technology. Initial steps are being taken to increase emphasis on interdisciplinary training, clinical research, and orientation to cultural competency in health professional education. The National Coalition for Health Professional Education in Genetics is promoting the incorporation of genetics, genomics, and proteomics into predoctoral programs to prepare future health professionals to integrate genetics into practice. Other developments that need to be addressed include HIV disease and other emerging and reemerging infectious diseases, increased understanding of gender health issues, management of chronic pain, and the growing numbers of aging baby boomers and older Americans with complex and chronic health problems. For example, instruction on the special needs of individuals whose oral health is compromised by systemic diseases or disease treatments and on the heightened quality-of-life expectations of young and middle-aged adults should be incorporated into the curricula. In addition, in Area Health Education Centers in some states, health profession students work together to care for patients in underserved, rural, or disadvantaged populations.

The HIV/AIDS Dental Reimbursement program assists dental education programs in meeting the HIV/AIDS community's significant need for oral health care services. This program trains dental students and residents in the care and treatment of those living with this chronic disease. A federal-

institutional partnership provides funds to dental education institutions to partially reimburse for the costs of providing oral health care services to people living with HIV and AIDS.

As the health professional curriculum evolves, so must efforts in K-12 education and beyond to improve the public's health literacy. Efforts directed toward improving science and health knowledge and attitudes and at implementing health-promoting practices have begun; these can contribute to an enhanced partnership between patients and their health care providers.

Taking Care of Those Most in Need

The capacity to care for those most in need requires not only an adequate number of individuals to provide the care, but also an equitable distribution of providers to ensure the availability of care. In addition, sufficient financial resources must be available to support the delivery of and reimbursement for care provided to those most in need. Attention must also be given to a quality of care that ensures that the services provided fulfill the needs and functional requirements of the patients. Although the proportion of the population that uses dental services continues to increase, disparities remain (see Chapter 4).

A recent review of the literature related to access to care has identified many of the factors associated with these disparities. The lack of dental insurance emerged as a highly significant factor (Isman and Isman 1997). A series of reports demonstrates that privately insured individuals of all ages are more likely to get dental care when they need it than are the uninsured (Bloom et al. 1997, Cohen et al. 1997, Simpson et al. 1997). Lack of insurance was found to be an even more significant barrier to gaining primary care access for children than either poverty or minority status (Newacheck et al. 1997).

Once access to care has been established, there is greater likelihood that individuals will adopt preventive practices. Although a causal relationship has not been established, Wagener et al. (1992) found that brushing with a fluoride dentifrice and using dietary fluoride supplements were more frequent among preschool children who had had a dental visit in the past year than among those who had not. In contrast, as discussed in Chapter 4, one of the most common reasons cited by individuals in all income and education groups for not having made a dental visit was that they did not perceive that they had a problem. This implies a lack of awareness that attaining and maintaining good oral health and preventing disease

require not only self-care but also professional oral health care.

Federal and state statistics show strong and consistent racial and ethnic disparities among U.S. children in disease occurrence and severity, untreated dental disease, access to dental care, and use of preventive services (see Chapter 4). Vulnerable child populations as well as the elderly, individuals with disabilities, people with HIV, migrant workers, and homeless persons pose an additional set of challenges. These populations require health care providers sensitive to cultural and social issues who are capable of addressing complex problems that demand integrated dental and medical care. The oral, dental, and craniofacial and medical care curricula are vital in preparing dental and other health care providers to coordinate and integrate care for these individuals.

The issues of oral health and the underserved have been addressed in a policy paper, *Oral Health for All: Policy for Available, Accessible and Acceptable Care* (Warren 1999). This report makes recommendations regarding financial barriers to care, integration of oral health services into health care delivery, capacity to meet oral health needs, cultural competency of health care providers, and education and oral professional practice requirements to meet the oral health care needs of underserved populations.

A survey of dental care reported that more than half of the responding private practice dentists provided some charitable care to low-income populations in 1996 (ADA 1998b). Although access-to-care dental programs for low-income populations are supported by many dental societies, this generosity falls well short of meeting the needs of these populations, which also require community-based programs (Waldman 1999) (see Chapter 7). Programs such as Community and Migrant Health Centers serve hard-to-reach populations. In 1996, more than half of such centers provided dental services, serving more than 1 million people (J. Anderson, personal communication, 1999).

TWENTY-FIRST CENTURY CHALLENGES: WHAT LIES AHEAD?

The United States is witnessing unprecedented changes in demography, patterns of disease and disorders, and the nature of health care. The imperative to keep abreast of advances in science and technology is already evident in dentistry and medicine, aided by access to multiple information

systems. In addition to the Internet and continuing dental education, the new century will see continued growth in imaging systems, computer-assisted technology, teledentistry and telemedicine, improved diagnostics and therapeutics, and new biomaterials and other biotechnology products. Genetic information will play an increasing role in assessing a patient's risk for disease and in planning treatments.

Although some information is available on the effectiveness, cost-effectiveness, and outcomes associated with health care treatment, further research will be needed to determine "best practices"—which treatments work for which patients, under what circumstances, and at what cost. Treatment planning will incorporate outcome measures and patient preferences. Systematic reviews of the existing literature will help promote an evidence-based approach to dental and medical care. In addition, comprehensive diagnostic and treatment codes, as well as a process by which new technologies can be incorporated appropriately, will be needed.

The dental profession has been at the forefront of efforts to prevent disease and enhance general health and the quality of life. Efforts such as community water fluoridation, over-the-counter fluoride products, and dental sealants represent a preventive orientation that has been associated with the dental profession for half a century. Dentistry is continuing to be responsive to the ever-rising expectations of patients. Increases in the provision of fee-for-service cosmetic dentistry, adult orthodontics, and dental implants are among the trends already in evidence and expected to grow.

As the knowledge base regarding the relationships between oral health and general health increases, so too will the need for greater coordination of dental and medical services. Efforts to improve cardiac care, for example, may include treatment of periodontal diseases. Prenatal care may come to include a dental evaluation and treatment to reduce the risk of preterm, low-birth-weight deliveries. Regular oral examinations and periodontal treatment for diabetic patients may become an important component in disease control. Partnerships will need to be expanded and new ones created among the private dental, medical, and public health components.

A challenge facing the health professions will be to expand community-based disease prevention and personal oral health care to meet the needs of populations. Questions of access and barriers to care must be addressed and satisfactory solutions found to ensure that there is care for all who seek it.

The extent to which these predicted structural, organizational, and thematic changes will affect the nation's capacity and commitment to provide oral health care is not certain. The nation's health promotion and disease prevention objectives, which include oral health objectives, serve as a critical guide. How successful a changed care system will be in addressing the oral health needs and wants of the nation can be measured in several ways. These include reductions in health disparities in the population, decreases in the overall incidence and prevalence rates of diseases for the entire population, improved functional status, lower costs, reduced mortality rates, and enhanced health and quality of life.

FINDINGS

- Dental, medical, and public health delivery systems each provide services that affect oral and craniofacial health in the U.S. population. Clinical oral health care is predominantly provided by a private practice dental workforce.

- Expenditures for dental services alone made up 4.7 percent of the nation's health expenditures in 1998—\$53.8 billion out of \$1.1 trillion. These expenditures underestimate the true costs to the nation, however, because data are unavailable to determine the extent of expenditures and services provided for craniofacial health care by other health providers and institutions.

- The public health infrastructure for oral health is insufficient to address the needs of disadvantaged groups, and the integration of oral and general health programs is lacking.

- Expansion of community-based disease prevention and lowering of barriers to personal oral health care are needed to meet the needs of the population.

- Insurance coverage for dental care is increasing but still lags behind medical insurance. For every child under 18 years old without medical insurance, there are at least two children without dental insurance; for every adult 18 years or older without medical insurance, there are three without dental insurance.

- Eligibility for Medicaid does not ensure enrollment, and enrollment does not ensure that individuals obtain needed care. Barriers include patient and caregiver understanding of the value and importance of oral health to general health, low reimbursement rates, and administrative burdens for both patient and provider.

- A narrow definition of "medically necessary dental care" currently limits oral health services for many insured persons, particularly the elderly.
- The dentist-to-population ratio is declining, creating concern as to the capability of the dental workforce to meet the emerging demands of society and provide required services efficiently.
- An estimated 25 million individuals reside in areas lacking adequate dental care services, as defined by Health Professional Shortage Area (HPSA) criteria.
- Educational debt has increased, affecting both career choices and practice location.
- Disparities exist in the oral health profession workforce and career paths. The number of under-represented minorities in the oral health professions is disproportionate to their distribution in the population at large.
- Current and projected demand for dental school faculty positions and research scientists is not being met. A crisis in the number of faculty and researchers threatens the quality of dental education; oral, dental, and craniofacial research; and, ultimately, the health of the public.
- Reliable and valid measures of oral health outcomes do not exist and need to be developed, validated, and incorporated into practice and programs.

REFERENCES

- American Association of Dental Schools (AADS). Report of the AADS President's Task Force on Future Dental School Faculty. Washington: American Association of Dental Schools; 1999.
- American Dental Association (ADA), Survey Center. 1990 survey of dental services rendered. Chicago: American Dental Association; 1990.
- American Dental Association (ADA). Dental manpower model: 1995-2020. Chicago: American Dental Association; 1996.
- American Dental Association (ADA). Distribution of dentists in the United States by region and state. Chicago: American Dental Association; 1997a.
- American Dental Association (ADA), Survey Center. Key dental facts. Chicago: American Dental Association, 1997b.
- American Dental Association (ADA), Survey Center. 1997 survey of dental practice. Characteristics of dentists in private practice and their patients. Chicago: American Dental Association; 1998a.
- American Dental Association (ADA), Survey Center. 1997 survey of current issues in dentistry: charitable dental care. Chicago: American Dental Association; 1998b.
- American Dental Association (ADA). 1998-99 survey of predoctoral dental educational institutions, [unpublished data]. Chicago: American Dental Association; 1999.
- Association of State and Territorial Dental Directors (ASTDD) Survey, 1999. [unpublished data].
- Association of State and Territorial Dental Directors (ASTDD). Building infrastructure and capacity in state and territorial dental programs (conference edition: 2000 May).
- Bader J, Ismail A. A primer on outcomes in dentistry. *J Public Health Dent* 1999;59:31-5.
- Beazoglou T, Brown LJ, Heffley D. Dental care utilization over time. *Soc Sci Med* 1993 Dec;32(12):1461-72.
- Beazoglou T. In: Jong A, Gluck GM, Morganstein WM, editors. *Jong's community dental health*. 4th ed. St. Louis: Mosby; 1998. p. 25-41.
- Bloom B, Gift HC, Jack SS. Dental services and oral health: United States, 1989. *Vital Health Stat* 10 1992 Dec;(183):1-95.
- Bloom B, Simpson G, Cohen RA, Parsons PE. Access to health care. Part 2: Working-age adults. *Vital Health Stat* 10 1997 Jul;(197):1-47.
- Brown LJ, Lazar V. Minority dentists: why do we need them? Closing the gap. Washington: Office of Minority Health, U.S. Department of Health and Human Services; 1999 Jul. p. 6-7.
- Burt BA, Eklund S. *Dentistry, dental practice and the community*. 5th ed. Philadelphia: W.B. Saunders; 1999.
- Cohen RA, Bloom B, Simpson G, Parsons PE. Access to health care. Part 3: Older adults. *Vital Health Stat* 10 1997 Jul;(198):1-32.
- Colby DC. Medicaid physician fees. 1993. *Health Affairs* 1994;255-63.
- Crall JJ, Szlyk CI, Schneider DA. Pediatric oral health performance measurement: current capabilities and future directions. *J Public Health Dent* 1999;59:136-41.
- Damiano PC, Kanellis MJ, Willard JC, et al. A report on the Iowa Title XIX Dental Program. Iowa City: Public Policy Center and College of Dentistry, The University of Iowa; 1996 Apr.
- Eklund SA, Pittman JL, Smith RC. Trends in dental care among insured Americans: 1980 to 1995. *J Am Dent Assoc* 1997 Feb;128(2):179-80.
- Employee Benefit Research Institute (EBRI). Facts from EBRI. 1998 Sep.
- Field MJ, editor. *Dental education at the crossroads. Challenges and change*. Washington: National Academy Press; 1995.
- Field MJ, Lawrence RL, Zwanzier L, editors. *Extending Medicare coverage for preventive and other services*. Committee on Medicare Coverage Extensions, Institute of Medicine. Washington: National Academy Press; 1999.

- Griffin SO, Gooch BF, Beltran E, Sutherland JN, Barsley R. Dental services, costs, and factors associated with hospitalization for Medicaid-eligible children, Louisiana 1996-97. *J Public Health Dent* 2000;60(1):21-7.
- Healthcare Cost and Utilization Project. Available from: <http://198.179.0.16/HCUPnet.asp>, Jan 2000.
- Health Care Financing Administration (HCFA). Available from: <http://www.hcfa.gov/stats/nhe-oact.tables/chart.htm> (2000a).
- Health Care Financing Administration (HCFA). National health expenditures, 1998. Washington: Health Care Financing Administration. Available from: <http://www.hcfa.gov/stats.NHE-Proj> (2000b Apr 25).
- Health Resources and Services Administration (HRSA). Health Personnel in the U.S. Ninth report to Congress. Washington: Health Resources and Services Administration, U.S. Department of Health and Human Services; 1993. Pub. no. P-OD-94-i. p. 33-4.
- Health Resources and Services Administration (HRSA). Bureau of Primary Health Care. Uniform data system 1998. Available from: <http://bphc.hrsa.gov/bphc/>.
- Health Resources and Services Administration (HRSA). Bureau of Health Professions, Office of Research and Planning. Dental supply model, 1999. Available from: <http://www.hrsa.dhhs.gov/bhpr/healthworkforce/factbook.htm>.
- Isman R, Isman B. Oral Health America white paper: access to oral health services in the U.S. 1997 and beyond. Chicago: Oral Health America; 1997.
- Kanellis MJ, Damiano PC, Momany ET. Medicaid costs associated with the hospitalization of young children for restorative dental treatment under general anesthesia. *J Public Health Dent* 2000;60(1):28-32.
- Kennedy JE. Faculty status in a climate of change. *J Dent Educ* 1990;54(5):268-70.
- Komaromy M, Grumbach K, Drake M, Vranizan K, Lurie N, Keane D, Bindman AB. The role of black and Hispanic physicians in providing health care for underserved populations. *N Engl J Med* 1996 May;334(20):1305-10.
- Levit K, Cowan C, Braden B, et al. National health expenditures in 1997: more slow growth. *Health Affairs* 1998;17(6):100-2.
- National Association of Dental Plans (NADP). 1998 Dental HMO/PPO Industry Profile. Dallas (TX): National Association of Dental Plans; 1998. Available from: <http://www.nadp.org>.
- National Center for Health Statistics (NCHS). Current estimates from the National Health Survey. 1992;10(1835).
- National Center for Health Statistics (NCHS). National Health Interview Survey (NHIS) 1995. Data tabulated by the Office of Analysis, Epidemiology, and Health Promotion. NCHS, Centers for Disease Control and Prevention; 2000.
- Newacheck PW, Stoddard JJ, Hughes DC, Pearl M. Children's access to health care: the role of social economic factors. In: Stein R, Brooks P, editors. *Health care for children: what's right, what's wrong, what's next*. New York: United Hospital Fund; 1997.
- Physician Payment Review Commission. Washington: Physician Payment Review Commission; 1997.
- Public Health Functions Steering Committee. *Public health in America*. Fall 1994. Available from: <http://www.health.gov.phfunctions/public.htm> (2000 Jan 1).
- Simpson G, Bloom B, Cohen RA, Parsons PE. Access to health care. Part I: Children. *Vital Health Stat* 10 1997 Jul;(196):1-46.
- Systemetrics, Inc. Strategic options to improve the PHS analytic capabilities in oral health. 1994 Sep.
- U.S. Bureau of the Census. The official statistics, statistical abstract of the United States, estimates for dental expenditures. Available from: <http://www.census.gov/Press-Release/www1999/cb99-189.html>. (cited 1998 Sep 17).
- U.S. Bureau of the Census. National population projections. 2000 Jan 13. Available from: <http://www.census.gov/population/www/projections/natsum-T5.html>.
- U.S. Bureau of Labor Statistics. Consumer Price Index, 1999.
- U.S. Department of Health and Human Services (USDHHS). Final report to the House of Representatives Appropriations Committee on oral health activities. 1989 May.
- U.S. Department of Health and Human Services (USDHHS), Office of Inspector General. Children's dental services under Medicaid. Access and utilization. San Francisco: Office of Evaluation and Inspection; 1996 Apr. Pub. no. OEI-09-93-00240.
- U.S. Department of Health and Human Services (USDHHS). *Healthy People 2010* (conference edition in two volumes). Washington: U.S. Department of Health and Human Services; 2000 Jan.
- Vargas CM, Isman RE, Crall JJ. Comparison of children's medical and dental insurance coverage by socioeconomic characteristics, U.S. 1995. Submitted for publication, 2000.
- Wagner DK, Nourjah P, Horowitz AM. Trends in childhood use of dental care products containing fluoride: United States, 1983-89. *Adv Data* 1992 Nov 20;(219):1-15.
- Waitzman N, Scheffler RM, Romano PS. The cost of birth defects: estimates of the value of prevention. Lanham (MD): University Press of America; 1996. p. 262.
- Waldman HB. Why not Medicaid dentistry. *NY State Dent J* 1999 Nov;42-4.
- Warren RC. Oral health for all: policy for available, accessible, and acceptable care. Washington: Center for Policy Alternatives; 1999 Sep. p. 33.
- York AK, Poindexter FR, Chisick MC. 1994 Tri-Service comprehensive oral health survey; active duty report. 1995 Jun. NDRI Report no. PR-9503.

What Are the Needs and Opportunities to Enhance Oral Health?

Many factors have been implicated in determining oral health, and they have varying effects across the life stages. These factors are discussed in Chapter 10, where the incorporation of determinants of health in major public health initiatives such as Healthy People 2010 is highlighted. Essential factors include individual biology and lifestyle, the physical and social environment (including whether a community supports health-promoting measures such as water fluoridation), and the organization of health care. These factors are not independent but interact. An individual with no inherent health problems and a healthy lifestyle also needs to live in a healthy environment with ready access to and ability to pay for health care services. Studies of oral health over the lifetime highlight the interaction of these factors. The chapter focuses primarily on America's most vulnerable populations—children and the elderly—where issues of access, insurance, and reimbursement are critical in determining oral health and limit the delivery of care for individuals with special needs and those residing in institutions.

Chapter 11 focuses on the future and the promise of research born of the revolutions in genetics, biotechnology, and biomimetics—the new science of tissue repair and regeneration. Global demographics and technologic innovations signal the need for health literacy and universal access to care if we are to enhance oral health for all Americans.

Chapter 12 highlights the major findings and recommendations of the report. Everyone—individuals, communities, policymakers, health care providers, educators, and researchers—has a role in improving and promoting oral health. The major conclusion of the report is that oral health is essential to general health and well-being. A National Oral Health Plan will facilitate the means to improve the nation's oral health. The chapter concludes with five actions proposed toward that end: strengthen understanding of oral health and disease by the public, practitioners, and policymakers; accelerate building the science and evidence base; enhance health infrastructure and program integration; reduce barriers to oral health care; and increase public-private partnerships to address health disparities.

Factors Affecting Oral Health over the Life Span

When the World Health Organization (WHO) expanded the definition of *health* in 1948 to mean a complete state of physical, mental, and social well-being, and not simply the absence of infirmity, the move stimulated research to define the major factors affecting health and well-being. Investigators developed model systems of “health-related quality of life” and “oral-health-related quality of life.” Chapter 6 describes such models and provides examples of indices and instruments used to measure quality of life. What these models have in common are factors that include biological or physiological measures of health, but also take into consideration an individual’s ability to function normally in the routines of daily living, experience symptom relief, and fulfill usual roles in personal relationships and in family, work, civic, and social interactions. The researchers note that the factors are not mutually exclusive, but interact, feeding back on one another. Often the measurements include an individual’s subjective assessment of quality of life before and after the onset of the disease or disorder and its treatment.

In the context of a broadened concept of health, there is clearly more to attaining and maintaining good health and quality of life than seeking regular medical and dental checkups and performing daily personal hygiene routines. Other factors that are important have been incorporated in a number of models of “determinants of health,” which are described in the next section. These models recognize that the determinants themselves are subject to change with changes in society and also vary in their salience over the lifetime of an individual. The concluding sections of the chapter illustrate this variability by examining oral health at successive stages of the life span, from childhood to old age. The vulnerabilities of selected subpopulations within each age group are highlighted, with particular emphasis on the plight of poor children and many older Americans.

HEALTH IN THE CONTEXT OF SOCIETY

Thinking about what makes people healthy has inspired philosophers and historians over the centuries. Following is a brief overview that points to commonalities among the models proposed.

Historical Models

As early as the fifth century B.C., Hippocrates considered it essential that physicians know each patient’s way of life, habitation, work, and dietary habits (Porter 1997). He counseled those who were considering a new city of residence to take into account the geography, water supply, and behavior of the citizens, specifically whether they drank and ate excessively, were lazy, or enjoyed exercise and hard work (Rose 1993).

Recent accounts of the history of medicine and public health similarly recognize the roles of environment, lifestyle, and the health care provider in determining health. Pine (1997) has described four phases in the history of public health. Phase 1, from the middle to the late 1800s, was characterized by urbanization and industrialization that significantly contributed to suboptimal living conditions for workers. Sanitary reforms were the hallmarks of public health achievement during this period. In addition, epidemiological studies began to demonstrate causal relationships between compromised health status and conditions such as malnutrition and poor hygiene.

The second phase, between 1880 and 1930, was characterized by advances in bacteriology and immunology. Increasingly, the prevention of disease was being applied to populations as well as individuals. The third phase, from 1930 through 1974, was a therapeutic period. The hospital became the essential base and focus for medical services, and medical treatment grew more complex. With the development

of vaccines and antibiotics, along with the success of surgical procedures, people began to rely on medical interventions as the source of health. The biomedical approach became paramount, and people began to believe that health was *delivered* to them by health professionals. The contributions of hygiene, sanitation, and living conditions to health were diminished. Doyal and Doyal (1984) point out that success depended on the maximal compliance of the patient.

Contemporary Models

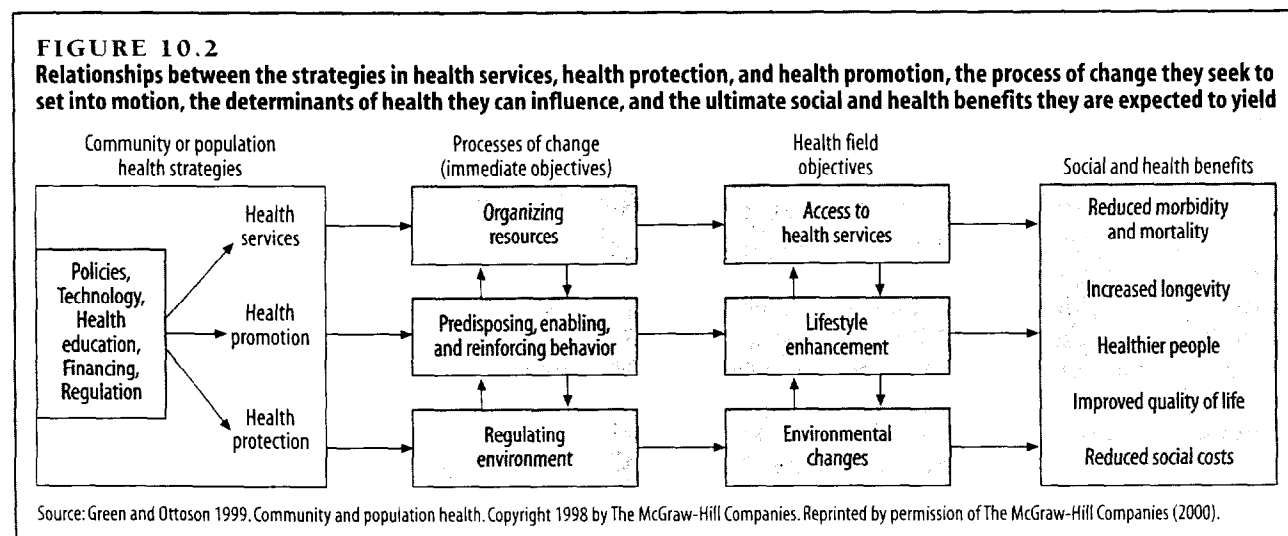
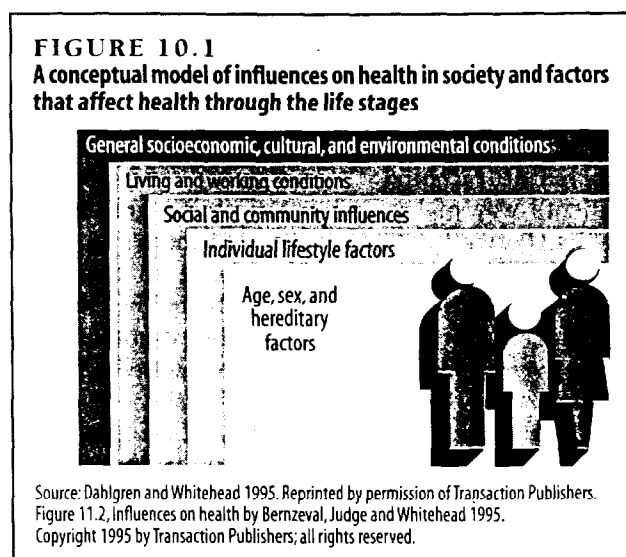
The fourth phase of public health, 1974 to the present, ushered in the modern era, referred to as “the new public health” (Ashton 1993). This phase developed out of a realization that health care costs were spiraling and there were few cures for an increasing burden of chronic diseases. The biomedical approach

alone could not solve all health problems. Rene Dubos (1979, 1990) stated that theories of specific etiology provided only a partial explanation for the development of diseases; they could not explain under what conditions a specific cause of disease could be determined and was able to flourish.

McKeown (1979) cited three factors he believed were responsible for the major reductions in disease: the environment, economics, and behavior. His analysis of data from numerous countries confirmed that the achievements of medicine alone could not explain improvements in health. The decline in mortality from many diseases, including tuberculosis, whooping cough, measles, scarlet fever, diphtheria, and smallpox, had begun well before the development of specific vaccines and therapies. He concluded that “the misinterpretation of the major influences, particularly personal medical care, on past and future improvements in health has led to misuse of resources and distortion of the role of medicine” (McKeown 1976).

Taking a similar critical view, Cochrane (1971), the physician in whose honor the Cochrane Collaboration of clinical trials was established, challenged the medical establishment to test medical procedures, including those long believed to be effective, with rigorous randomized controlled trials, paying particular attention to cost-benefit analyses. Long-held traditions of dental care have also been questioned, resulting in increased emphasis on clinical trials, systematic reviews of the oral health literature, and evidence-based practice (Chapter 8).

In 1974, Marc Lalonde, then Minister of Health of Canada, released a report that clearly articulated that human biology and health care organization are not the sole factors that determine health (Lalonde 1974). What is now known as the Lalonde Report, or



the Health Field Concept, emphasized that lifestyle and environment were of critical importance.

Lalonde defined four elements as determinants of health: human biology, lifestyle, environment, and the organization of health care. These elements were considered interdependent, and it was their dynamic interactions over the course of a lifetime that determined the level of health and well-being attained by an individual. As well, the elements and their interaction have implications for the level of health attained by larger aggregations of people—from neighborhoods to nations. Lalonde stated that most of society's efforts to improve health, and the bulk of direct health expenditures, have been focused on the fourth element—the organization of health care, yet the main causes of sickness and death are rooted in the other three elements.

At a subsequent WHO meeting in Ottawa, Canada, a set of five actions to promote health and quality of life, based on the four determinants, was proposed. Implementation of these actions clearly required going beyond the confines of a hospital, a medical office, or a home. Specifically, the Ottawa Charter for Health Promotion (WHO 1986) called for 1) creating supportive environments, 2) building healthy public policy, 3) strengthening community

action, 4) developing personal skills, and 5) reorienting health services.

In a model proposed by Dahlgren and Whitehead (1995), the individual is surrounded by lifestyle factors, social and community influences, living and working conditions, and general socioeconomic, cultural, and environmental conditions (Figure 10.1). Green and Ottoson (1999) integrate the Lalonde Health Field Concept into a framework of population health strategies, processes of change, determinants of health, and ultimate social and health outcomes (Figure 10.2).

Cohen and Gift (1995) acknowledge the role of multiple determinants and quote the medical historian, Henry Sigerist, who, in the mid-1940s, stated, "Health is promoted by providing a decent standard of living, good labor conditions, education, physical culture, means of rest, and recreation. The coordinated efforts of large groups are needed to this end, of the statesman, labor, industry, of the educator and of the [health care provider] who as an expert in matters of health must define norms and set standards" (Sigerist 1946).

McGoldrick (1997) provides an overview of several health behavior models in current use (Table 10.1). Some of these models have been applied to

oral-health-related behavioral research. Using the Health Belief Model in a study of dental patients, for example, Kuhner and Raetzke (1989) reported that motivation and perceived severity of the condition were the primary predictors of compliance with oral hygiene instruction. Perceived benefits and experience were also important.

The United States published a first set of national health goals for 1990 in 1979. The goals focused on the reduction of mortality in four different age groups and emphasized increased independence for older adults. Since then, national health goals have been established by the U.S. Department of Health and Human Services for each decade and published under the title "Healthy People." For Healthy People 2010 the broad goal is to increase the quality and years of healthy life. The conceptual framework, illustrated in Figure 10.3, features at the center determinants comparable to the

TABLE 10.1
Examples of theories and models in health behavior

Basic Theories	Authors	Major Elements
Group-Dynamic Model	Lewin, 1947, 1951	Role-playing approach
Social Learning Theory	Rotter 1954, Bandura 1969, 1977	Identification, reinforcement, feedback, and reward
Theory of Reasoned Action	Ajzen and Fishbein 1977, 1980	Attitude-behavior relations
Theory of Planned Behavior	Schifter and Ajzen 1985	Perceived behavioral control
Self-Efficacy Theory	Bandura 1982	Behavior determined by cognition of individuals about their behavior
Health Belief Model	Rosenstock 1966, 1974, Becker and Maiman 1975, Becker et al. 1974	Behavior determined by psychological readiness to take action
Health Action Model	Tones 1987, Tones et al. 1990	Interaction of knowledge, beliefs, values, attitudes, drives, and normative pressures
Theory of Social Behavior	Triandis 1979	Behavioral intention
Precede Framework	Green et al. 1980	Predisposing, reinforcing, and enabling causes in educational diagnosis and evaluation
Sense of Coherence Theory	Antonovsky 1979a,b	Salutogenic paradigm—focus on successful coping
Health Promotion Model	Pender 1987	Cognitive-perceptual factors

Source: Adapted from Pine 1997.

elements in the Lalonde Report. They include the interaction of individual biology, behavior, and the social and physical environment amidst policies, interventions, and access to quality health care.

The United States has invested heavily in elements of human biology and health care organization, but the nation has also readily embraced the notion that lifestyles influence health. Physical fitness and self-care are concepts that mesh with the individualistic spirit of U.S. society. Much attention has been given to health education, behavior change, and “healthy living.”

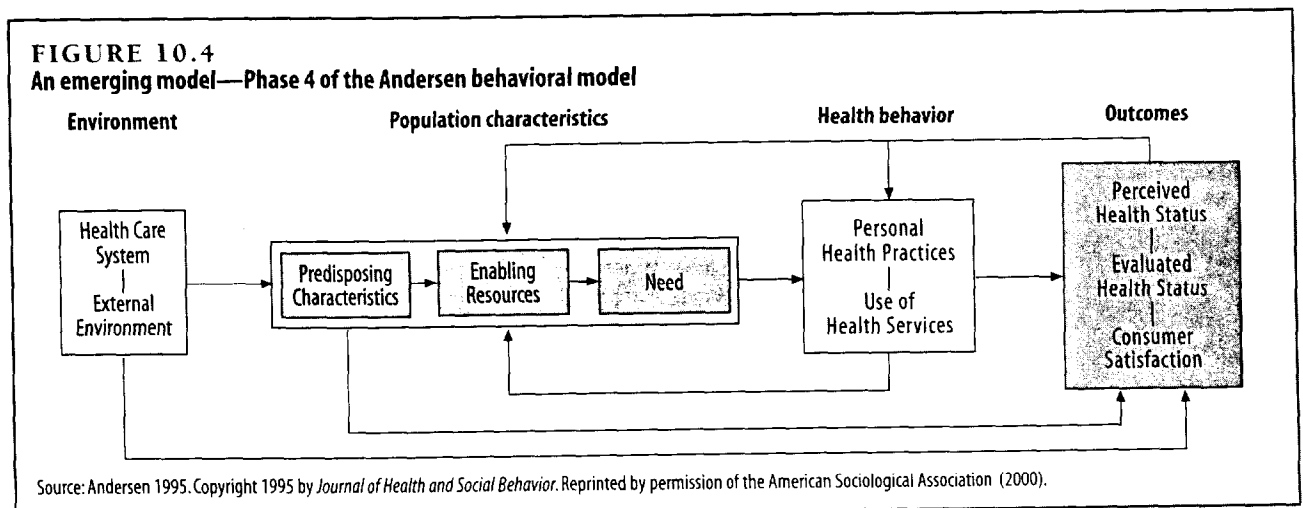
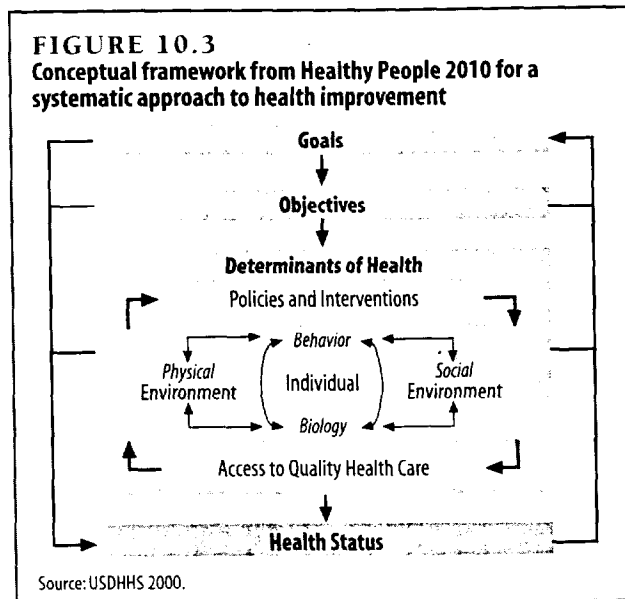
Applications to Oral Health

Within oral health, the self-care approach is best illustrated by the use of fluoride products for the reduction of dental caries. The successful adoption of

self-care regimens has been reinforced through the efforts of parents, caregivers, health educators, health professionals, advertisers, manufacturers, and early childhood programs such as Head Start that include oral health initiatives.

The Andersen Model. The oral health research community has begun to assess the behavior/lifestyle determinants of oral health as well. Andersen and colleagues point out that over the years concepts of health behavior have broadened from the biologic to the psychosocial (Andersen et al. 1988, Gochman 1988). Andersen has been a pioneer in the development of models of health determinants. The most recent refinement of the Andersen Behavioral Model (Figure 10.4) proposes that interactions among four major categories are critical to understanding the determinants of health. The first is the environment, described as the broader context in which populations live and behaviors occur. It includes the external environment and health care systems. The second category, population characteristics, includes three subsets: predisposing characteristics such as sociodemographic features, enabling resources such as those that enable the individual to pursue and achieve good health, and the need for care, which is defined by the individual’s perceptions of necessary preventive interventions or treatments. The third category is the health behaviors themselves, and the fourth category, outcomes, includes perceived and evaluated health status and consumer satisfaction (Andersen 1995).

In weighing the contributions of the various determinants of oral health, Andersen et al. (1995) suggest that the external environment, relating to both specific and general health, is the primary determinant of oral health behaviors and outcomes. Oral-health-specific environmental determinants



range from positive factors such as water fluoridation to negative factors such as lack of food policies to deal with frequent sugar and carbohydrate intake. They define general environmental factors as those that deal with the relative wealth of the society, general economic "climate," and the political and societal norms that affect the delivery of oral health services.

Workplace Effects. Several researchers (Karasek and Theorell 1990, Marmot and Theorell 1988, Marmot et al. 1984, Syme 1996) have found an association between the level of control and flexibility people have in their work setting and the types of health conditions they develop and the subsequent levels of severity of those conditions. Abegg et al. (1999) looked at the relationship between oral hygiene performances and levels of flexibility of work schedule. They found a highly statistically significant relationship between flexibility of work schedule and tooth-cleaning frequency, range of oral hygiene aids used, and level of dental plaque. These associations remained even after adjusting for age, sex, socioeconomic status, and marital status (Abegg et al. 1999).

Effects of Income Inequality

Investigators are also studying how socioeconomic status affects oral health (Chapter 4). The degree of income inequality between the richest and the poorest within a country, state, or neighborhood contributes to the overall health of the population (Kawachi et al. 1997, Kennedy et al. 1996). There is conjecture from this research that increased income inequality leads to decreased levels of social cohesion and trust, or what has been described as a "disinvestment in social capital" (Kawachi et al. 1997). This is defined as "features of social organization, such as civic participation, norms of reciprocity, and trust in others, that facilitate cooperation for mutual benefit." Results of other studies indicate that lower levels of social trust are associated with higher rates of coronary heart disease, cancer, stroke, and infant mortality. However, study of healthy versus unhealthy communities is a relatively new field and offers an opportunity for oral health to be included. Additional research is needed to determine the attributes of a community that either favor or diminish the health of residents, what factors influence their development, how attributes can be changed to improve the health of a community, and how communities can build social capital. There are indicators of differentials in oral health status when poor and nonpoor populations are compared.

Across numerous indicators, the poor are more likely to have oral diseases, disorders, and conditions. Poor children are less likely to have dental sealants. In addition, the poor are less likely to visit a dentist or dental hygienist in the course of a year. The differentials in oral health status between the poor and nonpoor cross the life span and are major social indicators of the current status of oral health in America today and provide a challenging baseline against which improvements can be measured.

CHANGING VULNERABILITIES THROUGHOUT LIFE

As all models of health determinants recognize, the health of individuals and of society at large is not static. Vulnerabilities and risks for diseases and disorders change over a lifetime and are affected by chance events as well as deliberate actions of individuals and communities, of the sort proposed in the Ottawa Charter. The remainder of the chapter describes how oral health plays out across major life stages and identifies selected aspects of biology, behavior, environment, and the organization of health care that affect oral health. The plight of vulnerable subpopulations, in particular, children and older Americans, are highlighted. The information presented includes data from national surveys, such as those presented in Chapter 4, as well as studies of convenience populations.

Children

In general, society gives special attention to the developing years of childhood, acknowledging that much of what happens to affect the health of a child bears directly on the health and well-being of the adult that child will become. In the case of oral health, there is enough known about health promotion and disease prevention to improve the oral health and well-being of *all* children, beginning with prenatal care. Adequate nutrition during pregnancy, including adequate folate intake, avoidance of substances of abuse and therapeutic agents that have teratogenic potential, and the elimination and control of microbial infections in the mother increase the likelihood of an infant's healthy start. (See Box 10.1 on the effects of nutrients on oral health.) As more becomes known about how the health of mothers and other caregivers can affect the oral health of children, additional services may be warranted during the prenatal period (Chapter 5). Subsequent nurturing of the infant includes the home and health professional care necessary to promote health and interventions that

limit the infant's exposure to infections that contribute to oral diseases.

Throughout the first two decades of growth and development, children and young people are deeply influenced by the social and environmental opportunities and constraints imposed by families, commu-

nities, and society. Although every healthy newborn has the potential for success and good health, there are profound disparities in children's experiences and opportunities, which often manifest in inequities in oral and general health, education, and well-being. Many children achieve excellent oral health—sound

BOX 10.1 **The Role of Nutrition in Oral Health**

General malnourishment impairs normal growth, development, and maintenance of the body's tissues and organs and impairs immune responses and wound healing. Reduced resistance of oral tissues to disease can lead to increased colonization by oral pathogens and more sustained and severe oral infections. Clinical signs of malnutrition often appear first in the oral cavity.

Craniofacial Development. Adequate maternal intake of folic acid during pregnancy has been shown to be essential in preventing neural tube defects (incomplete fusion of the neural tube in embryo), which results in spinal cord defects after birth (Botto et al. 1999). Folic acid also appears to be important in preventing clefting syndromes (in which there is incomplete fusion in utero of paired labial or palatal tissues at the midline) (Tolarova and Harris 1995).

Tooth Development. Protein/calorie malnutrition and deficiencies in ascorbic acid, vitamins A and D, calcium, phosphorus, and iodine affect the human dentition (DePaola et al. 1999). These deficiencies in development can lead to tooth defects after teeth erupt, manifesting as enamel hypoplasia and hypomineralization, either of which can increase susceptibility to dental caries. Premature and very-low-birth-weight infants frequently show enamel defects (Seow 1987). Since tooth enamel is acellular, and hence not subject to turnover and repair, enamel defects in development are permanent (Jonasson et al. 1999, Jeffcoat 1998, Talbot and Craig 1998, Payne et al. 1999).

Supporting Bone. Adequate calcium intake, along with vitamin D and other essential vitamins and minerals, is needed not only to build but also to maintain healthy teeth and bones. The teeth are supported in the jaws by projections of maxillary and mandibular trabecular bone known as alveolar processes. When teeth are lost, alveolar bone resorbs, reducing the height of the bony ridge supporting the teeth. When serum calcium levels fall, withdrawal of calcium from alveolar bone to meet other tissue needs may precede calcium withdrawal from skeletal bone elsewhere in the body. Hence reduction in alveolar bone mass may be an early indication of skeletal osteopenia (reduced bone volume) or frank osteoporosis (Jeffcoat 1998, Jeffcoat et al. 2000).

Oral Soft Tissues. Oral mucosa undergoes rapid turnover. In particular, the gingival lining between the gums and the teeth (the sulcular epithelium) is replaced every 3 to 7 days. Thus the tissue requires a steady supply of nutrients to support DNA, RNA, and protein synthesis (Alfano 1976). Diets poor in folate, ascorbic acid, iron, and zinc are associated with increased permeability and decreased integrity of the sulcular epithelium. Protein, vitamins A and C, and zinc are important for synthesis of connective tissue (largely collagen), which constitutes part

of the attachment apparatus supporting the teeth in the jaw (Alvares and Siegel 1981, Vogel et al. 1986). The classic signs of scurvy, caused by severe vitamin C deficiency, include gingival bleeding, tooth mobility, and loss of connective tissue attachment. Painful oral lesions, including inflammation and cracks at the corners of the mouth and vertical fissuring of the lips, are changes associated with riboflavin, iron, or protein deficiency. Inflammation, a burning sensation, and tenderness of the tongue or palate are associated with deficiencies in B-complex vitamins, protein, or iron.

Oral Defense Mechanisms. Chronic deficiencies of ascorbate and iron may impair the function of white blood cells (especially polymorphonuclear leukocytes) in moving to sites of infection and initiating immune defense mechanisms. Zinc is a component of many enzyme systems and is also important in leukocyte activity (Hsu et al. 1991).

General Oral Health. Foods rich in fiber aid digestion and stimulate salivary secretion. Salivary flow is important in initiating the digestion of starch, in facilitating food tasting and swallowing, and in ensuring a ready supply of components in the oral cavity that protect and maintain the oral tissues.

Nutrients Associated with Specific Diseases

Dental Caries. The role of sugars and other carbohydrates is critical. Nearly all carbohydrates have caries-promoting properties. Most sweet foods contain a mixture of sugars (predominantly sucrose) and starches, which can be fermented by cariogenic bacteria to dissolve tooth mineral. Bacteria also use sucrose to generate glucans—sticky extracellular molecules that promote their attachment to tooth surfaces. The physical consistency, frequency of consumption, and the order in which foods are eaten affect cariogenicity. For example, following a sweet with a nonsweet food such as an aged cheese may counteract the acid attack on enamel. The presence of calcium and phosphates in the cheese also is beneficial (Jensen 1999, Rugg-Gunn 1993). Caries in tooth roots is produced by the same process as in tooth crowns and involves the same dietary etiology (Papas et al. 1995), but may occur more rapidly because the root mineral, cementum, and underlying dentin are more soluble than enamel.

Periodontal Disease. Several studies have implicated deficiencies in ascorbate and folate with severity of gingivitis (Leggot et al. 1991, Pack 1984), but in general the role of nutrients in periodontal disease appears to be related to conditions that lead to increases in dental plaque, impaired host defenses, and weakened integrity of the periodontal tissues. More recently, surveys indicate that calcium intake for a large segment of the population is below recommended daily amounts (NHANES III) and that reduced calcium intake is associated with greater levels of periodontal disease in both men and women

(Nishida et al. 2000). This is consistent with the findings of Krall et al. (1997), who showed that calcium supplementation in postmenopausal women with deficient calcium intake protected against tooth loss.

Oral and Pharyngeal Cancers. High intakes of pickled vegetables, salted meat and fish, spicy foods, charcoal-grilled meat, and beverages served at very high temperatures have been found to be associated with oral cancers in some countries (Winn 1995). Malnutrition has also been found in association with the diagnosis of oral and pharyngeal cancers (Bassett and Dobie 1983). Whether the malnutrition was the cause or the effect is not clear.

Oral Cancer Prevention. A consistent finding across numerous studies is that a diet high in fruit and vegetables is associated with a reduced risk of oral cancer even when smoking and alcohol intake are taken into account (Steinmetz and Potter 1996). In a case-control study, risk of second primary tumors in oral and pharyngeal cancer patients was reduced in those with a high vegetable intake (Day et al. 1994). Fruits and vegetables contain fiber, carotenoids, and vitamin C, which may be important in cancer chemoprevention. Vitamin C may act as an antioxidant, protecting cell membranes and DNA from oxidative damage. Lack of vitamin C may interfere with collagen synthesis and permit tumor growth. Green leafy vegetables contain lutein, a carotenoid, xanthophyll, an antioxidant, and folic acid. Folic acid deficiency may interfere with DNA methylation and DNA repair (Winn 1995). The only prospective cohort study of diet and oral cancer (>25,000 persons in Maryland) showed that high serum total carotenoids and alpha tocopherol (vitamin E) reduced the risk of oral cancer, but high serum gamma tocopherol and selenium increased cancer risk (Zheng et al. 1993). The use of retinoids and β -carotene in controlled therapeutic doses shows protective effects. Fewer new primary tumors in persons with previous oral cancers and reversal or reduction in size of premalignant lesions have been reported (Khuri et al. 1997, Papadimitrakopoulou and Hong 1997). For example, high doses of 13-*cis*-retinoic acid, though causing significant toxicities, have been effective in the treatment of oral leukoplakia (Hong et al. 1990).

References

- Alfano MC. Controversies, perspectives and clinical implications of nutrition in periodontal disease. *Dent Clin North Am* 1976;20:519-48.
- Alvares O, Siegel I. Permeability of gingival sulcular epithelium in the development of scorbutic gingivitis. *J Oral Pathol* 1981;10:40-8.
- Bassett MR, Dobie RA. Patterns of nutritional deficiencies in head and neck cancer. *Otolaryngol Head Neck Surg* 1983;91:119-25.
- Botto LD, Moore CA, Khoury MJ, Erickson JD. Neural-tube defects. *N Engl J Med* 1999;341:1509-19.
- Day GL, Shore RE, Blot WJ, McLaughlin JK, Austin DL, Greenberg RS, Liff JM, Preston-Martin S, Sarkar S, Schoenberg JB, et al. Dietary factors and second primary cancers: a follow-up of oral and pharyngeal cancer patients. *Nutr Cancer* 1994;21:223-32.
- DePaola DP, Faine MP, Palmer CA. Nutrition in relation to dental medicine. In: Shils ME, Olson JA, Shike M, Ross CA, editors. *Modern nutrition in health and disease*. Baltimore: Williams & Wilkins; 1999. p. 1099-124.
- Hong WK, Lippman SW, Itri LM, Karp DD, Lee JS, Byers RM, Schantz SP, Kramer AM, Lotan R, Peters LJ, et al. Prevention of secondary primary tumors with isothetincin in squamous-cell carcinoma of the head and neck. *N Engl J Med* 1990;328:15-20.
- Hsu DJ, Daniel JC, Gerson SJ. Effect of zinc deficiency on keratins in buccal epithelial cells. *Arch Oral Biol* 1991;36:759-63.
- Jeffcoat MK. Osteoporosis: a possible modifying factor in oral bone loss. *Ann Periodontol* 1998;3:312-21.
- Jeffcoat MK, Lewis CE, Reddy MS, Wang CY, Redford M. Postmenopausal bone loss and its relationships to oral bone loss. *Periodontol* 2000; in press.
- Jensen ME. Diet and dental caries. *Dent Clin North Am* 1999;43:615-33.
- Jonasson G, Kiliaridis S, Gunnarsson R. Cervical thickness of the mandibular alveolar process and skeletal bone mineral density. *Acta Odontol Scand* 1999;57:155-61.
- Khuri FR, Lippman SM, Spitz MR, Lotan R, Hong WK. Molecular epidemiology and retinoid chemoprevention of head and neck cancer. *J Natl Cancer Inst* 1997;89:199-211.
- Krall EA, Dawson-Hughes B, Hannan MT, Kiel DP. Postmenopausal estrogen replacement and tooth retention. *Am J Med* 1997;102:536-42.
- Leggott PJ, Robertson PB, Jacob RA, Zambon JJ, Walsh M, Armitage GC. Effects of ascorbic acid depletion and supplementation on periodontal health and subgingival microflora in humans. *J Dent Res* 1991;70:1531-6.
- Nishida M, Grossi SG, Dunford RG, Ho AW, Trevisan M, Genco RJ. Calcium and risk for periodontal disease. *Periodontol* 2000;70(7):in press.
- Pack AR. Folate mouthwash: effects on established gingivitis in periodontal patients. *J Clin Periodontol* 1984;11:619-28.
- Papadimitrakopoulou VA, Hong WK. Retinoids in head and neck chemoprevention. *Proc Soc Exp Med* 1997;216:283-90.
- Papas AS, Joshi A, Palmer CA, Giunta JL, Dwyer JT. Relationship of diet to root caries. *Am J Clin Nutr* 1995;61(Suppl):425S-9S.
- Payne JB, Reinhardt RA, Nummikoski PV, Patil KD. Longitudinal alveolar bone loss in postmenopausal osteoporotic/osteopenic women. *Osteoporos Int* 1999;10:34-40.
- Rugg-Gunn AJ. Nutrition, diet and dental public health. *Community Dent Health* 1993;10(Suppl 2):47-56.
- Seow WK, Humphrys C, Tudehope DI. Increased prevalence of developmental defects in low birth-weight, prematurely born children: a controlled study. *Pediatr Dent* 1987;9(3):221-5.
- Steinmetz KA, Potter JD. Vegetables, fruit and cancer prevention: a review. *J Am Diet Assoc* 1996;96:1027-37.
- Talbot L, Craig BJ. Osteoporosis and alveolar bone loss. *Probe* 1998;32:11-3.
- Tolarova M, Harris J. Reduced recurrence of orofacial clefts after periconceptual supplementation with high dose folic acid and multivitamins. *Teratology* 1995;51:71-8.
- Vogel RI, Lamster IB, Wechsler SA, Macedo B, Hartley L, Macedo JA. The effects of megadoses of ascorbic acid on PMN chemotaxis and experimental gingivitis. *J Periodontol* 1986;57:472-9.
- Winn DM. Diet and nutrition in the etiology of oral cancer. *Am J Clin Nutr* 1995;61(Suppl):437S-45S.
- Zheng W, Blot WJ, Diamond EL, Norjus EP, Spate V, Morris JS, Comstock GW. Serum micronutrients and the subsequent risk of oral and pharyngeal cancer. *Cancer Res* 1993;53:795-8.

teeth, firm gums, healthy soft tissues, well-functioning bites, and beautiful smiles—but many do not.

One in every four U.S. children today is born into poverty (U.S. Bureau of the Census 1998b) with all of its associated barriers and constraints. Poverty is a key indicator of poor oral health status among children (Litt et al. 1995). Poor children suffer twice as much dental caries as their more affluent peers (Vargas et al. 1998). Studies have shown that the children with the most advanced oral disease are primarily found among America's most vulnerable groups: the poor, American Indians and other minorities, homeless and migrant populations, children with disabilities, and children with HIV (Isman and Isman 1997).

If untreated, oral diseases in children frequently lead to serious general health problems and significant pain, interference with eating, overuse of emergency rooms, and lost school time (Edmunds and Coye 1998). It has been estimated that 51 million school hours per year are lost because of dental-related illness alone (Gift 1997).

The Institute of Medicine reports that 70 percent of U.S. children are generally healthy and require only regular preventive and intermittent medical services. Twenty percent experience chronic problems, which may impose significant limitations on their ability to function effectively and require regular treatments for their conditions. Only the remaining 10 percent suffer from severe chronic conditions necessitating intensive health services (Edmunds and Coye 1998).

Similarly, the vast majority of America's children today enjoy excellent oral health, but a significant subset of children experience a high level of oral disease. Although it is no longer unusual to see children smiling with a full set of unmarred teeth, millions of other children have little to smile about. For them, the daily reality is persistent dental pain, endurance of dental abscesses, inability to eat comfortably or chew well, embarrassment at discolored and damaged teeth, and distraction from play and learning.

Like asthma, learning difficulties, and social problems, dental caries is highly correlated with low income, limited education, and social disadvantage. In this regard, it may serve as a sentinel disease for other pediatric conditions that are related to inadequate diet and hygiene and to family conditions and a social environment that do not support healthy lifestyles.

Some oral conditions, like other childhood illnesses, affect children randomly, regardless of social or economic status. Such conditions include cleft lip and palate and other craniofacial developmental dis-

orders, malocclusion, and unintentional injuries. Other oral conditions in children such as mucosal lesions may be a sign of risk behaviors such as tobacco use. All oral conditions may be exacerbated in children with other special health care needs.

Adults concerned about the health of children, particularly low-income and minority children, are regularly confronted by the reality and consequences of unmet oral health care needs. Although often viewed as innocuous by those who enjoy excellent dental health or have ready access to dental care, dental and oral problems impact the very life experience of affected children. Chronically poor oral health is associated with diminished growth in toddlers (Acs et al. 1992, Ayhan et al. 1996) and compromised nutrition (Acs et al. 1999). Dental disease in children also takes a personal and social toll. Observing disadvantaged inner-city schoolchildren, Kozol (1991) noted, "although dental problems don't command the instant fears associated with low birth weight, fetal death, or cholera, they do have the consequences of wearing down the stamina of children and defeating their ambitions."

In addition to the millions of children with extreme dental problems, many times more encounter more modest disease. For example, the review of the Healthy People 2000 objectives found that more than half of all second graders, children aged 6 to 8, still experience cavities (USDHHS 1997). Dental caries remains the single most common disease of childhood that is neither self-limiting, like the common cold, nor amenable to a simple course of antibiotics, like an ear infection (Edelstein and Douglass 1995).

The numbers of poor and minority children are increasing faster than other socioeconomic subsets of U.S. children (Waldman 1996), and dental caries is common in these children. Twenty-five percent of these children have never visited a dentist before entering kindergarten (USDHHS 1997), despite widespread understanding that the dental caries process is established before age 2 and the recommendation of experts that children as young as 1 may benefit from a dental visit (AAPD 1997, Green 1994, USDHHS 2000). Parents are consistently concerned about the dental needs of their children (Simpson et al. 1997), and studies conducted in hospital emergency rooms have found extensive dental needs among children (Sheller et al. 1997, Unkel et al. 1989, Wilson et al. 1997). Dental care has recently been noted as the most prevalent unmet health need among American children (Newacheck et al. 2000). These conditions are evident despite the advances in the oral health sciences and the growing capacity of

oral health care providers to prevent common pediatric oral diseases.

Children with disabilities present unique problems and are at increased risk for oral infections, delays in tooth eruption, periodontal disease, enamel irregularities, and moderate-to-severe malocclusion (Isman and Isman 1997). Their exposure to certain medications and therapies, special diets, and their difficulty in maintaining daily hygiene further compromise their oral health (Casamassimo 1996). Also, access to professional care is a particular problem for these children (see Chapter 4). Guides for dental professionals serving children with special health care needs are under development (USC 1999).

The Role of Insurance in Children's Oral Health

Disparities also occur in access to care. Medical insurance is a strong predictor of access to dental care. Children with no medical insurance are 2.5 times less likely than insured children to receive dental care (Bloom et al. 1992, Monheit and Cunningham 1992, Newacheck et al. 1997). Children with no dental insurance were 3.0 times more likely to have an unmet dental need than their counterparts with either public or private insurance (Newacheck et al. 2000, Waldman 1998). Dentists daily observe that insured children are more likely to obtain comprehensive, continuous, and coordinated care and are more likely to be followed regularly for semiannual preventive visits. It has long been recognized that dental plans with low cost-sharing requirements are likely to improve the oral health of young people, especially those with the poorest oral health (Bailit et al. 1985).

Children's general health also affects access to dental care. Children with "fair or poor" general health have nearly twice the unmet dental needs as children with "good or excellent" health, according to their parents (Simpson et al. 1997). As income rises, unmet treatment needs drop off dramatically. Children from families with annual incomes of \$10,000 to \$20,000 have 10 times more unmet dental needs than children whose families earn more than \$50,000 per year (Simpson et al. 1997).

White children are more likely than children in other ethnic and racial groups to have private dental insurance coverage. When last surveyed nationally in 1989, about half (52 percent) of white children had dental insurance, compared to only 39 percent of black children and 32 percent of Mexican American children. As family incomes increase, children are more likely to be covered by dental insurance (USDHHS 1992).

In the United States, most health insurance is provided through the workplace, and about 60 percent of children are covered by private health insurance through their parents' plans (U.S. Bureau of the Census 1998a). A smaller percentage, about 31 percent, enjoy dental insurance as well. There are at least 2.6 children without dental insurance for each child without medical insurance (Vargas et al. 2000).

Over the last decade, employer-based coverage for children has eroded, while publicly funded health insurance through Medicaid and the State Children's Health Insurance Program (SCHIP) has expanded to cover over 25 percent of all children (U.S. Bureau of the Census 1998a). The Congressional Budget Office estimates that 2.5 million children will be insured through SCHIP. However, even with this increase many children will remain without dental coverage.

Properly funded dental insurance works. When commercial-style, state-funded dental coverage became available to modest-income families in western Pennsylvania, the percentage of previously uninsured children (uninsured for more than 6 months) who saw a dentist during one year of coverage increased from 30 to 64 percent. The percentage of parents who reported that their child had a regular source of dental care increased from 51 to 86 percent. The percentage of parents who claimed that their children had unmet or delayed dental needs decreased from 52 to 10 percent. In addition, the number of dental visits fell as children's acute and episodic care decreased and they began programs of regular preventive and maintenance care (Lave et al. 1998).

Publicly Funded Insurance for Children

Medicaid. Although publicly funded programs such as Medicaid have succeeded dramatically in providing a "medical home" and regular medical care to children from low-income families (Newacheck et al. 1997), Medicaid's record of ensuring regular access to dentists and providing effective dental care is less successful. Fewer than one in five Medicaid-covered children received a single preventive dental visit during a recent year-long study period, according to the U.S. Inspector General (USDHHS 1996). The study indicated that three fourths of states provided preventive services to fewer than 30 percent of eligible children, and no state provided preventive dental care to more than 50 percent of all eligible children. More disturbing is the finding that few Medicaid children who receive dental care get any services beyond a cleaning and fluoride treatment, despite their need for dental repair and fillings (Solomon 1998).

Federal legislation enacted over three decades ago established a guarantee of dental care to Medicaid-eligible children through the Early and Periodic Screening, Diagnostic and Treatment Service (EPSDT; P.L. 90-284). Final regulations, effective in early 1972 (U.S. Bureau of the Census 1998a), ensure comprehensive dental services—prevention, diagnosis, and treatment for “teeth and associated structures of the oral cavity and disease, injury or impairment that may affect the oral or general health of the recipient”—and promise children access to dental services of sufficient “amount, duration, and scope” to ensure oral health. Federal law also requires provision of enabling services such as transportation and translation. In addition, revisions to the Social Security Act in 1989 (OBRA 89) made several changes to EPSDT services. States are now required to set a distinct periodicity schedule for the provision of dental services after consultation with recognized dental organizations involved in child health care. States are also required to provide any medically necessary dental service coverable under Medicaid to an EPSDT eligible child even if the service is not available to individuals age 21 and older under the Medicaid state plan. Despite these laws and regulations, inadequate funding, chronically poor payments to dentists, administrative burdens, and beneficiary utilization patterns have limited the effectiveness of this program (USDHHS 1996).

Increasingly, states are electing to purchase dental care for low-income populations through managed care organizations rather than to pay providers directly for Medicaid. As states take on the role of purchasers of care rather than claims payers, their focus has turned to a concern for health outcomes. However, participation of dentists in managed care programs is low (AAPD 1997, ADA 1998b, NADP 1998), and the effort to move dental Medicaid care into managed care programs may further constrain the availability of care.

A 1998 survey of state Medicaid authorities by the National Conference of State Legislatures reported that, on average, only 16 percent of dentists in the 35 responding states participate actively in Medicaid (i.e., were compensated more than \$10,000 in the preceding 12 months for dental care to Medicaid-enrolled patients). In 24 of these 35 states, fewer than 20 percent of active dentists participate actively (Guiden 1998). The study also raised awareness that common Medicaid payment rates for five typical children's dental procedures rarely exceed 65 to 70 percent of dentists' usual fees (Guiden 1998), a percentage that represents dentists' typical overhead

costs in delivering those services (ADA 1998b). A 1998 federally sponsored national meeting, “Building Partnerships to Improve Children's Access to Medicaid Oral Health Services,” also identified inadequate payments to dentists among multiple barriers in Medicaid program administration. Barriers identified by the conference were categorized as financing and funding issues, Medicaid policies and administrative procedures, supply and distribution of providers, parental valuation of oral health, and lack of a systematic approach to identifying and promoting successful interventions (Spizak and Holt 1999).

Medicaid expenditures for dental care are low. On average, state Medicaid agencies contribute only 2.3 percent of their child health expenditures to dental care (Yudkowsky and Tang 1997), whereas nationally, the percentage of all child health expenditures dedicated to dental care is more than 10 times that rate, almost 30 percent (Lewit and Monheit 1992). A 1998 actuarial study of health care costs for children (AAP 1998) calculated that 21 percent of expenditures for a comprehensive package of health services (including inpatient, outpatient, mental, dental, vision, hearing, and pharmacy services, but excluding orthodontic care) should be dedicated to dentists' services. This study suggests that fully \$21.35 per child per month must be expended in order to meet the dental care needs of covered children. A similar study conducted by the Reforming States Group (1999) determined that \$17 to \$18 per child per month is a necessary expenditure for dental care, assuming that providers accept a modest discount on their fees when serving low-income children. In FY 1995, Medicaid expended only \$4.44 per enrolled child per month (Yudkowsky and Tang 1997).

Although states vary widely in the percentage of children covered by Medicaid and in the income levels they require for eligibility, all states must entitle child beneficiaries to comprehensive dental services under EPSDT. A review of 15 state oral health and dental access surveys (Tinanoff 1998) noted the following recurrent themes about Medicaid in relation to children's oral health:

- States show similar dental care issues for Medicaid-enrolled children: high disease prevalence, low provider participation, and insufficient funding.
- Children at the highest risk of having dental caries are the least likely to have access to regular dental care.
- Barriers to provider participation include low reimbursement rates in a health care environment that has high overhead; perception of administrative

problems with Medicaid programs; and patients who do not fit the expectations of the dentist.

- Medicaid payments for dental care account for less than 3 percent of total state Medicaid child health expenditures in these states.
- The percentage of EPSDT eligibles with a dental visit (an initial measure of access to care) fails to reflect the insufficiency of reparative care to meet children's acute dental health needs.
- Lack of access to dental services for Medicaid recipients is perceived as the greatest pediatric health care problem in many states.
- Untreated dental problems get progressively worse and ultimately require more expensive interventions, often in a hospital emergency room or operating room.

State Children's Health Insurance Program. Thirty years after enacting Medicaid, the U.S. Congress in 1997 addressed the lack of medical coverage for over 10 million additional children by passing the State Children's Health Insurance Program (SCHIP). The Congressional Budget Office anticipates that this program will extend health insurance to at least 2.5 million more children and in the process will identify many additional children who are eligible for, but not enrolled in, Medicaid. SCHIP complements the Medicaid program by providing health insurance to children whose family income is above Medicaid eligibility standards, generally up to 200 percent of the federal poverty level. SCHIP differs from Medicaid in that it is not an individual entitlement, and states have broad latitude in designing and implementing insurance programs for modest-income children.

The law provides no direct mandate regarding services to be covered beyond immunizations and well-baby, well-child care. Dental coverage is specifically cited as one of 28 services that can be funded with SCHIP dollars. Although states are not required to provide dental coverage, congressional report language and presidential pronouncements are explicit in emphasizing the need for dental care (ADA 1998a,b). Prior to signing the bill in August 1998, President Clinton stated, "it is important that we have an adequate benefit package for children, recognizing that there are some problems that children have in a way that is more profound than adults, including problems with vision, with hearing and with dental health." Upon signing the bill, he said, "Because we have acted, millions of children all across the country will be able to get medicine, and have their sight and hearing tested and see dentists and doctors for the first time."

States can elect to apply federal SCHIP funds to expand Medicaid or they may use one of four options to provide services under a separate SCHIP program: 1) develop a new state program based on benchmark coverage, which is state employee coverage; 2) provide coverage under the SCHIP using benchmark-equivalent health coverage, which requires the use of an actuarial report to determine that coverage is at least equivalent to one of the benchmark plans; 3) apply existing comprehensive state-based coverage available in New York, Florida, and Pennsylvania; and 4) seek Secretary-approved coverage. Only 2 (Delaware and Colorado) of 56 states and territories have not included substantial dental care for most children covered by SCHIP. States implementing SCHIP have expanded access to dental care services through a variety of mechanisms. Expanding coverage through Medicaid ensures that newly enrolled children are entitled to dental coverage, although these children face the same barriers as other Medicaid children, as discussed previously. Even with current levels of commercial dental insurance and improved access through Medicaid and the new SCHIP program, almost one quarter of children will remain without dental coverage.

The Social and Professional Environment for Prevention

Although science continues to reveal new opportunities to prevent disease and promote health, sufficient understanding already exists to significantly reduce common oral diseases for all children. One of the most critical findings is that effective prevention requires an early start.

The American Academy of Pediatric Dentistry (AAPD 1997), the American Dental Association (ADA 1997), and the Bright Futures health supervision consensus project (Green 1994) all recommend that a toddler be seen by a dental professional at 12 months of age for an initial examination and risk assessment for common oral diseases and injuries. This first visit provides an opportunity for parents to learn about multiple oral health issues—dental caries, periodontal health, injury prevention, dental development, oral habits, common soft tissue sores, and bite development—as well as how to promote their child's complete oral health (Nowak 1997). Despite professional guidance and a Healthy People 2000 goal that 90 percent of children be seen by a dentist before entering kindergarten, only 63 percent of children have a dental visit before starting school (USDHHS 1997).

Because growth and development is so predictable, it can be anticipated and guided through education and carefully timed interventions. Applied to oral health, "anticipatory guidance" allows parents, children, and institutions to learn the stages of oral, facial, and dental development and how to care for the next stage of development (Nowak and Casamassimo 1995). Tables 10.2, 10.3, 10.4, and 10.5 provide examples of the risk and risk reduction methods related to periodontal diseases, dental caries, malocclusion, and injury, respectively (Casamassimo 1996). Physical, behavioral, socioenvironmental, and disease and treatment-related factors are addressed.

Anticipatory guidance allows the parent, dental team, other health care providers, and institutions that care for the child to ensure a child's good oral health, avoiding preventable pitfalls and problems by knowing how a child's mouth changes over time. For example, prevention of early childhood caries requires guidance to caregivers before the child's teeth erupt to prevent or limit the transmission of microbial infections from mother to child and to promote appropriate feeding practices even *before* the child has any teeth in place (Grindefjord et al. 1995, Kohler et al. 1984, 1988, Li and Caufield 1995, Tanzer 1995). Similarly, anticipatory guidance for oral health extends to safeguarding a house to prevent oral burns and injuries and to teach parents about the dangers of foreign objects in the mouths of toddlers and preschoolers. Anticipating a young person's interest in sports requiring mouth guards or head protection, discouraging smoking and smokeless tobacco before they are first used, and encouraging teens to adopt hygiene practices that prevent periodontal disease initiation also are examples of guidelines that need to be addressed by all individuals and organizations responsible for the child.

There is promise for further eradication of common childhood dental and oral infections. Education regarding oral infections in mothers and caregivers can con-

tribute to the infant's or toddler's general and oral health. Current investigations suggest that pathogenic exposures can be limited, children's resistance to acquiring disease-causing bacteria can be enhanced, physical and chemical barriers to transmission can be erected, and early-stage disease can be reversed with medications. Importantly, there is no one-size-fits-all solution to disease prevention and suppression. Most acquired dental and oral disease of childhood is preventable. The challenge today is to bring the promise of prevention to the most vulnerable of our children and youth. Meeting the challenge will require enhancing programs such as the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC), Head Start, along with early child care, and community and school-based centers.

Families have the capacity to support healthy oral health practices, as well as to support and

TABLE 10.2
Risk and protective factors for periodontal diseases

Risk Factors	Risk Reduction Methods
Physical Examples	
Anatomical variations (e.g., frenum)	Surgical correction
Malpositioned and crowded teeth	Orthodontic care
Gingivitis	Treatment of disease
Puberty	Preventive measures to address oral effects
Pregnancy	Preventive measures to address oral effects
Mouthbreathing	Management of mouthbreathing
Genetic predisposition (e.g., Down or Papillon Lefevre syndrome)	Preventive intervention to minimize effects
Behavioral Examples	
Inadequate oral hygiene	Improved oral hygiene
Tobacco use	Tobacco cessation
Socioenvironmental Examples	
Poor oral health and hygiene	Access to care and improved oral hygiene
Poverty	Access to care
Disease- or Treatment-related Examples	
Injury	Use of age-appropriate safety measures and treatment of injury
Nutritional deficiencies (e.g., vitamin C)	Healthy eating habits
Metabolic disease (e.g., diabetes, hypophosphatasia)	Treatment of disease
Neoplastic disease (e.g., leukemia or its treatment)	Treatment of disease and preventive intervention to minimize effects
Infectious disease (e.g., HIV/AIDS)	Treatment of disease and preventive intervention to minimize effects
Medications (e.g., Dilantin)	Preventive intervention to minimize effects
Poor-quality restorations	Restoration of carious lesions
Unrestored carious lesions	Properly contoured and finished restorations

Source: Modified from Casamassimo 1996.

encourage behaviors conducive to health and well-being, no matter their income. Communities that recognize children's oral health as an important public good can provide resources and ensure services, ranging from sealant programs, school education, and fluoridation programs to candy-free aisles in grocery stores and merchant campaigns to combat teen smoking and drinking.

At the state and federal levels, however, the good intentions of legislation have fallen short of adequate implementation. Nevertheless, by linking the power of growth and development with health promotion activities, the nation has the potential to bring excellent oral health to all children.

Health promotion covers a spectrum of efforts: anticipating problems, preventing them from occurring, and suppressing them when they first occur. These efforts can be targeted to individual children or entire communities of children, particularly children at high risk for dental and oral problems.

Adolescents and Young Adults

Data regarding oral health during adolescence and young adulthood are not abundant. However, most teenagers and young adults live healthy and active lives. Indeed, these years represent a peak period of biological fitness. This also is a time when individuals are exposed to and begin behaviors that may place them at risk, such as tobacco and alcohol use and poor dietary practices. For 12- to 17-year-olds who use smokeless (spit) tobacco, for example, 34.9 percent of current snuff users and 19.6 percent of current chewing tobacco users had tobacco-related oral lesions (Tomar et al. 1997). (See Box 10.2 on the effects of tobacco on oral health.) Adolescents become more mobile, traveling independently in cars, motorcycles, and other vehicles, where the use of safety belts and helmets is needed. Sexual practices begin during this time, further exposing individuals to infections that predispose them to general and oral health problems. Ideally, the prevention of risk behaviors begins earlier in life, but this stage of life brings such a cascade of events that even the most informed and well-supported adolescent may find it difficult to adhere to practices recommended by caregivers and institutions.

This period of life also is marked by rapid change as individuals move from school to work to marriage and parenting, possibly relocating far from their birthplace. Many young persons who were fortunate to have health insurance lose their coverage after they leave college or are no longer "dependents." Health status is largely determined by lifestyle behaviors and socioeconomic factors reflecting education, career, and income.

About one third of 15-year-olds have experienced dental caries in their permanent teeth, and another 20 percent have untreated dental decay. Poor adolescents have higher disease rates and more untreated disease. Periodontal diseases, as defined by having 4 mm or more of attachment loss, are seen in about 3 percent of 18- to 24-year-olds, although it is in the

TABLE 10.3
Risk and protective factors for dental caries

Risk Factors	Risk Reduction Methods
Physical Examples	
Variations in tooth enamel; deep pits and fissures; anatomically susceptible areas	Sealants (if possible) or observation
Gastric reflux	Management of condition
High mutans streptococci count	Reduction of mutans streptococci
Special health needs	Preventive intervention to minimize effects
Previous caries experience	Increased frequency of supervision visits
History of baby bottle tooth decay	Increased frequency of supervision visits
Behavioral Examples	
Bottle used at night for sleep or "at will" while awake	Prevention of bottle habit and weaning from bottle by 12 months
Frequent snacking	Reduction in snacking frequency
Inadequate oral hygiene	Improved oral hygiene
Eating disorders, including self-induced vomiting (bulimia)	Referral for counseling
Socioenvironmental Examples	
Inadequate fluoride	Optimal systemic and/or topical fluoride
Poor oral health and hygiene	Access to care and improved oral hygiene
Poverty	Access to care
High parental levels of bacteria (mutans streptococci)	Good parental oral health and hygiene
Diseases or Treatment-related Examples	
Special carbohydrate diet	Preventive intervention to minimize effects
Frequent intake of sugared medications	Alternate medications or preventive intervention to minimize effects
Reduced saliva flow from medication or irradiation	Saliva substitutes
Orthodontic appliances	Good oral hygiene for appliances

Source: Modified from Casamassimo 1996.

TABLE 10.4
Risk and protective factors for malocclusion

Risk Factors	Risk Reduction Methods
Physical Examples	
Congenital absence of teeth	Early intervention
Mouthbreathing	Management of mouthbreathing
Variations in development (e.g., tooth eruption delays and malpositioned teeth)	Early intervention
Muscular imbalances	Early therapy
Familial tendency for malocclusion	Early intervention
Conditions associated with malocclusion (e.g., cleft lip/palate)	Early intervention
Behavioral Examples	
Nonnutritive sucking habits	Elimination of habit
Disease- or Treatment-related Examples	
Injury	Use of age-appropriate safety measures (e.g., car safety seats, safety belts, stair gates, mouth guards) and treatment of injury
Acquired problem from systemic condition or its therapy	Dental intervention as a part of medical care
Loss of space due to caries	Early intervention for caries
Musculoskeletal conditions (e.g., cerebral palsy)	Dental intervention as a part of medical care
Skeletal growth disorders (e.g., renal disease)	Dental intervention as a part of medical care

Source: Modified from Casamassimo 1996.

TABLE 10.5
Risk and protective factors for injury

Risk Factors	Risk Reduction Methods
Physical Examples	
Lack of protective reflexes	Referral for appropriate therapy
Poor coordination	Referral for appropriate therapy
Protruding front teeth	Orthodontic care
Behavioral Examples	
Failure to use safety measures appropriate for infant/child/adolescent (e.g., car safety seats, stair gates, mouth guards, safety belts)	Use of age-appropriate safety measures
Participation in contact sports	Use of protective gear
Socioenvironmental Examples	
Substance abuse in family	Referral for counseling
Substance use by child or adolescent	Referral for counseling
Child abuse or neglect	Referral for counseling
Multiple family problems	Referral for counseling
Disease- or Treatment-related Examples	
Overmedication	Adjustment of medications
Hyperactivity	Management of condition

Source: Modified from Casamassimo 1996.

adolescent years that early-onset periodontitis is first diagnosed. Young non-Hispanic blacks have twice the proportion of periodontal disease than either white or Mexican Americans aged 30 to 49 years. Complete tooth loss is low in this age group, with only an estimated 0.4 percent of individuals aged 18 to 34 years having no teeth (see Chapter 4).

These years also mark the period of life when intentional and unintentional injuries take their greatest toll. Because many of these injuries affect the oral-facial region, they have special relevance to oral health. In particular, the example of oral-facial sports injuries illustrates the roles of behavior and socioeconomic environment as determinants of health, as well as pointing to several actions, such as use of protective head gear and mouth guards, that can serve as correctives.

Midlife Adults

Adults between 35 and 65 have been aptly called “the sandwich generation”—caring simultaneously for aging parents and dependent children, while trying to maintain their own health, careers, and family structure. This population cohort is growing in numbers in parallel with the ever-increasing numbers of the elderly. Although many older Americans will be self-sufficient for the rest of their lives, about one third will require higher levels of care because of chronic or terminal illness.

The demographic nature of these middle-aged adults is complex. In many families, both spouses work and have moved from their birthplaces. Many others have divorced, remarried, moved again, lost or changed jobs, and experienced a variety of midlife crises. Adding to the

BOX 10.2**The Effects of Tobacco on Oral Health**

The use of tobacco products—cigarettes, cigars, pipes, and smokeless (spit) tobacco products (snuff and chewing tobacco)—has emerged as a major preventable risk factor for a number of oral diseases and disorders.

Oral and Pharyngeal Cancers

Cigarettes. Tobacco smoke contains over 4,000 compounds, some of which are carcinogenic, toxic, or mutagenic (USDHHS 1989). An extensive review of the literature has clearly established a causal relationship between cigarette smoking and oral cancer (USDHHS 1982, 1989). Indeed, about 90 percent of oral cancer deaths are attributable to smoking (Shopland 1995, USDHHS 1989), and smoking cessation can significantly reduce the risk (USDHHS 1990).

Smokeless (Spit) Tobacco. These products are causally linked to oral and pharyngeal cancers (IARC 1985, Nash 1986, USDHHS 1986). About 30 carcinogens have been found in spit tobacco, including tobacco-specific N-nitrosamines, benzo[alpha]pyrene, and formaldehyde (Hoffman and Djordjevic 1997). Spit tobacco users have an oral cancer risk 4 to 6 times that of nonusers (Blot et al. 1988, Winn et al. 1981). Characteristic mucosal lesions are associated with spit tobacco use (Axéll et al. 1976, Holmstrup and Pindborg 1988, Peacock et al. 1960, Pindborg and Renstrup 1963) and can be found even among adolescent users (Greer and Poulson 1983, Offenbacher and Weathers 1985, Poulson et al. 1984, Tomar et al. 1997b, Wolfe and Carlos 1987). They are considered potentially premalignant (USDHHS 1986).

Cigars and Pipes. Cigar smoke contains the same toxic and carcinogenic compounds found in cigarette smoke (Hoffmann and Hoffmann 1998). A recent review of case-control and cohort studies also shows a consistent elevation in risk for oral and pharyngeal cancers among cigar smokers, with cigar smokers having 2 to 22 times the risk of non-smokers of cigars (USDHHS 1998). The risk of oral and pharyngeal cancers increases with the number of cigars smoked per day and the depth of inhalation.

Although data for pipe smoking and oral cancer risk are more limited than data for use of other forms of tobacco, relative risk estimates from longitudinal studies are similar for pipe smokers and cigarette smokers (USDHHS 1982, 1989).

Periodontal Diseases

Reviews of the literature have long implicated cigarette smoking as a risk factor for periodontal diseases. More recent studies such as Grossi et al. (1994, 1995) showed that smoking was a major risk factor for periodontal disease in a group of 1,500 adults. Measured either by radiographic bone height or probing attachment level, and after adjusting for age, sex, socioeconomic status, and plaque and calculus levels, the investigators found that smokers were 7 times more likely to develop periodontal disease than nonsmokers. They also found a direct linear dose-response relationship between the level of smoking, assessed by pack years (number of cigarettes smoked per day times years smoked), and destructive periodontitis. Smoking is also

a prognostic indicator: current smokers are at a significantly greater risk for further loss of periodontal attachment than are nonsmokers, with an odds ratio of 5.4 (95 percent confidence interval of 1.5 to 19.5).

Mechanisms explaining the association suggest that smoking depresses immune responses (Holt 1987, Sasagawa et al. 1985), including diminishing white blood cell activity (Gala et al. 1984, Kenney et al. 1977). Toxic and vascular effects as well as effects on the subgingival flora are also suggested. In addition, smokers do not heal as well as nonsmokers after periodontal disease therapy and experience less reduction in levels of periodontal pathogens (Grossi et al. 1997). The negative effects of smoking can be reversed with cessation of tobacco use. After 10 years, former smokers appear to be no more likely than nonsmokers to have severe loss of periodontal attachment (Tomar and Marcus 1998).

Spit Tobacco. Reports indicate that oral tobacco use results in gingival recession at the usual site of snuff or chewing tobacco placement. In a study of adolescent males, Offenbacher and Weathers (1985) found that 60 percent of users had gingival recession, compared with 14 percent of nonusers.

Dental Caries

The strongest evidence for an association of tobacco use and risk for dental caries relates to the use of chewing tobacco and increased risk for root caries. The causative factor relates to the sugar content of the product. Several popular brands of chewing tobacco have high levels of fermentable sugars (between 30 and 60 percent by weight). In a cross-sectional study of older adults in North Carolina, chewing tobacco users had a higher number and percentage of root surfaces affected by caries than those who used other forms of tobacco or had never or formerly used tobacco (Tomar et al. 1997a). This finding was confirmed in an analysis of data from NHANES III (Tomar and Winn 1998).

Trends in Tobacco Use

In 1995, 47 million adults—25 percent of the U.S. adult population—were smokers (CDC 1997). This figure represents a steady decline from the 52 percent of the population reported to be smokers in 1965, the year following the release of the first Surgeon General's Report on Tobacco (Giovinio et al. 1995). The prevalence of smoking in women was 34 percent in 1965, 30 percent in 1979 (Giovinio et al. 1994), and 23 percent in 1995 (CDC 1997).

In contrast, cigarette smoking in adolescents has been increasing. Daily smoking among high school seniors increased from 17 percent in 1992 to 22 percent in 1996 (Johnston et al. 1997). High school students who reported smoking in the preceding month increased from 27.5 percent in 1991 (USDHHS 1994) to 36 percent in 1997 (CDC 1998).

Spit tobacco use has also increased. Sales of moist snuff—the most popular form of spit tobacco used by young people (Tomar et al. 1995)—have increased every year since the mid-1970s (FTC 1997, Maxwell 1992, USDA 1997). About 20 percent of male high school students reported using spit tobacco during the previous month (CDC

(continues)

BOX 10.2 continued

1996, Johnston et al. 1997). About 6 percent of adult males use spit tobacco (CDC 1993). Nearly all regular users are male.

Aggressive marketing has also led to explosive growth of sales and consumption of cigars. Between 1993 and 1997, cigar consumption increased nearly 50 percent (Gerlach et al. 1998). In 1997, 22 percent of high school students smoked at least one cigar in the preceding 30 days (CDC 1998).

Implications of Trends

For Oral and Pharyngeal Cancers. The increases in spit tobacco and cigar use among young people do not bode well for the oral and general health of coming generations of Americans. Over the past 35 years the decline in the incidence and mortality rates of oral cancer has been attributable to declines in cigarette smoking primarily in adult white males. Cigarette smoking among African American males over the same time period was higher. This practice contributed to the higher rates of oral cancer among black males during these years. However, recent studies indicate precipitous declines in smoking among black males, so that their smoking rates are approaching the rates seen in white males (USDHHS 1998). Indeed, figures on smoking among adolescent and younger African American adults have even been lower than those for their white counterparts. These trends could result in substantial reductions in the risk for oral cancer among African Americans, were they to continue. Unfortunately, there is recent evidence that cigarette smoking among African American high school students is increasing (CDC 1998).

For Periodontal Diseases. The growing popularity of cigar smoking may counter the declines in cigarette smoking and maintain the percentage of periodontal disease attributable to tobacco use.

References

Axell T, Mornstad H, Sundstrom B. The relation of the clinical picture to the histopathology of snuff dipper's lesions in a Swedish population. *J Oral Pathol* 1976;5:229-36.

Blot WJ, McLaughlin JK, Winn DM, Austin DF, Greenberg RS, Preston-Martin S, Bernstein L, Schoenberg JB, Stemhagen A, Fraumeni JF Jr. Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Res* 1988;48:3282-7.

Centers for Disease Control (CDC). Use of smokeless tobacco among adults—United States, 1991. *MMWR Morb Mortal Wkly Rep* 1993(a);42:263-6.

Centers for Disease Control and Prevention (CDC). Tobacco use and sources of cigarettes among high school students—United States, 1995. *MMWR Morb Mortal Wkly Rep* 1996;45(20):413-8.

Centers for Disease Control and Prevention (CDC). Cigarette smoking among adults—United States, 1995. *MMWR Morb Mortal Wkly Rep* 1997;46:1217-20.

Centers for Disease Control and Prevention (CDC). Tobacco use among high school students—United States, 1997. *MMWR Morb Mortal Wkly Rep* 1998;47:229-33.

Federal Trade Commission (FTC). 1997 smokeless tobacco report to Congress. Washington: Federal Trade Commission; 1997.

Gala D, Kreilick RW, Hoss W, Matchett S. Nicotine-induced membrane perturbation of intact human granulocytes spin-labeled with 5-doxylstearic acid. *Biochim Biophys Acta* 1984;778:503-10.

Gerlach KK, Cummings KM, Hyland A, Gilpin EA, Johnson MD, Pierce JP. Trends in cigar consumption and smoking prevalence. In: National Cancer Institute. Cigars: health effects and trends. Smoking and Tobacco Control Monograph 9. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health; 1998. p. 21-53. NIH Pub. no. 98-4302.

Giovino GA, Schooley MW, Zhu BP, Chrismon JH, Tomar SL, Peddicord JP, Merritt RK, Husten CG, Eriksen MP. Surveillance for selected tobacco-use behaviors—United States, 1900-1994. *MMWR Morb Mortal Wkly Rep* 1994;43(SS-3):1-43.

Giovino GA, Henningfield JE, Tomar SL, Escobedo LG, Slade J. Epidemiology of tobacco use and dependence. *Epidemiol Rev* 1995;17:48-65.

Greer RO, Poulson TC. Oral tissue alterations associated with the use of smokeless tobacco by teen-agers. Part I. Clinical findings. *Oral Surg* 1983;56:275-84.

Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, Norderyd OM, Genco RJ. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* 1994;65:260-7.

Grossi SG, Genco RJ, Machtei EE, Ho AW, Koch G, Dunford R, Zambon JJ, Hausmann EE. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol* 1995;66:23-9.

Grossi SG, Zambon J, Machtei EE, Schifferle R, Andreana S, Genco RJ, Cummins D, Harrap G. Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. *J Am Dent Assoc* 1997;128:599-607.

Hoffmann D, Djordjevic MV. Chemical composition and carcinogenicity of smokeless tobacco. *Adv Dent Res* 1997;11:322-9.

Hoffmann D, Hoffmann I. Chemistry and toxicology. In: National Cancer Institute. Cigars: health effects and trends. Smoking and Tobacco Control Monograph 9. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health; 1998. p. 55-104. NIH Pub. no. 98-4302.

Holmstrup P, Pindborg JJ. Oral mucosal lesions in smokeless tobacco users. *CA Cancer J Clin* 1988;38:134-41.

Holt PG. Immune and inflammatory function in cigarette smokers. *Thorax* 1987;42:241-9.

International Agency for Research on Cancer (IARC). Tobacco habits other than smoking; betel-quid and areca-nut chewing; and some related nitrosamines. IARC Working Group. Lyon, 1984 Oct 23-24. *IARC Monogr Eval Carcinog Risk Chem Hum* 1985 Sep;37:1-268.

Johnston LD, Bachman JG, O'Malley PM. Monitoring the future: questionnaire responses from the nation's high school seniors, 1995. Ann Arbor (MI): Survey Research Center, Institute for Social Research, University of Michigan; 1997.

Kennedy EB, Kraal JH, Saxe SR, Jones J. The effect of cigarette smoke on human oral polymorphonuclear leukocytes. *J Periodont Res* 1977;12:227-34.

- Maxwell JC Jr. The Maxwell consumer report: the smokeless tobacco industry in 1991. Richmond (VA): Wheat, Butcher, and Singer; 1992.
- Nash DB. Health implications of smokeless tobacco: a National Institutes of Health Consensus Development Conference. *Ann Intern Med* 1986;104:436-7.
- Offenbacher S, Weathers DR. Effects of smokeless tobacco on the periodontal, mucosal and caries status of adolescent males. *J Oral Pathol* 1985;14:169-81.
- Peacock EE, Greenberg BC, Brawley BW. The effect of snuff and tobacco on the production of oral carcinoma: an experimental and epidemiological study. *Ann Surg* 1960;151:542-50.
- Pindborg JJ, Renstrup G. Studies in oral leukoplakias. II. Effect of snuff on oral epithelium. *Acta Derm Venereol* 1963;43:271-6.
- Poulson TC, Lindenmuth JE, Greer RO. A comparison of the use of smokeless tobacco in rural and urban teenagers. *CA Cancer J Clin* 1984;34:248-61.
- Sasagawa S, Suzuki K, Sakatani T, Fujikura T. Effects of nicotine on the functions of human polymorphonuclear leukocytes *in vitro*. *J Leuk Biol* 1985;37:493-502.
- Shopland DR. Tobacco use and its contribution to early cancer mortality with a special emphasis on cigarette smoking. *Environ Health Perspect* 1995;103(Suppl 8):131-42.
- Tomar SL, Marcus SE. Cigarette smoking and periodontitis among U.S. adults. *J Dent Res* 1998;77(Spec No B):830 [abstract 1585].
- Tomar SL, Winn DM. Coronal and root caries among U.S. adult users of chewing tobacco. *J Dent Res* 1998;77(Spec No A):256 [abstract 1205].
- Tomar SL, Giovino GA, Eriksen MP. Smokeless tobacco brand preference and brand switching among US adolescents and young adults. *Tob Control* 1995;4(1):67-72.
- Tomar SL, Weintraub JA, Gansky SA. Coronal and root caries among long-term users of chewing tobacco. *J Dent Res* 1997a;76(Spec No):372 [abstract 2872].
- Tomar SL, Winn DM, Swango GA, Giovino GA, Kleinman DV. Oral mucosal smokeless tobacco lesions among adolescents in the United States. *J Dent Res* 1997b;76(6):1277-86.
- U.S. Department of Agriculture (USDA). Tobacco situation and outlook report. TBS-239. Washington: U.S. Department of Agriculture, Commodity Economics Division, Economic Research Service; 1997.
- U.S. Department of Health and Human Services (USDHHS). The health consequences of smoking: cancer. Rockville (MD): U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health; 1982. DHHS Pub. no. PHS 82-50179.
- U.S. Department of Health and Human Services (USDHHS). The health consequences of using smokeless tobacco: a report of the Advisory Committee to the Surgeon General, 1986. Washington: U.S. Department of Health and Human Services, Public Health Service; 1986. NIH Pub. no. 86-2874.
- U.S. Department of Health and Human Services (USDHHS). Reducing the health consequences of smoking: 25 years of progress. A report of the Surgeon General. Rockville (MD): U.S. Department of Health and Human Services, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1989. DHHS Pub. no. CDC 89-8411.
- U.S. Department of Health and Human Services (USDHHS). The health benefits of smoking cessation: a report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1990. DHHS Pub. no. CDC 90-8416.
- U.S. Department of Health and Human Services (USDHHS). Preventing tobacco use among young people: a report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1994.
- U.S. Department of Health and Human Services (USDHHS). Tobacco use among U.S. racial/ethnic minority groups: African Americans, American Indians and Alaskan Natives, Asian Americans and Pacific Islanders, and Hispanics. A report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1998.
- Winn DM, Blot WJ, Shy CM, Pickle LW, Toledo A, Fraumeni JF. Snuff dipping and oral cancer among women in the southern United States. *N Engl J Med* 1981;304:745-9.
- Wolfe MD, Carlos JP. Oral health effects of smokeless tobacco use in Navajo Indian adolescents. *Community Dent Oral Epidemiol* 1987;15:230-5.

demands of a spouse and children, the care of older parents contributes yet another strain to caregivers "in the middle." These caregivers are predominantly female and may be dependent on their own income. They may be single and faced with dealing with their own "passages" (Sheehy 1984).

The baby boomers will be the first U.S. generation to age while maintaining their natural dentition. They are the first to benefit from the caries preventive effect of widespread community water fluoridation and fluoride dentifrices. As a result, the baby

boomers bring to the aging process higher expectations about oral health throughout the lifecycle.

Maintaining the family's oral health may require as many individual solutions as there are sandwich generation members (Sanders 1997, Stern 1994, Warner 1995). Healthy lifestyle decisions combined with preventive measures at home will be as important as regular professional care.

In addition to their own oral hygiene practices, a key component of maintaining the oral health of midlife Americans is the availability of dental bene-

fits. Six of 10 full-time employees are offered dental benefits by their employers, according to a survey by the U.S. Bureau of Labor Statistics ([www.e-dental.com/Virtual Community for the Dental Industry](http://www.e-dental.com/VirtualCommunityfortheDentalIndustry), 12/30/99). These data are from a 1997 survey of firms with 100 or more employees in private nonagricultural industries and are representative of benefits available to 46 million workers. The dental benefits, one of the less prevalent benefits for employees, vary by occupation group and are higher for professional and technical employees (64 percent) than for blue-collar or service employees (56 percent). Among the estimated 22.6 million employees with employer-provided dental benefits, most employees (81 percent) receive their care from traditional fee-for-service plans; 11 percent, from preferred provider organizations; and 8 percent, from health maintenance organizations.

Ensuring the oral health of the middle-aged generation must take into account the shifting patterns of need and the family's ability to cope, the education and training of health care workers about geriatric and family issues, general comprehensive community education programs about aging, estate and taxation issues, housing, and social policies and programs that support all individuals in their quest for self-sufficiency and individual responsibility.

Older Americans

Continued growth of the population 65 and older will have profound effects on health care in the twenty-first century (National Institute on Aging 1997). By 1994 the number of persons 65 and older had grown to 33.2 million and represented 13 percent of the population. Although the total U.S. population is expected to increase by 42 percent over the next half century, the number of men and women 65 and older will increase by 126 percent, those 85 and older by 316 percent, and centenarians by 956 percent—nearly 10 times the present number.

The baby boom generation currently makes up almost one third of the U.S. population. By 2011, when these men and women reach 65, they will swell the ranks of older Americans and significantly burden health care programs and organizations responsive to the needs of older Americans (National Institute on Aging 1997). Although members of this generation can look forward to continued good oral and general health, the challenge will be in providing effective oral health care for those who are not in good health, especially the oldest old, and those with limited financial support.

Oral Health Status

Chapter 4 provides selected oral health data for older Americans as a whole. There is great heterogeneity in oral health status among older Americans. The extent and severity of oral conditions varies across subpopulations of this age group, and many have unmet treatment needs. Even so, older Americans are retaining their teeth more than ever before and hence remain subject to oral diseases and disorders (Douglass et al. 1998). Indeed, with more teeth at risk, there will be an increase in coronal and especially root caries among the elderly, as well as periodontal diseases and inadequate or absent prostheses (Burt 1992). Oral and pharyngeal cancers are primarily diagnosed in older Americans.

For a closer look at the oral health of both institutionalized and homebound elderly, Dolan and Atchison (1993) compiled data based on a comprehensive review of the literature. Although the long-term care population is easily accessible in large groups, oral examinations for research purposes can be challenging. Patient consent and antibiotic premedication are issues, as well as the fact that convenience samples must be used because many patients are unable to cooperate. The authors' summaries of oral health status and perceived needs based on the most comprehensive homebound and long-term care oral health surveys are shown in Tables 10.6 and 10.7, respectively.

Table 10.6 describes eight studies, with 31 to 289 patients, with edentulous rates ranging from 23.8 to 62 percent. In these studies use of dental services within the past year ranged from 8 to 100 percent. In a 1994 Home Health and Hospice survey, only 1 percent of patients reported having a dental visit during that year (Dey 1996). Forty-three to 83 percent of persons in six of the homebound studies in Table 10.6 recognized that they had dental problems.

In the long-term care studies listed in Table 10.7, 45 to 65 percent of those surveyed were completely without their natural teeth. One study found that 17 percent required immediate or emergency dental care. By any standards in the United States, a high degree of dental disease and dental care needs was recognized in all four studies presented.

Daily oral care is an important and easily neglected service that should be offered to this population. Unlike many of the inevitable declines the frail elderly face with their various diagnoses, the decline in oral health can be stayed with good daily oral care. Nursing staff participation in the daily oral care of long-term care patients is crucial. Mouth care is often

considered an unpleasant task and is often delegated to nursing auxiliaries, who have even less oral health training than registered nursing staff. Seventy percent of patients in long-term care facilities had unacceptable levels of oral hygiene (Kiyak et al. 1993, McIntyre et al. 1986).

Barriers to such needed care include lack of knowledge about oral care by the nursing staff, perceived lack of time for care, and lack of perceived need for daily oral care by both caregivers and patients. The resulting failure to provide daily oral

care will often doom oral health that had been previously well maintained.

Data on the oral health status of hospice patients are scarce. Although not all hospice patients are elderly, data from the 1994 Home and Hospice Care Survey showed that 19.8 percent of those in hospice care wore dentures. The terminally ill often suffer from taste alterations, oral soreness, oral dryness, and oral candidiasis or thrush (Aldred et al. 1991). In most cases, the caregiver will perform daily oral care and palliative oral care measures. Palliative care can

TABLE 10.6
Summary of published reports and abstracts on the oral health status and barriers to dental care for homebound elders

	Sample Size	Sample Description	Mean Age (years)	Dental Utilization	Percentage Edentulous	Findings
Steifel et al. 1979	64	Two nonprofit visiting nurse services, Seattle	Approx. 75	25% visit in past year	Approx. 60%	60% reported dental needs; dentate subjects more likely to seek care Barrier: transportation
Yellowitz et al. 1988 [abstract]	107	Recipients of visiting nurse services, Utah	NA	34% visit in past year	NA	>50% reported mouth discomfort, painful tongue, dry mouth, difficulty chewing
Kaste et al. 1989 [abstract], Marcus et al. 1989 [abstract]	289	Homebound, >65 years, recipients of home care services, Boston	82.5	50% with no visit in 10 years	62%	43% perceived dental need Barriers: transportation, cost, needed physical assistance
Aponte-Merced et al. 1990 [abstract]	50	Recipients of home health services, county health departments, Alabama	79.0	8% visit in past year 28% no visit in 20 years	59%	37% had dental complaint; 60% perceived dental need; 84% wanted treatment
Strayer et al. 1990 [abstract], Strayer et al. 1991 [abstract]	67	Clients of urban social service agency, 60% homebound	NA	Not reported	44%	80% perceived dental need Barriers: transportation, cost, physical impairments
Yellowitz et al. 1991 [abstract]	123	Recipients, veterans hospital-based home care, Denver and Minneapolis	72.2	40% visit in past year	33%	50% reported dental health fair/poor; 50-83% perceived dental need Barriers: 53% no perceived need; 25% had no dentist; 22% transportation; 22% cost
Strayer and Ibrahim 1991	31	Chart audit, patients treated at Ohio State University	74.8	100%	23.8%	59.7% had periodontal/preventive/operative needs;
Williams and Butters 1992		Statewide survey to identify the number of homebound persons in Kentucky	68.6	53.8% visit in past year	NA	27.3% prosthodontic needs; 46.8% surgical needs; 2.7% of Kentucky households have a homebound resident

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include oral moisturizers such as artificial saliva, ice chips, a water atomizer, daily oral-cleaning or swabbing, and, if needed, treatment for yeast to relieve oral pain.

Americans have the potential to experience a lifetime of oral health rather than a lifetime of oral restorative care. Each of the following questions can be applied to the major oral problems of the elderly,

TABLE 10.7
Summary of four published reports on the oral health status and barriers to dental care for nursing home residents

	Sample Description	Mean Age (years)	Dental Utilization	Percentage Edentulous	Findings
Empy et al. 1983	242 residents of 12 skilled nursing facilities; stratified random sample; Washington state	81	mean time since last dental visit: 4.9 years	65.3%	Needing denture treatment: urban, semirural, rural; 63, 46, 39%, respectively Mean number of decayed teeth: 1.8, 3.0, 2.4, respectively Mean number of periodontally involved teeth: 1.9, 0.7, 1.1, respectively 80% who did not intend to visit dentist felt "no need" Median age of dentures: 15.5 years
California Dental Association (CDA) 1986	286 residents of a stratified random sample of nursing homes	81	22% visit in past year	57%	17% had immediate dental needs Dentate residents: mean number teeth: 17 12.9% carious 7.0% fractured 49.6% periodontal disease 75.8% needed 1+ quadrants scaling Prosthodontic needs: 25% maxilla, 28% mandible Reasons for not seeking care: 52% felt no need; 24% transportation; 9% finances, 9% illness; 43% oral mucosal disease
Veterans Administration (VA) 1989	634 residents of six VA facilities: Florida, Illinois, Massachusetts; regional convenience sample	71	Not reported	50%	Dentate residents: 3.7 decayed coronal surfaces (DFS = 18.6) 4.8 decayed root surfaces (DFS = 6.5) Average periodontal attachment loss: 2.5 mm, 27% pockets >4 mm Prosthodontic needs: 35% maxilla, 28% mandible 40% denture-related oral lesions
Kiyak et al. 1993	1,063 residents of 31 nursing homes in Washington state	range 72 to 98	Not reported	44.8%	Dentate residents: oral problems: 72% poor oral hygiene 36% root caries 26% coronal caries 24% retained root tips 18% significant tooth mobility 11% swelling, soft tissue lesions 10% dry mouth Edentulous residents: oral problems: 46% loose dentures 18% sore or bleeding gums 15% poor oral hygiene 10% dry mouth 5.4% soft tissue lesions 63.8% had dental treatment needs

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including coronal and root caries, periodontal diseases, oral cancer, oral-facial pain, tooth loss, salivary gland dysfunction, and oral mucosal diseases:

- How do we best identify elders at greatest risk for oral diseases? Who is not at risk? How can we improve diagnostic accuracy? When is increased accuracy not related to improved outcomes?

- Can these diseases be prevented or delayed? Which measures are most effective? Which have the greatest benefit for the least cost?

- Once a person has the disease, which treatments are most effective?

When measuring effectiveness, care should be taken to consider the proximal outcomes, that is, effects at the tissue level, as well as ultimate outcomes, that is, how the overall effects of the treatment affect a person's ability to function and be a productive, contributing member of society.

An important consideration in treating oral health problems in the elderly is the relationship between oral and general health. Too often, oral health care is ignored or takes second place in light of the high prevalence of such chronic and life-threatening conditions as heart disease, stroke, cancer, osteoporosis, and diabetes. Yet the evidence presented in Chapter 5 speaks to associations linking oral infectious diseases such as periodontitis to the increased risk for cardiovascular, cerebrovascular, and lung disease, to exacerbations of diabetes, and as an early indicator of osteoporosis. In turn, to ignore oral health care in the course of cancer radiation and chemotherapy predisposes the patient to serious oral infections, mucositis, severe pain, bone loss, and potential abscesses. The 1988 Surgeon General's Workshop on Health Promotion and Aging stated that all health care providers should be educated in the importance of oral health to overall health and well-being (USDHHS 1988).

Insurance Issues. In light of the oral care needs of the elderly and their vulnerability to systemic diseases, the lack of dental insurance poses a serious barrier (Jones et al. 1990, Niessen 1984). Medicare funds cover only a negligible and select amount of care. Many elders lose their dental insurance at retirement (Niessen 1984). The situation may be worse for older women. Because women overall have lower incomes than men, lack of insurance and high copayments for dental services may represent formidable obstacles to care. In addition, women assume a disproportionate burden as caregivers for family members of all ages: the young, the sick, and the elderly (Niessen 1984). This often disrupts employment and, consequently, insurance coverage.

Thus, the majority of dental care rendered to older patients is paid for out of pocket. Medicaid programs fund dental care for low-income and disabled adults, including elders, in some but not all states (ADA 1998b, Jones et al. 1990), but reimbursements are scant, even in emergency situations. Where there is reimbursement, it is often low and slow, adding yet another disincentive for provision of oral care. Medicaid funds the costs of the majority of patients in long-term care, which means that they either have spent their life earnings or were in poverty prior to admission.

This lack of dental coverage is occurring at a time when more and more of the new elderly will be dentate and both want and need care (Ettinger and Beck 1982). Thus, funding dental care for elders is a major obstacle.

Social Services. Decreased functional status and increasing levels of dependence add barriers to dental care for elders. It will be increasingly important for community and social service programs to respond to older residents' needs for assistance, including transportation to meet their oral care needs. For example, programs administered by the Administration on Aging (AOA) that integrate oral health into general health programs for the elderly raise awareness about the benefits of good oral health and its contribution to nutritional status and quality of life (National Policy and Resource Center 1998). For patients in long-term care settings, access to dental care is even more problematic. Lack of adequate compensation has been a barrier to increasing the number of dentists who choose to pursue this type of dental practice.

Trends

Despite advances in modern medicine that have greatly increased life expectancy in the twentieth century, there will be an increase in the number of persons with acute and chronic diseases, including arthritis, diabetes, osteoporosis, and senile dementia (U.S. Bureau of the Census 1998b). As always, it is necessary to distinguish between healthy elders who age normally and remain active and community dwelling and the frail elderly (Niessen and Jones 1991).

Most community-dwelling elders take both prescription and over-the-counter drugs (Chrischilles et al. 1992). Approximately 30 percent of all medications prescribed in the United States are for persons over the age of 65, with an average of 8.1 medications per patient in a long-term care facility (Gurwitz et al. 1990, Lamy 1989). Seventy-five to 94 percent of

Factors Affecting Oral Health over the Life Span

patients taking medications are taking at least one drug that may have an oral side effect (Baker et al. 1991, Levy et al. 1988, Lewis et al. 1993). The most common of these side effects is dry mouth, or xerostomia. Others include abnormal homeostasis, soft tissue lesions or reactions, taste changes, altered host resistance, gingival overgrowth, burning oral sensations, increased caries due to high sugar content, and involuntary oral movements.

At any given time, approximately 5 percent of the population 65 and older live in a long-term care facility, and an estimated 43 percent of these elders will require long-term care placement at some point in their lives (Murtaugh et al. 1990). As discussed earlier, one result of elders' increased disability and dependency is that middle-aged family members are confronted with increased parental care concerns and needs (U.S. Bureau of the Census 1990).

Determining the oral health status of homebound and hospice populations is challenging. Statistics are reported by evaluating persons who seek services for either home or hospice care. Obviously, this underrepresents both populations by leaving out those who refuse, are not aware of, or do not qualify for services. As with long-term care, most homebound are women, although the average age is younger than for those in long-term care facilities. This may represent a step in the continuum of care before long-term care is necessary. Fifty-five percent

of women are hospice patients, and hospice patients are a much younger population than either the homebound or those in long-term care.

Table 10.8 lists the 10 chronic conditions seen most frequently in the frail elderly. These health problems are important in relation to oral health because they, or their treatments, may worsen oral health or in turn be worsened in the presence of oral disease (see Chapter 5). Long-term care residents have an average of 3.3 chronic conditions per person (Adams and Marano 1995).

Although it is difficult to evaluate dementia patients following strict research protocol, several studies have noted high caries rates, poor oral hygiene, and a high percentage with unmet dental needs (Chapman and Shar 1991, Gordon 1988, Jones et al. 1993). Patients with dementia depend heavily on caregivers to provide daily oral care, and dental care can be most challenging.

The Impact on Women

Redford (1993) examined the effects of biological, behavioral, and societal factors on women's oral and general health and treatment needs. Throughout their lives, American women report more acute symptoms, chronic conditions, and short- and long-term disabilities than men; women's activities are limited by health problems 25 percent more days each year than men's (Verbrugge 1984, 1990). The

TABLE 10.8
Most common diagnoses of frail elderly (≥ 65) in nursing homes, receiving home health (homebound) and hospice care by percentage of the population, 1994 to 1995

Rank	Nursing Home Resident	Homebound	Hospice
1	Diseases of circulatory system	Diseases of the circulatory system	Neoplasms
2	Mental disorders	Endocrine, nutritional, metabolic, and immunity disorders	Diseases of the circulatory system
3	Diseases of nervous system and sense organs	Diseases of musculoskeletal and connective tissue systems	Diseases of the nervous system and sense organs
4	Injury and poisoning	Injury and poisoning	Diseases of the respiratory system
5	Endocrine, nutritional, metabolic, and immunity disorders	Diseases of the respiratory system	All other diagnoses
6	Diseases of the respiratory system	Neoplasms	AIDS and infectious or parasitic diseases ^a
7	Diseases of the musculoskeletal and connective tissue systems	Ill-defined conditions	AIDS and infectious or parasitic diseases ^a
8	Diseases of the digestive system	Diseases of nervous system and sense organs	
9	Diseases of the genitourinary system	Disease of skin and subcutaneous tissue	
10	Neoplasms	Diseases of the digestive system	

^a Rates of the two categories are equal.

Sources: Data are from 1994 Home and Hospice Care Survey and 1995 National Nursing Home Survey (Dey 1996, 1997, Haupt 1997).

“gender gap” in physical disability widens with advancing age (U.S. Bureau of the Census 1990). Women in nursing homes or personal care facilities outnumber men three to one (NCHS 1991).

In the course of aging, significant numbers of women experience compromised functional status, physical confinement, medical conditions, and cognitive impairments. The literature indicates that these factors have placed women’s oral health at risk. At the same time, they may limit a woman’s ability to maintain oral hygiene self-care regimens, seek professional dental services, tolerate dental treatment, and comply with postoperative instructions (Gift 1998).

Pharmacologic regimens common among women can promote xerostomia, thereby increasing the risk of caries, periodontal diseases, and atrophic/disease changes in oral mucosa (Atkinson and Fox 1992). As a consequence of chemotherapy for breast cancer, women may suffer inflammation and ulceration of the oral mucosa, oral infection, hemorrhage, neurotoxicity, and salivary gland dysfunction (McCarthy and Skillings 1992, National Institutes of Health Consensus Development Conference Statement: Oral Implications of Cancer Therapies 1990).

ACHIEVING ORAL HEALTH THROUGHOUT LIFE

Each life stage brings a unique set of issues and considerations. Ultimately, this overview identifies the need for research on health services, health promotion, and disease prevention specific to populations at different life stages and throughout the life span. Our nation’s young and old exemplify the complexities of the individual, family, community, and institutional interactions that shape health and well-being. The middle years are not without complexities, but represent a

time during which employment and responsibility for caring for others play a critical role. Overlying the age spectrum are other sociodemographic factors that intensify the need to address each group and each health issue in a manner that optimizes health outcomes. In the overview of special populations presented in Chapter 4, the impact of race and ethnicity, socioeconomic status, and issues in relation to the health of women and individuals with disabilities clearly cut across all life stages. The nation’s social and welfare programs, the organization of our private systems of health care, and the values of the many cultures that make up America contribute to

TABLE 10.9
Summary: Healthy People 2010 objectives—oral health

Objective	Age(s)	2010 Baseline	2010 Goal
21.1 Reduce dental caries experience in children	2-4	18%	11%
	6-8	52%	
	15	61%	
21.2 Reduce untreated dental decay in children and adults	2-4	16%	9%
	6-8	29%	
	15	20%	
	35-44	27%	
21.3 Increase adults with teeth who have never lost a tooth	35-44	31%	42%
21.4 Reduce adults who have lost all their teeth	65-74	26% ^a	20%
21.5a Reduce gingivitis among adults	35-44	48%	41%
21.5b Reduce periodontal disease among adults	35-44	22%	14%
21.6 Increase detection of Stage I oral cancer lesions	all	35%	50%
21.7 Increase number of oral cancer examinations	40+	9%	35%
21.8 Increase sealants in 8-year-old first molars and in 14-year-old first and second molars	8	23% (a 1st)	50%
	14	15% (a 1st&2nd)	50%
21.9 Increase persons on public water receiving fluoridated H ₂ O	all	62%	75%
21.10 Increase utilization of oral health (OH) system	2+	44%	56%
21.11 Increase preventive dental services for poor children	2-17	20%	57%
21.12 Increase number of school-based Health Centers with OH component	K-12	developmental unknown	
21.13 Increase number of Community Health Centers and local health departments with OH component	all	56%	75%
21.14 Increase utilization of dental service for those in long-term facilities, e.g., nursing homes	all	17%	25%
21.15 Increase states with system for recording and referring orofacial clefts	all	23	51
21.16 Increase the number of states with state-based surveillance systems	all	0	51
21.17 Increase the number of state and local dental programs with public health trained directors	all	developmental unknown	

^aBased on self-report, National Health Interview Survey, 1996 (NCHS 1996).
Source: USDHHS 2000.

the current status of health, including oral health, and are the basis for further improvements.

The models described at the beginning of this chapter provide a structure for designing strategies to improve and promote health. Any one approach can be used as a framework for action. The Healthy People 2010 objectives provide a useful template for driving many age-specific and disease/condition-specific outcomes. The multiple oral-health-related objectives outlined there emphasize the importance of risk behaviors and comorbidities that need to be addressed in order to further improve oral, dental, and craniofacial health (USDHHS 2000) (Table 10.9).

Recurrent themes in this chapter and other parts of the report underscore the importance of access to health care and health care services, the adoption of healthy behaviors, and the role of individuals and all health care providers in contributing to oral health. Public policies, institutional care guidelines, and community programs can reinforce what individuals can do by providing a health-promoting environment. Toward that end, a recently published report from the Center for Policy Alternatives (Warren 1999) examines and recommends health policies and related actions to improve the oral health status of the poor and underserved. Focus is placed on five dimensions of oral health—finance, sustainability of services, capacity to provide services, cultural competency of care providers, and infrastructure to support professional practice. Policy recommendations and proposed action steps are presented in terms of the availability, accessibility, and acceptability of care. Dental care services are emphasized over other aspects of oral health maintenance, because much of the unmet need warrants dental services for prevention and treatment.

Health care providers, program administrators, local, state, and government administrators, educators, scientists, and leaders, among others, have proposed ways of promoting health and preventing disease that respond to the principal health determinants presented in the chapter. Thus, efforts can be directed toward changing the environment to make it more life-enhancing; establishing new public health policies; enhancing health literacy to encourage healthy behaviors and lifestyles; working at the microlevel of neighborhoods and communities on health-related measures; and orienting health care to meet the needs of a changing society.

Building on programs and structures already in place that have contributed to the improvements in oral health is essential. Further advances in the oral health of all Americans cannot be made unless the health needs of the underserved and vulnerable pop-

ulations are addressed. The inability of federal and state programs that are the primary source of funding for services to these populations, specifically, Medicaid, SCHIP, and Medicare, to cover and adequately reimburse for dental services has been duly noted. The current review of access to dental care by the Government Accounting Office should add to an earlier review of EPSDT and further address barriers to access and other issues that warrant attention. The Institute of Medicine (IOM) study on the extension of Medicare services to include medically necessary dental services is an additional source of recommendations to better address the health needs of vulnerable populations and enhance health overall (Field et al. 1999).

Other critical reviews of the problems entailed in addressing the nation's oral health needs and proposing solutions include the 1989 Public Health Service Workshop on the Oral Health of Mothers and Children (USDHHS 1989). Recommendations covered the areas of public education, professional education, coalitions, advocacy and collaboration, health policy, and data collection, evaluation, and research. These recommendations formed the basis for the 2000 Surgeon General's Workshop on Children and Oral Health. Similarly, the 1988 Surgeon General's Workshop on Health Promotion and Aging (USDHHS 1988) provided guidance for steps to be taken to improve the oral health of the nation's elders, all of which are still relevant. This workshop provided the impetus to add objectives on oral health status in nursing homes to Healthy People 1990.

Ideally, organizations and agencies working together can resolve the issue of barriers to care. Concentrated efforts such as those focused on improving the access of children to Medicaid oral health services by the Health Care Financing Administration, Health Resources and Services Administration, American Dental Association, and National Center for Education in Maternal and Child Health are an example of how national organizations can unite to make a difference. Still, activities are needed at the local community level. The efforts of Milgrom and colleagues provide one such example for children eligible for Medicaid, with a focus on early childhood caries (Milgrom and Weinstein 1999, Milgrom et al. 1999). In implementing these efforts, however, the capacity of current national, state, and local programs as well as legislative mandates to meet the oral health needs of all Americans must be reviewed and strengthened, as necessary.

FINDINGS

- The major factors that determine oral and general health and well-being are individual biology and genetics; the environment, including its physical and socioeconomic aspects; personal behaviors and lifestyle; access to care; and the organization of health care. These factors interact over the life span and determine the health of individuals, population groups, and communities—from neighborhoods to nations.
- The burden of oral diseases and conditions is disproportionately borne by individuals with low socioeconomic status at each life stage and by those who are vulnerable because of poor general health.
- Access to care makes a difference. A complex set of factors underlies access to care and includes the need to have an informed public and policymakers, integrated and culturally competent programs, and resources to pay and reimburse for the care. Among other factors, the availability of insurance increases access to care.
- Preventive interventions, such as protective head and mouth gear and dental sealants, exist but are not uniformly used or reinforced.
- Nursing homes and other long-term care institutions have limited capacity to deliver needed oral health services to their residents, most of whom are at increased risk for oral diseases.
- Anticipatory guidance and risk assessment and management facilitate care for children and for the elderly.
- Federal and state assistance programs for selected oral health services exist; however, the scope of services is severely limited, and their reimbursement level for oral health services is low compared to the usual fee for care.

REFERENCES

- Abegg C, Marcenes W, Croucher R, Sheiham A. The relationship between tooth cleaning behaviour and flexibility of working time schedule. *J Clin Periodontol* 1999;26:448-52.
- Acs G, Lodolini G, Kaminsky S, Cisneros GJ. Effect of nursing caries on body weight in a pediatric population. *Pediatr Dent* 1992;14:302-5.
- Acs G, Shulman R, Ng MW, Chussid S. The effect of dental rehabilitation on the body weight of children with early childhood caries. *Pediatr Dent* 1999;21(2):109-13.
- Adams PF, Marano MA. Current estimate from the National Health Interview Survey, 1994. National Center for Health Statistics; 1995. *Vital Health Stat* 10 (193).
- Ajzen I, Fishbein M. Attitude-behavior relations: a theoretical analysis and review of empirical research. *Psychol Bull* 1977;84:888-918.
- Ajzen I, Fishbein M. *Understanding attitudes and predicting social behavior*. Englewood Cliffs (NJ): Prentice-Hall; 1980.
- Aldred MJ, Addy M, Bagg J, Finlay I. Oral health in the terminally ill: a cross sectional pilot survey. *Spec Care Dentist* 1991;11(2):59-62.
- American Academy of Pediatric Dentistry (AAPD). *Infant oral health care guidelines*. *Pediatr Dent* 1997; 1970.
- American Academy of Pediatrics (AAP). *An analysis of the costs to provide health care coverage to the children and adolescent population aged 0 to 21 years*. Elk Grove Village (IL): American Academy of Pediatrics; 1998.
- American Dental Association (ADA). *Dental practice survey. Your child's teeth*. Chicago: American Dental Association; 1997.
- American Dental Association (ADA), Edelstein BL. *Oral health services in the Child Health Insurance Program, Children's Dental Health Project Washington DC and the American Dental Association Task Force on CHIP resource packet*. Chicago: American Dental Association; 1998a.
- American Dental Association (ADA), Survey Center. *Survey of state dental programs in Medicaid*. Chicago: American Dental Association; 1998b.
- Andersen R. Revisiting the behavioral model and access to medical care: does it matter? *J Health Soc Behav* 1995;36:1-10.
- Andersen R, Davies JK, Kickbush I, et al. *Health behavior research and health promotion*. Oxford: Oxford Medical Publications; 1988.
- Andersen R, Marcus M, Mahshigan M. A comparative systems perspective on oral health promotion and disease prevention. In: Cohen LK, Gift HC, editors. *Disease prevention and oral health promotion: socio-dental sciences in action*. Copenhagen: Munksgaard; 1995. p. 307-40.
- Antonovsky A. *Health, stress and coping*. San Francisco: Jossey-Bass; 1979a.
- Antonovsky A. The sense of coherence as a determinant of health. In: Matarazzo JD, Weiss SM, Herd JA, et al., editors. *Behavioral health. A handbook of health enhancement and disease prevention*. New York: John Wiley and Sons; 1979b. p. 114-29.
- Ashton J. Institutes of public health and medical schools: grasping defeat from the jaws of victory? *J Epidemiol Community Health* 1993;47:165-8.
- Atkinson JC, Fox PC. Salivary gland dysfunction. *Clin Geriatr Med* 1992 Aug 8(3):499-511.
- Ayhan H, Suskan E, Yildirim S. The effect of nursing or rampant caries on height, body weight and head circumference. *J Clin Pediatr Dent* 1996;20:209-12.

- Bailit H, Newhouse J, Brook R, Duan N, Goldberg G, Hanley J, Kamberg C, Spolsky V, Black A, Lohr K. Does more generous dental insurance coverage improve oral health? *J Am Dent Assoc* 1985;110(5):701-7.
- Baker HA, Levy SM, Chrischilles EA. Medications with dental significance; usage in a nursing home population. *Spec Care Dentist* 1991;11:19-25.
- Bandura A. Principles of behaviour modification. New York: Holt, Rinehart and Winston; 1969.
- Bandura A. Social learning theory. London: Prentice Hall; 1977.
- Bandura A. Self-efficacy mechanism in human agency. *Am Psychol* 1982;37:122-47.
- Becker MH, Maiman LA. Sociobehavioral determinants of compliance with health and medical care recommendations. *Med Care* 1975;13:10-24.
- Becker MH, Drachman RH, Kirscht JP. A new approach to explaining sick-role behavior in low-income populations. *Am J Public Health* 1974 Mar;64(3):205-16.
- Bloom B, Gift HC, Jack SS. Dental service and oral health, United States, 1989. *Vital Health Stat* 1992;10:1-95.
- Burt BA. Epidemiology of dental diseases in the elderly. *Clin Geriatr Med* 1992 Aug;8(3):447-59.
- Casamassimo P. Bright futures in practice: oral health. Arlington (VA): National Center for Education in Maternal and Child Health; 1996.
- Chapman PJ, Shar RM. Normative dental treatment needs of Alzheimer patients. *Austr Dent J* 1991;36(2):141-4.
- Chrischilles EA, Foley DJ, Wallace RB, Lemke JH, Semla TP, Hanlon JT, Glynn RJ, Ostfeld AM, Guralnik JM. Use of medications by persons 65 and over: data from the established populations for epidemiologic studies of the elderly. *J Gerontol A Biol Sci Med Sci* 1992;47(5):M137-44.
- Cochrane AL. Effectiveness and efficiency; random reflections on health services. London: Nuffield Provincial Hospital Trust; 1971.
- Cohen LK, Gift HC. Introduction. In: Cohen LK, Gift HC. Disease prevention and oral health promotion: socio-dental sciences in action. Copenhagen: Munksgaard; 1995. p. 17-31.
- Dahlgren G, Whitehead M. Tackling inequalities: a review of policy initiatives. In: Benzeval M, Judge K, Whitehead M, editors. Tackling inequality in health: an agenda for action. London: Kings Fund Institute; 1995.
- Dey AN. Characteristics of elderly home health care users: data from the 1994 National Home and Hospice Care Survey. Advance data from vital and health statistics. Hyattsville (MD): National Center for Health Statistics; 1996. Abstract no. 279.
- Dey AN. Characteristics of elderly nursing home residents: data from the 1995 National Nursing Home Survey. Advance data from vital and health statistics. Hyattsville (MD): National Center for Health Statistics; 1997. Abstract no. 289.
- Dolan TA, Atchison KA. Implications of access, utilization and need for oral health care by the non-institutionalized and institutionalized elderly on the dental delivery system. *J Dent Educ* 1993 Dec;57(12):876-87.
- Douglass CW, Ostry L, Shih A. Denture usage in the United States: a 25 year prediction. *J Dent Res* 1998;77(SI A):209. Abstract no. 829.
- Douglass J, Wei Y, Zhang BX, Tinanoff N. Dental caries in preschool Beijing and Connecticut children as described by a new caries analysis model. *Community Dent Oral Epidemiol* 1994;22:94-9.
- Doyal L, Doyal L. Western scientific medicine: a philosophical and political prognosis. In: Birke L, Silvertown J, editors. More than the parts: biology and politics. London: Pluto Press; 1984. p. 82-109.
- Dubos R. Mirage of health. New York: Harper Colophon; 1979.
- Dubos RJ. The world of Rene Dubos: a collection of his writings. In: Piel G, Segerber O, editors. New York: Henry Holt and Company; 1990.
- Duperon DF. Early childhood caries: a continuing dilemma. *J Calif Dent Assoc* 1995;23(2):15-25.
- Edelstein BL, Douglass CW. Dispelling the myth that 50 percent of U.S. schoolchildren have never had a cavity. *Public Health Rep* 1995;110:522-30.
- Edmunds M, Coye MJ, editors. America's children: health insurance and access to care. Committee on Children, Health Insurance and Access to Care, Division of Health Care Services, Institute of Medicine. Washington: National Academy Press; 1998.
- Ettinger R, Beck J. The new elderly: what the dental profession can expect. *Spec Care Dentist* 1982;2:62-9.
- Field MJ, Lawrence RL, Zwanzier L, editors. Extending Medicare coverage for preventive and other services. Committee on Medicare Coverage Extensions, Institute of Medicine. Washington: National Academy Press; 1999.
- Gift HC. Oral health outcomes research—challenges and opportunities. In: Slade GD, editor. Measuring oral health and quality of life. Chapel Hill: University of North Carolina Department of Dental Ecology; 1997.
- Gift HC. Issues of aging and oral health promotion. *Gerodontology* 1998 Oct;4(5):194-206.
- Gochman DS. Health behavior: emerging research perspectives. New York: Plenum Press; 1988.
- Gordon SR. Survey of dental need among veterans with severe cognitive impairment. *Gerodontology* 1988; 4:158-9.
- Green LW, Ottoson JM. Community and population health. 8th ed. Boston: McGraw-Hill; 1999.
- Green L, Kreuter MW, Deeds SG, Partridge KB. Health education planning. A diagnostic approach.

- Mountain View (CA): Mayfield Publishing Company; 1980.
- Green M, editor. Bright futures: guidelines for health supervision of infants, children, and adolescents. Arlington (VA): National Center for Education in Maternal and Child Health; 1994.
- Grindefjord M, Dahllof G, Nilsson B, Modeer T. Prediction of dental caries development in 1-year-old children. *Caries Res* 1995;29:343-8.
- Guiden M. Dental health for kids moves to the forefront. *State health notes* 19(280). Forum for State Health Policy Leadership, National Conference of State Legislatures; 1998 Jul 6.
- Gurwitz JH, Soumerai SB, Avorn J. Improving medication prescribing and utilization in the nursing home. *J Am Geriatr Soc* 1990;38:542-52.
- Haupt BJ. Characteristics of patients receiving hospice care services: United States, 1994. Advance data from vital and health statistics. Hyattsville (MD): National Center for Health Statistics; 1997. Abstract no. 282.
- Isman R, Isman B. Access to oral health services in the U.S. 1997 and beyond. Chicago: Oral Health America; 1997.
- Jones JA, Adelson RA, Niessen LC, Gilbert GH. Issues in financing dental care for the elderly. *J Public Health Dent* 1990;50:268-75.
- Jones JA, Lavalley N, Alman J, Sinclair C, Garcia RI. Caries incidence in patients with dementia. *Gerodontology* 1993;10(2):76-82.
- Karasek R, Theorell T. *Healthy work: stress, productivity, and the reconstruction of working life*. New York: Basic Books; 1990.
- Kawachi I, Kennedy BP, Lochner K, Prothrow-Stith D. Social capital, income inequality, and mortality. *Am J Public Health* 1997 Sep;87(9):1491-8.
- Kennedy BP, Kawachi I, Prothrow-Stith D. Income distribution and mortality: cross-sectional ecological study of the Robin Hood index in the United States. *BMJ* 1996 Apr 20;312(7037):1004-7.
- Kiyak HA, Grayston MN, Crinean CL. Oral health problems and needs of nursing home residents. *Community Dent Oral Epidemiol* 1993;12:49-52.
- Kohler B, Andreen I, Jonsson B. The effect of caries-preventive measures in mothers on dental caries and the oral presence of the bacteria *Streptococcus mutans* and lactobacilli in their children. *Arch Oral Biol* 1984;29:879-83.
- Kohler B, Andreen I, Jonsson B. The earlier the colonization by mutans streptococci, the higher the caries prevalence at four years of age. *Oral Microbiol Immunol* 1988;3:14-7.
- Kozol J. *Savage inequalities: children in America's schools*. New York: Crown Publishers, Inc.; 1991.
- Kuhner MK, Raetzke PB. The effect of health beliefs on the compliance of periodontal patients with oral hygiene instructions. *J Periodontol* 1989;60:51-6.
- Lalonde M. *A new perspective on the health of Canadians*. Ottawa: Health and Welfare Canada; 1974.
- Lamy PP. Pharmacotherapeutics in the elderly. *Md Med J* 1989;38:144-8.
- Lave JR, Keene CR, Chyongchious JL, Ricci EM, Amersbach G, LaVallee CP. Impact of a children's health insurance program on newly enrolled children. *JAMA* 1998;279(22):1820-5.
- Levy SM, Baker HA, Semla TP, Kohout FJ. Use of medication with dental significance by a noninstitutionalized elderly population. *Gerodontology* 1988;14:119-25.
- Lewin K. Group decision and social change. In: Newcomb T, Hartley E. *Readings in social psychology*. New York: Holt; 1947. p. 197-211.
- Lewin K. *Field theory in social science*. New York: Harper; 1951.
- Lewis IK, Hanlon JT, Hobbins MJ, Beck JD. Use of medications with potential oral adverse drug reaction in community-dwelling elderly. *Spec Care Dentist* 1993;13(4):171-6.
- Lewit EM, Monheit AC. Expenditures on health care for children and pregnant women. U.S. health care for children. *Future Child* 1992;2(2):95-114.
- Li Y, Caulfield PW. The fidelity of initial acquisition of mutans streptococci by infants from their mothers. *J Dent Res* 1995;74:681-5.
- Litt MD, Reisine S, Tinanoff N. Multidimensional causal model of dental caries development in low income preschool children. *Public Health Rep* 1995;110:605-17.
- Marmot M, Theorell T. Social class and cardiovascular disease: the contribution of work. *Int J Health Serv* 1988;18:659-74.
- Marmot MG, Shipley MJ, Rose G. Inequalities in death: specific explanations of a general pattern. *Lancet* 1984;1:1003-6.
- McCarthy GM, Skillings JR. Orofacial complications of chemotherapy for breast cancer. *Oral Surg Oral Med Oral Pathol* 1992 Aug;74(2):172-8.
- McGoldrick PM. Principles of health behaviour and health education. In: Pine CM, editor. *Community oral health*. Oxford: Reed Educational & Professional Publishing Ltd.; 1997. p. 188-205.
- McIntyre RT, Jackson M, Shosenburg JW. Dental health status and treatment needs of institutionalized seniors. *Ont Dent* 1986;63:12-4,18-23.
- McKeown T. *The modern rise of population*. London: E Arnold; 1976.
- McKeown T. *The role of medicine: dream, mirage or nemesis*. Oxford: Basil Blackwell Publisher Ltd.; 1979. p. 1-207.
- Milgrom P, Weinstein P. Early childhood caries. A team approach to prevention and treatment. Seattle: University of Washington; 1999. p. 148.
- Milgrom P, Hujoel P, Grembowski D, Fong R. A community strategy for Medicaid child dental services. *Public Health Rep* 1999;114:528-32.
- Monheit AC, Cunningham PJ. Children without health insurance. *Future Child* 1992;2:154-70.

- Murtaugh C, Kemper P, Spillman B. The risk of nursing home use in later life. *Med Care* 1990;28(10):952-62.
- National Association of Dental Plans (NADP). 1998 Dental HMO/PPO industry profile. Dallas (TX): National Association of Dental Plans; 1998. Available from: <http://www.nadp.org>.
- National Center for Health Statistics (NCHS). Vital and Health Statistics, health United States, 1990. Hyattsville (MD): Public Health Service; 1991.
- National Center for Health Statistics (NCHS). Current estimates from the National Health Interview Survey 1996. Series 10, No. 200. Hyattsville (MD): Public Health Service; 1996.
- National Institute on Aging. Aging in the United States—past, present, and future. Washington: National Institute on Aging; 1997.
- National Policy and Resource Center on Nutrition and Aging. Oral health and older adults: community issues. Washington: National Policy and Resource Center on Nutrition and Aging; 1998.
- Newacheck PW, Stoddard JJ, Hughes DC, Pearl M. Children's access to health care: the role of social and economic factors. In: Stein RE, editor. Health care for children: what's right, what's wrong, what's next. New York: United Hospital Fund; 1997.
- Newacheck PW, Hughes DC, Hung YY, Wong S, Stoddard JJ. The unmet health needs of America's children. *Pediatrics* 2000 Apr;105(4 Pt 2):989-97.
- Niessen LC. Extending dental insurance through retirement. *Spec Care Dentist* 1984;4:84-6.
- Niessen LC, Jones JA. Oral health and aging: dental implications of a grayer America. *J Calif Dent Assoc* 1991 Aug;19(8):29-38.
- Nowak AJ. Rationale for the timing of the first oral evaluation. *Pediatr Dent* 1997;19:8-11.
- Nowak AJ, Casamassimo PS. Using anticipatory guidance to provide early dental intervention. *J Am Dent Assoc* 1995 Aug;126(8):1156-63.
- Pender NS. Health promotion in nursing practice. 2nd ed. East Norwalk (CT): Appelton and Lange; 1987.
- Pine CM. Introduction, principles and practice of public health. In: Pine CM, editor. Community oral health. Oxford: Reed Educational and Professional Publishing, Ltd.; 1997. p. 1-10.
- Porter R. The greatest benefit to mankind: a medical history of humanity. London: Harper Collins Publishers; 1997. p. 1-764.
- Redford M. Beyond pregnancy gingivitis: bringing a new focus to women's oral health. *J Dent Educ* 1993;57:742-8.
- Reforming States Group and Milbank Memorial Fund. Pediatric dental care in CHIP and Medicaid: paying for what kids need, getting value for state payments. New York: Reforming States Group, Milbank Memorial Fund Publishing; 1999. p. 23.
- Rose G. Strategy of preventive medicine. Oxford: Oxford University Press; 1993. p. 7-62.
- Rosenstock IM. Why people use health services. *Milbank Memorial Fund Q* 1966;44:94-127.
- Rosenstock IM. The health belief model and preventive behavior. *Health Education Monographs* 1974;2: 354-86.
- Rotter JB. Social learning and clinical psychology. New York: Prentice Hall; 1954.
- Sanders L. Sandwich generation: one family tries to ease the bite. [homepage on the Internet]. New York: McGraw-Hill Companies, Inc. Available from: <http://www.businessweek.com> [cited 1997 Jul 11].
- Schifter DE, Ajzen I. Intention, perceived control and weight loss: an application of the theory of planned behavior. *J Pers Soc Psychol* 1985;49:843-51.
- Sheehy G. Passages. New York: Bantam; 1984.
- Sheller B, Williams BJ, Lombardi SM. Diagnosis and treatment of dental caries-related emergencies in a children's hospital. *Pediatr Dent* 1997;19:470-5.
- Sigerist HE. The university at the crossroads: addresses and essays. New York: Henry Schuman; 1946. p. 127.
- Simpson G, Bloom B, Cohen RA, Parsons PE. Access to health care. Part I: Vital Health Stat 1997;197:1-47.
- Solomon LS. Rules of the game: how public policy affects local health care markets. *Health Aff (Millwood)* 1998 Jul-Aug;17(4):140-8.
- Spizak S, Holt K, editors. Building partnerships to improve children's access to Medicaid oral health services: national conference proceedings. Arlington (VA): National Center for Education in Maternal and Child Health; 1999.
- Stern PN. Variation on the sandwich generation. *Health Care Wom Int* 1994;15(1):v-vi.
- Syme SL. To prevent disease: the need for a new approach. In: Blane D, Brunner E, Wilkinson R, editors. Health and social organization: towards a health policy for the twenty-first century. New York: Routledge; 1996. p. 21-31.
- Tanzer JM. Dental caries is a transmissible infectious disease: the Keyes and Fitzgerald revolution. *J Dent Res* 1995;74:1536-42.
- Tinanoff N. State surveys of oral health needs and dental care access for children, summary of 15 state reports. Children's Dental Health Project. Washington: American Association of Dental Schools; 1998.
- Tomar SL, Winn DM, Swango GA, Giovino GA, Kleinman DV. Smokeless tobacco lesions among adolescents in the United States. *J Dent Res* 1997;76(6):1277-86.
- Tones K. Devising strategies for preventing drug misuse: the role of the health action model. *Health Educ Res* 1987;2:305-17.
- Tones K, Tilford S, Robinson YK. Health education: effectiveness and efficiency. London: Chapman and Hall; 1990.
- Triandis HC. Values, attitudes and interpersonal behavior. In: Page MM, editor. Nebraska symposium on motivation; beliefs, attitudes and values. Lincoln: University of Nebraska Press; 1979. p. 195-259.

- University of Southern California (USC) University Affiliated Program, Childrens Hospital Los Angeles. Planning guide for dental professionals serving children with special health care needs [draft]. 1999 Sep.
- U.S. Bureau of the Census. Current population reports, special studies. The need for personal assistance with everyday activities: recipients and caregivers. Washington: U.S. Department of Commerce; 1990. p. 70-19.
- U.S. Bureau of the Census. Department of Labor code of federal regulations, 42 EBRI. Washington: U.S. Department of Commerce; 1998a.
- U.S. Bureau of the Census. Washington: U.S. Department of Commerce; 1998b Jan 20.
- U.S. Department of Health and Human Services (USDHHS). In: Abdellah EG, Moore SR, editors. Surgeon General's workshop on health promotion and aging. Proceedings 1988. Washington: U.S. Department of Health and Human Services; 1988. p. 109.
- U.S. Department of Health and Human Services (USDHHS). Equity and access for mothers and children: strategies from the Public Health Service workshop on oral health of mothers and children. Washington: U.S. Department of Health and Human Services, Public Health Service, Health Resources and Services Administration, Maternal and Child Health Bureau; 1989. DHHS Pub. no. HRS-MCH-90-4.
- U.S. Department of Health and Human Services (USDHHS), Public Health Service (PHS). Current estimates from the national health interview survey, 1991. Series 10, no. 184. Hyattsville (MD): U.S. Department of Health and Human Services; 1992 Dec. p. 82, 83, 106. DHHS Pub no. PHS 93-1512.
- U.S. Department of Health and Human Services (USDHHS). Office of Inspector General. Children's dental services under Medicaid—access and utilization. San Francisco: Office of Evaluation and Inspections; 1996 Apr. OEI-09-93-00240.
- U.S. Department of Health and Human Services (USDHHS). Healthy People 2000 review 1997. Hyattsville (MD): National Center for Health Statistics; 1997.
- U.S. Department of Health and Human Services (USDHHS). Healthy People 2010 (conference edition, in two volumes). Washington: U.S. Department of Health and Human Services; 2000 Jan.
- Unkel JH, McKibben DH, Fenton SJ, Nazif MM, Mouri A, Schuit K. Comparison of odontogenic and non-odontogenic facial cellulitis in a pediatric hospital population. 1997 USDHHS. *Pediatr Dent* 1989;19:476-9.
- Vargas CM, Crall JJ, Schneider DA. Sociodemographic distribution of pediatric dental caries. NHANES III 1988-1994. Hyattsville (MD): National Center for Health Statistics; 1998.
- Vargas CM, Isman RE, Crall JJ. Comparison of children's medical and dental insurance coverage by socioeconomic characteristics, U.S. 1995. Submitted for publication, 2000.
- Verbugge LM. A health profile for older women with comparisons to older men. *Res Aging* 1984;6(3).
- Verbugge LM. Pathways of health and death. In: Apple RD, editor. *Women, health, and medicine in America. A historical handbook*. New York: Garland Publishing Inc.; 1990.
- Waldman HB. Mid-1990's profile of U.S. children and the conditions in which they live. *J Dent Child* 1996 Jul-Aug;63(4):285-90.
- Waldman HB. More children are unable to get dental care than any other single health service. *J Dent Child* 1998 May-June;65(3):204-8.
- Warner KL. The rewards of the sandwich generation. *J Pract Nurs* 1995;45(4):16-20.
- Warren RC. Oral health for all: policy for available, accessible, and acceptable care. Washington: Center for Policy Alternatives; 1999.
- Wilson S, Smith GA, Preish J, Casamassimo PS. Nontraumatic dental emergencies in a pediatric emergency department. *Clin Pediatr* 1997;36:333-7.
- World Health Organization (WHO). Ottawa charter for health promotion. Geneva: World Health Organization; 1986.
- Yudkowsky BK, Tang SFS. Medicaid state reports—FY 1995. Elk Grove Village (IL): American Academy of Pediatrics; 1997.

Facing the Future

The challenges for oral health in the twenty-first century are formidable. First and foremost is the need to ensure that all people have access to health care and can acquire the health literacy necessary to make use of health promotion and disease prevention information and activities.

The century offers the promise of a new era for health wrought by the convergence of six cultural movements, any one of which would be sufficient to transform the human condition:

- The biological and biotechnology revolutions.
- A redistribution of the world's people by rapid and sizable migrations within countries and across borders.
- Changing demographics in industrialized as well as developing nations.
- Changing patterns of disease, including the emergence and reemergence of infectious diseases, and changes in the organization of health care.
- Instant worldwide communication through the Internet, cable, satellite, and wireless technology.
- A continuing exponential rate of growth in information technology, specifically in computer speed, memory, and complexity.

These global currents are changing the way we live now and will have profound implications for the future of the oral and general health and well-being of all people.

THE PAST AND PRESENT AS PROLOGUE

The Pioneers

The history and intellectual activity of the eighteenth and nineteenth centuries set the seeds for the flowering of biology in the twentieth and early twenty-first centuries (Porter 1997). The scientific and techno-

logical discoveries of the early anatomists and embryologists—the founders of cell theory and brain research—were followed by the brilliant innovations of Pasteur, Koch, and Ehrlich, who established the new fields of microbiology and immunology. The cumulative achievements of these pioneers set the foundation for the diagnostic and therapeutic science and art of dentistry, medicine, nursing, and pharmacology in the twentieth century.

The seeds were also sown for the convergence of chemistry, physics, and biology in the field of molecular biology, as well as the convergence of Darwinism, fruit fly genetics, and population genetics into the modern evolutionary synthesis. These convergences inspired the current quest to identify all 100,000 genes of the human genome and to assign functional meanings to the motifs that are encoded within them.

Vital Statistics

The growth of the world population and the transcontinental movements of people are proving a dominant force for change. The twentieth century began with increased European and Asian migrations to the United States. By 1900 the U.S. population had reached 90 million residents and the Earth's population was approaching 1 billion people. Life expectancy in the United States was 47 years of age. Acute viral and bacterial infections were the primary causes of infant morbidity and mortality. Being edentulous, or "toothless," was a normal expectation for mature adults.

For most of recorded human history and the 100,000 years of human prehistory, life expectancy was very low. Life expectancy at the time of the Roman Empire was approximately 28 years of age. From the beginning of the first millennium A.D. to 1900, each year of history saw an average gain of 3 days in life expectancy. Each year since 1900,

however, has seen a gain of 110 days in average life expectancy (Rowe and Kahn 1998). Life expectancy at birth in the United States has increased from 47 years in 1900 to approximately 76 years today. While the entire population of the United States has tripled since 1900, the absolute number of older persons, currently 33 million, has increased elevenfold (Finch and Pike 1996, Rowe and Kahn 1998, p. 4). The U.S. population is 270 million and will reach 300 million in the next few decades. The Earth's population doubled by 1950, doubled again by 1975, and currently is 6 billion.

Health Improvement

Measures such as improved sanitation and housing, prenatal care, immunizations, health education and promotion, community water fluoridation, and dental sealants have greatly improved oral health for the majority of the population. Advances in science and technology, health professional education, the science of public health and clinical practice, and the health literacy of the public will continue to improve the health and well-being of Americans in the coming years (Kevles 1997, Schwartz 1998). Ever larger numbers of senior adults expect to retain a full or nearly complete dentition and to live well into their 70s, 80s, and 90s free of pain and discomfort (Slavkin 1997a).

DIVERSITY OF DISEASES AND PATIENTS

Those seeking care in the decades ahead will present with a wide range of diseases and disorders, unevenly distributed across populations. The very youngest patients include children with complex hereditary or congenital craniofacial defects in need of expert multidisciplinary teams to repair and restore form and function. Early childhood caries, one of the most severe forms of the disease, is especially prevalent among poor children in some racial/ethnic groups in America, such as American Indians and Mexican Americans. Young adults are particularly vulnerable to unintentional and intentional craniofacial injuries. Middle-aged and older generations typically experience chronic diseases affecting the heart or lungs as well as cancers, diabetes, and the various degenerative diseases of joints and bones and the nervous system, all of which may affect or be affected by oral diseases and their treatments.

TRANSFORMING TREATMENTS

The cultural movements that are changing the human condition will likely transform treatments for many of the complex disorders just described. The instrumentation used to detect subtle genetic variations in each of the 100,000 genes in the human genome will inexorably reveal which gene or genes are defective in hundreds of inherited and acquired craniofacial diseases or syndromes. On the horizon are promotion measures to enhance health and eliminate exposures to teratogens, as well as surgical techniques to correct the defects *in utero*, obviating the need for costly multiple surgeries and rehabilitation programs for affected children.

We are entering the "golden age of molecular oral health" with gene-based diagnostics, therapeutics, and biomaterials. Risk assessment for disease will be based in part on understanding the genetic variations that affect resistance or susceptibility, but also will be determined in part by environmental factors, socioeconomic status, personal behaviors, and lifestyle. The risk for early childhood caries is likely to be determined by a combination of all these factors, as well as cultural beliefs and practices within some populations. Elimination of all infections, whether in the oral cavity or elsewhere, will be seen as a critical part of health promotion.

Prevention of injuries will call for approaches that are both culturally and age sensitive, in addition to the coordinated efforts of policymakers and legislators to mandate protective gear in sports and other safety measures when necessary.

Gene therapy will be applied to treat oral and pharyngeal cancers and also will be used for the oral and systemic delivery of endogenous and synthetic molecules to treat salivary gland disorders, oral infections, and systemic disease. Highly specific drugs will be developed for the management of chronic facial pain such as trigeminal neuralgia and Bell's palsy.

Should additional evidence in the early years of the twenty-first century further indicate that oral infections actually cause some cases of heart disease, pulmonary disease, and stroke, or trigger the birth of premature, low-birth-weight babies, treatment approaches will be radically altered.

TRANSFORMING HEALTH PROFESSIONAL EDUCATION

The scientific and technological bases of dentistry, medicine, nursing, and pharmacy are expanding rapidly in parallel with the changing demographics of

the nation, the public's expectations for an enhanced quality of life, and changes in the management and financing of health care. Health professional schools, often organized around academic health science centers, are responding to these challenges and opportunities.

Students and clinicians alike need to be prepared to adopt evidence-based health care. Today and tomorrow, students must be well versed in epidemiology, biometry, bioinformatics, molecular biology, bioengineering, and much more. In addition, they must be prepared to adopt and implement new preventive strategies and comprehensive and molecular-based diagnostics and therapeutics; to support cost-effective community-based health programs; and to anticipate all the challenges that promotion of health entails. Clinical science or scientific evidence in the new millennium will continue to evolve in molecular dentistry and medicine with attendant opportunities for addressing the social, legal, and ethical implications. We must prepare clinicians for the nuances and complexities of modern clinical research-based results.

The previous chapters of this report provide the documentation that can be used to assess health professional education. Major progress in health promotion, disease prevention, diagnostics, therapy and therapeutics, and the socioeconomic and behavioral factors that influence oral, dental, and craniofacial health will further contribute to the transformation of health professional education.

TRANSFORMING HEALTH CARE

We are currently witnessing a significant transformation in the financing and management of health care, which is affecting all the health specialties. Care providers are assuming new responsibilities and functions, and changing employment patterns. Traditionally, the management of health care has been centered on the providers of services and hospitals. Recently, the center has enlarged to include additional marketplace stakeholders, the purchasers of health care and health care plans, and increasingly all segments of society. The interactions among all these participants will shape health and health care for the foreseeable future.

Risk assessment models are also being developed and used to design treatment options tailored to communities and to individual patients. Increased use of information technology, greater efforts to conduct community needs assessments, and greater emphasis on enhanced quality of life expectations of

patients, families, and communities are also in evidence (USDHHS 2000).

The responsibility for oral and craniofacial care involves all health professionals, so coordinated care delivery and reimbursement will be critical. Evidence-based systematic assessments and guidelines will contribute to clinical and public health decision making. In addition, the linkage between health care professions and public health and social service activities will need to be strengthened.

These trends are complemented by greater understanding of the psychosocial-behavioral aspects of oral diseases and disorders. These advances will continue to influence the nation's capacity to address the breadth and depth of diseases and conditions affecting oral health across the life span and their relationship to general health and well-being.

Access to the Internet and increased health and science reporting in print and broadcast media have created a more knowledgeable public motivated to understand the value of healthy choices. However, increasing numbers of patients are also questioning traditional practices and seeking alternative and complementary approaches.

ORAL HEALTH—NOT YET FOR ALL

Demographers predict that by 2050 there will be no single racial/ethnic majority in the United States. Rather, we will become an increasingly diverse nation with diverse patterns of disease and levels of health. This is especially evident for African American, Latino, Asian and Pacific Islander, and American Indian communities (Pamuk et al. 1998). Disparities in educational advancement, job opportunities, income and wealth, housing and neighborhood characteristics, health access and status, and involvement in the criminal justice system for various subpopulations will remain unless steps are taken to reverse the trends (Council of Economic Advisers 1998).

The proportion of school-aged children who are caries-free in their permanent teeth has more than doubled during the last 20 years. However, in states such as California, Texas, Louisiana, Alabama, Florida, and Georgia the trends are different; fewer than one third of the children are caries-free in their permanent dentition.

One attempt to come to the aid of poor children is the State Children's Health Insurance Program (SCHIP), federal legislation designed to help individual states meet the health needs of children (Council of Economic Advisers 1998, NRC 1998). As of 1998, more than 11 million children in America—1 in 7

children—are estimated to be uninsured. Most of these children live in families with working parents who have jobs that do not provide health insurance and who are unable to purchase health care insurance (NRC 1998). Nationally, 1 in 6 African American children and 1 in 4 Hispanic children are uninsured, compared with 1 in 10 white children (Council of Economic Advisers 1998, NRC 1998). This limited health care access is particularly significant in relation to oral health.

HOPE FROM SCIENCE AND TECHNOLOGY

The biological and biotechnology revolutions will accelerate, inspiring theory building and new models of miniaturization and speed that can be applied to improve oral health. The Human Genome Project will be completed no later than 2003. The entire human genetic lexicon will be accessible through the Internet. To date, more than several hundred mutated craniofacial regulatory and structural genes have been found to cause abnormal formation of oral, dental, and craniofacial tissues and structures.

In addition, the genomes of many significant viruses, bacteria, yeast, parasites, plants, and animals are currently being deciphered, and these are revolutionizing how we think about biology and human diseases (Bodmer and McKie 1995, Chambers 1995, Collins et al. 1998). At present, research is under way to decipher the genetic lexicon of 60 microbes, 6 of which are important oral pathogenic bacteria or fungi. The evolution of this knowledge will yield innovations in areas from clinical prevention to drug and biomaterials discovery. Figure 11.1 presents a

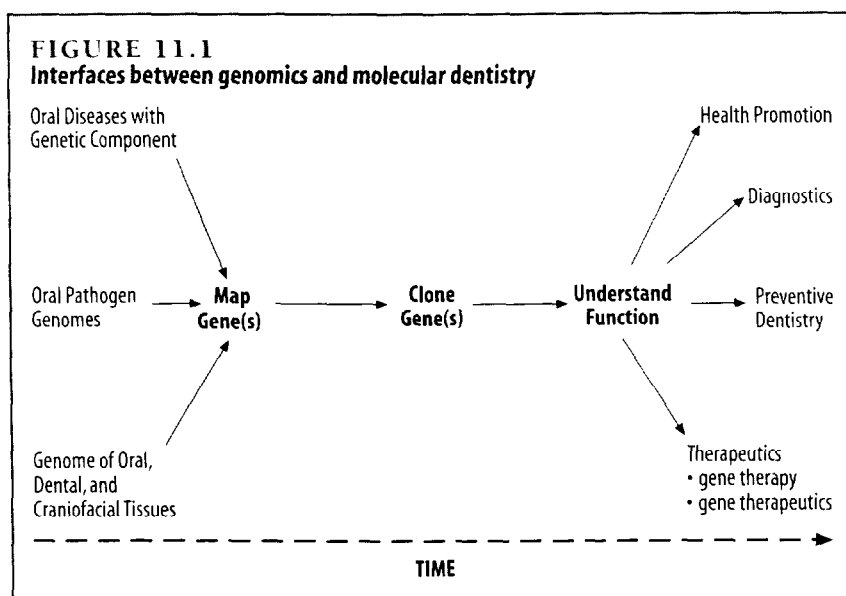
model of the possible interface between genomics and molecular dentistry.

Perhaps with the sole exception of trauma, all human diseases have a genetic component. Genetic dentistry and medicine are based on the paradigm that changes or mutations in individual nucleotides within genes or alleles result in variations or polymorphisms. These mutations are either inherited or acquired after birth. For example, inherited mutations in the amelogenin gene located on the human X (and Y) chromosome can produce X-linked dominant or recessive amelogenesis imperfecta, a painful disease characterized by defective tooth enamel (Backman 1997), and mutations in the fibroblast growth factor receptor 2 gene can produce serious craniofacial birth defects such as Crouzon's disease and other syndromes with premature fusion of cranial bones (craniosynostosis) (Cohen 1997). Mutations in a number of transcription factors that regulate development produce other craniofacial syndromes (Slavkin 1999).

The human genome contains approximately 100,000 genes or alleles. The genome consists of 3 billion nucleotides or bases. Mutations changing one or more bases, in one or more genes, can result in diseases or disorders. Many environmental factors termed mutagens, carcinogens, or teratogens can cause mutations in one or more genes resulting in human disease such as neoplastic diseases. The completion of the Human Genome Project in the next 2 years will afford an unprecedented opportunity to advance our understanding of inherited as well as acquired human diseases and disorders.

Scientific discoveries are rapidly defining single-gene mutations, mapping these individual genes in their precise positions on each of the 46 human chromosomes. These findings are being used to diagnose inherited and acquired clinical phenotypes as well as "at-risk populations" throughout the human life span.

These remarkable advances in human molecular genetics are identifying candidate genes for developing targeted gene-mediated therapeutic approaches to many oral health diseases, ranging from passive immunization for dental caries, induction of new bone and cartilage tissue, and regeneration of periodontal tissues, to the artificial synthesis of saliva for patients suffering from xerostomia.



Gene mutations also define the virulence of microbes (viruses, bacteria, yeasts, and parasites), as well as the fidelity of the human immune system. Microbial as well as human genes are extremely sensitive to environmental stress and can and do mutate, resulting in antibiotic resistance. The genetic variance within microbial genomes such as the genome of the yeast *Candida albicans* may be closely aligned with the host changes associated with immunologically compromised patients. The HIV viral genome is another particularly useful model for considering viral mutation frequency during pathogenesis (Slavkin 1996a). These discoveries provide the foundations for gene-based diagnostics for disease detection; therapeutic drug development; and individual predictors of drug response during the management of chronic facial pain, osteoarthritis as related to temporomandibular joint disease, and osteoporosis associated with periodontal diseases.

We are beginning to understand that polymorphisms (variations) in multiple genes confer susceptibility or resistance to chronic and disabling diseases and disorders such as osteoporosis, periodontal diseases, and temporomandibular disorders (Slavkin 1997b). In these examples, multiple genes and multiple gene-environment and gene-gene interactions are associated with the molecular etiology and pathophysiology of the disease process.

The function of most genes must inevitably be studied and understood at the level of their encoded proteins and protein-protein interactions, for these are the biologically active players of life. An enormous number of genes encode protein information that is highly conserved, that is, found in almost identical form in such diverse organisms as fruit flies and humans. Further scrutiny and analysis have determined that specific motifs encoded in larger domains of each protein serve as the "business" portion of the protein, binding to a cell surface, aggregating with other proteins, serving to catalyze a chemical reaction, binding to zinc or calcium ions, or serving other crucial functions in cell biology. The functional motifs are also being characterized in terms of structural biology. The scientific and educational communities are building large databases and then mining this information by using sophisticated information technology.

These genomic databases provide remarkable opportunities for the identification, design, and production of a new generation of biomarkers for diagnostics; innovative biomaterials for repair and regeneration; and the development of highly sensitive and specific drugs and vaccines to improve the health of all people (Baum et al. 1998, Slavkin 1996b,c,

1997a). Genomics has emerged as a major driver to realign academic, industry, and government science and technology to foster health, pharmaceutical, biotechnology, agricultural, food, chemical, environmental, energy, and computer science applications (Kaku 1997, Rifkin 1998). Many of these applications profoundly influence oral health (Slavkin 1996d, 1998a,b).

This epic period will also herald the advent of "biochemistry on a chip," used in connection with body fluids such as saliva, cells, and tissues to diagnose diseases and disorders. The so-called chip technology will enable identification, quantitation, and complex analyses on surfaces no larger than one-centimeter square coupled to laser optical reader systems and computer-assisted informatics. Prototypes are already available to be tested against samples of saliva, cervical fluids, buccal mucosal cells, and blood (Slavkin 1998b). This technology should revolutionize saliva-based diagnostics and prognostics in oral health (Table 11.1). Major progress is also anticipated in bioengineering through nanotechnology, miniaturization, and the innovations of design and fabrication of biomaterials. Anticipated advances include the repair and regeneration of cartilage, bone, muscle, nerve, salivary glands and saliva, and teeth (cementum, dentin, enamel, and periodontal ligament) (Slavkin 1996d, 1998a,b).

Additional scientific progress in the neurosciences will have broad implications for the diagnosis and treatment of diseases and disorders of the craniofacial complex including neuromuscular-related conditions (e.g., facial and dental trauma, bruxism, autism, Mobius syndrome, Bell's palsy, temporomandibular joint disorders, trigeminal neuralgia, Parkinson's disease, and disorders of speech, smell, and taste), the habilitation of craniofacial syndromes, and the management of facial pain.

The field of biomimetics is an example of the translation of human genomics into innovative developments in biotechnology. The idea is to use biological strategies to solve human diseases and disorders, essentially mimicking biological processes in the design and fabrication of new biomaterials to replace body parts or synthesize new drugs or reagents. For example, biological cartilage can now be designed and produced in artificial systems that present three-dimensional forms for nose and ear replacements as required in craniofacial birth defects, head and neck trauma, and oral and pharyngeal cancer patients (Slavkin 1996b, 1998a,b). Another approach is to design and fabricate bioceramics to be used in the replacement of human enamel or dentin on the surfaces of teeth.

A FRAMEWORK FOR ORAL HEALTH

At the most basic level, local, state, and national health care policies will continue to strive to improve the health status of all Americans. Major reforms will improve public health competency. Enlightened health literacy will continue to influence quality of life expectations. Many social, economic, and political influences will continue to influence local, state, and national priorities for health policies (Isaacs and Knickman 1999). Included in these reforms will be efforts to improve the oral and craniofacial health of the American people.

Oral and craniofacial health issues will continue to be diverse and complex. In this context, two major themes remain: the need and demand for oral and craniofacial health services; and the role, functions, and mix of health professionals (Casamassimo 1996, USDHHS 1998).

First, need and demand will continue to be the two drivers of the health service requirements of our society. Need is an epidemiologically based and clinically derived measure of the amount of disease and adverse conditions that require treatment in order for the population to be healthy. Demand measures a population's health literacy, willingness, and capacity to utilize and finance health services. Public health literacy or competency and proactive

oral health education will increase demand as well as delineate functions of oral health professionals for 2000 and beyond. Often, biomedical research advances in terms of new pharmaceuticals, devices, and procedures popularized in the media influence quality of life expectations, demand for health services, and the economy (Pardes et al. 1999). They can also lead to the creation of new types of health providers. Research also has the potential to reduce the need, demand, and costs for health services (McRae 1994). Thus outpatient surgery obviates the need for hospitalization; immunization or antibiotics control infections; and community water fluoridation, other fluorides, dental sealants, and related oral health policies help prevent dental caries.

Second, the major changes in demography, patterns of disease, and management of health care will continue to shape the roles and functions of health professionals. For example, significant increases in the numbers of senior citizens (65 years and older) with chronic facial pain, osteoarthritis, temporomandibular joint disorders, type 2 diabetes, dementia, osteoporosis, and oral and pharyngeal cancers will challenge health care providers for the next 50 years. These conditions will necessitate interdisciplinary and multidisciplinary approaches to care. Coordination of professional care with that of individuals, caregivers, and the community will be needed to control costs and ensure early diagnosis and prompt treatment.

To ensure that all people have access to health care and can acquire the health literacy necessary to make use of oral and craniofacial health promotion and disease prevention information and activities, a complete assessment of the nation's capacity to achieve access for all is warranted. Federal, state, and local government programs, legislation, and regulation; health professional societies and organizations; professional schools, colleges within universities, and K-12 education; patient groups; the private sector; and the larger society have the responsibility to achieve access to oral health care for all.

TABLE 11.1
Examples of saliva fluid diagnostics

Detection of exposure to viruses by measuring antibodies specific for a viral antigen

Measles	Hepatitis A	HIV-1
Mumps	Hepatitis B	HIV-2
Rubella		

Direct antigenic detection of microbes and biomarkers

- Influenza A and B (neuraminidase)
- Streptococcus* group A (N-acetylglucosamine)
- Salivary estradiol (pre-term labor indicator)
- CA-15, EGFR, cathepsin-D, and Waf 1 (proposed breast cancer biomarkers)
- Zinc-binding cystic fibrosis antigen (proposed biomarker)
- Glutamic acid decarboxylase autoantibody (proposed predictive biomarker in type-1 diabetes)

Culture of microbial organisms

- Streptococcus mutans*—dental caries
- Lactobacillus acidophilus*—dental caries
- Candida albicans*—oral candidiasis

Examples of hormones and drugs identified and measurable in saliva

aldosterone	progesterone	phenytoin	antipyrine	ethanol
cortisol	testosterone	theophylline		marijuana
estrogens	carbamazepine	caffeine		opiates
insulin	lithium	cocaine		
melatonin	methadone	continine		

Sources: Derived from Malamud 1992, AGD 1996, Malamud and Tabak 1993.

REFERENCES

- AGD. Saliva: a spitting image of the body. *AGD Impact* 1996;24(8):10-5.
- Backman B. Inherited enamel defects. In: Chadwick DJ, Cardew G. editors. *Dental enamel*. London: John Wiley and Sons Ltd; 1997. p. 175-96.
- Baum BJ, Atkinson JC, Baccaglioni LJ, et al. The mouth is a gateway to the body; gene therapy in 21st-century dental practice. *Can Dent Assoc J* 1998;26:455-60.
- Bodmer W, McKie R. *The book of man: the Human Genome Project and the quest to discover our genetic heritage*. New York: Scribner Publishers; 1995.
- Casamassimo P. editor. *Bright futures in practice: oral health*. Arlington (VA): National Center for Education in Maternal and Child Health; 1996.
- Chambers DA. editor. *DNA: The double helix: 40 years, prospective and perspective*. New York: New York Academy of Sciences; 1995.
- Council of Economic Advisers. *Changing America: indicators of social and economic well-being by race and Hispanic origin*. Washington: Council of Economic Advisers, Office of the President; 1998 1 Sep. Available from: US GPO, Washington, DC.
- Cohen MM Jr. Molecular biology of craniosynostosis with special emphasis on fibroblast growth factor receptors. In: Cohen MM Jr, Baum BJ, editors. *Studies in stomatology and craniofacial biology*. Amsterdam: IOS Press; 1997. p. 307-30.
- Collins F, Patrinos A, Jordan E, Chakravarti A, Gesteland R, Walters L. New goals for the U.S. Human Genome Project: 1998-2003. *Science* 1998;282:689.
- Finch CE, Pike MC. Maximum lifespan predictions from the Gompertz mortality model. *J Gerontol A Biol Med Sci* 1996;51(3):B183-94.
- Issacs SL, Knickman JR, editors. *To improve health and health care 2000*. San Francisco: Jossey-Bass Publishers; 1999.
- Kaku M. *Visions: how science will revolutionize the 21st century*. New York: Anchor Books, Doubleday; 1997.
- Kevles BH. *Naked to the bone*. New Brunswick (NJ): Rutgers University Press; 1997.
- Malamud D. Saliva as a diagnostic fluid. *BMJ* 1992;(305):207-8.
- Malamud D, Tabak L, editors. *Saliva as a diagnostic fluid*. *Ann NY Acad Sci* 1993;694.
- McRae H. *The world in 2020*. Boston: Harvard Business School Press; 1994.
- National Research Council (NRC). *America's children: health insurance and access to care*. Washington: National Academy Press, 1998.
- Pamuk E, Makuc D, Heck K, Rueben C, Lochner K. *Socioeconomic status and health chartbook*. Health, United States, 1998. Hyattsville (MD): National Center for Health Statistics; 1998.
- Pardes H, Manton KG, Lander ES, Tolley HD, Ullian AD, Palmer H. *Effects of medical research on health care and the economy*. *Science* 1999;283:36-7.
- Porter R. *The greatest benefit to mankind*. New York: W.W. Norton & Company; 1997.
- Rifkin J. *The biotech century*. New York: Penguin Putnam; 1998.
- Rowe JW, Kahn RL. *Successful aging*. New York: Pantheon Books; 1998. p. 182-3.
- Schwartz WB. *Life without disease*. Berkeley: University of California Press; 1998.
- Slavkin HC. An update on HIV/AIDS. *J Am Dent Assoc* 1996a;127:1401-4.
- Slavkin HC. Biomimetics: replacing body parts is no longer science fiction. *J Am Dent Assoc* 1996b;127:1254-7.
- Slavkin HC. Understanding human genetics. *J Am Dent Assoc* 1996c;127:266-7.
- Slavkin HC. Basic science is the fuel that drives the engine of biotechnology: a personal science transfer vision for the 21st century. *Tech Health Care* 1996d;4:249-53.
- Slavkin HC. Clinical dentistry in the 21st century. *Compendium* 1997a;18(3):212-8.
- Slavkin HC. Chronic disabling diseases and disorders. *J Am Dent Assoc* 1997b;128:1583-9.
- Slavkin HC. Biomimicry, dental implants and clinical trials. *J Am Dent Assoc* 1998a;129:226-30.
- Slavkin HC. Toward molecular based diagnostics for the oral cavity. *J Am Dent Assoc* 1998b;129:1138-43.
- Slavkin HC. Possibilities of growth modification: nature versus nurture. In: MacNamara JA Jr, editor. *Growth modification: what works, what doesn't and why*. Ann Arbor: University of Michigan Press; 1999. p. 1-16.
- U.S. Department of Health and Human Services (USDHHS), National Center for Health Statistics (NCHS). *1997 Healthy People 2000 review*. Hyattsville (MD): U.S. Department of Health and Human Services, National Center for Health Statistics; 1998.
- U.S. Department of Health and Human Services (USDHHS). *Healthy People 2010: understanding and improving health*. Washington: U.S. Department of Health and Human Services; 2000. Available from: US GPO, Washington, DC.

A Call to Action

The major message of this Surgeon General's report is that oral health is essential to the general health and well-being of all Americans and can be achieved by all Americans. However, *not all* Americans are achieving the same degree of oral health. In spite of the safe and effective means of maintaining oral health that have benefited the majority of Americans over the past half century, many among us still experience needless pain and suffering, complications that can devastate overall health and well-being, and financial and social costs that diminish the quality of life and burden American society.

To maintain the health and well-being of Americans already enjoying good oral health and to address the gaps in oral health status of others require actions at all levels of society, from individuals and neighborhoods to the nation as a whole. A coordinated effort can overcome the educational, environmental, social, health system, and financial barriers that have created vulnerable populations whose oral health is at risk.

MAJOR FINDINGS

Following are the major findings of the report. They reflect the detailed findings highlighted at the end of each chapter as well as the broad themes presented in Chapter 1.

Oral diseases and disorders in and of themselves affect health and well-being throughout life. The burden of oral problems is extensive and may be particularly severe in vulnerable populations. It includes the common dental diseases and other oral infections such as cold sores and candidiasis that can occur at any stage of life, as well as birth defects in infancy and the chronic facial pain conditions and oral cancers seen in later years. Many of these conditions and their treatments may undermine self-image and self-esteem, discourage normal social

interaction, cause other health problems, and lead to chronic stress and depression as well as incur great financial cost. They may also interfere with vital functions such as breathing, food selection, eating, swallowing, and speaking and with activities of daily living such as work, school, and family interactions.

Safe and effective measures exist to prevent the most common dental diseases—dental caries and periodontal diseases. Community water fluoridation is safe and effective in preventing dental caries in both children and adults. Water fluoridation benefits all residents served by community water supplies regardless of their social or economic status. Professional and individual measures, including the use of fluoride mouthrinses, gels, dentifrices, and dietary supplements and the application of dental sealants, are additional means of preventing dental caries. Gingivitis can be prevented by good personal oral hygiene practices, including brushing and flossing.

Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use, and poor dietary choices affect oral and craniofacial health as well. These individual behaviors are associated with increased risk for craniofacial birth defects, oral and pharyngeal cancers, periodontal disease, dental caries, and candidiasis, among other oral health problems. Opportunities exist to expand the oral disease prevention and health promotion knowledge and practices of the public through community programs and in health care settings. All health care providers can play a role in promoting healthy lifestyles by incorporating tobacco cessation programs, nutritional counseling, and other health promotion efforts into their practices.

There are profound and consequential oral health disparities within the U.S. population. Disparities for various oral conditions may relate to income, age, sex, race or ethnicity, or medical status.

Although common dental diseases are preventable, not all members of society are informed about or able to avail themselves of appropriate oral-health-promoting measures. Similarly, not all health providers may be aware of the services needed to improve oral health. In addition, oral health care is not fully integrated into many care programs. Social, economic, and cultural factors and changing population demographics affect how health services are delivered and used, and how people care for themselves. Reducing disparities requires wide-ranging approaches that target populations at highest risk for specific oral diseases and involves improving access to existing care. One approach includes making dental insurance more available to Americans. Public coverage for dental care is minimal for adults, and programs for children have not reached the many eligible beneficiaries.

More information is needed to improve America's oral health and eliminate health disparities. We do not have adequate data on health, disease, and health practices and care use for the U.S. population as a whole and its diverse segments, including racial and ethnic minorities, rural populations, individuals with disabilities, the homeless, immigrants, migrant workers, the very young, and the frail elderly. Nor are there sufficient data that explore health issues in relation to sex or sexual orientation. Data on state and local populations, essential for program planning and evaluation, are rare or unavailable and reflect the limited capacity of the U.S. health infrastructure for oral health. Health services research, which could provide much needed information on the cost, cost-effectiveness, and outcomes of treatment, is also sorely lacking. Finally, measurement of disease and health outcomes is needed. Although progress has been made in measuring oral-health-related quality of life, more needs to be done, and measures of oral health per se do not exist.

The mouth reflects general health and well-being. The mouth is a readily accessible and visible part of the body and provides health care providers and individuals with a window on their general health status. As the gateway of the body, the mouth senses and responds to the external world and at the same time reflects what is happening deep inside the body. The mouth may show signs of nutritional deficiencies and serve as an early warning system for diseases such as HIV infection and other immune system problems. The mouth can also show signs of general infection and stress. As the number of substances that can be reliably measured in saliva increases, it may well become the diagnostic fluid of choice, enabling the diagnosis of specific disease as

well as the measurement of the concentration of a variety of drugs, hormones, and other molecules of interest. Cells and fluids in the mouth may also be used for genetic analysis to help uncover risks for disease and predict outcomes of medical treatments.

Oral diseases and conditions are associated with other health problems. Oral infections can be the source of systemic infections in people with weakened immune systems, and oral signs and symptoms often are part of a general health condition. Associations between chronic oral infections and other health problems, including diabetes, heart disease, and adverse pregnancy outcomes, have also been reported. Ongoing research may uncover mechanisms that strengthen the current findings and explain these relationships.

Scientific research is key to further reduction in the burden of diseases and disorders that affect the face, mouth, and teeth. The science base for dental diseases is broad and provides a strong foundation for further improvements in prevention; for other craniofacial and oral health conditions the base has not yet reached the same level of maturity. Scientific research has led to a variety of approaches to improve oral health through prevention, early diagnosis, and treatment. We are well positioned to take these prevention measures further by investigating how to develop more targeted and effective interventions and devising ways to enhance their appropriate adoption by the public and the health professions. The application of powerful new tools and techniques is important. Their employment in research in genetics and genomics, neuroscience, and cancer has allowed rapid progress in these fields. An intensified effort to understand the relationships between oral infections and their management, and other illnesses and conditions is warranted, along with the development of oral-based diagnostics. These developments hold great promise for the health of the American people.

A FRAMEWORK FOR ACTION

All Americans can benefit from the development of a National Oral Health Plan to improve quality of life and eliminate health disparities by facilitating collaborations among individuals, health care providers, communities, and policymakers at all levels of society and by taking advantage of existing initiatives. Everyone has a role in improving and promoting oral health. Together we can work to broaden public understanding of the importance of oral health and its relevance to general health and well-being, and to ensure that existing and future preventive, diagnostic, and treatment measures for

oral diseases and disorders are made available to all Americans. The following are the principal components of the plan:

Change perceptions regarding oral health and disease so that oral health becomes an accepted component of general health.

- *Change public perceptions.* Many people consider oral signs and symptoms to be less important than indications of general illness. As a result, they may avoid or postpone needed care, thus exacerbating the problem. If we are to increase the nation's capacity to improve oral health and reduce health disparities, we need to enhance the public's understanding of the meaning of oral health and the relationship of the mouth to the rest of the body. These messages should take into account the multiple languages and cultural traditions that characterize America's diversity.

- *Change policymakers' perceptions.* Informed policymakers at the local, state, and federal levels are critical in ensuring the inclusion of oral health services in health promotion and disease prevention programs, care delivery systems, and reimbursement schedules. Raising awareness of oral health among legislators and public officials at all levels of government is essential to creating effective public policy to improve America's oral health. Every conceivable avenue should be used to inform policymakers—informally through their organizations and affiliations and formally through their governmental offices—if rational oral health policy is to be formulated and effective programs implemented.

- *Change health providers' perceptions.* Too little time is devoted to oral health and disease topics in the education of nondental health professionals. Yet all care providers can and should contribute to enhancing oral health. This can be accomplished in several ways, such as including an oral examination as part of a general medical examination, advising patients in matters of tobacco cessation and diet, and referring patients to oral health practitioners for care prior to medical or surgical treatments that can damage oral tissues, such as cancer chemotherapy or radiation to the head and neck. Health care providers should be ready, willing, and able to work in collaboration to provide optimal health care for their patients. Having informed health care professionals will ensure that the public using the health care system will benefit from interdisciplinary services and comprehensive care. To prepare providers for such a role will involve, among other factors, curriculum changes and multidisciplinary training.

Accelerate the building of the science and evidence base and apply science effectively to improve oral health. Basic behavioral and biomedical research, clinical trials, and population-based research have been at the heart of scientific advances over the past decades. The nation's continued investment in research is critical for the provision of new knowledge about oral and general health and disease for years to come and needs to be accelerated if further improvements are to be made. Equally important is the effective transfer of research findings to the public and health professions. However, the next steps are more complicated. The challenge is to understand complex diseases caused by the interaction of multiple genes with environmental and behavioral variables—a description that applies to most oral diseases and disorders—and translate research findings into health care practice and healthy lifestyles.

This report highlights many areas of research opportunities and needs in each chapter. At present, there is an overall need for behavioral and clinical research, clinical trials, health services research, and community-based demonstration research. Also, development of risk assessment procedures for individuals and communities and of diagnostic markers to indicate whether an individual is more or less susceptible to a given disease can provide the basis for formulating risk profiles and tailoring treatment and program options accordingly.

Vital to progress in this area is a better understanding of the etiology and distribution of disease. But as this report makes clear, epidemiologic and surveillance databases for oral health and disease, health services, utilization of care, and expenditures are limited or lacking at the national, state, and local levels. Such data are essential in conducting health services research, generating research hypotheses, planning and evaluating programs, and identifying emerging public health problems. Future data collection must address differences among the subpopulations making up racial and ethnic groups. More attention must also be paid to demographic variables such as age, sex, sexual orientation, and socioeconomic factors in determining health status. Clearly, the more detailed information that is available, the better can program planners establish priorities and targeted interventions.

Progress in elucidating the relationships between chronic oral inflammatory infections, such as periodontitis, and diabetes and glycemic control as well as other systemic conditions will require a similar intensified commitment to research. Rapid progress

can also occur with efforts in the area of the natural repair and regeneration of oral tissues and organs. Improvements in oral health depend on multidisciplinary and interdisciplinary approaches to biomedical and behavioral research, including partnerships among researchers in the life and physical sciences, and on the ability of practitioners and the public to apply research findings effectively.

Build an effective health infrastructure that meets the oral health needs of all Americans and integrates oral health effectively into overall health. The public health capacity for addressing oral health is dilute and not integrated with other public health programs. Although the Healthy People 2010 objectives provide a blueprint for outcome measures, a national public health plan for oral health does not exist. Furthermore, local, state, and federal resources are limited in the personnel, equipment, and facilities available to support oral health programs. There is also a lack of available trained public health practitioners knowledgeable about oral health. As a result, existing disease prevention programs are not being implemented in many communities, creating gaps in prevention and care that affect the nation's neediest populations. Indeed, cutbacks in many state budgets have reduced staffing of state and territorial dental programs and curtailed oral health promotion and disease prevention efforts. An enhanced public health infrastructure would facilitate the development of strengthened partnerships with private practitioners, other public programs, and voluntary groups.

There is a lack of racial and ethnic diversity in the oral health workforce. Efforts to recruit members of minority groups to positions in health education, research, and practice in numbers that at least match their representation in the general population not only would enrich the talent pool, but also might result in a more equitable geographic distribution of care providers. The effect of that change could well enhance access and utilization of oral health care by racial and ethnic minorities.

A closer look at trends in the workforce discloses a worrisome shortfall in the numbers of men and women choosing careers in oral health education and research. Government and private sector leaders are aware of the problem and are discussing ways to increase and diversify the talent pool, including easing the financial burden of professional education, but additional incentives may be necessary.

Remove known barriers between people and oral health services. This report presents data on access, utilization, financing, and reimbursement of oral health care; provides additional data on the extent of the barriers; and points to the need for public-private partnerships in seeking solutions. The data indicate that lack of dental insurance, private or public, is one of several impediments to obtaining oral health care and accounts in part for the generally poorer oral health of those who live at or near the poverty line, lack health insurance, or lose their insurance upon retirement. The level of reimbursement for services also has been reported to be a problem and a disincentive to the participation of providers in certain public programs. Professional organizations and government agencies are cognizant of these problems and are exploring solutions that merit evaluation. Particular concern has been expressed about the nation's children, and initiatives such as the State Children's Health Insurance Program, while not mandating coverage for oral health services, are a positive step. In addition, individuals whose health is physically, mentally, and emotionally compromised need comprehensive integrated care.

Use public-private partnerships to improve the oral health of those who still suffer disproportionately from oral diseases. The collective and complementary talents of public health agencies, private industry, social services organizations, educators, health care providers, researchers, the media, community leaders, voluntary health organizations and consumer groups, and concerned citizens are vital if America is not just to reduce, but to eliminate, health disparities. This report highlights variations in oral and general health within and across all population groups. Increased public-private partnerships are needed to educate the public, to educate health professionals, to conduct research, and to provide health care services and programs. These partnerships can build and strengthen cross-disciplinary, culturally competent, community-based, and community-wide efforts and demonstration programs to expand initiatives for health promotion and disease prevention. Examples of such efforts include programs to prevent tobacco use, promote better dietary choices, and encourage the use of protective gear to prevent sports injuries. In this way, partnerships uniting sports organizations, schools, the faith community, and other groups and leaders, working in concert with the health community, can contribute to improved oral and general health.

CONCLUSION

The past half century has seen the meaning of oral health evolve from a narrow focus on teeth and gingiva to the recognition that the mouth is the center of vital tissues and functions that are critical to total health and well-being across the life span. The mouth as a mirror of health or disease, as a sentinel or early warning system, as an accessible model for the study of other tissues and organs, and as a potential source

of pathology affecting other systems and organs has been described in earlier chapters and provides the impetus for extensive future research. Past discoveries have enabled Americans today to enjoy far better oral health than their forebears a century ago. But the evidence that not all Americans have achieved the same level of oral health and well-being stands as a major challenge, one that demands the best efforts of public and private agencies and individuals.

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