NIH State-of-the-Science Conference on Tobacco Use: Prevention, Cessation, and Control

June 12-14, 2006

William H. Natcher Conference Center National Institutes of Health Bethesda, Maryland

Sponsored by:

- National Cancer Institute, NIH
- Office of Medical Applications of Research, NIH

Co-sponsored by:

- Fogarty International Center, NIH
- National Heart, Lung, and Blood Institute, NIH
- National Human Genome Research Institute, NIH
- National Institute of Child Health and Human Development, NIH
- National Institute of Dental and Craniofacial Research, NIH
- National Institute of Mental Health, NIH
- National Institute of Nursing Research, NIH
- National Institute on Alcohol Abuse and Alcoholism, NIH
- National Institute on Deafness and Other Communication Disorders, NIH
- National Institute on Drug Abuse, NIH
- Office of Behavioral and Social Sciences Research, NIH
- Office of Research on Women's Health, NIH

Partners:

- Administration for Children and Families, DHHS
- Centers for Disease Control and Prevention, DHHS
- Health Resources and Services Administration, DHHS
- Indian Health Service, DHHS
- National Guideline Clearinghouse, AHRQ, DHHS
- U.S. Department of Veterans Affairs



Contents

Introduction	1
Agenda	3
Panel Members	9
Speakers	11
Planning Committee	13
Abstracts	17
Background and Overview Gary A. Giovino, Ph.D., M.S.	19
I. What Are the Effective Population- and Community-Based Interventions To Pr Tobacco Use in Adolescents and Young Adults, Including Among Diverse Popu	
Cultural Approaches to Community and School-Based Tobacco Prevention for Adolesc and Young Adults, Including Priority Populations Lourdes Baezconde-Garbanati, Ph.D., M.P.H.	
Programs and Policies for Prevention John P. Pierce, Ph.D.	37
Policy Interventions and Surveillance Jean Forster, Ph.D., M.P.H.	43
II. What Are the Effective Strategies for Increasing Consumer Demand for and U Proven Individually Oriented Cessation Treatments, Including Among Diverse Populations?	
Increasing Consumer Demand for Effective Tobacco Cessation Treatments: The Promise for Breakthrough Innovation C. Tracy Orleans, Ph.D.	49
Increasing Demand for and Use of Cessation Treatments Among Low-Income and Blue-Collar Populations Elizabeth M. Barbeau, Sc.D., M.P.H.	53
Evidence-Based Practice Center Presentation: Increasing Demand for and Use of Effec Tobacco Cessation Treatments Among Individuals Cathy L. Melvin, Ph.D., M.P.H.	tive

III. What Are the Effective Strategies for Increasing the Implementation of Proven Population-Level Tobacco-Use Cessation Strategies, Particularly by Healthcare Systems and Communities?

Health Systems Changes Michael C. Fiore, M.D., M.P.H.	65
Addressing Tobacco-Related Disease Health Disparities in the Delivery of Community-Based Interventions Phillip Gardiner, Ph.D.	71
QUITLINES: Public-Private Partnerships for Tobacco Control Tim McAfee, M.D., M.P.H.	77
IV. What Is the Effect of Smokeless Tobacco Product Marketing and Use on Populati Harm From Tobacco Use?	ion
Changing Product/New Tobacco Delivery System Dorothy Hatsukami, Ph.D.	83
Policy Perspective for Tobacco Risk Reduction Lynn T. Kozlowski, Ph.D.	91
Epidemiological Perspective for Tobacco Risk Reduction Scott L. Tomar, D.M.D., Dr.P.H.	97
Evidence-Based Practice Center Presentation: Smokeless Tobacco: The Effects of Product Marketing on Use and Population Harm Leah M. Ranney, Ph.D., M.A.	
V. What Is the Effectiveness of Prevention and of Cessation Interventions in Popular With Co-Occurring Morbidities and Risk Behaviors?	tions
Genetics and Smoking Caryn Lerman, Ph.D.	107
Treatment and Prevention of Tobacco Dependence in Individuals with Mental Health and Substance Abuse Disorders Sharon M. Hall, Ph.D.	111
Chronic Disease and Co-Occurring Risk	
Ellen R. Gritz, Ph.D.	115
Evidence-Based Practice Center Presentation: Effective Tobacco Cessation Strategies for Individuals With Co-Occurring Morbidities and Risk Behaviors	101
Leah M. Ranney, Ph.D., M.A.	141

Introduction

Background

Tobacco use remains the Nation's leading preventable cause of premature death. Each year, more than 440,000 Americans die from disease caused by tobacco use, accounting for one in every five deaths. Cigarette smoking alone is responsible for more than 30% of cancer deaths annually in the United States and smoking is also an important cause of death from heart disease, stroke, and chronic obstructive pulmonary disease. Currently, there are an estimated 44.5 million (21%) adult smokers and an estimated 3.75 million (22%) high school student smokers in the United States. Cigarettes are the predominant form of tobacco consumed; however, other forms of tobacco, such as smokeless tobacco, cigars, and pipes, are also used. In addition to the toll in human lives, tobacco use places an enormous economic burden on society. For 1995 to 1999, estimated annual smoking-attributable economic costs in the United States were \$75.5 billion for direct medical care for adults and \$81.9 billion for lost productivity.

Despite enormous progress in reducing the prevalence of tobacco use in the United States, it is unlikely that the *Healthy People 2010* objectives of reducing smoking prevalence to 12% or less in adults and 16% or less in youth will be reached on schedule. A better understanding of how effective strategies for prevention and treatment can be developed and implemented across diverse segments of the population is crucial to accelerate progress; meeting the *Healthy People 2010* prevalence goals is projected to prevent an additional 7.1 million premature deaths after 2010. For this reason, the National Cancer Institute and the National Institutes of Health's Office of Medical Applications of Research will sponsor a State-of-the-Science Conference on Tobacco Use: Prevention, Cessation, and Control, June 12–14, 2006, in Bethesda, Maryland. The key questions to be addressed at the State-of-the-Science Conference are:

- What are the effective population- and community-based interventions to prevent tobacco use in adolescents and young adults, including among diverse populations?
- What are the effective strategies for increasing consumer demand for and use of proven individually oriented cessation treatments, including among diverse populations?
- What are the effective strategies for increasing the implementation of proven population-level tobacco-use cessation strategies, particularly by healthcare systems and communities?
- What is the effect of smokeless tobacco product marketing and use on population harm from tobacco use?
- What is the effectiveness of prevention and of cessation interventions in populations with co-occurring morbidities and risk behaviors?
- What research is needed to make the most progress and greatest public health gains nationally and internationally?

At the conference, invited experts will present information pertinent to these questions, and a systematic literature review prepared under contract with the Agency for Healthcare Research and Quality (AHRQ) will be summarized. Conference attendees will have ample time to ask questions and provide statements during open discussion periods. After weighing the scientific evidence, an unbiased, independent panel will prepare and present a State-of-the-Science statement addressing the key conference questions. This conference is intended for researchers interested in tobacco prevention, cessation, and control; healthcare professionals; healthcare system professionals; health policy experts; public health practitioners; and interested members of the public.

General Information

Conference sessions will be held in the Natcher Conference Center, NIH, Bethesda, Maryland.

The conference may be viewed live via Webcast at http://videocast.nih.gov/. Webcast sessions will also be available after the conference.

The dining center in the Natcher Conference Center is located on the main level, one floor above the auditorium. It is open from 6:30 a.m. to 2:30 p.m., serving hot breakfast and lunch, sandwiches and salads, and snack items. An additional cafeteria is available from 7:00 a.m. to 3:30 p.m., in Building 38A, level B1, across the street from the main entrance to the Natcher Conference Center.

The telephone number for the message center at the Natcher Conference Center is 301–594–7302.

Financial Disclosure

Each speaker presenting at this conference has been asked to disclose any financial interests or other relationships pertaining to this subject area. Please refer to the material in your participant packet for details.

Panel members signed a confirmation that they have no financial or other conflicts of interest pertaining to the topic under consideration.

AGENDA

Monday, June 12, 2006

8:30 a.m. Opening Remarks

Mark Clanton, M.D., M.P.H.

Deputy Director

National Cancer Institute National Institutes of Health

8:40 a.m. Charge to the Panel

Barnett S. Kramer, M.D., M.P.H.

Director

Office of Medical Applications of Research

Office of the Director

National Institutes of Health

8:50 a.m. Conference Overview and Panel Activities

David F. Ransohoff, M.D.

Panel and Conference Chairperson

Professor of Medicine School of Medicine

University of North Carolina at Chapel Hill

9:00 a.m. Background and Overview

Gary A. Giovino, Ph.D., M.S.

Senior Research Scientist

Director, Tobacco Control Research Program

Division of Cancer Prevention and Population Sciences

Department of Health Behavior Roswell Park Cancer Institute

I. What Are the Effective Population- and Community-Based Interventions To Prevent Tobacco Use in Adolescents and Young Adults, Including Among Diverse Populations?

9:15 a.m. Cultural Approaches to Community and School-Based Tobacco Prevention for

Adolescents and Young Adults, Including Priority Populations

Lourdes Baezconde-Garbanati, Ph.D., M.P.H.

Director, Hispanic/Latino Tobacco Education Partnership and Southern California

Cancer Information Services Partnership Program Office (CIS)

Department of Preventive Medicine

University of Southern California Keck School of Medicine

Monday, June 12, 2006 (continued)

I. What Are the Effective Population- and Community-Based Interventions To Prevent Tobacco Use in Adolescents and Young Adults, Including Among Diverse Populations? (continued)

9:40 a.m. Programs and Policies for Prevention

John P. Pierce, Ph.D.

Sam M. Walton Professor for Cancer Research

University of California, San Diego

10:05 a.m. Policy Interventions and Surveillance

Jean Forster, Ph.D., M.P.H.

Professor

Division of Epidemiology and Community Health

University of Minnesota

10:30 a.m. Discussion

Participants with questions or comments for the speakers should proceed to the microphones and wait to be recognized by the panel chair. Please state your name and affiliation. Questions and comments not heard before the close of the discussion period may be submitted on the computers in the registration area. Please be aware that all statements made at the microphone or submitted later are in the public domain.

II. What Are the Effective Strategies for Increasing Consumer Demand for and Use of Proven Individually Oriented Cessation Treatments, Including Among Diverse Populations?

10:55 a.m. Increasing Consumer Demand for Effective Tobacco Cessation Treatments:

The Promise for Breakthrough Innovation

C. Tracy Orleans, Ph.D.

Distinguished Fellow and Senior Scientist

Robert Wood Johnson Foundation

11:20 a.m. Innovations and Outreach to Latino Populations in the United States and Abroad

Eliseo Perez-Stable, M.D.

Professor of Medicine

University of California, San Francisco

11:45 a.m. Increasing Demand for and Use of Cessation Treatments Among Low-Income

and Blue-Collar Populations

Elizabeth M. Barbeau, Sc.D., M.P.H.

Associate Professor

Dana Farber Cancer Institute

Harvard School of Public Health

Monday, June 12, 2006 (continued)

II. What Are the Effective Strategies for Increasing Consumer Demand for and Use of Proven Individually Oriented Cessation Treatments, Including Among Diverse Populations? (continued)

12:10 p.m. Lunch

Panel Executive Session

1:10 p.m. Evidence-Based Practice Center Presentation: Increasing Demand for and Use of

Effective Tobacco Cessation Treatments Among Individuals

Cathy L. Melvin, Ph.D., M.P.H.

Senior Research Fellow and Director

Child Health Services Research

Cecil G. Sheps Center for Health Services Research

Research Associate Professor

Department of Maternal and Child Health

School of Public Health

University of North Carolina at Chapel Hill

1:40 p.m. Discussion

III. What Are the Effective Strategies for Increasing the Implementation of Proven Population-Level Tobacco Use Cessation Strategies, Particularly by Healthcare Systems and Communities?

2:10 p.m. Health Systems Changes

Michael C. Fiore, M.D., M.P.H.

Professor of Medicine

Director, UW-Center for Tobacco Research and Intervention

University of Wisconsin

2:35 p.m. Addressing Tobacco-Related Disease Health Disparities in the Delivery of

Community-Based Interventions

Phillip Gardiner, Ph.D.

Social and Behavioral Sciences Research Administrator

University of California

3:00 p.m. QUITLINES: Public-Private Partnerships for Tobacco Control

Tim McAfee, M.D., M.P.H.

Chief Medical Officer

Free & Clear

3:25 p.m. Discussion

Monday, June 12, 2006 (continued)

IV. What Is the Effect of Smokeless Tobacco Product Marketing and Use on Population Harm From Tobacco Use?

3:55 p.m. Changing Product/New Tobacco Delivery System

Dorothy Hatsukami, Ph.D.

Professor

University of Minnesota

4:20 p.m. Policy Perspective for Tobacco Risk Reduction

Lynn T. Kozlowski, Ph.D.

Professor and Head

Department of Biobehavorial Health

Pennsylvania State University

4:45 p.m. Epidemiological Perspective for Tobacco Risk Reduction

Scott L. Tomar, D.M.D., Dr.P.H.

Associate Professor and Chair

Department of Community Dentistry and Behavioral Science

University of Florida, College of Dentistry

5:10 p.m. Evidence-Based Practice Center Presentation: Smokeless Tobacco: The Effects of

Product Marketing on Use and Population Harm

Leah M. Ranney, Ph.D., M.A.

Research Fellow and Research Associate

Cecil G. Sheps Center for Health Services Research

University of North Carolina at Chapel Hill

5:35 p.m. Discussion

6:00 p.m. Adjournment

Tuesday, June 13, 2006

V. What Is the Effectiveness of Prevention and of Cessation Interventions in Populations With Co-Occurring Morbidities and Risk Behaviors?

8:30 a.m. Genetics and Smoking

Caryn Lerman, Ph.D.

Professor, Department of Psychiatry Director, Tobacco Use Research Center University of Pennsylvania Cancer Center

Tuesday, June 13, 2006 (continued)

V. What Is the Effectiveness of Prevention and of Cessation Interventions in Populations With Co-Occurring Morbidities and Risk Behaviors? (continued)

8:55 a.m. Treatment and Prevention of Tobacco Dependence in Individuals With

Mental Health and Substance Abuse Disorders

Sharon M. Hall, Ph.D.

Professor in Residence, Psychiatry University of California, San Francisco

9:20 a.m. Chronic Disease and Co-Occurring Risk

Ellen R. Gritz, Ph.D.

Olla S. Stribling Distinguished Chair for Cancer Research Professor and Chair, Department of Behavioral Science University of Texas M.D. Anderson Cancer Center

9:45 a.m. Evidence-Based Practice Center Presentation: Effective Tobacco Cessation

Strategies for Individuals With Co-Occurring Morbidities and Risk Behaviors

Leah M. Ranney, Ph.D., M.A.

Research Fellow and Research Associate

Cecil G. Sheps Center for Health Services Research

University of North Carolina at Chapel Hill

10:15 a.m. Discussion

11:15 a.m. Adjournment

Wednesday, June 14, 2006

9:00 a.m. Presentation of the draft State-of-the-Science Statement

9:30 a.m. Public Discussion

The panel chair will call for questions and comments from the audience on the draft statement, beginning with the introduction and continuing through each subsequent section in turn. Please confine your comments to the section under discussion. The chair will use discretion in proceeding to subsequent sections so that comments on the entire statement may be heard during the time allotted. Comments cannot be accepted after 11:30 a.m.

11:00 a.m. Panel Meets in Executive Session

Panel meets in executive session to review public comments. Conference participants are welcome to return to the main auditorium to attend the press conference at 2:00 p.m.; however, only members of the media are permitted to ask questions during the press conference.

Wednesday, June 14, 2006 (continued)

2:00 p.m. Press Conference

3:00 p.m. Adjournment

The panel's draft statement will be posted to www.consensus.nih.gov as soon as possible after the close of proceedings and the final statement will be posted 4 to 6 weeks later.

Panel

Panel Chair: David F. Ransohoff, M.D.

Panel and Conference Chairperson

Professor of Medicine School of Medicine

University of North Carolina at Chapel Hill

Chapel Hill, North Carolina

Frederic C. Blow, Ph.D.

Director

Serious Mental Illness Treatment Research and Evaluation Center

Ann Arbor Veterans Administration Medical Center

Associate Professor

Department of Psychiatry

University of Michigan Medical School

Ann Arbor, Michigan

Marshall H. Chin, M.D., M.P.H.

Associate Professor of Medicine University of Chicago Chicago, Illinois

Bernadine Cimprich, Ph.D., R.N., F.A.A.N.

Associate Professor, Nursing University of Michigan Ann Arbor, Michigan

Lawrence Friedman, M.D.

Independent Consultant Rockville, Maryland

Christine Laine, M.D., M.P.H.

Senior Deputy Editor Annals of Internal Medicine Philadelphia, Pennsylvania

William F. Renn, M.S.W., L.C.S.W., L.C.A.S., C.C.S.

Associate Professor

Department of Psychiatry

University of North Carolina School

of Medicine

Director

University of North Carolina Health Care's Alcohol and Substance Abuse Program

Chapel Hill, North Carolina

Elena V. Rios, M.D., M.S.

President and CEO

National Hispanic Medical Association

Washington, DC

Mary E. Ropka, Ph.D., R.N., F.A.A.N.

Associate Member

Division of Population Science

Fox Chase Cancer Center

Cheltenham, Pennsylvania

Anita L. Sabichi, M.D.

Associate Professor

Department of Clinical Cancer Prevention

University of Texas M.D. Anderson

Cancer Center

Houston, Texas

Marilou G. Tablang-Jimenez, M.D., P.C.

General, Child, and Adolescent Psychiatry Germantown, Maryland

Stephen B. Thomas, Ph.D., FAAHB

Director Center for Minority Health Philip Hallen Professor of Community Health and Social Justice Graduate School of Public Health University of Pittsburgh Pittsburgh, Pennsylvania

Nancy S. Thomason

Founder and President Oklahoma Brain Tumor Foundation Oklahoma City, Oklahoma

Claudette Varricchio, D.S.N., R.N., F.A.A.N.

Rockville, Maryland

Speakers

Lourdes Baezconde-Garbanati, Ph.D., M.P.H.

Director, Hispanic/Latino Tobacco Education Partnership and Southern California Cancer Information Services Partnership Program Office (CIS)

Institute for Health Promotion and Disease Prevention Research and Norris Comprehensive Cancer Center Department of Preventive Medicine Keck School of Medicine University of Southern California Alhambra, California

Elizabeth M. Barbeau, Sc.D., M.P.H.

Associate Professor Dana Farber Cancer Institute Harvard School of Public Health Boston, Massachusetts

Michael C. Fiore, M.D., M.P.H.

Professor of Medicine
Director, UW-Center for Tobacco Research
and Intervention
University of Wisconsin
Madison, Wisconsin

Jean Forster, Ph.D., M.P.H.

Professor
Division of Epidemiology and Community
Health
University of Minnesota
Minneapolis, Minnesota

Phillip Gardiner, Ph.D.

Social and Behavioral Sciences Research Administrator University of California Oakland, California

Gary A. Giovino, Ph.D., M.S.

Senior Research Scientist
Director, Tobacco Control Research
Program
Division of Cancer Prevention and
Population Sciences
Department of Health Behavior
Roswell Park Cancer Institute
Buffalo, New York

Ellen R. Gritz, Ph.D.

Olla S. Stribling Distinguished Chair for Cancer Research Professor and Chair, Department of Behavioral Science University of Texas M.D. Anderson Cancer Center Houston, Texas

Sharon M. Hall, Ph.D.

Professor in Residence, Psychiatry University of California, San Francisco San Francisco, California

Dorothy Hatsukami, Ph.D.

Professor University of Minnesota Minneapolis, Minnesota

Lynn T. Kozlowski, Ph.D.

Professor and Head Department of Biobehavorial Health Pennsylvania State University University Park, Pennsylvania

Caryn Lerman, Ph.D.

Professor, Department of Psychiatry Director, Tobacco Use Research Center University of Pennsylvania Cancer Center Philadelphia, Pennsylvania

Tim McAfee, M.D., M.P.H.

Chief Medical Officer Free & Clear Seattle, Washington

Cathy L. Melvin, Ph.D., M.P.H.

Senior Research Fellow and Director Child Health Services Research Cecil G. Sheps Center for Health Services Research Research Associate Professor Department of Maternal and Child Health School of Public Health University of North Carolina at Chapel Hill Chapel Hill, North Carolina

C. Tracy Orleans, Ph.D.

Distinguished Fellow and Senior Scientist Robert Wood Johnson Foundation Princeton, New Jersey

Eliseo Perez-Stable, M.D.

Professor of Medicine University of California, San Francisco San Francisco, California

John P. Pierce, Ph.D.

Sam M. Walton Professor for Cancer Research University of California, San Diego La Jolla, California

Leah M. Ranney, Ph.D., M.A.

Research Fellow and Research Associate Cecil G. Sheps Center for Health Services Research University of North Carolina at Chapel Hill Chapel Hill, North Carolina

Scott L. Tomar, D.M.D., Dr.P.H.

Associate Professor and Chair Department of Community Dentistry and Behavioral Science University of Florida, College of Dentistry Gainesville, Florida

Planning Committee

Planning Chair: Scott J. Leischow, Ph.D.

U.S. Department of Health and Human Services

Washington, DC

and

Branch Chief

Tobacco Control Research Branch

National Cancer Institute National Institutes of Health

Rockville, Maryland

David Ashley, Ph.D.

Senior Scientist Officer Centers for Disease Control and Prevention Atlanta, Georgia

David Atkins, M.D., M.P.H.

Chief Medical Officer
Center for Practice and Technology
Assessment
Agency for Healthcare Research and Quality
Rockville, Maryland

Cathy L. Backinger, Ph.D., M.P.H.*

Tobacco Control Research Branch
Behavioral Research Program
Division of Cancer Control and Population
Sciences
National Cancer Institute
National Institutes of Health
Bethesda, Maryland

Rosalind A. Breslow, Ph.D., M.P.H.

Epidemiologist
Division of Biometry and Epidemiology
Division of Epidemiology and Prevention
Research
National Institute on Alcohol Abuse
and Alcoholism
National Institutes of Health
Bethesda, Maryland

* Institute Coordinator as of 5/05

Patricia S. Bryant, Ph.D.

Health Scientist Administrator
Behavioral and Social Science Research
Program
Clinical Research Branch, Division of
Clinical Research and Health Promotion
National Institute of Dental and
Craniofacial Research
National Institutes of Health
Bethesda, Maryland

Allison Chausmer, Ph.D.

Translational Research Branch National Institute on Drug Abuse National Institutes of Health Bethesda, Maryland

Monique Ernst, M.D., Ph.D.

Section of Developmental and Affective Neuroscience National Institute of Mental Health National Institutes of Health Bethesda, Maryland

Kenneth Fink, M.D., M.G.A., M.P.H.

Director

Evidence-based Practice Centers Program Center for Outcomes and Evidence Agency for Healthcare Research and Quality Rockville, Maryland

Gary A. Giovino, Ph.D., M.S.

Senior Research Scientist
Director, Tobacco Control Research Program
Division of Cancer Prevention and
Population Sciences
Department of Health Behavior
Roswell Park Cancer Institute
Buffalo, New York

Martha L. Hare, Ph.D., R.N.

Program Director National Institute of Nursing Research National Institutes of Health Bethesda, Maryland

Lynne M. Haverkos, M.D., M.P.H.

Medical Officer National Institute of Child Health and Human Development National Institutes of Health Bethesda, Maryland

Jack E. Henningfield, Ph.D.

Professor of Behavioral Biology Department of Psychiatry and Behavioral Sciences Vice President, Research and Health Policy Pinney Associates Bethesda, Maryland

Jared B. Jobe, Ph.D.

Health Scientist Administrator National Heart, Lung, and Blood Institute National Institutes of Health Bethesda, Maryland

Gary King, Ph.D.

Associate Professor Biobehavioral Health Pennsylvania State University University Park, Pennsylvania

Barnett S. Kramer, M.D., M.P.H.

Director

Office of Medical Applications of Research Office of the Director National Institutes of Health Bethesda, Maryland

Kelli K. Marciel, M.A.

Communications Director Office of Medical Applications of Research Office of the Director National Institutes of Health Bethesda, Maryland

Colleen M. McBride, Ph.D.

Chief, Social & Behavioral Research Branch National Human Genome Research Institute National Institutes of Health Bethesda, Maryland

Ernestine Murray, R.N., M.A.S.

Captain

U.S. Public Health Service Senior Health Policy Analyst Senior Advisor on Tobacco Use Center for Outcomes and Evidence Agency for Healthcare Research and Quality Rockville, Maryland

C. Tracy Orleans, Ph.D.

Distinguished Fellow and Senior Scientist Robert Wood Johnson Foundation Princeton, New Jersey

Terry F. Pechacek, Ph.D.

Associate Director for Science Office on Smoking and Health Centers for Disease Control and Prevention Atlanta, Georgia

Aron Primack, M.D.

John E. Fogarty International Center National Institutes of Health Bethesda, Maryland

Cynthia A. Rooney

Senior Advisor for Consensus Development Office of Medical Applications of Research Office of the Director National Institutes of Health Bethesda, Maryland

Susan Rossi, Ph.D., M.P.H.

Deputy Director Office of Medical Applications of Research Office of the Director National Institutes of Health Bethesda, Maryland

Charlotte A. Schoenborn, M.P.H.

Health Statistician National Center for Health Statistics Centers for Disease Control and Prevention Hyattsville, Maryland

Ed Trapido, Ph.D.

Associate Director Epidemiology and Genetics Program National Cancer Institute National Institutes of Health Bethesda, Maryland

Tatiana M. Foroud, Ph.D.[†]

Panel and Conference Chairperson Associate Professor Director, Hereditary Genomics Division Department of Medical and Molecular Genetics Indiana University School of Medicine Indianapolis, Indiana

15

[†] Panel and Conference Chairperson through 3/31/06

Abstracts

The following are the abstracts of the proposed speaker presentations at the NIH State-of-the-Science Conference on Tobacco Use: Prevention, Cessation, and Control. They are designed for use by the panelists and the participants in the conference, and as a reference document for anyone interested in conference deliberations. We are grateful to the authors.

The abstract for the following presentation does not appear:

Innovations and Outreach to Latino Populations in the United States and Abroad—Eliseo Perez-Stable, M.D.

Cynthia A. Rooney Senior Advisor for Consensus Development Office of Medical Applications of Research Office of the Director National Institutes of Health

Cathy L. Backinger, Ph.D., M.P.H.
Tobacco Control Research Branch
Behavioral Research Program
Division of Cancer Control and Population
Sciences
National Cancer Institute
National Institutes of Health

Background and Overview

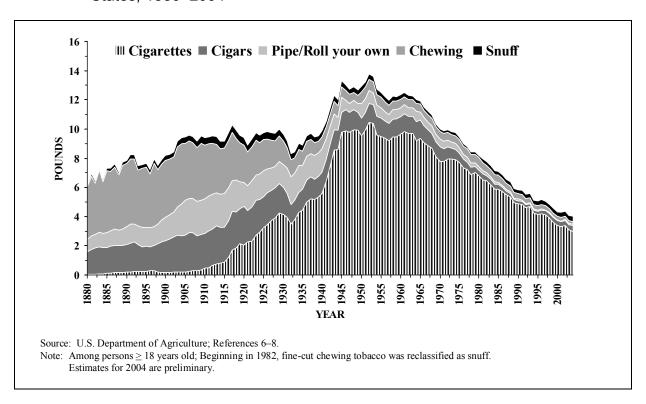
Gary A. Giovino, Ph.D., M.S.

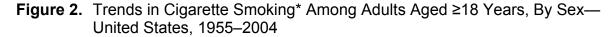
Introduction

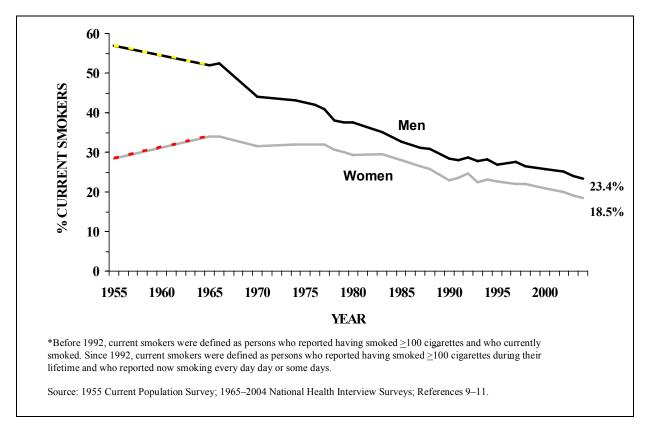
Tobacco use, primarily in the form of cigarettes, has caused more than 14 million premature deaths in the United States since 1964. Tobacco use remains the single leading preventable cause of death in the United States, with at least 8.6 million Americans living with serious disease(s) caused by their smoking, 400,000 current and former smokers dying annually from smoking-attributable diseases, and 38,000 nonsmokers dying annually because of exposure to tobacco smoke pollution. Peto and colleagues estimate that one-half of all smokers, especially those who began as teens, can expect to die of tobacco use. Of these, approximately one-half will die in middle age, losing on average 20–25 years of life expectancy.

Overall U.S. consumption of tobacco products has been declining for several decades (figure 1). ^{6–8} From 1995 through 2004, consumption (in pounds) declined for cigarettes (by 24%), smoking tobacco (i.e., pipe or roll-your-own) (by 23%), and chewing tobacco (by 64%); however, consumption increased for cigars (by 78%) and snuff (by 13%). The prevalence of cigarette smoking among U.S. adults has decreased substantially, from 42.4% in 1965 to 20.9% in 2004 (figure 2). ^{9–11} Consumption of cigarettes has been increasing in developing nations, while decreasing in the United States and most high-income countries. ¹²

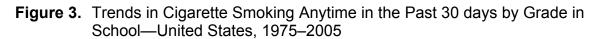
Figure 1. Trends in Per Capita Consumption of Various Tobacco Products—United States, 1880–2004

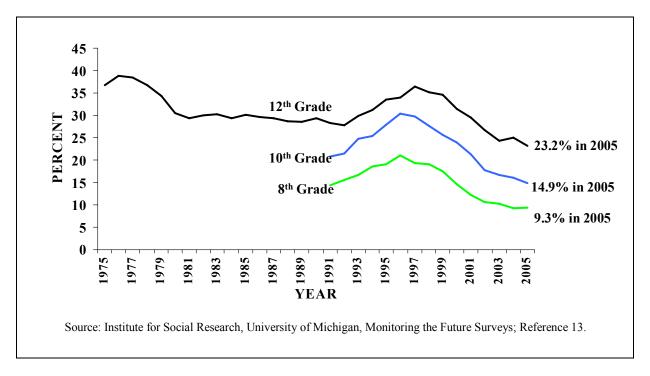






In 2004, the prevalence of cigarette smoking in the United States was higher for men (23.4%) than women (18.5%); for American Indians/Alaska Natives (33.4%) than for Hispanics (15.0%) and Asians (11.3%); for high school dropouts (34.0%) and those with a GED diploma (39.6%) than for those with an undergraduate (11.7%) or graduate (8.0%) degree; and for those living in poverty (29.1%) than for those living at or above the poverty line (20.6%). Among the estimated 42.4% (90.2 million) of persons who had ever smoked at least 100 lifetime cigarettes, 50.6% (45.6 million) were former cigarette smokers. Among U.S. secondary school students, cigarette smoking prevalence increased markedly in the 1990s, peaking in 1996 for 8th and 10th graders and in 1997 for 12th graders (figure 3) and then subsequently declining. The 2005 data suggest that progress toward fewer student smokers is slowing and may even be stopping. Patterns of prevalence suggest that future tobacco-attributable disease will be increasingly concentrated in socially disadvantaged populations, further exacerbating health disparities.

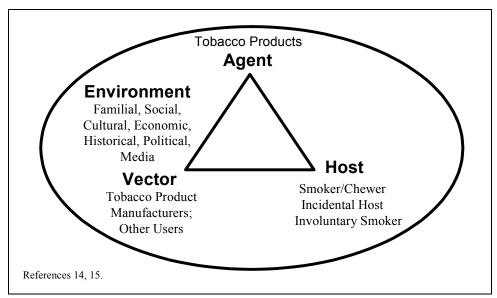




Tobacco use is a multilevel problem that is influenced by a number of factors. An epidemiologic model of tobacco use and nicotine addiction (figure 4) highlights the importance of understanding the roles of the Agent (tobacco product), Host (smoker/user or potential smoker/user), Vector (tobacco product manufacturers), and Environment (e.g., familial, social, economic, and media factors). ^{14–15}

Tobacco products have been changed substantially over the years to influence performance on standard machine tests and the bioavailability of nicotine. Light and ultralight cigarettes tacitly promise health benefits, but are as hazardous as full flavor varieties. Cigarette companies have studied the smoothness of their products, in response to young smokers' concerns about harsh taste. Research on traditional products and potential reduced-exposure products (PREPs) is needed to determine likely human exposures to nicotine and toxic/carcinogenic compounds. In addition, the price of the product influences use, with increasing prices leading to decreased use, both by reducing the number of users and decreasing consumption among continuing users.





The host is the person who uses the product. Some relevant host risk factors include biological susceptibility to addiction, in utero exposure to nicotine, motivation to start or quit, misperceptions, comorbidities, adverse childhood experiences, and self-esteem. This model also includes an incidental host, representing children and adults who are exposed to tobacco smoke pollution and are thus at increased risk of respiratory illnesses, lung cancer, coronary heart disease, and other diseases. 28,29

In epidemiology, the vector is the organism that transports the agent to susceptible individuals.³⁰ Tobacco companies market their products to maximize appeal and allay health concerns.^{26,27,31,32} They undermine public health efforts by resisting the implementation of health-promoting programs and policies.^{33–37} They attempt to manipulate the work of scientists studying the health effects of their products.^{34,38–40} Companies have used pricing strategies, such as discount coupons and multipack discounts, to offset the effects of tax increases.⁴¹

Environmental factors include familial, social, cultural, economic, historical, political, and media-based influences. For example, smoking by peers, siblings, and parents, as well as norms established in the home, can influence uptake. Tobacco growing and tobacco product manufacturing have in many countries become culturally established and economically powerful enterprises that greatly influence political decisions and even attitudes about use. Advice to quit from a health professional, and media influences, such as appearances of smoking in movies, pro-tobacco advertising and promotion, and anti-tobacco messages from the public health sector. The number of States passing smoke-free laws protecting all workers is increasing rapidly. As of April 2006, approximately 43% of U.S. adults live in an area where smoking is banned in private offices, restaurants, and/or bars. In many workers, especially those in the hospitality industry, remain unprotected. Laws protecting nonsmokers can also help smokers reduce consumption. In addition, substantial progress has been made in reducing children's exposure in homes.

Research activities and interventions can address one or more factors on the continuum from cells to society, all with the ultimate goal of minimizing tobacco use to the lowest level possible.⁵⁰

References

- 1. U.S. Department of Health and Human Services. *The Health Consequences of Smoking: A Report of the Surgeon General.* Atlanta, Ga: 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; 2004.
- 2. Armour BS, Woolery T, Malarcher A, Pechacek TF, Husten C. Annual smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 1997–2001. *MMWR Morb Mortal Wkly Rep.* 2005;54:625–628.
- 3. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA*. 2004;291:1238–1245.
- 4. Hyland A, Vena C, Bauer J, et al. Cigarette smoking-attributable morbidity—United States, 2000. MMWR Morb Mortal Wkly Rep. 2003;52:842–844.
- 5. Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr. *Mortality from Smoking in Developed Countries 1950–2000: Indirect Estimation from National Vital Statistics*. Oxford: Oxford University Press, 1994.
- 6. Milmore BK, Conover AG. *Tobacco Consumption in the United States, 1880–1955*. Addendum in, Haenszel W, Shimkin MB, Miller HP. U.S. Department of Health, Education, and Welfare. *Tobacco Smoking patterns In The United States: Public Health Monograph No. 45*. Department of Health, Education, and Welfare, Public Health Service. DHEW Publication no. (PHS) 463; May 1956: 107–111.
- 7. U.S. Department of Agriculture. *Tobacco Situation and Outlook Report*. U.S. Department of Agriculture, Commodity Economics Division, Economic Research Service, TS 228; 1994.
- 8. Capehart T. *Tobacco Outlook. Leaf Production Plummets With End of Program*. Washington, DC: U.S. Department of Agriculture, Economic Research Service, TBS-259; September 23, 2005. Available at: http://usda.mannlib.cornell.edu/reports/erssor/specialty/tbs-bb/2005/tbs259.pdf. Accessed April 5, 2006.
- 9. Haenszel W, Shimkin MB, Miller HP. U.S. Department of Health, Education, and Welfare. *Tobacco Smoking patterns In The United States: Public Health Monograph No. 45*. Department of Health, Education, and Welfare, Public Health Service. DHEW Publication no. (PHS) 463; May 1956.
- 10. National Center for Health Statistics. *Health, United States, 2005 with Chartbook on Trends of Health in Americans*. Hyattsville, Md: National Center for Health Statistics; 2005.

- 11. Maurice E, Trosclair A, Merritt R, et al. Cigarette smoking among adults—United States, 2004. *MMWR Morb Mortal Wkly Rep.* 2005;54:1121–1124.
- 12. Milenkovich Z. The global market for cigarettes. *Tobacco Journal International*. Oct/Nov 2004:70–79.
- 13. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. *Decline in teen smoking appears to be nearing its end*. December 19, 2005. Ann Arbor, Mich: University of Michigan News and Information Services. Available at: http://www.monitoringthefuture.org/data/05data.html#data-cigs. Accessed April 5, 2006.
- 14. Orleans CT, Slade J. *Nicotine Addiction: Principles and Management*. New York: Oxford Univeristy Press; 1993:ix.
- 15. Giovino GA. Epidemiology of tobacco use in the United States. *Oncogene*. 2002;21(48):7326–7340.
- 16. Fant RV, Henningfield JE, Nelson RA, Pickworth WB. Pharmacokinetics and pharmacodynamics of moist snuff in humans. *Tobacco Control*. 1999;8(4):387–392.
- 17. National Cancer Institute. *Risks Associated with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine*. Smoking and Tobacco Control Monograph No. 13. Bethesda, Md: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute; October 2001. NIH Publication No. 02-5074.
- 18. U.S. Food and Drug Administration. Regulations restricting the sale and distribution of cigarettes and smokeless tobacco to children and adolescents; Final rule. 61 *Federal Register* 44395–44618. August 28, 1996.
- 19. Hurt RD, Robertson CR. Prying open the door to the cigarette industry's secrets about nicotine: The Minnesota Tobacco Trial. *JAMA*. 1998;280(13):1173–1181.
- 20. Cummings KM, Morley CP, Horan JK, Steger C, Leavell NR. Marketing to America's youth: evidence from corporate documents. *Tobacco Control*. 2002;11(Supplement 1):i5–i17.
- 21. Wayne GF, Connolly GN. How cigarette design can affect youth initiation into smoking: Camel cigarettes 1983–93. *Tobacco Control*. 2002;11(Supplement 1):i32–i39.
- 22. Hatsukami DK, Giovino GA, Eissenberg T, Clark PI, Lawrence D, Leischow S. Methods to assess potential reduced exposure products. *Nicotine & Tobacco Research*. 2005;7(6):827–844.
- 23. Chaloupka FJ, Wakefield M, Czart C. Taxing tobacco: The impact of tobacco taxes on cigarette smoking and other tobacco use. In: Rabin RL, Sugarman S, eds. *Regulating Tobacco*. New York, NY: Oxford University Pres; 2001:chap 3.

- 24. Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. *Journal of the American Medical Association*. 1999;282:1652–1658.
- 25. Cummings KM, Hyland A, Giovino GA, Hastrup J, Bauer JE, Bansal MA. Are smokers adequately informed about the health risks of smoking and medicinal nicotine? *Nicotine & Tobacco Research.* 2004;6(Supplement 3):S333–S340.
- 26. U.S. Department of Health and Human Services. *Preventing Tobacco Use Among Young People: A Report of the Surgeon General.* Atlanta, Ga: 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.
- 27. U.S. Department of Health and Human Services. *Women and Tobacco: A Report of the Surgeon General*. Rockville, Md: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001.
- 28. California Environmental Protection Agency. *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant*. State of California, California Environmental Protection Agency, Air Resources Board, Office of Environmental Health Hazard Assessment; June 24, 2005. Available at: ftp://ftp.arb.ca.gov/carbis/regact/ets2006/app3exe.pdf. Accessed April 5, 2006.
- 29. Samet JM, Wang SS. Environmental tobacco smoke. In: Lippmann M, ed. *Environmental Toxicants: Human Exposures and Their Health Effects*. 2nd Ed. New York, NY: Wiley and Sons; 1999.
- 30. Last JM, ed. *A Dictionary of Epidemiology*. Fourth Edition. Oxford, UK: Oxford University Press; 2001.
- 31. Pollay RW, Dewhirst T. The dark side of marketing seemingly "light" cigarettes: Successful images and failed fact. *Tobacco Control*. 2002;11(Supplement 1):i18–i31.
- 32. Slade J. Marketing Policies. In: Rabin RL, Sugarman S, eds. *Regulating Tobacco*. New York, NY: Oxford University Press; 2001:chap 4.
- 33. U.S. Department of Health and Human Services. *Reducing Tobacco Use: A Report of the Surgeon General*. Atlanta, Ga: 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; 2000.
- 34. Kluger R. *Ashes to Ashes. America's Hundred-year Cigarette War, the Public Health, and the Unabashed Triumph of Philip Morris.* New York, NY: Alfred A. Knopf; 1996.
- 35. Glantz SA, Balbach ED. *Tobacco War. Inside the California Battles*. Berkeley, Calif: University of California Press; 2000.

- 36. Jamieson KH. "Tax and Spend" Vs. "Little Kids": Advocacy and Accuracy in the Tobacco Settlement Ads of 1997–8. Philadelphia, Pa: Annenberg Public Policy Center of the University of Pennsylvania; 1998.
- 37. Saloojee Y, Dagli E. Tobacco industry tactics for resisting public policy on health. *Bulletin of the World Health Organization*. 2000;78(7):902–910.
- 38. Garne D, Watson M, Chapman S, Byrne F. Environmental tobacco smoke research published in the journal *Indoor and Built Environment* and associations with the tobacco industry. *Lancet*. 2005;365(9461):804–809.
- 39. Muggli M, Hurt RD, Blanke DD. Science for hire: A tobacco industry strategy to influence public opinion on secondhand smoke. *Nicotine and Tobacco Research* 2003;5:303–314.
- 40. Ong EK, Glantz SA. Constructing "sound science" and "good epidemiology": Tobacco, lawyers, and public relations firms. *American Journal of Public Health*. 2001;91:1749–1757.
- 41. Chaloupka FJ, Cummings KM, Morley CP, Horan JK. Tax, price and cigarette smoking: evidence from the tobacco documents and implications for tobacco company marketing strategies. *Tobacco Control.* 2002;11(Suppl 1):i62–i72.
- 42. Wakefield MA, Chaloupka FJ, Kaufman NJ, Orleans CT, Barker DC, Ruel EE. Effect of restrictions on smoking at home, at school, and in public places on teenage smoking: Cross sectional study. *BMJ*. 2000;321(7257):333–337.
- 43. Noland MP, Kryscio RJ, Hinkle J, et al. Relationship of personal tobacco-raising, parental smoking, and other factors to tobacco use among adolescents living in a tobacco-producing region. *Addictive Behaviors*. 1996;21:349–361.
- 44. Von Gernet A. Origins of nicotine use and the global diffusion of tobacco. In: Ferrence R, Slade J, Room R, Pope M, eds. *Nicotine and Public Health*. Washington, DC: American Public Health Association; 2000.
- 45. World Bank. *Curbing the Epidemic: Governments and the Economics of Tobacco Control.* Washington, DC: The World Bank; 1999.
- 46. American Nonsmokers' Rights Foundation. *Summary of 100% Smokefree State Laws and Population Protected by State and Local Laws*. Available at: http://www.nosmoke.org/pdf/SummaryUSPopList.pdf. Accessed May 11, 2006.
- 47. Shopland DR, Gerlach KK, Burns DM, Hartman AM, Gibson JT. State-specific trends in smoke-free workplace policy coverage: The current population survey tobacco use supplement, 1993 to 1999. *Journal of Occupational and Environmental Medicine*. 2001;43(8):680–686.
- 48. Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces on smoking behaviour: systematic review. *BMJ*. 2002;325(7357):188–194.

- 49. Soliman S, Pollack HA, Warner KE. Decrease in the prevalence of environmental tobacco smoke exposure in the home during the 1990s in families with children. *American Journal of Public Health*. 2004;94(2):314–320.
- 50. National Cancer Institute. Tobacco Research Implementation Plan. *Priorities for Tobacco Research Beyond the Year 2000*. Bethesda, Md: National Institutes of Health, National Cancer Institute, Tobacco Research Implementation Group; 1998.

Cultural Approaches to Community and School-Based Tobacco Prevention for Adolescents and Young Adults, Including Priority Populations

Lourdes Baezconde-Garbanati, Ph.D., M.P.H.

The United States has experienced a dramatic reduction in adolescent and adult smoking, with remarkable health benefits. However, among some groups—such as racial/ethnic minorities; those of low socioeconomic status (SES), especially youth; those 18–24 years of age; and lesbian, gay, bisexual, and transgender individuals (LGBT)—tobacco use remains a significant and persistent problem. It is also a major contributor to health disparities. ³

Role of Culture

Although many multilevel factors contribute to adolescent and young adult smoking, ^{4,5} culture is one of the most powerful predictors of behavior and of the decision to smoke. ^{5–9} Considering the increased numbers of racial/ethnic and other cultural groups in the United States, communities are challenged in implementing evidence-based comprehensive tobacco prevention programs at the community and school levels and in proving their effectiveness.

Community-Based Cultural Approaches

California has been at the forefront in addressing diverse population tobacco prevention issues and in changing social and cultural norms that favor tobacco use in these communities. Programs developed for these purposes are not necessarily youth focused, but are more community focused in their approach to preventing smoking and developing social norms. Yet they have been successful in helping to reduce the prevalence rate among youth and those 18–24 years of age in part by changing social and cultural norms. To address the needs of particularly vulnerable populations at the community level, four ethnic networks/partnerships (African American [AA], American Indian [AI], Hispanic/Latino [H/L], and Asian/Pacific Islander [API]) were established in 1991 by the California Department of Health Services, Cancer Control Branch, Tobacco Control Section (TCS). Their purpose has been a common one: to address at a statewide level in culturally competent ways the technical assistance and training needs of TCS-funded programs that focus on particularly vulnerable populations.

The work has ranged from countering pro-tobacco influences on and targeting of the African American population with menthol cigarettes among the AA Tobacco Education Partnership to educating populations on the sacred use of tobacco by the AI Tobacco Education Partnership. The H/L Partnership has worked on smoke-free housing choice in various communities and inoculated Hispanic/Latino communities against industry sponsorship. The API Partnership has developed leadership strategies to move communities to readiness in tobacco control and focused on policies that promote Asian/Pacific Islander health. Since then, other programs have been established by the California TCS (the LGBT, low SES, and a program working with labor unions [BUILT]), forming a partnership with the ethnic networks known as California's Priority Populations Partnerships.

As a group, the California Priority Population Partnerships have informed and educated policy- and decisionmakers on tobacco use and continued to address tobacco industry marketing with a special focus on those 18–24 years of age, secondhand smoke workplace policies that protect all Californians from the deleterious effects of tobacco smoke, and other issues in highly vulnerable populations or groups with particularly high prevalence rates.³

Together with other competitive grantees, 68 Local Lead Agencies, a statewide and ethnic-specific media campaign, and a cadre of statewide programs, these efforts have formed part of the State of California's comprehensive tobacco-control movement. They have built local and statewide tobacco control capacity at the community level, increased cultural competence among those conducting prevention and cessation efforts, created cultural specific and language appropriate approaches by partnering with tribal and local governments, advocacy and community groups, schools and college campuses. They have worked with the ethnic and general media promoting social change, bringing ethnic communities to the table, and successfully passing policy initiatives that have made California's Tobacco Control Movement an example for the nation.

The success of California's comprehensive community-based approach is best evidenced by the reductions in tobacco prevalence in California among particularly vulnerable communities. Data from the California Tobacco Survey³ show significant decreases in adult, young adult, and adolescent smoking. Adult smoking prevalence declined from 16.6% in 2002 to 15.4% in 2004, the second lowest in the Nation. Among young adults, prevalence rates declined from 21.3% in 2002 to 18.3% in 2004, and among youth, prevalence rates declined from 16.0% in 2002 to 12.2% in 2004 in comparison to the national average youth smoking rate of 22.3% (http://www.dhs.ca.gov/tobacco).

School-Based Cultural Approaches

Out-of-school youth and young adults are at greatest risk for smoking. Many are reached via broad community-level interventions including some of the mechanisms presented above, and media campaigns such as the American Legacy Truth campaign, which has produced TV ads in conjunction with youth to counter pro-tobacco industry influences.

Schools have been another important vehicle for reaching young people, and are an ideal ground for accessing large numbers of individuals that reflect the economic and racial/ethnic diversity of their respective communities. Several school-based programs have been shown to be effective in particular age groups or in ethnic-specific versus multicultural school settings.

One such program is Project Fun Learning About Vitality Opinions and Respect (FLAVOR) developed for 6th graders in California. This project specifically targeted Asian and Hispanic/Latino students. The program is a culturally tailored eight-session tobacco prevention curricula with a strong theoretical base in social cognitive and social influence theories. The program was tested in three types of schools, mostly Asian, H/L and multicultural schools along with different conditions. Data show the program was most effective for Hispanic/Latinos in mostly Hispanic/Latino schools. The greatest impact was perceived among Hispanic/Latino boys in particular. Lessons from Project FLAVOR suggest that although nontailored programs can also be effective, there seems to be a greater effect in tobacco

prevention when, like Project FLAVOR, the program is not only age specific but also culturally tailored. At a time of cost containment, the key is to understand how much tailoring is needed, or whether the effect produced by the nontailored program is enough to create the desired changes in a targeted population over time.

The Keepin' It REAL (Refuse, Explain, Avoid, Leave) program developed by Michael Hecht et al. ¹³ is a culturally grounded, classroom-based prevention intervention targeting substance use among urban middle-school children (grades 6–8) that has been proven effective. The curriculum consists of 10 lessons promoting anti-drug norms, teaching resistance and other social skills. Booster activities and a media campaign complement the program. Three versions of the program have been produced. These include a Mexican American version, a Black/White version, and a multicultural version. The multicultural version was developed by incorporating five lessons each from the Mexican American and Black/White versions of the program. Its effectiveness in preventing substance use is endorsed by the U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration (SAMHSA), Center for Substance Abuse Prevention. SAMHSA features Keepin' it REAL on its Web site as a model program (http://www.samhsa.gov). According to SAMHSA, this program has been adequately tested and has provided evidence that it is effective in preventing or reducing substance use and other high-risk behaviors among Mexican American and Black/White students in grades 6–8.

Other cultural approaches have ranged from adaptations of the Life Skills Training (LST) program to Hispanic/Latino-centered and "Afrocentric" approaches developed for and with these communities. Some of the more successful culturally centered approaches have emphasized protective factors, such as identifying "anti-drug education," "extracurricular activities," and "sports participation." These factors seem to alter attitudes and behaviors about tobacco among African Americans. Other interventions have focused on cultural values ("familismo" [sense of family as a core value], "respecto" [respect towards elders, figures of authority, etc.]) among Hispanic/Latino adolescents or on group consensus building, onsistent with theories of interdependence among Hispanic/Latinos²⁰ and filial piety among Asian/Pacific Islanders. Sills

Investigators such as Sussman et al. ^{19,21,22,23} have developed effective prevention programs especially for high risk populations, such as those in alternative high school settings. Although these programs have been used in Hispanic/Latino samples, they have not been culturally tailored to this population. Sussman's Project Towards No Tobacco Use (TNT)²³ is a tobacco use prevention and cessation program aimed at young teens. Project EX is a tobacco prevention and cessation program aimed at older teens. Project EX was tested via a large field experimental design. It was found to be effective in preventing tobacco use and increasing cessation among older teens.

The same group of investigators also produced Towards No Drug Abuse (Project TND),²¹ a drug abuse prevention program conducted in south California alternative high school system for older teens, particularly for those at high risk for drug abuse (including tobacco). The program is composed of nine sessions, including health motivation, social skills, and decisionmaking. The curriculum has been scientifically tested among 21 schools that were randomly assigned to standard care (control), classroom only, or a classroom plus semester-long school–community component. Results of TND revealed significant positive long-term program

effects for the program interventions. For example, project TND reduced hard drug use in 46% of those who were successfully followed. TND was the first program to demonstrate long-term self-reported behavioral effects on hard drug use among high-risk youth by using a school-based, limited-session model. Project TND is now considered a model program by the Centers for Substance Abuse Prevention, Health Canada, Sociometrics, Inc., and the National Institute on Drug Abuse, the Centers for Disease Control and Prevention, Centers for Substance Abuse Prevention, and the Department of Education of California.

Also identified in the literature are other evidenced-based programs that were originally developed for non-Hispanic Whites but were later culturally adapted with various degrees of success among African Americans and Hispanic/Latinos. The success in implementation of these programs has varied in part due to the large differences among youth in identification and adherence to cultural norms, success of planned versus unplanned tailoring, type and degree of adaptation, and degree of cultural grounding desired to reach homogeneous or mixed groups in the same classroom setting.

Less scientific evidence is available on the effectiveness of culturally tailored tobacco control programs for American Indians and Asian/Pacific Islanders. Some research points to combining skills building with American Indian community conditions with varying effects. For Asian Pacific Islander Americans, programs that address delayed initiation and extend interventions through young adulthood (18–24 years old) have been favored.

Thomas, in a Cochrane Collaboration review on school-based programs for preventing smoking, identified 16 randomized controlled trials of school-based programs to prevent smoking among children who had never smoked.²⁷ A variety of approaches were identified, including programs that targeted ethnic minorities and particular cultural groups. Approaches ranged from information giving to social influence approaches, generic social skills training, and community interventions. In deciding what was most effective, the author did not find much evidence that information giving alone, for example, is effective. The review did find overall positive short-term effects on children's smoking behaviors. However, no long-term effects were identified. Limited evidence was found on the effects of interventions that included developing generic social competence, as well as among those with a multi-modal approach that included school and community-based initiatives. The author concludes that although those in the intervention groups usually smoked less than the control groups, many studies actually showed no effect of the intervention and that there is thus a lack of "high quality" evidence regarding the effectiveness of many of these interventions.

Sifting through the literature, however, one factor that seems to have influenced finding positive effects on school and community programs is their comprehensive nature as well as how they vary in the adoption of guidelines that lead to reductions in smoking and that increase opportunities for cessation. ^{28,29,30} The effects of school combined with community programs appear to have the greatest range and more long-term effects on substance abuse rates, and they are the only programs to show any effects on parental behavior. Nevertheless, definitive conclusions, especially regarding long-term effects of these programs, were not found. ²⁷ One other finding seems consistent in reviewing cultural approaches: an emphasis on cultural values ³¹ seems to have an impact on participation and retention in a variety of programs, especially when these interventions are implemented in culturally specific versus mixed environments.

Discussion

Several cultural approaches to community- and school-based tobacco prevention for adolescents and young adults, including priority populations, have been presented. Community-based culturally specific programs, such as the California Priority Populations Partnerships in California, have shown to be effective in countering pro-tobacco influences in communities specifically targeted by the tobacco industry, helping to reduce youth and young adult smoking rates, including among diverse populations. California's comprehensive tobacco control program has achieved changes in social and cultural norms that favor tobacco and has attained gains in reducing second-hand smoke exposure and motivating individuals to quit. Their achievements are best exemplified by policies that have passed at the local and State level, resulting in a 60% reduction in per capita consumption since the beginning of the program (from 1988 to 2004),³ and the impact of these policies in reducing the prevalence rates over time.

In addition to these community-based approaches, successful school-based programs have been reviewed. Model programs such as Keepin' It REAL, Project FLAVOR, and TND among others have been presented. These programs have been proven to be successful when administered at the appropriate school age level, even though in some, such as TNT and TND there has been no cultural tailoring. Other programs such as Keepin' It REAL and Project FLAVOR show the benefits of culturally tailoring a program to its specific target. TND was the only program to show long-term effects.

While multiple school-based interventions report positive effects and have been deemed suitable for dissemination, there is not a sufficiently strong body of evidence on cultural adaptation or the use of culturally grounded approaches to reach definitive conclusions regarding which is more effective in the short and long term. The greater effectiveness of nontailored versus tailored and of cultural adaptations or interventions created specifically for priority groups is still unclear or nonexistent. For example, we found no published curriculum that addresses the specific cultural needs of gay low SES youth of color. Nevertheless, research points to some of the highest rates of smoking among youth in these groups.

The discovery of new interventions, the testing of their effectiveness, and their dissemination in particularly vulnerable cultural groups is still needed. Needed also are long-term population intervention trials that assess the differential impact of culturally grounded programs when implemented along the acculturation spectrum or with different populations. We know from the literature that many youth and young adults prefer speaking English both at school and at home. This is less of an option for newcomers to America with limited English proficiency and who may need culturally and linguistic adaptation if we are to adequately reach them in a timely fashion. The extent to which cultural adaptations are needed likely depends on the extent to which youth identify with their families' cultures of origin and their ability to speak the English language.

Although we have some insights especially into short-term program effectiveness, more research is needed in order to obtain a precise picture of the most effective community- and school-based cultural approaches, especially if we are aiming for long-term effects. It is critical to also clarify that there are large differences between youth and adults in terms of needs for cultural tailoring, mostly based on acculturation status, and that the findings for youth cannot

necessarily be extrapolated to those of adult community-based approaches for example. Also needed are interventions that focus on approaches among American Indian and Asian/Pacific Islander populations and other vulnerable population groups. Large variations within the Asian/Pacific Islander community by nativity and acculturation will make this particularly challenging.

It seems that interventions at the middle school level are effective in the short term in reducing adolescent smoking. However, dramatic changes in the prevalence occur when children reach high school age. This is particularly marked among high-risk, low-SES White and Hispanic adolescents. It is still unclear what exactly occurs in that transition period and how we can best develop interventions for youth that will have the longest possible lasting effects. If we are to argue for comprehensive programs at the school and community level, it is particularly important to understand the effect of culture especially on interventions that incorporate a large environmental context and that aim at more permanent reductions in adolescent smoking over time.

Acknowledgement: I would like to thank Dr. Luanne Rohrbach, Dr. Jennifer Unger, Dr. Richard Clayton, Dr. Linda Jouridine Alexander, Marisol Romero, Enrique Ortega, and others who contributed with their ideas, reviews, and suggestions. Research was supported by grants from NCI-TTURC NIH CA-98-029 (2004–2009) renewal and the CDHHS, TCS-HLTEP #04-35309. Support was also provided by the Tobacco Research Network on Disparities (TReND) funded by the National Cancer Institute (NCI) and the American Legacy Foundation.

- 1. Centers for Disease Control and Prevention. *National Youth Tobacco Survey*. Atlanta, Ga: CDC; 2006.
- 2. Fichtenberg CM, Glantz SA. Association of the CA Tobacco Control Prog. with declines in cigarette consumption and mortality from heart disease. *N Engl J Med.* 2000;343(24):1772–1777.
- 3. Tobacco Education Research Oversight Committee (TEROC). *Confronting a Relentless Adversary: A Plan for Success*. California Department of Health and Human Services: TCS; 2006.
- 4. Appleyard J, Messeri P, Lyndon Haviland, M. Smoking among Asian American and Hawaiian/Pacific Islander youth: 2000 NYTS. *API J of Health*. 2001;8(1):5–14.
- 5. Unger JB, Cruz T, Shakib S, et al. Exploring the cultural context of tobacco use. *Nicotine and Tobacco Research*. 2003;5(1):S101–S117.
- 6. Unger JB, Baezconde-Garbanati L, Soto C. Family and peer risk and protective factors for tobacco use among Am Indian adolescents in CA. *Ethnicity in Substance Abuse*. 2004;3(4):1–15.

- 7. Unger JB, Cruz TB, Rohrbach LA, et al. English language use as a risk factor for smoking initiation among Hispanic and Asian American adolescents. *Health Psychology*. 2003;19(5):403–410.
- 8. Oetting ER, Beauvais F. Orthogonal cultural identification theory: The cultural identification of minority adolescents. *International Journal of the Addictions*. 1990–1991;25(5A/6A):655–685.
- 9. Baezconde-Garbanati L. Sociocultural framework for Hispanic/Latino adolescent tobacco use research. In: National Cancer Institute, ed. *Changing Adolescent Smoking Prevalence. Smoking and Tobacco Control. Monograph 14.* Bethesda, Md: National Cancer Institute; 2001.
- 10. Baezconde-Garbanati L, et al. Entering a new era: strategies of the H/LTEN for organizing and mobilizing Hispanic communities. In: Frost ML, ed. *Planning and Implementing Effective Tobacco Education and Prevention Programs*. Springfield, Ill: Charles C. Thomas Publishers; 1999.
- 11. American Legacy Foundation. Youth programs. Available at: http://www.americanlegacy.org/americanlegacy. Accessed April 15, 2006.
- 12. Ritt-Olson A, Lichtman K, Unger JB, et al. Developing Project FLAVOR: A Culturally Relevant Tobacco Prevention Program for Young Adolescents. *Journal of Health Promotion Practice*. Under review.
- 13. Hecht ML, Marsiglia FF, Elek E, et al. Culturally grounded substance use prevention: An evaluation of the Keepin' it R.E.A.L. curriculum. *Prev. Sci.* 2003;Dec 4(4):233–248.
- 14. Botvin GJ, Batson H, Witts-Vitale S, et al. A psychosocial approach to smoking prevention for urban Black youth. *Public Health Reports*. 1989;104:573–582.
- 15. Botvin GJ, Dusenbury L, Baker E, James-Ortiz S, Kerner J. A skills training approach to smoking prevention among Hispanic youth. *Behavioral Medicine*. 1989;12(3):279–296.
- 16. Cherry VR, Belgrave FZ, Jones W, Kennon DK, Gray FS, Phillips F. NTU: An Afrocentric approach to substance abuse prevention among African American youth. *The Journal of Primary Prevention*. 1998;18(3):319–339.
- 17. Litrownik AJ, Elder JP, Campbell NR, et al. Evaluation of a tobacco and alcohol use prevention program for Hispanic migrant adolescents: protective factor of parent-child communication. *Prev Med.* 2000;31:124–133.
- 18. Chen X, Unger JB, Cruz TB, Johnson CA. Smoking patterns of Asian-American youth in CA and their relationship with acculturation. *Journal of Adolescent Health*. 1999;24(5):321–328.
- 19. Sussman S, Yang D, Baezconde-Garbanati L, Dent C. Drug abuse prevention program development: Results among Latino and non-Latino White adolescents. *Evaluation and the Health Professions*. 2003;26(4):355–379.

- 20. Castro FG. Drug use and drug related issues. In: Molina CW & Molina MA, eds. *Latino Health in the U.S.: A Growing Challenge*. Washington, DC: American Public Health Association; 1994:425–446.
- 21. Sun W, Skara S, Sun P, Dent CW, Sussman S. Project Towards No Drug Abuse: Long-term substance use outcomes evaluation. *Prev Med.* 2006;42(3):188–192.
- 22. Dent CW, Sussman S, Ellickson P, Brown P, Richardson J. Is current drug abuse prevention programming generalizable across ethnic groups? *Am Behavioral Scientist*. 1996;39(7):911–918.
- 23. Sussman S, Dent CW, Stacy AW. Project towards no tobacco use: A review of the findings and future directions. *American Journal of Health Behavior*. 2002;26(5):354–365.
- 24. Ortega E, Romero M, Baezconde-Garbanati L, et al. *The Status of Tobacco Use Prevention Interventions in Priority Populations: A Critical Review of The Literature*. Unpublished manuscript USC TTURC, Institute for Health Promotion and Disease Prevention Research, University of Southern California (USC); 2003.
- 25. Pentz MA. *Preventing Drug Abuse Through the Community: Multicomponent Programs Make the Difference*. NIDA National Conference on Drug Abuse Prevention Research; 1996.
- 26. Hodge F. American Indian and Alaska Native Teen Cigarette Smoking: A Review. National Cancer Institute. Changing Adolescent Smoking Prevalence. Smoking and Tobacco Control Monograph No. 14. Bethesda, Md: U.S. DHHS, NIH, NCI, NIH Pub. 02-5086; November 2001.
- 27. Thomas R. *School-Based Programmes For Preventing Smoking (Review)*. The Chochrane Collaboration. Wiley Publishers, The Cochrane Library. 2006;Issue 1. Available at: http://www.theocochranelibrary.com. Accessed April 2006.
- 28. California Department of Education. Recommendations for an Effective Statewide Tobacco Use Prevention Education Program: The Report of the TUPE Recommendations Task Force. Sacramento, Calif: California Department of Education; 2005.
- 29. CDC. Guidelines for school health programs to prevent tobacco use and addiction. *MMWR Morb Mortal Wkly Rep.* February 25, 1994;43(RR-2):1–18.
- 30. Glynn TJ. Essential elements of school-based smoking prevention programs. *Journal of School Health*. May 1989;59(5):181–188.
- 31. Marin G, Van Oss Marin B. *Research With Hispanic Populations. Applied Social Research Methods Services.* Newbury, Calif: Sage Publications; 1991.

Programs and Policies for Prevention John P. Pierce, Ph.D.

Public Health and Smoking Behavior

Public health mandates to change smoking behavior in the population come from the overwhelming evidence of the health consequences of cigarette smoking. The major health consequence specifically linked to smoking is lung cancer, and its incidence has been demonstrated to be a power function of both duration of smoking and its intensity (daily consumption level). Thus, to reduce the health consequences, public health goals are:

- 1. Reduce the initial proportion of people who become dependent smokers
- 2. Reduce the consumption pattern of dependent smokers
- 3. Promote early successful cessation among dependent smokers

Smoking behavior starts with cognitions about smoking that are observable in U.S. residents as young as 10 years old.⁴ Early experimentation is common in adolescents from 12 to 14 years old,⁵ and many people are dependent smokers before they reach the legal age for purchase of cigarettes.⁵ The level of daily cigarette consumption of smokers tends to be higher in those who started smoking at younger ages, and this level has been modified by environmental and social rules.⁶ Lower levels of daily consumption are associated with more successful quit attempts, although chances of successful quitting are generally low even for those with high levels of motivation.

Effective Tobacco Control Strategies

There is good evidence to support the use of six tobacco control strategies by public health professionals to reduce dependent smoking. These six strategies are: (a) restricting the rights of the tobacco industry to market their products, (b) restricting the rights of smokers to expose others to secondhand smoke, (c) conducting a mass media campaign aimed at denormalizing tobacco use, (d) enforcing laws that ban sales of cigarettes to minors, (e) increasing excise taxes to increase cigarette price and reduce demand, and (f) including effective smoking education in schools. A comprehensive campaign combines these strategies into a single program that can achieve results that far outweigh those of the individual strategies alone. The first such comprehensive program in the United States was undertaken in California. This expert statement will limit its focus to two of the above approaches: (a) tobacco industry marketing and (b) comprehensive campaigns.

Strategy 1: Restrict Tobacco Industry Marketing

Cigarettes are a legal product in the United States and, as such, manufacturers are entitled to use commercial marketing practices to build demand. Marketing is well known to be categorized as having four elements: (a) product presentation, (b) unit price, (c) promotion, and (d) placement.

Product presentation includes the size of the package, its functioning in retrieving a cigarette, and the colors and text on the package. The standard package size in the United States has been 20 cigarettes, and that seems well suited to the size of pockets and purses. Providing smaller quantities for sale has been attempted in different countries, but in the United States this has been achieved by merchants breaking open packs and selling small quantities of cigarettes. Restrictions on the product have focused on warning labels: the content, minimum text size, and color presentation. Two countries are experimenting with requiring graphic depictions of health consequences and warnings in large black type on a white background that take up a large proportion of the packaging surface.

Unit price: All else being equal, higher prices lead to reduced demand. However, with highly addictive substances, higher prices often result in a resetting of priorities for disposable income. It is notable that only a small portion of smokers, apparently, use price as the critical determinant of where and which cigarettes they purchase. However, there is good evidence that the tobacco industry's use of price-subsidizing promotions in the United States resulted in significant increases in the initiation of smoking among 14–17-year-old adolescents.

Promotion: The right to advertise tobacco products on television and in other broadcast media in the United States was removed in 1971. Following this removal, there was a marked decline in incidence of initiation of adolescents 14–17 years old that remained in effect through the mid-1980s, suggesting that restricting advertising might be effective in decreasing product use in those under the legal age to purchase cigarettes. Studies of innovative tobacco industry campaigns established to launch products to a new demographic group also demonstrated that significant advertising in the mass media could bring new users into the smoking market, and that those new users were mainly between the ages of 14 and 17. ^{10,11}

Communication theories suggest that, to be effective, advertising needs to engage nonusers with the advertising message so that they become curious about trying the product. 12 These theories argue that there should be a hierarchy of effects in which the target individuals who were receptive to the advertising message were not only exposed to it, but liked the message and acted to demonstrate that liking. Such receptivity is correlated with smoking behavior. 13 It predicts which committed never smokers (CNS) will experiment with smoking over a 3-year period, ¹⁴ as well as which CNS will be adult smokers 6 years later. ¹⁵ As predicted by advertising theories, adolescents who are receptive to cigarette brand advertising were also more likely to be curious about smoking, increasing the probability of experimenting with smoking. ¹⁶ The growth of this evidence in the mid-1990s on the effectiveness of tobacco marketing in encouraging adolescents to start smoking led to further restrictions on the marketing practices of the tobacco industry as part of the negotiated Master Settlement Agreement between tobacco companies and State Attorneys General in 1998. 17,18 Ecological evidence of the effectiveness of these restrictions is that they coincided with a downturn in adolescent smoking following 10 consecutive years of increasing rates. This evidence is consistent in a number of surveillance data sets, the most popular of which is the Monitoring of the Future Surveillance System, which has monitored smoking behavior in a random sample of high school students every year since 1976.¹⁹

Placement in movies also is a well-recognized form of marketing. The price of this advertising varies with the character who uses the product (a hero using a product is priced the

highest) and whether or not use of the product helps with the plot.^{20–22} There are now two longitudinal studies that demonstrate that young adolescents whose favorite movie stars smoke on screen or who are exposed to a large number of movies portraying smokers are more likely to start smoking.^{23,24}

Strategy 2: Conduct Comprehensive Tobacco Control Programs

Tobacco control programs are community-wide programs aimed at reducing tobacco usage. Early evidence of success of community programs appeared in the 1970s with the Stanford Three Communities Project, in which multiple behaviors were targeted.²⁵ The first statewide program that focused only on tobacco was conducted in Sydney, Australia and used the majority of its budget to purchase mass media advertising. It was shown to effectively reduce prevalence. 26-28 Comprehensive statewide programs were introduced in the United States in the 1990s. The goal of the California program (1990–present) was to change the social norms surrounding tobacco so as to impact all smoking behavior. While the Massachusetts program (1993–2002) included youth programs, they were more focused on encouraging smokers to quit. Florida introduced a comprehensive program (1997–2002) aiming only to prevent youth from starting to smoke. All of these programs used mass media advertising as a central core of their interventions. California was the only program to use legislation requiring smoke free workplaces and school campuses to protect nonsmokers. Starting in 1996, California aggressively enforced laws restricting minors' access to tobacco. Massachusetts endeavored to protect nonsmokers through mass media messages and community action. Florida was set apart by its innovative use of media events to mobilize youth. Both California and Massachusetts provided support for cessation using smokers' helplines.

All three programs were carefully evaluated, and each was demonstrated to reduce smoking behavior. The campaigns in both California and Massachusetts have been evaluated as reducing smoking prevalence^{29,30} and all three programs have been evaluated as reducing adolescent smoking initiation.^{31–35}

- 1. U.S. Department of Health and Human Services. *Reducing Tobacco Use. A Report of the Surgeon General*, 2000. Atlanta, Ga: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 2000.
- 2. Peto, R. Influence of dose and duration of smoking on lung cancer rates. In: Zaridze DG, Peto R, eds. *Tobacco: A Major International Health Hazard*. Lyons, France: International Agency for Research on Cancer; 1986:23–33.
- 3. Flanders WD, Lally CA, Zhu BP, Henley SJ, Thun MJ. Lung cancer mortality in relation to age, duration of smoking, and daily cigarette consumption: Results from Cancer Prevention Study II. *Cancer Res.* 2003;63:6556–6562.
- 4. Choi WS, Gilpin EA, Farkas AJ, Pierce JP. Determining the probability of future smoking among adolescents. *Addiction*. 2001;96:313–323.

- 5. Gilpin EA, Choi WS, Berry C, Pierce JP. How many adolescents start smoking each day in the United States? *J Adolesc Health*. 1999;25:248–255.
- 6. Gilpin EA, White MM, Farkas AJ, Pierce JP. Home smoking restrictions: which smokers have them and how they are associated with smoking behavior. *Nic Tob Res.* 1999;1:153–162.
- 7. Klonoff EA, Fritz JM, Landrine H, Riddle RW, Tully-Payne L. The problem and sociocultural context of single-cigarette sales. *J Am Med Assoc.* 1994;271:618–620.
- 8. Emery S, Gilpin EA, White MM, Pierce JP. How adolescents get their cigarettes: implications for policies on access and price. *J Natl Cancer Inst.* 1999;91:184–186.
- 9. Pierce JP, Gilmer TP, Lee L, Gilpin EA, de Beyer J, Messer K. Tobacco industry pricesubsidizing promotions may overcome the downward pressure of higher prices on initiation of regular smoking. *Health Econ.* 2005;14:1061–1071.
- 10. Pierce JP, Lee L, Gilpin EA. Smoking initiation by adolescent girls, 1944 through 1988. An association with targeted advertising. *JAMA*. 1994;271:608–611.
- 11. Pierce JP, Gilpin EA. A historical analysis of tobacco marketing and the uptake of smoking by youth in the United States: 1890–1977. *Health Psychol*. 1995;14:500–508.
- 12. Smith RE, Swinyard WR. Cognitive responses to advertising and trial: Belief strength, belief confidence and product curiosity. *J Advertising*. 1988;17:3–14.
- 13. Evans NA, Farkas A, Gilpin EA, Berry C, Pierce JP. Influence of tobacco marketing and exposure to smokers on adolescent susceptibility to smoking. *J Natl Cancer Inst*. 1995;87:1538–1545.
- 14. Pierce JP, Choi WS, Gilpin EA, Farkas AJ, Berry C. Tobacco industry promotion of cigarettes and adolescent smoking. *J Am Med Assoc.* 279:511–515.
- 15. Gilpin EA, White MM, Messer K, et al. Receptivity to tobacco advertising and promotions as young adults predicts current established smoking as young adults. *Am J Pub Health*. In press.
- 16. Pierce JP, Distefan JM, Kaplan RM, Gilpin EA. The role of curiosity in smoking initiation. *Addict Behav.* 2005;30:685–96.
- 17. Pierce JP, Gilpin EA. How did the Master Settlement Agreement change tobacco industry expenditures for cigarette advertising and promotions? *Health Promot Pract*. 2004;5(3 Suppl):84S–90S.
- 18. Gilpin EA, Distefan JM, Pierce JP. Population receptivity to tobacco advertising/promotions and exposure to anti-tobacco media: effect of Master Settlement Agreement in California: 1992–2002. *Health Promot Pract.* 2004;5(3 Suppl):91S–98S.

- 19. Johnston LD, O'Malley PM, Bachman JG, et al. *Monitoring the Future National Survey Results on Drug Use, 1975–2004: Vol. 1.* Bethesda, Md: National Institute on Drug Abuse; 2004.
- 20. Distefan JM, Gilpin EA, Sargent JD, Pierce JP. Do movie stars encourage adolescents to start smoking? Evidence from California. *Prev Med.* 1999;28:1–11.
- 21. DeLorme DC. Moviegoers' experiences and interpretations of brands in films revisited. *J Advertising*. 1999;28:71–73.
- 22. Higgins KT. There's gold in silver screen product plugs. Advertising Age. 1985;19:6.
- 23. Distefan JM, Pierce JP, Gilpin EA. Do favorite movie stars influence adolescent smoking initiation? *Am J Pub Health*. 2004;94:1239–1244.
- 24. Sargent JD, Dalton M, Beach M, Bernhardt A, Heatherton T, Stevens M. Effect of cigarette promotions on smoking uptake among adolescents. *Prev Med.* 2000;30:320–327.
- 25. Farquhar JW, Maccoby N, Wood PD, et al. Community education for cardiovascular health. *Lancet.* 1977;1:1192–1195.
- 26. Pierce JP, Dwyer T, Frape G, Chapman S, Chamberlain A, Burke N. Evaluation of the Sydney Australia "Quit. For Life" anti-smoking campaign. Part 1. Achievement of intermediate goals. *Med J Australia*. 1986;144:341–344.
- 27. Pierce JP, Macaskill P, Hill D. Long-term effectiveness of mass media led antismoking campaigns in Australia. *Am J Public Health*. 1990;80:565–569.
- 28. Dwyer TJ, Pierce JP, Hannam CD, Burke N. Evaluation of the Sydney "Quit. For Life" antismoking campaign. Part 2. Changes in smoking prevalence. *Med J Aust*. 1986;144:344–7.
- 29. Pierce JP, Gilpin EA, Emery SL, et al. Has the California tobacco control program reduced smoking. *J Am Med Assoc.* 1998;280:893-899.
- 30. Biener LJ, Harris JE, Hamilton W. Impact of the Massachusetts tobacco control programme: population based trend analysis. *Br Med J.* 2000;321:351–354.
- 31. Bauer UE, Johnson TM, Hopkins RS, Brooks RG. Changes in youth cigarette use and intentions following implementation of a tobacco control program: findings from the Florida Youth Tobacco Survey, 1998–2000. *J Am Med Assoc.* 2000;284:723–728.
- 32. Sly DF, Trapido E, Ray S. Evidence of the dose effects of an antitobacco counteradvertising campaign. *Prev Med.* 2002;35:511–518.
- 33. Siegel M, Biener L. The impact of an antismoking media campaign on progression to established smoking: results of a longitudinal youth study. *Am J Pub Health*. 2000;90:380–386.

- 34. Hersey JC, Niederdeppe J, Ng SW, Mowery P, Farrelly M, Messeri P. How state counter-industry campaigns help prime perceptions of tobacco industry practices to promote reductions in youth smoking. *Tob Control*. 2005;14:377–383.
- 35. Pierce JP, White MM, Gilpin EA. Adolescent smoking decline during California's tobacco control programme. *Tob Control*. 2005;14(3):207–212.

Policy Interventions and Surveillance

Jean Forster, Ph.D., M.P.H.; Rachel Widome, Ph.D.; Debra Bernat, Ph.D.

1. Policies To Restrict Exposure of Youth and Young Adults to Environmental Tobacco Smoke

Clean indoor air (CIA) policies are Federal, State, local, and institutional policies that prohibit smoking in specified public places such as workplaces, schools, daycare centers, and healthcare facilities. While CIA policies have been in existence for more than 30 years, the number, strength, and breadth of these laws have dramatically escalated in recent years, even in such difficult locations as bars, restaurants, and blue-collar worksites. Almost 500 cities have adopted CIA policies covering workplaces, restaurants and/or bars, and 13 States have adopted 100% smoke-free workplaces, including bars and restaurants.¹

The primary purpose of these policies is to reduce the health risks of environmental tobacco smoke (ETS) exposure. The adverse health effects of ETS exposure are thoroughly discussed in a recent report from the California Environmental Protection Agency, which labeled ETS as a toxic air contaminant based on these effects. For children, ETS exposure is causally associated with developmental problems such as low birth weight and sudden infant death syndrome; respiratory illnesses, such as bronchitis and pneumonia; chronic respiratory symptoms; asthma induction and exacerbation; and middle ear infections. These problems are all less likely to occur and/or relieved by reducing exposure to ETS.

In addition, growing evidence suggests that CIA policies can have a powerful negative effect on smoking uptake by adolescents. These laws can reduce the visibility of role-models who smoke, limit the opportunities for youth to smoke alone or in groups and to exchange cigarettes with other smokers, and diminish the perceived social acceptability of smoking.³ A number of studies show that smoking bans in the home, at school, at work and in the community are associated with less progression to smoking, less consolidation of experimental into regular smoking, and more quitting among adolescents.⁴⁻⁶

2. Policies To Restrict Youth Access to Tobacco

In the 1992 Synar Amendment, Congress mandated that all States and territories must enact laws that prohibit the sale of tobacco to minors and enforce these laws with compliance checking. Despite delays in effectively implementing the requirements of Synar, sales to minors as measured by Synar-mandated compliance checks have decreased nationwide since State laws went into effect. Additionally, research has shown that youth sales can be reduced through active enforcement of these laws. However, research examining whether these policies reduce youth smoking has yielded mixed results. Several early studies found that active enforcement can reduce youth smoking; other examinations have found little no effect. Certainly, youth access policy is more likely to be effective if enforcement is consistent and uniform in geographically contiguous areas.

Some believe that, even if youth access policies limit adolescents' commercial access to cigarettes, youth will simply substitute social sources for commercial sources. ^{19,20} An increase in the use of social sources has been linked to greater commercial restrictions. ^{17,21} Of teens who smoke, it is the heavier smokers who are most likely to provide to other teens, either as a gift or for money. ²² Wolfson et al. postulates that this is due to the fact that being a heavy smoker may be at least partially based on having the greatest access to cigarettes, including commercial access. ²²

Another tactic aimed at reducing youth access is penalizing youth for possession, use, and purchase (PUP) of tobacco. Most tobacco control advocates do not favor emphasizing PUP policy because they feel it reinforces the tobacco industry messages that tobacco is for adults, and implies that the individual holds sole responsibility for choosing to smoke.²³ Currently there is no evidence that PUP enforcement reduces youth smoking rates.

3. Tobacco Excise Tax

Product-specific (excise) taxes are levied on tobacco products (in addition to applicable sales taxes) at the Federal, State, and some local levels. The Federal tax on cigarettes is currently 39 cents per pack; State excise taxes range from 7 cents (South Carolina) to \$2.46 (Rhode Island) and averages 92.3 cents per pack (median 79 cents per pack).²⁴ The range of State excise taxes reflects recent large increases enacted via statewide referenda and as a solution for recent budget shortfalls in many States. Since 2002, 41 States and the District of Columbia have adopted 57 excise tax increases, averaging 48 cents for each increase.²⁵

Decades of econometric research show that smokers are price-sensitive, and that increasing the price of cigarettes reduces demand. Most reports indicate that adolescents are at least as price-sensitive as adults; however, most of the studies were conducted over a narrow range of taxes considerably lower than current excise taxes. Also, youth smoking behaviors are often less intense and habitual compared to adult smoking, and thus their responses to price potentially less predictable. The largest effects of price are seen in heavier smokers, older age youth, and males, ^{26–29} which is consistent with reports that young, experimental, and female smokers obtain most of their cigarettes from social sources, and are least likely to purchase cigarettes. ³⁰

A series of papers using the Monitoring the Future longitudinal data from 1978 to 1994 found a stronger effect of price for young adult smoking than for adult or adolescent smoking. Daily, moderate, and heavy young adult smoking are all negatively correlated with the price of cigarettes, as are smoking cessation and regression to lighter smoking. ^{29,31}

4. Tobacco Use Surveillance Systems

Tobacco surveillance includes ongoing data collection to assess tobacco use at a given time and to monitor trends over time. These data are critical for tobacco use prevention, as they are used to guide research, public health programming, and public policy. Current best practices for tobacco control include participation in national and State surveillance systems.³²

Several surveillance systems track tobacco use among adolescents and young adults nationally. These surveillance systems include the Youth Risk Behavior Surveillance System, the

Behavioral Risk Factor Surveillance System, the National Survey on Drug Use & Health, and the Monitoring the Future Survey. The only national surveillance system devoted solely to assessing tobacco use and related attitudes and beliefs is the National Youth Tobacco Survey.³³ This was the first national survey to provide estimates of tobacco use for middle school students. This survey was administered in 1999, 2000, and 2002 and was a joint effort of the CDC and the American Legacy Foundation. All of these surveillance systems are designed to monitor tobacco use nationally, and several are also designed to provide State-specific estimates.

Several States have also implemented State surveillance systems to monitor youth tobacco use, primarily through the use of school-based surveys. For example, California administers the California Student Tobacco Survey, a biennial survey administered to 7th, 9th, and 11th grade students, to monitor statewide trends in tobacco use.³⁴ Minnesota also administers a statewide survey to 6th, 9th, and 12th graders every 3 years to assess tobacco use.³⁵

Given the importance of surveillance systems for tobacco use prevention, planning for continued surveillance is critical. Several issues warrant consideration. First, many surveillance systems rely on telephone surveys. Responses to telephone surveys, however, have declined in recent years, due to advances in telephone technology and cellular phone use. Thus, new methodologies for conducting surveillance may need to be considered. Another focus for future surveillance systems will be examining prevalence rates and trends in communities.

- 1. American Nonsmokers' Rights Foundation. *Municipalities with Local 100% Smokefree Laws*. Current in effect as of April 17, 2006. Berkeley, Calif: American Nonsmokers' Rights Foundation; 2006.
- 2. California Environmental Protection Agency. *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant.* Sacramento, Calif: CalEPA; Jun 24 2005.
- 3. Alesci NL, Forster JL, Blaine T. Smoking visibility, perceived acceptability, and frequency in various locations among youth and adults. *Prev Med.* Mar 2003;36(3):272–281.
- 4. Farkas AJ, Gilpin EA, White MM, Pierce JP. Association between household and workplace smoking restrictions and adolescent smoking. *JAMA*. Aug 9 2000;284(6):717–722.
- 5. Siegel M, Albers AB, Cheng DM, Biener L, Rigotti NA. Effect of local restaurant smoking regulations on progression to established smoking among youths. *Tob Control*. Oct 2005;14(5):300–306.
- 6. Wakefield MA, Chaloupka FJ, Kaufman NJ, Orleans CT, Barker DC, Ruel EE. Effect of restrictions on smoking at home, at school, and in public places on teenage smoking: cross sectional study. *BMJ*. Aug 5 2000;321(7257):333–337.
- 7. U.S. Department of Health and Human Services. *State Oversight of Tobacco Sales to Minors*. Atlanta, Ga: U.S. Department of Health and Human Services, Office of the Inspector General; 1995.

- 8. DiFranza JR, Dussault GF. The federal initiative to halt the sale of tobacco to children—the Synar Amendment, 1992–2000: Lessons learned. *Tob Control*. Apr 2005;14(2):93–98.
- 9. State Tobacco Activities Tracking and Evaluation (STATE) System. Available at: http://www.cdc.gov/tobacco/statesystem. Accessed Mar 30, 2005.
- 10. Altman DG, Wheelis AY, McFarlane M, Lee H, Fortmann SP. The relationship between tobacco access and use among adolescents: A four community study. *Soc Sci Med.* Mar 1999;48(6):759–775.
- 11. Feighery E, Altman DG, Shaffer G. The effects of combining education and enforcement to reduce tobacco sales to minors. A study of four northern California communities. *JAMA*. Dec 11 1991;266(22):3168–3171.
- 12. Gemson DH, Moats HL, Watkins BX, Ganz ML, Robinson S, Healton E. Laying down the law: Reducing illegal tobacco sales to minors in central Harlem. *Am J Public Health*. Jun 1998;88(6):936–939.
- 13. Jason LA, Ji PY, Anes MD, Birkhead SH. Active enforcement of cigarette control laws in the prevention of cigarette sales to minors. *JAMA*. Dec 11 1991;266(22):3159–3161.
- 14. Rigotti NA, DiFranza JR, Chang Y, Tisdale T, Kemp B, Singer DE. The effect of enforcing tobacco-sales laws on adolescents' access to tobacco and smoking behavior. *N Engl J Med*. Oct 9 1997;337(15):1044–1051.
- 15. Stead LF, Lancaster T. A systematic review of interventions for preventing tobacco sales to minors. *Tob Control*. Jun 2000;9(2):169–176.
- 16. Cummings KM, Hyland A, Perla J, Giovino GA. Is the prevalence of youth smoking affected by efforts to increase retailer compliance with a minors' access law? *Nicotine Tob Res*. Aug 2003;5(4):465–471.
- 17. Forster JL, Murray DM, Wolfson M, Blaine TM, Wagenaar AC, Hennrikus DJ. The effects of community policies to reduce youth access to tobacco. *Am J Public Health*. Aug 1998;88(8):1193–1198.
- 18. Widome R, Forster J, Hannan P, Perry C. Does tobacco youth access policy lead to fewer youth smokers? Data from the MACC study 2000–2003: Division of Epidemiology & Community Health, University of Minnesota; 2006.
- 19. Fichtenberg CM, Glantz SA. Youth access interventions do not affect youth smoking. *Pediatrics*. Jun 2002;109(6):1088–1092.
- 20. Harrison PA, Fulkerson JA, Park E. The relative importance of social versus commercial sources in youth access to tobacco, alcohol, and other drugs. *Prev Med.* Jul 2000;31(1):39–48.

- 21. DiFranza JR, Coleman M. Sources of tobacco for youths in communities with strong enforcement of youth access laws. *Tob Control*. Dec 2001;10(4):323–328.
- 22. Wolfson M, Forster JL, Claxton AJ, Murray DM. Adolescent smokers' provision of tobacco to other adolescents. *Am J Public Health*. Apr 1997;87(4):649–651.
- 23. Wakefield M, Giovino G. Teen penalties for tobacco possession, use, and purchase: evidence and issues. *Tob Control.* Jun 2003;12 Suppl 1:i6–13.
- 24. Campaign for Tobacco-Free Kids. State Cigarette Excise Tax Rates & Rankings. Available at: http://www.tobaccofreekids.org/research/factsheets/index.php?CategoryID=18. Accessed Apr 25, 2006.
- 25. Campaign for Tobacco-Free Kids. State Cigarette Tax Increases since January 1, 2002. Available at: http://www.tobaccofreekids.org/research/factsheets/index.php?CategoryID=18. Accessed Apr 25, 2006.
- 26. Cawley J, Markowitz S, Tauras JA. Lighting up and slimming down: The effects of body weight and cigarette prices on adolescent smoking initiation. *Journal of Health Economics*. 2004;23:293–311.
- 27. Gruber J. *Youth Smoking in the US: Prices and Policies*. Cambridge, Mass: National Bureau of Economic Research; 2000. Working Paper no. 7506.
- 28. Liang L, Chaloupka FJ. Differential effects of cigarette price on youth smoking intensity. *Nicotine Tob Res.* Feb 2002;4(1):109–114.
- 29. Tauras JA, Chaloupka FJ. Determinants of Smoking Cessation: An Analysis of Young Adult Men and Women. In: Grossman M, Hsieh CR, eds. *Economics of Substance Abuse*. Northampton, MA Edward Elgar Publishing Limited; 2001:337–364.
- 30. Ringel JS, Pacula RL, Wasserman J. Youth Access to Cigarettes: Results from the 1999 *National Youth Tobacco Survey.* Washington, DC: American Legacy Foundation; Nov 2000.
- 31. Tauras JA. Can public policy deter smoking escalation among young adults? *Journal of Policy Analysis and Management*. Fall 2005;24(4):771–784.
- 32. U.S. Department of Health and Human Services. *Best Practices for Comprehensive Tobacco Control Programs—August 1999*. Atlanta, Ga: 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; 1999.
- 33. Centers for Disease Control and Prevention. Tobacco use among middle and high school students—United States, 1999. *JAMA*. Mar 1 2000;283(9):1134–1136.
- 34. Austin G, Skager R. *Tenth Biennial California Student Survey on Drug, Alcohol, and Tobacco Use: 2003–2004.* Sacramento, Calif: California Attorney General's Office; 2004.

- 35. Minnesota Student Survey Overview. *Linking Health and Learning: Coordinated School Health*. Available at: http://www.mnschoolhealth.com/resources.html?ac=data. Accessed April 18, 2006.
- 36. Tuckel P, O'Neill H. The vanishing respondent in telephone surveys. *Journal of Advertising Research*. 2002;42(5):26–48.

Increasing Consumer Demand for Effective Tobacco Cessation Treatments: The Promise for Breakthrough Innovation

C. Tracy Orleans, Ph.D.

More U.S. adults have quit smoking than remain current smokers. But 44.5 million (20.9%) continue to smoke, with the highest rates among low-socioeconomic status (SES) and racial/ethnic minority populations. Although 70% of adult smokers want to quit, and as many as 40% make a serious quit attempt each year, only 20–30% report using an effective behavioral counseling or pharmacological treatment (e.g., nicotine replacement therapy, bupropion) able to double their quitting success rates. Lowest rates of treatment use are seen in populations with highest rates of tobacco use. Increasing smokers' demand for and use of evidence-based cessation treatments represents an extraordinary opportunity to extend lives and reduce healthcare costs and burden and represents our single best hope of reaching the *Healthy People 2010* adult smoking prevalence goal of 12%. The push-pull-capacity model offers a framework for understanding this opportunity in the context of broader cancer control research-to-practice efforts.

Science Push

The scientific platform for efforts to expand cessation treatment use and reach has never been stronger. Formal clinical practice guidelines based on over 6,000 articles have identified practical, evidence-based, and cost-effective interventions (psychosocial and pharmacologic) that can be delivered in a variety of settings and modalities (e.g., healthcare, community, quitline, or online) and individually tailored or targeted to the needs of priority populations.² This highly credible scientific evidence has been widely promoted to healthcare providers, health plans. policymakers, and advocates and has furnished a compelling rationale for new policies and treatment benefits. ⁴ But it has been much less well communicated to consumers—smokers and their families. Recent survey and focus group data reveal wide public uncertainty about the value of these treatments, difficulties discriminating effective and ineffective aids and approaches, and broad misconceptions about the harms of nicotine replacement therapy (NRT) use.³ The advent of new medications (i.e., varenicline, rimonabant), a 2007 guideline update (and the promotional opportunities both will bring), and the growth in consumer-directed health plans make this a critical time for innovative, theory-driven research to explore consumer treatment needs, expectations, and decisionmaking processes, and to develop communications strategies that will boost the appeal and use of treatments that work.

Delivery Capacity

Policymakers and healthcare and tobacco control leaders have made great strides in expanding the capacity to deliver effective treatments. An increasing number of national, State, and professional groups (medicine, nursing, pharmacy, dentistry, mental health, and cessation specialists) offer cessation-related training and assistance to deliver brief cessation advice and treatment. The proportion of health plans using some system to identify smokers has risen

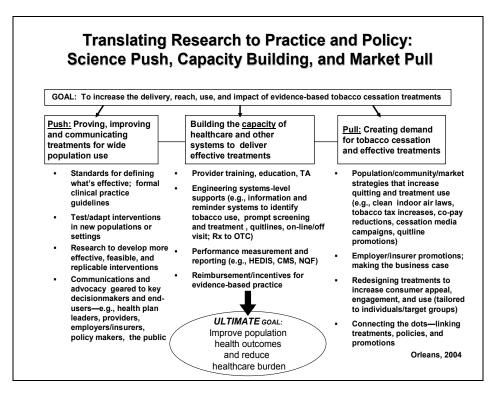
from 15% in 1997 to 71% in 2002. Roughly two-thirds of smokers currently report physician advice to quit, advice that is associated with increased use of effective therapies, though only 38% actually receive them.^{3,8–11} Advances in health information technology are rapidly expanding capacity for computerized reminder systems that boost delivery of counseling and medication in "5A" primary care interventions. ¹² Quitlines, now available nationwide through a single toll-free number, have given smokers and providers an unprecedented, barrier-free conduit to effective counseling.^{7,8,13} Growth in online services and the over-the-counter availability of NRT products also have increased treatment access.³ While declining public and private funding for State and local tobacco control threatens this progress, tobacco cessation advice and treatment are now metrics in national healthcare quality, and pay-for-performance initiatives will provide new incentives for their delivery as part of routine primary care and new opportunities for research to identify healthcare systems and policies that boost treatment use and quit rates.^{2,4,5,10,11,12}

Consumer and Market Demand

Without consumer demand, or "market pull," neither strengthening the science base nor increasing delivery capacity will yield optimal use of effective cessation treatments. Populationbased policies recommended by the Centers for Disease Control and Prevention¹² (CDC) to increase quitting and/or treatment use (i.e., clean indoor air laws, tobacco tax increases, reduced out-of-pocket treatment costs, cessation media campaigns) are reaching unprecedented numbers of smokers, strengthening the norms, supports, and incentives for treatment use. Comprehensive clean air laws now cover more than 25% of the population; average combined State and Federal cigarettes taxes have reached \$1.30 per pack and continue to rise; 41 State Medicaid programs and 98% of U.S. health plans provide coverage for some form of counseling or pharmacotherapy; and both Medicare and the Department of Veterans Affairs (VA) cover cessation counseling. ⁸ Cessation media campaigns, including those that target underserved minority smokers, have been found to increase quitting, quitline calls, NRT use, and the use of underused treatment benefits, and are a vital counterweight to the billions spent on tobacco advertising each year, including for products marketed as "reduced harm" products. 3,5,13-15 Huge demand can be unleashed when these strategies are combined. Aligning a strong clean indoor air law and high tobacco taxes with promotion of free quitline counseling and nicotine replacement, the New York City Department of Health achieved an 11% 1-year citywide guit rate and an immediate decline in heart attack rates.^{3,7} These results are compelling evidence for the promise of population-based efforts to boost consumer treatment use and demand, and a powerful incentive for investment in needed quitting resources and for research to understand how best to design, align, and even incentivize coordinated approaches combining policies, treatments, and promotions.

Boosting market demand also requires promotions and incentives aimed at employers, insurers, and health plans—powerful intermediary "consumers." New evidence and tools establishing the "business case" for tobacco dependence treatment and the inclusion of tobacco use screening and treatment in national pay-for-performance quality metrics bring new prospects. ^{8,9,16} But insurers and employers also place great weight on direct employee and enrollee request, ¹⁴ adding to a growing imperative for innovations to improve the ways in which evidence-based treatments are designed and delivered, so that they have greater consumer appeal and better engage quitters across their full quitting journeys. One of the most exciting new

frontiers in tobacco cessation research and practice involves applying design principles and processes used to build demand for other consumer products to meet this challenge.¹⁷ Early findings of the national Consumer Demand Roundtable suggest that this approach could lead to major breakthroughs in treatment use and impact, and in the Nation's prospects for achieving its *Healthy People 2010* tobacco goals.



- 1. Centers for Disease Control and Prevention. Annual smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 1997–2001. *MMWR Morb Mortal Wkly Rep.* 2005;54:625–628.
- 2. Fiore MC, Bailey WC, Cohen SJ, et al. *Treating Tobacco Use and Dependence: Clinical Practice Guideline*. Rockville, Md: U.S. Department of Health and Human Services, Public Health Service; 2000.
- 3. Cummings KM, Hyland A. Impact of nicotine replacement therapy on smoking behavior. *Amer Rev Public Health.* 2005;26:583–599.
- 4. Cokkinides VE, Ward E, Jemal A, Thun MJ. Under-use of smoking-cessation treatments. *Am J Prev Med.* 2005;28:119–122.
- 5. Orleans CT. Commentary on smoking cessation policy. In Isaacs S, ed. *VA in the Vanguard: Building on Success in Smoking Cessation*. San Francisco: UCSF School of Medicine; October 2005:31–40.

- 6. Kerner J, Rimer BK, Emmons K. Dissemination research and research dissemination: How can we close the gap? *Health Psychol*. 205;24:443–447.
- 7. Schroeder SA. What to do with a patient who smokes. *JAMA*. 2005;294:482–487.
- 8. Orleans CT, Woolf SH, Rothemich SF, Marks JS, Isham GJ. The top priority: Building a better system for tobacco cessation counseling. *Am J Prev Med*. In press.
- 9. McPhillips-Tangum C, Bocchino C, Carreon R, et al. Addressing tobacco in managed care: Results of the 2002 survey. *Prev Chronic Dis.* 2004;1:A04. Epub 2004 Sep 15.
- 10. Maciosek MV, Coffield AB, Edwards, et al. Priorities for improving utilization of clinical preventive services: Results. *Am J Prev Med.* In press.
- 11. Quinn VP, Stevens VJ, Hollis J, et al. Tobacco-cessation services and patient satisfaction in nine nonprofit HMOs. *Am J Prev Med.* 2005;29:77–84.
- 12. Hopkins DP, Briss PA, Ricard CJ, et al. Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. *Am J Prev Med*. 2001;20(S2);16–66.
- 13. Fiore M, Croyle R, Curry S, et al. Preventing 3 million premature deaths and helping 5 million smokers quit: A national action plan for tobacco cessation. *Am J Public Health*. 2004:94:205–210.
- Boyd NR, Sutton C, Orleans CT, et al. Quit Today—A targeted communications campaign to increase use of the Cancer Information Services by African American smokers. *Preventive Med.* 1998;27:S50–S60.
- 15. Biener L, Reimer RL, Wakefield, M, et al. Impact of smoking cessation aids and mass media among recent quitters. *Am J Prev Med.* 2006;30:217–224.
- 16. Bondi MA, Harris JR, Atkins D, et al. Employer coverage of clinical preventive services in the United States. *Am J Health Promotion*. In press.
- 17. Kelley T. The Art of Innovation. New York: Random House; 2001.

Increasing Demand for and Use of Cessation Treatments Among Low-Income and Blue-Collar Populations

Elizabeth M. Barbeau, Sc.D., M.P.H.

This abstract addresses the question: What are effective strategies for increasing consumer demand for and use of proven, individually oriented cessation treatments among low-income and blue-collar populations? Smoking prevalence remains high among individuals of lower socioeconomic position, whether measured by income, occupation, or educational attainment. In 2000, prevalence of current smoking among blue-collar workers was 35.4%, compared to 20.5% among white-collar workers. Likewise, smoking among individuals with incomes below the Federal poverty threshold was 34.7% compared to 20.7% for those earning three times the poverty threshold. Blue-collar and low-income smokers attempt to quit as often as their more advantaged counterparts, but they are less likely to succeed. The relative lack of success may be due, in part, to lower use of proven cessation treatments. According to data from the 2000 National Health Interview Survey, smokers with lower incomes and less education were less likely to use proven cessation treatments (counseling and/or pharmacotherapy as specified in the Public Health Service (PHS) guidelines) compared to those with higher incomes and more education (table 1). These patterns were evident for both genders, Blacks and Whites, but not for Hispanics, who were represented in small numbers in this survey.

A key assumption embedded in the question addressed in this abstract is that pharmacotherapy and counseling are proven cessation treatments across all social classes. This abstract thus focuses on synthesizing current knowledge and critical scientific gaps regarding effective strategies for increasing consumer use of and demand for these proven treatments. Potential strategies for increasing demand for and use of treatments can be divided into two broad categories: reducing barriers to use of treatments and promoting use of treatments.

Reducing Barriers to Use of Treatments Through Health Insurance Programs

Out-of-pocket expenditures for counseling and pharmacotherapy can pose financial barriers to their use. Insurance-based financial coverage for smoking cessation treatments is one way to lower this barrier, and empirical evidence to date indicates that insurance-based coverage increases use of smoking cessation treatments. In a comparison of different levels of coverage within a large health maintenance organization over 2 years, Curry et al. found that the use of all cessation services was greater with full coverage than with cost-sharing plans (11.6% vs. 3.5–3.7%). In a randomized control trial of 1,204 smokers who received either a self-help kit or fully covered benefits for nicotine replacement therapy (NRT)and behavioral counseling, Schauffler et al. found that full coverage of benefits resulted in significantly higher use of NRT (25% vs. 14%, p=0.001). In a similar study in the Netherlands, Kaper et al. arandomly assigned 1,266 smokers to an intervention group, which received an offer of reimbursement for nicotine replacement therapy, bupropion and behavioral counseling, or to a control group, which received no offer of reimbursement. During the reimbursement period, 10.8% of smokers in the intervention group reported having used a smoking cessation treatment, compared to 4.1% in the control group (Odds Ratio [OR]=2.9, 95% Confidence Interval [CI] 1.8–4.7). None of the possible effect

modifiers, including educational attainment and income, modified the effect of reimbursement on the use of treatments.

With respect to low-income smokers, Medicaid is an important insurance vehicle through which to assess strategies for increasing demand for and use of proven cessation treatment. In 2000, approximately 32 million low-income persons in the United States received their health insurance coverage through the Federal/State Medicaid program; approximately 11.5 million (36%) of these persons smoked. Cummings et al. reported that use of the NRT patch increased by 57% among Medicaid recipients when full financial coverage was provided, even after adjusting for other sociodemographic differences.

Labor-management health and welfare funds provide health insurance to 10 million unionized workers, largely in blue-collar occupations, and their dependents. Ringen et al. reported that, when a pilot smoking cessation program involving telephone counseling and pharmacotherapy was offered free of charge to a population of unionized blue-collar workers through their health and welfare fund, 944 of 7400 (13.4%) of smokers insured enrolled in the program within 2 years.⁷

In summary, a small, but convincing, empirically based literature indicates that increasing financial coverage of tobacco cessation treatments through insurance programs would likely lead to increased use of proven smoking cessation treatments. Medicaid and labor-management health and welfare funds are particularly important insurance channels through which to reach low-income and blue-collar workers, respectively. Important areas for future research are identifying effective intervention strategies at individual and organizational levels to: (1) increase demand for and purchase of these benefits among individual- and group-level *insurance purchasers and benefits administrators*, (2) increase demand for these services among *insurance plan participants*, and (3) increase utilization by *plan participants* once benefits are in place.

Promotions

Cessation treatments can be promoted in a variety of channels, including television, radio, and print media; the internet; and community-based settings (e.g., unions, worksites, churches, Women, Infants, and Children [WIC] programs). There is a dearth of studies using randomized controlled trial designs to evaluate the relative effectiveness of different promotion strategies (or promotion vs. no promotion) on the outcome of increasing demand for or use of treatments among low-income and blue-collar smokers. For example, according to a recent review of telephone quitline promotion practices, extant literature has described promotion as an important element of quitline programs, but promotion itself has not yet been studied as an independent variable. 8 A randomized controlled community-based trial of the effectiveness of a media campaign to increase use of the National Cancer Information Service among African Americans reported that use of the service was higher among residents in the intervention vs. control communities. No similar study exists for low socioeconomic (SES) groups. Likewise, promotion of pharmacotherapy treatments has not been well studied. Pharmaceutical firms that sell these medications may have information about the relative effectiveness of various promotion and advertising strategies on increasing sales, but these findings do not appear in the peer-reviewed literature.

A new area of research ought to be established to investigate the effectiveness of various promotion strategies for increasing demand for and use of cessation treatments for the general population of smokers and, most especially, for groups remaining at high risk of smoking, including low-income and blue-collar populations. Research is needed to identify effective messages and channels through which to disseminate those messages in order to achieve maximum impact on increasing demand for and use of proven cessation treatments. Such research will necessarily involve multidisciplinary teams of researchers with expertise in communication science, health education, behavioral science, and tobacco control, undertaking both qualitative and quantitative research using RCTs and quasi-experimental study designs.

- 1. Barbeau EM, Krieger N, Soobader M. Working class matters: Socioeconomic disadvantage, race/ethnicity, gender and smoking in the National Health Interview Survey, 2000. *Am J Public Health*. 2004;94:269–278.
- 2. Curry SJ, Grothaus LC, McAfee T, Pabiniak C. Use and cost effectiveness of smoking-cessation services under four insurance plans in a health maintenance organization. *New Engl J Med.* 1998;339:673–679.
- 3. Schauffler HH, McMenamin S, Olson K, Boyce-Smith G, Rideout JA, Kamil J. Variations in treatment benefits influence smoking cessation: results of a randomised controlled trial. *Tob Control*. 2001;10:175–180.
- 4. Kaper J, Wagena EJ, Willemsen MC, van Schayck CP. Reimbursement for smoking cessation treatment may double the abstinence rate: results of a randomized trial. *Addiction*. 2005;100: 1012–1020.
- 5. Kaiser Family Foundation. *Medicaid Enrollment: Kaiser commission on Medicaid and the uninsured.* Washington, DC: Kaiser Family Foundation; October, 2000.
- 6. Cummings K, Hyland A, Ockene J, Hymowitz N, Manley M. Use of the nicotine skin patch by smokers in 20 communities in the United States, 1992–1993. *Tob Control*. 1997;6:S63–S70.
- 7. Ringen K, Anderson N, McAfee T, Zbikowski SM, Fales D. Smoking cessation in a blue-collar population: results from an evidence-based pilot program. *Am J Ind Med*. 2002;42:367–377.
- 8. Feltracco A, Wilkerson T. *Knowledge synthesis report better practices in the promotion of smokers' helplines*. Ontario, Canada: November, 2004.
- 9. Boyd NR, Sutton C, Orleans CT, et al. Quit Today! A targeted communications campaign to increase use of the Cancer Information Services among African American smokers. *Prev Med.* 1998;27:S50–S60.

Table 1. Age-Adjusted Prevalence of Use of Tobacco Cessation Aids Among U.S. Current Adult Smokers Who Attempted To Quit for 1 Day or Longer by Sociodemographics Characteristics, National Health Interview Survey 2000.

		Use of Cessation Aids		
	Sample N=3,218	%	(s.e.)	Chi-square p-value
Overall prevale	nce	22.5	(0.9)	
Socioeconomic characteristics:				
Age:				0.00001
18–34 years	1,220	15.2	(1.2)	
35–54 years	1428	28.0	(1.4)	
>=55 years	568	26.2	(2.2)	
Gender*				N.S.
Male	1503	21.0	(1.3)	
Female	1715	24.8	(1.3)	
Years of completed education:*				0.0002
Less than a high school degree	722	17.0	(1.9)	
High school degree	926	23.4	(2.0)	
Some college or college graduate	1554	25.3	(1.4)	
Years of completed education and sex*				0.0002
Males	1494	21.0	(1.3)	
Less than a high school degree	335	14.4	(2.4)	
High School degree	437	20.3	(2.7)	
Some college or college graduate	722	24.4	(1.8)	
				0.07
Female	1708	24.8	(1.3)	
Less than a high school degree	387	19.7	(2.7)	
High School degree	489	25.9	(2.6)	
Some college or college graduate	832	26.1	(2.0)	

Table 1. Age-Adjusted Prevalence of Use of Tobacco Cessation Aids Among U.S. Current Adult Smokers Who Attempted To Quit for 1 Day or Longer by Sociodemographics Characteristics, National Health Interview Survey 2000. (continued)

		Us	Use of Cessation Aids		
	Sample N=3,218	%	(s.e.)	Chi-square p-value	
Race/ethnicity and education:					
White NH					
Less than a high school degree	371	22.6	(2.7)		
High school degree	654	26.0	(2.2)		
Some college or college graduate	1144	28.2	(1.6)		
Black NH					
Less than a high school degree	142	9.6	(2.6)		
High school degree	148	15.2	(3.7)		
Some college or college graduate	220	14.8	(2.5)		
Hispanic					
Less than a high school degree	185	4.2	(1.3)		
High school degree	97	3.4	(1.6)		
Some college or college graduate	132	5.5	(2.1)		
Family annual household income:*				0.0001	
\$20,000 or more per year	1045	25.1	(1.3)		
<\$20,000 per year	2051	17.0	(1.4)		
(Missing is less than 4%)					
Race/ethnicity and family household income:					
White NH					
\$20,000 or more per year	1487	27.9	(1.4)		
<\$20,000 per year	611	21.5	(1.9)		
Black NH					
\$20,000 or more per year	271	13.8	(2.1)		
<\$20,000 per year	218	9.6	(2.2)		
Hispanic					
\$20,000 or more per year	226	3.8	(1.3)		
<\$20,000 per year	178	4.8	(1.5)		

^{(%)*=}Age adjusted weighted prevalence

Recommended tobacco cessation therapies, includes counseling therapies alone and/or pharmacological therapies (nicotine replacement therapies, and Zyban/Bupriopion/Wellbrutrin), as recommended by the PHS Clinical Practice Guidelines

Table provided courtesy of Vilma Cokkinides, Ph.D.

Evidence-Based Practice Center Presentation: Increasing Demand for and Use of Effective Tobacco Cessation Treatments Among Individuals

Cathy L. Melvin, Ph.D., M.P.H.

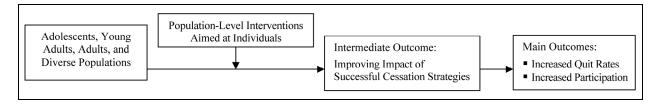
Background

For those individuals who become dependent on tobacco products, eventual disease, disability, and death for themselves and those around them can be avoided if they quit using these products. Users dependent on tobacco find that quitting is very difficult, although most of them would like to do so. Proven individual strategies for helping smokers to quit include counseling and behavioral therapy, and, except when contraindicated, the use of first-line and second-line medications. More often than not, individuals are trying to quit without assistance that can double their chances of success. A

Methods

Using the analytic framework in figure 1, we undertook a systematic review of the evidence for interventions aimed at increasing the number of smokers who attempt to quit and improving the impact of individually oriented treatments developed to help them quit using tobacco products ("key question 2" in the main evidence report).

Figure 1. Increasing Demand for and Use of Cessation Interventions Among Individuals: Analytic Framework



We searched standard electronic databases (MEDLINE®, the Cumulative Index to Nursing and Applied Health [CINAHL], Cochrane Collaboration libraries, Cochrane Clinical Trials Register, Psychological Abstracts, and Sociological Abstracts) between January 1, 1980, and June 10, 2005 (for this question, relevant inclusion dates were January 1, 1999, through June 10, 2005). We used Medical Subject Headings as search terms, when available, or key words when appropriate. We limited our review to (1) human studies conducted in developed countries and published in English; (2) studies with participants ages 13 and older, of both sexes, and diverse racial and ethnic populations; (3) randomized controlled trials (RCTs) with 30 or more individuals; (4) observational studies and other trials with 100 or more individuals; (5) studies with a minimum followup period of 6 months, with or without comparison groups. We excluded editorials, letters, and commentaries; articles that did not report outcomes related to our key questions; and studies that did not provide sufficient information to be abstracted. For earlier

work on these matters, we relied on prior systematic reviews.^{2–8} If studies already in those reviews addressed the same outcomes of interest (i.e., increased quit rates; greater number of smoking cessation participants), we did not re-abstract data. We reviewed and extracted data that met our inclusion criteria from studies published after the existing reviews; we entered the data into evidence tables and summarized them by descriptive methods. From our review of 639 abstracts, 22 studies addressed the demand for and use of effective tobacco cessation treatments; we rated 5 studies as poor quality and do not discuss them further. Seven eligible studies focused on strategies to increase the number of users who attempt to quit; 10 focused on interventions aimed at improving the success rate of quit attempts.

Results: Increasing Demand for Cessation Treatment

Our review of multicomponent strategies to increase the number of users who attempt to quit included telephone counseling as a strategy. Findings from recent reviews show that proactive telephone counseling is effective in increasing tobacco use cessation for adults, especially when combined with other counseling formats. Four studies of fair quality focused on telephone counseling with related print materials. ⁹⁻¹² As a group, these studies yielded insufficient and inconsistent evidence to draw conclusions about the efficacy of telephone counseling. When considered within the context of recent systematic reviews and meta-analyses, two studies ^{11,12} in our review were consistent with that body of evidence; each demonstrated a positive effect of telephone counseling along with relevant print materials on quitting smoking.

The evidence showing that telephone counseling targeting youth and young adults achieved quit rates comparable to those shown for adults is promising, but the small number (two) of fair studies is insufficient to draw conclusions on the effect of telephone counseling for this population. ^{10,11}

We found no studies evaluating multiple counseling formats. One study evaluating counseling enhanced by the provision of information on genetic susceptibility to lung cancer showed a short but un-sustained effect on cessation rates. Results of studies evaluating persistence of effect in the long term were inconsistent. Results of studies evaluating

Results: Improving Success of Cessation Interventions

Our review of strategies to improve the success of quit attempts identified 10 studies evaluating the efficacy of self-help strategies, counseling, and pharmacotherapies, including combination pharmacotherapy, fluoxetine, and bupropion in an indigenous population; all were of fair quality. Our findings for self-help strategies were consistent with those of earlier reviews: evidence of the effectiveness of self-help strategies was insufficient, given the inconsistency of effects and the small number of studies. Our findings for self-help strategies was insufficient, given the inconsistency of effects and the small number of studies.

Our review of interventions using counseling strategies produced mixed results. Two studies showed increased abstinence rates, ^{19,25} but two other studies showed no effect of counseling on cessation rates. ^{16,23} These findings were insufficient on their own to make a recommendation differing from earlier reviews that conclude that even brief individual cessation counseling is efficacious.

With respect to pharmaceutical approaches to increasing quit attempt success, we reviewed two trials of combination pharmacotherapy ^{17,20} that showed an increase in long-term (i.e., 12 months) cessation compared with one pharmacotherapy alone. Although insufficient in number and quality to draw definitive conclusions, these studies shed light on the use of combination therapies and suggest the need for additional research to replicate these findings. This is especially important because participants in these trials reported fewer side effects and withdrawal symptoms, both factors that could increase the likelihood of quitting smoking.

In one trial of fluoxetine (a selective serotonin-reuptake-inhibiting antidepressant) as an adjunct to cognitive behavioral counseling, investigators found that fluoxetine increased the likelihood of abstinence, as compared with placebo, among smokers with minor depression but not among those with little or no depression. Although these results may be promising for medication-compliant smokers with subclinical levels of depression, they need to be replicated in other studies that help us to understand the moderating effect of depression on fluoxetine responsiveness and the characteristics of those smokers most likely to benefit from fluoxetine.

In a study of whether bupropion combined with smoking cessation counseling was effective in treatment of tobacco use in indigenous Maori in New Zealand, the investigators found quit rates similar to those observed in other trials of bupropion²⁴ and reported no data to suggest that the Maori encountered any special problems related to bupropion use.

Conclusions and Needs for Future Research

Very few studies examine the relative population impact of various proven cessation interventions. For example, knowing how proactive telephone counseling support compares with a face-to-face intervention, or whether or not nicotine replacement therapy is offered, would be useful. Differential rates of enrollment and success on a population basis may offset or enhance each other. These research questions are especially important given the move toward increased provider referral to quitline services.

We found no studies comparing the specific aspects of telephone counseling with each other. Issues around the number and timing of calls and the role of feedback to the caller's primary provider have not been studied sufficiently.

We did not identify sufficient studies of the role of mass media in driving people to quitline and other cessation services. Research on specific messages and their effectiveness in reaching and motivating target audiences such as adolescents, young adults, and persons with low income and educational status should improve the impact of such interventions.

We found very few studies examining the effectiveness of multiple intervention formats, of combination pharmacotherapy, or of adjuncts other than pharmacotherapy to individual counseling in increasing the success of smoking cessation interventions. Similarly, very few studies examined differences in either withdrawal symptoms or side effects associated with continuation or success of pharmacotherapy. Persistence of effect was reported by only two studies; larger, prospective trials are likely needed to increase the evidence base for this issue. Finally, very few studies focused on ways to reach or treat special populations such as adolescents and young adults.

- 1. Centers for Disease Control and Prevention (CDC). Tobacco use, access, and exposure to tobacco in media among middle and high school students—United States, 2004. *Morb Mortal Wkly Rep.* 2005;54(12):297–301.
- 2. Fiore MC, Bailey WC, Cohen SJ, et al. *Treating Tobacco Use and Dependence, Clinical Practice Guideline*. Rockville, Md: U.S. Department of Health and Human Services, Public Health Service; 2000.
- 3. U.S. Department of Health and Human Services. Reducing Tobacco Use: A Report of the Surgeon General. Atlanta, Ga: US Department of Health and Human Services, Centers for Disease Control and Prevention, Office on Smoking and Health; 2000.
- 4. Stead LF, Lancaster T, Perera R. Telephone counselling for smoking cessation. *Cochrane Database Syst Rev.* 2003;(1):CD002850.
- 5. Task Force on Community Preventive Services. *The Guide to Community Preventive Services: What Works to Promote Health?* New York: Oxford University Press; 2005.
- 6. Lancaster T, Stead LF. Individual behavioural counselling for smoking cessation. *Cochrane Database Syst Rev.* 2005;(2):CD001292.
- 7. Lancaster T, Stead L. Self-help interventions for smoking cessation. *Cochrane Database Syst Rev.* 2005;(3):CD001118.
- 8. Stead LF, Lancaster T. Group behaviour therapy programmes for smoking cessation. *Cochrane Database Syst Rev.* 2005;(2):CD001007.
- 9. Bauman KE, Ennett ST, Foshee VA, et al. Influence of a family-directed program on adolescent cigarette and alcohol cessation. *Prev Sci.* 2000;1(4):227–237.
- 10. Lipkus IM, McBride CM, Pollak KI, et al. A randomized trial comparing the effects of self-help materials and proactive telephone counseling on teen smoking cessation. *Health Psychol.* 2004;23(4):397–406.
- 11. Rabius V, McAlister AL, Geiger A, et al. Telephone counseling increases cessation rates among young adult smokers. *Health Psychol.* 2004; 23(5):539–541.
- 12. Smith PM, Cameron R, McDonald PW, et al. Telephone counseling for population-based smoking cessation. *Am J Health Behav*. 2004;28(3):231–241.
- 13. McBride CM, Bepler G, Lipkus IM, et al. Incorporating genetic susceptibility feedback into a smoking cessation program for African-American smokers with low income. *Cancer Epidemiol Biomarkers Prev.* 2002;11(6):521–528.
- 14. Etter JF, Perneger TV. Post-intervention effect of a computer tailored smoking cessation programme. *J Epidemiol Community Health*. 2004;58(10):849–851.

- 15. Murray RP, Connett JE, Rand CS, et al. Persistence of the effect of the Lung Health Study (LHS) smoking intervention over eleven years. *Prev Med.* 2002;35(4):314–319.
- 16. Lancaster T, Dobbie W, Vos K, et al. Randomized trial of nurse-assisted strategies for smoking cessation in primary care. *Br J Gen Pract.* 1999;49(440):191–194.
- 17. Jorenby DE, Leischow SJ, Nides MA, et al. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *N Engl J Med.* 1999;340(9):685–691.
- 18. Hitsman B, Pingitore R, Spring B, et al. Antidepressant pharmacotherapy helps some cigarette smokers more than others. *J Consult Clin Psychol*. 1999;67(4):547–554.
- 19. Canga N, De Irala J, Vara E, et al. Intervention study for smoking cessation in diabetic patients: a randomized controlled trial in both clinical and primary care settings. *Diabetes Care*. 2000;23(10):1455–1460.
- 20. Bohadana A, Nilsson F, Rasmussen T, et al. Nicotine inhaler and nicotine patch as a combination therapy for smoking cessation: a randomized, double-blind, placebo-controlled trial. *Arch Intern Med.* 2000;160(20):3128–3134.
- 21. Jones C, Griffiths RD, Skirrow P, et al. Smoking cessation through comprehensive critical care. *Intensive Care Med.* 2001;27(9):1547–1549.
- 22. Clark MM, Cox LS, Jett JR, et al. Effectiveness of smoking cessation self-help materials in a lung cancer screening population. *Lung Cancer*. 2004;44(1):13–21.
- 23. Peterson W. A nurse managed smoking cessation and relapse prevention programme did not reduce smoking rates at 12 months beyond rates achieved by usual care in women with cardiovascular disease. *Evidence Based Nursing*. 2004;7(3):83.
- 24. Holt S, Timu-Parata C, Ryder-Lewis S, et al. Efficacy of bupropion in the indigenous Maori population in New Zealand. *Thorax*. 2005;60(2):120–123.
- 25. Hennrikus DJ, Lando HA, McCarty MC, et al. The TEAM project: the effectiveness of smoking cessation intervention with hospital patients. *Prev Med.* 2005;40(3):249–258.

Health Systems Changes

Michael C. Fiore, M.D., M.P.H.

In 1996, the Agency for Health Care Policy Research (AHCPR, now the Agency for Healthcare Research and Quality [AHRQ]) released the first Federal clinical practice guideline for smoking cessation. Both the 1996 AHCPR guideline and the 2000 Public Health Service update were innovative in that they identified six evidence-based strategies for healthcare systems to facilitate the institutionalization of tobacco dependence treatment. These strategies are:

- Implementing a tobacco-user identification system in every clinic
- Providing education, resources, and feedback to promote provider intervention
- Dedicating staff to provide tobacco dependence treatment and assessing the delivery of this treatment in staff performance evaluations
- Promoting hospital policies that support and provide tobacco dependence services
- Including all tobacco dependence treatments (both counseling and pharmacotherapy) identified as effective as paid or covered services for all subscribers or members of health insurance packages
- Reimbursing clinicians and specialists for delivery of effective tobacco dependence treatments and including these interventions among the defined duties of clinicians²

Systems-level strategies represented a new way of thinking about treating tobacco dependence. Typically, interventions have targeted either the smoker or the clinician. In contrast, systems strategies are designed to ensure that tobacco use, the leading cause of illness and death, is addressed in a more systematic way. The goal of these strategies is straightforward—to ensure that tobacco use is systematically assessed and treated at every clinical encounter. Since these recommendations were first released in 1996, new research has expanded the scientific basis for systems changes, including reviews conducted by the U.S. Public Health Service Task Force on Community Preventive Services as well as the Cochrane Collaboration. Moreover, an evaluation conducted by the Cancer Research Network found that the adoption of health plan policies can result in the implementation of systems-level changes and increased delivery of these services to patients. Examples of progress in some of these areas and future opportunities for research and implementation are summarized below.

Implementation of Tobacco-User Identification Systems in the Clinic Setting

There is significant evidence that implementing a clinic-based tobacco-user identification system increases the rate of smoker identification and facilitates provision of advice to quit and, possibly, assistance in quitting.^{8–11} Recent research further describes the positive impact of including tobacco use as a vital sign on rates of asking about smoking status.^{12,13} Findings from a

periodic survey of health plans conducted by America's Health Insurance Plans (AHIP) show that the percentage of health plans that were able to identify any individual members who smoke increased from 14.9% in 1997 to 71.7% in 2002 (p<0.001).¹⁴

Improvements in rates of clinician intervention also are seen in national data sets. The 2004 Health Employer Data and Information Set (HEDIS) data collected by the National Committee for Quality Assurance (NCQA) documented that 69.6% of commercial enrollees, 66.9% of Medicaid enrollees, and 64.7% of Medicare enrollees reported receiving advice to quit smoking, compared to 66.3% of commercial enrollees, 64.2% of Medicaid enrollees, and 59.3% of Medicare enrollees in 2000. 15

Providing Education, Resources, and Feedback to Clinicians

There is significant evidence that multicomponent interventions that incorporate both provider education and reminder systems facilitate delivery of evidence-based tobacco dependence treatments.³ A review on audit and feedback in clinical practice published by the Cochrane Collaboration found that these strategies can improve provider performance, but improvements are small to modest. The effects of audit and feedback were likely to be larger when initial performance was low.⁴ New research has helped expand the evidence base surrounding performance feedback, demonstrating both that providers will accept such feedback and that feedback can increase the performance of targeted behaviors.^{16–18}

Hospital Policies That Support Inpatient Cessation Services

A 2003 Cochrane review evaluated the effectiveness of smoking cessation interventions for hospitalized patients, finding that high-intensity behavioral interventions that included at least 1 month of followup were effective in increasing the delivery of smoking cessation treatments to inpatients. In 1992, the Joint Commission for Accreditation of Healthcare Organizations (JCAHO) issued a standard requiring that all accredited hospitals have a policy prohibiting smoking in the hospital; by 1994, more than 96% of hospitals surveyed complied with the JCAHO standard, and 41.4% had enacted policies that were stricter than the JCAHO standard. In 2002, for the first time, JCAHO added performance measures for adult smoking cessation advice and counseling for patients presenting with acute myocardial infarction (AMI), community-acquired pneumonia (CAP), and heart failure (HF) to its core performance measure set. From July 1, 2004, to June 30, 2005, national rates for providing advice or counseling were 89% (AMI), 78% (HF), and 75% (CAP).

Include Efficacious Tobacco Dependence Treatments in Insurance Packages

Over the last 15 years, there has been a substantial increase in the coverage of tobacco dependence treatments by publicly funded insurance programs. In 2005, Medicare began covering cessation counseling for recipients diagnosed with a tobacco-related illness, and in 2006, prescription cessation medications were covered through the Medicare Prescription Drug Act (Medicare Part D). A growing number of State Medicaid programs provide some coverage for tobacco cessation, with 37 States covering at least one evidence-based treatment in 2003. In 2006, the Department of Veterans Affairs (VA) eliminated copayments for cessation counseling. Increases in coverage are also seen in the private market. A periodic survey conducted by AHIP

found that plans reporting full coverage for any behavioral therapy or pharmacotherapy increased from 75% in 1997 to 98% in 2002. 14 A Cochrane review evaluated healthcare financing systems for increasing the use of tobacco dependence treatment. The authors found that there was some evidence that offering full coverage of tobacco dependence treatments can increase self-reported prolonged abstinence rates at relatively low costs when compared with a partial benefit or no benefit; however, methodological limitations require that these findings be interpreted with caution. 5

Lessons Learned and Future Directions

Systems changes have the potential to increase rates of tobacco-user identification and intervention, and subsequently to improve the health of patients by facilitating quit attempts. While much progress has been made, further work is needed to ensure that all tobacco users are identified and receive evidence-based treatment for tobacco dependence each time they present to the healthcare system. This goal is particularly salient given that 70% of smokers visit a primary care physician each year. A number of researchers have identified healthcare systems questions for further exploration, including: 3,22

- Systems approaches such as tobacco-user identification are successful in improving documentation of patients' tobacco use, but do not necessarily spur further intervention. What strategies can be implemented and evaluated to foster provision of advice and, particularly, the delivery of evidence-based treatment?
- What are the most and least effective combinations of services in multicomponent interventions?
- How effective are the HEDIS and JCAHO measures in improving patient receipt of advice to quit and patient tobacco use cessation? What would be the impact of a JCAHO requirement mandating that tobacco use be addressed for all hospital admissions?
- How does the base rate of tobacco use in a managed care organization or insurance plan affect implementation of systems-level changes and outcomes?
- How can mixes of tobacco use cessation interventions be most effectively integrated in managed care organizations?
- What are the costs, cost-benefit, and return on investment of systems-level interventions?
- How can technologies such as registries and electronic medical records be used to facilitate delivery of evidence-based tobacco dependence treatment?

- 1. Fiore MC, Bailey WC, Cohen SJ. *Smoking cessation: Clinical practice guideline No. 18*. Rockville, Md: U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; 1996.
- 2. Fiore MC, Bailey WC, Cohen SJ. *Treating tobacco use and dependence: Clinical Practice Guideline*. Rockville, Md: U.S. Department of Health and Human Services, U.S. Public Health Service; 2000.
- 3. Hopkins DP, Briss PA, Ricard CJ, et al. Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. *Am J Prev Med*. 2001;20(2 Suppl):16–66.
- 4. Jamtvedt G, Young JM, Kristoffersen DT, Thomson O'Brien MA, Oxman AD. Audit and feedback: Effects on professional practice and health care outcomes. *Cochrane Database Syst Rev.* 2003(3):CD000259.
- Kaper J, Wagena EJ, Severens JL, Van Schayck CP. Healthcare financing systems for increasing the use of tobacco dependence treatment. *Cochrane Database Syst Rev.* 2005(1):CD004305.
- 6. Rigotti NA, Munafo MR, Murphy MF, Stead LF. Interventions for smoking cessation in hospitalized patients. *Cochrane Database of Systematic Reviews*. 2003;1(CD001837):1–21.
- 7. Stevens VJ, Solberg LI, Quinn VP, et al. Relationship between tobacco control policies and the delivery of smoking cessation services in nonprofit HMOs. *J Natl Cancer Inst Monogr.* 2005(35):75–80.
- 8. Fiore MC, Jorenby DE, Schensky AE, Smith SS, Bauer RR, Baker TB. Smoking status as the new vital sign: Effect on assessment and intervention in patients who smoke. *Mayo Clinic Proceedings*. Mar 1995;70(3):209–213.
- 9. Chang HC, Zimmerman LH, Beck JM. Impact of chart reminders on smoking cessation practices of pulmonary physicians. *Am J Respir Crit Care Med.* Sep 1995;152(3):984–987.
- 10. Robinson MD, Laurent SL, Little JM, Jr. Including smoking status as a new vital sign: It works! *J Fam Pract*. Jun 1995;40(6):556–561.
- 11. Ahluwalia JS, Gibson CA, Kenney RE, Wallace DD, Resnicow K. Smoking status as a vital sign. *J Gen Intern Med.* Jul 1999;14(7):402–408.
- 12. Piper ME, Fiore MC, Smith SS, et al. Use of the vital sign stamp as a systematic screening tool to promote smoking cessation. *Mayo Clin Proc.* 2003;78(6):716–722.
- 13. Boyle R, Solberg LI. Is making smoking status a vital sign sufficient to increase cessation support actions in clinical practice? *Ann Fam Med.* Jan-Feb 2004;2(1):22–25.

- 14. McPhillips-Tangum C, Bocchino C, Carreon R, Erceg C, Rehm B. Addressing tobacco in managed care: results of the 2002 survey. *Prev Chronic Dis.* 2004;1(4):A04.
- 15. National Committee for Quality Assurance. The state of health care quality 2005. Available at: http://www.ncqa.org/Docs/SOHCQ 2005.pdf.
- 16. Swartz SH, Cowan TM, DePue J, Goldstein MG. Academic profiling of tobacco-related performance measures in primary care. *Nicotine Tob Res.* 2002;4 Suppl 1:S38–44.
- 17. McAfee T, Grossman R, Dacey S, McClure J. Capturing tobacco status using an automated billing system: Steps toward a tobacco registry. *Nicotine and Tobacco Research*. 2002;4 Suppl 1:S31–S37.
- 18. Bentz CJ, Bayley KB, Bonin KE, et al. Provider feedback to improve 5 A's tobacco cessation in primary care: A cluster randomized clinical trial. *Nicotine & Tobacco Research*. In press.
- 19. Longo DR, Feldman MM, Kruse RL, Brownson RC, Petroski GF, Hewett JE. Implementing smoking bans in American hospitals: Results of a national survey. *Tobacco Control*. 1998 1998;7:47–55.
- 20. Joint Commission on Accreditation of Healthcare Organizations. *University of Wisconsin Hospitals and Clinics Authority Quality Report*. Available at: http://www.qualitycheck.org.
- 21. Halpin HA, Bellows NM, McMenamin SB. Medicaid coverage for tobacco-dependence treatments. *Health Aff (Millwood)*. Mar–Apr 2006;25(2):550–556.
- 22. Taylor CB, Curry SJ. Implementation of evidence-based tobacco use cessation guidelines in managed care organizations. *Annals of Behavioral Medicine*. 2004;27(1):13–21.

Addressing Tobacco-Related Disease Health Disparities in the Delivery of Community-Based Interventions

Phillip Gardiner, Ph.D.

To address the question of tobacco-related disease health disparities and which community-based interventions have worked is no small task. Moreover, while tobacco prevention and cessation efforts are certainly necessary, ultimately they may be in and of themselves insufficient to fully address the chronic concentration of morbidity and mortality of tobacco-related disease in poor communities, special populations, and communities of color.

It should go without saying that the existing community interventions must not only collect data, but also bolster a community's capacity to tackle tobacco-related disease health disparities. Community-based researchers cannot just show up and "intervene" in communities; rather, researchers have to become part and parcel of the community and, together with community residents, transform the community's relationship with tobacco products and mitigate their impact. Toward this end, what we know about community-based interventions is addressed below, and one road forward that could get at the root of the disparities themselves is discussed.

What We Know

Two excellent review articles have assessed the effectiveness of tobacco control and cessation efforts in communities of color. Lawrence et al. discovered that interventions in communities of color were more or less equally divided between those taking place in clinical settings, on the one hand, and those located at specific locations (e.g. school) and/or community-wide interventions, on the other hand. Of the 36 interventions reviewed by Lawrence and her team, 14 (39%) reported significant reductions in the use of tobacco products. Additionally, she found that quasi-experimental studies were proportionately more successful than the randomized experimental studies. Lawrence also found that media and culturally tailored materials were used in the majority of interventions reviewed.

Okuyemi and colleagues, in their review of 43 studies that reported on smoking cessation in racial and ethnic populations, found a clear dose-response relationship; intensive interventions produced higher success rates and were more cost effective.² This team found that intensive interventions may include pharmacotherapy multiple individual or group sessions, telephone counseling, self-help materials, and followup assessments. Interestingly, both the Lawrence and the Okuyemi reviews showed that the majority of the studies have focused on African Americans and smoking, and Lawrence points out that it would be incorrect to generalize findings from experiences in this population linearly to other communities of color. While it is true that most studies dealing health disparities have focused on African Americans, it is also unfortunately true that African Americans suffer disproportionately from tobacco related diseases, including the highest incidence and death from lung, oral, pancreas esophagus and larynx cancers.³

There are, and continue to be, some successful community-based interventions to reduce smoking among communities of color. In the African American community, Ahluwalia et al.

have shown that community-based cessation programs using nicotine replacement therapy (NRT) or bupropion can decrease smoking rates. As Ricardo Munoz, working in the Latino/Hispanic community through a mood management program, cut smoking 25.4% for intervention subjects compared to 9.2% of controls. Similarly, Woodruff and colleagues were able to successfully use a promotores-modeled intervention where intervention participants abstinence rate was 20.5% compared to 8.7% for controls. Jenkins et al., implementing a community-based intervention, were successful in lowering the odds of being a smoker among Vietnamese Americans living San Francisco.

Not only are there successful community-based interventions taking place around the country to thwart the impact of tobacco use among disparate populations, but also the United States Federal Government, through the agency of the National Cancer Institute, has issued a lengthy list of research recommendations, including increased funding of community-based research, training and mentoring of researchers representing underserved racial and ethnic groups, and research on the impact of the tobacco industry's practices and policies in oppressed communities. Still, in all, model replicable interventions to tackle tobacco health impacts in separate communities are few and far between.

Undeniably, the "California experience" has conclusively demonstrated that a focus on changing social norms, prioritizing underserved communities, and establishing a statewide infrastructure that connects grassroots local organizations are key ingredients to reduce tobacco use. ^{10,11} Moreover, the California experiment has also shown that a focus on indoor and outdoor smoking policies, regular and systematic exposure of the deceitful and illegal practices of the tobacco industry, and an aggressive media campaign can have a tremendous impact. The fine work of the Tobacco Control Section of California's Department of Health Services, along with the efforts of both the Tobacco Use Prevention and Education program of the California Department of Education and the Tobacco Related Disease Research Program of the University of California Office of the President, has brought this State's smoking rates down to 15% of the adult population, second only to Utah. ^{12,13} It also must be noted that another societal-wide intervention has been shown to be effective in increasing the accessibility of cessation services and education to disparate groups—the quitlines that have proliferated throughout the country based on the California experience. ¹⁴

Yet, for all the advancements made by California, other States, and numerous successful community-level and State-level interventions, tobacco-related disease health disparities persist and endure. African Americans continue to die disproportionately from lung cancer, coronary heart disease, cerebrovascular disease, and oral cancer, to mention a few. For American Indians and Alaska Natives, smoking rates stubbornly remain around 40% of the adult population. Vietnamese male smoking rates continue above 50%, and, increasingly, Asian and Pacific Islander women coming to the United States have begun the long deadly journey to adopt cigarette smoking, although slowly.

It goes without saying that if it took years, nay decades, to produce and reproduce tobacco-related disease disparities, then it will take decades to overcome them. However, it will be of the utmost importance to tackle the problem correctly; band-aids and half steps will only perpetuate the problem. There must be a societal-level response to this issue; nothing less will be effective.

An Alternative Framework

To effectively and systematically identify, reduce, and ultimately eradicate tobacco-related disease health disparities, community capacities will have to be substantially increased; the participatory nature of research, regardless of the type of community, must become the norm; and environmental disparities including racialized marketing on the part of the tobacco industry stopped.

First and foremost, communities must have increased capacity to tackle runaway tobacco-related disease disparities. Robert Robinson has argued persuasively over the years that communities of color must have a mass infusion of resources if tobacco-related health disparities are going to be effectively addressed. It is no secret that large parts of the African American, Latino, American Indian, and immigrant Asian and Pacific Islander populations are mired in poverty. Communities lack cessation services, clinics, hospitals, pharmacotherapeutics, and general medical resources of all kinds. Even more broadly, communities lack health insurance and employment opportunities that ensure health coverage. True; there are, and should continue to be, successful prevention and cessation interventions taking place in the context of poor communities. It is also true that many communities of color have not and are not idly sitting by waiting for salvation; some have taken steps to right their own community's ship of health in a clearly unrelenting storm. However, unless and until these communities are lifted up and brought into the mainstream, the conditions that spawn and promote tobacco-related disease health disparities will persist.

Secondly, those interventions that, of necessity, take place in poor disparate communities must be participatory in nature. Gone are the days (or I should say the days *should* be gone) when researchers and investigators showed up in a community, shook a few hands, collected a lot of data, published their findings in narrowly read scientific journals and left. The involvement of the community in all aspects of the research is a must. From the identification of issues; the conceptualization of the research questions and hypothesis; writing and submitting grant applications; hiring of staff and the implementation of the intervention; collection of data and the analysis of the results; and the dissemination of findings across a broad array of venues and media—community members and researchers must jointly participate in the process. Moreover, community-based tobacco cessation and control interventions must take place in the actual context of the disparate conditions faced by many communities. Severe poverty, poor education, lack of fresh foods, nonexistent open areas, and deteriorated and segregated housing all are part of the milieu in which research must take. Simply said, larger sociostructural issues cannot be avoided, nay must be addressed, when tackling the question of tobacco-related disease health disparities in many communities of color.

Thirdly, major tobacco marketing and advertising restrictions must be implemented in communities that suffer disproportionately from tobacco-related diseases. If, for example, African Americans die disproportionately of lung cancer, then it behooves society to restrict as much as possible the promotion of tobacco products in that community. Tobacco company advertising in this context is like throwing gasoline on a raging fire. The tobacco industry's forays into special populations in the guise of market segmentation advertising must be severely curtailed. In an ideal world, each human being should be able to make rational decisions about what they consume. However, in the real world, communities of color are bombarded with

messages about the sexiness and/or manliness of tobacco use, and, coupled with the extraordinarily addictive character of cigarettes, it is nothing short of a recipe for disaster.

Some may feel that the some of the suggestions made above fall outside the realm or reach of community-level interventions to stem the tide of tobacco use; in some respects, they do. At the same time, those working in poor and marginalized communities must not only approach community residents about "their use of tobacco," but conversely must struggle to understand the conditions and issues that contextualize the use of tobacco products. Without doing this, any and all tobacco cessation projects will have limited success and be short lived at best; true community-based participatory research requires a long-term, societal-wide commitment.

References

- 1. Lawrence D, Graber JE, Mills SL, Meissner HI, Warnecke R. Smoking cessation intervention in U.S. racial/ethnic minority populations: An assessment of the literature. *Prev Med.* 2003 Feb;36(2):204–216.
- 2. Okuyemi KS, Sanderson-Cox L, Choi WS, Ahluwalia JS. Smoking cessation in U.S. ethnic minority populations. In Isaacs S, ed. *VA in the Vanguard: Building on Success in Smoking Cessation. Proceedings of a Conference Held September 21, 2004 in San Francisco, California.* Washington, DC: VA Press; 1995:103–133.
- 3. Edwards BK, Brown ML, Wingo PA, et al. Annual report to the Nation on the status of cancer, 1975-2002, Featuring Population-Based Trends in Cancer Treatment. *Journal of the National Cancer Institute*. 2005;97(19):1407–1427.
- 4. Ahluwalia JS, Richter KP, Mayo MS, Resnicow K. Quit for Life: Randomized trial of culturally sensitive materials for smoking cessation in African Americans. *JGIM*. 1999:14(2):6.
- 5. Ahluwalia JS, Harris KJ, Catley D, Okuyemi KS, Mayo MS. Sustained-release bupropion for smoking cessation in African Americans: a randomized controlled trail. *JAMA*. 2002;288(4):468–474.
- 6. Munoz RF, Marin BV, Posner SF, Perez-Stable EJ. Mood management mail increases abstinence rates for Spanish-speaking Latino smokers. *Am J Community Psychol*. 1997;25(3):325–343.
- 7. Woodruff SI, Talavera GA, Elder JP. Evaluation of a culturally appropriate smoking cessation intervention for Latinos. *Tob Control*. 2002;11(4):361–367.
- 8. Jenkins CN, McPhee SJ, Le A, Pham GQ, Ha NT, Stewart S. The effectiveness of a medialed intervention to reduce smoking among Vietnamese-American men. *Am J Public Health*. 1997;87(6):1031–1034.

- 9. King G, Fagan P, Lawrence D, et al. *Eliminating Tobacco-Related Health Disparities:* Summary Report of the National Conference on Tobacco and Health Disparities. Washington, DC: National Cancer Institute; 2005. NIH publication No. 05–5283.
- 10. California Department of Health Services/Tobacco Control Section. *A Model for Change: The California Experience in Tobacco Control*. Sacramento: California Department of Health Services/Tobacco Control Section; 1998. Available at: http://www.dhs.ca.gov/tobacco/documents/pubs/modelforchange.pdf.
- 11. California Department of Health Services/Tobacco Control Section. *California Tobacco Control Update 2004*. Sacramento: California Department of Health Services/Tobacco Control Section; 2004. Available at: http://www.dhs.ca.gov/tobacco/documents/pubs/2004TCSupdate.pdf.
- 12. Tobacco Education Research Oversight Committee (TEROC). *Toward a Tobacco-Free California: The Myth of Victory. Tobacco Education Research Oversight Committee Master Plan, 2003–2005.* Sacramento: Author; 2003. Available at: http://www.dhs.ca.gov/tobacco/documents/pubs/TobaccoMasterPlan2003.pdf.
- 13. TEROC. Confronting a Relentless Adversary; A Plan For Success. Tobacco Education Research Oversight Committee Master Plan, 2006–2008. Sacramento: Author; 2006. Available at: http://www.dhs.ca.gov/tobacco/documents/pubs/MasterPlan05.pdf.
- 14. Zhu SH. African Americans and the Quitline: Differential quitline sage, satisfaction and quit rates among minorities. Workshop presentation at: National Conference on Tobacco or Health; May 2005; San Francisco.
- 15. American Cancer Society. *Cancer Facts and Figures 2005–2006.* Available at: http://www.cancer.org/docroot/stt/stt_0.asp.
- 16. U.S. Department of Health and Human Services. Tobacco Use Among U.S. Racial/Ethnic Minority Groups—African Americans, American Indians and Alaska 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; 1998:140–141.
- 17. Robinson RG. Community development model for public health applications: Overview of a model to eliminate population disparities. *Health Promot Pract.* 2005;6(3):338–346.
- 18. Israel BA, Shulz AJ, Parker EA, Becker AB. Review of community-based research: Assessing partnership approaches to improve public health. *Annu Rev Public Health*. 1998;19:173–202.

QUITLINES: Public-Private Partnerships for Tobacco Control Tim McAfee, M.D., M.P.H.

Background

Quitlines (QLs) provide a broad range of cessation support services primarily via the telephone. Services range from a single reactive coaching session provided at the time a caller reaches the QL to multiple proactive counseling calls originating from the service provider over time. Most counseling is provided by paraprofessionals following a semi-structured protocol blending elements of several counseling theories (Stages of Change, Motivational Interviewing, Cognitive-Behavioral Therapy) with practical evidence-based advice and encouragement. Increasingly, cessation medication is being integrated into service provision, ^{1,2} ranging from screening and decision support to fulfillment via direct mail order or pharmacy vouchers. A strong evidence base supports QL efficacy and effectiveness. A 2004 Cochrane Review found a 1.56 odds ratio (OR), overlapping with the nicotine replacement therapy (NRT) OR confidence interval.

Table 1. Efficacy of NRT and Telephone Counseling (TC)—Cochrane Reviews

	Odds ratio (95% CI*)	Average sample size/trial		
NRT (n=98)	1.74 (1.64, 1.86)	385		
TC (n=13)	1.56 (1.38, 1.77)	1,100		

^{*}Confidence Interval

QLs often provide brief coaching to proxy callers such as friends and relatives, as well as healthcare providers. They also provide cessation materials and referral to local resources. Less research has been done on the effectiveness of these functions.

Significance

QLs help increase the reach of evidence-based services by increasing convenience and anonymity and by providing multilingual services. They improve linkages between pharmacotherapy and behavioral and adherence support. They enhance population quit rates in clinical practices (e.g. from 4.1% to 13%), worksites, and geographic regions, both by direct effects for those calling as well as by secondary effects such as inspiring quit attempts in noncallers exposed to promotion and encouraging legislators to continue funding multicomponent tobacco control programs. Some populations underrepresented in cessation treatment are overrepresented in State QL calls, such as the uninsured and African Americans.

How Delivered

There is wide variation in who QLs serve, and how they are delivered and financed. QLs have been set up to deliver services in municipalities, counties, and States and at the national level. They also have been set up to provide services to health plans, employers, and unions. QLs

are delivered by a wide variety of service providers, including academic and healthcare institutions, governmental and philanthropic organizations, and private companies. Financing for the services is quite varied, including: State funding from tobacco taxes, Master Settlement, and general funds; health plan benefit coverage; employer coverage as a "carve-out;" Federal funding to support State services; and philanthropic support underwriting indirect service costs, serving special populations and promotion. Based on evidence that end-user payment markedly decreases use, ¹¹ very few QLs charge users.

There are increasingly sophisticated public-private partnerships extending the depth and breadth of services offered. Integration occurs at the State level, with another layer at the national level. Currently, 46 States have operational QLs, with the remainder expected to be operational in 2006. However, some of these QLs are supported completely by limited Centers for Disease Control and Prevention (CDC) funding. The National Cancer Institute (NCI) and CDC have collaborated with States to create a national network of QLs, all with a single number (1-800-QUITNOW), available as a portal that routes calls back to their respective State QL. Some States are further collaborating with health plans and businesses that provide and/or fund indepth telephonic coaching services and full pharmacotherapy. In these States, callers are triaged by the State-level QL with warm transfer to the service provider for the health plan or employer. The State line conserves limited resources for the provision of full proactive service and pharmacotherapy for the uninsured. States that do not have this form of relationship frequently titrate the amount of promotion done to ensure they have sufficient funds to provide service to callers.

These ambitious, complex partnerships have been fostered by the creation of a North American Quitline Consortium¹² which includes Federal agencies such as NCI and CDC, State health departments and State-level foundations, service providers, and researchers. Limitations have included heterogeneity of funding at the State level, with some States providing limited services (i.e. a single call) with minimal promotion, while some provide robust promotion and services (either through direct provision such as Maine, or via public-private partnerships). Call rates range from less than 1% of smokers per year⁷ up to over 8%¹³ at the State level. Some employers have reached into the 20% level, when combined with incentives.

Another limitation of QLs is lack of awareness by smokers of their existence and effectiveness. ¹⁴ In 2006, the American Legacy Foundation will run several pilots of an ambitious mass media campaign that will include promotion of the 1–800–QUITNOW number. If successful, this will be followed by a national media campaign.

Challenges for the Future

The National Action Plan¹⁵ anticipates that with unfettered access to counseling and pharmacotherapy coupled with robust promotion, 10% of smokers per year would use services with a 20% quit rate. This is a 5- to 10-fold increase over current use rates. From experience, this number appears achievable with improved funding and infrastructure development.

There are promising innovations that could increase reach, effectiveness, and efficiency even further. These need additional development and research:

- 1. Using chronic care/disease management approaches with QLs:
 - Proactive outreach to high-risk populations where smoking status may be available. Initial results indicate >50% acceptance rates
 - Long-term follow-up and open-ended treatment, e.g. recurrent status checks with offers of re-enrollment for those who have relapsed 16
- 2. Integration of phone services with other remote communication vehicles:
 - Web-based tailored content and social interaction via chat and discussion functions, with iterative quit plan development tools
 - Text messaging, cell phones, e-mail
- 3. Further integration and innovation of pharmacotherapy and QLs:
 - Further experiments in promotion effects of brief courses of NRT
 - Tighter relationships with healthcare providers for prescription generation
 - Interrelationships between medication and coaching
 - Testing of safety, efficacy, and practicality of aggressive med approaches, including combination and higher-dose therapy, initiation while smoking, new medications, "test kits," and aggressive medication management
- 4. Impact of "Ask-Advise-Refer" model with QLs used for referral on clinic system adoption versus traditional "5-A" model 17
- 5. Developing and testing improved and new behavioral interventions:
 - Device-supported fading
 - Different theoretical approaches: CBT, MI, "3 Ts" 18
 - Increasing call frequency or call time
 - Relapse-sensitive vs. recycle-sensitive timing of calls
- 6. Incentive programs such as "quit & win" and decreased health insurance premiums
- 7. Cost-effectiveness of different promotional & service approaches:
 - Quantify quit attempt increases from mass media vs. healthcare promotion

- Cost Effectiveness Analysis and Return on Investment for likely high-yield populations such as Chronic Obstructive Pulmonary Disease and obstetrics
- 8. Development and testing of proxy interventions. ¹⁹ Can ex-smokers and nonsmokers improve basic support skills and serve as more competent resources to motivate and then support smokers attempting to quit?
- 9. Experiments in coordination of public-private benefits to increase reach:
 - Risk-based sharing of financing for service and promotion
 - Aggressive government-funded service—how does this impact health plan/employer financing and delivery?
- 10. Sub-population tailoring of promotion and service

Finally, QLs are creating a remarkable infrastructure for theoretical and applied research in numerous disciplines. They are collecting uniform minimum datasets on hundreds of thousands of tobacco users attempting to quit each year. There is wide heterogeneity in recruitment and service strategies from State to State and institution to institution, providing numerous natural experiments. Because of the high volume of participants and computerized coaching support, QLs also provide opportunities for "easy" large-scale social science, health services, pharmacologic effectiveness, and other randomized trials to test theoretical models as well as to conduct practical clinical trials relating to cost effectiveness and equivalency of different approaches.

References

- 1. Cummings KM, Fix B, Celestino P, Carlin-Menter S, O'Connor R, Hyland A. Reach, efficacy and cost-effectiveness of free nicotine medication giveaway programs. *J Public Health Management Practice*. 2006;12(1):37–43.
- 2. An L, Schillo BA, Kavanaugh A, et al. Access to nicotine replacement therapy as part of a statewide tobacco telephone helpline. *American Journal Health Promotion*. 2006;20(4):267–271.
- 3. Swan GE, McAfee T, Jack LM, et al. Effectiveness of buproprion SR for smoking cessation in health care setting. *Arch Intern Med.* 2003 Oct 27;163(19):2337–44.
- 4. Zhu SH, Anderson CM, Johnson CE, Tedeschi G, Roeseler A. A centralized telephone service for tobacco cessation: the California experience. *Tob Control.* Jun 2000;9:48–55.
- 5. Stead LF, Lancaster T, Perera R. Telephone counseling for smoking cessation. *The Cochrane Database of Systematic Reviews*. 2003;Issue 1. Art. No.: CD002850. DOI: 10.1002/14651858.CD002850.
- 6. Silagy C, Mant D, Fowler G, Lancaster T. Nicotine replacement therapy for smoking cessation. *Cochrane Database Syst Rev.* 2000(20):CD000146.

- 7. Ossip-Klein D, McIntosh S. Quitlines in North American: evidence base and applications. *Am J Med Sci.* 2003;326:201–205.
- 8. An L, Zhu SH, Nelson DB, et al. Benefits of telephone care over primary care for smoking cessation. *Arch Intern Med.* 2006;166:536–542.
- 9. Ossip-Klein DJ, et al. Effects of a smoker's hotline: results of a 10-county self-help trial. *J Consult Clin Psychol*. 1991 Apr;59(2):325–32.
- 10. El-Bastawissi AY, McAfee T, Zbikowski SM, et al. The uninsured and Medicaid Oregon tobacco user experience in a real-world phone-based cessation program. *Tobacco Control*. 2003;12:45–51.
- 11. Curry SJ, Grothaus LC, McAfee TA, Pabiniak C. Utilization and cost-effectiveness of smoking cessation services under four insurance coverage structures in a health maintenance organization. *NEJM*. 1998;339(10):673–679.
- 12. http://www.naquitline.org. Accessed April 2006.
- 13. Swartz SH, Tworek C, Welton M. Increasing use of quitline services in a State with comprehensive tobacco treatment: Maine. Submitted to *MMWR Morb Mort Wkly Rep.* April 2006.
- 14. Zhu SH, Anderson CM. Tobacco quitlines: where they've been and where they're going. In: Isaacs SL, Simon JA, Schroeder SA, eds. *VA in the Vanguard: Building on Success in Smoking Cessation*. Proceedings of a Conference Held September 21, 2004.
- 15. Fiore MC, et al. Preventing 3 million premature deaths and helping 5 million smokers quit: a national action plan for tobacco cessation. *Am J Pub Hlth*. Feb 2004;94(2):205–210.
- 16. Fu SS, et al. Promoting repeat tobacco dependence treatment: are relapsed smokers interested? *Am J Managed Care*. 2006 Apr; 235–243.
- 17. Bentz CJ, Bayley KB, Bonin KE, et al. Provider feedback to improve 5 A's tobacco cessation in primary care: a cluster randomized clinical trial 2006. *Nicotine and Tobacco Research*. (in press).
- 18. West R, Sohal T. Catastrophic pathways to smoking cessation: findings from national survey. BMJ,doi:10.1136/BMJ.38723.573866.AE; January 27, 2006.
- 19. Zhu S-H, Nguyen QB, Cummins S, Wong S, Wightman V. A preliminary study: Non-smokers seeking help for smokers. *Tob Control.* doi:10.1136/tc.2005.012401:2006;15;107–113.

Changing Product/New Tobacco Delivery System Dorothy Hatsukami, Ph.D.

Tobacco prevention and cessation are the most effective methods to reduce mortality and morbidity associated with tobacco use. However, because so many smokers are unable or unwilling to quit smoking at any one time, reducing—rather than eliminating—harm associated with tobacco use has been considered as a potentially viable alternative intervention method. 1 Methods to reduce exposure and potentially reduce risk for disease include reducing the amount of tobacco use, modifying tobacco products and/or using specialized filters for cigarettes to reduce toxicant levels, heating rather than burning a tobacco product, or switching from combustible to noncombustible products. Although the number of studies that have examined these potentially reduced exposure products (PREPs) are limited, 2 it is becoming apparent that the potential for harm reduction from combustible products is unlikely. Although products that contain solely nicotine (and that are regulated) will produce the least harm of all PREPs, oral noncombustible products may also lead to reduced harm relative to cigarettes. Compared to cigarette smoking, smokeless tobacco use is associated with significantly lower morbidity and mortality, has less potential for addiction, and is associated with higher rates of cessation than cigarettes.³ However, these results do not suggest that smokeless tobacco is safe and that it does not produce addiction. The use of these products has been associated with oral pathologies, including oral cancers, as well as pancreatic cancers. ^{4,5} Nonetheless, on an individual risk basis, smokeless tobacco has a significant potential to reduce harm. On the other hand, it is uncertain if, on a population basis, smokeless tobacco as a method to reduce disease might in fact increase potential harm by increasing the prevalence of tobacco use—particularly if smokeless tobacco is a gateway product for cigarette smoking—by encouraging continued use rather than cessation, or by resulting in high incidence of dual use of products with consequent greater toxicity. The fact that the public health impact depends not only on the physical characteristics of the products but also on how they are used, misused, and how their use affects other product use, is part of the reason that caution was urged by the Institute of Medicine Committee, and currently by the World Health Organization (http://www.who.int/tobacco/communications/events/wntd/2006/ rationale/en/index.html).

Nonetheless, research on the addiction potential and toxicity across different smokeless tobacco products, particularly reduced-nitrosamine products, is essential to determine the potential of these products for reduced exposure and reduced harm. Various smokeless tobacco products have also been evaluated for amount of nicotine. ^{6,7} Table 1 shows the studies that describe amounts of nicotine, the pH levels, and percent-free nicotine: the higher the pH, the greater the availability of free or bioavailable nicotine, which is more readily and quickly absorbed than ionized nicotine. To date, several studies have examined the amount of toxicants, particularly tobacco specific nitrosamines (TSNAs). ^{8,9} TSNAs are the most significant carcinogens in smokeless tobacco and cigarettes. ¹⁰ Two of these constituents, 4- (methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and *N*'-nitrosonornicotine (NNN), are carcinogenic in laboratory animals. ¹⁰ Both these carcinogens have been evaluated by the International Agency for Research on Cancer as carcinogenic to humans (Group 1). ⁵ A more recent study was conducted that shows TSNAs in more novel products as well as medicinal

nicotine (see table 2). The results show that medicinal nicotine contains minimal or nonexistent levels of TSNA. Ariva, a tobacco lozenge, also contains low levels of TSNA. Exalt and Revel, which are being marketed to smokers, contain relatively higher levels of TSNA, and the most popular brands of smokeless tobacco sold in the United States contain the highest levels among the products tested. A few studies have been conducted in humans. Figure 1 presents a summary of the various studies that examined the uptake of one of the TSNAs, as measured by 4-(methylnitrosamino)-l-(3 pyridyl) l-butanol (NNAL) plus its glucuronides (total NNAL), metabolites of the tobacco-specific carcinogen, 4-(methylnitrosamino)-l-(3-pyridyl)-l-butanone (NNK). The results in humans paralleled the TSNA product studies. Interestingly, the lowest levels of total NNAL were found with medicinal nicotine, and similar levels were observed for Ariva. As expected, the highest levels were found with Copenhagen and Skoal.

Several methods exist to determine addiction potential for tobacco products. These include examining nicotine pharmacokinetics, with the product producing the fastest and highest peak being associated with higher addiction potential. In addition, subjective responses to the products will also be informative. Figure 2 shows the nicotine pharmacokinetics across conventional smokeless tobacco products. The most popular products show the highest and fastest delivery of nicotine and also produce the most subjective responses. Another method to test addiction potential is to have individuals sample products and then provide them a choice of the product to use. Two studies were conducted, one examining Ariva vs. a medicinal nicotine lozenge and another examining Exalt vs. a medicinal nicotine lozenge. The results showed the Ariva was used more frequently than medicinal nicotine, however, no preference was demonstrated for Exalt over medicinal nicotine. The products and the products are producted to use.

More research examining these novel products that include the types of testing described in figure 3 is necessary. These studies should involve identifying and examining the effects of any new constituents. In addition, human clinical trials need to be conducted that are longer term and involve both naturalistic use of products—to determine patterns of use and resulting toxicant exposure—and sole use of the product. Conducting these trials would involve identifying reliable and valid biomarkers for exposure and toxicity. In addition, studies need to be conducted that assess consumer perception of these novel products and the impact of marketing claims on this perception. Research is also necessary to ensure the relative risk of products is conveyed in a way that is accurately interpreted. Finally, surveillance that examines prevalence of use of these products, the pattern of use, and consequences of use is necessary.

In summary, of all the novel PREPs and methods for reducing cigarette consumption, with the exception of medicinal nicotine, novel oral noncombustible products that contain low toxicant levels are likely to lead to reduced harm in smokers. Although medicinal nicotine is the safest product, because of the current regulatory environment, medicinal nicotine undergoes rigorous testing, is manufactured to be acceptable but not attractive in its own right, and thus is unlikely to produce addiction (and therefore sustained use) compared to the tobacco products, and tends to be high in cost. No such regulation is available for tobacco products. Therefore, regulation of tobacco products is essential to accurately inform consumers of the extent of toxicity of various tobacco products, to reduce toxicant levels in all tobacco products, and to render "the playing field" equivalent for tobacco and medicinal nicotine products. Research should be conducted toward this end and toward the end of providing the methodology and measures for tobacco regulation.

 Table 1. Nicotine Content of Smokeless Tobacco Products

Dose	Product	РН	Nicotine Content mg/g	Free Nicotine (%)
Low	Skoal Bandits ⁷ Wintergreen	6.9	7.5	7.0
	Skoal Bandits ⁶ Straight	5.4	10.1	0.2
	Hawken ⁶ Wintergreen	5.7	3.2	0.5
Medium	Skoal Long Cut ⁷ Straight Wintergreen Cherry	7.4–7.5	10.3–11.4	19.0–23.0
Medium High	Skoal Original Fine Cut Wintergreen	7.6 ⁷ 7.5 ⁶	10.4 11.9	28.0 22.0
High	Kodiak Wintergreen	8.2 ⁶	10.9	60.0
	Copenhagen	8.6 ⁷ 8.0 ⁶	11.4 12.0	79.0 49.0

Table 2. Tobacco-Specific Nitrosamines in Products⁹

Product		μg/g	product (w	et weight)	
	NNN	NNK	NAT	NAB	Total
New tobacco products					
Hard snuff					
Ariva	0.019	0.037	0.12	0.008	0.19 ^a
Stonewall	0.056	0.043	0.17	0.007	0.28 ^b
Spit-free tobacco packets					
Exalt					
Purchased in Sweden	2.3	0.27	0.98	0.13	3.7 ^c
Purchased in United States	2.1	0.24	0.68	0.05	3.1 ^b
Revel					
Mint flavored	0.62	0.033	0.32	0.018	0.99 ^b
Wintergreen flavored	0.64	0.032	0.31	0.017	1.0 ^b
Tobacco-free snuff					
Smokey Mountain	nd	nd	nd	nd	nd^{b}
Nicotine replacement therapy products					
NicoDerm CQ (patch, 4 mg nicotine) ^e	nd	0.008	nd	nd	0.008^{b}
Nicorette (gum, 4 mg nicotine) ^e	0.002	nd	nd	nd	0.002^{b}
Commit (lozenge, 2 mg nicotine) ^e	nd	nd	nd	nd	nd^b
Conventional tobacco products					
Smokeless tobacco					
General	0.98	0.18	0.79	0.06	2.0^{d}
Copenhagen					
Snuff	2.2	0.75	1.8	0.12	4.8 ^b
Long cut	3.9	1.6	1.9	0.13	7.5 ^b
Skoal					
Long cut straight	4.5	0.47	4.1	0.22	9.2 ^b
Bandits	0.9	0.17	0.24	0.014	1.3 ^b
Kodiak					
Ice	2.0	0.29	0.72	0.063	3.1 ^b
Wintergreen	2.2	0.41	1.8	0.15	4.5 ^b

 ^a Mean of 5 analyses, each performed in duplicate
 ^b Single analysis performed in duplicate
 ^c Mean of 3 analyses, each performed in duplicate
 ^d Mean of 2 analyses, each performed in duplicate
 ^e Values are expressed per piece

Figure 1. Total NNAL Concentrations Across Different Brands of Smokeless Tobacco^{12,14}

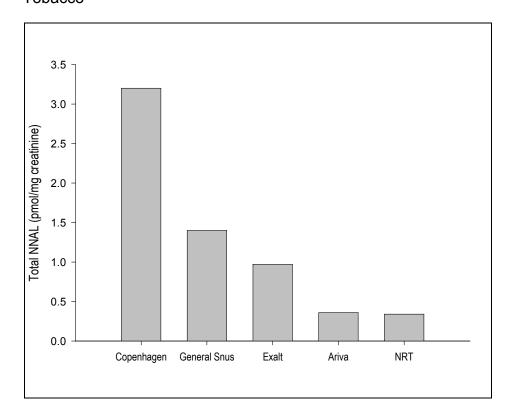
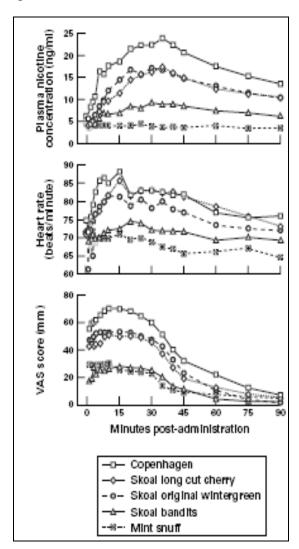


Figure 2. Mean Plasma Concentration, Heart Rate, and Total Visual Analogue Score for Drug Strength After Administration of Each of the Five Products¹¹



Step 1: Preclinical evaluation Step 2: Clinical evaluation of exposure reduction Step 3: Population effect, and health effects and market research regarding harm reduction evaluation labeling/advertising Comprehensive clinical trials assessing Post-marketing exposure reduction, health effects, abuse surveillance and liability, patterns of use (e.g., Phase I and II/III epidemiological studies). Outcomes: biomarkers relevant to surveys to examine carcinogen uptake and cardiovascular and population effects 1) Smoke chemistry pulmonary function. of PREP 2) Machine determined Long-term clinical vields outcome: Phase II/ Cell culture studies Market research assessing how best to III follow-up, Phase 4) Animal testing IV to assess communicate exposure or risk reduction without implying harm reduction. disease outcome. Goal: Goals: Goals: Determine that any PREP 1) Test products for exposure/risk reduction 1) Evaluate/monitor that proceeds has, as PREP effects on and health effects determined by an 2) Devise labeling/advertising strategies that tobacco use behavior. independent body of are appropriate for exposure/risk reduction 2) Determine effects on scientists, substantial claims. morbidity and potential to reduce risk of 3) Evaluate proposed advertisements mortality. disease, taking into account through consumer testing to ensure Assess for unintended the risk-reducing potential of appropriateness. consequences.

Figure 3. Steps to Evaluate Potential Reduced Exposure Products¹⁴

References

nicotine.

products such as medicinal

Pre-market

- 1. Stratton K, Shetty P, Wallace R, Bondurant S, eds. *Clearing the Smoke: Assessing the Science Base for Tobacco Harm Reduction*. Washington, DC: Institute of Medicine, National Academy Press; 2001
- 2. Hatsukami D, Hecht SS. *Hope or Hazard: What Research Tells Us About "Potentially Reduced-Exposure" Tobacco Products*. Minneapolis: University of Minnesota Transdisciplinary Tobacco Use Research Center; 2005.
- 3. Hatsukami D, Lemmonds C, Tomar S. Smokeless tobacco use: Harm reduction or induction approach? *Preventive Medicine*. 2004;38(3):309–317.
- 4. Hatsukami D, Henningfield J, Kotlyar M. Harm reduction approaches to reducing tobaccorelated mortality. *Annu Rev Public Health*, 2004;25:377–395.
- 5. International Agency for Research on Cancer. *Smokeless Tobacco and Tobacco-Specific Nitrosamines*. (Vol. 89). Lyon, France: International Agency for Research on Cancer; in press.

Post-market

- 6. Djordjevic M, Hoffman D, Glynn T, Connolly G. U.S. commercial brands of moist snuff, 1994. I. Assessment of nicotine, moisture, and pH. *Tobacco Control*. 1995;4:62–66.
- 7. Henningfield J, Radzius A, Cone E. Estimation of available nicotine content of six smokeless tobacco products. *Tobacco Control*. 1995;4(1):57–61.
- 8. Hoffmann D, Djordjevic MV, Fan J, Zang E, Glynn T, Connolly GN. Five leading U.S. commercial brands of moist snuff in 1994: assessment of carcinogenic N-nitrosamines. *Journal of the National Cancer Institute*. 1995;87(24):1862–1869.
- 9. Stepanov I, Jensen J, Hatsukami D, Hecht SS. Tobacco-specific nitrosamines in new tobacco products. *Nicotine & Tobacco Research*. In press.
- 10. Hecht SS. Biochemistry, biology, and carcinogenicity of tobacco-specific N- nitrosamines. *Chemical Research in Toxicology.* 1998;11(6):559–603.
- 11. Fant RV, Henningfield JE, Nelson RA, Pickworth W. Pharmacokinetics and pharmacodynamics of moist snuff in humans. *Tobacco Control*. 1999;8(4):387–392.
- 12. Mendoza-Baumgart M, Tulunay O, Jensen J, Hatsukami D. Smokeless tobacco vs. medicinal nicotine: Ssubjective and behavioral consequences. Presentation at: *The Society for Research on Nicotine and Tobacco Annual Meeting*. Prague, Czech Republic: 2005.
- 13. Hatsukami D, Giovino GA, Eissenberg T, Clark PI, Lawrence D, Leischow S. Methods to assess potential reduced exposure products. *Nicotine & Tobacco Research*. 2005;7(6):827–844.
- 14. Hatsukami D, Lemmonds CA, Zhang Y, Murphy SE, Le C, Carmella SG, Hecht SS. Evaluation of carcinogen exposure in people who used "reduced exposure" tobacco products. *Journal of the National Cancer Institute*. 2004;96(11):844–852.

Policy Perspective for Tobacco Risk Reduction

Lynn T. Kozlowski, Ph.D.

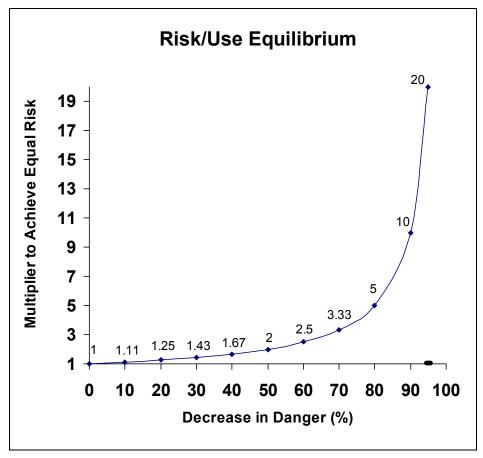
Leashing the "Pussycat" While the "Lion" Runs Free: The Problematic Mismatch of Regulations and Product Risks

In the United States, the least dangerous (and not very dangerous) form of nicotine delivery system, nicotine replacement therapy products (NRTs, or "medicinal nicotine"), has been subject to close, science-influenced oversight by the U.S. Food and Drug Administration (FDA), while the most dangerous (and extremely dangerous) nicotine delivery system, the cigarette, has been exempted from FDA and even Consumer Product Safety jurisdiction. Furthermore, cigarettes are advertised widely in magazines, their makers can add to their reputation in national advertising campaigns on television in prime time, and cigarettes can be readily, though illegally, bought at relatively low cost by even the marginally enterprising 12-year-old. Smokeless tobacco products, especially those with high levels of cancer-causing nitrosamines, are also exempted from drug and product laws. Much to the chagrin of many antitobacco advocates, the risks of manufactured smokeless tobacco products in the United States or Sweden, especially those with low nitrosamine levels, are much closer to the risks of NRTs than they are to cigarettes. Smokeless tobacco products are dangerous (NOT SAFE) products, but they are undoubtedly less dangerous (SAFER) products than cigarettes.

Population Harm and Smokeless Tobacco—Using the Risk/Use Equilibrium

Even though smokeless tobacco is *less* dangerous to individual users than cigarettes, if many more people start using the product, is it likely to produce a net loss for public health? This has become a frequent question when considering the population effects of less dangerous products for individuals. To get a sense of scale for the possible problems caused by increased use of a less dangerous product, we employ what we call *the risk/use equilibrium*—an equilibrium achieved by increasing use as risk decreases.³ Figure 1 plots the equilibrium line—the level at which a decrease in risk is made equal to the initial risk by virtue of an increased number of users. If the level of use rises faster than risk is decreased, public health would be hurt. If risk levels are decreased faster than use rises, public health would be helped. Figure 1 plots the relationship between level of risk and the increase in the number of users (as a multiplier) needed to achieve equilibrium, or, in other words, no increased population-level risks.

Figure 1. The Risk/Use Equilibrium



Each point on this curve indicates the multiplier needed to achieve a constant level of population risk, given specific levels of decreased danger per user. For example, if 100 individuals used a product with full danger (e.g., killing 100% of users), 10 times that number (1,000 individuals) would need to use a product that had 90% decreased danger, to achieve an equal health problem (100 dead in each instance). The formula is Y=100/100-X, where Y=Multiplier and X=Decrease in Danger, expressed in percentages. If danger is 0.1%, use would have to increase by 1,000 times to produce a problem of the same magnitude as the full-risk product (not plotted on figure). For a given risk on the curve, use that is increased by a smaller multiplier represents a public health benefit, and use that is increased by a larger multiplier represents a public health (population-level) cost.

Kozlowski LT, Strasser AA, Giovino GA, Erickson PA, Terza JV. Applying the risk/use equilibrium: Use medicinal nicotine now for harm reduction. *Tobacco Control*. 2001;10:201–203.

In 2003, a British Royal College of Physicians expert committee concluded that smokeless tobacco is from about "10–1,000 times less hazardous than smoking, depending on the product." While it is difficult to determine the precise level of risks to health from smokeless tobacco products, it is likely to be substantially less dangerous than cigarettes, especially for low nitrosamine products. It is reasonable to assume that very low nitrosamine products could be about 1,000 times less hazardous than smoking, which, according to the risk/use equilibrium would indicate very little chance for net public health harm.

The Specter of Smokeless Tobacco as a Causal Gateway to Cigarettes

Another concern has been that, even if smokeless tobacco is less dangerous than cigarettes, if smokeless tobacco use *leads to* (is a *causal* gateway) to cigarettes, then the reduced risk is, in a sense, only transitory. A "correlational gateway" between smokeless tobacco and cigarettes is easy to find evidence for, but a "causal gateway" in which prior use of smokeless tobacco causes an increased probability of smoking is much more challenging to prove. Some experts have argued that smokeless tobacco use in youth may lead to subsequent cigarette smoking; others have argued that smokeless tobacco may even act to prevent cigarette smoking in high-risk youth, and that much of the association between smokeless and smoking is not causally linked. But it is also true that marketing and prevention efforts to reduce cigarette smoking should be able to decrease the possible progression from smokeless to cigarettes. Mistaken public health information that smokeless tobacco is *as* dangerous or even *more* dangerous than cigarettes (see below) may actually promote the movement to cigarettes by smokeless tobacco users.

Health Communication Issues on Smokeless Tobacco Products

The health communication practices on comparative risks of tobacco products on some major Web pages have improved recently in the United States. A few years ago, for example, in 2001, the Centers for Disease Control and Prevention's (CDC's) Surgeon-General's Report for Kids asked the question "Is smokeless tobacco safer than cigarettes?" and answered "NO WAY!" and the Web page of the Substance Abuse and Mental Health Administration's (SAMHSA's) National Clearinghouse for Alcohol and Drug Information² contained this passage: "Q. Isn't smokeless tobacco safer to use than cigarettes? A. No. There is no safe form of tobacco." (accessed August 14, 2002). Kozlowski and O'Connor⁸ raised issues about the science base of such prominent public information on smokeless and questioned the ethical justification for this disinformation on comparative risks of smoking and smokeless tobacco use. There was also a formal complaint under the Data Quality Act that resulted in the National Institute of Aging changing the information on its Web site on smokeless tobacco. And in 2005 there was an analysis by Philips, Wang, and Guenzel on the "misleading and harmful public message about smokeless tobacco"¹⁰ to be found on the Internet. Accessing (or trying to access) these pages on March 28, 2006, however, demonstrated some major positive changes in the sites mentioned above and, for example, in the American Cancer Society's Web page. It seems to have become less controversial to indicate the established risks of smokeless tobacco products and to forgo hyperbole.

"Not Safe" Is Not Enough

Health communications should also do more than inform that "there is no safe tobacco product." The congressionally mandated rotating warning that "WARNING: This product is not a safe alternative to cigarettes" is, in effect, a "not safe" message. At present there is an alignment of the basic message from the National Cancer Institute ¹² and from the Philip Morris Tobacco Company ¹³ (on their Web site and in national advertising) that there is no safe tobacco product. This message is of course true, but it is also a truism, in that "nothing is completely safe," and it is of limited value in that the public largely knows that there is no safe tobacco product. ¹¹ One of the lessons of the low-tar-cigarette disaster is that, while the public understands

that "low-tar" cigarettes are "not safe," a public health tragedy has resulted from the simultaneous, non-contradictory belief that low-tar cigarettes were "safer" (when they are not). ¹⁴ Given the years of mistaken information about the comparative risks of smokeless and cigarettes in the United States, there may also be an active need to correct the belief that smokeless tobacco is more dangerous than cigarettes.

Policy Questions

How important is it to make more dangerous products harder to get?

Would it be good for population health to adjust the expense (e.g., by taxation), the marketing/promotion, and the availability of tobacco/nicotine delivery systems, such that it is harder to get more dangerous products and easier to get less dangerous products? Would such efforts reduce the sales of the most dangerous products?

How should cross-product comparisons be done to assess health risk differences?

Based on mortality, smokeless products are less dangerous than cigarettes. It is less common to compare on the basis of morbidity or quality-of-life differences. Premature death is part of the tragedy of chronic obstructive lung disease; but long-suffering, behaviorally impaired years of life are also part of the tragedy. More complete assessments of disease- *and* disability-risk differentials are needed and may help motivate smokers to turn away from cigarettes.

What is the best way to communicate risk differences?

Research is urgently needed to develop ways to communicate science-based risk differentials in ways that minimize deception and maximize the likelihood of choices that benefit public health. For example, is it better to avoid speaking of "safer" products and better to describe them as "less dangerous" or "less harmful"?

Why aren't all smokeless tobacco products sold in the United States meeting a very low nitrosamine standard?

If the United States ever were able to establish a strong, science-based, public-health-concerned regulatory authority, low nitrosamine levels would likely be required in all smokeless tobacco products, to help reduce cancer risks. Even without regulatory oversight, one wonders why more manufacturers are not voluntarily reducing levels of nitrosamines in their products. Given that some products on the market now contain extremely low levels of nitrosamines, might health communication campaigns related to levels of toxic ingredients in smokeless tobacco help discourage the use of the more toxic products and even have an effect on the manufacturing of such products?

References

- 1. Critchley JA, Unal B. The health effects associated with smokeless tobacco use: a systematic review. *Thorax.* 2003;58:435–443
- 2. Foulds J, Ramstrom L, Burke M, et al. The effect of smokeless tobacco (Snus) on smoking and public health in Sweden. *Tobacco Control*. 2003;12:349–359.
- 3. Kozlowski LT, Strasser AA, Giovino GA, et al. Applying the risk/use equilibrium: use medicinal nicotine now for harm reduction. *Tobacco Control*. 2001;10(3):201–203.
- 4. Tobacco Advisory Group of the Royal College of Physicians. Protecting smokers, saving lives. 2002. Available at: http://www.rcplondon.ac.uk/pubs/books/protsmokers/index.asp. Accessed 9 April 2006.
- 5. Tomar SL. Is use of smokeless tobacco a risk factor for cigarette smoking? The U.S. experience. *Nicotine and Tobacco Research*. 2003;5(4):561–570.
- 6. O'Connor RJ, Flaherty BP, Edwards BQ, et al. Regular smokeless tobacco use is not a reliable predictor of smoking onset when psychosocial predictors are included in the model. *Nicotine and Tobacco Research.* 2003;5(4):535–544.
- 7. Kozlowski LT, O'Connor RJ, Edwards BQ, et al. Most smokeless tobacco use is not a causal gateway to cigarettes: Using order of product use to evaluate causation in a national U.S. sample. *Addiction*. 2003;98(8): 1077–1085.
- 8. Kozlowski LT, O'Connor RJ. Apply Federal research rules on deception to misleading health information: An example on smokeless tobacco and cigarettes. *Public Health Reports*. 2003;118(3),187–192.
- 9. National Legal and Policy Center. National Institute on Aging forced to re-examine policy on smokeless tobacco products. *PR Newswire*. July 14, 2004. Available at: http://www.tobacco.org/news/170153.html. Accessed March 4, 2005.
- 10. Phillips CV, Wang C, Guenzel B. You might as well smoke; The misleading and harmful public message about smokeless tobacco. *BMC Public Health*. 2005,5:31. Available at: http://www.biomedcentral.com/1471–2458/5/31.
- 11. Kozlowski LT, Edwards, BQ. "Not safe" is not enough: smokers have a right to know more than there is no safe tobacco product. *Tobacco Control*. Aug 2005;14:ii3–ii7.
- 12. National Cancer Institute. Cancer Facts: The truth about "light" cigarettes. Reviewed 2003. Available at: http://cis.nci.nih.gov/fact/3_74.htm. Accessed February 24, 2004.

- 13. Philip Morris USA. Health issues: Cigarette smoking and disease in smokers. Available at: http://www.pmusa.com/health_issues/cigarette_smoking_and_disease.asp. Accessed February 24, 2004.
- 14. National Cancer Institute. *Risks Associate with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine*. Smoking and Tobacco Control Monograph 13. Bethesda, Md: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute. NIH Pub. No. 02–5074, October 2001.

Epidemiological Perspective for Tobacco Risk Reduction Scott L. Tomar, D.M.D., Dr.P.H.

Hundreds of millions of people worldwide are addicted to smokeless tobacco, and use by young people is increasing in many countries. The types of product vary widely around the world. The common defining characteristics of smokeless tobacco products are that they are not burned by the consumer at the time they are used; they deliver nicotine into venous circulation through passive absorption across oral or nasal mucosa; and virtually all products contain human carcinogens and toxins in levels substantially higher than are typically found in any non-tobacco consumer product.

There has been recent discussion within the tobacco control community concerning the role, feasibility, and supporting evidence of a harm-decreasing strategy in reducing the societal burden of tobacco use. Because of lower risks for morbidity or mortality compared with cigarettes, various smokeless tobacco products, particularly moist snuff, have been suggested as potential reduced-exposure products for smokers who are unable or unwilling to quit using tobacco. ^{2–4}

Compared with the large body of literature on the adverse health effects of cigarette smoking, the literature on adverse health effects associated with smokeless tobacco use is far smaller and, for some disease endpoints, less conclusive. In part, that limitation reflects the nature of smokeless tobacco use in most parts of the world. In general, smokeless tobacco use is far less prevalent than smoking in most developed nations, so observational studies that include an adequate sample size of exposed persons are more difficult to assemble. In addition, a large proportion of smokeless tobacco users in most countries also have a history of using burned types of tobacco products such as cigarettes, bidis, or hookahs, a factor that creates challenges in identifying cohorts whose only form of tobacco use is smokeless tobacco.

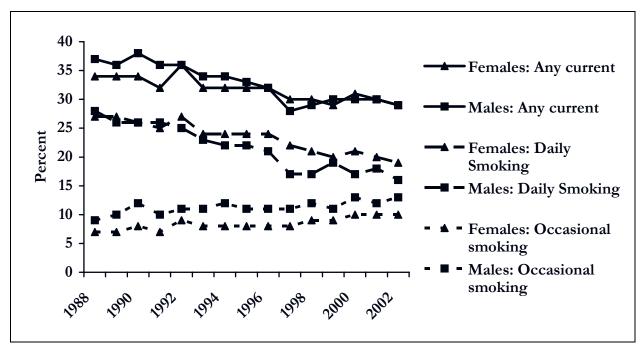
There have been several recent reviews on the health effects of smokeless tobacco. A working group recently convened by the International Agency for Research on Cancer reviewed the available epidemiological, animal, and chemical literature. The working group concluded there is sufficient evidence that smokeless tobacco causes oral cancer and pancreatic cancer in humans, and sufficient evidence of carcinogenicity from animal studies. The working group concluded that smokeless tobacco is "carcinogenic to humans." Several large prospective cohort studies suggest that smokeless tobacco use may elevate the risk of death from cardiovascular diseases, 6,7 though several case-control studies did not find a significant association. There is some evidence that smokeless tobacco use increases the risk for adverse reproductive health outcomes among pregnant women, including low birth weight, preterm delivery, stillbirth; and pre-eclampsia. Oral health effects include localized periodontal destruction, soft tissue lesions, and, possibly, dental abrasion. There is compelling evidence that smokeless tobacco use can result in nicotine addiction.

To date, only one study has explicitly examined the effectiveness of snuff use as a smoking cessation method. ¹⁵ That pilot study found that 16 of the 63 subjects (25%) in the study had quit smoking by using snuff at the 1-year followup, and 6 subjects (10%) had quit smoking

by using another method. However, this study did not have a control group. In a 7-year followup of 62 of the original 63 subjects, 28 (45%) had quit smoking, although fewer than half of the subjects (n=12) who had quit smoking in that uncontrolled study reportedly had done so by using snuff.¹⁶

Sweden has been cited as the single example in which moist snuff use apparently replaced smoking for a proportion of males. The prevalence of daily smoking dropped by 50% between 1980 and 2001 among 16–24-year-old males, while their snuff use increased, but the same rate of decline in smoking was seen for young women with almost no snuff use. ¹⁷ The pattern of smoking that appears to be emerging in Sweden is a declining but equal prevalence of current smoking for men and women, with a greater proportion of male current smokers than female smokers reporting smoking less often than daily (figure 1). Based on a cross-sectional study of current and former smokers in Sweden, ¹⁸ the apparent effectiveness of snuff in helping smokers to quit is modest at best. Among males, snuff was used in the most recent attempt to quit smoking by 28.7% of former smokers and 23.0% of current smokers (p=0.072). Only 4.8% of female current smokers in that study who had attempted to quit and 4.5% of female former smokers (p=0.85) reported using snuff during their most recent attempt to quit smoking. Switching from cigarettes to snuff in Sweden appears to occur primarily among men who had prior experience using snuff. ¹⁰

Figure 1. Prevalence of Daily or Occasional Smoking, Age 16–24 years, Sweden 1988–2002.



Source: Unpublished data from Swedish ULF Surveys, Statistics Sweden

Ecological patterns in Norway do not support a substitution effect for snuff on the prevalence of cigarette smoking (see figure 2). As in Sweden, snuff use is an almost entirely male behavior. The prevalence of snuff use among 16–24-year-old males increased from 9.3% in

1985 to 18.6% in 2003, while the prevalence of current smoking remained relatively stable at greater than 40% and generally equal for males and females during that period. Snuff use also increased among males older than 25 years of age since 1985, but snuff is rarely used by Norwegian women; rates of daily or any current smoking remain virtually equal for men and women ages 25–34 or 35–44.

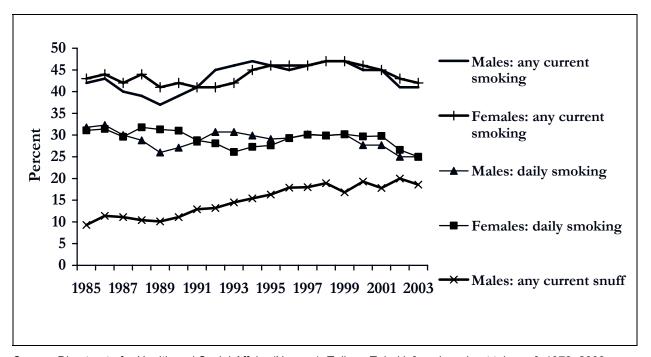


Figure 2. Smoking and Snuff Use, Norway, 1985–2003, Age 16–24.

Source: Directorate for Health and Social Affairs (Norway). Tall om Tobakk [number about tobacco]. 1973–2003. [Norwegian]. Oslo: Directorate for Health and Social Affairs; 2004.

There is little information on the proportion of U.S. smokers who have switched completely to the use of smokeless tobacco or have used smokeless tobacco as a method of quitting smoking. Fiore and colleagues¹⁹ reported findings from the 1986 Adult Use of Tobacco Survey on the methods smokers used to quit. That study reported that, in the mid-1980s, 6.8% of former smokers who successfully quit smoking for at least 1 year had substituted other tobacco products (including snuff, chewing tobacco, pipes, or cigars) during any attempt at quitting, and 4.0% of successful quitters substituted other products during their last attempt at quitting. However, the proportions were nearly the same among relapsers: 6.8% of smokers who made a serious quit attempt in the past year but were not successful in quitting tried substituting other tobacco products in any attempt, and 2.1% tried that strategy during their last attempt at quitting. More recent data come from the 2000 National Health Interview Survey, in which former smokers were asked what method they had used to quit smoking completely. ²⁰ Just 1% of male former smokers ages 36-47 reported switching to snuff or chewing tobacco to quit smoking. Of male current smokers in that age group who attempted to quit, 0.3% reported switching to smokeless tobacco on their last attempt to guit. In a birth cohort in which 16% of males, including 19% of those who had ever smoked, had used smokeless tobacco by age 34, smokeless tobacco use accounted for a very small proportion of quitting.

Smoking is relatively prevalent among smokeless tobacco users in the United States. In a nationally representative cross-sectional study, current smoking was reported by 19% of those who used snuff every day, 39% of occasional snuff users, and 39% of former snuff users.²¹

If smokeless tobacco were used as a substitute for cigarettes in some U.S. States, it might be expected that there would be a negative association between the prevalence of smokeless tobacco use and cigarette smoking. To the contrary, in multiple linear regression modeling, there was a significant positive association between State-level prevalence of smokeless tobacco use and cigarette smoking among males age 18 years and older in 1998–2000.

Smokeless tobacco use remains highly prevalent in India, where oral cancer vies with lung cancer as the most common type of malignancy.²² Smoking quit rates are very low in India, and there is no evidence that widespread use of the various forms of smokeless tobacco has reduced the use of burned tobacco products such as bidis. As in other countries, the initiation of smokeless tobacco use in India occurs primarily at young ages.²²

Use of various smokeless tobacco products is widespread throughout South Asia and parts of Africa. There is no evidence from these regions that these products have led to reductions in cigarette smoking or use of other burned tobacco products.

In conclusion, the available evidence suggests: (1) the uptake of moist snuff in the United States and several European countries during the past several decades has occurred primarily among adolescent and young adult males; (2) increased prevalence of snuff use is not consistently associated with a reduction in smoking initiation or prevalence; (3) moist snuff use apparently plays a very minor role in smoking cessation in the United States and an inconsistent role in Sweden; (4) U.S. States with the lowest smoking prevalence also tend to have the lowest prevalence of smokeless tobacco use and vice versa; (5) there are no data on the efficacy of snuff as a smoking cessation method; and (6) cigarette smoking is fairly prevalent among smokeless tobacco users in several countries. The evidence base for smokeless tobacco promotion as a public health strategy is weak and inconsistent.

References

- 1. Institute of Medicine. Stratton K, Shetty P, Wallace R, Bondurant S, eds. *Clearing the Smoke: Assessing the Science Base for Tobacco Harm Reduction*. Washington, DC: National Academy Press; 2001.
- 2. Rodu B. An alternative approach to smoking control [editorial]. *Am J Med Sci*. 1994;308(1):32–34.
- 3. Ramstrom LM. Snuff: an alternative nicotine delivery system. In: Ferrence R, Slade J, Room R, Pope M, eds. *Nicotine and PublicHealth*. Washington, DC: American Public Health Association, 2000:155–174.
- 4. Bates C, Fagerstrom K, Jarvis MJ, Kunze M, McNeill A, Ramstrom L. European Union policy on smokeless tobacco: a statement in favour of evidence based regulation for public health. *Tob Control*. 2003;12(4):360–367.

- 5. Cogliano V, Straif K, Baan R, Grosse Y, Secretan B, El Ghissassi F. Smokeless tobacco and tobacco-related nitrosamines. *Lancet Oncol.* 2004;5(12):708.
- 6. Bolinder G, Alfredsson L, Englund A, de Faire U. Smokeless tobacco use and increased cardiovascular mortality among Swedish construction workers [see comments]. *Am J Public Health*. 1994;84(3):399–404.
- 7. Henley SJ, Thun MJ, Connell C, Calle EE. Two large prospective studies of mortality among men who use snuff or chewing tobacco (United States). *Cancer Causes Control.* 2005; 16(4):347–358.
- 8. Huhtasaari F, Asplund K, Lundberg V, Stegmayr B, Wester PO. Tobacco and myocardial infarction: is snuff less dangerous than cigarettes? *BMJ*. 1992;305(6864):1252–1256.
- 9. Huhtasaari F, Lundberg V, Eliasson M, Janlert U, Asplund K. Smokeless tobacco as a possible risk factor for myocardial infarction: A population-based study in middle-aged men. *J Am Coll Cardiol*. 1999;34(6):1784–1790.
- 10. Rodu B, Stegmayr B, Nasic S, Cole P, Asplund K. Evolving patterns of tobacco use in northern Sweden. *J Intern Med.* 2003;253(6):660–665.
- 11. England LJ, Levine RJ, Mills JL, Klebanoff MA, Yu KF, Cnattingius S. Adverse pregnancy outcomes in snuff users. *Am J Obstet Gynecol*. 2003;189(4):939–943.
- 12. Gupta PC, Sreevidya S. Smokeless tobacco use, birth weight, and gestational age: population based, prospective cohort study of 1217 women in Mumbai, India. *BMJ*. 2004;328(7455):1538.
- 13. Christen AG, McDonald JL, Christen JA. *The Impact of Tobacco Use and Cessation on Nonmalignant and Precancerous Oral and Dental Diseases and Conditions*. Indianapolis: Indiana University School of Dentistry; 1991.
- 14. Hatsukami DK, Severson HH. Oral spit tobacco: addiction, prevention and treatment. *Nicotine Tob Res.* 1999;1(1):21–44.
- 15. Tilashalski K, Rodu B, Cole P. A pilot study of smokeless tobacco in smoking cessation [see comments]. *Am J Med.* 1998;104(5):456–458.
- 16. Tilashalski K, Rodu B, Cole P. Seven year follow-up of smoking cessation with smokeless tobacco. *J Psychoactive Drugs*. 2005;37(1):105–108.
- 17. Wicklin B. *Tobacco Statistics: Sales, Consumption and Lung Cancer* [Web page]. 2006. Available at http://www.statveca.com/english/index.html. Accessed 30 March 2006.
- 18. Gilljam H, Galanti MR. Role of snus (oral moist snuff) in smoking cessation and smoking reduction in Sweden. *Addiction*. 2003;98(9):1183–1189.

- 19. Fiore MC, Novotny TE, Pierce JP, et al. Methods used to quit smoking in the United States. Do cessation programs help? *JAMA*. 1990;263(20):2760–2765.
- 20. Tomar SL, Loree M. Errors in analyzing associations between use of smokeless tobacco and cigarettes. *Addiction*. 2004;99(2):260–2; author reply 262–264.
- 21. Tomar SL. Snuff use and smoking in U.S. men: implications for harm reduction. *Am J Prev Med.* 2002;23(3):143–149.
- 22. Gupta PC, Ray CS. Smokeless tobacco and health in India and South Asia. *Respirology*. 2003;8(4):419–431.

Evidence-Based Practice Center Presentation: Smokeless Tobacco: The Effects of Product Marketing on Use and Population Harm

Leah M. Ranney, Ph.D., M.A.

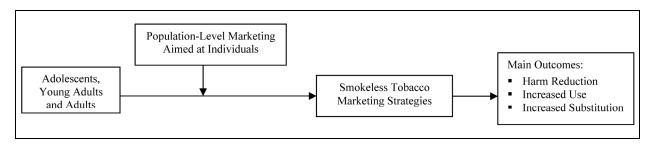
Background

Smokeless tobacco can lead to nicotine addiction and dependence.¹ Evidence from the Surgeon General and others has linked smokeless tobacco causally with oral leukoplakia and oral cancers.^{2,3} Two types of smokeless tobacco are sold in the United States: chewing tobacco (i.e., loose leaf tobacco, plug, or twist) and snuff (i.e., finely ground tobacco that can be dry, moist, or in sachets).^{4,5} An estimated 7% of high school students are current users of smokeless tobacco; males are the primary consumer.⁶ Adolescents who use smokeless tobacco are more likely to become smokers than adolescents who do not use smokeless tobacco products.⁷

Adolescents and young adults continue to be a strategically important market segment for the tobacco industry. During 2001, the largest tobacco manufacturers spent \$236.7 million on smokeless tobacco advertising and promotion using images that portray the attractiveness of tobacco products. Recently, tobacco companies have begun to market their smokeless tobacco products as less harmful alternatives to smoking tobacco, likening them to nicotine replacement products and emphasizing that smokeless tobacco does not carry risk for others as does smoking with second-hand, or environmental tobacco, smoke. We believe that it is too early to determine if these "harm reduction" approaches to smokeless tobacco marketing are effective in increasing its use.

We undertook a systematic review to investigate three concerns of smokeless tobacco product marketing ("key question 4" in the main evidence report), using the analytic framework in figure 1. We examined whether (1) substituting smokeless tobacco for smoking results in less smoking-related harm on a population basis, (2) smokeless tobacco marketing leads to greater use or substitution of smokeless tobacco for smoking (or both), and (3) data on harms and harm reduction associated with smokeless tobacco are used to model the potential health effects of substituting smokeless tobacco for smoking.

Figure 1. Marketing of Smokeless Tobacco Products: Analytic Framework



Methods

We searched standard electronic databases (MEDLINE®, the Cumulative Index to Nursing and Applied Health [CINAHL], Cochrane Collaboration libraries, Cochrane Clinical Trials Register, Psychological Abstracts, and Sociological Abstracts) between January 1, 1980 and June 10, 2005 using Medical Subject Headings as search terms when available or key words when appropriate. We limited our review to (1) human studies conducted in developed countries and published in English; (2) studies with participants ages 13 and older, of both sexes, and diverse racial and ethnic populations; (3) randomized controlled trials (RCTs) with 30 or more individuals; (4) observational studies and other trials with 100 or more individuals; and (5) studies with a minimum follow-up period of 6 months, with or without comparison groups. We excluded editorials, letters, and commentaries; articles that did not report outcomes related to our key questions; and studies that did not provide sufficient information to be abstracted.

We reviewed all eligible studies, extracted and entered relevant data into evidence tables, and summarized findings by descriptive methods. From our review of 639 abstracts, two articles addressed marketing of smokeless tobacco products.

Results: Smokeless Tobacco Product Marketing

We found no study evaluating whether substituting smokeless tobacco for smoking results in less smoking-related harm; similarly, no study can substantiate whether data on harm and harm reduction associated with smokeless tobacco are used to model the potential health effects of substituting smokeless tobacco for smoking. In addition, no prior systematic reviews addressed these issues. The absence of data on substituting smokeless tobacco for cigarettes is striking.

Two studies with quality ratings of fair focused on the smokeless tobacco use. ^{9,10} They investigated (a) how smokeless tobacco use affects smoking behaviors and (b) how exposure to smokeless tobacco advertising affects use. Tomar reported that smokers were more likely to quit smoking than become users of smokeless tobacco. ¹⁰ More importantly, users of smokeless tobacco were significantly more likely than nonusers of tobacco to become smokers. ¹⁰ Some evidence indicates that smokeless tobacco marketing leads to greater use at least for adolescents. ⁹ Choi et al. found that exposure to advertising increased adolescents' susceptibility to smokeless tobacco. One predictor of current use of smokeless tobacco is exposure to smokeless tobacco advertising, resulting in a sevenfold effect on current use. ⁹

Conclusions and Need for Future Research

The body of evidence available to address the three concerns noted above is insufficient, and no previous systematic reviews have been done on these topics. We cannot draw any conclusions without studies on whether smokeless tobacco product marketing results in substituting smokeless tobacco for smoking or whether data on harm and harm reduction associated with smokeless tobacco are used to promote potential health effects of substituting smokeless tobacco for smoking. Consistent with one Surgeon General report, we consider smokeless tobacco users to be more likely to become smokers; this study we identified, however, is insufficient to support definitive recommendations. Additionally, the body of

evidence is insufficient, given only one study, 9 to determine conclusively whether tobacco marketing increases smokeless tobacco use or whether smokeless tobacco is a gateway drug for smoking.

Topics in the area of smokeless tobacco product marketing that warrant future investigation are abundant. Several areas should be considered high priority for research: (1) whether new tobacco industry marketing strategies are increasing use of smokeless tobacco and, if so, whether the observed increase applies differentially to specific populations; (2) whether possible links between point-of-purchase tobacco promotion and advertising increase use of smokeless tobacco among adolescents and young adults; and (3) what strategies are effective to reduce use of smokeless tobacco among adolescents and young adults. Rigorous study designs (i.e., RCTs), longitudinal data, and longer follow-ups for cross-sectional data should be used to expand the smokeless tobacco product marketing and use evidence. Research has established links between use of smokeless tobacco and certain cancers; ¹¹ for that reason and the continuing development of effective smoking cessation treatment such as nicotine replacement therapy, future research should move away from investigating whether smokeless tobacco is a viable substitute for smoking and toward developing strategies to reduce its use. We see many opportunities in the area of smokeless tobacco research to build a stronger evidence base.

- 1. U.S. Department of Health and Human Services, P.H.S., Office of the Surgeon General. The Health Consequences of Using Smokeless Tobacco: A Report to the Advisory Committee of the Surgeon General. Bureau of Maternal and Child Health and Resources Development, Office of Maternal and Child Health; 1986.
- 2. U.S. Department of Health and Human Services. Reducing Tobacco Use: A Report of the Surgeon General. Atlanta, Ga: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Office on Smoking and Health; 2000.
- 3. Winn DM. Smokeless tobacco and aerodigestive tract cancers: recent research directions. *Adv Exp Med Biol.* 1992;320:39–46.
- Federal Trade Commission. Smokeless Tobacco Report for the Years 2000 and 2001. Washington, DC: Federal Trade Commission. Available at: http://www.ftc.gov/os/2003/08/2k2k1smokeless.pdf. Accessed July 2004, 2003.
- 5. National Cancer Institute. Smokeless Tobacco or Health: An International Perspective. Bethesda, Md: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute; 1992. Available at: http://cancercontrol.cancer.gov/tcrb/monographs/2/index.html. Accessed July 2004.
- 6. Substance Abuse and Mental Health Services Administration. Results from the 2004 National Survey on Drug Use and Health. Detailed Tables. Rockville, Md: Substance Abuse and Mental Health Services Administration, Office of Applied Studies. Available at: http://oas.samhsa.gov/nhsda/2k3tabs/Sect2peTabs1to56.htm#tab2.39b. 2005.

- 7. U.S. Department of Health and Human Services. Preventing Tobacco Use Among Young People: A Report of the Surgeon General. Atlanta, Ga: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Office on Smoking and Health; 1994.
- 8. Woodruff B. Smokeless tobacco: A good alternative to smoking? ABC News Original Report. November 17, 2005. Available at: http://abcnews.go.com/WNT/QuitToLive/story?id=1322995.
- 9. Choi WS, Farkas AJ, Rosbrook B. Does advertising promote smokeless tobacco use among adolescent boys? Evidence from California. *Tob Control*. 1995;4(Supplement 1):S57–S63.
- 10. Tomar SL. Is use of smokeless tobacco a risk factor for cigarette smoking? The U.S. experience. *Nicotine Tob Res.* 2003;5(4):561–569.
- 11. U.S. Department of Health and Human Services, P.H.S., Office of the Surgeon General. The Health Consequences of Smoking: A Report of the Surgeon General. Washington, DC: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2004.

Genetics and Smoking

Caryn Lerman, Ph.D.

Despite progress made in the treatment of tobacco dependence, currently available treatments are effective for only a fraction of smokers. Although current guidelines recommend the use of nicotine patch as a first-line treatment for tobacco dependence, about 70–80% of smokers treated with the patch relapse to their former smoking practices in the long term. Bupropion has been shown to produce higher quit rates than nicotine replacement therapies (NRTs), yet the majority of smokers do not quit and remain abstinent. Thus, research is needed to identify subgroups of smokers for whom particular smoking cessation pharmacotherapies will have the strongest beneficial effects. These efforts may someday help practitioners to individualize smoking cessation treatment based on genotype, thereby maximizing its efficacy.

Abundant data from animal model and human twin studies have established that smoking persistence is, in part, heritable. The emerging field of pharmacogenetics has the potential to advance the science of nicotine dependence treatment by generating new knowledge about genetic factors that influence the efficacy of different pharmacotherapies for smoking cessation. The basic premise of this approach is that inherited differences in drug metabolism and drug targets have important effects on treatment toxicity and efficacy. This presentation provides a brief overview of evidence supporting the potential utility of a pharmacogenetic approach to smoking cessation treatment.

Nicotine is metabolized to cotinine, and then to 3'-hydroxcotinine (3-HC), predominantly by the liver enzyme *CYP2A6*. Genetic variation in *CYP2A6* predicts eigarette consumption and smoking persistence, consistent with the premise that faster inactivation and elimination of nicotine requires higher levels of smoking to maintain the desired levels of nicotine in the body. The 3-HC:cotinine ratio derived from eigarette smoking (a phenotypic measure of *CYP2A6* activity) predicts the effectiveness of transdermal nicotine at the end of treatment and at 6-month followup. In a recent trial, the likelihood of abstinence was reduced by almost 30% with each increasing quartile of metabolic ratio (i.e., faster metabolism of nicotine). 5

With regard to genetic variation in drug targets, nicotine stimulates release of dopamine from neurons in the ventral tegmental area, an action thought to underlie its rewarding effects. Therefore, investigations have examined response to these alternate forms of NRT in relation to genetic variation in the dopamine pathway. Results of a large pharmacogenetic trial in the United Kingdom revealed greater efficacy of the nicotine patch (vs. placebo) among smokers with dopamine D2 receptor (*DRD2*) Taq1A genotypes associated with lower receptor density. Likewise, a recent open-label NRT trial reported that smokers with a *DRD2* polymorphism associated with lower transcriptional efficiency have significantly higher quit rates with the nicotine patch and nicotine nasal spray. Studies have also examined the role of the Catechol-O-Methyltransferase (COMT) Val/Met functional polymorphism in response to NRT, as COMT is the primary enzyme involved in the degradation and inactivation of dopamine. Female smokers with the Met/Met genotype have a significantly higher probability of abstinence with either nicotine nasal spray or nicotine patch.

Other pharmacogenetic analyses of NRT have focused on the role of the functional A118G variant in the mu-opioid receptor (OPRM1) gene. The mu-opioid receptor is the primary site of action for the rewarding effects of the endogenous opioid peptide, beta-endorphin, that is released following acute and short-term nicotine administration. Smokers carrying the low activity OPRM1 Asp40 variant are significantly more likely than those homozygous for the Asn40 variant to be abstinent following nicotine patch treatment (quit rates of 52% vs. 33% for Asp40 and Asn40 groups, respectively, odds ratio [OR]=2.4).

Pharmacogenetic analyses from bupropion trials have focused on drug metabolizing enzymes as well as polymorphisms in genes in the dopamine pathway. An initial report focused on the *CYP2B6* gene, which has been implicated in bupropion kinetics, as well as in brain metabolism of nicotine. In this placebo-controlled randomized trial, smokers with a decreased activity variant of *CYP2B6* (slower metabolizers) reported greater increases in cravings for cigarettes following the target quit date and had significantly higher relapse rates. ¹⁰ These effects were modified by a significant gender × genotype × treatment interaction, suggesting increased efficacy of bupropion among female smokers with a genetic liability to relapse.

Genetic variation in the dopamine pathway is also a plausible target for pharmacogenetic studies of response to bupropion treatment, since inhibition of dopamine reuptake is one putative mechanism for the beneficial effects of bupropion. Two pharmacogenetic trials suggest that smokers with the A2 allele (normal receptor density) for the *DRD2* gene (Taq1A variant) may have a better response to bupropion. Further, a third trial reported an interaction between *DRD2*–141C *Ins/Del* genotype and bupropion treatment indicating a more favorable response to bupropion among smokers with genotypes associated with increased transcriptional efficiency. Studies in this area suggest that smokers with genotypes associated with greater receptor density may have more D2 receptors available to bind dopamine, yielding a more rewarding experience of the nicotine-induced dopamine release. Blockade of dopamine reuptake by bupropion may be more effective in promoting abstinence in these genotype subgroups due to greater ability to bind dopamine.

Pharmacogenetics research on nicotine dependence treatment is in the very early stages. To date, only two pharmacogenetic trials of NRT and three pharmacogenetic trials of bupropion have been conducted. Although the results of these initial studies are promising, there are several stages of research needed prior to translation to clinical practice. First, initial results from these trials must be validated in independent investigations. Large-scale pharmacogenetic trials (e.g., 5,000 participants) are necessary to provide adequate power to conduct analysis of multiple genetic effects on treatment response simultaneously and to stratify for ethnic variation. Additional research should be conducted to examine the benefits, risks, and challenges of conveying genetic information about smoking predisposition to the patient, clinicians, and the public. Economic analyses of the cost-effectiveness of using genotype to tailor smoking treatment are also necessary. Clinical and ethical issues arising from the clinical use of genotype data in the smoking context must also be addressed, as many of the genetic variants that predict response to medications for smoking cessation may also portend an increased susceptibility to other drug addictions and psychiatric comorbidities. If these issues are addressed in parallel with the scientific research in pharmacogenetics, this work has the potential to improve public health by increasing the efficacy of smoking cessation treatment.

- 1. Sullivan PF, Kendler KS. The genetic epidemiology of smoking. *Nicotine Tob Res.* 1999;1 Suppl 2:S51–S57; discussion S69–S70.
- 2. Munafo MR, Shields AE, Berrettini WH, Patterson F, Lerman C. Pharmacogenetics and nicotine addiction treatment. *Pharmacogenomics*. 2005;6(3):211–223.
- 3. Evans WE, Relling MV. Moving towards individualized medicine with pharmacogenomics. *Nature*. 2004;429(6990):464–468.
- 4. Malaiyandi V, Sellers EM, Tyndale RF. Implications of CYP2A6 genetic variation for smoking behaviors and nicotine dependence. *Clin Pharmacol Ther*. 2005;77(3):145–158.
- 5. Lerman C, Tyndale RF, Patterson F, Wileyto EP, Pinto A, Benowitz N. Nicotine metabolite ratio predicts the efficacy of transdermal nicotine for smoking cessation. *Clin Pharmacol Ther.* In press.
- 6. Johnstone EC, Yudkin PL, Hey K, et al. Genetic variation in dopaminergic pathways and short-term effectiveness of the nicotine patch. *Pharmacogenetics*. 2004;14(2):83–90.
- 7. Lerman C, Jepson C, Wileyto EP, et al. Role of functional genetic variation in the dopamine D2 receptor (DRD2) in response to bupropion and nicotine replacement therapy for tobacco dependence: Results of two randomized clinical trials. *Neuropsychopharmacology*. 2006;31(1):231–242.
- 8. Colilla S, Lerman C, Shields P, et al. Association of Catechol-O-Methyltransferase functional variant with smoking cessation in two independent studies of women. *Pharmacogenetics & Genomics*. 2005;15(6):393–398.
- 9. Lerman C, Wileyto EP, Patterson F, et al. The functional mu opioid receptor (OPRM1) Asn40Asp variant predicts short-term response to nicotine replacement therapy in a clinical trial. *Pharmacogenomics Journal*. 2004;4(3):184–192.
- 10. Lerman C, Shields PG, Wileyto EP, et al. Pharmacogenetic investigation of smoking cessation treatment. *Pharmacogenetics*. 2002;12(8):627–634.
- 11. David SP, Brown RA, Papandonatos GD, et al. Pharmacogenetic clinical trial of sustained-release bupropion for smoking cessation. *Nicotine & Tobacco Research*. In press.
- 12. Swan GE, Valdes AM, Ring HZ, et al. Dopamine receptor DRD2 genotype and smoking cessation outcome following treatment with bupropion SR. *Pharmacogenomics J.* 2005;5(1):21–29.

Treatment and Prevention of Tobacco Dependence in Individuals With Mental Health and Substance Abuse Disorders

Sharon M. Hall, Ph.D.

Epidemiological studies have shown that 41% of individuals with current Axis I mental disorders smoke cigarettes. In the United States, 44.3% of cigarettes consumed are smoked by people with Axis I Psychiatric Disorders. Individuals with Axis I and II disorders are also more likely to be nicotine dependent when compared to the general population.²

Treatment

This disproportionate representation extends to individuals in treatment for mental health and substance abuse disorders. Multiple studies show extremely high rates of smoking among drug treatment patients, ranging from 74% to 88%.³ Rates among individuals engaged in mental health treatment vary depending on disorder, but are higher than in the general population.^{4–7}

Given this overrepresentation of smokers among those with psychiatric illnesses, it is lamentable that the treatment of nicotine dependence in both substance-abusing and psychiatric populations is in its infancy, and the research is sparse. This is especially acute in psychiatric settings. There are multiple reasons for both the failure to offer treatment and the related lack of outcome data. In the substance abuse field, barriers includes staff resistance, lack of training, and concerns about immediate critical needs such as housing and safety. Also, historically, there has been a belief in the substance abuse community that the preeminent task during recovery is abstinence from nonnicotinic drugs or alcohol, and that other health-related tasks should be delayed until that task is achieved. Finally, many drug treatment programs are poorly funded, and treatment of tobacco dependence has not been included in their mandate.

Some of the same barriers exist in mental health settings, including lack of training of providers, concern that other immediate needs take precedence, and reimbursement issues. In addition, there is concern that smoking cessation may cause a re-occurrence of the psychiatric disorder. Another important factor may be the relatively low status of tobacco dependence treatment among mental health policymakers and providers. 8,9,14

Despite the barriers to completing treatment studies and the paucity of treatment outcome data on the treatment of tobacco dependence in substance abuse settings, those available are promising. A recent meta-analysis has indicated that not only is smoking cessation effective in the treatment of tobacco dependence, but there also is no evidence that it increases relapse to other addictions. In fact, this meta-analysis hints that there is suggestive evidence that abstinence from tobacco is related to abstinence from other drugs. ¹⁵ At least one well-done clinical trial has suggested that timing of the intervention may be important, however, and that, although patients are more likely to accept smoking treatment early in treatment for alcohol, they may be less successful in their abstinence from alcohol if they participate in early treatment. ¹⁶

Data on the treatment of tobacco dependence in substance abuse treatment are sparse—even more so in the treatment of tobacco dependence in mental disorders. The few clinical trials that exist suggest that interventions used in the treatment of the general population are effective with populations with mental disorders. Further, there is evidence that these interventions are especially effective if integrated into mental health services, as opposed to provided independently. Frequently, absolute abstinence rates have been lower than those observed in the general population, this is not always the case. It seems quite reasonable that differences may be found as a function of the mental health disorder studied. Finally, despite concern about the recurrence of psychiatric disorders, especially Major Depressive Disorder, during and after smoking cessation and multiple case studies, the two large-scale studies of the issue produced conflicting results.

Prevention

So far as we could find in preparing this abstract, there have been no studies of primary prevention for tobacco dependence for individuals with substance abuse or mental health disorders. In part, this may stem from the fact that many of these disorders develop concurrently with tobacco dependence or after the first use of nicotine. This is not the case with at least one disorder where high rates of cigarette smoking have been reported: Attention Deficit-Hyperactivity Disorder (ADHD).²² Nevertheless, so far as we could find, no prevention studies with this subpopulation have been reported.

Research on smoking cessation in the treatment of substance-abusing individuals and individuals with mental health disorders is very much in its infancy. Core questions, such as whether the interventions used in the general population perform in a similar fashion in these populations, have yet to be answered. Questions of timing, tailoring of the intervention to the specific problems of mental health and substance abuse patients, and the possible effects of cessation on mental health problems need study. Research on targeted prevention efforts is lacking.

- 1. Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. Smoking and mental illness: A population-based prevalence study. *JAMA*. Nov 22–29 2000;284(20):2606–2610.
- 2. Grant BG, Hasin DS, Chou P, Stinson FS, Dawson DA. Nicotine Dependence and Psychiatric Disorders in the United States. *Archives of General Psychiatry*. 1107–1115 2004;61(Nov).
- 3. Kalman D. Smoking cessation treatment for substance misusers in early recovery: A review of the literature and recommendations for practice. *Substance Use and Misuse*. 1998;33:2021–2047.
- 4. Acton GS, Prochaska JJ, Kaplan AS, Small T, Hall SM. Depression and stages of change for smoking in psychiatric outpatients. *Addictive Behaviors*. 2001;26(621–631).
- 5. Hughes JR. Comorbidity and smoking. *Nicotine & Tobacco Research*. 1999;1:S149–S152.

- 6. Prochaska JJ, Gill P, Hall SM. Treatment of tobacco use in an inpatient psychiatric setting. *Psychiatric Services*. 2004;55:1265–1270.
- 7. Hughes JR, Hatsukami DK, Mitchell JE, Dahlgren LA. Prevalence of smoking among psychiatric outpatients. *Am J Psychiatry*. 1986;143(8):993–997.
- 8. Hughes JR. Treating nicotine dependence in mental health settings. *Journal of Practical Psychiatry and Behavioral Health*. July 1997:250–254.
- 9. Hall SM. Commentary. In Isaacs S, ed. *VA in the Vanguard: Building on Success in Smoking Cessation*. San Francisco: UCSF School of Medicine; October 2005:31–40.
- 10. Bobo JK, Slade J, Hoffman A. Nicotine addiction counseling for chemically dependent patients. *Psychiatric Services*. 1995;46(945–947).
- 11. Hahn EJ, Warnick T, Plemmons S. Smoking cessation in drug treatment programs. *Journal of Addictive Diseases*. 1999;18:89–101.
- 12. Bobo JK, Gilchrist L. Urging the alcohol client to quit smoking cigarettes. *Addictive Behaviors*. 1983;8:297–305.
- 13. Glassman AH. Cigarette Smoking: Implications for Psychiatric Illness. *Am J Psychiatry*. April 1993 1993;150(4):546–553.
- 14. Himelhoch S, Daumit G. To whom to psychiatrists offer smoking-cessation counseling? *Am J Psychiatry*. 2003;160(12):2228–2230.
- 15. Prochaska JJ, Delucchi K, Hall SM. A meta-analysis of smoking cessation interventions with individuals in substance abuse treatment or recovery. *Journal of Consulting and Clinical Psychology*. 2004;72(6):1144–1156.
- 16. Joseph AM, Willenbring ML, Nugent SM, Nelson DB. A randomized trial of concurrent versus delayed smoking intervention for patients in alcohol dependence treatment. *Journal of Studies on Alcohol*. 2004;65(6):681–692.
- 17. McFall M, Saxon AJ, Thompson CE, et al. Improving the rates of quitting smoking for veterans with posttraumatic stress disorder. *Am J Psychiatry*. 2005;162(7):1311–1319.
- 18. Hall SM, Tsoh JY, Prochaska JJ, et al. Treatment of depressed mental health outpatients for cigarette smoking: A randomized clinical trial. *American Journal of Public Health*. In press.
- 19. George TP, Vessicchio JC, Termine A, et al. A placebo controlled trial of bupropion for smoking cessation in schizophrenia. *Biological Psychiatry*. 2002;52(1):53–61.
- 20. Tsoh J, Humfleet G, Munoz RF, Reus VI, Hartz DT, Hall SM. Development of major depression following smoking cessation treatment. *Am J Psychiatry*. 2000;157(3):368–374.

- 21. Glassman AH, Covey LS, Stetner F, Rivelli S. Smoking cessation and the course of major depression: a follow-up study. *Lancet*. June 2001;357:1129–1932.
- 22. Humfleet GL, Prochaska JJ, Mengis M, et al. Preliminary evidence of the association between the history of childhood attention-deficit/hyperactivity disorder and smoking treatment failure. *Nicotine and Tobacco Research*. June 2005;3:453–460.

Chronic Disease and Co-Occurring Risk

Ellen R. Gritz, Ph.D.

For many years, smoking cessation efforts have been aimed primarily at healthy persons in the general population, as a form of primary prevention. Individuals at elevated risk for certain diseases, or who had already experienced an illness—for example, cardiovascular disease (CVD) or early chronic obstructive pulmonary disease (COPD)—were targeted as well. However, smoking and tobacco cessation among persons with other types of severe chronic illness has received less attention from healthcare practitioners and researchers. This lack of emphasis may be attributable to the focus on acute treatment of a life-threatening illness, a potential or actual high mortality rate, lack of cessation treatment tailored to these populations, and the perceived lack of relevance of smoking to treatment outcome and survival. Two major examples are cancer and human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS), both of which have seen increased survival over the last decade.

This presentation will focus on: (1) a brief overview of the evidence linking continued smoking to treatment complications, disease progression and adverse outcomes, including reduced survival in major smoking-related diseases including CVD and COPD, and the benefits of cessation; (2) other conditions in which smoking has a serious adverse impact—pregnancy, diabetes, and asthma; (3) evidence linking smoking behavior with increased complications and adverse outcomes for cancer and HIV/AIDS; and (4) the interest in quitting and motivation of persons with these two latter diseases to stop smoking. Best practices for healthcare providers treating such patients will also be reviewed.

Cardiovascular Disease

Cardiovascular disease encompasses a number of diseases, including coronary heart disease (CHD)—the leading cause of myocardial infarctions (MI)—cerebrovascular disease (stroke), aortic aneurysm, and peripheral vascular disease (PVD). Burns¹ reviews a body of evidence relating to continued adverse effects of smoking after the onset of documented disease and concludes that, overall, continued smoking results in disease progression, recurrent events, and higher mortality, while smoking cessation reduces disease risks over time, depending on the specific condition. Smoking cessation after MI or angiographically documented coronary artery disease leads to significantly lower rates of re-infarction within 1 year, compared to continuing smokers; rates remain much lower over time. Among those with CHD, continued smoking was associated with an increased risk for sudden coronary death. Following coronary artery bypass surgery, continued smoking at 5 years is associated with increased risk for MI, angioplasty, and development of angina. Cessation of smoking following bypass surgery resulted in a 10-year survival of 84% versus 68% among continuing smokers.² In patients with PVD, continued smoking is associated with much lower rates of improvement with medical management alone compared to nonsmoking patients, higher rates of amputation compared to quitters, and higher rates of obstruction following surgery. The Cochrane Collaboration meta-analysis (20 studies) estimated the magnitude of risk reduction when a patient with CHD stops smoking; it reported a pooled crude relative risk (RR) of 0.64 (0.58–0.71), representing a 36% reduction in all-cause mortality in ex-smokers compared to continuing smokers.³

Chronic Obstructive Pulmonary Disease

COPD describes a range of conditions involving damage to the lungs, which arises primarily from the inhalation of tobacco smoke: chronic obstructive bronchitis, emphysema, and chronic airflow obstruction. Permanent cessation of smoking by individuals with early COPD (mild to moderate airway obstruction) dramatically reduces the progression to clinically serious lung disease as found in the Lung Health Study. Intervening once lung disease has become disabling results in a slowing in the rate of decline of lung function, but the benefits are more limited in terms of symptomatology. Mortality risk from COPD declines following smoking cessation compared to continued smoking, but this decline is smaller than for heart disease and lung cancer, and risk remains elevated in former smokers even after 20 years of abstinence.

Diabetes

Both cross-sectional and prospective studies have consistently shown higher risk for micro- and macrovascular disease, as well as premature mortality in diabetic patients who smoke. Among adults with diabetes, smoking is associated with increased death by CHD, diagnosis of coronary artery disease, stroke, nephropathy, and neuropathy. Damage and constriction of blood vessels by smoking can lead to exacerbation of foot ulceration, blood vessel disease, and lower extremity amputation. The increased cardiovascular burden of diabetes among patients who smoke needs to be emphasized by healthcare providers; advice to quit is delivered to only about half of the smokers with diabetes. Special challenges involve smoking initiation by adolescent diabetics and, among smoking adults, problems of weight management, negative affect and interventions during hospitalization.

Asthma

Maternal smoking during pregnancy and secondhand smoke exposure during childhood are associated with the onset of asthma and, in the latter case, its severity (as measured by frequency of attacks, number of emergency room visits, and risk of intubation). Reduction or, preferably, elimination of exposure to smoke in the home must be a major objective in the care of asthmatic children. 13–17

Reproductive Outcomes

The risks of smoking on fertility and during pregnancy are well known and documented; these include reduced fertility, spontaneous abortion, premature delivery and low birth weight, and increased fetal and infant mortality. Research suggests that 17 to 25% of low birth weight births could be prevented by eliminating smoking during pregnancy. 18,19

Cancer²⁰

Tobacco use, including exposure to secondhand smoke, has been implicated as a causal or contributory agent in an ever-expanding list of cancers, including lung, head and neck, pancreas, liver, kidney, ureter, urinary bladder, uterine cervix, and myeloid leukemia. Independent of the etiologic effects of tobacco carcinogens in numerous cancers, a growing literature also documents the direct and indirect adverse effects of smoking on oncologic treatment efficacy (short- and long-term outcomes), toxicity and morbidity, quality of life

(QOL), recurrence, second primary tumors (SPT), and survival time. Despite the critical relevance of smoking to cancer outcomes, most oncology clinical trials do not collect data on smoking history and status unless the malignancy is widely acknowledged as smoking-related. Smoking history and status should be systematically collected as core data in all oncology clinical trials, from diagnosis and registration, throughout treatment and followup, to long-term survival or death. A set of items that are also useful in clinical practice is suggested for inclusion in clinical trial data sets. A much greater emphasis on smoking cessation for oncology patients is called for, and a treatment intervention literature is growing steadily. ^{21–23}

Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome (HIV/AIDS)

Highly active antiretroviral therapy (HAART) has resulted in a dramatic decrease in AIDS-related mortality. Given the current trends in incidence and mortality, the number of people living with HIV/AIDS will continue to grow. The prevalence of smoking in HIV/AIDS populations is far above the national average (50–70% vs. 21%). HIV/AIDS patients who smoke are at elevated risk for numerous adverse outcomes (e.g., elevated mortality, cardiovascular disease, pulmonary infections, cancers, oral conditions and decreased QOL) compared to HIV-positive non-smokers. HIV-associated malignancies, such as anal and cervical cancer, are also observed more frequently among patients with HIV/AIDS who smoke. Risk factors for CVD are adverse side effects of prolonged antiretroviral therapy, and the number of CVD cases observed in this patient population has been growing in recent years. Thus, it is vital that other major risk factors for CVD, particularly behavioral modifiable risk factors such as smoking, be targeted for intervention. To date, few efforts have been made to target HIV/AIDS patients for smoking cessation. A randomized controlled trial of a smoking cessation intervention tailored to a low-income, multiethnic population of individuals living with HIV/AIDS will be described. HIV/AIDS will be described.

Smoking Cessation Treatment for Persons Living with Chronic Disease

In the past, fatalistic assumptions about cancer and HIV/AIDS outcomes may have led both patients and providers to underemphasize the benefits of smoking cessation. However, several studies have documented interest in and motivation to quit despite disease- and treatment-related barriers and other psychosocial/socioeconomic disadvantages. ^{21,24,25,30–33} Guidelines for treating smoking in persons with chronic disease, including the need for specific targeting and tailoring, and the use of appropriate pharmacotherapy, will be discussed.

- 1. Burns DM. Epidemiology of smoking-induced cardiovascular disease. *Prog Cardiovasc Dis.* 2003;46:11–29.
- 2. Cavender JB, Rogers WJ, Fisher LD, Gersh BJ, Coggin CJ, Myers WO. Effects of smoking on survival and morbidity in patients randomized to medical or surgical therapy in the Coronary Artery Surgery Study (CASS): 10-year follow-up. CASS Investigators. *J Am Coll Cardiol*. 1992;20:287–294.

- 3. Critchley J, Capewell S. Smoking cessation for the secondary prevention of coronary heart disease. *Cochrane Database Sys Rev.* 2004;1:CD003041.
- 4. Anthonsien NR. Lessons from the Lung Health Study. *Proc Am Thorac Soc.* 2004;1:143–145.
- 5. Burns D. Chronic obstructive pulmonary disease. In: Boyle B, et al., eds. *Tobacco and Public Health: Science and Policy*. Oxford: Oxford University Press; 2004.
- 6. Haire-Joshu D, Glasgow RE, Tibbs TL. Smoking and diabetes. *Diabetes Care*. 1999;22:1887–1898.
- 7. American Diabetes Association. *Smoking* [American Diabetes Association Web site]. Available at: http://www.diabetes.org/type-1-diabetes/smoking.jsp. Accessed April 3, 2006.
- 8. American Diabetes Association. Smoking and diabetes. *Diabetes Care*. 2002;25:S80–S81.
- 9. Malarcher A, Ford E, Nelson D. Trends in cigarette smoking and physicians' advice to quit smoking among people with diabetes living in the U.S. *Diabetes Care*. 1995;18:694–697.
- 10. Mannino DM, Homa DM, Redd SC. Involuntary smoking and asthma severity in children: data from the Third National Health and Nutrition Examination Survey. *Chest*. 2002;122:409–415.
- 11. Arruda LK, Sole D, Baena-Cagnani CE, Naspitz CK. Risk factors for asthma and atopy. *Curr Opin Allergy Clin Immunol.* 2005;5:153–159.
- 12. DiFranza JR, Aligne CA, Weitzman M. Prenatal and postnatal environmental tobacco smoke exposure and children's health. *Pediatrics*. 2004;113:1007–1015.
- 13. Wahlgren DR, Hovell MF, Meltzer SB, Hofstetter CR, Zakarian JM. Reduction of environmental tobacco smoke exposure in asthmatic children. A 2-year follow-up. *Chest*. 1997;111:81–88.
- 14. Wahlgren DR, Hovell MF, Meltzer EO, Meltzer SB. Involuntary smoking and asthma. *Curr Opin Pulm Med.* 2000;2000:1.
- 15. Morkjaroenpong V, Rand CS, Butz AM, et al. Environmental tobacco smoke exposure and nocturnal symptoms among inner-city children with asthma. *J Allergy Clin Immunol*. 2002; 110:147–153.
- 16. Winickoff JP, McMillen RC, Carroll BC, et al. Addressing parental smoking in pediatrics and family practice: a national survey of parents. *Pediatrics*. 2003;112:1146–1151.
- 17. Klerman L. Protecting children: reducing their environmental tobacco smoke exposure. *Nicotine Tob Res.* 2004;6:S239–253.

- 18. U.S. Dept. of Health and Human Services, Public Health Service, Office of the Surgeon General. *Women and Smoking: a Report of the Surgeon General*. Washington, DC: U.S. G.P.O.; 2001. Available at: http://www.surgeongeneral.gov/library.
- 19. Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion. *Demographics and Health Effects* [Treatobacco.net Web site]. Available at: http://www.treatobacco.net/demographics/demographics.cfm. Accessed April 4, 2006.
- 20. Gritz ER, Dresler C, Sarna L. Smoking, the missing drug interaction in oncology clinical trials: ignoring the obvious. *Cancer Epidemiol Biomarkers Prev.* 2005;14:2287–2293.
- 21. Gritz ER, Fingeret MC, Vidrine DJ, Lazev AB, Mehta NV, Reece GP. Successes and failures of the teachable moment. Smoking cessation in cancer patients. *Cancer*. 2006;106:17–27.
- 22. Gritz ER, Vidrine DJ, Lazev AB. Smoking cessation in cancer patients: never too late to quit. In: Green CW, Given B, Champion VL, et al., eds. *Evidence-based Cancer Care and Prevention: Behavioral Interventions*. New York: Springer Publishing Co; 2003:107–140.
- 23. Cox LS, Africano NL, Tercyak KP, Taylor KL. Nicotine dependence treatment for patients with cancer: review and recommendations. *Cancer*. 2003;98:632–644.
- 24. Gritz ER, Vidrine DJ, Lazev AB, Amick BC 3rd, Arduino RC. Smoking behavior in a low-income multiethnic HIV/AIDS population. *Nicotine Tob Res.* 2004;6:71–77.
- 25. Niaura R, Shadel WG, Morrow K, Tashima K, Flanigan T, Abrams DB. Human immunodeficiency virus infection, AIDS, and smoking cessation: the time is now. *Clin Infect Dis.* 2000;31:808–812.
- 26. Crothers K, Griffith TA, McGinnis KA, et al. The impact of cigarette smoking on mortality, quality of life, and comorbid illness among HIV-positive veterans. *J Gen Intern Med*. 2005;20:1142–1145.
- 27. Patel N, Talwar A, Reichert VC, Brady T, Jain M, Kaplan MH. Tobacco and HIV. *Clin Occup Environ Med.* 2006;5:193–207.
- 28. Bozkurt B. Cardiovascular toxicity with highly active antiretroviral therapy: review of clinical studies. *Cardiovasc Toxicol*. 2004;4:243–260.
- 29. Sax PE, Kumar P. Tolerability and safety of HIV protease inhibitors in adults. *J Acquir Immune Defic Syndr*. 2004;37:1111–1124.
- 30. Lazev AB, Vidrine DJ, Arduino RC, et al. Increasing access to smoking cessation treatment in a low-income, HIV-positive population: the feasibility of using cellular telephones. *Nicotine Tob Res.* 2004;6:281–286.
- 31. Vidrine DJ, Arduino RC, Lazev AB, Gritz, ER. A randomized trial of a proactive cellular telephone intervention for smokers living with HIV/AIDS. *AIDS*. 2006;20:253–260.

- 32. Gritz ER, Carr CR, Rapkin D, et al. Predictors of long-term smoking cessation in head and neck cancer patients. *Cancer Epidemiology, Biomarkers and Prevention*. 1993;2:261–270.
- 33. Burkhalter JE, Springer CM, Chhabra R, Ostroff JS, Rapkin BD. Tobacco use and readiness to quit smoking in low-income HIV-infected persons. *Nicotine Tob Res.* 2005;7:511–522.

Evidence-Based Practice Center Presentation: Effective Tobacco Cessation Strategies for Individuals With Co-Occurring Morbidities and Risk Behaviors

Leah M. Ranney, Ph.D., M.A.

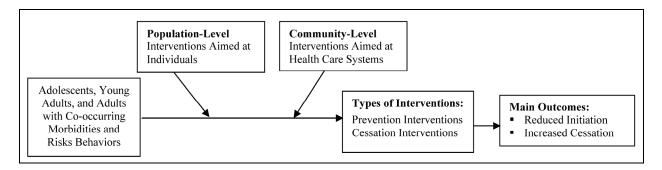
Background

Nicotine dependence among persons with co-occurring morbidities and risk behaviors occurs at an alarming rate.^{1,2} We define a person with co-occurring disorders as one who has a psychiatric condition and a nicotine addiction. Psychiatric conditions include schizophrenia, depression, anxiety, personality disorders, post-traumatic stress disorder, attention deficit disorder, eating disorders, and disruptive behavioral disorders. Risk behaviors trigger or exacerbate tobacco use such as alcohol and other chemical dependencies.

Individuals with psychiatric conditions and nicotine addiction are twice as likely to smoke as the general population and to smoke more heavily than other smokers. As many as 30% of smokers seeking cessation treatment have a history of depression. Smoking rates for alcohol and drug users are well above the average population, exceeding 70%. The risk of death is significantly higher for individuals with concurrent addictions of alcohol and nicotine than for individuals who abuse only alcohol or tobacco. ^{3,4}

Using the analytic framework in figure 1, we undertook a systematic review of the evidence for effective smoking prevention and cessation treatments for people with co-occurring morbidities and risk behaviors ("key question 5" in the full evidence report).

Figure 1. Cessation Strategies for Persons with Co-occurring Illness and Risk Behaviors: Analytic Framework



Methods

We searched standard electronic databases (MEDLINE®, the Cumulative Index to Nursing and Applied Health [CINAHL], Cochrane Collaboration libraries, Cochrane Clinical Trials Register, Psychological Abstracts, and Sociological Abstracts) between January 1, 1980 and June 10, 2005 using Medical Subject Headings as search terms when available or key words

when appropriate. We limited our review to (1) human studies conducted in developed countries and published in English; (2) studies with participants ages 13 and older, of both sexes, and diverse racial and ethnic populations; (3) randomized controlled trials (RCTs) with 30 or more individuals; (4) observational studies and other trials with 100 or more individuals; and (5) studies with a minimum follow-up period of 6 months, with or without comparison groups. We excluded editorials, letters, and commentaries; articles that did not report outcomes related to our key questions; and studies that did not provide sufficient information to be abstracted.

We reviewed all eligible studies, entered relevant data into evidence tables, and summarized them by descriptive methods. From our review of 639 abstracts, four addressed smoking cessation interventions for people with co-occurring morbidities and risk behaviors. We found no studies on prevention as a comorbid population by definition is already smoking.

Results: Psychiatric Populations

Approaches to increase quit rates among persons with psychiatric conditions include medications, educational strategies, and cognitive behavior modification. Although psychiatric populations had lower smoking cessation rates than nonpsychiatric populations, in the absence of relevant RCTs on smoking cessation for populations with psychiatric comorbidities, experts agree that clinicians should use smoking cessation treatments recommended for the general population such as pharmacotherapies and counseling. Prochaska et al. reported that multimodal strategies using nicotine replacement therapy in conjunction with psychosocial intervention strategies are effective in treating alcohol and other chemical substance users for tobacco addiction. Such interventions had positive short-term, but not long-term, effects. However, smoking cessation interventions did not interfere with recovery from chemical dependency, a finding consistent with recommendations from other reviews. Page 2.3

Among the four studies that addressed smoking cessation for people with co-occurring morbidities and risk behaviors, two evaluated smoking cessation for persons with psychiatric conditions. Hitsman et al. hypothesized that smokers with greater depressive symptoms would be more likely to achieve abstinence when receiving fluoxetine combined with cognitive behavioral therapy (CBT) than when receiving a placebo and CBT.⁵ Participants treated with fluoxetine had a higher likelihood of abstinence as compared with participants treated with placebo. At 3 months, fluoxetine benefited smokers with higher initial levels of depression. Conversely, placebo-treated participants with increasing depression scores were associated with decreasing likelihood of abstinence.⁵ Another trial focused on adolescent smokers ages 13–17 who had been hospitalized for psychiatric and substance use problems;⁶ neither motivational interviewing nor brief-advice tobacco cessation interventions was effective for this population.

Smoking cessation is challenging in populations with psychiatric disorders.¹ Evidence is insufficient and inconsistent about smoking cessation interventions in populations with psychiatric disorders.^{5,6} Thus, the gap in this evidence base for these populations remains significant.

Results: Alcohol and Substance Abuse

Two studies investigated smoking cessation among alcohol and substance abusers. ^{7,8} Covey et al. examined the influence of a history of alcoholism or major depression on smoking cessation rates in a 10-week RCT of clonidine on smokers who received individual behavioral counseling, or a placebo for 10 weeks in place of clonidine. ⁷ Rates of smoking cessation did not differ significantly between alcoholic and nonalcoholic participants. Male smokers who had both alcoholism and major depression demonstrated a severely impaired ability to stop smoking compared with those having alcoholism but not major depression. The results indicated equivalent smoking cessation rates for recovering alcoholics as compared with rates for the general population, but findings suggest smoking cessation strategies may need to be tailored for smokers with a history of alcoholism and diagnosis of depression.

Joseph evaluated the feasibility of a smoke-free policy and a nicotine treatment program implemented in a drug and alcohol treatment hospital. At 1-year follow-up, the number of patients who quit was greater among those admitted to the hospital after the start of the smoke-free and nicotine treatment policy than among patients admitted to the hospital before the policy changes. There was no effect on non-nicotine substance use.

We could not determine the effectiveness of smoking cessation interventions in populations with alcohol or substance abuse addictions. Two studies reported significant effects for smoking cessation without affecting abstinence for other addictive substances, ^{7,8} but abstinence was not maintained over time. ⁷ The studies supported findings from past reviews about the positive short-term effects of such interventions; ² however, the body of evidence is insufficient to point to further recommendations.

Conclusions and Need for Future Research

How best to approach smoking cessation treatment in populations with co-occurring morbidity and risk behaviors is controversial; successfully accomplishing therapy may require tailored smoking cessation treatments.^{2,7} Comprehensive research on concurrent treatment for smoking cessation and chemical dependency is warranted in light of recent data indicating no adverse effects on sobriety or other drug use.^{2,8} Investigators need to explain the interaction between depression and impaired ability to stop smoking among people diagnosed with clinical depression and among alcoholics or people with a history of alcoholism. Because populations with these additional ailments and problem behaviors smoke at higher rates than others,^{1,2} special attention should be directed toward treating nicotine addiction concurrently with recovery and management of other medical issues. Finally, barriers to smoking cessation treatment in patients with other health problems, such as contraindications of pharmacotherapy, and the validity of concerns on the part of clinicians about hindering sobriety, should also be investigated.

- 1. el-Guebaly N, Cathcart J, Currie S, et al. Smoking cessation approaches for persons with mental illness or addictive disorders. *Psychiatr Serv.* 2002;53(9):1166–1170.
- 2. Prochaska JJ, Delucchi K, Hall SM. A meta-analysis of smoking cessation interventions with individuals in substance abuse treatment or recovery. *J Consult Clin Psychol*. 2004;72(6):1144–1156.
- 3. Fiore MC, Bailey WC, Cohen SJ. Treating Tobacco Use and Dependence, Clinical Practice Guideline. Rockville, Md: U.S. Department of Health and Human Services, Public Health Service; 2000.
- 4. Cooney JL, Cooney NL, Pilkey DT, et al. Effects of nicotine deprivation on urges to drink and smoke in alcoholic smokers. *Addiction*. 2003;98(7):913–921.
- 5. Hitsman B, Pingitore R, Spring B, et al. Antidepressant pharmacotherapy helps some cigarette smokers more than others. *J Consult Clin Psychol*. 1999;67(4):547–554.
- 6. Brown RA, Ramsey SE, Strong DR, et al. Effects of motivational interviewing on smoking cessation in adolescents with psychiatric disorders. *Tob Control*. 2003;12 Suppl 4:IV3–IV10.
- 7. Covey LS, Glassman AH, Stetner F, et al. Effect of history of alcoholism or major depression on smoking cessation. *Am J Psychiatry*. 1993;150(10):1546–1547.
- 8. Joseph A. Nicotine treatment at the Drug Dependency Program of the Minneapolis VA Medical Center. A researcher's perspective. *J Subst Abuse Treat*. 1993;10(2):147–152.