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22

A Socioecological Approach to Addressing Tobacco-Related Health Disparities

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Contents

Figures and Tables

Figures

Tables

A Socioecological Approach to Addressing Tobacco-Related Health Disparities

Foreword

Use of tobacco products remains the leading preventable cause of death and disability for all population groups in the United States. The special effect of tobacco use on minority health and health disparities has received moderate attention over the past 30 years. National Cancer Institute (NCI)–funded programs have led many of these research efforts, and the Master Settlement Agreement energized subsequent public health mobilization efforts. This monograph is a comprehensive report covering cutting edge and state-of-the-art summaries of research on tobacco-related health disparities from the perspectives of epidemiology, individual behavior, biology, cultural context, and societal structures. This multilevel approach reflects the appropriate methodology to address the science of minority health and health disparities research and creates a foundation for future topics that the National Institute of Minority Health and Health Disparities will focus on. In consideration of advancing the field and adding emphasis to specific issues, I will comment on five areas.

The success of tobacco control in the United States over the past 50 years is unprecedented. Smoking rates have been decreased by more than 50% among men, and cardiovascular mortality has decreased across all populations by an even greater proportion. Reductions in secondhand smoke exposure have been found even when using the most sensitive measures of detectable cotinine in children under 5 years, although further reductions in exposure are needed, especially among African Americans and people living in poverty.[1] Despite this remarkable progress, tobacco smoking has been causally linked to about 4 out of 5 lung cancer deaths in the United States.[2] Fifty years after the landmark Surgeon General's report *Smoking and Health* of 1964, the 2014 Surgeon General's report stated that in the United States 83.7% of lung cancer deaths among men and 80.7% of those among women were attributed to tobacco smoking.[3] There is potential to further decrease the tobacco epidemic through implementation of evidence-based interventions to prevent uptake and promote cessation. A complementary proposal to require a gradual decrease in nicotine content of manufactured cigarettes over a decade would likely lead to even less tobacco dependence and lower overall use.[4] Indeed, on July 28, 2017, Food and Drug Administration Commissioner Dr. Scott Gottlieb announced that the agency will take a comprehensive approach to regulating nicotine, including an exploration of reducing nicotine in combustible cigarettes to render them minimally or non-addictive.[5]

The approach to smoking cessation for most of the past 30 years has been designed around the nicotine addiction paradigm. However, as has been well documented, nearly half of racial/ethnic minority smokers are either non-daily smokers or very light smokers (NDVL) who consume fewer than 5 cigarettes per day.[6] The addiction paradigm does not apply to this increasingly prevalent pattern of smoking because these smokers are not dependent on nicotine and do not have classic withdrawal symptoms when they try to quit. The research community has failed to focus on the challenge of how to assist non-daily and very light smokers in quitting, and by doing so, has ignored the most prevalent smoking behavior pattern of minority populations. In fact, eligibility criteria for most smoking cessation trials have included smoking 10 or more cigarettes per day, thus systematically avoiding empirical evidence on what intervention components may work in NDVL smokers. One possible approach would incorporate the availability of underused evidence-based cessation interventions such as quitline advice with clinician referrals and the electronic medical record. Clinician educational interventions have had limited but tangible benefits in promoting cessation using strategies based on the stages of change model and prescribing medication adjuncts.[7] Referral to a quitline through an electronic consultation platform

is now feasible and would continue to allow clinicians to motivate, advise, and assist with medication. Given that most smokers visit a clinician at least yearly, this approach would potentially expand cessation efforts to reach underserved and minority populations.

The immigrant paradox continues to present a perplexing observation that most scientists try to explain by endorsing the concept that as immigrants acculturate, behaviors will change and disease rates will go up. Among Asian and Latino immigrants to the United States, increasing acculturation among women is strongly associated with greater use of tobacco, although the patterns are either absent or reversed among men. Despite this, and the fact that over half of Latinos were born in the United States, overall smoking rates among Latina and Asian women are below 10%.[8] Although overall smoking rates are lower for both Latinos and Asians, much higher smoking rates have been found in some demographic subgroups, such as Cuban and Puerto Rican men and women and Vietnamese men. In considering the influence of acculturation on behavior, scientists need to take socioeconomic status into account in an integral way. Acculturation is not a linear process; it often results in a bicultural individual and is strongly influenced by the social class background of the immigrant family and the change in status and social mobility they experience in the United States.[9] This complex interaction has not been well studied and will require greater attention when evaluating tobacco-related health disparities.

Much discussion in the past has focused on the relative importance of race/ethnicity and social class in influencing health outcomes. Tobacco use behavior is an excellent example of how these factors interact, how they explain mutually independent variance and assist scientists and public health leaders in determining approaches. In tobacco-related health disparities, some demographic groups stand out as needing special emphasis in the future. First, people with co-incident chronic and severe mental disorders (SMD) smoke at exceedingly high rates,[10] and only recently have programs been developed to provide greater cessation assistance. Similarly, individuals with other substance use problems have excess smoking rates, and like those with SMD, suffer from societal marginalization and stigmatization that affect their quantity and quality of life. Second, the social class gradient in smoking behavior is quite striking as measured by smoking rates that approach 40% among persons with 9 to 11 years of education or even among those with general education diplomas (GEDs), compared to less than 5% among college graduates.[8] This disparity cuts across racial/ethnic groups but is most accentuated among poor whites. Finally, sexual and gender minorities (SGM) have higher smoking rates,[11] suffer from structural discrimination, and have not been well studied for long-term health outcomes; only recently have public health researchers begun to abandon the “Don’t ask, don’t know” mantra.

My last comment is to reflect on the importance of multilevel approaches that incorporate biological pathways. There is unequivocal evidence of the causal effect of tobacco smoking on lung cancer, even if not fully quantified in all population groups. The incidence of lung cancer does not completely mirror smoking behavior even after accounting for at least a 10-year lag time. An observation made in the Multi-Ethnic Cohort Study highlights the unknown factors in this causal pathway.[12] In that observational study of African Americans, Native Hawaiians, whites, Latinos, and Japanese participants, the relative risk of the 1,749 cases of lung cancer identified was calculated by level of cigarette smoking intensity. For a similar level of smoking, Latino, white, and Japanese participants had a 30% to 75% lower risk of lung cancer compared with African Americans and Native Hawaiians. It was not until a smoking intensity of 30 cigarettes per day was reached that the differences in relative risk became non-significant.[12] Multiple possible explanations may be considered, including greater use of mentholated brands by African Americans, nicotine metabolism differences influencing smoking behavior, genetic markers linked to ancestry that have not been discovered, gene–environment interactions that have not

been studied, and smoking topography. Although this is one smoking-related example, the underlying principle is that studying different racial/ethnic groups provides opportunities for scientific discovery that otherwise would not be available.

Minority health and health disparities research has been predominantly framed in a context of social disadvantage and social determinants of health. Without discounting these factors, this NCI monograph is an outstanding example of where the field needs to move to advance the science—that is, toward multilevel discovery that incorporates advances in behavioral, social, clinical, population, and biological sciences in addressing the determinants of health outcomes in minorities and other disparity populations. This tobacco-related health disparities monograph is an excellent illustration of this pathway.

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Abbreviations

Abbreviation/Acronym	Definition
Add Health	National Longitudinal Study of Adolescent to Adult Health
ASSIST	American Stop-Smoking Intervention Study
BRFSS	Behavioral Risk Factors Surveillance System
CARDIA Study	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
COPD	Chronic obstructive pulmonary disease
CPD	Number of cigarettes smoked per day
FDA	Food and Drug Administration
GED	General educational development diploma
HINTS	Health Information National Trends Survey
MSA	Master Settlement Agreement
MTF	Monitoring the Future study
NATS	National Adult Tobacco Survey
NCI	National Cancer Institute
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NIH	National Institutes of Health
NSDUH	National Survey on Drug Use and Health
NYTS	National Youth Tobacco Survey
PATH	Population Assessment of Tobacco and Health
POS	Point of sale
PRAMS	Pregnancy Risk Assessment Monitoring System
SAMHSA	Substance Abuse and Mental Health Services Administration
SEER	Surveillance, Epidemiology, and End Results program
SEM	Socioecological model
SES	Socioeconomic status
SHS	Secondhand smoke
TRHD	Tobacco-related health disparities
TUS-CPS	Tobacco Use Supplement to the Current Population Survey
YRBS	Youth Risk Behavior Survey

Section I
Overview and Epidemiology

Chapter 1
Introduction and Overview

Contents

Figure

Introduction

The 1964 Surgeon General's report, *Smoking and Health,* is now widely viewed as a transformative report that helped initiate concerted efforts to reduce tobacco use in the United States.[1,2] Decades of research and implementation of evidence-based measures have produced significant declines in cigarette smoking, reduced exposure to secondhand smoke (SHS), and reduced tobacco-related mortality.[3] For example, the overall prevalence of cigarette smoking among U.S. adults declined from 42% in 1965[3] to 15.1% in 2015.[4] Additionally, tobacco control efforts dating from 1964 are credited with averting an estimated 8 million premature deaths by 2012.[5]

However, progress in reducing tobacco use and related morbidity and mortality has not been equally distributed across population groups. Indeed, the 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*, concluded that "although cigarette smoking has declined significantly since 1964, very large disparities in tobacco use remain across groups defined by race, ethnicity, education level, and socioeconomic status (SES) and across regions of the country."[3,p.7] As of 2016, few groups have met the Healthy People 2020 objective of reducing adult cigarette smoking prevalence to 12.0%. Some racial/ethnic and other vulnerable population groups have made less progress toward meeting this objective than others,[6] and these population groups experience substantial disparities in smoking-related disease and death.

Tobacco use, particularly in the form of cigarette smoking, remains the leading preventable cause of death in the United States, causing nearly one-third of deaths from cancer.[7] Today, many of the major aggregate U.S. racial/ethnic groups, particularly African Americans or Blacks, American Indians and Alaska Natives, and Native Hawaiians and Other Pacific Islanders, continue to experience health disparities from the adverse effects of tobacco use and SHS exposure. Studies have also documented higher smoking prevalence among lesbian, gay, bisexual, and transgender (LGBT) populations.[8] As with racial/ethnic minorities, people who live in poverty or have low educational attainment, blue-collar and service workers, and other vulnerable groups continue to experience disproportionately greater adverse effects of tobacco use and SHS exposure.

The persistence of disparities in tobacco use and subsequent tobacco-related disease underscores the importance of focusing on understanding tobacco-related health disparities (TRHD). The goal of this NCI monograph, *A Socioecological Approach to Addressing Tobacco-Related Health Disparities,* is to synthesize the research literature on the many factors that influence and contribute to TRHD across the tobacco use continuum, and to provide guidance for future research studies.

Health Disparities

In 2002, Carter-Pokras and Baquet published what may be the first review of definitions of health disparities; these authors suggested that "a health disparity should be viewed as a chain of events signified by a difference in: (1) environment, (2) access to, utilization of, and quality of care, (3) health status, or (4) a particular health outcome that deserves scrutiny."[9,p.427] During the early 1990s, researchers, government agencies, and public health practitioners began referring to health differences in population groups in the United States as "health disparities,"[9] reflecting a focus on eliminating disparities at local, state, and national levels. Other authors have also contributed to our understanding of health disparities, health inequalities, and related concepts.[10–13]

As definitions of disparities have evolved in the scholarly literature, Healthy People, which delineates 10-year national objectives for improving the health of the U.S. population,[6] has also refined its definition of disparities and changed its goals in relation to them.[14] Healthy People 2000 established the goal of *reducing* health disparities,[15] which was expanded to *eliminating* health disparities in Healthy People 2010.[14] Healthy People 2020 includes addressing both *health equity* and *health disparity*:

1. Health equity: "attainment of the highest level of health for all people. Achieving health equity requires valuing everyone equally with focused and ongoing societal efforts to address avoidable inequalities, historical and contemporary injustices, and the elimination of health and health care disparities."[14]
2. Health disparity: "a particular type of health difference that is closely linked with social, economic, and/or environmental disadvantage. Health disparities adversely affect groups of people who have systematically experienced greater obstacles to health based on their racial or ethnic group; religion; socioeconomic status; gender; age; mental health; cognitive, sensory, or physical disability; sexual orientation or gender identity; geographic location; or other characteristics historically linked to discrimination or exclusion."[14]

The Healthy People 2020 goal combines both concepts: "to achieve health equity, eliminate disparities, and improve the health of all groups."[14]

History of Research on TRHD

Recognition of the importance of studying TRHD has grown over time. This section discusses major milestones in this effort: two landmark Surgeon General's reports, the first U.S. national conference devoted to this topic, and the Tobacco Research Network on Disparities, a research network funded by the National Cancer Institute (NCI) in partnership with the American Legacy Foundation (now known as the Truth Initiative).

The Surgeon General's Report on the Health Consequences of Smoking in the Workplace

The 1985 Surgeon General's report, *The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace*,[16] focused on the role of cigarette smoking and occupational exposures in the development of lung cancer and chronic lung disease. This report helped set the stage for more in-depth investigations of the relationship between social and occupational class and tobacco use, exposure to secondhand smoke, and disease outcomes. The report highlighted the intersection of racial disparities and occupational status, and drew several conclusions relevant to health disparities by race/ethnicity, sex, and employment, which include:

1. "Among men, a substantially higher percentage of blue-collar workers than white-collar workers currently smoke cigarettes. Operatives and kindred workers have the highest rate of current smoking (approaching 50 percent), with professional, technical, and kindred workers having the lowest rates of current smoking (approximately 26 percent)."[16,p.53]
2. "Blue-collar occupations have a lower percentage of former smokers than white-collar occupations; this difference is most pronounced among men. Among women, the pattern for homemakers closely parallels that of white-collar women."[16,p.55]

3. "Black workers have higher smoking rates than white workers, with black male blue-collar workers exhibiting the highest smoking rate. Black workers also have lower quit rates than white workers. In contrast, white workers of both sexes are more likely to be heavy smokers regardless of occupational category."[16,p.55]

One chapter of this Surgeon General's report highlighted research on workplace smoking intervention programs, concluding that they should be a major component of worksite-based health promotion efforts.

The Surgeon General's Report on Tobacco Use Among Racial/Ethnic Minority Groups

Although several previous Surgeon General's reports have addressed differences in tobacco use by various subgroups, the tobacco disease burden among racial and ethnic groups in the United States was the specific focus of the 1998 Surgeon General's report *Tobacco Use Among U.S. Racial/Ethnic Minority Groups: African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, Hispanics*.[17] This landmark report summarized information on risk factors and patterns of tobacco use as well as national and regional efforts to reduce tobacco use among four major racial and ethnic minority groups. The report reached five major conclusions:

1. "Cigarette smoking is a major cause of disease and death in each of the four population groups studied in this report. African Americans currently bear the greatest health burden. Differences in the magnitude of disease risk are directly related to differences in patterns of smoking."
2. "Tobacco use varies within and among racial/ethnic minority groups. Among adults, American Indians and Alaska Natives have the highest prevalence of tobacco use, and African American and Southeast Asian men also have a high prevalence of smoking. Asian American and Hispanic women have the lowest prevalence."
3. "Among adolescents, cigarette smoking prevalence increased in the 1990s among African Americans and Hispanics after several years of substantial decline among adolescents of all four racial/ethnic minority groups. This increase is particularly striking among African American youths, who had the greatest decline of the four groups during the 1970s and 1980s."
4. "No single factor determines patterns of tobacco use among racial/ethnic minority groups; these patterns are the result of complex interactions of multiple factors, such as socioeconomic status, cultural characteristics, acculturation, stress, biological elements, targeted advertising, price of tobacco products, and varying capacities of communities to mount effective tobacco control initiatives."
5. "Rigorous surveillance and prevention research are needed on the changing cultural, psychosocial, and environmental factors that influence tobacco use to improve our understanding of racial/ethnic smoking patterns and identify strategic tobacco control opportunities. The capacity of tobacco control efforts to keep pace with patterns of tobacco use and cessation depends on timely recognition of emerging prevalence and cessation patterns and the resulting development of appropriate community-based programs to address the factors involved."[17,p.6]

Recognizing the disproportionate burden of tobacco-related disease for the four major racial/ethnic groups, the 1998 report also concluded that "rates of tobacco-related cancers (other than lung cancer) vary widely among members of racial/ethnic groups, and they are particularly high among African American men."[17,p.185] The report also concluded that "levels of serum cotinine (a biomarker of tobacco exposure) are higher in African American smokers than in white smokers for similar levels of daily

cigarette consumption. Further research is needed to clarify the relationship between smoking practices and serum cotinine levels in U.S. racial/ethnic groups. Variables such as group-specific patterns of smoking behavior (e.g., number of puffs per cigarette, retention time of tobacco smoke in the lungs), rates of nicotine metabolism, and brand mentholation could be explored."[17,p.185]

Tobacco Use Among U.S. Racial/Ethnic Minority Groups was also the first Surgeon General's report to document the historical context of tobacco use for various groups. As the report describes:

- Blacks contributed to the British and American economies by working in tobacco fields as slaves; after emancipation, they farmed tobacco as a cash crop in the same southern states where slavery had previously been legal.
- Many North and South American Indians and Alaska Native groups cultivated and traded tobacco and used it for ceremonial, sacred, and medicinal purposes. Some American Indians continue these traditional practices, and some American Indians have come to rely on revenue derived from tobacco sales on reservations.
- Migrants to the United States bring with them the cultural attitudes and practices characteristic of tobacco use in their native countries, such as the custom of giving gifts of tobacco in some Asian countries.

This Surgeon General's report also discusses tobacco industry support for racial/ethnic minority communities, including direct employment, advertising revenue, support for community organizations, and financial support for education, cultural, civic, sporting, arts, and other programs and events.[17]

The National Conference on Tobacco and Health Disparities

The first comprehensive definition of TRHD was developed by the 2002 National Conference on Tobacco and Health Disparities: Forging a National Research Agenda to Reduce Tobacco-Related Health Disparities, co-sponsored by NCI, the Centers for Disease Control and Prevention (CDC), the American Legacy Foundation (now known as the Truth Initiative), the Robert Wood Johnson Foundation, the American Cancer Society, the Campaign for Tobacco-Free Kids, the National African American Tobacco Prevention Network, and the National Latino Council on Alcohol and Tobacco. The 2002 National Conference sought to follow up on recommendations in the 1998 Surgeon General's report and galvanize research aimed at reducing disparities. This conference brought together practitioners and researchers from multiple disciplines to review current research, identify gaps, and develop a comprehensive research agenda to eliminate TRHD, which resulted in more than 100 recommendations from the meeting participants.

The conference defined TRHD as "differences in patterns, prevention, and treatment of tobacco use; the risk, incidence, morbidity, mortality, and burden of tobacco-related illness that exist among specific population groups in the United States; and related differences in capacity and infrastructure, access to resources, and environmental tobacco smoke exposure."[18,p.211] Fagan and colleagues[19] later modified the definition to capture more details about patterns of tobacco use that affect prevention and treatment—that is, differences in the tobacco use continuum: exposure to tobacco, tobacco use initiation, current use, number of cigarettes smoked per day, quitting/treatment, relapse, and health consequences. In addition, the authors specified that differences in capacity, infrastructure, and access to resources include differences in access to care, quality of health care, socioeconomic indicators that impact health care, and psychosocial and environmental resources.[19] The definitions were intended to help guide empirical

inquiry into the proximal and distal determinants of tobacco use, nicotine addiction, and the health consequences of tobacco use among understudied and historically underserved populations in the United States.

The Tobacco Research Network on Disparities

In 2004, NCI in partnership with the American Legacy Foundation launched the first national research initiative focused on TRHD, the Tobacco Research Network on Disparities (TReND), with the mission of "eliminating tobacco related disparities through transdisciplinary research that advocates the science, translates this scientific knowledge into practice and informs public policy."[20,p.ii3] TReND's specific purposes were to advance the science on TRHD by stimulating new studies, challenging existing paradigms, and addressing significant gaps in research on understudied and underserved populations. TReND sought to:

- Encourage collaborations among multiple research disciplines
- Serve as a forum for generating new ideas and research projects focusing on TRHD
- Establish a translation mechanism for communicating and interacting with other networks and community advocacy groups
- Promote the involvement and training of junior investigators and the participation of senior researchers in health disparities research, and
- Provide scientific information and serve as a resource on tobacco and health disparities issues.

During its tenure, TReND engaged its core members as well as other U.S. and internationally based experts in its research mission. Among its many accomplishments, TReND was the first research network to study the effects and unintended consequences of tobacco control policies on low-SES women and girls.[21,22] TReND also played a critical role in providing scientific evidence on the potential harm of menthol cigarette smoking in relationship to initiation, current smoking, nicotine dependence, and quitting behaviors.[23]

Collectively, the aforementioned reports, conferences, and initiatives have demonstrated the complexity of TRHD. This monograph aims to summarize the extant literature so as to better understand the many factors associated with TRHD, as discussed below.

TRHD: A Multilevel Perspective

Conceptual Framework: The Socioecological Model

Many factors cause different population groups to experience the effects of tobacco use in different ways. This was recognized in the 1998 Surgeon General's report, *Smoking and Health*, which stated:

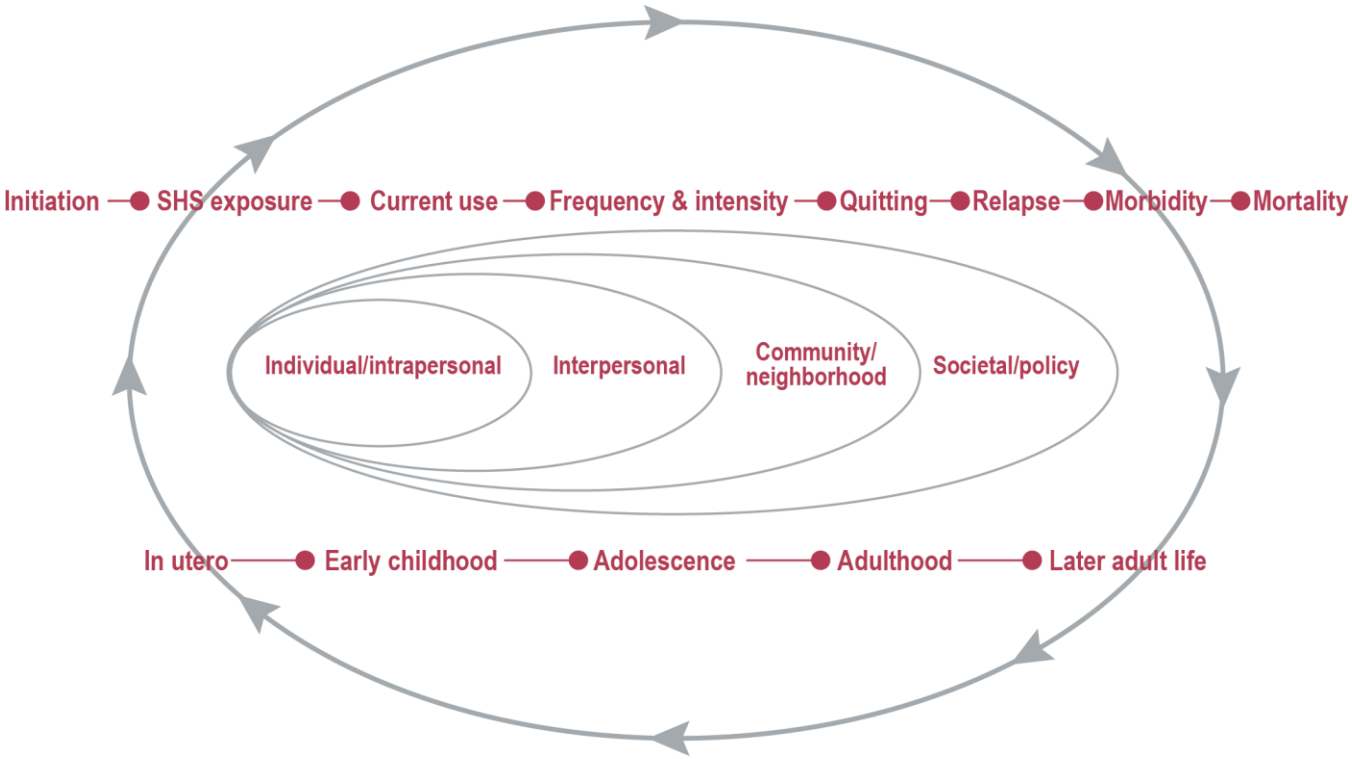
> No single factor determines patterns of tobacco use among racial/ethnic groups; the patterns are a result of complex interactions of multiple factors such as socioeconomic status, cultural characteristics, acculturation, stress, biological elements, targeted advertising, price of products, and varying capacities of communities to mount effective tobacco control initiatives.[17,p.6]

This monograph uses the socioecological model (SEM), which provides a framework for examining how multilevel factors can influence TRHD across the life span.[24] The SEM is a commonly used

framework for examining multiple levels and interrelated influences on human behavior and the health of individuals within a system.[24] In addition to explicating these multiple interrelated influences, the model has been used to design interventions to influence health behaviors and the health of individuals. The socioecological model evolved from Bronfenbrenner's conceptual ecological systems model and has undergone multiple iterations over the years.[25] Bronfenbrenner hypothesized that human behavior could be understood in terms of the individual's entire ecological system, made up of four subsystems that influence behaviors: individual; interpersonal; community/organizational; and policy/society.[25]

As shown in Figure 1.1, individual/intrapersonal, interpersonal, community/neighborhood, and societal/policy factors influence TRHD across the tobacco use continuum and across the life course. These systems do not operate in isolation, and the interactions between them are complex. Exposures to a number of factors may occur early in life, cumulatively, and chronically; they may help explain TRHD observed among racial/ethnic minority groups and low-SES groups, and at the intersection of these groups.

Figure 1.1 The Socioecological Model: Factors Influencing TRHD Across the Tobacco Use Continuum and Life Course



Notes: In addition to the experience of TRHD over time, there may be critical periods during development and throughout the life course when tobacco use or secondhand smoke exposure is significantly more detrimental than at other times. SHS = secondhand smoke.

The system within which many minority racial/ethnic and low-SES groups live and work incorporates substantial disparities. That is, the neighborhoods in which they live are often largely segregated[26,27] and have fewer neighborhood resources, including resources related to health care, education, economic opportunity and security, and political capital, relative to more advantaged groups. Among African Americans and Hispanics, poverty rates are approximately double those of whites and Asian Americans; these groups also have lower median incomes than whites and Asian Americans.[28] As the monograph

will describe, tobacco advertising is often disproportionately targeted toward low-income and minority communities, which typically lack the resources to prevent and treat tobacco use; some of these communities also have a cultural or economic connection to tobacco and/or to the tobacco industry. In 2014, African American men had the highest incidence of and mortality from several tobacco-related cancers, including cancer of the lung and bronchus, kidney and renal pelvis, pancreas, and larynx (see chapter 2).

The socioecological model underscores the interrelationships between tobacco use and multiple disparate circumstances—social, educational, health, residential, economic, and political disparities—and how each influences the other. This model makes it possible to critically examine the dynamic influences of factors (e.g., stressors, social or financial difficulties) on tobacco–disease trajectories, the timing of exposure to these factors, and the clustering of these factors at different points in relationship to disease outcomes. The socioecological model calls attention to the chronicity and incidence of disadvantages (e.g., discrimination, disenfranchisement, low SES) and how these disadvantages influence disparities even if conditions improve for individuals or population groups.

About This Monograph

This NCI monograph, *A Socioecological Approach to Addressing Tobacco-Related Health Disparities*, is the most comprehensive review of tobacco use among racial/ethnic minority and low socioeconomic status populations since publication of the 1998 Surgeon General's report. The monograph takes a socioecological approach to describe differences between population groups across the tobacco use continuum (i.e., initiation, secondhand smoke exposure, current tobacco use, number of cigarettes smoked per day, cessation, morbidity, and mortality) as well as differences in access to tobacco dependence treatment among minority racial/ethnic and low socioeconomic status groups.[18,19] Where possible, the monograph presents the evidence by age, sex, sexual orientation, and gender identity within the context of racial/ethnic and socioeconomic status groups. (The appendix to this chapter provides information about terminology regarding race/ethnicity and sexual orientation as used in this monograph.)

Additionally, where possible, the monograph takes an intersectionality approach, by focusing on the intersection or interrelationship among two or more demographic factors that are associated with TRHD, including race/ethnicity, sex, and socioeconomic status. Individuals and population groups experience all aspects of their identity simultaneously, and these social distinctions or systems may work together to produce health disparities. Researchers suggest that the interaction of sociodemographic factors shapes a person's unique life experiences, which ultimately affect their health status.[29,30]

It is important to note that this monograph is not a review of TRHD for all socially, economically, and otherwise disadvantaged populations in the United States, because no single report could adequately capture the entire range of tobacco use behaviors for all at-risk populations. For example, future reports might describe the complex associations surrounding tobacco use among people with mental health disorders, or examine tobacco use in the context of the addiction, relapse, and recovery cycle as it relates to other substance use (e.g., alcohol, marijuana, and other drugs).

The development of this monograph was informed by several previous efforts to study population differences, strengthen capacity to address differences, and develop interventions to eliminate or reduce TRHD. In particular, major recommendations from the 2002 National Conference on Tobacco and

Health Disparities resulted in a scientific blueprint for examining the complex nature of TRHD.[18] This evidence-based blueprint recommended a renewed focus on surveillance as well as a more comprehensive understanding of the roles that biology, psychosocial factors, socioeconomic factors, tobacco marketing, and tobacco control policy play in preventing tobacco use and treating tobacco dependence. This renewed focus and more complete understanding could help develop the research infrastructure needed to address TRHD.[18]

Other nationally funded initiatives that have addressed TRHD include NCI's Special Population Networks,[31] and a number of cooperative agreements with the CDC, including the Consortium of National Networks to Impact Populations Experiencing Tobacco-Related and Cancer Health Disparities,[32] A Comprehensive Approach to Good Health and Wellness in Indian Country,[33] Racial and Ethnic Approaches to Community Health,[34] Communities Putting Prevention to Work,[35] and Community Transformation Grants.[36]

Goals of the Monograph

The goal of this NCI monograph is to synthesize the research literature on the many factors that influence and contribute to TRHD across the tobacco use continuum so as to guide future research on TRHD, and to inform the design and implementation of interventions to improve the health of individuals and populations that bear the greatest burden of TRHD. It is hoped that such interventions can be applied at critical points along the tobacco use continuum, at the appropriate times and places during the lives of individuals, and for populations at risk for continued tobacco use and SHS exposure.

The review of the literature presented in this monograph is intended to inform researchers, policymakers, funding agencies, community-based organizations, faith-based institutions, stakeholders in diverse communities, institutions of higher education, and organizations that focus on reducing health disparities. Because this monograph was written to meet the different needs of these varied groups, some chapters are written in more technical language than others.

Preparation of the Monograph

This monograph underwent a rigorous development process that drew upon the expertise of many subject matter specialists—52 contributing authors, 8 section editors, a senior volume editor, and a scientific advisor—with extensive experience in the science of TRHD. The Senior Volume Editor and the Scientific Advisor led the editorial team, which developed the shared vision of the monograph's purpose, focus, and content; they also provided guidance and feedback on the monograph's content. Section editors were selected based on their expertise in areas aligned with the conceptual framework of the monograph. Given responsibility for specific topics, the section editors helped develop chapter outlines; identified authors and reviewers; and contributed to development, review, and editing of chapters. Chapter authors were selected by the editors and NCI based on their expertise and its relevance to the monograph. In addition to multiple internal reviews by the editorial team, each chapter was reviewed by external expert peer reviewers, and the monograph also underwent a full volume review. The monograph also received a final review by NCI before publication. In all, 47 reviewers participated in this process.

In general, the monograph examines the research literature through December 2013. Where necessary, key publications and reports published after 2013 were added to individual chapters. The landscape of tobacco use and tobacco control continued to change during the development of this monograph. For example, the use of emerging products such as electronic cigarettes and flavored cigars increased among some populations, and policies implemented by the Food and Drug Administration (FDA) under the Family Smoking Prevention and Tobacco Control Act of 2009 took effect. Because cigarette smoking is the leading cause of preventable death in the United States, this monograph focuses primarily on cigarette smoking and secondhand smoke exposure; where possible, studies on TRHD related to the use of other tobacco products are also included.

Major Conclusions of the Monograph

The five broad conclusions that emerge from this volume are as follows:

1. **Enormous progress has been made in reducing overall tobacco use. However, some population groups have benefited less or at a slower pace from efforts to reduce tobacco use. As a result, they experience higher tobacco-related morbidity and mortality, including mortality from cancer.** Progress in reducing tobacco use has been uneven in the United States, and substantially higher rates of tobacco use persist among population groups defined by race/ethnicity, occupation, socioeconomic status, sexual orientation, and other factors. Currently, individuals with low levels of education are at especially high risk of tobacco use, and African Americans have the highest incidence and mortality rates of tobacco-related cancer of all races/ethnicities.
2. **Many factors at multiple levels contribute to TRHD.** Our understanding of TRHD is enhanced by considering the interaction of factors at the individual, interpersonal, community/neighborhood, and societal/policy levels and by considering the impact of diverse factors across the tobacco use continuum over the life span.
3. **Research, including simulation modeling, indicates that broader implementation of known effective strategies to reduce tobacco use would contribute substantially to reducing TRHD. However, it is likely that additional strategies will be needed to accelerate reductions in tobacco use among all population groups.** The Family Smoking Prevention and Tobacco Control Act (2009), which gives the FDA the authority to regulate the manufacture, marketing, and distribution of tobacco products to protect public health and to reduce tobacco use, has strong potential to reduce TRHD. In addition, continued innovation in policies and programs at the state and local levels holds promise to address TRHD.
4. **Research to understand and address TRHD is of increasing importance to reducing the burden of tobacco use and tobacco-related cancer in the United States.** Disparities in tobacco use contribute substantially to disparities in the burden of cancer by race/ethnicity, SES, and other factors. As overall tobacco use rates have declined, the persistence of higher rates of tobacco use among groups based on race/ethnicity, socioeconomic status, sexual orientation, and other factors plays a larger role in slowing progress towards ending the tobacco epidemic.
5. **Improved surveillance of individual populations and factors that contribute to TRHD will increase our ability to understand and address TRHD.** The marketplace of tobacco products is increasingly diverse, and youth and adult patterns of tobacco use—including light and intermittent use and dual/poly use—are complex and dynamic. Communication technologies continue to evolve at a rapid pace, increasing the need for surveillance of tobacco industry

communication strategies. Enhancing surveillance to allow population-wide categories (such as race/ethnicity) to be disaggregated by sub-groups will facilitate research to understand TRHD.

Monograph Organization and Chapter Overviews

This monograph's organization in four sections reflects the socioecological model.

Section I: Overview and Epidemiology

Chapter 1: Introduction and Overview

Chapter 1 provides an introduction to TRHD research, gives a brief history of research in this area, and introduces the reader to the socioecological model, before describing the goals and preparation of this NCI monograph. The chapter also includes the overall monograph conclusions and brief descriptions of topics addressed by the other chapters of this volume, and discusses cross-cutting issues for future research.

Chapter 2: The Epidemiology of TRHD

Chapter 2 presents a detailed overview of the epidemiology of TRHD across the tobacco use continuum among youth (12–17 years old), young adults (18–25 years old), and adults in the United States. Using nationally representative data, the chapter highlights trends and current patterns of tobacco use for racial/ethnic minority groups, low-SES, and LGBT populations. This chapter presents the epidemiological data for sociodemographic groups, including trends in (1) youth and young adult susceptibility to cigarette smoking, cigarette smoking initiation, cigarette smoking prevalence and prevalence of other tobacco product use; (2) adult cigarette smoking prevalence, cigarette consumption, cigarette smoking duration, quitting behaviors, and other tobacco use; (3) secondhand and prenatal tobacco smoke exposure; (4) insurance coverage of tobacco dependence treatment; and (5) tobacco-related cancer morbidity and mortality. The chapter concludes with a discussion of some methodological limitations and challenges in the TRHD literature.

Section II: Intrapersonal/Individual Factors Associated With TRHD

Chapter 3: Genetics, Physiological Processes, and TRHD

Chapter 3 explores the relationships between genetic factors and tobacco use behaviors and tobacco-related cancers. First, the chapter discusses genetic factors associated with nicotine metabolism and smoking initiation, progression to established smoking, smoking prevalence, and smoking cessation. Genetic risk factors typically vary in prevalence across racial/ethnic populations and thus can contribute to TRHD among racial/ethnic groups. Second, the chapter describes genetic factors associated with tobacco-related cancers, specifically lung cancer, as well as genetic factors that may influence how the body responds to carcinogens in tobacco smoke. The chapter closes with a discussion of the current state of knowledge about genetic influences on TRHD, including critical knowledge gaps such as the contribution of genetic factors to TRHD in the context of complex socioeconomic environments.

Chapter 4: Flavored Tobacco and Chemosensory Processes

Chapter 4 focuses on the chemosensory effects of flavors in cigarettes and, in particular, on menthol. Flavor additives and ingredients are used to make the experience of smoking more palatable. Menthol, the most common characterizing flavor in cigarettes, has been added to cigarettes since the 1920s.

Menthol is the primary focus of this chapter because when used in cigarettes as a characterizing flavor, the compound affects multiple chemical senses, including the olfactory, gustatory, and trigeminal systems. The chapter describes the characteristics of the menthol compound, the role of the chemical senses in sensory response, the genetics of taste/sensory factors, the characteristics of flavor additives in tobacco, and menthol's effect on the chemical senses and how this may contribute to TRHD.

Chapter 5: Stress-Related Processes and TRHD

Chapter 5 provides an overview of stress processes and relevant conceptual frameworks, discusses physiological responses to stress, how perceived stress may influence tobacco use, and specific stressors such as racism and discrimination and their relationship to tobacco use. It also discusses how stressful events and stress-related processes, such as post-traumatic stress disorders resulting from childhood trauma, or stress as a function of interpersonal factors such as intimate partner violence, may also play a role in TRHD. Where possible, the research is presented separately by sex, race/ethnicity, and sexual orientation.

Section III: Interpersonal and Contextual Factors That Contribute to TRHD

Chapter 6: Social Relationships and TRHD

Chapter 6 addresses the various aspects of social relationships and how they may influence stages of the tobacco use continuum and potentially contribute to TRHD. The chapter begins with a discussion of the structural and functional characteristics of social relationships and then describes measures of social relationships and tobacco use, including social network structure, social influence and comparison, social control, social support, and discrimination. This chapter then reviews the evidence on how social relationships can create or exacerbate TRHD for youth and adults across racial/ethnic groups, SES groups, and sexual orientation groups over the tobacco use continuum.

Chapter 7: TRHD Among Immigrant Populations

Chapter 7 examines how immigration status, nativity, sex, SES, and ethnicity operate individually and synergistically to influence smoking behavior. Patterns of immigration to the United States are briefly discussed, and the literature on the smoking behavior of foreign-born people in the United States is reviewed, including differences within and between immigrant groups, comparisons between immigrant groups and the majority population, and differences between immigrants and their U.S.-born racial/ethnic counterparts. Where possible, the data for adolescents and adults are explored within the context of race/ethnicity, sex, and SES. Issues related to immigrant health generally and smoking behavior specifically are also discussed, and intersections of tobacco use, immigration, and demographic and socioeconomic factors are highlighted.

Chapter 8: Occupation, the Work Environment, and TRHD

Occupational status and the work environment help shape patterns of tobacco use. Chapter 8 examines occupational disparities across the tobacco use continuum and the causal pathway in the progression of smoking to disease, including initiation, current use and intensity, intentions to quit and quit attempts, cessation, relapse, and tobacco-related morbidity and mortality. This chapter also discusses the contributions of the work environment and job experiences to disparities in tobacco use. Because disparities by occupation can interact with other indicators of social disadvantage, this chapter explores the intersections between occupation and race/ethnicity, sex, age, and sexual orientation. Disparities by employment status are also reviewed, given the potential influence of the work environment, working conditions, and social status on tobacco use behaviors.

Chapter 9: Socioeconomic Status and TRHD

Socioeconomic status, whether measured by educational attainment or economic measures, influences health through multiple direct, indirect, and overlapping causal pathways. Chapter 9 provides an overview of the literature on the relationship between SES indicators and TRHD across the tobacco continuum; it reviews this evidence using nationally representative and non-nationally representative data sets for adolescents, racial/ethnic groups, LGBT groups, and pregnant women. The chapter also discusses neighborhood socioeconomic status and the influence of life-course socioeconomic status. Studies show that education—a key factor influencing other socioeconomic indicators such as occupation, income and wealth, and the neighborhood where people live—is closely linked with tobacco use across the continuum.

Section IV: Societal-Level Influences on Tobacco Use

Chapter 10: Communications, Marketing, and TRHD

As discussed in NCI Tobacco Control Monograph 19, *The Role of the Media in Promoting and Reducing Tobacco Use*, both pro-tobacco and anti-tobacco communications and marketing help shape the public's knowledge, attitudes, beliefs, and behaviors around tobacco. Chapter 10 expands and updates the literature to examine the effects of tobacco-related communications on population groups based on age, race/ethnicity, socioeconomic status, and sexual orientation. It begins by describing theoretical frameworks for understanding communications inequalities, then discusses research on the influence of diverse anti-tobacco and pro-tobacco communications on TRHD and on the role of the news media. Studies of how new communication technologies may serve as channels for anti- and pro-tobacco communications are discussed, recognizing that the rapid pace of change in communications technology poses a challenge for researchers.

Chapter 11: Federal, State, and Local Tobacco Control Policy and TRHD

As explained in the 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress,* "public health efforts to control tobacco use have been bolstered by policies at the national, state, and local levels."[3,p.788] Chapter 11 provides an overview of research on the ability of specific tobacco control policies (including those focused on restricting youth access to tobacco, tobacco tax and price, smoke-free environments, and tobacco treatment) and state-level comprehensive tobacco control programs to reduce TRHD. The chapter also discusses the potential for FDA regulation of the manufacturing, marketing, and distribution of tobacco products (authorized by the Family Smoking Prevention and Tobacco Control Act of 2009) to contribute to reducing TRHD. It also provides

examples of local approaches to reducing the prevalence of youth tobacco use, such as efforts to ban flavored tobacco products and to raise the minimum age of legal access to tobacco products to 21 years.

Chapter 12: Simulation Modeling of TRHD: SimSmoke

This chapter discusses a modified version of the *SimSmoke* tobacco control simulation model, a statistical model that examines trends in smoking and smoking-attributable death rates and projects the possible effects of various policies. The modified version was developed to examine trends in smoking rates related to income disparities and the potential effects of tobacco control policies on smoking trends. The modified version of *SimSmoke* considers policies in seven areas: cigarette taxes, smoke-free laws, mass media anti-tobacco campaigns, marketing restrictions, health warnings, cessation treatment policies, and preventing youth access. The model's predicted results are presented for recommended policies using the status quo scenario and scenarios with stronger policies. Best-case scenarios with a set of comprehensive policies are also described. This chapter illustrates the potential of broader implementation of evidence-based tobacco control policies to reduce tobacco use and tobacco-related mortality among low-income populations.

Future Directions in TRHD Research

Cross-Cutting Research Issues

Several cross-cutting issues are relevant to future research, particularly as the cultural, policy, and communications contexts of tobacco use and TRHD continue to change. Most studies of TRHD have focused on race/ethnicity, with an emphasis on the largest population groups: African Americans and Hispanics. Although research among these groups remains vital, research is also needed on less populous racial/ethnic groups with high smoking prevalence, including American Indians/Alaska Natives, Native Hawaiians/Pacific Islanders, and Asian American groups such as Filipinos, Koreans, and Vietnamese. In addition, it is now recognized that aggregating ethnic and nationality groups into a larger category can mask underlying differences in smoking prevalence. For example, the Asian American group includes people of Indian, Chinese, Filipino, Japanese, Korean, and Vietnamese origin, and the Hispanic group includes people from Central or South America, Cuba, Mexico, and Puerto Rico. Examining more specific ethnic or nationality groups is important, but can lead to smaller sample sizes, which limits statistical power and/or the generalizability of findings. Thus, the desirability of examining specific ethnic or nationality groups separately must be weighed against the benefits of aggregating groups into a larger category. In addition, although surveys indicate that LGBT groups are at increased risk for tobacco use, there is limited evidence on their tobacco use knowledge, attitudes, and behaviors, and disease-related disparities. The inclusion of questions about sexual orientation and gender identity in national and sub-national surveys will facilitate research in this area.

Most studies reviewed in this monograph have focused on the impact of membership in a single population group (by race/ethnicity, SES, or sexual orientation); however, people who are part of more than one vulnerable population group may be at especially high risk of experiencing TRHD. Currently, we lack sufficient data on which to base conclusions about how identification with multiple minority groups might create or exacerbate TRHD. How membership in more than one vulnerable group affects tobacco-related morbidity and mortality is an area requiring further research.

The degree to which tobacco control interventions should be adapted to different cultures and populations remains unclear. Research is needed to determine whether and to what extent programs that are effective among the general population are sufficient to address tobacco use among specific populations, or whether tailored programs are needed. Tailoring can be time-consuming and costly but may increase the effectiveness of the intervention. How to best develop culturally relevant programs to reduce tobacco use and TRHD among populations of interest (i.e., not simply tailoring existing programs) is also an important area for further inquiry and may help determine if such programs lead to faster declines in smoking prevalence.

Tracking trends in the use of new and emerging tobacco products, such as electronic cigarettes, among vulnerable population groups is important and may require expansion of existing surveillance systems or the creation of new ones. Future research on TRHD should also address the use of flavored tobacco products, including menthol products, particularly among youth and young adults. Research to prevent future TRHD related to the use of new and emerging tobacco products is an important area of focus.

Linking national studies and surveillance systems to systems for monitoring federal, state, and local policies would result in more robust surveillance systems and contribute to a more complete picture of tobacco use behaviors and TRHD. Multiple linked surveillance systems are critical to tracking the rapidly changing tobacco marketplace. As measures of emerging tobacco product use are fine-tuned, it will be important to standardize them across these studies and surveillance systems. Including population groups targeted by the tobacco industry in federal, state, and local surveillance systems will be critical to effectively monitor tobacco industry marketing practices across various levels. Surveillance systems should be augmented by the study of contextual factors that affect TRHD, including social norms, cultural values, and community factors, and how they interact with individual psychosocial, genetic, and biological factors.

New challenges to TRHD will continue to emerge. For example, changes in state marijuana laws (including laws that decriminalize or legalize marijuana use or permit the use of medical marijuana) may well influence tobacco use behaviors.[37–39] Understanding how changing marijuana laws may influence tobacco use initiation, progression to established tobacco use, successful cessation, and dual/poly product use across populations is likely to be increasingly important. Rapidly evolving communications technologies pose both challenges to and opportunities for tobacco control; these deserve attention from researchers.

Conclusion

As noted above, no single monograph can encompass the science of TRHD for all at-risk populations. This monograph focuses on TRHD among groups defined by race/ethnicity and socioeconomic status, presenting the evidence by age, sex, and sexual orientation where possible. It explains the complex and multifactorial nature of TRHD, gleaned by countless researchers and practitioners working to eliminate TRHD. This review of the evidence demonstrates that continued effort is needed to accelerate declines in tobacco use and SHS exposure in order to both reduce current TRHD and to prevent TRHD from increasing in the future.

Improving federal, state, and local infrastructure and resources for designing, delivering, and evaluating programs and policies aimed at reducing tobacco use and SHS exposure is critical to advancing our understanding of TRHD and to reducing the disproportionate burden of tobacco-related cancer among

at-risk populations. Training and mentoring the next generation of TRHD researchers is essential to accelerate progress in reducing TRHD. Collaborative networks and partnerships between researchers and community groups may contribute to this effort.

Over time, tobacco use has evolved from a mainstream behavior to one that is highly concentrated among population groups defined by socioeconomic status, race/ethnicity, sexual orientation, and other factors. Decades of research have documented the extraordinary hazards of tobacco products, helping to transform tobacco-related social norms, policies, and patterns of tobacco use behaviors. The result has been striking declines in tobacco-related deaths, including deaths from lung cancer. Indeed, it has been estimated that 20th-century tobacco control programs and policies are responsible for preventing more than 795,000 lung cancer deaths in the United States from 1975 through 2000.[40,41] As this monograph demonstrates, a central challenge for cancer control is to ensure that all Americans benefit from advances in tobacco control research and practice.

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Appendix I: Monograph Terms

Racial/Ethnic Groups

The term *race/ethnicity* recognizes both racial and ethnic identities. *Race* is a term that has been used to classify groups based on skin color, physical traits, ancestry, genetics, and social relations. The characterization of race has often been reduced to a simple biological construct; this monograph views race as a social, rather than a biological, construct that has historical and cultural context. In 1997, the U.S. Office of Management and Budget (OMB) provided guidelines for classifying individuals by race. The OMB standards permit the reporting of more than one race and rely on self-identification.[1] The five OMB categories are as follows:

- White – A person having origins in any of the original peoples of Europe, the Middle East, or North Africa.
- Black or African American – A person having origins in any of the black racial groups of Africa.
- American Indian or Alaska Native – A person having origins in any of the original peoples of North and South America (including Central America) and who maintains tribal affiliation or community attachment.
- Asian – A person having origins in any of the original peoples of the Far East, Southeast Asia, or the Indian subcontinent, including, for example, Cambodia, China, India, Japan, Korea, Malaysia, Pakistan, the Philippine Islands, Thailand, and Vietnam.
- Native Hawaiian or Other Pacific Islander – A person having origins in any of the original peoples of Hawaii, Guam, Samoa, or other Pacific Islands.

The term *ethnicity* refers to distinct groups that have a common culture, heritage, and place of origin. Ethnic groups can be characterized to some extent by the norms, values, and attitudes that arise from their culture of origin and may shape their behavior. However, culture and race/ethnicity are not equivalent, and cultures change over time as groups adapt to new settings and historical contexts. The distinctions between culture and race/ethnicity are particularly important when considering how historical context, psychosocial factors, and psychological processes influence health behaviors and outcomes.

Ethnic groups are also distinguished by more than their culture, and may have attributes associated with their status as minorities within the United States. For example, some ethnic groups are over-represented in lower socioeconomic strata and are subject to stereotypes and prejudices and discrimination related to social class or race. Thus, psychosocial processes related to their experiences, in addition to their culture of origin, may differentiate particular racial/ethnic groups. This monograph uses the term *racial/ethnic group* to encompass the full spectrum of identities within and between groups.

The categorization of blacks or African Americans includes the diverse terminology people use to identify themselves as members of the group. For example, people born in the United States might identify as African American, a social–political term used to affirm ancestry, culture, history in the United States, and U.S. citizenry that goes beyond skin color. People of Caribbean descent or immigrants from other countries might identify as black, the term used in their countries of origin. Where possible, this monograph uses black and African American or black/African American to capture different self-identities and racial/ethnic terms used in national surveys or, when standardizing terms is not appropriate, the terms appear as they do in the literature referenced.

The monograph uses Hispanic/Latino American, Hispanic/Latino, Hispanic, or Latino because various ethnic groups within this aggregate category self-identify with different terms.[2] This aggregate group includes people of Spanish origin, Puerto Ricans, Cubans, Chicanos, Mexican Americans, and ethnic groups from South or Central America.

American Indians and Alaska Natives include descendants of the original peoples of North, South, and Central America, many of whom maintain their tribal affiliation or community attachment.[1,3] In 1997, the U.S. OMB expanded its definition of American Indians to include people of South and Central American Indian descent. The approximately 566 federally recognized tribes[4] and non-federally recognized tribes have their own cultures, languages, beliefs, and practices. Over 200 tribes are located in Alaska alone.[5] These populations live across the United States in urban and rural areas and on or off reservations or land trust areas. Nearly 60% of American Indians and Alaska Natives live in metropolitan areas.[6] American Indians and Alaska Natives are the only groups whose tribes are recognized as sovereign nations within the United States. Unfortunately, individual tribal data are often masked within this aggregate category because as a whole, American Indians and Alaska Natives represent 2% of the U.S. population.[6]

The current categorization of Asian Americans and Native Hawaiians and Other Pacific Islanders differs slightly from what was reported in the 1998 Surgeon General's report. The 1997 OMB guidelines required that Asians be considered a separate group from Native Hawaiian and Other Pacific Islanders.[1] A distinction was made between these racial/ethnic groups because Native Hawaiians (descendants of the original people of the Hawaiian Islands, whose origins can be traced back to Polynesian and other cultures) made up only 3% of the Asian and Other Pacific Islander population. Other Pacific Islander is a broad term that may include Samoans, Guamanians, and Chamorros; Polynesians such as Tahitians, Tongans, and Tokelauans; Micronesians such as Chuukese, Palauans, Marshallese; or Melanesians such as Fijians, Guineans, or Solomon Islanders.[7]

All five minority racial/ethnic groups are aggregate categories that include multiple ethno-linguistic groups and multiple ancestries. These groups may have different histories of entry to the United States, settlement in this country, and evolution as racial, ethnic, and minority groups. Race and ethnicity are socially based constructs, but each category, whether in aggregate or disaggregate form, is used to describe the data and explain more about how people who identify as members of aggregate or disaggregate racial/ethnic groups experience TRHD. National, state, and local survey data rely on self-identification for each racial/ethnic group.

Lesbian, Gay, Bisexual, and Transgender Groups

LGBT is an aggregate category that includes groups that are defined by sexual orientation, gender identification, and gender expression. Sexual orientation includes three dimensions—self-identification, sexual behavior, and sexual attraction.[8] Sexual orientation is most often used to assess health behaviors among LGBT groups. The survey question most often asked to elicit respondents' identification of sexual orientation is, "Do you consider yourself to be . . . heterosexual/straight, gay or lesbian, or bisexual?"[9] The question that assesses sexual behaviors asks about the sex of the respondent's sexual partners (e.g., men only, women only, both, or no sexual partners). Sexual attraction is often assessed by asking respondents to describe their feelings related to their attractions (e.g., attractions to males and females). Gender identification questions assess the sex of the respondent at birth and the gender reported at the time of the survey.[8] According to analyses conducted by Flores and colleagues,[10]

individuals who identify as transgender are less likely to be white and more likely to be African American/black or Hispanic/Latino than the general U.S. adult population. Those who identify as transgender are often marginalized and are among the most disadvantaged groups in the United States. Many other terms are used to describe LGBT groups, which reflect diversity in identification within the aggregate category.[9]

Note: Socioeconomic status, an overarching theme of this monograph, is defined in chapter 9.

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Section I
Overview and Epidemiology

Chapter 2
The Epidemiology of Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

Tobacco use is the leading cause of preventable premature death in the United States, causing an estimated 480,000 deaths annually.[1] Although smoking prevalence has declined substantially over time,[1] 40 million U.S adults were current smokers in 2014.[2] The 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*,[1] noted that disparities in tobacco use persist by race, ethnicity, education level, socioeconomic status (SES), and U.S. geographical region, among other factors.

This chapter presents a detailed overview of the epidemiology of tobacco-related health disparities (TRHD) across the tobacco use continuum (defined as exposure to tobacco, tobacco use initiation, current use, number of cigarettes smoked per day, quitting/treatment, relapse, and health consequences) among youth (12–17 years old), young adults (18–25 years old), and adults (26+) in the United States. Using nationally representative data, this chapter highlights trends and current patterns for minority racial/ethnic groups; people of low SES; and lesbian, gay, bisexual, and transgender (LGBT) populations. This chapter presents the available epidemiological data for sociodemographic groups, including trends in (1) youth and young adult susceptibility to cigarette smoking, cigarette smoking initiation, and cigarette and other tobacco use prevalence; (2) adult cigarette smoking prevalence, consumption, smoking duration, quitting behaviors, and other tobacco use; (3) secondhand and prenatal tobacco smoke exposure; (4) insurance coverage of tobacco dependence treatment; and (5) tobacco-related cancer incidence and mortality. The chapter concludes with a discussion of some methodological limitations and challenges in the TRHD literature. The epidemiological data that describe disparities across the tobacco use continuum can inform prevention and cessation efforts to reduce the disproportionate burden of tobacco-related diseases and deaths on minority racial/ethnic, low-SES, and LGBT populations, and the intersection of these groups.

Data Sources

Table 2.1 describes the national and state surveys/studies that inform this chapter, with examples of measures used to report smoking prevalence and other smoking behaviors. Wording of survey questions can vary across surveys, which can lead to small differences in reported data; however, the trends across surveys are very consistent. (Note that this table does not include all surveys that measure tobacco use behaviors.)

In light of the limitations of aggregate data for explaining certain disparities, this monograph reports, where available, national data disaggregated for specific racial/ethnic groups. Although data are generally available for the larger racial/ethnic groupings (African Americans, Hispanics/Latinos, Asian Americans, Native Hawaiians/Other Pacific Islanders, and American Indian/Alaska Natives, as defined by the U.S. Office of Budget and Management), aggregate or disaggregated data may sometimes yield unstable estimates for individual years. In addition, poverty status variables in this monograph were constructed using the U.S. Census Bureau's poverty threshold, which accounts for family income, size, and number of children, adjusted annually for inflation (for additional information, see U.S. Census Bureau 2016[3]).

Table 2.1 Summary of State and National Surveys/Studies on Youth and Adult Tobacco Use Referenced in This Chapter

Survey	Description	Population and survey methodology	Example(s) of a measure(s)	Website
Monitoring the Future Study (MTF)*	MTF is an annual, ongoing study of beliefs, attitudes, and behavior of American secondary school students, college students, and young adults. Funded by the National Institute on Drug Abuse.	Nationally representative sample of 8th-, 10th-, and 12th-grade students take a group-administered school-based survey. Follow-up surveys are mailed to a randomly selected sample from each senior class, with biannual follow-up after high school on a continuing basis.	Current cigarette smoking: How frequently have you smoked cigarettes during the past 30 days?	http://monitoringthefuture.org
National Youth Tobacco Survey (NYTS)*	NYTS provides nationally representative data about middle and high school youths' tobacco-related beliefs, attitudes, behaviors, and exposure to pro- and anti-tobacco influences. Conducted biennially by the Centers for Disease Control and Prevention (CDC).	Nationally representative sample of middle and high school students (grades 6–12) Self-administered school-based survey	Current cigarette smoking: During the past 30 days, on how many days did you smoke cigarettes? Current cigar smoking: During the past 30 days, on how many days did you smoke cigars, cigarillos, or little cigars?	https://www.cdc.gov/tobacco/data_statistics/surveys/nyts
Youth Risk Behavior Survey (YRBS)*	YRBS is conducted biennially by the CDC to monitor priority health-risk behaviors and the prevalence of obesity and asthma among youth and young adults.	Nationally representative sample of high school students (grades 9–12) Self-administered school-based survey	Current cigarette smoking: Smoked on at least 1 day during the 30 days before the survey Current cigar smoking: Smoked cigars, cigarillos, or little cigars on at least 1 day during the 30 days before the survey	https://www.cdc.gov/mmwr/pdf/rr/rr6201.pdf

Table 2.1 continued

Survey	Description	Population and survey methodology	Example(s) of a measure(s)	Website
National Health Interview Survey (NHIS)†	NHIS is conducted annually by the CDC to monitor the health of the U.S. population. Collects and analyzes data on a broad range of health topics, including tobacco use, by various demographic and socioeconomic characteristics.	Representative sample of the U.S. population Cross-sectional in-person household interview survey	Current cigarette smoking: Persons who reported smoking ≥100 cigarettes during their lifetime and who, at the time of interview, reported smoking every day or some days. Interest in quitting: Current smokers who reported that they wanted to stop smoking completely Past-year quit attempt: Current smokers who reported that they stopped smoking for >1 day during the past 12 months because they were trying to quit smoking, and former smokers who quit during the past year Recent smoking cessation: Former smokers who quit smoking for ≥6 months during the past year	https://www.cdc.gov/nchs/nhis

Table 2.1 continued

Survey	Description	Population and survey methodology	Example(s) of a measure(s)	Website
National Health and Nutrition Examination Survey (NHANES)†	NHANES is a program of studies to assess the health and nutritional status of adults and children in the U.S. Conducted by the CDC. The survey is unique in that it combines interviews and physical examinations.	Nationally representative sample of the U.S. population of all ages Interviewer-administered home-based survey and physical examination by physicians	Home secondhand smoke exposure: A report of ≥1 household cigarette smokers and the number of cigarettes smoked per day Age of initiation: Age when first smoked a whole cigarette Ever tried: Ever tried cigarette smoking, even a few puffs Current cigarette smoking: Smoked a whole cigarette on at least 1 day during the 30 days before the survey	https://www.cdc.gov/nchs/nhanes
National Survey on Drug Use and Health (NSDUH)†	NSDUH is an annual survey sponsored by the Substance Abuse and Mental Health Services Administration. It provides national and state-level data on the use of tobacco, alcohol, illicit drugs (including non-medical use of prescription drugs) and mental health in the U.S.	Random sample of U.S. civilians age 12 or older Interviewer-administered home-based survey	Current cigarette smoking: During the past 30 days, have you smoked part or all of a cigarette?	https://www.samhsa.gov/data/population-data-nsduh
Population Assessment of Tobacco and Health (PATH) Study†	PATH is a national longitudinal study of tobacco use and how it affects the health of people in the U.S. Jointly conducted by the National Institutes of Health and the Food and Drug Administration.	Sample of people ages 12 and older in the U.S. 3 annual home-based in-person interviews including audio computer-assisted self-interviewing and biospecimen collection	Current cigarette smoking: Persons who reported smoking ≥100 cigarettes during their lifetime and who, at the time of interview, reported smoking every day or some days.	https://pathstudyinfo.nih.gov

Table 2.1 continued

Survey	Description	Population and survey methodology	Example(s) of a measure(s)	Website
Behavioral Risk Factors Surveillance System (BRFSS)‡	BRFSS, a project of the CDC, collects state data annually about U.S. residents regarding their health-related risk behaviors, chronic health conditions, and use of preventive services.	Representative sample of U.S. adults age 18 or older Home-based in person or phone administered interviews via random digit dialing	Cigarette smoking prevalence: Do you now smoke cigarettes every day, some days, or not at all?	https://www.cdc.gov/brfss
Pregnancy Risk Assessment Monitoring System (PRAMS)‡	PRAMS is a surveillance project of the CDC and state health departments. PRAMS collects state-specific, population-based data on maternal attitudes and experiences before, during, and shortly after pregnancy in 47 states, as of 2017.	Stratified samples of women who have recently given birth to live infants are selected from birth certificates in participating states. The survey is sent 2–6 months after delivery. Self-administered survey	Smoking status is recorded for the 3 months before pregnancy, the last 3 months of pregnancy, and postpartum: How many cigarettes did/do you smoke on an average day?	https://www.cdc.gov/prams
Tobacco Use Supplement to the Current Population Survey (TUS-CPS)‡	TUS-CPS is a National Cancer Institute-sponsored survey of tobacco use that is administered as part of the U.S. Census Bureau's Current Population Survey every 3–4 years.	Nationally representative sample of adults (youth ages 15–17 were included in 1992–2006 cycles) Telephone survey or in-person interview collection	Age of initiation: Age started smoking cigarettes "fairly regularly' (refers to age when respondent started smoking cigarettes on a routine basis, as opposed to age when tried first cigarette) Current cigarette smoking: Now smoking cigarettes every day or some days	https://cancercontrol.cancer.gov/brp/tcrb/tus-cps

*Survey/study includes youth only.
†Survey/study includes youth and adults.
‡Survey includes adults only.

Youth Tobacco Use Behaviors

Youth Susceptibility to Cigarette Smoking, by Race/Ethnicity

Susceptibility to smoking is often measured among never-smokers to predict the likelihood of smoking in the future.[4] Never-smokers who show a firm commitment not to smoke in the future and not to smoke cigarettes offered by a friend are less likely to ever smoke,[5] experiment,[4,6–8] or initiate smoking[6] than youth who do not make this commitment.[4,9] A few studies have examined racial/ethnic differences in susceptibility to smoking. Among youth ages 12–17 who have never smoked, Hispanic youths had the highest susceptibility to smoking (24.2%), followed by American Indian/Alaska Natives (19.7%), blacks/African Americans (19.4%), non-Hispanic whites (19.0%), Native Hawaiians or Other Pacific Islanders (16.0%), and Asian Americans (15.1%).[10] Among Asian Americans, susceptibility to smoking was highest among Filipinos (18.6%) and lowest among Chinese (11.7%). Among U.S. Hispanics, Mexicans were the most susceptible to smoking (25.8%) and Puerto Ricans were the least (18.3%).[10]

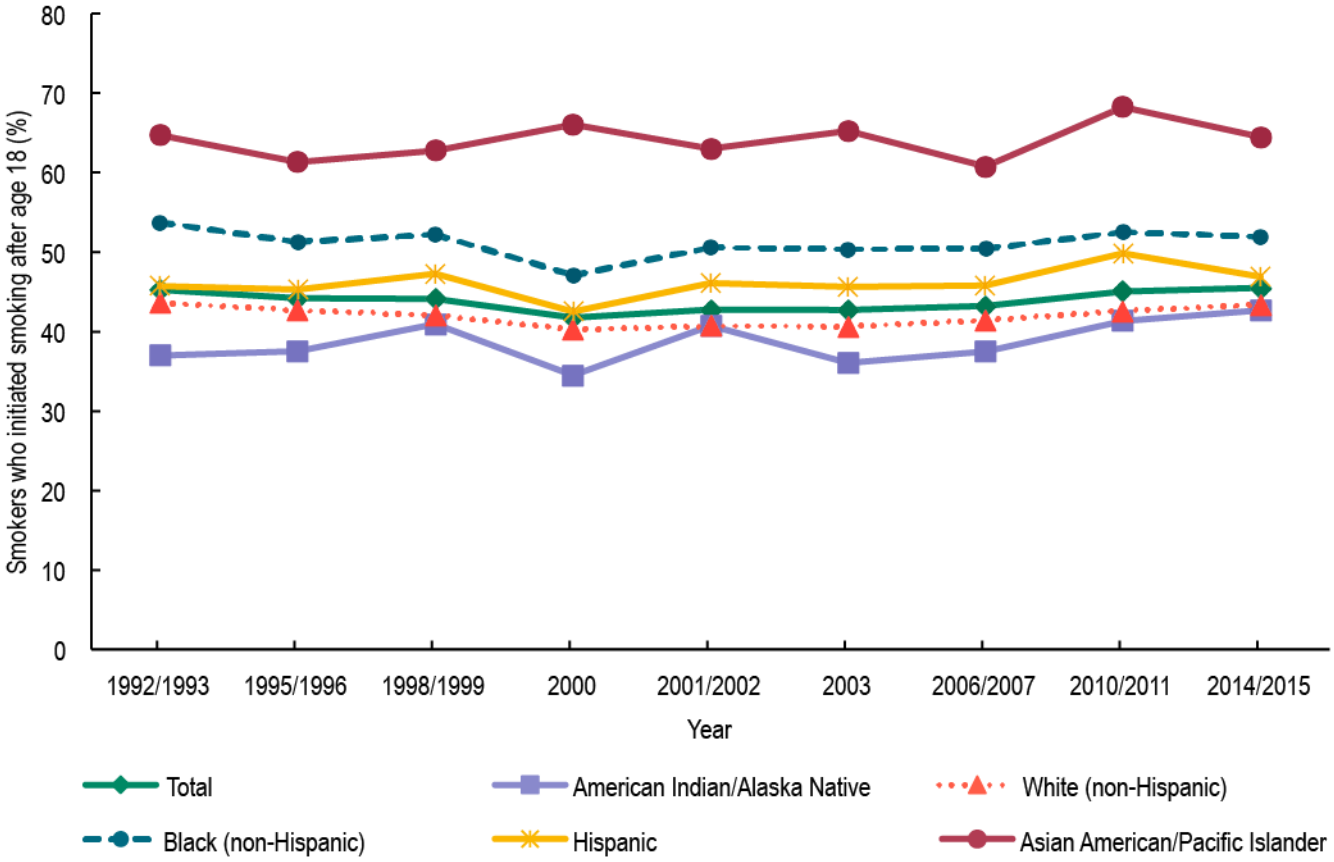
Youth Smoking Initiation, by Race/Ethnicity and SES

Age of smoking initiation is measured by asking smokers what age they were when they first smoked all or part of a cigarette (National Survey on Drug Use and Health [NSDUH]), or when they first smoked a whole cigarette (Youth Risk Behavior Survey [YRBS]), or when they first tried cigarette smoking, even one or two puffs (National Youth Tobacco Survey [NYTS], Population Assessment of Tobacco and Health [PATH] study). As highlighted in the 1994 Surgeon General's report *Preventing Tobacco Use Among Young People*,[9] most cigarette smokers began smoking during adolescence. According to data from the 2008–2010 NSDUH presented in the 2012 Surgeon General's report *Preventing Tobacco Use Among Young People*,[10] among adults ages 30–39 years who had ever tried cigarette smoking, 82% first tried before age 18, and nearly 99% first tried before age 25.

NSDUH data analyzed by Caraballo and colleagues[11] show that the age of initiation of smoking during adolescence varies by race/ethnicity. Among youths ages 12–17, American Indian/Alaska Native youths and Native Hawaiian and Other Pacific Islander youths initiated smoking at mean ages of 11.5 and 11.8 years, respectively, compared with 12.3 years among non-Hispanic white youths, 12.4 years among African American youths, 12.5 years among Hispanic youths, and 12.8 years among Asian American youths.[11] Data from the National Health and Nutrition Examination Survey (NHANES) also show that among 12- to 17-year-old youths, Mexican American and non-Hispanic black youths initiated smoking at older ages than non-Hispanic white youths.[12]

National-level data reported in the 2012 Surgeon General's report show a lower rate of smoking initiation among non-Hispanic black youths compared with non-Hispanic white and Hispanic youths.[10] This pattern parallels data from the Tobacco Use Supplement to the Current Population Survey (TUS-CPS) that show differences by race/ethnicity in the percentage of U.S. adult smokers who initiated regular smoking after age 18. As shown in Figure 2.1, the majority of Asian American/Pacific Islander and non-Hispanic black smokers initiated regular smoking after age 18, in contrast to other racial/ethnic groups.

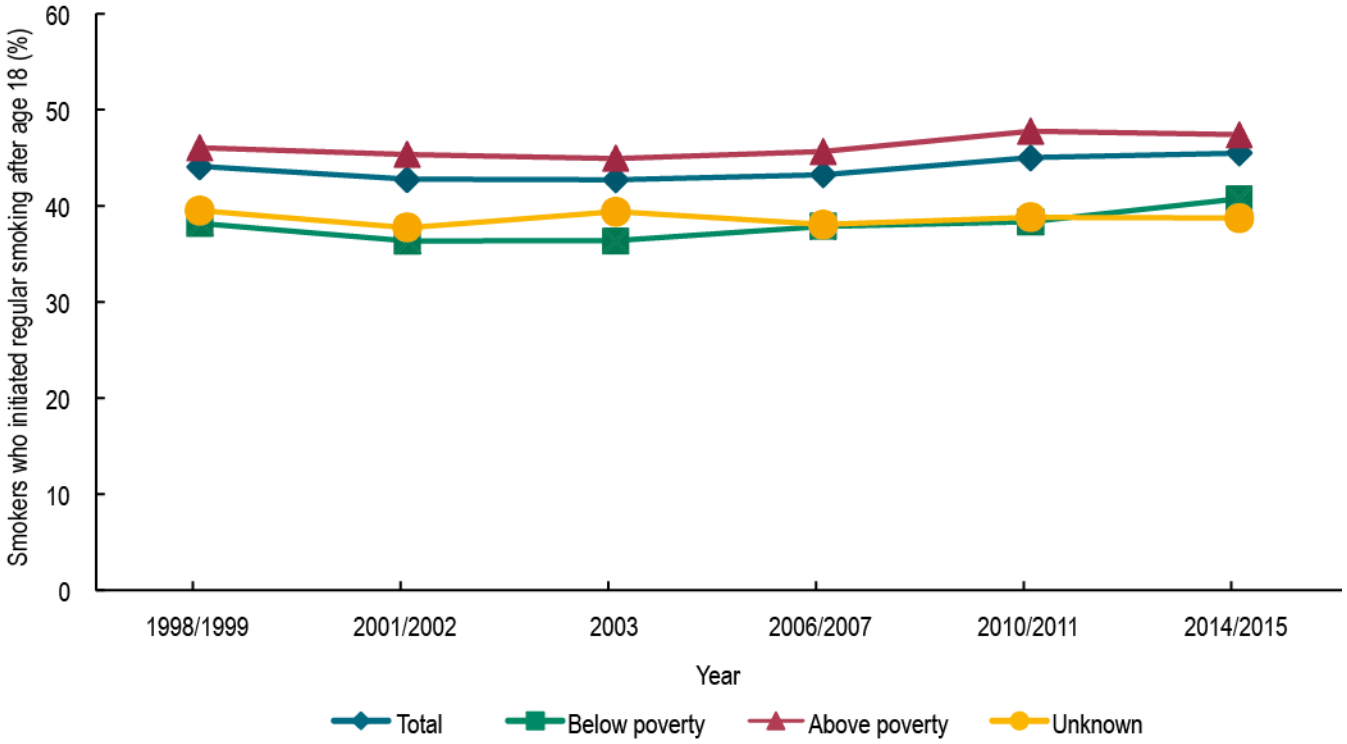
Figure 2.1 Percentage of U.S. Current Smokers Who Initiated Regular Smoking After Age 18, by Race/Ethnicity, 1992/1993–2014/2015



Note: Survey respondents were asked, "How old were you when you first started smoking cigarettes fairly regularly?"
Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 1992/1993–2014/2015.[13]

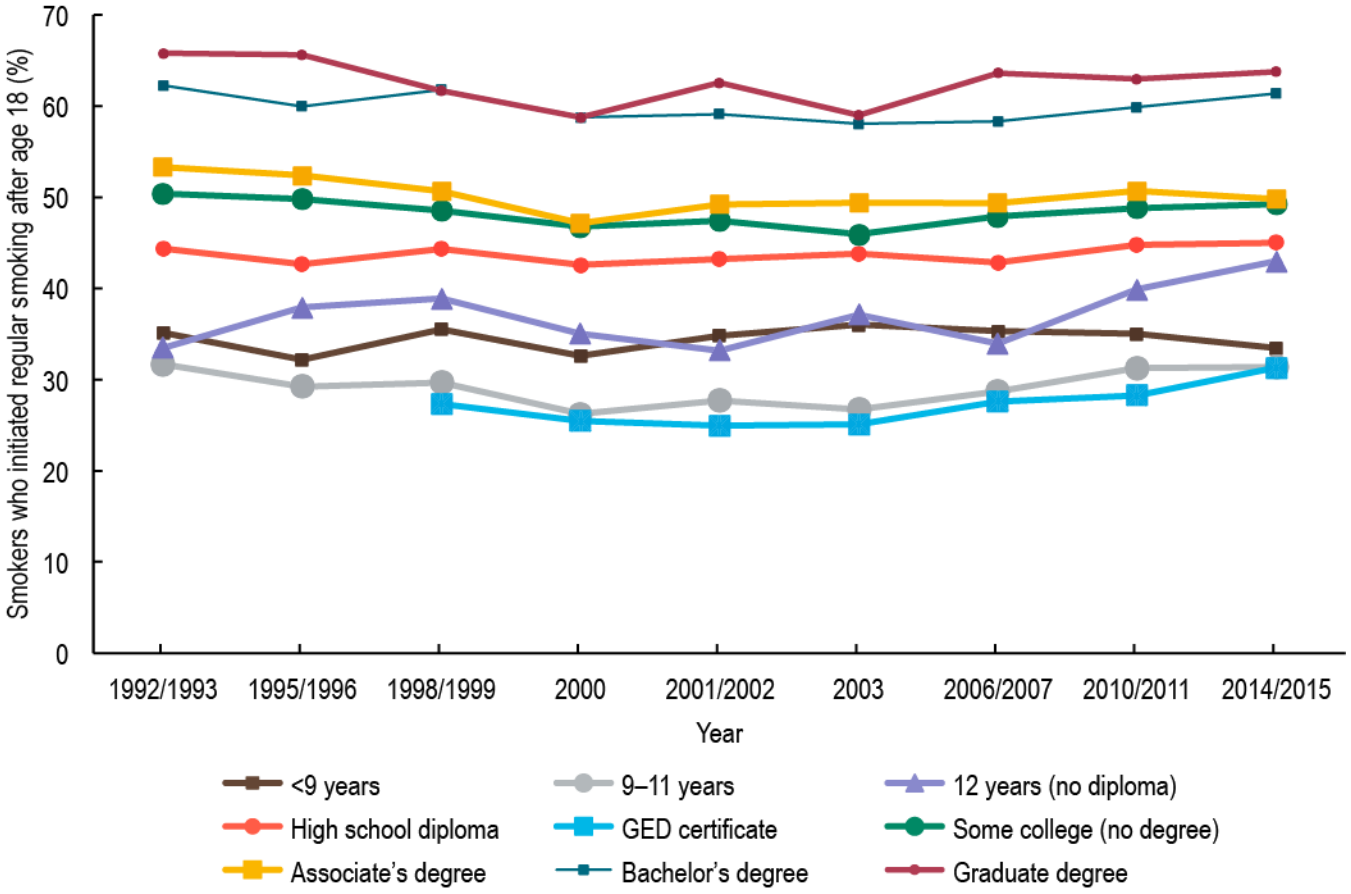
National data indicate that youths and young adults from low-SES backgrounds are at higher risk of smoking than their more advantaged counterparts.[10,12] Data from the 1999–2004 NHANES show that youth ages 12–17 living in poverty are significantly more likely to try smoking cigarettes and to report current smoking compared with more advantaged youth.[12] Trend data from the TUS-CPS also reveal differences in the age of onset of regular smoking by poverty status and educational attainment. Since 1998/1999, a higher percentage of current adult smokers living above the poverty line initiated regular smoking after age 18 (47% in 2014/2015) compared with those living below the poverty line (41% in 2014/2015) (Figure 2.2).[13] Similarly, since 1992/1993 more educated adults are more likely to have initiated smoking after age 18 than their less educated counterparts (Figure 2.3). The age of smoking initiation is an important behavior for surveillance and intervention efforts, because numerous studies have linked earlier initiation to greater nicotine dependence and longer duration of smoking.[14]

Figure 2.2 Percentage of U.S. Current Smokers Who Initiated Regular Smoking After Age 18, by Poverty Status, 1998/1999–2014/2015



Notes: Survey respondents were asked, "How old were you when you first started smoking cigarettes fairly regularly?" Unknown indicates that respondents were not part of a family to calculate poverty level (e.g., unmarried partners or roommates).
Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 1998/1999–2014/2015.[13]

Figure 2.3 Percentage of U.S. Current Smokers Who Initiated Regular Smoking After Age 18, by Educational Attainment, 1992/1993–2014/2015



Notes: GED = General Educational Development certificate. Data collection by GED certificate began in 1998/1999. Survey respondents were asked, "How old were you when you first started smoking cigarettes fairly regularly?"
Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 1992/1993–2014/2015.[13]

Cigarette Smoking Prevalence Among Youth

YRBS data show that cigarette smoking prevalence among high school youth reached a high of 36.4% in 1997, fell sharply to 21.9% in 2003,[15] and then declined to 15.7% in 2013[16] and 10.8% by 2015.[17] Data on middle and high school students collected by the NYTS between 2000 and 2015 also show a linear downward trend in current cigarette use—from 10.7% to 2.3% among middle school youths, and from 27.9% to 9.3% among high school youths.[18,19] Despite this overall progress, significant disparities in youth and young adult cigarette smoking rates persist by race/ethnicity and SES.[10,18]

Current Cigarette Smoking Among Youth, by Race/Ethnicity

Trends in youth cigarette smoking prevalence are typically reported only for the three largest racial/ethnic groups in the United States: non-Hispanic whites, Hispanics, and non-Hispanic blacks. Data from the YRBS show that historically, smoking prevalence has been highest among non-Hispanic white youth, followed by Hispanic and non-Hispanic black youth.[15]

As shown in Table 2.2, pooled NSDUH data from 2013–2015 show that the prevalence of current cigarette smoking among youth ages 12–17 was highest among American Indians/Alaska Natives (7.0%) followed by non-Hispanic whites (6.3%), Native Hawaiians or Other Pacific Islanders (3.4%), Hispanics (3.4%), non-Hispanic blacks/African Americans (2.7%), and Asians (1.6%). NSDUH[20] also collects cigarette smoking prevalence data for Asian and Hispanic subgroups. Among Asians, Asian Indian youth reported the highest prevalence of smoking (3.2%), and Chinese youth reported the lowest (0.7%). Among Hispanic youth, Mexicans reported the highest prevalence of smoking (3.6%), and Central or South American youth reported the lowest (2.4%).[20] Overall, and for most racial/ethnic groups, current smoking prevalence was higher among males than females.

Table 2.2 Prevalence of Current Cigarette Smoking Among U.S. Youth Ages 12 to 17, by Race/Ethnicity and Sex, 2013–2015

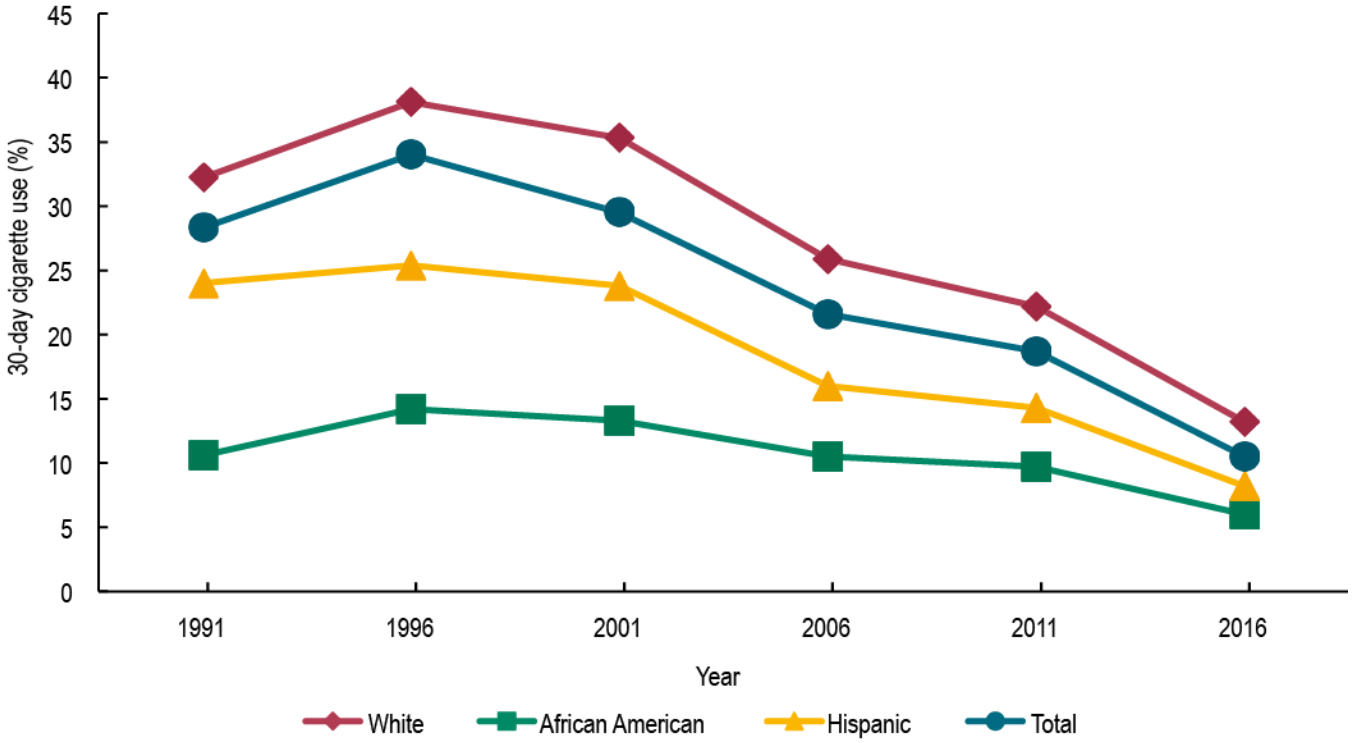
Race/Ethnicity	Total % (95% CI)	Males % (95% CI)	Females % (95% CI)
Total*	4.9 (4.7–5.1)	5.1 (4.8–5.4)	4.6 (4.3–5.0)
Not Hispanic or Latino*	5.3 (5.1–5.6)	5.6 (5.2–5.9)	5.1 (4.7–5.4)
White	6.3 (6.0–6.7)	6.5 (6.1–7.0)	6.1 (5.6–6.6)
Black/African American	2.7 (2.3–3.1)	3.1 (2.5–3.9)	2.2 (1.7–2.9)
American Indian/Alaska Native	7.0 (4.9–10.0)	6.5 (3.5–11.7)	7.7 (4.8–12.0)
Native Hawaiian or Other Pacific Islander	3.4 (1.4–8.1)	2.9 (1.3–6.1)	—
Asian*	1.6 (1.1–2.5)	2.0 (1.2–3.4)	1.2 (0.6–2.5)
Chinese	0.7 (0.2–2.1)	—	0.8 (0.2–3.4)
Filipino	0.9 (0.3–2.7)	0.3 (0.1–1.3)	1.6 (0.4–6.1)
Japanese	—	—	—
Asian-Indian	3.2 (1.7–6.1)	4.8 (2.3–10.0)	1.9 (0.5–7.1)
Korean	3.1 (1.4–7.1)	—	—
Vietnamese	—	—	—
Hispanic*	3.4 (3.0–3.9)	3.6 (3.0–4.2)	3.2 (2.7–3.9)
Mexican	3.6 (3.1–4.2)	4.0 (3.3–4.9)	3.2 (2.5–4.0)
Puerto Rican	3.3 (2.4–4.6)	3.1 (1.9–4.8)	3.6 (2.3–5.6)
Central or South American	2.4 (1.6–3.7)	1.9 (1.1–3.4)	3.0 (1.7–5.2)
Cuban	2.5 (1.1–5.4)	4.0 (1.6–9.8)	1.1 (0.3–4.2)

Notes: Based on responses to the question, "During the past 30 days, have you smoked part or all of a cigarette?" Respondents who chose "Yes" were classified as current smokers. CI = confidence interval. Em dash (—) = low precision; no estimate reported.
*Totals include data on respondents who reported being of racial or ethnic subgroups not shown and on respondents who reported being of more than one racial or ethnic group.
Source: Based on data from the National Survey on Drug Use and Health 2013–2015.[20]

An analysis of the 2014 NYTS reported cigarette smoking prevalence for high school and middle school students by race/ethnicity.[19] Current cigarette use among middle school youth was highest among Hispanic youths (2.8%) followed by non-Hispanic whites (2.1%), and non-Hispanic blacks (1.0%). Among high school students, non-Hispanic whites had the highest cigarette smoking prevalence (10.2%), followed by Hispanics (9.0%) and non-Hispanic blacks (5.7%).

Data from the Monitoring the Future (MTF) study show that 30-day smoking prevalence estimates have consistently been higher among non-Hispanic white and Hispanic high school seniors compared with African American high school seniors (Figure 2.4). Between 1991 and 2016, smoking prevalence declined from 32.2% to 13.2% among non-Hispanic white high school seniors, from 24.0% to 8.2% among Hispanic high school seniors, and from 10.6% to 6.0% among African American high school seniors.[21] These data show a narrowing of the difference in smoking prevalence for African American compared with non-Hispanic white and Hispanic youth. Data are not reported for other racial/ethnic groups due to small sample sizes.[22]

Figure 2.4 30-Day Prevalence of Cigarette Use Among U.S. 12th Graders, by Race/Ethnicity, 1991–2016



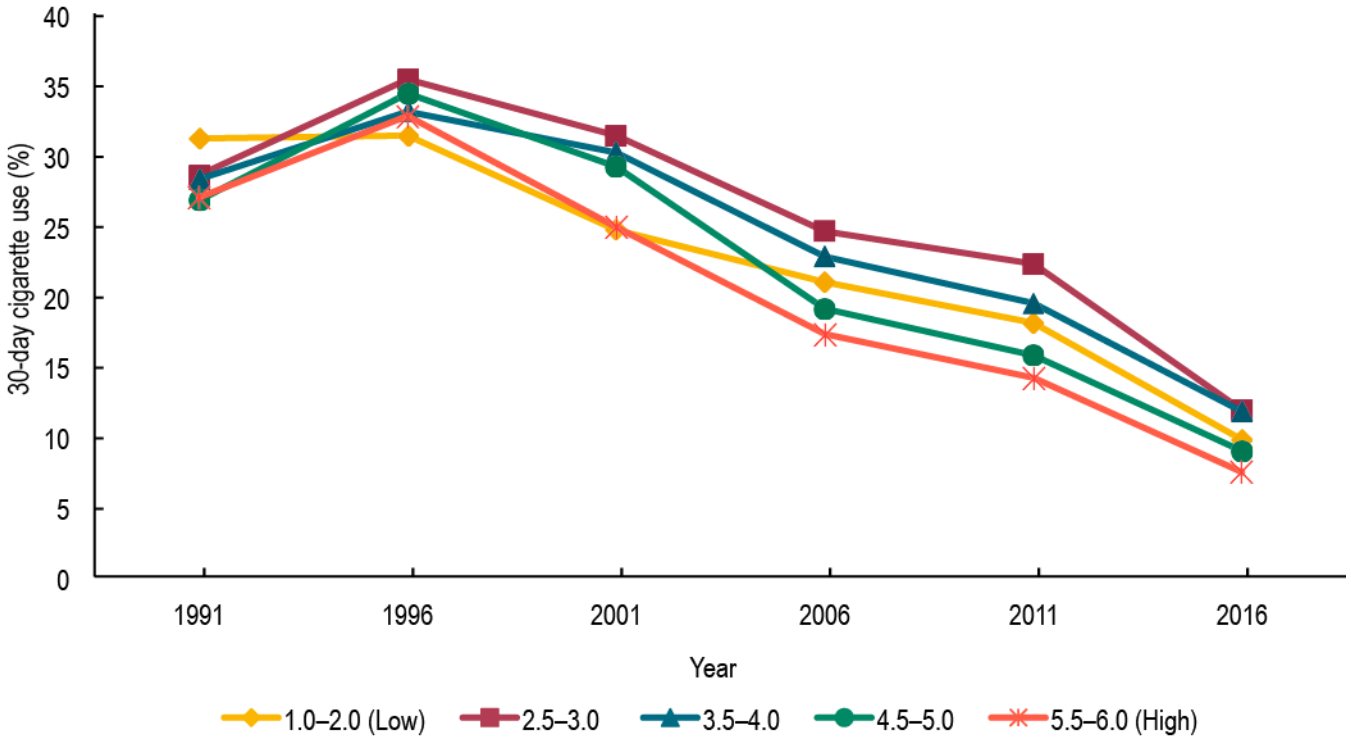
Source: Miech et al. 2016.[21]

Current Cigarette Smoking Among Youth, by SES

Parental educational attainment, often used as a proxy for SES, is also associated with disparities in youth smoking prevalence. As shown in Figure 2.5, data from the MTF study show that differences in youths' smoking prevalence by parental educational status have changed over time.[21] In 1991, smoking prevalence was fairly similar among youth whose parents were in the highest educational group compared to youths with parents in the lowest and second-lowest educational groups (27.1% compared with 31.3% and 28.7%, respectively). However, by 2006, differences in youth smoking prevalence by

parental educational attainment had increased, particularly between the second-lowest parental educational group (24.7%) and the highest educational group (17.4%). As of 2016, differences between these two groups appear to be converging (12.0% for the second- and third-lowest parental educational group and 7.6% for the highest educational group).[21]

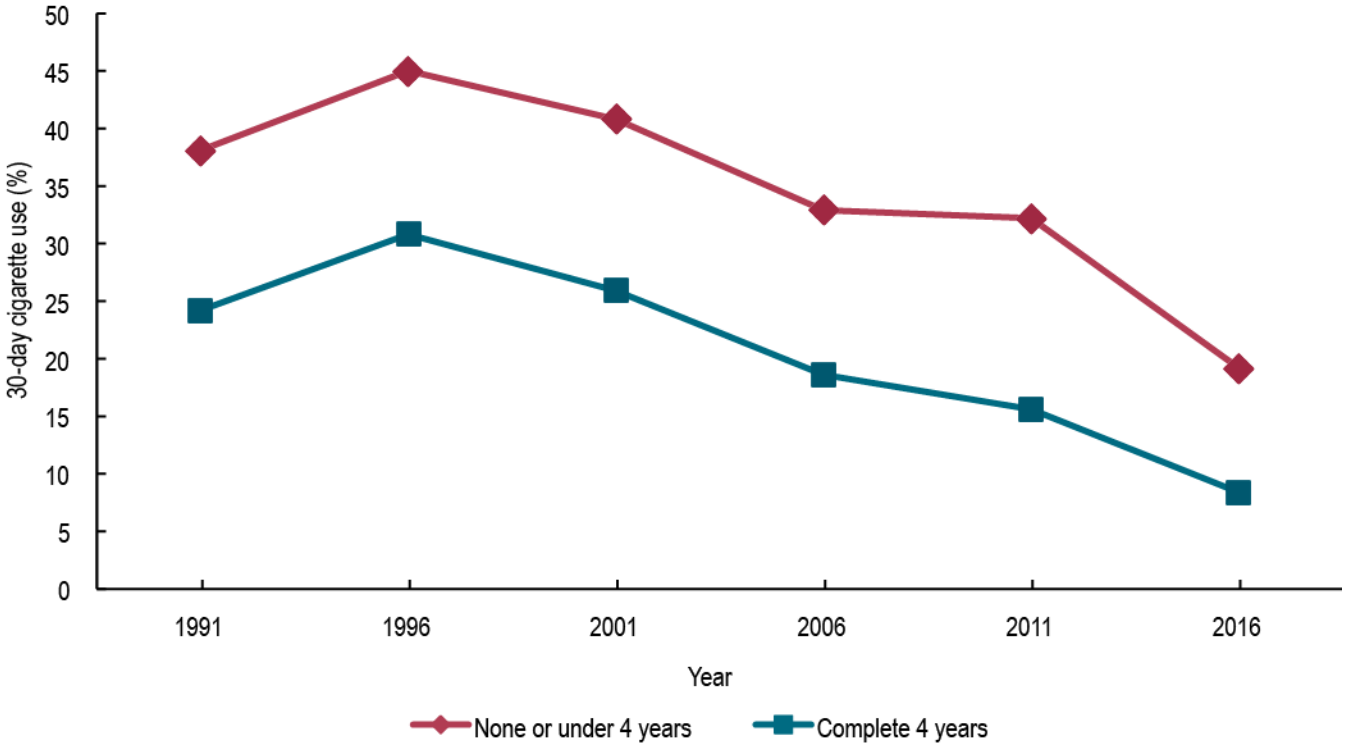
Figure 2.5 30-Day Prevalence of Cigarette Use Among 12th Graders, by Parental Educational Attainment, 1991–2016



Notes: Parental educational attainment was assessed by taking the average of the mother's reported education and the father's reported education and was categorized as follows: 1 = completed grade school or less, 2 = some high school, 3 = completed high school, 4 = some college, 5 = completed college, and 6 = graduate or professional school after college.
Source: Miech et al. 2016.[21]

Youths' own plans for educational attainment are also strongly associated with disparities in current smoking.[10] Data from MTF (Figure 2.6) show that between 1991 and 2016 there have been striking differences in the smoking prevalence of 12th graders who plan to pursue a 4-year college degree compared with those who do not. The difference in smoking prevalence increased slightly until 2011, after which it began to decrease.[21] Additionally, based on data from NSDUH (2006–2010), prevalence of current smoking among adolescent school dropouts ages 16–19 was far greater than that of adolescents of the same age who were currently enrolled in 12th grade (57.0% versus 18.6%, respectively).[10]

Figure 2.6 30-Day Prevalence of Cigarette Use Among 12th Graders, by College Plans, 1991–2016



Source: Miech et al. 2016.[21]

Some research suggests that the effects of SES on cigarette smoking among youths could be moderated by race, ethnicity, and cultural factors.[10] For example, data from the 1994–2002 National Longitudinal Study of Adolescent to Adult Health (Add Health) suggest that neighborhood poverty might be associated with smoking among non-Hispanic white but not black adolescents,[23] and an analysis of 1999–2008 MTF data by Bachman and colleagues[24] found that the effects of parental education on cigarette smoking were strongest among non-Hispanic whites compared with Hispanics and non-Hispanic blacks. The authors note that the weaker association between educational attainment and smoking among minority youth might be partially explained by the higher percentage of black and Hispanic youths whose parents are in the lower educational attainment categories.[24]

Current Cigarette Smoking Among Youth, by Sexual Orientation

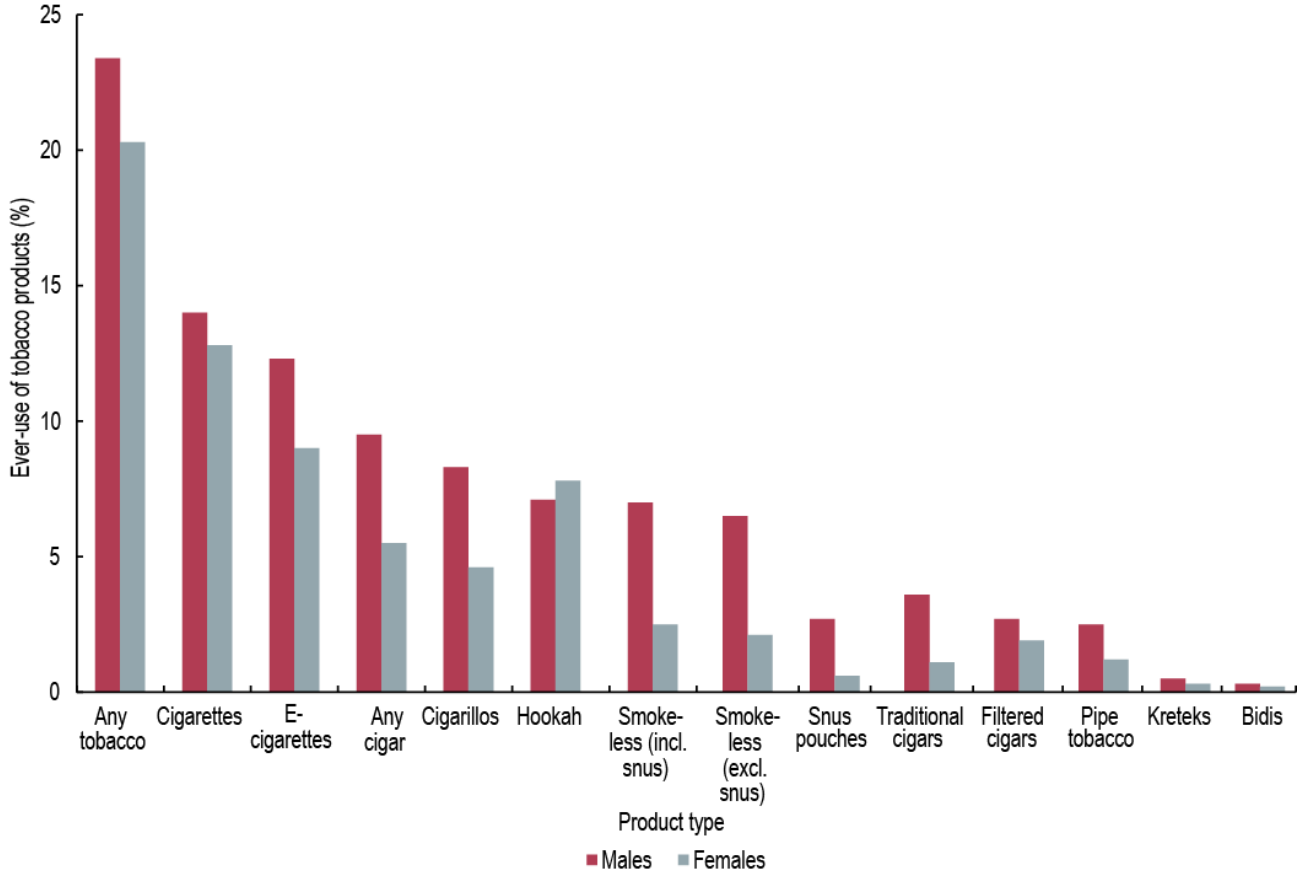
Differences in current smoking prevalence are also seen among adolescents by sexual orientation. Nationally representative data for lesbian, gay, and bisexual populations from the PATH study show that gay/lesbian and bisexual youths ages 14–17 have a significantly higher prevalence of cigarette smoking and of any tobacco use. Prevalence of cigarette use was highest among bisexual youth (20.1%; 95% CI 15.8–25.3), as was prevalence of any tobacco use (29.8%; 95% CI 24.4–35.8), compared to heterosexual youth (cigarette use: 5.8%; 95% CI 5.3–6.4) (any tobacco use: 11.8%; 95% CI 10.9–12.7).[25]

Use of Other Tobacco Products Among Youth

As novel tobacco products and marketing strategies emerge, and tobacco control policies alter the social environment, patterns of tobacco consumption among youth may become more complex and challenging to study. Other tobacco products discussed in this section include smokeless tobacco and combustible products such as cigars (including cigarillos and little cigars), hookah (waterpipe), and pipe tobacco. Another group of products has emerged more recently, often called electronic nicotine delivery systems (e.g., electronic cigarettes [e-cigarettes], e-hookah, vape pens, tank systems). These products are battery-powered devices designed to heat a liquid, which typically contains nicotine and a variety of flavors, into an aerosol for inhalation by the user.[26] Use of these and other tobacco products by youth may contribute to TRHD in the future.

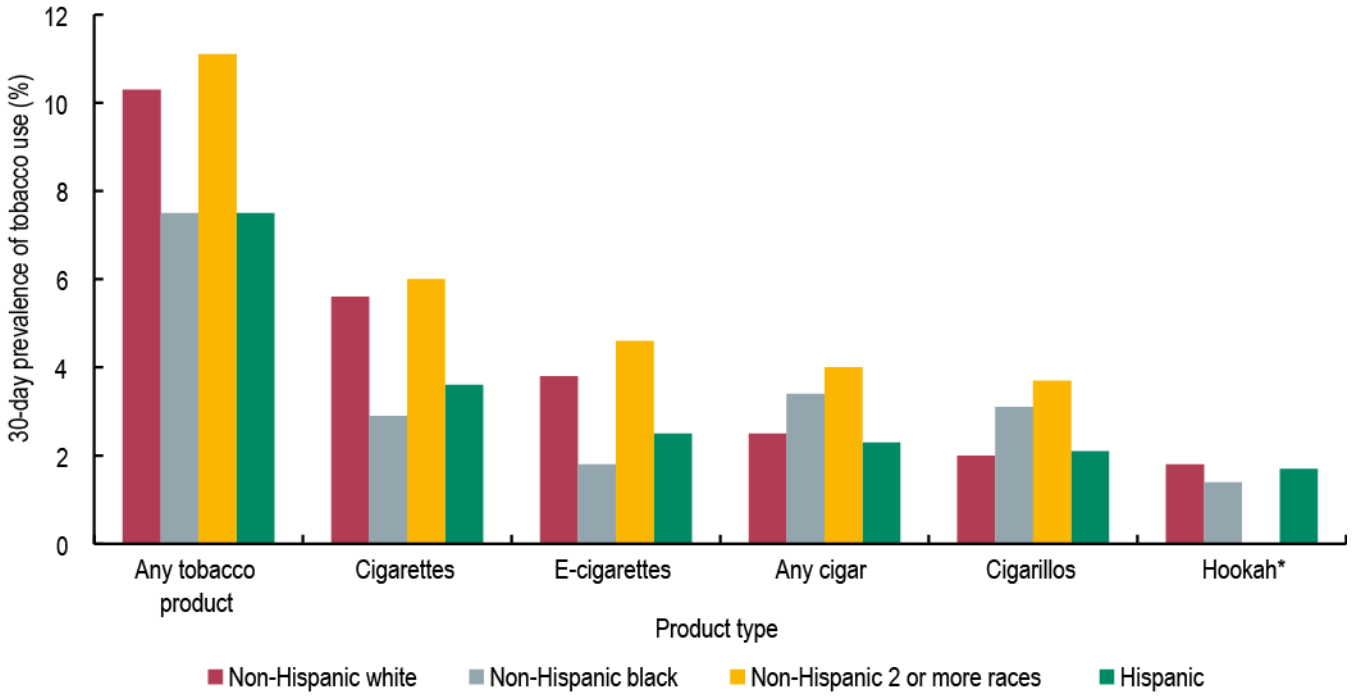
Data from the 2013-2014 PATH study show that patterns of using other tobacco products among youths (12–17 years) differ by sex, race/ethnicity, and sexual orientation. With the exception of hookah, males were more likely than females to ever use other tobacco products, including e-cigarettes, traditional cigars, cigarillos, smokeless tobacco, snus pouches, and pipe tobacco (Figure 2.7).[25] Non-Hispanic white youth and multiracial (≥2 races) youth reported the highest current use of any tobacco product, of cigarettes, and of e-cigarettes; multiracial youth also reported the highest current use of cigars and cigarillos (Figure 2.8). Non-Hispanic white youth reported the highest ever-use of smokeless tobacco and snus.[25]

Figure 2.7 Ever-Use of Tobacco Products, by Product Type and Sex, 2013-2014



Source: Kasza et al. 2017.[25]

Figure 2.8 30-Day Prevalence of Tobacco Product Use, by Product Type and Race/Ethnicity, 2013-2014



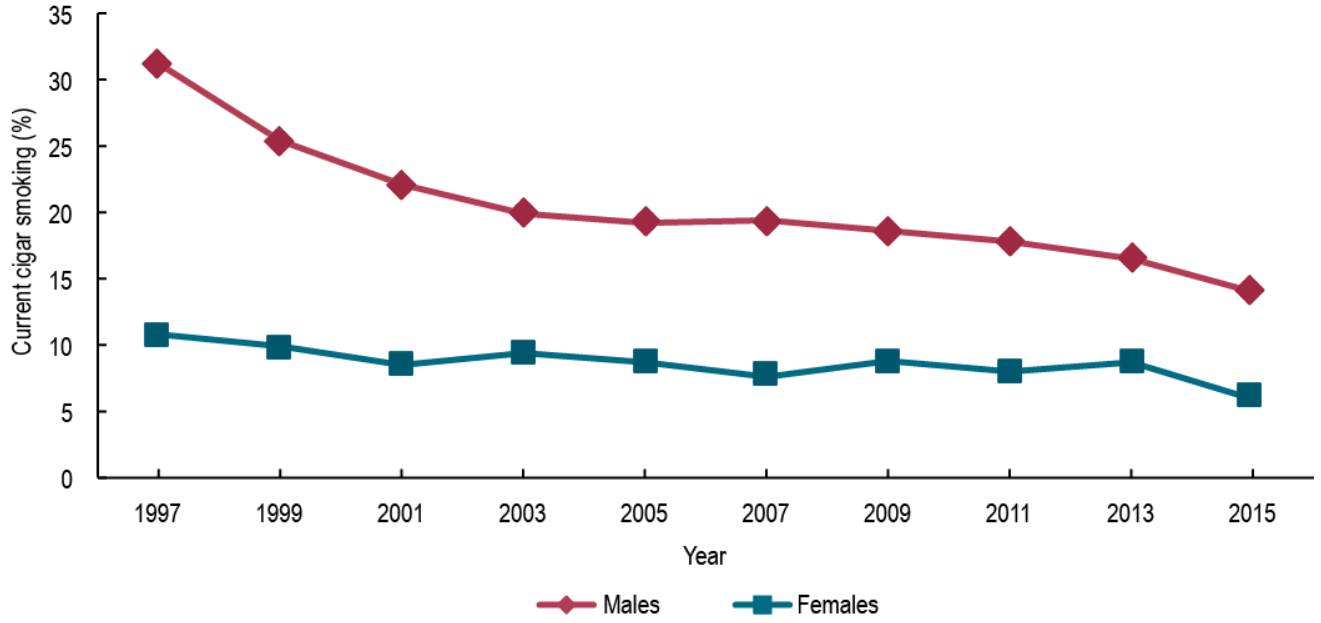
*Data not shown for hookah use by people who were non-Hispanic 2 or more races because the relative standard error was greater than 30%.
Source: Kasza et al. 2017.[25]

Gay/lesbian, and bisexual youth reported higher ever-use of any tobacco product compared to heterosexual youth, with the highest use among bisexual youth, according to 2013-2014 PATH study data. Gay/lesbian youth reported the highest prevalence of current e-cigarette use (13.4%).[25]

National Cancer Institute (NCI) Tobacco Control Monograph 9, *Cigars, Health Effects and Trends*, noted that "promotional activities for cigars have increased the visibility of cigar consumption, normalized cigar use, and broken down barriers to cigar use."[27,p.217] Research has shown that various cigar products are popular among youth and young adults.[28] According to NYTS data, past-month cigar use by youths in the United States increased during 2011-2012, declined for 2013-2014, and remained unchanged for 2014-2015.[29] Among high school students, the prevalence of current cigar use was similar across racial/ethnic groups: 8.3% among non-Hispanic whites, 8.8% among non-Hispanic blacks, and 8.0% among Hispanics.[29] Among middle school students, Hispanic youths had the highest proportion of current cigar use (2.9%), followed by non-Hispanic blacks (2.0%) and non-Hispanic whites (1.4%).[29] Other research shows that from 2012 to 2014 among middle and high school students, ever-use of cigars declined overall combined racial/ethnic groups (21.2% to 17.6%) and particularly among Hispanic youth (23.1% to 18.1%) and black youth (27.8% to 20.8%).[30]

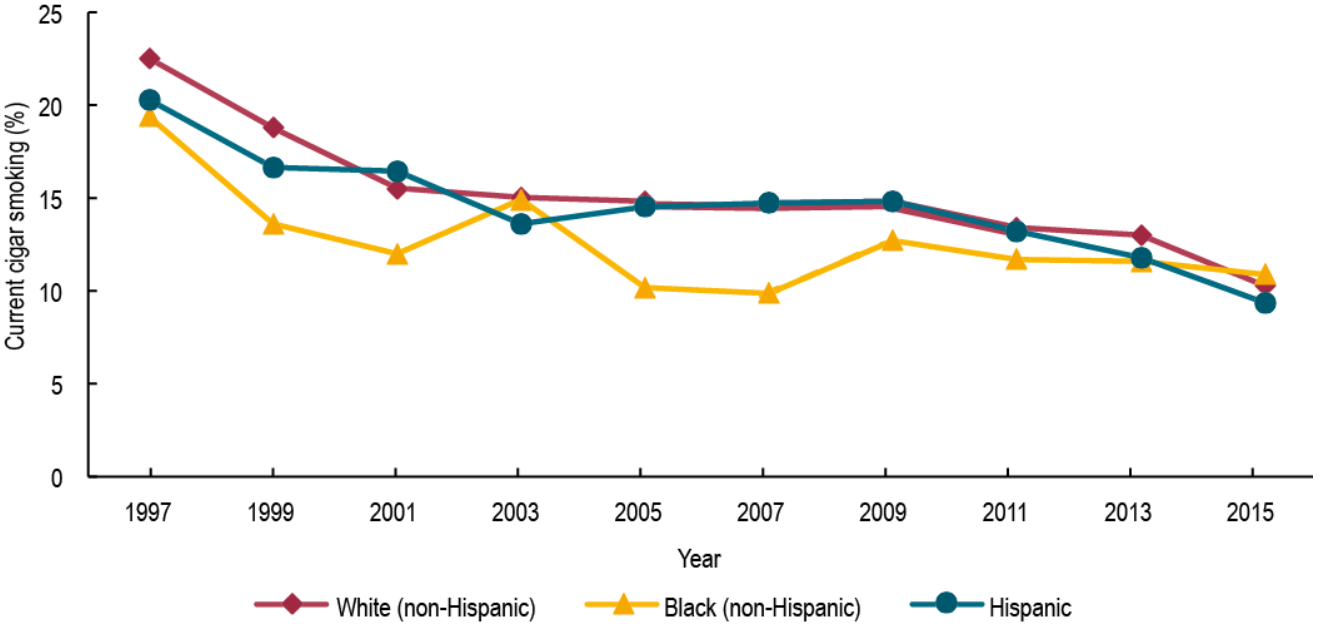
Results from the 1997–2015 YRBS show that cigar use was relatively stable among female high school students and generally declined among male students (Figure 2.9). Cigar use among Hispanic and non-Hispanic white high school students generally declined, whereas a fluctuating pattern was seen among non-Hispanic black high school students (Figure 2.10).[10,16,17,31]

Figure 2.9 Prevalence of Current Smoking of Any Type of Cigar Among U.S. High School Students, by Sex, 1997–2015



Notes: Based on responses to the question, "During the past 30 days, on how many days did you smoke cigars, cigarillos, or little cigars?" Respondents who reported that they had smoked any of these tobacco products on 1 or 2 days or more were classified as current cigar smokers.
Sources: Data based on the National Youth Risk Behavior Survey 1997–2009[10]; 2011[31]; 2013[16]; 2015.[17]

Figure 2.10 Prevalence of Current Smoking of Any Type of Cigar Among U.S. High School Students, by Race/Ethnicity, 1997–2015



Notes: Based on responses to the question, "During the past 30 days, on how many days did you smoke cigars, cigarillos, or little cigars?" Respondents who reported that they had smoked any of these tobacco products on 1 or 2 days or more were classified as current cigar smokers.
Sources: Data based on the National Youth Risk Behavior Survey 1997–2009[10]; 2011[31]; 2013[16]; 2015.[17]

Flavored Tobacco Products

Research has found that youth and young adult cigar smokers are more likely than cigar smokers in other age groups to report having a usual cigar brand that is flavored.[28] Other research shows that the majority of youth tobacco product ever-users report that their first tobacco product was flavored.[32] Overall, 70% of middle and high school students who were current users of any tobacco product—or nearly 3.3 million youth—reported past-month use of at least one flavored tobacco product.[33] Among high school students overall, e-cigarettes (8.8%) were the most commonly used flavored tobacco product, followed by hookah (6.0%), cigars (5.3%), menthol cigarettes (5.0%), any smokeless tobacco (4.1%), and pipes (0.7%).[33] Non-Hispanic black students reported lower use of flavored tobacco products than non-Hispanic whites, except that use of flavored cigarettes was highest among non-Hispanic black students (see the section "Menthol Cigarette Smoking Among Youth and Young Adults").

Cigarette Smoking Prevalence Among Young Adults

Current Cigarette Smoking Among Young Adults, by Race/Ethnicity

The patterns of current cigarette smoking among young adults ages 18–25 are generally similar to the patterns among youths. Smoking prevalence is highest among American Indian/Alaska Native young adults (41.8%), followed by non-Hispanic whites (33.3%), Native Hawaiian and Other Pacific Islanders (24.5%), non-Hispanic blacks/African Americans (23.2%), Hispanics (22.5%), and Asians (15.1%). Among Asian American young adults, smoking prevalence is highest among Koreans (21.0%) and lowest among Chinese (10.0%). Among Hispanic young adults, smoking prevalence is highest among Cubans (25.7%) and lowest among Central or South Americans (19.6%) (Table 2.3).[20]

Table 2.3 Prevalence of Current Cigarette Smoking Among U.S. Young Adults Ages 18–25, by Race/Ethnicity and Sex, 2013–2015

Race/Ethnicity	Total % (95% CI)	Males % (95% CI)	Females % (95% CI)
Total*	28.6 (28.0–29.1)	33.5 (32.7–34.3)	23.6 (22.9–24.3)
Not Hispanic or Latino*	30.2 (29.6–30.8)	34.7 (33.8–35.6)	25.7 (24.9–26.4)
White	33.3 (32.6–34.1)	37.6 (36.6–38.7)	29.0 (28.1–29.9)
Black/African American	23.3 (22.0–24.7)	29.2 (27.2–31.2)	17.9 (16.4–19.6)
American Indian/Alaska Native	41.8 (36.3–47.6)	41.3 (33.2–50.0)	42.4 (35.3–49.9)
Native Hawaiian or Other Pacific Islander	24.5 (19.1–30.8)	27.8 (19.7–37.8)	20.5 (14.3–28.5)
Asian*	15.1 (13.4–16.9)	20.3 (17.7–23.3)	9.9 (8.0–12.2)
Chinese	10.0 (7.4–13.3)	13.1 (9.3–18.1)	7.1 (4.2–11.8)
Filipino	20.4 (15.7–26.1)	25.7 (18.5–34.4)	15.6 (10.0–23.5)
Japanese	—	—	—
Asian-Indian	12.7 (9.7–16.5)	17.7 (13.1–23.5)	7.1 (3.6–13.4)
Korean	21.0 (15.1–28.3)	30.6 (20.9–42.3)	12.0 (6.9–19.8)
Vietnamese	13.8 (9.4–19.9)	19.0 (12.2–28.4)	9.0 (4.5–17.2)

Table 2.3 continued

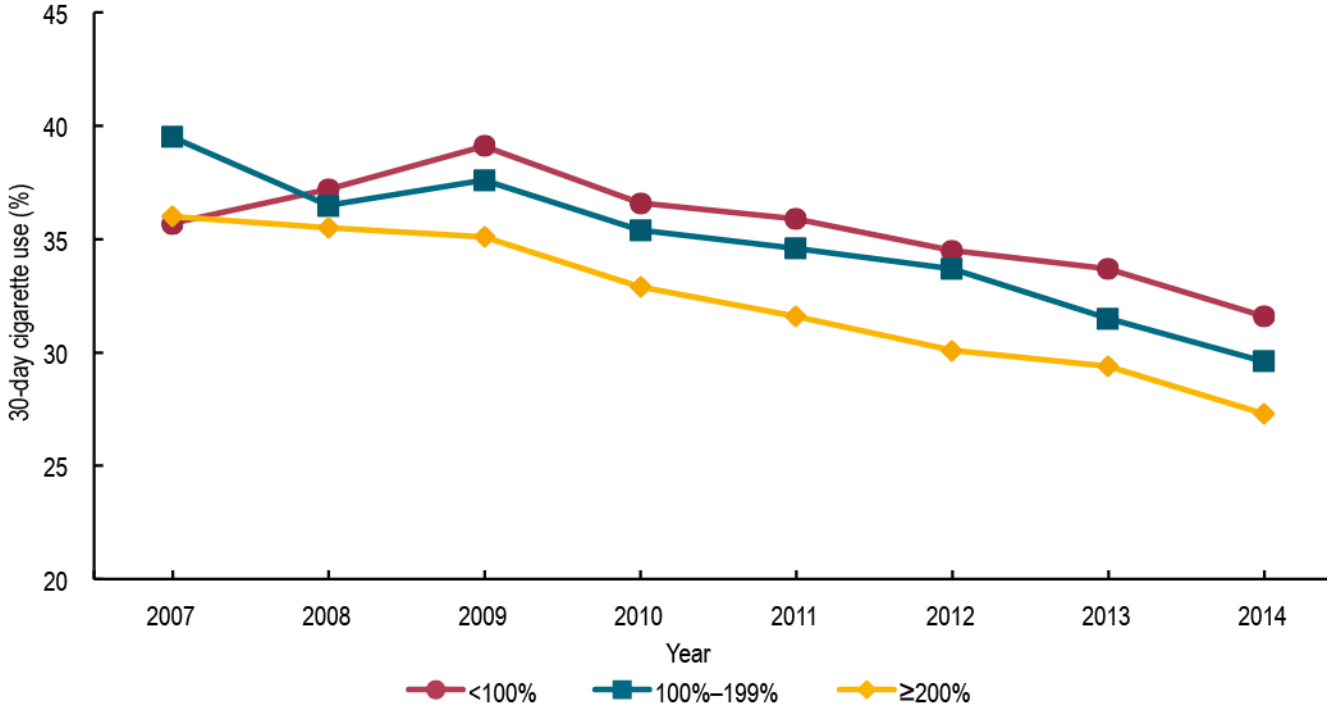
Race/Ethnicity	Total % (95% CI)	Males % (95% CI)	Females % (95% CI)
Hispanic*	22.5 (21.4–23.7)	29.2 (27.5–31.0)	15.5 (14.2–16.8)
Mexican	22.2 (20.8–23.6)	29.7 (27.6–32.0)	14.2 (12.7–15.8)
Puerto Rican	24.5 (21.3–28.0)	27.2 (22.5–32.5)	21.6 (17.6–26.2)
Central or South American	19.6 (17.0–22.4)	25.9 (22.0–30.2)	12.3 (9.7–15.5)
Cuban	25.7 (19.8–32.6)	34.9 (24.9–46.5)	17.0 (11.9–23.8)

Notes: Based on responses to the question, "During the past 30 days, have you smoked part or all of a cigarette?" Respondents who chose "Yes" were classified as current smokers. CI = confidence interval. Em dash (—) = low precision; no estimate reported.
*Totals include data on respondents who reported being of racial or ethnic subgroups not shown and on respondents who reported being of more than one racial or ethnic group.
Source: Based on data from the National Survey on Drug Use and Health 2013–2015.[20]

Current Cigarette Smoking Among Young Adults, by SES

Data from the NSDUH show a slow but steady decline in smoking prevalence for all three poverty level groups between 2009 and 2014 (Figure 2.11). However, there was no narrowing of the gap in prevalence between young adults living at 200% above the poverty threshold compared with those living at less than 100% of the poverty line during this period.

Figure 2.11 30-Day Prevalence of Cigarette Use Among Adults Ages 18–25, by Poverty Level, 2007–2014

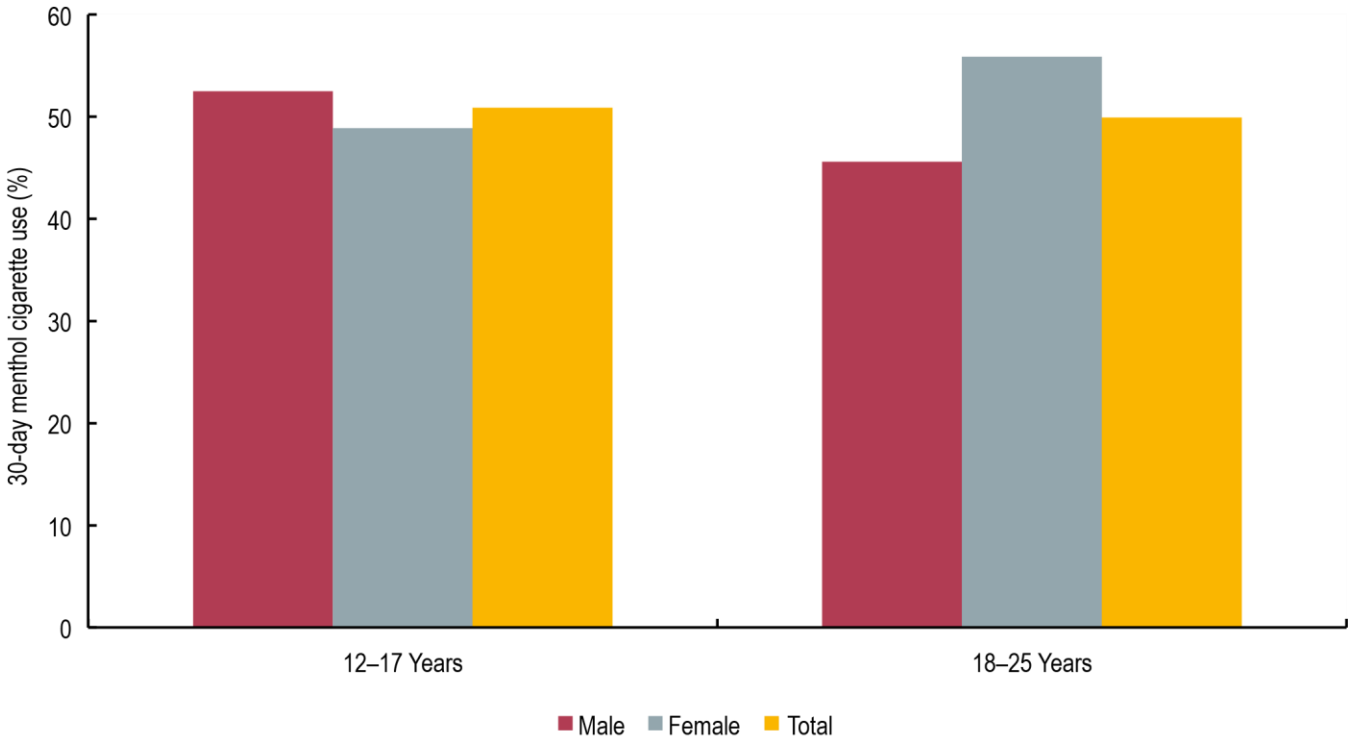


Source: Based on data from the National Survey on Drug Use and Health 2007–2014.[20]

Menthol Cigarette Smoking Among Youth and Young Adults

Various studies have documented the use of menthol cigarettes by youth and young adults over time. For example, an analysis of NSDUH data from 2007 to 2010 found that more than half (51.7%) of new cigarette smokers smoked menthol cigarettes, compared with 41.7% of new smokers between 2004–2006.[34] Additionally, NSDUH data shows that, in 2015, an estimated 50.9% of youth ages 12–17 and 49.9% of young adults ages 18–25 reported smoking menthol cigarettes; among young adults aged 18–25, menthol smoking prevalence was highest among females (56%) (Figure 2.12).[20] An analysis of 2014 NYTS data found that for current cigarette-smoking youth, prevalence of menthol cigarette smoking was 70.5% among non-Hispanic blacks, 52.3% among Hispanics, and 51.4% among non-Hispanic whites.[33]

Figure 2.12 30-Day Prevalence of Menthol Cigarette Smoking Among Youth and Young Adults, by Age Group and Sex, 2015



Source: Based on data from the National Survey on Drug Use and Health 2015.[20]

Giovino and colleagues[35] estimated the use of menthol cigarettes using data from the 2004–2010 NSDUH, adjusting for self-reported menthol status for selected brands either exclusively menthol or non-menthol. Their study found that menthol cigarette smoking was more common among youth (56.7% among 12- to 17-year-olds) and young adults (45.0% among 18 to 25-year-olds) than among older adults (30.5%–34.7%). Additionally, between 2004 and 2010, the rate of non-menthol cigarette use decreased among youth, but the rate of menthol cigarette use remained constant. Among young adults, non-menthol cigarette use also declined, but menthol smoking rates increased. The authors concluded that "young people are heavy consumers of mentholated cigarettes. Progress in reducing youth smoking has likely been attenuated by the sale and marketing of mentholated cigarettes, including emerging varieties of established youth [non-mentholated] brands."[35,p.28]

A number of factors may contribute to the high rates of menthol cigarette use among youth. As discussed in chapter 4, menthol produces a variety of sensory effects. Beyond serving as a flavorant, the multisensory effects of menthol—which acts on the olfactory, gustatory, and trigeminal systems—may appeal to youth, and may contribute to the addictive potential of cigarettes.[36,37] Current young adult menthol smokers may perceive menthol cigarettes as safer than non-menthol cigarettes.[38] An analysis of tobacco industry documents found that cigarette companies carefully researched the menthol segment of the market and tracked menthol cigarette use by age, sex, and race; this analysis concluded that "menthol is a prominent design feature used by cigarette manufacturers to attract and retain new, younger smokers."[39,p.ii12]

Adult Tobacco Use Behaviors

Cigarette Smoking Prevalence Among Adults

Table 2.4 presents National Health Interview Survey (NHIS) prevalence data on adult smoking between 1994 and 2015 by sex, race/ethnicity, and SES. As with youth, current smoking among adults has decreased substantially over time. In 1994, 25.5% of U.S. adults reported current smoking,[40] compared with 15.1% in 2015.[41] Although declines in smoking prevalence have occurred among adults of both sexes, from all racial/ethnic groups, and at all poverty and educational levels, disparities in smoking prevalence remain. For example, in 2015, males continued to have a higher prevalence of current smoking than females (16.7% versus 13.6%).[41]

Table 2.4 Prevalence of Current Cigarette Smoking Among U.S. Adults Age 18 and Older, by Sex, Race/Ethnicity, Poverty Status, and Educational Attainment, 1994–2015

Category	1994	1998	2002	2006	2010	2011	2012	2013	2014	2015
Total	25.5	24.1	22.5	20.8	19.3	19.0	18.1	17.8	16.8	15.1
Sex										
Male	28.2	26.4	25.2	23.9	21.5	21.6	20.5	20.5	18.8	16.7
Female	23.1	22.0	20.0	18.0	17.3	16.5	15.8	15.4	14.8	13.6
Race/Ethnicity*										
White	26.3	25.0	23.6	21.9	21.0	20.6	19.7	19.4	18.2	16.6
Black	27.2	24.7	22.4	23.0	20.6	19.4	18.1	18.3	17.5	16.7
Hispanic/Latino	19.5	19.1	16.7	15.2	12.5	12.9	12.5	12.1	11.2	10.1
American Indian/ Alaska Native	42.2	40.0	40.8	32.4	31.4	31.5	21.8	26.1	29.2	21.9
Asian/Pacific Islander	13.9	13.7	—	—	—	—	—	—	—	—
Asian	—	—	13.3	10.4	9.2	9.9	10.7	9.6	9.5	7.0
Multiple race	—	—	—	—	25.9	27.4	26.1	26.8	27.9	20.2
Poverty Status										
At or above	24.1	23.5	22.2	20.4	18.3	17.9	17.0	16.2	15.2	13.9
Below	34.7	32.3	32.9	30.6	28.9	29.0	27.9	29.2	26.3	26.1
Unknown	28.8	22.5	19.7	18.3	16.0	15.0	13.6	16.0	16.4	10.5
Educational Attainment†										
≤8	23.7	21.9	19.3	17.4	16.2	15.0	15.2	15.4	13.7	14.4
9–11	38.2	36.8	34.1	35.4	33.8	34.6	32.1	33.2	29.5	31.6
0–12 (no degree)	—	—	27.6	26.7	25.1	25.5	24.7	24.2	22.9	24.2
12 (no degree)	—	—	31.0	25.6	21.7	25.1	24.7	19.7	25.7	26.3
GED certificate	—	—	42.3	46.0	45.2	45.3	41.9	41.4	43.0	34.1

Table 2.4 continued

Category	1994	1998	2002	2006	2010	2011	2012	2013	2014	2015
12 (degree)	29.8	27.4	25.6	23.8	23.8	23.8	23.1	22.0	21.7	19.8
Associate's degree	—	—	21.5	21.2	18.8	19.3	17.9	17.8	17.1	16.6
Some college	—	—	23.1	22.7	23.2	22.3	20.9	20.9	19.7	18.5
Undergraduate degree	—	—	12.1	9.6	9.9	9.3	9.1	9.1	7.9	7.4
Graduate degree	—	—	7.2	6.6	6.3	5.0	5.9	5.6	5.4	3.6
13–15	25.7	24.6	—	—	—	—	—	—	—	—
≥16	12.3	11.3	—	—	—	—	—	—	—	—

Notes: Em dash (—) = data not collected in a category for a particular year. GED = general educational development certificate. Current smokers include those who smoked 100 cigarettes per day and who smoked every day or some days. Data were not collected in 1996. NHIS was redesigned in 1997, and trend analysis and comparison with data years before 1997 should be conducted with caution.

*All racial/ethnic groups are non-Hispanic except those categorized as Hispanic. In 1997 the Office of Management and Budget changed its data collection guidelines to require that Native Hawaiian and Other Pacific Islander data be collected separately from Asian. Limited data were collected on American Indians/Alaska Natives, and data for a single year could be unstable or unreliable due to a small sample size. Data on current smoking among Native Hawaiians/Pacific Islanders are not reported.

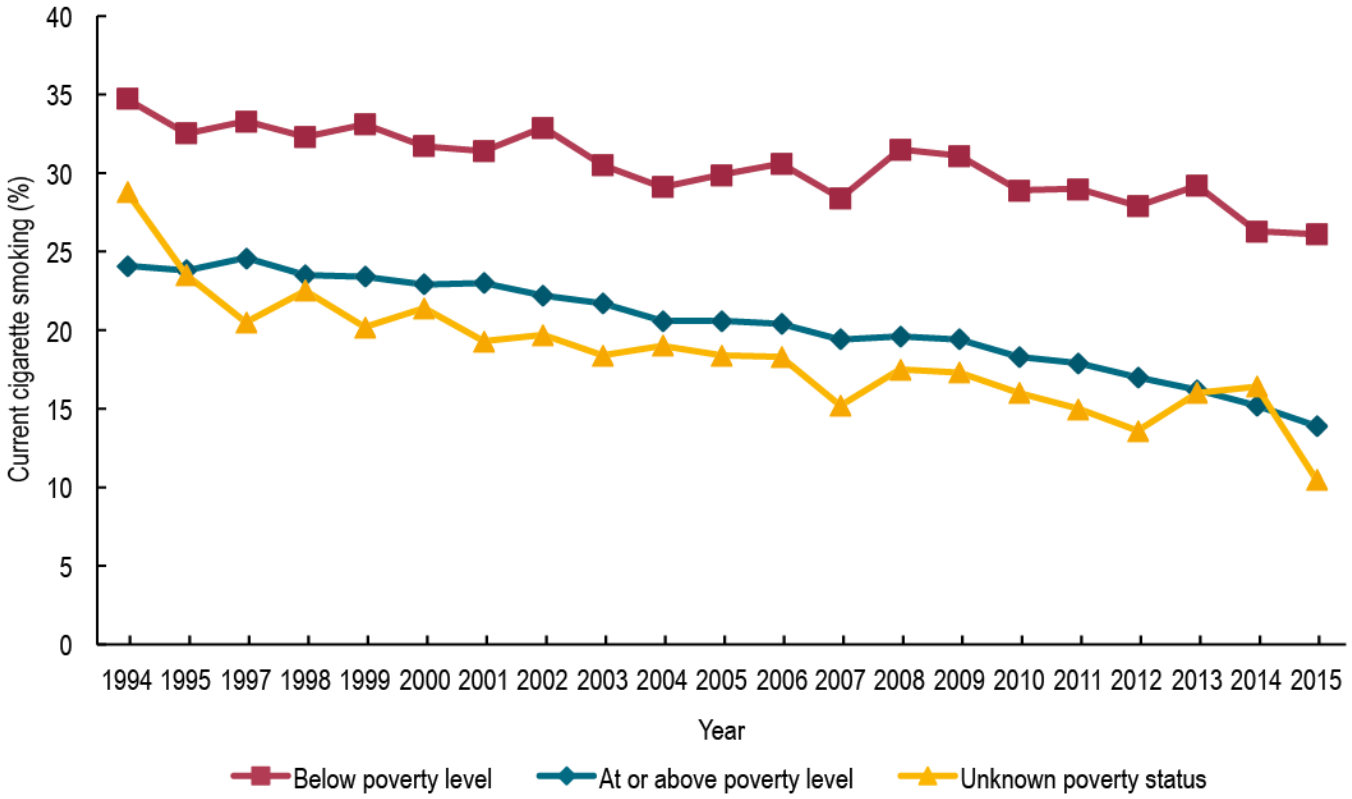
†Additional categories were added to education in 1999.

Source: Based on data from the National Health Interview Survey 1994–2015.[2,40,41,44,47,132–135]

Current Smoking Among Adults, by Race/Ethnicity and SES

American Indian/Alaska Native adults have long had the highest prevalence of current smoking of all U.S. racial/ethnic groups.[9,42] NHIS data show that in 2015, 21.9% of American Indian/Alaska Native adults reported current smoking compared with 16.7% of blacks, 16.6% of non-Hispanic whites, 10.1% of Hispanics, and 7.0% of Asian adults (Table 2.4).[41] Significant disparities in cigarette smoking also persist among adults with lower educational attainment compared with those with higher educational attainment. Additionally, smoking prevalence has long been higher among adults living below the poverty level, and is declining at a slower pace among these adults, compared with those living at or above poverty (Figure 2.13). In 1994, 34.7% of adults living below the poverty level smoked cigarettes, compared to 24.1% of those at or above poverty.[40] In 2015, 26.1% of adults living below the poverty line smoked cigarettes compared to 13.9% of adults living at or above poverty.[41]

Figure 2.13 Current Smoking Among U.S. Adults, by Poverty Status, 1994–2015



Note: Data not reported for 1996. NHIS was redesigned in 1997, and trend analysis and comparison with data prior to 1997 should be conducted with caution.
Source: Based on data from the National Health Interview Survey 1994–2015.[2,40,41,44,47,132–145]

Current Smoking Among Adults, by Sexual Orientation

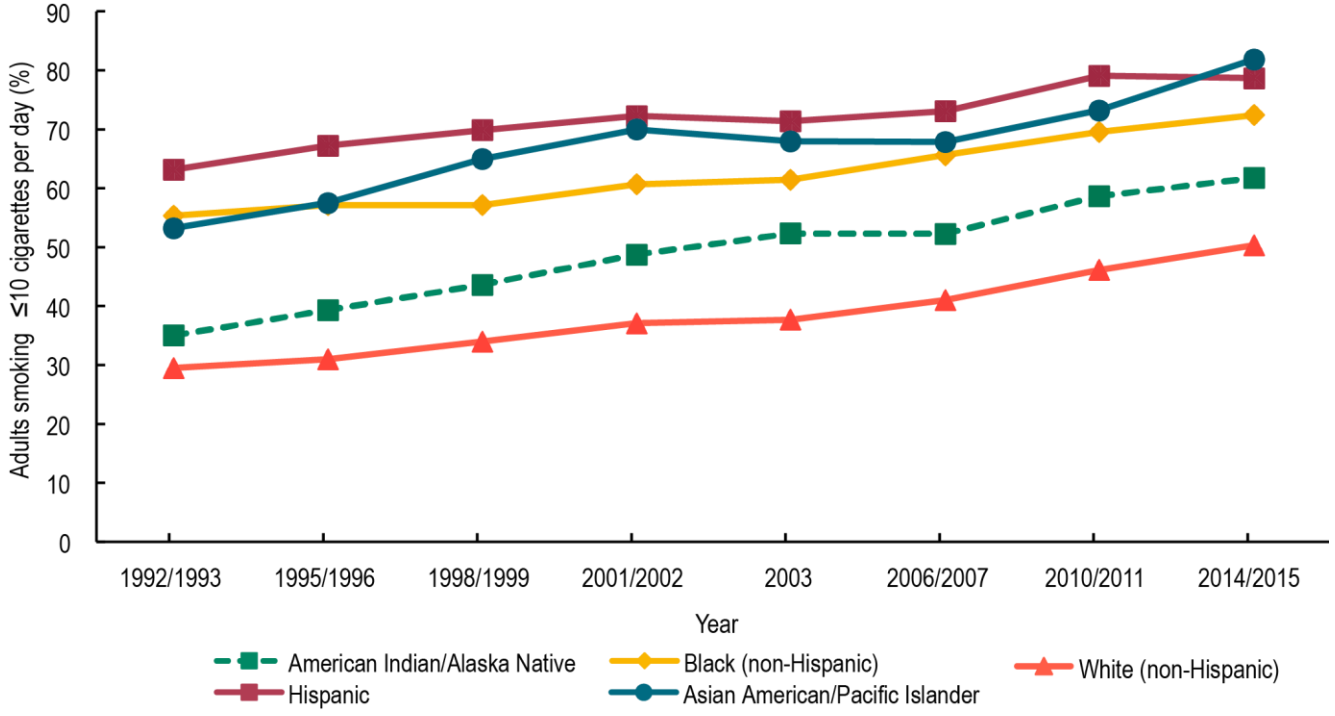
The first nationally representative study to show a higher prevalence of smoking among LGBT adults ages 18 and older compared with heterosexual/straight adults (32.8% vs. 19.5%), used data from the 2009-2010 NATS.[43] Data on sexual orientation has been collected by NHIS beginning in 2013, and by NSDUH beginning in 2015. NHIS data show that, as with other populations, the prevalence of smoking among lesbian, gay, and bisexual individuals has declined over time. However, significantly higher

smoking levels are found among lesbian, gay, and bisexual populations for both men and women, compared to heterosexuals. In 2013, 26.6% of individuals who identified as lesbian, gay, or bisexual reported current smoking compared to 17.6% of heterosexuals.[44] In 2015, 20.6% of individuals who identified as lesbian, gay, or bisexual reported smoking compared to 14.9% of heterosexuals.[41] NSDUH data show a similar trend: 32.8% of those who identified as bisexual and 30.4% of lesbians reported smoking within the past month compared to 20.7% of heterosexuals.[20] Data collected by the PATH study in 2013-2014 show a higher prevalence of current smoking among gay, lesbian, and bisexual adults compared to heterosexual adults; smoking was highest among bisexual individuals (32.6%).[25]

Adult Cigarette Consumption—Light and Intermittent Smoking

Recent trends in smoking patterns and behaviors indicate a higher prevalence of light smoking (variously defined as less than 9 or 10 cigarettes/day) and intermittent (non-daily) smoking in the United States and abroad.[45,46] National-level data show declines in the percentage of daily smokers who smoke 30 or more cigarettes per day (from 12.6% in 2005 to 6.8% in 2015) and a significant increase in the proportion of daily smokers who smoke 9 or fewer cigarettes per day (from 16.4% in 2005 to 25.1% in 2015).[41,47] As shown in Figure 2.14, the trend toward light (≤10 cigarettes/day) smoking is seen among all racial/ethnic groups, but historically, the prevalence of light smoking has been higher among racial/ethnic minority groups compared with non-Hispanic whites. A similar pattern is also seen among low-income adult light smokers, by race/ethnicity; nearly 80% of low-income Hispanic smokers consume 10 or fewer cigarettes per day.[13]

Figure 2.14 Percentage of U.S. Adults Smoking ≤10 Cigarettes per Day, by Race/Ethnicity, 1992/1993–2014/2015



Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 1992/1993–2014/2015.[13]

Other research also indicates that the trend toward increased light and intermittent smoking is significantly more pronounced among smokers from racial/ethnic minority groups than non-Hispanic white smokers.[46,48,49] Data from the 2003 TUS-CPS show that African American and non-Hispanic white smokers reported a higher prevalence of current daily smoking (49.2% and 43.9%, respectively), regardless of smoking intensity (assessed by cigarettes per day [CPD]) compared with Hispanic/Latino and Asian/Pacific Islander smokers (36.9% and 38.1%, respectively).[49] However, the prevalence of current intermittent smoking was significantly higher among African Americans (15.9%), Asians/Pacific Islanders (16.1%), and Hispanics/Latinos (20.8%) compared with non-Hispanic whites (8.5%). In other research modeling the odds of being a light (≤10 CPD) and/or intermittent smoker (adjusting for other characteristics), Hispanics (odds ratio [OR] 5.38; 95% CI 4.38–6.61), non-Hispanic African Americans (OR 3.67; 95% CI 2.92–4.60), and people of other races (OR 1.81; 95% CI 1.40–2.34), were much more likely to be light/intermittent smokers compared to non-Hispanic whites. A similar pattern was observed among light/daily smokers but with more attenuated risk estimates.[46]

Smoking Duration Among Adults

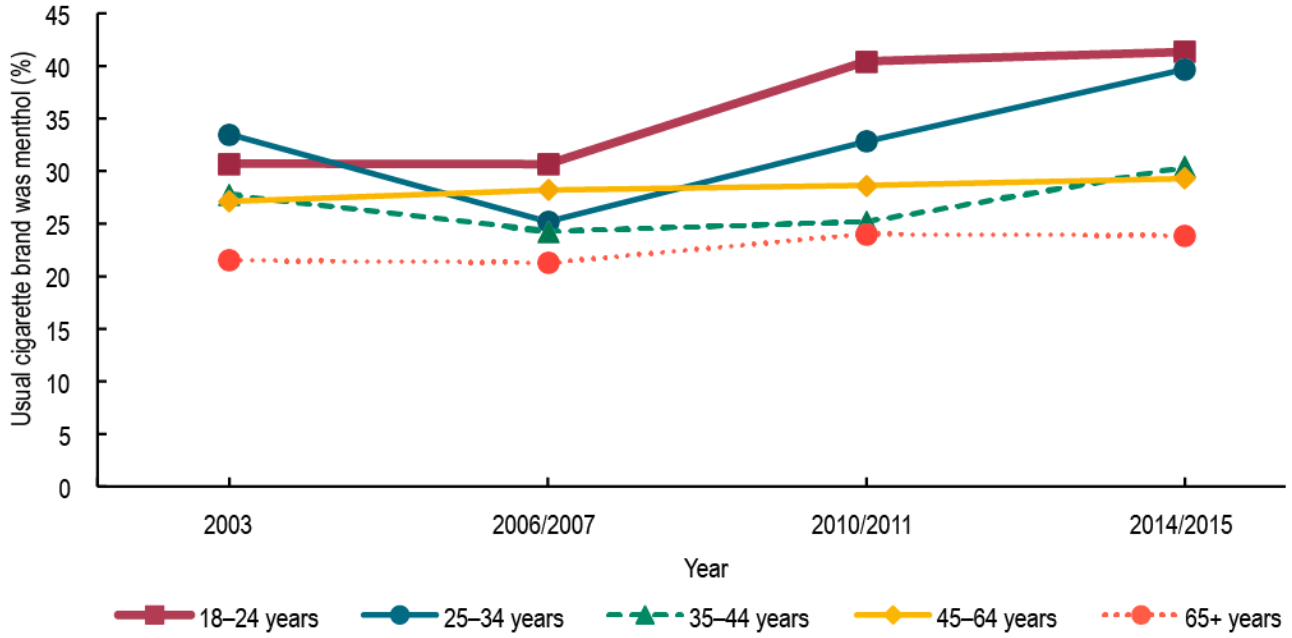
Using data from the 2003 and 2006/2007 TUS-CPS, Siahpush and colleagues[50] found substantial differences in smoking duration by race/ethnicity, SES, and other demographic factors. The highest median duration of smoking was found among American Indians (32 years), followed by blacks and "other" races (30 years), whites (28 years), and Hispanics (24 years). The authors also found a strong gradient based on SES; for example, the median duration of smoking among people living at or below the poverty level was 40 years, compared with 22 years among people living at least three times above poverty. Median duration of smoking also differed by occupation, employment status, age at smoking initiation, and region of the country.[50]

Menthol Cigarette Smoking Among Adults

The NSDUH has collected nationally representative data on menthol cigarette smoking among people age 12 years and older annually since 2004. The TUS-CPS has collected nationally representative data on menthol cigarette smoking among adults periodically since 2003. In addition, questions about menthol cigarette use have sometimes been included in other survey instruments. (See chapter 4 for information about menthol as an ingredient in cigarettes.)

Based on the four nationally representative surveys of U.S. adults (NHANES [1999–2010], NHIS [2005 and 2010], TUS-CPS [2003 and 2006/2007], and NSDUH [2000–2009]), it was estimated that approximately 26%–30% of all adult smokers smoke menthol-flavored cigarettes.[51] TUS-CPS data from 2014/2015 showed that 32.5% of U.S. smokers reported typically smoking menthol-brand cigarettes.[13] According to NSDUH data, 35.5% of adult smokers age 26 or older reported current smoking of menthol cigarettes in 2015.[20] An analysis by Villanti and colleagues[52] comparing 2008–2010 and 2012–2014 NSDUH data found that while the prevalence of current menthol cigarette smoking increased across all age groups, the largest increase was among 26- to 34-year-olds (34.6% in 2008–2010 to 43.9% in 2012–2014). TUS-CPS data (Figure 2.15) also show increased use of menthol cigarettes since 2006/2007, especially among young adults (18–24) and adults ages 25 to 34.

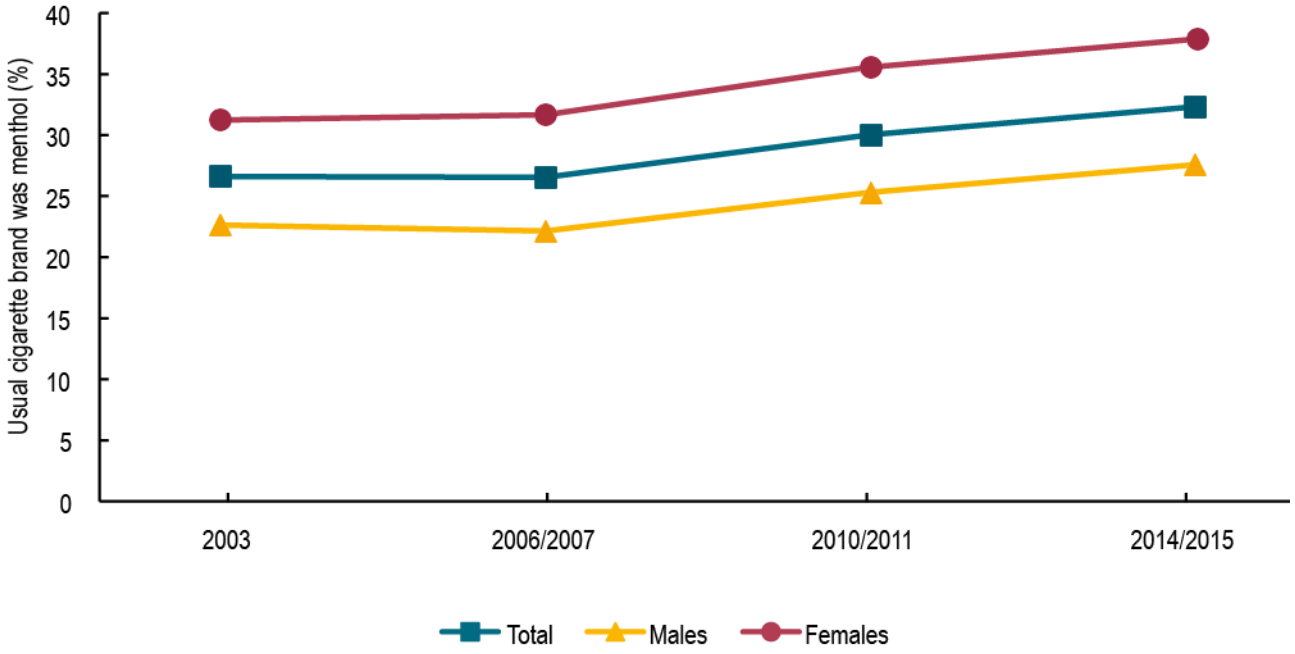
Figure 2.15 Percentage of U.S. Adult Smokers Whose Usual Cigarette Brand Was Menthol, by Age, 2003–2014/2015



Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 2003–2014/2015.[13]

An upward trend in menthol cigarette smoking is seen for both sexes, with a higher prevalence of menthol cigarette smoking among women than men (38.1% vs. 27.7% in 2014/2015) (Figure 2.16).[13]

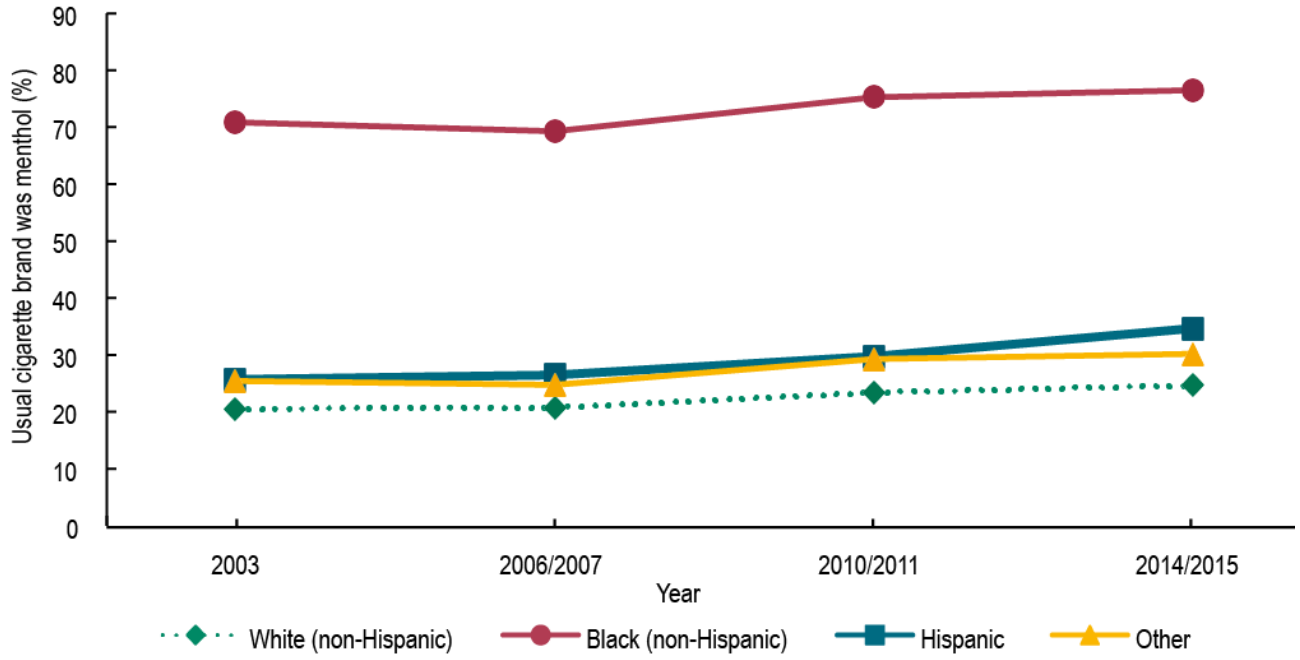
Figure 2.16 Percentage of U.S. Adult Smokers Whose Usual Cigarette Brand Was Menthol, by Sex, 2003–2014/2015



Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 2003–2014/2015.[13]

As shown in Figure 2.17, African Americans consistently report the highest prevalence of menthol cigarette smoking of any racial/ethnic group. TUS-CPS data from 2014/2015 suggest that the prevalence of menthol cigarette smoking may be increasing among Hispanics (Figure 2.17).

Figure 2.17 Percentage of U.S. Adult Smokers Whose Usual Cigarette Brand Was Menthol, by Race/Ethnicity, 2003–2014/2015



Source: Based on data from the Tobacco Use Supplement to the Current Population Survey 2003–2014/2015.[13]

The most recent TUS-CPS data also show differences in current use of menthol cigarettes by employment and educational attainment. In 2014/2015, 42.0% of current smokers who were unemployed smoked menthol cigarettes, compared with 32.2% of smokers who were employed and 30.8% of smokers not in the labor force. Additionally, service industry workers who currently smoke reported a higher prevalence of menthol cigarette smoking (41.0%) than smokers who were white-collar workers (31.3%), blue-collar workers (30.6%), or workers in other industries (30.9%). Smokers with 9–11 years of education reported a higher prevalence of current menthol cigarette smoking (35.4%) than a high school degree (31.6%), those with some college (31.5%), a college education or greater (24.7%), or 8 years or less of education (23.5%).[13]

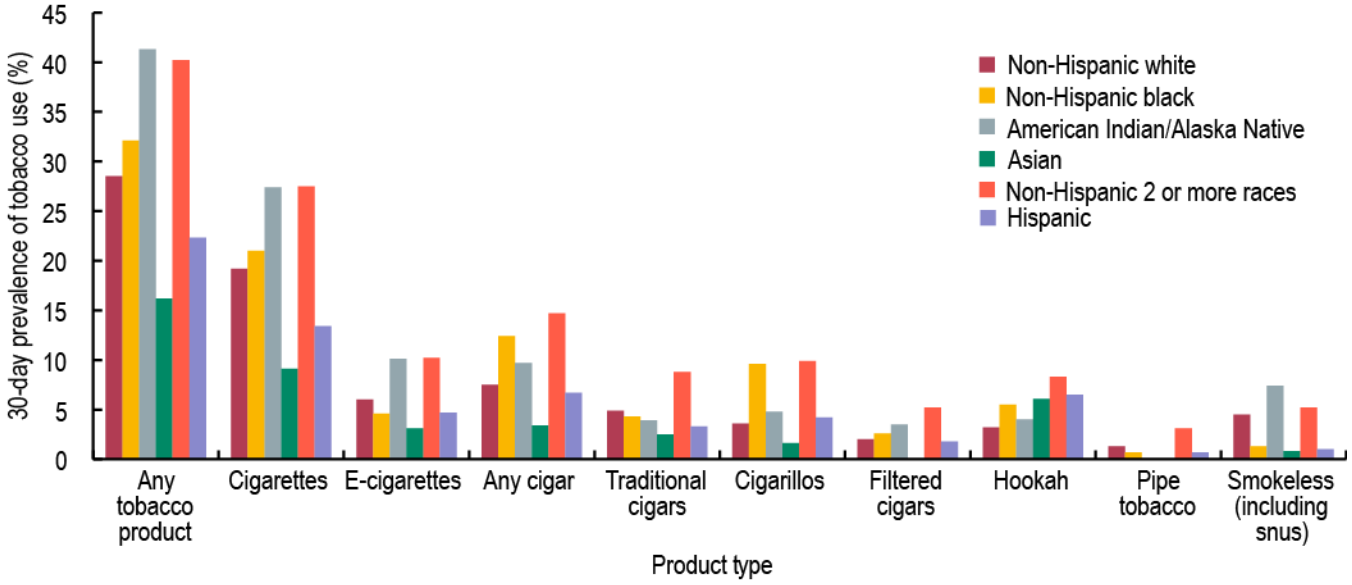
Use of Other Tobacco Products Among Adults

Use of other (non-cigarette) tobacco products is common among adults. Data from the PATH study found that, in 2013-2014, nearly 28% of adults were current users of at least one tobacco product, and approximately 40% of these adults currently used multiple tobacco products. Use of traditional cigarettes and e-cigarettes was the most common tobacco product combination.[25] According to the 2013-2014 PATH study data, young adults (18- to 24-years-old) reported a higher proportion of ever-use of e-cigarettes, cigarillos, hookah, filtered cigars, and snus pouches compared to adults age 25 years and older. Young adults also reported more frequent use of e-cigarettes, cigarillos, hookah, and

smokeless tobacco compared to older adults. Men were more likely than women to use any type of non-cigarette product.

As shown in Figure 2.18, the prevalence of current use of other tobacco products (as well as use of cigarettes) varies by race/ethnicity. Adults reporting multiple races had the highest rates of use of many different tobacco products, except for smokeless products. Among people of a single race, American Indian/Alaska Natives had the highest use of e-cigarettes (10.1%); non-Hispanic blacks had the highest use of cigarillos (9.6%); and Asians had the highest use of hookah (6.1%). Bisexual adults reported the highest current use of any type of cigars (6.2%, traditional cigars; 11.4%, cigarillos; 5.3%, filtered cigars). Prevalence of current e-cigarette use was around 12% for both bisexual and gay adults. Prevalence estimates of use of any type of tobacco product were higher among lesbian, gay, and bisexual adults than among heterosexual adults.[25]

Figure 2.18 30-Day Prevalence of Tobacco Product Use Among U.S. Adults, by Product Type and Race/Ethnicity, 2013-2014



Source: Kasza et al. 2017.[25]

Adults with a General Educational Development (GED) certificate reported the highest current use of e-cigarettes (10.6%), any type of cigar (13.1%), and smokeless tobacco (5.9%) compared to people of other education levels.[25]

Electronic Cigarettes

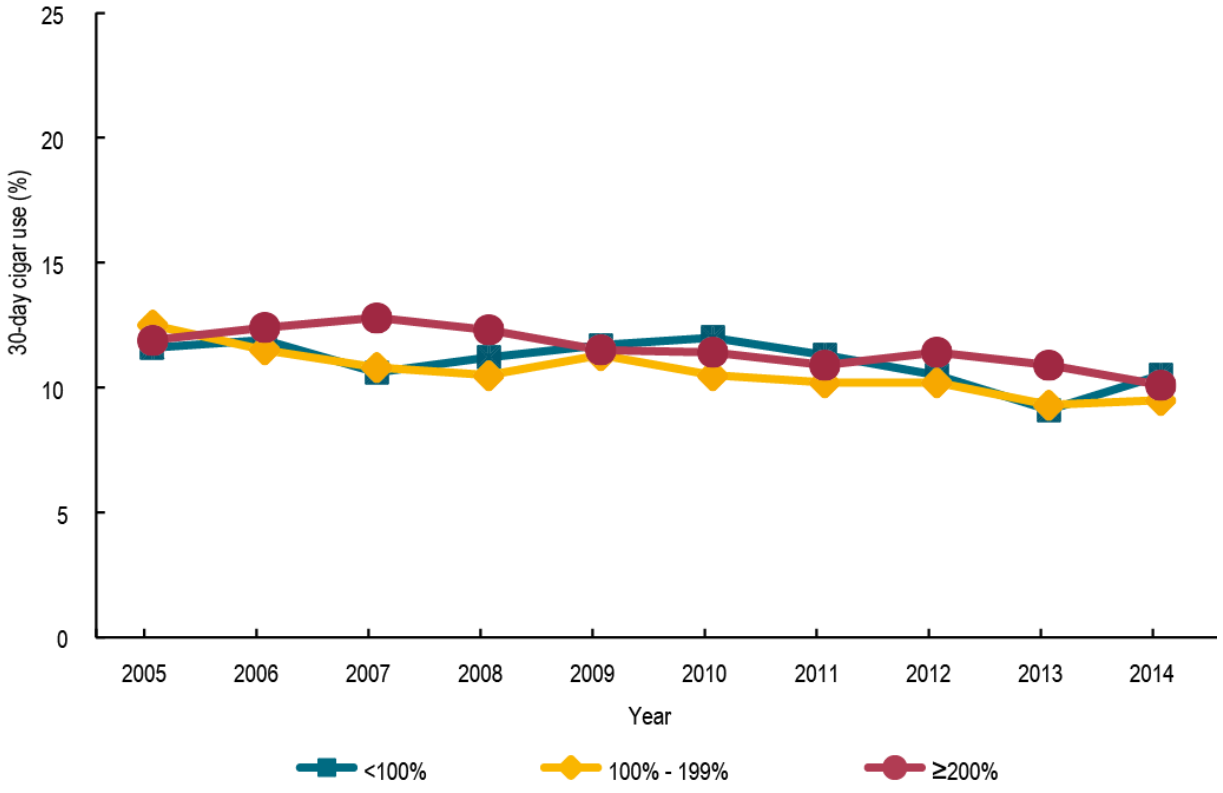
Both awareness and use of e-cigarettes have increased over time among adults: HealthStyles survey data for the years 2010–2013 found increased awareness (from 40.9% to 79.7%), ever-use (3.3% to 8.5%), and current use (1.0% to 2.6%).[53] NATS, conducted in 2013-2014, found that overall, 3.3% of adults age 18 or older used e-cigarettes every day or some days.[54] Use was higher among men (4.0%) than women (2.8%) and was also higher among young adults ages 18–24 (5.5%) than other age groups. E-cigarette use was also high among people with a GED (8.0%), and lesbian, gay, or bisexual

individuals (6.9%). Analysis of data from the 2013-2014 PATH study found that overall, 5.5% of adults were current e-cigarette users; of these, 21.3% reported using e-cigarettes on a daily basis.[55]

Cigars

In the United States, there is wide variation in the landscape of cigar products in relation to cigar type (traditional/premium/large cigars, cigarillos, little filtered cigars [LFC]), flavor, pack size, and brand.[28] Data from the 2012-2013 NATS show that 7.3% of U.S. adults smoke cigars "every day," "someday," or "rarely." Of these, 61.8% reported usually smoking cigarillos; 19.9%, premium cigars; and 18.4%, LFCs.[56] A majority of male and female cigar smokers reported cigarillos as their usual cigar type (61.6% and 59.4%, respectively); 23.9% of men reported premium cigars as their usual cigar type, and LFCs were the usual cigar type of 35.3% of women compared to 14.5% of men. Additionally, 72.1% of adults ages 18–29 reported cigarillos as their usual cigar type, but 15.1% of people in this age group smoked premium cigars and 12.8% smoked LFCs. Differences in cigar type are also found by race/ethnicity: 82.6% of non-Hispanic blacks reported cigarillos as their usual type, whereas 26.7% of non-Hispanic whites reported premium cigars as their usual type. Generally, adults with higher educational levels and annual household incomes had a lower prevalence of usual use of cigarillos and of LFCs and a higher prevalence of usual use of premium cigars. The prevalence of LFCs as the usual type was higher among lesbian, gay, and bisexual adults (35.6%) than among heterosexual adults (17.6%).[53] Figure 2.19 shows NSDUH data on trends in cigar use among young adults (18–25 years) by poverty level; these data reveal a generally decreasing trend.[20]

Figure 2.19 30-Day Prevalence of Cigar Use Among Young Adults Ages 18–25, by Poverty Level, 2005–2014



Source: Based on data from the National Survey on Drug Use and Health 2005–2014.[20]

Flavored Tobacco Products

Non-cigarette tobacco products are increasingly common and available in a growing number of flavors.[57] Research analyzing data from the 2013-2014 NATS found that reported prevalences of using flavored tobacco products in the past month by U.S. adults were: hookah, 82.3%; e-cigarette, 68.2%; smokeless tobacco, 50.6%; cigar, 36.2%; and pipe smoking, 25.8%.[58] Specific flavors varied by product type, but overall, the most commonly used flavors were menthol or mint; clove, spice, or herb; fruit; alcohol; and candy, chocolate, or other sweet flavors. Disparities in flavored tobacco product use were observed by age, sex, income, education, and sexual orientation, with more use of flavored products among young adults (ages 18–24), women, gay/lesbian and bisexual individuals, and people of less income or education. Among e-cigarette users, non-Hispanic blacks reported the highest prevalence of flavored use (87.5%).[58]

Quitting and Cessation Behaviors Among Adults

Quit attempts and smoking cessation behaviors vary by racial/ethnic group and SES. As discussed in the 1998 Surgeon General's report *Tobacco Use Among U.S. Racial/Ethnic Minority Groups*[59] and elsewhere, more white ever-smokers than African American ever-smokers report successfully quitting for at least 30 days.[59–61] Data from the 2015 NHIS show that non-Hispanic black adult smokers report greater interest in quitting smoking (72.8%; 95% CI 68.2–77.4) than Asians (69.6%; 95% CI 59.5–79.8), non-Hispanic whites (67.5%; 95% CI 65.0–70.0), Hispanics (64.7; 95% CI 61.9–72.8), and American Indians/Alaska Natives (55.6%; 95% CI 35.8–75.4).[62] The highest rate of past-year quit attempts was made by Asians (69.4%; 95% CI 62.1–76.7), followed by non-Hispanic blacks (63.4%; 95% CI 59.0–67.9), Hispanics (56.2%; 95% CI 51.6–60.9), non-Hispanic whites (53.3%; 95% CI 50.8–55.7), and American Indians/Alaska Natives (52.1%; 95% CI 32.1–72.2). However, rates of recent smoking cessation (for 6 months or more during the past year) were lower among non-Hispanic blacks (4.9%; 95% CI 3.2–6.6) compared with Asians (17.3%; 95% CI 10.1–24.5), Hispanics (8.2%; 95% CI 5.5–10.9), and non-Hispanic whites (7.1%; 95% CI 6.0–8.2).[62]

Trinidad and colleagues[49] conducted an in-depth examination of quitting and cessation behaviors across U.S. racial/ethnic groups using data from the 2003 TUS-CPS. Among current daily smokers, they found that members of racial/ethnic minority groups were significantly less likely than non-Hispanic whites to report a quit attempt lasting at least 1 day in the past year. Only 58.6% (±2.3) of African Americans, 59.6% (±5.8) of Asians/Pacific Islanders, and 60.3% (±3.1) of Hispanics/Latinos reported a quit attempt that lasted 1 day or longer in the past year, compared with 69.4% (±1.0) of non-Hispanic whites. Among current intermittent smokers, the rate of quit attempts was even lower across racial/ethnic groups, and significantly lower among Hispanic/Latino smokers compared with members of other racial/ethnic groups. In this same study, multivariable analyses found that African American smokers were only about 50% as likely to achieve smoking cessation for at least 6 months compared with non-Hispanic whites (OR = 0.51; 95% CI 0.36, 0.72), after age, sex, education, income, and nicotine dependence were controlled for. No statistically significant differences in 6-month smoking cessation were reported for Asians/Pacific Islanders or Hispanic/Latino smokers compared with non-Hispanic whites. Trinidad and colleagues[49] also found that the prevalence of former smoking among ever-smokers was lower among African Americans (30.4% ± 1.6), Hispanics (36.6% ± 1.8), and Asians/Pacific Islanders (39.8% ± 3.6) compared with non-Hispanic whites (42.9% ± 0.6); however, the difference was statistically significant only for African American and Hispanic smokers.

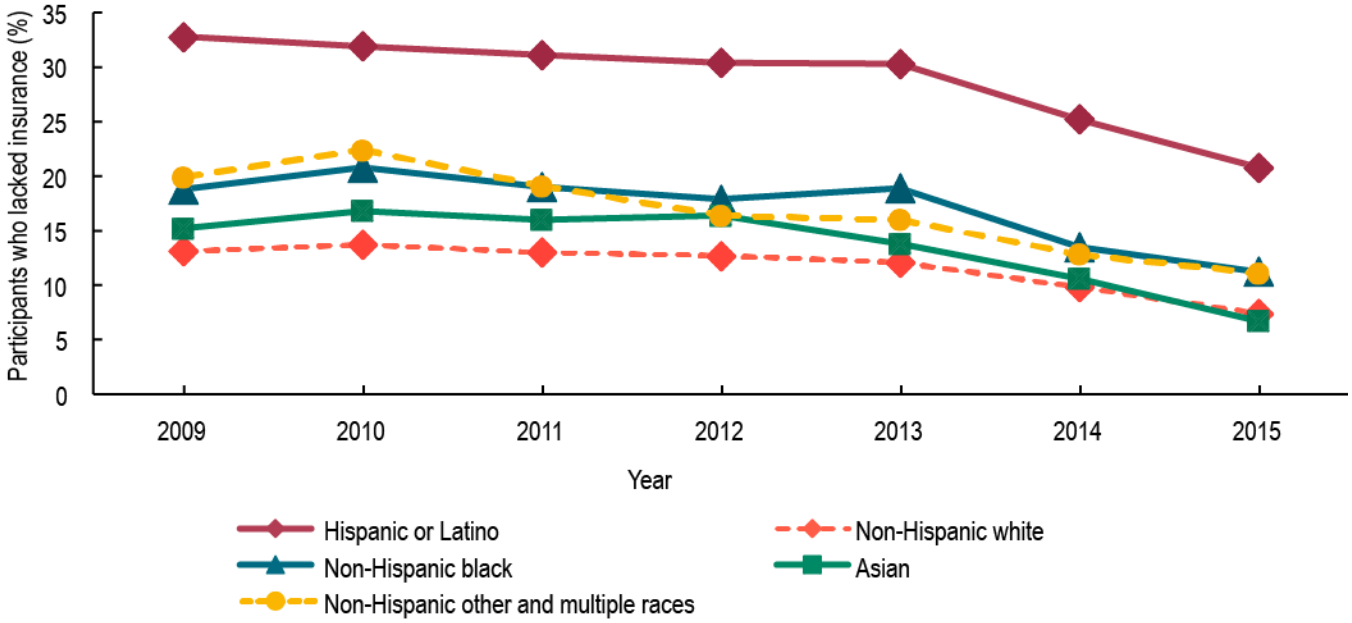
Several nationally representative studies show that low-income smokers are less likely to quit than higher income smokers.[49,63,64] Trinidad and colleagues[49] conducted multivariable analyses using data from the 2003 TUS-CPS and found that those with annual household incomes two times below the U.S. Census Bureau poverty threshold were significantly less likely than more advantaged smokers to achieve 6 months of smoking abstinence. Data from the 2015 NHIS did not show differences in quitting interest, recent quit attempts, or smoking cessation by poverty status, but did find differences by health insurance coverage. Recent smoking cessation was higher among smokers with private insurance (9.4%; 95% CI 7.9–10.9), compared with smokers covered through Medicaid (5.9%; 95% CI 4.1–7.7) and those who were uninsured (5.2%; 95% CI 3.3–7.0).[62]

National-level data also show significant disparities in quitting and smoking cessation behaviors by educational attainment.[49,63,65] In an analysis of 2003 TUS-CPS data, Trinidad and colleagues[49] found that smokers with a college degree were 1.7 times (95% CI 1.39–2.12) more likely to report a 6-month smoking cessation period than those without a high school diploma. Reid and colleagues[63] also reported that smokers with higher education were more likely to intend to quit, make a quit attempt, and be abstinent for at least 1 month or 6 months. Data from the 2015 NHIS show that a lower percentage of smokers with less than a high school education report recent smoking cessation (4.4%; 95% CI 2.7–6.1) compared with those with an associate degree (9.2%; 95% CI 7.4–15.0) and those with an undergraduate college degree (11.2%; 95% CI 7.4–15.0).[62]

Insurance Coverage of Tobacco Dependence Treatment

Health insurance coverage is associated with increased access to medical care, including preventive services such as smoking cessation treatment. Disparities in health care access and quality might contribute to higher smoking initiation and SHS exposure rates, higher current smoking prevalence, and lower quitting success among members of racial/ethnic minorities and people with lower incomes. The percentage of the overall U.S. population who are uninsured declined from 22.3% in 2010 to 12.8% in 2015.[66] However, as shown in Figure 2.20, there are substantial differences in uninsurance rates, with Hispanic/Latino adults the least likely to have health insurance of any racial/ethnic group. Among adults younger than 65, higher rates of uninsurance are also found among younger age groups and among those who are poor or near poor.

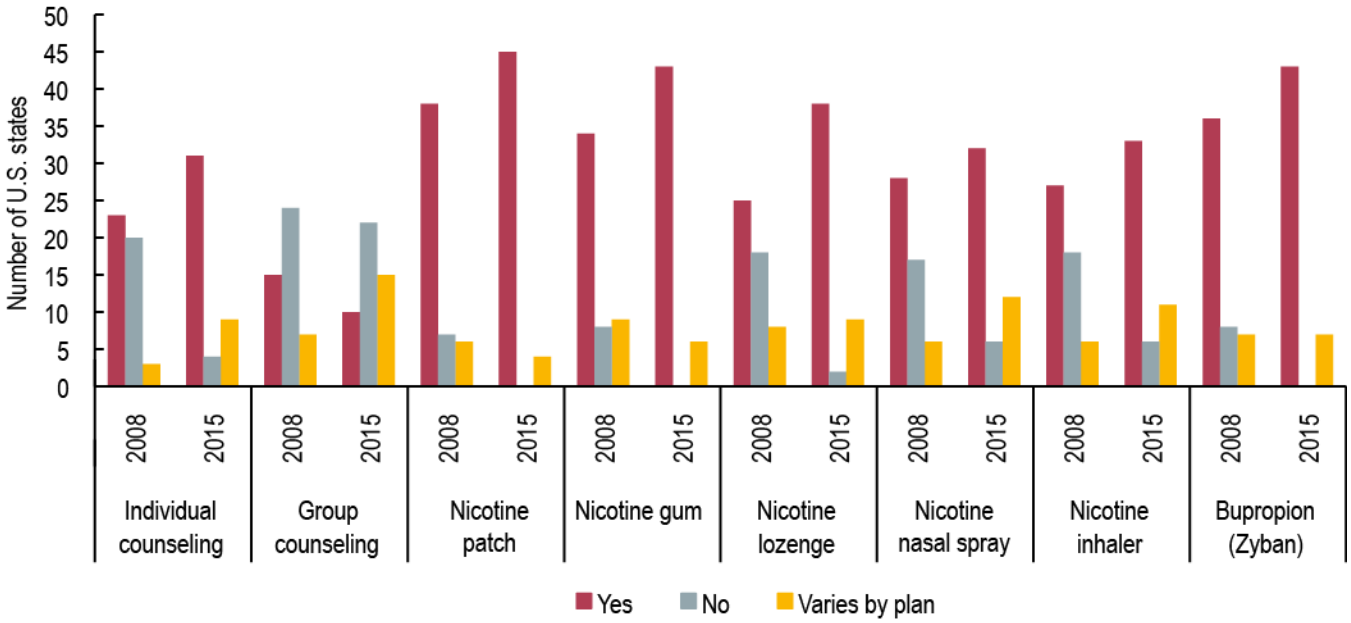
Figure 2.20 NHIS Participants Under Age 65 Who Lacked Health Insurance Coverage at Time of Interview, by Race/Ethnicity, 2009–2015



Sources: Martinez and Cohen 2014[146]; Cohen et al. 2016.[66]

Various steps have been taken to provide tobacco dependence treatments for Americans, including low-income Americans, through insurance coverage. The 2008 Public Health Service Clinical Practice Guidelines recommended that all state Medicaid insurance programs provide coverage for tobacco dependence treatment medications (i.e., gum, patch, lozenge, nasal spray, inhaler, varenicline, bupropion hydrochloride) and behavioral counseling (i.e., individual, group, telephone).[67] The Affordable Care Act (ACA) mandated that all United States Preventive Services Task Force A and B level recommendations must be covered by private health plans without cost-sharing, which includes tobacco cessation interventions.[68] Additionally, in October 2010, the ACA mandated Medicaid coverage of tobacco dependence treatments for pregnant women. As of January 2014, state Medicaid programs were required by the ACA to cover the costs of FDA-approved tobacco dependence medications for all Medicaid recipients. The 2014 report on state Medicaid coverage for tobacco dependence treatments finds that while all states cover tobacco dependence treatments for some enrollees, only nine states cover all nine evidence-based cessation methods (excluding telephone counseling) (Figure 2.21).[69] While this is a marked improvement from previous years, barriers to access still exist, including duration limits (applicable in 40 states), annual limits on quit attempts, pre-authorization requirements, and co-pays. In addition, studies indicate that many smokers with Medicaid insurance are unaware of programs that provide coverage for smoking cessation pharmacotherapies,[70,71] and that Medicaid programs that offer treatment lack the necessary outreach efforts to inform clients of those benefits.[72] Increasing coverage of tobacco dependence treatment and awareness of this coverage by both smokers and health care providers can increase quit attempts, use of effective treatment, and quit rates, and contribute to reducing TRHD.[67,68]

Figure 2.21 State Medicaid Coverage of Tobacco Dependence Treatments, 2008 and 2015



Notes: Yes = state Medicaid coverage for treatment; No = no state Medicaid coverage for treatment; Varies by plan = varies by state Medicaid insurance plan.
Source: Singleterry et al. 2015.[69]

Secondhand Smoke and Prenatal Tobacco Exposure

The 2006 Surgeon General's report, *The Health Consequences of Involuntary Exposure to Secondhand Smoke*, concluded that "secondhand smoke exposure causes premature death and disease in children and in adults who do not smoke"[73,p.11] and that, among nonsmoking adults, SHS exposure is causally related to heart disease and lung cancer. The 2014 Surgeon General's report confirmed a causal relationship between secondhand smoke (SHS) exposure and stroke.[1] Children exposed to SHS are at a higher risk of sudden infant death syndrome (SIDS), acute respiratory infections, ear problems, and poor lung function.[73] Prenatal smoke exposure is causally linked to reduced fertility, pregnancy complications, and poor birth outcomes, including impaired lung development, low birth weight, and preterm delivery.[73,74]

The burden of SHS exposure is experienced disproportionately among nonsmoking racial/ethnic minority individuals and people from low-SES backgrounds, including nonsmoking pregnant women, as detected by biomarkers of exposure (e.g., cotinine). From 1999 to 2012, the percentage of the nonsmoking population age 3 and older with detectable serum cotinine levels ≥0.05 ng/mL declined across all racial/ethnic groups.[75] However, a significantly higher proportion of non-Hispanic black nonsmokers continued to have serum cotinine levels of ≥0.05 ng/mL, compared to Mexican American and non-Hispanic white nonsmokers. For example, in 2011-2012, nearly 50% of non-Hispanic black nonsmokers had serum cotinine levels of ≥0.05 ng/mL, compared with 22% of non-Hispanic white and 24% of Mexican American nonsmokers.[75] Also between 1999 and 2012, serum cotinine levels of ≥0.05 ng/mL declined significantly among nonsmokers age 3 years and older regardless of poverty status. However, in 2011-2012, a significantly greater percentage of nonsmokers living in poverty had serum cotinine levels of ≥0.05 ng/mL compared with their higher income counterparts (43.2% vs. 31.7%).[75]

Data from the Pregnancy Risk Assessment Monitoring System (PRAMS) show that the prevalence of maternal smoking during pregnancy declined significantly between 2000 and 2010.[76] However, PRAMS data also show differences in the prevalence of smoking during pregnancy by race/ethnicity. In 2010, smoking during pregnancy was highest among American Indians/Alaska Natives (26.0%), followed by non-Hispanic whites (14.3%), non-Hispanic blacks (8.9%), Hispanics (3.4%), and Asians/Pacific Islanders (2.1%).[76] Birth certificate data from 2014 show a similar trend: American Indians/Alaska Natives had the highest prevalence of smoking during pregnancy (18.0%) followed by non-Hispanic whites (12.2%); lower prevalence rates were found for non-Hispanic blacks (6.8%), Hispanics (2.0%), and Asians (0.7%).[77]

Birth certificate data for 2014 show that, overall, about 8.4% of women smoked at any time during their pregnancy, and differences between groups in the prevalence of maternal smoking during pregnancy closely followed differences between groups in the prevalence of smoking before pregnancy. Higher rates of smoking during pregnancy were seen in women with fewer than 12 years of education (14.1%), women with Medicaid coverage (14.0%), women ages 20–24 (13.0%), unmarried women (14.7%), and non-Hispanic American Indian/Alaska Native women (18.0%).[77] National data also show that a mother's educational level and smoking during pregnancy independently increase the risk of smoking among her offspring.[78] Additionally, being black non-Hispanic (adjusted prevalence ratio [aPR] = 1.25; 95% CI 1.14–1.38) compared with being white non-Hispanic and having 12 years of education (aPR = 1.09; 95% CI 1.01–1.17) compared with having more than a high school education were found to be associated with postpartum relapse to smoking.[79] Data from the 2010 PRAMS also indicate disparities in the prevalence of smoke-free home rules postpartum.[76] Overall, 93.6% of women with a recent live birth reported having a complete smoke-free home rule; women who smoked during pregnancy and postpartum had the lowest percentage of smoke-free home rules (77.6%). Lower percentages of smoke-free home rules were also found among non-Hispanic black women (86.8%), American Indian/Alaska Native women, women with an annual income below $15,000 (87.6%), women with fewer than 12 years of education (88.6%), women with Medicaid coverage during pregnancy or delivery (89.7%), and women enrolled in the Special Supplemental Nutrition Program for Women, Infants and Children (WIC) (90.6%).[76]

Disparities also exist regarding SHS exposure among children and adolescents. While overall SHS exposure, measured by serum cotinine, declined from 52.5% in 1999-2000 to 25.3% in 2011-2012, declines have been slower and rates of exposure have remained higher among children ages 3 to 11 (40.6%) and adolescents ages 12 to 19 (33.8%) compared with adults (21.3%).[75] NHANES data from 2011-2012 show that 67.9% of non-Hispanic black children (3–11 years old) were exposed to SHS compared with 37.2% of non-Hispanic white and 29.9% of Mexican American children.[75] Using NHANES data from 2003 to 2006, Marano and colleagues[80] found that 24.1% of non-Hispanic black youth (3–19 years old) were exposed to SHS in the home compared with 19.4% of non-Hispanic white and 6.6% of Mexican American youth. Even among children and youths who were not exposed to SHS in the home, non-Hispanic blacks had significantly higher serum cotinine levels compared with non-Hispanic whites.[80]

NHANES data from 2003 to 2006 also show that SHS exposure in the home was significantly higher among children and adolescents from families with annual family incomes of less than $20,000 compared with those from families with annual family incomes of $20,000 or more (26.4% vs. 15.5%, respectively).[80]

Prevalence of SHS exposure in the home among children and adolescents also varied by the educational attainment level of the household reference person, defined as an adult resident 18 years old or older owning or renting the residence sampled. When the household referent had less than a high school education, prevalence of exposure was 24.9%; with a high school education or equivalent, 19.7%; and with more than a high school education, 11.8%. These data also show significantly higher serum cotinine levels among children and youths from families with lower annual family incomes and lower householder educational levels, regardless of SHS exposure in the home.[80]

Tobacco-Related Cancer Incidence and Mortality

Approximately half of all people who continue to smoke will die from tobacco-related diseases,[81] and smoking contributes to at least 30% of all cancer deaths in the United States.[82] Cigarette smoking and exposure to SHS are estimated to result in more than 480,000 premature deaths in the United States each year.[1] Annual smoking-attributable costs for the years 2009–2012 are estimated at $289–$332.5 billion, which includes $132.5–$175.9 billion for adult direct medical care, $151 billion for lost productivity due to premature deaths, and $5.6 billion for lost productivity due to exposure to SHS.[1]

There are at least 7,000 chemicals in tobacco smoke, and at least 69 are known to cause cancer.[83] Tobacco smoking, SHS, and smokeless tobacco were listed as human carcinogens in the U.S. Department of Health and Human Services *Report on Carcinogens, 9th edition* (2000).[84] The International Agency for Research on Cancer (IARC) has designated tobacco smoking, SHS exposure, and smokeless tobacco as carcinogenic to humans.[85,86] As of 2014, the Surgeon General has causally linked cigarette smoking to 12 different cancers: acute myeloid leukemia, and cancers of the lung, trachea, and bronchus; oropharynx; esophagus; larynx; stomach; bladder; kidney and ureter; pancreas; uterine cervix; colon and rectum; and liver. The evidence for a causal relationship between active smoking, SHS exposure, and breast cancer was found to be suggestive but not sufficient.[1] The 2014 Surgeon General's report, as well as many previous reports, confirms a causal link between smoking and many serious chronic diseases, including chronic obstructive pulmonary disease (COPD), coronary heart disease, stroke and atherosclerotic peripheral vascular disease, diabetes, and overall diminished health status. The 2014 Surgeon General's report estimated that the leading causes of annual average smoking-attributable mortality (SAM) among adults age 35 and older between 2005 and 2009 were lung and other cancers (163,700 deaths), followed by cardiovascular diseases (160,600 deaths) and respiratory diseases (113,100 deaths). Lung cancer alone contributed to 158,530 deaths annually (88,730 deaths among men, 69,800 deaths among women).[1]

The 2010 Surgeon General's report, *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease*,[83] summarizes the science on how tobacco smoking harms health. The conclusions of the 2010 Surgeon General's report include:

> Inhaling the complex chemical mixture of combustion compounds in tobacco smoke causes adverse health outcomes, particularly cancer and cardiovascular and pulmonary diseases, through mechanisms that include DNA damage, inflammation, and oxidative stress.
>
> Through multiple defined mechanisms, the risk and severity of many adverse health outcomes caused by smoking are directly related to the duration and level of exposure to tobacco smoke.[83,p.9]

Cigar smokers have an increased risk of oral, esophageal, laryngeal, and lung cancer[85,87] and "regular cigar smokers who inhale, particularly those who smoke several cigars per day, have an increased risk of coronary heart disease and chronic obstructive pulmonary disease."[87,p.i] As with cigarettes, cigar smoking involves the burning of tobacco; cigars have the potential to deliver as much nicotine and may contain the same or higher levels of carcinogens and toxicants as cigarettes.[27] A systematic review of the literature on the health risks of cigar smoking concluded that mortality from cigar smoking varies by level of smoke exposure (measured by cigars per day, inhalation level) and can equal or exceed the mortality risk of cigarette smoking; even among cigar smokers who do not inhale, mortality risk from oral, esophageal, and laryngeal cancers was elevated.[88] In another study examining the SAM of regular cigar smoking, cancers of the trachea, lung, and bronchus were the leading causes of premature death, followed by cancers of the larynx and lip, oral cavity, and pharynx.[89] This study estimated that in 2010, cigar smoking caused more than 9,000 premature deaths among adults age 35 years and older, with lung cancer as the leading cause of premature death. SAM estimates for men (>8,000) were higher than for women (>1,000), reflecting men's higher cigar smoking rates.

The estimated number of new cancer cases and deaths (in 2017) for selected tobacco-related cancers, based on incidence data from the North American Association of Central Cancer Registries (1999–2013) and mortality date from the National Center for Health Statistics, Centers for Disease Control and Prevention (2000–2014), is shown in Table 2.5.[90] Among men, the highest number of new cases were lung/bronchial, bladder, and kidney/renal cancers, and the highest number of deaths were lung/bronchial, pancreatic, and esophageal cancers. Among women, both the highest number of new cases and the highest number of deaths were lung/bronchial, pancreatic, and kidney/renal cancers.[90] The proportion of cancer deaths attributed to cigarette smoking varies by cancer site, from an estimated 80% of lung, bronchus, and trachea cancer deaths to 10% of deaths from colorectal cancer.[91]

Table 2.5 Tobacco-Related Cancers: Estimated New Cases and Deaths in 2017

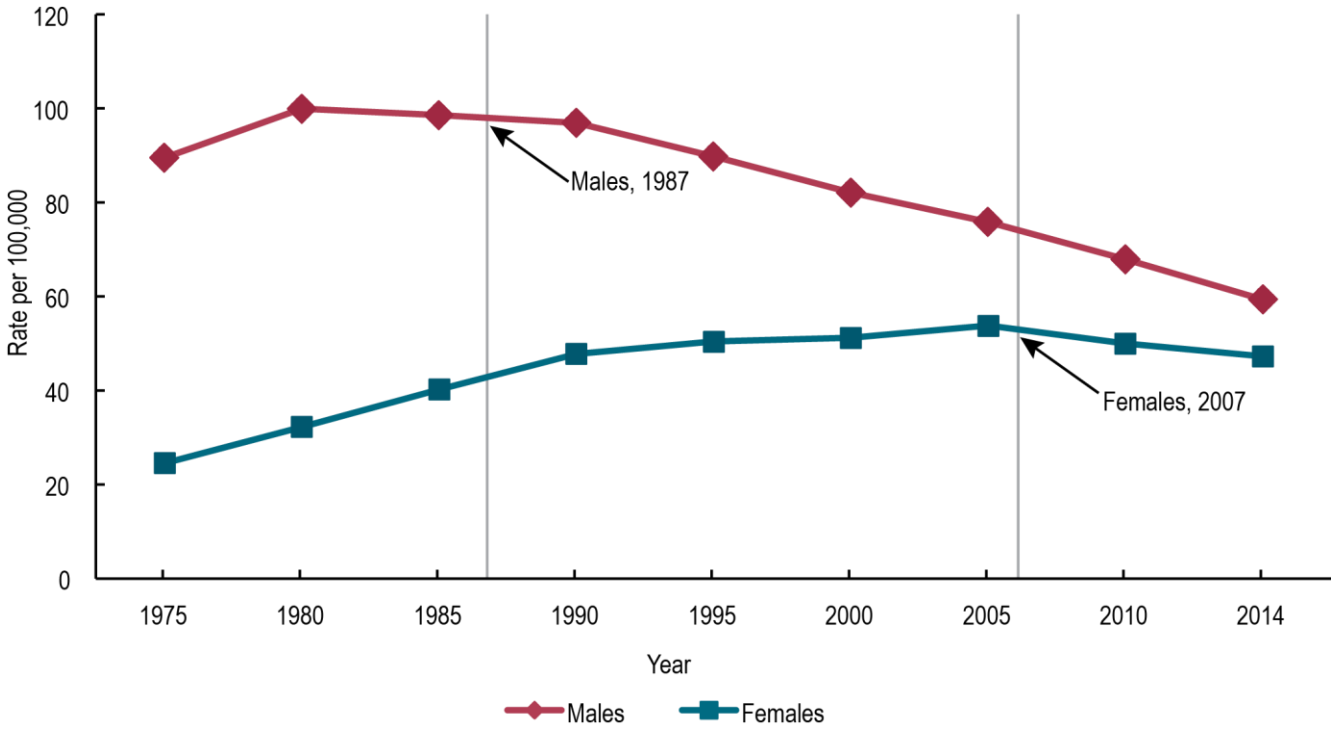
	Expected New Cases in 2017			Estimated Deaths in 2017		
Tobacco-Related Cancer	Total	Men	Women	Total	Men	Women
Lung and bronchus	222,500	116,990	105,510	155,870	84,590	71,280
Bladder	79,030	60,490	18,540	16,870	12,240	4,630
Kidney and renal pelvis	63,990	40,610	23,380	14,400	9,470	4,930
Pancreas	53,670	27,970	25,700	43,090	22,300	20,790
Cervix/uterus	12,820	N/A	12,820	4,210	N/A	4,210
Oral cavity and pharynx	49,670	35,720	13,950	9,700	7,000	2,700
Stomach	28,000	17,750	10,250	10,960	6,720	4,240
Esophagus	16,940	13,360	3,580	15,690	12,720	2,970
Acute myeloid leukemia	21,380	11,960	9,420	10,590	6,110	4,480
Larynx	13,360	10,570	2,790	3,660	2,940	720

Note: N/A = not applicable.
Source: American Cancer Society 2017.[90]

Tobacco-Related Cancer Incidence and Mortality, by Sex

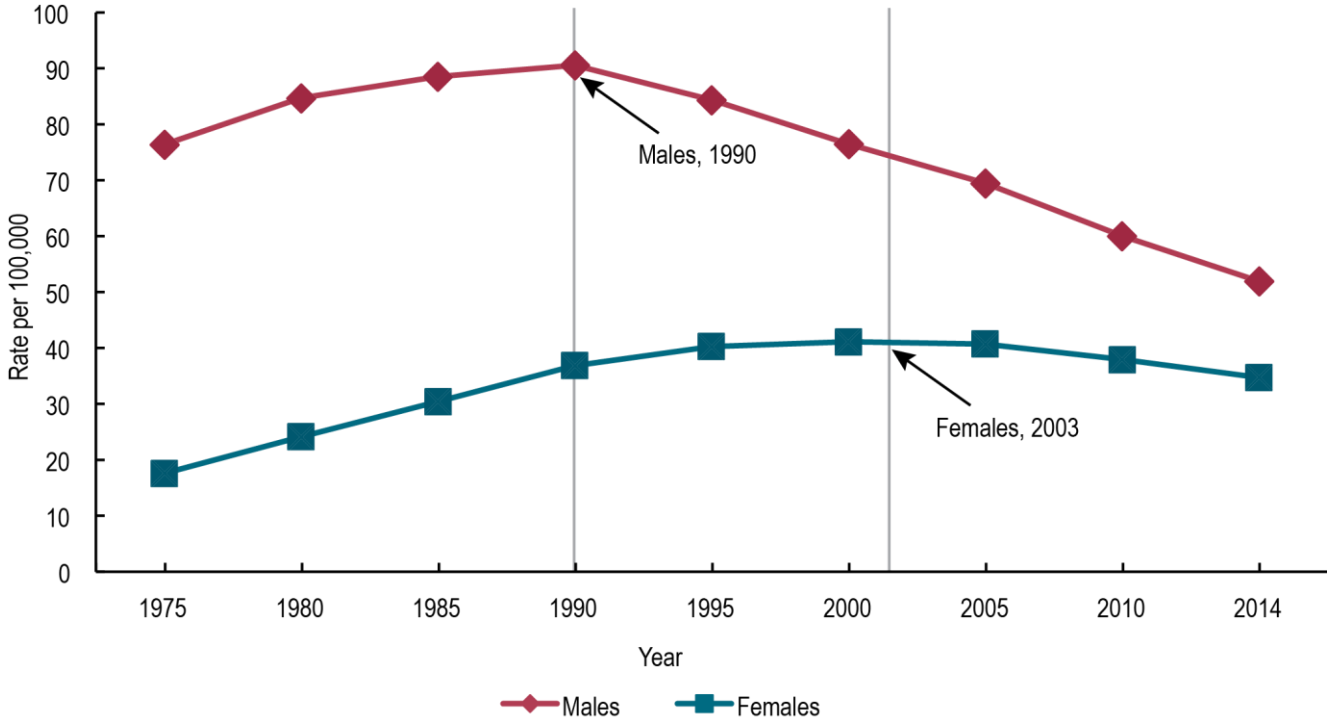
Lung cancer is the leading cause of cancer-related deaths among both men and women in the United States.[92] Lung cancer deaths also account for the largest fraction of smoking-attributable cancer deaths.[1] In 2014, lung/bronchial cancer incidence and mortality per 100,000 people were higher among men (59.3 and 51.9, respectively) than women (47.2 and 34.7, respectively).[93] (See Figure 2.22 for incidence data, and Figure 2.23 for mortality data.) However, lung cancer incidence and mortality among men have been steadily declining since the 1980s and 1990s, respectively. Lung cancer incidence and mortality among women have now begun to decrease as well. Differences in lung cancer incidence and mortality trends for males and females largely reflect historical patterns in smoking prevalence, which began falling more quickly among men than women beginning in the 1950s.

Figure 2.22 Age-Adjusted U.S. Incidence of Lung and Bronchus Cancers, by Sex, 1975–2014



Note: Vertical lines denote the year in which incidence peaked, by sex.
Source: Based on data from the National Cancer Institute, Surveillance, Epidemiology, and End Results Program 1975–2014.[93]

Figure 2.23 Age-Adjusted U.S. Mortality from Lung and Bronchus Cancers, by Sex, 1975–2014



Note: Vertical lines denote the year in which mortality peaked, by sex.
Source: Based on data from the National Cancer Institute, Surveillance, Epidemiology, and End Results Program 1975–2014.[93]

Tobacco-Related Cancer Incidence and Mortality, by Race/Ethnicity

The 1998 Surgeon General's report concluded that African Americans currently bear the greatest health burden of disease and death from cigarette smoking.[59] In 2014, African American men had the highest incidence of and mortality from several tobacco-related cancers, including cancers of the lung and bronchus, kidney and renal pelvis, pancreas, and larynx, compared with men from other racial/ethnic backgrounds (Tables 2.6 and 2.7).[93] As noted in the 1998 Surgeon General's report, "the higher lung cancer incidence and death rates among African American men have not been fully explained,"[59,p.140] and this remains true today. Factors that have been proposed to explain the higher rate of tobacco-related cancer mortality in African American men, given their lower level of cigarette smoking than men of other races/ethnicities, include: historical patterns of cigarette smoking[59,94]; genetic factors (discussed in chapter 3); smoking topography[59]; the disproportionate use of mentholated cigarettes by African Americans[95,96]; barriers to receiving timely, appropriate, and high-quality medical care[97]; as well as the many other social and environmental factors discussed in this monograph. However, DeSantis and colleagues[97] note that disparities in lung cancer death rates between African American men and white men have decreased substantially over time (from >40% in the early 1990s to 20% in 2012) and have been eliminated in adults younger than 40. In 2014, white women had a higher lung cancer incidence and death rate than African American women, and both had higher rates than women of other races/ethnicities (Table 2.7).[93]

Table 2.6 Tobacco-Related Cancer Incidence per 100,000 People in the United States, by Race/Ethnicity and Sex, 2014

	White (Non-Hispanic)		African American		Hispanic/Latino		Asian/Pacific Islander		American Indian/ Alaska Native*	
Cancer Type	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Lung and bronchus	66.4	53.8	79.0	46.9	32.5	22.4	41.6	26.9	39.5	22.8
Bladder	39.1	9.5	19.8	6.5	18.8	4.7	14.5	3.9	16.1	~
Kidney and renal pelvis	22.2	10.8	25.2	12.1	20.7	11.2	10.9	6.0	17.3	10.7
Pancreas	14.7	11.1	17.4	14.4	12.1	10.1	10.3	8.8	11.4	7.7
Cervix/uterus	N/A	7.1	N/A	8.2	N/A	8.8	N/A	6.0	N/A	7.4
Oral cavity and pharynx	19.7	6.9	14.0	5.1	9.5	4.1	10.7	4.9	9.6	4.4
Stomach	7.9	3.5	13.5	7.1	12.6	8.2	13.7	7.3	13.0	7.8
Esophagus	8.0	1.9	5.8	2.0	4.6	1.1	3.5	0.8	5.2	~
Acute myeloid leukemia	5.5	3.9	4.6	3.1	3.9	2.9	3.7	3.1	~	~
Larynx	5.2	1.3	8.5	1.5	3.1	0.4	1.5	~	~	~

Notes: Rates are per 100,000 population and are age-adjusted to the 2000 U.S. Standard Population (19 age groups – Census P25-1130). N/A = not applicable. ~Indicates less than 16 cases; statistic not displayed.
*Rates are higher for American Indians/Alaska Natives when analyses are restricted to Contract Health Service Delivery Areas (CHSDA).[147]
Source: Based on data from the National Cancer Institute Surveillance, Epidemiology, and End Results Program 2014.[93]

Table 2.7 Tobacco-Related Cancer Mortality per 100,000 People in the United States, by Race/Ethnicity and Sex, 2014

	White (Non-Hispanic)		African American		Hispanic/Latino		Asian/Pacific Islander		American Indian/ Alaska Native*	
Cancer Type	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Lung and bronchus	54.6	38.4	62.5	32.8	25.4	13.4	29.7	17.6	44.3	29.5
Bladder	8.3	2.2	5.6	2.5	3.7	1.2	2.8	1.0	2.8	2.3
Kidney and renal pelvis	5.7	2.3	5.9	2.2	5.0	2.2	2.8	1.2	8.0	3.1
Pancreas	12.9	9.6	15.0	11.8	9.3	7.5	7.8	7.2	9.9	7.5
Cervix/uterus	N/A	2.1	N/A	3.6	N/A	2.6	N/A	1.5	N/A	2.3
Oral cavity and pharynx	4.1	1.4	4.9	1.3	2.4	0.8	3.2	0.9	4.2	~
Stomach	3.3	1.7	8.2	3.7	6.7	4.0	6.5	4.1	8.7	3.9
Esophagus	7.9	1.5	5.5	1.8	3.9	0.8	2.9	0.7	7.3	~
Acute myeloid leukemia	3.8	2.4	2.8	1.8	2.5	1.7	2.4	1.6	~	~
Larynx	1.7	0.4	3.3	0.5	1.4	0.1	0.6	~	2.5	~

Notes: Rates are age-adjusted to the 2000 U.S. Standard Population (19 age groups - Census P25-1130). N/A = not applicable. ~Indicates less than 16 cases; statistic not displayed.
*Rates are higher for American Indians/Alaska Natives when analyses are restricted to Contract Health Service Delivery Areas (CHSDA).[147]
Source: Based on data from the National Cancer Institute Surveillance, Epidemiology, and End Results Program 2014.[93]

Lung cancer incidence and mortality were lowest among Hispanic/Latino men and women in 2014 (Tables 2.6 and 2.7). However, lung cancer was the leading cause of cancer death for Hispanic men and the second leading cause of cancer death for Hispanic women.[98] In addition, a larger fraction of lung cancers are diagnosed at distant stage among Hispanics (59%) than among non-Hispanic whites (52%), and fewer cases are diagnosed at localized stage among Hispanics (13%) than among non-Hispanic whites (17%), contributing to a lower survival rate for Hispanics than for non-Hispanic whites.[98]

Lung cancer incidence rates among American Indian/Alaska Native and Asian/Pacific Islander men were 39.5 and 41.6, respectively, in 2014 (Table 2.6), but mortality was higher among American Indian/Alaska Native males (43.9) than Asian/Pacific Islander males (29.7) (Table 2.7). After Hispanic women, lung cancer incidence and mortality were lowest among Asian/Pacific Islander women, and Indian/Alaska Native women.[93] Despite lower lung cancer incidence, the 5-year survival rate was lower among American Indian/Alaska Natives than non-Hispanic whites, and American Indian/Alaska Native populations were more likely to be diagnosed with advanced stage cancers and less likely to undergo resection compared with whites.[99]

As discussed earlier in the chapter, smoking prevalence may vary significantly among populations within the broad categories of Hispanics/Latinos, Asian Americans, and American Indian/Alaska Native populations, a fact that has important implications for the burden of tobacco-related cancer.

Tobacco-Related Cancer Incidence and Mortality, by SES

Significant disparities in lung cancer incidence/mortality also exist by SES. Analysis of data from the SEER–National Longitudinal Mortality Study (NLMS) project show that between 1979 and 1998, men with a high school education or less had significantly higher lung cancer incidence rate ratios (high school, 2.32; less than high school, 3.01) than men with a college education.[100] Women who had a high school education or less had significantly higher lung cancer incidence rate ratios (high school, 1.74; less than high school, 2.02) compared with women with at least a college degree.[100] According to national data for 2003–2007 for all major cancers combined, the largest SES disparity was seen for lung cancer.[101] Among all races, people who completed 12 years or less of high school were much more likely to develop lung cancer (five times more likely for men, and four times more likely for women) than those who completed a college degree or more.[101] Other research indicates that lung cancer incidence increases with decreasing SES, except among Hispanic men and women, where there is an inverse effect of SES.[102]

NLMS data also show that people with lower incomes are at higher risk of lung cancer. Incidence of lung cancer among men and women with annual family incomes of less than $12,500 was more than 1.7 times higher than lung cancer incidence among those with incomes of $50,000 or higher.[100] Unemployed men and women also had a higher lung cancer incidence than employed people (rate ratios = 1.83 and 2.09, respectively).[100] Research also shows that low SES is associated with lower survival rates among lung cancer patients.[103,104] The disparities in smoking prevalence between low SES and high SES undoubtedly contribute to disparities in rates of lung and other tobacco-related cancers.

Tobacco-Related Cancer Incidence and Mortality and HIV

Infections such as HIV are associated with certain cancers, which may also contribute to TRHD. Data from the United States suggest that tobacco use is higher among persons living with HIV/AIDS (PLWHA) compared with their uninfected counterparts.[105,106] Smoking is also more prevalent among

population subgroups that are highly affected by the HIV epidemic. For example, lower SES, including lower income and education, is associated with both HIV morbidity and mortality[107–111] and with tobacco use. Additionally, men who have sex with men are the population most affected by HIV in the United States[112,113]; they also have high smoking rates.[114,115] This convergence of smoking and HIV among vulnerable populations could further contribute to cancer-related health disparities. As the use of highly active antiretroviral therapy has significantly prolonged the lives of PLWHA, more PLWHA are reaching ages where chronic diseases such as cancer are more common. Moreover, lung cancer is the leading cause of cancer death in HIV-infected individuals.[113] This is largely due to higher smoking rates in PLWHA, but even after controlling for smoking status, HIV-infected individuals still have a 2 to 3 times higher risk of developing lung cancer than the overall population.[116–119] Evidence also suggests that HIV-related immunosuppression and inflammatory processes can further increase cancer risk in PLWHA.[118–124]

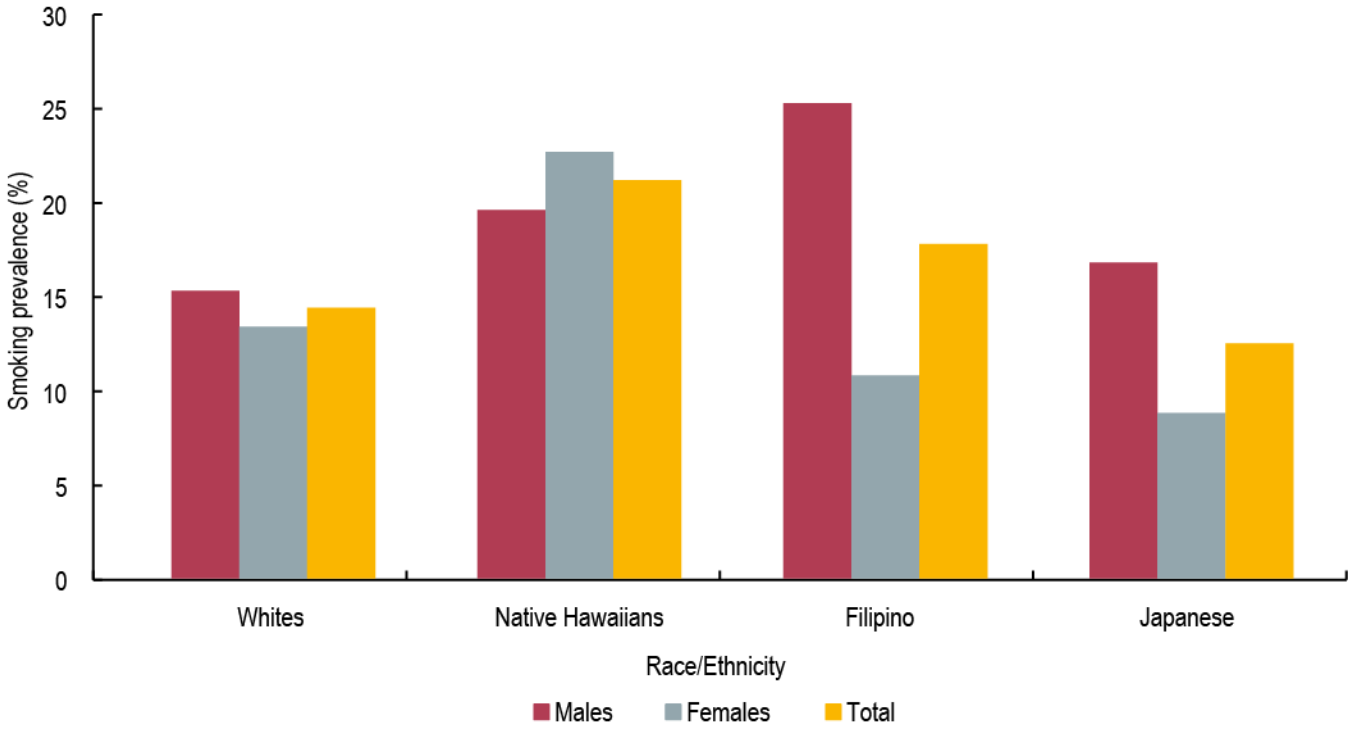
Methodological Limitations and Challenges in the TRHD Literature

The 1998 Surgeon General's report delineated four main categories of methodological limitations in the TRHD literature: (1) nongeneralizability, (2) noncomparability, (3) sample size and aggregation problems, and (4) nonreporting.[63] These and other methodological limitations and challenges remain relevant today, as discussed below.

Aggregate data can mask significant disparities in smoking prevalence and cancer outcomes both within and across racial/ethnic and other population groups. National data are not available in disaggregate form for some races/ethnicities, and for many populations trend data cannot be reported. A lack of disaggregated data often makes it difficult to report TRHD by sex, race/ethnicity, and sexual orientation, or stratified by SES indicators such as poverty status, education, and occupation. In some cases, surveillance data for groups known to be at higher risk of tobacco use, such as LGBT groups, are limited.

Examples of disaggregated data show the type and value of the information that can be gained. For example, Hawaii's Behavioral Risk Factors Surveillance System (BRFSS) surveys are unique because they disaggregate race/ethnicity and report smoking by Native Hawaiians, Filipinos, and Japanese as separate groups. The data show marked differences in smoking prevalence between males and females in non-white racial/ethnic groups, with Filipino males and Native Hawaiian females reporting the highest smoking prevalence, and Japanese women and Filipino women reporting the lowest smoking prevalence (Figure 2.24). Disaggregated data show that lung cancer incidence and mortality rates in Hawaii are higher among Native Hawaiians and Filipinos than among whites.[125] In addition, data from the Multiethnic Cohort Study of Diet and Cancer show that Native Hawaiians and African Americans have an elevated risk of lung cancer compared with other racial/ethnic groups when light smoking (fewer than 10 cigarettes a day) is considered.[126]

Figure 2.24 Smoking Prevalence in Hawaii, by Ethnicity and Sex, 2008



Source: Adapted from Pobutsky and Lowery St. John 2010.[125]

Confidence intervals may be wide for some populations when group-specific data are reported. Wide confidence intervals reflect a lack of precision of the population parameter estimate, such that if the survey were conducted again in a different sample of the population, a different estimate might be observed, resulting in diminished reliability of the findings. In addition, the data are subject to misinterpretation if a finding is not statistically significant when it is (or vice versa). Regional survey data may result in better estimates for some aggregate and disaggregated groups, and may reflect more stable estimates of tobacco use and a more accurate picture of the presence or absence of disparities, but these advantages come at the cost of being representative of only that region. One strategy to report data on small populations and increase statistical stability is to collect and combine survey data across years, but this strategy has limitations as well.

Studies may not adequately examine how contextual factors contribute to disparities in tobacco use and related disease outcomes. For example, American Indians/Alaska Natives have had consistently higher smoking prevalence and longer durations of smoking compared with blacks/African Americans.[41] Other intrapersonal, interpersonal, community/neighborhood, or societal/policy-level factors may help explain the disparities that exist between racial/ethnic groups.[127,128] The constructs of race/ethnicity and culture may differentially influence psychosocial processes that lead to harmful health behaviors or outcomes.[129,130]

Studies may not collect adequate data on aspects of tobacco use that are important or unique to specific groups, or collect adequate data among specific populations, such as LGBT groups. Researchers have recently (2015) suggested expanding data collection on cigarette type (menthol vs. non-menthol) to improve our understanding of how menthol tobacco products may contribute to disparities among

youths and adults.[33,131] National surveys have recently begun collecting data on emerging tobacco products, including e-cigarettes. In addition, national data are increasingly being collected on flavors in tobacco products, especially related to premium cigars, LFCs, cigarillos, and hookah. However, not all national surveys collect data on these products, distinguish by type of product, or monitor the type of flavors used by different racial/ethnic or socioeconomic groups.

Chapter Summary

This chapter presents information on tobacco use behaviors among youths and adults, including young adults and pregnant women, using data from a number of state and national surveys. Cigarette smoking has declined substantially over time among adults of both sexes, among all racial/ethnic groups, and among adults at all poverty and educational levels. However, there are approximately 40 million current smokers in the United States, and significant disparities in prevalence persist by race/ethnicity, level of educational attainment, income, sexual orientation, and other factors.

Among youth, cigarette smoking prevalence has steadily declined since the mid-1990s, but the research still finds evidence of disparities by race/ethnicity and SES. For example, pooled NSDUH data from 2013 to 2015 show that the prevalence of current cigarette smoking among youth ages 12–17 was highest among American Indians/Alaska Natives (7.0%) followed by non-Hispanic whites (6.3%), Native Hawaiians or Other Pacific Islanders (3.4%), Hispanics (3.4%), non-Hispanic blacks/African Americans (2.7%), and Asians (1.6%). Cigarette smoking prevalence is also far higher among youth who do not plan to complete 4 years of college compared with those who do (19.2% vs. 8.4% in 2016). Additionally, nationally representative data from the PATH study show that gay/lesbian and bisexual youth ages 14–17 have a significantly higher prevalence of cigarette smoking and of any tobacco use, compared with heterosexual youth.[25] Patterns of cigarette smoking among young adults (ages 18–25) are generally similar to patterns found among youth. In addition to differences by race/ethnicity, substantial differences by poverty level are found among young adults.

Use of other tobacco products, including e-cigarettes, cigars, hookah, and smokeless tobacco, is also found among youth. Research suggests that flavored tobacco products (including menthol), are especially attractive to youth and young adults; indeed, a majority of youth who have ever used tobacco report that their first tobacco product was flavored.[32] Based on NSDUH data from 2004 to 2010, menthol cigarette use is especially common among youths and young adult cigarette smokers (56.7% and 45.0%, respectively), compared with adult cigarette smokers over the age of 25 (range 30.5% to 34.7%).[35] (Menthol as a tobacco flavorant is discussed in chapter 4.)

In 2015, 21.9% of American Indian/Alaska Native adults reported current smoking compared with 16.7% of non-Hispanic blacks, 16.6% of non-Hispanic whites, 10.1% of Hispanics, and 7.0% of Asian adults.[41] Significant disparities in cigarette smoking also persist among adults with lower educational attainment compared to those with higher educational attainment. Smoking prevalence is also higher and is declining at a slower pace among adults living below the poverty level, compared with adults living at or above poverty. In 2015, 26.1% of adults living below the poverty level smoked cigarettes compared to 13.9% of adults living at or above poverty.[41]

Among adults, light and intermittent (non-daily) smoking is increasingly common in the United States. A trend towards light smoking ($\leq$ 9 or 10 cigarettes per day) is seen among all racial/ethnic groups, with levels of light smoking highest among racial/ethnic minorities. Additionally, it is estimated that

approximately one-third (32.5%) of all adult smokers use menthol-flavored cigarettes, and African American cigarette smokers report the highest prevalence of menthol cigarette smoking of any racial/ethnic group, with levels of menthol smoking consistently exceeding 70%.[13] About two-thirds of all adult smokers are interested in quitting, but rates of recent smoking cessation (≥6 months during the past year) were lower among non-Hispanic blacks (4.9%; 95% CI 3.2–6.6) compared with non-Hispanic Asians (17.3%; 95% CI 10.1–24.5), Hispanics (8.2%; 95% CI 5.5–10.9), and non-Hispanic whites (7.1%; 95% CI 6.0–8.2) in 2015.[62] Quit rates were also lower among low-income smokers and those with lower levels of educational attainment, compared with their more advantaged counterparts.

Secondhand smoke exposure is causally linked to premature death and disease in nonsmoking youths and adults.[73] Although SHS exposure has been decreasing overall, a disproportionate burden of SHS exposure remains among nonsmoking racial/ethnic minority groups and people from low-SES backgrounds, including nonsmoking pregnant women, as detected by biomarkers of exposure (e.g., cotinine). These disparities are particularly evident among children and adolescents compared with adult nonsmokers. In 2014, an estimated 8.4% of mothers smoked at some time during their pregnancy, but higher rates of maternal smoking were seen among less-educated and low-income women, young women, American Indian/Alaska Native women, and white women, compared with women overall.[77]

As summarized in the 2010 Surgeon General's report, "inhaling the complex chemical mixture of combustion compounds in tobacco smoke causes adverse health outcomes—particularly cancer, and cardiovascular and pulmonary disease—through mechanisms that include DNA damage, inflammation, and oxidative stress"[83,p.9] and "through multiple defined mechanisms, the risk and severity of many adverse health outcomes caused by smoking are directly related to the duration and level of exposure to tobacco smoke."[83,p.9]

Lung cancer deaths comprise the largest fraction of smoking-attributable cancer deaths. Largely because of declines in smoking prevalence, lung cancer incidence and mortality among men have been steadily declining since the 1980s and 1990s, respectively, and have recently begun to decline among women as well. However, disparities persist in tobacco-related cancer incidence and mortality by race/ethnicity, SES, and other factors. As noted in the 1998 Surgeon General's report, "the higher lung cancer incidence and death rates among African American men have not been fully explained,"[59,p.140] and this remains true today. In 2014, African American men had the highest incidence of and mortality from several tobacco-related cancers including cancers of the lung and bronchus, kidney and renal pelvis, pancreas, and larynx. However, disparities in lung cancer death rates between African American men and white men have decreased substantially over time (from >40% in the early 1990s to 20% in 2012) and have been eliminated in adults younger than 40 years.[97]

Finally, this chapter points to a number of methodological limitations and challenges in the TRHD literature: aggregate data can mask significant disparities in prevalence and cancer outcomes both within and across racial/ethnic and other population groups; confidence intervals may be wide for some populations when group-specific data are reported; studies have not adequately examined how contextual factors (e.g., community/neighborhood factors) contribute to disparities in tobacco use and related disease outcomes; and studies may not collect adequate data on aspects of tobacco use that are important or unique to specific population groups, such as use of menthol versus non-menthol tobacco products, or on specific populations, such as LGBT groups.

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Section II
Intrapersonal/Individual Factors Associated With Tobacco-Related Health Disparities

Chapter 3
Genetics, Physiological Processes, and Tobacco-Related Health Disparities

Contents

Figures

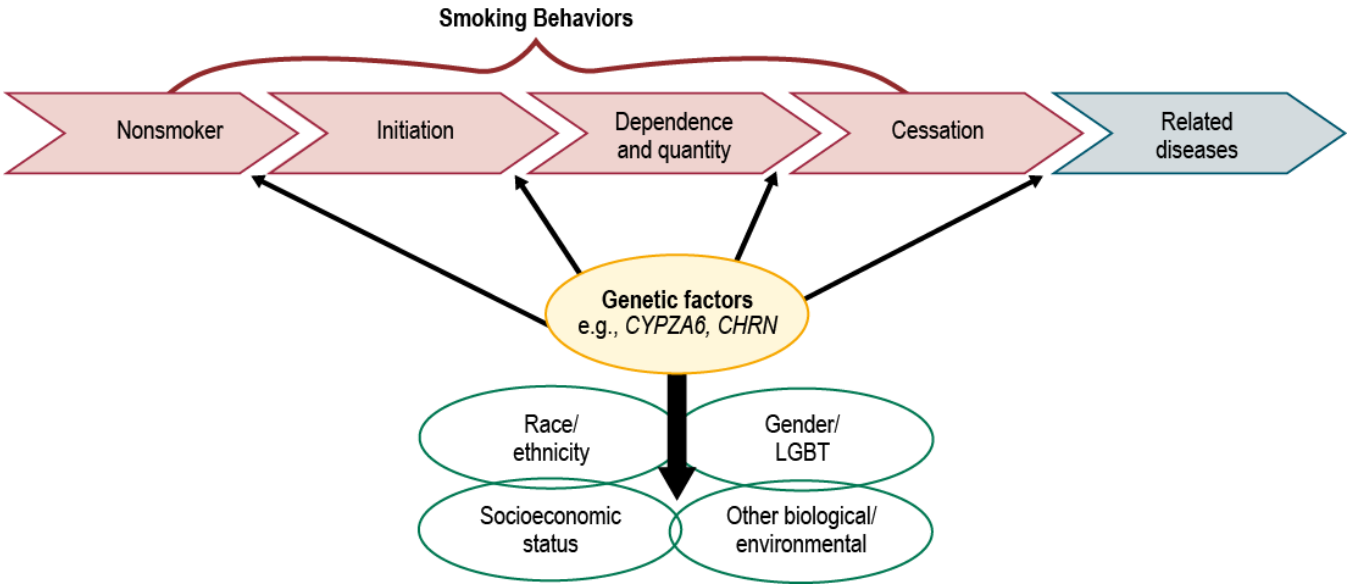
Introduction

This chapter explores the relationships between genetic factors and tobacco use behaviors and tobacco-related cancers. First, the chapter discusses genetic factors associated with nicotine metabolism and smoking initiation, progression to established smoking, smoking prevalence, and smoking cessation. Genetic risk factors typically vary in prevalence across racial/ethnic populations and thus can contribute to the manifestation of tobacco-related health disparities (TRHD) among racial/ethnic groups. Second, the chapter describes genetic factors associated with tobacco-related cancers, specifically lung cancer, and genetic factors that may influence how the body responds to carcinogens in tobacco smoke. It is important to keep in mind that complex interactions between genetic and environmental factors, many of which are correlated, influence interindividual susceptibility to harmful smoking behaviors and to the risk of tobacco-related diseases (Figure 3.1). More data are needed on high-risk segments of the population to pursue important leads about the relative role of genetic factors in TRHD.

The information presented in this chapter is based on a survey of the genetic factors for which the evidence is stronger (i.e., larger, more powered studies and/or replicated associations), not a meta-analysis of each gene investigated with respect to tobacco use and smoking behaviors. Broad search terms pertaining to genetics and smoking were first used to identify specific genetic factors, which were then individually investigated using the relevant gene name or gene region. The absence of a description of a specific genetic factor should not be interpreted as a negative result. Given that the overall purpose of this monograph is to explore and understand TRHD, there is a focus on discussing genetic factors that have been investigated across multiple racial/ethnic groups.

The chapter closes with a discussion of the current state of knowledge about genetic influences on TRHD, including critical knowledge gaps, such as the contribution of genetic factors in the context of complex socioeconomic environments.

Figure 3.1 Contribution of Genetic Factors to TRHD



Note: LGBT = lesbian, gay, bisexual, and transgender.

Genetic factors influence each stage of the tobacco use continuum, from initiation to cessation and to tobacco-related diseases. Tobacco use, smoking behaviors, and tobacco-related diseases are not seen uniformly across populations, and specific racial/ethnic and socioeconomic groups tend to bear a disproportionate burden of tobacco-related health outcomes. For example, African Americans report smoking fewer cigarettes per day compared with Americans of European descent, yet they are less likely to quit smoking and more likely to develop lung cancer.[1,2] Smoking initiation and progression to daily smoking also differ across racial/ethnic groups even after differences in socioeconomic status (SES) are accounted for.[3,4] In addition to race/ethnicity and SES, another important marker of tobacco-related health risk is sexual orientation: Smoking prevalence is significantly elevated in lesbian, gay, bisexual, and transgender (LGBT) groups.[5] The importance of these factors for different racial/ethnic groups is highlighted, where known. As of 2017, little research evidence is available on genetic factors and smoking behaviors stratified by SES or LGBT status.

Data Limitations on Genetic Factors Related to Disparate Populations

The evidence for a genetic contribution to smoking behaviors has largely been established in twin studies conducted mostly in European and European American populations. These twin studies have estimated that the heritability of smoking initiation is 36%–75%, and the heritability of cigarette consumption is 51%–86%.[6–9] Genetic factors also play a strong role in nicotine dependence (59%–75%) and smoking cessation (50%–58%).[6–11] The broad range for estimated heritability of a given smoking measure reflects the differing relative impacts of genetic and environmental influences and depends on multiple variables, including time (cohort), age, race/ethnicity, and societal and cultural context.[12–16]

It is noteworthy that the genetic components of each smoking measure are only modestly correlated with each other, indicating that there are unique and common genes contributing to each measure.[8,11,16] Specific genes and gene variants associated with smoking measures have been identified in candidate gene studies, which investigate variants chosen for their purported biological role, and genome-wide linkage and association studies have identified genomic regions of interest by testing the association of single nucleotide polymorphisms (tag SNPs) that label intervals across the genome with smoking measures.

Due to the complexity of smoking as a behavior, large-scale genetic association approaches involving data from many thousands of individuals have been favored to facilitate the detection of individual genetic signals. Out of necessity, such approaches tend to simplify or disregard the influence of the socioenvironmental context on the manifestation of genetic factors. Furthermore, investigations have largely been carried out within epidemiology studies primarily or exclusively conducted with people of European descent. Hence, for reasons of statistical power and the avoidance of population stratification, analyses have typically been restricted to the European subgroup, which is an impediment to our understanding of genetic factors in other racial/ethnic groups. In situations where genomic regions were first identified in a population of European descent and then were subsequently investigated in additional racial/ethnic populations, e.g., African Americans, Chinese, the tag SNPs tested in other populations might not be inherited with the same causal genetic variants because patterns of genetic variation are different across racial/ethnic populations.[17–22] Until the causal variants are identified, it can be difficult to compare the relative impact of variations in a gene across different racial/ethnic groups or to investigate the role of genetic variants between sociodemographic and environmental contexts relevant to TRHD.

Genetic Factors Associated With Nicotine and Smoking

Overview

Genetic factors associated with smoking behaviors include genetic variations in neurotransmitter systems within the brain reward pathways, neuronal plasticity and connectivity, and nicotine metabolism. In particular, and as expected, genetic variations in the nicotinic cholinergic system have been associated with a range of smoking behaviors, including smoking quantity and intensity, the risk of being nicotine dependent, and the level of nicotine dependence. The binding of nicotine to nicotinic acetylcholine receptors in the brain results in the release of neurotransmitters, such as dopamine, which is thought of as a neurotransmitter that signals reward-related events. For this reason, the genes coding for nicotinic receptor subunits have been the subject of intensive research efforts.[23,24] The α4 and β2 nicotinic receptor subunits, coded by the *CHRNA4* and *CHRNB2* genes, respectively, were leading candidates; these subunits are the most populous in the brain, form receptors with the highest affinity for nicotine, and alter nicotine self-administration in animals (as investigated using genetic and pharmacological manipulations).[23,25] However, genetic variations in other nicotinic receptor subunits are the most strongly associated with smoking behaviors.[23]

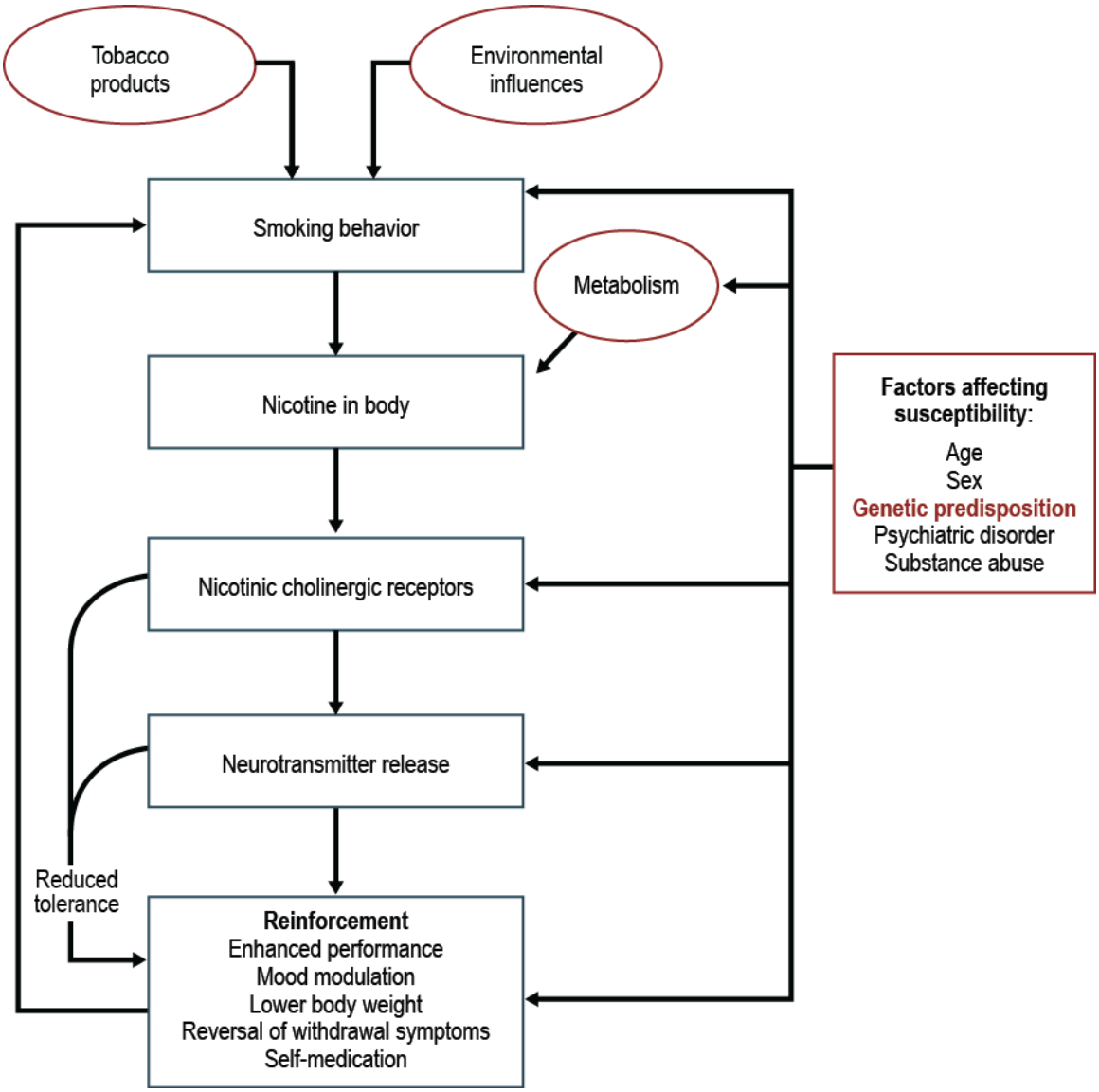
Genetic variations in the dopaminergic system also modulate smoking behaviors. Cigarette smoking increases dopamine in the brain,[24] and genes in this neurotransmitter system have been investigated as potential modifiers of smoking behaviors. However, genetic associations in the dopaminergic system are less universally reproducible than variations in nicotinic receptor subunits, possibly because dopamine release is a downstream consequence of nicotine binding to nicotinic receptors in the brain. In addition, the dopaminergic system is not specific to smoking; this system is a convergent pathway for many addictive (and other) behaviors, and genetic variations have been associated with multiple substance dependencies.[26,27]

Associations between genetic variations in other neurotransmitter systems and smoking behaviors are more equivocal. However, the glutamate receptor subunit gene *GRIN3A* is associated with smoking quantity and nicotine dependence scores (as determined by the Fagerström Test for Nicotine Dependence [FTND], a 6-item questionnaire scored from 0 to 10, which is predictive of relapse and primarily assesses aspects of withdrawal and the urge to smoke).[28–30] Moreover, the gamma-aminobutyric acid (GABA) receptor subunit genes *GABRA2* and *GABRA4* are associated with an increased risk of being a dependent (FTND $\geq$ 4) versus a nondependent (FTND = 0) smoker.[31] Studies focused on genetic variations in the serotonergic system, which are generally centered on a serotonin transporter variant with reduced expression, have largely failed to demonstrate significant associations with smoking initiation, behaviors, or cessation.[32–37]

Aside from genes in neurotransmitter pathways, genes involved with the formation and strengthening of neural connections are also associated with smoking behaviors. *NRXW1*, which codes for the neurexin 1 cell surface protein, was the strongest signal in a genome-wide association study (GWAS) on the risk of being a dependent (FTND $\geq$ 4) versus a nondependent (FTND = 0) smoker.[38] In addition, *NTRK2*, which codes for the brain-derived neurotrophic factor (BDNF) receptor, is associated with smoking quantity and FTND scores.[39] The association between variations in these genes, smoking quantity, and nicotine dependence suggests that genetic differences in learning and memory processes influence smoking behaviors.[40]

Genetic factors that influence nicotine metabolism are also associated with smoking behaviors. Nicotine is the main psychoactive ingredient in cigarettes that establishes and maintains dependence.[41] The complex biology of nicotine addiction is shown in Figure 3.2. Nicotine from inhaled cigarette smoke is rapidly and extensively metabolized by the liver. On average, less than 10% of absorbed nicotine is excreted in the urine unchanged.[42] Smokers adapt their smoking behaviors to maintain a preferred level of nicotine. The manipulation of nicotine clearance, through changes in nicotine metabolism or renal elimination, is associated with compensatory changes in smoking behavior.[43,44] Thus, genetic factors that affect the amount of nicotine available to bind to receptors in the brain, such as variations in the main nicotine metabolism gene, *CYP2A6*, are associated with cigarette consumption patterns, nicotine dependence, and smoking cessation.

Figure 3.2 Biology of Nicotine Addiction



Source: Benowitz 2010.[24]

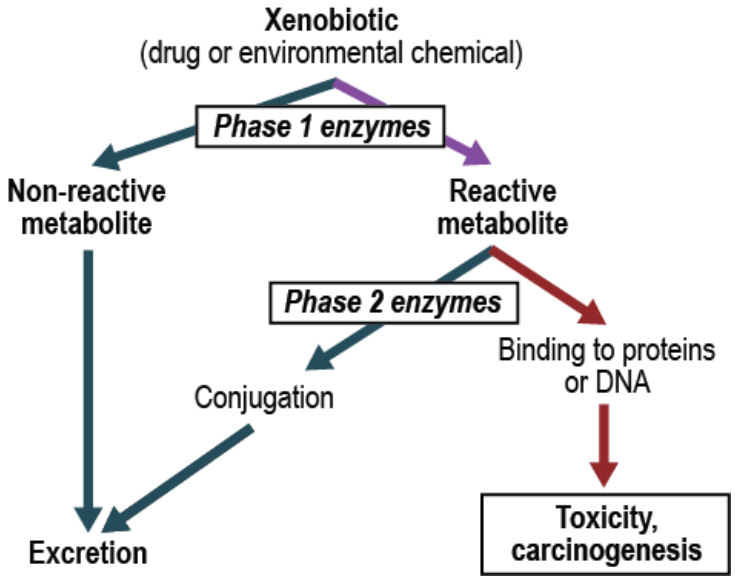
Nicotine Metabolism

Based on the existing evidence, overall rates of nicotine metabolism tend to be faster among populations of European descent; slower in Africans; and slowest in Asians, with Japanese as the slowest characterized population.[45–47] Early evidence in the Yupik population, a subgroup of Alaska Natives, indicates that nicotine metabolism could be highest in that population.[48] Within a population, rates of nicotine metabolism also show large interindividual variations.[46,49,50]

Genetic factors can account for a substantial proportion of the variability observed in the rate of nicotine clearance among individuals and racial/ethnic populations.[45,50,51] A twin study in European Americans estimated that additive genetic factors explained 59% of the variability in nicotine clearance.[52]

Cytochrome P450 genes produce enzymes whose action forms (synthesizes) chemicals or breaks them down (metabolizes them) to either non-reactive or reactive metabolites (Figure 3.3). In humans, names of the many different cytochrome P450 genes and their enzymes begin with "CYP." These CYP enzymes are extensively involved in metabolizing the carcinogens and toxicants such as nicotine found in cigarettes, and other forms of tobacco, drugs, and environmental chemicals typically influence whether the metabolism is fast or slow. These genes can be classified as Phase 1 and Phase 2 genes. Phase 1 genes can activate carcinogens by creating a reactive metabolite that binds to proteins or DNA or metabolize them to metabolites that are excreted.[53] Phase 2 genes generally de-activate these reactive substances, which are also then excreted, as shown in the figure.

Figure 3.3 Phase 1 and Phase 2 Drug-Metabolizing Enzymes



Source: Sozzani et al. 2005.[53]

Most nicotine (80%) is metabolically inactivated to cotinine,[54] and the *CYP2A6* enzyme mediates approximately 90% of this inactivation pathway.[55] In individuals who lack functional *CYP2A6*, there is a dramatic reduction in the rate of nicotine clearance.[46,51,56,57] In addition to *CYP2A6*, other CYPs (e.g., *CYP2B6*) make a minor contribution to the inactivation of nicotine to cotinine.[58]

Further metabolism of cotinine to trans-3'-hydroxycotinine is exclusively mediated by *CYP2A6*.[51,59] The ratio of trans-3'-hydroxycotinine to cotinine, often referred to as the nicotine metabolic ratio, is correlated with nicotine clearance and serves as a phenotypic marker for the rate of nicotine metabolism and for *CYP2A6* activity.[51] Nicotine is also inactivated to several minor metabolites, including nicotine N-oxide (~4%) and nicotine N-glucuronide (~4%); these pathways of inactivation are catalyzed by flavin-containing monooxygenases (FMOs) such FMO3, and by uridine diphosphate (UDP) glucuronosyltransferases (UGTs) such as UGT2B10, respectively.[42,60–63]

Genetic variants in *CYP2A6* are the most established genetic factors associated with nicotine clearance owing to the substantial contribution of *CYP2A6* to nicotine metabolism and to the characterization of numerous *CYP2A6* alleles (variant forms of the gene) with altered activity. The *CYP2A6* genotype influences many measures of nicotine metabolism and clearance, such as total and nonrenal clearance, clearance to cotinine, nicotine half-life, and the ratio of trans-3'-hydroxycotinine to cotinine.[50,51] Genetic variants in *CYP2A6* that affect activity include SNPs as well as gene deletions, duplications, and conversions.[64]

Examples of *CYP2A6* alleles encoding *CYP2A6* enzymes that are inactive toward nicotine include the *CYP2A6*2*, **4*, **7*, and **17* alleles.[64–66] The *CYP2A6*4* allele is an example of a deletion allele; it has 0% enzymatic activity relative to the nonvariant (wild-type) allele—*CYP2A6*1*. Individuals generally possess two alleles for each gene; thus, individuals possessing two copies of *CYP2A6*4* have no *CYP2A6* enzymatic activity, resulting in nearly undetectable levels of cotinine and no detectable trans-3'-hydroxycotinine, and the small amount of cotinine formation that takes place is catalyzed by other enzymes.[46,57,67] Possession of one copy each of the 0% activity (i.e., inactive) *CYP2A6*4* allele and the 100% activity *CYP2A6*1* allele is associated with a 50% reduction in both *CYP2A6* activity and total nicotine clearance, on average, compared with the possession of two copies of the *CYP2A6*1* allele.[50,68]

Some *CYP2A6* alleles have decreased, rather than absent, nicotine metabolic activity; as examples, the *CYP2A6*9* and **12* alleles have approximately 50% lower activity compared with *CYP2A6*1*.[64] On average, individuals possessing one copy of the 50% activity *CYP2A6*9* or **12* allele and one copy of the 100% activity *CYP2A6*1* allele have 75% of the *CYP2A6* activity and 80% of the total nicotine clearance of *CYP2A6*1/*1* individuals.[50,68]

Due to the large number of low-frequency *CYP2A6* alleles, individuals with reduced-activity alleles are commonly grouped together as *CYP2A6*-reduced (<75% activity) metabolizers for data analyses.[69] *CYP2A6*-reduced metabolizers can be further subdivided by genotype into *CYP2A6*-slow (≤50% activity) and *CYP2A6*-intermediate (~75% activity) metabolizers.[68,70,71] A small number of *CYP2A6* alleles with increased activity, such as *CYP2A6*1B* and the duplication allele *CYP2A6*1X2*, have also been characterized.[72–74]

Thus, genetic variations in *CYP2A6* result in a wide range of enzyme activity and, consequently, are associated with a wide range of nicotine metabolism and clearance rates. Consistent with the prominent role of *CYP2A6* in the metabolic inactivation of nicotine and total nicotine clearance, genetic variations

in *CYP2A6* are strongly associated with multiple smoking behaviors. The most robust genetic associations pertain to cigarette consumption, followed by nicotine dependence and smoking cessation (discussed in subsections below).

Variations in *CYP2A6* contribute to the racial/ethnic differences observed in nicotine metabolism. Although *CYP2A6* alleles have a similar impact on *CYP2A6* activity and nicotine metabolism among different racial/ethnic populations,[46,57,71] the frequency of alleles varies significantly across populations, resulting in large differences in the relative rates of nicotine metabolism. The collective frequency of reduced-activity genetic variants parallels the population rates of nicotine metabolism; overall, ~20% of whites, ~40% of African Americans, ~55% of Chinese, and ~80% of Japanese populations have *CYP2A6*-reduced metabolism genotypes.[64] Recent investigations into populations that are less well characterized in terms of *CYP2A6*, such as Alaska Natives, suggest that novel increased-activity *CYP2A6* gene variants could be contributing to the comparatively high rates of nicotine metabolism that persist after accounting for known genetic variants.[48]

Genetic variants such as the *CYP2A6*4* and *9* alleles are found in all populations tested to date, albeit at different allele frequencies. For example, the frequency of the *CYP2A6*4* deletion allele ranges from 0% to 4% in white, 0% to 2% in African American, 5% to 15% in Chinese, and 17% to 24% in Japanese populations. Other variants are found predominantly in one population; the *CYP2A6*7* allele is typically only detected in Asian populations (6%–13% frequency),[46,75–77] and the *CYP2A6*17* allele is typically only detected in African American or black populations (7%–11% frequency).[46,71] On a population level, the overall percentage of individuals possessing altered-activity *CYP2A6* gene variants (i.e., the portion of the population with reduced activity) matters more to the overall rate of nicotine metabolism than the specific gene variants that are found in that racial/ethnic population, because the impact of characterized *CYP2A6* variants is similar across populations, as assessed using genotype-to-phenotype measurements.

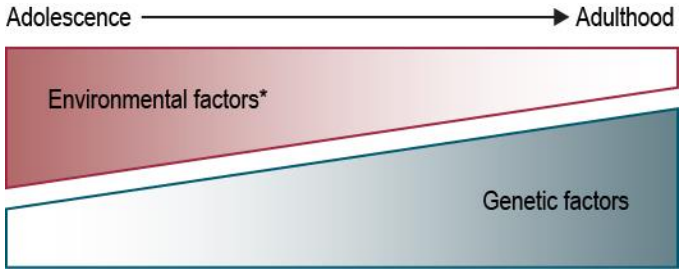
In addition to *CYP2A6*, *CYP2B6* also converts nicotine to cotinine, and there are *CYP2B6* genetic variants with increased and decreased enzymatic activity.[78] However, *CYP2B6* plays a comparatively minor role in nicotine metabolism, and variations in the *CYP2B6* gene have not been associated with differences in nicotine metabolism when activity at the adjacent *CYP2A6* gene was accounted for.[79,80] Similarly, variations in the genes coding for other enzymes involved with the metabolic inactivation of nicotine, such as FMOs and UGTs, have a small influence on the total removal of nicotine. Therefore, although not studied extensively, these genes have not been shown to significantly alter smoking behaviors.[63,64,81] However, although much of the research has focused on clearance of nicotine in major body compartments, some of these non-*CYP2A6* pathways such as FMO may affect the clearance of nicotine from the central nervous system.[82]

Smoking Initiation

Smoking initiation occurs predominantly in adolescence, a time when environmental influences account for a greater proportion of risk of initiation, in contrast with early and middle adulthood, when genetic factors increase in importance (Figure 3.4).[16,83] Investigating the role of genetic versus environmental factors in smoking initiation is made more challenging by the fact that the relative contributions of these factors change with age. Smoking initiation in adolescence is particularly characteristic of populations of European descent; as outlined in chapter 2, a greater proportion of African Americans and Asian Americans initiate regular smoking after age 18. For a review of twin studies in adolescents, refer to

chapter 6 of National Cancer Institute (NCI) Monograph 20, *Phenotypes and Endophenotypes: Foundations for Genetic Studies of Nicotine Use and Dependence*.[84]

Figure 3.4 Relative Contributions of Genetic and Environmental Factors to Smoking Initiation



*Societal and cultural contexts influence the role of environment.

Specific genetic factors involved with smoking initiation have been less well characterized than genetic influences on later stages of the tobacco use continuum. Studies conducted in adult populations are hampered by recall bias and might fail to account for important environmental influences; thus, prospective longitudinal studies in adolescents are better suited to investigating genetic risk factors for smoking initiation in the context of changing socioenvironmental influences. However, these studies are often difficult to conduct, and few have been undertaken to date. (For recommendations on the genetic modeling of smoking trajectories, see NCI Monograph 20, chapter 6.[84]) Nevertheless, one longitudinal study of adolescents assessed tobacco use over a period of years and found that having a higher genetic risk score based on variants in genes involved in tobacco dependence was associated with more rapid progression to tobacco dependence and heavier smoking and more failures in cessation attempts, but was not related to smoking initiation.[85]

Smoking initiation has been associated with variations in the dopaminergic system in particular. Variations in the *TTC12-ANKK1-DRD2* gene cluster and in the dopamine receptor gene *DRD4* are associated with an increased risk of ever smoking.[86–89] Of note, the relationship between smoking initiation and variations in the *TTC12-ANKK1-DRD2* gene cluster and in the *DRD4* gene is influenced by novelty-seeking and depressive symptoms.[86,88,90] This finding highlights the importance of incorporating endophenotypes (heritable observable characteristics) such as personality traits, through which gene variants could be operating, to influence the risk for smoking initiation.[91] Not all studies in adolescents find that *TTC12-ANKK1-DRD2* variations are implicated in smoking initiation; these variations are associated with continued smoking, progression to higher levels of smoking, and daily smoking, but not initiation, in other adolescent populations.[32,86,90]

Variations in genes involved with neural connectivity and plasticity are also associated with smoking initiation. Cell adhesion genes such as *CDH13* and the BDNF receptor gene *NTRK2* are associated with adults' risk of ever having been a smoker versus never having been a smoker, as are variations in the glutamatergic receptor subunit genes *GRIN2B, GRIN2A, GRIK2,* and *GRM8*.[35,92] These studies were mainly conducted in European and European American populations,[93] thus data are currently insufficient to determine whether genetic factors contribute to racial/ethnic differences in the epidemiology of smoking initiation.

Smoking Quantity, Dependence, and Age of Smoking Initiation

Genetic factors influence cigarette consumption and nicotine dependence, which differ by race/ethnicity and SES and are important contributors to tobacco-related health outcomes. This section describes the most robust genetic associations and discusses genetic factors within the nicotinic cholinergic and dopaminergic neurotransmitter systems and variations in the nicotine metabolism gene *CYP2A6*. These genetic factors currently do not explain differences in average cigarette consumption by race/ethnicity, but they contribute to differences among individual smokers of a given race/ethnicity.

Nicotinic Cholinergic System Genetic Factors

Within the nicotinic cholinergic system, genetic variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster are most strongly associated with nicotine dependence and daily cigarette consumption; large-scale meta-analyses of GWASs confirm the association.[20–22] Although the genetic association is strong, the effect size of variations in the gene cluster is modest; each minor allele of the most significant genetic marker accounts for only 0.5% of the variance in cigarettes per day, for an increase in daily cigarette consumption of approximately one cigarette.[21] However, using cigarette consumption as a surrogate for daily nicotine dose could underestimate the influence of variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster on nicotine intake. Even at a given level of cigarette consumption, individuals differ in how intensively they smoke each cigarette.[94] Compared with cigarettes per day, biomarkers of cigarette exposure, such as cotinine and urinary total nicotine equivalents, better reflect smoking level and nicotine intake and can be used to estimate smoking intensity per cigarette.[94–96]

There are caveats to comparing cotinine levels among individuals, however since nicotine metabolism varies by age, sex, race, diet, and pregnancy status.[45,97,98] Variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster are also associated with differences in cotinine levels among smokers and account for comparatively more of the variation in cotinine than cigarettes per day, which is indicative of more intensive smoking.[99–101] For instance, Keskitalo and colleagues discovered that each copy of the genetic variant was associated with an increase in serum cotinine of 77 ng/mL, which would be equivalent to ~6 cigarettes per day, assuming 12–13 ng/mL of cotinine per cigarette, whereas an increase in only 1.2 cigarettes per day per allele was reported, suggesting that the increase in cotinine largely reflected more intensive smoking.[99,102] Further evidence for an association of variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster and greater smoking intensity comes from a multiracial/ethnic population study of heavy smoking (greater than 10 cigarettes per day), which assessed total nicotine equivalents adjusted for cigarette consumption.[103]

Variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster are also associated with nicotine dependence, as indicated by smoking heaviness and the FTND. Genetic variations in this chromosomal region increase the risk for being a heavy smoker (25 or 30 cigarettes per day) rather than a light smoker (fewer than or equal to 5 cigarettes per day).[104,105] (The typical cutoff employed for a light smoker is less than or equal to 10 cigarettes per day.) FTND scores and the risk for being a dependent (FTND $\geq$ 4) versus a nondependent (FTND = 0) smoker also increase with genetic variations in the cluster.[38,106,107]

The GWAS that identified the *CHRNA5-CHRNA3-CHRNB4* gene cluster were conducted in European and European American populations.[20–22,108,109] Important risk variants in populations of European origin include a specific variation in SNP rs16969968 in exon 5 of *CHRNA5*, in linkage disequilibrium with rs1051730 in *CHRNA3*, as well as rs578776 in *CHRNA3*, a separate SNP.[110] Tag SNPs within the region have been subsequently tested in additional racial/ethnic populations. They were found to be a

significant risk factor for cigarette consumption and nicotine dependence in Hispanics, African Americans, and Asians,[18,19,111–113] although the risk SNPs may differ between race/ethnic groups.[112] The cluster was also associated with a biomarker of smoking quantity (total nicotine equivalents) among Alaska Native smokers.[114] Moreover, multiple distinct loci within the *CHRNA5-CHRNA3-CHRNB4* gene cluster have been associated with smoking behaviors, but the precise functional variants in the region remain to be confirmed.[20,112,115] Once the causal genetic variants have been identified and characterized, it will be feasible to evaluate the role of nicotinic receptor variants in the context of environmental factors that are important to TRHD.

Variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster might also interact with age to influence smoking behaviors. Certain variations in the *CHRNA5-CHRNA3-CHRNB4* are also associated with age of onset of smoking.[116] In another study, Weiss and colleagues[117] found that the association between the genomic region and the severity of nicotine dependence was limited to individuals who began daily smoking at or before age 16, whereas Ducci and colleagues[88] found that variations in the region conferred the same degree of risk for smoking at age 14 (regular versus nonsmoker) as at age 31 (heavy versus nonsmoker). A meta-analysis of the *CHRNA5-CHRNA3-CHRNB4* SNP rs16969968 compared smoking heaviness among individuals with one risk allele and found that those who started smoking by age 16 were at greater risk for heavy smoking compared with those who started smoking after age 16.[118] Given that the age of smoking initiation varies substantially by race/ethnicity and SES (see chapter 2), age could be an important consideration when interpreting the association of the *CHRNA5-CHRNA3-CHRNB4* gene cluster with smoking behaviors.

Additional nicotinic receptor subunit genes associated with smoking include *CHRNB3* and *CHRNA4*. Variations in *CHRNB3* are associated with smoking quantity and the risk of being a dependent versus a nondependent smoker.[22,38,106] Variations in *CHRNA4* are associated with nicotine dependence, as defined by the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* (DSM-IV)[119] and the FTND.[120–122] Variations in *CHRNB2* might also influence the risk for being a dependent smoker but only through interactions with variations in other genes, such as *CHRNA4*.[123] Finally, other variants in the cholinergic receptor subunit genes may also be important in African Americans and European-origin populations.[108]

Aside from nicotinic receptor subunits, variations in the choline acetyltransferase gene (*ChAT*), which codes for a key enzyme in the synthesis of endogenous acetylcholine, are also associated with smoking quantity and FTND scores.[124,125] Other chromosomal regions and genes might also be important risk factors for cigarette consumption and nicotine dependence in populations of non-European descent. For example, in one study, risk variants for a population of African American but not European origin were found in regions on chromosome 8 and chromosome 14, but not with the *CHRNA5-CHRNA3-CHRNA4* cluster.[126] In an Asian population, variants in *FRMD4A* genes were identified which were associated with nicotine dependence and also age of initiation.[127]

Dopaminergic Neurotransmitter System Genetic Factors

Within the dopaminergic system, genetic variations in the dopamine receptor subunit gene *DRD2* have received much attention because of this gene's central role in the dopamine reward system.[26,27] The *DRD2* gene is part of the *TTC12-ANKK1-DRD2* gene cluster, and variations in the cluster are associated with cigarette consumption and the degree of nicotine dependence, as assessed by FTND scores and the

heaviness of smoking index.[32,128–130] In addition, variations in *TTC12-ANKK1-DRD2* are also associated with the risk for being nicotine dependent as assessed by the DSM-IV.[131]

The cluster likely influences smoking behaviors through dopamine receptor expression. The commonly investigated Taq1A variant, which resides in the *ANKK1* gene, is associated with reduced *DRD2* mRNA and dopamine D2 receptor density.[87,130] This genetic region is associated with smoking quantity and nicotine dependence in African Americans and European Americans.[125,130] However, different locations within the *TTC12-ANKK1-DRD2* gene cluster are associated with dependence risk, possibly owing to differences in the underlying pattern of genetic variations among these racial/ethnic populations.[130,131]

Few studies have investigated genetic variations in dopamine receptor genes aside from *DRD2*, in part because these other receptors and their genetic variants are not as well characterized.[26] However, functional genetic variants in the *DRD1* and *DRD3* dopamine receptor subunit genes have also been associated with smoking quantity and FTND scores.[128,132,133] The relative importance of variations in dopamine receptor genes differs across racial/ethnic populations; family studies suggest that genetic variations in *DRD1* modulate smoking quantity and FTND scores preferentially in African Americans, whereas genetic variations in *DRD3* modulate smoking quantity and FTND scores preferentially in European Americans.[132,133]

Nicotine Metabolism Gene CYP2A6 Genetic Factors

It is well established that variations in the nicotine metabolism gene *CYP2A6* affect the amount of cigarette consumption. Cigarette smokers who possess *CYP2A6* genotypes that are associated with reduced rates of nicotine metabolism smoke fewer cigarettes per day compared with those who possess normal *CYP2A6* metabolizer genotypes, particularly among racial/ethnic groups characterized by heavier smoking.[68,70,72,76,134,135] Among European American smokers, those with *CYP2A6*-reduced metabolizers smoked an average of 20 cigarettes per day, compared with those with *CYP2A6*-normal metabolizers, who smoked 26 cigarettes per day.[135] Among Japanese smokers, cigarette consumption ranged from approximately 15 cigarettes per day in those without functional *CYP2A6* (*CYP2A6*4/*4*) to as many as 30 cigarettes per day in predicted normal metabolizers (*CYP2A6*1/*1*).[76,134] Chinese smokers with *CYP2A6*-reduced metabolizers also smoked fewer cigarettes per day, but the role of genetic factors in smoking behaviors has not been investigated as extensively in this population.[77] In contrast to heavy-smoking populations, cigarette consumption is not associated with *CYP2A6* genotype groups among African American light smokers[71,136] or Alaska Native light smokers.[137] Light smoking is also prevalent among Hispanic Americans and in the Asian American aggregate group,[138] but less is known about *CYP2A6* and smoking in these populations.

Among light smokers, biomarkers that operate as reliable indicators of smoking levels in European heavy-smoking populations, such as plasma cotinine, and exhaled carbon monoxide, have limited utility.[94,136] The relationship between self-reported cigarette consumption and either exhaled carbon monoxide or plasma cotinine is substantially weaker in African American light smokers, making it difficult to investigate the role of *CYP2A6* genotype groups.[136] As of 2015, it was unclear whether differences in the utility of biomarkers could be ascribed to light smokers generally or to African American light smokers specifically.[96,136] Further complicating the utility of cotinine, the most commonly used marker of nicotine intake, it has been demonstrated that the relationship between cotinine and nicotine dose is affected by *CYP2A6* genetics and sex.[97] Future studies employing more reliable biomarkers of nicotine intake, such as urinary total nicotine equivalents, are necessary to

determine whether variations in *CYP2A6* are an important determinant of smoking behaviors in light-smoking populations. Another biochemical indicator of consumption, carbon monoxide, appears to yield strong associations with *CHRNA5-CHRNA3-CHRNB4* as well as *CYP2A6*.[139]

As with the *CHRNA5-CHRNA3-CHRNB4* gene cluster, the use of cigarette consumption as a surrogate for daily nicotine dose can underestimate the influence of variations in *CYP2A6* on nicotine intake. Smokers might titrate (i.e., adjust) nicotine levels through their cigarette smoke inhalation patterns as well as change the number of cigarettes smoked. The *CYP2A6* genotype is associated with smoking intensity among European American smokers, with *CYP2A6*-reduced metabolizers taking smaller volume puffs compared with *CYP2A6*-normal metabolizers.[140] Nicotine titration was also evident in an open-label clinical trial of nicotine replacement therapy (NRT), where *CYP2A6*-reduced metabolizers achieved similar nicotine plasma levels when compared with normal metabolizers by using fewer daily sprays of nicotine nasal spray.[68]

Variations in *CYP2A6* also influence smokers' progression toward nicotine dependence and final level of dependence. In European American adolescents, *CYP2A6*-slow metabolizers progress in nicotine dependence at a slower rate and reach a stable level of dependence more quickly compared with normal metabolizers.[141–143] Slow *CYP2A6* metabolism, however, is a risk factor for acquiring nicotine dependence in adolescence, and the existence of one to two copies of the inactive alleles, *CYP2A6*2* or **4* increases the risk of conversion to nicotine dependence.[141] Once dependent, slow metabolizers consume fewer cigarettes compared with normal metabolizers.[141,142] Thus, longitudinal cohort studies of adolescents have suggested that *CYP2A6*-slow metabolizers acquire nicotine dependence sooner (after initial exposure to nicotine), reach a plateau in their degree of dependence earlier, and have lower levels of cigarette consumption and nicotine dependence compared with normal metabolizers.[143] Given the importance of environmental influences in adolescence, the association of *CYP2A6* and dependence trajectories must be assessed by race/ethnicity and SES.

Lower levels of nicotine dependence in *CYP2A6*-reduced versus *CYP2A6*-normal metabolizers are also seen in adult European American cigarette smokers, with *CYP2A6*-reduced (<75% activity, as predicted by genotype) versus *CYP2A6*-normal metabolizers having significantly lower FTND scores.[135] A component of the FTND is the time to first cigarette in the morning; *CYP2A6*-slow metabolizers trend toward a reduced likelihood of smoking within the first 5 minutes of waking compared with normal metabolizers.[68] Japanese smokers with *CYP2A6*-reduced metabolism genotypes also have lower FTND scores and are less likely to smoke their first cigarette within 5 minutes of waking compared with *CYP2A6*-normal metabolizers.[144]

Smoking Cessation

Genetic factors have been demonstrated to affect smoking cessation. Disparities in the ability to quit smoking and the ability to quit smoking with and without nicotine pharmacotherapy are both important risk factors for tobacco-related adverse health outcomes. As outlined in chapter 2, the frequency and success of quit attempts differ by race/ethnicity and SES. For instance, African Americans are less likely to achieve smoking cessation than European Americans. In addition, light smokers appear to have lower abstinence rates compared with moderate-to-heavy smokers on either placebo or pharmacotherapy; however, this finding has not been tested directly.[145,146]

As with smoking behaviors, multiple neurotransmitter systems are implicated in the ability to quit smoking, with the most replicated associations found among genetic variants in the dopaminergic system. In this system, two variants in the *TTC12-ANKK1-DRD2* gene cluster, Taq1A (*ANKK1* rs1800497 C>T) and *DRD2*-141 Ins/Del C, are associated with smoking cessation in clinical trials and in a general care setting,[37,147–149] although an earlier meta-analysis found no association between variations in the *TTC12-ANKK1-DRD2* gene cluster and smoking cessation.[32] Individuals with the Taq1A T/T (also known as A1/A1) genotype are more likely to be abstinent, regardless of the type of treatment, compared with those with the Taq1A C/C (A2/A2) genotype,[149] but individuals with the Taq1A C/C genotype benefit more from bupropion versus placebo.[37,148,149] Regarding the *DRD2*-141 Ins/Del C variant, individuals with a Del C allele have higher quit rates with transdermal NRT compared with those with the InsC/InsC genotype in an open-label NRT study. Individuals with the InsC/InsC genotype benefit more from bupropion versus placebo.[37,147] In addition to the *TTC12-ANKK1-DRD2* gene cluster, variations in *DRD4* are associated with a reduced likelihood of abstinence, regardless of therapy.[150] In contrast, genetic variations in *DRD2* were not associated with spontaneous cessation in a large population-based sample of smokers.[151] It is noteworthy that sex might modify the influence of genetic factors in the dopaminergic system; the strength of the association between variations in *DRD2* and smoking cessation is related to the proportion of men in the study population.[87,152]

The catechol-O-methyltransferase (COMT) enzyme, which metabolizes catecholamines, including dopamine, is also associated with transdermal NRT quit rates. Specifically, individuals homozygous for the decreased-activity rs4680 A variant (rs4680 G>A, also known as Val[108,158]Met) have a greater probability of quitting with NRT than placebo compared with individuals with the G/G or G/A genotype.[37,153,154] Variations in the COMT gene have also been associated with responses to bupropion in which only individuals with the rs4680 A allele benefit from bupropion rather than placebo.[155] Moreover, variations in the dopamine transporter gene, *SLC6A3*, were associated with cessation in a meta-analysis[156] but not in a more recent population-based study.[151] In addition to clinical studies of drug therapies, an association between the *CHRNA5-CHRNA3-CHRNB4* gene cluster and smoking cessation in large human population studies has been observed.[157–160]

In the cholinergic system, variations in the nicotinic acetylcholine receptor subunit *CHRNB2* are associated with the ability to quit smoking on either bupropion or placebo even 6 months following treatment.[161] Variations in *CHRNB2* were also associated with the ability to remain abstinent for a longer period on transdermal NRT versus placebo in a crossover study.[162] Variations in the choline acetyltransferase gene *ChAT* are associated with cessation outcomes on transdermal NRT in open-label studies.[124] Initially, variations in the *CHRNA5-CHRNA3-CHRNB4* gene cluster appeared to have a negligible role in smoking cessation.[124,163–165] However, subsequent investigations revealed an association between SNPs in this cluster and a reduced ability to quit unaided, which was mitigated by pharmacological treatment (NRT in particular).[159,166] One study also suggested that variants in *CHRNB2*, *CHRNA4*, and *CHRNA7* and in the *CHRNA5-CHRNA3-CHRNB4* gene cluster influence abstinence while an individual is taking varenicline, a pharmacological treatment that acts at nicotinic receptors by partially mimicking the effects of nicotine.[167] Variation in the *CHRNA5-CHRNA3-CHRNB4* gene cluster may also influence response to the investigational cessation aid selegiline.[168] Importantly, variation in this gene cluster is also associated with smoking abstinence on active pharmacotherapy among African American smokers.[169]

Genetic variations in the μ-opioid receptor have also been implicated in smoking cessation. Nicotine stimulates the release of endorphin, which binds to μ-opioid receptors, and these receptors mediate

feelings of withdrawal.[170] Hence, candidate gene studies have investigated the association between variations in the μ-opioid receptor gene *OPRM1* and smoking cessation—in particular, the rs17999971 A>G variant, because the G allele is associated with reduced receptor expression.[37] In an open-label study of NRT, individuals with the rs17999971 G/G or G/A genotype had higher abstinence rates on transdermal NRT,[171] whereas in a placebo-controlled trial of NRT, individuals with the rs17999971 A/A genotype had higher abstinence rates on active treatment.[172] These observations, coupled with the *DRD2* and *CHRNA5-CHRNA3-CHRNB4* findings, highlight the importance of treatment condition in genetic association studies of smoking cessation; the effect of a genetic variant that reduces general quit ability might be mitigated by pharmacotherapy.

Genetic variations in *CYP2A6* are associated with smoking cessation, both unassisted and assisted by pharmacotherapy. *CYP2A6*-reduced metabolizers appear to have higher levels of smoking cessation, as European, European American, and Japanese individuals possessing the inactive *CYP2A6*4* allele have a lower likelihood of being a current smoker and have a greater likelihood of quitting compared with *CYP2A6*-normal metabolizers.[70,173,174] Consistent with these findings, the proportion of *CYP2A6*-slow metabolizers (<50% activity, as predicted by genotype) among current smokers decreases as smoking duration increases.[70] The influence of *CYP2A6* is even apparent in adolescent ever-smokers, as a greater proportion of slow versus normal metabolizers had quit smoking for at least 1 year.[175] Additional evidence for an increased ability to quit smoking among *CYP2A6*-slow metabolizers comes from the placebo arm of clinical trials. The slowest quartile of nicotine metabolizers, as assessed by the nicotine metabolic ratio (a biomarker of *CYP2A6* activity and genotype), have higher quit rates on placebo compared with the fastest three quartiles of nicotine metabolizers in European Americans and African Americans.[71,176]

Rates of nicotine metabolism, which influence how nicotine levels fluctuate after a cigarette, can affect the development of conditioned responses among smokers; functional brain imaging has demonstrated greater neural responses to smoking cues in faster versus slower *CYP2A6* genotypes.[177] Slow versus fast nicotine metabolism is also predictive of higher abstinence rates on transdermal NRT and of less intense cigarette cravings in the week following the target quit date in populations of predominantly European descent.[178,179] Furthermore, in a clinical trial comparing normal and extended NRT, only those with *CYP2A6*-reduced metabolism genotypes were found to benefit from extended therapy.[69] *CYP2A6* is related to cessation success in those getting NRT. There is evidence that cessation success is related to NRT and not to bupropion pharmacotherapy, and that the contribution of *CYP2A6* is independent of that of *CHRNA5-CHRNA3-CHRNB4*.[180] Together, these studies suggest that variations in *CYP2A6* modulate smoking cessation outcomes; *CYP2A6*-reduced metabolizers benefit more from transdermal NRT compared with normal metabolizers and, more importantly, are able to quit more easily even without the use of pharmacotherapy. A clinical trial found that normal nicotine metabolizers were more successful in smoking cessation on varenicline than on nicotine patches, but this was not the case among the slow nicotine metabolizers who also experienced more side effects on varenicline than did the normal metabolizers. This suggests the potential for nicotine metabolism testing to help in identifying who may benefit most from specific cessation therapies.[181]

Genetic variations in *CYP2B6* also appear to influence smoking cessation outcomes. *CYP2B6* genetic variants could be a risk factor for a reduced likelihood of quitting smoking, as reduced-expression variants are associated with increased cigarette craving and reduced abstinence rates in the placebo arm of clinical trials.[80,182,183] Importantly, *CYP2B6* is the main enzyme that metabolizes the smoking cessation drug bupropion,[184] and variations in *CYP2B6* are associated with bupropion treatment response

and with altered levels of hydroxybupropion (an active metabolite) in pharmacogenetic studies of smoking cessation.[167,185] Thus, although variations in *CYP2B6* might not be a significant genetic factor in nicotine metabolism and cigarette consumption, they might predict the likelihood of quitting smoking unassisted or assisted by bupropion, but possibly not assisted by nicotine replacement therapy.[183]

Summary for Tobacco Smoking Initiation, Nicotine Dependence, and Cessation

Smoking is a complex behavior, with genetic and environmental influences operating at each stage along the tobacco use continuum. The relative contribution of genetic and environmental factors to a given smoking behavior differs by cohort, age, race/ethnicity, and sociocultural context, and genetic factors characterized in one context cannot be readily extrapolated to another. Most of the evidence concerning genetic factors comes from studies in populations of European descent.

This section described genetic factors in neurotransmitter systems, neuronal connectivity and plasticity, and nicotine metabolism and their effects on smoking behaviors. Common and unique genetic risk factors in neurotransmitter systems and neural connectivity are associated with smoking behaviors along the tobacco use continuum, from initiation to cessation.[186] Genetic factors related to smoking initiation include variations in dopamine receptor genes and neuronal connectivity genes. Nicotine dependence and smoking levels are strongly associated with variations in nicotinic receptor genes, in particular the *CHRNA5-CHRNA3-CHRNB4* gene cluster. Variations in dopaminergic and neuronal connectivity genes are also associated with nicotine dependence, and variations in dopaminergic genes are associated with smoking cessation. Although racial/ethnic populations appear to share certain genetic factors, such as variations in the *CHRNA5-CHRNA3-CHRNB4* and *TTC12-ANKK1-DRD2* gene clusters, the specific risk SNPs are sometimes different in the different population groups. Studies encompassing each racial/ethnic group are necessary to determine the precise genetic variants operating within each population and the importance of genetic factors relative to sociocultural factors.

Variations in nicotine metabolism genes are also associated with smoking behaviors. *CYP2A6* is the main enzyme that metabolically inactivates nicotine, and variations in the *CYP2A6* gene are an established genetic factor affecting nicotine metabolism and, consequently, nicotine clearance. In line with evidence that cigarette smokers titrate their nicotine intake from cigarettes to maintain a preferred level of nicotine, individuals with *CYP2A6*-reduced metabolism genotypes smoke fewer cigarettes per day and take smaller puff volumes compared with *CYP2A6*-normal metabolizers. *CYP2A6*-reduced metabolizers progress more slowly in their level of nicotine dependence as youths and have lower nicotine dependence scores as adults. Variations in *CYP2A6* are also associated with increased smoking cessation, both the ability to quit without pharmacotherapy and the ability to quit on transdermal NRT. Although the frequency of *CYP2A6*-reduced metabolism genotypes varies according to race/ethnicity, the functional impact of *CYP2A6* genotypes on nicotine metabolism appears to be consistent across racial/ethnic groups. In heavier smoking populations, principally men of Japanese, Chinese, and European descent, a consistent association between variations in *CYP2A6* and smoking behaviors such as cigarette consumption is emerging. Less is known about the effects of *CYP2A6* genetic variations in lighter smoking populations, such as African Americans. Genetic factors that influence smoking behaviors among racial/ethnic populations characterized by lighter smoking are not well understood in part due to the inadequacy of common cigarette smoke exposure biomarkers in light smokers.

Genetic Factors Associated With the Risk for Lung Cancers

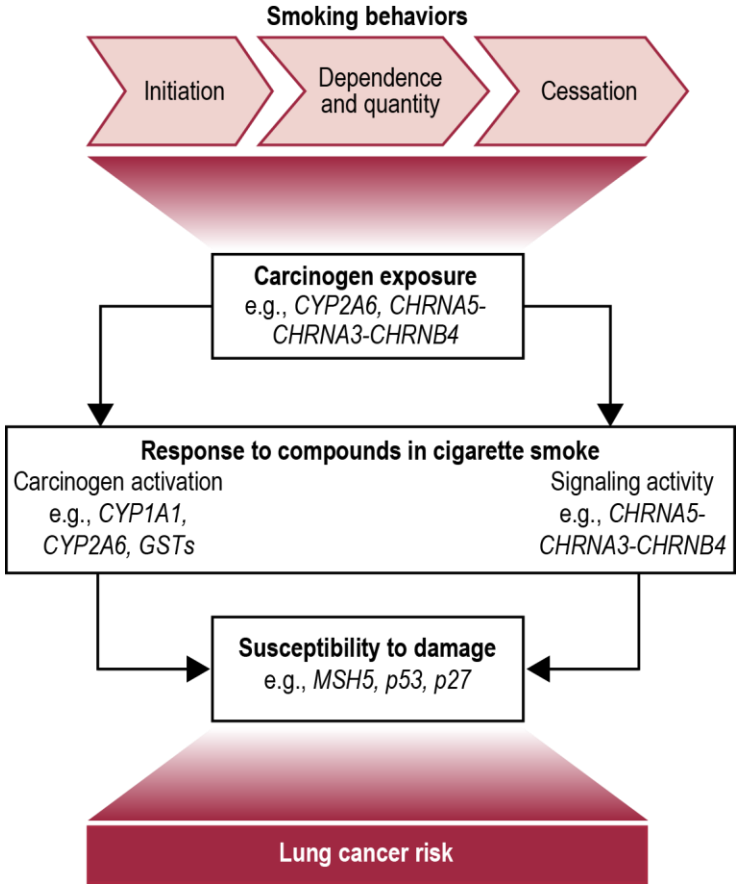
Multiple factors contribute to the risk for developing tobacco-related cancers and to population disparities in risk such as differences in smoking behaviors (e.g., the prevalence and/or amount of smoking) and socioeconomic and environmental influences. One such factor, genetic factors,[2,187] is the focus of this section. Cigarette smoking and secondhand smoke exposure are associated with numerous cancers, including those of the oral cavity, oropharynx, nasopharynx, and hypopharynx, oesophagus (adenocarcinoma and squamous cell carcinoma), stomach, colorectum, liver, pancreas, nasal cavity and paranasal sinuses, larynx, lung, uterine cervix, ovary (mucinous), urinary bladder, kidney (body and pelvis), ureter, and bone marrow (myeloid leukemia).[188–190]

Cigarette smoking is the leading risk factor for lung cancer: 80% of all lung cancer deaths in the United States are attributable to tobacco smoke exposure.[191] The risk for lung cancer increases with the level of daily cigarette consumption and the duration of cigarette smoking.[192] Despite a strong association between increasing cigarette exposure and increasing lung cancer risk, susceptibility to lung cancer varies greatly among smokers,[193] particularly among racial/ethnic, low SES, and LGBT groups,[194,195] and genetic factors have been shown to modulate the risk of developing lung cancer.[196–198] Although the biological impact of individual genetic variants is predicted to be similar, the frequency of risk factors varies across populations.[199] Furthermore, although heavier cigarette smoking is itself such a strong predictor of lung cancer risk, genetic risk factors are often more pronounced and detectable at lower levels of cigarette smoke exposure.[196,200]

This section introduces the general mechanisms by which genetic factors influence cancer risk among smokers, with a specific focus on lung cancer risk, and provides examples, where possible, of genetic factors and their importance by race/ethnicity. As of 2015, there was limited, if any, evaluation of genetic factors and lung cancer risk stratified by SES or LGBT status. The intersection of those environments affects health and health services use[201] and could obscure the detection and understanding of genetic factors.

Genetic factors modulate cancer risk among smokers via three general mechanisms (Figure 3.5). First, genetic factors influence cigarette smoke exposure levels by modifying smoking behaviors and smoking cessation outcomes, as discussed in the previous section. Second, genetic factors influence the body's response to carcinogenic compounds in cigarette smoke, such as the processing of carcinogens for removal, and the ability of carcinogens to interfere with endogenous growth-signaling pathways. The genetic influence on both of these factors, which act proximate to carcinogen exposure, will be the focus of this section. Lastly, genetic factors influence the body's underlying susceptibility to the damage caused by carcinogens. Gene variations that regulate DNA repair, the cell cycle, and apoptosis are associated with the risk of developing lung cancer—for example, the cell cycle genes *p53* and *p27*; the chromosome 6p21.33 region, which contains a DNA mismatch repair gene, *MSH5*; and an apoptosis and DNA damage response gene *BAT3*.[202–204] Such genetic factors are beyond the scope of this review, but have been discussed by others.[205,206] Genetic factors might also modulate cancer risk through more than one of the mechanisms described above. For example, genetic variations in *CYP2A6* and in *CHRNA5-CHRNA3-CHRNB4*, which will be discussed, modulate lung cancer risk both indirectly, by influencing smoking behaviors, and directly, by influencing the carcinogenicity of tobacco smoke compounds.

Figure 3.5 Genetic Factors Influence Cancer Risk by Modulating Smoking Behaviors, Activity of Carcinogens, and Susceptibility to Damage Caused by Carcinogens



Cigarette smoke delivers at least 69 carcinogens,[207] of which nitrosamines, polycyclic aromatic hydrocarbons (PAHs), aromatic amines, volatile organic chemicals, and heavy metals are among the most potent.[208–210] Many of the chemicals in cigarette smoke are procarcinogens—chemicals that exert their full carcinogenicity following metabolic activation. In general, chemicals that enter the body, including cigarette smoke carcinogens entering through the lungs, undergo sequential steps of biotransformation (metabolic processing). Initially, such enzymes as CYPs typically make chemical substrates more hydrophilic and more reactive by adding polar chemical groups. These more polar metabolites then become the substrates of classes of transferase enzymes, such as UDP-glucuronosyl transferases (UGTs) and glutathione S-transferases (GSTs), which facilitate elimination by conjugating large, bulky hydrophilic groups onto the metabolites. Alternatively, these transferases could also directly interact with procarcinogens. Thus, in general, CYPs metabolically activate procarcinogens into reactive species capable of damaging DNA, and genetic variations that result in reduced CYP activity are anticipated to reduce cancer risk. In contrast, transferase enzymes metabolically inactivate (i.e., detoxify) carcinogens, and genetic variations that result in reduced transferase activity are anticipated to increase cancer risk by allowing carcinogens to reside in the body for longer periods.[197] In addition to DNA damage, nitrosamines might also foster carcinogenesis by interfering with endogenous nicotinic receptor signaling, which is vital to managing proper cell growth,[211,212] and genetic variations in nicotinic receptor subunits are associated with lung cancer risk among smokers.[213]

Genetic factors associated with lung cancer risk could offer insight into disparities in the smoking-related risk for lung cancer among racial/ethnic populations. Differences in cigarette consumption cannot readily explain racial/ethnic susceptibility to lung cancer, as African American smokers, a typically light-smoking population, appear to be at a higher risk of lung cancer than European American and Japanese smokers, both of whom are generally heavier smoking populations.[2,187] The excess risk observed among African Americans may result from the interplay of genetic and environmental factors that need more detailed study.

Lung cancer is a heterogeneous disease defined by histological subtypes and increasingly by molecular signatures.[214] Lung cancers are broadly classified as small cell carcinomas and non-small-cell carcinomas; the latter includes two of the most prevalent histological subtypes of lung cancer—adenocarcinoma and squamous cell carcinoma.[215] Adenocarcinoma is the most prevalent histological subtype overall and within smokers (40% of all lung cancers in the United States), followed by squamous cell carcinoma (20% of lung cancers).[216] Smoking is a risk factor for all histological subtypes of lung cancer.[217] The heterogeneity of lung cancer can confound genetic investigations, as genetic risk factors are unlikely to be uniformly associated with all histological subtypes of lung cancer and could predominantly influence a single subtype. There is a trend in this direction. For example, in a GWAS of lung cancer involving almost 30,000 cases and 56,000 controls, certain susceptibility loci were specific to lung adenocarcinoma.[218]

In addition to smoking behavior, cigarette brand could affect lung cancer histology. The shift from a predominance of squamous cell carcinoma to adenocarcinoma over the past 50 years has coincided with changes in cigarette composition and design, resulting in greater relative exposure to tobacco-specific nitrosamines than PAHs and fostering deeper inhalation.[219] Thus, product preference and trends in use might further complicate the relationship between genetic factors and lung cancer and could possibly contribute to TRHD.

PAHs and nitrosamines are the procarcinogens most strongly associated with lung cancer.[208] Nitrosamines can also foster carcinogenesis by binding to nicotinic receptors, and evidence for the association of variations in nicotinic receptor signaling with lung cancer follows. The next section discusses (1) genetic variations in enzymes that metabolize PAHs and are associated with lung cancer risk, then (2) variations in enzymes that metabolize nitrosamines. Aromatic amines are also strong procarcinogens, but they are more strongly associated with bladder cancer than with lung cancer,[207] therefore genetic variations in metabolizing enzymes that predominantly interact with aromatic amines (such as N-acetyltransferases) are not discussed here.[220,221]

Genetic Factors Associated With Carcinogen Metabolism

Variations in genes involved with the metabolism of PAHs are associated with differences in lung cancer risk. PAHs such as benzo[a]pyrene are metabolically activated into epoxides and further into diol epoxides capable of reacting with DNA.[208] *CYP1A1* and myeloperoxidase (MPO) are among the enzymes capable of metabolically activating benzo[a]pyrene intermediates and have been studied widely as potential genetic risk factors in lung cancer.[196,222,223] Genetic variations in *CYP1A1* have been associated with lung cancer in several populations, including African Americans, Asians, Europeans, European Americans, and Indians.[224–228] They have also been associated with other respiratory cancers, such as oral and pharyngeal cancers.[229] Large-scale meta-analyses supported an association between the *CYP1A1* Msp1 variant and increased lung cancer risk in Asian and non-Asian populations, whereas the

CYP1A1 Ile[462]Val variant is predominantly associated with increased lung cancer risk in Asian populations, probably because of the lower prevalence of this variant in non-Asian populations.[230–232] Variations in *CYP1A1* appear to be predominantly associated with squamous cell carcinoma.[230,231] MPO genetic variants with decreased transcription are associated with a reduction in lung cancer risk in populations of European descent,[233] but not all studies have supported this association.[234] Variations in *CYP1B1* have also been investigated, albeit to a lesser extent, and study results have been mixed.[196,235–237]

Detoxification enzymes for PAHs include GSTs.[223] Genes coding for GSTs, such as *GSTM1*, *GSTT1*, and *GSTP1*, have been widely studied as genetic factors in cancer risk,[196] and many studies have found them to be associated with lung cancer and head and neck cancer risk in smokers,[238,239] but not all studies report an association.[240] Some investigations have confirmed the association of the *GSTM1* deletion allele with increased lung cancer risk in Asian populations.[226,241,242] A meta-analysis of the *GSTT1* deletion allele also reported a significant association between GSTT1 and lung cancer risk in Asians but not in Europeans, which was likely due to the lower frequency of the deletion allele in people of European descent, thereby reducing the power to detect the association.[243] The results were more equivocal for a reduced-activity *GSTP1* allele in a combined meta-analysis and pooled analysis in Asian and European populations.[244,245]

Variations in genes involved with the metabolism of nitrosamines are also associated with lung cancer risk. The drug-metabolizing enzymes *CYP2A6* and *CYP2A13* activate tobacco-specific nitrosamines such as 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and N'-nitrosonornicotine (NNN).[246] Cigarette smokers with *CYP2A6*-reduced metabolism genotypes are associated with a reduction in lung cancer risk compared with *CYP2A6*-normal metabolizers; this finding has been observed in Japanese, European, and European American populations.[134,135,198,247–249] In contrast, one study found that the loss-of-activity *CYP2A6*4* allele was associated with increased lung cancer in a Chinese population[250]; however, a subsequent larger Chinese study found no association between *CYP2A6*4* and lung cancer,[251] and a small study in a Thai population also failed to find an association between *CYP2A6*4* and nasopharyngeal cancer.[252] The relevance of these findings to smokers is unclear; the Chinese studies included a significant proportion of never-smokers, whereas the Thai study provided no details on the smoking status of participants.

Earlier studies among European populations also did not find an association between *CYP2A6* and the risk for developing lung cancer, but these studies assessed only one or two variants of low frequency in Europeans (~1%).[246] In terms of histology, Japanese studies have reported an association between variations in *CYP2A6* and squamous and small cell carcinomas, whereas in a European American population a stronger association with adenocarcinoma was noted.[134,135,247] Some researchers offer the caveat that genetic association studies in cancer are typically not powered to assess genetic risk within each lung cancer subtype, and they can be further confounded by differences in smoking level, which contribute unequally to the risk of each histological subtype of cancer.[253,254]

CYP2A6-reduced metabolism genotypes are also associated with lower cigarette use and lower nitrosamine exposure.[134,135,255] However, even after controlling for cigarette exposure, the association between *CYP2A6* variations and lung cancer risk remains significant, suggesting that genetic differences in the *CYP2A6*-mediated activation of nitrosamines contribute to differing lung cancer risk in addition to *CYP2A6*-mediated influences on smoking behavior.[134,135]

The involvement of *CYP2A6* metabolic activation in carcinogenesis is corroborated by human data showing that the inhibition of *CYP2A6* in cigarette smokers is associated with increased routing of NNK to the metabolite NNAL (i.e., evidence of the reduced activation of NNK),[256] and by mouse data demonstrating that the inhibition of the mouse version of *CYP2A6* reduces the occurrence of NNK-induced adenomas.[257] Variations in *CYP2A6* are also associated with other respiratory tract cancers—oral cancer in North Indians,[258] head and neck cancers in Sri Lankans,[259] and upper aerodigestive cancers in Europeans.[260]

Genetic variations in *CYP2A13* are less well characterized, but two different functional variants have been associated with altered lung cancer risk in Chinese and European populations.[251,261] Variations in another nitrosamine-activating gene, *CYP2E1*, have been extensively investigated as potential genetic risk factors for lung cancer, but results have largely been equivocal.[262]

Although *CYP2A6* and *CYP2A13* metabolically activate nitrosamines, UGTs typically detoxify the metabolites of nitrosamines and PAHs into noncarcinogenic glucuronide conjugates, and there is growing evidence that genetic variations in UGTs can influence the risk of lung cancer and other tobacco-related cancers. A *UGT2B17* deletion variant with a reduced ability to detoxify nitrosamines was associated with increased lung cancer risk in European American female smokers.[263] Reduced-activity variants in the *UGT1A7* gene were associated with an increased risk for lung cancer among Japanese,[264] increased orolaryngeal cancer among European Americans and African Americans,[265] and increased proximal digestive cancers (e.g., esophageal, orolaryngeal, and gastric cancers) among Europeans,[266] presumably because of the reduced ability of these gene variants to detoxify PAHs. However, increased activity *UGT1A7* variants were associated with an increased risk, as opposed to a decreased risk, of head and neck cancers,[267] underscoring the need to better characterize the functional and biological consequences of variations in UGT genes.

Variations in *UGT1A10*, another UGT involved with the detoxification of PAHs, are also associated with the risk for orolaryngeal cancer in African Americans but are unlikely to be detected as a risk factor for cancer in Europeans or Asians due to the lower prevalence of the variants in these other populations (less than 1%).[268] Genetic variations in UGTs could also be an important consideration in biomarker studies. The ratio of NNAL-glucuronide to NNAL (the main metabolites of NNK) is a proposed biomarker for nitrosamine detoxification[269] and for cancer risk[270]; thus, variations in UGTs could confound the interpretation of this biomarker.

Hereditary factors, in addition to diet and cigarette design, are hypothesized to contribute to lower lung cancer incidence in Japanese men compared with American men, despite a higher prevalence of smoking and heavier smoking.[271] Genetic variations that result in reduced metabolic activation of carcinogens could contribute to the lower lung cancer susceptibility. For example, the *CYP2A6* deletion allele is significantly more prevalent in Japanese compared with Europeans, European Americans, and African Americans,[272] and cigarette smokers homozygous for the deletion allele have a substantially lower risk of lung cancer compared with *CYP2A6*-normal metabolizers, with an odds ratio of 0.29 (95% confidence interval 0.15–0.56).[134] Therefore, the high prevalence of the *CYP2A6* deletion allele could contribute to the lower average lung cancer risk observed in Japanese smokers. However, genetic variants in *CYP1A1* and GSTs, which are associated with increased lung cancer risk, are also more prevalent in Asian versus non-Asian populations.[196,243] Thus, variations in metabolic genes associated with increased and decreased cancer risk are prevalent in Japanese populations, underscoring the need to assess the concurrent impact of variations in multiple genes.

In African Americans, genetic variations in the glucuronidation detoxification pathway of carcinogens could increase the risk of lung cancer, as a greater proportion of African Americans have a slow glucuronidation phenotype compared with European Americans.[45,273,274] On the other hand, a greater proportion of African Americans are *CYP2A6*-reduced metabolizers compared with populations of European descent,[64] which should confer protection from lung cancer. Thus, the impact of *CYP2A6*-reduced activity might be opposed by an increased risk conferred by impaired glucuronidation. Finally, among smokers *CYP2A6* faster-metabolizing genotypes increase smoking intensity, thus increasing their exposure to carcinogens. This is another mechanism by which *CYP2A6* may affect lung cancer risks.[275]

Nonmetabolic Genetic Risk Factors

In addition to metabolic genes, genes involved with nicotinic receptor signaling have also been associated with lung cancer risk. GWASs of lung cancer have been conducted. GWASs examine many single nucleotide polymorphisms (SNPs) across the genome of thousands of lung cancer cases and thousands of controls and are useful in identifying SNPs associated with lung cancer and help rule out chance findings. Lung cancer GWASs have found more than two dozen common loci associated with this cancer.[276]

Chromosome 15p25.1

GWASs have found a strong association between genetic variations in the chromosome 15q25.1 region and lung cancer.[277–279] The 15q25.1 region encompasses the nicotinic receptor subunit gene cluster *CHRNA5-CHRNA3-CHRNB4*. Variations in this cluster are associated with multiple histological subtypes of lung cancer, including adenocarcinoma, squamous cell carcinoma, and small cell carcinoma[204,280] and with an earlier age of cancer onset.[281] Genetic association studies of the 15q25.1 region initially were conducted in European and European American populations. The tag SNP (genetic variant) most highly associated with increased lung cancer risk within *CHRNA5-CHRNA3-CHRNB4* occurs at a frequency of 37% in European populations but only 10% in African Americans and ~1% in Asian populations.[282,283] A two-fold elevated risk is associated with being homozygous for the risk alleles at rs588765-rs16969968 compared to being wildtype at those SNPs.[284]

Subsequent association studies in Japanese, Chinese, and African American populations have also implicated the region in lung cancer risk.[211,282–286] Many of the SNPs in the *CHRNA5-CHRNA3-CHRNB4* gene cluster contribute to lung cancer susceptibility in African-Americans and Asian populations as well as European/American white populations. However, there are many other variants of potential importance in the *CHRNA5-CHRNA3-CHRNB4* region among African Americans and Asians.[276,285–288]

In addition to lung cancer risk, genetic variations in the 15q25.1 region are associated with a modest increase in cigarette consumption and an increased nitrosamine exposure,[20–22,103] potentially increasing the risk of lung cancer through increased carcinogen exposure.[101] However, cancer risk remains elevated after controlling for cigarette smoke exposure,[107,135,281] which suggests that genetically determined alterations in nicotinic receptor signaling might contribute directly to the risk of lung cancer as well as indirectly through increased cigarette consumption. The *CHRNA5-CHRNA3-CHRNB4* subunits are expressed in the lung and participate in nicotinic receptors with a high affinity for nitrosamines in addition to nicotine and the endogenous ligand acetylcholine.[211,289] Nitrosamines could foster the development and progression of cancer by binding to nicotinic receptors, thereby disrupting the balance

between inhibitory and stimulatory receptor signaling and resulting in increased cell proliferation, invasion, angiogenesis, and reduced apoptosis.[212]

GWASs found that variations in the chromosome 15q25.1 region, but not specifically the *CHRNA5-CHRNA3-CHRNB4* subunits, were implicated in lung cancer risk. Thus, other genes in this region, such as *IREB2* (iron-sensing response element) and *PSMA4* (proteasome α 4 subunit isoform 1), could contribute to lung cancer risk in addition to or instead of the nicotinic receptor subunit genes.[107,277–279,290]

Because the *CHRNA5-CHRNA3-CHRNB4* region has been associated with both tobacco smoking behaviors and lung cancer, investigators have examined whether the region influences smoking behaviors and is thus associated with lung cancer, or whether the region is associated with lung cancer independent of its effect on smoking behaviors. Evidence on this question among African Americans is inconsistent.[112,276,284]

Chromosome 5p15.33 and Other Loci

Another region on chromosome 5, specifically 5p15.33, is near the gene telomerase reverse transcriptase (TERT) and is associated with lung cancer.[291] Among African Americans, a variant located on 5p15.33, specifically at locus rs2853677, is associated with adenocarcinoma of the lung, but not other lung cancer types, with an odds ratio of 1.32 (95% CI 1.20–1.44),[276] confirming previous studies of this SNP and lung adenocarcinoma in African Americans.[288] Variants in *CHRNA5* (rs 2036527) are associated with lung cancer risk and smoking frequency in African Americans.[276,288] In African Americans there are other loci in the nicotinic cholinergic receptor genes and in others, such as TERT.[286,288,292] In studies of nonsmoking Asian women, associations were observed with multiple variants that are associated with longer telomere length and loci for lung cancer[293] or lung adenocarcinoma[294] that are different from those found in other populations. GWASs of lung cancer among Asians have identified a number of SNPs of importance to this population group.[293–297]

Other common genes associated with lung cancer based on GWASs include *PRPF6*[298] and *NEXN-AS1*.[299] In addition to common genes, rarer genes such as *BRCA2* and *CHEK2*, are also associated with lung cancer risk.[300]

Several other developments have provided further insights about the genetics of lung cancer. The GWAS approaches used to identify loci for specific cancers have been extended to examine whether some SNPs are associated with multiple cancers including lung cancer. For example, both lung cancer and breast cancer are associated with variants at 1q22.[301] Risk prediction models that include tobacco smoking behaviors as well as genetic factors are being developed to help identify subgroups of people who are at greatest risk of lung cancer. For example, lung cancer risk prediction models have been developed, but the inclusion of top genetic hits in the model have not improved its utility for African Americans.[302] And finally, research studies are exploring the functional implications of various risk alleles.[303–305]

Summary for Lung Cancer

Genetic factors, along with smoking behaviors and socioeconomic and environmental influences, contribute to population disparities in the risk for developing tobacco-related cancers. This section introduced the general mechanisms by which genetic factors influence cancer risk among smokers, discussed lung cancer risk specifically, and provided key examples of genetic risk variants.

Variations in the genes involved with carcinogen processing and carcinogen signaling pathways modulate lung cancer risk. Variations in the *CYP2A6* gene, which metabolically activates nitrosamines, are an important genetic risk factor for lung cancer, particularly in Asian populations in which the frequency of *CYP2A6*-slow metabolism is high.[198,246] The evidence for other drug-metabolizing enzymes, such as *CYP1A1* and GSTs, which metabolize PAHs, is more equivocal, possibly owing to fewer characterized functional variants.[198] The association between *CYP2A6* and cancer in European populations only became significant as more prevalent functional genetic variants were identified and evaluated. The association between lung cancer risk and variations in *CYP2A6* has two potential mechanisms—altered carcinogen exposure through smoking behaviors and altered metabolic activation of nitrosamines.

The nicotinic receptor subunit genes *CHRNA5-CHRNA3-CHRNB4* have also emerged as significant candidate genetic risk factors for lung cancer across multiple racial/ethnic populations. Recent GWASs have found loci in African Americans and nonsmoking Asian women that were previously identified in European populations; these studies have also found loci unique to those populations. Variations in these genes could influence lung cancer risk by modulating smoking behaviors by influencing the degree to which nitrosamines and/or nicotine interfere with endogenous signaling pathways. A number of other loci have emerged as important in lung cancer risk, but limited evidence about them in populations other than people of European origin is available. Some genetic loci are more important for adenocarcinoma than for other histologic forms of lung cancer. Until the specific cancer-causing genetic variants are determined and the frequencies of these variants (as opposed to tag SNPs) are assessed in each racial/ethnic population, it is difficult to determine whether variations in nicotinic receptor subunits could be contributing to population differences in lung cancer risk.

As more genetic variants are identified and characterized within different genes and among diverse populations and are investigated in the context of smoking behaviors, a clearer picture of disparities in the genetic risk for smoking-related cancers should emerge.

Genetics and TRHD: Current Knowledge and Future Directions

Genetic factors can contribute to tobacco use and its consequences by affecting:

- Use of tobacco including amounts used, tobacco dependence, age of onset, and reduced cessation success, and
- Risk of lung and other cancers.

Some major classes of genes that have been found through candidate gene and GWASs and affect use of tobacco are involved in:

- Nicotine metabolism, especially *CYP2A6*
- The dopaminergic system, especially relating to smoking initiation
- The nicotinic cholinergic system, especially the *CHRNA5-CHRNA3-CHRNB4* gene cluster, related to tobacco quantity and dependence as well as cessation.

Many of the earlier types of studies involved selecting genes thought to be involved in a pathway and studying that set (i.e., candidate gene approach). Technological advances enabled GWASs that compare loci (specifically SNPs) across hundreds of thousands of loci in groups of people with particular diseases

or characteristics and those without them to identify SNP alleles that differ in the compared groups. These studies need to be very large, involving thousands of people, to help rule out chance as a reason for differences, given the very large number of statistical comparisons being made. Many loci have been identified through this approach. However, most of the evidence has come from populations of European origin. The relatively few studies of African American populations and studies of Asian women who did not smoke find both similarities and differences in the genetic regions involved.

As of this writing (2017), the contribution of genetic factors to TRHD, specifically tobacco-related cancers, cannot be estimated precisely because of the insufficient evidence available on this subject particularly about non-European-origin populations. Progress has been made in identifying and characterizing specific genetic variants that influence stages along the tobacco use continuum as well as in estimating the risk of developing tobacco-related cancers, as detailed in this section, but the inventory of genetic risk factors is far from complete for different population groups.[306] In addition, these genetic studies have been undertaken primarily to determine disease etiology rather than to address TRHD[307]; therefore, these studies have typically been performed within a restricted range of sociodemographic groups, which has limited the ability to translate findings to TRHD. Also, genetic investigations have favored methodological approaches that circumvent the heterogeneity of smoking behaviors and cancer risk in order to be able to detect genetic signals—an approach that minimizes the complexity of interactions between genes and environmental factors that lead to disease as opposed to investigating these interactions.[307]

As of 2017, genetic factors do not readily explain TRHD because genetic variants account for a modest proportion of the heritable variance in smoking behaviors, and because of the scarcity of genetic studies in relevant populations such as racial/ethnic and LGBT groups. Large, replicated studies conducted primarily with populations of European origin have clearly established genetic susceptibility loci as involved in tobacco dependence, quantity of use, cessation, and lung cancer risk, both tobacco-related and not tobacco-related. These genetic factors have a modest effect on risk of tobacco behaviors and cancer risk, yet help elucidate the biological mechanisms underlying tobacco behaviors and lung cancer risk and may have important implications for stratifying groups for interventions, such as cessation treatments, that may be most effective for them.

Some large-scale studies of African American populations and studies of Asian women who did not smoke find both similarities and differences compared to populations of European origin in the genetic regions involved in both smoking behaviors and lung cancer risk. Similarities include the importance of loci in *CYP2A6* involved in nicotine metabolism and the nicotinic cholinergic system, especially the *CHRNA5-CHRNA3-CHRNB4* gene cluster. The latter is related to tobacco quantity and dependence as well as cessation and may also be involved in lung cancer risk independent of its role in tobacco use behaviors. However, African Americans and Asians each have other distinct loci, both in those and other genetic regions. These genetic factors are only part of a complex web of behavioral, biological, environmental, social, and other characteristics that contribute to TRHD.

The state of knowledge about the genetic risk for smoking behaviors and lung cancer continues to be characterized by significant gaps and a need for further research. To more fully understand TRHD, more research is needed, as described below.

More Large Studies in a Wider Range of Populations

Data on the numerous genetic regions and genes that have been investigated as potential risk factors in the etiology of lung cancer come primarily from Asian, European, and European American populations; additional genetic association studies are needed for minority populations.[308]

Many of the studies of African Americans have achieved the large sample sizes needed by pooling data from multiple studies. The associations observed in a number of studies of nonsmoking women in Asia, specifically China,[293,294] may or may not apply to women in the United States. It would be very difficult to obtain an adequate sample size of Asian Americans for GWASs. There are very few studies in Hispanic populations or in populations defined by sexual orientation. It is important to conduct studies in other ethnic groups because they account for the majority of the human population (Asians) and genetic variation (Africans). There are also few studies in these populations that have examined in detail smoking phenotypes such as age of initiation and cessation. And most studies that break ground in new areas, such as the functional implications of these variants, are done in populations of European origin first because the large number of study participants needed are easier to assemble, and studies in other populations come much later.

Some ways to help achieve the large sample sizes needed for assessing genetic risks in these relevant populations include fostering pooling data whenever possible. Although pooling projects have been successfully conducted and are critical to research progress, racial and ethnic minority individuals are still significantly under-represented in existing human population studies. Large-scale cohorts and case-control studies with good representations by race/ethnicity and SES are necessary to further our understanding of racial/ethnic disparities in the risk for lung cancer. One such cohort, described in the Southern Community Cohort Study, offers the potential to assess genetic risk factors among African Americans and European Americans of similar socioeconomic backgrounds while incorporating biomarkers of cigarette exposure and accounting for environmental factors such as menthol smoking.[309]

Furthermore, to understand underlying risk differences among populations and the specific role of genetic factors, analyses should address interacting and interrelated environmental factors, such as SES, education level, diet, smoking behaviors, and other carcinogen exposures.[310] Most importantly, a concrete understanding of the prevalence, amount, and intensity of smoking within each high-risk group (e.g., race/ethnicity, SES, sexual orientation) is necessary because smoking is the predominant risk factor for lung cancer; thus all other risk factors (genetic or environmental) need to be understood in the context of smoking. However, it will be challenging to obtain the large sample sizes needed for such studies.

By furthering our understanding of the biological basis of smoking behaviors and by disaggregating high-risk populations by interindividual risk, genetic factors will help guide novel treatments and help tailor intervention strategies. Although there is a small body of research that suggests that having certain genotypes may influence smoking cessation success with different cessation treatment approaches, this has not been examined within different population groups.

Most of the effects of individual gene variants on lung cancer risk found to date have been modest. However, it is possible that risk models incorporating genetic variations across multiple genes could capture more of the variance in lung cancer susceptibility than is captured by any of these factors acting alone. For example, a greater proportion of lung cancer risk could be accounted for by the combined effect of: (1) two metabolic-activating enzymes, *CYP1A1* and *MPO*; or (2) a metabolic-activating

enzyme, *CYP1A1*, and a metabolic-inactivating enzyme, *GSTM1*; or (3) a metabolic-activating enzyme, *CYP2A6*, and nicotinic receptors, *CHRNA5-CHRNA3-CHRNB4*.[135,240,311]

Lung Cancer Heterogeneity

The heterogeneity of lung cancer might also confound investigations. Lung cancer is a disease with multiple histological subtypes, each having a different relationship with smoking behaviors.[253] For example, adenocarcinoma is currently the most prevalent lung cancer type among smokers,[215] but adenocarcinoma shows a more modest association with cigarettes smoked per day and with years of smoking relative to other subtypes.[253] As such, the smoking patterns of a population under study and the proportion of a given histological lung cancer subtype could influence a researcher's ability to detect genetic and nongenetic associations, and future research on TRHD would benefit from separate studies of cancer subtypes or from correcting for cancer subtypes by using procedures analogous to those employed to correct for population stratification.[312] Fortunately, investigators are increasingly publishing genetic data associations for specific lung cancer histologic types and finding that some susceptibility loci are specific for certain histologic types.[218]

Although lung cancer has been the focus of this chapter, a better understanding of genetic contributions to other cancers would be valuable. Studies on never-smokers could also shed light on understanding disparities in racial/ethnic, sex, and LGBT groups.

Gene–Environment Interactions

The importance of environmental factors is highlighted by migration studies, which find that the cancer risks of generations born after the original immigration resemble the risks found in the adopted country rather than the ancestral land.[310]

In addition to combined gene risk models, further progress in understanding the genetic factors associated with cancer risk requires a diligent assessment of gene–environment interactions. Such studies are challenging to conduct because they require large sample sizes. The degree of interaction between variations in metabolic genes and cigarette smoke exposure typically varies with the level of exposure.[200] For instance, at lower versus higher levels of cigarette exposure, variations in genes such as *CYP2A6*, *CYP2A13*, *CYP1A1*, *GSTM1*, and *MPO* are more prominently associated with lung cancer risk.[135,224,225,251,313,314] In contrast, the association of variations in the *CHRNA5-CHRNA3-CHRNB4* subunit genes with lung cancer changes little with the level of cigarette smoke exposure.[107,135] Future genetic association studies employing biomarkers of smoking dose and quantitative records of historical cigarette exposure will help characterize gene–environment interactions and their relationship to lung cancer risk.[197] In addition to smoking dose, environmental factors that are unique to high-risk groups such as LGBT and lower socioeconomic groups must be investigated.

In the future, as more genetic association studies employ analytical approaches that incorporate and evaluate gene–environment interactions, population heterogeneity, biomarkers, and more precise behavioral measures, more of the genetic variance in smoking behaviors will be explained.[315] Multigene models that assess the combined effect of multiple genetic variants on smoking characteristics may be more informative than studying genetic factors one at a time.

Biomarkers

Biomarkers of smoking dose could offer insight into the paradox of worse health outcomes among African American smokers despite their greater proportion of light smoking. Differences in lung cancer risk between African Americans, Native Hawaiians, and other racial/ethnic groups are greatest at lower levels of cigarette consumption (e.g., no more than 10 cigarettes per day).[2] However, there is emerging evidence that African Americans smoke more intensively at lower levels of daily cigarette consumption than other racial/ethnic groups.[96,316] The higher rates of lung cancer could be explained by greater carcinogen exposure despite consuming fewer cigarettes daily, coupled with the fact that a greater proportion of African Americans have slower carcinogen detoxification capabilities.

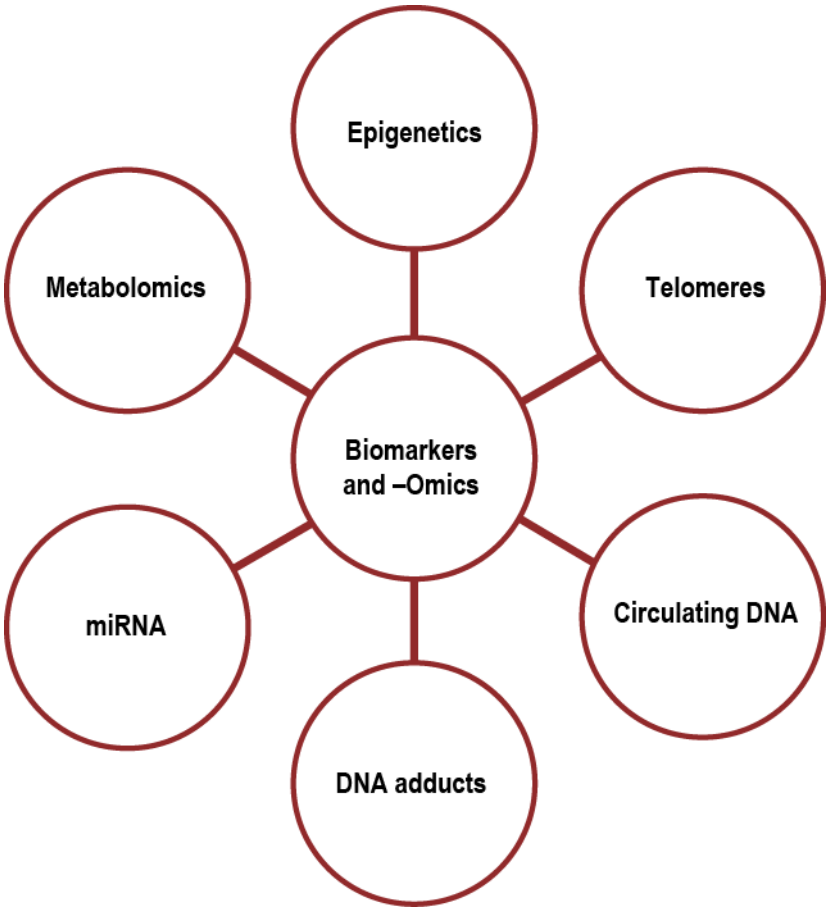
In addition to amassing genetic data on the relevant populations of interest, it is critical to incorporate better biomarkers and surrogates for cigarette smoke exposure. As smoking is the predominant risk factor for lung cancer, smoking biomarkers must be properly validated among different groups of interest and incorporated and/or controlled for to investigate the contribution of other risk factors, genetic or nongenetic, to TRHD. For example, African American smokers would be predicted to have lower cigarette exposure compared with European American smokers based on self-reported number of cigarettes smoked; however, using such biomarkers as total nicotine equivalents, it becomes apparent that African American smokers can achieve comparable levels of cigarette exposure despite smoking fewer cigarettes per day.[96,316] The fact that this amount of cigarette exposure can be achieved despite smoking fewer cigarettes suggests differences in smoking topography. Topography could also interact with genetic factors to influence disease risk, given that the depth of inhalation, an aspect of topography, is believed to contribute to disease risk.[317] Thus, without properly accounting for cigarette exposure, relevant genetic and nongenetic factors could be obscured by differing self-reported smoking behaviors among populations.

Expanded Genetic Characterization and Other High-Throughput Characterization Approaches ("-Omics")

Cigarette smoke exposure is causally associated with numerous cancers and is the leading risk factor for lung cancer.[188–191] Cigarette smoke contains more than 69 carcinogens, among which PAHs and nitrosamines have the strongest causal association with lung carcinogenesis.[207] Variations in the genes that influence smoking behaviors might indirectly influence lung cancer risk by altering carcinogen exposure (i.e., tobacco intake). Variations in genes such as DNA repair genes can also increase individuals' underlying susceptibility to the damage caused by carcinogens.

Increasingly, more sophisticated data is being generated: exome sequencing, gene expression, epigenetic data from shared online databases and consortia. It will be important to study the role of epigenetic changes and gene expression in addition to the genetic changes. Also, functional studies are helping unravel the basis for the effect of the genes involved. It will be very important that these studies are performed in multiple population groups to better understand TRHD. Epigenetics is another promising research avenue to better understand the environmental component of gene–environmental interactions influencing TRHD as both smoking and social environmental stressors appear to influence epigenetic patterns.[318–320] These and many other examples of -omics and biomarkers approaches are shown in Figure 3.6.

Figure 3.6 Types of Biomarkers and -Omics Technologies That Could Help Understanding of TRHD



Complex Interrelationships

It is important to understand the impact of all other environmental and host factors of smoking. Sexual orientation is important and understudied. Poverty and many associated exposures (diet, obesity, work/home environment, geographic location), pollution, health care access, education, and many other factors contribute as well. Comorbidities, such as chronic obstructive pulmonary disease and immune-related conditions such as HIV, and family history of cancers and other conditions are also important.

The challenges in applying genetic findings from one population to another, even when dealing with a causative variant as opposed to a tag SNP, are numerous. There is also the issue of the potential contribution of correlated and as yet poorly defined risk factors. For example, race/ethnicity, which genetic researchers typically use to represent geographic/ancestral origin and genomic variability, also encompasses correlated environmental, economic, and sociocultural factors that can influence disease risk.[321,322] Likewise, the effect of a gene variant on a particular outcome (e.g., smoking persistence) is likely influenced by multiple interacting genetic and environmental components that could differ across populations.[15] Similarly, other populations experiencing TRHD, such as lower SES groups and LGBT groups, may experience environmental risk factors that could interact with genetic risk factors. Thus, without a better understanding of genetic and nongenetic risk factors and their potential interactions in the manifestation of tobacco-related diseases, it is not straightforward to extrapolate genetic findings

from one population to another, even when the impact of a genetic variant on the function of a gene is known.

As our knowledge of genetic and nongenetic factors and their interactions with each other and with smoking behaviors increases, a clearer picture of TRHD will emerge. In addition, as key genetic and nongenetic risk factors become uncoupled from race/ethnicity, SES, and LGBT status, it should become feasible to predict whether a particular individual will suffer disproportionately from TRHD without having to rely on these demographic categories.

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Section II
Intrapersonal/Individual Factors Associated With Tobacco-Related Health Disparities

Chapter 4
Flavored Tobacco and Chemosensory Processes

Contents

Figures and Tables

Introduction

Flavor additives such as menthol, ginger, vanilla, nutmeg, licorice, cocoa, and sugars are examples of ingredients that are added to cigarettes.[1,2] This chapter focuses on the chemosensory effects of flavors in cigarettes and, in particular, on menthol. The most common characterizing flavor in cigarettes, menthol has been added to cigarettes since the 1920s.[3] Menthol is the primary focus of this chapter because when used in cigarettes as a characterizing flavor, the compound affects multiple chemical senses, including the olfactory (smell), gustatory (taste), and trigeminal (burning, tingling, touch, temperature, nociception) senses.[4–6]

Three months after the date of its enactment, the Family Smoking Prevention and Tobacco Control Act of 2009 (Tobacco Control Act) banned characterizing flavors, other than menthol and tobacco, in cigarettes.[7] The Tobacco Control Act also required that within 1 year after its establishment, the U.S. Food and Drug Administration (FDA) Tobacco Products Scientific Advisory Committee (TPSAC) submit a report and recommendations on menthol in cigarettes and public health, including use among children, African Americans, Hispanics, and other racial and ethnic minorities. In its report *Menthol Cigarettes and Public Health: Review of the Scientific Evidence and Recommendations*, the FDA TPSAC concluded that "the availability of menthol cigarettes has led to an increase in the number of smokers and that this increase does have adverse public health impact in the United States."[8,p.220] (Other provisions of the Tobacco Control Act and their relationship to tobacco-related health disparities [TRHD] are discussed in chapter 11.)

Background

"Flavored" tobacco was made popular with the inadvertent invention of menthol cigarettes in 1924 by Lloyd F. (Spud) Hughes, a resident of Mingo Junction, Ohio. Hughes used menthol for medicinal purposes, inhaling the menthol crystals to treat his asthma. After hiding his cigarettes in a tin can that contained menthol crystals and baking powder, Hughes discovered that the menthol cigarette flavor created a cooling and soothing effect.[9] In 1924, he filed for a U.S. patent that specified the treatment of cigarettes with menthol, alcohol, and cassia oil derived from the *Cinnamomum cassia* tree. In his patent application, Hughes stated:

> This invention relates to a process of treating tobacco for use in the production of cigarettes, and it has for its object to provide a cigarette tobacco which, while cooling and soothing to irritated membranes of the mouth and throat of the smoker, is absolutely non-injurious and is pleasant to taste. The process consists in spraying upon the tobacco which is to be rolled into cigarettes a solution consisting of menthol ($C_{10}H_{20}O$), cassia oil, and alcohol.[3]

The patent was granted on September 29, 1925, and production of the new product began soon after. Hughes formed the Spud Cigarette Corporation in Wheeling, West Virginia, and Spud cigarettes were manufactured for Hughes's corporation by Bloch Brothers Tobacco Company (Figure 4.1). Hughes sold his cigarettes door to door, out of his car, and to railroad and mill workers who frequented his father's restaurant.[10] In 1926, Hughes sold his patent to the Axton Fisher Tobacco Company of Louisville, Kentucky, for $90,000. Spud was the fifth largest selling tobacco company in the United States until Brown and Williamson introduced two cheaper menthol cigarettes, Penguin in 1931 and Kool in 1933[11] (see Figure 4.1).

Figure 4.1 Cigarette Packs: Spud Menthol Cooled Cigarettes, 1924, and Kool Cigarettes, 1950

Sources: Trinkets & Trash.[186,187]

The pleasing mint flavor and cooling sensation of menthol in tobacco were used to market menthol cigarettes as "healthy," and they increased in popularity in the 1950s.[12] In 1956 R.J. Reynolds Tobacco Company (RJR) introduced Salem, the first filter-tipped menthol cigarette. RJR sold the Kool and Salem brands to Imperial Tobacco Company in 2015.[11] In 1957, Lorillard Tobacco introduced the Newport menthol brand, which Reynolds America, RJR's parent company, purchased in 2015.[11] According to 2016 sales data, Newport is the second most popular cigarette brand in the United States, having 13% of the market share. The domestic share of menthol cigarettes increased from 16% in 1963 to 30% in 2014.[13,14]

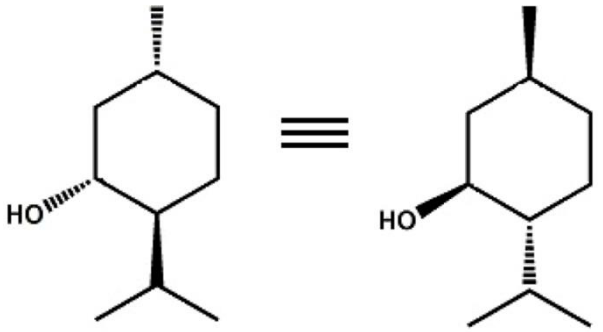
As described in chapter 2, menthol cigarettes are disproportionately smoked by youth, women, and African Americans. For example, the prevalence of menthol cigarette use in the past 30 days among black adolescent smokers is 95%.[15] Some populations groups, such as African Americans and Native Hawaiians and Pacific Islanders, have higher rates of tobacco-caused morbidity and mortality than others, and it has been suggested that menthol in cigarettes may play a role in the chronic disease pathway.[16–20]

The effects of menthol as a characterizing flavor can be immediately perceived by the consumer, whether the product is inhaled, chewed, smoked, or comes in contact with the skin. Other additives and constituents, such as cocoa and licorice, which are common additives in menthol cigarettes and other tobacco products, also act on the chemical senses.

The Menthol Compound

Menthol is a complex compound ($C_{10}H_{20}O$, molecular weight 156.27 g/mol) that has multiple biological effects on the human body. The chemical structure of menthol is shown in Figure 4.2. Menthol is a white or colorless crystalline substance that is solid at room temperature, partly soluble in water, and freely soluble in alcohol, diethyl ether, or chloroform.[21,22] This cyclic monoterpene alcohol has three asymmetric carbon atoms[23,24] and is present as four pairs of optical isomers: (+) and (–) menthol; (+) and (–) neomenthol; (+) and (–) isomenthol; and (+) and (–) neoisomenthol.[22–24] The menthol isomer (–) menthol (L-menthol), the isomer most widely found in nature,[23] is known for its flavor and cooling properties.[22]

Figure 4.2 Chemical Structure of Menthol



Menthol is found naturally in peppermint (*Mentha piperita*)[25] and cornmint plant oils (*Mentha arvensi*).[23] Menthol constitutes 50% of peppermint oil, and it can be extracted or synthesized from other essential oils like citronella, eucalyptus, and Indian turpentine oil.[23]

Menthol has been added to food and used in cosmetics and pharmaceutical products. Mint teas and peppermint candy and gum are widely used around the world. Menthol is commonly used in hygiene products such as toothpaste, mouthwash,[23,26–28] shampoo, and soap.[29,30] Menthol has been used as a local analgesic and an anesthetic, and for its antibacterial, antifungal,[22] and antipruritic properties. As an analgesic, menthol is an ingredient in topical rubs. Products that involve inhaling menthol are used to reduce respiratory discomfort due to colds and flu, because they inhibit airway irritation that leads to coughing.[31] Cough drops containing menthol are often used as an anesthetic to soothe throat irritation. Menthol inhibits the growth of bacterial strains[32–34] such as *Streptococcus* pneumonia.[35] It also has synergistic effects with antibiotics such as oxacillin and erythromycin.[35] As an antifungal agent, menthol compounds such as peppermint oil[36] have been known to be effective against *Candida albicans.*[37]

Tobacco industry documents suggest that menthol is the primary additive that creates multiple sensory effects.[4,5] Menthol is the only flavor additive that, when added at different concentrations, is known to act on the olfactory, gustatory, and trigeminal systems[30,38–41] to produce "desired" sensory effects for different types of smokers. Unlike strawberry, grape, or cherry characterizing flavors, menthol when used in cigarettes produces sensory effects that go beyond taste, flavor, and aroma; certain concentrations of menthol create cooling/tingling, analgesic, and smoothing effects. These sensory effects may serve as positive reinforcement for behavioral abuse of nicotine[6,42,43] and may affect the abuse liability of menthol.[44] As the World Health Organization Study Group on Tobacco Product Regulation has stated, "menthol is not only a flavouring agent but also has drug-like characteristics that modulate the effects of nicotine and tobacco smoke."[45,p.30]

Brief Review of the Chemical Senses

Physiology and psychology meet in the study of the chemical senses.[46] To understand how menthol's use in cigarettes influences experimentation, current use, and nicotine dependence, it is important to understand the complexities of the chemical senses and menthol's effects on them. Much is known and much is still to be learned about how the chemical senses operate, interact, and signal each other to produce unique flavor sensations and experiences among smokers.[47]

The perception of chemical stimuli by sensory means is called chemosensation or chemoreception.[48] Flavor results from the complex interaction of the chemical senses[49] and will not be discussed in detail in this chapter. The primary chemical senses for distinguishing flavors include the olfactory and gustatory systems.[50] The trigeminal somatosensory system (cooling and pain) also plays a role in chemosensation and how flavor is experienced.[48] No compound activates only one sensory channel,[51] and a single compound may not have the same smell, taste, and cooling or pain thresholds either in different individuals or on each of the independent sensory channels in the human trigeminal system.[52,53]

Olfaction allows us to detect odors such as the minty smell of menthol cigarettes. Odors stimulate a series of biochemical activities within the cell when the odor molecule binds to an odor receptor in the ciliary membrane.[54] Olfaction is not, strictly speaking, an oral sense; however, olfactory sensations that arise from odorants in the mouth are perceptually localized to the oral cavity. Much of the sensation of taste is olfactory.[55] Olfactory receptors facilitate a sequence of events that lead to flavor sensation, perception, and cognition.[49]

Gustation, or taste, is another well-known chemical sense. When chemical stimuli come in contact with taste cells embedded in the taste buds in fungiform papillae on the surface of the tongue, taste is detected, and it is experienced in different ways. There are five basic classes of taste: salty, sour, sweet, bitter, and umami.[50] For example, compounds such as sugar may stimulate multiple receptors that translate into a sweet taste.[50] Bitter taste is evoked by more receptors than sweetness, and some of the bitter receptors have been identified, such as TAS2R.[56,57] Research suggests that bitter taste prevents mammals from ingesting potentially harmful food constituents.[58,59] Sensations arising from the oral and nasal cavities vary considerably; some of this variation is attributable to genetics, and some to common pathologies. This variation in oral sensations plays an important role in health by affecting dietary choices, drinking alcohol, and smoking cigarettes.

The capacity of the trigeminal nerve to detect chemicals is called chemesthesis.[48] The sensory properties evoked by smoking result from stimulation of the cranial nerves that innervate the oral and nasal cavities. Sensations from the tongue include taste and somatosensation (irritation/pain, temperature, touch). Taste is mediated by the chorda tympani nerve (CN VII) on the anterior tongue and the glossopharyngeal nerve (CN IX) on the posterior tongue. Somatosensation is mediated by the trigeminal nerve (CN V) on the anterior tongue and is mediated along with taste by the glossopharyngeal nerve on the posterior tongue. The endings of the trigeminal nerve can also be activated by physical stimuli and chemical agents[60] and can evoke sensations of touch, temperature, and pain[48] even in the absence of olfactory perceptions.[61] The trigeminal system produces protective responses through salivation, tearing, coughing, respiratory depression, and sneezing.[48] The trigeminal system is the least understood of the chemical senses, but this system is known to play an important role in the consumption of food and other substances.

Cigarette Smoking and the Chemical Senses

Cigarette smoking impairs the senses of smell and taste. Studies have shown that, compared to nonsmokers, smokers have less ability to identify the presence of a taste (i.e., low odor threshold), to identify a particular taste, and to discriminate between tastes.[62,63] Number of pack-years (number of packs smoked per day multiplied by number of years smoking occurred), a measure of cigarette dose, is inversely associated with odor thresholds, discrimination, and identification.[62]

The mechanisms by which smoking influences olfaction are under investigation, but several studies suggest that smoking damages the nasal epithelium and increases cell apoptosis, thus causing nasal congestion.[64] Some studies have found that smoking impairs olfaction,[62] but other data suggest that olfaction returns to normal in smokers who quit.[63] Some researchers have found that smokers are less likely than nonsmokers to perceive bitter taste.[65] Few studies, however, have examined the relationship between olfaction and smoking, particularly as it relates to menthol cigarette smoking. Little research has examined how menthol cigarettes' effects on olfaction differ from the effects of non-menthol cigarettes or how this might affect the likelihood of smoking initiation and continuation.

Characteristics of Flavor Additives and Constituents

Cigarette smoke is irritating,[66,67] and nicotine has a bitter flavor.[68] The chemosensory effects of menthol make menthol cigarettes easier to smoke and may contribute to continued smoking. Analysis of tobacco industry documents shows that the industry has conducted research to understand consumers' perception of menthol cigarettes for many decades.[69]

There are over 7,000 chemicals in cigarette smoke.[70] Flavor additives and constituents of tobacco products can act on the chemical senses to create specific expectations of the product, entice new users, neutralize the negative experiences of nicotine and tobacco, and create positive experiences that make it easier for current users to continue to use a product that causes chronic disease and death. The number of flavor additives and constituents in tobacco that stimulate the chemical senses is unknown. A 1994 report from the six major American cigarette companies listed 599 ingredients used in cigarettes; many of these—including vanillin, valerian root extract, rosemary oil, raisin juice concentrate, honey, cocoa, coriander, basil oil, almond bitter, licorice, and ginger—appear to be used as flavor additives.[2] Few studies have investigated how these and other known flavor ingredients affect the chemical senses and impact TRHD. The following sections describe the use of cocoa, licorice, and menthol as additives to cigarettes and other tobacco products.

Cocoa as an Additive

Derivatives of cocoa beans have been used for different purposes throughout history, and research is still being conducted on their pharmacological and phytochemical properties.[71] Records from the 1500s show that cocoa beans, derived from the *Theobroma cacao* tree, were used as a medicine by Maya and Aztec civilizations of South America to treat gastrointestinal, cardiovascular, and nervous system ailments.[71] Twentieth-century studies have suggested that cocoa has pharmaceutical value as a flavor to improve the taste and facilitate delivery of medicines.[71]

Cocoa powder, cocoa butter, and cocoa liquor, derived from cocoa beans, have been used as both characterizing and non-characterizing flavors in cigarettes since as early as 1932,[42,72] and analysis of tobacco industry documents shows that the industry has "experimented with manipulating cocoa levels as a means of achieving sensory properties that appeal to women and youth."[42,p.984] These products can contain protein, amino acids, polyhydroxy phenols, starch, sugars, theobromine, caffeine, or fatty acid triglycerides when processed.[73] Cocoa enhances the taste and reduces the harshness of cigarettes when burned. Cocoa and cocoa extract are often used in the cigarette casing[74] to enhance the aroma and flavor of cigarettes and improve the overall smoking quality of blended cigarettes, but used in this way, cocoa is not detected as cocoa flavor by the smoker.[75] Tobacco industry documents state that cigarette companies have found cocoa useful because cocoa butter in tobacco products creates a smoother, enhanced tobacco flavor.[75]

Like menthol, cocoa derivatives are added to tobacco during cigarette manufacturing,[76,77] and industry documents suggest that levels typically do not exceed 0.5% (5,000 ppm total weight of tobacco) for cocoa and 0.1% (1,000 ppm) for cocoa extract.[77] Cocoa is used as a characterizing flavor in little cigars or chocolate-flavored electronic cigarette juice/liquid, which are advertised and marketed as flavored products.

Other than enhancing the taste of tobacco, it is not clear that cocoa as a characterizing or non-characterizing flavor in cigarettes has other sensory or pharmacological effects. A few in vivo and in vitro studies suggest that *Theobroma cacao* bean extract, known for its polyphenols, can suppress trigeminal nerve activity[78] and reduce inflammatory responses that cause pain,[78–80] but it is unclear what the effects of cocoa are on the trigeminal nerve system when cocoa is added to cigarettes. One study suggests that cigarettes do not contain enough theobromines, the primary bitter-tasting compound in cocoa, to have an effect on trigeminal nerve activity[81]; evidence from tobacco industry documents supports this as well.[42]

Licorice as an Additive

Although not a common characterizing flavor, licorice as a flavor additive has been used since the late 1800s in pipe tobacco and snuff.[82] The licorice plant is used for medicinal purposes, and licorice extract is also used as a sweet flavorant. Most of the sweet flavor comes from glycyrrhizin, which is found in the plant's root. A single company manufactures 70% of all licorice in the world, and almost 63% of its sales are to the tobacco industry.[83]

Unlike menthol, licorice is a non-volatile material added to cigarettes both as a flavorant and casing material.[84] Available in block, powder, and liquid forms, licorice has various effects when used in cigarettes. It is thought to enhance the smoke flavor, reduce dryness in the mouth and throat, reduce irritation, improve the absorption of flavors uniformly in tobacco, and minimize rough smoke by balancing the overall flavor of tobacco smoke.[84]

Licorice has been investigated for its potential health effects, such as its anti-inflammatory and immunoregulatory effects,[85] but it is also thought to raise blood pressure and induce hypertension.[86] Little is known of the health effects of licorice as an additive to cigarettes or how the amounts of licorice in sub-brands differentially influence the three chemical senses.

Menthol as an Additive

Research on menthol's effects on the olfactory, gustatory, and trigeminal chemical senses is more developed than research on the effects of other flavors. This research continues to clarify the role of menthol as a characterizing flavor in cigarettes and its multiple effects upon sensory processes. Enactment of the Tobacco Control Act in 2009 stimulated renewed interest in how this flavor additive may influence the harm of tobacco products.

As explained earlier in this chapter, menthol has been used in its natural and synthetic forms[87] in cigarettes since 1924. Menthol can be added by spraying it on tobacco during blending, applying menthol to the foil or filter,[88–90] injecting it into the tobacco stream in the cigarette maker, placing a menthol thread into the filter, inserting it into a crushable capsule (e.g., Camel Crush), or by a combination of these methods.[8] Regardless of the application process used, the volatility of menthol

ensures that it diffuses through the cigarette, creating flavors and sensations that appeal to some smokers.

Manufacturers add menthol to an estimated 90% of cigarettes sold in the United States.[91] A study of 45 U.S. cigarette brands found menthol content varied widely; as expected, the menthol content of brands labelled as "menthol" (2.9–19.6 mg menthol/cigarette) was far higher than that of brands not labelled as menthol (0.002–0.07 mg/cigarettes).[92] Menthol, interacting with other compounds in tobacco smoke, can produce a variety of physiological effects. Nicotine and tobacco are bitter, irritating, and harsh, causing sensations of burning or pungency, which may signal the user to refrain from using the product.[66] Menthol and nicotine activate the olfactory, gustatory, and trigeminal systems, and menthol can greatly alter the sensory properties of tobacco smoke.

In their review of published research analyzing the tobacco industry documents, Kreslake and Yerger conclude that "the tobacco industry has conducted extensive research on the chemosensory and physiological effects of menthol in tobacco smoke and has actively promoted menthol's sensory characteristics,"[93,p.S98] and "the industry has established internally that menthol's effects extend far beyond its use as a characterizing flavor, and have used it to ease inhalation and reduce irritation from smoking."[93,p.S98] They note that previous studies of internal tobacco industry documents have described tobacco industry research on a variety of menthol's properties including stimulation of nociceptors and cold receptors in the trigeminal nerve and stimulation of olfactory and gustatory receptors. The researchers also find evidence that menthol is added to cigarettes in concentrations to achieve "desired" effects and to appeal to smokers with different chemosensory perceptions. The properties of menthol have also been studied by other authors. For example, menthol has been shown to reduce irritation and sensitivity to nicotine.[94] Its analgesic and anesthetic effects reduce irritation from nicotine on the tongue[95] to make it easier to smoke. A study found that applying menthol to the side of the tongue of study participants significantly diminished the irritation from nicotine, compared with the non-treated side.[94] Menthol flavor additives may also influence the self-administration of nicotine.[96,97]

Four possible mechanisms by which menthol may alter tobacco smoking are highlighted in a review by Wickham: (1) menthol may reduce the initially aversive experiences of tobacco smoking; (2) menthol may serve as a highly reinforcing sensory cue when associated with nicotine and thus may promote smoking behavior; (3) menthol's actions on nicotinic acetylcholine receptors may alter the reinforcing value of nicotine; and (4) menthol may alter nicotine metabolism and increase nicotine bioavailability.[12] Regarding chemical sensation, the review states,

> Recent publicly available data from tobacco company records strongly suggested the reason for including menthol as an additive was to minimize the aversive experiences associated with tobacco smoking and, thus, decrease smoking's perceived health risk. These documents revealed that smokers of mentholated cigarettes report using them because they have less harsh, less irritating, and more soothing sensory profiles. Moreover, the flavor profile of mentholated cigarettes [was] reported to be improved compared to non-mentholated cigarettes, likely due to the appetitive minty flavor of menthol as well as its ability to mask aversive flavors of tobacco.[12,p.280]

How Menthol Produces Chemical Sensations

Menthol reduces the negative sensations of the smoking experience through its interaction with the chemical senses. When it is added to the cigarette and sprayed on the foil and package of cigarettes,[88–90] menthol likely acts on the olfactory system before, during, and after combustion. Odorants like menthol can reach the olfactory cleft from the mouth to the nasal cavity,[50] and even low concentrations of menthol, just above detection level, can activate the olfactory receptors, which results in odor sensation.[38,52,53] Medium concentrations evoke both the smell and the cooling sensation.[41,52,53] Because menthol itself is bitter, higher concentrations can result in the sensation of pain in addition to the smell and cooling sensation.[52] Menthol may independently affect each of the senses of smell, cooling, and pain.[53]

Menthol produces these various sensations by acting on transient receptor potential (TRP) ion channels. The ions TRPM8, TRPV1, and TRPA1 are primarily expressed in the neurons of the trigeminal and dorsal root ganglia.[98] TRPM8 is associated with cooling and easing of pain sensations. Menthol also stimulates heat-activated TRPV3,[30,99] which is mainly expressed in keratinocytes (skin cells)[98] and also has thermal and nociceptive properties, activating TRPV1.[30] At 16 ppm, which is less than the amount in menthol cigarettes, menthol can activate TRP receptors and halt irritant responses via TRPA1 and TRPV1.[31]

The menthol isomer (–) menthol (L-menthol) is known for its flavor and cooling properties.[22] Whether at low or high concentrations, menthol produces a cooling sensation when it is applied topically, ingested, inhaled, or chewed,[100] and this cooling sensation alters smokers' sensory perceptions. The cooling and refreshing effects are experienced when the concentration of menthol is high enough to activate TRPM8 ion channels[101–103] and when menthol is inhaled. Menthol increases intracellular calcium influx through the channels. One study showed that the cooling effects can last up to 70 minutes in about 65% of study participants.[100] The cooling effect is not a result of lowering of body temperature; studies have not shown that menthol causes any change increase in body temperature.[104]

The cooling sensation of menthol distracts from the pain of nicotine and blocks pain by inhibiting TRPA1.[105] It also reduces irritation and sensitivity to nicotine,[106] an irritant known to act on TRPA1 receptors as menthol does,[107,108] and reduces sensitivity to tobacco smoke.[107–109] If, by stimulating cold receptors, menthol results in the smoker holding his or her breath for extended periods, exposure to nicotine and the particulate matter of cigarette smoke would be increased.

Menthol's analgesic effects are a result of TRP activity as well. L-menthol can induce analgesia via TRPM8.[110] Because menthol cigarette brands vary in their analgesic effects, it is important to understand the levels of menthol used in particular tobacco products. It has been suggested that menthol's analgesic properties may mask early respiratory problems caused by smoking cigarettes.[18,19] The cooling effect plus the analgesic properties of mentholated cigarettes may give the smoker a false sense of well-being and reduce the likelihood of seeking medical attention for respiratory distress.[18]

Menthol's induction of various sensations depends not only on the concentration of menthol, but also on the part of the body to which it is applied.[111–113] Although at high concentrations menthol itself is an irritant, studies show that menthol reduces irritation from nicotine when applied to the tongue,[94] and menthol desensitizes the oral cavity to irritation.[112] Menthol may be a more effective stimulus to the mouth than it is to skin.[111]

Menthol may increase the bioavailability of nicotine.[12] Menthol has been shown to inhibit the metabolism of nicotine[114] and may also increase nicotine absorption.[115] If menthol's cooling effects facilitate smoke inhalation[31] or its smell reinforces smoking, these sensory effects could help explain higher levels of nicotine dependence and smoking maintenance among smokers of menthol cigarettes. Modern psychophysical tools now permit accurate assessment of sensory variability and thus have made it possible to link such sensory variation with specific health risks such as risk for smoking. The next section describes what is known about sensory variability and its importance to TRHD.

Chemical Senses and Variation

Variations in taste physiology, particularly in relation to gender and race/ethnicity, have been the subject of research on preference for menthol cigarettes. One source of this variation in taste makeup is the ability to taste the bitterness of 6-*n*-propylthiouracil (PROP) or phenylthiocarbamide (PTC).

Genetic variation in taste was discovered in the 1930s thanks to an accident in the laboratory of Arthur Fox at DuPont. Fox was synthesizing PTC when some of it blew into the air. A colleague nearby noted a bitter taste, which Fox did not perceive. A test revealed other "tasters" who could perceive the bitter taste of PTC (and other chemically related compounds like PROP, a less toxic bitter compound) and "nontasters" who could not.[116] A test of attendees at a meeting of the American Association for the Advancement of Science found that 28% of the 2,550 individuals tested were nontasters.[117] Snyder[118] tested families and concluded that nontasting was due to a single recessive gene. In the 1960s, Fischer and colleagues began to relate this genetic variation to health issues (e.g., nontasters were more likely to be smokers).[119] PROP sensitivity has also been associated with sweet preferences among children.[120,121] Studies show that there are fewer nontasters among children than among adults because taste perception changes over time[122,123]; with age, experience, and diseases, people become less sensitive to PROP.[123]

Multiple studies have further documented the finding that sensitivity to bitter tastes is a genetic trait[124,125] mediated by TASR38 and possibly 25 other bitter taste receptors expressed on the tongue.[125] PTC and PROP are perceived as bitter by 70%–75% of the population.[126–128] PTC and PROP have been used as markers of genetic variability in perceptions of taste[129] and to help distinguish three taster groups. Although earlier studies using PTC suggested that taste was bimodal, substantial evidence shows that taste sensitivity is a continuous measure of intensity extending from nontasters, to medium tasters, to supertasters.[126,127,130]

Earlier work on taste sensitivity used thresholds to classify individuals as nontasters (high thresholds) and tasters (low thresholds). In the 1960s, the pioneering work of S.S. Stevens introduced direct scaling methods (especially magnitude estimation) that enabled researchers to assess the rate at which the bitterness of PTC and PROP grew with concentration. In the 1970s, a new method (ultimately called "magnitude matching")[131,132] permitted comparisons of taste intensities across individuals with varying genetic abilities.[133,134] Magnitude matching is based on cross-modality matching, a phenomenon studied by Stevens and his students[135,136] and extended in the modern era by the work of Luce and colleagues.[137] Essentially, cross-modality matching refers to matching sensations for intensity across different qualitative continua. This permits an investigator to select a standard from a continuum unrelated to the continuum of primary interest. For example, nontasters and tasters of PROP were asked to compare PROP bitterness to loudness. This rests on the assumption that taste and loudness are not related; thus, any variation in the perception of loudness should be similar across nontasters and tasters of PROP. Surprisingly, three groups emerged. Nontasters of PROP matched the bitterness they perceived in PROP

to a very soft sound. Tasters of PROP fell into two groups. One group (later called supertasters of PROP) matched their bitterness to a very loud sound; another group (medium tasters of PROP) matched their bitterness to an intermediate sound. Since loudness and taste intensity are not related, average loudness for the three groups is assumed to be the same, which permits a comparison of PROP bitterness across the three groups. Subsequent research using magnitude matching has provided considerable information about chemosensory variation across these three groups (see Table 4.1).

Table 4.1 Sample Characteristics of Taster Types

Highly sensitive tasters (supertasters)	Moderately sensitive tasters (medium tasters)	Mildly sensitive tasters (nontasters)
Strong sensations from PROP as a bitter flavor; strong sensation from mint, which is more pleasant	Moderate to strong bitterness from PROP; moderate sensation from mint	Weak or no bitterness from PROP; weak sensation from mint
High FPD	Less FPD than supertasters	Less FPD than medium tasters
Less likely to smoke than nontasters	Less likely to smoke than nontasters	More likely to smoke than tasters
Higher perception of irritation and pain from oral irritants; higher tactile perception in mouth	Moderate perception of irritation and pain from oral irritants; moderate tactile perception in mouth	Lower perception of irritation and pain from oral irritants; lower tactile perception in mouth
Food flavors important	Food flavors important	Food flavors not that important
Smell perception very strong	Smell perception moderately strong	Smell perception not very strong

Notes: PROP = 6-n-propylthiouracil; FPD = density of fungiform papillae on the tongue.

The three taster groups can be distinguished by examining variations in the density of fungiform papillae, structures that hold the taste buds on the anterior dorsal surface of the tongue. Supertasters have more fungiform papillae than medium tasters or nontasters. Studies show that PROP sensitivity is highly correlated with fungiform papillae density: Supertasters have more than twice as many taste buds per square centimeter as medium tasters.[138–142] Fungiform papillae are the primary sensor of chemesthetic stimuli on the front of the tongue[143]where cigarettes are smoked.

It is important to note that supertasting is not limited to bitter taste.[133] In addition to bitter compounds such as PTC and PROP, Bartoshuk suggests that supertasters perceive stronger taste intensities from sweet compounds.[126,144] Compared to the perceptions of medium tasters and nontasters, supertasters perceive virtually all tastes as more intense.

Supertasters who have the most fungiform papillae[145] experience more intense sensations from oral burn (e.g., chili peppers, ethanol) and oral touch (e.g., fats, thickeners in foods).[144] These properties of supertasting presumably result from anatomy; fungiform papillae are innervated by nerve fibers mediating oral burn and touch as well as by those mediating taste.

Olfactory sensations can be evoked in two different ways. (1) Sniffing odorants from the outside world (orthonasal olfaction) draws odorants through the nostrils into the olfactory cavity where turbinate bones cause a sample to be directed upward through the olfactory cleft and onto the olfactory mucosa. There, odorants contact the olfactory receptors; this is called "smell." (2) When food is placed in the mouth, chewing and swallowing forces any odors emitted from the food up behind the palate into the nasal cavity from the rear (retronasal olfaction). Taste combined with retronasal olfaction make up what is

usually called "flavor." As predicted by Rozin[146] and confirmed by functional magnetic resonance imaging (fMRI) studies,[147] orthonasal and retronasal olfaction do not project to identical central areas, and these areas apparently do not interact in the same way with taste. Taste can enhance retronasal olfaction without enhancing orthonasal olfaction.[148] Thus, supertasters experience more intense retronasal olfaction (i.e., perception of flavor).[149] In other words, supertasters live in a "neon food world" compared to the "pastel food world" of those who have the fewest fungiform papillae.

Confusion Between Individual Bitter Genes and Supertasters

Although supertasters were originally discovered in the context of PROP research, supertasting cannot be explained by PROP genetics. It is now known that the PROP gene expresses a receptor that is quite specific to PROP. This receptor cannot be responsible for supertasters' perception of more intense non-bitter tastes, oral burn, oral touch, and flavor. Clearly, density of fungiform papillae is a crucial part of supertasting. The density of fungiform papillae is essentially independent of the PROP genotype.[57] To clarify the terminology, "nontaster" should only be used in the context of PTC or PROP. Nontasters are not the opposite of supertasters. This point is important to understanding associations between smoking and chemosensory genetics.

Taster Group and Variance Across Populations

In addition to the existence of three taster groups in the world's populations, prior data show that perceptions of taste vary by gender,[150] age,[123,151] and ethnicity.[142,152,153] Studies suggest that about 75% of the population are tasters (medium tasters or supertasters) and 25% are nontasters[144,154–157] and that 35% of women and 15% of men are supertasters.[50] Asians and African Americans may be more likely than whites to be supertasters.[151] Since the early research on this variability, studies have shown that women are more responsive to the bitter taste of PROP and PTC.[145]

As discussed above, analysis of tobacco industry documents indicates that menthol is added to cigarettes in part to reduce the negative sensory characteristics of smoking. Does menthol facilitate smoking among African Americans and women? The targeting of blacks and women through advertisements for menthol cigarettes may have encouraged smoking among people who would be less likely to smoke, based on their chemosensory physiology. To examine this possibility, the next section discusses some chemosensory issues related to the addition of menthol to cigarettes.

Smoking Among Taster Groups

The idea that variation in the unpleasant sensory properties of cigarette smoke as it affects users' ability to perceive these properties may lead to differences in smoking behavior is an old one. Nicotine and tobacco are generally perceived as bitter tastes.[68,158] Studies suggest that PTC/PROP tasters are likely to find cigarettes adversely bitter, and taster status may protect against smoking bitter toxic compounds like tobacco.[159–162] In the 1960s, investigators studying individual differences in taste perception observed that heavy smokers were less sensitive to the bitterness of PTC/PROP than nonsmokers.[119,160] Subsequent studies have produced similar findings, indicating that being a "taster" of PTC or PROP may protect against consuming bitter toxic compounds like tobacco.[50,142,150,159,161] Differences in smoking and taster status have been found among American Indians as well. American Indian nonsmokers and social smokers tend to be PTC/PROP tasters, and regular smokers tend to be nontasters.[150]

Variations in the bitter taste receptor TAS2R38 in particular are associated with smoking behaviors. Black women expressing the "nontaster" form of this gene are especially likely to smoke,[56] and whites expressing the "taster" variant report that tobacco-related sensations do not drive their motivation to smoke.[161] Smoking-related links with other oral sensory receptor genes are likely to generate interest as sequence analysis for those genes becomes available. Recent data suggest that variations in the TRPA1 irritant receptor gene are linked to stronger preferences for menthol cigarettes among heavy smokers.[163]

Two oral sensations associated with menthol—bitter and burn—can lead to rejection by the user if they are sufficiently intense. To better assess the potential role of menthol cigarettes in TRHD, bitter and burn should be further studied.

Inhibition of oral burn is commonly invoked as one of the reasons why menthol is added to cigarettes.[164] On the tongue, menthol desensitizes polymodal nociceptors responsive to heat and to mechanical and chemical irritation,[52] similar to its inhibitory action on respiratory irritation leading to cough.[31] At first glance, menthol's effects on oral irritation would appear unrelated to any effects menthol might have on bitterness, but this is not actually the case. Bitter taste receptors would not be expected to respond to irritants, but bitterness and irritation are connected through supertasting. Supertasters perceive bitter taste and oral irritation more intensely because they express the most fungiform papillae. Thus, if investigators use genotyping to classify PROP nontasters and tasters, they will not capture the full range of variation in bitterness or irritant perception. Attempts to relate sensory variability to variability in smoking behavior would profit from an examination of multiple sources of sensory variability.[57]

To illustrate, the authors compared white smokers and nonsmokers in terms of TAS2R38 genetics (which differentiates tasters from nontasters) and suprathreshold PROP bitterness (which identifies supertasters among tasters). Consistent with earlier reports,[161] genetic analysis alone showed no relationship with smoking behavior. However, a study that combined genetic and psychophysical analysis found that smokers are less likely to perceive PROP bitterness, attributing this finding largely to an absence of supertasters among smokers.[165] In other words, using methods that capitalize on the full range of oral sensory variation revealed that differences in bitter taste perception predict tobacco use in whites[166] just as they do in other racial/ethnic groups.[56,150]

Alexander and colleagues have suggested "that there is an interactive effect of age, race/ethnicity, bitter taste sensitivity, and trigeminal sensitivity related to menthol" which could help explain low rates of smoking among African American youth, followed by transitions to regular smoking as young adults.[16,p.S94] As these authors note, this hypothesis remains to be tested.

Chemosensation and TRHD

Chemosensory alterations that result from radiation therapy for head and neck cancer are of particular interest. Radiation therapy for head and neck cancer typically damages the glossopharyngeal nerve because the radiation is directed toward the rear of the oral cavity, the location of many head and neck tumors. Although some studies claim that any damage to taste by radiation for head and neck cancer is of short duration, other studies contradict this conclusion.[167,168] Damage produced by radiation is generally limited to the glossopharyngeal nerve, leaving the chorda tympani intact. These two taste nerves project to the brain where they interact via inhibitory connections.[169–171] Damage to one nerve releases inhibition on the intact nerve, thus intensifying the sensations mediated by the intact nerve. Thus, many survivors of head and neck cancer may experience changes in chemosensory experience that

could not only influence their quality of life but also affect future behavior so as to increase risk factors for other health problems. For example, damage to the glossopharyngeal nerve by tonsillectomy is associated with enhanced fat preference produced by release of inhibition on fat sensations[172]; increased fat intake is hypothesized to lead to the weight gain associated with tonsillectomy.[173] Similar changes among survivors of head and neck cancer might not lead to weight gain (given eating problems among head and neck cancer survivors) but might increase fat intake, leading to increased risk for cardiovascular disease.

A second phenomenon involving interactions between taste and pain in non-oral body locations may be of special interest with regard to head and neck cancer. In patients with more extensive taste damage (e.g., damage to both cranial nerves VII and IX), pain sensations may be intensified in a variety of body locations.[174] A study of head and neck cancer patients found that current smokers reported higher pain levels than never-smokers and former smokers; the authors hypothesize that smoking may have analgesic properties and that pain management may enhance smoking cessation in this population.[175]

A similar interaction may induce long-term obesity risk early in life. Perinatal tobacco exposure is linked to childhood obesity,[176] and both early tobacco exposure and childhood obesity promote ear infection.[177,178] In severe cases, ear infection can damage the chorda tympani and compromise anterior taste sensation.[179] Based on the disinhibition model described above, such damage appears to elevate fat sensation and preference in a progressive manner. Consequently, overweight children tend to become overweight adults,[180] but data show that childhood ear infection is also linked to obesity in adulthood.[181,182] In similar fashion, children of smokers tend to become smokers themselves,[183] and data have shown that adult male smokers raised in homes with multiple smokers have higher body mass. Consistent with the idea that nontasters are more likely to smoke overall, these men also gain the most weight when they quit smoking,[184] suggesting that sensory cues play a significant role in their tobacco use.[161] A direct link between menthol cigarette smoking, its sensory characteristics, taste sensitivity, and cancer risk has not been identified; this subject deserves greater attention from investigators.

Chapter Summary

The tobacco industry uses flavor additives and ingredients to make the experience of smoking more palatable. This chapter discusses three common additives that affect the chemical senses—cocoa, licorice, and menthol—and the evidence of menthol's effects on the chemical senses—the olfactory, gustatory, and trigeminal systems. Menthol is added to an estimated 90% of cigarettes sold in the United States.[91] It has multiple effects on the chemical senses that may make it easier for consumers to smoke menthol cigarettes; for example, menthol can reduce the pain and irritation of tobacco smoke. These and other factors may help explain the widespread use of menthol in cigarettes, both those that are labelled as menthol and those that are not.

Studies have shown that taste perception is associated with smoking status; the ability to detect bitter taste may help protect individuals from tobacco use. Tasters, including supertasters, who make up approximately 75% of the world's population,[145,154–157] are more likely to reject the bitter taste of tobacco and nicotine. Studies also show that supertasters are more likely to smoke menthol cigarettes than medium and nontasters, and that African Americans, Asians, and women are more likely to be supertasters than whites and men. Supertasters are more likely to perceive bitter flavors, but also perceive stronger taste intensities from PTC/PROP than medium and nontasters. It is possible that

menthol helps mask the bitter, irritating, and painful effects of nicotine/tobacco and in doing so, makes cigarettes and other tobacco products more palatable for supertasters.

The sensory effects of menthol could increase the risk of smoking among African Americans, who are more likely than whites to be supertasters; menthol could also contribute to TRHD if it increases the risk for nicotine dependence and the difficulty of quitting. Marketing menthol to African Americans, women, youth, and other groups, may be more than a marketing strategy. Rather, it may encourage groups with a genetic tendency to reject bitter taste to smoke a tobacco product that they are likely to find more palatable than other tobacco products.

By 2050, over 300,000 cumulative excess deaths are expected to result from menthol smoking in the United States alone.[8] The congressionally mandated 2011 FDA Tobacco Product Scientific Advisory Committee report on menthol cigarettes found that "the evidence is insufficient to conclude that it is more likely than not that smokers of menthol cigarettes have increased risk for disease caused by smoking compared with smokers of non-menthol cigarettes."[8,p.218] However, the 2011 TPSAC report also found that it "is more likely than not that the availability of menthol cigarettes increases the likelihood of addiction and the degree of addition in youth smokers," and that it "is more likely than not that the availability of menthol cigarettes results in lower likelihood of smoking cessation success in African Americans, compared to smoking non-menthol cigarettes."[8,p.216-217] These factors could contribute to the disease burden of lung cancer among groups with high rates of menthol smoking, such as African Americans.

Research Needs

The effects of menthol on TRHD should be studied in relation to the entire tobacco use continuum, smoking initiation through chronic disease outcome.[185] It has been hypothesized that menthol cigarettes increase and maintain smoking in part through menthol's sensory qualities. Further study of the chemical senses may lead to a greater understanding of smoking and quitting behaviors among menthol smokers. The hypothesis that smoking rates would be lower among groups with high rates of menthol cigarette use—such as African Americans, Asians, women, and youth——if menthol cigarettes were removed from the market requires further study. Studies are also needed to determine how other ingredients with effects similar to menthol may influence smoking behaviors, including smoking initiation and maintenance. The chemosensory effects of other flavor additives in cigarettes, such as cocoa, licorice, nutmeg, ginger, and sugar, as both non-characterizing and characterizing flavors, merits further examination. Tobacco industry documents may be a useful source of information on flavor additives and their impact on the chemical senses. It is also important to focus on flavor additives in other tobacco products, including cigars, smokeless tobacco, and electronic cigarettes, as well as those used in conventional cigarettes.

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Section II
Intrapersonal/Individual Factors Associated With Tobacco-Related Health Disparities

Chapter 5
Stress-Related Processes and Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

Tobacco dependence is a complex disorder with interacting biological, behavioral, and psychosocial determinants. Psychosocial determinants that contribute to tobacco-related behaviors and health outcomes are multifaceted and can be both distal (e.g., socioeconomic status [SES]) and proximal (e.g., knowledge, attitudes, affect, and stress). Psychosocial processes acting within the individual include cognitive factors (e.g., knowledge, attitudes toward smoking and quitting, self-efficacy) and affective factors (e.g., mood states, perceived stress). These psychosocial determinants and processes may operate at each stage of the tobacco use continuum (smoking initiation, prevalence, frequency and intensity, cessation, secondhand smoke exposure, morbidity, and mortality) and result in disparities.

This chapter describes the role of stress-related processes and specific stress indicators in tobacco-related health disparities (TRHD). *Stress* may be defined as the effects of threats, real or implied, to the psychological or physiological integrity of an individual.[1] A *stressor* is the actual or perceived threat to the organism, which leads to the individual's *stress response*. Because stress processes are components of the social-ecological experience of smokers, this chapter focuses on individual/intrapersonal and interpersonal stress processes, while considering community and neighborhood influences on the experience of stress. This review aims to understand what is known about the role of stress at each point along the tobacco use continuum.

This chapter first presents several conceptual frameworks that may help to explain the relationship between stress processes and TRHD. Second, the chapter discusses physiological stress responses and their relationship to disparities in minority racial/ethnic and lesbian, gay, bisexual, and transgender (LGBT) groups. Third, the chapter provides an overview of the influence of perceived stress on the tobacco use continuum and in specific racial/ethnic, gender, and LBGT groups. Fourth, the chapter discusses the relationship between TRHD and the specific stressors of racism and discrimination, psychological disorders (i.e., trauma and post-traumatic stress disorder [PTSD]), and intimate partner violence (IPV) because of their association with smoking prevalence, motivation to quit, and disparate health consequences. More information on the relationship between TRHD and distal factors like SES is provided in chapters 8 (occupation) and 9 (education and income).

Stress Processes and TRHD: Literature and Conceptual Frameworks

Stress Processes Literature Search Strategy

The literature search covered four areas: (1) physiological (sympathetic nervous system [SNS] and hypothalamic-pituitary-adrenal [HPA] axis stress responses), (2) perceived stress, (3) discrimination and racism as stress contributors, and (4) other stress-related processes (e.g., post-traumatic stress). The search included all published literature on U.S. participant samples through April 2014. Research investigating perceived neighborhood stress from the individual perspective was eligible for inclusion in the review, but ecological-level stressors related to environmental hazards, high rates of violence and crime, and other variables related to neighborhood disadvantage and SES were not included.

Searches were conducted in the PubMed, PsychInfo, and Web of Science databases. The Boolean search terms for the physiological section of the chapter included smoking, physiological stress, hypothalamic-pituitary-adrenal axis (HPA), parasympathetic nervous system, adrenocorticotropic hormone (ACTH), neuroendocrine, allostatic load, cortisol, race (substituting each of the following for race: ethnic, African American, black, white, Hispanic, Asian, Native Hawaiian, Pacific Islander, Native American Indian, low SES, lesbian, gay, bisexual, transgender, LGBT, sexuality, disparities, minority), and each level of

the tobacco use continuum. The results of these searches yielded many articles investigating cardiovascular health and obesity that included smoking status as a covariate. The studies were further examined to determine whether they reported findings for stress associations with smoking by racial/ethnic or LGBT status. The search was considered exhaustive when various search term combinations resulted in duplicate or irrelevant studies. Articles were excluded if analyses based on both smoking status or race/ethnicity/sexuality/disparities were not reported.

Search terms for the perceived stress and racial discrimination sections included smoking, race (substituting each of the following for race: ethnicity, low SES, LGBT, sexuality, disparities, minority, discrimination, racism), stress, distress, acculturative stress, and each level of the tobacco use continuum.

In addition to searching electronic databases, the reference lists were also reviewed for published studies to identify additional sources. The final search yielded 47 articles related to tobacco smoking and disparity populations (physiological stress: 0; perceived stress: 26; racism and discrimination: 21).

Conceptual Frameworks and Physiological Stress Response

This first section provides an overview of conceptual models related to stress processes. The next section briefly reviews the literature on how the effects of stress on health are mediated by the central and peripheral nervous systems, which coordinate the behavioral and physiological changes that enable an individual to respond to stress.

Conceptual Frameworks

Several models have been introduced to address the role of stress in shaping behavior, the factors that increase susceptibility to the effects of stress, and the factors that mediate the effects of stress on health. This section reviews four models (the diathesis stress model, transactional model of stress and coping, biospychosocial model, and allostatic load model) and the extent to which the disparity-related literature has applied these models to examine the impact of stress on smoking and health.

The *diathesis stress model* emphasizes the role of dispositional or vulnerability factors in shaping the influence of adverse environmental events on the risk for psychopathology or other health problems.[2] This vulnerability, which could be biological, genetic, or environmental, may magnify the negative quality of stressful events that occur.[3] In the context of smoking behavior, having a genetic or biological predisposition may increase the impact of psychosocial and stress factors on smoking initiation, maintenance, relapse, and disease risk.

The *transactional model of stress and coping,* introduced in the late 1970s, defines stress as a demand exacted by the interaction of both external components of a stressful or challenging situation and the person's appraisal of the situation.[4] In this model, the impact of a stressful event is determined not only by the properties of the situation itself but also by the person's perception of it, which includes the person's perception of his/her ability to overcome this challenge as well as previous experiences with similar situations. Thus, the impact of the stressor will be determined by how much the person perceives the situation as threatening and overwhelming relative to his/her coping ability. This model introduced concepts related to coping, including primary and secondary appraisals of stress. Primary appraisals focus on evaluating the stressor itself and its characteristics. Secondary appraisals focus on evaluation of

one's abilities and resources to cope with the stressor. The balance of these two types of appraisals determines the perception of the stressor as a threat or as a mere challenge to be overcome.

The transactional model conceptualizes coping as problem or emotion focused. Problem-focused coping refers to the use of behavioral and problem-solving strategies to address the sources of stress directly and attempt to modify the situations that produce stress. Emotion-focused strategies concentrate efforts on the person, addressing his/her cognitive or emotional reactions to the stressful events or actions to mitigate the impact of the stressor. Cigarette smoking is a behavioral method that is widely used to cope with stress and negative affect. To the extent that stress levels vary across groups, high stress levels in some groups may increase the likelihood of cigarette smoking as a coping method and therefore affect smoking consequences.

The *biopsychosocial model* integrates multiple influences—psychological (e.g., thoughts and emotions), biological (e.g., genetic), and social factors (e.g., cultural and environmental)—and considers the interaction of these factors in increasing risk for diseases or behavioral disorders.[5] According to this model, stress is a psychological condition that could influence tobacco use, along with biological factors such as the pharmacological effects of tobacco, other psychological conditions (e.g., emotional status), and social conditions (e.g., peers, modeling, and neighborhood). These factors may influence not only initiation, but also maintenance of smoking and the harm caused by smoking. The biopsychosocial model has been widely used in advocating a multimodal approach to conceptualizing, assessing, and treating disease. The influence of this approach has extended across multiple disciplines, although some concerns about its limitations have been expressed.[6]

Finally, the *allostasis model of stress* highlights the importance of neuroendocrine adaptation in response to stressors.[7] In normal, healthy neuroendocrine–immune function, adaptive systems are activated and deactivated infrequently over short time periods. Chronic activation over the life span can result in dysregulation (i.e., hyporesponsivity or hyperresponsivity) of the physiological stress response, referred to as allostatic load. McEwen[7] suggests that allostatic load can compromise immune system functioning and facilitate pathogenesis. To the extent that racial/ethnic minority groups and LGBT individuals may experience greater allostatic load, this model has implications for disparities in tobacco-related outcomes.

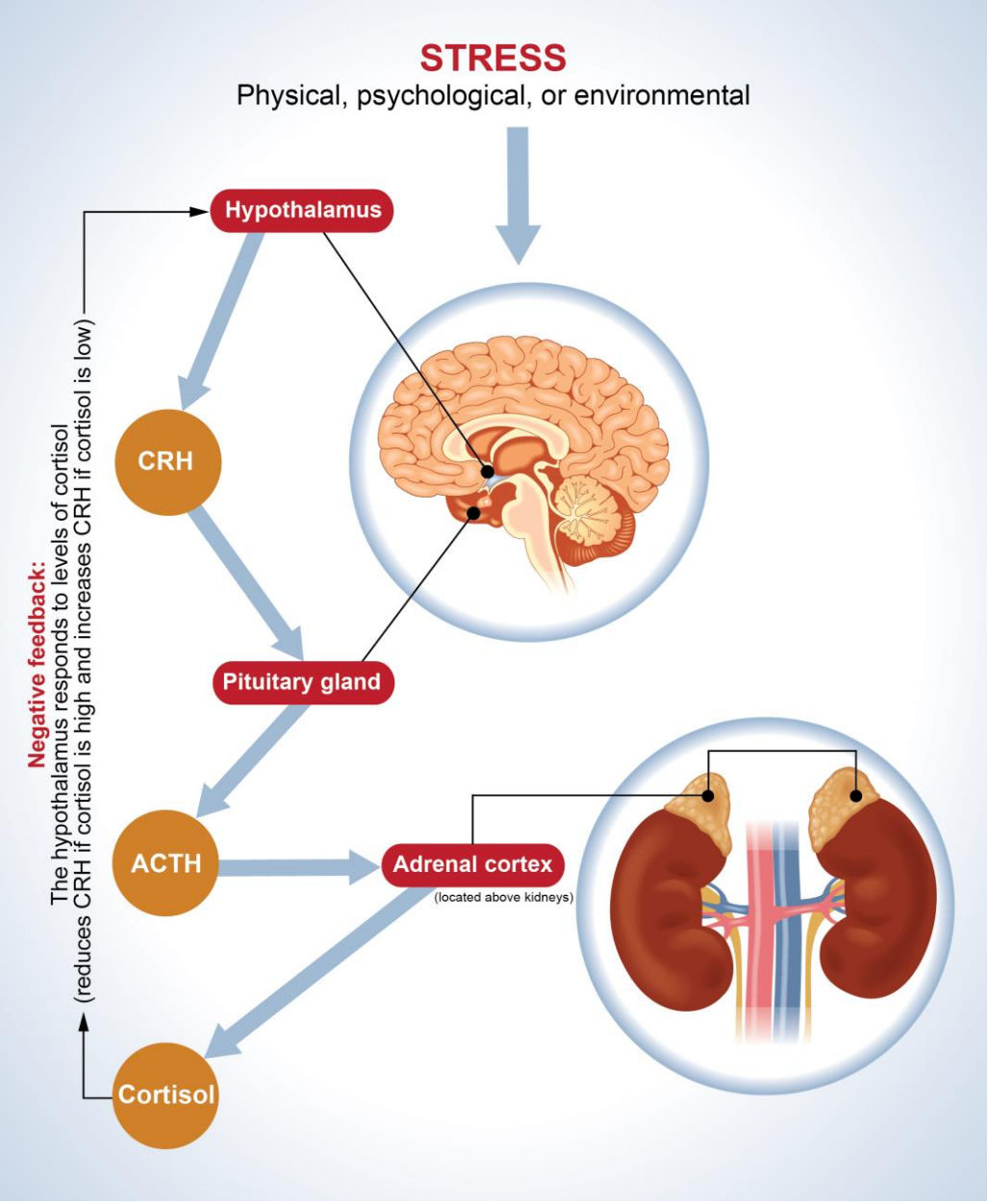
This review of the relationships between stress and smoking in disadvantaged populations identified very few studies grounded in one or more of the above models of stress. The extent to which these established models can be generalized to the tobacco use continuum in these racial/ethnic, gender, and LGBT groups is unknown. Research incorporating these stress frameworks as well as new conceptualizations is needed to better understand the relationship between stress and the tobacco use continuum.

Nicotine and Physiological Stress Response Systems

The effects of stress on health are mediated by the central and peripheral nervous systems, which coordinate the behavioral and physiological changes that enable an individual to respond to stress. This section presents information on the effects of nicotine, one of the primary constituents responsible for tobacco addiction, on physiological stress response.

Several brain structures are involved in the perception of external and internal events as threatening or stressful and in regulating the stress response. The two neurobiological systems that directly regulate the physiological stress response are the HPA axis (see Figure 5.1) and the sympatho-adrenomedullary system. The HPA axis is also involved in mediating the effects of nicotine.[8–11] The stimulating effects of acute doses of nicotine on stress-related biological systems, including the HPA axis, the SNS, and the endogenous opioid system, have been documented in several laboratories.[12–16] The SNS plays an essential role in the body's acute adjustments to normal demands, and is involved in integrating and expressing the physiological and behavioral responses known as the fight-or-flight response.

Figure 5.1 Hypothalamic-Pituitary-Adrenal Axis



Notes: CRH = corticotropin-releasing hormone. ACTH = adrenocorticotropic hormone.

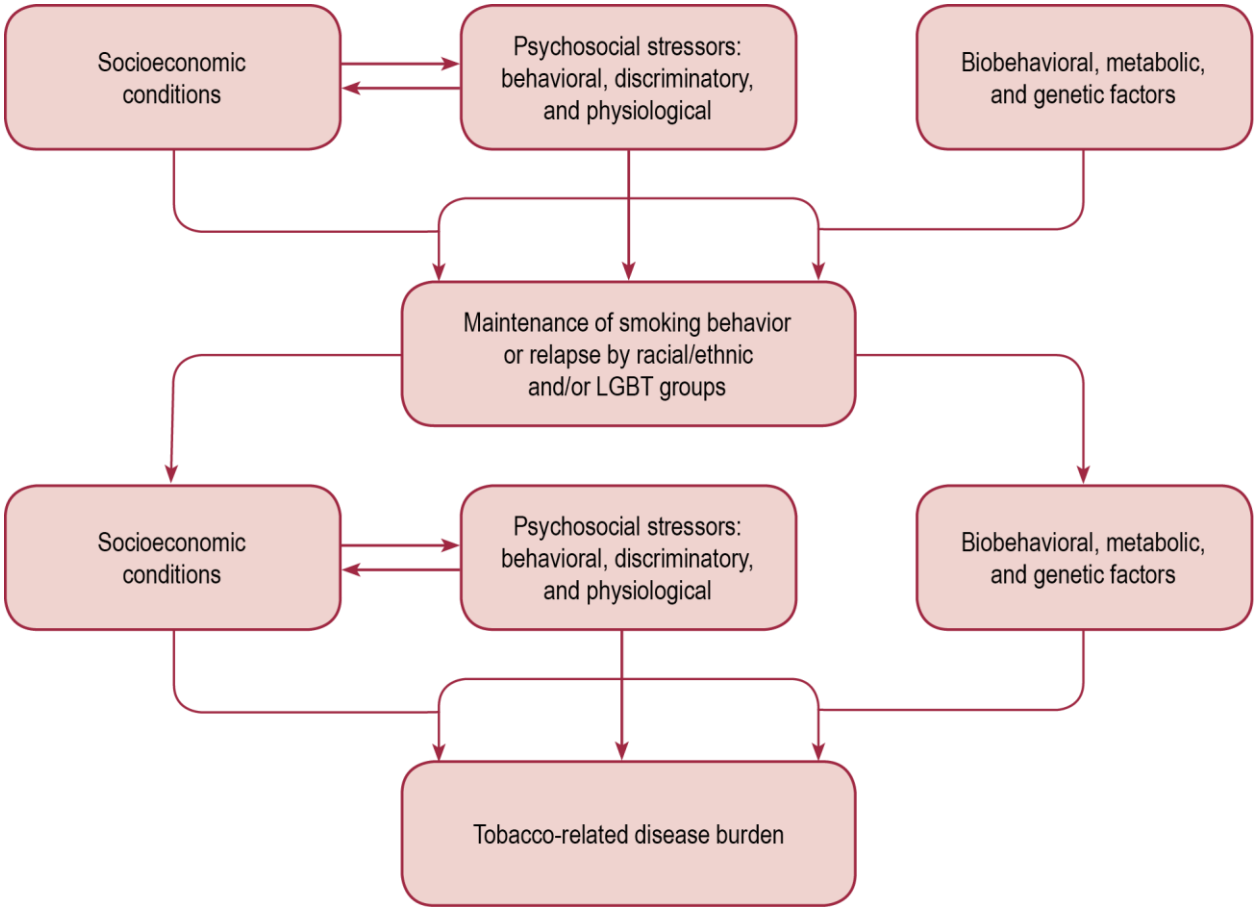
Nicotine's effects on neurobiological stress response systems are centrally mediated, although the specific pathways have not been fully elucidated. Nicotine stimulates dopaminergic, noradrenergic, and serotonergic neurotransmission.[17–19] Vreeburg and colleagues[20] found that current smoking was associated with higher levels of cortisol, a hormone released at awakening, in the evening and in response to stress. Experimental evidence is consistent with other research examining the impact of stress on tobacco smoking. Using a within-subjects design, McKee and colleagues[21] exposed nicotine-deprived smokers to either neutral-personalized or stress-personalized imagery across a 2-day period. The stress-inducing imagery diminished the ability to delay smoking for a short time period compared to neutral imagery. Stress also led to greater HPA axis reactivity, cravings, puffing intensity, and perceived positive reinforcement in comparison to the neutral condition. This research has implications for the role of stress in smoking cessation disparities across racial/ethnic groups. For example, the greater allostatic load among racial/ethnic minority groups due to the cumulative effects of daily stress might be related to the greater difficulty in quitting observed in these populations.

Physiological Stress Processes and Health in Racial/Ethnic and LGBT Groups

An individual's manner and intensity of stress response are influenced by several dispositional, environmental, and situational factors. Variables related to membership in racial/ethnic and LGBT groups could influence the impact of stress and the individual's ability to cope with it. Accounting for these factors is important, considering the potential harmful effects of stress on smoking behavior and related health outcomes.

In accordance with the allostasis model, greater exposure to stress may contribute to racial/ethnic health disparities.[22] Evidence indicates greater risk of allostatic load for African Americans compared to whites.[23] Relationships between psychosocial factors, smoking, and health burden in racial/ethnic and LGBT groups are multifaceted and are mediated in part by stress processes (Figure 5.2). Race/ethnicity is independently associated with the prevalence of both recent and lifetime stressful events.[24] LGBT groups also experience stressors that impact overall quality of life, such as structural discrimination and stigmatization.[25] Chronic activation of the stress response system can result in pathogenic processes.[26] Studies show that the cumulative effects of high-stress circumstances robustly predict morbidity and mortality from chronic obstructive pulmonary disease (COPD) and lung cancer.[27,28]

Figure 5.2 Relationships Between Biopsychosocial Factors and Tobacco Use Among Racial/Ethnic and LGBT Groups and Their Effects on Health



Note: LGBT = lesbian, gay, bisexual, and transgender.

Racial/Ethnic Differences in Sympathetic Nervous System and Hypothalamic-Pituitary-Adrenal Axis Functioning

The few studies that have examined physiological stress processes in relation to smoking or tobacco use for each racial/ethnic group provide initial evidence of differential SNS and HPA functioning by race/ethnicity. Most of these studies compare African Americans and whites; they do not include or report outcomes for other racial/ethnic minority groups, nor do they examine LGBT groups.

In one comprehensive investigation, Skinner and colleagues[23] examined patterns of HPA regulation among African American and white young adults and assessed smoking behavior over a 3-day period. These researchers hypothesized that HPA functioning varied for the two groups as a function of the African Americans' exposure to stressors (e.g., discrimination, racism, daily race-related hassles, financial strain, past-year life problems, stressful life events, neighborhood quality, personal experiences with violence, and family conflict). Results confirmed the greater stress experiences of African Americans relative to whites. Blunted diurnal rhythms were related to smoking among whites but not among African Americans. Although the authors did not offer an interpretation of the latter finding, the relationship between smoking and diurnal rhythms in African Americans might have been obscured by the slower overall cortisol decline. In a study of pregnant women, Suglia and colleagues[29] found that

African Americans who reported high cumulative stress (interpersonal violence, discrimination, negative family-related life events, and community violence) also had lower cortisol levels on awakening and a flatter slope throughout the day compared to Hispanics.

The existing research has considered multiple indexes of HPA axis responses, such as cortisol reactivity at different points throughout the day and adrenocorticotropic hormone (ACTH) levels. Within the half-hour following a public-speaking stressor, Chong and colleagues[30] found a 36% greater cortisol response (95% confidence interval [CI] 10%–67%) and 35% higher mean plasma ACTH levels (95% CI 16%–58%) in white men and women compared to African American men and women. Compared with white women, African American women have shown greater plasma ACTH immunoreactivity (ACTH-IR) following intense exercise[31] and lower allopregnanolone (ALLO) following psychological stress.[32] A study of the relationship between race and HPA response in African American and white men found greater ACTH concentrations in African Americans than whites after equal intravenous administration of corticotropin-releasing hormone (CRH).[33] In another study, Cohen and colleagues[34] found that African Americans in the Coronary Artery Risk Development in Young Adults (CARDIA) study were more likely to have higher evening cortisol levels than whites. These findings suggest there may be racial differences in HPA functioning.

Discrimination and Hypothalamic-Pituitary-Adrenal Responses

Research suggests that neuroendocrine functioning may be directly impacted by perceived discrimination. (Discrimination and its role in TRHD are also discussed in chapters 6 and 8.) Racism refers to a pejorative reaction to an individual or group based on the belief that all members of a racial category possess characteristics that render them inferior to another race (or races). Discrimination refers to intentional acts based on racial, ethnic, or sexual identification that are unfair or injurious, and that benefit the "in" versus "out" group. Landrine and Klonoff[35] conceptualized racial discrimination (i.e., racist events) as culturally specific stressors. Individuals and groups with racial/ethnic minority status experience negative stressors because of their group membership. These stressors may be direct and explicit acts of discrimination or indirect and subtle attitudes or behaviors.[36] Racial and ethnic minority groups have experienced greater racial discrimination than whites historically and currently, in the 21st century.[37,38]

Jamieson and colleagues[39] exposed African American and white participants to social rejection by same-race or cross-race partners (conceptualized as discrimination) in a computer-based experiment. These researchers found that for both African American and white participants, cross-race rejection led to lower cortisol reactivity, greater anger, and risky behavior compared to rejection by partners of the same race, suggesting that racial discrimination influences physiological responses, cognition, and risk behaviors.

Hypothalamic-Pituitary-Adrenal Axis Regulation and TRHD

Findings from studies comparing HPA axis regulation by race/ethnicity have implications for TRHD. Among participants in the Boston Puerto Rican Health Study, allostatic load and smoking were among the strongest correlates of poor self-rated health.[40] Indeed, chronic diseases may be a consequence of maladaptive elevation or attenuated responses of the HPA system.[41] This differential HPA functioning across vulnerable populations may contribute to significant disparities in smoking-related morbidity. Multiple stress response processes influence how individuals respond to acute and chronic nicotine exposure. It is possible that the blunting effect of habitual nicotine exposure on neuroendocrine

responses combined with minority populations' greater difficulty maintaining abstinence[42] leads to variations in HPA axis activity that may contribute disproportionately to adverse health outcomes. However, no studies have explicitly compared HPA axis hormones in plasma, urine, or saliva of smokers across racial/ethnic or LGBT groups. Thus, there are large gaps in what is known about how much physiological stress contributes to disparities in tobacco cessation and health.

Perceived Stress and Tobacco Use: Overview

A large body of research has demonstrated the clear link between perceived stress and all phases of the nicotine addiction process, including initiation, maintenance, and relapse.[43] Stress is widely cited by smokers as the reason they continue to smoke or relapse to cigarette smoking after cessation. Tobacco dependence is a complex phenomenon that involves multiple bio-neurological, psychosocial, and behavioral processes, but growing evidence suggests that perceived stress and expectation of relief are important motivators for smoking.

Perceived stress is a multidimensional construct, and research has considered the effects of different types of stress on smoking status. Mulder and colleagues[44] demonstrated that multiple types of stressors (financial problems, low perceived health, low perceived life control, and lack of social support) were directly related to smoking, and partially mediated the association between education level and smoking. Moreover, residing in highly stressful communities is associated with increased risks of smoking-related illnesses, such as cancer and COPD.[27] Slopen and colleagues[45] examined the relationships between psychosocial stress and smoking among a national cohort of 4,938 U.S. adults at baseline and at follow-up, 9 to 10 years later. They found that high levels of psychosocial stress (related to relationships, finances, employment, perceived inequality, past-year family problems, and overall stress) at baseline and follow-up were associated with increased odds of persistent smoking (odds ratio [OR] 1.40; 95% CI 1.08–1.81). In a sample of low-income smokers in a randomized trial, Bock and colleagues[46] found that elevated stress levels predicted lower abstinence rates over a 1-year period. Qualitative findings suggest that high-stress environments may also negatively influence smokers' attempts to seek help with cessation, even when cessation assistance is available at no financial cost.[47]

Several studies address the relationship between stress and smoking behaviors in various racial/ethnic groups. Most of this research has been conducted among African Americans, but there is evidence that stress also affects smoking in other groups. Among young adults (ages 18 to 36) of various racial/ethnic backgrounds, stress represents a risk factor for smoking initiation[48]; among adults age 25 and older, psychosocial stress was a mediating factor in current smoking.[49] In multiethnic samples, perceived stress/negative affect has been found to indirectly mediate relationships between low SES and smoking relapse through low self-agency.[50,51]

Qualitative findings from primarily minority racial/ethnic groups in a disadvantaged community in South Bronx, New York, highlight the high stress levels associated with a number of factors (financial problems, housing concerns, personal and family health problems, safety issues, and employment conditions) and the possibility that smoking is a pathway linking stress to disparities.[52] Other studies have found that racial/ethnic differences in daily stressors may contribute to physical and mental health disparities.[53–55] Table 5.1 summarizes research on perceived stress and smoking in minority communities.

Table 5.1 Studies Examining Perceived Stress and Tobacco Use Among Racial/Ethnic and LGBT Groups, 1991–2013

Author (year)	Population(s)	Study design	Stress constructs	Findings
African American (majority of sample)				
Beech and Scarinci 2003[58]	Low-income African Americans	Qualitative		Focus groups explored the sociocultural factors associated with smoking attitudes and practices among low-income African American young adults. One focus group theme was that smoking is perceived as relieving stress.
Berg et al. 2010[65]	African American smokers, treatment-seekers	Prospective	Global perceived stress	Positive association with smoking reduction*; lower perceived stress predicted cessation at week 26*
Businelle et al. 2009[59]	African Americans, treatment-seekers	Cross-sectional	Global perceived stress	Light smokers reported lower perceived stress than moderate to heavy smokers*
Manfredi et al. 2007[67]	Low-income, women, African American, treatment-seekers	Cross-sectional	Global perceived stress	Indirectly mediated intentions and cessation through low self-efficacy*
Manning et al. 2005[66]	Low-income African American smokers, treatment-seekers	Longitudinal, prospective	Global perceived stress, daily hassles	Baseline levels not predictive of cessation; concurrent stress inversely associated with abstinence*; reductions from baseline to EOT positively predicted abstinence*
Romano et al. 1991[56]	African Americans	Cross-sectional, random sampling, community survey	Daily hassles	Positive association with smoking status*
Sheffer et al. 2011[47]	African Americans	Qualitative	Stress related to low SES, occupation, environment; daily hassles	Stress identified as a “root cause” of tobacco use
Slopen et al. 2012[57]	African Americans	Cross-sectional	11 stress domains: psychological work stress, physical work stress, work–family conflict, perceived inequality, relationship stress, neighborhood stress, discrimination, financial stress, problems in immediate family during the past year, stressful life events, and childhood adversity	7 of 11 stressors exhibited positive associations with being a current smoker*: neighborhood, financial, relationship, and psychological work stress, perceived inequality, stressful events, childhood adversity
Webb and Carey 2008[61]	Low-income African American women	Cross-sectional	Global perceived stress	Positive associations with light, moderate, and heavy smoking*
Webb and Carey 2009[69]	Low-income, African Americans, treatment-seekers	Cross-sectional	Global perceived stress	Positive association with smoking-related symptoms*; positive associations with cardiovascular* and gastrointestinal symptoms*

Table 5.1 continued

Author (year)	Population(s)	Study design	Stress constructs	Findings
African American and other groups				
Kiviniemi et al. 2011[64]	Whites, African Americans, Hispanics	Cross-sectional, nationally representative telephone survey	Psychological distress (e.g., sad, nervous)	Positive association with current smoking and smoking intensity among whites only*
Ludman et al. 2002[60]	Low-income white and African American women	Cross-sectional	Global perceived stress	Positive association with smoking dependence among African American women only*
Martinez et al. 2010[72]	African Americans, Hispanics, others, treatment-seekers	Cross-sectional	Global perceived stress	Inverse association with cessation self-efficacy in full sample*; Hispanics reported cessation self-efficacy when facing internal stimuli*
Maxson et al. 2012[63]	White and African American pregnant women	Cross-sectional	Global perceived stress	Positive associations with current* and former smoking*
Schulz et al. 2008[49]	African Americans, whites, Hispanics	Cross-sectional, probability sampling	Psychosocial stress	Positive association with current smoking*
Non-African American				
Borrelli et al. 2011[70]	Dominicans, Puerto Ricans, non-Hispanic whites	Cross-sectional	Daily hassles	Dominicans reported greater stress than non-Hispanic white smokers
Friis et al. 2006[78]	Cambodian Americans living in California	Qualitative	Psychosocial (family and relationship problems) and stress related to home environment	Positive association between stress and smoking intensity; smoking to cope with family-related stress
Hayes and Borrelli 2013[71]	Hispanics	Cross-sectional	Global perceived stress	Positive association with smoking intensity*
Hodge et al. 1996[75]	American Indians	Descriptive, cross-sectional	Daily hassles	Positive association with living in urban vs. rural areas*
Honda 2005[73]	Non-Hispanic whites, non-Hispanic blacks, Hispanics, others, ages ≥ 60 years	Cross-sectional, nationally representative survey	Psychological distress (e.g., sad, nervous)	Positive association with current smoking*; Hispanics less likely to be former/never-smokers than non-Hispanic whites*
Spigner et al. 2005[77]	Chinese American and Vietnamese American men	Qualitative		One focus group theme was smoking to alleviate stress
Lesbian, Gay, Bisexual, and Transgender				
Blosnich 2012[82]	Lesbian, gay, and bisexual	Cross-sectional	Physical violence, stress related to sexual orientation	Violence victimization positively associated with current smoking,* particularly among bisexuals*; gay-related stress positively associated with smoking among males*

Table 5.1 continued

Author (year)	Population(s)	Study design	Stress constructs	Findings
Johns et al. 2013[123]	LGBT women	Cross-sectional, observational	Daily hassles	Positive association with current smoking*; no association with smoking frequency or intensity
Rosario et al. 2008[83]	Lesbian and bisexual youth, ages 14–21 years	Longitudinal, prospective	Gay-related stressful life events, internalized gay-related stress (internalized homophobia), emotional distress (e.g., depressive symptoms)	Gay-related stress, internalized homophobia; emotional distress mediated an association between butch/femme identification and smoking*
Rosario et al. 2011[84]	Lesbian, bisexual, and gay youth	Longitudinal, prospective	Gay-related stressful life events, interpersonal stress, psychological distress	Stressful life events positively associated with current smoking,* smoking status moderated association between stress and distress*

Notes: SES = socioeconomic status. EOT = end of treatment. LGBT = lesbian, gay, bisexual, and transgender. Gay-related stress refers to gay-related stressful events, negative attitudes toward homosexuality, and discomfort with homosexuality.
*Finding was statistically significant.

Perceived Stress and Tobacco Use: African Americans

Current Smoking and Smoking Intensity

Perceived stress appears to be associated with smoking risk and maintenance among African American smokers. Using a random sampling design to survey African American households in the San Francisco Bay area, Romano and colleagues[56] found that high levels of stress were associated with current smoking, compared to lower stress levels. Slopen and colleagues[57] conducted a comprehensive assessment of the stress–smoking relationship in African Americans, considering 11 distinct stressors (e.g., work–family conflict, perceived inequality, relationship stress, and stressful events). Seven of the 11 stressors and a cumulative stress score were significant risk factors for current smoking versus never smoking. In addition, the risk of smoking increased more than threefold, with scores in the highest quartiles on five or more stressors. In a qualitative study with 18- to 35-year-old low-income African American smokers, Beech and Scarinci[58] concluded that relief from stress may be a contextual factor related to smoking in this group. Perceived stress is also associated with smoking intensity (number of cigarettes smoked per day) among African American treatment-seekers, such that light smokers (5–10 daily cigarettes) report less stress.[59] Thus, high-stress environments may have implications for smoking risk among African Americans.

Current Smoking and Smoking Intensity Among Women

Several studies have examined perceptions of stress and smoking among African American women. Ludman and colleagues[60] compared low-income African American and white female smokers and found that perceived stress was related to smoking intensity among African American women but not among white women. Webb and Carey[61] found a positive association between perceived stress and current smoking in a community-based sample of African American women. Two studies that focused on correlates of smoking status among pregnant African American women found positive associations between perceived stress and smoking.[62,63]

Current Smoking and Distress

One study has examined the relationship between psychological distress and current smoking in a nationally representative sample. Using data from the 2007 Health Information National Trends Survey (HINTS), Kiviniemi and colleagues[64] demonstrated an interaction between race/ethnicity and generalized psychological distress, such that past 30-day psychological distress was positively related to smoking among whites, but not African Americans or Hispanics. The authors note limitations of their study including its cross-sectional nature and that the HINTS survey measured overall psychological distress.

Smoking Cessation

Perceptions of stress are predictive of smoking cessation in the general population, and some research has examined this phenomenon among African Americans. Berg and colleagues[65] considered perceived stress as a factor influencing trajectories of smoking-related behavior changes among treatment-seekers over 6 months, including reductions in daily smoking, cessation, or no behavior change. Contrary to expectations, reductions in smoking were not predicted by lower baseline perceived stress. However, consistent with previous literature, this study found that lower perceived stress levels predicted smoking cessation at week 26.

Manning and colleagues[66] examined the association between perceived stress and smoking cessation among African Americans in the placebo arm of a bupropion randomized controlled trial. Baseline perceived stress levels were not predictive of end-of-treatment or 6-month cessation, but greater concurrent stress was associated with failure to quit. Two other studies included perceived stress in conceptual models to understand smoking cessation among African Americans in the treatment context. Manfredi and colleagues[67] found that perceived stress was a cessation barrier among women (sample was 77% African American) at 6 months after treatment, acting indirectly through low self-efficacy, which in turn impacted quitting plans and cessation. Reitzel and colleagues[68] found that the relationship between social cohesion and 6-month continuous abstinence was indirectly mediated by perceived stress.

Tobacco-Related Morbidity

Few studies have specifically investigated the influence of perceived stress on tobacco-related morbidity among African Americans. Webb and Carey[69] focused on stress as a psychosocial factor that may influence early health symptoms in a sample of African American treatment-seekers. Results confirmed a high prevalence of the early health consequences of smoking, such as shortness of breath (66%) and coughing (50%). After adjustment for sociodemographics, smoking, and medical history, perceived stress was independently associated with smoking-related symptoms. As suggested by models for examining TRHD,[53] health outcomes among smokers are a function of multilevel influences, including perceived stress.

Summary of African Americans, Stress, and Smoking

African American smokers appear to exhibit high levels of global, specific, and unique stressors, which may contribute to health disparities. An abundant literature supports the association between perceived stress and smoking risk and maintenance among African Americans. For relationships with the other stages along the tobacco use continuum, such as cessation and morbidity, there is less evidence, and no studies have examined stress and mortality among African Americans with a tobacco use history. The

literature is characterized by methodological limitations: With few exceptions, studies of the association between perceived stress and smoking status in this population have relied on convenience and/or treatment-seeking samples and cross-sectional designs. Perceived stress, in particular, is positively associated with current smoking, smoking intensity, and specific types of stressors (environmental or neighborhood).

Perceived Stress and Tobacco Use: Hispanics/Latinos

Although the prevalence of cigarette smoking in the aggregate Hispanic category is relatively low, the evidence suggests that as immigrants acculturate to mainstream attitudes, beliefs, and practices, current cigarette smoking may be affected, although the direction of the relationship depends on multiple factors (discussed further in chapter 7). Little is known regarding the relationship between stress and smoking in this heterogeneous ethnic group.

In a sample of Hispanic and white smoking caregivers for children with asthma, Borrelli and colleagues[70] found that Dominicans reported greater stress, and factors such as lower nicotine dependence were positively associated with smoking cessation. Hayes and Borrelli[71] examined differences in psychosocial characteristics among treatment-seeking Hispanics who were light smokers (3–9 cigarettes per day) and moderate/heavy smokers (10 or more cigarettes per day). Perceived stress was greater among heavier smokers compared to light smokers, yet there were no differences in smoking cessation between groups at 2- and 3-month follow-ups.

Additionally, two studies have also investigated psychosocial processes associated with smoking cessation among multiethnic samples that included Hispanics. Martinez and colleagues[72] demonstrated that perceived stress was inversely related to cessation self-efficacy in a multiethnic sample that included a representative proportion of Hispanics. They found that compared to other ethnic groups, treatment-seeking Hispanics reported lower cessation self-efficacy upon encountering internal stimuli, although they did not test the interaction between ethnicity and stress on self-efficacy specifically. Honda[73] examined correlates of smoking cessation among older ever-smokers using data from the 2000 National Health Interview Survey (NHIS). This study found that psychological distress was lower among former smokers in general, and suggested that Hispanic ethnicity was an independent barrier to cessation. Studies have not reported data on other specific Hispanic groups.

Perceived Stress and Tobacco Use: American Indians/Alaska Natives

American Indians/Alaska Natives as an aggregate group have the highest smoking prevalence of all racial/ethnic groups in the United States,[74] but little is known about the association between perceived stress and smoking in this aggregate group. Hodge and colleagues[75] assessed daily hassles, an indicator of stress, in a sample of American Indian primary care patients, and found that those who lived in urban rather than rural areas had higher smoking prevalence rates and reported greater stress and less social support. No studies have examined associations between stress and other dimensions along the tobacco use continuum. Studies have not reported data for specific American Indian or Alaska Native groups.

Perceived Stress and Tobacco Use: Asian Americans

No quantitative studies have examined the relationship between perceived stress and smoking among the Asian American aggregate group. Acculturative stress—measured using 7 items most relevant to South Korean students taken from the 36-item Acculturative Stress Scale—has been associated with an

increase in smoking among students from South Korea studying at U.S. universities.[76] Spigner and colleagues[77] conducted a qualitative investigation of tobacco-related perceptions among 30 Chinese and Vietnamese immigrants. Among the themes participants discussed during focus groups was the use of smoking as a method of alleviating stress. Friis and colleagues[78] assessed predisposing, reinforcing, and enabling as theoretical factors that may be associated with smoking status in a sample of Cambodian Americans in California. Using qualitative methods, they concluded that smoking is used to manage stress. Participants described increased smoking frequency during stressful times, when they experienced financial problems, family and relationship difficulties, or home environment problems. These few studies suggest that some Asian American groups may use smoking as a coping strategy during stressful periods.

Perceived Stress and Tobacco Use: Native Hawaiians and Other Pacific Islanders

The relationship between perceived stress and smoking among Native Hawaiians and Other Pacific Islanders has not been extensively examined by researchers. Hickman and colleagues[79] found that psychological distress was positively associated with menthol cigarette smoking in this population. As noted earlier, however, distress is more closely aligned with depressive symptoms than perceptions of stress.

Perceived Stress and Tobacco Use: LGBT Populations

As discussed in chapter 2, smoking rates among individuals who identify as LGBT exceed the national average. Blosnich and colleagues,[80] in a systematic review focusing on the etiology of tobacco-related disparities among LGBT groups, confirmed that few studies have examined the stress–smoking association in these vulnerable populations. Some evidence has suggested that these individuals experience greater risk factors for smoking, including stress, than heterosexuals. Since the publication of Blosnich and colleagues' review,[80] a study by Johns and colleagues[81] found that perceived stress was positively associated with current smoking among LGBT groups, while discrimination was not associated with current smoking. In addition, perceived stress was not associated with the frequency of smoking (daily versus some day) or intensity (number of cigarettes per day), but discriminatory events increased the likelihood of daily (versus some days) smoking. Another study by Blosnich[82] focused on the relationship between unique stressors (being the victim of violence or discrimination) and smoking among LGBT individuals ages 18–24. Current smoking was positively associated with experiences of physical violence, and gay-related stress (gay-related stressful events, negative attitudes toward homosexuality, and discomfort with homosexuality) was associated with greater likelihood of smoking among males than among females. The positive association between smoking and gay-related stress has also been found among young (ages 14–21) ethnically diverse lesbian and bisexual women in a community-based sample.[83] Rosario and colleagues[84] showed that stress moderated the relationship between smoking and psychological distress in a sample of 14- to 21-year old lesbian, gay, and bisexual individuals: No differences in distress were found between smokers and nonsmokers in low-stress conditions, but high stress among smokers (but not nonsmokers) was related to elevated distress. In short, perceived stress and sexuality-specific stress have been associated with smoking status. No studies have considered the role of perceived stress in smoking cessation, relapse, or morbidity in LGBT populations.

Racism and Discrimination and Their Relationship to Disparities

Because of the social categories to which they belong, members of racial/ethnic, gender, and LGBT groups in the United States can experience racism and discrimination. This section discusses the evidence on the relationship between racism and discrimination and TRHD. Racism and discrimination can be direct but can also be subtle and elusive, making it difficult to identify these events in some situations. This section describes the research focused on racism, discrimination, and health, and discusses studies that examined the associations between racism, discrimination, and smoking. Most of this research has focused on the experiences of African Americans, although literature on this topic is emerging for other groups.

Racism, Discrimination, and Health

The influence of racism and discrimination on biological indicators of health has been investigated in many studies. Perceived racial discrimination jeopardizes the physical well-being of members of racial/ethnic minority groups.[85] For instance, perceived racial discrimination is associated with changes in blood pressure among African Americans, particularly when social support is low.[86] Fang and Myers[87] found that when African American and white men viewed video excerpts illustrating neutral, anger-provoking, and racist situations, diastolic blood pressure increased in both groups following both racist and anger-provoking stimuli. Although there was no racial difference in blood pressure, the authors suggested that cardiovascular reactivity resulting from African Americans' long-term exposure to stress-inducing racist situations might lead to health disparities. Guyll and colleagues[88] found that African American women had greater diastolic blood pressure reactivity than white women after completing a speech task in which they defended themselves against an accusation of shoplifting. African American women who perceived the accusation as racial discrimination exhibited even greater reactivity compared to those who did not. Kwate and colleagues[89] found an inverse relationship between lifetime experiences with racism and perceived health status, and a positive association between racist events and lifetime physical disease. Cruz[90] found significantly elevated salivary cortisol levels among African Americans and Hispanics who viewed videos of racist acts compared to baseline and the control group. Findings from this study suggest that racist experiences have a negative influence on stress processes, specifically physiological processes in African Americans and Hispanics.

Mechanisms Linking Racism and Discrimination to Tobacco Use

Several models have been proposed to explain the relationships between discrimination and smoking. Pascoe and Richman's model[36] suggests that mental and physical health and engagement in unhealthy behaviors contribute to the positive association between perceived racial discrimination and smoking. The stress-process model described by Pearlin and colleagues[91] suggests that individual variability in response to a stressor (especially cognitive and emotional responses) and the availability of adaptive coping resources (such as resilience, social support, and self-efficacy) determine subsequent behavioral and physical outcomes.

The prevailing perspective, however, is that the stress response to racist or discriminatory experiences is the primary factor explaining the association between racial discrimination and smoking.[92,93] It is likely that the effects of racism and discrimination vary from person to person, depending on the individual's appraisal processes. For example, Cuevas and colleagues[94] examined stress as a mediator of the relationship between discrimination and current smoking in a sample of 1,363 African American adults; they found that higher levels of perceived discrimination were associated with higher stress and

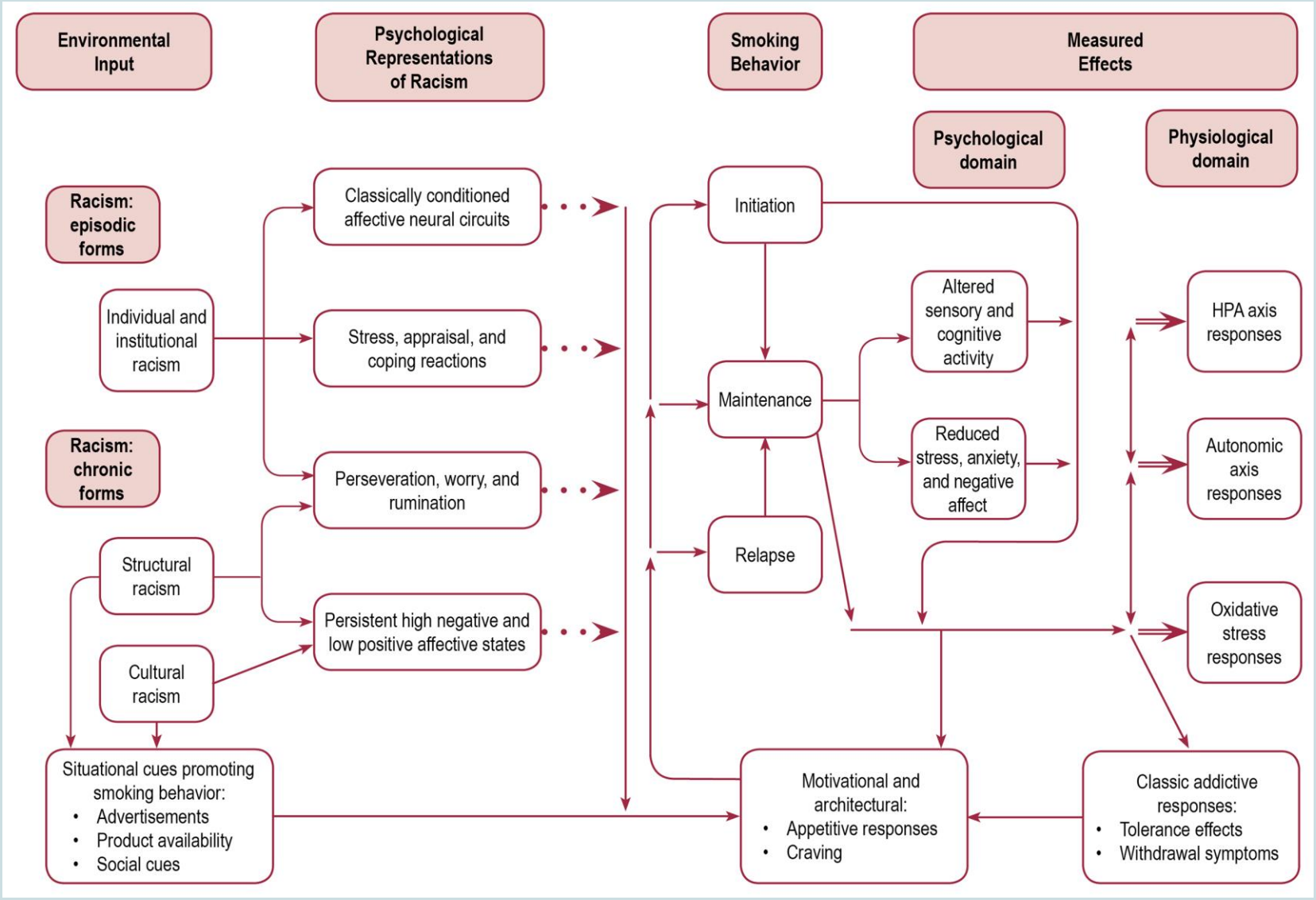
depressive symptoms, which were associated with current smoking. Consistent with the transactional model of stress,[95] this study suggests that individual appraisals of discrimination as stressful may in turn increase smoking risk. Using data from the Reactions to Race component of the 2004–2008 Behavioral Risk Factor Surveillance System (BRFSS), Purnell and colleagues[96] concluded that, across racial and ethnic groups, perceptions of discrimination increased the likelihood of current smoking, a relationship that was mediated by psychological distress. The existing research indicates that cessation and relapse prevention interventions should take into account reactions to perceived discrimination and provide adaptive coping strategies. A description of the existing literature on the relationship between racial discrimination and smoking is provided in Box 5.1.

Box 5.1: Psychological, Biological, and Environmental Effects of Racism on Smoking Behavior

The conceptual model in the figure that follows is based on the existing literature analyzing the potential relationship between smoking and racism experienced by African Americans. This model depicts the potential influence of a number of factors on smoking and health. The figure shows that smoking is influenced by the addictive properties of nicotine and its psychological effects on emotions and cognition, as well as by environmental cues. Racism contributes to a portion of the affective and cognitive processes that smoking may impact. In its chronic forms, racism through environmental cues may encourage smoking more directly. Other links on the figure connect smoking to the mitigation of negative affective states induced by racism and to the enhancement of cognitive acuity that is required by racism. Racism and smoking traverse a second common pathway illustrating their potential shared physiological impact. Double-lined arrows on the right side of the figure mark paths where smoking and racism bring about significant physiological reactions that may develop into disease states.

Some components of the pathways would benefit from further investigation. For example, the underlying neural mechanisms through which smoking and racism affect particular physiological systems are important to explore. Much is known of the neural circuits activated by nicotine when psychological tasks are performed, but little is known about how the myriad forms of racism impact the central nervous system. Further, studies are needed to determine the conditions or individuals in which the acute physiological changes associated with smoking and racism are likely to progress to chronic levels, and eventually to disease processes.

The Converging Paths of Racism and Smoking



Racism, Discrimination, and Tobacco Use

Accumulating evidence indicates that racism and discrimination are associated with smoking status (Table 5.2). This section examines the relationship between racial discrimination and smoking status among racial/ethnic and LGBT groups. Landrine and Klonoff[35] developed the Schedule of Racist Events, a self-report inventory of the frequency of lifetime and past-year racism and discrimination. This measure is correlated strongly with smoking and psychological distress. (Note: The literature search did not identify any studies examining the relationship between racial/ethnic discrimination and smoking for American Indian/Alaska Native groups or for Native Hawaiians/Other Pacific Islanders.)

Table 5.2 Summary of Studies on Racial Discrimination and Smoking Status Among Racial/Ethnic and LGBT Groups, 2000–2014

Author (year)	Population(s)	Study design	Stress constructs	Findings
African American (majority of sample)				
Bennett et al. 2005[203]	African Americans	Cross-sectional	Racial harassment	Positive association with daily smoking*
Corral and Landrine 2012[98]	African Americans	Cross-sectional	Recent racial/ethnic discrimination	Positive association with current smoking*
Cuevas et al. 2014[94]	African Americans	Cross-sectional	Daily unfair treatment/ discrimination, global perceived stress	Association between discrimination and current smoking mediated by perceived stress*
Fernander et al. 2007[53]	African American women	Cross-sectional	Race-related stress	Race-related events positively associated with global stress, which was, in turn, related to smoking*
Greene 2012[100]	African Americans	Cross-sectional, nationally representative survey	Perceived discrimination	Positive association with smoking among men,* inversely related among women
Kwate et al. 2003[89]	African American women	Cross-sectional	Past-year racism, lifetime racism	Positive associations with psychological distress*; past-year racism positively associated with number of cigarettes per day
Landrine and Klonoff 2000[93]	African Americans	Cross-sectional	Racial discrimination	Positive associations with current smoking,* frequent (vs. infrequent) discrimination,* and severity of discrimination-related stress*
Landrine and Klonoff 2000[99]	African Americans	Cross-sectional, random sampling	Racial discrimination, racial segregation	No association with smoking status; segregation positively associated with smoking prevalence*
African American and other groups				
Borrell et al. 2007[97]	African Americans, whites	Prospective	Racial discrimination	Positive associations with current tobacco use among African Americans only*
Borrell et al. 2013[37]	African Americans, whites	Prospective	Racial discrimination	Positively predicted smoking among African Americans and whites*

Table 5.2 continued

Author (year)	Population(s)	Study design	Stress constructs	Findings
Fernander et al. 2010[92]	African American pregnant women	Cross-sectional	Race-related stress, institutional race-related stress	Overall frequency and negative perceptions of race-related stress positively associated with current smoking*; no association with institutional race-related stress
Kendzor et al. 2014[38]	African Americans, Hispanics, and whites	Cross-sectional	Everyday discrimination	Positive associations with indicators of nicotine dependence,* particularly among Hispanics*
Nguyen et al. 2012[101]	Low-income, pregnant African Americans and Hispanics	Longitudinal, prospective	Ethnic discrimination	High (vs. moderate) levels of discrimination positively associated with prenatal smoking among African American women*
Wiehe et al. 2010[105]	African Americans and Hispanics, ages 12–19 years	Cross-sectional	Perceived discrimination	Positive association with current smoking among boys only*
Non-African Americans				
Todorova et al. 2010[102]	Hispanics of Puerto Rican origin	Cross-sectional	Perceived discrimination, global perceived stress	Positive association with ever-smoking*; perceived stress not a mediator of discrimination–smoking association
Chae et al. 2008[106]	Asian Americans	Cross-sectional	General unfair treatment, race/ethnicity specific discrimination	Unfair treatment positively associated with current smoking*; discrimination positively associated with current smoking,* moderated by ethnic identification*
Yoo et al. 2010[108]	Asian Americans	Cross-sectional	Racial discrimination	Positive association with current smoking*
Kam et al. 2010[204]	Hispanic youth, Mexican heritage	Cross-sectional	Perceived discrimination	Indirectly associated with substance use (cigarettes, alcohol, and marijuana composite) through increased acculturative stress*
Ornelas 2010[104]	Hispanic immigrant men	Cross-sectional	Perceived ethnic discrimination	No association with current smoking
Purnell et al. 2012[96]	U.S. multiethnic sample	Cross-sectional, nationally representative survey	Perceived discrimination	Positive association with current smoking,* mediated by psychological distress*
Tran et al. 2010[107]	African-born blacks, Southeast Asians, Hispanics	Cross-sectional	Perceived discrimination	Positively associated with current smoking in full sample,* and among Southeast Asians*

Note: No studies were found for American Indian and Alaska Native groups, Native Hawaiians or Other Pacific Islanders, and LGBT groups.
*Finding was statistically significant.

Race, Discrimination, and Smoking in African American Populations

Research evidence demonstrates an association between racial discrimination and cigarette smoking among African Americans. This robust relationship has been found across sample types, including representative national surveys, community-recruited samples, and convenience samples recruited in medical settings. Data from the CARDIA study indicate that African Americans who had experienced at least three types of discrimination had greater odds of current tobacco use (adjusted odds ratio [aOR]

1.87; 95% CI 1.18–2.96) and previous tobacco use (aOR 2.28; 95% CI 1.19–4.36) than those who had not experienced discrimination.[97] In a separate study analyzing data from the CARDIA study independent of individual and neighborhood socioeconomic conditions and segregation, self-reported discrimination positively predicted smoking and alcohol use 8–15 years later.[37]

In a sample of 2,118 African American adults, Corral and Landrine[98] also found a positive association between racial discrimination and smoking (OR 1.33; 95% CI 1.04–1.67). Because smoking status is confounded with societal status indicators, it is important to determine the independent effect of racial discrimination. In one of the few studies to adjust for social status, racial discrimination was shown to be a stronger predictor of smoking among African Americans than social status variables.[93] Smoking rates were higher among participants who reported experiencing frequent (versus infrequent) discrimination (26.7% and 6.4%, respectively), and who appraised these experiences as extremely (versus mildly) distressing (42.2% and 20.8%, respectively).[93]

In contrast, another study by Landrine and Klonoff[99] did not find a relationship between racial discrimination and smoking, although smoking was more common among African Americans in highly segregated communities than in less segregated areas. Smoking intensity has also been positively linked to past-year racist experiences.[89] A study by Greene[100] considered gender differences in the relationship between racial discrimination and smoking. Using data from the National Survey of American Life on a representative sample of adult African Americans, this researcher concluded that perceived discrimination increased the likelihood of smoking among men but not women.[100]

Racial discrimination may also influence smoking during pregnancy. In a study of pregnant African American and Hispanic women, Nguyen and colleagues[101] found that African Americans were more likely to smoke while pregnant if they had experienced a high degree of discrimination compared with a moderate amount. A study by Fernander and colleagues[92] with a sample of pregnant African American women found that the likelihood of current smoking was positively related to the emotional impact and frequency of race-related stress. In contrast, smoking status is not related to institutionalized racial discrimination (i.e., stress from policies and practices of an institution that have been intentionally based on the idea that some racial groups are superior to others).

Race, Discrimination, and Smoking in Hispanic Populations

A few studies have assessed the health consequences of ethnic discrimination among Hispanic populations. Among middle-aged Puerto Ricans, ethnic discrimination has been positively associated with perceived stress, self-reported medical diagnoses, and lifetime smoking history.[102] Kendzor and colleagues[103] found that U.S. Hispanics are more likely than non-Hispanics to report experiences of perceived discrimination related to ethnicity and national origin. In a sample of African Americans, Hispanics, and whites, these investigators also found that discrimination in everyday life was most strongly related to nicotine dependence among Hispanics. Some research, however, has not observed a relationship between perceived racial discrimination and smoking among Hispanics.[104]

Racial harassment has been associated with smoking risk among African American and Hispanic adolescents. Wiehe and colleagues[105] found that perceived racial/ethnic discrimination was associated with smoking among boys, particularly when the discrimination occurred in shops or through interactions with police. One study considering the role of discrimination and smoking cessation among Latinos over a 6.5-month period found that the frequency of major discriminatory events was inversely

associated with cessation.[38] No other studies have focused on specific stages of the tobacco use continuum.

Race, Discrimination, and Smoking in Asian American Populations

Little research has been conducted on the association between racial discrimination and smoking among Asian Americans. Chae and colleagues[106] demonstrated a positive correlation between unfair treatment or discrimination and current smoking among Asian Americans. Strong ethnic identity emerged as a protective factor for people with more discrimination experiences. Tran and colleagues[107] assessed perceived discrimination in a multiethnic sample and found a positive association between discrimination and current smoking among Southeast Asian immigrants to the United States. Being treated as a foreigner was positively correlated with tobacco use among Asian Americans, according to a study by Yoo and colleagues.[108]

Race, Discrimination, and Smoking in LGBT Populations

Smoking prevalence is higher among LGBT individuals than among heterosexuals.[80,109] The LGBT population experiences uniquely stressful circumstances, including more frequent discrimination compared to heterosexuals,[109] but little research has specifically investigated the role of sexual identity–based discrimination on smoking behavior. Johns and colleagues[81] found that discrimination was unrelated to smoking status, yet discriminatory events increased the likelihood of daily smoking versus smoking on some days. Physical violence toward LGBT individuals is a form of discrimination and increases the likelihood of current smoking.[110] Research has not considered how the intersection of race/ethnicity and LGBT identification influences the stress–smoking association and TRHD.

Psychological Disorders and TRHD

This section examines other psychosocial factors, such as psychological disorders and trauma, and their effect on TRHD.

Psychological Disorders and Minority Racial/Ethnic and LGBT Groups

The links between a variety of psychological disorders and tobacco use are well established at each point along the tobacco use continuum for both adolescents and adults,[43,111–115] but there is less evidence on their association with TRHD. Prevalence of smoking and higher rates of smoking are associated with psychological disorders such as depressed mood, anxiety disorders, PTSD, attention deficit disorder, alcohol disorders, and disruptive behavior disorders. Lifetime psychopathology symptoms are associated with smoking initiation (e.g., Rohde et al. 2003[116]), and individuals with psychiatric symptoms and disorders are more likely to proceed along the tobacco use uptake continuum[117,118] and become nicotine dependent.[119] Symptoms of psychopathology are also correlated with difficulties in quitting, particularly in smokers with comorbid depressive symptoms or disorders or comorbid substance use.[120–123]

To understand whether the presence of these disorders plays a role in TRHD, several questions need to be addressed: Do disadvantaged groups vary in the prevalence of these disorders in a way that might explain behavioral differences in tobacco use patterns or in health outcomes? Might the presence of these disorders in disadvantaged groups impair access to treatment for smoking? Are disadvantaged groups differentially hindered by psychological disorders or symptoms (e.g., depression or depressive

symptoms) in their attempts to stop smoking? Are some disorders more likely to bring about a cascade of negative physiological effects?

Patterns of psychiatric morbidity across racial/ethnic groups have been examined with appropriate controls and samples. Using data from the National Comorbidity Study, along with appropriate consideration of the role of SES, Breslau and colleagues[124] found that both Hispanics and African Americans have a lower lifetime risk of psychiatric disorders than non-Hispanic whites, but those who do have a disorder tend to have more persistent illnesses. Williams and colleagues[125] also found that chronicity of major depressive disorder is higher for both African Americans and Caribbean blacks compared to whites, suggesting that the burden of mental disorders may be higher among blacks than whites in the United States.

Using data from the U.S. National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), two studies[126,127] also found that the prevalence of mental health disorders varies significantly by racial/ethnic group, as do patterns of comorbidities, although these differences did not necessarily replicate those found by Breslau and colleagues.[124] Most notably, Huang and colleagues[126] found significantly greater rates of alcohol use disorders, drug use disorders, mood disorders, anxiety disorders, and personality disorders among American Indians compared with other race/ethnic groups in the U.S. population. Asian Americans had the lowest rates of mood, anxiety, and substance use disorders. Disparities in comorbidities do not necessarily mirror disparities in tobacco use prevalence rates across racial/ethnic groups, further complicating the picture of understanding racial/ethnic patterns of disorders.[126]

It is also important to note that large epidemiological surveys of mental health disorders rarely include formal psychiatric diagnostic interviews, but tend to reflect a combination of symptoms that map onto diagnostic criteria that together are highly suggestive of formal disorders; as such, they are often labeled as the disorder in the investigations addressing psychological disorders or symptomatology and smoking. It may be more appropriate to consider that the literature reflects links between the tobacco use continuum and psychological symptoms rather formal diagnostic assessments.

The finding that African Americans have lower rates of depression than whites may appear contrary to expectations, given their higher rate of exposure to stressful events and discrimination. One potential explanation for this discrepancy is that blacks engage in unhealthy behaviors, such as tobacco or alcohol use, to cope with stress[128] rather than experience depression. Keyes and colleagues[129] tested the hypothesis that the black–white depression paradox is due to the protective effects of unhealthy behaviors (alcohol consumption, smoking, and high body mass index) at high stress levels. Using longitudinal, nationally representative data from NESARC, these researchers found that African Americans were less likely than whites to smoke cigarettes at low, moderate, or high levels of stress, and that the stress pathways to depression did not operate differentially by race.

The evidence for LGBT groups having higher rates of mental disorders is much stronger and more consistent than that for racial/ethnic minority groups. In a meta-analysis, King and colleagues[130] found that lesbian, gay, and bisexual people are at high risk of mental disorders, suicidal ideation, substance misuse, and self-harm than heterosexuals. Similarly, a study of a community sample of 246 LGBT youths ages 16–20 years old, found that one-third of the youth met criteria for any mental disorder, a higher prevalence than that found among youths in national samples.[131]

Persistence of mental health disorders may place individuals at greater risk for continued smoking and increased difficulty quitting. Studies examining relationships between mental disorders, tobacco use, and cessation for specific population groups tend to echo the general finding that mental illnesses may be a hindrance to cessation, but there is a relative paucity of studies specifically examining how mental disorders influence smoking cessation among minorities or disadvantaged groups. Hickman and colleagues,[132] analyzing data from a nationally representative sample of black adults participating in the National Survey of American Life, found recent mental illness (past year and past month) was associated with lower odds of cessation. Castro and colleagues[133] also found that depressive symptoms predicted lower cessation rates for both whites and African Americans, but there was no relationship between depression and cessation for Latinos.

One way in which mental disorders might influence TRHD is through access to care. Racial/ethnic minority groups have more barriers to mental health care than non-minority groups do[134,135] and are more likely to underuse mental health services.[136,137] Disparities in mental health care may leave minority groups more vulnerable to tobacco use as a way of coping with mental health symptoms and may also translate into barriers to treatment for smoking cessation.

Trauma and Post-Traumatic Stress Disorder in Minority Racial/Ethnic and LGBT Groups

Studies have investigated the relationship between exposure to trauma and other adverse life events and TRHD. PTSD is a psychiatric condition characterized by exposure to trauma (e.g., events such as natural disasters, war, violence, and abuse) and subsequent symptoms of re-experiencing, avoidance, emotional numbing, and physiological hyperarousal.[138] Whether defined by race, ethnicity, sexual orientation, or SES, a person's demographic status may be an important factor in the risk of experiencing exposure to trauma and PTSD, and together, these factors may be associated with TRHD. Racial minority status is associated with higher rates of exposure to certain types of traumatic events, such as community violence[139] and violence in the household.[140] A meta-analysis across racial groups found that low SES, but not racial/ethnic minority status, was associated with increased rates of PTSD.[141] Table 5.3 summarizes some of the research conducted in this area.

Table 5.3 Studies Examining Stress/Trauma and Tobacco Use Among Racial/Ethnic Groups, 2003–2012

Author (year)	Population(s)	Study design	Stress construct	Findings
Beaudoin 2011[163]	African Americans	Cross-sectional	PTSD	Positive association with current smoking*
Dickerson et al. 2009[157]	American Indians	Cross-sectional	PTSD	Positive association with lifetime nicotine dependence*
Flory et al. 2009[162]	African Americans, whites, others	Cross-sectional	Psychosocial stress, PTSD symptoms	Stress positively associated with current smoking*; nicotine dependence positively associated with PTSD symptoms*
Jessup et al. 2012[151]	Women of African American, American Indian, Asian/Pacific Islander, Hispanic, white, and mixed ethnicity	Cross-sectional	PTSD	Positive association with current smoking*

Table 5.3 continued

Author (year)	Population(s)	Study design	Stress construct	Findings
Lopez et al. 2011[154]	Pregnant women, African Americans	Cross-sectional	Lifetime and current PTSD symptoms and diagnosis	Positive association with current smoking*
Sawchuk et al. 2012[205]	American Indians	Descriptive, cross-sectional	PTSD	Positive association with lifetime smokeless tobacco use among Northern Plains vs. Southwest Tribal members*
Stephens et al. 2010[155]	African Americans, whites, American Indians, Asian/Pacific Islanders, Hispanics, others	Cross-sectional	PTSD symptoms	No association with tobacco use in full sample; positive association with tobacco use among African Americans, American Indians, and Asian Americans*
Weaver and Etzel 2003[159]	African Americans, whites, Hispanics, others	Cross-sectional	PTSD, trauma severity	No association with smoking status; positive association with nicotine dependence*

Note: PTSD = post-traumatic stress disorder.
*Finding was statistically significant.

Exposure to traumatic events in childhood may differentially affect minority youth and lead to early tobacco use. A study using data from the National Survey of Adolescents found that African American and Hispanic youth were at greater risk for developing PTSD than white youth.[142] In a 12-year longitudinal study following 585 children (82% European American, 16% African American, 2% other) from pre-kindergarten to grade 11, childhood physical maltreatment (e.g., physical abuse) was associated with more school absences, suspensions, and behavioral problems.[143] The authors note that their study provides support for the persistent and long-term effects of early physical maltreatment. Although this study did not measure tobacco use as an individual outcome variable, or find evidence for differential effects of trauma on substance abuse more generally, the results suggest that early trauma exposure could lead to TRHD by impacting some populations more than others. In another study, which focused on a predominantly (85%) African American sample of adolescent girls, both the number of lifetime traumas and levels of PTSD symptoms independently predicted current smoking status, such that a greater number of traumas and/or more severe PTSD were associated with a greater likelihood of smoking.[144]

In a comprehensive study examining the relationship between adverse childhood experiences and different aspects of smoking behavior along the tobacco use continuum, and adjusting for age, sex, race, and education, Anda and colleagues[145] found a strong and cumulative relationship between adverse childhood experiences and smoking behavior. Compared with adults who reported no adverse childhood experiences, adults who reported five or more categories of adverse events (emotional abuse; physical abuse; sexual abuse; a battered mother; parental separation or divorce; growing up with a substance-abusing household member, mentally ill household member, or incarcerated household member) had significantly higher risks of early smoking initiation (OR 5.4; 95% CI 4.1–7.1), ever smoking (OR 3.1; 95% CI 2.6–3.8), current smoking (OR 2.1; 95% CI 1.6–2.7), and heavy smoking (OR 2.8; 95% CI 1.9–4.2).[145]

Two other meta-analyses have also found that prevalence rates of tobacco use were lowest among people with no trauma exposure; elevated among persons with trauma exposure but without PTSD; and highest among individuals with both trauma exposure and PTSD.[146,147] Moreover, trauma and PTSD were most clearly implicated in the initiation and continuation—as opposed to cessation—of tobacco use.[146] Roberts and colleagues[148] examined the relation between self-reported exposure to various types of trauma and smoking behaviors in the National Longitudinal Study of Adolescent to Adult Health (Add Health study), a population-based, longitudinal study of more than 15,000 adolescents followed into young adulthood. The study found that reported past-year exposure to trauma was associated with up to a two-fold risk of regular smoking in the past year (exposure to physical assault: OR 1.91; 95% CI 1.25–2.92; exposure to interpersonal violence: OR 1.46; 95% CI 1.01–1.07).[148]

Additional insights come from studies exploring smoking behaviors after trauma exposure. In a large sample of New York City public high school students, black and Hispanic students were less likely than whites to report increases in smoking 6 months following the World Trade Center attacks in September 2001.[149] Although demonstrating that post-trauma tobacco use trajectories may differ across racial groups, this study did not utilize a non-trauma-exposed control group. Among a sample of sexual assault victims, racial minority adults were less likely to have a heavy and increasing smoking trajectory compared to whites,[150] but this study also lacked a non-traumatized control group, so the changes in behavior may not necessarily reflect responses to trauma. Despite methodological limitations, these studies of changes in tobacco use behaviors can provide important information about longitudinal changes in smoking in response to traumatic events.

In a study of the relationship between smoking and PTSD in an ethnically diverse sample of women recruited from health clinics, mental health agencies, substance abuse treatment centers, and senior communities, Jessup and colleagues[151] found smoking rates of 58% among African Americans, 56% among American Indians, and 53% among bisexual women. Smoking was positively related to PTSD, although the interaction with race/ethnicity was not reported. In a study of residents of Central Harlem in New York City, current smokers (87% black; 55% female) reported having experienced greater lifetime exposure to traumatic events than nonsmokers.[152] Amos and colleagues[153] studied a sample of African American college males and found that those who reported past sexual abuse in or around campus were more likely to report tobacco use in the past 30 days. Lopez and colleagues[154] reported that African American pregnant women who smoked were significantly more likely to have a current or past PTSD diagnosis than women who did not smoke and were not African American. Stephens and colleagues[155] concluded that tobacco use was not an independent predictor of PTSD symptoms in acutely injured trauma center inpatients, but African American and Native American trauma survivors had a higher prevalence of tobacco use than whites.

In a sample of Native American adults, rates of current smoking were higher among individuals with PTSD than those without it.[156] Dickerson and colleagues[157] found a positive relationship between lifetime nicotine dependence and PTSD among American Indian male veterans. Importantly, when comparing rates of smoking between trauma/PTSD groups, these studies did not control for other risk behaviors: They did not examine whether trauma or PTSD was associated with increases in other health risk behaviors, such as other substance or alcohol use, in addition to smoking.

A relationship between trauma and current smoking has also been found for LGBT groups. Gay, lesbian, and bisexual young adults from a large epidemiological sample who reported having been victims of violence were more likely to smoke than those who reported they had not been victimized.[110]

Victimization was also associated with smoking among heterosexuals, indicating that the relationship between violence exposure and smoking was not specific to LGBT groups. However, compared to the heterosexual participants, LGBT individuals reported higher levels of both smoking and victimization. The authors conclude that "risks for elevated smoking may stem from stressful events like discrimination and victimization, which sexual minority populations experience at disparately higher rates their heterosexual peers. As found in other studies, it is not simply being gay, but the stressful events…and resulting emotional or psychological distress that can be predictive of cigarette smoking."[110,p.1291-92]

In addition, associations between trauma or PTSD and level of nicotine dependence have been found. In a community sample of adult smokers (over half of whom were African American), Thorndike and colleagues[158] found that levels of nicotine dependence were positively associated with levels of PTSD symptoms among men but not among women. One limitation of this study is that it did not examine the relationship by race. These findings that report differences by sex contrast with previous work that showed that both violence exposure and PTSD symptoms were positively associated with nicotine dependence among a predominantly African American sample of severely battered female smokers.[159] Thorndike and colleagues[158] noted that their own sample might have experienced less severe trauma, possibly showing a weaker relationship between trauma/PTSD and smoking. This finding highlights the importance of considering trauma severity when making comparisons between traumatized and non-traumatized groups.

Very few studies have examined the relationship between trauma/PTSD and smoking cessation among minority groups. The pattern of PTSD symptoms and their recurrent nature may, however, place newly abstinent smokers at greater risk for relapse. Zvolensky and colleagues[160] found that smokers with a history of PTSD had a higher risk of early relapse than smokers without psychiatric problems, but this study's sample was too small and non-diverse to lead to any conclusions about whether the PTSD–relapse risk is similar in different minority groups.

Studies of the relationship between PTSD and tobacco use might benefit from an examination of types of PTSD symptoms. Among a sample of 66 adult Bosnian war refugees presenting for primary health care services, current smokers reported higher levels of PTSD hyperarousal, but not re-experiencing, avoidance, or numbing.[161] Hyperarousal symptoms were also associated with greater nicotine dependence in this sample.

Flory and colleagues[162] found a high rate of smoking (53%) (as well as high rates of alcohol consumption and hazardous/harmful alcohol use) among adult survivors of Hurricane Katrina. Current smoking was associated with psychosocial stressors, and nicotine dependence was associated with PTSD symptoms. Most participants in the study were African American (57%), but analyses by race/ethnic group were not reported. In a study examining post-disaster trends in alcohol and tobacco use among African Americans in New Orleans, Beaudoin[163] found comparable levels of cigarette smoking before and after Hurricane Katrina; however, logistic regression analysis found that PTSD was significantly associated with cigarette smoking (OR 1.78; 95% CI 1.05–3.44)

The existing literature suggests that PTSD is related to the prevalence of smoking, smoking intensity, and dependence among racially/ethnically diverse smokers. No studies on this subject have included representative proportions of Hispanics, Asian Americans, or LGBT groups, and none have examined differences in smoking across racial/ethnic groups. Because these traumatic processes are associated

with socioeconomic indicators—due to displacement, for example—additional empirical studies are needed to examine how these factors may contribute to TRHD.

Examining Specific Psychological Stress, Trauma, and Smoking: Women and Intimate Partner Violence

The final section in this chapter focuses on a specific category of stress, trauma, and violence: intimate partner violence (IPV) experienced by women. The Centers for Disease Control and Prevention (CDC) defines IPV as "physical violence, sexual violence, stalking and psychological aggression (including coercive tactics) by a current or former intimate partner," which can include one's spouse or former spouse, current or former partner, boyfriend, or girlfriend, or dating partner, whether heterosexual, lesbian, bisexual, gay, or transgendered.[164,p.11] The United Nations' 1993 Declaration on the Elimination of Violence Against Women defines violence against women generally as "any act of gender-based violence that results in, or is likely to result in, physical, sexual or psychological harm or suffering to women, including threats of such acts, coercion or arbitrary deprivation of liberty, whether occurring in public or private life."[165,Article 1] Article 2 of this declaration refers more specifically to IPV, stating that "violence against women shall be understood to encompass . . . physical, sexual and psychological violence occurring in the family."[165,Article 2]

This section describes the relationship between IPV and the tobacco use continuum, using evidence from the literature. Types of IPV are described in Box 5.2. Although both men and women can experience IPV, this chapter focuses on women because they are the subject of most of the available literature on this topic.

Box 5.2: Four Types of Intimate Partner Violence

There are four main types of IPV: physical violence, psychological/emotional violence, sexual violence, and stalking.[164]

Physical violence is the intentional use of physical force with the potential for causing death, disability, injury, or harm. Physical violence includes, but is not limited to, scratching, pushing, shoving, throwing, grabbing, biting, choking, shaking, slapping, punching, burning, use of a weapon, and use of restraints or one's body, size, or strength against another person.

Psychological/emotional violence involves trauma to the victim caused by acts, threats of acts, or coercive tactics. Psychological/emotional abuse can include, but is not limited to, humiliating the victim, controlling what the victim can and cannot do, withholding information from the victim, deliberately acting to make the victim feel diminished or embarrassed, isolating the victim from friends and family, denying the victim access to money or other basic resources, and threats of physical or sexual violence (using words, gestures, or weapons to communicate the intent to cause physical harm, injury, or death).

Sexual violence is divided into three categories: (1) use of physical force to compel a person to engage in a sexual act against his or her will, whether or not the act is completed; (2) an attempted or completed sex act involving a person who is unable to understand the nature of the act, to decline participation, or to communicate unwillingness to engage in the sexual act because of intimidation or pressure, illness, disability, or the influence of alcohol or other drugs; or (3) abusive sexual contact, including unwanted touching or fondling.

Stalking generally refers to harassing or threatening behavior that an individual engages in repeatedly, such as following a person, appearing at the person's home or place of business, making harassing phone calls, leaving written messages or objects, or vandalizing personal property.[206]

Estimates of the prevalence of IPV in the United States vary significantly, in part because of under-reporting and differences in definitions and data collection methods. The CDC estimates that as of 2010, more than 35% of women and 28% of men have experienced physical violence, sexual violence, or stalking by an intimate partner at some time in their lives.[166] Estimates from the national- and state-level National Intimate Partner and Sexual Violence Survey (NISVS) for different types of IPV (including psychological/emotional violence) are presented in Table 5.4. For women, estimates of the prevalence of types of IPV range from 6% to 64%, depending on the specific population; data from the NISVS suggest that individuals who are multiracial or American Indian/Alaska Native are more likely to suffer from multiple forms of IPV than whites, blacks, Hispanics, or Asian or Pacific Islander populations.[167] Approximately half of the women who experience IPV are physically injured by their partners, and most sustain multiple injuries.[168]

Table 5.4 Weighted Prevalence of Type of Intimate Partner Violence for Men and Women During Their Lifetimes and in Past 12 Months, 2011

	Women		Men	
Type of IPV	Lifetime (%, 95% CI)	Past 12 months (%, 95% CI)	Lifetime (%, 95% CI)	Past 12 months (%, 95% CI)
Physical violence	31.5 (29.9–33.2)	4.0 (3.2–4.8)	27.5 (25.8–29.3)	4.8 (4.0–5.8)
Psychological/emotional violence	47.1 (45.3–48.8)	14.2 (12.9–15.5)	46.5 (44.6–48.4)	18.0 (16.5–19.6)
Sexual violence (not including rape)	15.8 (14.6–17.1)	2.1 (1.6–2.6)	9.5 (8.4–10.8)	2.1 (1.7–2.7)
Sexual violence (rape only)	8.8 (7.8–9.8)	0.8 (0.5–1.2)	0.5 (0.3–0.8)	N/A
Stalking	9.2 (8.2–10.3)	2.4 (1.9–3.0)	2.5 (1.9–3.3)	0.8 (0.6–1.2)

Notes: IPV = Intimate partner violence. CI = Confidence interval. N/A = Not applicable.
Source: Breiding et al. 2014.[167]

IPV has been found to occur in all regions of the world among all socioeconomic, religious, and cultural groups.[169] In a 10-country study of women's health and domestic violence, between 15% and 71% of women reported experiencing physical or sexual violence by a partner.[170] Justifications for IPV are similar around the world, and include disobeying or arguing with the man, not being a good wife (not caring adequately for him, the children, or the home; refusing sex), and suspected infidelity. While IPV is a universal phenomenon, women may be more vulnerable to IPV in cultures with social norms that enforce gender roles, particularly male dominance over women, and where inequalities between men and women are pronounced.[171]

Smoking and smoking-attributable disease rates are lower among women than men, but women more often experience IPV. As described in chapter 2, although the overall prevalence of smoking among women in 2015 was 13.6%, prevalence varied by race and ethnicity: 24.0% of American Indian/Alaskan Natives, 16.0% of whites, 13.3% of African Americans, 7.1% of Hispanics, and 2.6% of Asians.[172] In an

analysis of data from the 1999 Rhode Island BRFSS, women who reported having experienced IPV had higher rates of cigarette smoking than those who did not report having experienced IPV. The study authors note that the cross-sectional design of their study did not allow for causality to be inferred.[173]

Significant evidence has demonstrated that IPV poses a serious threat to public health, with impacts reaching beyond the initial occurrence. Women who have been victims of IPV report lower quality of life in terms of their mental and physical health. They have increased rates of stress compared with women who have not experienced violence; studies have suggested that IPV should be treated as a chronic disease.[174,175]

Research has linked the high levels of stress by women who have experienced IPV to increased rates of substance abuse.[174,176] Women who have experienced psychological, physical, or sexual abuse are consistently more likely than non-abused women to smoke.[177,178] Researchers have also suggested that smoking is used as a coping mechanism, albeit maladaptive, for stress associated with IPV, relationship distress, and traumatic events.[174,179,180]

IPV has the potential to interfere with women's efforts to stop smoking. In general, research has found that the health behaviors of romantic partners influence one another.[181] Systematic reviews acknowledge the potential importance of partner support for promoting smoking cessation,[182,183] but this line of inquiry does not appear to consider whether women are currently experiencing IPV. The relationship dynamics in couples with IPV may mean that traditional smoking cessation goals, such as establishing a smoke-free household or simply refusing to smoke with a partner, could lead to physical harm. These findings highlight the importance of understanding the relationship between tobacco use and IPV. Evidence on this relationship and how it affects subsequent health outcomes may be valuable when developing tobacco prevention and cessation interventions targeted toward women and others at risk of experiencing IPV. Additionally, women with a history of experiencing IPV or other trauma may require more-focused interventions and alternative coping resources and strategies to replace smoking.

To inform future research, a description of data sources that may be used to explore the relationship between IPV, the tobacco use continuum, and TRHD is provided in Table 5.5.

Table 5.5 Data Sources on Intimate Partner Violence and Smoking

Data source (sponsoring organization)	Focus of survey	Questions on IPV	Questions on smoking status
Behavioral Risk Factor Surveillance System (CDC) http://www.cdc.gov/brfss	Health conditions and risk behaviors in the United States	8-question module on sexual violence 7-question module on IPV	Tobacco and smoking cessation (since 2005) questions included annually
Pregnancy Risk Assessment Monitoring System (CDC) http://www.cdc.gov/prams	U.S. state-specific, population-based data on maternal attitudes and experiences around pregnancy	Questions on physical abuse during and after pregnancy	Questions on tobacco use during pregnancy

Table 5.5 continued

Data source	Focus of survey	Questions on IPV	Questions on smoking status
Youth Risk Behavior Surveillance System (CDC) http://www.cdc.gov/HealthyYouth/yrbs/index.htm	Health risk behaviors that contribute to death and disability among middle and high school students in the United States	Questions around teen dating abuse 1) Have you ever been physically forced to have sexual intercourse when you did not want to? 2) During the past 12 months, how many times did someone you were dating or going out with physically hurt you on purpose? 3) During the past 12 months, how many times did someone you were dating or going out with force you to do sexual things that you did not want to do?	Includes eight questions on smoking behaviors including age of first smoke, smoking frequency, and quitting smoking
National Longitudinal Study of Adolescent to Adult Health NICHD http://www.cpc.unc.edu/projects/addhealth	Causes of health-related behaviors of U.S. adolescents in grades 7–12 and the outcomes into young adulthood	Questions on IPV included during Wave 3	Includes nine questions on smoking behaviors including age at first cigarette, smoking frequency, quitting smoking, and peer smoking
Multi-Country Study on Women's Health and Domestic Violence Against Women (WHO) http://www.who.int/gender/violence/who_multicountry_study/en	Information on IPV and its association with women's physical, mental, sexual, and reproductive health in 10 countries	Detailed questions regarding IPV	Two questions on smoking, including current smoking behavior and lifetime smoking behavior

Notes: IPV = Intimate partner violence. CDC = Centers for Disease Control and Prevention. NICHD = National Institute of Child Health and Human Development. WHO = World Health Organization.

Intimate Partner Violence and the Tobacco Use Continuum

Intimate Partner Violence and Current Smoking

Although studies have examined smoking and IPV in different populations and with different measures, research has consistently found that adult women who have experienced psychological, physical, or sexual abuse were more likely to smoke than non-abused women.[173,177,178,184–187] Studies of the association between smoking and IPV in adolescents have produced inconsistent results.[188–190] Exner-Cortens and colleagues[190] did not identify an association between teen dating violence and smoking, while Silverman and colleagues[189] identified a strong association. Both studies relied on large national data sets.

In a meta-analysis of 31 peer-reviewed publications (with a total of 271,192 study participants, of whom 95% were women), Crane and colleagues[174] found that those who had experienced IPV were significantly more likely to smoke than those who had not (d = 0.41; 95% CI 0.35–0.47). Moderators in this study included pregnancy status, relationship type, ethnicity, and SES. Previous studies have suggested that the association between IPV and smoking is stronger among pregnant women than in the non-pregnant IPV population; the meta-analysis detected a trend toward significance based on pregnancy status.[174] Crane and colleagues[174] found no significant differences in the strength of the victim–smoking relationship in regard to relationship type, SES, or ethnicity. Other studies found that

pregnant women who had experienced IPV (before and/or during pregnancy) were more likely to have smoked during their pregnancy than those who had not experienced IPV.[191–193]

A meta-analysis by Caleyachetty and colleagues[194] evaluated the degree of association between IPV and smoking using data on women of reproductive age (15–49 years; n = 231,892) from the Demographic and Health Surveys administered in 29 low- and middle-income countries. Smoking rates in this population ranged from 0.1% to 16.7%, with a rate higher than 5% in 31% of the countries surveyed. Domestic violence prevalence ranged from 8.7% to 62.5%; the rate exceeded 20% in 62% of the surveyed countries. Just under half of the countries involved in the study were found to have a statistically significant association between exposure to IPV and smoking after controlling for age, education, occupation, household wealth, religion, and pregnancy status. When the data were pooled across countries, the relationship between IPV and current smoking was significant (aOR 1.58; 95% CI 1.38–1.79); this association was found to be moderately consistent across the 29 countries involved in the study (I^2 = 55.3%, p < 0.0001).[194]

Several other notable studies that addressed the IPV–smoking association were not included in the 2013 meta-analysis by Crane and colleagues. Gerber and colleagues[185] looked at the rates of IPV among smokers and nonsmokers in the Boston area. This cross-sectional study surveyed 2,386 women in eight health care settings and found that (1) women who reported no smoking or drinking had a 10% probability of having experienced IPV in the previous year and a 30% probability of having experienced IPV in their lifetimes; (2) women who reported smoking had a 14% past-year probability and a 49% lifetime probability; and (3) women who reported both smoking and drinking had a past-year probability of 27% and a lifetime probability of 54%. These relationships were found to be statistically significant after adjusting for age, race, education, marital status, health care site, and depression.

Jun and colleagues[184] examined the relationship between IPV and smoking in the context of psychological abuse with or without the co-occurrence of physical and sexual abuse in a sample of nurses in the United States. Using data from the Nurses' Health Study II (n = 54,200), these researchers found that women experiencing only psychological abuse were 33% more likely (95% CI 13%–57%) to smoke than non-abused women. Women who reported psychological abuse with a single occurrence of physical or sexual abuse were 50% more likely to be smokers (95% CI 30%–80%) than non-abused women, and those with repeat co-occurrences of physical or sexual abuse were found to be 90% more likely (95% CI 70%–230%) to be smokers.

The largest study (as of 2014) of the association between smoking and IPV analyzed data from the BRFSS on 42,566 women and 27,590 men.[195] Of women who had not experienced IPV, 14.9% were smokers (95% CI 14.1%–15.6%), whereas 33.8% of females who had experienced IPV reported smoking (95% CI 32.2%–35.4%). Among males, 19.9% of non-victims were smokers (95% CI 18.9%%–20.9%); in contrast, 36.5% of those who experienced IPV were smokers (95% CI 33.8%–39.1%). After controlling for age, race/ethnicity, income, and education, women who had experienced IPV were found to be 2.3 times more likely to smoke (95% CI 2.07–2.54) than non-victims, and males who experienced IPV were 1.92 times more likely be smokers (95% CI 1.66–2.23) than non-victims.

The previously mentioned study by Jessup and colleagues[151] found similar results. The 997 study participants included 322 smokers and 675 nonsmokers; 65.1% of the smokers had experienced IPV compared to 31.3% of the nonsmokers (p < 0.001).

As of November 2014, only one longitudinal study has investigated teen dating violence victimization and adverse health outcomes, including smoking.[190] This is an important area of focus because studies of teen dating violence have found that 20% of teens report psychological violence victimization, and between 0.8% and 12% report any physical violence victimization.[190,196–198] Exner-Cortens and colleagues[190] analyzed data from the National Longitudinal Study of Adolescent Health on a population of 5,681 middle and high school students (ages 12–18) who completed three waves of surveys over an 8-year period. Controlling for sociodemographics, child maltreatment, and pubertal status, the study found students who reported teen dating violence victimization at wave 2 were more likely than non-victims to smoke when interviewed at wave 3 (aOR 1.53; 95% CI 1.13–2.06).

Additional evidence is available on the relationship between smoking and other psychosocial factors that can in turn be associated with IPV. For example, one study found that a cumulative psychosocial index, which included indicators related to family relationships, negative life events, financial stressors, and problematic alcohol use, had a significant relationship with likelihood of smoking in women in their 60s (aOR = 1.53; 95% CI 1.30–1.81).[199] Another study found an association between smoking rates among LGBT individuals and psychosocial factors such as perceived parent and partner support, psychological distress, and victimization related to LGBT lifestyle factors.[200]

Several studies have made recommendations about screening and interventions with the IPV population that could reduce future smoking-related illnesses.[174,194] It remains unclear whether IPV leads to stress and poor health outcomes such as smoking, or if high levels of stress could potentially trigger IPV.[177]

Intimate Partner Violence and Frequency and Intensity of Tobacco Use

A 2011 study by Ashare and colleagues[201] examined the relationship of smoking and PTSD in individuals who had experienced IPV based on the theory that smoking rates among IPV victims would be higher because they would be self-medicating due to PTSD. These researchers followed a group of 83 women who had experienced IPV and found smoking expectancies and PTSD symptoms to be correlated with the number of cigarettes smoked per day by women in this group. No other studies that examined the relationship between IPV and smoking frequency and intensity were found in the published literature.

Intimate Partner Violence and Tobacco Cessation and Relapse

Studies of an association between IPV and tobacco cessation and relapse are limited, and the researchers were unable to identify any peer-reviewed studies. Most studies that examined the association between IPV and tobacco use concluded by discussing the need to adapt tobacco cessation programs for this population. One study that used a convenience sample of women arrested for IPV perpetration found that 62% of the women smoked, and 65% of those expressed a desire to quit,[202] but there was no discussion of barriers to smoking cessation.

Chapter Summary

This chapter reviews studies on the relationships between stress-related processes, psychological disorders, trauma, and intimate partner violence and how these are related to smoking and TRHD across racial/ethnic, gender, and sexual orientation groups.

Research on the effects of stress in other areas of health suggests that physiological indicators of stress may differ for African Americans compared with whites. However, no evidence links physiological processes, such as HPA axis and sympathetic responses, to TRHD, and no studies have specifically examined differences between smokers across racial/ethnic groups. Thus, the role of physiological stress in TRHD remains an empirical question.

The literature reviewed does offer evidence of an association between perceived stress and smoking in racial/ethnic and LGBT populations. Most of these studies involved low-income African Americans; the evidence is less clear for other racial/ethnic groups and those with better financial resources. The preponderance of the research on perceived stress and current smoking consistently demonstrates a positive association. A few studies also found a positive association between perceived stress and smoking intensity; one study found stress to be a factor in smoking relapse following a smoking cessation attempt.

The few studies that have examined stress–smoking relationships among Hispanics provide an indication of an association. Only one study focused on Native Americans/Alaska Natives. Studies of LGBT groups suggest that current smoking is positively related to stress involving sexual orientation, although the data about this population were limited.

The literature, although scant for several racial/ethnic groups, suggests that racism and discrimination play a role in tobacco smoking. As hypothesized earlier, discrimination is one of the factors that may increase the risk of unhealthy behaviors such as smoking, decrease the chances of quitting, and ultimately increase health risk. Studies of a potential association between racial discrimination and current smoking were primarily cross-sectional and focused on African Americans. The data indicate that perceived racial discrimination (recent and past), race-related stress, and daily hassles are associated with current smoking. Specifically, the risk of current smoking increases with racist and discriminatory experiences, and perceived stress may mediate the association between racial discrimination and smoking. Race-related distress may be a factor contributing to the higher rate of smoking initiation among African American adults compared with white adults. The few studies focused on Hispanics provide some evidence of an association between discrimination and smoking status, although more research is needed. The data are scant concerning Asian Americans, and no studies included samples of American Indians/Alaska Natives or examined the role of racial discrimination in relation to aspects of the tobacco use continuum other than current smoking.

The evidence suggests that blacks, Hispanics, and Asians have a lower prevalence of some psychological disorders, and that American Indians tend to have higher rates. Members of LGBT groups are at higher risk of psychological disorders, substance use disorders, suicide, and self-harm compared with heterosexuals.[130] Persistent psychological disorders may confer greater risk for continued smoking. Evidence for an association between psychological symptoms or disorders and tobacco use varies along the tobacco use continuum and by population. In general, there is strong evidence that tobacco use is associated with psychological symptoms (especially mood disorders and other substance use disorders) and influences all points along the tobacco use continuum, notably for escalation of use and failure to quit. However, this evidence is strongest and most consistent for whites, and less consistent for other racial/ethnic groups. The lack of consistency in findings on the relationship between tobacco use and psychological symptoms and disorders for other groups may be a function of smaller sample sizes and fewer longitudinal studies. An association between mood symptoms and tobacco use has good theoretical plausibility, given the mood management properties of nicotine. It is not yet clear whether

the associations between psychological symptoms related to mood disorders and tobacco use vary by race/ethnicity or other groups.

The association between trauma/PTSD and tobacco use has such a strong theoretical potential to explain disparities that it may be useful to consider trauma/PTSD separately from other psychological disorders. There is good evidence for a relationship between trauma/PTSD and tobacco use in minority samples, and some minority groups are more likely to be exposed to traumatic events, but the literature on trauma/PTSD and tobacco use in minority groups is relatively sparse and is primarily limited to cross-sectional studies. As a result, the available evidence does not extend far beyond the basic observation that trauma exposure or PTSD may contribute to TRHD. Although the general literature supports a causal relationship in which trauma/PTSD lead to tobacco use, this relationship has yet to be demonstrated across a variety of minority groups. Perhaps most importantly, few studies have examined minority group differences in the strength of the association between trauma/PTSD and smoking. Some minority groups are exposed to more traumatic experiences yet smoke at lower rates, and among these groups, trauma and PTSD may be less strongly associated with smoking. Protective factors related to minority status may buffer the relationship between trauma/PTSD and tobacco use. Nevertheless, the role of trauma/PTSD in TRHD will remain unclear until further studies examine these associations across minority groups.

An association between smoking and IPV against women is supported by the available evidence, but few studies have investigated the relationship between IPV and TRHD, and most have not analyzed differences by race/ethnicity and other factors. Almost all studies have focused on IPV broadly rather than how specific types might increase the risk of smoking; women frequently experience overlapping types of IPV (e.g., physical, sexual, psychological, stalking, and coercive control), and the combined effects of these experiences are difficult to measure and analyze in relation to smoking. Further, few studies have measured the duration and intensity of women's experience with IPV and their perceptions of its impact on their lives, or have looked for a dose–response relationship between IPV and adverse health outcomes. The literature review did not find information on interventions that considered IPV as a factor affecting smoking cessation. Understanding partner dynamics, including partner smoking behaviors, may be useful to helping women with histories of IPV quit smoking.

In conclusion, the existing evidence indicates that stress plays a role in current smoking and maintenance of smoking behavior. To the extent that stress processes are exacerbated in minority populations, the potential result is an increased risk of adverse health consequences. Cessation rates may be improved by interventions that include stress management techniques with demonstrated efficacy, and by acknowledging the stress-inducing structural barriers faced by these populations.

Research Needs

This review identified significant gaps in the literature regarding the relationship of stress and TRHD in minority racial/ethnic, gender, and LGBT groups. The literature search conducted for this section found that most studies included different racial/ethnic groups in their samples but did not examine effects within specific groups. Few studies examined the relationship between stress, trauma, and TRHD among LGBT groups.

An improved understanding of potential links between stress, trauma, and psychological processes and the tobacco use continuum may provide more insight into TRHD. These relationships may not be

straightforward or direct, and the field would benefit from more specific attention to clarifying potential mechanisms that may differ by subpopulation. In addition, research should examine the implications of the relationships between stress, trauma, and psychological processes in order to improve tobacco control policies and practices aimed at reducing TRHD.

Links between psychosocial processes, tobacco use at all points along the continuum, and population differences may help explain TRHD. Understanding these links is challenging and requires clear, theoretically driven hypotheses, strong measurement of constructs, and large samples. To date, much of the evidence has been based on post hoc secondary analyses. It is important that considerations of population differences be built into study designs from the onset. Additionally, harmonizing variables and constructs across existing studies would allow samples to be pooled, resulting in more opportunities to explore potential explanations of population differences. Finally, involving key stakeholders, including representatives of diverse populations, in all stages of the research process, from conceptualization and measurement of constructs to interpretation of results, will enrich the understanding of TRHD.

More research is needed that focuses on stress processes in populations that experience disparities across the entire tobacco use continuum. The literature on sympathetic nervous system and HPA axis responses to stress among racial/ethnic and LGBT smokers is quite limited. For example, no published studies examine stress–response physiological patterns in diverse groups of smokers and report outcomes by group. Alterations in neural circuitry and attenuated responses to stress, acute and chronic nicotine exposure, and smoking cessation have implications for health and possibly TRHD. Existing research on these issues is limited to understanding relationships with smoking status (current smoking and, in some cases, past smoking). More research is needed about the impact of stress processes on smoking frequency, intensity, cessation, relapse, morbidity, and mortality across racial/ethnic and LGBT populations.

Increasing the understanding of mechanistic contributors, mediators, and moderators of the stress–smoking relationship is also important. Little is known about the factors that may explain the association between stress and current smoking in racial/ethnic minority populations. There is some suggestion that global and unique stressors are explanatory factors, and that perception of stress and acculturative stress also contribute to smoking behavior. However, theoretically grounded investigations with hypotheses based on established or new models are needed to explain how stress impacts tobacco use and TRHD. Many of the risk factors for increased stress, such as low SES and racial discrimination, are difficult to disentangle from one another.

To date, most studies of perceived racial discrimination and smoking have focused on African Americans. Future research should consider the impact of racism and discrimination on tobacco use across other racial/ethnic groups.

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Section III
Interpersonal and Contextual Factors That Contribute to Tobacco-Related Health Disparities

Chapter 6
Social Relationships and Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

The link between social relationships and health outcomes is well established.[1–6] Prospective studies in diverse cultural settings have shown that people who are integrated into supportive social networks are at reduced risk of all-cause mortality and disease-specific mortality[1,3–5] and have fewer biomarkers of disease.[4] Although social relationships can influence health outcomes via several pathways, health behaviors (including tobacco use) represent a major mediating mechanism through which these influences can occur. Social relationships are relevant at all stages of the tobacco use continuum, the causal pathway in the progression of smoking to disease which includes initiation, current use and intensity, intentions to quit and quit attempts, cessation, relapse, and tobacco-related morbidity and mortality. They influence the risk of early experimentation with tobacco and progression to higher levels of tobacco use, as well as the likelihood of successful smoking cessation.[7] Although numerous studies have shown strong associations between social relationships and health outcomes and have identified tobacco use as a significant mediator of those associations, few of these studies have focused on the role of social relationships in creating or exacerbating disparities.

Several review articles have summarized the cross-sectional and longitudinal associations between aspects of social relationships—including structural aspects such as social network structure, and functional aspects such as social influence, social control, and social support—and various tobacco-related behaviors such as early experimentation with smoking, progression to nicotine dependence, and smoking cessation.[8–20] However, these reviews have not focused on the associations between social relationships and tobacco-related behaviors and variations across sociodemographic groups (i.e., racial/ethnic groups, socioeconomic status [SES] groups, and sexual orientation groups). Because social relationships are so closely linked to tobacco use in general, it is likely that they are also involved to some extent with the development and maintenance of tobacco-related health disparities (TRHD). This chapter reviews the evidence on how social relationships can create or exacerbate TRHD across racial/ethnic groups, SES groups, and sexual orientation groups.

Social Relationships and Disparities Across the Tobacco Use Continuum

The broad term "social relationships" encompasses both *structural* and *functional* characteristics of an individual's social network.[21] The structural aspect represents the person's position in a social network, including the number of ties with other people in the network, the strength of those ties, and interconnections among those ties. The functional aspect represents the social interactions that occur across those ties. The nature, source, amount, and relative importance of structural and functional characteristics likely vary across racial/ethnic groups, SES groups, and sexual orientation groups. This chapter will review how structural and functional characteristics of the social network can influence TRHD.

Structural Characteristics of Social Relationships

Structural characteristics of social relationships include measures of social integration, such as the number of social ties from an individual to other individuals and groups and the interconnections among those ties. People with numerous, densely connected social ties are considered highly socially integrated, popular, or central. People with few or no social ties are considered socially isolated.[22]

Structural aspects of social networks have been shown to influence tobacco use behavior across the tobacco use continuum and throughout the life course. Studies of adolescents have found that popular

students and, conversely, socially isolated students, are at increased risk of smoking.[22] Social isolation also has been associated with smoking among middle-aged adults[23,24] and older adults.[25] The contagion effects of smoking (i.e., smoking behavior spreading like a virus from one person to another) have been documented using social network analyses. Research indicates that smoking behaviors can be spread through close and distant social ties and that smoking initiation and cessation patterns typically occur at the same time among interconnected groups of people.[26,27]

Functional Characteristics of Social Relationships

The functional mechanisms by which social relationships influence health can be divided into three broad categories: (1) social influence and social comparison, (2) social control, and (3) social support.[21]

The first category, *social influence and social comparison*, refers to the process by which people adjust their own behavior to conform with the behavior of others. People make social comparisons with similar others to obtain guidance about which behaviors are normative and which behaviors are likely to be socially reinforced.[28] Although social influence and social comparison processes could operate at any point in the tobacco use continuum, they can be especially relevant during the early stages of smoking uptake. Smoking initiation typically occurs during adolescence and young adulthood, when conformity to peer norms and acceptance by peers are especially salient. Through social learning processes, nonsmokers observe their friends and family members receiving or not receiving social, physical, or emotional reinforcement after smoking; if such reinforcement occurs they may then emulate the smoking behaviors with the expectation of receiving the same reinforcement. Although any member of an individual's social network can exert social influences to smoke or not to smoke, social influences typically originate from close relationships with admired and/or similar individuals.[29]

After smoking has become a behavior that is maintained by physiological dependence on nicotine, social influence and social comparison processes might become less important. However, these processes could still influence other smoking-related behaviors, such as the types or brands of tobacco used (e.g., menthol cigarettes, dual use of cigarettes and other tobacco products), the settings in which smoking occurs, and the likelihood and timing of cessation attempts. Smokers might look to their peers, people they admire, or the idealized images in cigarette advertisements to decide which cigarette brands are consistent with the self-image they wish to project.[30] Although numerous studies have documented social influences on tobacco use, a smaller subset of those studies has focused on social influences as a contributor to TRHD.

The second category, *social control*, refers to explicit attempts by social network members to encourage people to practice healthy behaviors.[31] Social control is a mechanism to influence the individual to engage in normative, non-deviant behavior; in the health behavior context, social control is viewed as a mechanism to encourage healthy (normative, non-deviant) behavior.[31] Social control of smoking can be a direct (e.g., a spouse or child convincing a smoker to quit) or indirect (e.g., a smoker autonomously deciding to quit to be able to fulfill obligations to family members).[32] Early in smoking uptake, social control can occur when parents forbid their children from smoking or establish no-smoking rules in the home. At later stages of use, social control can occur when social network members encourage the smoker to quit or to refrain from exposing others to secondhand smoke. Social control generally moves people in the direction of performing healthier behaviors, although it can have the opposite effect if it is perceived as overly intrusive and creates reactance.[21]

The third category, *social support,* can be subdivided into emotional support (e.g., caring, empathy, commitment), informational support (providing information about resources and services), and instrumental support (performing actions that facilitate the individual's behavior change) to promote health and well-being.[21] Social support may be especially helpful to disadvantaged populations in facilitating smoking cessation[33]; social network members can provide encouragement and empathy during the quit attempt (emotional support); offer information about smoking cessation strategies and resources (informational support); and/or perform actions that facilitate cessation efforts, such as driving the smoker to appointments or purchasing pharmacological cessation aids (instrumental support).

In addition to the functional aspects of social relationships described by Thoits,[21] this chapter also focuses on *discrimination* as a social influence. Discrimination encompasses a variety of negative social interactions experienced by disadvantaged and minority populations. It refers to differential treatment based on one's membership in a minority or disadvantaged group. Discrimination includes overt acts, such as name-calling, violence, harassment, or discourteous treatment, as well as more subtle microaggressions, such as speaking in a manner that implies that a person is uneducated, unintelligent, or untrustworthy.[34,35]

Discrimination is included as a social influence in this chapter because numerous studies have identified associations between discrimination and smoking among adults[36–45] and youth.[46–48] Discrimination is far more common among members of disadvantaged groups; the prevailing perspective is that the stress reaction to discriminatory experiences primarily explains its association with smoking among these groups.[42,49] Further discussions of the associations between discrimination and smoking among adults are presented in chapter 5.

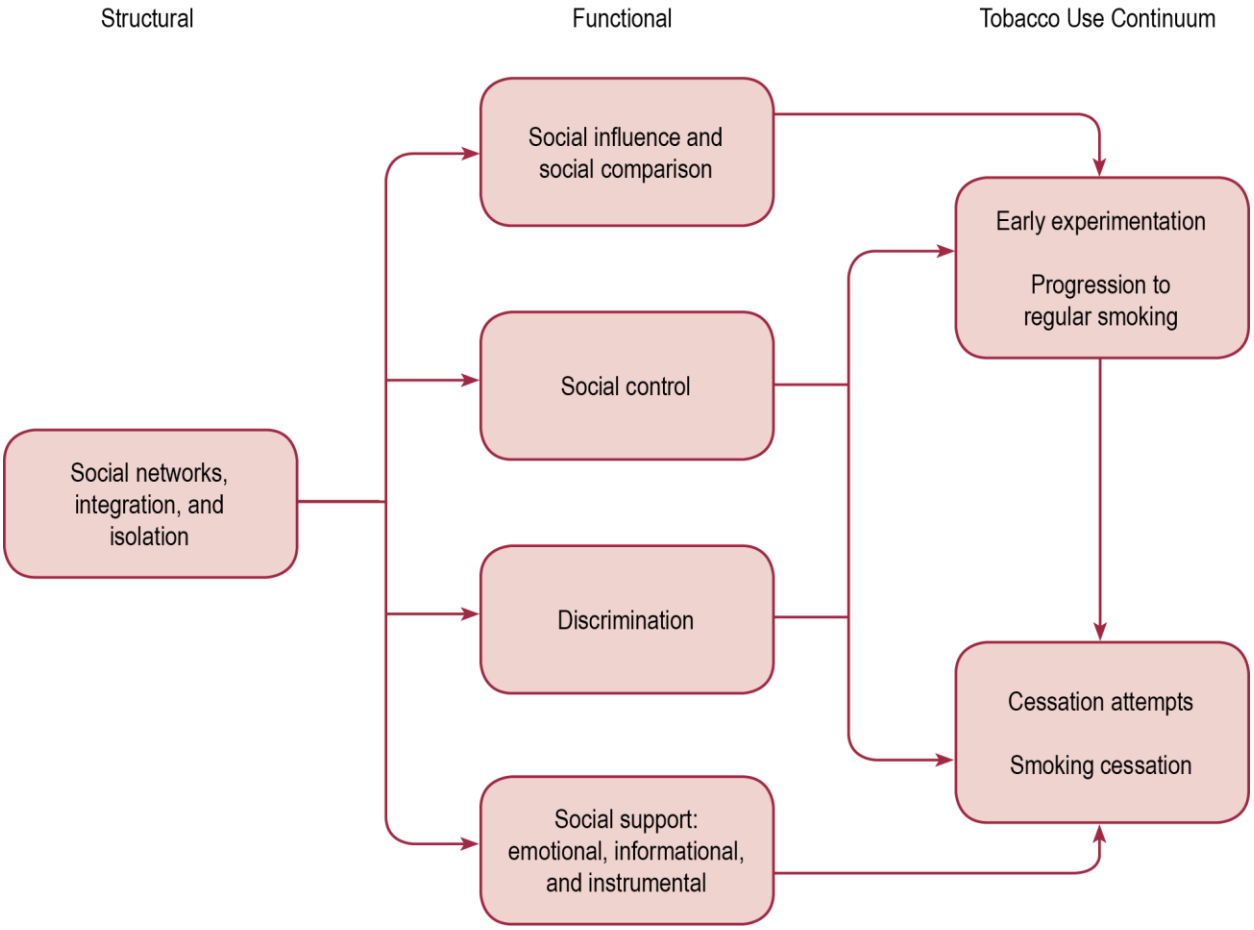
Discrimination could be conceptualized as a structural characteristic of social relationships rather than as a functional characteristic, because discrimination can cause specific individuals to be excluded from social networks, preventing them from receiving social influences, social control, or social support from those networks. However, this chapter conceptualizes discrimination as a functional characteristic because most of the studies of discrimination and tobacco use have examined associations between an individual's perceptions of discrimination and his or her tobacco use behaviors, without considering the potential mediating influences of exposure to social networks. This is an interesting direction for future research, but the pathway from discrimination to social network structure to tobacco use is likely to be difficult to disentangle.

The Tobacco Use Continuum and Social Relationship Characteristics

In general, research has focused on the social relationship factors thought to be most relevant for particular stages of the tobacco use continuum. For example, nearly all the research on social relationships and early experimentation has focused on social influence and social comparison processes; little research has focused on social support as a predictor of experimentation. Conversely, nearly all the research on social relationships and smoking cessation has focused on social support and social control; little smoking cessation research has focused on social influence and social comparison. Discrimination has been examined in relation to lifetime and recent smoking among adolescents and in relation to current smoking among adults, but its role in the other stages of the tobacco use continuum has not been thoroughly addressed. The available research becomes even more limited when it is restricted to studies that also examine disparities.

This chapter focuses primarily on the pathways between social relationships and behaviors along the tobacco use continuum, as shown in Figure 6.1.

Figure 6.1 Influences of Social Relationships Across the Tobacco Use Continuum



Social Relationships and TRHD: Two Types of Mechanisms

Causal mechanisms by which social relationships can give rise to TRHD can be seen as falling into two broad categories: (1) simple differences in the prevalence of risk and protective factors and (2) moderator effects.

Simple differences across groups in the prevalence of risk and protective factors can lead directly to differences in the prevalence of smoking. For example, if members of one racial/ethnic group, SES group, or sexual orientation group experience more social influence that supports smoking than members of another group, the group experiencing more social influence to smoke would be expected to engage in more tobacco use, which would lead to TRHD. If another group receives less social support to quit smoking, that group might have fewer quit attempts. This approach assumes that the effect of a given social interaction on smoking behavior is similar across groups, which may or may not be true. Moderation effects make it possible to test this assumption.

Moderation effects occur when the effects of a given risk factor are stronger among one group than among another group. If one group is more susceptible to a given risk factor, and another group is more resilient to the same risk factor, the risk factor will result in more smoking among the susceptible group than among the resilient group. For example, if a specific group is especially vulnerable to social influences to smoke, the same level of social influence would cause more smoking among that group than among another group that is more resilient to social influences. The most rigorous test of such a moderation effect would be a study that assesses statistical interactions between social influences and group membership in the prediction of tobacco-related outcomes. For example, a study of the effect of family support on smoking cessation outcomes could recruit smokers of diverse racial/ethnic and SES groups and directly test whether the strength of the association between family support and smoking cessation is significantly different across groups. However, to date, most published research has focused on a single demographic group (e.g., moderate-income African Americans) rather than comparing across racial/ethnic and SES groups. This chapter reviews studies that concentrate on single groups, but it focuses on studies that make explicit comparisons across groups, because those studies are most informative about disparities.

Measures of Social Relationships and Tobacco Use

The methods for measuring social relationships vary widely across studies; this fact makes comparisons across studies difficult and precludes a formal meta-analysis. This section describes the measures of structural aspects of social relationships (social networks) and functional aspects of social relationships (social influence and social comparison, social control, social support, and discrimination) that have been used most frequently in studies of tobacco use and TRHD.

Social Network Structure

Formal social network analysis techniques[50] can be used to diagram an individual's social network, quantify his or her position in the social network and connections with other members of the network, and examine similarities in behavior among members who share social ties.[51] In this social network approach, researchers typically collect data from a closed system of individuals (e.g., classroom, school, workplace) and survey all members of the system about their smoking and their social relationships with other members of the system. For example, school-based studies[22] have asked students to report their smoking behavior and list the other students in the school who are their friends. The matrix of nominations can be used to calculate the number of friends each student nominated, number of friend nominations each student received, and the structure of interconnections among the friends. The friends' self-reports of their own smoking behavior also can be used to examine each student's exposure to smoking peers and the diffusion of smoking behavior throughout the social network.

One advantage of this method is that it removes the problem of respondents misperceiving their peers' behavior, because the social influence data are obtained from the peers themselves. Although this method can yield more accurate data on the peers' behavior, it omits the possible social influences from people not nominated by the respondent as friends and from people who were nominated as friends but did not provide data. It also omits influences from social network members who are outside the closed system under study (e.g., friends who attend different schools).

Social Influence and Social Comparison

Social influence is typically defined operationally in tobacco use survey research as the number of people in the respondent's social network who smoke or approve of smoking. The most practical way to assess social influence in a survey is simply to ask the respondents how many of their friends or family members smoke. For example, the National Youth Tobacco Survey (NYTS)[52] asks, "How many of your four closest friends smoke cigarettes?" and the National Longitudinal Study of Adolescent to Adult Health (Add Health)[53] asks, "Of your 3 best friends, how many smoke at least 1 cigarette a day?" Some adolescent surveys also ask whether the respondent's parents and/or siblings smoke. Adult surveys typically ask how many of the respondent's household members and friends smoke. For example, the International Tobacco Control Survey[54] asks, "Does your partner or spouse currently smoke?" The National Adult Tobacco Survey[55] and several statewide tobacco surveys ask, "How many of your friends use any tobacco products?"[56]

These measures are based on an assumption that mere exposure to people who smoke is sufficient to influence the respondent's smoking behavior. They do not assess other theoretically important aspects of the social learning process, such as whether the respondent witnessed the smoker receiving reinforcement for smoking[29] or whether the respondent is motivated to comply with perceived social norms.[57]

Social Control

Social control can include a variety of behavioral attempts to constrain an individual's smoking behavior. The nature of the social control likely depends on the relationship between the smoker and the person exerting the social control. A parent might exert social control on a child by forbidding or punishing the child's smoking. A child might exert social influence on a parent by asking the parent not to smoke. If the relationship is between spouses, friends, or people of equal status, the social control might involve advising or pressuring the person to quit smoking.

Measures of adolescents' perceptions of social control against smoking typically ask about their parents' rules about smoking. For example, the NYTS[52] asks, "Which statement best describes the rules about smoking inside your home? Smoking is not allowed anywhere inside my home. Smoking is allowed in some places or at some times. Smoking is allowed anywhere in my home. There are no rules about smoking in my home." Measures of social control among adults also include questions about rules against smoking in the home or workplace and questions about whether the respondent was asked not to smoke. For example, the California Adult Tobacco Survey includes the question, "About how many times in the past 12 months has anyone asked you not to smoke when you were smoking or were about to smoke?"[56]

Social Support

Numerous measures of social support exist, and several comprehensive articles and books describe their theoretical perspectives and psychometric properties. Some studies of social support and smoking have measured general social support, and others have measured social support specific to an individual's efforts to quit smoking. General social support scales typically include measures of frequency of social interaction, the perceived availability of support and help in times of crisis, satisfaction with social support, membership in social groups, and the availability of confidants.[58–60] Measures of social support specific to smoking cessation[61–63] ask whether a spouse, partner, or other social network member

performed behaviors that were supportive (e.g., complimented you on not smoking, congratulated you for your decision to quit smoking) or unsupportive (e.g., criticized your smoking, commented on your lack of willpower). In addition to the specific actions the partner performed, it is also important to assess the extent to which the smoker perceived those actions as helpful or unhelpful.[64] The social support literature has drawn a strong distinction between received support (occurrence of specific supportive actions) and perceived support (perceptions of the availability of support)[65]; however, this distinction has rarely been discussed in the literature on social support for smoking cessation.

Discrimination

Although many different survey measures have been used to assess discrimination,[66] most studies of discrimination and smoking focus on individuals' perceptions of everyday hassles and negative social interactions that are based on the individuals' membership in one or more minority groups. For example, the Everyday Discrimination Scale[67] asks about such experiences as being treated with less courtesy or respect and receiving poorer service at restaurants. The Behavioral Risk Factor Surveillance System Reactions to Race module assesses perceived discrimination by asking whether the individual was treated worse than, the same as, or better than people of other races while seeking health care and being in the workplace. When using these measures, it is important to remember that individuals likely differ in the extent to which they attribute these events to discrimination or to other factors unrelated to their minority status. For example, some people will attribute a waiter's poor service to discrimination, whereas others will attribute it to the waiter's incompetence or workload. More research is needed to determine how individuals' personalities, past experiences, and social contexts influence the extent to which they attribute negative experiences to discrimination.

Literature Search Strategy

A literature search was conducted in PubMed, Google Scholar, and PsycINFO® to gather research on the role of social relationships in TRHD. Key search terms included smoking, tobacco, or cigarette; minority, disparity, race, racial, ethnic, ethnicity, socioeconomic, income, gay, lesbian, bisexual, transgender, LGBT, homosexual, heterosexual, or sexual orientation; Hispanic, Latino, African American, black, or Asian; social, peer, friend, sibling, spouse, parent, family, discrimination, or social network.

Searches were limited to studies conducted with U.S. samples, published in English through 2011, and involving humans. The searches yielded 3,498 articles. An examination of the abstracts of these articles revealed 442 potentially relevant articles, which were then examined for potential inclusion in this chapter. Articles were included if they addressed disparities across racial/ethnic, income, or sexual orientation groups regarding the association between social relationships and smoking. A total of 84 studies met this criterion.

Social Network Structure and Smoking

Adolescents

Although several studies have found that popular adolescents, those with numerous, densely connected social ties, are more likely to smoke,[27,68] studies have also found that socially isolated adolescents are more likely to smoke.[69,70] Other studies have found complex curvilinear associations between popularity and smoking[71] or complex interactions between popularity and other measures of social network

position.[72] Although several studies have examined associations between social networks and smoking, most of those studies have not assessed disparities in those associations. A recent review of 10 school-based studies of social networks and adolescent smoking concluded that adolescents who are socially isolated are at increased risk for smoking.[70] However, this review did not address disparities concerning the association between social isolation and smoking, and most of the studies reviewed were conducted among predominantly white, average-SES samples. The few social network studies that focused on vulnerable populations are reviewed below.

A study of a multiethnic sample of middle school students in Los Angeles (56% Hispanic, 27% Asian American, and 23% white)[27] found that the positive association between popularity and smoking was significant among Hispanic students but not among other racial/ethnic groups. When the sample was stratified by ethnicity and gender, popularity was a significant predictor of susceptibility to smoking only among Hispanic girls. However, because Hispanics represented more than half of the sample, there may not have been sufficient statistical power to detect associations between popularity and smoking among the other racial/ethnic groups.

One study[73] assessed social network influences on smoking among a predominantly Hispanic sample of students attending an alternative high school. Although low-SES students are overrepresented at alternative schools, students attend alternative schools for numerous reasons (e.g., disciplinary problems, pregnancy, work schedules that preclude attendance at traditional schools), so not all students in this study were in the low-SES group. In this study, 54% of the students had mothers with less than a high school education, suggesting they are from low-SES households. In this sample, 40% of students reported past-month smoking, and the most consistent predictor of past-month smoking was in-degree centrality (the number of other students who nominated the respondent as a friend, which is an indicator of popularity). These findings suggest that the association between popularity and past-month smoking may generalize to low-SES Hispanic students in a high-risk social context.

A study of Hispanic 8th-grade students at a single low-SES middle school in Los Angeles found that students who spoke Spanish with more of their social network members were less likely to have social network members who used substances, and lower substance use among the social network members was associated with lower substance use among the respondents.[74] However, this study used a composite measure of tobacco, alcohol, and other drugs, so it is not clear whether social network characteristics were specifically associated with tobacco use in this sample. In addition, the significant predictor of substance use in this study was the number of Spanish-speaking friends, not the number of friends overall, which suggests that acculturation could have confounded or moderated the association between social networks and substance use. Acculturation is discussed in more detail in chapter 7.

A large longitudinal study found that among white and African American adolescents, those who were socially isolated in 7th grade were at increased risk of being smokers in 11th grade.[69] The association between social isolation and smoking was similar among whites and African Americans. However, the exact nature of this association is difficult to determine because social isolation was defined as a lack of several different types of social support, including satisfaction with the level of popularity and other related constructs.

Adults

Most of the research on sociometric position and smoking has focused on adolescents. The few studies that have examined this association among adults have not examined disparities or minority populations. For example, an analysis of the Framingham Heart Study data from 1971 to 2000[26] showed that connected clusters of smokers within a large social network tended to quit smoking around the same time. However, the Framingham sample was predominantly white and middle class, so this study does not provide information about disparities, and few other studies of smoking among adults include assessments of entire social networks. A study of changes in smokers' social networks after a quit attempt found that quitting was associated with a shift to a larger social network and to less contact with and exposure to smokers; the sample was predominantly white (83%) and majority female (58%), so differences by race/ethnicity and other demographic factors could not be determined.[75] Similarly, no studies have examined differences across racial/ethnic or lesbian, gay, bisexual, and transgender (LGBT) groups in the association between sociometric position and smoking behavior among adults.

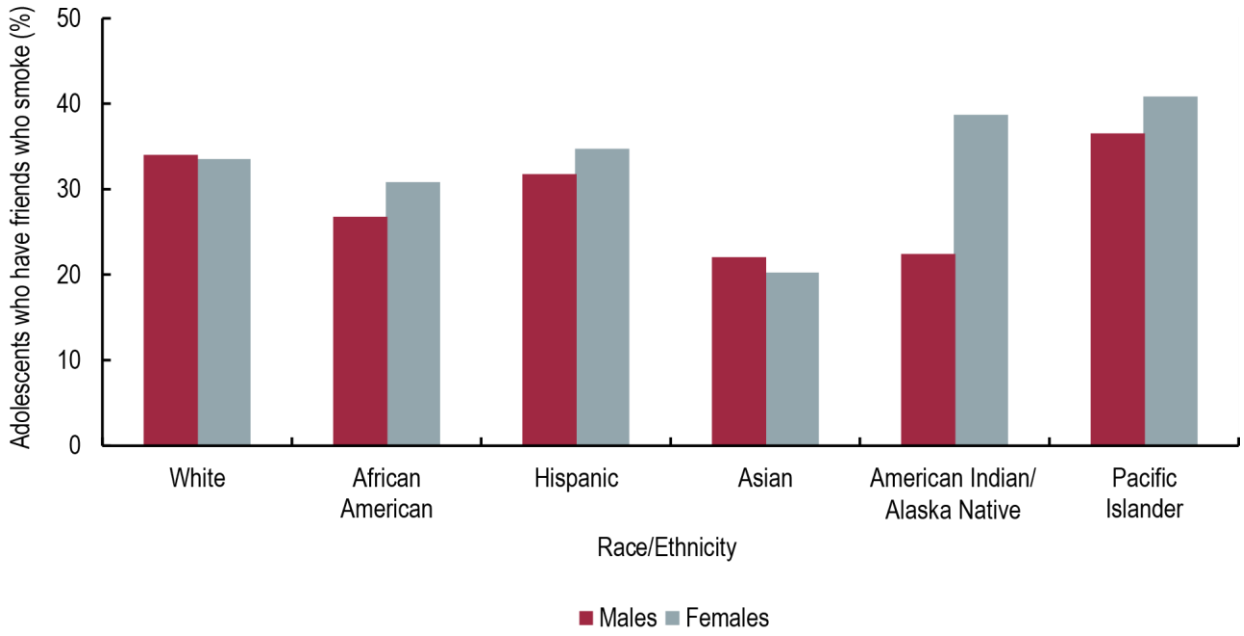
Social Influence, Social Comparison, and Smoking

Adolescents

Disparities in the Prevalence of Peer Influences on Smoking

Various studies have provided data on the proportion of adolescents who report that they have friends who smoke; however, many are not nationally representative. Figure 6.2 shows nationally representative estimates from the 2013 NYTS of the proportion of adolescents who have one or more friends who smoke.[52] The proportion of adolescents who had at least one friend who smoked varied across racial/ethnic groups. Pacific Islander adolescents were most likely to have a friend who smoked (38%), followed by whites (34%), Hispanics (33%), African Americans (29%), American Indian/Alaska Natives (28%), and Asians (21%). Information is not available on the proportion of adolescents, by sexual orientation or SES, who have friends who smoke.

Figure 6.2 Percentage of Adolescents Who Report Having One or More Friends Who Smoke, by Race/Ethnicity and Gender, 2013



Source: Centers for Disease Control and Prevention 2013.[52]

Disparities in the Prevalence of Family Social Influences

Although nationally representative data are not available on how many adolescents report having parents or other family members who smoke, data are available on how many adolescents live with a smoker. In the 2013 NYTS,[52] American Indians/Alaska Natives were most likely to report that they lived with a smoker (35%). The proportion of adolescents who lived with a smoker was lower among other racial/ethnic groups (whites: 33%; African Americans: 29%; Hispanics: 28%; and Asians: 25%), despite the fact that the prevalence of smoking among adults varies more across these racial/ethnic groups.[76] Data are not available by sexual orientation.

Disparities in the Strength of Peer Influences on Smoking Initiation and Progression

The literature presents conflicting evidence regarding racial/ethnic differences in the effect of friends' smoking on adolescents' experimental smoking behaviors. Several studies have examined associations between friends' smoking and adolescent smoking in multicultural samples. The vast majority of these studies have found that friends' smoking is a very strong predictor of smoking initiation and progression across all racial/ethnic groups studied.[77–81] These studies did not report significant differences across racial/ethnic groups in the strength of the association between friends' smoking and respondents' smoking.

Other studies have found differences across racial/ethnic groups in the strength of peer influences on adolescent smoking. Landrine and colleagues[82] found that peer smoking was a strong predictor of smoking among white adolescents, accounting for 23.5% of the variance, but it explained a much smaller proportion of the variance in smoking among Hispanic, Asian American, and African American adolescents. An analysis of the Add Health Wave III data[83] on participants whose average age was 21.8 years found that friends' smoking was associated with ever-smoking and nicotine dependence

among white young adults (odds ratio [OR] 1.97; 95% confidence interval [CI] 1.60–2.40) and Hispanic young adults (OR 2.12; 95% CI 1.30–3.50) but not among African American young adults. In a large sample of California adolescents, whites were more likely than Chinese Americans to initiate smoking if their friends smoked, although the effect was significant for both.[84] In a longitudinal study of the predictors of change in smoking status from 7th to 12th grades,[69] friends' smoking was a risk factor for progression from never-smoking to monthly smoking among whites and African Americans; however, African Americans were at risk for smoking if only a few of their friends smoked, whereas whites were at risk for smoking if most of their friends smoked. Several other studies have found that the association between friends' smoking and adolescent smoking is stronger among whites than among African Americans.[81,85,86]

Studies also have examined racial/ethnic differences in the association between perceptions of peer norms and adolescent smoking. Perceptions of peer norms represent a more generalized, albeit more speculative, measure of social influence. These measures ask adolescents to estimate the percentage of their peers who smoke, their perceptions of the pressure to smoke, or their perceptions of the acceptability of smoking among their general peer group. Analyses of the National Longitudinal Survey of Youth (NLSY) data[87] found that perceived peer pressure to smoke was a risk factor for smoking among whites (OR 20.4; $p \leq 0.001$) and Hispanics (OR 6.1; $p \leq 0.05$) but not among African Americans. However, analyses of the Add Health data[88] found that perceived peer pressure to smoke predicted smoking initiation equally among white, African American, and Hispanic adolescents. Siddiqui and colleagues[89] found that the association between peer approval of smoking and adolescent smoking was stronger among whites than among African Americans, Hispanics, and Asians. A study of adolescents from four Asian American groups in California[90] found that the association between perceived peer norms about smoking and smoking behavior was significant and consistent across Asian groups and genders.

Prior summary reports, including both the 1994 Surgeon General's report, *Preventing Tobacco Use Among Young People*,[91] and the 2012 Surgeon General's report, *Preventing Tobacco Use Among Youth and Young Adults*,[7] have concluded that adolescents are more likely to smoke if they have friends who smoke. For example, *Preventing Tobacco Use Among Youth and Young Adults* concluded that "the evidence is sufficient to conclude that there is a causal relationship between peer group social influences and the initiation and maintenance of smoking behaviors during adolescence."[7,p.460] While the causal mechanisms are not fully understood, the association is likely due to a combination of peer influence (i.e., adolescents smoking because their friends exert informational or normative influences for them to smoke), and peer selection (i.e., adolescents selecting friends with similar smoking behaviors).[11,16]

Several studies have found that peer influence effects are stronger among whites than among most racial/ethnic minority groups. African American adolescents, in particular, appear to be affected less strongly by peer influence to smoke. However, there is inconsistency across studies regarding racial/ethnic differences in the effects of peer influence. No studies were identified that examined differences in peer effects on adolescent smoking across sexual orientation or SES groups.

Parental Influences on Adolescent Smoking

Studies have reported both similarities and differences across racial/ethnic groups in the strength of the association between parents' smoking and adolescent smoking. An analysis of the Add Health Wave I and II data[81] found that parents' smoking was a risk factor for transition from ever-smoking to daily

smoking among whites, African Americans, and Hispanics over a 1-year period; however, there were no significant ethnic differences in this association, and parents' smoking did not predict smoking initiation among never-smokers. A later analysis of the Add Health Wave III data (participants' mean age was 21.8 years)[83] found that parents' smoking was a risk factor for lifetime nicotine dependence among whites (OR 1.89; 95% CI 1.43–2.49) and Hispanics (OR 2.02; 95% CI 1.02–4.02) but not among African Americans. However, parents' smoking was not a significant predictor of ever-smoking in this sample. Another analysis of the Add Health Wave II data that focused on mothers' influences on girls[92] found that mothers' smoking was a risk factor for adolescent girls' smoking among whites but not among Hispanics or African Americans. Similarly, an analysis of data from the 1992 NLSY[87] found that mothers' smoking was associated with greater adolescent lifetime smoking among whites but not among African Americans or Hispanics. Other analyses of large, nationally representative samples of adolescents[88,93] and smaller, more in-depth studies of geographically localized samples[78,94] have found that the positive association between parents' smoking and adolescents' smoking was similar across racial/ethnic groups.

Most studies have not had sufficient statistical power to include Asian Americans as a separate category in analyses of racial/ethnic differences as predictors of smoking. An analysis of California data[84] found that the positive association between parents' smoking and adolescent smoking initiation was stronger among Chinese Americans (relative risk [RR] 3.01; $p \leq 0.003$) than among whites (RR 1.68; $p \leq 0.001$). It is not known whether the association between parental smoking and adolescent smoking varies across SES groups or by sexual orientation.

Adults

Most studies of social relationships and smoking cessation among adults have focused on social networks, social control, or social support rather than social influence. These studies are reviewed elsewhere in this chapter in the corresponding sections.

Social Control and Smoking

Adolescents

Social control by parents includes communicating with children about not smoking, prohibiting them from smoking, or restricting their access to cigarettes. A review of 19 studies[95] concluded that the evidence suggests that parental rules against household smoking reduced adolescent smoking behaviors; however, the reviewed studies did not focus on differences by race/ethnicity or other variables.

One line of research on parental social control on adolescent smoking has explored the hypothesis that African American parents feel more empowered than parents of other races/ethnicities to prevent their children from smoking, and that they are more likely to set and enforce clear rules against smoking.[14,94,96,97] Therefore, even if African American parents are smokers, they might be more likely to limit their children's smoking with firm rules. Differences in parenting practices and rules about smoking could protect African American youth from experimenting with smoking, even in the presence of other risk factors.

If African American parents are more likely to set and enforce rules against smoking, one might expect the associations between parental monitoring and no-smoking rules and adolescent smoking to be stronger among African Americans than among other groups. However, several large studies have

reported opposite findings. For example, Bohnert and colleagues[98] conducted a study in southeast Michigan and found that parental monitoring was protective against smoking initiation between ages 11 and 17 among white adolescents (OR 0.89; 95% CI 0.83–0.96) but not among African American adolescents (OR 0.98; 95% CI 0.93–1.04). A cross-sectional analysis of the Add Health Wave I sample produced a similar finding: Parental control was protective against smoking among white adolescents but not among African American adolescents.[99] It is possible that commonly used parenting measures do not adequately capture parental monitoring related to tobacco use. More research is needed to understand the specific smoking-related messages that parents of different racial/ethnic groups convey to their children, how children perceive these messages, and how these messages influence children's tobacco use behaviors.

Several other studies have compared the associations between parenting practices and adolescent smoking in racial/ethnic groups other than African Americans and whites. Shakib and colleagues[100] reported on several such associations, finding that parental monitoring was more protective against smoking among whites (OR 0.30; 95% CI 0.15–0.60) than Hispanics (OR 0.68; 95% CI 0.54–0.85), and that adolescent communication with parents was more protective among Hispanics (OR 0.63; 95% CI 0.50–0.78) than whites (OR 1.48; 95% CI 0.70–3.13). Neither parental monitoring nor communication was significantly associated with smoking among Asian Americans. Another study[87] found that positive parenting practices (monitoring and closeness) were protective against lifetime smoking among white (OR 0.6; $p \leq 0.001$) and African American adolescents (OR 0.5; $p \leq 0.001$) but not among Hispanics.

Home smoking bans protect family members, including infants and children, from the serious health hazards of exposure to SHS[101] and reduce youth smoking and progression from initiation to regular smoking.[7,102,103] For this reason, the American Academy of Pediatrics supports promoting smoke-free homes.[104] However, as noted in the 2012 Surgeon General's report, "more information is needed on how home smoking policies vary by sociodemographic characteristics."[7,p.709]

Adults

Studies conducted among the general population have shown that people with home smoking bans are less likely to be smokers, and smokers with home smoking bans are more likely to make cessation attempts.[105] Low-income families are less likely to have home smoking bans,[106–108] so low-income smokers might be less likely to experience this type of social control.

Only one study was identified that compared the association between home smoking bans and smoking behavior across racial/ethnic groups and among SES groups. This study, which analyzed data on employed women from the Tobacco Use Supplement to the Current Population Survey[107] found that across all racial/ethnic and SES groups, respondents with a home smoking ban were less likely to be current smokers, compared with those without a home smoking ban.

Several studies have focused on social control and adult smoking among specific populations. Most of these studies focused on associations between home smoking bans and smoking status, intentions to quit, or cessation. A study of African American and Puerto Rican young adults[109] found that respondents in homes where smoking was banned were less likely to be smokers than those in homes where smoking was allowed; the study did not control for whether there was a smoker in the household. A study of LGBT adult smokers in Colorado[110] found that those who had smoking restrictions in the home were

more likely to be preparing to quit in the next month (OR 2.42; 95% CI 1.54–3.80). Similarly, among a sample of Chinese American smokers in New York, those with complete smoking bans reported smoking fewer cigarettes per day and were 3.4 times more likely to report a quit attempt in the past year than those with no home smoking ban (95% CI 1.51–7.05).[111] A study of male Vietnamese smokers in California[112] reported a similar finding, but the association between home smoking bans and quit intentions was confounded by family conflict about smoking. A study of American Indian adults who had filled a prescription for nicotine replacement therapy[113] found that those with home smoking bans were more likely to report 7-day abstinence 8 months later compared to those without home smoking bans.

Social Support and Smoking

Adolescents

No studies were identified that focused on disparities in the effects of social support on adolescent smoking initiation or progression. The few studies that approach similar topics have focused on specific aspects of parenting that are difficult to disentangle from other parenting practices, such as social control and monitoring. For example, Nowlin and colleagues[99] found that high-quality parent–child relationships were protective against smoking among white and African American adolescents, but the association was significantly stronger among whites. Only the association between high-quality mother–child relationships and smoking among whites remained significant in a 1-year follow-up. A growth curve study of family interactions and substance use among white and African American adolescents[114] found that negative family interactions were associated with increases in smoking during adolescence among African American males and white females but not among African American females and white males. Studies have not assessed disparities in the influence of social support on smoking initiation and progression among adolescents of other racial/ethnic groups, across SES groups, or across sexual orientation groups. As explained above, the lack of research in this area could be attributable to the assumptions that social influence is a stronger determinant of smoking initiation and that social support is a stronger determinant of smoking cessation, which usually occurs among adults.

Adults

Most studies of social relationships and smoking cessation have focused on social support, including emotional, informational, and instrumental support. The evidence indicates that people who have social support are more successful in quitting and achieving long-term abstinence than those who lack social support[115] and for this reason many smoking cessation programs include components to provide social support or to enhance the individual's existing support networks.[64] Reviews[64,116] have concluded that interventions to make smokers' existing social networks more supportive had not yet demonstrated efficacy, but interventions that deliver additional social support via repeated counseling sessions can be effective. Although numerous studies have focused on racial/ethnic differences in the effectiveness of pharmacological smoking cessation treatments,[117–121] very few have examined disparities in the effects of social support interventions on smoking cessation outcomes.[122]

In reviewing this literature, it is important to distinguish between studies that provide additional social support as a smoking cessation treatment (e.g., support groups, group counseling, ongoing contact with a professional or paraprofessional counselor) from studies that examine whether smokers who already have supportive social networks are more likely to quit than are those who lack social support. Additionally, the 2008 U.S. Public Health Service Clinical Practice Guideline, *Treating Tobacco Use*

and Dependence,[117] distinguished between practical counseling, defined as providing problem-solving skills/skills training, and providing general support and encouragement to quit. For reviewed studies that provide both practical counseling and social support it can be difficult to distinguish between the effects of the two. The Guideline also distinguished between "intra-treatment social support" (providing support during contact with a clinician) and "extra-treatment social support" (intervening to increase social support in the smokers' environment). The Guideline panel recommended the former but not the latter, citing literature indicating the difficulty of helping smokers identify and use support outside of the treatment setting.[117]

Smoking Cessation Interventions That Include Social Support Components

In a review of smoking cessation interventions among racial/ethnic minority groups, Cox and colleagues[118] located a total of 64 studies. These focused on African Americans (n = 28), Hispanics (n = 10), American Indians (n = 4), Asian Americans (n = 3), and multiple racial/ethnic minority groups (n = 19). Studies that used social support interventions from the Cox and colleagues review, along with additional studies published between 2011 and 2012, are discussed here and summarized in Table 6.1. Specific criteria for study inclusion were: extensive use of social support (i.e., more than one session) but no use of pharmacotherapy, media campaigns, or community-wide programs. Various study designs and interventions were used. Most studies involved counseling and support provided by health professionals or trained laypeople, either in person or by telephone. The interventions typically included emotional support and counseling on motivation, goal setting, and/or relapse prevention. In addition to individual and group support, study interventions sometimes included other social support components such as buddy interventions, culturally tailored or nontailored self-help materials, and motivational enhancement. Some studies provided structured and directive interventions; others offered general check-in contacts and left the content of the conversation to the discretion of the counselor and client.

Most of these studies found that smoking cessation interventions that included individual and/or group support and counseling were more effective than control conditions or interventions that did not include support and counseling. Only a few studies[123–126] compared the effects of a social support intervention across demographic groups. Three studies found higher quit rates among African Americans than among whites,[123–125] and one study found equally strong intervention effects among Chinese Americans, Korean Americans, and Vietnamese Americans.[126] Although Audrain-McGovern and colleagues[123] found that African Americans had a higher quit rate than whites (8.0% vs. 2.0%), the intensive motivational interviewing intervention was less successful than structured brief advice (OR 0.41; 95% CI 0.17–0.97). Also, the higher quit rate among African Americans compared with whites reported by Cluss and colleagues[124] cannot be attributed to the social support intervention because the study lacked a control group.

Table 6.1 Studies of Social Support Smoking Cessation Interventions Among Specific Populations

Author (year)	Sample	Social support intervention	Outcomes
Studies that compared social support intervention effects across demographic groups			
Audrain-McGovern et al. 2011[123]	355 adolescents (45% African American, 40% white, and 12% Hispanic)	Five sessions of MI or structured brief advice	African Americans were more likely to attempt to quit than whites,* and MI intervention was *less* effective than structured brief advice.* No interactions between treatment condition and ethnicity were reported.
Cluss et al. 2011[124]	856 low-income pregnant women (59% white, 37% African American, and 4% other)	Four to eight sessions of MI, goal setting, and counseling	African Americans were more likely to attempt to quit than whites,* but this difference could not be attributed to the social support intervention because the study lacked a control group.
Windsor et al. 1993[125]	814 pregnant adult smokers (52% African American and 48% white)	Individual and group counseling, social support, and buddy intervention (control condition was no intervention)	Abstinence at 32 weeks was higher among the treatment group than among the control group.* A significant treatment effect was found among African Americans* but not among whites.
Zhu et al. 2012[126]	2,277 Asian American adults (including those of Chinese, Korean, and Vietnamese origin) in California	As many as six quitline counseling sessions (control condition was self-help materials)	Abstinence at 6 months was higher for those who received quitline counseling than those who received self-help materials only.* The intervention effect was significant among Chinese Americans,* Korean Americans,* and Vietnamese Americans.*
Studies that involved different populations but did not compare intervention effects across demographic groups			
Hennrikus et al. 2005[162]	2,095 hospital inpatients (78% white and 16% African American)	Physician advice plus three to six phone calls incorporating MI, action planning, and relapse prevention (control conditions were physician advice only or modified usual care)	No significant intervention effects or racial/ethnic differences were reported.
Jason et al. 1988[163]	165 adults (96% African American)	Weekly support meetings and supportive phone calls (control condition was no intervention)	Four-month abstinence was higher among the treatment group than among the control group.
Malchodi et al. 2003[164]	142 pregnant women (63% Hispanic and 12% African American)	As many as eight contacts with trained peer counselors (control was usual prenatal clinic care)	The intervention group smoked fewer cigarettes per day,* but there were no group differences in cessation, and no racial/ethnic differences were reported.
Nevid & Javier 1997[165]	93 Hispanic adults	Eight group sessions and telephone support (control condition was one session and self-help materials)	No significant intervention effect was found.
Voorhees et al. 1996[166]	292 African American adults recruited at churches	Churches randomly assigned to intensive program with individual counseling and group sessions or to a minimal self-help condition	No difference in 12-month abstinence was found between the groups.
Wetter et al. 2007[167]	297 Spanish-speaking Hispanic adults	One helpline phone counseling session plus three proactive phone calls (control was one helpline counseling session only)	The intervention condition produced significantly higher 12-week abstinence.*
Woodruff et al. 2002[168]	313 Hispanic adults	Four home visits and three phone calls from trained lay health advisors known as *promotores* (control was referral to helpline)	A significant intervention effect on 7-day abstinence was found.*

Notes: Studies were included in this table if they included multiple counseling or support sessions but did not include pharmacotherapy, community-wide programs, or media campaigns. MI = motivational interviewing.
*Significant finding.

Studies of Naturally Occurring Social Support and Tobacco Use Behaviors

Studies have examined individuals' preexisting level of social support as a predictor of smoking cessation success. Several of these studies focused on diverse populations, including Hispanics,[127,128] African Americans,[128–135] Filipino immigrants,[136] and Korean Americans.[137,138] All the studies found that people were more successful in quitting smoking if they had support for their quit attempts from spouses, other family members, or friends.

Some studies have examined the association between social support and current smoking. A study of low-income African American women in Detroit[139] found an inverse relationship between social support (defined as having someone they could count on to help run errands, lend money, watch their children, lend a car or give a ride, and provide encouragement if needed) and current smoking. Overall, the evidence suggests that naturally occurring social support is associated with more successful smoking cessation across demographic groups.

Discrimination and Smoking

Disparities in the Prevalence of Discrimination

Among adults, African Americans are most likely to report discrimination, followed by Hispanics, Asians, and whites.[38,140,141] Among young adults, LGBT groups report more discrimination than heterosexuals.[37] The prevalence of reported discrimination among adolescents has not been well studied.

Discrimination and Smoking Initiation and Progression Among Adolescents

Several studies have documented associations between discrimination and smoking initiation or progression within specific racial/ethnic minority groups of adolescents, but little research exists examining this association across groups (Table 6.2). A study that compared this association across racial/ethnic groups[78] found that perceived discrimination was associated with light smoking among older Puerto Rican and African American adolescents, with no racial/ethnic differences in the strength of the association. A study of Hispanic adolescents[47] found that discrimination was associated with the increased odds of lifetime and past-month smoking (OR 1.73; 95% CI 1.30–2.31 and OR 2.54; 95% CI 1.73–3.72), and a study of African American adolescent girls[46] found that discrimination was correlated with the odds of lifetime smoking ($r = 0.35$; $p \leq 0.001$) but did not assess progression to higher levels of smoking. A study of Hispanic adolescents in Southern California[142] found that discrimination predicted smoking initiation among girls but not among boys. A study of Oregon adolescents[143] found that the disparity in smoking across sexual orientation groups was smaller in communities that had more supportive social environments for lesbian, gay, and bisexual youth (e.g., higher proportion of same-sex couples, presence of gay–straight alliances in schools, nondiscrimination and antibullying school policies). A study of American Indian adolescents living on or near one of three reservations in the upper Midwest found high levels of reported discrimination among the youth; the study also found that adolescents who had experienced discrimination tended to respond with anger and delinquent behaviors, which in turn were associated with substance use, including tobacco.[48]

Most studies have found that the risk of smoking increases as the level of discrimination increases. However, a study of low-income African American and Hispanic adolescents found that discrimination was a risk factor for smoking among boys (OR 1.9; 95% CI 1.2–3.0) but was protective among girls (OR 0.6; 95% CI 0.3–1.1).[144] This study analyzed data from the Moving to Opportunity Study in which low-income public housing residents were randomly assigned to remain in public housing, move to any

neighborhood outside of public housing, or move to a low-poverty neighborhood. Among adolescents, the positive association between discrimination and smoking was strongest among the boys who remained in public housing, suggesting a socioeconomic disparity in the effects of discrimination on smoking. The inverse association between discrimination and smoking among girls did not vary across experimental conditions. In post hoc analyses, the inverse association between discrimination and smoking among girls appeared to be driven by girls who had become pregnant and had dropped out of school. Among girls who remained in school and/or did not become pregnant, there was no association between discrimination and smoking. These findings illustrate the complexity of the association between discrimination and smoking and underscore the importance of examining confounding and moderating variables.[145]

Overall, the evidence suggests that discrimination can increase the risk of smoking, but it is not clear whether specific populations of adolescents are particularly vulnerable or resilient to the effects of discrimination. More research is needed to understand variation in the association between discrimination and adolescent smoking by race/ethnicity, sexual orientation, SES and other factors.

Table 6.2 Studies of Discrimination and Smoking

Author (year)	Discrimination measure	Sample	Findings
Studies of adolescents			
Fagan et al. 2009[78]	"How much have you experienced discrimination by the police or security guards?"	550 older adolescents (mean age = 19 years); 52% African American and 48% Hispanic	Discrimination in late adolescence was significantly associated with light smoking (relative to nonsmoking) in late adolescence,* which in turn was significantly associated with smoking in early adulthood.* No significant differences were found between African Americans and Hispanics in the strength of the association between discrimination and smoking.
Guthrie et al. 2002[46]	Williams Everyday Discrimination Scale	105 African American adolescent girls (mean age = 15 years)	Discrimination was correlated with cigarette smoking.*
Lorenzo-Blanco et al. 2011[142]	Williams Everyday Discrimination Scale	1,124 Hispanic 9th-grade students in Southern California	Perceived discrimination was associated with past-month smoking among girls* but not among boys.
Okamoto et al. 2009[47]	Williams Everyday Discrimination Scale	1,332 Hispanic 9th-grade students in Southern California	Perceived discrimination was associated with lifetime smoking* and past-month smoking.*
Whitbeck et al. 2001[48]	Williams Everyday Discrimination Scale	195 American Indian 5th- to 8th-grade students living on or near reservations	Discrimination was associated with a composite measure of substance use (multiple substances, including cigarette smoking).*
Wiehe et al. 2010[144]	"Can you think of 1 or more occasions in the past 6 months when you felt you were treated unfairly because of your race or ethnicity in the following places?"	2,561 African American and Hispanic adolescents ages 12 to 19 years who participated in the Moving to Opportunity Study	Discrimination was associated with increased odds of smoking among boys* and decreased odds among girls.* No racial/ethnic differences in the association between discrimination and smoking were reported.

Table 6.2 continued

Author (year)	Discrimination measure	Sample	Findings
Studies of adults			
Albert et al. 2008[145]	"Ever discriminated against due to race/ethnicity?"	1,475 adults in Dallas (54% African American, 33% white, and 13% Hispanic)	Hispanics who reported discrimination had a higher prevalence of smoking compared to those who did not, but no statistical significance was reported. There was no association between discrimination and smoking among African Americans or whites.
Bennett et al. 2010[151]	Williams Everyday Discrimination Scale "For unfair reasons, do you think that you have ever not been hired for a job?" "Have you ever been unfairly stopped, searched, questioned, physically threatened or abused by the police?"	4,454 low-income, inner-city pregnant women at public health centers in Philadelphia (67% African American, 21% Hispanic, 9% white, and 3% other)	A high level of everyday discrimination was significantly associated with smoking.* No significant differences were found across racial/ethnic groups regarding the strength of the association.
Blosnich & Horn 2011[37]	"Within the last 12 months, have any of the following affected your academic performance?"	College students ages 18–24 years (4,286 heterosexual, 1,825 gay/lesbian, 2,545 bisexual, and 1,545 unsure)	Discrimination was more prevalent among gay/lesbian, bisexual, and unsure students than among heterosexuals,* but discrimination was not associated with smoking. Among gay/lesbian, bisexual, and unsure students, being in a physical fight was associated with an increased risk of smoking.*
Borrell et al. 2010[38]	Williams Everyday Discrimination Scale	6,680 adults participating in the Multi-Ethnic Study of Atherosclerosis in California, Illinois, Maryland, Minnesota, New York, and North Carolina during 2000 and 2002 (39% white, 28% African American, 22% Hispanic, and 12% Chinese American)	Discrimination was associated with increased odds of being a current smoker among African Americans and whites* but not among Hispanics or Chinese Americans.
Borrell et al. 2007[146]	Williams Everyday Discrimination Scale	3,320 adult participants in the CARDIA study (45% African American and 45% white)	African Americans experiencing racial discrimination in at least three domains in both years of this study had higher odds of reporting current* and former smoking* than did those experiencing no discrimination. The association between discrimination and smoking was not significant among whites.
Burgess et al. 2007[39]	Krieger Experiences of Discrimination (EOD) measure	Adults in Minnesota (472 LGBT individuals and 7,412 heterosexuals)	Discrimination* and smoking* were each more prevalent among LGBT individuals than among heterosexuals, but discrimination was not associated with smoking.
Chae et al. 2008[40]	Williams Everyday Discrimination Scale	1,977 Asian Americans in the National Latino and Asian American Study (2002–2003)	Odds of current smoking were higher among Asian Americans who reported high levels of racial/ethnic discrimination compared to those who reported no discrimination. This finding was not significant.

Table 6.2 continued

Author (year)	Discrimination measure	Sample	Findings
Corral & Landrine 2012[152]	"How much racism or discrimination have you personally experienced in the past year?"	2,118 African American adults in California participating in a door-to-door survey in random census tracts	High discrimination was associated with higher odds of current smoking.*
Gibbons et al. 2004[41]	Schedule of Racist Events	897 African American parent-adolescent dyads	Among parents and adolescents, discrimination was associated with higher scores on a combined substance use index (tobacco, alcohol, and other drugs combined).*
Horton & Loukas 2013[147]	Schedule of Racist Events	984 technical/vocational school students in Texas (41.8% white, 27.8% African American, and 30.4% Mexican American)	Discrimination increased the likelihood of current use of cigarettes* and cigars/cigarillos* among African American students, and current cigar use among white students.* There were no associations between discrimination and tobacco use among Mexican Americans.
Krieger et al. 2005[140]	Krieger Experiences of Discrimination (EOD) measure	616 working-class adults in Boston (26% African American, 40% Hispanic, and 34% white)	The association between discrimination and current smoking approached statistical significance among African Americans and Hispanics but not among whites.
Landrine et al. 2006[43]	Schedule of Racist Events	1,569 college students and community adults (49.7% white, 25.9% Hispanic, 11.1% African American, and 6.0% Asian American)	Among whites and racial/ethnic minority groups, those who experienced moderately frequent* or frequent* discrimination were more likely to be current smokers than those who experienced low discrimination. All racial/ethnic minority groups were combined into a single group.
Landrine & Klonoff 2000[42]	Schedule of Racist Events	453 African American adults	Smoking prevalence was higher among participants who reported frequent discrimination than among those who reported infrequent discrimination.*
Li & Delva 2012[148]	Krieger Experiences of Discrimination (EOD) measure	998 Asian American men who participated in the 2002-2003 National Latino and Asian American Study (28% Chinese American, 24% Filipino American, 24% Vietnamese American, and 24% Other [Asian groups])	Discrimination was associated with current smoking among the whole sample, but the association was significant only among the other Asian groups in stratified analyses.*
Maxson et al. 2012[150]	Krieger Experiences of Discrimination (EOD) measure	1,518 pregnant women (78% African American and 22% white)	Discrimination was associated with current smoking versus never-smoking among African Americans* but not among whites.
Nguyen 2012[149]	Krieger Experiences of Discrimination (EOD) measure	677 pregnant women (39% African American and 61% Hispanic)	Discrimination was associated with smoking among African Americans* but not among Hispanics.
Purnell et al. 2012[141]	Perceived racial discriminations assessed in 2 domains (health care, work) as measured by the Reactions to Race module	85,130 adult respondents in the 2004–2008 Behavioral Risk Factor Surveillance Surveys (81% white, 11% African American, and 4% Hispanic)	Current smoking was more prevalent among respondents who reported being treated worse than people of other races in health care settings* or in the workplace,* relative to those who reported equal treatment. Racial/ethnic differences in the association between discrimination and smoking were not assessed.

Table 6.2 continued

Author (year)	Discrimination measure	Sample	Findings
Todorova et al. 2010[153]	"Have you ever experienced discrimination as a result of your race, ethnicity or language?" "Have you ever experienced discrimination as a result of your race, ethnicity or language [in a 'healthcare setting']?"	1,122 Puerto Rican adults in Boston	Former smokers were more likely to report discrimination than never-smokers or current smokers.*
Tran et al. 2010[44]	Krieger Experiences of Discrimination (EOD) measure	1,384 immigrant adults in the Midwest (40% African-born black, 31% Southeast Asian, and 29% Latino/Hispanic)	Perceived discrimination was significantly related to being a current smoker for Southeast Asian immigrants* but not among those in the other racial/ethnic groups.
Yoo et al. 2010[45]	Asian American Racism-Related Stress Inventory and the Perceived Ethnic Discrimination Questionnaire—Community Version	271 Asian American adults participating in the 2008 Asian Pacific Arizona Initiative Survey	Asian Americans treated like they were not American because of their race were at increased risk of tobacco use.*

Notes: Hatzenbuehler et al. 2011,[143] not included in this table, used a measure of social environment to conclude that a more supportive social environment for lesbian, gay, and bisexual youth was significantly associated with reduced tobacco use (OR 0.92; 95% CI 0.90–0.94). LGBT = lesbian, gay, bisexual, and transgender.
*Significant finding.

Discrimination and Current Smoking Among Adults

Many studies have examined the strength of the association between discrimination and smoking among adults across racial/ethnic groups (Table 6.2). Most of the studies that included multiple racial/ethnic groups compared African Americans, Hispanics, and whites. Most of these studies found significant associations between discrimination and smoking among one or more racial/ethnic groups, but the specific associations differed between studies.

A study of adults ages 45–84 years in six U.S. states[38] found that African Americans and whites who reported racial/ethnic discrimination were more likely to be current smokers, compared to those who did not report discrimination (OR 1.34; 95% CI 1.00–1.81 and OR 1.88; 95% CI 1.02–3.44, respectively); this association was not significant among Hispanic or Chinese American participants. Conversely, a study of adults in Texas[145] found that Hispanics who experienced discrimination had a higher prevalence of current smoking, but not African Americans or whites. In a longitudinal study of young adults,[146] African Americans who reported high levels of discrimination were at increased risk of being current or former smokers (OR 1.87; 95% CI 1.18–2.96), compared with those who did not report discrimination.

A study of adults in Boston[140] did not find significant associations between discrimination and smoking among whites, African Americans, or Hispanics, although the association approached statistical significance among the latter two groups. A study of technical/vocational students (mean age = 25) found that discrimination was significantly associated with increased cigarette and cigar smoking among African Americans ($r = 0.17$ and $r = 0.29$, respectively), associated only with cigar smoking among whites ($r = 0.13$), and not significantly associated with smoking among Mexican Americans.[147]

In an ethnically diverse sample of college students and community adults, experiencing moderately frequent or frequent discrimination was associated with an increased risk of being a current smoker among whites (OR 1.56; 95% CI 1.09–2.24 and OR 1.76; 95% CI 1.09–2.82) and members of racial/ethnic minority groups (OR 1.99; 95% CI 1.14–3.48 and OR 2.32; 95% CI 1.38–3.91).[43] However, the odds ratios were similar for whites and racial/ethnic minority groups, and the minority groups were not subdivided into specific racial/ethnic groups (the racial/ethnic minority group was 25.9% Hispanic, 11.1% African American, and 6.0% Asian). An analysis of data from the 2004–2008 Behavioral Risk Factor Surveillance Surveys found that across racial/ethnic groups, adults who reported that they had been treated worse than others in health care or workplace settings because of their race/ethnicity had an elevated risk of being current smokers (OR 1.18; 95% CI 1.09–1.26 and OR 1.13; 95% CI 1.03–1.23).[141] However, this study included race/ethnicity as a covariate rather than a moderator, so it does not indicate whether the association between discrimination and smoking was stronger among one group than another.

A study of Asian American men in the 2002–2003 National Latino and Asian American Study compared current smokers with current nonsmokers and found that current smokers had significantly higher scores on an everyday discrimination measure (OR 1.41; 95% CI 1.06–1.89).[148] Stratifying the sample by Asian groups revealed that the association between discrimination and smoking was not significant among Vietnamese American, Filipino American, or Chinese American men, but was significant among the "Other" group (OR 2.67; 95% CI 1.52–4.71).

Although most studies of discrimination and smoking have included U.S.-born respondents and immigrants, one study[44] focused only on immigrants. This study found that discrimination was associated with increased odds of being a current smoker among Southeast Asian immigrants (OR 1.60; 95% CI 1.02–2.51) but not among Hispanic and African immigrants.

Three studies of discrimination and smoking among multiple racial/ethnic groups focused on pregnant women. Nguyen and colleagues[149] found that discrimination was a risk factor for smoking among pregnant black women (OR 3.36; 95% CI 1.23–9.19) but not among pregnant Hispanic women. Maxson and colleagues[150] found that discrimination was associated with increased smoking among pregnant African American women (OR 1.15; 95% CI 1.01–1.31) but not among pregnant white women. A study of low-income pregnant women (67% African American and 21% Hispanic) found that a high level of everyday discrimination was associated with an increase in smoking during pregnancy (OR 1.41; 95% CI 1.15–1.74); race/ethnicity did not moderate this association.[151]

Additional studies have focused on the association between discrimination and smoking among single racial/ethnic groups; the majority have focused on African Americans. Several studies[41,42,152] found that African American adults who reported high levels of discrimination were more likely to smoke than African Americans who did not report high levels of discrimination. Similar associations between discrimination and current smoking have been reported among other racial/ethnic groups, including Asian Americans.[40,45,148] A study of Puerto Rican adults living in Boston[153] found that perceived discrimination was associated with a higher probability of having ever smoked. However, there were no significant differences in discrimination between current smokers and current nonsmokers.

Although LGBT individuals experience more discrimination and have a higher prevalence of smoking than heterosexuals, few studies have compared the association between discrimination and smoking across sexual orientation. The two studies that assessed the association between discrimination and

smoking among LGBT groups[37,39] did not find that discrimination was a risk factor for smoking. No studies were identified that assessed SES-based discrimination and smoking.

Evidence Summary

Table 6.3 summarizes the evidence discussed in this chapter on disparities in associations between aspects of social relationships, the continuum of smoking behavior, and TRHD.

Table 6.3 Summary: Social Relationships, Smoking Behavior, and TRHD

Characteristics of social relationships	Early experimentation and progression to regular smoking	Cessation attempts and smoking cessation
Social networks	*The evidence strongly supports that:* ▪ Social connections with smokers in a social network influence smoking initiation and progression. ▪ Social network isolation (few or no social ties) is associated with smoking initiation and progression. ▪ Popularity in school-based social networks is associated with smoking initiation and progression, although this can vary according to the social context of the school. ▪ The effect of popularity on smoking generalizes to some vulnerable populations, such as Hispanics. *The evidence is insufficient to determine* the effect of popularity on smoking for most other vulnerable populations and for Hispanics in social contexts that are not primarily Hispanic. *The evidence is insufficient to determine* differences across groups concerning the effects of social networks on smoking.	*There is limited evidence suggesting that* smoking cessation diffuses through social networks. *The evidence is insufficient to determine* if differences exist by demographic group.
Social influence	*The evidence strongly supports that* social influence is associated with smoking initiation and progression among most racial/ethnic groups. *The evidence strongly supports that* living with a smoker is associated with smoking initiation and progression. *There is limited evidence suggesting that* the effect of social influence on smoking initiation and progression is stronger among white adolescents than among adolescents of other racial/ethnic groups. *The evidence is insufficient to determine* whether there are differences in the amount of social influence across SES or sexual orientation groups. *The evidence is insufficient to determine* whether the effect of social influence on smoking initiation and progression varies across SES or sexual orientation groups. *The evidence is insufficient to determine* whether the effect of living with a smoker varies across demographic groups.	*The evidence is insufficient to determine whether:* ▪ There is a causal relationship between social influence and smoking cessation. ▪ There are differences across demographic groups in the effects of social influence on smoking cessation.

Table 6.3 continued

Characteristics of social relationships	Early experimentation and progression to regular smoking	Cessation attempts and smoking cessation
Social control	*The evidence strongly supports that:* ▪ Home smoking bans are protective against adolescent smoking initiation and progression. ▪ Low-income families are less likely to have home smoking bans than higher income families. *The evidence is insufficient to determine whether*: ▪ The effects of home smoking bans vary across demographic groups. ▪ Other parenting practices to prevent youth smoking are differentially effective across demographic groups.	*The evidence strongly supports that* low-income families are less likely to have home smoking bans. *There is limited evidence suggesting that* the association between home smoking bans and cessation-related behaviors exists in multiple racial/ethnic groups and among LGBT groups. *The evidence is insufficient to determine* whether the strength of the association between home smoking bans and cessation differs across demographic groups. *The evidence is insufficient to determine* whether home smoking bans are causally associated with lower smoking prevalence, higher intentions to quit, and more successful quit attempts among adults.
Social support	*There is limited evidence suggesting that* social support from parents is protective against adolescent smoking initiation and progression. *The evidence is insufficient to determine* whether the effects of social support vary across demographic groups.	*The evidence strongly supports that:* ▪ Smokers with higher levels of naturally occurring social support have better smoking cessation success. ▪ The association between naturally occurring social support and smoking cessation success exists in multiple racial/ethnic groups. ▪ Social support interventions (in the absence of pharmacotherapy) are more effective than control conditions in producing abstinence among smokers trying to quit. ▪ Social support interventions (in the absence of pharmacotherapy) are effective in producing abstinence in multiple racial/ethnic groups. *There is limited evidence suggesting that* social support interventions are more effective among African Americans than among other groups. *The evidence is insufficient to determine* whether the strength of the association between naturally occurring social support and smoking cessation success varies across demographic groups.
Discrimination	*There is limited evidence suggesting that* discrimination is associated with smoking initiation and progression among adolescents. *The evidence is insufficient to determine* which demographic groups of adolescents report the most discrimination. *The evidence is insufficient to determine* whether the strength of the association between discrimination and smoking initiation or progression varies across demographic groups.	*The evidence strongly supports that* discrimination is associated with current smoking among African Americans, Hispanics, and some Asian groups. *There is limited evidence suggesting that* the association between discrimination and current smoking is stronger among racial/ethnic minority groups than among whites. *The evidence is insufficient to determine* whether the association between discrimination and smoking varies across SES groups.

Notes: SES = socioeconomic states. LGBT = lesbian, gay, bisexual, and transgender.

Chapter Summary

Social relationships exert powerful influences on numerous human behaviors, including tobacco use behaviors. Both structural aspects of social relationships (social networks) and functional aspects (social influence and social comparison, social control, social support, and discrimination) have been studied in relationship to TRHD. This chapter has reviewed the evidence that social relationships contribute to TRHD across the tobacco use continuum, for both youth and adults, and across groups based on race/ethnicity, SES, and sexual orientation. It is likely that different aspects of social relationships influence different stages of the tobacco use continuum; as a result, some relationships have been studied in greater depth than others. The depth of the literature also differs across race/ethnicity, SES, and sexual orientation, and is especially limited for the latter two demographic categories. A summary of the findings from the literature reviewed in this chapter is provided in Table 6.3.

Indicators of sociometric position (i.e., a person's pattern of connections to others in the social network), such as popularity and social isolation, are risk factors for smoking initiation among adolescents; this finding likely generalizes to disparate populations. However, the direction and mechanism of this association remain unclear, and there is little evidence about whether there are racial/ethnic, gender, or sexual orientation differences in this association. Additional research is needed to determine why two opposite social network statuses—popularity and social isolation—are both risk factors for adolescent smoking. It would also be informative to explore whether the overall composition of the school moderates the association between social network variables and smoking. The influence of sociometric position on smoking cessation among adults and how this may differ based on race/ethnicity, SES, and sexual orientation is an area for future research.

Studies show that social influences (peers, parents, and other family members) are associated with smoking initiation and progression among adolescents across most demographic groups that have been studied. Parents (including those who are smokers themselves) may exert social control over youth smoking through a variety of means, including talking with their children about smoking, prohibiting their children from smoking, restricting youth's access to tobacco products, and by completely banning smoking in the home (implementing home smoking bans that apply to all). Few studies have examined differences in the effectiveness of these social control mechanisms by groups. The evidence shows that home smoking bans are associated with decreased adolescent smoking initiation and progression and that smokers with home smoking bans are more likely to have cessation intentions and make quit attempts. However, low-income families—who are more likely to include people who smoke—are less likely to have home smoking bans than families of higher SES. There is no evidence that the association between home smoking bans and reduced smoking behaviors differs across demographic groups.

Social support is associated with increased quitting success among smokers across racial/ethnic groups. Smokers who have more established social support systems and those who are provided social support during treatment have an increased likelihood of successfully quitting. No evidence is available about the relative effectiveness of social support interventions across SES groups or sexual orientation groups. In addition, there is insufficient research to determine whether culturally tailored social support interventions are superior to culturally generic interventions. Research is needed to compare the effectiveness of different types of social support interventions in different populations, alone and in combination with pharmacotherapy. Understanding which types of social support interventions are most effective for various populations may contribute to increasing cessation success.

Studies find that discrimination is associated with smoking initiation and progression among racial/ethnic minority adolescents and current smoking among racial/ethnic minority adults. However, findings about which minority groups experience the strongest effects of discrimination on smoking vary considerably. It is likely that some of these studies had insufficient statistical power to detect associations between discrimination and smoking among some groups studied, so a failure to detect significant effects should not be taken as evidence that no effects exist. The larger studies generally found significant associations between discrimination and current smoking among adults of most racial/ethnic minority groups. Although LGBT groups have high levels of smoking and experience high levels of discrimination, an association between discrimination and smoking among LGBT groups has not been found. However, only a few studies have examined this relationship.

Overall, relatively few studies of social relationships distinguish among groups by race/ethnicity, SES, and/or sexual orientation. At times, studies find that social relationships were associated with reductions in TRHD. For example, the presence of friends who smoke may be a stronger risk factor for smoking among white adolescents than among racial/ethnic minority adolescents, and the effects of social support interventions for smoking cessation appear to be stronger among African Americans than among whites. These patterns would be expected to reduce, not exacerbate, TRHD.

Research Needs

Research is needed to fill gaps in the literature relating to understudied areas of the intersection between social relationships, tobacco use, and TRHD, and where appropriate, should consider both cigarettes and other types of tobacco products. Although TRHD have been recognized for decades,[154] most studies on social relationships and tobacco use do not focus on disparities. Many studies focus on homogenous populations and do not address whether social relationships have different effects for different groups. To date, most research on disparities in social relationships and smoking has focused on racial/ethnic disparities, and on the largest population groups: whites, African Americans, and Hispanics. Research should also be directed toward examining social influences on smoking among ethnic groups who represent smaller segments of the population, especially those who have high smoking prevalence, including American Indians/Alaska Natives and Native Hawaiians/Pacific Islanders.

Although LGBT populations are at increased risk of smoking, very few studies have addressed the potential impact of social influences on TRHD by sexual orientation. A better understanding of social relationships and smoking among LGBT groups might be especially informative considering the recent attention paid to adolescent bullying based on sexual orientation. More research is also needed about the nature of social support and social control among same-sex couples and how the dynamics of these social interactions can influence tobacco use. Many large national surveys now assess sexual orientation (e.g., the National Adult Tobacco Survey as of 2009,[55] NYTS as of 2014,[52] Youth Risk Behavior Surveillance Survey as of 2015[155]) which can inform this research.

Most of the studies reviewed in this chapter focused on membership in only one type of minority group—racial/ethnic, SES, or sexual orientation. Individuals who are members of more than one minority group could be at especially high risk for TRHD. Very few studies focused on intersections across multiple minority statuses, such as LGBT and racial/ethnic minority groups.[156–159] Research is needed to help understand how social relationships create or exacerbate TRHD across the various intersections of minority groups.

Most existing research studies include race/ethnicity and SES as confounders in larger, multivariate prediction models of smoking, making it difficult to discern possible disparities in the strength of the effects of social relationships on smoking. When studies have sufficient statistical power, researchers should conduct analyses of the interactions (moderator effects) of social influences and race/ethnicity, gender, and sexual orientation to determine whether specific predictors of smoking are stronger in specific groups.

Studies of social influence on adolescent smoking initiation and progression have generally focused on peers who are close in geographic proximity (e.g., friends in classrooms and schools). Given the large and growing importance of online social networks, the extent to which these influences differ across racial/ethnic, gender, or sexual orientation groups is increasingly important to consider.[160] Similarly, studies may now use online and mobile technologies to deliver cessation interventions. Research studies should evaluate whether these technologies are equally effective at promoting cessation across different racial/ethnic groups, SES groups, genders, and sexual orientations.

Finally, this chapter summarizes evidence regarding associations of social influences with TRHD. However, it is also likely that tobacco use behaviors influence the types of social relationships that individuals form. Similarities in smoking behavior between adolescents and their friends are likely due to a combination of peer influence effects (adolescents emulating their friends' smoking behavior) and peer selection effects (adolescents befriending others who have similar smoking behaviors).[11,161] To the degree that smokers are more likely to affiliate with smokers and nonsmokers to affiliate with nonsmokers, these selection effects could contribute to and perpetuate disparities across groups. In addition, as smoking becomes a more stigmatized behavior, individuals may experience discrimination due both to their membership in a minority demographic group and to their smoking behavior. These questions warrant further research attention.

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Section III
Interpersonal and Contextual Factors That Contribute to Tobacco-Related Health Disparities

Chapter 7
Tobacco-Related Health Disparities Among Immigrant Populations

Contents

Figures and Tables

Introduction

Variations in smoking by age, gender, race/ethnicity, education, income, and region are well documented in the scientific literature. A small but growing body of evidence further suggests intersections between health outcomes and immigrant or nativity status (i.e., U.S.-born versus foreign-born) and disparities in these outcomes. In 2015 nearly 42 million people, or 13% of the total U.S. population, were foreign-born[1]—the largest absolute number of immigrants ever recorded and the highest proportion of foreign-born people since the 1920s. With diversity among immigrants in terms of national origin, language, religion, social class, reasons for migration, and processes of migration also greater than ever before,[2] the evidence on smoking behavior among immigrants is similarly complex. Social and structural determinants, including the processes of assimilation and acculturation for new immigrants, appear to play different roles within and across immigrant groups and across different aspects of smoking behavior (i.e., initiation, cessation, daily smoking, cigarette consumption, tobacco-related disease/mortality).

The chapter begins with a brief overview of immigration to the United States, including the processes of immigrant adaptation and an introduction to the countries sending the largest share of immigrants to the United States, to frame the discussion of smoking behavior of immigrants. Because immigrants largely come from cultures that are different from mainstream U.S. culture, policymakers, program planners, and researchers often ask whether the prevalence of certain behaviors and the social factors associated with them are similar or different for immigrants compared with U.S.-born individuals, who also come from diverse cultures. By placing immigrants in a broader social and cultural context, the complex interplay between factors affecting smoking behavior in both sending countries and the United States can be better understood, and the tobacco-related health and health care concerns of different immigrant groups can be more effectively addressed.[3]

This chapter reviews the literature on the smoking behavior of foreign-born people in the United States, including differences within and between immigrant groups, comparisons between immigrant groups and the majority population (U.S.-born, non-Hispanic white), and differences between immigrants and their U.S.-born racial/ethnic counterparts. Issues related to immigrant health generally and smoking behavior are also discussed, and unexpected intersections of tobacco, immigration, and demographic and socioeconomic factors are highlighted. The chapter concludes by identifying theoretical, methodological, and empirical gaps in the literature and opportunities for future research.

U.S. Immigration Patterns 1800–2010

American Indians were the first inhabitants of what became the United States, and with the exception of people brought to the country as slaves, all other racial/ethnic groups immigrated to the United States. Before 1800, migration to the United States consisted of a small but influential flow of European settlers whose preindustrial plantations required large amounts of cheap labor[4] from workers and slaves to secure their profit share. The most important source of plantation labor became the forced migration of more than 10 million African slaves. Diseases brought by European colonists and others sharply reduced the number of indigenous American Indians, who had not developed immunity to what were, to them, new diseases.[4]

Valuable insight into the demographic transformation of the United States can be derived from census counts, despite their notable limitations. Early censuses used inconsistent definitions of racial/ethnic categories, undercounted some groups, and, before 1860, did not count some groups at all. According

to the first U.S. Census in 1790, approximately 3.9 million people resided in the United States. Only two racial/ethnic categories were enumerated at that time: 81% of the population were classified as white, and the remaining 19% were classified as black.[5] By the 1850 census, the first year in which immigrant status was indicated, foreign-born individuals totaled 2.2 million, or 9.7% of the total U.S. population.[6] Mass movements of people from Europe and, to a lesser extent, Canada increased the U.S. immigrant population rapidly through the early 1920s as part of what historians often term the age of mass migration. The United States alone absorbed about 60% of Europe's total outflow between 1800 and 1929.[4] By 1910 approximately 87% of U.S. immigrants were from Europe, and another 9% were from Canada.[7]

The Immigration Act of 1924 (The Johnson-Reed Act) included strict limitations on the number of immigrants allowed to enter the United States. It established a national origins quota, which provided immigration visas to 2% of the total number of people of each nationality in the United States as of the 1890 national census. Between 1930 and 1948, migration to the United States was greatly decreased as a result of the Great Depression, the onset of the Second World War, and stricter enforcement of existing U.S. immigration policies.[8] Whereas an annual average of 621,000 immigrants entered the United States between 1900 and 1930, only 53,000 immigrants came to the United States during the decade of the 1930s, and only slightly more during the 1940s.[9] The Cold War continued to stifle migration through the mid-1960s, but by the 1970s immigration to the United States was again on the rise.[6] Unlike earlier migration patterns, however, more immigrants arrived from Latin America, especially Mexico, and Asia than from Europe and Canada, a pattern that continued through 2010 (Table 7.1). This trend was still in evidence as of 2015.[10] At the beginning of the 21st century, the five countries from which the largest proportions of foreign-born people in the United States originated were: Mexico (29.5%), the Philippines (4.4%), China (3.8%, excluding Hong Kong and Taiwan), India (3.3%), and Vietnam (3.2%). By 2010 the breakdown was similar, although India provided slightly more immigrants to the United States than either China or the Philippines (Figure 7.1).

Countries of Origin and Smoking Behavior

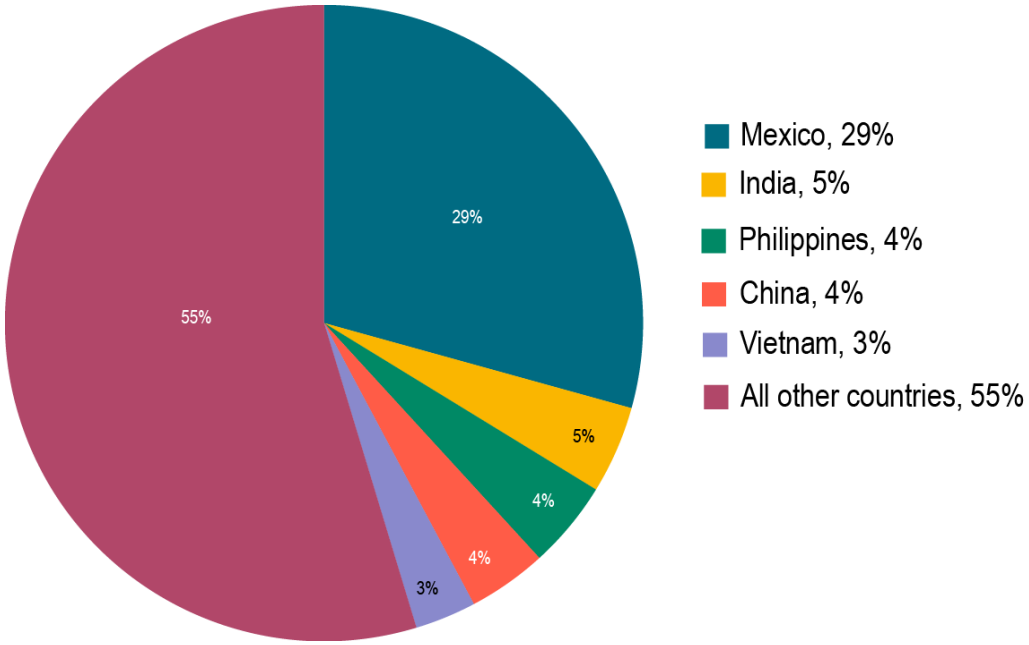
Immigrants arrive from diverse cultures shaped by unique social and cultural factors that inevitably transcend national borders. Not surprisingly, the prevalence of some health behaviors, including smoking, and specific factors associated with those behaviors, vary by country. For example, of the five countries with the largest number of immigrants in the United States in 2010, the prevalence of current tobacco smoking in 2009 was highest in the Philippines (28.3%)[11] and China (28.1%),[12] followed by Vietnam (23.8%),[13] Mexico (15.9%),[14] and India (14.0%),[15] which has roughly half the rate of the leading two countries (Table 7.2). A more striking pattern is apparent when these prevalence rates are examined by gender. In all five countries, a large proportion of tobacco users are men, with 52.9% of Chinese,[12] 47.7% of Filipino,[11] 47.4% Vietnamese,[13] 24.8% of Mexican,[14] and 24.3% of Indian[15] men classified as current smokers according to the 2009 Global Adult Tobacco Survey. In contrast, the average smoking rate among women in these countries is 4.7%.[11–15] However, in India, where as many as one in four people use smokeless tobacco (ST), men are only 1.7 times more likely to be ST users than women (32.9% versus 18.4%).[15] By way of comparison, the smallest male-to-female ratio among current tobacco smokers (as opposed to ST users) in the top five sending countries is 3.2 to 1 in Mexico,[14] and the largest ratio is 33.8 to 1 in Vietnam[13] (Table 7.2).

Table 7.1 Total and Country-Specific Foreign-Born Populations Living in the United States, 1960–2010

Year	Total Foreign-born	Mexico			India			Philippines			China			Vietnam		
		Number	%	Rank	Number	%	Rank	Number	%	Rank	Number	%	Rank	Number	%	Rank
1960	9,738,091	575,902	5.9	7	12,296	0.1	42	104,843	1.1	21	314,226	3.2	9	—	—	—
1970	9,619,302	759,711	7.9	4	51,000	0.5	30	184,842	1.9	12	299,202	3.1	9	—	—	—
1980	14,079,906	2,199,221	15.6	1	206,087	1.5	16	501,440	3.6	8	363,277	2.6	10	231,120	1.6	12
1990	19,797,316	4,298,014	21.7	1	450,406	2.3	12	912,674	4.6	2	583,513	2.9	7	543,262	2.7	9
2000	31,107,889	9,177,487	29.5	1	1,022,552	3.3	4	1,369,070	4.4	2	1,192,437	3.8	3	988,174	3.2	5
2010	39,955,854	11,711,103	29.3	1	1,780,322	4.5	2	1,777,588	4.4	3	1,601,147	4.0	4	1,240,542	3.1	5

Note: Rank refers to the order of countries having the largest number of foreign-born immigrants living in the United States.
Sources: Gibson and Jung 2006[96] and the U.S. Census Bureau 2010.[36]

Figure 7.1 Five Source Countries With the Largest Populations in the United States as Percentages of the Total Foreign-Born Population, 2010



Source: U.S. Census Bureau 2010.[36]

Table 7.2 Tobacco Use Behaviors and Knowledge Among Adults (%), by Country, 2009

	Mexico			India			Philippines			China			Vietnam		
Category	**All**	**Men**	**Women**	**All**	**Men**	**Women**	**All**	**Men**	**Women**	**All**	**Men**	**Women**	**All**	**Men**	**Women**
Tobacco use behaviors															
Current tobacco smokers	15.9	24.8	7.8	14.0	24.3	2.9	28.3	47.7	9.0	28.1	52.9	2.4	23.8	47.4	1.4
Daily tobacco smokers	7.6	11.8	3.7	10.7	18.3	2.4	22.5	38.2	6.9	24.1	45.4	2.0	19.5	38.7	1.2
Average number of cigarettes consumed	9.4	9.7	8.4	—	—	—	10.6	11.3	7.0	—	—	—	13.5	13.6	10.9
Smokeless tobacco users	0.3	0.3	0.3	25.9	32.9	18.4	2.0	2.8	1.2	—	—	—	4.8	—	—
Cessation															
Quit during past year among daily ever-smokers	32.0	31.6	33.1	12.6	12.1	16.2	21.5	20.9	25.0	—	—	—	23.5	23.3	28.6
Are interested in quitting	72.1	71.1	75.2	—	—	—	60.6	60.5	61.1	—	—	—	—	—	—
Secondhand smoke															
Exposed to tobacco smoke at work	19.7	23.3	13.9	29.9	32.2	19.4	36.9	43.3	28.8	63.3	71.1	53.2	55.9	68.7	41.4
Exposed to tobacco smoke at home	17.3	17.2	17.4	52.3	52.2	52.5	48.8	50.9	46.7	67.3	70.5	63.9	73.1	77.2	69.2
Media															
Noticed any advertisements for cigarettes	50.6	—	—	64.5	68.5	58.5	71.2	74.7	67.7	19.6	24.7	17.6	16.9	19.1	16.2
Knowledge															
Believe that smoking causes serious illness	98.1	—	—	90.2	91.5	88.8	94.0	93.1	94.9	—	—	—	95.7	—	—

Notes: Adults refer to people 15 years old and over. Values presented in table are percentages.
Sources: World Health Organization 2010[11–14] (country reports for the Philippines, China, Vietnam, and Mexico, respectively). International Institute for Population Studies 2010[15] (country report for India).

Impact of Acculturation and Assimilation to the United States

The prevalence rates of tobacco use and cessation and the proportions exposed to secondhand smoke and tobacco marketing, shown in Table 7.2, provide a broad indication of the often gendered tobacco-related contexts from which immigrants originate. In many Southeast Asian countries, smoking is an expected behavior among men, particularly older men, and an indicator of social status, whereas female smoking is socially unacceptable.[16,17] Similarly, smoking among females may not be fully accepted in some Latin American countries, but is often a sanctioned social activity among male peers.[18,19]

On arriving in the United States, immigrants are inevitably confronted with a different set of smoking-related norms and expectations. For example, among the overall U.S. population an estimated 15.1% of adults were current cigarette smokers in 2015, including 16.7% of men and 13.6% of women.[20] The U.S. prevalence of current smoking falls at the lower end of the range found in the top five source countries, and gender disparities are considerably less pronounced in the overall U.S. population than in those countries.

The effects on health and health behavior of navigating these varied and potentially conflicting norms and expectations are often discussed in the context of acculturation.[21] Although definitions vary, *acculturation* has commonly been defined as "the process of change that occurs within populations or societies because of interaction with other populations or societies, specifically with respect to [the] evolution of cultural traditions, customs, beliefs, or artifacts."[2,p.112] Acculturation does not just apply to individual immigrants, but has often been measured among immigrant populations. Acculturation can be posited as either a salutary process of increased economic and social mobility as immigrants more fully integrate into mainstream society or, conversely, as a harmful force in which residual protective effects of immigrant status (presumably related to "cultural orientation" and strong social networks) decline over the time spent in the United States.[22] In practice, of course, the incorporation patterns of recent immigrants are far more complex and multidimensional than the dichotomy just presented. What constitutes healthy versus unhealthy acculturation largely depends on which health outcomes are examined, for whom, and on the conditions in which acculturation occurs. Some initial discussion of both extremes, however, is instructive.

On the one hand, research has long documented a strong and consistent direct relationship between social position and health—often referred to as the *health gradient*—in which individuals of higher socioeconomic status (SES) have better health than those of lower SES.[23–31] For example, some immigrants have higher rates of poverty and lower educational attainment compared to non-immigrants,[1] although there are significant exceptions to this generalization. Poverty and educational attainment are some of the most common indicators of SES. Furthermore, immigrants often endure the added burden of living in hostile environments or resource-poor neighborhoods[31]; experience difficulties finding good, secure jobs in safe work settings[31]; and have inadequate access to social networks that could provide instrumental and emotional support.[30] All of these conditions have been linked to poor health, either directly (e.g., via stress processes or epigenetic changes[25,30,31]) or indirectly (e.g., via access to and mobilization of resources[31]). Taken together, these findings suggest that some immigrants will suffer from worse health than non-immigrants. A more in-depth discussion of the relationship between SES, smoking, and health is provided in chapter 9.

On the other hand, researchers often find that immigrants, especially Latino immigrants, are healthier (e.g., lower morbidity, mortality, and rates of low birthweight) than non-immigrants with similar socioeconomic profiles—what is commonly referred to as the *healthy immigrant effect* or, more specifically, the *Latino health paradox*.[2,32] Research has documented that Latino immigrants often appear to have a health advantage over non-Latinos and their U.S.-born counterparts, and that for certain outcomes, the protective effect of immigrant status also extends to immigrants of other racial/ethnic groups.[2] For some researchers, these better-than-expected health outcomes are rooted in the "cultural orientation" (presumably related to engagement in healthy behaviors) and strong social networks attributed to immigrants' countries of origin. In the process of acculturation, however, immigrants could be exposed to different risk factors or could adopt unhealthy behaviors and lifestyles (e.g., poor diet and physical inactivity) that result in shifts in morbidity and mortality for various diseases, leading in turn to declines in their overall health status.[33–35]

Although neither of the above scenarios is intended to capture all the complexities of immigrant adaptation or its effects on health, together they might provide clues about the potential smoking behavior of immigrants in the United States and differences within and across groups. For example, there is considerable variation in SES among immigrants from the top five source countries. Mexican immigrants tend to report low SES; in 2010, 60% of Mexican immigrants had less than a high school diploma. In contrast, almost one-quarter of Vietnamese immigrants (23%), nearly one-half of both Chinese and Filipino immigrants (44% and 50%, respectively), and about three-quarters of Indian immigrants (74%) had a bachelor's degree or higher, according to the 2010 American Community Survey.[36] In contrast, about 27% of all foreign-born and 28% of the total U.S. population had attained a bachelor's degree or higher.[36]

Hypothetically, immigrants from Vietnam, China, the Philippines, and India (especially those from the latter three countries) could adopt norms and expectations associated with higher SES in the United States, leading in turn to lower rates of tobacco use among men and higher rates among women as their smoking behavior becomes more consistent with that of their socioeconomic counterparts in the general U.S. population. Among Mexican immigrants, however, stronger orientation toward the culture of their home country could provide some initial protection against lower SES, but the combination of greater acculturation, norms linking smoking and social integration, and tobacco marketing targeted toward immigrants might serve to reinforce smoking behavior among Latino men while increasing tobacco use among women.

The remainder of this chapter examines whether the above conjectures reflect the existing empirical reality of immigrants and their smoking behavior.

Literature Search Strategy

A literature search was conducted to identify the current state of the science on smoking behavior of immigrants. Empirical studies examining the smoking behavior of immigrants were identified using the PubMed and Web of Science database search engines. Key search terms included immigration, nativity, acculturation, assimilation, foreign-born, "smoke or tobacco," "frequency or intensity," cancer, gender, age, "psychographic or lifestyle," and SES. For this literature review, searches were limited to articles published between 2000 and 2011, although several papers included in this review reference earlier studies on the smoking behavior of Asian American and Latino immigrants.[37–41] All journals in the databases were searched. When searches yielded many results, the fields were limited to more specific

subfields (e.g., title). After the searches were completed, all databases were merged (n = 1,282), and duplicate (n = 366) and irrelevant (n = 832) articles were removed, leaving a final sample of 84 articles. Of these, 59 articles focused specifically on studies that addressed the intersections of acculturation, gender, SES, and/or race/ethnicity with nativity and, to the extent possible, different aspects of smoking behavior. These 59 articles are summarized in Table 7.3 and discussed throughout the remainder of the chapter. An article may be discussed in more than one section if it examines multiple relationships within immigrant smoking behavior.

Acculturation, Immigrant Status, and Smoking Behavior

The process of immigrant acculturation is difficult to capture in most surveys available for health research, which are primarily cross-sectional. Thus, many studies use proxy measures for acculturation, such as nativity (U.S.-born versus foreign-born), length of U.S. residence, language preference, and generational status, whereas other studies use more formal acculturation scales often involving some combination of the above measures. The studies included in this review employed a range of measures for acculturation, and most of these studies documented an association between acculturation and smoking behavior, and in particular, current smoking.

Acculturation, Immigrant Status, and Current Tobacco Use – Adolescents

In the nine studies that involved adolescent samples,[38,42–49] acculturation was most often indirectly associated with tobacco use. For example, Allen and colleagues[42] found that Latino adolescents who spoke mostly Spanish with social network members were less likely to use tobacco, alcohol, and other drugs; however, when parental monitoring and the demographics of network members were controlled for, the association was no longer significant. Such findings suggest that less language acculturation could be indirectly associated with less substance use, including tobacco, through protective social network characteristics such as greater parental monitoring and more extended family members versus peers, and more adults versus peers of middle school age or younger in an adolescent's network.[50] Similarly, Castro and colleagues[43] found that higher ethnic pride and traditional family values had indirect effects on decreased cigarette and alcohol use among Latino adolescents. Unger and colleagues[49] reported that English language use was associated with an increased risk of lifetime smoking among both Latino and Asian American youths, but not after controlling for access to cigarettes, perceived consequences of smoking, friends' smoking, and cigarette offers. Studies by Lorenzo-Blanco and colleagues[44] and Trinidad and colleagues[48] further suggested that discrimination and emotional intelligence, respectively, could mediate the acculturation–smoking relationship during adolescence.

Rosario-Sim and O'Connell[45] found a positive relationship between greater English language acculturation and current smoking among Asian American adolescents. Choi and colleagues[38] also showed that acculturated Asian adolescents were two times more likely to smoke than non-acculturated youths. These studies suggest that acculturation is more often indirectly associated with smoking behavior among Hispanic/Latino adolescents but is more directly associated with smoking behavior among Asian adolescents.

Table 7.3 Summary of Reviewed Studies Examining Smoking Behavior Among Immigrants (n = 59)

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Abraido-Lanza et al. 2005[52]: To test the health behavior and acculturation hypotheses on smoking, alcohol use, exercise activity, and body mass index	National Health Interview Survey	Latino Americans	3,100	After adjusting for age and SES, higher acculturation was associated with current smoking, alcohol use, high body mass index, and more exercise.	—	—	—
Acevedo-Garcia et al. 2005[79]: To investigate the relationship between immigrant generation and daily smoking	Tobacco Use Supplement to the Current Population Survey (TUS–CPS)	U.S. general	221,798	Being foreign-born and being second generation with two immigrant parents had a protective effect. Being foreign-born was especially protective for females, low-income individuals, and racial/ethnic minority groups.	—	—	—
Allen et al. 2008[42]: To identify Spanish-language-sensitive individual and social network attributes associated with substance use, including tobacco, alcohol, and drug use	Original	Latino adolescents	258	Use of Spanish within an adolescent's social network was associated with a substance use scale in bivariate, but not multivariate models.	—	—	—
Al-Omari and Scheibmeir 2009[58]: To describe the relationship between tobacco dependence and acculturation	Original	Arab Americans	96	There was a significant inverse association between acculturation and tobacco dependence.	—	—	—
An et al. 2008[66]: To examine ethnic- and gender-specific smoking prevalence and quitting status and the effects of three acculturation indicators	California Health Interview Survey	Chinese, Filipino, South Asian, Korean, Japanese, and Vietnamese American adults	8,192	Men who used only English at home had lower current smoking prevalence and higher quit rates, except for Filipino and South Asian men. Women who used only English at home had higher current smoking prevalence, except Japanese women.	Women's current smoking prevalence was lower than men's in all six Asian American groups.	Less-educated men and women had higher smoking prevalence and lower quit rates.	Current smoking was higher, and the quit rate was lower for Korean, Filipino, and Vietnamese American men compared with Chinese American men.

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Bennett et al. 2008[82]: To examine the association between nativity and cigarette smoking	Harvard Cancer Prevention Program Project	Black Americans	667	Language acculturation was positively associated with cigarette smoking. U.S.-born blacks were more likely to be smokers than those born in the Caribbean or Africa.	—	—	—
Bethel and Schenker 2005[37]: To conduct a systematic review of published studies investigating the association of acculturation and smoking patterns	N/A	Hispanic Americans	26,611	9 of 11 studies showed a positive association between acculturation and smoking among women, and one study involving men showed a negative association.	—	—	—
Blue and Fenelon 2011[61]: To test whether different levels of smoking-related mortality can explain part of the "healthy immigrant effect" or the "Hispanic paradox"	Multiple Cause-of-Death Public-Use Microdata Files, U.S. Census 5% Public Use Microdata Sample Files, CDC tabulations of smoking-related mortality	Hispanic and non-Hispanic white Americans	2,392,452	Smoking explained >50% of the difference in life expectancy at 50 years between foreign- and U.S.-born men and >70% of the difference between foreign- and U.S.-born women.	—	—	Smoking explained >75% of the difference in life expectancy at 50 years between U.S. Hispanic and non-Hispanic white men, and close to 75% of the Hispanic advantage among women.
Bock et al. 2005[62]: To examine differences in cognitive and behavioral characteristics relevant to smoking cessation	Original	Latino Americans	615	Less-acculturated Latinos had higher cessation rates than bicultural and non-Latino whites.	—	—	Nicotine dependence and smoking rates were lower among Latinos than non-Latinos.

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Borrelli et al. 2011[84]: To examine differences in smoking attitudes and behavior	Original	Latino Americans	225	Compared to Dominicans, Puerto Ricans were more acculturated, more nicotine dependent, less motivated to quit, and identified more benefits of smoking.	—	—	Compared to non-Latino whites, Puerto Ricans smoked fewer cigarettes per day and reported greater pros of smoking, while Dominicans were less nicotine dependent, more confident of quitting, reported greater cons of smoking, and were more likely to have a home smoking ban.
Castro et al. 2009[43]: To examine the influence of ethnic pride, traditional family values, and acculturation on cigarette and alcohol use	Original	Latino adolescents	945	Higher ethnic pride and traditional family values had indirect effects on cigarette and alcohol use (i.e., they resulted in decreases of both). Greater ethnic pride had a direct effect on cigarette and alcohol use among girls (i.e., it decreased them). Greater acculturation predicted more cigarette and alcohol use among girls but not boys.	—	—	—
Castro et al. 2009[71]: To examine the influence of gender, acculturation, and their interaction on smoking cessation	Original	Latino Americans	271	Greater acculturation predicted higher abstinence rates but only among men.	—	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Chae et al. 2006[65]: To estimate the prevalence of current and lifetime smoking by gender, nativity, and other sociodemographic factors	National Latino and Asian American Study	Asian Americans	2,073	The prevalence of current smoking was higher among foreign-born vs. U.S.-born men, whereas U.S.-born women had a higher prevalence than foreign-born women.	—	—	—
Choi et al. 2008[38]: Meta-analysis to describe the extent to which acculturation affects smoking behavior	N/A	Asian Americans	16,759	Acculturated men were 53% less likely to smoke than non-acculturated men. Acculturated women were five times more likely to smoke than non-acculturated women.	—	—	—
Chou et al. 2010[97]: To examine indicators of health status and health care use by immigrant status	National Health Interview Survey (United States and Taiwan)	Chinese (in Taiwan) and Chinese Americans	1,217 (United States) 15,549 (Taiwan)	Chinese in Taiwan had higher odds of having ever smoked than recent Chinese immigrants. U.S.-born Chinese were more likely to report having ever smoked compared to new Chinese immigrants who had been in the U.S. <5 years.	—	—	—
Constantine et al. 2009[18]: To examine the relationship between acculturation, knowledge of smoking and health, and perception of the benefits of smoking	Original	Latino Americans	804	Greater acculturation was a significant predictor of perceiving the benefits of smoking.	Men perceived more benefits of smoking than did women.	Less than a high school education was a significant predictor of perceiving the benefits of smoking.	—
Constantine et al. 2010[17]: To explore the relationship between smoking and acculturation	Original	Americans of Hmong, Vietnamese, Lao, and Cambodian origin	1,628	Less-acculturated male respondents and more-acculturated female respondents were more likely to be smokers. Most male Hmong respondents started smoking after immigration.	—	Less education was associated with greater odds of being a smoker.	Vietnamese and Cambodian men smoked at higher rates than men in the general U.S. population.

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Cooper et al. 2011[55]: To examine smoking-related behaviors among young adult college students	Original	Hispanic Americans	174	Smokers were more likely to be less acculturated than nonsmokers.	—	—	—
de Castro et al. 2010[78]: To examine smoking prevalence by occupational classification, gender, and nativity	National Latino and Asian American Study	Asian Americans	1,528	Among Asian immigrants, smoking was highest among blue-collar workers.	Smoking prevalence was higher among males than among females.	Blue-collar employment was associated with being a current smoker.	—
Detjen et al. 2007[98]: To explore whether higher levels of acculturation were associated with higher rates of cigarette smoking during pregnancy	Latina Gestational Diabetes Mellitus Study	Hispanic women	1,231	Acculturation was associated with elevated smoking rates in pregnant women. U.S.-born women who preferred English had more than twice the odds of smoking compared with Puerto Rican or foreign-born women who preferred Spanish.	—	—	—
Elo and Culhane 2010[81]: To examine relationships between nativity and tobacco, alcohol, and marijuana use and measures of physical and mental health during pregnancy	Original	Black women	3,101	Foreign-born black women were less likely to engage in substance use and had better self-rated physical and mental health than U.S.-born black women. The foreign-born advantage was somewhat stronger for African-born women than for Caribbean-born women.	—	Women with higher levels of education were less likely to report use of tobacco.	—
Fitzgerald et al. 2006[99]: To study associations of acculturation and SES with obesity and lifestyle traits that could be risk factors for diabetes and cardiovascular disease	Original	Puerto Rican women	200	Less-acculturated women were 57% less likely to smoke than their more-acculturated counterparts.	—	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Fu et al. 2003[68]: To assess the relationship between linguistic aspects of acculturation and cigarette smoking	Original	Chinese Americans	541	Increased English proficiency was associated with decreased current smoking among men.	Smoking prevalence was higher for men than for women.	—	—
Gollenberg et al. 2008[100]: To assess dietary behaviors, physical activity, and cigarette smoking during pregnancy	Latino Gestational Diabetes Mellitus Study	Puerto Rican women	1,231	Spanish language preference was associated with approximately 40% less likelihood of smoking.	—	College education was associated with a lower likelihood of smoking.	—
Gonzales et al. 2006[101]: To assess the prevalence of home and automobile smoking bans on children's exposure to secondhand smoke by nativity	Original	Mexican American women	269	Children of U.S.-born mothers had increased odds of exposure to secondhand smoke indoors but not in automobiles.	—	—	—
Guevarra et al. 2005[83]: To reconfirm relationships between acculturation and cigarette smoking	Original	Black women	66	Results replicated the negative association between acculturation and lifetime smoking among African American women.	—	—	—
Hofstetter et al. 2004[67]: To examine the relationship between tobacco use and acculturation	Original	Korean Americans	2,830	Less-acculturated men and more-acculturated women reported higher present and predicted future rates of smoking.	More men than women reported smoking >100 cigarettes during their lifetime and smoking in the past 30 days.	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Hu et al. 2010[102]: To analyze the impact of immigration status on current tobacco use	Original	Chinese Americans	1,054	Smoking rates among recent immigrant men (<5 years in the U.S.) were significantly higher than in the general Texas population. U.S.-born men initiated smoking 4 years earlier than their immigrant counterparts.	Men's smoking rates were much higher than women's.	Lower household income and education increased smoking among males, but more-educated females had a tendency to smoke more than less educated females.	—
Juon et al. 2003[103]: To examine the prevalence and correlated factors of cigarette smoking	Healthy Korean American Project	Korean American men	333	Men in the U.S. for more than 20 years were less likely to be current smokers than those in the U.S. for less than 10 years.	Men were far more likely to be current smokers than women.	—	—
Kim et al. 2007[39]: To conduct an integrative review of the literature on tobacco use and dependence	N/A	Asian Americans	N/A	Smoking prevalence was higher among Asian American men with low acculturation, but the reverse pattern was observed among Asian American women.	Smoking rates were higher for men than women, regardless of country of origin.	—	—
Lee et al. 2000[104]: To examine how acculturation is related to smoking, physical activity, fat intake, body weight, and reported health	Original	Korean Americans	356	Bicultural men were least likely to smoke, while acculturated and bicultural women were more likely to smoke than traditional women.	Fewer women smoked and were former smokers than men.	—	—
Lorenzo-Blanco et al. 2011[44]: To examine whether perceived discrimination explained the associations of acculturation with depressive symptoms and cigarette smoking	Original	Hispanic adolescents	1,124	Discrimination explained the relationship between acculturation and cigarette smoking among girls (effect only marginally significant).	—	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Loury and Kulbok 2007[56]: To examine the relationship among sociodemographic, cultural, and psychological factors associated with alcohol and tobacco use in the rural South	Original	Mexican immigrants	173	Pre-immigration use of tobacco was significantly associated with current tobacco use. Acculturation level was not a significant single predictor of current tobacco use.	—	—	—
Ma et al. 2003[70]: To assess the impact of demographics and acculturation on stages of change in smoking behavior	Original	Asian Americans	1,174	Time living in the U.S. showed a significant positive correlation with the stages of smoking behavior change.	Women were much more likely than men to be in the precontemplation stage, while men were somewhat more likely than women to be in the preparation stage.	Education was negatively associated with stages of smoking behavior change.	Chinese respondents had the highest rate of those not wanting to quit (38%), while Cambodians (9%) had the lowest rate of those not wanting to quit.
Ma et al. 2004[73]: To examine the relationship between acculturation and smoking in homes	Original	Asian Americans	1,374	Living in the U.S. 5 or fewer years, experiencing less acculturation, and being foreign-born predicted smoking in the home and visitors being allowed to smoke there.	Being female predicted smoking in the home.	Being more educated protected against smoking in the home and against visitors being allowed to smoke in the home.	Being Korean, Vietnamese, or Cambodian predicted smoking in the home and visitors being allowed to smoke in the home.
Ma et al. 2004[69]: To assess knowledge, attitudes, and behaviors related to tobacco use and tobacco-related cancer issues	Original	Asian Americans	1,374	More-acculturated youths and less-acculturated male adults had higher smoking rates. Acculturated adult females had a higher smoking rate than those who were less acculturated.	Smoking rates for all females were generally lower than those for males regardless of acculturation status.	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Masel et al. 2006[54]: To determine if acculturation is associated with smoking, alcohol use, and physical activity	Hispanic Established Populations for the Epidemiologic Studies of the Elderly	Mexican Americans	4,901	Those who were more proficient in English or who had more contact with Anglo Americans were more likely to be former or current smokers than nonsmokers.	Many more males than females were smokers (former or current).	—	—
Maxwell et al. 2005[80]: To report prevalence rates and correlates of cigarette smoking in California	California Health Interview Survey	Chinese and Filipino Americans	53,907	Smoking rates were higher among foreign-born than U.S.-born Asian males. Acculturation is associated with increased smoking rates among women. Effect of acculturation was stronger for foreign-born Chinese men and for foreign-born Filipino women.	—	Having more than a high school education was protective except for Chinese females and Filipino males.	—
Maxwell et al. 2007[72]: To examine tobacco-related knowledge and attitudes, cessation efforts, and preferences for smoking cessation programs	Original	Filipino American men	318	Smokers were defined as less acculturated based on language use and English fluency. More than half of current smokers requested smoking cessation activities in Tagalog or a combination of Tagalog and English.	—	Smokers had lower levels of education and income compared with nonsmokers.	—
Myers et al. 2009[105]: To investigate baseline influences on initial smoking and the transition to established smoking among college students who had not smoked prior to college	Original	Chinese and Korean Americans	267	Acculturation was not a significant predictor of experimentation or established smoking.	Overall, men were significantly more likely than women to experiment and progress to established smoking.	—	Students of Korean ethnicity were more likely to become established smokers.
Parker et al. 2010[106]: To examine differences in tobacco use associated with acculturation	Electronic medical records	U.S. general	100,329	More-acculturated Hmong and Mexican women were more likely to be tobacco users. Among those who did not speak English, current tobacco use was more prevalent among men than women.	—	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Perez-Stable et al. 2001[53]: To examine differences in cigarette smoking behavior by gender and country of origin	Original	Latino Americans	8,882	Foreign-born respondents were less likely to be smokers than U.S.-born respondents. High acculturation was associated with more smoking in women and less smoking in men.	Current smoking was more prevalent among men than women.	Respondents with 12 years or fewer of education had increased odds of smoking.	Central American men and women had the lowest smoking rates; Puerto Rican women had the highest smoking rates.
Perreira and Cortes 2006[107]: To examine race/ethnicity and nativity correlates of substance use during pregnancy	The Fragile Families and Child Wellbeing Study	U.S. women	4,185	Foreign-born women were less likely to smoke during pregnancy than their U.S.-born counterparts.	—	Tobacco use during pregnancy was concentrated among less-educated and poorer white and black women, but not among Hispanic women.	Newborns with white mothers were most at risk of tobacco exposure. Black and Hispanic mothers were less likely than whites to smoke during pregnancy.
Reitzel et al. 2010[108]: To identify individual- and neighborhood-level variables predicting the association of subjective social status with acculturative and socioeconomic variables among immigrant smokers	Adiós al Fumar	Latino Americans	297	Less acculturation predicted low subjective social status among immigrant Latino smokers.	—	Low income and low education predicted lower subjective social status.	—
Rosario-Sim and O'Connell 2009[45]: To explore the correlates of smoking status	Original	Asian American adolescents	328	More English language acculturation was significantly associated with current smoking.	—	Having poor academic performance was significantly associated with current smoking.	Asian American adolescents initiated smoking later than non-Asian Americans.

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Saint-Jean et al. 2008[46]: To identify and evaluate sociopsychological factors that are associated with substance use.	Florida Youth Substance Abuse Survey	U.S. adolescents	63,000	Acculturation status was a strong predictor of substance use among adolescents.	—	—	—
Shankar et al. 2000[57]: To describe the prevalence of cigarette smoking	Original	Salvadoran immigrants	1,458	Smoking behavior exclusively represented the smoking pattern that the Salvadorans had adopted before immigration to the U.S.	Men were significantly more likely than women to have ever smoked.	—	—
Shelley et al. 2004[109]: To examine the relationship between acculturation and tobacco use behaviors	Original	Chinese Americans	712	Acculturation was positively associated with never smoking among men, but not with smoking cessation.	Being male was positively correlated with ever smoking.	Having less than a high school education was associated with ever smoking relative to never smoking.	—
Stoddard 2009[110]: To estimate the relationship between nativity and risk of initiation of regular smoking in children and young adults ages 10 to 30	National Health Interview Survey	Mexican Americans	61,358	Odds of smoking initiation declined among Mexican immigrants after immigration, relative to the risk before immigration to the U.S.	—	—	Mexican Americans and those born in Mexico were significantly less likely to ever initiate regular smoking than other racial and ethnic groups, except Asian Americans.

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Stoddard and Adler 2011[47]: To assess whether associations between education and smoking and leisure-time physical activity depend on nativity and age at immigration	National Health Interview Survey	Hispanic and Asian American adolescents	13,345 Hispanic 2,528 Asian American	For both Hispanics and Asians, smoking prevalence was higher among U.S.-born individuals than among foreign-born individuals. Associations between education and smoking among foreign-born Hispanics who had immigrated at an early age more closely resembled those of U.S.-born Hispanics than did education associations among Hispanics who had immigrated at an older age.	—	The association of education with smoking and physical activity was weaker for foreign-born Hispanics but did not vary by nativity for Asian Americans.	—
Sussman and Truong 2011[11]: To examine the effects of acculturation and gender on smoking attitudes and smoking prevalence	Original	Chinese and Russian immigrants	215 Chinese immigrants 149 Russian immigrants	More years living in the U.S. and more use of English led to more negative attitudes toward smoking. Acculturated immigrants were less likely to date, befriend, or marry smokers. Acculturated females had a more positive attitude toward smoking and were more likely to smoke, which is similar to U.S.-born females.	—	—	—
Tong et al. 2008[112]: To examine how the interaction between having a smoke-free home rule and immigrating to the U.S. is associated with quitting smoking	California Health Interview Survey	Asian Americans	1,050	Smoke-free home rules were associated with status as a former smoker, especially among recent immigrants (<10 years in the U.S.), and with lighter smoking in long-term residents (>10 years in the U.S.).	—	—	—
Trinidad et al. 2005[48]: To examine the effects of emotional intelligence and acculturation on smoking in early adolescents	Original	U.S. adolescents	416	A significant interaction between emotional intelligence and acculturation suggests that adolescents with high emotional intelligence could perceive more social consequences from smoking.	—	—	—

Table 7.3 continued

Study and aim(s)	Data source	Population(s)	Sample size	Acculturation findings	Gender findings	SES findings	Race/ethnicity findings
Unger et al. 2000[49]: To examine associations between English language use and smoking	Independent Evaluation Consortium	Hispanic and Asian American adolescents	4,167 Hispanic Americans 2,836 Asian Americans	English language use was associated with increased risk of lifetime smoking in both groups but not after access to cigarettes, perceived consequences of smoking, friends' smoking, and cigarette offers were controlled.	—	—	—
Wilkinson et al. 2005[51]: To investigate the effects of nativity, age at migration, and acculturation on smoking	Original	Mexican Americans	5,030	Higher acculturation predicted a history of smoking among U.S.- and Mexican-born participants. Younger age at migration predicted a history of smoking among those who are Mexican-born.	Male gender predicted a history of smoking among U.S.- and Mexican-born respondents.	Having more than a high school education predicted a history of smoking among the U.S.-born.	—
Zhang and Wang 2008[41]: A systematic meta-review to examine factors associated with smoking	N/A	Asian Americans	N/A	Acculturation was negatively associated with men's smoking but was positively associated with women's smoking.	Men were more likely to smoke than women.	Education was uniformly found to be negatively related to smoking.	—
Zinser et al. 2011[63]: To determine the extent to which smokers are using effective interventions for smoking cessation, especially NRT	Original	Latinos and non-Latino whites	1,010 Latino 519 white	Latinos reported using NRT less often than whites, and this difference was more pronounced among less-acculturated Latinos. The daily smoking rate was higher among highly acculturated Latinos than low-acculturated Latinos.	—	Latinos who had graduated from college were more likely to report NRT use, but this effect was significant only for the low-acculturation group.	Non-Latinos reported smoking significantly more cigarettes per day than Latinos.

Notes: Due to study design, two studies discussed in this chapter are not included in this table. A "—" indicates no findings on that topic. SES = socioeconomic status. NRT = nicotine replacement therapy.

Acculturation, Immigrant Status, and Current and Ever Smoking – Adults

The association between acculturation and smoking behavior was generally stronger and more persistent among adults than among adolescents. Studies of this association have reached different conclusions about whether greater acculturation is associated with greater likelihood of smoking. On the one hand, greater acculturation and younger age at migration were significant predictors of ever smoking among Mexican-born respondents in Wilkinson and colleagues'[51] study of smoking behavior. Abraido-Lanza and colleagues[52] found that higher acculturation among Latino adults was associated with current smoking after adjusting for age and SES. Perez-Stable and colleagues[53] showed that foreign-born Latinos were less likely to smoke than their U.S.-born counterparts. Similarly, Masel and colleagues[54] suggested that Mexican Americans who were more proficient in English or who had more contact with Anglo Americans were more likely to be former or current smokers than nonsmokers.

On the other hand, in a study of Hispanic college students, Cooper and colleagues[55] reported that current smokers were more likely to be less acculturated than nonsmokers. Similarly, Loury and Kulbok[56] suggested that tobacco use among Mexican immigrants in the rural South might be related to pre-migration behavior rather than the process of acculturation, a finding consistent with Shankar and colleagues,[57] who concluded that the smoking behavior of Salvadoran immigrants represented patterns adopted before immigration.

Another study by Al-Omari and Scheibmeir[58] found that less-acculturated Arab Americans were more tobacco dependent than acculturated Arab Americans. The prevalence of current tobacco use in some Arab countries (including Iraq, Egypt, the Palestinian Territories, Lebanon, Jordan, Syria, Tunisia, and Yemen) was as high as 63.6% for men in 2012 (Jordan)[59] and 34.0% for women in 2014 (Lebanon).[60] According to the Al-Omari and Scheibmeir[58] study, in many Middle Eastern countries cigarette smoking is an acceptable social and cultural behavior, and the offer of a cigarette is considered a sign of hospitality.

Acculturation, Immigrant Status, Current Smoking, and Mortality

Estimates by Blue and Fenelon[61] suggest that, in 2000, smoking explained at least 50% of the difference in life expectancy at age 50 between foreign- and U.S.-born men and at least 70% of this difference in women (i.e., the "healthy immigrant effect"). Smoking explained greater than 75% of the difference in life expectancy at age 50 between U.S. Hispanic and non-Hispanic white men and close to 75% of this difference in women (i.e., the "Latino health paradox"). Therefore, the authors concluded that lower smoking-related mortality was the main reason for this longevity advantage enjoyed by immigrants and U.S. Hispanics. Such an advantage could be attributable to lower rates of current smoking among less-acculturated Hispanic immigrants, as documented by many of the aforementioned studies. Another possible cause of this longevity advantage among Hispanic immigrants, as reported by Constantine and colleagues,[18] could be the fact that more-acculturated Latino Americans perceive more benefits from smoking, which could lead in turn to higher rates of smoking and greater smoking-related mortality among these more-acculturated Latinos.

Acculturation, Immigrant Status, and Smoking Cessation

Smoking cessation among less-acculturated immigrants could also contribute to their low rates of current smoking. Bock and colleagues[61] found higher overall cessation rates among less-acculturated Latinos compared with bicultural and non-Latino whites. Regarding specific smoking cessation

techniques, Zinser and colleagues[63] found that Latinos, especially less-acculturated Latinos, were less likely than non-Latino whites to use nicotine replacement therapy. However, Wetter and colleagues[64] found that a telephone-based smoking cessation intervention could reach, retain, and deliver efficacious treatment to a sample of low-SES, Spanish-speaking smokers, most of whom (90%) were immigrants.

Gender, Acculturation, Immigrant Status, and Smoking Behavior/Outcomes

Gender, Acculturation, Immigrant Status, and Current Smoking

Among adults of Asian descent, the effects of acculturation on smoking behavior appear more gender-specific, although similar patterns emerged in one systematic review[37] and a study of Latino and Asian Americans.[65] For example, according to An and colleagues,[66] greater English language acculturation appeared to increase the risk of cigarette smoking among women from China, the Philippines, South Asia, Korea, and Vietnam but not from Japan, and it decreased current smoking prevalence among men of Chinese, Japanese, Korean, and Vietnamese backgrounds, but not among Filipino or South Asian men. Moreover, Hofstetter and colleagues[67] showed that among Korean Americans in California, less-acculturated men but more-acculturated women reported higher current and predicted future rates of smoking (the latter based on a measure of smoking uptake). Similarly, Constantine and colleagues[17] showed that less-acculturated Hmong, Vietnamese, Cambodian, and Laotian men were more likely to be current smokers, whereas the opposite was observed among their female counterparts. However, these authors also noted that most Vietnamese, Cambodian, and Laotian men started smoking prior to immigration to the United States, whereas most Hmong men initiated smoking after immigration. In their study of Chinese Americans in Philadelphia, Fu and colleagues[68] found that greater English language proficiency was associated with decreased current smoking among men, but there were too few current smokers to conduct a similar analysis among females.

In a meta-analysis of nine studies published between 1994 and 2005, Choi and colleagues[38] showed that acculturated Asian men were 53% less likely to smoke than non-acculturated (traditional) men, whereas acculturated Asian women were five times more likely to smoke than traditional women. This general pattern was echoed in studies by Kim and colleagues,[39] Ma and colleagues,[69] and Zhang and Wang.[41]

Gender, Acculturation, Immigrant Status, and Smoking Cessation

In terms of behaviors other than current smoking, Ma and colleagues[70] showed that among Korean, Chinese, Vietnamese, and Cambodian smokers in the Delaware Valley region, time living in the United States (0–2 years versus 3 or more years) was positively correlated with being further along in three stages of smoking behavior change (precontemplation, contemplation, and preparation), particularly among men. Women were much more likely than men to be in the precontemplation stage, whereas men were somewhat more likely than women to be in the preparation stage (i.e., more inclined to plan to quit within 1 month). Immigration status (i.e., U.S. citizen, permanent resident, noncitizen) did not have a statistically significant effect on readiness to quit smoking in this study.

Castro and colleagues[71] showed that among Latino smokers, time living in the United States, proportion of life in the United States, and preference for English language media significantly predicted higher rates of abstinence, but only among men. However, despite the seemingly positive association between acculturation and inclinations to quit, Maxwell and colleagues[72] found that more than half of Filipino American men requested smoking cessation interventions in Tagalog or a combination of Tagalog and English.

Gender, Acculturation, Immigrant Status, and Smoking at Home

Ma and colleagues[73] showed that the following characteristics predicted smoking in the home: 5 or fewer years living in the United States; lower level of acculturation; being female; being foreign-born; having a family size of four or more; being Korean, Vietnamese, or Cambodian (versus Chinese); and being smokers. These same variables, except female gender, also predicted allowing visitors to smoke in the home.

Gender, Acculturation, Immigrant Status, Smoking, and Cancer Mortality

A study by Gomez and colleagues[74] showed that, among Asian/Pacific Islander and Latina women who had never smoked, immigrant women had a slight advantage in their rates of surviving lung cancer compared with their U.S.-born counterparts, but not compared to non-Hispanic whites.

Socioeconomic Status, Acculturation, Immigrant Status, and Smoking Behavior

It is widely accepted that higher SES is associated with improved health outcomes, including lower morbidity and mortality and better self-rated health and quality of life.[75–77] As indicated in chapter 9, which explores the relationships between SES and smoking in greater detail, educational attainment is one of the most widely used indicators of SES and consistently demonstrates a positive association with health. Education is often the route to economic and social rewards. Progressing through the educational pipeline often leads to better quality jobs, more secure jobs in safe work environments, more opportunities to enhance income, greater capacity to increase wealth, and access to a wider range of social networks that provide instrumental and emotional support, all of which can influence smoking behavior.

Education, Acculturation, Immigrant Status, and Current Smoking

Few studies have examined the relationships between education, acculturation, and smoking. An and colleagues,[66] in a study of Californians of Asian descent, found that less-educated men and women had higher prevalence of current smoking than their more-educated counterparts, when indicators of acculturation such as the following were controlled for: generational status (first generation: born in foreign countries; second generation and above: born in the United States), time living in the United States, and language spoken at home. Constantine and colleagues[17] showed a similar relationship: Having less than a high school education significantly increased the odds of being a smoker, when controlling for acculturation as measured by fluency with U.S. culture. However, Stoddard and Adler[47] show a slightly different association between education and smoking in their study of Hispanic and Asian Americans. They found that the association between education and smoking held for foreign- and U.S.-born Asian Americans but was considerably weaker among foreign- versus U.S.-born Hispanics.

Education, Acculturation, Immigrant Status, and Smoking Cessation

The above-mentioned study by An and colleagues[66] found, after controlling for indicators of acculturation such as generational status, time living in the United States, and language spoken at home, that less-educated men and women also had lower quit rates than their more-educated counterparts.

Occupation, Employment Status, Acculturation, Immigrant Status, and Current Smoking

Examining occupational status, another commonly used measure of SES, de Castro and colleagues[78] showed that, among Asian and Latino immigrants in the labor force, smoking prevalence was highest among blue-collar workers and lowest among white-collar workers. Notably, immigrants with blue-collar jobs had the highest smoking rates of any occupational status, and individuals who were unemployed had the highest rate among those who were U.S.-born. The relationship between occupation and smoking is explored in greater detail in chapter 8.

Income, Acculturation, Immigrant Status, and Current Smoking

Little research has focused on relationships between income, acculturation, and smoking frequency. Acevedo-Garcia and colleagues[79] reported that being foreign-born and being second generation with two immigrant parents were protective against daily smoking, especially among low-income individuals.

Immigrant Ethnicity and Smoking Behavior

Ethnicity and Current Smoking

Measures of health and health behavior, including tobacco use, vary in important ways for different ethnic groups. More-nuanced analyses by ethnicity or country of origin provide an imperfect but useful means of understanding immigrants in the context of the cultural norms of their countries of origin, and of linking immigrants to appropriate social services. The current review included several studies that delineated smoking behavior by ethnicity, primarily among Asian groups. For example, in the previously mentioned comprehensive analysis of acculturation, gender, ethnicity, and smoking behavior among Chinese, Filipino, South Asian, Japanese, Korean, and Vietnamese adults in California, An and colleagues[66] showed that current smoking prevalence varied from 14.6% to 36.7% among men and from 1.7% to 13.2% among women. Men of Korean, Vietnamese, and Filipino descent reported higher smoking prevalence than Chinese American men, while women of Filipino, Japanese, and Korean ancestry reported higher smoking prevalence compared with Chinese American women. Constantine and colleagues[17] also found that Vietnamese and Cambodian (but not Hmong or Laotian) men in Minnesota smoke at higher rates than men in the general U.S. population and, as previously mentioned, that most Hmong men started smoking after immigrating to the United States.

According to Maxwell and colleagues,[80] the positive effect of acculturation on smoking among men was stronger for foreign-born Chinese men than Filipino men, and the negative effect of acculturation on smoking among women was stronger for Filipina women than Chinese women. Elo and Culhane[81] further examined ethnic variation among black women in Philadelphia in terms of tobacco, alcohol, and marijuana use during pregnancy and showed that foreign-born black women were less likely to engage in substance use than U.S.-born black women. Similar findings were also reported by Bennett and colleagues[82] and Guevarra and colleagues.[83] Regarding ethnicity more specifically, this foreign-born advantage was somewhat stronger for African-born women than for Caribbean-born women.[81]

Among Hispanics/Latinos in the United States, Perez-Stable and colleagues[53] found that Puerto Rican and Cuban respondents were significantly more likely to be current smokers and to smoke more than 20 cigarettes per day compared with Mexican American respondents, controlling for age, gender, SES, nativity status, and acculturation. No statistically significant differences were observed between Central or South American respondents and Mexican American respondents.

Ethnicity and Smoking Cessation

American men of Korean, Vietnamese, and Filipino descent reported lower quit rates than Chinese American or Japanese American men, while women of Korean or Chinese ancestry reported lower quit rates than Vietnamese, Filipina, or Japanese American women.[66] Borrelli and colleagues[84] found that compared to Dominicans, Puerto Rican caregivers who smoke and have a child with asthma were significantly more acculturated, more nicotine dependent, less motivated and confident of quitting, and identified more benefits of smoking, although Puerto Ricans smoked fewer cigarettes per day than their non-Latino white counterparts.

Ethnicity and Secondhand Smoke Exposure

Differences in the potential for secondhand smoke exposure can be seen in results of the study by Ma and colleagues,[73] which showed that Americans of Korean, Vietnamese, and Cambodian descent were more likely than Chinese Americans to smoke in the home and to allow visitors to smoke in the home.

Chapter Summary

In 2015 almost 42 million people, representing 13% of the U.S. population, were born outside the United States. In 2010 about half of all foreign-born people in the United States came from five countries (Mexico, India, Philippines, China, and Vietnam). In these countries, social norms and behaviors regarding tobacco use are quite different from those in the United States. For example, the prevalence of current smoking is generally lower in the United States than in these five countries, and male/female smoking rates are far more similar. In addition to differences in norms and behaviors regarding tobacco use, immigrants to the United States bring diverse cultures, languages, religions, social classes, and reasons and processes for migration. These and other factors make the study of immigrant status, the tobacco use continuum (initiation, current use and intensity, intentions to quit and quit attempts, cessation, relapse, and tobacco-related morbidity and mortality), and tobacco-related health disparities quite complex.

In this review of 59 studies, most of the evidence on different aspects of immigration status, gender, SES, and race/ethnicity focused on the relationship between immigration status and current smoking. Some studies reported data by the aggregate race/ethnicity category (e.g., Asian) or by specific ethnic group (e.g., Mexican). Some studies examined acculturation using formal scales, while others used proxy measures such as foreign-born versus U.S.-born, English language use, and ethnic identity. It should be noted that this review did not include refugee populations as a specific focus.

The literature demonstrates that, in general, foreign-born men are more likely to smoke than their U.S.-born counterparts; conversely, foreign-born women are less likely to smoke than U.S.-born women. The evidence suggests that for youth and adults, acculturation is both a risk and a protective factor for current cigarette smoking behavior, but the direction of the relationship depends on gender, ethnicity, and the intersection of gender and ethnicity. The relationship between acculturation and the intensity and frequency of tobacco use is not yet clear. Few studies have examined the relationship between SES and smoking among foreign-born individuals; studies on SES identified for this review focused primarily on the Asian and Hispanic aggregate racial/ethnic groups. These studies suggest that high SES is protective against smoking in some immigrant ethnic groups but a risk factor for smoking in other groups, with the relationship depending on whether education, income, or occupation is examined.

As described further below, there are many gaps in the literature. Few studies have examined whether immigration status is associated with smoking initiation among youth from different ethnic groups. Research is needed to examine the relationship between acculturation and smoking cessation and the direction of the relationship among different immigrant groups, as well as the question of whether there is a relationship between acculturation and smoking relapse. Studies are also needed to determine the relationship between a person's country of origin (that is, whether born in the United States or another country) and tobacco-related cancer morbidity and mortality. Finally, studies are needed on the relationship between sexual identity, immigrant status, and smoking status.

Research Needs

Research on immigration and tobacco use would benefit from further study of the social/contextual factors that are especially relevant to immigrants. This section presents three research topics that are particularly important. First, *the issue of time spent in the United States* should be investigated more systematically in longitudinal studies of smoking. Only a handful of the 59 studies reviewed for this chapter examined smoking behavior, usually smoking cessation, over time. A few more studies analyzed cross-sectional data from longitudinal data sets. A life-course perspective argues that the timing of events can shape future events, especially as they relate to social mobility, marriage and having children, and health trajectories. Time spent in the United States is often conceptualized as a critical factor for immigrants, especially as they adjust to life in a new country. A widely held assumption is that the longer immigrants stay in a new society, the more likely they are to expand their social networks, which in turn can promote greater social and economic opportunities. Other temporal factors are also critical in determining how immigrants manage life in their communities.[85–87] One particularly neglected dimension is the age at which immigrants come to the United States, although this is sometimes captured as part of acculturation scales. Age at immigration can be conceptualized as the developmental context of an individual's experiences at the time of arrival in the United States. Seen in this light, age at immigration has the potential to play a powerful role in delineating how a person negotiates life in a new country and a different culture. In addition, consideration of arrival cohort is important because of changes in smoking patterns over time in both the United States and country of origin.[88]

The contexts in which people immigrate help shape language ability and use, density and heterogeneity of social networks, place of residence, and exposure to risky behaviors and stressful environments. The immigrant's generation further frames the immigration experience into unique opportunities and challenges occurring during discrete developmental periods. The social institutions that affect peoples' lives can vary depending on their generational status, leading to different life-course trajectories[85,86]; different social institutions provide access to unique types of social networks and relationships. Social networks provide a mechanism by which structural characteristics of society exert influence on individuals.[89,90] Social networks can be seen as opportunity structures because when a person's social ties have access to societal resources and opportunities, the person has greater access to those resources and opportunities. The number of social groups and institutions (e.g., schools, clubs, friendship networks, family ties) geared toward teaching children about the new society is far greater than those available for adults, giving children greater access to the opportunity structures in a new culture. Conversely, because immigrant children can have a larger set of social groups available to them, they could also experience a greater amount of negative stressors and influences which could lead to negative social and health outcomes as they mature. Chapter 5 provides an in-depth discussion of stressors, and chapter 6 discusses the influence of social relationships on tobacco use and disparities.

A second priority research topic is *the link between SES and smoking in immigrant populations* (SES and TRHD are explored in greater depth in chapter 9). Some studies show that the relationship between education and smoking has a more pronounced effect for U.S.-born individuals than for immigrants. It is possible that educational attainment, country of educational attainment, and other SES indicators might not fully measure social advantage or inequalities among immigrants or ethnic minorities. Other studies have shown that SES measures, including education, do not have the same predictive power for immigrants and ethnic minorities that they have for whites in the United States.[91,92] As researchers refine current measures of SES, it might also be useful to examine alternative indicators of SES that could describe social hierarchies in different groups. Some of these possibilities include studies of wealth, spatial stratification, and perceptions of social status. Studies of alternative indicators could enhance our understanding of how social inequalities are associated with smoking and could also provide insights about targeted smoking cessation and prevention efforts.

The final priority research topic is *tobacco industry marketing strategies that target immigrants*. Acevedo-Garcia and colleagues[93] describe three distinct marketing strategies aimed at immigrants: (1) geographically based marketing directed toward immigrant communities; (2) segmentation based on assimilation status; and (3) coordinated marketing based on immigrant country of origin. Each of these strategies has the potential to maintain the high levels of smoking among immigrant men, reduce the quit rate among immigrants overall, and encourage the initiation of smoking among immigrant women. These strategies could also encourage smoking initiation among immigrant adolescents. Systematic research is needed to determine the effects of tobacco marketing approaches within immigrant communities and across immigrant groups. An extensive discussion of the role of communications and marketing in TRHD is presented in chapter 10.

Given the lack of evidence on the relationship between immigration status and smoking behavior, to better understand disparities and inform appropriate interventions, additional research is needed on tobacco use by immigrants across the tobacco use continuum. Research and data are especially limited on immigrants and TRHD. Moreover, although immigration status is relatively easy to measure, the mechanisms linking immigrants to smoking and TRHD, including acculturation and assimilation, are considerably more difficult to measure and interpret. In particular, although articles and data can be found for smoking behavior, few published studies are available on how or to what extent smoking is linked to different health outcomes among immigrants. More research on immigration and tobacco-related morbidity and mortality is appropriate, particularly in light of the relatively high rates of tobacco use among some immigrant groups. Most studies on immigrants and tobacco use tend to be cross-sectional; longitudinal studies are needed in order to investigate the process of migration and adaptation that leads to smoking, especially among immigrant women.

Prevention and cessation programs are needed for all populations, including immigrant populations who may not have access to resources because of their immigrant status. Barriers could include a lack of (1) bilingual clinicians, (2) adequate financial resources, (3) access to mental health services, (4) culturally congruent treatment approaches, and (5) ethnically congruent counselors.[94] The enduring nature of these barriers reflects the complexities that confront any intervention. As noted in the 2008 Public Health Service Guidelines, additional studies are needed on the ability of culturally tailored interventions, compared with generic interventions, to enhance cessation interventions.[95] Finally, research is needed to determine how to prevent increased smoking among immigrant women, whose use rates are often low, so as to protect the health of women, children, and families.

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Section II
Intrapersonal/Individual Factors Associated with Tobacco-Related Health Disparities

Chapter 8
Occupation, the Work Environment, and Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

Occupation is an important indicator of socioeconomic status (SES) that independently affects health.[1–6] Occupation provides a measure of SES for those in the labor force, and is an important source of income and social status along with other benefits, such as a means to build social networks, social support, and self-esteem.[7–9] Occupation might also introduce threats to health through exposure to hazardous working conditions or harmful psychosocial experiences on the job.[7,10–16] Healthy People 2020 provides science-based, 10-year national objectives for improving the health of Americans, including objectives to address the social determinants of health and reduce tobacco use and secondhand smoke (SHS) exposure. Specific Healthy People 2020 goals aim to increase the proportion of individuals covered by indoor worksite policies that prohibit smoking.[17]

This chapter examines occupational disparities across the tobacco use continuum, the causal pathway in the progression of smoking to disease including initiation, current use and intensity, intentions to quit and quit attempts, cessation, relapse, and tobacco-related morbidity and mortality. This review also examines the contributions of the work environment and job experiences to disparities in tobacco use. Differential exposures to toxicants on the job are possible mediators through which the work environment and job experience, which are differentially patterned and imbued with advantages and disadvantages based on social class, are associated with tobacco-related health disparities (TRHD).

Disparities by occupation can interact with other indicators of social disadvantage so that workers who experience multiple sources of disadvantage could be at greater risk for tobacco use and tobacco-related diseases. Accordingly, this chapter explores the intersections between occupation and race/ethnicity, gender, age, and sexual orientation. Given the influence of the work environment, working conditions, and the social status that can be related to employment, employment status is also reviewed. Conclusions are based on a systematic review of the literature and may inform future research needs and steps to ameliorate TRHD.

Occupation as an Indicator of SES

Occupation connects two other key indicators of SES, education and income, because educational attainment provides the qualifications that often result in one's occupation, which in turn influences the level of income one is likely to earn.[1] The advantages of using occupation as a measure of SES include the fact that it reflects both income across the life span as well as job characteristics, such as power (authority and decision-making) and working conditions.[18] However, the use of occupational status as a unique SES measure in research has been criticized because of the potential for reverse causation with health (i.e., health status may influence occupational status); the potential for change across the life span; its inutility as a measure of SES for those outside the formal work sector; and its interplay with other demographic factors, such as race, ethnicity, and gender.[18] Nonetheless, although income and education have also been related to health across multiple studies, occupation has been shown to be an independent determinant of health status and health behaviors.[1–3,19–21]

Multiple plausible pathways link occupation to health. Occupational class can influence health by differential access to quality health care/health services and by varying levels of material deprivation and psychosocial resources.[18,22] Occupation can also cause poor health, by exposing workers to environmental, chemical, ergonomic, and psychosocial hazards. Workers in lower class jobs often have higher levels of exposure to some of these hazards, which can lead to the higher rates of mortality from cardiovascular disease and cancer.[22,23] Finally, occupation and the work context are associated with

health behaviors, including tobacco use. For example, blue-collar workers are more likely to smoke than white-collar workers.[1,24]

Occupation can affect tobacco use patterns and health outcomes in several ways. Some occupations involve risks of hazardous exposures and harmful psychosocial experiences on the job. The effects of working in occupations that pose health risks can interact with tobacco use either synergistically or additively, to substantively increase health risks.[7,10–12,25–28] For example, smoking and exposure to asbestos interact synergistically to increase the risk of lung cancer above what would have occurred from either exposure separately.[29,30] In addition, a study suggests workers exposed to hazards on the job are less likely to intend to quit smoking, perhaps in part because they perceive cessation as futile in the face of other health hazards posed by the work environment.[31] Much of the literature on the influence of the psychosocial work environment on tobacco use focuses on a stress-mediated pathway; smoking can represent a perceived means of coping with on-the-job stressors.

Some workplace benefits are differentially distributed by occupation and industry; for example, social norms supporting nonsmoking can be more prevalent among white-collar workers than blue-collar workers and more prevalent among some blue-collar workers than others. Additionally, white-collar workers are less likely to be exposed to SHS at their workplaces than blue-collar or service workers.[32,33] Yet occupation alone does not paint a full picture of tobacco-related disparities at worksites, as occupations can be dominated by a certain gender or race/ethnicity. For instance, whites and Asian Americans are more often in management or professional jobs compared with African Americans and American Indians/Alaska Natives.[34] Further exploration of these additional and interacting influences on tobacco-related disparities is needed.

Literature Search Strategy

Using the PubMed and Web of Science Social Science Citation Index (SSCI) databases, a literature search was conducted of articles published in English between 2005 and 2011, with some exceptions to the date limit, as noted below. The main search terms included "blue-collar," cessation, cigar, "electronic cigarettes," employment, job, occupation, quitting, "smokeless tobacco" (ST), smoking, "smoking initiation," "smoking and work," snus, tobacco, unemployment, and workers. Income and education, which also relate to occupation, are addressed in chapter 9.

The same search terms were used to search both databases. Most searches were conducted across all fields in PubMed and in the title, keyword, and abstract fields in SSCI. When search terms retrieved many irrelevant articles, the search was limited to certain fields only, such as title or keyword. If a search revealed few results, older articles were reviewed for relevance. Additional key references were added for the years 2012 to 2015. After a database search was completed, irrelevant and duplicate articles were removed, resulting in a total of 515 articles.

For purposes of this chapter, "occupational class" generally refers to blue-collar, white-collar, and service jobs. "Occupational profession and industry" represents a finer breakdown of occupational class by profession and industry, which tends to vary among studies. Examples of professions include construction workers, laborers, fabricators, food service personnel, health-diagnosing occupations, and teachers; examples of industries include construction, food service, retail trade, mining, finance, and educational services. "Employment status" describes the labor force participation of individuals.

Employed individuals are people who worked for pay in the last week, whereas unemployed individuals did not work in the last 4 weeks but are actively looking for jobs.

As this monograph focuses on documenting TRHD in the United States, the bulk of the studies reported here pertain to U.S. populations. To improve relevance to the overall U.S. population, this review focuses on studies using nationally representative data, with some exceptions, especially in areas with fewer studies and those that focus on morbidity and mortality. National surveys on health typically exclude the military in their sampling plans, so these populations are not necessarily represented in this review. Previous reviews of the literature are also used. Where available, reporting focuses on multivariate analyses as indicative of the most robust associations. Some studies are mentioned in more than one category because they analyzed multiple outcomes.

Disparities Across the Tobacco Use Continuum, by Occupational Characteristics

The purpose of this section is to describe disparities by occupational class, occupational profession and industry, and employment status across the tobacco use continuum of initiation, current use, quit attempts, cessation, and relapse. To the extent possible, information is included on the intersections between occupation and employment and other population characteristics, such as gender, age, race/ethnicity, and sexual orientation. Although most research focuses on cigarette use, research on use of other tobacco products and exposure to SHS are included as available. Gaps in the existing literature are noted.

This chapter reviews several main studies that used nationally representative data to investigate cigarette use by occupational class across the tobacco use continuum between 2004 and 2015; these studies are summarized in Table 8.1. This section focuses on cigarette use and occupational class because studies of these factors are the most robust and numerous and make use of comparable occupational groupings. Relatively few studies have addressed this area of research. As Table 8.1 shows, the reviewed studies found that blue-collar and service workers have significantly higher odds ratios of initiation and current cigarette use than white-collar workers. The literature is less consistent about differences in quit attempts and cessation by occupation, as fewer studies have reported multivariable regression findings by occupational class, and their definitions of quitting vary. The existing studies suggest that white-collar workers have somewhat higher odds of making a quit attempt and quitting successfully than blue-collar workers; the data on service workers are less clear. One study found that construction workers had significantly higher rates (53.3%) of persistent smoking compared to all other occupations combined (41.7%).[35] No studies looked at relapse by occupational class.

Table 8.1 Cigarette Use Across the Tobacco Use Continuum, Nationally Representative Data, by Occupational Class

Continuum phase Author(s)	Data source, year, n, ages	Prevalence (%)				Odds ratio/relative risk (95% confidence interval)			
		White-collar	Blue-collar	Service	Other	White-collar	Blue-collar	Service	Other
Initiation (cigarette smoking prevalence)									
Ham et al. 2011[35]	TUS-CPS 2006-2007 n = 106,604 Ages 18–64	12	24	20	26*	1.00†	1.18 (1.15–1.22)	1.07 (1.04–1.10)	—
Current use (cigarette smoking prevalence)									
Barbeau et al. 2004[1]	NHIS 2000 n = 24,276 Ages 18–64	20	35	31	24‡	1.00	1.28 (1.15–1.41)	1.19 (1.05–1.36)	0.72 (0.55–0.94)
Ham et al. 2011[35]	TUS-CPS 2006-2007 n = 106,604 Ages 18–64	12	24	18	26*	1.00†	1.41 (1.34–1.49)	1.25 (1.18–1.32)	—
Fagan et al. 2007[43]	TUS-CPS 1998-1999, 2001-2002 n = 288,813 Ages 18–64	18	33	27	24‡§	1.00	1.31 (1.27–1.35)	1.15 (1.10–1.20)	—
Lawrence et al. 2007[45]	TUS-CPS 1998-1999 n = 15,394 Ages 18–24	23	35	32	21‖	1.00	1.50 (1.32–1.70)	1.62 (1.42–1.84)	1.11 (0.88–1.41)
Asfar et al. 2016[49]	NHIS 2010 n = 1,531 Ages 18–24	18	25	24	—	1.00	1.40	1.36	—
Quit attempts (stopped smoking for 1 day+ in last 12 months)									
Alexander et al. 2010[71]	TUS-CPS 2006-2007 n = 30,176 Ages ≥18¶	52	51	56	—	1.00	0.87 (0.73–1.10)	0.80 (0.69–0.94)	—
Barbeau et al. 2004[1]	NHIS 2000 n = 24,276 Ages 18–64	45	42	47	43	—	—	—	—
Ham et al. 2011[35]	TUS-CPS 2006-2007* n = 106,604 Ages 18–64	46	39	42	38*	1.00†	0.94 (0.88–1.01)	0.99 (0.92–1.06)	—
Asfar et al. 2016[49]	NHIS 2010 n = 1,531 Ages 18–24	58	58	54	—	1.00	0.70 (0.46–1.06)	0.85 (0.51–1.40)	—

Table 8.1 continued

Continuum phase / Author(s)	Data source, year, n, ages	Prevalence (%) White-collar	Blue-collar	Service	Other	Odds ratio/relative risk (95% confidence interval) White-collar	Blue-collar	Service	Other
Cessation (prevalence of former smokers)									
Barbeau et al. 2004[1]	NHIS 2000 n =,276 Ages 18–64	20	18	14	17‡	—	—	—	—
Fagan et al. 2007[43]	TUS-CPS 1998-1999, 2001-2002 n = 288,813 Ages 18–64	19	18	14	16‡§	1.00	0.80 (0.76–0.83)	0.81 (0.77–0.85)	—
Relapse									
None									

Notes: TUS-CPS = Tobacco Use Supplement to the Current Population Survey. NHIS = National Health Interview Survey. Em dash (—) = not applicable, or insufficient data.
*Other industry was construction.
†Relative risk presented.
‡Other industry was farming.
§Other industry was forestry and fishing.
‖Unknown or refused to answer.
¶Prevalence measured the intention to quit in the next 6 months.

Initiation of Tobacco Use

In general, blue-collar and service workers are more likely to initiate smoking at younger ages than white-collar workers.[34,36–39] Some evidence suggests that service workers may have initiation patterns similar to white-collar workers, as was observed in a trend analysis of pooled cross-sectional data from the Tobacco Use Supplement to the Current Population Survey (TUS-CPS) from 1993, 1999, 2001, and 2006-2007.[35] This analysis also found that construction workers began smoking at earlier ages than workers in other blue-collar occupations. Early initiation of tobacco use could also apply to smokeless tobacco use, as skilled laborers such as production, construction, farming, or transportation workers have reported beginning ST use before age 16.[40] No literature was found that examined the initiation of tobacco use by employment status or worksite exposure to SHS. The literature on occupational class, profession and industry, and employment status did not analyze initiation by gender, race/ethnicity, or sexual orientation.

Current Use and Intensity

Cigarette Use, by Occupational Class and by Occupational Profession and Industry

There is consistent agreement in the literature that overall, blue-collar and service workers are more likely to be ever-smokers, current daily smokers, and heavier smokers than white-collar workers.[1,34,35,38,41–48] In a cross-sectional analysis of the 2000 National Health Interview Survey (NHIS), Barbeau and colleagues[1] found that 35% of blue-collar and 31% of service workers currently smoked, compared with 20% of white-collar workers. A study that pooled cross-sections of the NHIS and compared trends between 1987 and 1994 with those between 1997 and 2004 reported that many

blue-collar and service worker occupational groups had larger declines in smoking rates between 1997 and 2004 than in the previous time period.[46] However, these authors and others have noted that the historical gap in smoking prevalence rates between blue- and white-collar workers persists and may be widening.[38,46,48,49]

Studies have found disparities in smoking prevalence by occupational profession and industry, although comparisons across studies are difficult because each study tends to define the industries in slightly different ways, with some providing more of a breakdown in categories than others.[35,46,50] For example, the prevalence of smoking is significantly higher among workers in the following industries: mining (30.0%; 95% confidence interval [CI] 24.6–35.4), accommodation and food service (30%; 95% CI 28.3–31.6), construction and extraction occupations (31.4%; 95% CI 29.7–33.1), and food preparation and serving-related occupations (30%; 95% CI 28.4–31.7) compared with workers in management (10.9%; 95% CI 3.3–18.4), the educational services industry (9.7%; 95% CI 9.1–10.4), and education, training, and library occupations (8.7%; 95% CI 7.9–9.5).[51] The message from the studies is generally consistent: Workers in food services and in construction and extraction trades (e.g., rotary drill operator) have the highest rates of smoking prevalence, whereas those in education and library services, legal, and some health occupations have the lowest rates.[35,46,50,51] Disparities along class lines within an industry are illustrated by two studies of health care providers.[52,53] Licensed practical nurses (LPNs), respiratory therapists, and nursing assistants (NAs) had much higher smoking prevalence rates than registered nurses (RNs), who had higher rates than physicians, dentists, and pharmacists.

Syamlal and colleagues[54] analyzed data from the 2004–2012 National Health Interview Study (NHIS) to determine smoking prevalence among working adults by industry and occupation. Between 2004 and 2012, cigarette smoking prevalence among working adults declined from 22.4% to 18.1%. Declines varied by industry and occupation; the highest smoking prevalence was found among adults ages 18 to 44, non-Hispanic whites, individuals with a high school education or less, people with annual household incomes below $35,000, and people who lacked health insurance. Similar to patterns found in earlier studies, the highest smoking prevalence was found among workers in accommodation and food services (28.9%), construction (28.7%), and mining (27.8%). The authors concluded that "although in a majority of industries and occupations the age-adjusted smoking prevalence declined significantly over time, the current decline rates indicate that the smoking prevalence in certain industries and occupations may not reach the 2020 Healthy People goal."[54,p.605] Using data from the 2006-2007 TUS-CPS, Ham and colleagues[35] found that blue-collar and construction workers were more likely than other workers to be ever-smokers and current smokers, and construction workers were particularly heavy smokers (>20 cigarettes per day).

Cigarette Use, by Occupational Class or Industry and Demographic Characteristics

Further disparities can be seen when tobacco use is examined in terms of both occupational class/industry and demographic characteristics such as age, gender, and race/ethnicity. In the overall U.S. population, men have higher smoking prevalence rates than women. Although few recent studies have specifically analyzed gender differences in smoking within occupational class or industry, there are a handful of exceptions. One report on the Asian American workforce analyzed a sample of Chinese, Filipinos, Vietnamese, and others (e.g., Japanese, Koreans, Cambodians, Laotians, and Indians) from the cross-sectional data of the National Latino and Asian American Study.[42] As in the overall U.S. population, this study found that in each occupational class men had higher rates of smoking prevalence than women, although the study did not analyze by each national origin group. For both men and

women, blue-collar and service workers had higher rates of smoking than white-collar workers. These differences were especially pronounced for women.

In an analysis using a nationally representative sample of U.S. adults from the 2000 NHIS, men in blue-collar occupations had higher rates of current smoking than women in the same occupations.[1] In another study, among health care workers, gender differences for smoking were not significant in adjusted models for RNs, LPNs, and NAs; other clinical staff members; nonclinical staff members; or wage-grade personnel.[53] The only significant difference by gender was for an occupational grouping of physicians, physician assistants, and nurse practitioners, with men having higher rates of smoking prevalence than women.

Most studies in the current review adjusted for age, analyzed adults age 18 and older, and did not examine occupational differences by age category. A cross-sectional study by Lawrence and colleagues[45] was an exception in that it analyzed adults ages 18–24 using data from the 1998-1999 TUS-CPS. Like the overall literature on adults, this study found that young adult blue-collar and service workers were more likely to smoke, smoke daily, and be heavy smokers than those who worked in white-collar occupations. Blue-collar and service workers were also less likely to be light smokers (smoking 1–9 cigarettes per day) than their white-collar counterparts. Service workers ages 18–24 had the highest likelihood of smoking (odds ratio [OR] 1.62; 95% CI 1.42–1.84) of all occupational groups.

As described in chapter 2, disparities exist in smoking prevalence rates by race/ethnicity, with American Indians/Alaska Natives having the highest rates of smoking, followed by whites and blacks, Hispanics, and Asian Americans.[55] For nearly all occupational groups, American Indians/Alaska Natives had the highest smoking rates and Asian Americans the lowest, while whites had the most pronounced differences in rates between occupational groups. In terms of smoking prevalence, Barbeau and colleagues[1] found that for whites, blacks, Hispanics, and Asian Americans, blue-collar workers had higher prevalence rates than white-collar workers. For American Indians/Alaska Natives this pattern was different: Service workers had the highest rates of smoking, followed by white-collar workers. In another study focused on health care workers, adjusted prevalence rates of smoking were higher for Hispanic than non-Hispanic physicians, physician assistants, and nurse practitioners.[53] Adjusted smoking rates were higher among whites than nonwhites among RNs, LPNs, NAs, and nonclinical staff members.

Similarly, in multivariate logistic regression analyses of a nationally representative sample of employed U.S. adults ages 18–64 from the 1998-1999 TUS-CPS, occupational class was significantly associated with current smoking for non-Hispanic whites and American Indians/Alaska Natives but not for African Americans, Asian Americans/Pacific Islanders, or Hispanics.[47] For American Indians/Alaska Natives, workers in sales and administrative support were less likely to be current smokers (24.9%) than those in professional and managerial occupations (26.7%). For non-Hispanic whites, laborers (32.9%) and service workers (36.4%) were more likely to be smokers than those in professional or managerial occupations (16.7%). This study also examined racial/ethnic differences in smoking by industry and found significant differences by industry for all racial/ethnic groups except African Americans. Among non-Hispanic whites, workers in nearly all other industries had significantly higher odds of current smoking (odds range from OR 1.31 to 1.66; 95% CI) when compared with those in professional and related services. Asian Americans/Pacific Islanders working in agriculture, forestry, farming, and fishing (OR 1.81; 95% CI 1.01–3.25); wholesale and retail trades (1.62; 95% CI 1.11–2.37); finance, insurance, and real estate (1.61; 95% CI 1.00–2.58); and other service professions (2.05; 95% CI 1.37–3.07) had

higher odds of current smoking than Asian American/Pacific Islander workers in professional and related services. Hispanics working in transportation and common public utilities (1.50; 95% CI 1.09–2.07) and American Indians/Alaska Natives working in finance, insurance, and real estate (2.64; 95% CI 1.20–5.80) had significantly higher odds of smoking compared with professional and related service industries for these race/ethnicities. In adjusted models, Shavers and colleagues[47] reported that work performed in labor and/or service occupations was associated with lower odds of occasional (i.e., non-daily) smoking among African Americans, Hispanics, and non-Hispanic whites when compared with professional groups; however, occasional smoking did not vary significantly by occupation for American Indians/Alaska Natives or Asian Americans/Pacific Islanders.

Occasional smoking varied by industry for African Americans, American Indians/Alaska Natives, and non-Hispanic whites, but it did not vary by either occupation or industry for Asian Americans/Pacific Islanders. African American and non-Hispanic white service workers had higher odds of heavy smoking (i.e., more than 20 cigarettes per day) than those working in professional or managerial occupations—the only significant differences by occupation and race/ethnicity.[47] Heavy smoking did not vary by industry and race/ethnicity, except for Hispanic private or household services workers and non-Hispanic whites working in public administration, who were more likely to be heavy smokers than Hispanics and non-Hispanic whites working in the professional and related services industry. The authors noted that the reasons for this lack of consistency in smoking patterns by industry, occupation, and race/ethnicity are not clear, and they suggested that differential job experiences, such as job demand and stress by race/ethnicity, could account for some of these findings.[47]

In an analysis that added multiple levels of social deprivation (having less than a 4-year college degree, working in a blue-collar occupation, and having an income below 200% of the federal poverty level), Barbeau and colleagues[1] found higher prevalence of current smoking among white males with all three levels of social deprivation (52%) than for white males with less than a college degree (35%). A similar comparison showed that experiencing multiple levels of social deprivation increased smoking prevalence among white and black women by 12–13%. Multiple levels of deprivation were not associated with an increase in smoking prevalence for black and Hispanic men or Hispanic women.[1]

In summary, disparities in patterns of smoking vary by occupation and industry in the United States. Generally, compared with white-collar workers, blue-collar and service workers are more likely to initiate smoking earlier and to be ever-smokers, current daily smokers, and heavier smokers. The highest prevalence rates are found among workers in the construction and extraction and food preparation and service industries, whereas those in education and library services had the lowest rates. Overall, the finding that men have higher smoking rates than women generally holds for all racial/ethnic and occupational groups. Although there is consistent evidence for an association between occupational class and smoking by race/ethnicity for non-Hispanic whites and Asian Americans/Pacific Islanders, evidence for Hispanics, African Americans, and American Indians/Alaska Natives is less consistent. The existing literature has only begun to highlight where disparities exist and where they do not. Augmenting this largely epidemiologic and quantitative literature with qualitative studies could shed light on how gender, age, and race/ethnicity could act as barriers to and protect against smoking. No literature was found on patterns of smoking by occupation/industry and sexual orientation.

Cigarette Use, by Employment Status

Little recent research comparing smoking patterns by employment status is available for the United States. The few nationally representative studies that have focused on employed versus unemployed status have not generally compared tobacco use rates.

Cigarette Use, by Employment Status and Demographic Characteristics

A literature review on smoking patterns by employment status as it interacts with demographic characteristics (such as age, gender, race/ethnicity, and sexual orientation) found no clear pattern in relationships between smoking, employment, and gender. Fagan and colleagues[56] used cross-sectional data from the 1998-1999 and 2001-2002 TUS-CPS and found that unemployed men had increased odds for smoking compared to unemployed women. By contrast, a 19-year longitudinal study using data from the 1979–1998 National Longitudinal Survey of Youth (participants were ages 14–21 in 1979) found that joblessness was more strongly associated with smoking among adolescent/young adult women than men.[57]

Although most studies controlled for age, two studies focused separately on age group categories and employment status. Falba and colleagues[58] conducted a longitudinal study of smoking intensity using data from two waves (1992 and 1994) of the Longitudinal Health and Retirement Survey on individuals ages 52–62 who had suffered an involuntary job loss, defined as loss of a job due to layoff or to a business or plant closing. Older adults who had suffered an involuntary job loss and who remained unemployed at wave 2 reported a significantly higher daily cigarette consumption (about seven more cigarettes per day) compared with those who had not suffered a job loss. There was no significant difference by occupation. The cross-sectional study by Lawrence and colleagues[45] investigating cigarette smoking patterns among young adults ages 18–24 found that those who were unemployed (in the labor force but not currently working) were more likely than those not in the labor force to be current, daily, and heavy smokers.

Race/ethnicity may also interact with employment status to affect smoking behavior. Fagan and colleagues[56] reported that unemployed whites and American Indians/Alaska Natives had nearly three times the odds of current smoking compared with unemployed Asian Americans/Pacific Islanders. Unemployed Hispanics were about 33% less likely to be current smokers than unemployed Asian Americans/Pacific Islanders. Unemployed blue-collar and service workers had slightly greater odds ratios of current smoking compared with unemployed white-collar workers. De Castro and colleagues,[42] in multivariate analyses, found that unemployed Asian Americans had greater odds of smoking than their employed counterparts. Braun and colleagues[57] used employment and occupational prestige scores as outcomes rather than smoking, based on five waves of data (1985–1995) from the longitudinal Coronary Artery Risk Development in Young Adults (CARDIA) Study conducted on a sample of black and white men and women in four major U.S. cities (Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California). These researchers found that among blacks (but not whites), current smokers were less likely to be employed as professionals than nonsmokers.

Use of Other Tobacco Products, by Occupational Class or Industry and Employment Status

Cigarettes are the main tobacco product used in the United States, but ST, cigars, and pipe tobacco have also been used for many decades. Other forms of tobacco use, including clove cigarettes, bidis (small hand-rolled cigarettes), waterpipes, and, more recently, e-cigarettes, have entered the U.S. market.

Studies on these newer products continue to emerge, and many were published after the initial literature search for this chapter was conducted. For example, a Centers for Disease Control and Prevention (CDC) study used data from the 2014 NHIS to examine e-cigarette use among working adults by industry and occupation. Overall, for working adults age ≥18 years, an estimated 3.8% were current (every day or some days) e-cigarette users. By industry, e-cigarette use was highest in the accommodation and food services industry (6.9%) and lowest among education services workers (1.8%). By occupation, prevalence was highest among food preparation and serving-related occupations (6.8%) and lowest among architecture/engineering/computer/mathematical workers (1.9%).[60]

As described in chapter 2, ST use is low overall in the United States. However, data from the National Survey on Drug Use and Health (2003–2008) indicates that although most ST users are white males, ST users vary more in terms of education, occupation, and place of residency than is commonly thought.[40] Additionally, a study estimating ST use among employed workers age 18 and older from the 1987–2005 NHIS found that about 3.5% used an ST product, with no statistically significant overall upward or downward trend in use during the time period.[61] When the authors investigated pooled prevalence rates, they found significant differences by occupation: Farm workers had the highest rates (11%), followed by blue-collar (7%), service (2%), and white-collar (2%) workers.[61]

Few disparities-focused studies have looked at individuals who used more than one tobacco product (called dual users or poly-tobacco users). A nationally representative study that looked at concurrent tobacco use in the United States by occupation, employment, and other sociodemographic characteristics was conducted by Backinger and colleagues[62] using data from the 1995-1996, 1998, 2000, and 2001-2002 TUS-CPS. Concurrent users were predominantly white males. Generally, blue-collar workers had higher prevalence rates of concurrent use of tobacco products, ranging from about 6% to 11% over the years studied. In a breakdown by type of concurrent use, blue-collar workers generally had higher prevalence rates of pipe smoking, cigar smoking, and use of chewing tobacco and snuff than white-collar and service workers. In multivariate analyses, occupation was not a significant predictor of current and daily smokers' concurrent tobacco use, although blue-collar workers who were intermittent smokers had a 22% increased risk of concurrent use compared with white-collar workers.[62]

Backinger and colleagues[62] also found that concurrent use of tobacco products was similar among the employed and the unemployed and was slightly lower for those not in the labor force. Pipe use among smokers was similar by employment status, and cigar use among smokers was somewhat lower for those not in the labor force compared with the employed and unemployed. This latter pattern also held for smokers who used chewing tobacco and snuff, both separately and together. Multivariate models showed no significant difference by employment status for concurrent tobacco use.

Influence of Worksite Smoking Bans or Restrictions on Smoking, by Occupation, Industry, and Employment Status

Exposure to SHS has been causally linked to a wide variety of adverse health effects in both adults and children and is estimated to account for more than 41,000 deaths in the United States annually.[63] Reviews of the evidence have concluded that worksite smoking restrictions, especially those that ban smoking everywhere on the worksite, reduce the number of cigarettes workers smoke per day.[64–67] National Cancer Institute (NCI) Tobacco Control Monograph 21, *The Economics of Tobacco and Tobacco Control*, concluded that "comprehensive smoke-free policies in workplaces reduce active smoking behaviors including cigarette consumption and smoking prevalence."[68,p.12]

Although state and local laws prohibiting smoking in workplaces and public places are now common, employees in some worksites, especially those in the hospitality industry (casinos, bars, restaurants, and hotels) continue to be exposed to SHS.[69] Compared with white-collar workers, blue-collar and service workers were less likely to report having a smoking ban in place where they work,[32–35,46] a trend that has continued over time.[35] Occupational class and exposure to SHS have been shown to be inversely related.[34,44]

Shopland and colleagues[33] found that smoking bans are associated with a reduction in the use of cigarettes, but these reductions have been found more in white-collar professions than in the service industry. A study analyzing data from the National Health Interview Survey (1987–2005) found no evidence that worksite smoking restrictions prompted workers to substitute ST products for cigarettes; the prevalence of ST use was stable over this period, despite substantial increases in worksite smoking restrictions.[61] Additionally, the authors found that ST use was lower among workers employed in workplaces where smoking was banned, suggesting that smoking restrictions contribute to lower ST use.[61]

No studies were found that investigated the association between exposure to SHS and tobacco use by occupation and by gender, age, race/ethnicity, or sexual orientation.

Use of Other Tobacco Products, by Occupation, Industry, and Employment Status and by Demographic Characteristics

The study by Dietz and colleagues[61] reported that for all occupational classes and racial/ethnic groups, men had higher rates of ST use than women. Among both black and white men, the rates of ST use varied by occupation and were highest among farm workers (black: 14.65%; white: 12.70%). Black women had relatively high rates of ST use among farm (9.01%) and blue-collar workers (3.07%). No national data were found by industry/employment status and demographic group.

Quit Attempts and Intentions to Quit Smoking

This section examines the literature on quit attempts and intentions to quit smoking by occupational class or industry and employment status. Relatively few studies have examined quit attempts and intentions to quit by occupation. Although the studies reviewed in this chapter use various definitions of quit attempts (ranging from stopping smoking for 1 day to stopping for longer periods), this chapter defines a quit attempt as stopping smoking for 3 months or less, and successful cessation as stopping smoking for a period longer than 3 months.

Attempts and Intentions to Quit Smoking, by Occupational Class or Industry

Before becoming a former smoker, most smokers make multiple quit attempts.[70] Given the differences in smoking prevalence by occupation, it is important to understand whether cessation varies by occupation. Most studies conclude that quit attempts and intentions to quit smoking did not vary by occupational class—that is, there were no significant differences in attempts to quit smoking among white-collar, blue-collar, or service workers.[1,34,35,43]

Barbeau and colleagues[1] found that typically between 40% and 50% of smokers in each occupational class, including farm workers, had attempted to quit at least once in the past year, although these data were not from multivariate regression models. Two studies reported some differences in quit attempts by

occupational class or industry,[35,71] but the differences found in one of these studies[35] were no longer significant in multivariate models. Alexander and colleagues[71] performed a cross-sectional analysis of the 2006 TUS-CPS to investigate the relationships between smoking mentholated cigarettes, occupational class, and quit attempts; using multivariate models that controlled for menthol use, they found that service workers were significantly less likely to make a quit attempt than white-collar workers (OR 0.8; 95% CI 0.69–0.94). These authors noted that service employees are less likely to be protected by smoke-free laws or regulations, which could contribute to fewer quit attempts.

Attempts and Intentions to Quit Smoking, by Occupational and Demographic Characteristics

One study provided descriptive data on the percentages of members of different occupational classes and racial/ethnic groups who attempted to quit, which generally ranged from about 40% to 50%.[1] The authors found no social patterning in quit attempts by SES and racial/ethnic group or, among whites, by occupational class. Among Hispanic workers, service workers had the highest percentage of quit attempts (51%), farm workers the lowest (37%). Among Asian Americans, service workers had the highest percentage of quit attempts (52%), white-collar workers the lowest (43%). Among blacks, white-collar workers had the highest percentage of quit attempts (51%) and farm workers the lowest (42%).[1] Reports of quit attempts by occupational class or industry and by age, gender, or sexual orientation were not located.

Attempts and Intentions to Quit Smoking, by Employment Status and by Demographic Characteristics

NCI Tobacco Control Monograph 12, *Population Based Smoking Cessation*, concluded that as restrictions on workplace smoking are implemented, they increase the rate at which smokers attempt to quit.[72] Similarly, a review of workplace smoking restrictions conducted by Brownson and colleagues[65] concluded that smokers who are employed in workplaces with smoking bans are more likely to consider quitting than smokers employed in workplaces with weaker policies or no policies at all. No studies were found on smoking restrictions and quit attempts by occupation; quit attempts by differential employment status or by employment status intersected with age, gender, race/ethnicity, and sexual orientation; or quitting the use of other forms of tobacco, by occupation.

Cessation

Cessation, by Occupational Class

Research has consistently shown that white-collar workers are more successful at quitting smoking than blue-collar and service workers,[1,34,43,56,73] with multivariate analyses finding lower odds of former smoking among blue-collar and service workers than among their white-collar counterparts.[43,71]

For example, in an analysis of secondary data from a nationally representative sample of employed adults, Fagan and colleagues[43] found that former smokers made up about 19% of white-collar workers; 18% of blue-collar workers; 16% of workers in farming, forestry, and fishing; and 14% of service workers. Barbeau and colleagues[1] reported similar prevalence rates for these categories. Using multivariate analyses, Fagan and colleagues[43] reported that the odds of being a former smoker were about 20% lower for workers in blue-collar (OR 1.31; 95% CI 1.27–1.35) and service occupations (OR 1.15; 95% CI 1.10–1.20) than in white-collar occupations. Former smokers were defined as people who had smoked at least 100 cigarettes in a lifetime and responded “not at all” to the survey question “Do you now smoke every day, some days, or not at all?” Former smokers were asked how long it had been since they had quit smoking, and “successful quitters” were those who had not smoked for at least

12 months. In multivariate analyses, occupational class was not associated with successful quitting of 12 months or more.

Cessation, by Occupational Class or Industry and by Demographic Characteristics

Only one of the studies discussed in the preceding section also reported smoking cessation results by occupation and by gender and race/ethnicity. The study by Barbeau and colleagues[1] detailed prevalence rates of former smoking (defined as not currently smoking) by occupational class and race/ethnicity. White-collar workers had the highest rates of former smoking of any occupational group for whites, Hispanics, and Asian Americans. Among blacks, blue-collar workers had the highest prevalence rates of former smoking; white-collar and service workers had similar rates of former smoking. Barbeau and colleagues'[1] previously described analysis using increasing levels of social deprivation also analyzed gender and racial/ethnic group (white, black, and Hispanic) differences. This analysis found that the pattern of former smoking by gender and race/ethnicity followed the inverse gradient found in the overall population: White, black, and Hispanic women and men who had higher levels of social deprivation were less likely to be former smokers. The authors noted that this pattern was especially pronounced among white men and among women in all three racial/ethnic groups. Of women at the most socially deprived level, only 3% of Hispanics and 4% of blacks were former smokers.

Cessation, by Employment Status

Reviews of the evidence conclude that worksite smoking restrictions significantly increased cessation rates (defined as 3 months or more without smoking), although the effects by occupation status were not discussed.[65–67,72] A few studies have investigated whether employment status influences cessation; most studies adjust for employment status or include only employed workers. In a study of smoking and quitting behaviors among the unemployed, Fagan and colleagues[56] found that unemployed workers whose last or usual job was in blue-collar or service work had lower odds of being former smokers (defined as not currently smoking) compared with unemployed white-collar workers. However, unemployed blue-collar workers were 1.83 times as likely (95% CI 1.17–2.87) to be successful quitters (defined as former smokers who stopped smoking completely for at least 12 months) compared with unemployed white-collar workers. The authors note that "contrary to expectations, unemployed blue-collar workers were more likely than white-collar workers to quit successfully."[56,p.245] The authors suggest that unemployed blue-collar workers may no longer have worksite social influences to smoke, and may be able to develop better coping strategies for quitting when removed from the worksite.

Cessation, by Employment Status and by Demographic Characteristics

One study investigated the relationship of employment status to cessation among different age groups using longitudinal data. Weden and colleagues[57] analyzed a nationally representative sample of young men and women who were 14–21 years old when first surveyed in 1979 and who were interviewed annually or biannually for about 20 years. Cessation was defined as the age at which respondents stopped smoking daily. (Note that this definition of cessation cannot distinguish between smokers who have quit completely and those who now smoke on only some days or intermittently.) The authors found that European American women who were unemployed or out of the labor force were significantly less likely to quit smoking than their employed counterparts, but that employment status and cessation had no significant relationship for African American or Hispanic women. Although there were no significant associations between employment status and cessation among European American and Hispanic men, unemployed African American men were more likely to quit smoking than employed African American

men. The authors speculate that these gender and racial/ethnic differences in smoking and quitting patterns by employment status may be related to differences in smoking and quitting patterns by occupation, and by differential exposures to worksite stresses and demands.[57]

No studies were found that looked at employment status and cessation by sexual orientation or compared quitting use of other tobacco products by different occupational classes.

Relapse

No nationally representative studies were found that directly addressed relapse in terms of occupational class, industry, or employment status. No studies were found that addressed relapse in relation to use of other tobacco products by different occupational classes, or the impact of worksite smoking restrictions on relapse by occupational class.

Effect of Occupation and Tobacco Smoking on Cancer Risk

Tobacco use, exposure to SHS, and occupation have each been found to be related to cancer incidence and mortality and to other diseases.[24,38,74–79] Howard[38] identified six interactions between tobacco smoking and occupational exposures that can produce cancer and other negative health effects:

1. Chemicals contained in tobacco smoke (e.g., carbon monoxide, benzene, acetone, formaldehyde) can also be present in work processes, thereby increasing a worker's cumulative exposure and risk for disease.
2. A workplace toxicant combined with tobacco smoking can produce negative health effects that neither produces alone.
3. If the surface of a cigarette is contaminated with an occupational substance such as lead, the substance can enter the body through ingestion, inhalation, or absorption through the skin.
4. Tobacco smoke and exposure to workplace toxicants can have additive health effects on the body.
5. Tobacco smoke and occupational toxicants could have multiplicative or synergistic effects. For example, research has documented that the combined risk of smoking and exposure to asbestos is greater than the sum of the independent risks of either factor (see Box 8.1)
6. Tobacco smoking has been associated with traumatic occupational injuries. Possible explanations include distraction associated with the act of smoking, the impact of smoking on human performance, and confounding by higher rates of use of alcohol and other drugs among smokers compared with nonsmokers.

By these mechanisms, workers who smoke or are exposed to SHS and are also exposed to workplace toxicants may experience greater cancer risks than those not exposed to workplace toxicants. Box 8.1, on asbestos-related cancer, and the sections on radon and diesel fuels and lung cancer, illustrate that these agents and tobacco smoke have independent and strong effects on cancer risks, and the workplace exposure can interact with tobacco smoke exposure in complex ways to affect cancer risks.

Box 8.1 Asbestos, Smoking, and Cancer Risk

Asbestos has been mined and used commercially in North America since the late 1800s. Its use increased greatly during World War II, and since then, asbestos has been used in many industries. Millions of American workers have been exposed to asbestos since the 1940s, and health hazards from asbestos fibers have been recognized in workers exposed in the shipbuilding trades, asbestos mining and milling, manufacturing of asbestos textiles and other asbestos products, insulation work in the construction and building trades, and a variety of other trades. Demolition workers, dry wall removers, asbestos removal workers, firefighters, and automobile workers also may be exposed to asbestos fibers. As a result of government regulations and improved work practices, today's workers who have no previous asbestos exposure are likely to face smaller risks from asbestos than workers in previous decades.[180]

Asbestos and all commercial forms of asbestos are known to be human carcinogens, based on sufficient evidence of carcinogenicity in humans.[30] The International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence for the carcinogenicity of asbestos in humans.[181–183] Many studies have shown that the combination of smoking and asbestos exposure is particularly hazardous. Interactions on both the additive and multiplicative scale have been suggested from co-exposure to asbestos and tobacco and subsequent risk of lung cancer.[184] There is also evidence that quitting smoking will reduce the risk of lung cancer among asbestos-exposed workers.

Studies Investigating the Interactions Between Occupation and Smoking and Lung Cancer

Some studies have tested possible interactions between exposures to occupational hazards and tobacco smoking. This section discusses studies that examined interactions between occupational lung carcinogens (radon and diesel engine exhaust) and smoking and risk of lung cancer. The data from these different occupational settings suggest that the observed interactions with smoking and known occupational carcinogens are not the same for all agents, and thus, detailed studies with combined information are needed to understand the health impacts of these co-exposures.

Radon

Miners may be exposed to radon via inhalation in uranium or other mines. A number of studies have assessed the association of radon decay products (known as radon progeny or radon daughters) and lung cancer, and have considered the effects of smoking.[80] Additionally, a report of the National Research Council's Committee on the Biological Effects of Ionizing Radiation provided an in-depth review of the combined effect of smoking and radon on lung cancer risk and detailed evidence from the epidemiologic studies of underground miners showing differing patterns of the effect of exposure to radon progeny on never-smokers and ever-smokers.[81] Relative to the overall effect of exposure to radon progeny on lung cancer risk, the risk estimate in ever-smokers was proportionally lower by a factor of 0.9, whereas the relative effect in never-smokers was proportionally higher by 1.9, a sub-multiplicative interaction.

Diesel Engine Exhaust

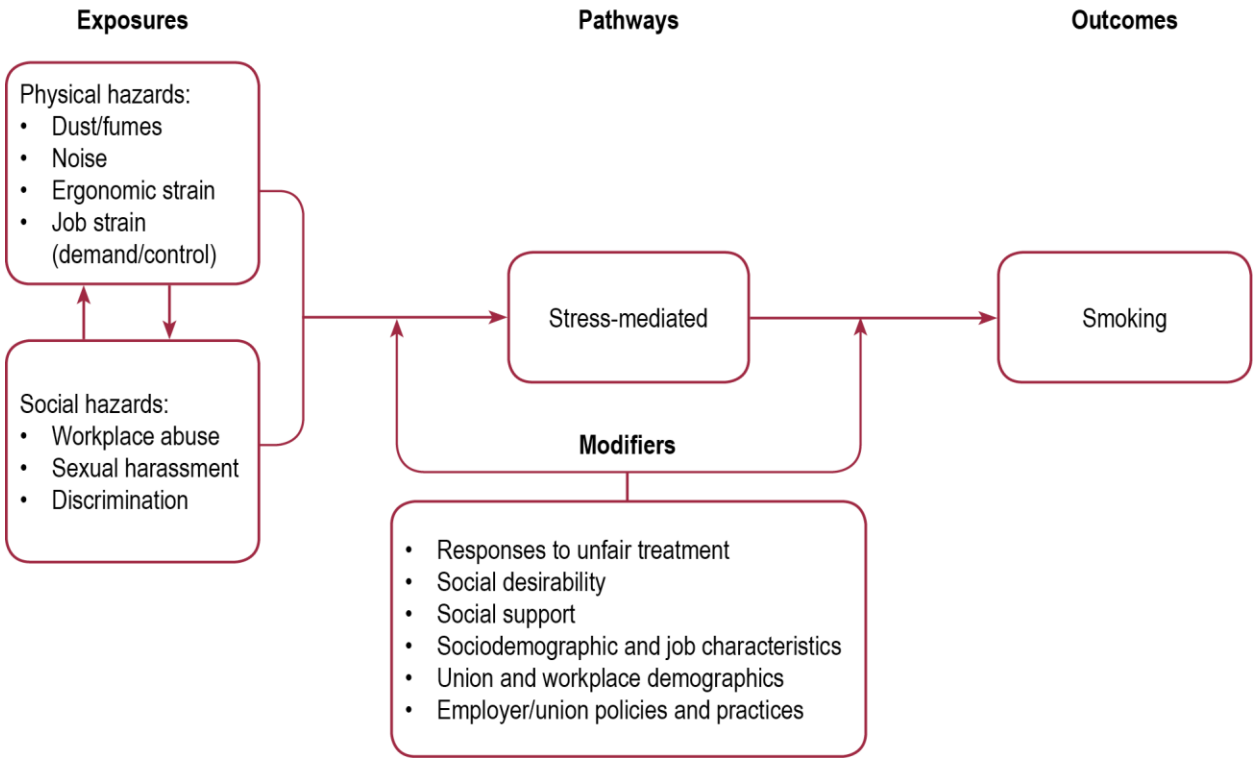
Studies of non-metal miners from the United States have demonstrated a relationship between diesel engine exhaust and lung cancer.[82] In this large cohort of 12,315 workers, the combined effect of diesel exposure and intensity of cigarette smoking was also evaluated. The authors observed that risk associated with smoking intensity was modified by diesel exposure. Among workers in the lowest third

of cumulative respirable elemental carbon exposure, smokers of at least two packs per day had a risk 27 times that of nonsmokers, whereas among those in the highest third of cumulative respirable elemental carbon exposure, heavy smokers had about 2.5-fold the risk of nonsmokers.

Contributions of the Work Environment to Disparities Along the Tobacco Use Continuum

Empirical and theoretical evidence suggests that work environments influence on-the-job and off-the-job health behaviors and may contribute to disparities along the tobacco use continuum.[31,83,84] The link between occupational exposures and smoking is attenuated but remains after controlling for education.[83] Most research studies on the influence of the work environment on tobacco use posit a stress-mediated pathway from exposures in the work environment to tobacco use (Figure 8.1).[85] Evidence from the larger literature on stress and health shows that one mechanism through which stress can influence health is its contributions to changes in health behavior.[86–89] As discussed in chapter 5, low-SES individuals experience multiple sources of stress, and smoking is well known as a source of perceived stress relief.[90,91]

Figure 8.1 Conceptual Model of Stress-Mediated Pathways to Smoking



Source: Adapted with permission from Barbeau et al. 2007.[85]

The work environment can also influence smoking in ways that do not involve stress. Work-related discrimination can influence health via stress, but it can also influence health behaviors through other pathways. Job discrimination can lead to lower wages, which may be associated with an increased use of substances such as cigarettes. Job discrimination can also affect job assignment and, hence, exposure to workplace hazards that may increase the risk of smoking initiation, continuation, and/or the severity of its consequences.[92] Another theory is that self-selection plays a role: smoking may be associated with

lower occupational aspirations, which may increase the likelihood of working in more hazardous jobs.[59] This theory has been hard to evaluate, given that many of the social disparities associated with smoking disproportionately occur early among less advantaged members of societies, who also have fewer career opportunities. A longitudinal study of occupational attainment and smoking found evidence that smoking was associated with less occupational attainment but only among African American workers.[59]

The growing body of work on occupational health disparities shows that socially disadvantaged workers—such as less educated groups; racial/ethnic, gender, and lesbian, gay, bisexual, and transgender (LGBT) groups; immigrants; migrants; and blue-collar and service workers—bear a disproportionate burden of adverse work conditions.[23,93–98] This unequal distribution of working conditions means that any association between adverse work environments and smoking is likely to translate into disparities across the tobacco use continuum. The following sections explore the current evidence for the contribution of adverse work environments to tobacco use. The contribution of these factors to disparities in the tobacco use continuum is also discussed.

Psychosocial Work Environment

Job Control and Effort–Reward Imbalance

Most studies of the relationship between the psychosocial work environment and smoking have used either Karasek's demand–control model or the effort–reward imbalance model to define the psychosocial work environment.

According to the demand–control model, work environments can be categorized into one of four quadrants, based on whether they are high or low in physical and psychological demands and in decision latitude/control.[99] The most stressful working condition—high demands and low control (job strain)—has consistently been shown to be detrimental to health.[99–101] The literature on psychosocial work environments has established that there are occupational disparities in these job characteristics and that socially disadvantaged populations are more likely to have jobs with either or both low control or high demands.[101,102] Studies have yielded inconsistent results, with some studies finding positive associations between job strain and smoking,[11,103–108] and others finding no significant association.[13,106,109–115]

Most of the early studies on job strain and smoking focused on men. For example, using a U.S. sample of 389 male chemical plant workers, Green and Johnson[11] found that those who experienced job strain were more likely to smoke cigarettes. They also found that the proportion of heavy smokers increased with increasing job strain. Most of their study participants were white men (93%), and thus they were unable to examine gender or racial differences in the association. A study of 46,190 public sector workers (81% female) in Finland, found that job strain, as defined by high effort–reward imbalance, was associated with smoking among both women and men, but the association was significant only among women (1.28; 95% CI 1.19–1.39).[105]

Some studies have examined associations with other quadrants of the job demand–control model. Brisson and colleagues[110] found that smoking prevalence was highest among men and women whose working conditions were high in both psychological demands and control. Another study did not find an association between job strain and smoking status but found that smoking status was associated with demands but not control.[111]

Other studies and reviews of the literature have found that the strongest evidence on the influence of the psychosocial work environment on smoking comes from studies of the effect of psychological and physical job demands on increased smoking intensity.[83,116]

Some studies have examined the association between the psychosocial work environment and smoking cessation, but with inconsistent results.[13,117] Of the 4,928 public sector workers (77% female) who were smokers at baseline in 2000–2002, Kouvonen and colleagues[105] found that, at the level of the work unit, low job strain (1.43; 95% CI 1.17–1.75) and high job control (1.61; 95% CI 1.31–1.96) were associated with greater likelihood of smoking cessation at follow-up in 2004-2005.[105] Other studies and reports on occupational disparities have shown that racial and ethnic minority and immigrant workers are more likely to work in units with higher job strain.[7,118,119]

Another model examining job stress is the effort–reward imbalance model, which evaluates jobs based on the degree to which workers are rewarded (in terms of income, opportunities, and prestige) for the physical and psychological efforts required for their jobs.[84,120] Kouvonen and colleagues[105] examined this model in addition to job strain and smoking intensity, as described above. They found that effort–reward imbalance was significantly associated with both smoking intensity and the prevalence of smoking. (The odds of smoking were 1.28 higher for women with a high effort–reward imbalance and 1.13 higher for men with a high effort–reward imbalance compared with female and male employees with low effort–reward imbalance.) A report from the United Kingdom explains that socially disadvantaged populations may be more likely to have jobs in which there is an imbalance in terms of efforts and rewards.[118]

How these working conditions might differentially influence smoking cessation attempts and success among socially disadvantaged workers has received scant empirical attention. One study, which prospectively examined working conditions and smoking among 654 transit operators in San Francisco across 10 years, found that scoring high on an index of job problems (including aspects of psychological demands along with conflict with customers) predicted initiating, maintaining, or increasing smoking.[121] The study also found that black workers were more likely than workers in other racial/ethnic groups to initiate, maintain, or increase their smoking, even after controlling for gender, age, occupational factors, and alcohol use. In this study, black workers had a higher prevalence of smoking than any other racial/ethnic group. The study did not have sufficient power to examine interactions between race and existing job problems.

Job-Related Social Norms, Social Support, and Social Capital

Work-related social norms, social support, and social capital have all been linked to disparities in tobacco use behaviors. As noted previously, studies consistently find that blue-collar workers have higher smoking rates than workers in other occupational classes.[1] Blue-collar workers also reported less pressure to quit and lower support for smoking cessation, both of which were associated with less motivation to quit in this group.[16,122,123] Blue-collar workers also reported receiving less support for smoking cessation from their work environments.[124] A systematic review of the literature on the studies addressing the impact of the work environment on smoking behavior concluded that workplace social support increases cessation and decreases relapse and the amount smoked.[83] The role of workplace social capital may be to act as a buffer between the psychosocial work environment and smoking.[125] In addition, organized labor groups may assist in making smoking cessation programs more relevant and effective for their members.[126]

Job Insecurity

Job insecurity, generally defined in the studies referenced as work status (temporary or permanent), has been established as a source of stress among workers and a source of social inequalities in health.[127,128] Using a cross-sectional community sample of U.S. workers, Muntaner and colleagues[129] found an association between job insecurity and smoking status. Two studies did not find any associations with job security and smoking: a study conducted in Turkey, which defined job insecurity as having temporary employment,[130] and a longitudinal examination of job security and change in smoking behavior among the Whitehall II study cohort in England.[127]

Job loss, another aspect of job insecurity, has potential implications for disparities in tobacco use. As explained earlier, studies have generally found a higher proportion of smokers among the unemployed compared to employed individuals[131,132] and compared to students or participants in labor market programs (e.g., job training, assistance finding a job).[132] Studies of the implications of job loss for tobacco use have shown inconsistent results. Some studies found associations between job loss and smoking,[58,133] whereas others did not.[134,135] Two of these studies were longitudinal observations of workers who had lost their jobs versus those who had not.[133,135] Both studies reported that participants who later experienced job loss had significantly higher levels of smoking at the beginning of the study, but only one study found that job loss preceded increased smoking.[133] Both studies were of British men and did not examine their findings by markers of social disadvantage.

One study found that unemployment was more strongly associated with smoking among young adult women than among young adult men.[57] Further analyses found that the influence of joblessness for women was related to fewer social and economic resources; after adjusting for reductions in resources, the significant association of joblessness with smoking was eliminated for African American and Hispanic women.

Organizational and Physical Work Environment

Work Hours and Schedule

Work hours and schedule can play a role in smoking and smoking cessation. A longitudinal study of Norwegian nursing assistants found a tiered association between work hours and smoking cessation at follow-up;[136] the lowest odds of smoking cessation occurred among those who worked more than 36 hours per week, followed by those who worked 19–36 hours per week, and then by those who worked 1–9 hours per week. A cross-sectional study, which used an Australian sample, found that among men, working 50 or more hours per week was associated with an increased likelihood of smoking, compared with working 35 or fewer hours per week.[137] Another longitudinal study found that those who did not work day shifts were less likely to report smoking cessation than those who did.[104] This study also found that those who worked variable shifts had decreased odds of smoking cessation compared with those who worked only day shifts. The one U.S. study on this issue analyzed nationally representative, cross-sectional data from the 1998-1999 and 2001-2002 TUS-CPS and used multivariate analyses controlling for occupational class; this study found that individuals who worked variable hours were more likely to be smokers than those who worked part time.[43]

With one exception, the studies on work hours and schedules used European and Australian samples and were not able to examine the role of race/ethnicity, immigration status, and social advantages. Studies using nationally representative samples from the United States have shown that socially disadvantaged workers, especially those with low education and income and those who are black and Hispanic, are less

likely to have control over their work hours and schedules.[138,139] As a result, they are likely to be most at risk for any negative influences of work hours and schedules on smoking behaviors.

Discrimination, Workplace Bullying, Abuse, and Harassment

Key sources of workplace stress are experiences such as workplace discrimination, bullying, abuse, and harassment.[118,119,140] Of these, discrimination has received the most attention in the research literature and is discussed further in chapter 5. Meta-analytic reviews indicated that racial discrimination was associated with unhealthy behaviors, especially among racial/ethnic minority groups,[141,142] but this association has received less attention in the workplace literature, and the few studies on the topic have been cross-sectional. Some of these studies used instruments that included questions specifically about having experienced racial discrimination on the job.[140,143–147] Okechukwu and colleagues[148] analyzed a cross-sectional, multiethnic lower class worker sample and reported an association between exposure to workplace racial discrimination and smoking that was strongest for black workers; these authors did not find significant effects for either sexual harassment or workplace bullying.

Workplace discrimination, bullying, abuse, and harassment could play important roles in disparities in tobacco use behaviors. These exposures are significant sources of stress, and they are often specifically targeted toward socially disadvantaged groups, whether based on SES, race/ethnicity, or sexual orientation. The scant workplace-based literature on this topic suggests the need for further research, using prospective designs and improved methods to determine whether these factors contribute to disparities along the tobacco use continuum.

Occupational Hazards

The broader literature on occupational disparities has found that racial/ethnic minority groups, immigrants, migrants, and workers with low wages and/or low education can face more occupational hazards than other workers.[93,95,96,98] Exposure to these hazards does not appear to be directly associated with smoking prevalence. A cross-sectional study of blue-collar and service workers from several workplaces did not find any associations between exposure to occupational hazards and smoking.[148] Other studies, which did include a multivariable analysis of the association, have found a higher prevalence of smoking among workers exposed to occupational hazards.[31] One study found higher smoking prevalence in occupations in which workers were exposed to irritating dust and fumes,[149] and one Japanese and two U.S. studies found a relationship between smoking and exposure to noise.[150–152] A multivariable analysis of more than 7,000 Swedish workers found that shift work and piece work were associated with higher odds of smoking in men, while physical load and a greater ability to interact with co-workers were associated with higher odds of smoking in women.[12]

There is evidence for an association between exposure to occupational hazards and smoking cessation and relapse. As Sorensen has noted, "workers may perceive changes in their individual health behaviors to be futile in the face of significant occupational exposure."[153,p.S197] Indeed, workers' exposure to job-related hazards has been shown to be associated with diminished interest in quitting smoking.[31,154] Results from a longitudinal study of 3,606 smokers found that, controlling for social class, the probability of smoking cessation decreased with exposure to occupational hazards.[116] A study of Norwegian nursing assistants found that a poor workplace social climate and workplace violence predicted smoking relapse.[155] However, evidence suggests that workplace health programs can be effective in improving cessation rates. For example, the WellWorks 2 study reported a doubling of smoking cessation rates among blue-collar workers in manufacturing worksites that were randomly

assigned to an intervention consisting of health promotion and occupational health protection compared with blue-collar workers in worksites that received only health promotion.[154]

Management efforts to support a healthy work environment can bolster workers' motivations to quit and can increase receptivity to messages from management about worker health.[156] This principle of integrating worksite health protection with worksite health promotion has been adopted as a research to practice priority by the National Institute for Occupational Safety and Health in its Total Worker Health™ Program.[157] To date, studies of this integrated approach have focused particularly on manufacturing workers; innovative approaches are needed that address the hazards presented in other industries. Such integrated approaches have been endorsed by the American Heart Association for cardiovascular health promotion,[158] the American College of Occupational Medicine,[159] the International Association for Worksite Health Promotion,[160] and the National Research Council.[156]

Workplace Policies

Policies that restrict smoking in the workplace have been shown to decrease exposure to SHS and produce population-wide reductions in smoking prevalence.[65,161–163] More than half of all states now provide comprehensive protection (100% smoke-free workplaces) to workers.[164] Generally, Hispanics are less likely to work in places that have formal policies banning smoking.[165–167] The gender inequality in coverage by workplace policies that restrict smoking is more complex. Overall, women are more likely than men to be covered by a comprehensive ban on smoking in the workplace,[168,169] but the coverage rate differs within female-dominated occupations. For example, in 1999, 91% of teachers were covered by such policies, compared with only 43% of food service employees.[33] Compared with white-collar workers, blue-collar and service workers are less likely to be covered by smoke-free policies that restrict or ban smoking in workplaces.[32,65,71]

Health insurance coverage for smoking cessation has been shown to increase the odds of long-term abstinence from smoking,[170,171] but health insurance coverage for smoking cessation is unequally distributed by occupational class. Historically, blue-collar workers were less likely than other occupational categories to have health insurance that includes coverage for smoking cessation.[172] A study found that, after controlling for worksite smoking policies and programs, the higher cessation rate among white-collar workers was significant when compared to the cessation rate among service workers but was not significant compared to the cessation rate for blue-collar workers.[71] The literature search did not locate studies that examined whether the Affordable Care Act has influenced insurance coverage for smoking cessation based on occupation.

Intersections of Work, Family, and Neighborhood

Although the work and family environments may be significant sources of stress for many workers, no empirical evidence associates the combination of environments with smoking.[173] However, there are indications that the disparities that exist in the tobacco use continuum by occupational class also exist in workers' households. A study of a community-based sample of blue-collar construction workers found that those who smoked were 13 times more likely to have partners who smoked.[174] Also, having a blue-collar head of household or parent has been shown to be associated with increases in the risk of smoking among adolescents and the risk of later smoking in a cohort study of U.S. women.[175,176] A study using data from the Health Behaviour in School-aged Children study (collected in 2002), found that compared with high parental occupational status, low parental occupational status was associated with a higher risk of adolescent smoking in 14 of 28 countries.[177]

In a study of public sector workers in Helsinki, Finland, higher smoking prevalence was found among women residing in areas with high levels of unemployment, single households, and single-parent families.[178] Adjusted smoking rates were between 21% and 35% higher in these areas. Smoking was more prevalent in the area where the highest percentage of manual workers lived. This study highlights the potential impact of neighborhood on smoking, and especially how neighborhood could intersect with occupation, gender, and other demographics.

Evidence Summary

This section summarizes the evidence (overall and by subgroup) on associations between occupational class, industry, or profession; employment status; the work environment; and indicators along the tobacco use continuum. The consistency and strength of results across studies were considered, along with the strength of the study designs and methodologies used, and gaps in the evidence are discussed.

Some U.S. studies and studies from other countries have found higher rates of smoking and lower rates of quitting among the unemployed compared to the employed. Apart from one study on poly-tobacco use, nationally representative studies among adults ages 18–65 in the United States did not compare associations between employment status, occupation, and tobacco use along the tobacco use continuum.

Initiation

Evidence from nationally representative studies indicates that blue-collar and service workers initiate smoking at younger ages than white-collar workers, and they may also begin using ST at younger ages. The evidence suggests that service workers might be more like white-collar workers than blue-collar workers with regard to initiation of smoking. No nationally representative studies were found that analyzed smoking initiation by race/ethnicity, gender, occupation, or exposure to SHS.

Current Use and Intensity

The evidence across nationally representative studies suggests that blue-collar and service workers are more likely to be ever-smokers, current daily smokers, and heavier smokers compared with white-collar workers. There also appear to be disparities by industry, with construction and extraction trades having the highest rates of smoking, and education and some health professions having the lowest rates. Although only two cross-sectional studies reported an interaction between occupation and gender, these studies suggested that males have higher rates of smoking than females among both blue-collar and service workers. The evidence consistently indicates an inverse association between smoking level and occupation level by race/ethnicity for non-Hispanic whites and Asian Americans/Pacific Islanders. The evidence for tobacco use by occupation among Hispanics, African Americans, and American Indians/Alaska Natives is more mixed; the evidence regarding the intensity of use by occupation and race/ethnicity is also mixed.

Few studies investigated the use of other tobacco products by occupation. Although overall use of ST is relatively low, studies show that blue-collar and farm workers have much higher rates of ST use than white-collar and service workers. In one methodologically strong study of poly-use of multiple tobacco products, no significant relationship was found between occupation or employment status and poly-tobacco use among current cigarette smokers in multivariable models.[62]

Disparities in the use of ST by occupational class and by gender and race/ethnicity have been reported, with white men having higher rates of use in all occupational classes except farm workers, where black men have the highest rates of use. Among female blue-collar and farm workers, black women were found to have higher prevalence rates than white women.

Little research was found that investigated differences by occupational class in the relationship between SHS exposure and current tobacco use. Evidence suggests that reductions in the number and use of cigarettes related to the presence of smoking restrictions were stronger among white-collar workers compared with service workers. However, no studies explored exposure to SHS and tobacco use by occupation and by gender or race/ethnicity.

The strongest evidence for the impact of the psychosocial work environment on smoking is found for the influence of job strain and physical demands on smoking intensity; those with more job strain and physical demands are more likely to be heavier smokers compared with those with less job strain. Those in lower occupational classes have more job strain and physical demands, but the literature has not examined job strain in relation to smoking by different occupational classes or industries. Evidence on the relationship between job strain and smoking by gender, age, race/ethnicity, or sexual orientation is also inadequate. The literature was inconsistent about the relationship of job insecurity to tobacco use and did not analyze the data by occupational class.

In terms of organizational factors in the work environment, the strongest evidence was found for a positive relationship between worksite racial discrimination and smoking; this relationship was not analyzed by different occupational levels.

Quit Attempts

The evidence suggests that the number of quit attempts does not differ significantly by occupational class; however, additional nationally representative studies spanning more than 1 year are needed to determine whether the rate of quit attempts varies by occupation and gender or race/ethnicity. The evidence is insufficient to draw conclusions about quit attempts by exposure to SHS or use of other tobacco products by occupation. Similarly, few studies have looked at how the psychosocial work environment affects quit attempts, and the studies that exist do not compare by different occupations.

Cessation

The evidence suggests that white-collar workers are more successful in quitting than blue-collar and service workers; this might be particularly true of quitting for 12 months or less. More nationally representative studies are needed to conclude whether quitting varies by occupational class and by gender and race/ethnicity. Studies reviewing cessation related to smoke-free policies did not discuss differences by occupation.

Exposures to occupational hazards at the worksite have been associated with decreased quitting. Workers who have higher exposures to workplace hazards have been found less likely to be successful at quitting smoking compared with those with lower exposures. No conclusions can be drawn about the relationship of job strain to cessation by occupational class or industry. There is evidence to suggest that blue-collar workers experience less pressure to quit smoking and lower social support for smoking cessation than white-collar workers. Working in jobs with greater imbalance between effort and reward is significantly associated with higher rates of smoking.

Morbidity and Mortality

Tobacco use and SHS exposure can interact with occupation to influence the risk of cancer and other diseases in a variety of ways. Research has established that the combination of cigarette smoking and exposure to asbestos is particularly hazardous. Additionally, studies have focused on the interaction of tobacco use with exposure to radon and diesel exhaust (both occupational lung carcinogens). These studies demonstrate that the interactions of occupational toxicant exposure with tobacco use are complex, and likely to differ among occupational carcinogens.

Study Design and Methodological Issues

The literature on occupational disparities across the tobacco use continuum often relied on small cell sizes in some populations, particularly when making comparisons across occupational classes or industries within demographic subgroups, such as racial/ethnic groups. Information about interactions between occupation and other markers of social disadvantage, such as low SES or sexual orientation, was not consistently available. Many studies were conducted with either one or multiple cross-sections of data. Some case-control or cohort studies have focused on specific subgroups of workers; the generalizability of findings based on subsets of a population is limited. Analyses of cessation used inconsistent definitions of quit attempts, quitting, successful quitting, and cessation.

Similarly, several issues pose challenges to understanding the contribution of the psychosocial work environment to disparities in the tobacco use continuum. Further study of measurement and operationalization of concepts of the psychosocial work environment (e.g., social norms) that remain stable across different industries and occupational classes could be valuable.[179] Also, most studies have been cross-sectional, making causal inferences difficult. Most studies of associations between the psychosocial work environment and tobacco use have examined only cigarette smoking and no other tobacco products. Finally, most of the studies were conducted in Europe or with non-diverse samples; few studies assessed the potential implications of the results for health disparities in the United States.

Chapter Summary

The 1985 Surgeon General's report, *The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace*, focused on the interaction between occupation and tobacco use.[39] More than 30 years after the report's publication, significant disparities in tobacco use persist across the tobacco use continuum by occupation. Blue-collar and service workers are more likely to be ever-smokers, current daily smokers, and heavier smokers than white-collar workers, and are less likely to quit successfully, although intentions to quit and quit attempts do not differ by occupation. Tobacco use is especially prevalent in certain industries, notably construction and extraction, mining, and hospitality and food services—settings that offer few worksite cessation programs and often lack comprehensive smoke-free policies. The work environment influences patterns of tobacco use prevalence, intensity, and cessation by occupation. Plausible pathways mediating this relationship include work-related stress, work hours, racial discrimination, pro-tobacco social norms, and lack of social support for cessation. Workers' exposure to job-related hazards appears to be associated with lower interest in quitting and less likelihood of quitting.

Worksite smoke-free policies and tobacco control programs can play a protective role in influencing workers' tobacco use patterns and their potential for SHS exposure. Workers in certain industries are less likely to be covered by smoke-free policies on the job, including construction workers who are

employed outdoors, and service workers who are employed in restaurants, bars, and casinos. Enacting and implementing smoke-free laws with no exemptions for construction sites and hospitality and gaming venues would both support workers' cessation efforts and ensure all indoor workers are protected from secondhand smoke. In addition, worksite tobacco use cessation programs can help ensure that all workers have access to such programs. Labor unions, which have a long-standing commitment to worker health and safety, may be effective partners in efforts to reduce tobacco use among workers.

Research Needs

As this chapter has explained, the relationships between occupation, demographic factors (including gender, age, race/ethnicity, and sexual orientation), and tobacco initiation, prevalence, and cessation are complex and warrant further study. Much of the literature on occupational disparities in tobacco use has examined differences by occupational category, with little attention to interactions by other sociodemographic characteristics. Future qualitative and quantitative research could examine interactions between occupation and race/ethnicity, gender, and sexual orientation to assess the extent to which occupational disparities in tobacco use, quit attempts, and cessation differ across these groups.

Qualitative and quantitative research on differences in tobacco use patterns by specific industry, in addition to broad occupational categories, can provide a more nuanced view of the impact of work experiences on tobacco use in specific settings. To facilitate accurate comparisons across industries, consistent categories of industries should be used (e.g., by Standard Industrialization Classification code). Occupation is an important social determinant of health and TRHD; inclusion of occupation in national health surveys would facilitate research.

In general, studies of populations occupationally exposed to toxicants that have the potential to increase cancer risks have focused on men. There are fewer studies of women occupationally exposed to toxicants, and limited or no studies of interactions between tobacco smoking, occupational toxicant exposures, gender, race, ethnicity, and sexual orientation as they may affect cancer risk; understanding these potentially complex interactions is a research need.

This review highlights the importance of the work environment in helping to shape patterns of tobacco use by occupation. Whenever possible, it is important that investigations examine the pathways by which occupation and on-the-job experiences influence tobacco use patterns. Research is needed to explore the potential impact of the changing nature of work, including increasing contract and contingent work as well as work performed remotely, on patterns of tobacco use and TRHD. Changes in how and where work is performed may modify the impact of the work experience on tobacco use.

This review revealed that most of the research on occupation and smoking in the United States has focused on cigarette smoking; future research could explore the use of other tobacco products by occupation, industry, and employment status. Explorations of the use of other tobacco products in the United States are particularly important given increased poly-tobacco use and the marketing and use of new and emerging tobacco products such as electronic cigarettes.

Research to develop and test multilevel interventions (i.e., at the individual, interpersonal, organization/worksite, community, and policy levels) to address occupation, the work environment, and TRHD is important. It will also be helpful to design and test the feasibility of interventions aimed at ameliorating working conditions (e.g., job strain) that may contribute to TRHD, taking worksite size into account.

Research can explore the extent to which changes in the work environment interact with supports for tobacco control and contribute to reductions in smoking. Future research can build on the successes of programs integrating tobacco control into occupational health and safety initiatives. To date, studies of this integrated approach have focused particularly on manufacturing workers; innovative strategies and studies are needed that address the hazards present in other industries.

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Section III
Interpersonal and Contextual Factors That Contribute to Tobacco-Related Health Disparities

Chapter 9
Socioeconomic Status and Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

The importance of social determinants of health, including socioeconomic status (SES), is now widely recognized.[1] The United States' Healthy People 2020, which sets 10-year objectives to improve the health of all Americans, includes as one of its four overarching goals for the decade, "create social and physical environments that promote good health for all."[2,3] Socioeconomic status is an important risk factor across the tobacco use continuum, the causal pathway in the progression of smoking to disease including initiation, current use and intensity, intentions to quit and quit attempts, cessation, relapse, and tobacco-related morbidity and mortality. The Healthy People objective for cigarette smoking is that by the year 2020, only 12% of U.S. adults will be current smokers.[3] However, trends in smoking prevalence indicate that the national benchmark of 12% will be challenging to reach, especially for groups of lower socioeconomic status, including both lower educational attainment and lower income groups. As of 2015, about 15% of U.S. adults age 18 and older were cigarette smokers. However, 26.1% of adults living below the poverty level were smokers, compared with only 13.9% of adults living above the poverty level.[4] Moreover, mortality from lung cancer is estimated to be 77% higher among adults without a high school diploma, compared to those with at least a high school diploma.[5,6]

In the United States, SES is typically measured with indicators of income (e.g., annual family income in dollars) or educational attainment (years of schooling or credentials earned). SES is a complex multidimensional construct indicative of assets available or not available to individuals, including power, prestige, and economic resources,[7] all of which confer different health advantages and disadvantages and help determine individuals' life chances. The positive association between SES and health—that higher SES translates into better health—is among the most persistent and consistent epidemiological relationship researchers have observed.[8–10]

Historically, the relationship between SES and tobacco use was reversed: Individuals with higher income were at increased risk for tobacco use and related diseases. For example, in 1940, individuals with less than a high school education were least likely to smoke (35.8% smokers), whereas those with a high school education, some college course work, or a college education had a smoking prevalence of about 40%.[11] However, by 2000, only 14.2% of those with at least a college education were smokers, whereas the prevalence rates for the lower education groups were above 25%.[11] The dynamic relationship between SES (at least as measured by educational attainment) and smoking is thought to be related in part to changes in the social meaning of smoking over time, supported by the diffusion of innovation theory.[12,13] In the early 1900s, higher SES groups had the resources to adopt innovations such as the then-fashionable trend of cigarette smoking, while lower SES groups could not. As information became available about the health consequences of smoking (e.g., the 1964 Surgeon General's report) and about cessation approaches, higher SES groups initiated smoking at lower rates, and the smokers among them quit at higher rates compared with lower SES groups. Smoking trends eventually diffused to lower SES groups and have remained more concentrated there. Several factors may help explain the concentration of smoking in low-SES groups, including greater exposure to pro-tobacco messages and access to tobacco products, combined with higher levels of stress and lower access to health care (see chapter 10). This line of reasoning is very much in line with Link and Phelan's argument regarding social factors as fundamental causes of poor health.[14] Published data support the diffusion of innovation theory and demonstrate that lower SES groups have not yet benefitted equally from the anti-tobacco messages and policies[13,15,16] discussed in chapters 10 and 11.

SES and the Tobacco Use Continuum

SES influences health in general through multiple direct, indirect, and overlapping causal pathways. For example, low SES increases the risk of direct exposure to material deprivation, such as inadequate shelter, health care, clothing, or diet, and to hazardous environmental conditions such as violence. These factors, in turn, are associated with a range of health problems. Psychosocial stress (discussed in greater detail in chapter 5), which is generally higher among people with low versus high SES, has also been shown to have direct physiological effects on the body.[17] Specific health risk behaviors and fewer social resources (perceptions of control and lower social strain) associated with low SES may also mediate the pathway by which SES influences health outcomes.[18–20]

The association between SES and tobacco-related outcomes is multifactorial in nature. Individuals with low socioeconomic resources not only have a higher prevalence of smoking, they are also more likely than higher SES individuals to be exposed to secondhand smoke (SHS) in utero,[21] in the workplace, and at home.[22–25] They are less likely to live in homes where smoking is banned[26,27] and more likely than higher SES individuals to have peers and family members who smoke.[28,29] Lower SES people are less likely to have social networks that support quitting, thus influencing their exposure to SHS and risk of smoking initiation, and contributing to poor cessation outcomes.[30,31] Chapter 6 discusses the role of social relationships in tobacco-related health disparities (TRHD).

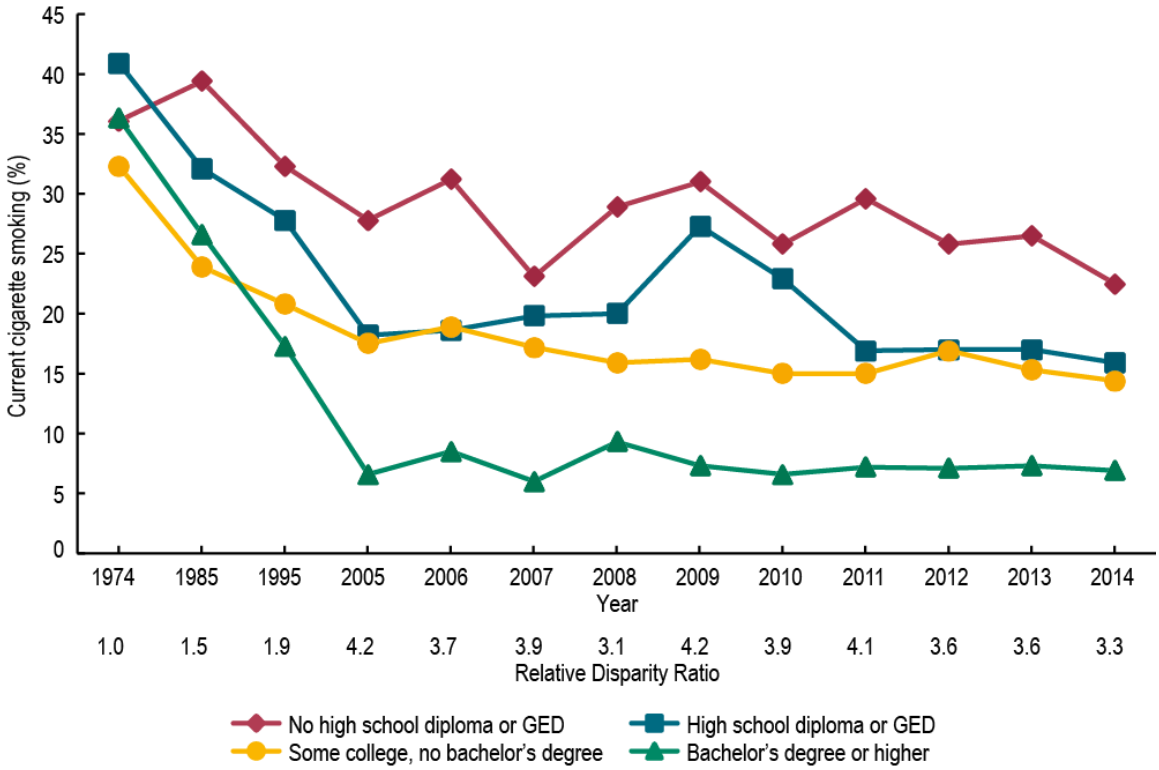
Psychosocial stress associated with low social status (both absolute and relative deprivation) combined with limited material and psychosocial resources could lead to smoking as a perceived coping mechanism or make it more difficult to quit.[13] In addition, compared with higher SES individuals, lower SES individuals, are more likely to live in lower SES neighborhoods and may be exposed to more tobacco advertising and hazardous environmental conditions (i.e., pollution) which could amplify the harmful health effects of smoking[32,33] and make it more difficult to quit.[34] Other psychosocial factors, including negative emotions, self-efficacy, and cognitive ability have been tested as mechanisms linking SES to smoking or cessation.[30,31,34,35]

Although people are generally aware that smoking is harmful to health, those health risks often compete with other, more immediate concerns, especially among lower SES groups.[31] Some evidence suggests that smoking may be more harmful to health for lower versus higher SES individuals, supporting a social vulnerability or double jeopardy model. As Pampel and Rogers argue, "the health of low status groups may be harmed most by smoking because lifestyle choices exacerbate the health problems created by deprived material conditions."[36,p.306] Although some evidence suggests that use of tobacco can result in lower wages and lower net wealth,[37,38] such reverse causation is likely to play a relatively limited role compared with the effects of SES on tobacco use.

Figures 9.1 through 9.4 present prevalence rates from 1974 to 2014 by educational attainment, stratified by race (non-Hispanic black, non-Hispanic white) and gender, using data from the National Health Interview Survey (NHIS). Although the data do not indicate what proportions of the population fall into each category of educational attainment, the relative disparity ratios for all four gender–racial/ethnic groups have increased over time among those with at least a bachelor's degree compared with those without a high school diploma or general education development (GED) (i.e., relative disparity ratios in 1974 for the four groups begin between 1.0 and 1.9; by 2014 those ratios are in the 3.2–3.9 range). This indicates a steeper rate of decline in smoking among the most highly educated compared with the least educated. Levy and colleagues[39] report, however, that between 1992 and 2002, the rate of smoking decline among women was greater for those without a high school education than for women with

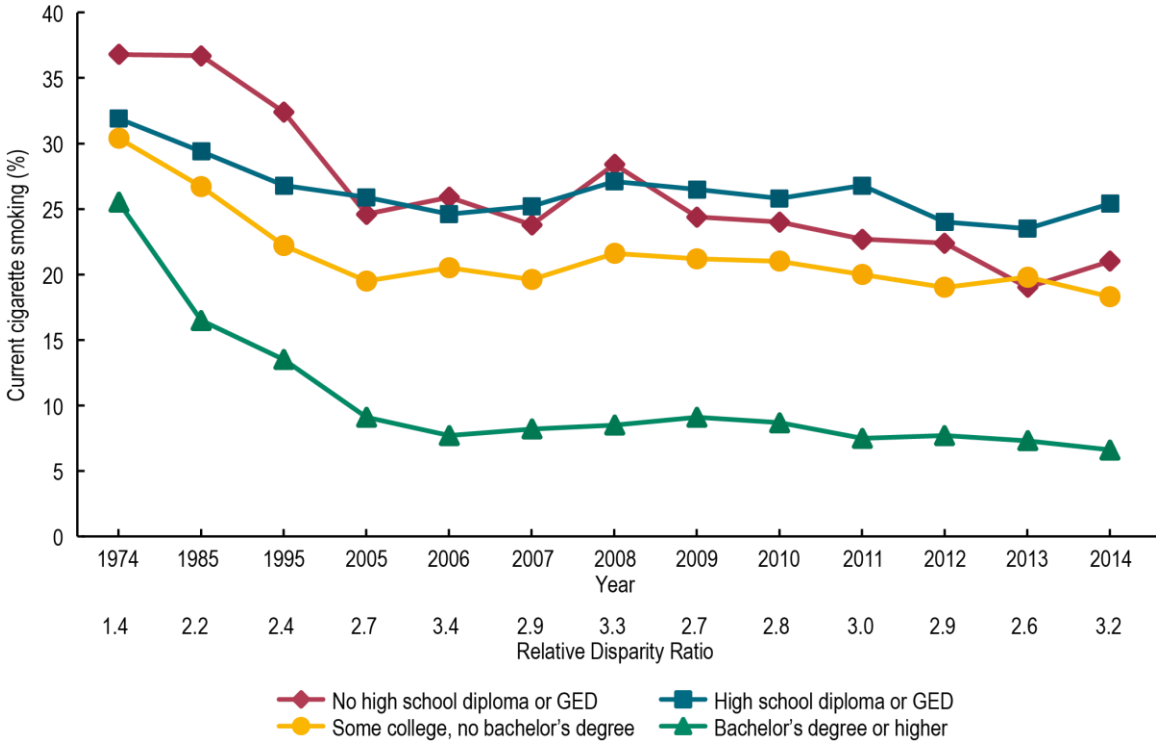
higher educational attainment. These findings might differ from those presented in the following figures because Levy and colleagues present estimates for all race/ethnicity groups combined (including white, black, Asian/Pacific Islander, Hispanic, and other), whereas the current analysis stratifies the data by white versus black/African American using data from a different time period.[39] Pampel[40] reports substantial declines in smoking among Hispanics with less education, for example, which may affect overall estimates.

Figure 9.1 Current Cigarette Smoking Among Black or African American Women, by Educational Attainment, Selected Years, 1974–2014



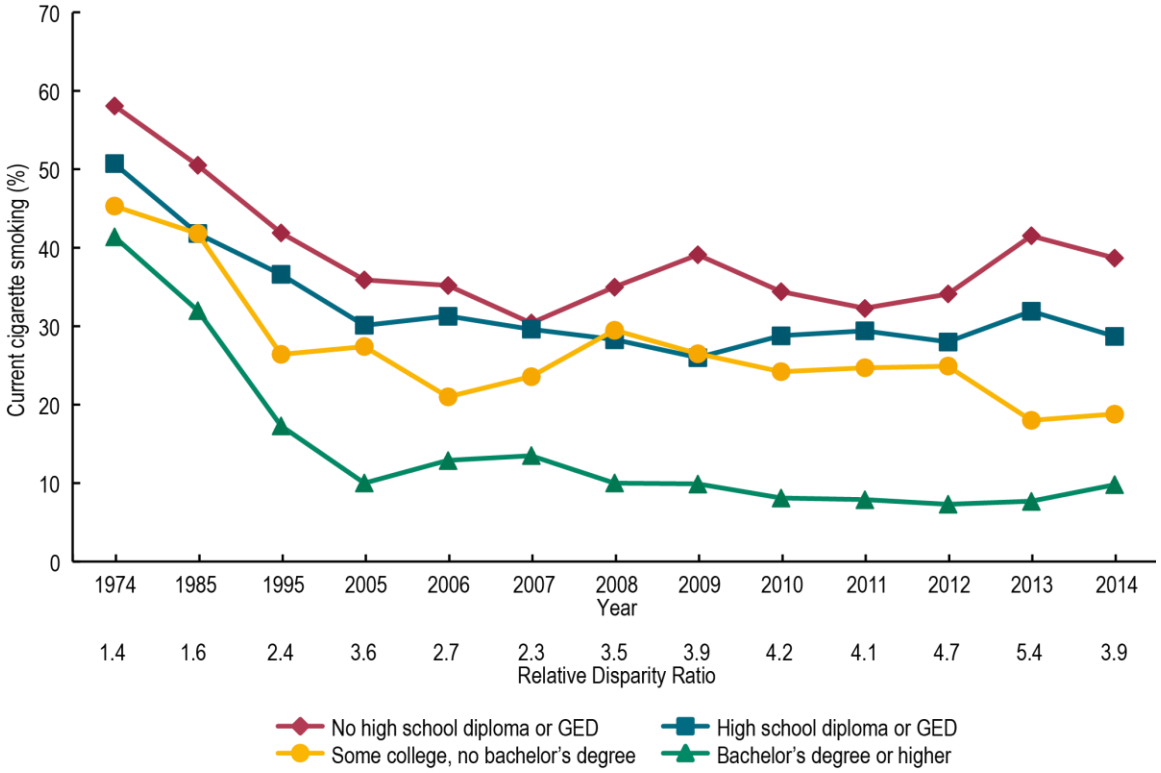
Notes: Includes people of Hispanic and non-Hispanic origin. Data prior to 1997 are not strictly comparable with data for later years due to the 1997 questionnaire redesign. See Appendix I, National Health Interview Survey. Estimates are age-adjusted to the year 2000 standard population using four age groups: 25–34 years, 35–44 years, 45–64 years, and 65 years and over. The following estimates have large standard errors (20–30% relative standard error) and are not considered reliable: bachelor's degree or higher in 1974, 2005, and 2010. Relative disparity ratios were calculated by dividing number with high school diplomas or GEDs by number with bachelor's degree or higher.
Source: Data were obtained from Centers for Disease Control and Prevention 2015 [Table 48],[145] based on National Health Interview Survey data.

Figure 9.2 Current Cigarette Smoking Among White Women, by Educational Attainment, Selected Years, 1974–2014



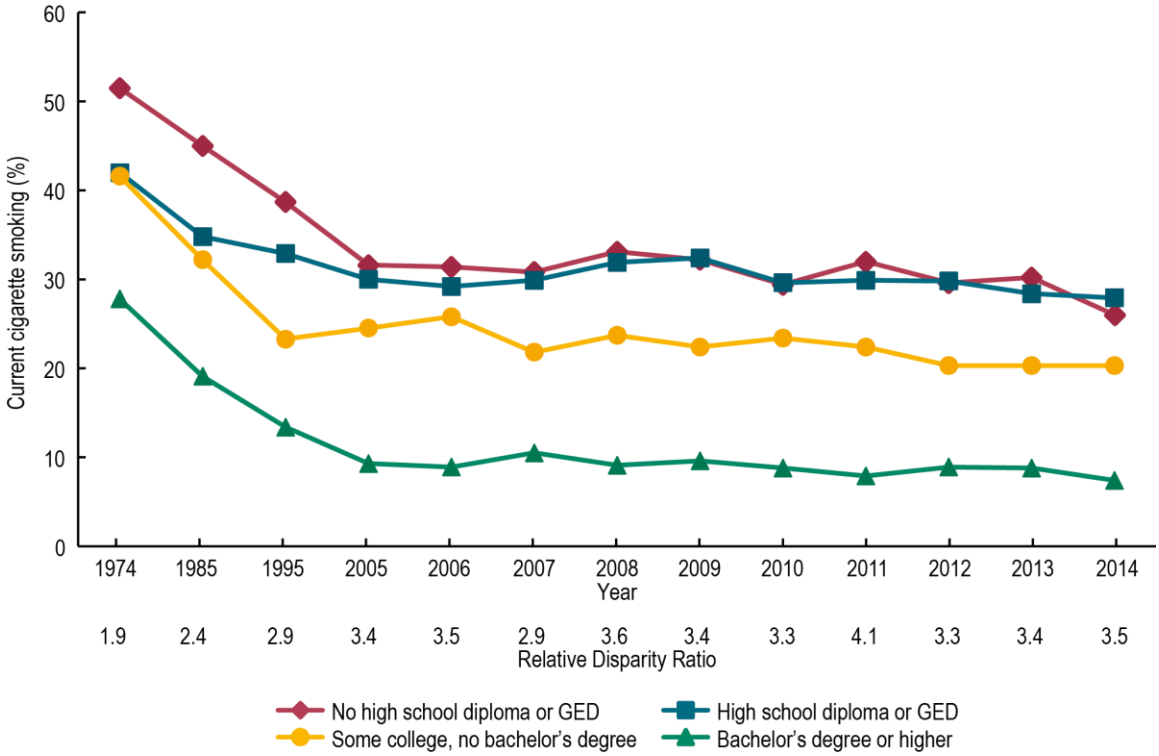
Notes: Includes people of Hispanic and non-Hispanic origin. GED = general education development. Data prior to 1997 are not strictly comparable with data for later years due to the 1997 questionnaire redesign. See Appendix I, National Health Interview Survey. Estimates are age-adjusted to the year 2000 standard population using four age groups: 25–34 years, 35–44 years, 45–64 years, and 65 years and over. Relative disparity ratios were calculated by dividing number with high school diplomas or GEDs by number with bachelor's degree or higher.
Source: Data were obtained from Centers for Disease Control and Prevention 2015 [Table 48],[145] based on National Health Interview Survey data.

Figure 9.3 Current Cigarette Smoking Among Black or African American Men, by Educational Attainment, Selected Years, 1974–2014



Notes: Estimates are age-adjusted to the year 2000 standard population using four age groups: 25–34 years, 35–44 years, 45–64 years, and 65 years and over. GED = general education development. The following estimates for black women and men have large standard errors (20–30% relative standard error) and are not considered reliable: high school diploma or GED in 1974; some college, no bachelor's degree in 1974; bachelor's degree or higher in 1974, 1985, 1995, 2002, 2003, 2007, 2008, and 2012. Relative disparity ratios were calculated by dividing number with high school diplomas or GEDs by number with bachelor's degree or higher.
Source: Data were obtained from Centers for Disease Control and Prevention 2015 [Table 48],[145] based on National Health Interview Survey data.

Figure 9.4 Current Cigarette Smoking Among White Men, by Educational Attainment, Selected Years, 1974–2014



Notes: Includes people of Hispanic and non-Hispanic origin. GED = general education development. Estimates are age-adjusted to the year 2000 standard population using four age groups: 25–34 years, 35–44 years, 45–64 years, and 65 years and over. Data prior to 1997 are not strictly comparable with data for later years due to the 1997 questionnaire redesign. See Appendix I, National Health Interview Survey. Relative disparity ratios were calculated by dividing number with high school diplomas or GEDs by number with bachelor's degree or higher.
Source: Data were obtained from Centers for Disease Control and Prevention 2015 [Table 48],[145] based on National Health Interview Survey data.

SES measurement has important implications for conclusions regarding the nature and extent of socioeconomic, and especially racial/ethnic, disparities in health.[7,8,41–43] Indicators of SES (e.g., educational attainment, income, occupational status, wealth) are not interchangeable, reflecting the multidimensional nature of the construct. Further, evidence demonstrates the different associations between SES and health—and between race/ethnicity and health—when different SES measures are used in analyses.[7,41] To illustrate, wealth refers to total financial resources amassed over a lifetime, versus income, which refers to the capital obtained during a specified period of time (e.g., annual earnings in dollars).[7,44,45] Wealth can buffer the effects of temporary low income, as in the event of illness or unemployment, and compared with income, wealth can better reflect long-term family resources and, hence, the resources available across an individual's lifetime. Wealth might be particularly important to understanding racial/ethnic disparities in health because differences in wealth by racial/ethnic group are far greater than the corresponding differences in income.

In addition, standard SES measures are often quite limited and might not always fully capture relevant aspects of the construct. For example, educational attainment reflected in credentials earned does not take into account the quality of the education attained, and there is debate as to whether education should be measured as years of schooling or credentials earned.[9] In addition, studies often combine individuals with less than 9 years of education and those with 9–11 years, although the former group has

much lower rates of smoking compared with the latter group.[46] Wealth data are also difficult to collect; the topic is considered sensitive, the collection of reliable information is laborious, and the values of assets and debts vary over time and may require professional appraisal.[44]

It is also important to take both levels and time into account when considering measurement of SES. Individual-, household- or family-, and neighborhood-level SES could each independently contribute to TRHD. Independent of an individual's SES, residence in low-SES neighborhoods, typically measured at the level of census tracts, is thought to influence health through the decreased availability of health-promoting goods and services and/or increased exposure to health-damaging residential environments (e.g., crime, noise, delinquency, tobacco and alcohol advertising and availability). In addition, normative values and behaviors, psychological stress, social cohesion among neighbors, and access to information can vary according to neighborhood deprivation and can influence the health of all residents.[47–49] The point in the life course when SES is measured (e.g., at birth, adolescence, or adulthood) may also matter. For example, parental SES during early childhood could theoretically affect the likelihood of smoking initiation among adolescents[13] and continuation of smoking during adulthood, independent of the adult's own SES.

In this chapter, multiple socioeconomic factors will be examined in relation to the tobacco use continuum, with a particular focus on the intersections between SES and race/ethnicity. Throughout this monograph, race/ethnicity is conceptualized as a social construct that reflects differences in social environments shaped by the economic and historical experiences of groups.[50,51] The distribution of socioeconomic factors such as education, income, and wealth differs substantially by race/ethnicity in the United States[43]; it is therefore critical to consider race/ethnicity and SES jointly when examining the tobacco use continuum. Given the high prevalence of tobacco use among lesbian, gay, bisexual, and transgender (LGBT) populations[52,53] and advertising by the tobacco industry targeted to them, LGBT groups were examined in relation to the tobacco use continuum if the data were further classified by SES.

Notwithstanding issues of residual confounding by SES (i.e., the inability to measure SES perfectly),[42] it is important to note that racial/ethnic TRHD cannot be reduced to SES differences alone. This is particularly relevant for current smoking because whites smoke at higher rates than most other racial/ethnic groups, except the American Indian/Alaska Native aggregate group, despite the overall socioeconomic advantage of whites compared with most other groups.[54] In addition to SES, the experiences of racism at all levels, including internalized, interpersonal, and institutional,[55] must be considered. Racism, although related to socioeconomic disparities among racial/ethnic groups (e.g., residential segregation—one form of institutional racism—influences socioeconomic attainment through the availability of high-quality education and employment opportunities) is covered in chapter 5.

Literature Search Strategy

This chapter presents a literature review on socioeconomic factors that may contribute to TRHD. Five primary socioeconomic factors were examined: (1) educational attainment, (2) income, (3) wealth, (4) neighborhood SES, and (5) life-course measures of SES. Each factor was examined across the tobacco use continuum—smoking initiation; current smoking; intensity, frequency, and duration of smoking; quitting and cessation; treatment; SHS exposure; and tobacco-related cancer morbidity and mortality. Each primary SES factor was used as a search term and combined with each tobacco use continuum indicator. Studies that examined relationships overall as well as within racial/ethnic groups were also included. Occupational status is not included as an SES indicator because the topic is discussed in chapter 8.

The search was limited to studies published between 2000 and 2011 and those using data from the United States only. For studies of neighborhood SES, the search was limited to multilevel studies using individual measures of tobacco-related outcomes (that is, ecological studies were not included).

The literature search was conducted in the PubMed, Web of Science, EconLit, and PsychInfo databases. The same search terms were used to search all databases, and multiple search terms were used for socioeconomic factors and stages on the tobacco use continuum. When searches yielded many results, more specific fields were used. After a database search was complete, all search results were merged, and duplicates, irrelevant articles, and abstract-only publications were removed. Studies such as the following were excluded: those identified in the income search that were conducted in a low-income sample but did not investigate income as an independent variable; studies identified in the neighborhood-SES search by the word *community* because they were community-based intervention trials; and studies examining neighborhood characteristics other than SES (e.g., neighborhood disorder, collective efficacy, built/physical environment).

In addition to studies identified by the initial search, studies were included that had been reviewed by Fagan and colleagues[56] or Schapp and Kunst[57] and were published (1) between 2000 and 2011 or (2) before 2001 and explicitly examined socioeconomic disparities in tobacco-related outcomes by race/ethnicity. Reference lists in articles identified by the initial search were also examined. The literature was also searched for articles that explicitly examined SES factors among LGBT populations in relation to the tobacco use continuum.

To supplement the literature review, data from the 2010 NHIS Cancer Control Supplement (CCS) were analyzed in terms of variables on the tobacco use continuum by educational attainment and income (based on federal poverty levels [FPL]) for non-Hispanic black, Hispanic/Latino, and non-Hispanic white adults. Tobacco use continuum variables included age of initiation, current smoking (smoked every day or some days), number of cigarettes smoked per day, quit attempts, years quit, use of cessation treatments, SHS exposure, and smoking-related cancer diagnosis. The sample included 27,157 respondents ages 25–64 years, of which 10,884 were ever-smokers, 5,147 were current smokers, 5,737 were former smokers, 16,083 were never-smokers, and 3,326 were current smokers who had made a quit attempt in the past year. Data on the 3,326 current smokers who had made a quit attempt were combined with data on former smokers for the cessation treatment analysis. The sample also included 7,529 respondents age 60 and older. Educational attainment was divided into four groups (less than high school, high school graduate or GED, some college, college graduate). Using the imputed income files provided by the National Center for Health Statistics for NHIS respondents with missing income data, income was defined as the ratio of total family income to the Federal poverty threshold

(<100%, 100% to <200%, 200% to <400%, and ≥400%). Prevalences that were adjusted to the age distributions from the 2000 Census were estimated using SUDAAN (version 10.0.1); means of tobacco use variables among adults ages 25–64 years and, for tobacco-related cancers, adults 60 and older were also estimated. (Results of these analyses are discussed in a subsequent section and presented in Table 9.1.)

Educational Attainment and TRHD

The initial literature search identified 36 articles that examined associations between educational attainment and the tobacco use continuum and 4 studies that examined associations between education and tobacco outcomes within LGBT populations. Fourteen studies (2000–2011) that were not identified in the initial search were also included. Because an extensive literature exists on current smoking, this review is organized into nationally representative studies of adults, non-nationally representative studies of adults, studies of tobacco outcomes during pregnancy and the post-partum period, studies of adolescents, and studies that stratified by race/ethnicity or LGBT identification.

Education and Smoking Initiation

Three studies demonstrated associations between lower education and higher prevalence/younger age of smoking initiation. One study reported that years of education were significantly correlated with smoking initiation measured as an affirmative response to the question "Have you smoked at least 100 cigarettes in your life?" with a higher prevalence of smoking initiation among those with fewer years of education.[58] Kandel[59] found that the age of smoking initiation increased as education level increased, from 14.9 years among those with less than a high school education to 16.2 years for college graduates. In a 1-year follow-up study of Houston-area students, Gritz and colleagues[60] found that having at least one parent with at least a high school diploma was protective against starting smoking among white students (odds ratio [OR] 0.48; 90% confidence interval [CI] 0.27–0.84) but was associated with higher odds of starting smoking among African American students (OR 2.12; 90% CI 1.18–3.84).

Education and Current Smoking Among Adolescents

Several studies examined associations between parental education and adolescent current smoking. Among 1,250 adolescents ages 12 to 17 in the 1993 Massachusetts Tobacco Survey (a probability sample of Massachusetts households), Soteriades and DiFranza[61] found that each decrease in the education level of a parent was associated with 31% higher odds of adolescent smoking. Education levels were categorized as some college, high school graduate, and not a high school graduate; bachelor's degree or higher was the reference category. This association was only attenuated by 10% when parental smoking was taken into account.

In addition, data from the Monitoring the Future (MTF) study—a national survey of about 50,000 students—indicate an approximately inverse gradient between parental education and prevalence of smoking in the past 30 days among 8th-, 10th-, and 12th-graders in 2011.[62] Parental education (an average score of mother's and father's education) was divided into categories ranging from completed grade school or less to graduate or professional school after college. For example, among 10th-graders with parents in the lowest education category, 14.6% reported having used cigarettes in the past 30 days compared with 16.2%, 12.7%, 8.6%, and 8.0%, respectively, for 10th-graders with parents in the increasingly higher education categories.[62]

Conversely, in a prospective study of 1,004 5th-, 8th-, and 12th-graders from Houston area schools who were followed for 1 year, Gritz and colleagues[60] found that the prevalence of ever smoking was slightly, but not significantly, higher at baseline for students whose parents had less than a high school education (23%) compared with students whose parents had a high school education (20%). Cubbin and colleagues[63] found no associations between parental education (<9th grade/some high school, high school graduate/GED, some college, college graduate) and smoking within the last 30 days among adolescents ages 12 to 17 years in the Youth Assets Study.

Unger and colleagues[64] examined associations between various indicators of SES and having ever tried smoking among 1,847 8th-grade students in Los Angeles. Four measures of SES (ZIP code, median household income, parental education, and rooms per person) were combined into a summary score. Higher SES was associated with lower odds of lifetime smoking.

Finally, data from the National Survey on Drug Use and Health (NSDUH) show that among youth ages 12–17, those who had dropped out of high school were more likely to be current smokers (45.7% in 2006-2008; 46.4% in 2009-2010) than youth who remained in school, regardless of their grade level.[65]

Education and Current Smoking Among Adults (Nationally Representative Data)

Studies using nationally representative samples of the U.S. population include the NHIS, the Behavioral Risk Factor Surveillance System (BRFSS), the Tobacco Use Supplement to the Current Population Survey (TUS-CPS), the Midlife Development in the United States (MIDUS) survey, and the Health Information National Trends Survey (HINTS). Studies using these nationally representative samples reported a strong educational gradient in current smoking.[11,40,56,59,66–73] In the 2000 NHIS data, for example, 36.7% of respondents with less than a high school education reported smoking compared with 31.9% of those with a high school diploma and 24.2% of those with some college or an associate's degree; individuals with a GED had the highest rates of smoking (53.1%).[66] A college degree, in particular, was protective against current smoking compared to having a high school degree or some college.[11,59,66,74] For example, individuals with a college degree or higher had the lowest rates of current smoking (12.5%) in the 2000 NHIS.[66]

Several other studies have also documented an inverse association between years of education and the probability of current smoking.[40,68,69,71,73] Using data from the 1992-1993 TUS-CPS, Hersch reported that years of education were associated with decreased probability of smoking, but these associations were stronger among people with high incomes (top quartile, >$54,000) than those with middle and low incomes (bottom quartile, <$17,400).[72]

Lawrence and colleagues[75] reported that young adults not currently enrolled in school were more than twice as likely to report current and daily smoking compared with those currently enrolled in school.

Kandel and colleagues,[59] using data from the 2006 National Survey of Drug Use and Health, the National Longitudinal Survey of Adolescent Health (NLSAH) (Wave III, 2001-2002), and the 2005-2006 National Health and Nutrition Examination Survey (NHANES), reported that women with less than a high school education were less likely than all other education groups to have ever smoked, but women in this category who smoked were most likely to smoke currently and persistently.[59]

Education and Current Smoking Among Adults (Non-Nationally Representative Data)

Research using non-nationally representative populations also demonstrates a strong inverse gradient in the prevalence of current smoking from lowest to highest educational attainment.[60,61,76–80] Several of these studies also demonstrated markedly lower prevalence of smoking among the college educated compared with all other groups.[77,78,81] One study of 1,699 individuals in six Chicago neighborhoods (the Sinai Health System's Improving Community Health survey, 2002-2003) demonstrated that the educational gradient in smoking differed by neighborhood in Chicago, with some neighborhoods having a strong inverse gradient and others having no gradient or a positive gradient (i.e., a lower prevalence of smoking among individuals with lower educational attainment),[76] suggesting that neighborhood characteristics are also important to consider.

Education and Current Smoking During Pregnancy

Using data on 4,911 pregnancies in the National Longitudinal Survey of Youth between 1983 and 2004, Kandel and colleagues[59] found that 45.0% of women with less than a high school education smoked during pregnancy, compared with 34.1% of high school graduates, 17.4% of those with some college education, and 5.1% of college graduates. Kahn and colleagues[21] examined predictors of smoking during pregnancy in a national cohort study of pregnancy outcomes, the 1988 National Maternal and Infant Health Survey (NMIHS, n = 9,953). This study also demonstrated a strong educational gradient, with odds ratios for smoking during the 12 months prior to delivery increasing from 2.1 (95% CI 1.6–2.8) for women with some college education (compared to college graduates), to 3.2 (95% CI 2.5–4.2) for women with 12 years of education, to 4.1 (95% CI 3.0–5.6) for women with less than 12 years. Finally, using data from the Pregnancy Risk Assessment Monitoring System (PRAMS), Tong and colleagues[82] found that women who reported smoking before or during pregnancy or after delivery were more likely to have 12 or fewer years of education (24.9% vs. 16.9%) than non-smoking women.

Education and Current Smoking, by Race/Ethnicity

Eight studies examined whether associations between education and current smoking differed by race/ethnicity.[40,64,66,73,77,79,83,84] Using 2000 NHIS data, Barbeau and colleagues[66] reported that the education gradient in smoking was strongest among whites, followed by blacks, but was less evident in Hispanic and Asian subgroups; Malmstadt[77] reported similar findings from the Wisconsin BRFSS. Kimbro and colleagues[84] used data from the 2000–2006 NHIS to determine how the relationship between education and current smoking differs by race and nativity. Gradients were less steep among foreign-born compared to U.S.-born non-Hispanic blacks, Hispanics, and Asians.[84] In an analysis of trends in educational disparities in smoking using NHIS data from 1976 to 2006, Pampel[40] found that educational disparities in smoking prevalence have narrowed over time among Hispanics; the author suggests this is due to the influx of Hispanic immigrants with low levels of both smoking and education. In contrast, the study found that educational disparities in smoking among whites and blacks have not narrowed over time.[40]

Based on 2000–2008 NHIS data, Stoddard and Adler[73] reported that years of completed schooling were associated with reduced odds of smoking, but this association was weaker among foreign-born Hispanics compared with U.S.-born Hispanics; the association between education and smoking did not differ by nativity for Asians. In addition to nativity, the authors found that years of education were more strongly associated with reduced odds of smoking based on age at immigration. The association for Hispanics who immigrated to the United States when younger than 15 years of age was stronger

compared with those who immigrated after age 15. While nativity had no effect on the education and smoking association for Asian immigrants, this group showed a similar pattern to Hispanic populations in terms of age at immigration. However, three studies reported no difference in the educational gradient by race/ethnicity.[64,79,83]

Education and Current Smoking Among LGBT Populations

Four studies were identified that examined the association between education and current smoking among LGBT populations or that compared education gradients in smoking between LGBT and heterosexual populations.[52,53,85,86] Greenwood and colleagues[52] compared data from the Gay Men's Tobacco Study, a cross-sectional survey conducted in 1999, on 1,780 men who have sex with men (MSM) to 1999 NHIS data on men in the general population of similar age and geographic residence. At all levels of education, MSM had a higher prevalence of smoking than men overall in the NHIS, and a strong inverse gradient in prevalence from low to high education was observed. Approximately 39% of MSM with less than a college education were current smokers, compared with 31% of those with a college degree and 23% of those with an advanced degree.[52]

Hughes and colleagues[85] investigated correlates of current smoking among lesbian (n = 550) and heterosexual (n = 279) women from Chicago, New York City, and Minneapolis/St. Paul, Minnesota. An inverse educational gradient in current smoking prevalence was seen among both groups. Thirty-nine percent of lesbian women with a high school education or less were current smokers compared with 20% of women with a bachelor's degree or some college and 11% of women with an advanced degree. Heterosexual women with a high school education or less were more likely to be current smokers (43%) than lesbian women (39%) with the same education, but heterosexual women with an advanced degree were less likely to smoke (7%) than lesbians (11%).[85]

Tang and colleagues[53] used data from the 2001 California Health Interview Survey to examine sociodemographic predictors of smoking among self-identified gay males (n = 593), bisexual males (n = 282), lesbian females (n = 343), and bisexual females (n = 511). The prevalence of smoking among lesbian and bisexual women without a college degree (36.6% and 32.1%, respectively) was higher than the prevalence among heterosexual women without a college degree (17.3%) and that of lesbian, bisexual, and heterosexual women with a college degree (14.6%, 18.8%, and 9.0%, respectively). A similar pattern was seen among gay men compared with heterosexual men, although the prevalence of smoking by education among bisexual men was similar to that of heterosexual men.[53]

Matthews and colleagues[86] explored predictors of current smoking among women identifying as lesbian, gay, or bisexual, or women who reported having sex with women (n = 171). Education (high school or less and some college, compared with a college or graduate degree) was not significantly associated with smoking.

Education and Current Smoking: Efforts to Estimate Causal Association

Several authors have sought to determine whether the well-documented association between educational attainment and smoking is causal or due to unobservable (i.e., confounding) factors associated with both education and smoking. Three of these studies used the Vietnam War draft as an instrumental variable for college attendance.[11,69,71] Studies using data from the 1978–2000 NHIS,[11] the 1983–1995 NHIS,[69] and the 1992–2000 TUS-CPS[71] found evidence suggestive of a causal association between college attendance and reduced smoking. Gilman and colleagues[87] compared the sibling offspring of women

in the National Collaborative Perinatal Project (NCPP) born between 1959 and 1966 (n = 1,311) with different levels of education to adjust for the effects of familial vulnerabilities to smoking; these researchers also found evidence suggestive of a causal association between education and smoking. In contrast, Tenn and colleagues[88] did not find evidence of a causal effect of each additional year of education on current smoking using data from the TUS-CPS; they concluded that the strong relationship between education and smoking is likely due to other unobserved factors correlated with both variables.

Education and Intensity, Frequency, and Duration of Smoking

Eight studies examined educational attainment and patterns of smoking; of these, all but one[58] reported that individuals with lower levels of education smoked more heavily, more frequently, or for a longer duration.[59,72,78,87,89–91] A cross-sectional survey of 2,641 ever-smokers found that college graduates had higher odds of being intermittent rather than daily smokers compared with those with less than a high school education.[89] Gilman and colleagues[87] reported that individuals with less than a high school education smoked approximately 50% more pack-years than those with college degrees, even after adjusting for multiple childhood factors; however, this association was attenuated after controlling for sibling fixed effects, which controlled for familial vulnerability to smoking. Hersch[72] reported that years of education were inversely correlated with the number of cigarettes smoked per day, and this correlation was stronger among those with high incomes compared with those with middle and low incomes.

Kandel and colleagues[59] measured the prevalence of having ever smoked daily, number of cigarettes smoked per day, nicotine dependence (a binary variable created from the continuous Nicotine Dependence Syndrome Scale), and concentrations of cotinine per cigarette smoked among women across educational categories (less than high school, high school graduate, some college, and college graduate). All measures exhibited an inverse educational gradient, women with lower educational attainment being the heaviest and most dependent smokers. In adjusted analyses, people in the higher educational groups had lower odds of nicotine dependence than those with less than a high school education. Among pregnant women, the percentage of those smoking a pack of cigarettes or more per day decreased across education levels, from 13.6% among women with less than a high school education to 7.6% among women with a high school education, 3.6% among women with some college, and 0.3% among women with a college education.[59]

Solberg and colleagues[78] also reported that people with a high school education or less had a higher prevalence of daily smoking, smoking at least two packs of cigarettes per day, and smoking within 5 minutes of waking, compared with those who had 2 or 4 or more years of college. Siahpush and colleagues[91] analyzed data from the 2003, 2006, and 2007 TUS-CPS (n = 117,168) using survival analysis to predict the duration of smoking (in years). Individuals with less than a high school education smoked for approximately 50% longer than those with at least a bachelor's degree, whereas those with a high school diploma or some college education smoked for approximately 30% longer than those with at least a bachelor's degree.[91] McCaffery and colleagues,[58] on the other hand, found no association between education and nicotine dependence (measured using the Mental Health Diagnostic Interview Schedule, Version III, Revised).

A cross-sectional study of 3,360 Mexican American and non-Hispanic white adolescents 12 to 21 years old compared mean number of cigarettes smoked per day among high school dropouts, academically

at-risk students, or students with significantly lower grade point averages than controls, and in-school controls, and found that the association between education and the number of cigarettes differed by ethnicity. In both groups, dropouts smoked the highest mean number of cigarettes, followed by at-risk students and then controls. However, means were higher across all categories of education, and differences among categories were larger among non-Hispanic whites than Mexican American youths.[90]

Education and Quitting/Cessation

Many studies have documented a strong positive gradient from lowest to highest education in the percentage of former smokers or the probability of success in quitting. These include both studies using nationally representative data[11,59,66,68–71,74] and studies using data from special populations.[34,59,79,81,87,92–96] Again, a college education appeared to be strongly associated with the increased probability of quitting successfully.[11,56,69,81]

Gilman and colleagues[87] reported that those with less than a high school education had lower adjusted rates of short- and long-term quit attempts and lower adjusted odds of cessation compared with college graduates; this finding was, however, attenuated in sibling fixed-effects models. Barbeau and colleagues[66] also found no educational gradient in quit attempts but reported that the prevalence of former smokers increased across the educational gradient; this association was true overall and among all racial/ethnic groups.

Watson and colleagues[79] found that vocational/some college and college/post-college education were associated with higher odds of being a former smoker compared with high school or less education; this finding did not differ by race/ethnicity. Data from the 2003, 2006, and 2007 TUS-CPS showed that the prevalence of quitting was positively associated with educational attainment: Only 42.5% of ever-smokers with less than a high school education were former smokers, compared with 74.1% of those with a bachelor's degree or higher.[91]

Piper and colleagues[93] assessed differences in cessation rates and treatment response by education among participants in two smoking cessation trials evaluating quit aids in Wisconsin (n = 2,850). They reported an educational gradient (less than high school, high school, and greater than high school) in initial cessation and 8-week abstinence. After 6 months, there was no difference in abstinence between those with a high school or greater than high school education, but those with less than a high school education were still least likely to be abstinent. In another study, individuals in an outpatient smoking cessation program who had a bachelor's degree or higher had a statistically significant 81% increase in odds of remaining abstinent at a 4-week follow-up, compared to individuals with a high school education, GED, or less.[92] Businelle and colleagues[34] also examined associations between an SES latent variable, including education, income, insurance status, and employment status, and cessation and found that SES was both directly and indirectly associated with cessation. The significant indirect pathways included neighborhood disadvantages, social support, negative affect/stress, and agency as mediators.

In one longitudinal study, students from 30 California and Oregon schools were recruited in grade 7 and followed up in grade 12 and at ages 23 and 29 years (n = 360).[94] Tucker and colleagues[94] examined the predictors of quit attempts and 6-month abstinence for individuals between the ages of 23 and 29 and found that higher education (a categorical variable from 1 to 11, not further described) was not associated with quitting after controlling for income and other demographic variables. Kendzor and colleagues[97] analyzed individual- and neighborhood-level socioeconomic determinants of remaining quit

for 26 weeks among 379 African American smokers in a smoking cessation intervention study. These authors found that educational attainment was *not* associated with abstinence after controlling for individual income and unemployment status. Solberg and colleagues[78] did not find a difference in quit attempts over a 12-month period by education (high school or less, 2-year college, and 4-year college) but reported that those with 2 or more years of college had a higher prevalence of reporting quitting or reducing smoking compared with those with only a high school education.

Education and Quitting/Cessation During Pregnancy

Several studies examined educational differences in smoking during pregnancy, demonstrating strong educational gradients. The first, a clinical trial (n = 316) focused on cessation in pregnant women,[81] found that among women who reported being smokers at the time they learned of their pregnancy, women with either 12 years or more than 12 years of education had higher odds of being abstinent upon entering prenatal care, at the end of pregnancy, and 24 weeks post-partum, compared with women with less than 12 years of education. This study also found that women with more than 12 years of education had the highest odds of being abstinent at each time point.

Using data on pregnant women from the 1998 NHIS supplement on pregnancy and smoking (n = 5,288), Yu and colleagues[96] found that pregnant women with less than 12 years of education who had attempted to quit smoking had approximately 12 times the odds of being an unsuccessful quitter compared with women who had 16 or more years of education; women with 12 to 15 years of education had higher odds (at least 4.5) of unsuccessful quitting than those who had a college education, while those with less than 12 years of education had the highest odds of unsuccessful quitting (12.1) compared to the highest education group.[96] Women with 12 years of education had 4.4 times the odds of being abstinent at the end of pregnancy if they smoked when entering prenatal care compared to women with less than 12 years of education, although women with more than 12 years of education did not differ significantly from those with less.[81] Educational gradients in the odds of quitting during pregnancy and relapsing post-partum were also seen in the data from the NMIHS.[21]

Data from PRAMS also show the inverse relationship between education and smoking. Among women who quit smoking during pregnancy, 46.8% had more than 12 years of education, 37.2% had 12 years, and 16% had less than 12 years of education.[82] Businelle and colleagues[35] examined multiple mechanistic models explaining the relationship between SES and post-partum smoking relapse among 251 women in a randomized trial. This study found that SES influenced post-partum relapse via increased post-partum negative affect/stress, reduced sense of agency or self-efficacy, and increased cravings, with cravings identified as being a proximal determinant of relapse.[35]

Education and Quitting/Cessation Among LGBT Populations

Burkhalter and colleagues[98] examined predictors of intention to quit among 101 LGBT individuals recruited from an LGBT community center in Manhattan in 2005. There were no differences in intention to quit between LGBT respondents with a high school education or less compared with those with more than a high school education. Comparing proportions of current and former smokers to provide estimates of cessation, Greenwood and colleagues[52] found that education was significantly associated with current versus former smoking among urban MSM. A higher proportion of men without a college degree were current smokers (62%) compared with those with a college degree (55%) and those with an advanced degree (43%).[52]

Education and Cessation Treatment

Findings from three studies have documented educational differences in the use of some (but not all) treatment/cessation aids. Solberg and colleagues[78] found no educational differences in the reported use of nicotine replacement products, use of bupropion, or support from friends, books, or groups. However, a clear education gradient emerged in receiving advice from a physician to quit: 71% of those with some 4-year college experience reported being advised to quit compared with 61% of those with some 2-year college experience and 56% of those with a high school education or less. Piper and colleagues[93] reported an educational gradient in the success of quitting with bupropion after 6 months. The results of this trial also showed that those with less than a high school education benefited more from combination therapy for quitting compared to monotherapy. In a 3-year follow-up to the 1993 Massachusetts Tobacco Survey (n = 481), Honjo and colleagues[99] reported that educational attainment was positively associated with the use of resources such as printed materials, quitlines, nicotine replacement therapy, or quitting programs.

Education and Secondhand Smoke Exposure

Tong and colleagues[27] examined educational differences in exposure to SHS among 1,879 Chinese American and Korean American women in California in 2003, using self-reports of smoke-free policies at home and work and exposure to SHS at home and work. Although the study found no educational differences in smoke-free policies, a higher percentage of women with a high school education or less reported anyone smoking at home and exposure to SHS in the last 2 weeks in an indoor workplace, compared with women who had at least some post-high school education. The less-educated group of women also had a lower probability of setting the smoking policy in their home and had more household members who smoked.

Scarinci and colleagues[100] reported that among a community sample of black and white women who had never smoked (n = 416), those with a vocational education had the highest numbers of days per week of exposure to SHS, followed by those with a college education, those with a high school education, and finally, those with post-college education. There were no significant differences in these associations by race/ethnicity. Using data from the nationally representative Women's Determinants Study (n = 2,326 nonsmokers), Stamatakis and colleagues[25] reported that women who had 8 years of education or less or were high school graduates had the highest odds of exposure to SHS at home compared with those with at least a college degree, whereas those with some high school or some college were no different from those with a college degree. Some high school education or a high school diploma was associated with higher odds of SHS exposure at work compared with at least a college degree. Honjo and colleagues[99] also found that educational attainment in years was negatively associated with hours of SHS exposure at home and work as well as with the number of peers who smoke. Data from the 2007 National Survey of Children's Health (NSCH) (n = 90,853) showed that the adjusted prevalence of exposure to SHS inside the home for children younger than 18 was 16.4% in households where the highest education was less than 12 years; where it was 12 years, 12.7%; 13–15 years, 9.1%; and at least 16 years, only 2.0%.[24]

Education and Cancer Morbidity and Mortality

Using data from the Multiethnic Cohort Study in California and Hawaii, Haiman and colleagues[101] found that both vocational training and attending some college were associated with decreased risk of lung cancer, compared with completing no more than 8 years of education. Using pooled data from 37 studies examining associations between education and oral cancer, Conway[102] calculated that low

education was associated with 1.85 times higher odds of oral cancer compared to high education. Clegg and colleagues[103] used the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) data matched to the National Longitudinal Mortality Study (NLMS) to estimate age-adjusted incidence rates for lung cancer and found a strong inverse educational gradient (from 11 years or less, 12 years, 13–15 years, and 16 or more years) among both men and women.

Siegel and colleagues[104] estimated age-adjusted lung cancer death rates (ages 25–64 years) by educational attainment and race/ethnicity. Across all racial/ethnic groups, lower educational attainment (≤12 years and 13–15 years) was associated with higher cancer death rates compared with 16 or more years of education. Non-Hispanic African American men with 12 years of education or less had the highest death rates, followed by non-Hispanic white men with 12 years of education or less. The inverse gradient was more pronounced among men than women and among non-Hispanic African Americans and whites compared with Hispanics.

Steenland and colleagues[6] used data from two American Cancer Society cohort studies, each of which enrolled more than 1 million participants, to examine associations between education and mortality due to cancer and other causes: Cancer Prevention Study I (participants enrolled in 1959) and Cancer Prevention Study II (participants enrolled in 1982). These authors categorized educational attainment as grammar school, some high school, high school graduate, some college, and college graduate. They found that, for men in both cohorts, lung cancer death rates exhibited a gradient, with the highest rates among those with the lowest education. For women in the 1959 cohort, lung cancer death rates were similar across all educational levels, except that those with a college education had lower death rates than all other women. In the 1982 cohort, however, evidence of an educational gradient in lung cancer death rates emerged. In both men and women, those with some high school had higher death rates than those with only grammar school.[6] In another study using almost 1.5 million person-years of data from the NLMS cohort the authors also found higher mortality rates from lung cancer among those with less than a high school education compared with those with at least a high school diploma.[5] Additionally, a study examining the disparity in cancer incidence by composite SES score (which included an education index) according to racial/ethnic group for five major cancer sites found that lung cancer incidence increased with lower SES, except among Hispanics, who showed an inverse effect of SES.[105] In another study, which included a group area-level SES index (including an education variable) using data from the 2000 U.S. Census to estimate total cancer mortality, including mortality from lung cancer, cancer mortality was found to be 77% higher in the lowest SES areas compared with the highest.[106]

Education: Analyses of 2010 NHIS Cancer Control Supplement Data

Data from the 2010 NHIS Cancer Control Supplement (CCS) were analyzed to augment the literature review. Table 9.1 presents age-adjusted prevalence and means for behaviors on the tobacco use continuum, stratified by educational attainment, for the three largest racial/ethnic groups in the United States. Several patterns can be seen in these data. Compared with blacks and Hispanics, whites have the highest rates of current smoking, begin smoking at a younger age, and smoke the most cigarettes per day; stepwise education gradients in the expected direction were also generally found among all groups with few exceptions. In contrast, quit attempts are highest among blacks, but no clear educational gradient was evident. For whites, quit attempts increased with increased education, but for Hispanic/Latino adults, quit attempts decreased with increased education. Among former smokers, it appears that a threshold exists for number of years quit. For each racial/ethnic group, college graduates reported the highest number of years quit; mean years quit were roughly similar for respondents at all

other education levels. Among nonsmokers, blacks and whites reported more smoking inside the home than Hispanics/Latinos, and a clear gradient by education was seen in these two groups, whereas no education gradient was seen among Hispanics. No clear pattern by education was found for the use of any type of cessation treatment for any quit attempt, whether successful or not, or for smoking-related cancer among those age 60 and older. However, differential mortality by SES may have impacted the education-cancer relationship.

Table 9.1 Age-Adjusted Percentages and Means for Indicators on the Tobacco Use Continuum Among Adults, by Educational Attainment and Race/Ethnicity, 2010

Category	Black (non-Hispanic) (n = 3,103)	Hispanic/Latino (n = 3,861)	White (non-Hispanic) (n = 10,320)
Current smokers (%)			
<High school	38.0	15.0	52.7
High school graduate	28.9	16.9	38.4
Some college	22.5	13.6	28.7
College graduate	8.0	9.2	10.3
Age of initiation among ever-smokers (mean)			
<High school	18.1	17.4	16.0
High school graduate	19.2	18.8	17.0
Some college	19.6	18.9	17.7
College graduate	20.3	19.0	18.4
Number of cigarettes per day among current smokers (mean)			
<High school	11.0	7.9	18.4
High school graduate	9.9	8.6	15.1
Some college	8.8	7.4	14.2
College graduate	7.6	5.8	10.3
Quit attempt in past year among current smokers (%)			
<High school	56.0	52.4	36.6
High school graduate	54.7	50.2	43.1
Some college	57.5	49.6	48.6
College graduate	50.7	36.4	49.9
Years quit among former smokers (mean)			
<High school	8.6	10.9	10.2
High school graduate	9.0	11.1	10.2
Some college	8.1	10.5	10.7
College graduate	11.6	11.8	12.0

Table 9.1 continued

Category	Black (non-Hispanic) (n = 3,103)	Hispanic/Latino (n = 3,861)	White (non-Hispanic) (n = 10,320)
Use of any type of treatment* during any quit attempt, among current smokers with a quit attempt in the past year and former smokers who had ever used cessation treatment (%)			
<High school	22.0	15.0	41.6
High school graduate	17.3	19.4	35.4
Some college	27.1	19.5	39.0
College graduate	32.1	†	37.9
Smoking reported inside the home by nonsmokers (%)			
<High school	29.6	6.0	35.0
High school graduate	20.8	5.1	23.0
Some college	17.5	6.5	15.2
College graduate	4.9	2.1	4.6
Ever diagnosed with a smoking-related cancer, age 60 and over (%)‡			
<High school	1.7	1.3	3.2
High school graduate	2.4	1.1	2.9
Some college	3.9	5.8	3.3
College graduate	1.4	1.2	2.4

Notes: Participants in this study were ages 25–64 (n = 17,284) or 65 and over (n = 7,067).
*Treatments included nicotine patch, gum, lozenge, nasal spray, or inhaler; prescription drugs varenicline (Chantix), bupropion (Zyban, Wellbutrin); telephone quitlines, one-on-one counseling, and cessation clinics, classes, or support groups.
†Not enough data to estimate.
‡Cancer sites include bladder, cervix, blood or bone marrow, lung, mouth/tongue/lip, throat/pharynx, kidney, stomach, pancreas, esophagus, and larynx/windpipe (adapted from Fagan et al. 2007[56]).
Source: Created using data from the National Health Interview Survey Cancer Control Supplement 2010.[144]

Income and TRHD

The initial literature search identified 29 articles examining associations between income and the tobacco use continuum. Additional studies identified after the initial search, including studies of associations between income and tobacco outcomes within LGBT populations, were also included.

Income and Smoking Initiation

Only one national study examined associations between income and smoking initiation. Using BRFSS data from 1994 through 2007, and focusing on young adults ages 18–30 and people ages 31–50, this study found that the probability that individuals in both groups would start smoking decreased with increasing income.[107] Among study participants age 51 or older, however, initiation was similar across income groups, except that the lowest income group was most likely to have started smoking.

Income and Current Smoking

The majority of studies examined associations between income and current smoking.[56,66,68,70,72,74,75,79,99,108,109] Most studies examined either household income in dollars or household income as a percentage of the federal poverty level (FPL), which takes into account the number of people in the household. Studies using both nationally representative data and non-nationally representative populations consistently reported that lower income was associated with a higher prevalence of smoking.

Income and Current Smoking Among Young Adults

Using nationally representative data from the 1998-1999 TUS-CPS, Lawrence and colleagues[75] reported that young adults ages 18–24 with a household income of less than $20,000 had higher odds of being daily smokers than young adults with higher household income. Fagan and colleagues[74] also found decreasing prevalence of smoking with increasing income (under $25,000; $25,000–$49,999; $50,000 or more) among adults ages 18 to 30 in the 2003 TUS-CPS (n = 7,912). Cubbin and colleagues[63] reported that adolescents ages 12 to 17 from households with incomes between 301% and 400% of the federal poverty line had lower odds of smoking within the last 30 days compared with adolescents from households with incomes 401% or more of the FPL; adolescents from poorer households did not differ significantly from the top income group.

Income and Current Smoking Among Adults (Nationally Representative Data)

Barbeau and colleagues,[66] using data from the 2000 NHIS, found a clear income gradient in rates of current smoking, with smoking prevalence of 34.7% for those living in poverty (<100% of the FPL), 34.2% for those near poverty (100–199% of the FPL), 31.4% for those in the middle-income group (200–299% of FPL), and 20.7% for those in the highest income group (≥300% of FPL). Data from the 1994-2004 NHIS[56] and the 2007-2008 NHIS[67] showed that the prevalence of smoking among individuals with household incomes below the FPL was approximately 10% higher than the prevalence of those with household incomes at or above poverty. Data from the 2008 BRFSS collected in 13 states[110] also documented an income gradient in cigarette smoking: 28.8% of those earning less than $15,000 per year were current smokers, in contrast to 16% among those earning $50,000–$74,999 and 12% among those earning more than $75,000 per year. Hersch[72] reported that annual family earnings are negatively correlated with the probability of smoking, and this association is stronger among low-income families (bottom quartile, <$17,400) compared to middle- and high-income families (top quartile, >$54,000). Two of these studies also demonstrated inverse associations between income and current smoking after adjusting for education and other sociodemographic variables.[66,72] In the nationally representative Health Information National Trends Survey, 54% of individuals with less than $35,000 in household income were current smokers, compared with 32% of those with $35,000–$74,999 per year and 14% of those with $75,000 or more.[70]

In contrast, Chapman and colleagues[68] reported that household income was not significantly associated with current smoking after adjusting for education, wealth, and personality factors among 2,429 adults in the 1995 Midlife Development in the United States survey.

Using data from the TUS-CPS (years 1998-1999 and 2001-2002; n = 13,480), Fagan and colleagues[109] found that among unemployed adults, current smoking prevalence decreased with increasing family income, from under $25,000 to $25,000–$49,999 to $50,000 or more; associations were robust after adjustment for education and other sociodemographic variables.

Income and Current Smoking Among Adults (Non-Nationally Representative Data)

Watson and colleagues[79] reported a nonlinear association between income and smoking among women. Women from households earning $20,000–$40,000 annually had higher odds of current smoking than those from households earning more than $40,000 (even after adjusting for education), but those in the lowest income households were not statistically different from those in the highest income households. Dell and colleagues[76] found lower prevalences of smoking among individuals with incomes above $30,000 compared with those with incomes at or below $30,000.

Honjo and colleagues[99] found that household income did not have a direct relationship with smoking. Rather, the association between income and smoking was mediated by the use of resources to quit smoking and by restrictive home environments (i.e., home smoking bans).

Income and Current Smoking During Pregnancy

Adams and colleagues[108] reported that pregnant women with annual family incomes under $16,000 had a higher prevalence of smoking during pregnancy than those with family incomes of $16,000 or more (PRAMS, 2002; n = 34,346). Tong and colleagues[82] reported similar findings using PRAMS data from 2000 to 2005. Yu and colleagues[96] also reported that pregnant women with incomes below the poverty level had higher odds of continuing to smoke (versus not smoking) compared with women who had incomes above the poverty level. The 1988 NMIHS data revealed that women with household incomes under $35,000 were more likely to smoke during pregnancy than women with incomes over $50,000 or more; odds ratios were as follows: for women with incomes under $10,000, OR was 1.9 (95% CI 1.4–2.7); for women with incomes of $10,000–19,000, OR was 1.6 (95% CI 1.1–2.1); and for women with incomes of $20,000–34,999, the OR was 1.5 (95% CI 1.1–2.0).[21]

Income and Current Smoking, by Race/Ethnicity

Barbeau and colleagues[66] reported that the income gradient was strongest among whites and was relatively strong among blacks. Among Hispanics, however, only the lowest income group had higher rates of smoking than the other groups; no gradient was evident among Asians. In their study of 715 women (43% black, 57% white) recruited from the community between 1994 and 1997, Watson and colleagues[79] did not find an interaction between race/ethnicity and income in relation to current smoking among women.

Data from the TUS-CPS show that across all racial/ethnic groups, smoking prevalence is higher among people with an annual family income of less than $25,000, but disparities remain by racial/ethnic group. For example, in 2010, among low-income adults, about 36% of American Indian/Alaska Natives reported current smoking, compared with 28% of non-Hispanic whites, 22% of non-Hispanic blacks, 13% of Hispanics, and 11% of Asian Americans.[111]

Income and Current Smoking Among LGBT Populations

Tang and colleagues[53] found that, among both male and female LGBT individuals, annual household incomes under $30,000 were associated with higher odds of smoking than were incomes of $80,000 or more. A cross-sectional survey of 580 young MSM (ages 13–29 years) from the New York City metropolitan area by Storholm and colleagues[112] found that perceived low family SES was associated with lower odds of current smoking (OR = 0.56; 95% CI 0.36–0.88) than middle family SES. In contrast, Matthews and colleagues[86] found that income was not associated with current smoking among lesbian and bisexual women.

Income and Intensity, Frequency, and Duration of Smoking

Five studies reported greater intensity of smoking among lower income smokers than higher income smokers. Ackerson and Viswanath[89] demonstrated increasing odds of being an intermittent versus a daily smoker as annual household income increased. Individuals with less than $20,000 in annual household income also had higher odds of being daily or heavy smokers compared with people with more income.[75] Monthly income was negatively correlated with cigarettes smoked per day among 263 black female participants in a randomized trial of a sexual health risk reduction program.[113] However, Hersch[72] reported that annual family earnings were not correlated with the number of cigarettes smoked per day except among high-income (top quartile) women. Siahpush and colleagues[91] reported that, compared to individuals at or above 300% of the FPL, those at or below 100% of the FPL smoked approximately 40% longer (in years), those at 100–200% of the FPL smoked almost 25% longer, and those between 200 and 300% of the FPL smoked about 12% longer.

Income and Quitting/Cessation

Income and Quitting/Cessation (Nationally Representative Data)

Four studies using nationally representative data sets reported positive associations between income and smoking cessation or the probability of being a former smoker. Binkley[107] found that the probability of quitting rose steadily with increasing household income; this gradient was steeper in middle and older age groups compared with younger smokers. Barbeau and colleagues[66] analyzed data from the 2000 NHIS and documented a strong income gradient in the prevalence of former smoking. They found that 13.3% of poor survey participants, 13.9% of near-poor participants, 16.0% of middle-income participants, and 22.3% of higher income participants were former smokers; the overall percentage of survey participants who were former smokers was 18.6%. Data from the 2003, 2006, and 2007 TUS-CPS also showed that the percentage of those who had quit increased with income in relation to the FPL.[91] Alternatively, and similar to their findings for current smoking, Chapman and colleagues[68] reported no association between income and former smoking after adjusting for education and wealth.

Income and Quitting/Cessation (Non-Nationally Representative Data)

Among young adult smokers ages 18–30, incomes between $25,000 and $49,999 were associated with lower odds of a serious intention to quit compared with those with higher incomes.[74] Unemployed individuals with family incomes under $25,000 also had lower odds of being former smokers or successful quitters compared with those with higher incomes.[109] Results from two longitudinal smoking trials—one following 424 participants for 2 years[114] and one following 6,603 participants for 13 years[115]—demonstrated that increasing household income was associated with higher odds and probability of quitting. Cui and colleagues[116] analyzed data from 1999 to 2002 on participants in a

smoking cessation program for veterans in Tennessee (n = 189) and found that veterans with annual incomes above $10,000 had a lower hazard of relapse than those in the lowest income group. One study reported no association between income and quitting among women,[79] and a study among young adults (ages 18–24) reported no association between income and being a former smoker.[75] Tucker and colleagues[94] found that household income adjusted for the number of persons supported was not associated with quit attempts or 6-month abstinence among adults ages 23–29. In their study of African American smokers in a smoking cessation intervention, Kendzor and colleagues[97] found that participants with $30,000 or more in annual household income had 2.4-times higher odds of staying quit for 26 weeks than those with incomes less than $10,000; however, this association was not significant after controlling for other individual-level measures of SES, such as unemployment.

Income and Quitting/Cessation During Pregnancy

Yu and colleagues[96] reported that pregnant women with incomes below the FPL had lower odds of initiating a quit attempt compared with women with incomes above the FPL, although the odds of quitting successfully did not differ by poverty level. Analyzing data from the NMIHS, Kahn and colleagues[21] found that women with less than $50,000 in total household income had lower, but not significantly different, odds of quitting for at least a week during pregnancy compared to women with $50,000 or more in total household income. Women with total household incomes under $10,000 had significantly higher odds (OR 2.3; 95% CI 1.1–4.8) of relapsing by 17 months post-partum compared to women with incomes of $50,000 or more.[21] Tong and colleagues[82] found that women with annual incomes of $15,000 or more were more likely to quit smoking during pregnancy (67.0%) compared to women with lower annual incomes (47.8%).

Income and Quitting/Cessation Among LGBT Populations

Limited data were available on the relationship between income and quitting smoking among LGBT populations. Among 101 LGBT individuals in New York City, Burkhalter and colleagues[98] found no differences in intention to quit among individuals with less than $50,000 in annual income compared to those with more than $50,000 in annual income.

Income and Cessation Treatment

A study using data from the 2001 NHIS found that a higher percentage of current smokers with $20,000 or more in household income reported being offered assistance in quitting from a provider compared with lower income current smokers.[117] In a clinical trial (n = 619) Cooper and colleagues[118] found no association between income and adherence to transdermal nicotine treatment (nicotine patch). On the other hand, Honjo and colleagues[99] reported that income was positively associated with the probability of using resources (e.g., printed materials, quitlines, nicotine replacement therapy, smoking cessation programs) to quit smoking.

Income and Secondhand Smoke Exposure

A number of studies have found that higher income is associated with lower exposure to SHS. Honjo and colleagues[99] found that income was negatively correlated with SHS exposure at home and work as well as with peer smoking. Another study created a combined SES variable based on both income and education using data from the 2006 and 2007 International Tobacco Control Four-Country Survey (n = 8,245); this study found that high-SES smokers had increased odds of having bans on smoking in

the home compared with low-SES smokers.[26] No associations were found, however, between income and having bans on smoking in the workplace or at bars or restaurants in the participant's area of residence.

Pyle and colleagues,[22] using a sample of parents of pediatric patients (n = 1,770), found that parents in families with less than $41,000 in annual income had higher odds of allowing smoking in the home compared to parents with more than $41,000 in income. A higher percentage of lower income parents (compared with parents with income >$41,000) also allowed smoking in a car, reported sitting in smoking areas in restaurants and trains, and allowed smoking around children. Data from the 2007 NSCH (n = 90,853) showed that the adjusted prevalence of exposure to smoke inside the home for children younger than 18 was 14.5% in households below 100% of FPL, 10.6% for households at 100–199% of FPL, 6.3% in households at 200–399% of FPL, and 2.5% in households at or above 400% of FPL.[24]

Income and Tobacco-Related Cancer Morbidity and Mortality

Conway and colleagues[102] conducted a systematic review of case-control studies to examine the association between SES and risk of oral cancer; based on five studies, the authors calculated that low income was associated with 2.41 times higher odds of oral cancer compared with high income. Clegg and colleagues[103] found evidence of increasing rates of lung cancer incidence with a decreasing ratio of family income to FPL using SEER and NLMS matched data; however, this gradient was not as strong as the gradient for educational attainment. Using NLMS data, Lewis and colleagues[5] also found that income below $60,000 was associated with an increased risk of lung cancer mortality compared with income above that amount.

Income: Analyses of 2010 NHIS Cancer Control Supplement Data

Table 9.2 presents age-adjusted prevalence and means for behaviors on the tobacco use continuum, stratified by income, for the three largest racial/ethnic groups in the United States. Several patterns can be seen in these data. As with findings for education, the data for income show stepwise increases in the expected direction for current smoking among blacks and whites and a more moderate gradient among Hispanics/Latinos. An income gradient for cigarettes smoked per day was observed only among whites. An income gradient in quit attempts was observed only among Hispanics/Latinos, but quit attempts decreased as income increased. Among former smokers, the number of years quit generally increased with higher income among blacks and whites, but no gradient was observed among Hispanics/Latinos. Similarly, the percentage of nonsmokers reporting smoking inside the home generally decreased with increasing income among blacks and whites, whereas no gradient was seen among Hispanics/Latinos. No clear pattern by income was found for age of smoking initiation, use of any type of cessation treatment, or smoking-related cancer among those age 60 and older.

Table 9.2 Age-Adjusted Percentages and Means for Indicators on the Tobacco Use Continuum Among Adults, by Poverty Level and Race/Ethnicity, 2010

Category	Black (non-Hispanic) (n = 3,103)	Hispanic/Latino (n = 3,861)	White (non-Hispanic) (n = 10,320)
Current smokers among all adults (%)			
<100%	33.9	15.9	46.1
100–<200	28.8	16.0	39.2
200–<400	18.9	14.2	28.0
400+	12.3	10.5	15.3
Age of initiation among ever-smokers (mean)			
<100%	19.1	18.4	17.2
100–<200	19.3	18.0	17.1
200–<400	19.3	18.4	17.6
400+	19.1	18.9	17.7
Number cigarettes per day among current smokers (mean)			
<100%	9.5	8.4	16.9
100–<200	10.2	8.1	15.6
200–<400	9.6	7.5	14.2
400+	9.8	7.6	12.6
Quit attempt in past year among current smokers (%)			
<100%	54.9	53.6	44.9
100–<200	58.8	50.5	43.1
200–<400	57.7	49.2	45.7
400+	55.1	33.2	46.3
Years quit among former smokers (mean)			
<100%	7.0	9.4	8.6
100–<200	9.8	11.5	9.8
200–<400	8.9	9.8	10.7
400+	10.7	12.4	11.8
Use of any type of treatment* during any quit attempt, among current smokers with a quit attempt in the past year and former smokers who had ever used cessation treatment (%)			
<100%	18.5	13.7	37.0
100–<200	22.9	20.3	30.9
200–<400	24.2	19.3	38.1
400+	23.8	13.7	42.4

Table 9.2 continued

Category	Black (non-Hispanic) (n = 3,103)	Hispanic/Latino (n = 3,861)	White (non-Hispanic) (n = 10,320)
Smoking reported inside the home by nonsmokers (%)			
<100%	7.0	1.7	6.4
100–<200	6.7	2.3	7.7
200–<400	4.1	2.6	3.2
400+	3.2	1.8	2.6
Ever diagnosed with a smoking-related cancer, age 60 and over (%)†			
<100%	3.4	1.9	2.2
100–<200	1.3	0.8	5.1
200–<400	2.7	3.0	3.5
400+	2.8	3.4	2.8

Notes: Participants in this study were ages 25–64 (n = 17,284) or 65 and over (n = 7,067).
*Treatments include nicotine patch, gum, lozenge, nasal spray, or inhaler; prescription drugs: varenicline (Chantix), bupropion (Zyban, Wellbutrin); telephone quitlines, one-on-one counseling, and cessation clinics, classes, or support groups.
†Cancer sites include bladder, cervix, blood or bone marrow, lung, mouth/tongue/lip, throat/pharynx, kidney, stomach, pancreas, esophagus, and larynx/windpipe (from Fagan et al. 2017[56]).
Source: Created using data from the National Health Interview Survey Cancer Control Supplement 2010.[145]

Wealth and TRHD

The initial literature search identified seven articles examining associations between measures of wealth, which varied from study to study, and the tobacco use continuum (specifically, current smoking as well as quitting and cessation); one additional study was later identified. No studies were identified that examined relationships between wealth and smoking initiation, intensity and frequency, treatment, SHS exposure, or cancer morbidity and mortality, nor were studies found that examined relationships between wealth and the tobacco use continuum by race/ethnicity or LGBT status.

Wealth and Current Smoking

Of the seven studies that examined measures of wealth and current smoking, four reported that higher levels of wealth were associated with lower risks of current smoking, and three did not find associations between wealth and smoking.

Wealth and Current Smoking Among Adolescents

Cubbin and colleagues[63] used data from the Youth Assets Study to examine associations between family wealth (i.e., ownership of home; savings, checking, or money market accounts or savings bonds; IRAs; tax-deferred plans [e.g., 401K]; CDs; personal loans to others; held mortgages; and stocks, bonds, or mutual funds) and smoking by adolescents ages 12–17 in the last 30 days. Wealth was not associated with smoking after adjusting for age, race/ethnicity, family structure, parents' and grandparents' education, and parents' occupation. Unger and colleagues[64] found that adolescents' self-reported possession of large amounts of personal spending money was associated with increased odds of lifetime smoking among 1,847 8th-grade students in a Los Angeles study; however, perceived ability to afford

basic necessities, family wealth relative to others, and family wealth relative to last year were not associated with smoking.[64]

Wealth and Current Smoking Among Adults

Data on 2,249 adults from the MIDUS survey (1995) demonstrated that wealth (assets minus debts) was associated with a 1% increase in the relative risk of never (versus current) smoking after adjusting for household income, education, and personality traits.[68] Cubbin and colleagues[64] examined associations between net worth and current smoking, using data from the 2004 Survey of Consumer Finances (respondents ages 25–64 years) and the 2004 Health and Retirement Survey (respondents age 50 years and older) and found an inverse gradient between net worth (measured in quartiles) and smoking after adjusting for education and income.

Grafova[119] used data from the 1999–2005 waves of the Panel Study of Income Dynamics (PSID) (n = 19,389) to examine associations between smoking and financial strain, using three measures to reflect the availability of financial resources when income is interrupted—assets, the availability of emergency funds, and financial solvency. Individuals in households without access to emergency funds were more likely to smoke than those in households with adequate emergency funds; the association between smoking and financial strain was stronger in lower income quartiles than in the top income quartile. Men and women in households without at least 3 months of income in liquid assets or at least 6 months of income in non-pension financial assets were, on average, 10% more likely to smoke than adults in families who had emergency funds available. Financial insolvency (i.e., having more debt than assets) was not associated with smoking after adjusting for individual and family characteristics. Among men only, the onset of financial strain was associated with an increase in the probability of smoking.[119]

Using data from the 1999, 2001, 2003, and 2005 PSID (not weighted to be nationally representative in this analysis), Hajat and colleagues[120] examined associations between family wealth (i.e., total assets minus debt) and current smoking. The risk of smoking declined as wealth increased; the risk for those with debt or no wealth was 2.1 times greater than the risk for those in the highest quintile of wealth. Being in the two lowest wealth quintiles was also significantly associated with an increased risk of smoking compared with being in the highest wealth quintile, after adjusting for education and income.

Wealth and Quitting/Cessation

Chapman and colleagues[68] found that wealth was associated with a 1% increase in the relative risk of former (versus current) smoking in the MIDUS survey. Grafova[119] reported that the onset of household financial strain was associated with relapse after quitting, although only among men.

Neighborhood SES and TRHD

Twelve articles were identified examining associations between neighborhood SES and the tobacco use continuum. No studies were identified that examined relationships between neighborhood SES and cessation treatment or cancer morbidity and mortality or between neighborhood SES and the tobacco use continuum by LGBT status. Individual and neighborhood SES are known to be highly correlated, so individual SES represents an important confounder in studies of neighborhood SES and tobacco outcomes. Therefore, studies that controlled for individual-level SES are noted. This review includes multilevel studies only; ecological studies were not examined. One of the primary pathways through which neighborhood SES is thought to influence tobacco use is through access to goods and services; in

general, low-SES neighborhoods have greater access to tobacco and less access to cessation resources than higher income neighborhoods. For this reason, U.S. studies that examined the built environment of neighborhoods in relation to the tobacco use continuum were reviewed.

Neighborhood SES and Smoking Initiation

Reardon and colleagues[121] analyzed data from the Project on Human Development in Chicago Neighborhoods, a multilevel, prospective, longitudinal study of children living in 79 neighborhoods. The authors conducted a multilevel event history analysis to examine the age of cigarette use initiation among 1,979 youths ages 11 to 18 years. Neighborhood poverty was not associated with age of initiation; no individual-level measure of SES was controlled for in these analyses.

Neighborhood SES and Current Smoking

Most studies examining neighborhood SES and variables along the tobacco use continuum focused on current smoking. Results were inconsistent across studies; some studies found no association between neighborhood measures of SES and current smoking, whereas others found that individuals in lower SES neighborhoods had higher risks or odds of current smoking. Associations between neighborhood SES and current smoking were also found to differ by gender and race/ethnicity, although these differences were not consistent across studies.

Neighborhood SES and Current Smoking Among Adolescents (Nationally Representative Data)

Lee and Cubbin[122] examined data on 8,165 youths ages 12 to 21 in the 1992 Youth Risk Behavior Survey, which included children from households in the nationally representative NHIS. Neighborhood SES variables—median family income at the 1990 census tract level, proportion of residents below 175% of the FPL, proportion of adults with less than a high school education, median value of owner-occupied housing, proportion of housing with more than one person per room, and proportion of people employed in blue-collar occupations—were not associated with youth smoking after adjusting for parental education.

Neighborhood SES and Current Smoking Among Adults (Non-Nationally Representative Data)

Baseline data from 1995 on 41,726 women in the Black Women's Health Study were used to examine associations between neighborhood poverty (percentage of poverty in the census tract, based on the 1990 Census) and current smoking.[123] In multilevel models adjusting for individual education and occupation as well as state-level poverty rates, increasing neighborhood poverty was associated with increasing odds of current smoking. Galea and colleagues[124] examined associations between neighborhood income and income distribution and current smoking using data from a cross-sectional survey of New York City residents in 2002 (n = 1,355). Neighborhood median household income was based on the 2000 U.S. Census and was used to calculate the Gini coefficient to measure income inequality; neither of these measures was associated with cigarette smoking after adjusting for individual income and education. Ross[125] examined associations between current smoking and SES variables including neighborhood (census tract, based on the 1990 Census), poverty (percentage of households in poverty), and education (percentage of population older than 25 with a college degree) using the 1995 Community, Crime, and Health survey, a probability sample of Illinois households. For men, neighborhood poverty was significantly associated with an increased likelihood of smoking, whereas there was no association between neighborhood poverty and smoking for women after adjusting for

education, household income, and poverty. Neighborhood education level was not associated with smoking for either gender.

Data from control participants in the Carolina Breast Cancer Study (1993–1996, n = 648) were used to assess associations between area-level characteristics from census block groups (based on the 1990 Census), including education, poverty, unemployment, vehicle ownership, home ownership, and crowding.[126] The odds of current smoking did not differ by area-level characteristics after adjusting for individual-level education. A study using data from the Black Women's Health Study (n = 41,726) linked to census tract data found that the prevalence of smoking increased as neighborhood poverty increased, even after adjustment for individual-level education, marital status, age, and occupation.[123]

Neighborhood SES and Current Smoking, by Race/Ethnicity

Cubbin and colleagues[127] examined data from NHANES III (1988–1994) linked to a neighborhood deprivation index (from 1990 Census tract variables) to examine associations with current smoking among black, Mexican American, and white women and men ages 25–64. After adjusting for education and income, they found increased odds of current smoking with each unit increase in neighborhood deprivation among black women, black men, and white women but not among Mexican Americans or white men.

In 1995-1996 Diez Roux and colleagues[128] examined associations between current smoking and six neighborhood variables at both the census tract and census block level using data from the 10-year follow-up to the Coronary Artery Disease Risk Development in Young Adults Study (CARDIA). Among whites, the following area SES variables were all significantly associated with higher odds of smoking even after adjusting for individual-level variables: lower median house value; lower percentage of college graduates; lower percentage in executive, managerial, and professional occupations; and a lower neighborhood summary score, which combined six area variables (median household income; median value of housing units; percentage of households receiving interest, dividend, or net rental income; percentage of adults who completed high school; percentage of adults who completed college; and percentage of persons in managerial or professional specialty occupations). For example, whites living in areas in the lowest quartile of median house value had 1.8 times higher odds of smoking compared to those living in the highest quartile. Among blacks, however, the odds of smoking did not differ by area characteristics after adjusting for individual variables.[128]

Scarinci and colleagues[23] used data from the 1994 baseline survey of the Memphis Health Project, a prospective study of risk factors for cigarette smoking, to examine associations between ZIP code–level educational attainment, income, and current smoking among 3,813 white and African American adolescents ages 11 to 19. No measures of individual-level SES were included in this study. Associations between neighborhood SES and smoking differed by race/ethnicity and SES indicator. African American youths living in high-income neighborhoods (above $26,500 per year) had 2.1 times higher odds of smoking than those in moderate-income neighborhoods ($20,001 to $26,500 per year) and 3.1 times higher odds of smoking than those in low-income neighborhoods ($20,000 or less per year). The authors did not find an association between neighborhood income and smoking among white youth. Overall, high neighborhood education (some college or more) was associated with reduced odds of smoking (0.60) compared to low neighborhood education (high school degree or less).[23]

Tseng and colleagues[126] analyzed a sample (n = 648) using data from the Carolina Breast Cancer Study (1993–1996) and found that area-level low education and poverty were associated with increased odds of smoking among white but not black women. Area-level low education was defined as more than 25% of residents with less than a high school education, and high education as 25% or less of residents without a high school diploma; area-level poverty was defined as more than 20% of residents having household incomes below the FPL, versus 20% or fewer having incomes below the FPL.[126]

Neighborhood SES and Intensity and Frequency of Smoking

Chuang and colleagues[129] assessed associations between neighborhood SES and cigarettes smoked per day using data from the Stanford Heart Disease Prevention Program, a cross-sectional survey of four cities in California from 1979 to 1990. Neighborhood SES was a summary score of five variables from the 1980 and 1990 Censuses: (1) percentage with less than a high school education, (2) percentage in blue-collar occupations, (3) percentage unemployed, (4) median annual family income, and (5) median housing value. Lower neighborhood SES was associated with higher levels of individual smoking, after adjusting for individual SES (based on educational attainment and household income). The results also demonstrated that the reduction in cigarettes per day associated with high individual SES was weaker in low-SES neighborhoods than in high-SES neighborhoods.

In a study using structural equation modeling to examine mediators between neighborhood SES and adolescent cigarette smoking, Chuang and colleagues[130] analyzed data on 959 adolescents ages 12 to 14 years in a nationwide randomized trial targeting family risk factors for alcohol and tobacco use through informational mailings. Adolescent smoking was measured using a scale assessing the number of cigarettes ever smoked. Low neighborhood SES was measured by the proportion of residents with family income under $12,500, proportion of unemployed males, and proportion of residents below the poverty line; high neighborhood SES was measured by the proportion of residents with family income greater than $75,000, proportion of residents in managerial occupations, and proportion of residents with more than 12 years of education. Low neighborhood SES was associated with increased parental monitoring, which was associated with decreased levels of smoking; high neighborhood SES had no direct or indirect effects on adolescent smoking.[130]

In a study of adolescents younger than age 18 in the National Longitudinal Survey of Adolescent to Adult Health (Add Health, n = 9,463), high levels of neighborhood poverty were associated with moderate increases in smoking frequency and quantity for white, but not black, adolescents.[131] In longitudinal follow-up analyses at 1 and 6 years, however, neighborhood poverty was not a strong predictor of adolescent smoking. Neighborhood poverty was measured using a combined score of the proportion of families below the FPL, median family income, and the proportion of single-parent families; analyses controlled for family income.

Stimpson and colleagues[132] assessed associations between neighborhood deprivation and cotinine levels using data from NHANES III (n = 20,050). Neighborhood deprivation, based on the Singh composite index[133] of indicators in the U.S. Census, was associated with increased odds of cotinine concentrations greater than 14ng/mL, indicative of smoking; a gradient in odds of more than 14ng/mL of cotinine was shown across increasing quartiles of deprivation. The analyses controlled for individual-level education, income, and employment status.

Neighborhood SES and Quitting/Cessation

Tseng and colleagues[126] used data from the Carolina Breast Cancer Study to examine associations between census block group characteristics and former smoking. Neighborhood-level characteristics were not associated with former smoking after controlling for individual-level education. Kendzor and colleagues[97] found that the percentage of unemployment at the census tract level was significantly negatively associated with staying quit for 26 weeks in a smoking cessation intervention.

Neighborhood SES and Secondhand Smoke Exposure

In a sample of 416 never-smokers from a large 2-year prospective evaluation of the determinants of weight gain among black and white women, Scarinci and colleagues[100] reported that women living in ZIP codes with a median income of less than $21,152 and between $21,152 and $35,377 reported approximately 4.5 days per week of SHS exposure compared with 3.7 days per week for those living in ZIP codes with incomes above $35,377, but these differences were not statistically significant. These associations also did not differ significantly by race/ethnicity.

Neighborhood: The Built Environment and the Tobacco Use Continuum

Literature on neighborhood socioeconomic status and the tobacco use continuum hypothesizes that the built environment can represent a primary pathway through which neighborhood SES affects tobacco outcomes. The built environment has been defined as the human-made space in which people live, work, and recreate on a day-to-day basis.[134] Features of the built environment that could be relevant for tobacco outcomes include the density and accessibility of tobacco outlets, availability and accessibility of cessation resources, and prevalence of pro-tobacco advertising and anti-tobacco messaging.

The study by Chuang and colleagues[129] examining interactions between individual and neighborhood SES and cigarettes smoked per day sought to understand the role of convenience store concentration, an indicator of cigarette availability, on smoking prevalence. The authors examined the concentration of convenience stores within a participant's census tract, distance between participants' households and the nearest convenience store, and number of convenience stores within a 1-mile radius of a participant's home. Higher convenience store density and shorter distance to a convenience store were associated with higher average cigarettes per day after adjusting for individual characteristics. Furthermore, an interaction was found between density and neighborhood SES; convenience store density was positively associated with cigarettes per day in high-SES neighborhoods but not in low-SES neighborhoods.

Novak and colleagues[135] also examined tobacco outlet density and smoking among a sample of 2,116 youths in Chicago. Youths living in neighborhoods in the highest quartile in terms of tobacco outlet density had 13% higher odds of smoking in the past month compared with those in the lowest quartile. The authors used propensity score matching to account for potential neighborhood-level confounders.

Reitzel and colleagues[136] examined associations between tobacco outlet density and residential proximity to tobacco outlets on smoking abstinence at 6 months in a longitudinal cohort study of smoking cessation in Houston. Density and proximity were measured using spatial analysis tools, and individual-level characteristics were controlled for in the models. Study participants living less than 250 meters or less than 500 meters from the closest tobacco outlet were less likely to remain abstinent than those living farther away. The density of tobacco outlets, however, was not associated with abstinence.

Life-Course SES and TRHD

The initial literature search identified four articles examining associations between socioeconomic status over the life course and the tobacco use continuum, and one additional article was identified in the Fagan review.[56] No studies were identified that examined relationships between life-course SES and cessation treatment or between life-course SES and tobacco use outcomes by race/ethnicity or LGBT status.

Life-Course SES and Smoking Initiation

To investigate associations between childhood SES and smoking in adulthood, Gilman and colleagues[137] used data on the offspring of mothers enrolled in the Providence, Rhode Island–Brown University site of the National Collaborative Perinatal Project (NCPP) (n = 1,057); the subjects were between the ages of 30 and 39 years at the time of this follow-up study. Childhood SES was measured at the time of the offspring's birth and at age 7 (combined scores were created) using maternal education in years, parental occupation (defined as either manual or non-manual according to 1960 U.S. Census categories), and household poverty status. In multivariable models, childhood parental occupation and household poverty were associated with smoking initiation (not adjusting for adult SES variables). Specifically, parental manual occupation was associated with a 49% increase in the risk of initiation, and household poverty was associated with a 33% increase in the risk of initiation. Childhood maternal education was also associated with progressing to regular smoking at an earlier age (adjusting for adult educational attainment).

Life-Course SES and Current Smoking (Nationally Representative Data)

In a prospective study of 10,142 young adults from the Add Health study, McDade and colleagues[138] found that higher parental education (highest level of either mother or father, categorized as less than high school, high school graduate, some college, college graduate, and more than college), as assessed when the child was in the 7th through 12th grades, was associated with reduced likelihood of smoking between ages 18 and 26. This study did not control for participants' current (adult) education.[138]

Life-Course SES and Current Smoking (Non-Nationally Representative Data)

Fagan and colleagues[139] analyzed data from a longitudinal study of 603 adults first interviewed around age 5 in 1975 and followed to a mean age of 27 in 1997 to examine associations between parental education/occupation and later life smoking. Smoking was measured along a scale from never smoked, former smoker, less than daily smoker, 1–5 cigarettes per day, about half a pack per day, about 1 pack per day, and 1½ or more packs per day. In structural equation models, higher parental education (a latent variable based on continuous measures of both maternal and paternal education) was directly associated with lower levels of smoking in adulthood and was indirectly associated with adult smoking via improved parent-child relationships and lower levels of adolescent smoking. Parental occupation was associated with adult smoking only through these mediated pathways. No measures of the respondent's educational attainment in adulthood were included in the models.

Tehranifar and colleagues[140] examined a sample of female participants in the NCPP for associations between parental education and occupation during the participants' early childhood and the participants' education measured during adolescence and early adulthood, controlling for income in adulthood. Participants were born in New York City between 1959 and 1974 and were followed into adulthood between 2001 and 2006 (n = 262). Having a parent in a blue-collar occupation when the child was born

was associated with 2.7 greater odds of the child being a current smoker during adulthood compared with having a parent in a white-collar occupation; other childhood and adolescent measures of SES were not associated with current smoking.

Life-Course SES and Intensity and Frequency of Smoking

McDade and colleagues[138] also found that higher parental education (highest education of either mother or father), as assessed when the child was in the 7th through 12th grades, was associated with reduced numbers of cigarettes smoked per day between ages 18 and 26. For example, young adults whose parents were college graduates smoked 0.94 fewer cigarettes per day compared with those whose parents were high school graduates.

Life-Course SES and Quitting/Cessation

Gilman and colleagues[137] also examined NCPP data on smoking cessation and found that maternal education and parental occupation in childhood were not associated with odds of quitting for at least 1 year, after adjusting for adult educational attainment and occupation.

Life-Course SES and Cancer Morbidity and Mortality

Singh and colleagues[141] used data from the National Cancer Institute's SEER database combined with area-level poverty rates from the U.S. Census to investigate associations between census tract poverty rates and lung cancer incidence by race. Age-adjusted lung cancer incidence rates increased with increasing neighborhood poverty (<10%, 10–20%, and ≥20% poverty) among men but not women. When stratified by race/ethnicity, this gradient was observed among both non-Hispanic white men and women and black men and women and, to a lesser extent, among Asian/Pacific Islander men. However, lung cancer incidence was lowest in the highest poverty census tracts for American Indian/Alaska Native men and women, and a moderate inverse gradient was observed among Hispanic men and women.

Evidence Summary

A summary of the findings of studies reviewed in this chapter is provided in Table 9.3.

Table 9.3 Summary of Findings on SES Measures, Stage of the Tobacco Use Continuum, and TRHD 2000–2011

Measure/Stage	Lower SES, less severe TRHD*	Lower SES, more severe TRHD†	Null findings
Education			
Current smoking			
Nationally representative – adults		Barbeau et al. 2004[66]; CDC 2009[67]; Chapman et al. 2009[68]; de Walque 2004,[11] 2007[69]; Fagan et al. 2007[56]; Grimard & Parent 2007[71]; Hersch 2000[72]; Kandel et al. 2009[59]; Kimbro et al. 2008[84]; Pampel 2009[40]; Stoddard & Adler 2011[73]	Tenn et al. 2010[88]
Non-nationally representative – adults		Dell et al. 2005[76]; Gilman et al. 2008[87]; Higgins et al. 2009[81]; Kahn et al. 2002[21]; Karter et al. 2008[83]; Malmstadt et al. 2001[77]; Solberg et al. 2007[78]; Tong et al. 2009[82]; Watson et al. 2003[79]; Wetter et al. 2005[80]	
Adolescents		Johnston et al. 2012[62]; Soteriades & DiFranza 2003[61]; Unger et al. 2007[64]; Garrett et al. 2013[65]	Cubbin et al. 2011[63]; Gritz et al. 2003[60]
Smoking initiation	Gritz et al. 2003[60]	Gritz et al. 2003[60]; McCaffery et al. 2008[58]	
Age of initiation		Kandel et al. 2009[59]; McCaffery et al. 2008[58]	
Heaviness/frequency/ duration of smoking		Ackerson & Viswanath 2009[89]; Aloise-Young et al. 2002[90]; Gilman et al. 2008[87]; Hersch 2000[72]; Kandel et al. 2009[59]; Siahpush et al. 2010[91]; Solberg et al. 2007[78]; Webb & Carey 2008[113]	McCaffery et al. 2008[58]
Quitting/cessation			
Nationally representative – adults		Barbeau et al. 2004[66]; Chapman et al. 2009[68]; de Walque 2004,[11] 2007[69]; Fagan et al. 2007[74]; Finney Rutten et al. 2005[70]; Grimard & Parent 2007[71]; Kandel et al. 2009[59]; Siahpush et al. 2010[91]	
Non-nationally representative – adults		Businelle et al. 2010[34]; Foulds et al. 2006[92]; Gilman et al. 2008[87]; Higgins et al. 2009[81]; Piper et al. 2010[93]; Tucker et al. 2005[94]; Watson et al. 2003[79]; Wetter et al. 2005[95]; Yu et al. 2002[96]	Kendzor et al. 2012[97]
Treatment		Honjo et al. 2006[99]; Piper et al. 2010[93]; Solberg et al. 2007[78]	Solberg et al. 2007[78]
Exposure to SHS		Honjo et al. 2006[99]; Scarinci et al. 2000[100]; Singh et al. 2010[24]; Stamatakis et al. 2002[25]; Tong et al. 2009[27]	
Cancer morbidity/ mortality		Clegg et al. 2009[103]; Conway et al. 2008[102]; Haiman et al. 2006[101]; Lewis et al. 2009[5]; Siegel et al. 2011[104]; Steenland et al. 2002[6]	
Income			
Current smoking			
Nationally representative – adults		Barbeau et al. 2004[66]; Fagan et al. 2007,[74] 2007,[56] 2007[109]; Finney Rutten et al. 2005[70]; Hersch 2000[72]; Kahn et al. 2002[21]; Tong et al. 2009[82]	Chapman et al. 2009[68]

Table 9.3 continued

Measure/Stage	Lower SES, less severe TRHD*	Lower SES, more severe TRHD†	Null findings
Non-nationally representative – adults		Adams et al. 2008[108]; Dell et al. 2005[76]; Fagan et al. 2007[56]; Malmstadt et al. 2001[77]; Watson et al. 2003[79]; Yu et al. 2002[96]	Honjo et al. 2006[99]
Young adults		Cubbin et al. 2011[63]; Lawrence et al. 2007[75]	
Initiation		Binkley 2010[107]	
Heaviness/frequency/ duration of smoking		Ackerson & Viswanath 2009[89]; Hersch 2000[72]; Lawrence et al. 2007[75]; Siahpush et al. 2010[91]; Webb & Carey 2008[113]	
Quitting/cessation			
Nationally representative – adults		Binkley 2010[107]; Barbeau et al. 2004[66]; Fagan et al. 2007,[74] 2007[109]; Siahpush et al. 2010[91]	Chapman et al. 2009[68]; Lawrence et al. 2007[75]
Non-nationally representative – adults		Burkhalter et al. 2009[98]; Cui et al. 2006[116]; Fagan et al. 2007[56]; Hyland et al. 2004[115]; Kahn et al. 2002[21]; Kendzor et al. 2010[114]; Tong et al. 2009[82]; Yu et al. 2002[96]	Kendzor et al. 2012[97]; Tucker et al. 2005[94]; Watson et al. 2003[79]; Yu et al. 2002[96]
Treatment		Browning et al. 2008[117]; Honjo et al. 2006[99]	Cooper et al. 2004[118]
Exposure to SHS		Honjo et al. 2006[99]; King et al. 2011[26]; Pyle et al. 2005[22]; Singh et al. 2010[24]	
Cancer morbidity/ mortality		Conway et al. 2008[102]; Lewis et al. 2009[5]	
Wealth			
Current smoking			
Nationally representative – adults		Chapman et al. 2009[68]; Cubbin et al. 2011[44]; Grafova 2011[119]	
Non-nationally representative – adults		Hajat et al. 2010[120]	
Adolescents			Cubbin et al. 2011[63]; Unger et al. 2007[64]
Quitting/cessation		Chapman et al. 2009[68]; Grafova 2011[119]	
Neighborhood SES			
Current smoking			
Nationally representative – adults		Cubbin et al. 2001[127]	Cubbin et al. 2001[127]; Lee & Cubbin 2002[122]
Non-nationally representative – adults		Diez Roux et al. 2003[128]; Datta et al. 2006[123]; Ross 2000[125]	Diez Roux et al. 2003[128]; Galea et al. 2007[124]; Ross 2000[125]; Tseng et al. 2001[126]
Adolescents	Scarinci et al. 2002[23]	Scarinci et al. 2002[23]	Lee & Cubbin 2002[122]; Scarinci et al. 2002[23]
Initiation			Reardon et al. 2002[121]

Table 9.3 continued

Measure/Stage	Lower SES, less severe TRHD*	Lower SES, more severe TRHD†	Null findings
Heaviness/frequency/ duration of smoking	Chuang et al. 2005[130]	Chuang et al. 2005[129]; Nowlin & Colder 2007[131]; Stimpson et al. 2007[132]	Chuang et al. 2005[130]; Nowlin & Colder 2007[131]
Quitting/cessation			Tseng et al. 2001[126]
Exposure to SHS			Scarinci et al. 2000[100]
Life-Course SES			
Current smoking			
Nationally representative – adults		McDade et al. 2011[138]	
Non-nationally representative – adults		Fagan et al. 2005[139]; Tehranifar et al. 2009[140]	
Adolescents			
Initiation		Gilman et al. 2003[137]	
Heaviness/frequency/ duration of smoking		McDade et al. 2011[138]	
Quitting/cessation			Gilman et al. 2003[137]

Notes: Some publications are listed in multiple columns due to findings differing by specific population characteristics. SES = socioeconomic status. TRHD = tobacco-related health disparities. SHS = secondhand smoke exposure.
*Finding indicates that a lower level of SES is associated with a better tobacco-related outcome in terms of health, such as lower levels of smoking, older age of initiation, higher levels of quitting or using treatment, and lower levels of exposure to secondhand smoke or cancer.
†Finding indicates that a lower level of SES is associated with a worse tobacco-related outcome in terms of health, such as higher levels of smoking, younger age of initiation, lower levels of quitting or using treatment, and higher levels of exposure to secondhand smoke or cancer.

Among SES factors, the evidence is strongest for an association between adult educational attainment and indicators along the tobacco use continuum: current smoking, quantity smoked, quitting, SHS exposure, and tobacco-related cancer. There were strong, consistent educational gradients overall and among blacks and whites for current smoking; gradients were less pronounced among Hispanics/Latinos, Asian/Pacific Islanders, and American Indians/Alaska Natives, although small sample sizes make these analyses less certain. In addition to overall gradients, with each increasing year or degree of education being associated with a lower prevalence or likelihood of smoking, a college education appears to confer a particularly protective benefit in terms of current smoking.

Associations between education and tobacco use were found in data from both nationally representative and non-nationally representative samples. The majority of the studies, however, employed cross-sectional designs. Prospective designs were found largely among studies examining initiation,[60] quitting and cessation,[78,81,93–95] and cancer mortality.[6,101] Studies also find that higher maternal education is linked to a much lower likelihood of smoking during pregnancy.[21,59,81,82]

Findings on the relationship between education and current smoking among adolescents were mixed; three studies found an inverse association between education and smoking, while two found no association. The limited evidence on the association between education and smoking initiation also had mixed findings. Three studies found that higher education was protective against initiation or was

associated with an older age of initiation, while one study found that among black adolescents, higher parental educational attainment was associated with higher odds of initiation. An analysis of NHIS 2010 data conducted for this chapter revealed differences as large as 40 percentage points (unadjusted, except for age) in the prevalence of current smoking among respondents who did not complete high school compared to those who graduated from college. The findings were also mixed for cessation treatment; the inconsistency could be due to the variety of ways in which treatment (e.g., health professional advice, quitlines, prescription drugs) is measured.

One proposed causal pathway linking education and tobacco use suggests that people with less education have fewer resources or are less able to take advantage of the resources they do have to quit smoking.[14] The literature reviewed in this chapter, which demonstrates that higher education is associated with a higher rate of receiving advice about quitting from health professionals and greater use of quitting resources, is consistent with this hypothesis. The normative behavior of peers may also have a role; individuals with higher education may be less likely to have peers who smoke or less likely to be exposed to SHS in the workplace.

Studies on the relationship between income and the tobacco use continuum most commonly examined current smoking (overall and within racial/ethnic groups), quantity smoked, quitting, or SHS exposure. Except for quitting, most of the studies suggest an inverse association with income, regardless of the measure of income that is used. For quitting, nine studies found an inverse association (three based on nationally representative data), but five studies suggested a null association (one based on nationally representative data). More variables were used to measure income than were used to measure education, which could account for the mixed findings. Most of the studies were based on cross-sectional designs, except for three prospective studies of quitting[94,115,142] and one study of treatment.[118] Only one study examined smoking initiation, and the findings on treatment were mixed. Three studies found an inverse association between income and cancer outcomes. Prevalence rates of current smoking did not vary as dramatically across income groups as they did for levels of education. For example, in the 2000 NHIS, the prevalence of smoking in the group with the lowest income was only 14% higher than that in the group with the highest income.[66]

Seven studies were identified on the relationship between wealth and indicators along the tobacco use continuum. Four of the five studies among adults found inverse associations between wealth and current smoking, with two of these studies reporting modest or strong associations. Two studies also found that wealth was inversely related to quitting smoking. Two studies among adolescents reported no association between family wealth and current smoking. Three of the seven studies on wealth used prospective study designs, and all studies adjusted for other measures of SES (e.g., education, income). Similar to income, there are theoretically plausible links between wealth and tobacco-related outcomes; low levels of wealth may be associated with psychosocial stress, pro-tobacco peer or community norms, increased exposure to tobacco advertising, less availability of tobacco dependence treatment, and other factors.

Although 13 studies were identified which examined measures of neighborhood SES and indicators along the tobacco use continuum, few consistent findings emerged. In nationally representative data sets, two studies found no association between neighborhood deprivation and current smoking, whereas one found an inverse association. Findings from studies using non-nationally representative data on adolescent smoking were also mixed, and findings within studies differed by race/ethnicity and gender.

Only one study examined the initiation of smoking, reporting no association.[121] Studies looking at quitting[126] and SHS exposure[100] both reported null associations.

Findings from the four studies examining neighborhood SES and intensity of smoking were mixed. One study found that although neighborhood SES was associated with smoking frequency and quantity in the cross-sectional sample, the association was not present in a longitudinal design.[131] This finding highlights a challenge with the literature: It is difficult to determine whether neighborhood SES is positively correlated with tobacco outcomes or whether the selection of individuals into neighborhoods introduces bias. The inconsistency of findings in the literature on neighborhood SES might be attributable to the variety of measures of neighborhood SES; different definitions of neighborhoods (e.g., census tracts, ZIP codes); and different control variables, including individual SES measures, in the models. The strength of association between neighborhood SES variables and outcomes on the tobacco use continuum was generally modest. Neighborhood SES could theoretically be a causal determinant of tobacco-related outcomes because such factors as the availability of tobacco, tobacco advertising, cultural norms, and stress could differ by neighborhood SES. In light of the different methods used and inherent selection and measurement issues in neighborhood effects research, it might be best to consider the magnitude of an observed neighborhood effect as representing a range, with the true value lying somewhere between the crude and adjusted association.[143]

Only four studies were identified that examined associations between life-course SES measures and outcomes on the tobacco use continuum. These studies reported associations between parental education in childhood and smoking in adulthood in both nationally representative and non-nationally representative data. Studies also reported associations between parental occupation in childhood and adult smoking in non-nationally representative data. Parental education and occupation were also found to be associated with initiation of smoking, progression to regular smoking, and intensity of smoking, but not with quitting. Although all studies were prospective designs, only two of four studies controlled for adult measures of SES. These two studies reported strong associations between life-course measures of SES and adult smoking and initiation, but associations between parents' education and the number of cigarettes smoked were more modest.

Few studies examined the tobacco use continuum by race/ethnicity or sexual orientation, and most of these examined current smoking. Little evidence on socioeconomic disparities by race/ethnicity or LGBT status exists for other indicators across the tobacco use continuum. Although LGBT populations have higher rates of current smoking than heterosexual populations, the evidence is insufficient to determine whether SES has a differential impact on current smoking or other tobacco-related indicators in the LGBT population.

Several limitations in this review and in the literature should be noted. The search, although thorough, might not have identified every relevant study. Most of the studies identified were based on self-reported data and cross-sectional study designs. Because of the nature of SES, it is difficult to firmly establish a high degree of internal validity. Despite these limitations, the majority of studies examined, for all socioeconomic factors across the tobacco use continuum, found that people of low SES have more negative outcomes, suggesting that these factors contribute to TRHD.

Chapter Summary

As described by Healthy People 2020 "social determinants of health are conditions in the environments in which people are born, live, learn, work, play, worship and age, that affect a wide range of health, functioning and quality-of-life outcomes and risks."[3] Socioeconomic status, the focus of this chapter, relates to each of the five Healthy People 2020 areas of social determinants of health: economic stability; education; social and community context; health and health care; and neighborhood and built environment.

As the chapter describes, there is very strong evidence showing that educational attainment is closely linked with tobacco use across the continuum; this evidence is strongest for white and black populations. Small sample sizes and lack of focus on acculturation and nativity make conclusions regarding the relationship between education and tobacco use for other racial/ethnic groups less firm. Educational gradients appear to exist among LGBT populations as well, although fewer studies have examined this population group. Education is closely associated with cognition and social capital and also helps determine other socioeconomic factors, such as occupation, income and wealth, and type of neighborhood. As Link and Phelan have said, "social factors such as socioeconomic status…are likely 'fundamental causes' of disease."[14,p.80] Given the consistent association with tobacco use, and strong theoretical plausibility, this statement may be extended to say that social factors such as low educational attainment may be a "fundamental cause" of tobacco use.

Diverse efforts to increase educational attainment within and across different racial/ethnic groups may contribute to reducing tobacco use. Population-wide strategies that increase educational attainment may have considerable multiplier effects for improving population health and reducing TRHD over the long term. These efforts will be especially important for individuals and population groups with lower overall levels of educational attainment. As of 2015, adults with a 4-year college degree or greater were the only educational group to have reached the Healthy People 2020 target of reducing cigarette smoking by adults to 12%. To reach the Healthy People 2020 target, smoking must decrease among all educational groups, but at a faster rate among individuals with lower educational levels. Policy interventions that broadly improve educational attainment may contribute to this effort.

Many studies also show a strong inverse association with income, regardless of the measure of income used. Income could be linked to tobacco-related outcomes through a variety of indirect pathways. For example, lower income might be associated with higher levels of psychosocial stress, leading to tobacco use as a perceived coping behavior, or with occupational exposure to SHS. Those with less income may live in communities where smoking is more normative or where tobacco advertising or the availability of tobacco products is more prevalent than in other communities.

Studies that examine tobacco use along the life course tend to find an association between parental education and occupation, and tobacco use among the offspring in adulthood. Causal pathways between life-course SES and measures along the tobacco use continuum in adulthood are inherently indirect and therefore difficult to measure. Parental education could affect many factors, such as parent–child relationships, which influence adolescent smoking and in turn adult smoking. Early-life SES could also influence trajectories for later-life education, occupation, income, wealth, and neighborhood SES and could subsequently influence tobacco outcomes through the pathways previously described for these measures.

Research Needs

The research described in this chapter, and in other studies, calls attention to the many health challenges faced by population groups with low levels of education and income, both primary social determinants of health. Identifying mechanisms through which socioeconomic status influences tobacco use may result in new potential targets for interventions that could ultimately reduce TRHD. Research to examine factors that contribute to low smoking rates among some groups despite their low educational attainment, such as Hispanics/Latinos, would also be informative. Research to identify other effective strategies targeted toward individuals with low levels of education are needed; these may include strategies to change pro-smoking social norms, promote cessation, reduce SHS exposure, and others. Interventions to improve educational attainment may also play a role in reducing disparities in relation to the tobacco use continuum, and research studies should address this possibility. As this chapter has identified, there remain gaps in the evidence base regarding socioeconomic status and TRHD, including studies to examine neighborhood SES and life-course SES and TRHD, as well as gaps focused on specific understudied population groups, such as LGBT individuals.

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Section IV
Societal Level Influences on Tobacco Use

Chapter 10
Communication, Marketing, and Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

Both pro-tobacco and anti-tobacco communication and marketing have helped shape the public's knowledge, attitudes, beliefs, and behaviors around tobacco. National Cancer Institute (NCI) Tobacco Control Monograph 19, *The Role of the Media in Promoting and Reducing Tobacco Use*, systematically documented the evidence related to the effectiveness of these efforts on the general population, and identified research questions about the mechanisms through which media operate to influence behavior.[1]

This chapter expands that body of evidence to examine the effects of tobacco-related communication initiatives on various populations, exploring how communication processes may differentially influence population groups to create, exacerbate, or reduce tobacco-related health disparities (TRHD). Communication inequalities may be defined as differences between social groups in their ability to generate, manipulate, and distribute information at the macro level and to access, process, and act on information at the individual level.[2] These communication inequalities may in turn play a role in poor health outcomes, including tobacco-related health outcomes. This chapter examines the evidence on the effects of pro- and anti-tobacco communication among disadvantaged groups, particularly racial/ethnic and low-socioeconomic-status (SES) groups.

Numerous mass media campaigns have been implemented with the goal of reducing tobacco use initiation among youth and encourage cessation among smokers. The literature provides strong evidence that anti-tobacco media campaigns can effectively reduce smoking prevalence among the general population. For example, NCI Monograph 19 concludes that "evidence from controlled field experiments and population studies shows that mass media campaigns designed to discourage tobacco use can change youth attitudes about tobacco use, curb smoking initiation and encourage adult cessation."[1,p.12] These conclusions were confirmed and extended in NCI Tobacco Control Monograph 21, *The Economics of Tobacco and Tobacco Control*,[3] the *Community Guide to Preventive Services*,[4] and the 2014 Surgeon General's report, *The Health Consequences of Smoking—50 years of progress*.[5] The CDC's *Best Practices for Comprehensive Tobacco Control* considers mass-reach health communication interventions one of the five key components of a comprehensive tobacco control program.[6]

However, less evidence is available about the effectiveness of mass media campaigns among specific population groups. Some campaign effects among whites, African Americans, Hispanics, Asians, and low-SES groups have been documented, but few studies have assessed campaign effects among lesbian, gay, bisexual, and transgender (LGBT), American Indian/Alaskan Native, or Native Hawaiian/Pacific Islander groups. Evidence on the effects of campaigns among specific population groups is somewhat mixed but overall indicates promising strategies for ensuring the effectiveness of health communication campaigns among disadvantaged groups. In addition, as discussed in NCI Monograph 19, campaigns that are complemented by additional state, community, or school-based tobacco control programming are most effective in supporting behavior change among youth and adult groups that experience disparities.[1]

An extensive literature shows that pro-tobacco marketing promotes tobacco use and related attitudes among the general population.[1] However, it remains unclear whether and to what extent these effects differ by race/ethnicity or SES. The few existing studies show that positive attitudes about tobacco advertising predict tobacco use among various subgroups. There is evidence that the tobacco industry uses event sponsorship, audience segmentation, and product development to effectively reach particular groups.[1] For example, the tobacco industry promotes tobacco products at the point of sale (POS) more

heavily in low-income and racial/ethnic minority communities, and makes pricing and placement decisions based on demographics. Studies show that African American and American Indian/ Alaska Native youths have more exposure to smoking imagery in television and movies, which may more strongly influence perceptions of smoking among these groups. On the other hand, some research suggests that racial minority youth may be more resistant to the influence of depictions of smoking in movies than white youths.[7–9] This chapter will discuss these and other examples of pro- and anti-tobacco communication, marketing, and promotion in detail.

The first section of this chapter outlines how communication-related factors fit within a broader theoretical framework of health inequalities to help explain differential health outcomes. Next, the chapter summarizes the evidence on how both anti-tobacco and pro-tobacco communication and marketing efforts could influence TRHD. Later, the chapter discusses the rise of online and digital technologies, which provide novel pathways to reach, amplify, and engage target audiences with pro- and anti-tobacco messages, and describes how TRHD may persist due to groups' varying ability to generate, manipulate, and distribute information as well as to access, process, and act on that information. Future directions for research and program implementation are identified.

Understanding Communication Inequalities

Theoretical Frameworks

The study of communication inequalities can be traced to theoretical developments in the fields of social epidemiology and media studies. This section discusses how social epidemiology, fundamental cause theory, social determinants framework, and the knowledge gaps hypothesis provide the basis for the Structural Influence Model (SIM) which helps explain communication inequalities. This section also discusses how communication inequalities operate at the individual and institutional levels, consistent with the socioecological model discussed in chapter 1. Communication inequality refers to differences in groups' ability to generate, manipulate, and distribute information as well as to access, process, and act on that information.[2] These communication inequalities might in turn play a role in poor health outcomes, including tobacco-related outcomes. What are the factors that underlie these inequalities?

Social epidemiology is the branch of epidemiology that studies the various mechanisms and pathways through which a person's social and environmental structures, such as SES, get "under the skin," leading to either health or illness.[10] The social-epidemiological approach contrasts with approaches that focus more narrowly on the biological causes of disease as well as with theories that emphasize the influence of individual lifestyles and stress profiles on health outcomes.[11,12] In recent years, researchers have recognized that peoples' ability to live healthy lives is influenced by social determinants of health, including SES, race/ethnicity, and gender; the social and physical quality of their neighborhoods, schools, transportation options, and workplaces; and their access to affordable, healthy food and appropriate medical care.[13–15]

Fundamental Cause Theory

Fundamental cause theory (FCT), which is consistent with a socio-epidemiological approach, postulates that persistent socioeconomic differences in health and mortality arise because people of higher SES possess a wide range of resources, including money, knowledge, and power, that can be used to their advantage.[16,17] This theory accounts for the observation that in social systems where diseases, risks, treatment options, and knowledge are constantly changing, people with greater access to social and

financial resources use them to avoid the risk of disease or to minimize its consequences. For example, scholars using FCT have demonstrated the emergence of a strong SES gradient in smoking behavior in the years following the release of scientific evidence on the adverse health effects of smoking. Those with greater resources were able to use this new scientific information to support their own cessation efforts, which disproportionately reduced their smoking prevalence, compared with people of lower SES.[18] FCT offers a clear advantage in explanatory power over earlier theories based on simple associations between individual risk factors and disease outcomes. Public health research has pushed this model further by reintegrating biological explanations for health outcomes within an even broader array of social and environmental influences on health.

Social Determinants Framework

Social epidemiology also encompasses the social determinants framework for understanding health inequalities. Social determinants, factors embedded in our social environments that determine the health status of individuals or populations[19] include social class, social networks, neighborhood conditions, and social cohesion. A social determinants approach can improve our understanding of the various pathways that lead to disease outcomes in certain population groups.[20,21] Although more research is needed on the causal pathways that connect social determinants with health outcomes, it is generally agreed that social determinants exert their influence through both proximal and distal factors such as access to material and intellectual resources, social support and living conditions, the unequal distribution of knowledge, and exposures to environmental stressors.[20,22]

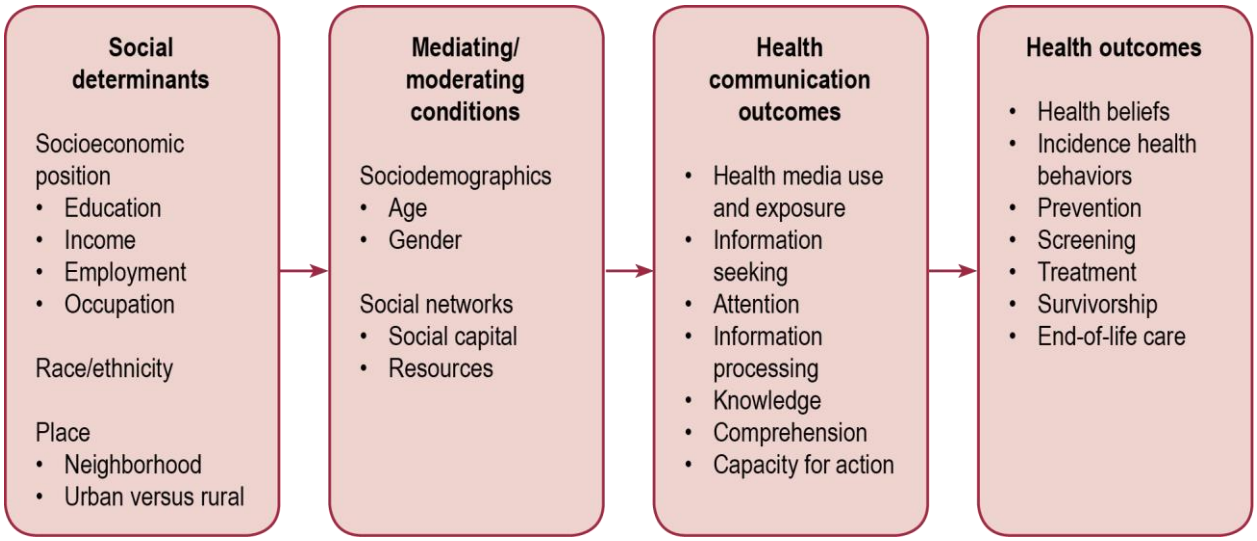
Knowledge Gap Hypothesis

Media theories also inform present-day research on communication inequalities. Central to this tradition is the knowledge gap hypothesis, which emphasizes the role of the social environment in shaping how individuals are affected by content from the news and entertainment media.[23] Proponents of this hypothesis maintain that an increasing flow of information into a social system (e.g., from a media-based anti-tobacco campaign) is more likely to benefit groups of high SES than those of low SES.[24] Specifically, the knowledge gap hypothesis reinforces the concern that social group differences in income, education, and other factors could lead to disparities in health, such as those resulting from differences in tobacco initiation, use, and cessation, as well as disparities in the morbidity and mortality associated with tobacco consumption.[23] Differential access to knowledge by high- and low-SES groups is one mechanism that could mediate the link between SES and health disparities; that is, disparities in health can occur in tandem with disparities in access to information and knowledge.[2]

Structural Influence Model

The social epidemiological and media theories that help explain health disparities in general provided the foundation for the SIM that was developed to help explain communication inequalities. The initial definition of communication inequalities[2] was further developed as the SIM, shown in Figure 10.1, which posits that health communications are a critical pathway through which the larger social environment, particularly social determinants, influence proximal predictors of health, such as knowledge, attitudes, beliefs, and behaviors.[25,26]

Figure 10.1 The Structural Influence Model



Source: Adapted from Viswanath et al. 2007.[26]

SIM proposes that social determinants (such as SES and geographic location) act through social networks and demographic characteristics (such as age and gender) to influence how individuals access and comprehend health information. Communication inequalities can arise from the ways in which members of different communities pay attention to, process, and act on health information.[27] Some groups could see certain kinds of health information as more relevant and attend to it sooner than others because of prior exposure. To the extent that such actions are influenced by the varying social capacity of the groups, health communication inequalities are more likely to emerge or be reinforced.

This model suggests that communication inequalities unfold over time, in line with the life-course perspective on health inequality. Life-course effects are the ways in which a person's health status at any given age reflects not only contemporary conditions but also prior living circumstances and the cumulative effect of biological and physical insults to the body over time.[28–30] For example, a number of studies have found that low SES during childhood can have long-term effects on smoking behavior. A prospective study of a multiethnic cohort of women found that blue-collar parental occupation at birth increases the risk of smoking, particularly for current smoking relative to former smoking.[31] Graham and colleagues[32] explain that children's socioeconomic circumstances strongly influence educational trajectories, which in turn are associated with knowledge about the harms associated with tobacco use and smoking uptake in adolescence, current smoking, heavy smoking, and quitting in adulthood. Graham and colleagues[32] also point out that education eliminates the effect of childhood circumstances on these dimensions of smoking status, which supports the idea that childhood conditions can be modified by education.

Individual- and Institutional-Level Inequalities

Communication inequalities operate at two levels that are integral to the socioecological model presented in chapter 1. At the individual level, communication inequalities refer to differences in individuals' ability to access and use information channels and services, attend to and process health information, and act on the information provided. At the systems or institutional level, communication

inequalities refer to differences among social groups in their ability to generate, disseminate, and use information.

Individual-Level Inequalities

Individuals may have differing abilities to access information insofar as they are members of groups that are characterized by different abilities to access information, use different media channels, and afford communication service subscriptions. In general, groups with more education and higher incomes are more likely to access and use the Internet, read newspapers, and actively seek information on health, all of which can increase exposure to more comprehensive and detailed information regarding health issues. Different racial/ethnic groups also use and rely on media differently. For example, data from 2014 show that, on average, African Americans watched more television and read more print magazines than the general population.[33] In 2015, 18% of Hispanic adults reported daily readership of a newspaper compared to 31% of whites and 27% of African Americans[34]; however, 97% of U.S. Hispanic adults reported listening to the radio weekly.[35] While there were no differences by race/ethnicity for Internet use in 2016, greater percentages of Hispanics (23%) and African Americans (15%) used smartphones to access the Internet compared to whites (9%). Individuals living in rural areas are less likely to use the Internet compared with those living in urban or suburban areas.[36] These media use patterns undoubtedly influence the likelihood of exposure to both pro-tobacco and anti-tobacco messages and thus influence tobacco use, initiation, and cessation.

Individual differences in attention to information and processing of information are an important dimension of individual-level communication inequality. Research reveals that in a cluttered information environment, advertisers often compete by selecting particular channels and developing messages that will "cut through the noise" and influence their intended audience.[2] Campaign planners, both pro-tobacco and anti-tobacco, focus their marketing research on the intended audience and the media outlets they use, and sometimes on the psychological characteristics of individual audience members.[37] A little more than a decade ago, scholars adopted a social–contextual view that suggested that audiences attend and react to mediated content based on the individual's location in the environment and the social roles they play.[2,38] Social characteristics, including SES, occupation, race and ethnicity, gender, and geography, may mediate or moderate the impact of messages through such factors as collective experiences, group membership, media access, and media preferences (see Figure 10.1).[2,26]

People also differ in terms of the literacy and numeracy skills they bring to the task of processing information.[39] Complicated language and the presentation of contradictory scientific findings in health communication messages can hinder information processing,[40,41] and elevated levels of chronic stress in disadvantaged population groups can amplify these difficulties.[42] A heavy burden of stress can undermine one's ability to learn new information and can influence decisions about seeking advice and support from medical professionals, family, and friends.[43] An individual's ability to process health information can also be impaired by information overload and perceived ambiguity about the information received—that is, the individual may feel uncertain or lack clarity about this information. The rise of the patient-centered "informed consumer" model of health care has been paralleled by a tremendous increase in the coverage of health-related information by the media and on the Internet.[44] For some people, the quantity of health information could overwhelm information-processing capabilities and lead to confusion.[40,45] In addition, health information in the media can be presented in a confusing or contradictory manner and could produce uncertainty and confusion about health recommendations. There is some evidence that ambiguity promotes pessimistic judgments about health

risks and risk-reducing behaviors and lowers rates of adopting healthy behaviors.[46] Research also suggests that people of older age, lower education levels, and non-white race are more likely to perceive the information they receive as ambiguous.[40,45,47] Because these same characteristics also identify population groups at risk for poor health outcomes, it is important to understand the mechanisms, direct and indirect, underlying the observed associations.[47,48]

Finally, differential ability to take appropriate action based on the successful processing of health information is a critical outcome of individual-level communication inequality (see Figure 10.1). The sheer complexity of many health communication topics poses a significant challenge for individuals not only in trying to learn and understand information, but also in acting on that information. The capacity to act on health information is also subject to an opportunity structure, including the built environment. For example, individuals may have difficulty acting on cessation information when they do not have access to cessation counseling in their community or when they are heavily exposed to tobacco advertising where they live.

Institutional-Level Inequalities

Important communication inequalities also exist at the institutional level. Although these inequalities have received less research attention than individual-level inequalities, there is evidence of system-level differences in the capacity to learn, use, and produce information.[2,49] For example, compared with academic medical centers serving higher SES populations, community-based health organizations that serve vulnerable populations might not have the cessation resources and information to support smokers interested in quitting. The effects of such system-level inequalities are compounded by the tobacco industry's targeted marketing to vulnerable populations.[1] Populations at greater risk of tobacco dependency are in greater need of community-based support.

Ultimately, both individual- and institutional-level communication inequalities need to be considered if TRHD are to be successfully addressed. The SIM provides a framework for understanding communication inequalities that may operate at the individual or institutional levels. This model presents a broad conceptual roadmap of how communication efforts related to tobacco use may differentially impact disadvantaged communities. The next sections focus on specific types of tobacco-related communication initiatives and the evidence of their efficacy among different populations.

Anti-Tobacco Communication, Marketing, and TRHD

Anti-tobacco communication and marketing campaigns are one type of public health communication campaign. Public health communication campaigns can be described as "purposive attempts to inform or influence behaviors in a large audience … using an organized set of communication activities and featuring an array of mediated messages in multiple channels generally to produce noncommercial benefits to individuals and society."[50,p.3] Anti-tobacco communication and marketing efforts are a critical component of comprehensive tobacco control programs designed to counter the marketing and promotional efforts of the tobacco industry. Although anti-tobacco campaigns primarily focus on changing individual behavior, such as motivating smokers to quit or encouraging youths to reject tobacco use, campaigns can also seek to shift attitudes and beliefs to modify social norms[51] or increase public support for tobacco control and related policies.[52] Anti-tobacco campaigns use mass media (TV, radio, print, etc.) to reach large numbers of target audience members and do not depend on person-to-person contact.[53] These campaigns are implemented at various geographic levels, from the local

neighborhood to the national level. Increasingly, campaigns employ websites, digital advertising, interactive social media, and mobile channels to disseminate messages and expand reach.[50]

As noted in NCI Tobacco Control Monograph 21, "well-designed and -implemented anti-tobacco mass media campaigns are effective in improving understanding about the health consequences of tobacco use, building support for tobacco control policies, strengthening social norms against tobacco use, and reducing tobacco consumption among youth and adults."[3,p.13] Similarly, the Community Preventive Services Task Force recommends "mass-reach health communications interventions based on strong evidence of effectiveness in: decreasing the prevalence of tobacco use; increasing cessation and use of available services such as quitlines; and decreasing initiation of tobacco use among young people."[4]

Anti-tobacco campaigns that use television can be a powerful tool to reduce TRHD, particularly since low-SES and racial/ethnic minority individuals generally have higher rates of television viewing, which increases their likelihood of exposure to anti-tobacco messaging compared with other groups.[54,55] However, the available evidence is inconsistent about the degree of effectiveness of media campaigns among socioeconomically disadvantaged populations, particularly the most highly disadvantaged.[56,57] For example, in their review of studies that analyzed the effectiveness of media campaigns by SES, Niederdeppe and colleagues found that "media campaigns to promote smoking cessation are often less effective, sometimes equally effective, and rarely more effective among socioeconomically disadvantaged populations relative to more advantage populations. Disparities in the effectiveness of media campaigns between SES groups may occur at any of three stages: differences in meaningful exposure, differences in motivational response, or differences in opportunity to sustain long-term cessation."[56,p.1343]

There is also less evidence on the efficacy of specific components of messages among those at highest risk of initiation and unsuccessful cessation. Evidence related to message effects—how the content and style of messages affect cognitive, attitudinal, and behavioral outcomes[38]—is inconclusive for the effectiveness of specific tobacco control message components among racial/ethnic minority and low-SES youth (see Box 10.1).[58–64] Studies indicate that graphic and emotionally arousing messages that evoke fear are associated with strong responses among adults in general[65] and may resonate more strongly among lower SES populations.[56,66–69] Additional research is needed on how the various elements of message construction influence the effectiveness of anti-tobacco advertising among disadvantaged groups, particularly among youth.[38]

Box 10.1: Message Effects Research

Most of the literature on message effects consists of forced exposure studies that examine self-reported perceived effectiveness of different types of advertisements or other cognitive measures of impact, such as memorability, liking, or attitude and belief changes. Other studies examine associations between different message types and behavioral outcomes such as quitline calls, quit attempts, or sustained cessation. As discussed in NCI Tobacco Control Monograph 19, studies consistently show that advertising using strong negative messages about health consequences is more effective compared with other message types, such as those using humor or emotionally-neutral content.[1] Research findings from studies that have examined message effects among low-SES adults or other diverse groups conclude that members of low-SES groups or other groups perceive advertisements portraying negative health consequences with graphic or emotional elements as equally or more effective and equally or more able to encourage quitting behavior.[66,68,69,459–461] A review of the literature on media campaigns aimed at reducing youth tobacco use

found that advertisements using personal testimonials; advertisements with a surprising narrative; intense images, sound, and editing were especially effective among youth. Evidence for the effectiveness of health consequences messages was mixed; anti-industry messages were effective when combined with health consequences messages. The evidence was insufficient to determine whether secondhand smoke or social norm messages were effective.[462] The authors conclude that youth "[anti-tobacco] media campaigns can be effective across racial/ethnic populations, although the size of the campaign effect may differ by race/ethnicity."[462,p.e71] A study examining youth smoking prevalence in the United States from 1998 to 2005 found that youth-targeted anti-smoking advertisements emphasizing health consequences to self and others and advertisements featuring deceptive tobacco industry practices were independently associated with reduced youth smoking rates.[463] Another study found that advertisements containing a personal testimonial or graphic visceral theme were more likely to be recalled, discussed with others, and thought about by 8th-, 10th-, and 12th-grade youth; these effects did not differ by race/ethnicity.[464]

Review of the Effects of Anti-Tobacco Media Campaigns on TRHD

The evidence presented in this section reflects a review of the literature on the impact of anti-tobacco campaigns among racial/ethnic minority and low-SES groups—specifically, campaigns aimed at preventing smoking among youth and promoting cessation among adult smokers. This review used two primary approaches. First, it examined major published campaign literature reviews and reviews of interventions among populations that experience TRHD. Second, additional information on published studies of specific campaigns was obtained from an online search using standard search tools and databases. Other potentially relevant articles were identified from the reference lists of review articles.

Literature searches were conducted in PubMed, Embase, Scopus, Web of Science, Communication & Mass Media Complete, Humanities and Social Science Indices, Humanities Full Text, Humanities International Index, and Academic Search Complete for the period 1990–July 2014, with a focus on studies conducted in the United States. Search terms included (adults OR youth OR "young adults") AND ("smoking cessation" OR "quit smoking" OR "prevent smoking" OR tobacco) AND ("community intervention" OR "targeted media" OR media OR "campaign" OR "self-help") AND (disadvantaged OR "socioeconomic status" OR SES OR "low education" OR "low income" OR poverty OR "blue collar" OR minority OR "racial group" OR "ethnic group" OR "African-American" OR black OR Latino OR Hispanic OR "Asian-American" OR "Native American" OR "Alaska Native" OR "foreign" OR "foreign-born" OR gay OR lesbian OR bisexual OR transgender OR homosexual).

This literature search yielded 792 articles, of which, 78 met the inclusion criteria (see Box 10.2). Published studies that examined the effects of general population campaigns on specific populations, campaigns targeted to specific populations, and campaigns that included unusually diverse samples in the evaluation were sought. All study designs, including controlled field trials and population-based studies, were included. Controlled field trials are campaigns designed as experimental or quasi-experimental research studies conducted specifically to carefully test the efficacy or effectiveness of mass media on certain outcomes, alone or with other program components (i.e., school-based programs). Population-based studies are large-scale interventions mounted on a regional or national scale, often funded by state or national government, and do not include planned experimental assessments, such as control or comparison groups.[1,70]

Box 10.2: Inclusion and Exclusion Criteria for the Review of Literature on Anti-Tobacco Communication and Marketing Campaigns

Campaigns

The review defined campaigns as "purposive attempts to inform or influence behaviors in a large audience … using an organized set of communication activities and featuring an array of mediated messages in multiple channels generally to produce noncommercial benefits to individuals and society."[50,p.3]

Channels include: television, radio, newspapers, billboards, posters, leaflets, booklets, and direct marketing intended to reach large numbers of people. Channels could not depend on person-to-person contact. They could have a digital component but could not be solely digital.

Inclusion Criteria

- Conducted in the United States
- Published between 1990 and July 2014
- Published in English
- Focused on some aspect of youth tobacco smoking prevention or adult tobacco smoking cessation. For prevention campaigns, youth must be the primary audience. For cessation campaigns, adults must be the primary audience.
- Should examine general population campaign effects among groups of interest, campaigns among unusually diverse populations, or targeted campaigns among groups of interest, which include any racial/ethnic minority groups (African American, Hispanic, Asian American, American Indian/Alaska Native, Pacific Islander, etc.), any socioeconomically disadvantaged groups (low income or SES, homeless, blue collar), foreign-born, or gay, lesbian, bisexual, or transgender identity

Exclusion Criteria

- Conducted outside the United States
- Published outside the stated time frame
- Published in languages other than English
- Focused on:
  - secondhand smoke, a policy objective, or media advocacy
  - smokeless tobacco
  - outcomes related to anti-tobacco advertising or exposure but not specific to an individual campaign
  - variation in receptivity to effects of varying messages across groups (i.e., either forced exposure or population-based message effects studies) rather than campaign impact or outcomes
  - campaigns that targeted tobacco as part of a broader anti-substance-abuse message, cardiovascular health, or cancer prevention or cancer screening campaign
  - campaigns targeted solely toward pregnant women
  - campaigns to encourage health care providers to help smokers quit
  - campaigns conducted solely on the Internet, via mobile phones, or as entertainment education

To understand the potential mechanisms through which communication disparities could arise across groups, the analysis focused on differences in (1) meaningful exposure to media messages (e.g., recall, awareness, comprehension), (2) motivational response (e.g., receptivity or perceived effectiveness,

information-seeking, treatment-seeking such as through quitline calls, impact on beliefs or attitudes, discussion about the campaign), and (3) opportunities to act (e.g., sustained abstinence, lower initiation, and other tobacco-related behavior change).[56] The following sections summarize the included studies and present data on these three potential sources of disparities for specific campaigns where data were available. This section also includes a discussion of several campaigns implemented after July 2014 (the cutoff date of the literature review).

Youth-Focused Anti-Tobacco Communication and Marketing Campaigns

Youth-focused anti-tobacco campaigns have been conducted at the national, state, and municipal levels with varying audiences, strategies, and outcomes. Common outcomes have included knowledge about the effects of smoking, attitudes toward smoking, beliefs about the tobacco industry, discussion of campaign messages with others, perceptions of peer smoking and smoking approval, intentions to smoke, and smoking behavior. Campaigns found to be effective among youth include those targeting the general population, including adults, and those that target children and youth ages 6 to 18 years. Most campaigns focus on the 12- to 18-year-old age group. Some research suggests that anti-tobacco campaigns have more reliable positive effects on youths in preadolescence and early adolescence,[61] and that different age-appropriate messages might be needed for older youths.

Although evaluations have found significant declines in smoking associated with several population-based youth campaigns, little peer-reviewed, published research has examined the impact of youth anti-tobacco campaigns among groups defined by race/ethnicity or SES. However, evidence of anti-tobacco campaigns directed toward specific population groups is available from studies of a city-based campaign in Chicago; state-based campaigns in Florida, Massachusetts, Indiana, and Minnesota; the national "truth" campaign; and several controlled field experiments that spanned multiple cities or states and included diverse populations. Table 10.1 summarizes the youth-focused anti-tobacco communication and marketing campaigns reviewed.

Table 10.1 Summary of Youth-Focused Anti-Tobacco Communication and Marketing Campaigns Reviewed

Group	Study Type and Number of Studies for Each Type	Effects on Targeted Smoking Behaviors*	Conclusions
Low SES	General population campaigns: 0 studies	N/A	No evidence
Low SES	Targeted campaigns in low-SES communities: 4 studies from one campaign	▪ Positive effect overall (media+school vs. school only)[86,88,89,90]	Evidence of benefit for the media+school arm
Racial/ethnic groups	General population campaigns: 5 studies	▪ Mixed effects for a media-only condition[102,103] and for a media+state tobacco control program[73,80] ▪ Positive effect overall and no differences for media+state tobacco control program[79]	Some evidence that large population-based media campaigns combined with comprehensive state tobacco control programs may benefit racial/ethnic minority groups

Table 10.1 continued

Group	Study Type and Number of Studies for Each Type	Effects on Targeted Smoking Behaviors*	Conclusions
Demographically diverse communities	Campaigns in racially/ethnically diverse communities: 3 studies	▪ No effect overall and no difference by racial/ethnic groups (media only vs. no media)[94] ▪ No effect overall and no analyses by specific group (media vs. no media)[92] ▪ Positive effect overall (media+community and school interventions)[93]	No evidence of benefit for media campaigns only. Limited evidence for media+community and school interventions
African Americans	Campaign targeted to a specific population: 2 studies	▪ Media-only vs. media+school arms cannot distinguish media effect[71] ▪ Media in one community vs. no media in another community showed reduction in both communities[72]	Inconclusive evidence of benefit

Note: Some studies' campaigns are listed in several categories because they focused on several groups (e.g., racial/ethnic group, low-SES groups). n = 14. SES = socioeconomic status.
*Smoking behaviors considered among youth: amount or frequency of recent smoking and initiation.

Chicago Youth Campaign

A campaign to prevent smoking initiation among African American youth was aired in Chicago in 1989.[71] A study to evaluate its impact established two conditions: a media+school-based condition and media message–only condition. The media component for both conditions included a smoking prevention curriculum printed in the children's weekly section of a newspaper with a predominantly African American readership, eight public service announcements on a local radio station with a largely African American audience, billboards, and a community Smoking Prevention Rap and poster contest. Messages focused on raising awareness of the health risks of smoking and environmental influences that encourage youth to smoke.[71]

The intervention was carried out in all 6th- and 7th-grade classrooms from three public elementary schools located in largely African American neighborhoods. Schools were randomly assigned to conditions: two schools were assigned to the media+school arm (n = 175) and one school to the media-only arm (n = 101). Pre- and post-test surveys were conducted 1 week before and 1 week after the intervention, as well as an additional 6-month post-test, with follow-up rates of 94% for the 1-week post-test survey and 83% for the 6-month follow-up.

Ninety-nine percent of student respondents were African American. A relatively high proportion of youth in both arms were aware of some part of the media campaign, although awareness was higher among the media+school group, which prompted youth to engage with the media component as part of the school curriculum. Cigarette smoking knowledge was significantly higher from pre- to post-test in the media+school group compared with the media-only group. The two arms did not differ in smoking behavior at 1-week or 6-month follow-up, but results demonstrated that both arms significantly decreased smoking from pre-test to 6-month follow-up. The effects of the media intervention could not be determined due to the study design, and decreases over time in both arms could not be distinguished from broader secular time trends because the study did not include a no-media control arm for comparison purposes.[71]

Baltimore Youth Campaign

A media campaign with the goal of reducing cigarillo use among African American youth was implemented in Baltimore, Maryland.[72] The 18-month campaign was modeled after the national "truth" campaign described later in this section and was based on the theory of reasoned action and social inoculation theory. Messages were designed to increase awareness of both tobacco industry targeting and the health risks of cigarillos to promote negative attitudes toward the tobacco industry and toward cigarillos. Advertisements were run on radio, television, billboards, the Internet, on the sides of buses, at subways stops or in subway cars, and on abandoned buildings. Most of the advertising featured an 18-year-old African American teen, with language reflecting the vernacular of urban areas as determined by focus groups conducted with the target audience.

For the evaluation, a quasi-experimental design was used with an exposed city (Baltimore) and a comparison city with similar demographics (Philadelphia). The comparison city received one campaign advertisement randomly per day, and the exposed city received over 10 advertisements per day during after-school hours. The researchers selected a random sample of schools and public places frequented by the target audience and administered in-person pre- and post-surveys to youth ages 10–19. A significant decrease in cigarillo use (from 3.8 to 1.1 cigarillos per day) was found among respondents in the exposed city; cigarillo use also decreased significantly in the comparison city, although by a lesser amount (from 2.3 to 1.5 cigarillos per day). Given the design, effects could not be distinguished from broader secular time trends.[72]

Florida "truth" Youth Campaign

The Florida "truth" campaign, first implemented in 1998 to help teens reject smoking, included a comprehensive statewide anti-tobacco effort with a primary message highlighting the deceptive behavior of the tobacco industry (Figure 10.2).[73–78] (The national "truth campaign" is discussed later in this section.)

Figure 10.2 Advertising Image, Florida "truth" Campaign, 2001

Source: Centers for Disease Control and Prevention 2013.[465]

Cross-sectional analyses from the Florida Youth Tobacco Surveys from 1998 to 2000 demonstrated that non-Hispanic white and African American middle school students who previously experimented with tobacco products reported stronger intentions not to smoke, compared to Hispanic middle school students.[73] Overall rates of current smoking (having smoked within the past 30 days) and of frequent smoking (having smoked on 20 of the past 30 days) declined significantly among middle school and high school students during the campaign. However, this analysis found no significant declines in current smoking among African American high school students, and no significant declines in frequent smoking among Hispanic high school students or African American middle school or high school students. All population groups showed significant increases in never smoking as well as decreases in experimenting with tobacco products.[73] Whether these effects on smoking-related behaviors resulted from the campaign or from other components of the Florida Tobacco Control Program could not be determined.[73,75] Other longitudinal studies have demonstrated that the campaign lowered the risk of smoking initiation and progression to established smoking among students overall, but these results were not presented by race/ethnicity or SES.[77]

Massachusetts Campaign

Initiated in 1993 as a component of the Massachusetts Tobacco Control Program,[79] this media campaign was conducted primarily through advertisements on television, radio, newspapers, and billboards.[80] Rather than targeting youth specifically, this campaign was designed to expose a broad cross-section of the population to anti-smoking messaging, which highlighted tobacco industry practices and the health effects of tobacco use. Cross-sectional analyses indicated that 733 of the youth participants (96%) self-reported that they had been exposed to a Massachusetts anti-smoking advertisement in the past month, but exposure levels were not provided for specific groups. Results indicate that the advertisements were perceived as highly effective by the overall youth population.[58]

A subsequent longitudinal study of the Massachusetts campaign (592 youths, 4-year follow-up) found that exposure to the campaign was high, and was associated with less likelihood of progressing to established smoking. However, this effect was found only among youths who were ages 12–13 years old at baseline and not among those who were 14–15 at baseline. Differences in advertising awareness or in the likelihood of progression to established smoking were not found between non-Hispanic whites and other racial/ethnic groups.[79]

Another study of youth smoking prevalence in the state from 1996 to 1999 found significant decreases in lifetime and current use of cigarettes, cigars, and smokeless tobacco.[81] This study showed that cigarette use declined at a greater rate in Massachusetts than in the Northeast or in the nation as a whole, but declines in use differed across racial/ethnic groups. Lifetime and current use declined significantly among whites and African American middle and high school students. Declines in usage rates among Hispanic middle or high school students were not significant, possibly due to small sample sizes. This study could not distinguish the effects of the media campaign from other components of the Massachusetts Tobacco Control Program.[81]

Indiana Youth Campaign

In 2001, Indiana implemented an anti-tobacco campaign targeted toward youth that was designed to prevent smoking, encourage cessation, and change social norms. Campaign messages varied over time; early messages used anti-industry themes or emphasized negative health consequences of tobacco use. For example, the “Rick Stoddard” advertisement, originally created for the Massachusetts Department of

Public Health, told the story of a man who lost his wife to cancer.[82,83] After an early campaign run, a follow-up survey of 3,858 middle school students was conducted in four rural, three suburban, and three urban schools. Residents of the rural counties in the study had lower income and education levels than residents of the suburban and urban counties. Eighty-nine percent of youth reported awareness of the campaign; suburban and urban youths were more than three times as likely to report awareness compared with rural youths. The study did not control for the overall level of media exposure, so it was not possible to determine whether the differences were due to variation in actual exposure levels, given fewer information sources in rural areas, or to variation in the recall and processing of campaign messages across groups. Among youths who were aware of the campaign, there were no significant differences in receptivity; rural, suburban, and urban youths were equally likely to say the advertising made them think about not using tobacco in the future.[84]

A later study of 391 rural Indiana youth, including 58 American Indian/Alaska Native youth, found high levels of self-reported recall of and receptivity to graphic campaign messages, with no differences between white and American Indian/Alaska Native youth.[63]

Minnesota Youth Campaign

In 2000, the state of Minnesota implemented a multi-faceted anti-tobacco campaign ("Target Market") directed at youth. The campaign included three main components: paid advertising, a youth organization, and a website targeted to youth. Messages were based on an anti-industry, youth empowerment theme that encouraged youth to learn about and fight against tobacco industry marketing to teens. The campaign ended in July 2003 when funding for Minnesota's Tobacco Control Program was reduced from $23.7 million to $4.6 million.[85] Four cross-sectional surveys conducted from July 2002 to December 2003 found that awareness increased to 84.5% from July 2002 to July 2003, with a plateau during the summer of 2003 and a significant decline to 56.5% from July 2003, when funding was reduced, to December 2003. These changes in awareness were similar across urban and rural areas of the state. Additionally, after funding was reduced (July 2003–December 2003), youth susceptibility to smoking increased overall and by geographic area.[85]

Youth Campaigns in Vermont, New York, and Montana

In addition to the population-based studies described above, youth campaigns have included field-based experimental trials of mass media campaigns, some of which were paired with school-based interventions. From 1985 through 1989, researchers conducted a controlled non-randomized trial in which media messages were aired in matched pairs of lower income communities in the northeastern United States (Vermont and New York) and in Montana. The media campaign focused on reducing youth tobacco use by changing youth attitudes toward the advantages and disadvantages of smoking, teaching cigarette refusal skills, and altering perceived norms of peer smoking based on theories of health behavior change, including the theory of reasoned action and social learning theory and information processing models.[86,87] By using a variety of message styles, advertisements were customized for specific gender and age groups, and the diverse formats included comedies, cartoons, rock videos, and testimonials. There was also a strong focus on messages and media targeting high-risk adolescent girls.[88] The campaign did not use a specific logo or sponsoring agency name, on the hypothesis that young people at higher risk for smoking tend to shun authority. This unbranded campaign presented 12–18 different advertisements during each airing and ran for 4 years, with the goal of promoting widespread perceptions of a positive lifestyle.[86]

Two matched communities received media messages coupled with a school-based intervention, which was compared with a school-only intervention in the other two matched communities. The media and school interventions were not linked programmatically except in terms of general campaign objectives. Two pairs of standard metropolitan statistical areas were selected from a regional sample, with community samples matched through the selection of specific school districts and related demographic characteristics. "High-risk" communities were chosen based on census data indicating lower adult educational attainment and household income at the tract level for the catchment areas serving the schools.[86]

To evaluate the campaign's impact, a combined cohort of 5,458 students was surveyed at baseline in grades 4–6 and followed annually for 4 years. Individual- and community-level analyses at the end of the 4-year campaign found significant reductions in smoking and consistent effects on targeted mediating variables, including smoking attitudes and social norms, for the combination media+school intervention group compared with the school-only intervention group.[86] An additional follow-up survey, conducted 2 years after the campaign ended, found that the media+school intervention group had a 38% lower risk of smoking than the school-based-only intervention group, and the difference was significant.[89] The campaign had a stronger impact on attitudes, beliefs, and reductions in smoking among adolescent girls at both the end of the campaign and at the 2-year follow-up compared with boys.[88] Further analyses indicated that smoking prevalence was significantly lower among high-risk students in the media+school communities than for high-risk students in the school-only communities, with high risk defined as ever smoking prior to baseline or having two or more family members who smoked. The effects on low-risk students were similar but were only marginally significant.[90] The generalizability of the campaign results was limited primarily to lower income white populations, as the communities in which the interventions occurred were 96.5% white.[90]

California Youth Campaign

In 1986, researchers implemented the Television, School and Family Project (TVSFP), a large-scale school- and media-based tobacco use prevention and cessation project in Southern California, which was grounded in social psychological theories such as social influence theory.[91] The study included five conditions: a social resistance classroom curriculum, a media (TV) intervention, a social resistance classroom curriculum plus mass media intervention, and two control groups consisting of a health-information attention-control curriculum and a no-control condition. Forty-seven schools in Los Angeles and San Diego were randomly assigned to conditions within six school districts; the television conditions existed only in Los Angeles. The intervention conditions were designed to raise youths' awareness of social influences to smoke and social consequences of smoking. The TV conditions included broadcast of educational segments of *Feeling Fine*, a health issues component of the local evening news in Los Angeles. The segments focused on resistance skill modeling for students and cessation strategies for adults. All conditions included a family involvement component using homework assignments that required parent participation.

The evaluation included a pre-test of 7,351 7th-grade students in 340 classrooms from the 47 schools, an immediate post-intervention follow-up, and 1- and 2-year follow-ups. Respondents were 35.5% Hispanic, 33.3% white, 13.9% African American, and 17.3% other. A priori comparisons included a television versus no-television condition, as well as other condition comparisons. Findings at post-test follow-up demonstrated that the television campaign had a significant main effect compared with no campaign on targeted knowledge, attitudes, and beliefs, including awareness of peer and media

influences to smoke, strategies for resisting social influences, and perceived prevalence of adult and youth smoking. These effects did not persist at 1- and 2-year follow-ups, however. Neither the TV condition nor any other study condition had an effect on intentions to smoke or on current smoking behavior. The authors suggested that poor execution of the television programming component may have contributed to the limited effects.[92]

Texas Youth Campaign

The evaluation of a Texas youth prevention campaign based on cognitive social influence theory was designed to examine how the varying intensity of anti-smoking media campaigns and differing types of community- and school-based anti-smoking programs influenced tobacco use and attitudes among young adolescents (ages 11–12).[93] The quasi-experimental study was conducted in the year 2000 across 14 sites (each with a population of approximately 100,000 people), which were chosen based on having ethnically diverse populations and high rates of tobacco-related disease. Messages were delivered via an animated duck character and were focused on the addictiveness and unattractiveness of smoking. Approximately one-third of the cross-sectional pre-evaluation and post evaluation sample was white, one-third was Hispanic, 20% was African American, and 5% was Asian American. Results were not reported for specific racial/ethnic groups, but overall findings indicated that the most consistent decreases in tobacco use, susceptibility to smoking, and pro-smoking attitudes were achieved by combining an intensive media campaign with comprehensive community programs, including school-centered and community-oriented activities.[93]

The Program to Reduce Youth Smoking Through Media

The Program to Reduce Youth Smoking Through Media was an experimental trial designed to examine the influence of a mass media campaign on youth smoking prevalence across multiple states.[94] The audience was segmented into three age groups, grades 4–6, 7–8, and 9–12, with age-specific messaging and media. A prevention campaign was targeted toward each age group from 2002 to 2005, and a cessation campaign was targeted toward students in grades 9–12 from 2002 to 2004. The campaign included TV and radio messages that featured Hispanic, African American, and white youths, and it aired on targeted media channels popular among these racial/ethnic groups. Campaign objectives and advertising were based on behavior change theories such as social cognitive theory. The campaign objectives were to: decrease perceptions of smoking prevalence among young people; increase perceptions of smoking disapproval; increase confidence in the ability to refuse cigarettes; decrease positive outcome expectations for smoking; and increase negative outcome expectations for smoking. The campaign was not linked to interventions in schools or communities. One-third of messages reflected Hispanic casting and lifestyle, one-third African American, and one-third white, and media programming was targeted by age, gender, and race/ethnicity. Advertising formats included dramas, comedies, testimonials, and cartoons.[94]

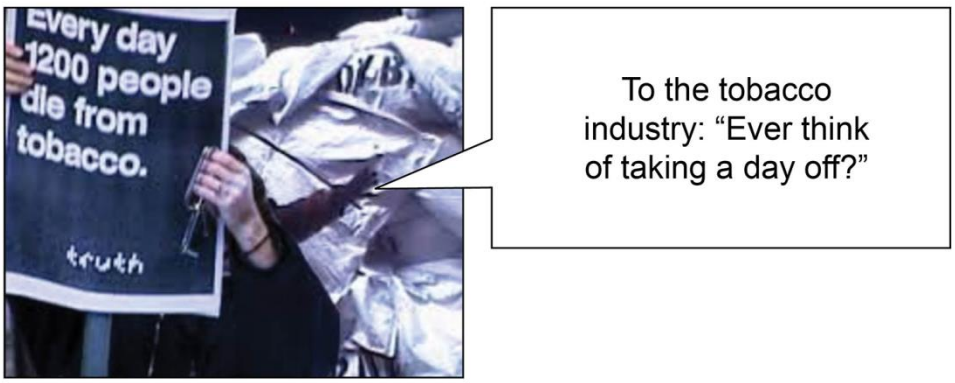
Using an experimental community trial design, the campaign was conducted in four matched pairs of communities across four states (Florida, South Carolina, Texas, and Wisconsin) from 2001 to 2005, with communities and study samples selected for their racial/ethnic and socioeconomic diversity. Communities were matched, and one community of each pair was randomized to receive the intervention. School districts within the Designated Media Areas were recruited based on low-income and education populations, and surveys for grades 7–12 were conducted at baseline and 4 years later. The study samples ranged from 53%–58% non-Hispanic white, 23%–27% African American, 13%–15% Hispanic, and 5%–10% “Other” across conditions.[94]

Evaluation studies demonstrated favorable and significant changes in tobacco-related beliefs among Hispanic and African American youths but not among white youths. A significant favorable effect for some campaign-related beliefs was found for students in grades 7 and 8, and a trend was found for students in grades 9–12, but these effects did not translate into behavioral changes in most cases. No differences were found in intentions to smoke, in past-30-day and 7-day smoking, and in overall smoking prevalence between intervention and comparison communities at the 4-year follow-up. The findings suggested a trend in reduction of past-30-day smoking among Hispanics only. Researchers speculated that the effects among Hispanic students could have been due to the novelty and relevance of campaign messages specific to a traditionally underserved population. The overall lack of campaign effects on behavior might have been due to a ceiling effect of mass-media-based interventions, given that this trial was conducted during the same time as the period of highest exposure for the national "truth" campaign.[94] Further, the authors suggest that the absence of a school or community component may have undermined the campaign's impact.[93,94] Such additional program components may be needed, especially when targeting disadvantaged or diverse youth populations.

The National "truth" Youth Campaign

The national "truth" campaign of the Truth Initiative (formerly the American Legacy Foundation), created in 1998, was a branded campaign aimed at preventing youth smoking by influencing youth "sensation-seekers," ages 12 to 17 years, at greatest risk of smoking (Figures 10.3 and 10.4). Early campaign messages were delivered primarily via television, with supplemental advertisements on radio, on the Internet, and at other locations (e.g., on outdoor billboards, street furniture, transit), and were characterized by an edgy and rebellious message strategy with an anti-tobacco-industry theme.[95,96] The campaign was successful in reaching young people: 75% of all U.S. youths reported awareness of at least one "truth" message 10 months after the campaign first launched,[95] and awareness averaged approximately 70% over the first 3 years of the campaign.[97]

Figure 10.3 A "truth" Body Bags Campaign Message, 2000



Source: Truth Initiative 2000.[466]

Figure 10.4 A "truth" Singing Cowboy Campaign Message, 2006

Source: Truth Initiative 2006.[467]

Evaluation studies of the "truth" campaign examined differences by SES and geographical location. One study pooled seven waves of repeated cross-sectional data from the 2002–2004 Legacy Media Tracking Survey (LMTS) to assess the impact of "truth" by SES. The study found that youths from low-income ZIP Codes had lower awareness of the campaign but similar levels of receptivity compared with youths from higher income communities.[98] A small cross-sectional survey of youth in five rural high schools in western Pennsylvania found relatively high awareness of the campaign (56%) several years after the campaign ended. Of those who were aware, 88% perceived the campaign to be effective.[99]

Duke and colleagues[100] examined the impact of an enhanced media delivery initiative of the "truth" campaign in a quasi-experimental study in rural and low-population-density communities. Eight communities were assigned to receive supplemental "truth" advertising, and another eight comparison communities received less than the national average of "truth" messages. A longitudinal analysis of 2,618 youths over more than 5 months found that rural youths in the supplemental media markets had significantly higher confirmed awareness of "truth" than youths in comparison rural markets receiving lower doses of "truth." Rural youths were also found to be highly receptive to the advertisements. These results suggest that targeted supplemental media efforts in rural communities could increase the awareness of anti-smoking advertising and could overcome potential limitations in media delivery sources for rural youths. Enhanced media targeting of youths in low-income areas could have a similar effect. These data also suggest that exposure to campaign messages was one of the key elements influencing the success of the campaigns.

Evaluation studies of "truth" have also analyzed campaign outcomes with respect to race/ethnicity. Advertisements featured youths from all racial/ethnic groups and targeted a portion of advertising toward media channels popular among racial/ethnic minorities.[96] Analyses from 2002 based on a pre-campaign wave of the LMTS and a 9-month cross-sectional follow-up found high levels of confirmed awareness and receptivity to the campaign among youths and young adults but did not analyze by race/ethnicity.[95] A subsequent analysis using LMTS data pooled across seven waves from 1999 to 2003 revealed some differences in the influence of the campaign on targeted attitudes, beliefs, and intentions to smoke by race/ethnicity. Specifically, associations between campaign exposure and changes in industry-related beliefs and attitudes were significant among whites and African American youths, but not among Hispanic or Asian American youths.[101] Further analyses found variations in the impact of specific messages[101] and the processes through which campaign messages influenced attitudes and

smoking behavior.[102] Further, the impact of the campaign on intention to smoke among never-smokers was strongest for African American youth.[101]

A study by Hersey and colleagues[102] found that exposure to the "truth" campaign negatively affected progression to established smoking by strengthening counter-industry attitudes and beliefs, and this relationship was significantly stronger among African Americans than among all other racial/ethnic groups evaluated. Evans and colleagues[103] also looked at mediators of "truth" messages. Controlling for peer influence, cigarette price, and personal independence, these researchers found that having a positive attitude toward being tobacco-free and toward nonsmoking social imagery made progression to smoking less likely. This pathway differed by race/ethnicity: The role of peer influence on the formation of nonsmoking social imagery was significantly stronger among whites and African Americans than Hispanics.[103] Together, these studies suggested that although exposure had a favorable impact on tobacco use across demographic groups, the salience of messages and the pathways of influence can vary across racial/ethnic groups.

In 2014, the Truth Initiative launched *truth FinishIt*, which targets youth and young adults ages 15–21 via social media. The campaign was designed to develop a relationship with the 92% of nonsmoking teens to reduce their intentions to smoke and affect longer term smoking behavior.[104]

Food and Drug Administration Youth and Young Adult Education Campaigns

The Real Cost

The Food and Drug Administration (FDA) launched its first federally funded U.S. anti-tobacco public education campaign in February 2014, targeting youth ages 12–17 who are open to trying smoking or already experimenting with cigarettes. Advertisements appeared on television, radio, print, digital, and out-of-home displays and focused on the cosmetic health effects of smoking, loss of control caused by addiction, and the toxic mix of chemicals found in cigarette smoke.[105] An evaluation of the campaign conducted between 2014 and 2016 found that high levels of campaign exposure during this time were associated with a 30% decrease in the risk of smoking initiation, and prevented nearly 350,000 youth ages 11–18 from initiating smoking.[105] In April 2016, The Real Cost brand expanded to include new advertising targeting rural male youth ages 12–17 at risk of smokeless tobacco use in 35 targeted U.S. markets.[106] At the time of this writing (2017), the campaign was ongoing and an outcome evaluation measuring the impact of campaign exposure on tobacco-related knowledge, attitudes, and beliefs was under way.[107]

Fresh Empire

The Fresh Empire campaign was launched in May 2015 in select southeast U.S. markets and expanded to additional markets in October 2015. The goal of the campaign was to prevent and reduce tobacco use among at-risk youth ages 12–17 who identify with hip-hop culture, specifically targeting African Americans, Hispanics, and Asian Americans/Pacific Islanders.[108] The campaign messaging highlighted the disconnect between the ideal image hip-hop culture promotes (e.g., fashionable, authentic) and the consequences of tobacco use. Traditional media advertisements aired during programs most popular among the hip-hop peer crowd. The campaign also engaged with the target audience through multiple digital platforms and outreach at the local level. Brand ambassadors attended local hip-hop events that linked back to social media promotions to increase campaign reach.[109] At the time of this writing (2017),

the campaign was ongoing and an outcome evaluation measuring the impact of campaign exposure on tobacco-related knowledge, attitudes, and beliefs was under way.[108]

This Free Life

FDA launched its This Free Life campaign in May 2016 to prevent and reduce tobacco use among LGBT young adults ages 18–24 who use tobacco occasionally. The campaign connected LGBT young adult shared values and the desire to be "free" as it related to their lives and experiences to the importance of being tobacco free. Print, digital, and out-of-home advertisements and local outreach events were the primary campaign dissemination vehicles in 12 U.S. markets. At the time of this writing (2017), the campaign was ongoing and an outcome evaluation measuring the impact of campaign exposure on tobacco-related knowledge, attitudes, and beliefs was under way.[110]

Adult-Focused Anti-Tobacco Communication and Marketing Campaigns

As with youth-focused campaigns, adult-focused campaigns have directed campaign strategies toward diverse segments of the smoking population in varying geographic areas. Adult cessation campaigns commonly address outcomes such as effects on knowledge, campaign-related beliefs, quit intentions, information-seeking or treatment-seeking (e.g., calling a quitline), reductions in cigarettes smoked per day, quit attempts, sustained abstinence, and reductions in smoking prevalence. The adult campaigns reviewed in this section include large national or state-led general population campaigns with paid mass media and extensive reach as well as smaller, targeted, community-based campaigns with earned media (unpaid coverage) or direct-marketing efforts combined with local campaign activities (Table 10.2).

Table 10.2 Summary of Adult-Focused Anti-Tobacco Communication and Marketing Campaigns Reviewed

Group	Study Type and Number of Studies for Each Type	Effects on Targeted Smoking Behaviors*	Conclusions
Low SES	General population campaigns: 15 studies	▪ No difference in effects between low and high SES[143,178,179,192] ▪ Positive effect for low SES[68,118,153,160,174,190,191] ▪ Mixed effects[171,468] ▪ Negative effect for low SES[138,154]	Evidence of benefit for campaigns with graphic themes or campaign components targeted to low-SES groups, but studies vary widely No evidence of benefit for campaigns with primarily printed self-help-focused components
Diverse populations	Targeted campaigns: 14 studies	▪ No effect[111,128,129,133,134] ▪ Positive effect[114,115,120,121,122,126,132,139,140]	Some evidence of benefit for intensive, multicomponent, culturally- and language-appropriate community-based campaigns targeting specific populations Relevant social support (e.g., counseling, peer support) may also be beneficial as part of these campaigns

Table 10.2 continued

Group	Study Type and Number of Studies for Each Type	Effects on Targeted Smoking Behaviors*	Conclusions
Racial/ethnic groups	General population campaigns: 11 studies	▪ No difference between racial/ethnic minority groups and other groups[192] ▪ Positive effect for racial/ethnic minority groups[174,175,178,190] ▪ Mixed findings[171,172,179,191] ▪ Negative effect for racial/ethnic minority groups[127,138]	Some evidence of positive effects for large mass media campaigns combined with additional tobacco control program or policy components. Effectiveness varied by campaign and by racial/ethnic group.
Racial/ethnic groups	Targeted campaigns: 18 studies	African American ▪ No effect[111,133,134] ▪ Positive effect[114,115,130] Hispanic ▪ No effect[129] ▪ Positive effect[113,126,132,139] ▪ Mixed effects[125,131] Asian American ▪ No effect[128] ▪ Positive effect[120,121,122,140]	Some evidence of benefit for intensive, multicomponent, culturally- and language-appropriate community-based campaigns targeting specific populations, including immigrant populations Relevant social support (e.g., counseling, peer support) may also be beneficial No evidence of benefit for campaigns with primarily self-help focused components Some evidence of benefit for Spanish-language media campaigns promoting quitlines combined with enhanced phone counseling and/or nicotine replacement therapy

Notes: Some studies' campaigns are listed in several categories because they focused on several groups (e.g., racial/ethnic minority group, low-SES group). n = 36. SES = socioeconomic status.
*Smoking behaviors considered among adults: quit attempts, abstinence, and smoking prevalence.

Large-Scale Anti-Tobacco Campaigns and Racial/Ethnic Minority Adults

A number of large-scale anti-tobacco campaigns provide insights into campaign effects among racial/ethnic minority adults.[111–140] Most of these campaigns were conducted between 1990 and 2000, with some exceptions,[113,122,127,139,140] and many were community-based. They included mass media combined with a variety of local intervention activities.[111,112,114–117,119–138,140] Many also focused on lower socioeconomic groups. Most of these campaigns were targeted to or included large racial/ethnic minority populations, including African Americans,[111,112,114–117,119,130,133–138] Latinos,[113,123–127,129,131,132,135,139] and Asian American/Pacific Islanders.[120–122,128,135,140] The campaigns employed broadcast TV, radio, and out-of-home advertising; self-help materials, including audiotapes and videotapes; print materials for telephone and group cessation counseling; peer support networks; and community advocacy efforts.

Although evaluation studies varied in terms of study design, intervention components assessed, and outcomes measured, some common themes emerged. Findings from several studies suggested sufficient exposure to campaign messages[117,119,120,123–125,132,134,135,137,139] as well as strong indicators of motivational response among low-SES and racial/ethnic minority target audiences, as evidenced by increased calls to informational and smoking cessation counseling quitlines.[112,113,121,139] Studies also found receptivity to

counseling calls[111] or intervention materials,[131] intervention engagement,[115,118] increased quit attempts,[116] quit knowledge,[123,125,139] and movement along the stages of change for quitting—for example, with more intensive interventions moving smokers from pre-contemplation to contemplation and from preparation to action.[134,136] In terms of opportunities to act or actual behavior change, several studies found modest increases in cessation rates and declines in prevalence among the target audience.[113–115,120–122,126,127,131,138] One study found a reduction in disparities,[140] and two provided evidence that tailored smoking cessation counseling via telephone quitlines increased quit rates compared with standard counseling for smokers recruited through media campaigns.[130,139]

Campaigns to Promote Cessation Among Low-SES Adults

A 2008 review of media campaigns by Niederdeppe and colleagues[56] documented the evidence related to the effectiveness of campaigns to promote cessation among socioeconomically disadvantaged adults age 18 years and older. The review primarily covered studies conducted in the United States, as well as a few studies from Australia, Great Britain, and Canada. In a sample of 50 published studies that used different study designs to evaluate 31 separate mass media campaigns, the analysis examined several intermediate- and long-term campaign outcomes and identified three potential sources of disparities in response to smoking cessation media campaigns: message exposure or recall, motivated response (i.e., quitline calls, quit attempts), and opportunities to act (i.e., abstinence, quit success). The authors used these three stages of campaign response to assess whether a study was more, less, or equally effective in reducing disparities across SES groups.

Specifically, studies that showed lower levels of exposure/recall, motivated response, or quit success among low-versus high-SES populations were considered to increase disparities and thus be less effective. Studies that showed equivalent levels of response at all three stages among low- and high-SES populations were considered to maintain disparities and thus be equally effective. Studies that showed higher levels of response in at least one of the three stages without showing lower levels in another stage were considered likely to reduce disparities and thus be more effective.[56] Of the 18 campaigns designed for a general audience,[138,141–157] Niederdeppe and colleagues concluded that 9 were less effective, 6 were equally effective, and 5 were more effective among a lower SES audience. Of the 13 campaigns that specifically targeted a low-SES audience,[111,115,125,126,129,133,134,139,158–162] 8 generated mixed or inconclusive results in effectiveness for reducing disparities, and 5 were less effective among a low-SES audience.[56]

Niederdeppe and colleagues[56] emphasized the importance of sufficient exposure to the campaign to ensure awareness and enhance effectiveness among low-SES populations, including utilizing multiple strategies such as paid, earned, and donated media as well as direct marketing to reach smokers. Simple self-help or quit-to-win contests, in isolation, were not found to benefit low-SES populations or attain sufficient reach. To increase awareness and improve low-SES smokers' motivational response to campaigns, the authors emphasized the need for formative research to understand the preferences of low-SES smokers, including literacy needs, language preferences, and cultural values of targeted groups. The authors also state that "media campaigns appear most effective among low SES smokers when they are implemented alongside larger tobacco control programs that include community mobilization, free NRT [Nicotine Replacement Therapy], telephone counseling, social support, or policy changes to change the social and structural context of cigarette use."[56,p.1352] In addition, Garrett and colleagues[161] note that the literature suggests that mass media campaign advertisements "featuring emotional/personal testimonies and graphic images of the health effects of tobacco that evoke strong negative emotions are

more likely to be effective in promoting smoking cessation among low-SES populations in comparison to ads that solely provide information on how to quit without the use of testimonials."[163,p.895]

Cessation Campaigns Analyzed by Race/Ethnicity or SES: Massachusetts, California, and New York

Reviews by Bala and colleagues[53,164] on the effectiveness of media cessation campaigns identified only two general campaigns with relevant, if minimal, analyses by race/ethnicity or SES among adult smokers: the Massachusetts and California campaigns. Additionally, relevant findings from campaigns implemented in New York State and New York City are described below.

Massachusetts. The Massachusetts campaign, implemented in 1993, was focused primarily on television media and utilized graphic and emotional advertising to relay information on the health consequences of tobacco use. Data indicated that awareness of the campaign was high among the overall population, but awareness levels were not examined for specific population groups.[165] A longitudinal population-based study found that smokers with lower educational attainment were somewhat more receptive to the campaign than more highly educated smokers.[165] However, a cross-sectional study of recent quitters found that smokers with a high school education or less and Hispanic smokers were not more likely than more highly educated and non-Hispanic smokers to report an anti-smoking TV ad as helpful in quitting.[142] In terms of actual behavior change, time-series analyses comparing smoking prevalence in Massachusetts with 41 other states that had limited tobacco control programming found that declines in prevalence among Massachusetts smokers were more pronounced among those who had graduated from high school but not college, and among non-Hispanic whites compared with smokers of other racial/ethnic groups.[53,164] These analyses could not separate the impact of the media campaign from the other components of Massachusetts's tobacco control program.

California. The California media campaign, launched in early 1990, was designed to promote a social norm of "not accepting tobacco" and included messages on the role of the tobacco industry in promoting tobacco use, the hazards of secondhand smoke exposure, addiction, and other topics.[166] Over time, the campaign also directed advertising and other efforts to specific ethnic populations, incorporating culturally relevant messages in a number of languages.[167] Early analyses indicated higher campaign awareness among Hispanics than other racial/ethnic groups analyzed.[168] Analysis of data (1992–2009) showed higher use of the California Smokers' Helpline (state quitline) by African Americans compared with other racial/ethnic groups.[169] Quitline use was also higher among ethnic minority or low-income young adults.[170] When the campaign targeted Hispanic and Asian American/Pacific Islander populations with language-specific media, quitline call rates among these groups increased.[157] This strategy also resulted in more calls from proxies—individuals who called on behalf of family and friends. In general, non-English-speaking populations were referred to the quitline at much higher rates than English-speaking populations, but low-education populations were less likely to call the quitline than more highly educated groups.[157]

Increased quitline call volume is an important indicator of interest in cessation, but data are not available to determine how quitline calls from smokers or proxies translate into successful quit attempts for specific population groups. Men's smoking prevalence in California between 1989 and 2000 declined equally across racial/ethnic groups, but smoking prevalence declined at a greater rate among Hispanic and white women than among African American women during the same period.[53] In contrast, a 1996–2002 study estimating changes in cigarette consumption using cigarette sales and self-reported survey data found greater increases in quitting among non-Hispanic whites and African Americans, and these

quits were more likely to be among women in these groups.[171] Another study, a cross-sectional analysis, compared declines in prevalence in 1992-1993 and 2001-2002 among non-Hispanic whites and African Americans in California with declines in prevalence in states that did not have comprehensive tobacco control programs. This study found significant declines among non-Hispanic whites in California only, but African American prevalence declined similarly across all states.[172] Differences in cessation by education varied by sex as well; the greatest declines in prevalence were among college-educated men and among women who did not graduate from high school.[53,164] In California, as in Massachusetts, these analyses could not separate the effect of the media campaign from other tobacco programming efforts delivered by the state health department.

New York. From 2003 to 2009 the New York State Tobacco Control Program invested $75 million in paid advertising on television and radio, in print, on the Internet, and in other venues, with messages designed to encourage smokers to quit by increasing their awareness of the health effects of smoking and the dangers of secondhand smoke. The campaign primarily used advertisements with strong emotional and graphic elements, such as those from the Massachusetts Department of Public Health "Pam Laffin" series (which shows the family of a young mother who died from emphysema due to smoking) and advertisements from Australia's "Every Cigarette Is Doing You Damage" campaign (which features stark, graphic images of the health effects of smoking). These graphic and emotional advertisements were supplemented by advertisements intended to enhance self-efficacy for quitting by providing resources and information on how to quit.[173]

Analyses comparing 6 years of cross-sectional data from New York State found that smokers' exposure to the state's anti-tobacco advertising increased from 6% to 45% over time, and quit attempts increased from 46% to 62%.[173] During that same period (2003–2009), smoking prevalence declined at a higher rate in New York (18%) than in the United States as a whole (5%). These data were not analyzed by specific group and, as with campaigns in states such as Massachusetts and California, the analyses could not distinguish the effects of the media campaign from effects of other components of the state's tobacco control program.[173] However, later analyses of cross-sectional data from the 2003–2010 New York Adult Tobacco Surveys demonstrated that exposure, as measured by confirmed awareness and gross rating points (GRPs) in separate models, was positively associated with increased odds of making a quit attempt. GRPs are a measure of the percentage of the population potentially exposed to advertisements (reach) and the average number of times they may have seen the advertisements (frequency) over a time period. The positive association between awareness and GRPs with quit attempts held true for all smokers, smokers who wanted to quit, smokers in low-income (<$30,000 per year) and high-income brackets (≥$30,000 per year), and smokers at lower education levels (high school degree or less and at least some college). Exposure to advertisements without graphic images or strong emotions had no effect among adult smokers.[68]

Additional analyses based on cross-sectional data from 2003 through 2011 among 9,408 smokers found that anti-smoking advertising, as measured by GRPs and confirmed awareness, was associated with increased quit attempts among non-Hispanic black and Hispanic smokers and those with lower levels of income and education. Nonnemaker and colleagues[174] noted that this was partially attributable to emotionally arousing and graphic advertisements. Findings also indicated that anti-smoking advertising, including graphic advertising, did not promote quit attempts among individuals with poor mental health.

The Roswell Park Cancer Institute, in Buffalo, New York, collaborated with local organizations in Erie and Niagara counties in upstate New York to run a Quit & Win contest and a NRT voucher giveaway

program to encourage cessation. This program was extensively promoted through the media and tailored to racial/ethnic minority group smokers. Studies conducted during the 2002 campaign found that the local minority population was receptive. A higher percentage of racial/ethnic minority group individuals chose to participate in the three components of the program (i.e., the Quit & Win contest, the NRT voucher giveaway, and a combination of the two) relative to their proportion in the local population. While behavioral outcomes were not examined by specific group, quit rates across the three intervention groups were high, ranging from 26% to 29% at 4- to 7-month follow-up.[175]

New York City also implemented public education campaigns as part of the city's comprehensive tobacco control program. In addition to intensive media promotions and anti-tobacco advertising, free NRT products were widely distributed to smokers. Studies show these efforts had significant success in increasing awareness and prompting responses from disadvantaged smokers. In 2003, a 6-week NRT giveaway program via the New York state quitline gave free NRT to an estimated 5% of all eligible smokers in New York City; 64% of these recipients were non-white, foreign-born, or resided in low-income neighborhoods. Foreign-born smokers in this program had higher quit rates than any other group.[148] During a 2006 NRT giveaway campaign, approximately 60% of the city's smokers reported program awareness, with awareness above 50% in each racial/ethnic, education, income, and nativity-based group and fairly evenly distributed across all groups.[176] A cross-sectional survey of 1,000 randomly selected city residents was conducted after the campaign and assessed whether those who were not aware of the campaign would have been receptive if they had been aware. The researchers found that receptivity among those who were not aware was highest for Spanish-speakers and the foreign-born.[148,176] A later 2008 NRT distribution campaign reached an estimated 3% of the city's adult smoking population; in areas with high smoking prevalence, uptake was higher in low-income neighborhoods, compared to higher income neighborhoods.[177]

Studies also examined smoking prevalence during the period when the New York City graphic anti-tobacco advertising campaign was conducted in conjunction with the city's multicomponent tobacco control program. Analyses from 2002 to 2003 found that smoking prevalence declined significantly overall and among all age, race/ethnicity, and educational groups, including U.S.-born and foreign-born individuals. Most of this decline was attributed to tobacco tax increases and smoke-free policies implemented as part of the broader city program.[178] Data collected after expansion of the campaign in 2006 demonstrated a significant decline in smoking among men and Hispanics in that year but not among other groups or overall.[179]

National Anti-Smoking Campaigns

Following the Fairness Doctrine period (1967–1971), which ended with passage of the Public Health Cigarette Smoking Act banning cigarette advertising on television and radio, there was little or no national anti-smoking advertising in the United States.[151,180] The following sections describe several recent large-scale efforts to use mass media to highlight the hazards of tobacco use, with encouraging results for groups that experience TRHD.

The "BecomeAnEX" Campaign

In 2008 the Truth Initiative, along with the National Alliance for Tobacco Cessation, launched the "BecomeAnEX" (EX) campaign, the first national branded adult cessation mass media campaign. This campaign targeted the general population but included an explicit focus on promoting cessation among

lower income and blue-collar smokers of diverse race/ethnicity who were thinking about quitting (Figure 10.5).[181]

Figure 10.5 Print Advertisement, EX Campaign, 2007

Source: Truth Initiative 2007.[469]

The campaign's message strategy was to empathically encourage smokers to "relearn" life without cigarettes by disassociating certain daily activities, such as driving or drinking coffee, with smoking. This strategy was based on recommendations from the literature regarding effective mass media campaigns and behavior change theory.[181–189]

After extensive formative research and a pilot study, the profile of the target audience was refined to smokers ages 25–49 of low-to-moderate income who were thinking about quitting. Subtle visual and behavioral cues were used to realistically portray the challenges of a lower income smoker's daily routine while attempting to quit (Figure 10.6). Media plans for the campaign focused on airing messages on networks and during programming popular among the target audience.[181,190]

Figure 10.6 EX Advertisement: Image of a Blue-Collar Worker Trying To "Relearn" Drinking Coffee Without Cigarettes, 2007

Source: Truth Initiative 2007.[470]

An evaluation of the EX campaign's effect by race/ethnicity and level of education found evidence that the campaign was effective at increasing smokers' favorable cognitions about quitting and quit attempts.[190] This evaluation was based on a national cohort of 4,067 smokers, of which 74% were non-Hispanic white, 11.5% were non-Hispanic African Americans, 7.4% Hispanic, and 7.0% were classified as "Other." Participants were interviewed at baseline and 6-month follow-up. African Americans reported the highest levels of campaign awareness. Over the study period, campaign exposure markedly increased favorable cessation-related cognitions among Hispanics and quit attempts among African Americans; campaign exposure also increased cognitions and quit attempts among respondents with lower educational attainment.[190]

In a subsequent path analysis of EX campaign effects based on the same data, the results for the sample overall indicated that campaign awareness had a direct effect on quit attempts and that campaign awareness also indirectly affected quit attempts by creating positive changes in how participants thought about cessation.[191] The effects differed, however, when examined by race/ethnicity and education. Only among African Americans did awareness of the EX campaign have positive, significant effects, both direct and indirect, on quit attempts. Within educational strata, positive and significant direct and indirect effects were found only among individuals with less than a high school education.[191] Later analyses that attempted to control for differences in awareness of the campaign via propensity score matching found that campaign awareness was not significantly associated with cessation-related cognitions or quit attempts at 6-month follow-up among the sample overall. Excluding the 217 smokers who had quit at follow-up, analyses indicated a positive and significant effect on both outcomes. No differential effects were found after examining the data by race/ethnicity and education, contradicting results from the earlier studies.[192]

Tips From Former Smokers™

In 2012, the U.S. Centers for Disease Control and Prevention (CDC) launched "Tips From Former Smokers" (*Tips*™), the first-ever paid national tobacco education campaign in the United States. This multi-year campaign was developed to increase public awareness of the health consequences of smoking

and exposure to SHS, encourage smokers to quit, and make free help available; encourage smokers not to smoke around others; and encourage nonsmokers to protect themselves and their families from SHS exposure[193] through powerful emotional messaging, a national quitline portal, and a smoking cessation website. The campaign featured testimonials, or stories told by real people, from former smokers who described real-life experiences in graphic and realistic terms, including the consequences of living with diseases and disabilities caused by smoking. The advertisement development process and media purchasing strategy were designed to address TRHD and reach at-risk populations of smokers, including American Indian/Alaskan natives, members of the military, people with mental health conditions, people from LGBT communities, and others. The initial *Tips* campaign television advertisements ran for three months (March to June 2012) complemented by print, radio, billboard, digital, and website advertisements in English and Spanish (see Figure 10.7).[194]

Figure 10.7 Advertising Image, CDC's *Tips From Former Smokers*™

Source: Centers for Disease Control and Prevention 2017.[193]

An evaluation of the 2012 campaign included baseline assessment and 3-month follow-up among a longitudinal cohort of smokers (n = 3,051) and nonsmokers (n = 2,220) from a probability-based nationally representative online sample. Seventy-eight percent of smokers and 74% of nonsmokers recalled seeing at least one *Tips* advertisement on television, and quit attempts among smokers increased by about 12%, from 31.1% to 34.8%, during the broadcast period. An estimated 1.64 million additional smokers made a quit attempt as a result of the campaign, with an estimated 220,000 remaining abstinent at follow-up, and approximately 100,000 were estimated to stay quit for at least 6 months. There were no interaction effects between pre–post changes in quit attempts and smokers' characteristics before and after the 2012 *Tips* campaign, but stratified models indicated significantly more quit attempts among African American smokers than white smokers, and among those with less education compared to those with at least some college education.[194] Additionally, an analysis of the 2012 *Tips* campaign found that it succeeded in reducing smoking-attributable morbidity and mortality, and was a highly cost-effective mass media intervention.[195]

The 2013 *Tips* campaign aired for 16 weeks with similar creative content to the 2012 campaign but also included supplemental media buys in randomly selected local markets to increase exposure to campaign advertising. These higher dose markets were exposed to three times the advertising of standard-dose markets. Overall, the incidence of quit attempts was greater in higher dose markets relative to standard-dose markets. Researchers found that the relative increase in quit attempts associated with the additional dose was markedly higher among African American smokers, with those in higher dose markets reporting a significantly higher rate of quit attempts than those living in standard exposure markets (50.9% vs. 31.8%).[196]

After the launch of the 9-week-long 2014 *Tips* campaign, 1.83 million smokers attempted to quit smoking and an estimated 104,000 Americans quit smoking for good. The quit attempt rate among smokers increased by 17%, and an additional 1.73 million intended to quit within 6 months.[197] The authors concluded that "these data provide further justification for the continued use of tobacco education campaigns by federal and state health agencies to accelerate progress toward the goal of reducing adult smoking in the United States."[197,p.5]

In addition, an evaluation of 2014 *Tips* campaign advertisements found that the advertisements' perceived effectiveness, a measure of audience receptivity calculated by taking the mean of respondents' advertisement ratings on 6 items (memorable, attention-grabbing, informative, powerful, meaningful, and convincing) varied by race/ethnicity. Non-Hispanic black and Hispanic smokers responded significantly more favorably to the advertisements than white smokers, irrespective of the race/ethnicity of the person in the advertisement. As the authors note, the study "provides further support for previous research showing that hard-hitting, general population anti-smoking media campaigns can be used across a variety of demographic subpopulations," and that "in developing antismoking ads, a greater focus on compelling message content irrespective of the race/ethnicity of ad participants is prudent."[198,p.6–7]

Evaluating the Effectiveness and Methodology of Cessation Campaigns

A review by Guillaumier and colleagues[57] examined the effectiveness and methodological quality of adult cessation mass media campaigns among socially disadvantaged groups —including racial/ethnic minorities and people who were mentally ill, homeless, low income, and less educated—and by occupation. The authors reviewed 17 relevant studies (including many reviewed in this chapter) from the United States, Australia, and New Zealand. Eleven of these studies used specific group analyses in their evaluations of general anti-tobacco campaigns, and six studies focused on campaigns that targeted disadvantaged groups. The authors concluded that, "while socially disadvantaged smokers may be less likely to recall general population campaigns compared with more advantaged groups, they may be equally likely to perceive these campaigns as effective and to quit in response."[57,p.705] The researchers also noted that when general-population and targeted campaigns were aired nationally, disadvantaged smokers were more likely to recall and respond favorably to them, suggesting that these campaigns have the potential to be effective with disadvantaged groups. Another finding was that most studies examined campaign effects among low-to-moderate SES groups, rather than highly disadvantaged groups or those who experience multiple forms of disadvantage (i.e., indigenous populations, people who are homeless, and people with substance abuse disorders). Guillaumier and colleagues[57] also examined the methodological strength of the evidence for these campaigns. According to the criteria of the Effective Public Health Practice Project Quality Assessment Tool,[199] only 4 of the 17 studies were rated as "strong" or "moderate" for all applicable assessment items. Guillaumier and colleagues[57] determined

that weak study designs and selection bias limited strong conclusions regarding campaign effectiveness. While acknowledging the practical limitations of implementing more rigorous designs, the authors emphasized the need to raise the minimal level of evaluative evidence required to assess the effectiveness of cessation campaigns among disadvantaged populations. Specifically, the authors recommended the use of controlled time-series, sequential randomized trials, and pilot randomized controlled trials, where feasible, before widespread dissemination.

Evidence Review: Anti-Tobacco Communication and Marketing Campaigns

Overall, studies find that media campaigns aimed at the general population and those targeted toward racial/ethnic groups and socioeconomically disadvantaged populations are effective, especially when combined with state, community, and/or school-based programs that complement campaign efforts.

Youth Campaigns

Among youth, population-based and controlled field-based studies undertaken with specific group analyses or targeted toward specific groups provide limited evidence about the effectiveness of campaigns on reducing smoking behavior across racial/ethnic groups. The national "truth" campaign evaluations suggested that anti-tobacco campaigns can be effective in reaching and engaging various groups of youths, and in influencing knowledge, attitudes, beliefs, and behaviors; however, the types of campaign messages and pathways that would effectively influence attitudes and behaviors may differ by race/ethnicity. The strongest effects on attitudes and intentions were found among African American youth. The findings on awareness of the "truth" campaign and receptivity to its messages among low-SES and rural youths are promising, but given the limited work in this area, further research and analysis on behavioral outcomes among SES groups are needed.

Field-based controlled trials with sufficient duration and intensity conducted in low-income white or racially/ethnically diverse communities have shown mixed results. The literature search of studies published between 1990 and July 2014 did not identify studies on youth prevention mass media campaigns with analyses among LGBT youth or among foreign-born youth. However, for a number of national campaigns launched between 2014 and 2016 targeting LGBT youth and youth of specific racial/ethnic groups, outcome evaluations are planned or under way.

Studies also find that young people living in rural areas are receptive to anti-tobacco youth prevention campaigns, but supplemental media efforts may be required to overcome media delivery challenges in these areas. Studies evaluating the influence of youths' exposure to any anti-smoking advertising provide evidence that anti-smoking advertising can be effective among racial/ethnic groups; in some cases, anti-smoking advertising may be more effective among racial/ethnic youth than among white youth. However, these studies are unable to determine the effects of specific campaigns or types of advertising on smoking behavior among youth.

Adult Campaigns

The numerous studies examining the influence of adult cessation campaigns on diverse populations provide a nuanced and multifaceted view of the effects of anti-tobacco mass media campaigns. Data suggest that targeting specific populations with linguistically appropriate media can enhance receptivity to campaigns and stimulate treatment seeking, although the extent to which this activity translates into behavior change is not well established. Campaigns targeting specific low-SES and racial/ethnic groups

with materials in their own language and tailored to their culture have shown some positive effects, particularly among African American, Hispanic, and some Asian ethnic groups. Evaluation of the CDC's *Tips From Former Smokers* campaign has shown that emotional and graphic testimonials about living with the health consequences of smoking are broadly effective and may have a greater impact on quit attempts by African Americans and people with less education. In contrast to these campaigns, studies of the national EX campaign provided promising but somewhat mixed findings regarding the role of a supportive how-to-quit message in generating awareness, receptivity, attitude changes, and behavioral outcomes among racial/ethnic minority and low-SES populations.

In addition,

- Among adults, some evidence indicates that cessation campaigns with graphic themes or those targeted toward low-SES populations are effective for these groups. Mass media campaigns combined with state or community-based programs that complement campaign efforts may be most effective in increasing cessation among low-SES adult populations.
- There is little to no evidence of benefit for cessation campaigns consisting primarily of printed self-help materials for low-SES or racial/ethnic minority populations.
- There is some evidence of effectiveness for intensive, multicomponent, culturally and linguistically appropriate community-based campaigns that target specific populations, such as low-SES and/or specific racial/ethnic minority groups (African American, Hispanic, Asian American), including immigrant populations. Relevant social support via telephone counseling or peer support may also be needed to ensure campaign effectiveness.
- There is some evidence of benefit from large media campaigns combined with additional tobacco control program or policy components for racial/ethnic minorities, but effectiveness may vary by campaign and by racial/ethnic group.
- Some evidence supports the use of Spanish-language media campaigns promoting quitlines, combined with enhanced phone counseling and/or NRT.
- Strongly promoted NRT giveaway campaigns may be effective in reaching low-SES and racial/ethnic minority populations, including immigrant groups, but studies are needed to examine quit outcomes resulting from these campaigns.
- Differences in public awareness of campaigns, such as among non-English-speaking immigrant groups, may reflect structural differences in access to media and health-related information due to geographic, language, education, or income-related factors. Such disparities align with the knowledge gap hypothesis, which holds that social environment and social group differences in income, education, and other factors can lead to disparities in information that can influence longer term health behavior and health.
- Variation in campaign effects across racial/ethnic groups may reflect differing pathways through which campaigns influence behavioral outcomes among minority groups. These differences may be influenced by social or cultural factors and may result in communication inequalities if campaign messages do not resonate with specific groups. Formative research to examine factors that influence racial/ethnic minority groups' receptivity to media campaigns is critical to ensuring campaigns have the intended effect.
- As suggested by fundamental cause theory, low-SES and racial/ethnic minority populations may be least likely to benefit from health education campaigns, not only because of differential access to knowledge but also because of limitations in material and social resources that can support

behavior change. Media campaigns combined with multicomponent state, community, or school-based activities, including relevant social support components, show some benefit among low-SES and racial/ethnic minority populations, suggesting that additional social and structural support can help address the fundamental causes that limit health behavior change efforts among disadvantaged groups.

Pro-Tobacco Communication, Marketing, and TRHD

Advertising and promotion of tobacco products, brands, and corporate identities are intended to increase sales, influence social norms about tobacco use, and foster positive attitudes about tobacco companies.[1,200,201] An extensive body of research demonstrates that the industry's use of advertising and marketing practices does indeed have an effect. For example, NCI Tobacco Control Monograph 21 concludes that "the weight of the evidence from multiple types of studies done by researchers from a variety of disciplines and using data from many countries indicates that a causal relationship exists between tobacco company marketing activities and tobacco use including the uptake and continuation of tobacco use among young people"[3,p.258] and NCI Tobacco Control Monograph 19 concludes that "targeting various population groups…has been strategically important to the tobacco industry."[1,p.170]

This review of the literature on the effects of pro-tobacco communication and marketing effects on specific groups was carried out in two phases: an examination of major published literature reviews in this area, and a search using standard search tools and databases to identify any other relevant publications. In the first phase, reference lists of the review articles were searched and potentially relevant articles were examined. The second phase consisted of a search of MEDLINE, Embase, PsycINFO, Web of Science, Academic Search Complete, and the Cochrane Library for articles published between January 1, 2000, and July 1, 2014. The search was limited to publications in English, with a focus on studies conducted in the United States. Search terms included: (tobacco OR cigarette OR smoke OR smoking) AND (marketing OR media OR advertising OR channel OR newspaper OR magazine OR movies OR television OR industry OR company OR targeting OR promotion OR regulation OR control OR discount OR coupon OR purchase OR sponsor OR sport OR concert OR event OR "point of sale" OR pack OR packaging OR "warning label" OR labeling) AND (youth OR adolescent OR ethnic OR disparity OR disparities OR inequality OR disadvantage OR race OR racial OR minority OR "African American" OR black OR Latino OR Hispanic OR Asian OR "Native American" OR "Alaska Native" OR economic OR socioeconomic OR SES OR low-income OR poverty OR gay OR lesbian OR bisexual OR transgender OR homosexual). This literature search yielded 946 articles, 45 of which met the inclusion criteria (Box 10.3).

Box 10.3: Inclusion and Exclusion Criteria for the Review of Literature on Pro-Tobacco Communication

Inclusion Criteria

- U.S. studies only
- Published between January 1, 2000, and July 1, 2014
- Published in English
- Focused on the effects of pro-tobacco communication and marketing among groups of interest, which include any racial/ethnic minority groups (African American, Hispanic, Asian American, American Indian/Alaska Native, Pacific Islander, etc.), any socioeconomically disadvantaged groups (low income or SES), or foreign-born, or gay, lesbian, bisexual, or transgender identity

Exclusion Criteria

- Conducted outside the United States
- Published outside the stated time frame
- Published in languages other than English

A 2011 Cochrane Collaboration review identified 19 longitudinal studies that followed more than 29,000 youths (age 18 or younger) who were not regular smokers at baseline. In 18 of the 19 studies, the nonsmoking youths who were more aware of tobacco advertising, or were receptive to it, were more likely to experiment with cigarettes or become smokers by the 30-month follow-up.[202] Only one study, however, examined effects by race or ethnicity; it concluded that exposure to tobacco advertising was associated with susceptibility to smoking among white and African American youths but not among Hispanic youths.[203] Similarly, research demonstrates statistically significant relationships between adults' exposure to advertising and cigarette cravings (including urges to start smoking among recent ex-smokers), impulse purchasing,[204] cigarette consumption by adults,[205–207] increased market share,[208] and intentions to quit.[209] However, little existing literature specifically and rigorously examines these effects by race/ethnicity or SES. One of the few studies with an adequate sample size to report results by racial/ethnic groups found that media exposure (i.e., exposure to commercials or Internet advertisements) was an important factor influencing smoking initiation among all racial/ethnic groups.[210]

Greater exposure to advertising has been associated with higher perceived prevalence and positive attitudes about tobacco use among adolescent populations overall, but the evidence is limited on racial/ethnic minority youths and adults.[211] Research has also demonstrated links between advertising exposure and youth susceptibility,[207,212,213] experimentation,[202] initiation,[214,207] and smoking status.[202] Exposure to tobacco advertising has a well-established association with smoking attitudes and behavior among both youth and adults.[1,202,207,209,215–220] The few studies among targeted populations (i.e., African American, American Indian/Alaska Native, youth of Mexican origin, younger audiences) have found that positive attitudes about tobacco advertising,[221] exposure to pro-tobacco messages,[222] and increased perceptions of smoking prevalence among others[223] are predictors of tobacco use. It is important to better understand how exposure to industry advertising and promotion practices influences tobacco use behavior, particularly among vulnerable groups.

Both the Surgeon General's report *Tobacco Use Among U.S. Racial/Ethnic Minority Groups*[200] and NCI Tobacco Control Monograph 19, *The Role of the Media in Promoting and Reducing Tobacco Use*,[1] describe tobacco industry advertising and promotional practices that target or disproportionately expose low-income individuals, racial/ethnic minorities, and other minority populations. This section draws from these important sources and summarizes more recent evidence on how pro-tobacco communication and marketing efforts could influence TRHD among these groups.

The following sections highlight a process of advertising and promotion that is extremely responsive to the changing economic, policy, and social environment as well as to the changing tobacco consumer. A key component of the industry's strategy is its use of audience segmentation to effectively reach particular groups, such as youths, African Americans, Hispanics, and women, as well as the development of tobacco products with appeal to particular market segments.[200,224–226] As NCI Tobacco Control Monograph 19 notes, "targeting various population groups—including men, women, youth and young adults, specific racial and ethnic populations, religious groups, the working class, and gay and lesbian populations—has been strategically important to the tobacco industry."[1,p.11] Internal tobacco industry documents describe a sophisticated, data-driven process through which manufacturers identify a target audience, come to intimately understand the audience's experiences and needs, and use that information to develop and target products, brands, advertising, and promotions toward that audience.[1]

Pro-Tobacco Advertising and Promotional Channels

The tobacco industry's process of advertising and promotion makes effective use of a variety of communication channels,[227,228] each selected based on its ability to reach an identified audience.[1,229] Because race/ethnicity, SES, and geography influence exposure to tobacco marketing,[1,19,230,231] these factors are specifically considered within an overall media plan. Variations in tobacco prices, products, placement, and promotional strategies are employed based on detailed information related to the targeted demographic groups.[1] Evidence indicates systematic differences in the strategies used, particularly in the marketing of menthol products for urban, low-income, and often predominantly African American communities.[232] Industry terms such as "focus communities" have sometimes been used in internal tobacco industry documents to refer to these communities.[233,234]

Over time, restrictions have been placed on tobacco advertising and promotion (see Box 10.4). With each limitation on its ability to reach consumers, the tobacco industry has placed greater emphasis on other forms of product and brand promotion. The following sections address how the landscape of industry advertising and promotions has changed and what is known about how these practices affect different racial/ethnic and low-SES communities and populations.

Box 10.4: Regulation of Tobacco Industry Advertising and Promotion in the United States

As described below, restrictions on the tobacco industry's advertising and promotion practices have been implemented over time. Restrictions on industry practices often result in the transfer of resources from regulated to unregulated venues. The tobacco industry's advertising and promotion practices have also changed in response to the evolving consumer marketplace.

1965: Congress passes the Federal Cigarette Labeling and Advertising Act, requiring a health warning on all cigarette packages.[180,p.671]

1970: Congress enacts the Public Health Cigarette Smoking Act of 1969 (passed in 1970), banning cigarette advertising on television and radio and requiring a stronger health warning on cigarette packages.[180,p.672]

1973: Congress enacts the Little Cigar Act of 1973, banning little cigar advertisements from television and radio.[180,p.674]

1984: Congress enacts the Comprehensive Smoking Education Act, requiring rotational health warnings on cigarette packages and advertisements.[180,p.677]

1986: Congress enacts the Comprehensive Smokeless Tobacco Health Education Act of 1986. Requires rotation of three health warnings on smokeless tobacco packages and advertisements and bans smokeless tobacco advertising on broadcast media.[180,p.678]

1998: The Master Settlement Agreement (MSA) between 46 states, 5 U.S. Territories, and the District of Columbia imposes restrictions on participating manufacturers' marketing practices, including: (1) forbidding direct or indirect tobacco marketing to youth; (2) prohibiting tobacco advertisements on public transit and on billboards; (3) prohibiting the use of cartoon characters in cigarette advertising, promotion, and packaging; (4) eliminating paid tobacco product placement in media outlets; (5) restricting tobacco company sponsorship of sports, arts, and cultural events; and (6) restricting free samples to adult-only facilities.[266]

2009: The 2009 Family Smoking Prevention and Tobacco Control Act gives the Food and Drug Administration broad authority to regulate the manufacture, marketing, and distribution of tobacco products. The legislation required FDA to reissue a regulation it had issued in 1996 which, among other things, prohibits manufacturers, distributors, and retailers from sponsoring sporting and other cultural events with the brand name or other indicia of product identification similar to, or identifiable with, that used for any brand of cigarette or smokeless tobacco, while permitting sponsorship of these events in the name of the corporation.[471]

Television, Movies, and Tobacco Imagery

Studies indicate that many young people in the United States are exposed to tobacco imagery on television in the context of programming and movie trailers.[235–238] One longitudinal study found that youths' recall of people smoking in television programs was associated with increased odds of ever smoking at baseline.[239] Other research demonstrates that exposure to images of tobacco use in movies or by celebrities has a clear causal link with youth smoking.[1,217,240,241] Specifically, studies have shown that exposure to smoking in movies is associated with trying smoking but not with smoking intensity or faster progression to established smoking following initiation.[242–245] Studies have also indicated that exposure to smoking in movies has increased cravings and smoking behavior in adults.[242,246–249] Finally, the 2012 Surgeon General's report *Preventing Tobacco Use Among Youth and Young Adults* concluded "that there is a causal relationship between depictions of smoking in the movies and the initiation of smoking among young people."[19,p.10]

Television and movie consumption differs among adults and youths by race/ethnicity and income, which can influence exposure to certain types of tobacco products and imagery. For example, a 2015 national study of media use among youths ages 8 to 18 illustrated the potential for televised tobacco images to affect low-SES and racial/ethnic minority youths more than white youths. On average, among 8- to 12-year-olds, African Americans and Hispanics spent significantly more time watching television or videos (3 hours and 40 minutes and 3 hours and 14 minutes, respectively) each day, compared with

white youth (2 hours and 29 minutes).[250] Among 13- to 18-year-olds, African Americans reported 4 hours and 33 minutes of television watching compared to whites (2 hours and 56 minutes). Significant differences were seen in the average time per day watching television between youth with family incomes of less than $35,000 (3 hours and 40 minutes among 8- to 12-year-olds and 4 hours and 14 minutes among 13- to 18-year-olds) and those with incomes of $100,000 or more (2 hours and 9 minutes and 2 hours and 41 minutes, respectively). Significant differences were also observed between youth whose parents had a high school education (3 hours and 20 minutes among 8- to 12-year-olds and 4 hours and 4 minutes among 13- to 18-year-olds) and those whose parents had a college degree (2 hours and 22 minutes and 2 hours and 42 minutes, respectively).[250]

There is some evidence that the effects of depictions of tobacco use in television and movies on adolescent smoking behavior can vary by race/ethnicity and can be moderated by environmental variables. Some research has suggested that African American youths, and to a lesser degree Hispanic youths, are more resistant to the influence of smoking imagery overall than white youths.[7–9] The results of one study indicated that although the smoking behavior of white youths was affected by seeing white and African American actors smoking, African American youth smoking behavior was affected only by seeing African American actors smoking (and showed reduced media effects overall).[8] A national study found that African American and American Indian/Alaska Native youths reported significantly more exposure to smoking imagery on television and in movies and reported watching more hours of television per day compared with white youths.[223] These factors contributed to perceptions among African American and American Indian/Alaska Native youths of higher smoking prevalence,[221] and cross-sectional and longitudinal studies provide evidence that perceived smoking prevalence is highly predictive of smoking initiation among youths.[223,251–253]

Tobacco Advertising in Magazines and Newspapers

Tobacco advertising in magazines and newspapers decreased after the Master Settlement Agreement (MSA) was reached in 1998, with magazine advertising expenditures in particular declining steadily between 1999 ($377.4 million) and 2011 ($23.3 million).[227] However, recent years have seen an increase in magazine advertising ($50.0 million in 2014).[225] By 2005 the only major cigarette brands advertised in magazines were menthols (e.g., Newport, Salem, and Kool) or products with a prominent menthol brand presence (e.g., Camel).[254] Between June 2012 and January 2013, Newport and American Spirit spent an estimated $9.4 million on print advertising for menthol cigarettes.[255] Magazine advertising for smokeless products increased from to $7.9 million in 2009 to $11.1 million in 2010, then fell to $4.9 million in 2011, before increasing to $18.9 million in 2014.[228]

Youth

Healthy People 2020, which delineates 10-year national objectives for improving the health of the U.S. population, set a goal of decreasing the proportion of youth in grades 6 through 12 who are exposed to tobacco marketing in magazines and newspapers from 48.6% to 19.3%.[256] National data from the 2011 National Youth Tobacco Survey showed that 48.2% of middle school students and 54.0% of high school students reported being exposed to pro-tobacco advertising in magazines. The rate of exposure to magazine advertising among middle school students who were categorized as susceptible to smoking (22.5% of all middle school students) declined from 71.8% in 2000 to 46.1% in 2009, then increased to 55.4% in 2011.[257] Overall prevalence of exposure to pro-tobacco advertisements in newspapers and magazines among middle and high school students decreased from 65% in 2000 to 36.9% in 2012.[212,257]

Available data on magazine readership suggest that young people in racial/ethnic minority groups are more likely to be exposed to tobacco advertising in magazines than whites. Kaiser Family Foundation data from a 2008-2009 nationally representative survey of students ages 8–18 show that African American youths spend, on average, 11 minutes per day reading print (rather than online) magazines, whereas Hispanic youths spend 10 minutes, and white youths spend 8 minutes.[55] As of 2013, 46.2% of non-Hispanic whites, 46.8% of Hispanics, and 48.3% of African American youth reported exposure to pro-tobacco advertisements through these channels.[256]

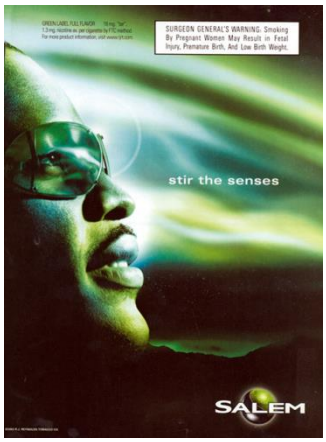
Morrison and colleagues[258] used national magazine advertising and readership data from 1992 to 2002 to assess the level of smokeless tobacco advertising in popular magazines with a large youth-based audience. Despite the reduction in industry magazine advertisement spending, they concluded that youths' exposure to smokeless tobacco advertisements remained high and might even have increased post-MSA partly due to advertising in adult magazines. A study of national magazine advertising and readership data from 1998 to 2006 supported the conclusion that youths continued to be exposed to smokeless tobacco advertisements through adult and men's magazines but suggested that youths' exposure had declined since the MSA.[259] A 2013 study on smokeless tobacco products found that discount snuff advertising tended to be published in magazines with a high youth readership and roughly corresponded to the increased popularity of this product type among male adolescents.[260]

Adults

As of 2014, 52 percent of African Americans read magazines, a figure that is far higher than the general population (22%).[33] GfK Mediamark data from 2010 also indicate differences in magazine reading by race/ethnicity; African American adults read, on average, 3.9 magazines per week, Hispanics read 3.1, and whites read 2.6.[54] Respondents in each education group read, on average, three magazines per week.

Researchers found that magazines with high African American readership had more cigarette advertisements overall (Figure 10.8) and more advertisements for menthol cigarettes than magazines with larger white readership.[261,262] Magazines tailored to Hispanics were also found to have more menthol advertising than those tailored to whites.[262] In the late 1990s, Philip Morris and R.J. Reynolds (RJR), launched their own "lifestyle" magazines, which used style and content elements to promote smoking and their own products.[263]

Figure 10.8 Advertisement for Salem Menthol Cigarettes, *Maxim* Magazine, March 2004



Source: Rutgers School of Public Health 2004.[472]

Newspaper advertising has declined dramatically since the 1970s and early 1980s, when newspapers represented a major channel of communication for the tobacco industry, accounting for 20% or more of advertising spending in any given year.[227] Since 1992, newspaper advertising has represented less than 1% of all tobacco industry cigarette advertising and promotional spending; in 2011 the industry spent $549,000 on newspaper advertising. Newspaper spending data are not available for 2012 through 2014 because only one company reported spending in this category.[227]

Evidence from the period leading up to 1990 indicates that spending on newspaper advertising may have been aimed at creating support in the African American community for policies favorable to the industry. A 2012 study analyzed information from the archives of the National Newspaper Publishers Association and tobacco industry documents from 1968 through 2004, and concluded that "in exchange for advertising dollars and other support, the tobacco industry expected and received support from Black newspapers for tobacco industry policy positions" prior to 1990.[264,p.739] Indeed, historically, African American newspapers have received revenue from tobacco industry advertising and other forms of support to sustain circulation,[264] and contemporary readers of publications with a large African American audience can experience a higher level of exposure to tobacco advertising than readers of publications addressed to a more general audience.[265] A content analysis of African American and general audience newspapers from 2004 through 2007 showed that African American newspapers were more likely to include tobacco product advertising than general audience newspapers, although in both types of newspapers, advertising promoting commercial goods/services to stop smoking was more common than advertising for tobacco products.[265]

Outdoor Advertising

The MSA (signed in 1998) banned what was then the primary form of outdoor tobacco advertising—tobacco advertisements on outdoor billboards larger than 14 square feet.[266] As a consequence, spending for this category decreased significantly, from $294 million in 1998 to $53.9 million in 1999.[267,268] Industry spent $2.2 million in 2014 on outdoor cigarette advertising[227] compared to $1.1 million on outdoor advertising for smokeless tobacco products.[228]

There is some evidence that outdoor tobacco advertising may be disproportionately targeted to minority and low-income communities. Studies conducted before the MSA found more tobacco billboards in African American and low-income areas than in other areas,[269–273] and determined that a large proportion were placed near public schools.[274] Studies have also found that cigarette advertising in African American, Latino, and low-income communities[232,275] tends to be larger and more likely to be located within 1,000 feet of a public school than in other communities; it also tends to promote menthol products and display lower prices.[234]

Packaging

The importance of the cigarette package itself as a form of cigarette advertising has increased, as restrictions on advertising through traditional media have become more common.[276] Cigarette packaging is used to communicate certain characteristics of a brand or product to consumers.[225,276–281] In turn, consumers use the pack to communicate their brand choice to peers.[278,281] A recent study suggests that packaging contributes to brand selection among youths,[279] an important consideration given that brand selection is highly correlated with race/ethnicity.[282] In addition, the color and shape of a cigarette pack can indicate a masculine or feminine product, luxury or value product, and menthol or non-menthol product. Product displays have been heavily used in urban areas to promote menthol products.[233]

Attractive and prominent packaging displays may also undermine cessation among adults.[180] For example, in a study of tobacco purchasing, 25% of current smokers reported purchasing cigarettes on impulse after seeing cigarette displays, and more than one-third of former smokers and those attempting to quit reported experiencing an urge to buy cigarettes on encountering a retail display.[283]

Packaging design also influences the consumer's perceptions of risk. In the United States, a provision of the Family Smoking Prevention and Tobacco Control Act of 2009 bans use of the terms "light," "mild," or "low," or similar descriptors, without a marketing authorization from the FDA.[19] The court in *United States of America v. Philip Morris USA, Inc.* also prohibited the defendants and other covered persons and entities from using misleading descriptors such as "low-tar," "light," "mild," and "natural."[284,p.938] In response, tobacco companies have moved to distinguish among brands by color; studies have shown that consumers are able to distinguish between "regular" and "light" cigarette products in the absence of text labels.[279,285–288] Indeed, numerous studies have shown that the color of cigarette packaging is associated with risk perceptions among smokers, with lighter packages conveying reduced risk.[285,287–293] Adults and youth are significantly more likely to rate "silver" and "gold" packs as lower tar and lower health risk; adults are significantly more likely to say it is easier to quit smoking these "silver" and "gold" packs; and youths are significantly more likely to say a "silver" or "gold" pack is their top choice if trying smoking for the first time.[291,294,295]

Health warnings on cigarette packages, particularly warnings with large pictorial images, are effective across diverse populations.[296] For example, a 2012 experimental study involving a large diverse population found that graphic pictorial warnings were more effective than text-only versions, and smokers indicated that the warnings were more impactful and credible and had a greater effect on their intentions to quit. The stronger impact of pictorial warnings was consistent across race/ethnicity, education, and income.[297] Similarly, a study by Thrasher and colleagues[298] found that labels with graphic imagery were more effective for groups of various races/ethnicities and levels of health literacy than text or other types of imagery. NCI Monograph 21 noted that:

> Studies have assessed the ability of health warnings to reduce differences in knowledge and smoking behaviors between population subgroups, particularly between advantaged and disadvantaged groups within countries. In general, these studies indicate that pictorial warning messages have very wide reach, and can be a broadly effective tool in improving knowledge and reducing health disparities. For example, a study comparing the impact of pictorial warning labels with text-only labels among U.S. adult smokers found that the pictorial warnings were more effective across diverse racial/ethnic and socioeconomic groups, concluding that 'pictorial health warning labels may be one of the few tobacco control policies that have the potential to reduce communication inequalities across groups'.[3,p.290,297,p.1]

In addition, the importance of revising warnings over time to avoid "wear-out" is now well recognized.[3,299]

Australia became the first country to implement plain (standardized) packaging for all tobacco products (December 2012). Under the law, all tobacco products sold in Australia must have a standardized "drab dark brown" package with the brand name and variant name shown in a standard font, style, and size on the front of the package.[300] The law also standardized the appearance and color of the tobacco products and increased the size of the required pictorial health warnings.[301] As noted in NCI Monograph 21,

"plain (standardized) packaging (i.e., devoid of logos, stylized fonts, colors, designs or images, or any additional descriptive language) reduces the appeal of tobacco products, enhances the salience of health warnings, minimizes consumers' misunderstanding of the hazards of tobacco, and has contributed to a decline in tobacco use in Australia, the first country to implement plain packaging."[3,p.303] As other countries implement plain packaging, this will provide the opportunity to examine its effects among diverse population groups.[302]

Advertising at the Point of Sale, Price Discounts, and Other Promotional Channels

As other venues for tobacco advertising have been increasingly restricted, the tobacco industry has directed its marketing dollars to point-of-sale (POS) advertising, promotions, and price discounts. In the 10 years after the MSA was implemented, tobacco companies spent a total of $110 billion, or 92% of their total marketing expenditures for cigarettes and smokeless tobacco products on advertising, promotions, and price discounts in convenience stores, gas stations, grocery stores, and other retail outlets that sell tobacco.[303] In the face of increasing advertising regulation, the retail context has become an important channel through which tobacco companies communicate with their target audience.[233,304]

Point-of-Sale Advertising

Tobacco companies and retailers often use POS displays, along with complementary tactics such as promotional discounts, to attract consumers (Figure 10.9).[280] Tobacco industry spending on POS advertising, separate from price discounts and promotions, totaled $138.2 million for cigarettes and $33.0 million for smokeless tobacco products in 2014.[227,228] Analysis of the 2011 National Youth Tobacco Survey found that 81.5% of middle school students and 86.9% of high school students reported exposure to pro-tobacco advertising in stores.[212] The study also found that African American students were somewhat less likely than white students to report seeing store advertisements. Overall, middle school and high school students' exposure to pro-tobacco advertisements at retail stores declined from 87.8% in 2000 to 76.2% in 2012.[257]

Figure 10.9 Displays of Tobacco Brand Prices at the Point of Sale, Including Special Discounts, 2011

Source: Truth Initiative 2011.[473]

Data from other studies suggest that advertising at retail POS has increased disproportionately in disadvantaged neighborhoods since the passage of MSA restrictions.[230,231] Numerous studies of individual communities have demonstrated a greater concentration of stores selling and advertising

cigarettes in African American and Hispanic communities,[305–313] although not all studies were consistent.[312,314] One study of New York City community districts found that the density of tobacco retailers in the community and their proximity to schools co-varied with population density, commercial land use, and indicators of social disadvantage such as health insurance coverage.[315] A national study examining density and sociodemographic factors across 64,909 census tracts in the continental United States found that tobacco outlets were more concentrated in urban areas and in tracts with larger proportions of African Americans, Hispanics, and women with low levels of education.[316]

Retailer density has in turn been associated with young people's self-reported exposure to point-of-sale advertising.[307] Research has also shown a greater amount of in-store tobacco advertising in neighborhoods that are predominantly lower income and African American.[317–320] One study found a greater proportion of menthol advertisements in neighborhoods with larger African American student populations.[317] Census block groups with larger African American,[321] Asian, low-income, and young populations have also been shown to have more advertisements for menthol brands.[322]

The results of a longitudinal school-based study of an urban, racially diverse California community showed that African American youths were three times more likely than youth of other racial groups to recognize the Newport brand and less likely than other racial groups to recognize the Marlboro brand. After adjustment for shopping frequency and other risk factors, youths who recognized the Newport brand at baseline were more likely to have initiated smoking at 12-month follow-up, regardless of race.[323] Research on other tobacco products has found that little cigars and cigarillos are more likely to be available, advertised, and less expensive in Washington, D.C., communities with a greater proportion of African Americans than in other communities.[324]

More than one in four African Americans are younger than age 18 (27.8%) compared with about one in five among the white population (21.7%); this indicates that a larger fraction of the African American population than the white population is at risk for tobacco marketing aimed at youth.[325] Studies have shown that POS cigarette displays are associated with greater brand recall by youths[326] and with unplanned or impulse purchases.[283,327] For example, a study conducted in New York State using observational estimates of exposure found that, for youths, living in counties with more retail cigarette advertisements was associated with having positive attitudes toward smoking.[328] Other studies have shown that youths' exposure to POS advertisements was associated with more positive perceptions of people who use the product,[329] a weakened resolve not to smoke in the future,[202,323] and experimental smoking and smoking initiation.[308,330–332]

Another youth-focused study found that exposure to retail advertising was linked with increased odds of ever smoking at baseline and that pro-tobacco media and advertising at the POS increase susceptibility to smoking over time.[239] One study showed that Hispanic youths were more likely to be exposed to retail tobacco advertising than other youths (76% vs. 60%, respectively) and that the odds of ever smoking increased 50% among youths who were exposed to retail tobacco advertising, after controlling for other factors.[333]

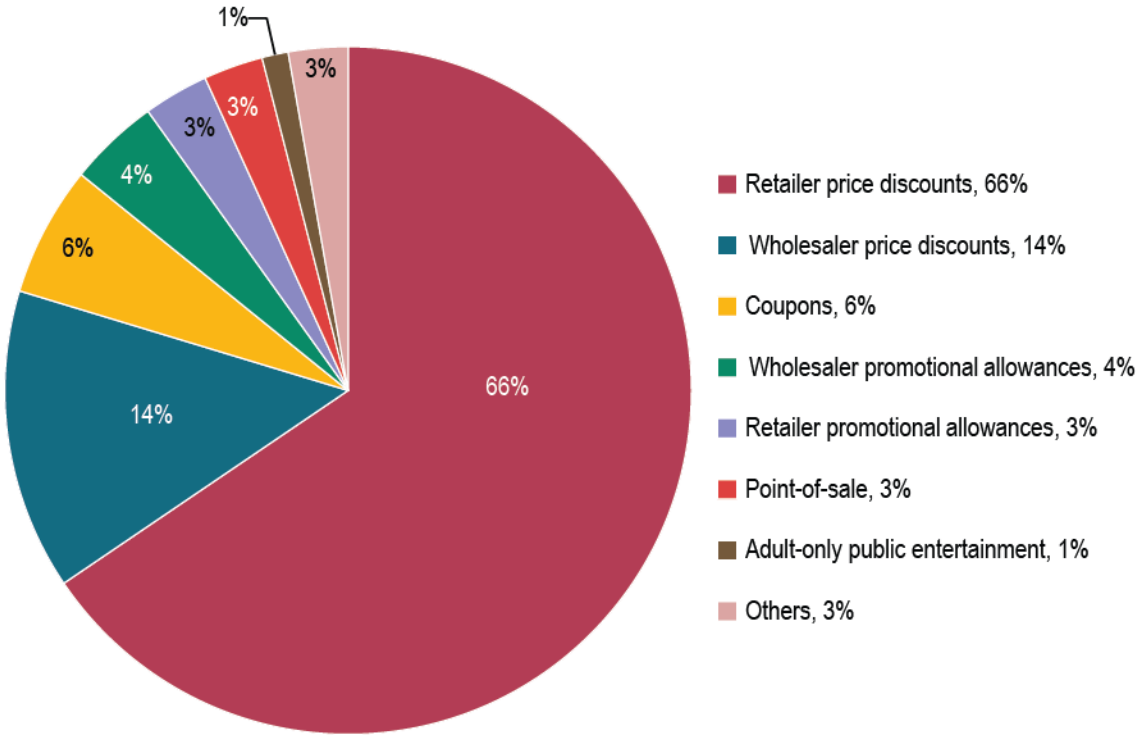
Price Discounts and Promotions

Price discounts are defined as payments made to the retailer or wholesaler to reduce the price consumers pay for tobacco products; in doing so, they counteract the impact of significant tax and price increases to reduce smoking and encourage cessation. Price promotions to retailers (e.g., payments for cigarette

stocking, shelving, displaying, incentives) and wholesalers (e.g., payments for volume rebates)[227] help maintain a pro-tobacco environment by ensuring prominent selling space for tobacco products and by creating strong financial bonds with retailers.[3]

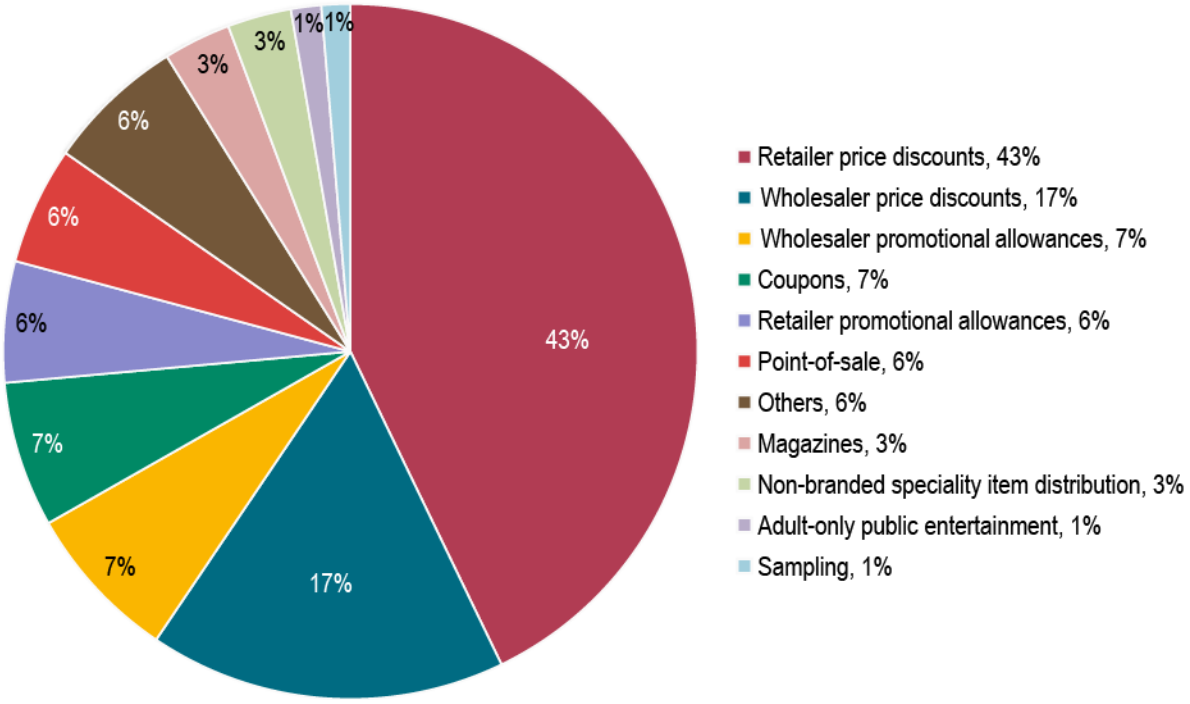
Price discounts have represented the largest category of spending on cigarette advertising and promotion since 2002 when the FTC began reporting these expenditures as a separate category.[227] Price discounts accounted for approximately 80% ($6.8 billion) of total U.S. tobacco industry spending on cigarette advertising and promotion in 2014, with 66% ($5.6 billion) spent on retailer price discounts and 14% ($1.2 billion) on wholesaler price discounts.[227] Similarly, for smokeless tobacco products, price discounts were the largest category of promotional expenditures, accounting for 59% ($357.2 million) of total promotional spending for smokeless products in 2014, with 43% ($257.3 million) spent on retailer price discounts and 17% (99.8 million) on wholesaler price discounts.[228] Promotional allowances paid to wholesalers, coupon expenditures to lower the cost of tobacco products, and promotional allowances paid to retailers are the next largest advertising and promotional expenditures reported by U.S. cigarette and smokeless tobacco manufacturers (Figures 10.10 and 10.11).

Figure 10.10 Distribution of U.S. Cigarette Advertising and Promotional Expenditures, 2014



Note: "Others" include magazines, direct mail, non-branded specialty item distribution, company website, outdoor, branded specialty item distribution, other promotional allowances, telephone, and all others (newspapers, sampling distribution, and other Internet).
Source: Adapted from Federal Trade Commission 2016.[227]

Figure 10.11 Distribution of U.S. Smokeless Tobacco Advertising and Promotional Expenditures, 2014



Note: "Others" include direct mail, company website, outdoor, Internet – other, other promotional allowances, and all others (newspapers, retail-value-added—bonus smokeless tobacco product, and social media).
Source: Adapted from Federal Trade Commission 2016.[228]

Price discounts disproportionately affect low-income and racial/ethnic minority smokers, who are more sensitive to price[334] and more likely to take advantage of promotional offers.[282] Tobacco companies have used price discounts to increase the menthol cigarette market in low-income, predominantly African American urban communities (Figure 10.12).[233,317] Interviews with a former Brown & Williamson trade marketing manager revealed systematic differences in the application of price discounting; the former employee could offer retail outlets in low-income African American urban communities—referred to as "focus" communities—greater price discounts than would be offered to outlets in "non-focus" or white suburban communities. These discounts resulted in lower prices for consumers, primarily for the purchase of menthol products.[233]

Figure 10.12 Salem Menthol Print Advertisement With Coupon, 2003

Source: Rutgers School of Public Health 2003.[474]

In addition, the availability of store-advertised promotional offers (multipack discount, other discount, or gift with purchase) for Newport cigarettes was related to school/neighborhood demographics: Promotional offers were more available and Newport cigarettes were less expensive in neighborhoods near high schools with more African American students.[317] Less evidence is available regarding differences in price discounts by race/ethnicity for little cigars and cigarillos. A study of tobacco retailers in Washington, D.C., found that price per cigarillo decreased significantly with increasing proportion of African American residents.[324] Price discounts and promotions and possible differential exposure and response to these strategies are important examples of the structural-level communication inequalities that can lead to TRHD.

Other Promotional Channels

Direct-to-consumer tobacco marketing, through mail, Web, email and mobile marketing platforms, allows tobacco companies to reach consumers to distribute price promotions (coupons and "give-aways"), to offer brand-loyalty programs, and to target specific market segments.[255,335–339] A 2014 study found that 12% of 15- to 17-year-olds and 26% of 18- to 23-year-olds were exposed to direct-to-consumer tobacco marketing, and racial/ethnic minority nonsmoking respondents were more likely than nonsmoking whites to see tobacco websites.[340]

The tobacco industry may also reach consumers through sponsorship of a variety of different events (although both the MSA and the Family Smoking Prevention and Tobacco Control Act limit event sponsorship) and through promotions at venues such as bars and nightclubs. Studies analyzing tobacco industry documents find that the tobacco industry expects bar promotions to help develop or maintain brand equity among young adults.[345–346] Research indicates this tactic is successful; young adults report a high level of exposure to direct marketing practices in nightclubs and bars, including in-person interactions with tobacco marketers and the distribution of free gifts. Initiation[344] and progression to established smoking are significantly more likely among youths who attend adult-only venues and report being exposed to tobacco marketing.[345,346] Results from a 2005 cross-sectional study of young adults from a Web-enabled Knowledge Networks panel found that advertising in bars was associated with current smoking and having not made a serious quit attempt, independent of alcohol use.[347] Essentially no research describes the prevalence of exposure or the effects of these events by SES or race/ethnicity, but some campaigns, such as the Kool Mixx campaign, appear specifically designed to appeal to African Americans and followers of hip-hop culture.[1,348]

Industry Advertising and Promotion to the LGBT Community

It is well established that LGBT populations are at elevated risk for tobacco use relative to their heterosexual peers.[349–353] And although most literature on pro-tobacco advertising and promotion and most surveys of media consumption do not report specifically on the LGBT community, evidence supports the idea that tobacco company targeting of this group contributes to this disparity.[354]

The tobacco industry was among the first large industries to advertise in LGBT publications and collaborate with LGBT organizations through sponsorship and philanthropy, beginning in the early 1990s.[355,356] For example, researchers interviewed leaders of 74 LGBT organizations and publications in the United States and found that 22% had accepted tobacco industry funding.[357] In part as a result, LGBT individuals are more likely than heterosexuals to encounter tobacco advertisements and promotions and may be more receptive to such marketing efforts (Figure 10.13).[358] The response of the LGBT population to tobacco industry targeting has been mixed: Although some people have expressed concern, others have viewed it as a positive development for LGBT equality and inclusivity.[355,356]

Figure 10.13 Advertisement in *OUT* Magazine, January 2002

Note: Text at the top of the page states: "Whatever the approach, each of these companies has decided to demonstrate its commitment to gay and lesbian Americans by speaking directly to us in … Companies that Care."
Source: Rutgers School of Public Health 2002.[475]

Tobacco industry documents clearly show the industry's interest in the LGBT population. The Phillip Morris and RJR document collections include information on several LGBT publications, such as *Out*, *The Advocate*, and *Venus*, with data on readership demographics, circulation, advertisement prices, and other related information. These collections also include letters from publications (e.g., *Venus*, *HeatStroke*) thanking the tobacco company for its interest in advertising in their magazines. Direct mentions of targeting the LGBT community appear in marketing strategy documents, such as a marketing document for the Eclipse brand; Project SCUM (subculture urban marketing), which targeted gay and homeless populations[359]; and PRISM, a gay and lesbian employees group, which proposed activities such as building awareness of gay and lesbian marketing data and fostering relationships with gay and lesbian business associations.

Based on their study of industry documents and marketing materials produced by tobacco companies, Stevens and colleagues outlined four key strategies used by the industry to market to LGBT populations: (1) direct advertising in LGBT publications (Figure 10.14); (2) indirect advertising in mainstream publications with high LGBT readership, such as alternative newsweeklies; (3) community outreach and promotions (e.g., LGBT bar nights featuring cigarette brands and support of HIV/AIDS causes); and (4) event sponsorships (e.g., LGBT film festivals and pageants).[359]

Figure 10.14 Advertisement from a 1995 Issue of *OUT* Magazine

Note: A 1995 issue of *Rolling Stone* contained an ad nearly identical to this one except that it omitted the seated man.
Source: Rutgers School of Public Health 1995.[476]

A 2002 article identified other key industry strategies for targeting LGBT communities, particularly youths, and described the industry's targeting of geographical areas popular among LGBT young people[357] and its attempts to take advantage of the LGBT bar culture through its marketing investment (e.g., distributing free cigarette samples, buying free drinks).[356,360–363]

LGBT communities are also exposed to pro-tobacco messaging through movies. One study found that 87% of movies with LGBT themes or characters depicted tobacco use, showing an average of four occurrences of tobacco use per hour. Only 3% of these incidents conveyed a sense that any harm was caused by tobacco use.[364]

Evidence Review: Pro-Tobacco Communication and Marketing

An extensive body of research and the conclusions of many major reports document a causal relationship between tobacco industry advertising and promotion and increased tobacco use.[1,3,19] The discussion below highlights the information available on the effects of tobacco advertising and promotion on smoking behaviors by race/ethnicity or SES.

- There is some indication that the effects of depictions of smoking on television and movies on youth smoking may differ by race/ethnicity; differences may be attributable to groups' varying degrees of exposure and to attributes of the characters engaged in smoking. This is consistent with the SIM, which holds that social determinants and sociodemographic factors can impact media use and exposure as well as information processing, which in turn can affect health outcomes.

- Studies show that youth (overall, and of all races/ethnicities) continue to be exposed to advertisements for cigarettes and smokeless tobacco products. African American youths' exposure to tobacco advertising in adult magazines is greater than that of white youths. Studies have also documented that outdoor tobacco advertising at retail outlets is more common in African American and low-income communities compared with predominantly white or higher SES communities. These institutional-level inequalities may well exacerbate TRHD; few studies have been conducted in the United States that examine links between outdoor retail advertising and youth or adult smoking behavior. A longitudinal study of outdoor advertising in combination with indoor advertising suggested a dose–response relationship between the frequency of exposure to branded cigarette advertising at retail outlets and smoking initiation.
- Pictorial health warnings on cigarette packages are effective across diverse populations. Research on the potential effects of components of packaging other than warning labels on various racial/ethnic groups is limited.
- Studies of the density of POS advertising have had mixed results, but evidence suggests that stores selling tobacco products are more concentrated in urban areas and in neighborhoods with larger proportions of African Americans, Hispanics, and women with low levels of education. These institutional-level inequalities likely intersect with individual-level inequalities to contribute to TRHD. Evidence is accumulating that low-income neighborhoods that are predominantly African American or Hispanic tend to have more in-store tobacco advertising, including more advertisements for menthol brands. Studies have shown that exposure to retail tobacco advertisements is linked to a variety of outcomes among youth, from positive attitudes and improved perceptions of people who use the tobacco product (brand user imagery) to increased susceptibility and experimental smoking and higher odds of ever smoking.
- Price discounts, defined as payments made to the retailer or wholesaler to reduce the price consumers pay for tobacco products, are an important promotional strategy for the tobacco industry. Because low-income smokers are more sensitive to price, they are disproportionately affected by price discounts.
- Research on the impact of event sponsorship by the tobacco industry is limited. Some evidence suggests that initiation and progression to established smoking are more likely among young adults who attend bars and nightclubs and report being exposed to tobacco marketing. However, the prevalence of exposure to industry-sponsored events or their effects by SES or race/ethnicity is not known.

The News Media and Tobacco Communications

Like anti-tobacco media campaigns, the news media can draw attention to the negative effects of tobacco use, promote smoking cessation, and affect tobacco-related knowledge, attitudes, and behaviors in a multitude of ways. For example, one study found that each time a newspaper's number of tobacco-related articles increased by 10 over a 5-month period, the likelihood would increase that readers would perceive great harm from smoking and disapprove of smoking, and the odds of perceiving most or all friends as having smoked in the past 30 days would decrease.[365] A study examining newspaper coverage of the Florida Tobacco Control Program found that news coverage of the program, particularly coverage of youth advocacy efforts, contributed to observed declines in current smoking after controlling for alternate explanations, leading the study authors to conclude that newspaper coverage of health communication campaigns might represent a meaningful indirect source of campaign effects.[366] Research on news coverage about drunk driving suggests that the impact of news coverage on behaviors

is often indirect rather than direct: News coverage can drive policy actions which in turn impact behaviors.[367]

The strategic use of news-making through TV, radio, and newspapers can increase awareness about the health effects of tobacco, promote public debate, and generate community support for changes in tobacco-related community norms and policies. However, health journalism often fails to meet these goals. A study of news coverage (newspapers, new magazines, and TV newscasts) in the United Sates over a 2-year period (2002–2003) found that coverage of tobacco topics was only modest and that tobacco's negative health effects were rarely mentioned; however, when newspapers did cover a tobacco story, it was accorded relatively high prominence.[368] Additionally, an extensive analysis of cancer coverage in the media by Stryker and colleagues suggested that although articles often discuss tobacco as a major risk factor for disease, only 8% of these stories treat tobacco as a primary focus of prevention.[369]

These findings highlight the role that individuals and organizations play in shaping news about the link between tobacco and health outcomes such as cancer.[370] Stories offered by media channels are products of the interaction between news sources and media professionals. Journalists routinely gather information that is used to create news[371] from established or organized sources, including spokespersons for government agencies, businesses, diverse professionals, organized community groups, and others.[372,373] These sources can perform the key role of identifying topics and bringing them to the media's attention.[374] Sources also compete for public attention and for the chance to define and increase the public profile of their issue. Community-based, grassroots efforts often lack the resources and media savvy to compete with tobacco industry–funded efforts, which in effect limits their ability to influence whether and how tobacco-related issues are covered as a broader public health concern.[375] The tobacco industry has also used diverse strategies to influence media coverage of smoking and health in ways favorable to their interests.[264,376–379]

Evidence Review: The News Media and Tobacco Communications

- News media coverage can contribute to promoting or preventing tobacco use. Both the tobacco industry and tobacco control advocates attempt to influence news media coverage.
- Limited studies show that increased coverage of tobacco issues or anti-tobacco campaigns can have positive effects on tobacco-related attitudes and behaviors, though it is possible these effects may disproportionately benefit those of higher SES, in accordance with fundamental cause theory and the knowledge gap hypothesis. However, health journalism often fails to reinforce the health reasons for tobacco control efforts or to feature tobacco use as a primary focus of prevention.
- Studies have shown that the tobacco industry often makes claims that are featured in news stories or deploys industry-supported consultants who respond in the news media to stories related to tobacco. Communication inequality frameworks suggest that disadvantaged groups may have less ability than other groups to distinguish between objective news sources and claims made by the tobacco industry.

New Communications Technologies: The Web and Beyond

Over the past decade, digital media and communications technologies have evolved to offer novel ways to reach diverse audiences. The terms "new media," "social media," and "Web 2.0" are often used

interchangeably to refer to these new technologies.[380,381] Web 2.0, the term used in this chapter, may be described as "a set of economic, social, and technological trends that collectively form the basis for the next generation of the Internet, a more mature, distinctive medium characterized by participation, openness, and network effects."[381,p.1,382]

Compared with the more static and unidirectional focus of the first generation of the Internet (Web 1.0), Web 2.0 is multidirectional and interactive, enabling previously unimaginable degrees of user-generated content and sharing. Web 2.0 applications that are being used to promote smoking prevention and cessation include websites, social networking platforms[383] such as Facebook and Twitter, photo and video creation and sharing platforms, discussion forums, blogs, video conferencing, mobile applications, online and mobile games, and combinations of these channels. These same channels are also being used extensively by the tobacco industry and others to promote tobacco use, and pro-tobacco norms.[384–393]

This section reviews evidence on access and use of Web 2.0 applications, and how the effects of pro-tobacco and anti-tobacco content accessed through new media channels are mediated by age, gender, race/ethnicity, and SES. The literature on these subjects was searched through electronic databases such as PubMed, Embase, PsycINFO, and Web of Science. Search terms related to Web 2.0 included ("social media" OR "digital media" OR "new media" OR online OR mobile OR YouTube OR Facebook OR "video games" OR "online gaming" OR "mobile gaming") AND ("public health" OR "tobacco prevention" OR "tobacco control" OR "tobacco promotion" OR "tobacco industry" OR "tobacco companies") AND (minority OR "African American" OR black OR Latino OR Hispanic OR Asian OR "Native American" OR "Alaska Native"). Information on different definitions and terms related to Web 2.0 was derived from systematic reviews and overview articles on social network sites and on new media. Due to the interdisciplinary nature of the topic, the literature search was supplemented with additional resources: national media use surveys by the Pew Research Center, the U.S. Department of Commerce, the Kaiser Family Foundation, and others. This literature review focuses on evidence published between 2008 and 2014. Where applicable, sections have been updated to reflect trends seen at the time of publication (2017).

Internet and Social Media Access and Use Patterns

Internet access has increased rapidly in the United States; as of 2016, only 13% of Americans reported they do not access the Internet. Analyses have found that Internet non-access is correlated with age, educational attainment, household income, and community type. For example, rural Americans are twice as likely to report never using the Internet compared to their urban or suburban counterparts.[394] Although progress has been made in shrinking the digital divide, disparities persist, particularly among low-income groups[395] as shown in Table 10.3.

Table 10.3 Internet Access and Use Patterns in the United States, 2015–2016

Access Vehicle	General Population	White	African American	Hispanic	Annual Household Income <30K
Home high-speed broadband Internet penetration, 2016[36]	73%	78%	65%	58%	53%
Mobile phone ownership, 2016[396]	cell phone: 95% smartphone: 77%	cell phone: 94% smartphone: 77%	cell phone: 94% smartphone: 72%	cell phone: 98% smartphone: 75%	cell phone: 92% smartphone: 64%
Social networking site use (e.g., Facebook, Twitter, Instagram, LinkedIn), 2016[401]	69%	69%	63%	74%	60%
Gaming, 2015[403,477,478]	adults: 49% teens: 72%	adults: 48% teens: 71%	adults: 53% teens: 83%	adults: 51% teens: 69%	adults: 46% teens: 70%

Web 2.0 platforms can be accessed from a multitude of channels and devices in this digitally connected landscape. Smartphone ownership rates are comparable across races/ethnicities, with rates among non-Hispanic African Americans and Hispanics being slightly lower than rates for non-Hispanic whites.[396] However, 12% of Americans access the Internet only via their smartphones because they do not have home high-speed broadband Internet.[396] Among smartphone users, higher percentages of racial/ethnic minorities, low-SES groups, and younger age groups say that they usually access the Internet via their cell phones.

Researchers have cautioned against assuming that increasing mobile access lessens the digital divide, pointing out that a variety of services, such as video on demand, telemedicine, and Internet classrooms, require reliable high-speed connections rarely found through wireless and mobile channels.[397] In addition, activities that require a great deal of typing can be difficult on a hand-held device, and monthly data caps can prohibit such activities as downloading large video files; all of these factors contribute to meaningful differences in what various demographic groups are able to do online.[397] Moreover, although technology ownership requires only a one-time purchase, continuing access to services such as data plans are a recurring expenditure, and maintaining a subscription can be a challenge for disadvantaged groups.[398,399] In 2015, 48% of smartphone-dependent people reported they have had to cancel or turn off their cell phone service because the financial cost to maintain it was too great.[400]

Use of social networking sites (e.g., Facebook, Twitter, LinkedIn) generally is more prevalent among females and young people, but more older adults are also adopting use of these sites, particularly Facebook. Overall, 69% of U.S. adults use social networking sites[401] and 68% use Facebook, the most popular social networking platform, followed by Instagram (28%), Pinterest (26%), LinkedIn (25%), and Twitter (21%).[402] These sites vary in their uptake among certain audiences; for example, Twitter and LinkedIn tend to be more popular among those with college degrees—29% of Internet users with college degrees use Twitter and more than 50% use LinkedIn.[402]

A greater percentage of African American teens report playing video games compared to white and Hispanic teens.[403] Another survey found that gaps can be seen in relation to parents' education level among groups in time spent playing games on computers, gaming consoles, or mobile devices. On average among youth ages 8–12, those with high-school-educated parents spent an average of 2 hours

and 17 minutes per day playing any type of video game compared to an average of 1 hour and 42 minutes per day among those with college-educated parents.[250]

Use of Web 2.0 for Anti-Tobacco Communications and Marketing

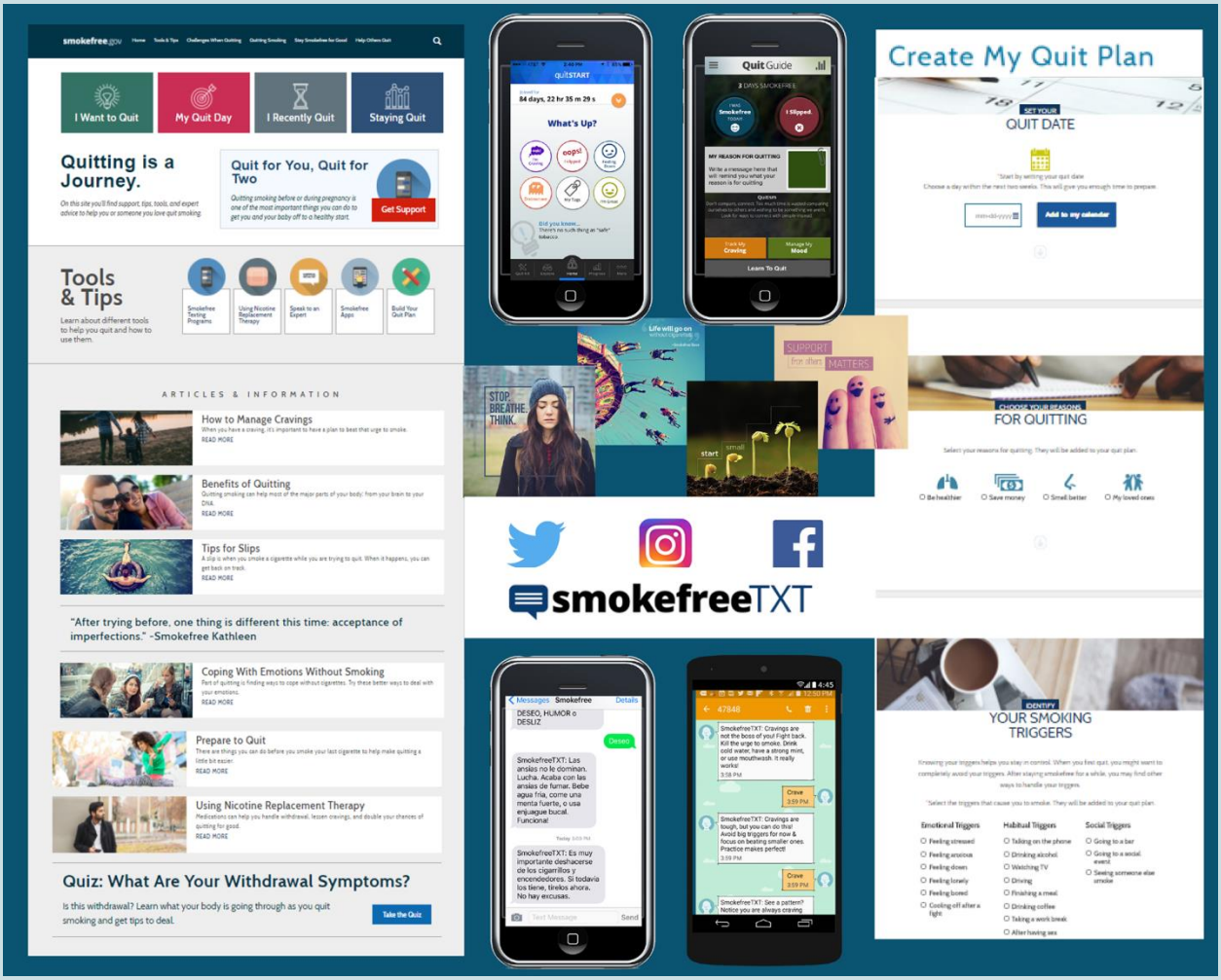
Web 2.0 applications are currently used as a broadcasting platform to amplify messages from traditional media sources, such as television or radio, and as a new way to reach and engage target audiences.[404] In the realm of health promotion, Web 2.0 applications can allow practitioners more direct access to target populations, enabling the consumer to be part of the promotional process. For example, tobacco control organizations may hold contests in which participants develop commercials to promote tobacco prevention and smoking cessation, and the winning entry is produced and aired.[405] They may also use viral or word-of-mouth marketing, through which friends encourage their social network to participate in a wide array of health-promotion interventions.[405] Web 2.0 applications can also be used to gauge audience members' beliefs about a subject, such as tobacco use, or to provide an outlet for audience members to support one another in achieving a behavior change, such as smoking cessation.[406]

Web 2.0 can facilitate and mobilize direct action supporting or opposing particular policies. A series of case studies emerging from a tobacco control Facebook group illustrate how tobacco control advocates used Web 2.0 applications to mobilize people to take action.[407] In one example, advocates encouraged group members to post messages urging a celebrity to drop tobacco sponsorship from her concert and to join a Facebook page with the same message; ultimately, the sponsorship was withdrawn. In another example, advocates created a petition on the Web that requested a major music festival drop tobacco sponsorships, and encouraged people to make their messages to band members immediate and public by posting their comments on the band members' Facebook and Twitter pages.[407]

Usage demographics make it clear that advanced mobile applications are a promising channel for reaching underserved groups, because they incorporate social network and other Web 2.0 tools and are increasingly prevalent among minority populations who generally have less access to high-speed broadband Internet.[400,408] Public health–related mobile applications are often used with websites to support behavioral monitoring, social support networks, and feedback,[409] all of which can play a role in tobacco control and cessation efforts. A systematic review of one-way and Web 2.0 mobile phone interventions for smoking cessation conducted in 2010 indicated that mobile phone–only interventions can be effective both in the short and long term, but three of the five interventions examined included studies that found no effect.[410]

The Smokefree.gov Initiative (SFGI) developed by NCI provides free Web- and mobile-based quit smoking information to the public, including targeted resources specific to populations with unique information needs and/or higher smoking rates (see Box 10.5).

Box 10.5: The National Cancer Institute's Smokefree.gov Initiative



Source: National Cancer Institute 2017.[479]

The Smokefree.gov Initiative (SFGI) offers a variety of traditional Web-based and interactive tools including 6 websites, 2 mobile applications, 6 social media accounts, as well as 15 text messaging–based programs to help teen and adult smokers quit using tobacco and live healthier lives. Across these platforms, the SFGI serves the general public and audiences with specific information needs including women of reproductive age and pregnant women, teens, veterans, Spanish speakers, LGBT groups, and older adults.

Smokefree.gov Websites

The Smokefree.gov website (https://smokefree.gov) anchors the SFGI and provides smokers with evidence-based cessation advice and support, including information about preparing to stop smoking, effective quit methods, and challenges to quitting. The website serves as an entry point for all SFGI mHealth resources and tools, as well as NCI's telephone and online smoking cessation counseling services (https://smokefree.gov/tools-tips/speak-expert).

Smokefree.gov employs a variety of interactive features, tools, and resources to provide highly relevant information to smokers seeking cessation information and support. A quit plan builder guides smokers through the steps to prepare for and undertake a successful quit attempt. Quizzes help users assess topics

such as their nicotine dependence, stress level, or withdrawal symptoms to inform their quit experience. Videos and user-generated testimonials from social media accounts offer firsthand advice and encouragement for smokers and former smokers to quit permanently. The SFGI encourages smokers to join supportive online Smokefree.gov communities on Facebook, Instagram, and Twitter to get inspiration and motivation from smokers and former smokers.

Text Messaging Programs

The SFGI offers general and audience-specific text messaging programs for smoking cessation and other health behavior changes. The Smokefree.gov text messaging programs are designed as interactive tools to provide personalized, on-demand support and information. SmokefreeTXT (https://smokefree.gov/Smokefree-Text-Messaging-Programs) offers a 6-8 week text messaging–based smoking cessation intervention to smokers trying to quit. Smokers can choose from a variety of other cessation-focused text messaging programs, depending on their support needs (e.g., building quitting skills vs. cessation). Smokers can also choose programs to address healthy eating, physical activity, or weight management.

Smartphone Apps

The SFGI supports two free interactive smoking cessation smartphone apps (https://smokefree.gov/tools-tips/apps). QuitGuide for adults and quitSTART for teens are designed to help users prepare to quit smoking and build the skills needed to become and stay smoke free. These tools offer personalized cessation support by allowing users to track their cravings and moods, tag specific locations and times of day that trigger their tobacco use, get personalized information that matches their smoking history and quitting goals, request on-demand help, and monitor their progress toward smoke-free milestones. Both apps are available for download in iOS and Android.

Other types of mobile phone interventions are also being used for tobacco prevention and control. One emerging area is the combined use of video, online, and mobile games to encourage smoking prevention and health promotion. Games across a variety of platforms are now a dominant form of media that is enjoyed by a variety of demographic groups,[411] and evidence indicates that playing games designed for prevention and health promotion purposes can lead to positive health-related changes.[412]

Researchers are beginning to explore the use of video, online, and mobile gaming for tobacco prevention and control. For example, *QuitIT*, attempts to integrate the principles of smoking behavior change and relapse prevention.[413] The "truth" campaign launched a free-to-play iOS and an Android mobile game, *Flavor Monsters*, which attempts to prevent youth smoking by revealing the tobacco industry's use of appealing flavors to entice young people to initiate tobacco use (Figure 10.15).[407] An evaluation of the game found that after adjusting for age, gender, and whether someone had ever tried cigarettes, player status was a significant positive predictor of tobacco-related knowledge, and level of engagement was a positive predictor of the number of correct responses. Playing *Flavor Monsters* was found to be a significant positive predictor of anti-tobacco industry attitudes and beliefs, after controlling for baseline anti-industry attitudes and other factors, but player status was not a predictor of intention to smoke at follow-up.[414]

Figure 10.15 Screenshot from *Flavor Monsters* Game

Source: Truth Initiative 2013.[480]

Other mobile applications are also being used for tobacco prevention and control purposes, as evidenced by a 2011 study that examined 47 iPhone applications for smoking cessation. This study found, however, that these applications rarely adhered to evidence-based guidelines for smoking cessation interventions,[415] suggesting that efforts to use mobile tools to affect tobacco-related behavior are in the early stages. More strategic development of these tools and more extensive evaluation of their impact are necessary to determine how mobile devices may complement tobacco prevention and control programs and campaigns.

Few studies examine the use of Web 2.0 for anti-tobacco efforts targeting specific populations (see Box 10.6). One Web-based intervention, SmokingZine, was found to have promising results: Intentions to try a cigarette declined from 16% to 0% among nonsmokers in the intervention group and increased from 8% to 25% in the control group.[416] A version of this game was adapted to influence smoking-related attitudes and intentions among American Indian/Alaska Native youths, who indicated in focus groups that they wanted a website oriented toward their cultural images.[417] Another study examined online advertising for the evidence-based BecomeAnEX Internet cessation program to reach and engage Spanish-speaking Latino smokers. Although this study found that the online advertisements were effective and cost-efficient, the advertisements' message-framing and cultural-targeting efforts did not make a significant difference in terms of clicks, click-through rates, and registrants.[418]

Box 10.6: Web 2.0 and Message Tailoring

Web 2.0 applications can also point to new directions in message tailoring—that is, computer- or Web-based individualization of messages to correspond to the user's personal data. Evidence suggests that tailored messages in computer-driven applications can be effective for underserved populations.[481,482] As with message effects research, research on message tailoring has focused on individual psychological or cognitive factors, including health behavior, stages of change, risk factors, and information needs.[481] It is important to understand how these individual-level factors are moderated by racial/ethnic, social, and structural variables to influence message awareness, receptivity, and response. Lastly, the Internet makes it possible to target cessation interventions to many different demographic groups at low incremental cost.[483]

Use of Web 2.0 for Pro-Tobacco Communications and Marketing

The tobacco industry has turned to Web 2.0 for many of the same reasons that public health professionals have—to engage consumers and influence their attitudes, behaviors, and purchases.[384] Researchers have noted the potential impact of the increased interactivity available online, observing that "a viewer would probably spend far more time browsing and interacting with a pro-smoking website than viewing a static cigarette advertisement in a magazine."[419]

Tobacco imagery and other forms of advertising and promotion are common across a variety of Web 2.0 applications, which have been created and posted by both the tobacco industry and other often difficult-to-identify sources (see Box 10.7). A literature review of the effect of the Internet on teen and young adult tobacco use noted that most descriptive studies found that (1) pro-smoking Internet content was more prevalent than anti-smoking content, (2) most smoking content was viewed on what appeared to be user-generated Web pages as opposed to explicitly industry-generated sites, and (3) evidence on a relationship between exposure to smoking imagery online and tobacco use behavior was limited. Also, Internet content appeared to suggest that minors could easily obtain tobacco products online.[420]

Box 10.7: E-Cigarette Advertising on the Internet

Interest in electronic cigarettes (e-cigarettes) is driven at least in part by Web 2.0 platforms. Use of the term "electronic cigarettes" in Google searches increased by more than 5,000% between January 2007 and January 2010. Beyond typical Internet advertising and promotion (see example below), e-cigarette companies have used marketing strategies through which product users become distributors and earn profits by recruiting customers. Companies provide promotional materials, Web forums for distributors to share strategies to maximize online presence, and podcasts on search engine optimization for their websites. A study found that the vast majority of top Web search results for "electronic cigarette" are e-cigarette shops.[484]

E-cigarette virtual user communities may also be contributing to the increased interest in this product. E-cigarette users (who often refer to themselves as "vapers") and retailers indicate that such forums are invaluable for new users; communities formed online are often complemented by in-person communities.[485]

Website Promotion of Blu E-Cigarettes, October 2011

Source: Rutgers School of Public Health 2011.[486]

An analysis of data from the 2011 National Youth Tobacco Survey found that about 40% of middle school and high school students were exposed to tobacco advertising online, and that tobacco advertising seen online by high school students who had never smoked but were open to trying cigarettes increased from 26% in 2000 to 45% in 2011.[212] Another study found that racial/ethnic minority status and younger age were associated with receiving tobacco promotions via Facebook/MySpace and text message.[390] Overall prevalence of exposure to pro-tobacco advertisements online among middle and high school students increased from 22.3% to 43.0% between 2000 and 2012.[257] On occasion, tobacco products have been promoted on social media through celebrity endorsements, such as Snoop Dogg's promotion of Executive Branch cigars on Instagram.[391] The FTC issued a compulsory order in 2011 requiring tobacco companies to report social media expenditures for tobacco products since 2009.[228] Social media expenditures reported to the FTC show no spending on cigarette advertising through this medium.[227] Expenditures were reported for smokeless tobacco but were not published separately because only one tobacco company reported spending in this category.[228]

Tobacco industry websites can serve as a direct form of promotion and advertising and can include forums with product reviews and other commentary that can urge buyers to purchase certain brands or try certain tobacco products.[384] In 2014, the U.S. tobacco industry's expenditures for advertising on company websites was $16.6 million for cigarettes[227] and $6.4 million for smokeless tobacco.[228] Tobacco industry websites also encourage user-generated content and mimic social media sites in format and features, using online participation to generate offline engagement and purchases.[421] On their websites, tobacco companies have used other marketing strategies, such as "open-source marketing," in which companies engage with consumers online to design new tobacco product flavors and packages; this practice illustrates how Web 2.0 can blur the lines between market research and marketing.[422] Niche pro-tobacco websites and blogs are another source of online tobacco marketing.[384] The most far-reaching impact online, however, is probably achieved by tobacco brand and product promotions on widely accessed websites such as Facebook, Instagram, YouTube, and Twitter.[384,392,423] A study of a small, representative longitudinal panel of 200 young adults in Connecticut found that viewing social media depictions of tobacco use predicted future smoking, even after controlling for exposure to television and movie depictions of smoking.[424]

Several studies have also conducted content analyses of YouTube videos, which collectively have generated millions of pro-tobacco message viewings.[425–429] YouTube is intended as a forum to share consumer-generated content, but the authenticity of these tobacco-related videos has sometimes been questioned. A study of tobacco-related videos on YouTube found 200 "smoking-fetish" videos, which eroticize smoking.[429] Researchers have also found YouTube videos (n = 163) that depict tobacco brand images or a brand name, most of which (71%) could be characterized as "pro-tobacco" in tone.[426] In 2011, a study found 78 YouTube videos showing smokeless tobacco, and 74% of these portrayed smokeless tobacco in a positive light.[425] In 2012, a study found 56 YouTube videos about little cigars and cigarillos; of these, 43 were categorized as in favor of little cigars and cigarillos, 11 as neutral toward them, and only 2 as against use of little cigars and cigarillos.[392] A study of African American new smokers suggested that being exposed to Internet advertisements for tobacco was positively associated with experimental smoking.[210]

Eighty-eight percent of adolescents ages 8–18 play video games at least occasionally.[430] In interviews, teen and young adults who play video games recalled regularly seeing smoking imagery in games. Unlike movies, where the viewer watches characters smoking, video games provide opportunities for players to interact with tobacco; for example, a player's avatar may be given special advantages for

chewing tobacco or may sell tobacco for profit in the game.[431] An analysis of games listed in the Entertainment Software Rating Board's online database found that the prevalence of tobacco-related content increased from 0.8% in 2005 to 12.6% in 2011 for games rated as appropriate for young people age 10 and older, and from 1.0% in 1994 to 5.7% in 2011 for games rated appropriate for teens.[432] Additionally, adolescents recalled exposure to tobacco in games rated for their use, yet few of those recalled games (8%) had a descriptor warning of tobacco-related content.[433] The first systematic review of the literature demonstrated that tobacco imagery is present in video games, and notes that the relationship between video game playing and smoking needs further study.[433]

In addition to the Internet, YouTube, and video and online games, the tobacco industry and other pro-tobacco interests are exploring the use of mobile channels, which have demonstrated reach among low-SES and minority populations.[400] A 2012 study identified 107 pro-smoking applications for smartphones, include some with explicit images of cigarette brands. The authors concluded that tobacco products were being promoted via smartphone applications, a Web 2.0 channel with "global reach, a huge consumer base of various age groups, and underdeveloped regulation."[434,p.e4] Analyses have also found that most pro-smoking applications are assigned to entertainment and games categories, with some placed in categories directed specifically to children.[435]

Effectiveness of Web 2.0 Anti-Tobacco Communications and Marketing

Although Web 2.0 is increasingly being used by pro-tobacco and anti-tobacco interests, the empirical evidence for the efficacy of these approaches is just emerging. A cross-sectional analysis of quit-smoking messages on Twitter found that posted content was largely inconsistent with clinical guidelines, with less than 5% of posts recommending evidence-based approaches, and 48% of the messages were linked to commercial sites selling quit-smoking products. In addition, nearly half of the activated Twitter quit-smoking accounts (153) from 2007 were inactive by August 2010.[436] Another study found that websites focusing on smoking prevention were less likely to use evidence-based components compared with websites focusing on smoking cessation.[437] Research also indicates that cessation websites and social media sites are used as information portals in public health rather than as places to offer behavior-change strategies[437] and dynamically engage and interact with their intended audience.[438,439] In general, efforts by tobacco control advocates to effectively employ digital strategies have not taken full advantage of Web 2.0's unique characteristics—a high-level of interactivity that enables multidirectional communication and meaningful engagement.

Pro- and Anti-Tobacco Messaging: The Role of Interpersonal Communication

In addition to mass media, online and new media technologies, and advertising in the retail environment and elsewhere, individuals' communication ecologies also include interpersonal communication channels. Communication with friends, family, and others may impact tobacco use behaviors independently as well as in conjunction with other communication channels. For instance, interpersonal communication may moderate the impact of a mass media campaign.[440]

A number of studies have examined the impact of interpersonal communication in the context of smoking cessation campaigns, although these studies have not typically analyzed results for specific groups. Studies have found that ad-stimulated interpersonal pressure from family and friends is associated with increased recent quit attempts[441]; smokers with some intention to quit are more likely to share anti-smoking messages than those with little or no intention; and novelty and positivity of the message are associated with smokers' intention to share messages.[442] Peer support, particularly in the

context of smoking cessation websites, has been found to have a positive effect on cessation.[443] Beyond peer-to-peer support online, research on an in-person peer-to-peer tobacco education and advocacy program focused on helping smokers with mental illness found that 40% of participants reported seriously thinking of quitting in the next 30 days upon completion of the peer-to-peer session.[444]

Interpersonal communication is directly linked to health-enhancing behaviors in general, and can also mediate the influence of the multichannel media environment on health-enhancing behaviors, according to a study using data from the Annenberg National Health Communication Survey. This study, one of the few to examine findings by SES, found that the mediating role of interpersonal health communication was only significant for less-educated individuals, suggesting that interpersonal health communication may play a role in reducing TRHD.[440]

Other studies have found that the quality and frequency of communication on smoking, received by adolescents from parents, were associated with lower risk of adolescent smoking and were found to influence whether adolescents associate with friends who smoke.[445] Another investigation examined self-reported information regarding college students' social networks and found that "social network risk," a measure of close friends' alcohol use, increased the odds of using tobacco, especially among whites.[446]

Websites and online social networks are increasingly important channels through which interpersonal communication occurs. For example, an analysis of the Camel Snus website message board found that participants were using the space to share perceptions and experiences with the product and interact with each other.[447] This analysis further noted that, with increasing restrictions and decreasing social acceptance of smoking, online message boards may provide an outlet for interpersonal communication that tobacco users are unable to find elsewhere.

Further research is needed to determine differences in the role of interpersonal communication, both online and offline, across socioeconomic and racial/ethnic groups for pro- and anti-tobacco marketing.

Evidence Review: New Communications Technologies

Novel Web 2.0 technologies such as social networking websites and mobile applications have evolved over the past decade and could play a role in exacerbating or reducing TRHD. The evidence on Web 2.0 and tobacco communication and marketing suggests the following:

- Access to and use of Web 2.0 is increasing rapidly. Although the broadband access digital divide has important consequences, disparities in the ownership and use of mobile technologies such as smartphones are narrowing, which has important implications for the potential of different communication channels to reach low-SES and racial/ethnic minority populations, in terms of both access to information and the ability to process and act upon information.
- In general, Web 2.0 applications are used in anti-tobacco communications to amplify messages from traditional media sources and to reach and engage audiences in a new way. These Web 2.0 anti-tobacco efforts include online contests to engage youths in creating their own anti-tobacco messages; online petitions aimed at convincing celebrities or events to drop tobacco sponsorships; interactive websites, texting interventions, and mobile applications to assist with smoking cessation; and mobile games for cessation and prevention purposes. Very few studies examine the use of Web 2.0 for anti-tobacco efforts targeting specific populations; these initial studies indicate Web 2.0 platforms may be a promising way to communicate with racial/ethnic

minority populations. Additionally, the Internet makes it possible to tailor anti-tobacco communications to a variety of demographic groups at low incremental cost, suggesting that Web 2.0 platforms may help mitigate knowledge gaps and communication inequalities.

- Web 2.0 applications are used by pro-tobacco interests to engage consumers and influence their attitudes, behaviors, and purchases. Examples of these uses include components of industry-owned brand websites, other pro-tobacco websites and blogs, pro-tobacco content on social networking sites such as Facebook and YouTube, tobacco imagery in games and other online content, and pro-smoking mobile applications. Media use patterns suggest that Internet tobacco advertisements may disproportionately affect racial/ethnic minority youth, and the ability to tailor communications enhances the tobacco industry's ability to target minorities as well. Further research is needed to determine the degree to which Web 2.0 pro-tobacco communications may exacerbate TRHD.
- Although Web 2.0 is increasingly used by both pro-tobacco and anti-tobacco interests, the empirical evidence for the impact of these approaches is just emerging. Efforts by tobacco control advocates to employ digital strategies have thus far been limited in scope.
- Studies described in this section have indicated that Web 2.0 applications have immense potential to facilitate changes in tobacco-related knowledge, attitudes, beliefs, and behaviors. Adoption of best-practice guidelines and ongoing research would help to maximize our ability to use this platform to address TRHD.

Research has shown that health behavior change is often best achieved using multimodal interventions and a combination of different communication channels in conjunction with environmental changes.[448–450] Thus, Web 2.0 should not be viewed as a replacement for conventional mass media interventions or other public health initiatives. Rather, Web 2.0 is an additional tool to enhance public health efforts when public health efforts capitalize on Web 2.0's unique strengths, conform to best-practice evidence, and are strategically integrated with other intervention products and services.

Chapter Summary

Several public health theories and approaches postulate that health is shaped by a wide range of determinants, including SES, race/ethnicity, gender, and geography; the social and physical quality of neighborhoods and workplaces; and access to resources such as healthy food and medical care. When examined through this lens, evaluating the impact of pro-tobacco and anti-tobacco communications requires moving beyond simply assessing whether communication efforts change the behavior of the population at large. It becomes necessary to account for the fact that differences exist among groups in their ability to access, process, and act upon different types of information, and that pro-tobacco and anti-tobacco communications target different groups in different ways.

A review of the anti-tobacco communication literature as it pertains to youth indicates that anti-tobacco TV campaigns can effectively reduce smoking prevalence among the general population, but there is less evidence about their effectiveness among different population groups. For youth, communication inequalities may contribute to differences in awareness of tobacco prevention campaigns across groups but may not affect receptivity to campaigns or the impact of campaign messaging on attitudes, beliefs, and behaviors. Research from several campaigns suggests that geography may contribute to a knowledge gap between urban/suburban versus rural youth regarding tobacco-related information unless

supplemental efforts are undertaken to ensure that campaign messages reach youth in rural areas. However, because of the rapid changes in the media landscape since these studies were conducted in the early 2000s, media access and thus campaign awareness by geography may be more similar than in the past. Low-SES youth and racial/ethnic minorities are receptive to campaign messages, and campaigns can influence knowledge, attitudes, and beliefs among diverse groups. However, pathways by which campaigns influence attitudes and beliefs may vary, suggesting that differences in message processing should be considered in campaign development. Further, campaigns with the strongest short- and long-term behavioral effects among low-SES and racially diverse youth were often complemented by community, school, or state programs that supplemented campaign messaging with other tobacco control programming.

A review of the anti-tobacco communication literature as it pertains to adults indicates that campaigns with (1) high exposure, targeted media efforts; (2) additional tobacco-related program components; or (3) language-appropriate and/or culturally tailored messaging can be effective and may reduce potential communication inequalities that lead to gaps in tobacco-related knowledge. Additionally, campaigns with graphic and emotionally arousing messages can also stimulate quitting among racial/ethnic minorities and low-SES groups. By ensuring that additional supportive resources are available, such as quitline support, free NRT, and other community-based programs and policies, campaign effectiveness can be improved among diverse populations. These findings support the concept of fundamental causes, in that disadvantaged populations may benefit less from health education campaigns due to a lack of resources to support behavior change. Providing additional community, school, or other tobacco-related services may be especially important for groups with limited resources to help ensure that campaigns do not inadvertently contribute to disparities.

A review of the pro-tobacco communication literature finds strong evidence that pro-tobacco imagery and marketing influence tobacco use and related attitudes, but evidence on how these effects differ by race/ethnicity or SES is limited. As posited by the SIM, differing levels of exposure to television and movies, as well as differing levels of identification with characters who smoke, may contribute to variation in the effects of television and movies on youth smoking based on race/ethnicity. Such racial/ethnic differences are also seen in terms of exposure to tobacco advertisements in magazines, perhaps driven by the higher density of tobacco advertising in magazines with high African American readership or by the greater amount of time African Americans spend reading magazines. The tobacco industry (1) uses event sponsorship, audience segmentation, and product development to effectively reach particular groups, and (2) promotes tobacco products at the point of sale more heavily in low-income and minority communities. These findings are in line with theories that hold that the unequal distribution of resources, including political and financial power to oppose tobacco industry interests, can cause disadvantaged groups to experience disproportionately high risks. In addition, evidence indicates that the tobacco industry's use of price discounts as a promotional strategy disproportionately affects low-income and racial/ethnic minority smokers.

News media coverage of tobacco has been both anti-tobacco and pro-tobacco in nature, with parties from both sides of the issue attempting to influence news coverage. Some evidence suggests that greater coverage of tobacco-related health problems and anti-tobacco campaigns can positively affect tobacco-related attitudes and behaviors, but fundamental cause theory and the knowledge gap hypothesis suggest that such communication efforts may disproportionately benefit those of higher SES. Although there is some anti-tobacco news coverage, health journalism overall often fails to underscore the health reasons for tobacco control efforts or to highlight the need for a preventive approach. On the pro-tobacco

communications side of news coverage, news stories often feature claims made by the tobacco industry or by industry-supported consultants. Though there is little if any evidence on how use of such information or sources impacts disadvantaged groups, it is possible these groups are less able to distinguish between objective news coverage and claims made by the tobacco industry.

Both pro- and anti-tobacco communications have been drastically altered in recent decades by the rise of online and digital technologies (i.e., Web 2.0). Minority and low-SES groups continue to experience challenges in accessing and using the Internet, but use of mobile devices among these groups is increasing. This increasing access has important implications for the potential of different Web 2.0 communication channels to reach disadvantaged populations. Differences in literacy and numeracy skills may undermine the impact of Web 2.0 anti-tobacco communications among racial/ethnic minority and low-SES groups, but the ability to easily tailor interventions and the reach of mobile applications may enhance the impact of these efforts. In addition, some evidence indicates that Web 2.0 platforms can be a promising way to both recruit and communicate with minority populations.

Pro-tobacco imagery and promotion are common on Web 2.0 platforms, ranging from pro-tobacco websites, blogs, and social networking content to tobacco imagery in games and other online content as well as pro-smoking mobile applications. Although research on the topic is limited, some data indicate that racial/ethnic minorities receive more tobacco promotions through these means, again illustrating how a variety of factors contribute to disproportionately high risks for certain groups. Web 2.0 platforms' enhanced ability to tailor communications enables the industry to fine-tune its targeting of racial/ethnic minorities and low-SES groups; such institutional-level inequities also have the potential to worsen TRHD.

As tobacco use becomes increasingly concentrated among people who have the least resources, our ability to communicate effectively with groups that bear a disproportionate burden of the tobacco epidemic becomes ever more important.

Research Needs

When examined in light of communication and health inequality frameworks, pro-tobacco and anti-tobacco communication efforts and their impact are characterized by key gaps in the literature. Social epidemiology and media studies theories inform communication inequalities and suggest a number of pathways through which pro- and anti-tobacco communication may disproportionately impact racial/ethnic minorities and low-SES groups—empirical tests of these pathways could identify the degree to which communication inequalities contribute to TRHD as well as to identify potential points of intervention.

Coordinated efforts are needed to develop surveillance systems for tracking pro-tobacco and anti-tobacco advertising and promotion over time using studies with sample sizes adequate to test effects among different population groups. Multiple forms of surveillance are critical to track the rapid changes in the tobacco marketplace that are expected over the coming years, including the introduction of new tobacco products. Communications surveillance systems should also be linked to systems for monitoring evolving policies related to tobacco marketing in order to adequately evaluate these policies.

Pro-tobacco and anti-tobacco marketing exposure and industry targeting of groups experiencing TRHD should be monitored. Studies have shown that the tobacco industry drives consumer demand by

selectively marketing particular types of products, such as mentholated brands, in low-income, minority communities, where menthol is the brand of choice.[234,451,452] It is important to understand if and how new restrictions affect industry strategies regarding new product marketing and how such marketing, in turn, shapes the perceptions and purchasing behaviors of disparate groups. It is also important that populations be involved in monitoring marketing practices in their communities as regulations are implemented and new products are introduced into the marketplace.

Improving our understanding of the relationship between tobacco industry advertising and promotion and TRHD requires further research in several key areas, including: the prevalence and types of tobacco industry marketing; levels of exposure to tobacco marketing across demographic groups; the impact of marketing on tobacco use attitudes, beliefs, and behaviors among racial/ethnic minority and low-SES groups across the life course; and the ultimate impact of tobacco marketing on TRHD. Although there are many examples of tobacco industry targeting of specific demographic groups through advertising, tobacco packaging, and other avenues,[224,348,453] there are no systematic analyses that quantify or assess the impact of these strategies in a comprehensive way or among various subpopulations. Few studies include specific group analyses, and among those that do, small sample sizes and lack of consistency in study design, analytic approaches, and outcomes make it difficult to draw overarching conclusions. Further use of methods such as ecological momentary assessment[454] and objective (versus self-reported) measurement of exposure would be informative.

The anti-tobacco campaign literature is characterized by heterogeneity in study designs and inconsistency in outcomes and analytic approaches. Heterogeneity within groups (e.g., nativity status, level of acculturation among Hispanic and Asian populations) and multiple levels of disadvantage add to the complexity of interoperating differences in outcomes.[2] It is important for campaign developers and evaluators to specify the mechanisms by which a campaign is expected to affect behavior and to consider all the points along the communication continuum where variations can arise for different groups across the life course. An additional challenge is the use of study designs that do not make it possible to separate the effects of media campaigns from other community- or state-based interventions. Collection of larger samples will be needed for specific group analysis. New methods of analyses combining small samples across studies to understand intervention effects among different groups may be one option for utilizing the available data.[455,456]

Experimental or quasi-experimental research is also needed to compare different targeting or segmentation strategies for specific populations. More research is needed that (1) uses explicit, controlled comparisons of different campaign types (e.g., general versus segmented audience) among specific populations, or (2) tests specific comparisons in real-world population-based campaigns, such as examining how varying campaign strategies reduce disparities among specific populations over time.[457]

Moreover, further research is needed to examine the extent to which news media coverage influences tobacco-related knowledge, attitudes, and behaviors. Community-based and public health groups have fewer resources than the tobacco industry.[375] This resource inequality can influence how public health issues such as tobacco use are defined and what solutions are recommended. Strategies for training community-based groups to become effective suppliers of information to the media should be investigated.[458] Such strategies could have long-term impact on news media coverage of tobacco and TRHD and thus on how the public perceives the problem.

Studies described in this chapter indicate that Web 2.0 applications have immense potential to facilitate changes in tobacco-related knowledge, attitudes, beliefs, and behaviors, but the use of these tools would benefit from a careful adoption of best-practice guidelines and ongoing research to understand how to use this increasingly important platform to address TRHD. Most of the existing evidence on the impact of Web 2.0 tobacco control interventions focuses on tobacco cessation which, though important, is only one component of a comprehensive tobacco control effort. Overall, more research, experimentation, and evaluation is needed to determine the best use of Web 2.0 applications for tobacco control across diverse population groups. Further research is needed to understand the degree to which Web 2.0 anti-tobacco efforts might create, exacerbate, or decrease TRHD.

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Section IV
Societal Level Influences on Tobacco Use

Chapter 11
Federal, State, and Local Tobacco Control Policy and Tobacco-Related Health Disparities

Contents

Figures and Tables

Introduction

A variety of tobacco control policies and programs at the federal, state, and local levels have strong potential to address many current tobacco-related health disparities (TRHD). This chapter considers the scope of comprehensive tobacco control policies and programs at the federal, state, and local levels and their differential impact on various populations. The impact of specific tobacco control policies such as cigarette taxation and price, smoke-free policies, efforts to reduce youth access to tobacco, and policies to increase access to smoking cessation services are also explored.

Comprehensive Tobacco Control Programs in States

In 1989 California became the first state to create a comprehensive statewide tobacco control program. In 1988 voters in California passed Proposition 99, which raised the cigarette excise tax by 25 cents per pack.[1] A portion of the revenue generated from the tax increase was allocated to anti-tobacco initiatives, including anti-tobacco media campaigns, community education programs, school-based education programs, as well as tobacco control research, surveillance, and evaluation activities. Massachusetts followed suit in 1992 by passing Proposition 1, which raised the cigarette tax by 25 cents per pack.[2] Tax revenues in Massachusetts were used to fund initiatives, including a large anti-smoking media campaign, school and community anti-smoking education programs, and increased enforcement of local tobacco ordinances. Subsequent to these two programs, initiatives to raise tobacco taxes were successful in several other states, and a portion of the revenues generated from these taxes were used to fund large-scale tobacco control programs.[3]

Tobacco control programs in some states are funded by those states' individual settlements with cigarette manufacturers or through the Master Settlement Agreement (MSA) reached on November 23, 1998, with the major tobacco companies.[3–5] Under the MSA, 46 states, five U.S. territories, and the District of Columbia settled their lawsuits against the nation's major tobacco companies, giving up certain future legal claims, and the tobacco companies agreed to make annual payments to the states to compensate them for health care costs for tobacco-related illness.[4,5] (Four states reached earlier individual settlements with the tobacco companies.) The total amount of the annual payments required of the tobacco companies is estimated at $246 billion over the first 25 years.[6] To finance the settlement, the tobacco companies raised the wholesale price of cigarettes by 45 cents/pack.[7] More information on the Master Settlement Agreement is provided below.

In its *Best Practices for Comprehensive Tobacco Control Programs—2014*, the Centers for Disease Control and Prevention (CDC)[8] recommended that states annually spend a combined $3.3 billion, or $10.53 per capita, to maintain comprehensive tobacco control programs. However, states have typically appropriated significantly less than the CDC's recommended amounts. For example, in fiscal year (FY) 2017, it is estimated that the states and the District of Columbia combined will collect $26.6 billion from the tobacco settlement and tobacco taxes, but will spend only $494 million (1.8%) on tobacco control programs.[6] Table 11.1 shows state-level FY 2017 tobacco control appropriations in relation to CDC-recommended expenditures.[6,8] In comparison, total marketing expenditures for the major U.S. cigarette companies in 2014 were $8.49 billion.[9]

Table 11.1 FY 2017 Funding for State Tobacco Prevention Programs

State	FY 2017 Current Annual Funding ($M)	CDC Annual Recommendation* ($M)	FY 2017 Percentage of CDC's Recommendation	Current Rank (Based on Percentage of CDC's Recommendation)
Alabama	1.5	55.9	2.7	42
Alaska	9.5	10.2	93.0	2
Arizona	18.4	64.4	28.6	16
Arkansas	9.0	36.7	24.5	19
California	75.7	347.9	21.8	21
Colorado	23.2	52.9	43.8	10
Connecticut	0.0	32.0	0.0	50
Delaware	6.4	13.0	48.9	7
District of Columbia	1.0	10.7	9.3	31
Florida	67.8	194.2	34.9	14
Georgia	1.8	106.0	1.7	43
Hawaii	6.8	13.7	49.3	5
Idaho	2.9	15.6	18.4	23
Illinois	9.1	136.7	6.7	35
Indiana	5.9	73.5	8.0	34
Iowa	5.2	30.1	17.4	25
Kansas	0.8	27.9	3.0	40
Kentucky	2.4	56.4	4.2	37
Louisiana	7.0	59.6	11.7	27
Maine	7.8	15.9	49.1	6
Maryland	10.6	48.0	22.0	20
Massachusetts	3.9	66.9	5.8	36
Michigan	1.6	110.6	1.4	45
Minnesota	22.0	52.9	41.7	11
Mississippi	10.7	36.5	29.4	15
Missouri	0.1	72.9	0.1	49
Montana	6.4	14.6	44.1	8
Nebraska	2.6	20.8	12.4	26
Nevada	1.0	30.0	3.3	39
New Hampshire	0.1	16.5	0.8	48
New Jersey	0.0	103.3	0.0	51
New Mexico	5.7	22.8	24.9	18
New York	39.3	203.0	19.4	22

Table 11.1 continued

State	FY 2017 Current Annual Funding ($M)	CDC Annual Recommendation* ($M)	FY 2017 Percentage of CDC's Recommendation	Current Rank (Based on Percentage of CDC's Recommendation)
North Carolina	1.1	99.3	1.1	46
North Dakota	9.9	9.8	100.9	1
Ohio	13.5	132.0	10.3	28
Oklahoma	23.5	42.3	55.6	3
Oregon	9.8	39.3	25.0	17
Pennsylvania	13.9	140.0	9.9	29
Rhode Island	0.4	12.8	2.9	41
South Carolina	5.0	51.0	9.8	30
South Dakota	4.5	11.7	38.5	13
Tennessee	1.1	75.6	1.5	44
Texas	10.2	264.1	3.9	38
Utah	7.5	19.3	38.9	12
Vermont	3.4	8.4	40.2	9
Virginia	8.2	91.6	9.1	33
Washington	2.3	63.6	3.6	47
West Virginia	3.0	27.4	11.1	24
Wisconsin	5.3	57.5	9.2	32
Wyoming	4.2	8.5	49.4	4

*CDC annual recommendations are based on the CDC's *Best Practices for Comprehensive Tobacco Control Programs 2014*.[8]
Source: Campaign for Tobacco-Free Kids 2016.[6]

A combination of federal and private funding has been used to support multistate efforts to reduce tobacco use. The first major multistate effort was the American Stop-Smoking Intervention Study for Cancer Prevention (ASSIST), a partnership between the National Cancer Institute (NCI), the American Cancer Society (ACS), and 17 state health departments, conducted between 1991 and 1998. ASSIST focused on tobacco control policies in four areas: eliminating SHS exposure, increasing the price of tobacco products, restricting tobacco advertising and promotions, and reducing youth access to tobacco products.[10] During this same period, the CDC funded the remaining states (excluding California) and the District of Columbia under its Initiatives to Mobilize for the Prevention and Control of Tobacco Use (IMPACT) program.[11] In 1999 these two programs were replaced by the CDC-funded National Tobacco Control Program, which currently (2017) supports tobacco control efforts in all 50 states, the 5 U.S. territories, and the District of Columbia.[12] In addition, from 1993 to 2004 the Robert Wood Johnson Foundation's SmokeLess States program, administered by the American Medical Association, funded coalitions to strengthen tobacco control policies in nearly all states.[13]

Impact on Tobacco Use

Evaluations of major individual state programs provide compelling evidence that these programs are associated with reduced tobacco use.[10,14–18] In California, for example, as a result of the California Tobacco Control Program (CTCP) adult smoking prevalence declined by 51.1% between 1988 (the year before the program began) and 2014, from 23.7% to 11.6%. This represents approximately 3.3 million fewer adult smokers in the state. The decline in prevalence was most pronounced during the early years of the program.[19]

After adopting large-scale, comprehensive state tobacco control programs, Arizona, Florida, Massachusetts, and Oregon saw large reductions in smoking.[20–28] The Community Preventive Services Task Force[18] found that states with a comprehensive tobacco control program saw a median additional annual reduction in adult tobacco use prevalence of 45% (range: 18% to 89% reduction) compared to the rest of the country. Similarly, U.S. states or localities with comprehensive tobacco control programs had greater reductions in smoking prevalence among young people than states or localities without such programs.[18]

The health benefits of reduced tobacco use after the implementation of comprehensive tobacco control programs are increasingly apparent. For example, in California the estimated rate of death caused by heart disease and lung cancer has fallen sharply.[28–31] A study by Pierce and colleagues[31] concluded that, as a direct result of the CTCP, California will have faster declines in lung cancer than the rest of the nation for the next several decades. In addition, several studies have examined the economic effects of the CTCP. Lightwood and Glantz[32] estimated that this program saved $134 billion in health care expenditures between 1989 and 2008. These authors found that reductions of one percentage point in current smoking prevalence and a one-pack per year reduction in cigarette consumption per smoker in California are associated with $35.4 (standard error [SE] $9.85) and $3.14 (SE $0.786) reductions in per capita health care expenditure, respectively (2010 dollars). A study by Max and colleagues[33] found that if tobacco control funding in California were increased from the 2011 level to the CDC-recommended level, health care savings would reach $4.7 billion for the years 2012–2016.

A few national-level analyses have examined the impact of state tobacco control programs on cigarette smoking. An early analysis that compared per capita cigarette sales in ASSIST states to sales in non-ASSIST states found that sales declined 28% faster in the ASSIST states in the first several years after the program began (sales trends in the two groups of states were similar in the years before the ASSIST program).[34]

Farrelly and colleagues examined the effect of state-level per capita tobacco control expenditures on state-level per capita cigarette sales for the period from 1981 through 2000.[35] After controlling for potential confounding covariates, they concluded that investments in tobacco control programs had reduced overall cigarette consumption. Their findings suggested that if states had funded tobacco control efforts at the minimum CDC-recommended levels, the rate of decline in cigarette consumption would have doubled from 1994 through 2000.

A 2008 study by Farrelly and colleagues employed survey data from 1985 to 2003 to examine the impact of tobacco control expenditures on adult smoking prevalence rates.[36] They found that increases in state per capita tobacco control spending were associated with significant declines in smoking prevalence. Tobacco control expenditures were found to be more effective in reducing smoking rates among people age 25 and older than among those ages 18–24. The authors concluded that if states had

spent the minimum CDC-recommended level of expenditures on tobacco control efforts, there would have been 2.2 million fewer adult smokers than observed between 1985 and 2003.

Impact on TRHD

Evaluations of state-specific tobacco control programs show that comprehensive tobacco control programs have substantial effects on smoking prevalence rates among youths and young adults. For example, a study by Pierce and colleagues[37] found it likely that the comprehensive CTCP kept new adolescent cohorts from experimenting with cigarettes, and this decline in experimentation translated into a decline in California young adult smoking prevalence that was not observed in the rest of the United States. Another report published by the CTCP found that smoking prevalence among California adolescents was similar to that in the rest of the nation at the onset of the CTCP, but by 2005 California adolescents smoked 50% less than adolescents in the rest of the country.[38]

Similarly, in Massachusetts, current smoking among high school students declined by 27% between 1995 and 2001, whereas the nationwide rate dropped by only 18%.[39] During this time, the Massachusetts Tobacco Control Program was extremely active in tobacco control efforts. A New York State Department of Health report documenting trends in youth smoking following the implementation of New York's tobacco control program concluded that current smoking among middle school students declined by 68.6% (from 10.2% to 3.2%) between 2000 and 2010. In addition, current smoking among high school students declined by 53.5% (from 27.1% to 12.6%), and current smoking by young adults ages 18–24 declined by 30.0% (from 33.0% to 23.1%) between 2000 and 2009.[40]

A nationally representative multivariate econometric study conducted by Tauras and colleagues[41] examined the impact of state-level tobacco control expenditures on youth smoking prevalence and on smoking intensity, as measured by average number of cigarettes smoked. This study used data on 8th-, 10th-, and 12th-grade students collected as part of the Monitoring the Future (MTF) surveys conducted from 1991 through 2000. While controlling for cigarette prices, smoke-free laws, youth access laws, and demographic and socioeconomic factors, the researchers found a strong inverse relationship between per capita tobacco control funding at the state level and smoking prevalence rates among adolescents.[41] They also found that per capita tobacco control expenditures had a strong negative impact on the average number of cigarettes smoked by adolescent smokers. The researchers estimated that adolescent smoking prevalence would have been 3.3% to 13.5% lower than that observed in the 1990s if states had funded their tobacco control efforts at the minimum CDC-recommended levels.

Many evaluations have found that state-specific comprehensive tobacco control programs have had significant effects on adult smoking prevalence rates across various socioeconomic status (SES) and racial/ethnic subgroups. For example, in California all racial/ethnic groups experienced large declines in smoking prevalence rates (>25%) between 1990 and 2005, except Asian American/Pacific Islander women, whose smoking prevalence declined by only 11%.[38]

Studies show that in California, as elsewhere in the United States, smoking is inversely related to income and educational attainment. Smoking prevalence among adults in California declined between 1990 and 2005 across all age groups, racial/ethnic groups, education levels, and for both sexes. The largest declines in prevalence were observed among young adults, college graduates, individuals with the highest incomes, and non-Hispanic whites. In 2005, smoking prevalence among college graduates was less than half the prevalence among people who did not attend college: Only 7.4% of college

graduates in California were current smokers in 2005, the lowest level reported for any educational group.[38] Between 1990 and 2005, smoking prevalence among college graduates declined by 40%, a greater decrease in smoking prevalence than was found in any other educational group; this reduction was consistent for both genders, with women's prevalence declining by 42.9% and men's prevalence declining by 37.3%.[38]

Given the strong association between education and income, people with higher incomes are expected to have lower smoking prevalence rates. Indeed, in 2005, California households that reported annual incomes greater than $50,000 had the lowest smoking prevalence (14.4% for $50,000–$75,000, and 11.9% for >$75,000), and those with the lowest incomes had the highest smoking prevalence (20.7% for <$10,000, and 19.3% for $10,000–$20,000).[38] Individuals with the highest annual incomes also had the largest declines in smoking prevalence between 1990 and 2005—a 23.8% decline for people with incomes of $50,000–$75,000 (from 18.9% in 1990 to 14.4% in 2005), and a 33.2% decline for those with incomes over $75,000 (from 16.9% in 1990 to 11.3% in 2005); smoking prevalence declined by 17.6% for people with incomes under $10,000 (from 25.1% in 1990 to 20.7% in 2005), and 12.6% for those with incomes between $10,000 and $20,000 (from 22.1% in 1990 to 19.3% in 2005).[38]

Similar patterns can be found in other states and localities after the implementation of comprehensive tobacco control programs. For example, Frieden and colleagues[42] examined changes in smoking prevalence in New York City one year after the 2002 implementation of a comprehensive tobacco control program, which included increased cigarette excise taxes, enhanced smoke-free policies, increased cessation services, and public education efforts. This study found that smoking declined among all age groups, race/ethnicities, and education levels, among both men and women, and among both native- and foreign-born populations. However, declines in prevalence were larger among young people, women, people in both the lowest and highest income groups, those with higher educational levels, and heavy smokers. Almost half (45.3%) of all smokers reported that the tax increase influenced their motivation to quit smoking.[42]

In 2006, the New York City Department of Health and Mental Hygiene implemented an extensive television-based anti-tobacco media campaign. At the same time, the New York State Department of Health aired a statewide anti-tobacco media campaign. A CDC report found that overall, the prevalence of smoking among New York City residents decreased from 18.9% in 2005 to 17.5% in 2006.[43] While the overall change in prevalence was not statistically significant, the decline was significant among men and among Hispanics. The CDC report also found significant decreases in smoking prevalence between 2002 and 2006 for many population groups in New York City, including young adults (34.9% decline; from 23.8% in 2002 to 15.5% in 2006), Asian Americans/Pacific Islanders (30.1% decline; from 15.3% in 2002 to 10.7% in 2006), women (22.7% decline; from 19.8% in 2002 to 15.3% in 2006), college graduates (20.7% decline; from 16.4% in 2002 to 13.0% in 2006), individuals with some college but no degree (20.6% decline; from 24.3% in 2002 to 19.3% in 2006), Hispanics (20.5% decline; from 21.5% in 2002 to 17.1% in 2006), non-Hispanic whites (17.2% decline; from 23.9% in 2002 to 19.8% in 2006), non-Hispanic blacks (14.9% decline; from 20.8% in 2002 to 17.7% in 2006), and men (15% decline; from 23.4% in 2002 to 19.9% in 2006). Smoking prevalence among New York City residents with only a high school education declined by 10% (from 23.9% in 2002 to 21.5% in 2006), and smoking prevalence among people who did not graduate from high school declined by 6.1% (from 24.5% in 2002 to 23.0% in 2006), although neither decline was statistically significant.

Levy and colleagues[44] examined the effects of cigarette prices and comprehensive tobacco control programs with significant media campaigns on smoking among women of low SES, as defined by educational attainment. Using four waves of the Tobacco Use Supplement to the Current Population Survey (TUS-CPS) between 1992 and 2002, the authors found women in the low education group (less than a high school diploma) were particularly responsive to price and to anti-smoking media campaigns; state media campaigns were associated with a 14% lower likelihood of being a current smoker among women in the low education group, compared to 11% for women in the medium education group (high school degree or GED through bachelor's degree); no effect of media campaigns was seen among high-education women (graduate-level education).

Master Settlement Agreement

As noted above, in November 1998, the Attorneys General of 46 states, 5 U.S. territories, and the District of Columbia entered into the Master Settlement Agreement with the nation's five major tobacco companies. In addition to financial compensation, the MSA imposed restrictions on participating manufacturers' marketing practices, including: (1) forbidding direct or indirect tobacco marketing to youth; (2) prohibiting tobacco advertisements on public transit and on billboards; (3) prohibiting the use of cartoon characters in cigarette advertising, promotion, and packaging; (4) eliminating paid tobacco product placement in media outlets; (5) restricting tobacco company sponsorship of sports, arts, and cultural events; and (6) restricting free samples to adult-only facilities. Tobacco companies agreed to finance the creation of the American Legacy Foundation (renamed the Truth Initiative in 2015), a national nonprofit entity focused on reducing death and disease caused by tobacco use. The MSA also required companies to make available online millions of previously internal company documents (initially made public as a result of Minnesota's 1998 settlement with major cigarette manufacturers) and to disband industry-funded research groups that misled the public about the harms associated with tobacco use.[45]

A number of studies show that the MSA and the four separate state settlements were associated with a significant decrease in smoking, largely due to the accompanying increase in cigarette prices. For example, Sloan and Trogdon[46] used national data from the Behavioral Risk Factor Surveillance System (BRFSS) and estimated that by 2002, the settlements had decreased smoking among young people ages 18–20 by 3.5%, and among people age 21 and older by 1–2%. Another study found that the California state tax increase plus the price increase resulting from the MSA reduced cigarette consumption, although this study could not distinguish the separate effects of the MSA price increase.[7]

A number of observers have expressed disappointment in the overall effect of the MSA.[47–49] According to Jones and Silvestri, "the MSA has not resulted in a clear and straightforward intensification of state tobacco control efforts. . . . MSA resources have been significantly diverted from tobacco control and treatment into other state policy activities."[50,p.697] Links between the MSA and changes in TRHD are possible but have not been examined. For example, MSA advertising restrictions may have reduced youth exposure to print tobacco advertisements,[51] but this may have been offset by shifts of promotional spending to point-of-sale locations.[52] To the extent that the concentration of tobacco retail outlets with point-of-sale tobacco advertising is higher in low-SES than in high-SES communities, MSA advertising restrictions may have inadvertently led to increased tobacco promotion in low-income neighborhoods and exacerbated TRHD.[53–55]

Federal Tobacco Control Policy

Food and Drug Administration Regulation of Tobacco

On June 22, 2009, the Family Smoking Prevention and Tobacco Control Act (Tobacco Control Act)[56] was signed into law, granting the Food and Drug Administration (FDA) the authority to regulate tobacco products "for the protection of the public health" and creating the Center for Tobacco Products (CTP) within FDA.[56] One purpose of the Tobacco Control Act is to ensure that FDA "has the authority to address issues of particular concern to public health officials, especially the use of tobacco by young people and dependence on tobacco."[57] The Tobacco Control Act allows FDA to adopt tobacco product standards if appropriate for the protection of the public health, requiring the agency to consider scientific evidence concerning:

> "(i) the risks and benefits to the population as a whole, including users and nonusers of tobacco products; (ii) the increased or decreased likelihood that existing users of tobacco products will stop using such products; and (iii) the increased or decreased likelihood that those who do not use tobacco products will start using such products."[58]

The Tobacco Control Act also established a 12-member advisory committee, known as the Tobacco Products Scientific Advisory Committee (TPSAC).[59]

The Tobacco Control Act has the potential to reduce TRHD by decreasing the toxicity, addiction potential, and/or attractiveness of products favored by specific population groups or by affecting the production and marketing of tobacco products targeted toward vulnerable populations.[60] In particular, the Tobacco Control Act provides FDA with the regulatory authority to reduce tobacco's impact on different populations via several mechanisms, including (1) disclosure and communication of harmful and potentially harmful constituents of tobacco products; (2) required graphic health warnings on cigarette packaging and advertising and larger text warnings for smokeless tobacco packaging and advertisements (FDA is also authorized to require larger health warnings on other tobacco products); (3) premarket evaluation of new tobacco products; (4) provisions restricting the marketing and sales of tobacco products; and (5) promulgation of tobacco product standards, including the authority to ban menthol as a characterizing flavor in tobacco products.

In general, the Tobacco Control Act does not preempt local jurisdictions' additional or more stringent regulations related to tobacco products. The statute does preempt state and local laws in several discrete topic areas reserved for federal regulation (e.g., product standards and premarket review). However, as described further below, it explicitly preserves the authority of local jurisdictions to establish requirements in a range of other areas (e.g., sales, distribution, and advertising of tobacco products) that are "in addition to, or more stringent than" the requirements of the Tobacco Control Act or regulations promulgated under its authority. Specific provisions of the Tobacco Control Act and their potential to reduce TRHD are discussed below.

Disclosure of Harmful and Potentially Harmful Constituents in Tobacco Products

The Tobacco Control Act requires tobacco manufacturers and importers to report the levels of harmful and potentially harmful constituents (HPHCs)—chemicals or chemical compounds in tobacco products or tobacco smoke that cause or could cause harm to smokers or nonsmokers—found in their tobacco products and in tobacco smoke. FDA must publish HPHC quantities by brand and sub-brand of tobacco product in a way that people find understandable and not misleading.[61]

The Tobacco Control Act enables FDA to request documents concerning research activities related to health, behavioral, or physiologic effects of tobacco products or their ingredients, components, or additives. FDA can also request documents on marketing research or marketing practices and the effectiveness of such practices.[56] Scientific analysis of tobacco industry documents have produced findings relevant to TRHD, including evidence that (1) the companies targeted marketing to African American smokers and other groups, (2) they used the civil rights movement to promote menthol cigarettes to African Americans, and (3) they "manipulate menthol content in cigarettes"[62,p.ii20] to make them more appealing, especially to young people, and easier to smoke.[62–66]

Graphic Warning Labels on Cigarettes

The Tobacco Control Act mandates the inclusion of graphic health warning labels covering 50% of the front and back of the cigarette pack, and warnings covering 20% of advertisements. Additionally, graphic warning labels on smokeless tobacco products must cover 30% of the front and back of the packaging and 20% of advertisements. In August 2012, the U.S. Court of Appeals for the D.C. Circuit upheld a lower court decision that the particular graphic warnings adopted by FDA violated the First Amendment.[67] The court remanded the matter to the agency, and FDA has undertaken research to support a new rulemaking.[68] A more recent lawsuit was filed by public health organizations challenging the legality of the time it is taking FDA to promulgate this new rule.[69] As of July 2017, this case is still in litigation.

Abundant evidence demonstrates that graphic health warning labels—typically the most visible health information presented to smokers—are a powerful informational intervention. For example, NCI Tobacco Control Monograph 21, *The Economics of Tobacco and Tobacco Control*, concluded that "large pictorial health warning labels on tobacco packages are effective in increasing smokers' knowledge, stimulating their interest in quitting, and reducing smoking prevalence. These warnings may be an especially effective tool to inform children and youth and low literacy populations about the health consequences of smoking."[70,p.305] Research suggests that the effectiveness of graphic warning labels is mediated by certain contexts, such as the tobacco control environment and social norms surrounding tobacco use.[71] Some messages can be more effective with some groups than with others, supporting the rotation of graphic warning labels to effectively communicate with multiple population groups.[71] Research conducted in Canada finds that graphic warning labels decrease the odds of being a smoker and increase the odds of making a quit attempt.[72]

Graphic warning labels also have strong potential to decrease youth smoking initiation.[71] For example, research conducted in Australia suggests that graphic warnings increase youths' cognitive processing of anti-smoking messages, decrease intention to smoke among youths who have not initiated smoking, and increase thoughts about cessation among smoking youths.[73] Similarly, American youths who see pictorial warning labels are more likely to notice and talk about the labels, and young smokers who see warning labels are more likely to report considering cessation.[74]

American consumers rate graphic warning labels as the most effective way to convey information about the health risks of smoking, with no differences by race/ethnicity or education.[75] However, smokers with lower education levels are less likely to recall the messages in text-only warnings than smokers with higher education levels, which is particularly concerning given the inverse relationship between educational attainment and smoking status.[71] Pictorial warnings more effectively convey information about the health effects of smoking than text-only warnings in populations with low literacy rates.[74]

New and Modified-Risk Tobacco Products

Historically, the tobacco industry has introduced new products targeted to specific groups as a strategy to attract those market segments—in particular, women, young adults, and racial/ethnic groups.[77–80] For example, in 1990, R.J. Reynolds planned to test-market a cigarette brand (Uptown) explicitly targeted to African Americans; after substantial public outcry from local groups, the planned test market was shelved.[78,81] "Slim" cigarettes, with a longer cigarette shaft length and light-colored packaging, is another example of products meant to appeal to a market segment—in this case, women.[74] FDA regulation of tobacco products has the potential to reduce TRHD by preventing the introduction of products tailored to appeal to specific populations.

In addition, analysis of internal tobacco industry documents and other sources finds evidence that as early as the 1960s, tobacco companies sought to create products that would increase the social acceptability of smoking, reduce secondhand smoke exposure to bystanders, or be perceived as reducing the harm to consumers of the product. This included products that were purported to heat rather than burn tobacco, such as the Ariel cigarette (British American Tobacco), and Premier and Eclipse cigarettes (both by R.J. Reynolds).[82] Although none of these "heat not burn" tobacco products were commercially successful, the introduction of these and other "potential reduced harm products" highlights the importance of tobacco product regulation to ensure that misleading health claims will not harm public health.[83,84] More recently, the expansion of the e-cigarette market has raised similar challenges.[82,85,86]

As noted in the Tobacco Control Act, "the dangers of products sold or distributed as modified risk tobacco products that do not in fact reduce risk are so high that there is a compelling governmental interest in ensuring that statements about modified risk tobacco products are complete, accurate, and relate to the overall disease risk of the product."[87,88[Finding 40]] Therefore, the Tobacco Control Act requires FDA to conduct a premarket review for all new and "modified risk" tobacco products; FDA has the authority to review new tobacco product applications and to reject products that do not meet the statutory requirement. Under Section 911, FDA-CTP will not allow products to be marketed as "modified risk" unless the applicant company can provide evidence that such a product, when actually used by consumers, will significantly reduce the risk of tobacco-related disease to individuals and will benefit public health.[88]

Another example of tobacco industry product diversification was the introduction of "light" and "low-tar" cigarettes. As noted in the Tobacco Control Act, Congress found that "studies have demonstrated that there has been no reduction in risk on a population-wide basis from 'low tar' and 'light' cigarettes, and such products may actually increase the risk of tobacco use."[88[Finding 39]] Also, "many smokers mistakenly believe that 'low tar' and 'light' cigarettes cause fewer health problems than other cigarettes. As the National Cancer Institute has also found, mistaken beliefs about the health consequences of smoking 'low tar' and 'light' cigarettes can reduce the motivation to quit smoking entirely and thereby lead to disease and death."[88[Finding 38]] For this reason, the Tobacco Control Act prohibits tobacco companies from making reduced harm claims like "light," "low," or "mild" without filing an application for a modified risk tobacco product and obtaining an authorization to market as such.[88] Following FDA's ban on descriptors (light, low, mild) tobacco companies have made use of pack colors, numbers, and other means to convey a message of reduced harm to consumers; studies suggest these alternate means are effective at conveying false beliefs about product risk.[75,89]

Restricting Marketing and Sales

The Tobacco Control Act grants FDA the power to impose restrictions on the advertising and promotion of tobacco products. Specifically, the Act states that FDA "may by regulation require restrictions on the sale and distribution of a tobacco product, including restrictions on the access to, and the advertising and promotion of, the tobacco product," if it determines this to be appropriate for the protection of the public health.[90] Additionally, FDA "may by regulation impose restrictions on the advertising and promotion of a tobacco product consistent with and to full extent permitted by the first amendment to the Constitution."[90] Given the role of targeted marketing in promoting tobacco use by vulnerable populations, restrictions on tobacco advertising and promotion have the potential to substantially reduce TRHD.

Restrictions on marketing and sales have strong potential to reduce low-income, urban, and racial/ethnic minority youths' exposure to tobacco advertising. As described in chapter 10, a number of studies have found that tobacco advertising, especially exterior advertising and advertising that promotes menthol cigarettes, is most common in low-income and minority neighborhoods.[54,62,91–95] Similarly, studies have found that point-of-sale cigarette advertising has increased, especially in predominantly African American neighborhoods.[92,96] Because greater exposure to cigarette advertising is associated with increased receptivity to such advertising[97] and with youth smoking initiation,[98] restricting marketing and advertising has the potential to reduce TRHD.

Tobacco Product Standards

The Tobacco Control Act banned cigarettes with characterizing flavors, other than menthol and tobacco, as of 3 months after the date of enactment of the law. In addition, the Tobacco Control Act allows FDA to adopt product standards appropriate for the protection of public health, regarding the construction, components, ingredients, additives, constituents (including smoke constituents), and properties of the tobacco product. FDA has the ability to require the reduction or elimination of an additive, constituent, or component of a tobacco product, and may set limits on the nicotine yield of tobacco products, but cannot require the nicotine yields of a tobacco product be reduced to zero.[58]

A number of studies have suggested that mandating reductions in a tobacco product's nicotine content could improve public health by reducing the rate of transition from experimentation to nicotine dependence and by facilitating cessation.[99,100] Research suggests that cigarettes with very low nicotine content lead to several positive outcomes, including reduced nicotine exposure, reduced smoking intensity, and reduced cigarette dependence.[101,102] However, it is not known if mandating reduced cigarette nicotine yields would have a differential impact on youth, or by race/ethnicity, SES, or other factors. Additionally, support for mandating reduced cigarette nicotine yields may vary by racial/ethnic status and SES. Pearson and colleagues found that Hispanics and African Americans are 1.6–2.6 times more likely than whites to support requiring nicotine reductions; support for requiring nicotine reductions was 2.3–2.9 times greater among high school graduates or people with some high school education than among otherwise similar people with a college degree.[103]

Although the Tobacco Control Act's ban on characterizing flavors did not include menthol, FDA has the authority to issue a product standard on menthol. As described in chapter 2, menthol cigarette use differs substantially by population group and is highest among African Americans. The Tobacco Control Act required the Tobacco Products Scientific Advisory Committee to study the impact of the use of menthol in cigarettes on the public health, including such use among children, African Americans, Hispanics, and

other racial and ethnic minority groups, and issue a report and recommendations within one year.[56] The resulting TPSAC report, *Menthol Cigarettes and Public Health: Review of the Scientific Evidence and Recommendations*, concluded that "removal of menthol cigarettes from the marketplace would benefit public health in the United States,"[104,p.225] Surveys of menthol smokers suggest that a ban on menthol cigarettes would reduce smoking prevalence; a substantial fraction (35% to 40%) of menthol smokers report that if menthol cigarettes were banned, they would quit smoking.[103,105] Additionally, simulation modeling examining the potential impact of a menthol ban suggests that a menthol ban that resulted in 30% of menthol smokers quitting and a 30% reduction in initiation among those who would have started as menthol smokers would save between 323,000 and 633,000 lives, almost one-third of these being among African Americans.[106]

Regulation of Other Tobacco Products

The Tobacco Control Act provided FDA with the immediate authority to regulate cigarettes, cigarette tobacco, roll-your-own tobacco, and smokeless tobacco. The Act also provides that other tobacco products, such as cigars (including little cigars and cigarillos), pipe tobacco, waterpipe tobacco (hookah), and electronic cigarettes, may be brought under FDA authority by regulation.[107] In 2014, FDA signaled its intention to extend authority to these previously unregulated products via a proposed rule; the final "deeming" rule was published on May 10, 2016, and became effective on August 8, 2016.[108]

The deeming rule's provisions extend FDA's regulatory authority to all tobacco products, including e-cigarettes and other electronic nicotine delivery systems (ENDS), cigars, waterpipe tobacco, pipe tobacco, nicotine gels, and dissolvables that did not previously fall under the FDA's authority.[108] It requires health warnings on roll-your-own tobacco, cigarette tobacco, and certain newly regulated tobacco products and also bans free samples. In addition, manufacturers of newly regulated tobacco products that were not on the market as of February 15, 2007, will be required to show that products meet the applicable public health standard set by the law, and those manufacturers will have to receive marketing authorization from the FDA. The rule also restricts youth access to newly regulated tobacco products by (1) not allowing products to be sold to people younger than 18 and requiring age verification via photo ID; and (2) not allowing tobacco products to be sold in vending machines (unless in an adult-only facility). Finally, the rule provides a foundation for future FDA actions related to newly deemed tobacco products.[108] Some requirements went into effect on August 8, 2016 (such as the minors' access provisions, the ban on free samples, and the requirement for premarket review for any modified risk claims), but there were staggered deadlines for other provisions (such as the health warnings and the reporting of ingredients and HPHCs). As of July 2017, six pending lawsuits were challenging the deeming rule.[109]

To the extent that other tobacco products are differentially used by various population groups, the provisions of the deeming rule have the potential to reduce TRHD. For example, consumption of little cigars and cigarillos is concentrated among African Americans, males, young adults, youths, and at the intersections of these race, gender, and age groups.[110–113] Surveillance of these products can be challenging because of the diversity of products on the market.[111,114] Little cigars often have filters, are sold in packs of 20, and may be similar in size to cigarettes; cigarillos more closely resemble cigars and come in a wide variety of sizes, filter tips, and wrapper types.

With passage of the Tobacco Control Act, cigarettes with characterizing flavors other than tobacco or menthol are banned. However, as of 2017, characterizing flavors were not prohibited in non-cigarette

tobacco products, such as e-cigarettes, hookah, and cigars (premium cigars, cigarillos, and little filtered cigars). Data from the 2013-2014 National Adult Tobacco Survey show that more than one-third of cigar smokers (36.2%) used flavored cigars in the past 30 days; flavored cigar use was highest among young adults (48.3%).[115] Differences in the preference for flavored tobacco products, including cigars and cigarillos, have also been observed by gender, race/ethnicity, income, education, sexual orientation, and transgender identification.[115,116] In 2016, FDA took action against four companies for "selling flavored cigarettes that are labeled as little cigars or cigars, which is a violation of the Family Smoking Prevention and Tobacco Control Act."[117]

The lower price and the availability of flavorings could contribute to the popularity of little cigars and cigarillos among youths and young adults. As described in chapter 2, cigar use by U.S. youths increased during 2011-2012 and then declined for 2013-2014 and remained unchanged for 2014-2015.[118] Results from the 1997–2015 YRBS suggest a relatively stable trend in cigar use among females and fluctuating trends among non-Hispanic black high school students. From 2013 to 2015, cigar smoking increased among African American high school students.[118] These statistics could reflect promotional efforts in that little cigars and cigarillos are disproportionally available, advertised, and priced lower in some lower income, younger, and predominantly African American neighborhoods.[119,120] Variation in state-level taxation and tobacco industry marketing highlights the need for federal regulation of little cigars and cigarillos to help reduce TRHD.

Preservation and Expansion of State and Local Authority

The Tobacco Control Act preserves nearly all authority of states and local governments to regulate various aspects of tobacco products. States may impose specific bans or restrictions on the time, place, and manner—but not the content—of cigarette advertising. They may prohibit or restrict the distribution of free samples of smokeless tobacco in any location (FDA may prohibit or restrict them to only "qualified adult-only facilities.") States may adopt or continue to enforce all requirements pertaining to tobacco products that are in addition to, or more stringent than, the requirements of the new law relating to or prohibiting the sale or distribution of tobacco products; the possession, exposure, or access to tobacco products; the advertising and promotion of tobacco products; the use of tobacco products by individuals of any age; information reporting to the state; and measures relating to fire safety standards for tobacco products.

Thus, the Tobacco Control Act gives local authorities expanded power to address local problems, particularly among the most at-risk populations. As described in the Center for Public Health Systems Science publication *Point-of-Sale Strategies: A Tobacco Control Guide*:

> The 1965 Federal Cigarette Labeling and Advertising Act (FCLAA) and its amendments preempted states and communities from imposing requirements related to cigarette advertising or promotion based on concerns about smoking or health. The Tobacco Control Act changed this provision by allowing states and communities, where allowed by state law, to restrict or regulate the time, place, and manner (but not the content) of cigarette advertising and promotion. For example, states and many communities are now authorized to enact advertising restrictions, limit the size of product ads, and regulate the location of ads in stores. However, states and communities considering these strategies will need to make sure that the policies pursued do not violate the First Amendment.[121,p.6]

As a result, a number of communities have moved to address tobacco product sales through zoning, licensing, and other ordinances.[122] Additionally, the Tobacco Control Act does not constrain states' authority to engage in many other tobacco control activities, including raising tobacco taxes; enacting and enforcing smoke-free laws; funding comprehensive tobacco control programs; implementing counter-marketing campaigns; restricting the sale, distribution, and possession of tobacco products; and implementing measures to counter smuggling and tax evasion.[123]

Youth Access Policies and Gender and Race/Ethnicity

Limiting young people's ability to purchase tobacco products is a well-established component of a comprehensive strategy to prevent youth smoking.[8] Youth obtain cigarettes from social sources (e.g., friends or family members) and from commercial sources. As explained in NCI Tobacco Control Monograph 21, "youth access policies are intended to reduce opportunities for minors to obtain tobacco products from commercial sources, with the goals of preventing youth from beginning to smoke, decreasing cigarette consumption, changing social norms with respect to smoking, and decreasing young people's overall smoking prevalence."[70,p.402]

The 1992 Synar Amendment to the Alcohol, Drug Abuse and Mental Health Services Administration (ADAMHA) Reorganization Act (Public Law 102-321, Section 1926)[124] required that all states and territories legally prohibit the sale of tobacco to minors by the middle of 1995. Prior to passage of the Synar Amendment, youths obtained cigarettes from commercial sources with relative ease.[125–129] The Synar Amendment, which requires state legislative action, has been supplemented by local (city and county) ordinances in the 27 states where such ordinances are not restricted by state preemption.[130] The Synar Amendment requires states to enforce youth access laws through compliance checks and to report progress to the Secretary of the U.S. Department of Health and Human Services (HHS). States that do not comply with Synar annual goals can have their Substance Abuse Prevention and Treatment block grant monies reduced.[131]

The Tobacco Control Act gave FDA the authority to regulate the distribution of tobacco products at the federal level in order to reduce tobacco use by minors.[56] This youth access regulation, in effect as of June 2010, established a federal age of 18 for tobacco purchase, required photo identification for buyers younger than age 27, banned vending machines and self-service tobacco displays, except in adult-only establishments, and authorized state contracts for compliance-check inspections.[56] The FDA youth access regulations also apply to areas previously exempt from the federal Synar Amendment requirements, such as American Indian reservations.

FDA has implemented a robust retailer enforcement program, through which the agency conducts inspections, and when violations are found, takes enforcement action such as issuing Warning Letters; for repeat violations, FDA may seek Civil Money Penalties or No-Tobacco-Sale Orders. The Food, Drug, and Cosmetic Act also authorizes FDA to contract with states, territories, and tribes to inspect retail establishments, including the undercover use of minors to attempt to purchase tobacco products, and to determine whether retailers are in compliance with other youth access restrictions. In addition, FDA conducts surveillance of promotion, advertising, and labeling, including online marketing, to monitor compliance with the Food, Drug, and Cosmetic Act and FDA regulations.

A study by DiFranza and colleagues[132] examined the association of youth tobacco use rates from the 2003 Monitoring the Future (MTF) survey and merchant compliance from the 1997–2003 state Synar

reports. These authors found that the odds of daily smoking among 10th graders in 2003 were reduced by 2% for each 1% increase in merchant compliance. Johnston and colleagues,[133] also using MTF data, found that the percentage of students who reported that getting cigarettes was "fairly or very easy" declined from 77.8% in 1992 to 49.9% in 2013 for 8th graders, and from 89.1% in 1992 to 71.4% in 2013 for 10th graders. Data from the 2001–2015 Youth Risk Behavior Surveys (YRBS), presented in Table 11.2, also show a general decline in the proportion of youth smokers who reported usually buying their own cigarettes commercially—from 19.0% in 2001 to 12.6% in 2015.[134] NCI Tobacco Control Monograph 21 concluded that "youth access policies, when consistently enforced, can reduce commercial access to tobacco products among underage youth," and that "strongly enforced youth access policies that successfully disrupt the commercial supply of tobacco products to underage youth can reduce youth tobacco use, although the magnitude of this effect is relatively small."[70,p.419]

Table 11.2 Percentage and Number of U.S. High School Students Who Usually Obtained Their Own Cigarettes by Buying Them in a Store or Gas Station, 2001–2015

Category	2001 (%, 95% CI, n)	2003 (%, 95% CI, n)	2005 (%, 95% CI, n)	2007 (%, 95% CI, n)	2009 (%, 95% CI, n)	2011 (%, 95% CI, n)	2013 (%, 95% CI, n)	2015 (%, 95% CI, n)
Total	19.0 (16.8–21.4) 2,586	18.8 (16.3–21.7) 2,512	15.2 (12.7–18.2) 2,152	16.0 (12.8–19.9) 1,939	14.1 (11.7–17.0) 2,266	14.0 (11.5–16.9) 1,835	18.1 (14.4–22.4) 1,344	12.6 (9.7–16.1) 1,198
Gender								
Female	13.1 (10.8–15.9) 1,306	13.7 (9.9–18.6) 1,208	11.7 (8.6–15.6) 1,077	11.3 (8.0–15.6) 893	9.6 (6.9–13.2) 1,098	10.2 (7.6–13.7) 832	15.6 (11.4–21.0) 595	7.7 (4.8–12.2) 564
Male	25.4 (22.3–28.9) 1,273	24.3 (20.8–28.1) 1,298	18.8 (15.7–22.5) 1,072	20.0 (16.0–24.8) 1,043	18.3 (15.6–21.5) 1,159	17.1 (13.5–21.3) 996	20.4 (16.2–25.5) 748	16.5 (12.7–21.2) 625
Race/Ethnicity								
Black	24.0 (16.5–33.5) 245	22.1 (16.7–28.7) 384	21.6 (12.1–35.5) 280	19.3 (13.3–27.3) 227	19.7 (12.5–29.8) 187	13.7 (8.5–21.2) 167	23.5 (13.2–38.3) 161	N/A 69
Hispanic	16.6 (12.9–21.0) 607	23.1 (17.8–29.4) 655	17.4 (11.5–25.5) 495	13.8 (8.8–21.2) 516	13.3 (9.3–18.6) 625	14.9 (11.2–19.5) 534	21.1 (15.5–28.0) 311	17.5 (12.7–23.7) 302
White	19.2 (16.6–22.1) 1,509	17.5 (14.5–21.0) 1,331	14.1 (11.6–17.1) 1,196	15.9 (21.1–20.6) 992	14.1 (11.3–17.5) 1,248	13.9 (10.6–18.0) 902	17.2 (12.7–22.9) 739	9.7 (6.9–13.6) 696

Notes: Data shown are: percentage, confidence interval, and cell size for respondents who reported smoking in the previous 30 days. N/A = fewer than 100 respondents for the subgroup.
Source: Centers for Disease Control and Prevention 2001-2015.[134]

Youth access laws have largely focused on restricting minors' ability to purchase cigarettes in person from legal businesses. However, among the provisions of the Prevent All Cigarette Trafficking (PACT) Act of 2010 is a requirement for Internet and other mail-order tobacco retailers to check the age and identification of all customers at the time of purchase and at the time of delivery.[135] Purchases via the

Internet and vending machines represented a small proportion of total cigarette purchases by youths (about 9%, depending upon age) as reported in the 2014 National Youth Tobacco Survey (NYTS).[136]

Whether all youth benefit equally from youth access laws is unclear, and enforcement of these laws is likely to have changed over time. Some evidence suggests that race/ethnicity and gender may be associated with differential likelihood of being able to purchase tobacco. Landrine and colleagues[137] examined combined data on all purchase attempts (n = 3,361) reported annually from 1999 to 2003 in California to meet the Synar Amendment requirements. They found that black 15-year-olds and Asian 16-year-olds were more likely to be sold cigarettes (22.2% and 22.6%, respectively) and that black girls were more likely to successfully purchase cigarettes (23.2%) compared with the overall purchase rate of 14.7% for California youths in these years. These differences were almost entirely explained by clerk behavior: 25% of the clerks failed to request youth age identification, and they were responsible for 95% of the cigarette sales.[137] In 2014, Lipperman-Kreda and colleagues[138] found that at the community level, higher percentage of minors, higher education, and a greater percentage of African Americans were associated with increased likelihood of a successful purchase of cigarettes by young buyers.

The 2015 NYTS obtained details about all the sources from which youths obtained tobacco products. Table 11.3 shows that the most common source for high school students who used tobacco products was buying the products themselves, which was reported by 14.8% to 30.7% of students, depending on demographics. Substantial proportions of students reported obtaining tobacco products by giving others money to buy them (18.2%–24.2%) and borrowing or "bumming" them (11.9%–16.9%).[139] Overall, the data suggest that youths who use tobacco, including youths of color, are able to obtain tobacco products from a variety of sources.

Table 11.3 High School Students' Usual Source of Tobacco Products in the Past Month, NYTS, 2015

Sex, race/ethnicity	Bought them myself (%)	Had someone else buy them for me (%)	Asked someone to give me some (%)	Someone offered them to me (%)	Bought them from another person (%)	Took them from a store or another person (%)	Other (%)
Male	30.7	19.9	11.9	18.0	6.7	3.3	9.5
Female	14.8	23.3	16.9	27.5	5.3	3.3	8.8
Race/Ethnicity							
White, Non-Hispanic	24.2	22.3	13.7	22.6	5.9	3.6	7.8
Black, Non-Hispanic	26.4	24.2	13.5	15.5	7.0	3.0	10.5
Hispanic	22.0	18.2	15.1	23.9	6.7	3.3	10.9
Asian, Non-Hispanic	21.5	22.3	14.0	18.2	5.8	7.4	10.7

Note: NYTS = National Youth Tobacco Survey.
Source: Centers for Disease Control and Prevention 2015.[139]

Several studies of commercial access have tested the hypothesis that neighborhood context is an important factor that might be related to differential sources of cigarettes for youth. In the 2010 California study by Landrine and colleagues cited above, neighborhood ethnicity (white versus Latino)

had no effect on purchase success.[137] Similarly, Widome and colleagues[140] failed to find neighborhood demographic characteristics that predicted the likelihood of tobacco sales to minors. In a study of 50 California cities, Lipperman-Kreda and colleagues[138] found that several city characteristics were associated with compliance with underage sale laws (i.e., percentage of minors, education level, percentage of African Americans in the population). However, the units of analysis were entire midsize cities (50,000 to 150,000 people), so the results cannot be extrapolated to individual neighborhoods.

Few studies have looked at the context of neighborhood influences on youth access to cigarettes. A qualitative study of young adult smokers in African American neighborhoods in Baltimore revealed an informal but ubiquitous market for single cigarettes, or "loosies."[141] Although the sale of single cigarettes is prohibited by federal law,[142] single cigarettes were reported to be available for purchase in stores; outside stores, bars, clubs, and subway stations; and from individuals who had purchased or stolen a pack for that purpose. Although not expressly marketed to youths, single cigarettes were likely available to them, and probably reduced the initial cost of smoking. Sales of single cigarettes have also been documented in Philadelphia and New York City and likely occur in other jurisdictions as well.[143,144]

Additionally, some localities have implemented ordinances to ban the sale of flavored tobacco products to youths. New York City prohibited all sales of flavored tobacco products (cigars/cigarillos/little cigars, smokeless tobacco, and others) in 2009, with enforcement beginning in 2010. An analysis of the impact of the ban found that in addition to decreased sales of flavored tobacco products, youth use of both flavored and any tobacco product declined significantly after enforcement of the ban.[145] Chicago became the first U.S. city to restrict the sale of all flavored tobacco products, including menthol tobacco products.[4] This ordinance resulted from the Healthy Chicago Initiative, a multidimensional approach to achieving public health goals, including reducing tobacco use, which engaged the public health community, clergy, educational institutions, charitable organizations, local elected officials, concerned residents, and advocates from around the country. Although the ordinance was later rescinded and replaced with a measure that increased the age of sale for tobacco products from 18 to 21,[146] the effort illustrates the value of using a multidimensional approach to identifying and developing public health policies at the local level. In June 2017, the city of San Francisco banned the sale of flavored tobacco products, as well as flavored electronic cigarette liquids.[147] The legislation prohibited the sale of all flavored tobacco products, including menthol-flavored products.

The 2009 Tobacco Control Act directed FDA to convene an expert panel to study the implications of raising the minimum age to purchase tobacco products. The resulting report, *Public Health Implications of Raising the Minimum Age of Legal Access to Tobacco Products*, published in 2015 by the Institute of Medicine (now known as the National Academy of Medicine), analyzed the potential effects of raising the minimum legal age of access to 21 and to 25 and concluded that increasing the minimum age of legal access to tobacco will "likely prevent or delay initiation of tobacco use by adolescents and young adults"[136,p.2] and "will likely lead to substantial reductions in smoking-related mortality."[136,p.3] Although FDA is not permitted to raise the minimum legal age of sale, states and localities are free to do so. As of August 2017, more than 250 localities and 5 states had raised the legal age of sale of tobacco products to 21 years.[3]

Tobacco Tax Policies and Price

The price of tobacco products is a major factor determining their use[100,148,149]; although many factors affect the price of cigarettes paid by the consumer, the factor that is most amenable to the influence of policy is cigarette taxes. NCI Tobacco Control Monograph 21 concluded that:

- A substantial body of research, which has accumulated over many decades and from many countries, shows that significantly increasing the excise tax and price of tobacco products is the single most consistently effective tool for reducing tobacco use.[70,p.151]
- Significant increases in tobacco taxes and prices reduce tobacco use by leading some current users to quit, preventing potential users from initiating use, and reducing consumption among current users.[70,p.151]
- Tobacco use by young people is generally more responsive to changes in taxes and prices of tobacco products than tobacco use by older people.[70,p.151]
- Lower income populations often respond more to tobacco tax and price increases than higher income populations. As a result, significant tobacco tax and price increases can help reduce the health disparities resulting from tobacco use.[70,p.585]

In the United States, the federal government, all 50 states, the District of Columbia, Puerto Rico, Guam, Northern Marianas, and many local governments impose taxes on cigarettes. There are two types of excise taxes on tobacco products in the United States—specific and *ad valorem*. A specific excise tax is a fixed monetary amount of tax per quantity, volume, or weight of tobacco products (e.g., dollars per pack, carton, or kilogram). Specific excise taxes do not fluctuate with the price of tobacco. *Ad valorem* taxes, on the other hand, are a fixed percentage of the value of the tobacco product (e.g., a percentage of the wholesale or retail price). Thus, *ad valorem* taxes increase or decrease as cigarette and other tobacco prices change. In the United States, cigarettes are taxed through specific excise taxes.[70] However, states with sales tax also apply their sales tax (i.e., a non-excise, *ad valorem* tax) to cigarettes; most apply it to the cigarette price inclusive of the excise tax.

Federal Cigarette Tax Policy

The federal tax on cigarettes increased from $0.39 per pack to $1.0066 per pack on April 1, 2009, a level that was still in effect as of 2017. The revenue generated from the 2009 tobacco tax increase is being used to fund an expansion of the State Children's Health Insurance Program (SCHIP). The 2009 federal tax increase was the first federal cigarette tax increase since the two-stage increase mandated by the Balanced Budget Act of 1997, which raised the federal cigarette tax from $0.24 per pack to $0.34 per pack on January 1, 2000, and from $0.34 per pack to $0.39 per pack on January 1, 2002. (See Table 11.4.)

Table 11.4 Federal Cigarette Excise Taxes for Selected Dates, 1993–2016

Effective Date	Tax per Pack of 20 Cigarettes
January 1, 1993	$0.24
January 1, 2000	$0.34
January 1, 2002	$0.39
April 1, 2009	$1.0066

Sources: Orzechowski and Walker 2009[285]; Campaign for Tobacco Free Kids 2017.[3]

In addition to raising the federal cigarette excise tax, the 2009 increase also applied the same tax rate to cigarette-like small cigars (from $0.037 cents per pack to $1.0066 cents per pack) and to roll-your-own tobacco (from $0.045 cents per pack to $1.0066 cents per pack).[150] Substantial differences remain between federal taxes on cigarettes, small cigars, roll-your-own tobacco, and other tobacco products, including regular cigars and smokeless tobacco; non-cigarette tobacco products are generally taxed at lower rates than cigarettes.

State and Local Tobacco Tax Policy

All 50 states, the District of Columbia, Puerto Rico, Guam, and Northern Marianas currently impose an excise tax on cigarettes. As of April 1, 2017, state excise tax rates ranged from a low of $0.17 per pack in Missouri to $4.35 per pack in New York[3] (see Table 11.5). In 2017 the average state cigarette tax was $1.69 per pack across all 50 states and the District of Columbia, although the average tax remains much lower ($0.485 per pack) in tobacco-growing states.[3] State excise taxes have increased considerably over time; from 2002 to 2017, 48 states and the District of Columbia increased their excise taxes on cigarettes at least once. Combined, the states and the District of Columbia have passed more than 128 separate excise tax increases since January 1, 2002.[3] Since the federal tax increase in 2009, some states have increased excise tax rates on non-cigarette tobacco products, although in most states the tax rate on these products remains markedly lower than the cigarette excise tax rate.[151] These changes in federal and state taxes, combined with manufacturers' efforts to adjust product weight to qualify for lower taxes, are believed to partially account for increased cigar use, and could contribute to TRHD among groups that favor these products, notably African Americans, youths, and young adults.[110,112,152,153]

Table 11.5 State/Local Cigarette Excise Tax, 2017

State/Locality	Excise Tax ($)	Rank	State/Locality	Excise Tax ($)	Rank
Alabama	0.675	40	Nebraska	0.64	41
Alaska	2.00	14	Nevada	1.80	20
Arizona	2.00	14	New Hampshire	1.78	21
Arkansas	1.15	34	New Jersey	2.70	10
California	2.87	9	New Mexico	1.66	24
Colorado	0.84	38	New York	4.35	1
Connecticut	3.90	2	North Carolina	0.45	47
Delaware	1.60	25	North Dakota	0.44	48

Table 11.5 continued

State/Locality	Excise Tax ($)	Rank	State/Locality	Excise Tax ($)	Rank
District of Columbia	2.50	13	Ohio	1.60	25
Florida	1.339	30	Oklahoma	1.03	36
Georgia	0.37	49	Oregon	1.32	31
Hawaii	3.20	5	Pennsylvania	2.60	11
Idaho	0.57	45	Rhode Island	3.75	3
Illinois	1.98	19	South Carolina	0.57	45
Indiana	0.995	37	South Dakota	1.53	27
Iowa	1.36	29	Tennessee	0.62	42
Kansas	1.29	32	Texas	1.41	28
Kentucky	0.60	43	Utah	1.70	22
Louisiana	1.08	35	Vermont	3.08	6
Maine	2.00	14	Virginia	0.30	50
Maryland	2.00	14	Washington	3.025	8
Massachusetts	3.51	4	West Virginia	1.20	33
Michigan	2.00	14	Wisconsin	2.52	12
Minnesota	3.04	7	Wyoming	0.60	43
Mississippi	0.68	39	Puerto Rico	3.40	
Missouri	0.17	51	Guam	3.00	
Montana	1.70	22	Northern Marianas	1.75	

Note: Table shows all cigarette tax rates in effect as of April 1, 2017.
Source: Campaign for Tobacco-Free Kids 2017.[3]

Hundreds of municipalities and counties also tax cigarettes, with most applying a relatively small tax compared with state tax rates. However, on occasion, cities and counties have implemented large tax increases. For example, on January 1, 2011, the city of Anchorage, Alaska, increased the tax on cigarettes by $0.75 per pack, yielding a total city cigarette tax of $2.21. In 2002, New York City increased its tax on cigarettes from 8 cents per pack to $1.50 per pack. Similarly, the city of Chicago and Cook County, Illinois, raised taxes on cigarettes. In 2017, the combination of federal, state, and local taxes meant that individuals purchasing cigarettes in New York City paid $6.86 per pack in taxes, and Chicagoans paid $7.17 per pack in taxes.[3]

Impact of Cigarette Tax Policy and Price on Cigarette Demand

One of the fundamental principles of economics—the downward-sloping demand curve—holds that as the real price of a good increases, the consumption of that good falls. At one time, some researchers believed that cigarettes would be an exception to this fundamental principle, given the addictive nature of nicotine. However, numerous econometric studies conducted over the past four decades, including several studies that explicitly modeled the addictive properties of cigarettes, have confirmed that an inverse relationship exists between cigarette prices and cigarette consumption.

The years 1997–2002 witnessed one of the largest increases in the inflation-adjusted price of cigarettes in the United States. The real price of cigarettes increased by 71.1% during this period, partly because of significant increases in the wholesale price of cigarettes. Between 1998 and 2003, wholesale cigarette prices increased 122%,[154] largely as a result of the costs associated with the four individual state tobacco settlement expenses and expenses related to the MSA. The increased retail price was also partly the result of the two federal tax increases and numerous increases in state excise taxes. Cigarette tax increases led to proportional or more than proportional increases in the retail price of cigarettes. It is important to note that because excise taxes are per-unit taxes, the inflation-adjusted value of the tax will fall over time unless these taxes are increased regularly; in fact, given the importance of tax in determining the price of cigarettes, infrequent increases in the tax will likely result in declines in inflation-adjusted cigarette prices over time.

Economists measure how responsive cigarette consumption is to changes in the real price of cigarettes using a concept known as the price elasticity of demand, defined as the percentage change in the amount of cigarettes consumed that results from a 1% change in the price of cigarettes. For example, a price elasticity of –0.5 means that a 10% increase in price will result in a 5% decrease in consumption.

A substantial body of economic research has estimated the price elasticity of demand for cigarettes. The findings from this research clearly demonstrate that the consumption of cigarettes is inversely related to the price of cigarettes. The consensus estimate from reviews of the literature is that the overall price elasticity of demand for cigarettes in the United States falls in a narrow range of –0.3 to –0.5.[14,148,155] These reviews suggest that approximately one-half of the effect of price increases on cigarette consumption comes from decreases in smoking prevalence rates, with the remaining effect of price increases coming from decreases in smoking intensity among those who continue to smoke. Among adults, much of the impact of price increases on smoking prevalence is driven by increased cessation; among youth, much of the impact of price increases on smoking is attributable to decreased initiation.[14,148,155]

Impact of Price on Cigarette Demand by Age

Economic theory predicts that adolescents and young adults will be much more responsive to price changes than older adults; that is, the demand for cigarettes among adolescents and young adults is expected to be more price elastic than the demand among older adults. There are several reasons for this prediction. First, the fraction of disposable income spent on cigarettes by young smokers is likely to be larger than that spent by adult smokers.[156] Economic theory predicts that the greater the share of disposable income a good takes up, the more responsive individuals will be to price changes. Second, adolescents and young adults typically have a greater propensity to discount the future than adults[156]—that is, young people place a relatively greater importance on short-term costs of smoking such as the purchase price of cigarettes, than on long-term costs of smoking such as its future health effects. Third, young smokers are likely to be more sensitive to price because they are likely to have shorter smoking histories than adults and therefore may be less addicted than long-time smokers.[157] Fourth, adolescents and young adults are likely to be more influenced by their peers (friends, siblings) than adults are; compared with older adults, young people are more likely to smoke if their peers also smoke.[157] This finding implies that an increase in the cigarette price would not only reduce youth smoking directly but would also reduce it indirectly by decreasing peer smoking.

Numerous econometric studies examining the determinants of youth and young adult cigarette use have been conducted since the 1980s. With few exceptions, these studies have taken advantage of natural experiments occasioned by government changes in cigarette taxes and/or prices. Consistent with economic theory, these studies have found demand for cigarettes among youth and young adults is inversely related to cigarette prices, and most studies have found that the absolute value of the price elasticity of demand is larger for youth and young adults than for older adults. Higher prices appear to deter smoking initiation, decrease the probability of smoking escalation, and increase smoking cessation by young adults.

Lewit and colleagues[157] were the first to assess the impact of cigarette prices on smoking by U.S. youth. Using data from the nationally representative 1966–1970 National Health and Nutrition Examination Survey (NHANES)(Cycle III), this study estimated an overall price elasticity of –1.44. The strongest impact of price was seen on prevalence, for which the price elasticity was estimated to be –1.20; the price elasticity for consumption among young smokers was –0.25. A follow-up study by Lewit and Coate used the 1976 Health Interview Survey to estimate smoking prevalence and intensity of smoking for populations defined by gender and age.[158] This study found that young adults (ages 20–25 years) were the most responsive to changes in price, with an estimated total price elasticity of demand equal to –0.89. The authors estimated the total price elasticity of demand for adults ages 26 through 35 at –0.47, and for adults ages 36 through 74 at –0.45.

In the early 1990s, two other U.S. studies cast doubt on these early studies by finding that price elasticity among young people was not greater than among adults. Chaloupka[159] used data from the second NHANES, conducted from 1976 to 1980, to estimate the cigarette demand equations. Applying Becker and Murphy's theoretical model of rational addiction, he found young adults ages 17–24 to be insensitive to changes in price, whereas individuals age 25–64 showed a significant long-run response to a change in price, with an estimated long-run price elasticity of demand in the range of –0.46 to –0.31.

Similarly, Wasserman and colleagues[160] used data on 1,891 adolescents ages 12–17 from the second NHANES (1976–1980) and data on adults taken from several waves of the National Health Interview Survey (NHIS) conducted in the 1970s and 1980s to estimate cigarette demand equations.[160] They focused on the effects of cigarette prices while controlling for state policies restricting smoking in public places and a host of socioeconomic and demographic variables. These researchers found no statistically significant differences in the price elasticity of demand among youth versus adult smokers. They attributed much of the difference between their findings and those of Lewit and colleagues' previous studies[157,158] to their inclusion of a measure reflecting the magnitude of restrictions on smoking in public places and its correlation with cigarette prices. When Wasserman and colleagues excluded the smoking restrictions measure from their models, they obtained price elasticities similar to those found by earlier studies. Thus, they argued that price elasticity estimates were affected by an omitted variables bias when the smoking restrictions measure was not included.

Beginning in the mid-1990s, however, a series of papers were published that supported the early findings of Lewit and colleagues[157,158] that young people are indeed more price responsive than older people. These studies were based on data covering a period of greater variation in tax and price, which allowed for more precise estimates of the impact of price. For example, Chaloupka and Grossman[156] used data on 8th-, 10th-, and 12th-grade students taken from the 1992, 1993, and 1994 MTF longitudinal surveys and calculated a total price elasticity for youth smoking of –1.31. However, in contrast to Lewit and colleagues' finding[157] that most of the impact of price was on smoking prevalence, Chaloupka and

Grossman[156] found that approximately one-half of the effect of price was on smoking prevalence and one-half was on smoking intensity. This study controlled for other smoking-related interventions, such as restrictions on smoking in public places and schools as well as youth access restrictions, and refuted Wasserman and colleagues' earlier contention that previous studies reporting price effects had been affected by an omitted variables bias.

A CDC report authored by Farrelly and Bray[161] estimated cigarette demand equations in the United States using data from the National Health Interview Surveys that had included tobacco smoking items (conducted in 1976–1980, 1983, 1985, and 1987–1993). They estimated total price elasticities of demand for different age groups: –0.58 for young adults ages 18–24 years, –0.42 for adults ages 25–39 years, and –0.10 for adults age 40 and older. The total price elasticity of demand for young adults, –0.58, is more than double the total price elasticity of demand for all respondents, which was –0.25.

Lewit and colleagues[162] assessed the impact of cigarette prices on youth smoking prevalence and intentions to smoke using data from cross-sectional surveys of 9th-grade students in the 22 U.S. and Canadian sites that were part of the Community Intervention Trial for Smoking Cessation in 1990 and 1992.[162] They concluded that the price elasticity of demand for 9th-graders was –0.87 and that intentions to smoke among nonsmokers were somewhat more price-elastic, with an estimated elasticity of –0.95.

Tauras and Chaloupka[163] followed individuals from each cohort in the 1976–1993 MTF longitudinal surveys of high school seniors through a maximum of seven follow-ups. Using an individual fixed effects model, they linked increases in cigarette prices to significant reductions in both the number of young adults who smoked and the intensity with which they smoked. The estimated total average price elasticity of demand was found to be –0.79, with price having a smaller effect on smoking prevalence than on smoking intensity.

Almost all econometric research conducted in the 2000s confirmed earlier findings of an inverse relationship between price response and age. Ross and Chaloupka[164] examined the effect of cigarette prices on smoking among high school students in the United States and estimated total price elasticities of demand of –0.67 when using average state prices, and 1.02 when using perceived prices among youth. Tauras and colleagues[165] investigated the impact of cigarette prices and tobacco control policies on youth and young adult smoking from the late 1990s through the early 2000s in the United States, a period characterized by significant changes in cigarette prices and taxes. Using the first five waves of data (1997–2001) from the 1997 National Longitudinal Survey of Youth (NLSY) and controlling for unobserved year and individual characteristics, they also found a strong negative impact of cigarette prices and taxes on young people's smoking prevalence and intensity. Specifically, they estimated a total price elasticity of cigarette demand of –0.83, a price elasticity of demand of smoking prevalence of –0.31, and a price elasticity of demand of smoking intensity of –0.52.

Sloan and Trogdon[46] used data from the Behavioral Risk Factor Surveillance System from the 1990s and early 2000s to estimate smoking prevalence equations among young adults (ages 18–20) and older adults (21 and older). Using both state and year fixed effects, the authors estimated smoking prevalence elasticity of demand for young adults at –0.27 and concluded that smoking prevalence was more price-elastic in young adults than in older adults. They also found that the absolute value of the price elasticity of smoking prevalence declined monotonically with age, with the exception of individuals age 65 and older.

Unlike several previous studies that had used dichotomous state indicators to indirectly control for state-level anti-smoking sentiment, DeCicca and colleagues[166,167] used factor analysis to evaluate attitudinal and belief items from the TUS-CPS administered during the 1990s to develop a direct measure of state-specific anti-smoking sentiment. By linking this anti-smoking sentiment measure to youth smoking data from the 1992 and 2000 waves of the National Education Longitudinal Study (NELS), these authors found price to have a strong, significant, negative impact on smoking prevalence and intensity in young smokers. Estimated price elasticities of smoking prevalence and intensity ranged from –0.59 to –0.76 for the 1992 data and from –0.3 to –0.66 for the 2000 data. Although price consistently had a strong negative influence on the intensity of cigarette use by young smokers in the 2000 cross-section, the price effects lost statistical significance when the direct measure of anti-smoking sentiment was included in the smoking prevalence equations. Using the 2000 wave of data, the authors compared models using the various measures of anti-smoking sentiment. Price was found to have a negative, significant impact on smoking prevalence among youths only when the direct measure of anti-smoking sentiment was excluded. Thus, DeCicca and colleagues question the adequacy of other proxy measures to control for anti-smoking sentiment. However, some caution should be used in interpreting models that include a direct measure of anti-smoking sentiment because state smoking prevalence is likely to influence the state level of anti-smoking sentiment and vice versa, resulting in simultaneity bias.

Carpenter and Cook[168] used national, state, and local YRBS data from 1991 to 2005 to test three alternative methods of dealing with anti-smoking sentiment. First, they estimated a cross-sectional model that relied on intra-state variation in cigarette taxes to identify the impact of price on youth smoking. Second, they estimated a fixed effects model that controlled for area (i.e., state, local) fixed effects and year fixed effects to discern whether the effects were influenced by time or location. Year fixed effects were simply dichotomous indicators for each year of data that was employed, less one year that is intentionally omitted from the equation as the reference category. The same formula was applied for area fixed effects; dichotomous indicators were developed for each location, less one location that is intentionally omitted as the reference category. Employing the same measure of anti-smoking sentiment used by DeCicca and colleagues,[167] Carpenter and Cook found clear and consistent evidence of a negative, significant effect of cigarette taxes on smoking prevalence in the cross-sectional and fixed effects models. Even more importantly, using DeCicca and colleagues' measure of anti-smoking sentiment as well as tax effects from national and state samples, they found a strong negative effect of taxes on smoking prevalence among youths, alleviating concerns raised by that earlier work; estimated price elasticities for youth smoking prevalence were –0.56 in the national sample and –0.25 in the state sample.[168]

Differentiating the Impact of Price on Cigarette Smoking Initiation, Prevalence, and Cessation Efforts

Many researchers examining the impact of price on adolescent smoking prevalence have found that much of the effect of price is felt in relation to smoking initiation. For young adults and adults, on the other hand, price seems to have its strongest effects in the areas of smoking escalation and cessation. Several recent studies have attempted to directly quantify the impact of price on smoking initiation among youths and on smoking escalation and cessation among young adults. Most recent studies, including those described below, have used longitudinal data that track individuals' smoking behavior and other determinants over time. For modeling smoking transitions, longitudinal data is preferred over cross-sectional data with retrospective information.

The first study to examine the impact of price on youth smoking initiation using longitudinal data was conducted by Tauras and colleagues.[169] This study used data from three cohorts of students enrolled in the 8th and 10th grades in 1991, 1992, and 1993 as part of the longitudinal component of the MTF project. The authors examined three alternative measures of smoking initiation, including the transitions from not smoking to smoking any positive amount, smoking at least 1–5 cigarettes per day on average, and smoking at least one-half pack per day on average. The average price elasticity of initiation of any smoking was estimated at –0.27; for smoking an average of at least 1 to 5 cigarettes per day, the estimate was –0.81; and for smoking an average of at least one-half pack per day, the estimated price elasticity was –0.96. These estimates imply that youth smoking initiation is quite responsive to changes in cigarette prices.

As Cawley and colleagues[170,171] conclude, smoking initiation among adolescent girls is significantly influenced by weight-related factors (e.g., self-described overweight, body mass index, reports of trying to lose weight), while smoking initiation among adolescent boys is significantly affected by cigarette prices, with neither factor being significant for the opposite gender. For boys, Cawley and colleagues[170] found that price had a greater impact on the initiation of more regular smoking, estimating elasticities of –0.86 for any smoking initiation, and –1.49 for initiation of frequent smoking. However, for girls, Cawley and colleagues[171] found a price elasticity of initiation of –0.24 compared to –1.2 for boys. These findings suggest that gender-specific differences in the impact of price may account for the mixed findings about price and initiation from previous studies.

DeCicca and colleagues[166] also examined the influence of price and tax on smoking initiation by youths and young adults, using data from the 1988, 1990, 1992, and 2000 waves of the NELS. They found price to have a strong, significant, negative influence on smoking initiation when state fixed effects were excluded from the model. However, when state fixed effects were included in the regressions, price failed to reach significance at conventional levels. The authors concluded that unobserved state-level heterogeneity (possibly anti-smoking sentiment), not price, drove youth and young adult smoking initiation.

In another analysis, DeCicca and colleagues[167] used data from the 1992 and 2000 waves of the NELS to examine the influence of cigarette excise taxes on smoking initiation by young adults (ages 18 and 26). The researchers used three strategies in their identification of effects: (1) they considered intra-state variation in cigarette taxes to identify the impact of price on smoking initiation; (2) they included the direct measure of anti-smoking sentiment developed by DeCicca and colleagues[166] in their equations; (3) they compared the effect of cigarette taxes among young adults who had moved across state lines between 1992 and 2000 to those young adults who had had remained in the same state. Cigarette taxes were found to have a significant negative impact on young adult smoking initiation only among those who had remained in the same state during this time period. The authors concluded that cigarette prices have little impact on smoking initiation. The results of this study should be considered in light of several factors. First, models that rely solely on intra-state variation in taxes give only weak evidence of a negative effect of taxes on smoking prevalence; that is, the price effect fails to reach 5% significance levels of a two-tailed test. Second, anti-smoking sentiment might develop simultaneously with smoking. Third, these results reflected a sample of individuals who had initiated smoking later in life (nonsmokers in high school but smokers by modal age 26); most adults who have ever smoked initiate smoking before this age. The smoking initiation process for older individuals might be significantly different from that for younger people.

A few studies have examined the impact of cigarette price on cessation behavior among young adults. Tauras and Chaloupka[172] were the first to model quit attempts using the longitudinal component of the MTF study by employing a semiparametric Cox regression to assess the probability that smokers would make a transition from smoking to nonsmoking. The authors found that the likelihood that both men and women would make a quit attempt increased significantly when cigarette prices were higher. The estimated price elasticity of transitioning to a nonsmoking status ranged between 0.27 and 0.92 for men and between 0.34 and 0.71 for women; thus, for example, a 10% increase in price would raise the probability of quitting by as much as 10%.

Expanding on the original study, Tauras[173] used the longitudinal component of the MTF study and employed a stratified Cox regression to model multiple quit attempts by young adults. Findings confirmed the positive relationship between cigarette prices and quit attempts, suggesting that a 10% increase in the price of cigarettes increases the likelihood of quit attempts among young adults by approximately 3.5%.

The previously mentioned study by DeCicca and colleagues[167] looked at the influence of cigarette excise taxes on cessation behavior among young adults, using data from the 1992 and 2000 waves of the NELS. Using intra-state variations in cigarette excise taxes, they found young adults to be responsive to tax changes. The price elasticity of prompting cessation behavior was estimated to be 0.93. The price elasticity of cessation declined to 0.47 when the direct measure of anti-smoking sentiment was added. Again, the authors compared the effect of variations in cigarette taxes on the smoking behavior of young adults who had moved across state lines between 1992 and 2000 with that of young adults who had remained in the same state in these 2 years. This analysis found that cigarette taxes had a positive impact on young adult smoking cessation only for those who had moved to a different state between 1992 and 2000. The price elasticity of cessation behavior among young adult movers was relatively large—1.49. The authors concluded that despite the lack of significance of price in this specification, which was most likely attributable to the small sample size (n = 321), price is likely to play a strong role in cessation behavior among young adults.

Tauras[174] examined the impact of cigarette prices on progression to established smoking among young adults in the United States—specifically, transitions from non-daily to daily smoking and from light smoking intensity (defined as 1–5 cigarettes per day on average) and moderate smoking intensity (an average of 10 cigarettes per day) to higher intensities of smoking. Baseline surveys from the 1976–1993 longitudinal components of MTF were used, with follow-up surveys through 1995. The data were adjusted for residence in a tobacco-producing state or in the state of Utah and for area fixed effects to capture census division differences in anti-smoking sentiment. Cigarette prices were found to have a strong negative impact on all smoking transitions estimated. For example, the estimated mean price elasticities were –0.646 for daily use, –0.576 for moderate intensity, and –0.412 for heavy intensity, which would mean that a 10% increase in cigarette prices would decrease daily use by approximately 6.46%, moderate intensity by about 5.76%, and heavy intensity by about 4.12%. These findings clearly demonstrate that cigarette price increases will prevent many young adults from progressing to higher intensities of smoking.

Impact of Price on Cigarette Demand by Socioeconomic Status

The Surgeon General's report *Reducing Tobacco Use*[14] identified the elimination of TRHD as a major goal for the field. Similarly, Healthy People 2020, which delineates 10-year national objectives for

improving the health of the nation, includes as one of its four overarching goals: "achieve health equity, eliminate disparities, and improve the health of all groups."[175] Although tobacco use causes disease and preventable death in all population segments, individuals from lower SES groups bear a disproportionate share of the overall health burden. As described below, the empirical evidence from the United States and other countries on demand for cigarettes among low-SES individuals, as defined by lower incomes and education, indicates that lower SES groups have a stronger response to cigarette price changes than higher SES groups.

A growing number of studies have examined differences in the price responsiveness of individuals by various SES measures. Chaloupka's 1991 study, previously mentioned, was the first to investigate the price sensitivity of different socioeconomic groups in the United States. He estimated cigarette demand equations using the Becker–Murphy model of rational addiction and data from the second NHANES. This study found that individuals with fewer years of formal education were more responsive to changes in cigarette prices than individuals with more formal education. The price elasticity of demand ranged between –0.62 and –0.57 for those with fewer years of education. More-educated individuals were found to be unresponsive to changes in cigarette prices.

Using NHIS data from the 14 years of surveys between 1976 and 1993, Farrelly and colleagues estimated smoking prevalence and smoking intensity equations for various U.S. populations.[176] They found lower income adults to be much more price responsive than higher income adults. Total price elasticity of demand among adults with incomes at or below the median income for the sample was –0.43, whereas those with incomes above the median in the sample had a total price elasticity of demand of –0.10. The price effect of the high-income group was imprecise, however, and the estimate was not significantly different from zero. If the imprecise elasticity for the high-income group is accepted, these findings imply that low-income individuals are more than four times as responsive to changes in cigarette prices as high-income individuals.

Hersch[177] used data from the September 1992, January 1993, and May 1993 waves of the TUS-CPS to estimate smoking demand by income and gender. Hersch found the total price elasticity of demand for low-income women to be –1.71 (prevalence elasticity was –0.99; intensity elasticity, –0.72), and total price elasticity of demand for low-income men, –1.18 (prevalence elasticity, –0.58; intensity elasticity, –0.60). Among middle-income respondents, the corresponding estimated total price elasticity of demand for women was –0.61 (prevalence elasticity, –0.06; intensity elasticity, –0.55) and for men, –0.84 (prevalence elasticity, –0.40; intensity elasticity, –0.44). High-income males and females did not have a significant price response. Hersch concluded that cigarettes do not constitute a large enough share of the household budget for high-income individuals to influence their smoking behavior.

Using U.S. survey data, studies by Gruber and Koszegi,[178] Stehr,[179] and DeCicca and McLeod[180] confirmed the finding that price responsiveness and income are inversely related. A study by Franks and colleagues,[181] however, sparked a debate on the effects of cigarette prices on cigarette consumption and the role that SES plays in price responsiveness. This study extracted data from the 1984–2004 BRFSS and estimated the price elasticity of smoking prevalence among different socioeconomic groups during the years 1984–1996, before the MSA was signed, and in 1997–2004, after the MSA was signed. The authors found that the price elasticity of smoking prevalence in the pre-MSA years was –0.45 in the lowest income group and –0.22 in the highest income group—a finding consistent with previous studies. However, in the post-MSA period, they found that none of the income groups were responsive to

cigarette price changes. These researchers concluded that "increasing cigarette prices [might] no longer be an effective policy tool and [could] impose a disproportionate burden on poor smokers."[181,p.1873]

Farrelly and Engelen[182] reevaluated Franks and colleagues' cigarette-demand equations using slightly different periods to identify the pre-MSA (1990–October 1998) and post-MSA (November 1998–2006) periods and came to a different conclusion. They found that although price effects were smaller in the post-MSA period than in the pre-MSA period for middle- and high-income individuals, the lowest income group was still responsive to price.

A 2008 study by Colman and Remler[183] using U.S. survey data from 1992 to 2003 also suggested that low-income individuals are relatively more price-elastic. Using TUS-CPS data, they found the price elasticity of demand to be –0.37 for the low-income group, –0.35 for the middle-income, and –0.20 for the high-income group.

Evidence from other high-income countries also supports an inverse relationship between SES and price responsiveness. For example, Townsend and colleagues[184] found that individuals in the highest social class in Great Britain exhibited little price responsiveness, whereas individuals in the lowest social class showed a significant response to changes in cigarette prices, with a price elasticity of demand close to –1.0. Similarly, Siahpush and colleagues,[185] using monthly survey data from the 1991–2006 Australian population surveys, concluded that smoking prevalence of low-income individuals was significantly more responsive to price changes than that of medium- or high-income individuals. Specifically, they found the prevalence price elasticity of demand to be –0.32 for low-income groups, –0.04 for medium-income, and –0.02 for high-income groups. Hill and colleagues, in a 2014 review of the literature, examined English-language articles from countries at advanced stages of the tobacco epidemic and found strong evidence that increases in tobacco prices have a pro-equity effect on socioeconomic disparities in smoking.[186]

The World Bank's report *Curbing the Epidemic: Governments and the Economics of Tobacco Control*[187] presented evidence suggesting that low-income, developing countries have larger price elasticities in absolute value when compared with high-income, developed countries. This finding was extended and confirmed by NCI Tobacco Control Monograph 21, which stated:

> Much of the recent evidence indicates that demand for tobacco products in LMICs is at least as responsive to price as demand in HICs, and likely more responsive. In HICs, most estimates of elasticities of demand range from –0.2 to –0.6, clustering around –0.4. In LMICs, elasticity estimates range from –0.2 to –0.8, clustering around –0.5. Thus, in HICs a 10% increase in the price of cigarettes may be expected to decrease tobacco consumption by 4%, while in LMICs a 10% increase in price may be expected to decrease consumption by 5%.[70,p.150]

Researchers have also considered the question of tobacco taxes and equity, noting the importance of ensuring that tobacco control measures reach those who are most burdened by TRHD. Tobacco taxes are often seen as regressive—that is, the poor who are more likely to smoke and have less income to spend pay a larger share of their income on tobacco taxes—and this argument is often used by the tobacco industry to oppose tobacco tax increases.[188] However, because lower income populations are generally more responsive to tobacco tax and price increases than higher income populations, a tax increase will have a greater impact on, and thus provide a greater benefit to, low-income smokers than higher income

smokers. Indeed, research demonstrates that increased tobacco taxes can reduce inequities in tobacco use.[70,189–191] Allocating a portion of the revenue from higher tobacco taxes to programs that serve the needs of low-income populations, including tobacco control and other health programs, can increase the pro-equity effect of tobacco taxes.[189]

Impact of Price on Cigarette Demand by Race/Ethnicity

Many studies find that African American and Hispanic adolescents and adults are more responsive to changes in cigarette prices than white adolescents and adults. This finding is supported by the significant decline in smoking prevalence rates among African American and Hispanic adolescents and adults between 1997 and 2004, a period of dramatic price increases in the United States. In some instances, the estimated price response was found to be extremely large. For example, Chaloupka and Pacula[192] were the first to examine racial differences in the impact of cigarette prices, youth access laws, and smoke-free policies on adolescent smoking prevalence. Using 1992, 1993, and 1994 MTF survey data and controlling for demographic and socioeconomic variables such as parental education and youth income from all sources, the researchers found that African American adolescents were more responsive to price changes than white adolescents. The estimated prevalence price elasticity of demand was –1.11 for black compared to –0.64 for white adolescents, which suggests that black adolescent smoking prevalence is nearly twice as responsive to changes in cigarette prices as the smoking prevalence of white adolescents.

Using several different youth surveys, Gruber and Zinman[193] estimated separate cigarette demand equations for white and non-white youths and for white and black youths. Using the MTF survey, these authors found that both white and non-white 8th- and 10th-graders were unresponsive to price changes. White 12th-graders were also unresponsive to price changes, but non-white 12th-graders were extremely responsive; their price elasticity of demand was an estimated –4.35. This extremely high price elasticity estimate among the older non-white group could not be further analyzed because the non-white racial category could not be disaggregated into different racial/ethnic categories. Using the YRBS, Gruber and Zinman compared the price responsiveness of black and white youths by age. Younger teenagers were defined as 8th-, 9th-, or 10th-graders; 12th-grade students were classified as older teenagers. Again, both younger and older white teenagers as well as younger black teenagers were found to be unresponsive to price changes. Older black teenagers, however, were responsive to price changes, with an extremely high estimated price elasticity of demand of –17.51, a prevalence elasticity of –9.26, and a conditional demand elasticity of –8.25. Lastly, using the Vital Statistics Natality data, these researchers found that black teen mothers were unresponsive to cigarette price changes compared with white teen mothers, who significantly decreased cigarette consumption when prices increased. These conflicting findings and the extremely high price elasticity estimates for non-whites and blacks are thought to reflect the relative stability of cigarette taxes—that is, lack of within-state variation in cigarette prices—during the period when the data were collected (1991–1997).

DeCicca and colleagues[194] used NELS data to examine the impact of cigarette prices on smoking among white, black, and Hispanic adolescents. Unlike previous studies of the determinants of smoking prevalence or average smoking, this study examined the determinants of smoking initiation. Cigarette price was found to be an insignificant determinant of smoking initiation by white adolescents, but it had a negative effect on smoking initiation by black and Hispanic adolescents.

Farrelly and colleagues[176] used NHIS data (1976–1993) to analyze the effect of cigarette prices on adult smoking prevalence and intensity by race/ethnicity. They found that cigarette prices were an important

determinant of black and Hispanic smoking prevalence and average consumption. Although cigarette prices were inversely related to average cigarette consumption among whites, prices were not found to be associated with smoking prevalence among whites. The estimated total price elasticity estimates of demand suggested that black adults were more than twice as price responsive as white adults, and that Hispanics were more than six times as price responsive as white adults. Specifically, the total estimated price elasticity of demand was –0.15 for whites, –0.35 for blacks, and –0.93 for Hispanics.

Several other studies conducted between 2000 and 2017 found similar effects of cigarette price by age and race/ethnicity.[186,195] Using data from the 1991 through 2010 MTF surveys, Tauras and colleagues[196] found that higher cigarette prices will reduce smoking prevalence rates among black, Hispanic, female, and low-SES youth groups faster than the overall youth population and other population groups. The researchers found that black and Hispanic youth had the strongest response, with price elasticities estimated to be −0.718 and −0.444, respectively.

The Community Preventive Services Task Force[197] conducted extensive reviews on cigarette price elasticity among various populations; these studies consistently showed larger price elasticity estimates among low-income tobacco users and among young people. The Task Force concluded that interventions to increase the price of tobacco products are an effective approach to reducing TRHD by income and age. The Task Force also found that price elasticity estimates were greatest for Hispanic populations, followed by African Americans, and concluded that price interventions may have the potential to reduce TRHD associated with race and ethnicity.

Hawkins and colleagues[198] assessed the effects of tobacco control policies, including state cigarette taxes, on pregnant women and infants. These researchers found that white and black mothers with the least amount of education (0–11 years) had the strongest responses to cigarette taxes; they also had the highest prevalence of maternal smoking during pregnancy (42.4% and 20.0%, respectively) and the poorest birth outcomes. Among these mothers, tax increases also reduced the risk of having low-birth-weight, preterm, and small-for-gestational-age babies, but increased the risk of having large-for-gestational-age babies.

Overall, econometric research has generally found Hispanic and black adolescents and adults to be more responsive to changes in cigarette prices than whites. However, it is possible that the differential response to price could reflect unobserved socioeconomic differences or other factors.

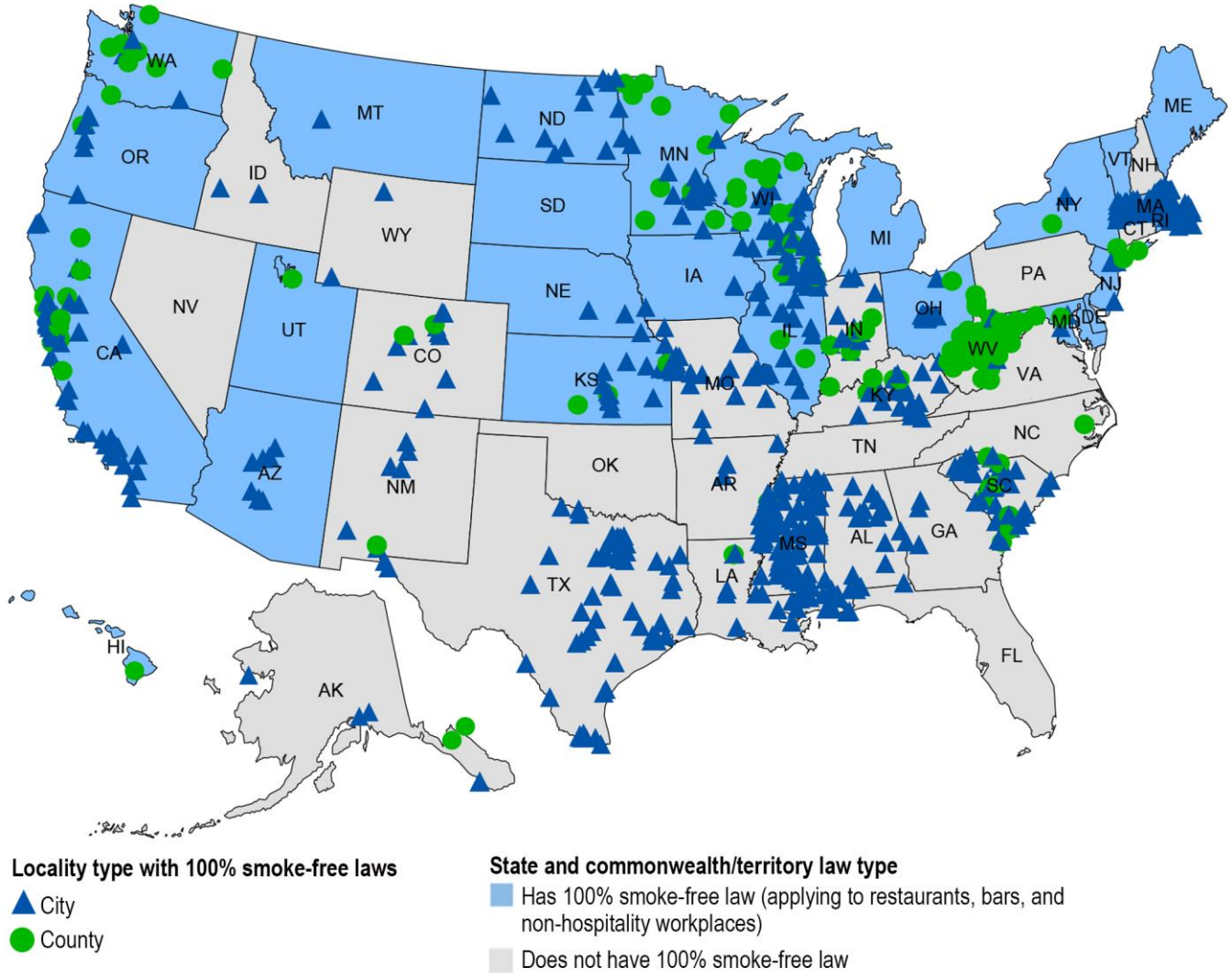
Smoke-Free Policy

Strong progress has been made over the past three decades in protecting the public from exposure to secondhand smoke (SHS). Legislation restricting smoking in public places and worksites has been enacted in many jurisdictions across the United States, and existing laws have been made stronger and more comprehensive.[199]

The earliest laws restricting smoking were intended to prevent fires and food contamination, but as evidence emerged about the adverse health consequences of SHS exposure, laws were adopted to protect public health. Momentum to establish new policies increased significantly with the release of the 1986 Surgeon General's report *The Health Consequences of Involuntary Smoking*, which concluded that exposure to SHS causes disease, including lung cancer, in healthy nonsmokers; that children of parents who smoke were at increased risk of respiratory diseases; and that simply separating smokers from

nonsmokers does not eliminate nonsmokers' exposure to SHS.[200] This report contributed to passage of federal regulations banning smoking on domestic airplane flights of 2 hours or less and eventually on virtually all domestic flights (in 1990) and all international flights (in 2000) departing from or arriving in the United States.[201] Findings from *The Health Consequences of Involuntary Smoking* were confirmed and expanded by many subsequent reports, including a second Surgeon General's report[202] and reports by the Environmental Protection Agency,[203] the California Environmental Protection Agency,[204] and the International Agency for Research on Cancer.[205,206] To protect children from exposure to SHS, federal law (the Pro-Children Act of 1994) prohibits smoking in indoor facilities which regularly provide services to children, including education, day care, health care, and early childhood development (e.g., Head Start, the Special Supplemental Nutrition Program for Women, Infants, and Children).[207,208] As shown in Figure 11.1, as of April 2017, about half of U.S. states had laws requiring workplaces, restaurants, and bars to be 100% smoke free.

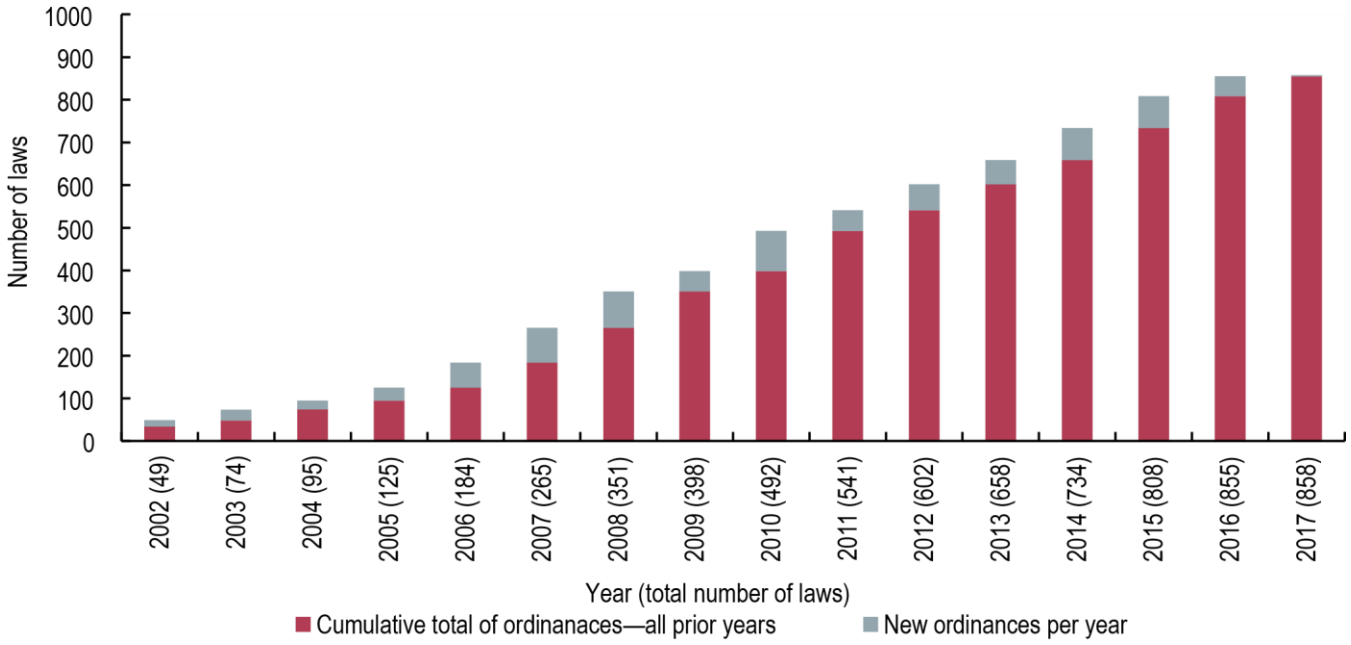
Figure 11.1 100% Smoke-Free Policies in the United States, 2017



Source: Adapted from Americans for Nonsmokers' Rights 2017.[209]

According to the Americans for Nonsmokers' Rights Foundation (ANRF), as of July 3, 2017, 36 states and the District of Columbia had laws in effect requiring non-hospitality workplaces, restaurants, bars, and/or state-run gambling establishments to be 100% smoke free.[209] Additionally, 1,395 municipalities had a 100% smoke-free provision in effect at the local level in workplaces, restaurants, and/or bars, and 915 municipalities required all three venues—workplaces, restaurants, and bars—to be 100% smoke free. ANRF estimates that as of July 3, 2017, 58.3% of the total U.S. population was covered by a state or local policy that made restaurants, workplaces, and bars 100% smoke free, and 65.5% of the population is covered by a policy that required restaurants and bars to be 100% smoke free.[209] Figure 11.2 illustrates the growth in the number of these comprehensive policies at the local level over the past two decades.

Figure 11.2 Local Smoke-Free Laws Covering Workplaces, Restaurants, and Bars, 2002–2017



Notes: This figure only includes ordinances or regulations that have effective dates through 2017, do not allow smoking in attached bars or separately ventilated rooms, and do not have size exemptions. The jurisdictions affected by county-level laws vary widely. Workplaces are defined as both public and private non-hospitality workplaces, including, but not limited to, offices, factories, and warehouses. Restaurants include any attached bar in the restaurant.
Source: Adapted from Americans for Nonsmokers' Rights 2017.[209]

In general, research has demonstrated that smoke-free state and local policies are self-enforcing and that compliance is high within a short time after implementation.[210–212] As a result, these policies are highly effective in reducing nonsmokers' exposure to SHS.[202,213]

In addition to protecting nonsmokers from exposure to SHS, smoke-free policies contribute to reducing cigarette smoking because they strengthen social norms against smoking, limit opportunities for smoking, and increase the inconvenience of smoking.[206,214] Comprehensive reviews of the research on the impact of smoke-free workplace policies by NCI,[215] the Community Preventive Services Task Force,[18,213] the Surgeon General,[202] and the International Agency for Research on Cancer,[206] among others, have found that these policies are effective in prompting cessation and in reducing cigarette consumption overall. For example, NCI Tobacco Control Monograph 21 concludes that "comprehensive smoke-free policies reduce exposure to SHS; compliance with these policies is generally high, and

public support for them is strong," and that "comprehensive smoke-free policies in workplaces reduce active smoking behaviors including cigarette consumption and smoking prevalence."[70,p.221]

A 2015 report by the CDC[216] estimated the percentage of the U.S. nonsmoking population exposed to SHS by evaluating levels of serum cotinine, a metabolite of nicotine and marker of SHS exposure. This report concluded that the prevalence of serum cotinine greater than or equal to 0.05 ng/mL in the nonsmoking U.S. population (ages 3 and older) declined significantly from 1999-2000 (52.5%) to 2011-2012 (25.3%). Much of this decline can be attributed to a significant increase in the number of state and local laws prohibiting smoking in indoor workplaces and other public places and to a decrease in smoking prevalence rates.[216] Increasingly, smoke-free policies are being extended to previously unregulated areas, including outdoor spaces (such as beaches, public parks, and outdoor dining areas of restaurants), private cars (especially when children are present), and multiunit housing.[70,p.204]

In addition, a growing number of households, including those with smokers, have adopted rules banning smoking in the home. In November 2016, the U.S. Department of Housing and Urban Development (HUD) finalized a rule that requires the nation's public housing agencies to implement a smoke-free policy for all public housing indoor areas. HUD has estimated that, as of 2016, over 600 U.S. public housing agencies have voluntarily implemented smoke-free policies in at least some of the properties they manage.[217] Such policies have the potential to protect the more than 7 million people who are served by public housing in the United States, many of them families with children.[218] By 2011, about 83% of U.S. households had smoke-free home rules in place, although a much higher percentage of households with no smokers had smoke-free home rules (95%) compared to households with smokers (61%).[217] Studies have also documented disparities in rates of adoption of home smoking bans. For example, using data from the TUS-CPS, Zhang and colleagues,[220] found that in homes with children, complete home smoking bans were less likely among households of single parents, low-income people, smokers, parents with less than a college education or without an infant in the home. Mills and colleagues[221] found that home smoking bans were less common in African American households than in other racial/ethnic households.

Impact of Smoke-Free Policy by Age

Numerous studies have shown that smoke-free policies effectively reduce smoking in specific age cohorts. To assess the association between statewide smoke-free policies and smoking prevalence among adolescents ages 12–17, Chriqui[222] used an index developed by the ImpacTeen Project (a partnership of nationally recognized health experts that focuses on youth substance use, obesity, and physical activity) that reflects the number of places covered by these policies and the extent of their restrictions, which range from no restrictions to a complete ban. Although the analysis did not control for other factors that affect smoking prevalence or for the potential for reverse causality between prevalence and state policies, the inverse relationship found by the authors between smoking prevalence and smoke-free laws is consistent with the growing body of multivariate research, discussed below, that does take confounding into account.[222]

Wasserman and colleagues[160] were among the first researchers to employ multivariate regression techniques to assess the impact of public smoking restrictions on teenage smoking decisions. Using data from the second NHANES (conducted 1976–1980) on 1,891 adolescents ages 12–17, the authors concluded that restrictions on smoking in public places have a strong, negative, and statistically significant ($p<0.05$) impact on the total number of cigarettes smoked by adolescents; restrictions on

smoking were found to have a much larger effect on adolescents' decision on whether to smoke or not than on the average number of cigarettes they consumed.

Chaloupka and Grossman[156] used data from the 1992, 1993, and 1994 MTF surveys of 8th-, 10th-, and 12th-grade students to estimate smoking prevalence and smoking intensity demand equations. They found that relatively strong restrictions on smoking in public places significantly reduced the probability that adolescents would smoke but had little impact on the average number of cigarettes adolescents smoked. However, these researchers found that restrictions on smoking in schools reduced average cigarette consumption among young smokers.

Tauras and Chaloupka[163] used longitudinal data from the 1976–1995 MTF surveys to estimate cigarette demand equations for young adults in the United States. Using an individual fixed effects approach, this study concluded that restrictions on smoking in public places and private worksites had a strong negative impact on young adults' intensity of smoking and likelihood of smoking. Using data from the 1991–2000 MTF surveys of 8th-, 10th-, and 12th-grade students, Tauras[174] found that smoking restrictions in public places had a strong negative effect on the prevalence of smoking by young people and on smoking intensity among young smokers.

Chaloupka and Wechsler[223] used data from the 1993 Harvard Alcohol Study to examine the determinants of cigarette smoking among U.S. college students. In addition to estimating a total price elasticity of demand of –1.11 for college students, they found that laws limiting smoking in restaurants and schools significantly lowered smoking prevalence rates among college students and decreased their average cigarette consumption.

Using data from the 1992-1993 TUS-CPS for males of different age groups (16–24, 25–44, and >44 years), Ohsfeldt and colleagues[224] found that restrictions on smoking in public places and private worksites negatively affected the likelihood of current use of cigarettes or smokeless tobacco products among all three age groups, but the effect varied by age. Workplace laws were found to have a greater impact on males older than age 24 than on males ages 16–24, and restrictions on smoking in "other" places were also found to have more impact on the probability of smoking among males older than age 24 than among younger males.

Farkas and colleagues[225] also used data from the TUS-CPS but, in contrast to Ohsfeldt and colleagues, they used all the waves between 1992-1993 and 1995-1996 to examine the association between smoking restrictions in the workplace and at home and smoking among adolescents ages 15–17. They found that adolescents who lived in smoke-free households were significantly less likely to be smokers than adolescents who lived in households without a smoking ban, even after controlling for the presence of smokers in the household. Moreover, adolescents who worked in smoke-free worksites were significantly less likely to be smokers compared with adolescents whose workplaces had no smoking restrictions.

Other studies suggest that stronger smoking restrictions reduce the probability of smoking escalation among young people. For example, Wakefield and colleagues[226] used 1996 data on U.S. high school students to determine the relationship between smoking uptake and restrictions on smoking at home, at school, and in public places. Levels of smoking uptake were defined by individual smoking histories, current smoking levels, and intentions to smoke in the future. The authors found that more restrictive policies on smoking at home, more comprehensive bans on smoking in public places, and enforced bans

on smoking in schools were associated with a greater likelihood of being at an earlier stage of smoking uptake.[226]

Siegel and colleagues[227] examined the effect of local restaurant smoking regulations on the progression to established smoking among adolescents, starting with a cohort of 2,623 Massachusetts youths (ages 12–17) at baseline and following up with them 2 years later. Adolescents living in towns with complete smoking bans in restaurants had less than one-half the odds of progression to established smoking compared with youths living in towns with weak restaurant regulations.[227]

Tauras[174] used the longitudinal component of the MTF survey from 1976 to 1995 to examine the determinants of smoking uptake transitions among adults ages 18–32 in the United States. He examined the transition from non-daily to daily smoking and from light smoking intensity (defined as 1–5 cigarettes per day) and moderate smoking intensity (10 cigarettes per day on average) to higher intensities of smoking. Private worksite restrictions and restrictions on smoking in other public places were found to have a significant negative impact on the probability of progression to moderate smoking intensity levels among the age group studied.[174]

A series of papers by Tauras and colleagues[169] estimated the effect of tobacco policies on the smoking cessation decisions of young adults in the United States. Using the longitudinal component of MTF from 1976 to 1995, they found that stronger restrictions on smoking in private worksites and other public places increased the probability of successful smoking cessation by young adults. The average hazard ratio across the alternative specifications, which included private worksite restrictions as a covariate, indicated that individuals who resided in states that regulated smoking in private worksites had a 4.55% greater probability of quitting smoking than individuals who resided in states that did not regulate smoking in private worksites. Using the same MTF data, Tauras and Chaloupka[172] examined the impact of smoking restrictions on quit attempts by gender. They found that an index variable designed to capture the overall magnitude of smoke-free laws in each state had a positive, significant effect on attempts by young women to stop smoking, but the effect on quit attempts by males failed to reach significance.

Smoke-free laws and policies might also contribute to different age-related declines in exposure to SHS; overall, self-reported exposure to SHS declined by approximately 70% from the late 1980s through 2002 in the United States.[228] Similarly, studies show declines in nonsmokers' SHS exposure, as measured by serum cotinine levels. For example, the CDC estimates that the proportion of U.S. nonsmokers exposed to SHS declined from 52.5% in 1999-2000 to 25.3% in 2011-2012.[216] Despite reductions in exposure for all the groups that were examined, significant disparities in SHS exposure by age still existed during 2011-2012. SHS exposure was highest among children ages 3–11 (40.6%) and lower among children ages 12–19 (33.8%). The prevalence of SHS exposure among adults (age 20+) in 2011-2012 was 21.3%. Among children ages 3–11 years, 67.9% of non-Hispanic blacks were exposed to SHS compared with 37.2% of non-Hispanic whites, and 29.9% of Mexican Americans.[216]

The primary source of SHS exposure for children is the home.[228] A CDC study found that the proportion of households that have rules against smoking increased from 43% during 1992-1993 to 83% in 2010-2011.[229] This increase in households with smoke-free home rules coincided with a decline in self-reported home SHS exposure among nonsmokers age 4 and older from 20.9% in 1988–1994 to 10.2% in 1999–2004, and a decline in any SHS exposure (as measured by serum cotinine) from 83.9% to 46.4% over the same period.[230] However, the percentage decrease in home SHS exposure was smaller in

children than adults. Self-reported SHS exposure in the home declined 37.7% for respondents ages 4–11, 44.9% for those ages 12–19, and 59.8% for those age 20 and older.[230]

Children may also be exposed to SHS in cars and in locations such as private daycare centers, restaurants, shopping malls, grocery stores, and other locations. One study found that geometric mean cotinine levels decreased from 0.12 ng/mL in 1988–1994 to 0.05 ng/mL in 2003–2006 among children ages 4–16 who were not exposed to SHS in the home. [231] In comparison, mean cotinine levels did not change among those ages 4–16 who were exposed to SHS at home. The decline in cotinine levels among children without home SHS exposure may be attributed to the increase in state and local smoke-free laws.

Using data from the 1999–2006 NHANES, Dove and colleagues[231] examined the association between smoke-free policies and serum cotinine levels among nonsmoking children and youth (ages 3–19) with and without home SHS exposure. These researchers found that among nonsmoking youth who reported no exposure to SHS in the home (about 80% of the sample), those who lived in counties with extensive smoke-free policies were significantly less likely (0.61 times) to have detectable serum cotinine compared with youths living in counties without smoke-free policies. Almost all youth exposed to SHS in the home (about 20% of the sample) had detectable serum cotinine, and level of serum cotinine did not differ by county smoke-free policy status after adjusting for individual and county covariates (e.g., demographics, region of the country). These results emphasize the importance of both smoke-free policies and home smoking restrictions to reduce children's and youths' SHS exposure.

Impact of Smoke-Free Policy by Socioeconomic Status and Race/Ethnicity

Studies show that substantial disparities exist in nonsmokers' SHS exposure. For example, during 2011-2012, the CDC[118] found a significantly higher prevalence of cotinine concentrations (0.05–10 g/mL) among individuals living below the poverty level (43.2%) than for those at or above the poverty level (21.2%). Similarly, during 2011-2012, Homa and colleagues[216] found a higher prevalence of cotinine concentrations (0.05–10 ng/mL) among those with less than a high school degree (27.6%), a high school diploma or equivalent (27.5%), or some college or associate degree (21.2%) than among college graduates (11.8%). Moreover, during 2011-2012, the CDC found higher prevalence of cotinine concentrations (0.05–10 ng/mL) among non-Hispanic blacks (46.8%) than among Mexican Americans (23.9%) and non-Hispanic whites (21.8%).[216]

Between 1999-2000 and 2011-2012, declines in SHS exposure (defined as a serum cotinine level) were observed for all groups defined by race/ethnicity and socioeconomic status, but disparities in declines were also observed. In terms of race/ethnicity, declines in SHS exposure during this period were smallest among non-Hispanic blacks (36.6% decline; from 73.8% in 1999-2000 to 46.8% in 2011-2012), followed by Mexican Americans (46% decline; from 44.3% in 1999-2000 to 23.9% in 2011-2012), and non-Hispanic whites (56.2% decline; from 49.8% in 1999-2000 to 21.8% in 2011-2012).[216] By poverty status, declines in SHS exposure during this period were smallest among those living below the poverty level (39.7% decline; from 71.6% in 1999-2000 to 43.2% in 2011-2012) and larger for those living at or above the poverty level (56.6% decline; from 48.8% in 1999-2000 to 21.2% in 2011-2012).[216] Finally, by education, smaller declines in SHS exposure were generally observed for those with lower levels of educational attainment.[216]

Some studies have examined the differential effects of smoke-free policies on smoking by SES and racial/ethnic groups. Some evidence suggests that smoke-free laws have a weaker effect on smoking by low-SES individuals and racial/ethnic minority groups, compared to members of other groups. Chaloupka and Pacula[192] were the first to examine differences in the impact of tobacco control policies on adolescent smoking prevalence by race. Using data from the 1992, 1993, and 1994 MTF surveys, they found that smoke-free laws had a significant negative impact on smoking prevalence rates among adolescent white males, but no effect on adolescents of any other racial group. As an indicator of smoke-free laws, this study used the fraction of the population in the person's county of residence that was subject to state or local smoke-free restrictions in private worksites, restaurants, retail stores, schools, and other public places. In their models, the authors controlled for the existence of smoke-free laws but not their level of enforcement. If differential policy enforcement were correlated with race/ethnicity, this could account for the lack of impact of smoke-free policies on some groups.

Workplace smoke-free restrictions reduced smoking prevalence and smoking intensity across all demographic groups in nearly all industries studied by Farrelly and colleagues[232] using data from the 1992-1993 TUS-CPS. With respect to race/ethnicity, the authors found that a 100% smoke-free workplace policy decreased the prevalence of smoking by 6.2% for non-Hispanic whites, 3.5% for non-Hispanic African Americans, 1.5% for Hispanics, and 5.7% for other race/ethnicity groups. The percentage declines in smoking prevalence associated with a smoke-free environment were observed across various levels of educational attainment, with the largest effects found among workers with a college degree (28.4%), and the smallest effects among high school dropouts (13.7%).

Several studies have considered the combined effects of smoke-free laws and cigarette prices on groups of different SES and race/ethnicity. Dinno and Glanz's 2009 study[233] concluded that smoke-free laws and cigarette prices reduce smoking prevalence and intensity in a similar fashion for all socioeconomic and racial/ethnic groups. Using the February 2002 wave of the TUS-CPS, they estimated models that not only controlled for smoke-free laws, cigarette prices, and individual characteristics but also included interaction terms among the policy variables (e.g., smoke-free laws, prices) and among educational attainment, household income, and race/ethnicity. Both smoke-free laws and cigarette prices were found to have a significant negative influence on smoking prevalence and intensity for the overall population. In general, the interaction terms indicated that the effects of smoke-free laws and cigarette prices did not change with educational attainment, household income, or race/ethnicity. The one exception was the significant interaction term between smoke-free laws and being black, implying that blacks could have a differential response to smoke-free laws than the general population. The authors further stratified the regression by employment status and found that the interaction term (between being black and smoke-free laws) became nonsignificant for employed as opposed to unemployed blacks. Some caution should be used when interpreting the results of this study because the analysis used only 1 month of survey data.

The response is also likely to depend on whether an individual's workplace is covered by a smoke-free law; disparities in workplace coverage have been documented. For example, Shopland[234] found that Hispanic males (58.8%) and Hispanic females (69.9%) were less likely to be covered by workplace smoking policies compared with white males (63.5%), white females (74.1%), black males (63.5%), and black females (72.2%). Occupation also plays a role; blue-collar workers were less likely to be protected by smoke-free laws than white-collar workers.[235]

Tobacco Treatment Policy

The health benefits of smoking cessation have been well established,[236–239] and results from the 50-year follow-up to the British Doctors' Study have demonstrated the importance of quitting smoking before age 30 to avoid most tobacco-related mortality.[236,237] Additionally, research has clearly shown that quitting smoking is beneficial regardless of age or health status. The 1990 Surgeon General's report *The Health Benefits of Smoking Cessation* concludes that "smoking cessation has major and immediate health benefits for men and women of all ages. Benefits apply to persons with and without smoking-related disease."[239,p.8]

The first U.S. Clinical Practice Guideline, *Smoking Cessation Clinical Practice Guideline, No. 18*, was published in 1996 and updated in 2000 and 2008.[240–242] These guidelines highlight the "5 A's" (Ask, Advise, Assess, Assist, Arrange) and recommend counseling and pharmacotherapies as effective interventions for tobacco cessation. The 2008 update to the Guideline states that the chances of successful quitting could be significantly improved by use of additional smoking cessation pharmacotherapies; stronger support for counseling interventions, including quitlines; and health care coverage of tobacco dependence treatments.[242]

To complement the Clinical Practice Guidelines, the Task Force on Community Preventive Services recommended (1) increasing the unit price of tobacco products, (2) reducing client out-of-pocket costs for effective cessation therapies, (3) developing multicomponent interventions that include client telephone support, (4) using mass media education campaigns combined with other interventions, and (5) incorporating provider reminder systems and provider education programs.[213] However, the use of evidence-based treatments for smoking cessation remains low, despite these strong recommendations, meta-analyses of clinical studies demonstrating the efficacy of cessation treatments,[243] and the increasing availability of low-cost cessation treatments. In 2015, approximately 55.4% of adult smokers reported a past-year quit attempt, and of those, nearly 70% attempted to quit without counseling or a pharmacologic treatment.[244]

NHIS data show that use of evidence-based cessation treatments among adults also varies by age, race/ethnicity, and access to health care. In 2015, young adults (ages 18–24), non-Hispanic blacks, Hispanics, and people without health insurance were less likely to use counseling and/or medications to quit smoking compared with older adults, non-Hispanic whites, and insured individuals.[244] A study of patients served by health centers supported by the Health Resources and Services Administration found that in 2009, approximately 31% of adult health center patients were current smokers; 83% of current smokers reported wanting to quit smoking, and 68% reported receiving some form of tobacco cessation counseling.[245] Patients with two or more chronic health conditions were twice as likely to report receiving counseling, compared with patients with no chronic health conditions. Additionally, Hispanic patients were less likely to receive cessation counseling, compared with smokers of other race/ethnicities. These results support a growing body of evidence demonstrating that younger smokers, racial/ethnic minorities, individuals with socioeconomic disadvantages, and those without health insurance are less likely to receive advice to quit from a health professional.[229,246–250]

The low rate of using evidence-based cessation treatments has also been shown to persist more among racial/ethnic minority groups than non-Hispanic whites, regardless of insurance status.[247,251] Of particular concern is the limited use of evidence-based treatments by young adults[252] and non-Hispanic blacks. Although the frequency of quit attempts is greater among these groups than among older adults and non-Hispanic whites, members of these groups tend to experience lower rates of cessation

success.[229,253] Also, low-SES smokers are less likely to make a quit attempt and to quit successfully, compared with other smokers.[229] As discussed in chapter 2, smoking prevalence among LGBT populations is higher than the national average, but no studies of cessation rates and use of evidence-based cessation treatment among LGBT populations were identified.

Data from the National Ambulatory Medical Care Survey and NHIS indicate potential disparities in screening for tobacco use; 62.7% of outpatient visits during 2005–2008 included screening, with Hispanic patients less likely to receive screening than non-Hispanic white patients.[248] Tobacco use screening also varied by health insurance status: Patients covered by private insurance, Medicare, Medicaid, or SCHIP, and self-payers were more likely to be screened than patients with another source of coverage or unknown insurance status, or patients who had a no-charge visit or whose visits were paid for by charity.

Building Consumer Demand for Cessation Services

The 2006 NIH State-of-the-Science Conference on Tobacco Use included a focus on increasing the reach, use, and impact of evidence-based cessation services by building consumer demand for these services, including among diverse populations.[254] The conference detailed effective strategies to promote cessation products and services to smokers, and emphasized the need to make treatments more attractive to smokers so as to create sustained demand. The 2007 Institute of Medicine report *Ending the Tobacco Problem: A Blueprint for the Nation*[17] further emphasized the need to expand treatment use by aligning cessation treatments and the policies that support their use and delivery across all levels of the health care and public health systems. This report called for a coordinated, comprehensive strategy to dramatically increase the number of smokers who quit each year.[17]

The data indicate significant differences in the use of tobacco cessation counseling and treatments by type of insurance coverage, SES, race/ethnicity, and age.[229,246,247,249,250] A number of strategies have been suggested to ensure that tobacco cessation is consistently integrated into health care delivery:

- Using a tobacco user identification system in every clinic[241,242,255–257]
- Providing education, resources, and feedback to promote provider intervention[241,255,257]
- Dedicating staff to provide tobacco dependence treatment and assessing its delivery in staff performance evaluations[241,255,257]
- Promoting hospital policies that support and provide inpatient tobacco dependence services[241,255,257]
- Including tobacco dependence treatments (counseling and medication) identified as effective in the guidelines as paid or covered services for all subscribers to health insurance packages.[241–243,255,257]

Workplace interventions have the potential to reduce TRHD by reducing barriers to treatments in certain populations, including blue-collar and service workers, who have higher smoking rates[258,259] and lower levels of protection from SHS.[230] Cessation interventions such as individual counseling and pharmacological treatments have been shown to be as effective in the workplace as in other settings.[260] The evidence on interventions addressing social and environmental support, workplace competitions and incentives, and comprehensive programs is limited and does not show a clear benefit in helping smokers quit at work, largely because a significant difference in cessation between treatment and control groups has not been found.[260] Nine of ten studies of worksite social support, environmental support, and

incentives failed to detect any differences between treatment and control groups, which was also the case in seven of eight studies of multicomponent programs aimed at improving employees' health, including smoking cessation programs.[260]

In 2010 the U.S. Department of Health and Human Services published *Ending the Tobacco Epidemic, A Tobacco Control Strategic Action Plan for the U.S. Department of Health and Human Services,* a strategic action plan to coordinate tobacco control efforts across the Department.[261] The report noted the significant disparities in smoking prevalence that exist by race/ethnicity, SES, geographic area and many other factors, and proposed a number of strategies to reduce TRHD, including: (1) expanding Medicaid and Medicare health insurance coverage to include comprehensive, evidence-based cessation treatment; (2) targeting interventions to locations serving high-risk populations—for example, public housing, substance abuse facilities, mental health facilities, and others; and (3) expanding research and surveillance related to high-risk populations, including American Indians/Alaska Natives and other racial/ethnic minority groups, LGBT groups, low-SES individuals, and individuals with mental illnesses.[261]

The Patient Protection and Affordable Care Act of 2010 (ACA)[262] includes tobacco cessation in several sections related to disease prevention, including (1) prohibiting states from excluding coverage for tobacco cessation medications from their Medicaid programs, (2) covering the cost of tobacco dependence treatments for pregnant women without requiring cost-sharing or co-pays, and (3) eliminating copayments for Medicare preventive services that are rated A or B by the U.S. Preventive Services Task Force, which includes tobacco use counseling and treatment for all adults.[263] These changes were also part of the August 2010 Medicare expansion of coverage for smoking and tobacco use cessation counseling to beneficiaries who use tobacco but do not have signs or symptoms of tobacco-related disease.[264]

Evidence for the potential impact of broad access to cessation services comes from the United Kingdom, whose National Health Service (NHS) "stop smoking services" provide behavioral support and medication to adult smokers. In 2010-2011 these services were provided to 700,000 adult smokers, representing 8% of the 9 million smokers in the United Kingdom, with 54% of stop smoking service users eligible for free prescription medication.[265] Subsequent fully adjusted analyses of the effectiveness of the NHS provision of specialist behavioral support plus medication indicated that smoking abstinence in this group was more than three times higher than in users who were not in treatment, though smokers in the two lowest social grades made less use of the NHS specialist services than the other treatment options (medication and brief advice, over-the-counter nicotine replacement therapy [NRT], and no treatment).[266]

In the United States, expanded coverage of cessation treatments at the state level has produced substantial population-level effects on smoking prevalence. In Massachusetts, mandated health care insurance and the inclusion of tobacco cessation medications and behavioral interventions into the MassHealth Medicaid program have helped reduce access barriers, resulting in a 26% decrease in state smoking prevalence over approximately 2 years (July 1, 2006–December 31, 2008). Broad promotion of tobacco cessation coverage led 37% of Medicaid smokers to use the cessation benefit during this period.[267] Longitudinal analyses demonstrated significant annual declines in Massachusetts Medicaid claims for hospitalizations for acute myocardial infarction and acute coronary heart disease during these years.[268] Expanding Medicaid coverage for cessation treatments helped Massachusetts reach low-income, low-education smokers, who are less likely to quit successfully. Richard and colleagues[269]

found that the short-term return on investment for the smoking cessation program in Massachusetts was between $1.63 and $1.84 per person.

Similar results were found in New York City, where large-scale distribution of free nicotine patches in 2003 was shown to reach a diverse population of smokers. Of the recipients, 64% were non-white, foreign-born, or resided in low-income neighborhoods.[270] Abstinence at 12-months among New York City quitline callers who received the free nicotine patches was shown to be 1.78 times higher compared to quitline callers a year earlier who did not receive the free nicotine patches,[271] providing further support for the potential of widespread cessation services to improve cessation success in lower income, racial/ethnic minority smokers.

In 2012, Athar and colleagues[272] estimated the increase in the number of quitters and the savings in Medicaid medical expenditures associated with expanding Medicaid coverage of nicotine replacement therapy to the entire adult Medicaid population in Alabama, Georgia, and Maine. By expanding Medicaid coverage of nicotine replacement therapy from only pregnant women to all adult Medicaid enrollees, the state of Alabama could expect 1,873 to 2,810 additional quitters ($526,203 and $789,305 in savings of annual Medicaid expenditures); Georgia, 2,911 to 4,367 additional quitters ($1,455,606 and $2,183,409 in savings); and Maine, 1,511 to 2,267 additional quitters ($431,709 and $647,564 in savings).

Additional Policy Interventions to Close the Access Gap

Additional types of policy interventions, including adequate funding for the use and promotion of state-sponsored telephone quitlines,[273] could also help narrow the gap in access to cessation treatments. Studies of quitlines in California[274] and other states[273] have shown that more African Americans than white smokers used quitlines, indicating that quitlines could play an important role in reducing disparities in cessation. A randomized controlled trial of quitline effectiveness conducted by the American Cancer Society also showed that African American users had quit rates similar to non-Hispanic whites, demonstrating the potential of using telephone counseling to reduce disparities in cessation by race/ethnicity.[275]

Facilitating access to quitlines might not automatically result in increased demand for quitline services among all smokers. A study of the Colorado quitline showed that Latino smokers, LGBT smokers, those with private insurance, and those with no insurance were more likely to report never intending to call the quitline, compared with non-Latino smokers, heterosexual smokers, and smokers with Medicaid insurance.[276] This study noted that the predominant reason for not intending to call the quitline was "no need for assistance,"[276] underscoring the need to increase smokers' awareness of the quitting process and to improve their knowledge of available treatment options.

Other policy interventions that may improve the use of cessation treatments include health care system changes to prompt, guide, and incentivize tobacco treatments; promotion of evidence-based treatments via the Internet; and research to improve the long-term effectiveness of evidence-based treatments via individually tailored, stepped-care approaches.[272] Recent studies modeling the implementation of these policies to improve the reach, use, and impact of smoking cessation treatments show that in isolation, each policy could increase the population quit rate between 4% and 40%.[277] However, implementing all five policies in combination was projected to increase the quit rate 150% over the baseline population quit rate.

The 2009 American Recovery and Reinvestment Act, which included the Health Information Technology for Economic and Clinical Health (HITECH) Act, could also enhance smoking cessation efforts. One of the major goals of the HITECH Act was to accelerate the adoption of electronic health records (EHR) through the creation of Medicaid and Medicare EHR Incentive Programs, with payments totaling $27 billion over 10 years to "meaningful EHR users."[278] The HITECH Act defines three stages of "meaningful use," with Stage 1 (2011-2012) focusing on capturing patient data and sharing that data with the patient or other health care professionals.[279] In line with the 2008 Public Health Service recommendations,[242] one of the 15 core objectives constituting "meaningful use" in Stage 1 is to "record [the] smoking status for patients 13 years or older."[279,p.42]

Currently (2017), few studies address the influence of EHR tobacco screening on health care provider and patient behaviors related to smoking cessation,[280] but some have shown increases in the documentation of tobacco use status,[280] delivery of the "5A's"[281,282] or other provider counseling, referrals to a quitline,[283,284] and the proportion of patients setting a quit date. Tobacco screening via EHR systems has strong potential to reduce disparities in ascertaining smoking status and delivering evidence-based treatments, but additional research is needed to address whether widespread EHR systems will translate into increased cessation-promoting behaviors among providers and patients.[280]

Chapter Summary

As this chapter describes, tobacco control policies at the federal, state, and local levels have considerable potential to reduce TRHD.

In 1989, California became the first U.S. state to implement a comprehensive tobacco control program. The experience of California and other states has demonstrated that these programs reduce tobacco use, reduce tobacco-related mortality, and reduce health-care spending. Strong empirical evidence also shows that comprehensive tobacco control programs have had significant effects on smoking prevalence rates across SES and racial/ethnic groups. Although the CDC provides guidance for appropriate spending on state-level programs, states typically appropriate considerably less than what is recommended.

The 1998 Master Settlement Agreement between the Attorneys General of 46 states, 5 U.S. territories, and the District of Columbia with the nation's major tobacco companies, provided financial compensation to the states, imposed some restrictions on the marketing practices of the participating manufacturers, created the American Legacy Foundation (renamed the Truth Initiative in 2015), broadened access to previously internal company documents, and disbanded two industry-funded research groups. The specific impact of the MSA on TRHD has not been well studied.

A variety of federal policies also have the potential to reduce TRHD. The 2009 Family Smoking Prevention and Tobacco Control Act provided the FDA with the authority to regulate the manufacture, marketing, and distribution of tobacco products. The Act has strong potential to lessen tobacco's impact on diverse populations and to reduce TRHD, through steps such as requiring graphic health warnings on cigarette packaging and advertising, and through promulgating tobacco product standards. In addition, the Act preserves the ability of states and localities to enact requirements in certain domains that are more stringent than those promulgated by FDA. This is especially important given the leading role often played by states and localities in putting forward innovative tobacco control policies, including those focused on reducing TRHD.

Youth access policies, when consistently enforced, can reduce commercial access to tobacco products among underage youth, and can help reduce youth smoking prevalence. Cigarettes are accessible to youths by a variety of means, with retail stores still an important source despite local and state laws banning sales to minors, and despite several relevant federal laws (the 1992 Synar Amendment, the 2009 Tobacco Control Act, and the 2010 Prevent All Cigarette Trafficking Act). Some evidence suggests that compliance with youth access laws depends on the demographic characteristics of the young purchasers; differential compliance with these laws can be an important factor contributing to disparities in adult tobacco use because today's youth consumer is tomorrow's adult consumer. Although curtailing commercial sources of cigarettes for youths is necessary, it is not sufficient to eliminate the supply of cigarettes to youths, because social sources of cigarettes often remain available. This chapter includes a discussion of efforts by states and localities to reduce tobacco use, especially among youth, by banning the sale of flavored tobacco products, and by increasing the minimum legal age of sale of tobacco products to 21 years.

A very strong body of evidence demonstrates that significant increases in tobacco taxes are the most consistently effective policy tool to reduce tobacco use among both youth and adults. Youth, young people, and low-SES populations are especially sensitive to significant tax and price increases, highlighting the potential for tobacco taxes to contribute to reducing TRHD.

The empirical evidence demonstrates that smoke-free policies are effective at protecting nonsmokers from exposure to SHS and in reducing cigarette smoking among adolescents and adults alike. As of 2017, nearly 60% of the U.S. population was covered by a state or local policy prohibiting smoking in all workplaces, including restaurants and bars. Despite this, disparities in coverage remain, especially by geographic region. Some evidence suggests that the impact of smoke-free policies may differ by SES and by race/ethnicity; smoke-free laws appear to have a weaker effect on smoking among low-SES individuals and/or racial/ethnic minority groups, in part because individuals in these groups are less likely to work in venues covered by these policies. Adoption of smoke-free home rules, often with the purpose of protecting infants and children in the home, are increasingly common. As of 2011, more than 80% of all U.S. households had adopted such rules, although nonsmoking households were far more likely to have adopted them than smoking households (95% vs. 61%). Differences in adoption of smoke-free home rules were also found by SES, race/ethnicity, and other demographic variables, contributing to differential exposure to SHS among children and later TRHD.

Quitting smoking is beneficial, regardless of the individual's age or health status, and effective tobacco dependence treatments are available. However, the use of evidence-based treatments is quite low, and varies by age, race/ethnicity, and access to health care; in general, younger smokers, racial/ethnic minority groups, low-SES people and people without health insurance are less likely than others to receive advice to quit from a health professional. As the experience of Massachusetts has shown, expanded access to tobacco dependence treatment through a state's Medicaid program can have a substantial impact on reducing tobacco use and tobacco-related disease among low-SES smokers.

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Section IV
Societal Level Influences on Tobacco Use

Chapter 12
Simulation Modeling of Tobacco-Related Health Disparities: *SimSmoke*

Contents

Tables

Introduction

Racial/ethnic disparities and socioeconomic status (SES), at both the individual and the community levels, are generally related to poorer health outcomes such as higher risks of heart disease and cancer.[1,2] Smoking is an important risk factor for these and other chronic diseases, and as discussed in chapter 2, smoking rates remain high among people with low education levels and income.[3–10] Low education and income have also been linked to a lower rate of quit attempts and quit success.[11–13] Additional information on relationships between race/ethnicity, SES, and tobacco-related health disparities (TRHD) is presented in chapters 2 through 9. Chapters 8 and 9 discuss the relationship between SES and TRHD in detail.

Extensive research has demonstrated that tobacco control policies are an important tool in reducing smoking rates. Since 1997, smoking rates in the United States have declined substantially, with much of this reduction attributable to public policies such as tobacco product price increases, mass media anti-tobacco campaigns, and smoke-free laws.[4,8,14–16] States with strong tobacco control policies, such as California, Massachusetts, Minnesota, Hawaii, and Arizona, have seen particularly large reductions in smoking rates.[17–20] There is evidence that most traditional policies are effective in reducing smoking rates among low-SES groups, but smoking rates are still high among low-SES groups and certain racial/ethnic groups (see chapter 9).[4,5,8,9] Increasingly, national and state programs have focused on reducing smoking among particular sociodemographic groups.[5,21,22]

To examine the effects of tobacco control policies on tobacco use, many investigators have used simulation models. Statistical evaluations have limited ability to distinguish the effects of policies on smoking rates, thus most statistical evaluation studies have examined the effect of only one or, at most, two policies (e.g., studies by Hu and colleagues,[23,24] and Farrelly and colleagues 2003[25]). Simulation models combine information from different sources, such as various policy evaluation studies, and information on policy levels to examine how the effects of public policies unfold over time in complex social systems.[26,27] Simulation models examining the effects of tobacco control policies have been developed by Mendez and Warner,[28,29] Tengs and colleagues,[30–32] Ahmad,[33] Ahmad and Billimek,[34,35] and Levy and colleagues.[15,27,36,37] Levy and colleagues' *SimSmoke* model simultaneously considers a broad array of public policies[38] and has been applied in many countries[15,16,39–48] and U.S. states.[18–20,49–51]

This chapter discusses a modified version of the *SimSmoke* tobacco control simulation model that was developed to examine trends in smoking rates related to SES disparities and the potential effect of tobacco control policies on smoking trends in the United States. *SimSmoke* is easily programmed to project outcomes for the total population and for subpopulations (by age and gender), thereby identifying target groups that may need special policy attention. *SimSmoke* shows likely trends in rates of smoking and rates of smoking-attributable deaths in the absence of policies, and how specific policies or groups of policies may alter these rates.[15,37,52–58] The modified version of *SimSmoke* discussed in this chapter examines policies in seven areas: cigarette taxes, smoke-free laws, mass media anti-tobacco campaigns, marketing restrictions, health warnings, cessation treatment policies, and enforcement of youth access laws. This chapter also examines the effect of a combination of these policies.

As this monograph describes, many sociodemographic, psychosocial, environmental, and biological factors may help explain TRHD. As discussed in prior chapters, disparities may be related to SES or race/ethnicity, among other factors. As reviewed in chapter 9, there is an inverse relationship between smoking status and the two primary measures of SES: education and income. The relationship of smoking to race/ethnicity is more complex; Hispanics, African Americans, Native Hawaiians and Other

Pacific Islanders, and American Indian and Alaska Natives are disproportionately represented in lower SES strata compared to whites and Asian aggregate groups. Consequently, this chapter focuses on SES; the potential for variation by race/ethnicity is considered in the Conclusions section.

The Modified *SimSmoke* Model: Methods

The *SimSmoke* model is designed to project smoking rates and deaths attributable to smoking.[36,38] This section first describes the income quintiles used in the development of the models, and particularly the income quintiles developed for the two lower income quintiles. This section then describes the populations of interest: smokers, never-smokers, and former smokers by age and gender. Next is a discussion of the two *SimSmoke* models, a population model and a smoking model. In the following section, policy modules for the input of policy parameters are described. A discrete-time, first-order Markov process is employed to project future population growth and changes in smoking rates over time that could be caused by tobacco control policies and prior smoking patterns.

Income Quintiles

SES is often categorized by income quintiles or levels of education (e.g., less than high school, high school, some college, undergraduate degree, some graduate training, graduate degree). The models used in this chapter are defined in terms of income quintiles because education levels are generally increasing, whereas income quintiles are a relative measure and thus a more stable measure over time. Further, for youth and young adults, family income can be expected to more closely reflect SES than education.

Two income disparities models, pertaining to the two lower income quintiles in the United States, were created. The policies used in the models are the same since both were applied at the national level during the same time span. However, although the same procedures are used to derive smoking prevalence and rates of initiation and cessation, these rates differ because the models pertain to different income groups with different rates. Both models begin in 2006, using the 2006-2007 Tobacco Use Supplement to the Current Population Survey (TUS-CPS).[59–61] This survey had a sufficiently large sample size to distinguish smoking rates by age and gender as well as SES classifications. The 2010-2011 TUS-CPS was used to calibrate the model.[60]

The basic *SimSmoke* model of population, smoking, and policies was programmed using Excel software.

Population Model

U.S. population data for 2006 were obtained from the Census Bureau for both males and females for each age from 0 through 84, and in the 85-and-older age group. The data were not modified for use in the models. Given that the models pertain to income quintiles, the population estimate for the second-lowest income quintile was first estimated by simply dividing the U.S. population by 5. This method may impart a bias, since age distributions may vary by income quintile. Accordingly, the 2006-2007 TUS-CPS populations by income quintile were used to adjust the age groups 15 and above,[59] and Census income-specific population data on children per household were used to adjust the age group 0–14.[62] After categorizing the data by quintile, the percentage difference by age group in the lowest and second-lowest income quintiles (also referred to as the first and second quintiles relative to the total population) was obtained. Compared to other income quintiles, the first and second quintiles generally had larger proportions below age 24 and at or above age 55.

Fertility and mortality rates were obtained from the Centers for Disease Control and Prevention (CDC).[63] To reflect an average value over time, the 2010 fertility and mortality rates were used for all years. Average fertility rates, calculated by income quintile relative to the average fertility rates for all quintiles, were 1.4 for the lowest quintile and 1.07 for the second-lowest quintile.[64] (Similar variations have been found using education groups.[65]) Fertility rates differentiated by age groups (e.g., 14, 15–17, 18–19, 20–24, 25–29, etc.) were adjusted to reflect the overall higher fertility rates in these two income quintiles. Mortality rates were distinguished by age and gender. Studies generally find that people at lower income and education levels, particularly those of working age, have higher mortality rates,[66,67] with the differential from higher levels of income and education increasing over time. Canadian studies conducted in 2012 and 2013 on data collected between 1991 and 2006 found that mortality rates in the lowest income quintile were 31% higher for males and 26% higher for females than average mortality rates in the general population; and mortality rates in the second-lowest income quintile were 6% higher for males and 4% higher for females than population rates.[68–70] Similar results were obtained by a 2008 U.S. study (NHANES data collected 1988–1994 and 2001)[71] and in a study examining 2002 mortality rates of people younger than age 65 (i.e., premature mortality).[72]

For the modified version of *SimSmoke*, mortality rates by age and gender for ages 25 through 75 were adjusted upward, using the relative difference in the Canadian mortality rates to reflect the higher rates for the first and second income quintiles compared with the average death rates of the population.

Smoking Model

SimSmoke divided the population into the number of smokers, former smokers, and never-smokers in the 2006 baseline year. Smokers were defined as individuals who have smoked more than 100 cigarettes in their lifetime and are currently smoking either daily or on some days. Former smokers were defined as individuals who meet the 100 lifetime cigarettes threshold but are not currently smoking, and never-smokers are those who have not smoked 100 cigarettes in their lifetimes. Former smokers were further categorized by years since quitting (<1, 1, 2,…, 15, >15 years). Baseline estimates of smoking status were obtained from the 2006-2007 TUS-CPS.[59] These data using self-response weights were aggregated by smoking status (never, current, former smokers, and former smokers by years quit), gender, age group (15–17, 18–20, 21–24, 25–34, 35–44, 45–54, 55–64, 65–74, 75–84, and 85 and older), wave of the survey, and income category ($19,999 or less, $20,000–$39,999, $40,000–$59,999, $60,000–$99,999, and $100,000 or more). For former smokers, the years-quit categories in the data before converting to single years quit were: less than 1 year, 1 year to less than 3 years, 3 years to less than 5 years, 5 years to less than 10 years, 10 years to less than 15 years, and 15 years or more.

Next, the data for each wave were broken into income quintiles based on the 2006 Census Bureau figures. For 2006, the upper limits for the first four income quintiles (in 2006 dollars) were $20,035, $37,774, $60,000, and $97,032. The highest income quintile for each year did not have an upper cutoff (i.e., the highest quintile included any household earning above the upper limit for the fourth quintile). For 2007, the upper limits for the income quintiles increased to $20,291, $39,100, $62,000, and $100,000. Since the TUS-CPS was conducted in May and August 2006 and in January 2007, the 2006 and 2007 data were weighted to arrive at an estimated income distribution such that TUS-CPS income categories would be the closest match to the 2006 Census income quintiles. This resulted in an upper limit of $20,078 for the first quintile, $37,995 for the second quintile, $60,333 for the third quintile, $97,527 for the fourth quintile.

Separate data sets for each year were then created by age group and smoking status counts, including former smokers by years quit, for the 10 gender/income quintile categories. Because the income categories outlined in the TUS-CPS do not coincide with these income quintiles, linear interpolation was used to estimate the number of cases in the different income quintiles. Since the lowest TUS-CPS income response category was $0–$19,999, the lowest income quintile for 2006-2007 was estimated as $0–$20,078. To correct for the difference in brackets between the income quintile and the TUS-CPS classifications, the number of smokers in the lowest income quintile was estimated using the formula:

$$N_{Q1,2006/07} = N_{[\$0,\ \$19,999]} + [(\$20,078–\$20,000)/(\$39,999–\$20,000)] * N_{[\$20,000,\ \$39,999]}$$

where N represents the count for the respective age group/smoking status category in the income category denoted by the given subscript. Since the second-lowest TUS-CPS income category was $20,000–$39,999 and the second-lowest income quintile was $20,079–$37,995, the following formula was used for the second-lowest quintile:

$$N_{Q2,2006/07} = N_{[\$20,000,\ \$39,999]} * [1 – (\$20,079–\$20,000)/(\$39,999–\$20,000) – (\$39,999–\$37,995)/(\$39,999–\$20,000)]$$

The data from the lowest and second-lowest income quintiles were distributed into the three smoking status categories (never, current, and former smokers) by the age groups above, and the former smokers were distributed into the six years-quit categories mentioned above. Interpolation (moving average [MA] smoothing) was then used to distribute the age group smoking rates to single ages within each smoking status category as follows: 3-year MA for ages 15 to 26 and 5-year MA for ages 27 and older. Individuals younger than 15 years were considered never-smokers. The age group rates for current smokers, former smokers, and former smokers by years-quit were distributed to each single age within the respective age group. Cessation was tracked from age 24, since the relative mortality risks from smoking are not discernible for those quitting before that age.[73,74] Therefore, former smokers under age 25 were reclassified as never-smokers. With the above procedure, the age group estimate became the estimate for the mid-point age of each age group. To ensure that the prevalence rates of all smoking categories combined was 100%, the never-smoking rate was recalculated as 100% minus the sum of the current- and former-smoking rates.

Within the smoking model, individuals may evolve from never-smokers to smokers through smoking initiation. People are classified as never-smokers from birth until and unless they initiate smoking. Individuals may evolve from smoker to former smoker through cessation and may return to smoking through relapse. Relapse rates are proportional to the cessation rate (although independent of it), but are specific to age and the number of years since quitting.

Because estimating initiation and cessation rates at young ages is difficult, and to ensure stability and internal consistency of the model, initiation is measured net of quitting (i.e., as new smokers minus those who quit at each age) by computing initiation as the smoking prevalence at a particular age in the base year minus the smoking prevalence at the previous age in the base year. Initiation into the lowest and second-lowest income quintile model occurs until age 24.

Data on quit rates for individuals age 26 and older were obtained from the TUS-CPS. The measure of annual quit rates was based on the number of smokers who quit in the last year. The 2006 age group cessation data were initially interpolated by using 5-year MA smoothing from age 27. Data aggregated over all SES quintiles from the *SimSmoke* model were used to measure relapse rates for the lowest and

second-lowest income quintiles by duration of cessation for each of the years' quit groups.[73,75–77] However, in calibrating the model, relapse rates were checked by SES group.

Predicting Smoking-Attributable Deaths

Smoking-attributable deaths in *SimSmoke* were predicted using smoking prevalence rates and the relative risks of smokers and former smokers relative to nonsmokers, similar to standard attribution measures.[78,79] Specifically, the relative risks and prevalence rate of smokers and former smokers and the death rate in a particular age group were used to distinguish the death rates of never, current, and former smokers. The number of smokers at each age was multiplied by the death rate of smokers minus the death rate of never-smokers to obtain the excess deaths due to being a smoker. The same procedure was applied to each former smoker group using the former smoker death rate, and the results were summed over smoking groups for all ages to obtain the number of smoking-attributable deaths.

Deaths rates were distinguished by age, gender, and smoking type (never, current, and the six former smoker groups based on years quit, as above) using the data on mortality rates and smoking rates (as described above) and relative risk estimates for current and former smokers from the Cancer Prevention Study II.[77,80,81] While the mortality rates by smoking status used in this study are not adjusted for demographic and behavioral factors, such as diet and physical activity, other studies have found that the estimates are robust after controlling for these factors.[82,83] The relative risks may, however, vary for those groups; for example, relative risks may be higher if poor diet and other behavioral factors create greater risk from smoking (as has been found for lung cancer), or may be reduced to the extent that background risk is higher. Although no studies were found that specifically distinguish relative risks by income, Thun and colleagues[84] found that "the relative-risk estimates associated with current and former smoking among smokers with only a high school education are generally similar to or larger than those among smokers who are college graduates."[84,p.363] As a conservative measure, the relative risks for the two lowest income quintiles were assumed to be the same as the average relative risk for the entire population, but note that the estimates of smoking-attributable deaths from the two low-income groups reflect the higher mortality rates, especially in the lowest income group.

Policy Modules Methods

The model begins with policies in effect in 2006. Using the policy modules, the model then incorporated the effects of policies that changed between 2006 and 2014 by taking into account the changes in policies that were newly implemented and the effects of those changes. Seven policies were considered: cigarette taxes, smoke-free laws, mass media anti-tobacco campaigns, marketing restrictions, health warnings, cessation treatment policies, and youth access policies.

The effects of policies were estimated in terms of the percentage change (PC) in the smoking, initiation, and cessation rates relative to the initial rates:

$$[PC = (\text{post-policy rate} - \text{initial rate})/\text{initial rate}]$$

with $PC < 0$. Policies have their most immediate effect on smoking prevalence directly through cessation—that is,

$$Smokers_{t,a} * (1 + PC)$$

which may vary by age **a** and is assumed to occur in year **t** of the policy change. During each year after the first year in which the policy was in effect, the percentage reduction was also applied to the initiation rate as (1 + PC) and as a percentage increase (1 – PC) to the cessation rate. First-year quit rates continued to be elevated for each of the policies, because they reduce quantity smoked, which tends to increase cessation.[85] When more than one policy is in effect, the effects are multiplicatively applied—that is, $(1 + PC_i)*(1 + PC_j)$ for policies **i** and **j**—implying that the relative effect is independent of other policies, but the absolute effect is smaller when another policy is also implemented.

Policy descriptions and effect sizes are shown in Table 12.1. The effect sizes by income group were modified from those in the previous United States *SimSmoke* model[15,16] informed by the studies presented in chapter 11.

Table 12.1 Policy Inputs and Effect Size for *SimSmoke* Projection

Policy	Description	Effect Size*
Cigarette taxes		
Cigarette taxes	The state-level average price for a pack of cigarettes was computed as the weighted average of single pack, carton, and vending machine cigarette prices, including state excise taxes. Prices of both branded and generic cigarettes were used in the average.	Elasticity: ages 10–17: –0.60 ages 18–24: –0.45 ages 25–34: –0.30 ages 35–64: –0.15 ages 65 and above: –0.25
Smoke-free policies		
Worksite ban, well enforced	Cigarette use banned in all indoor worksites in all areas, with strong public acceptance and enforcement	4.5% reduction
Restaurant and bar ban, well enforced	Ban in all indoor restaurants in all areas	2.25% reduction
Bans in other places	Ban in 3 out of 4 of the following: government buildings, retail stores, public transportation, and elevators	0.75% reduction
Mass media anti-tobacco campaigns		
Highly publicized media campaign	Campaign publicized heavily on TV (for at least 2 months of a year) and on at least some other media, with a social marketing approach	6.5% reduction
Moderately publicized media campaign	Campaign publicized sporadically on TV and in at least some other media, and a local program	3.75% reduction
Low-publicity media campaign	Campaign publicized only sporadically in newspaper, on billboards, or some other medium	1.65% reduction
Marketing restrictions		
Comprehensive marketing ban	Advertising banned on television, radio, billboards, and in print; in-store displays, sponsorships, and free samples also banned	Prevalence: 5% reduction Initiation: 6% reduction Cessation: 3% increase
Total advertising ban	Advertising banned on television, radio, billboards, and in print	Prevalence: 3% reduction Initiation: 4% reduction Cessation: 2% increase

Table 12.1 continued

Policy	Description	Effect Size*
Enforcement	A government agency is designated to enforce the laws.	Effect size is reduced 50% if no enforcement
Health warnings		
Strong health warnings	Warning is bold and graphic, and covers at least 50% of the package	Prevalence: 4% reduction Initiation: 4% reduction Cessation: 8% increase
Weak health warnings	Warning does not include graphics, and covers less than one-third of the package	Prevalence: 1% reduction Cessation: 4% increase
Quitlines	A proactive quitline with nicotine replacement therapy and a campaign of publicity through the media	~1% reduction in prevalence, but a greater effect on cessation rates depending on level of publicity through the media
Youth access enforcement		
Youth access restrictions, strongly enforced and publicized	Regular compliance checks, heavy penalties, high visibility; vending machine and self-service bans	Prevalence and initiation only: ages <16: 30% reduction ages 16–17: 20% reduction
Youth access restriction, moderate enforcement	Compliance checks are conducted at least once per year per outlet, penalties are moderate, and the program receives some publicity	Prevalence and initiation only: ages <16: 15% reduction ages 16–17: 10% reduction

*Unless otherwise indicated, the effects are on prevalence in the first year, and on initiation and first-year quit rates during the ensuing years that the policy is in effect. The effect sizes are based on previous *SimSmoke* models, with modifications informed by the studies presented in chapter 11.

The effect of changes in U.S. policies was tracked from 2006 through 2014. Data pertaining to tobacco control policies were simulated at the state level for smoke-free laws, tobacco control campaigns, cessation treatment programs, and youth access enforcement. Since the 2006-2007 TUS-CPS data are from May, August, and January, smoking rates that represented the midpoint month, August, were used. To be consistent, policy data were set to their August levels of the particular year.

Cigarette Tax Module

In the tax module, prices were modeled as having constant proportional effects, derived from studies of demand elasticities.[55] The studies reviewed consistently obtained higher elasticities among people of low SES compared with high SES, in terms of both income and education; elasticities were generally between 50% and 100% higher for low- than for middle- and high-income individuals. Based on these studies, the model for the lowest and second-lowest income quintiles assigns a prevalence elasticity of –0.60 for both males and females younger than age 18; –0.45 for individuals ages 18 to 24; –0.30 for individuals ages 25 to 34; –0.15 for those ages 35 to 64; and –0.25 for those 65 and older.

U.S. prices (2006–2014) were measured by a retail price index weighted by brand sales, which includes generic cigarettes.[86] The prices were deflated using the Bureau of Labor Statistics' Consumer Price Index,[87] and the deflated prices were adjusted to the first quarter of 2006. From the average state price of a pack of cigarettes of $3.92 in 2006 prices, pack price rose slightly in 2007 and 2008, reaching $4.33, but with the federal tax increase in 2009, the average price increased sharply, to $5.15. Prices continued to rise in 2010 and 2011 but not as sharply as in 2009, reaching $5.60. In 2014, prices were at $6.03.

After adjusting for inflation (with base year 2014), prices increased from $4.60 to $6.03 between 2006 and 2014—a 26% increase. From 2006 to 2014, the average state and federal tax increased from $1.98 to $2.55 per pack, with the largest increases ($0.70 and $0.15) in 2008 and 2009. The model assumes that prices increase in absolute terms with the amount of the cigarette tax after the year 2014, and the non-taxed price increases over time with general price inflation.

Smoke-Free Laws Module

The smoke-free policy module examines the effect of smoking restrictions in three locations: worksites, restaurants, and other public places.[56] The module incorporated an interaction with publicity through the mass media anti-tobacco campaign module. The module takes into account the effect of enforcement, which is measured on a scale of 0–10, where 10 represents complete enforcement. The review in chapter 11 found that most studies obtained larger effects for whites than other racial groups, and Farrelly and colleagues[25] found much smaller effects for people with less education.

Based on the review in chapter 11 and the lower likelihood that low-SES smokers participate in the workforce or frequent restaurants, the effects for low-SES individuals were reduced by 25% compared to the population as a whole. The module predicted that prevalence rates would be 7.5% lower in locations with complete smoking bans that are strongly enforced and publicized through the media than in locations without smoke-free laws. Worksite laws were assumed to have the largest effect, reducing prevalence 4.5%; smoke-free laws in restaurants, pubs, and bars were assumed to produce a 2.25% effect, and laws covering other places each were assumed to have about a 0.75% effect. Partial worksite and restaurant bans were assumed to have one-third the effect of total bans.

Information on smoking bans that distinguish between venues in which they are imposed (private worksites, restaurants, and free-standing bars) was obtained for the years 2006 through 2009.[88] Each location was given a value of 3 if covered by smoke-free restrictions, a value of 2 if separate ventilated areas were required for smokers, 1 if only separate areas were required, or 0 if no smoke-free restrictions were in place. Locations with a value of 3 were given full weight, those with a value of 2 were given 0.5 weight, and those with a value of 1 were give 0.25 weight. The state data were aggregated to the U.S. level weighted by adult population. These data were updated using information from the Americans for Nonsmokers' Rights website: By 2014, smoke-free policies covered 65% of the U.S. population in the workplace, 77.3% of restaurant patrons, and 65.1% of customers in bars.[89]

Mass Media Anti-Tobacco Campaigns Module

The mass media anti-tobacco campaigns policy module[52] was based largely on experiences in California, Massachusetts, and several European nations, where media campaigns are part of a comprehensive policy (including local initiatives and other policies). Mass media campaigns were categorized based on campaign expenditures, a large part of which in most states is devoted to mass media campaigns publicized on TV and radio, and to local grassroots educational efforts. State per capita expenditures include revenues distributed to state health departments from state and federal government agencies such as the CDC, through its National Tobacco Control Program, as well as funding through nongovernmental organizations such as the Truth Initiative (formerly known as the American Legacy Foundation) and the Robert Wood Johnson Foundation, through its SmokeLess States Program. These data were updated with state expenditures data obtained from the Campaign for Tobacco-Free Kids.[90] The expenditure data were divided by yearly population from the Census and by the annual Bureau of Labor Statistics Consumer Price Index[87] so that they would represent inflation-

adjusted per capita measures and thus be comparable over time. States were categorized based on CDC-recommended levels, with <25% being low intensity, 25% to <75% medium intensity, and ≥75% high intensity.

Studies such as those by Al-Delaimy and colleagues[91] and Friedan and colleagues[92] found that these campaigns had greater effects with high-income smokers, but Levy and colleagues[93] found greater effects among females with less than a high school education. Estimates from the previous U.S. *SimSmoke* model were used.[16] Mass media policies directed at all smokers are assumed to yield up to a 6.5% reduction in smoking rates (relative to the initial level) for low-SES smokers, the same as for the entire population.[16]

Marketing Bans Module

The marketing bans policy module in *SimSmoke* corresponds to the bans on advertising, promotion, and sponsorship discussed in the World Health Organization's *Report on the Global Tobacco Epidemic: The MPOWER Package*.[94] This report distinguished four levels of marketing bans: (1) no policy, (2) minimal policy (banning some direct advertising), (3) moderate policy (banning direct advertising and some indirect promotions), and (4) complete policy (a total ban on direct and indirect marketing). The basis for policy effect estimates is described in studies by Levy and colleagues[95] and Blecher.[96] Where a complete policy is in effect (total ban), it is assumed that prevalence is reduced by 6%, cessation is increased by 3%, and initiation is reduced by 8%. With a moderate policy (direct advertising and some indirect promotions are banned), it is assumed that prevalence is reduced by 4%, cessation is increased by 2%, and initiation is reduced by 6%. With a minimal policy, it is assumed that prevalence is reduced by 1%, cessation is not affected, and initiation is reduced by 1%. The *SimSmoke* module also incorporates the effect of enforcement, which is measured on a scale of 0–100%, where 100% represents complete enforcement. The effects are reduced by up to 50% if the enforcement level is zero.

Marketing may be particularly effective among people of low SES, as indicated by evidence presented in National Cancer Institute (NCI) Tobacco Control Monograph 21, *The Economics of Tobacco and Tobacco Control*,[97] that advertising and marketing are targeted to minority groups, and that advertising bans are particularly effective in low- and middle-income countries.

In the United States, cigarette advertising on radio and television has been banned since 1971, but tobacco advertising is still allowed at the point of sale and in newspapers and magazines. Other forms of marketing, such as sponsorships, branding, and mail giveaways, are also still allowed. For the purposes of the marketing module, marketing restrictions are considered moderate, and enforcement is set at level 8.

Health Warnings Module

In the United States, health warnings were first placed on cigarette packs in 1966 as "Caution: Cigarette Smoking May Be Hazardous to Your Health." The current four rotating health warnings on cigarette packages and advertisements were mandated by the Comprehensive Smoking Education Act, enacted in 1984. In this module, health warnings are considered minimal.

The effect of implementing a strong warning that covers at least 50% of the principal display area of the pack and contains graphic images was considered. Evidence on the effects of health warnings on cessation behaviors is provided by Levy and colleagues[95] and has been strengthened by findings from

studies conducted since 2004[98–100] and two studies of Canadian health warnings completed in 2013 and 2014.[101,102] Evidence presented in chapter 11 indicates that health warnings can be as effective for low-income groups as for the rest of the population. With strong health warnings, prevalence is reduced by 4%, cessation is increased by 8%, and initiation is reduced by 4%. When the level is set to moderate, prevalence is reduced by 2%, cessation is increased by 4%, and initiation is reduced by 2%. When the level is minimal, prevalence is reduced by 0.5%, cessation is increased by 1%, and initiation is reduced by 0.5%.

Cessation Treatment Module

The cessation treatment policy module considered the effect of increased access to pharmacotherapies and behavioral therapies through quitlines that are well publicized (e.g., through a media campaign), including those that encourage follow-up with multiple sessions.[103,104] The module allowed for a direct prevalence effect, as well as a continuous effect on future 1-year quit rates. The effect on future 1-year quit rates was halved to reflect the greater use of treatments and effectiveness of interventions in the first year of the program. It was estimated that use of either behavioral therapy or pharmacotherapies alone doubles quit rates, and that their combined use quadruples quit rates. Proactive quitlines with follow-up can double the quit success rate of those making a quit attempt.

A study by Abrams and colleagues[103] indicates that quitlines that are highly publicized and provide free nicotine replacement therapy (NRT) to the qualified smoker attract 4% of smokers in the first year (range: 2% to 6%). Of those who used these quitlines and the free NRT, 30% were new quit attempts. The authors estimated that 50% of those who use treatments as a result of cessation-based policies would not otherwise have made a quit attempt.[103]

Quitline data for 2006 to 2014 obtained from the North American Quitline Consortium's Annual Survey of Quitlines indicated that all states had proactive quitlines with follow-up by 2006. By 2006, 24 states provided free NRT; 40 states were offering free NRT by 2009.[105] The number of states offering quitlines and free NRT has stayed relatively constant since 2009. This module did not consider other aspects of cessation treatment policies, such as financial access outside of quitlines and the role of brief health care provider interventions. Health care provider interventions are surveyed in the TUS-CPS, and information on Medicaid coverage for these interventions is provided in the American Lung Association's 2010 report on cessation coverage in the states.[106]

Youth Access Policies Module

For the minimum legal purchase age of 18, the model considered three levels of enforcement: (1) strongly enforced and publicized, (2) medium enforcement, and (3) weak enforcement. The module also incorporated the role of self-service and vending machine bans. When all policies are in full force, it was estimated a 20% reduction in prevalence and initiation for 16- and 17-year-olds and a 30% reduction for ages 10–15.[107] The review in chapter 11 did not find obvious differences in purchase rates and compliance by race or education. Enforcement and compliance estimates from previous models were used. Based on average compliance rates of about 90%, it was estimated that there has been a medium level of enforcement since 2006. Vending machine and self-service bans are both considered at 90% compliance since 2006.

Calibration Methods

To calibrate the model, predictions of smoking prevalence by age and gender from the model for 2010 and 2011 were compared to corresponding estimates from the 2010-2011 TUS-CPS.[60] Based on this comparison, the first-year cessation rate was adjusted. For those age 55 and older, relapse rates were generally lowered, leading to higher cessation rates and lower smoking prevalence over time. For those under age 55, relapse rates generally increased, yielding higher smoking rates.

Predicted Results of the Recommended Policies Compared With the Status Quo

This section presents estimates of smoking prevalence by income quintile from the TUS-CPS, then estimates the status quo scenario for the two lower income quintiles, and then discusses the differential effects of varying levels of tobacco control policies, in isolation and in combination, as a comprehensive tobacco control strategy.

Smoking Prevalence by Income Quintile

Smoking prevalence rates from the TUS-CPS are shown in Table 12.2 (for 2006-2007) and Table 12.3 (for 2010-2011), by income quintile. Except for the age category 75 to 84, smoking prevalence declined as income increased. For ages 18 and above in 2006-2007, smoking prevalence among males fell from 30.2% for the first income quintile to 10.6% for fifth quintile, and among females, from 22.7% for the first quintile to 8.3% for fifth quintile. For ages 18 and above in 2010-2011, smoking prevalence for males fell from 28.0% for the first income quintile to 8.8% for fifth quintile, and for females, from 20.8% for the first quintile to 6.7% for fifth quintile. The smoking rate declined for all income quintiles between 2006-2007 and 2010-2011—for example, the prevalence rates for males fell from 30.2% to 28.0% in quintile 1, and the rates fell from 10.6% to 8.8% in quintile 5.

Table 12.2 Smoking Prevalence by Age and Income Quintile, TUS-CPS, 2006-2007 (Percentages)

	Income Quintiles – Male					Income Quintiles – Female				
Age Group	1	2	3	4	5	1	2	3	4	5
18–20	24.3	28.0	20.7	13.0	8.8	21.4	13.6	18.6	10.5	5.7
21–24	29.3	23.1	27.5	22.0	11.1	26.2	19.3	16.5	21.6	11.7
25–34	30.7	31.2	24.2	19.0	13.6	28.9	23.7	16.5	13.4	6.9
35–44	41.8	30.6	25.4	15.2	11.5	31.8	22.6	21.8	14.5	8.9
45–54	43.1	31.4	24.8	19.7	11.1	33.5	26.7	22.3	15.8	9.5
55–64	31.4	26.4	21.4	15.8	8.0	24.9	16.9	14.9	12.9	7.6
65–74	23.3	12.5	10.6	7.9	5.0	14.7	10.5	9.0	6.3	2.9
75–84	11.5	7.8	6.9	3.9	10.7	5.2	3.3	4.5	1.7	5.4
85+	2.3	0.1	0.0	0.0	0.0	2.5	3.8	3.1	0.0	0.0
18+	30.2	25.3	22.3	16.7	10.6	22.7	18.4	17.2	13.8	8.3

Note: Quintile 1 is the lowest income group; quintile 5 is the highest.
Source: U.S. Department of Commerce 2008.[59]

Table 12.3 Smoking Prevalence by Age and Income Quintile, TUS-CPS, 2010-2011 (Percentages)

	Male Income Quintile					Female Income Quintile				
Age Group	1	2	3	4	5	1	2	3	4	5
18–20	22.3	15.7	15.6	12.1	9.5	16.5	11.8	10.8	9.5	3.3
21–24	26.1	24.6	22.0	16.0	14.0	21.9	19.4	14.5	10.0	7.7
25–34	32.2	26.1	19.5	17.3	12.0	26.1	21.2	15.2	10.4	6.8
35–44	32.9	25.2	20.4	13.9	7.5	27.0	21.2	15.0	11.1	6.1
45–54	38.1	27.7	22.5	15.1	9.0	31.3	24.0	19.2	13.7	8.5
55–64	30.7	24.1	18.6	13.0	8.5	22.6	19.1	13.3	9.3	6.2
65–74	19.3	12.3	10.2	10.0	5.0	14.8	10.1	7.5	7.0	4.5
75–84	7.4	6.6	4.9	2.6	4.5	6.0	4.4	4.0	2.4	4.2
85+	3.3	2.3	1.7	0.0	1.1	1.7	2.6	1.9	1.6	0.6
18+	28.0	21.8	18.1	14.0	8.8	20.8	17.1	13.7	10.5	6.7

Note: Quintile 1 is the lowest income group; quintile 5 is the highest.
Source: U.S. Department of Commerce 2012.[60]

The Status Quo Scenario

The model begins with the policy levels in effect in 2006 as the baseline. Changes in policy through 2014 were entered into the model. The status quo scenario maintains policies at the 2014 level through 2064. Data presented for years after 2006 are predictions. Table 12.4 shows results for the status quo scenario by gender, income quintile, and age group.

Table 12.4 Smoking Prevalence by Income Quintile (Lowest and Second-Lowest) and by Age, Sex, and Year, as Predicted by *SimSmoke's* Status Quo Scenario (Percentages)

	Lowest Income Quintile					2nd Lowest Income Quintile				
Age Group	2006	2011*	2015*	2045*	2064*	2006	2011*	2015*	2045*	2064*
Males										
18–24	26.4	22.7	22.3	22.1	22.0	23.2	19.7	19.7	19.6	19.6
25–44	36.7	28.2	24.4	20.9	20.8	30.9	24.6	22.0	18.1	18.1
45–64	36.8	32.2	30.9	14.5	14.4	28.5	24.1	21.8	10.6	9.9
65+	17.6	15.9	15.4	8.5	5.3	10.4	10.9	11.5	5.8	3.5
18+	30.5	25.7	23.6	16.9	15.9	24.6	20.7	19.1	13.2	11.7
Females										
18–24	21.7	18.8	18.5	18.3	18.2	18.4	15.7	15.6	15.5	15.5
25–44	30.5	23.6	20.8	18.0	17.8	23.2	19.3	17.5	14.2	14.2
45–64	28.6	25.6	24.4	12.0	11.8	21.2	20.6	20.8	12.0	11.5
65+	9.8	9.7	10.2	6.9	4.7	7.0	8.0	9.%	8.9	6.0
18+	22.5	19.5	18.3	14.1	13.3	17.6	16.2	15.8	12.5	11.2

*Predicted smoking prevalence using the *SimSmoke* model.

For the lowest income quintile in 2006, smoking prevalence for males age 18 and over was 30.5%. Predicted prevalence declined slowly in subsequent years: to 25.7% in 2011, 23.6% in 2015, 16.9% in 2045, and 15.9% in 2064. For the second-lowest income quintile model, adult male smoking prevalence was at 24.6% in 2006, with predictions falling to 20.7% in 2011, 19.1% in 2015, 13.2% in 2045, and 11.7% in 2064. Smoking prevalence among women age 18 and over in the lowest quintile model also decreased gradually over these years: 22.5% in 2006 and predicted to be 19.5% in 2011, 18.3% in 2015, 14.1% in 2045, and 13.3% in 2064. Smoking prevalence for women in the second-lowest quintile model was 17.6% in 2006, then predicted to be 16.2% in 2011, 15.8% in 2015, 12.5% in 2045, and 11.2% in 2064. The model predicted a slow downward trend in the absence of policy change, as reflected in 2006 prevalence, initiation, and cessation rates. Fluctuations from that trend are due to policy changes, primarily explained by increases in cigarette prices between 2006 and 2014 and the implementation of additional smoke-free laws.

Table 12.4 shows that by 2011, smoking prevalence rates also declined for most age groups, except for women in the 65-and-older age group in both income quintiles, and men ages 65 and older in the second-lowest income quintiles. The 45–64 age group showed the largest declines, followed by the 25–44 group and the 18–24 group. Among adults ages 45–64, the 2006 prevalence for males was 36.8% for the first income quintile and 28.5% for the second, and for females, 28.6% for the first income quintile and 21.2% for the second. The larger declines in the 25–44 and 45–64 age groups may reflect a need to further calibrate the model once data are available for later years. This calibration will allow for initiation in later age groups and lower cessation rates at the younger ages.

Smoking-attributable deaths predicted for age 18 and older according to *SimSmoke's* status quo scenario are shown in Table 12.5. With the policies implemented and maintained in future years, smoking-attributable deaths predicted for the lowest income quintile in 2006 were 74,778 among men and 38,916 among women, or 113,694 combined. In 2011, estimated smoking-attributable deaths in this income quintile increased to 78,190 for men and 40,233 for women, or 118,423 combined. In 2015 these estimates rose again, to 78,181 deaths among men and 40,970 deaths among women (119,151 combined); the number of deaths declined by 2064 to 38,492 deaths (men), 23,716 (women), and 62,208 (combined). For the years 2015 through 2064, a total of 4,382,226 premature deaths were predicted.

For the second-lowest quintile, the status quo model predicted 54,400 smoking-attributable deaths among males and 33,159 smoking-attributable deaths among females (87,559 combined) in 2006, and predictions increased in 2011 to 59,119 deaths among males and 33,802 deaths among females (92,921 combined). The number of deaths was predicted to increase again in 2015, to 60,867 among males and 35,499 among females (96,366 combined), then decline in 2064 to 29,573 deaths among males and 24,822 deaths among females (54,395 combined). For the years 2015 through 2064, a total of 3,842,548 premature deaths were predicted. The lower number of deaths in the second income quintile reflects lower smoking rates at that income level. The increase in smoking-attributable deaths over time reflects the aging of the large number of former smokers as well as general population growth.

Table 12.5 Smoking-Attributable Deaths by Income Quintile (Lowest and Second-Lowest) and by Sex and Year, as Estimated by *SimSmoke's* Status Quo Scenario

Quintile and Sex	2006	2011*	2015*	2025*	2045*	2064*	2015–2064*
Lowest income quintile							
Men	74,778	78,190	78,181	70,661	45,867	38,492	2,746,847
Women	38,916	40,233	40,970	40,619	28,804	23,716	1,635,379
Total	113,694	118,423	119,151	111,280	74,671	62,208	4,382,226
Second-lowest income quintile							
Men	54,400	59,119	60,867	57,500	37,065	29,573	2,215,841
Women	33,159	33,802	35,499	38,129	30,658	24,822	1,626,707
Total	87,559	92,921	96,366	95,629	67,723	54,395	3,842,548

*Predicted smoking prevalence using the *SimSmoke* model.

Stronger Policy Scenarios

Next, the effect of strengthening current policies, both individually and in combination, was considered. These stronger policies—which might be viewed as the desired set of policies, similar to those recommended in the Healthy People 2010 objectives[37]—included:

- Tax increases of $1.00, $2.00, and $3.00 per pack, with the assumption that these taxes are indexed to inflation so that their value is maintained over time
- Extending coverage of smoke-free laws to cover worksites, restaurants, and bars in all 50 states, with high compliance
- Increasing mass media anti-tobacco campaign expenditures to a high intensity level in all states from their average current medium-high intensity level
- Increasing restrictions from current advertising on TV and radio, to include newspapers, point of sale, sponsorship, branding, and mail giveaways, with stronger enforcement; and implementing strong graphic health warnings
- Implementing a well-publicized cessation policy involving multi-session quitlines and free NRT
- Strengthening youth access policies to a high level of enforcement.

The incremental effects of these stronger policies (referred to below as *SimSmoke*-Recommended Policies) depend on the level of policies in effect in 2014. The effects of policies are presented relative to the status quo level for smoking prevalence in the same year (**t**), that is:

$$[\text{Policy rate}_t - \text{status quo rate}_t]/\text{status quo rate}_t$$

and in terms of lives saved:

$$[\text{Deaths in status quo}_t - \text{Deaths with policies in place}_t]$$

for smoking-attributable deaths.

The comprehensive best-case strategy includes the predicted simultaneous implementation (in the year 2015) of each of the above policies together with a tax increase of $1.00, $2.00, or $3.00 per pack.

New, more rigorous policies were modeled as if implemented and maintained from 2015 through 2064. The predicted effects on male and female smoking prevalence are shown in Tables 12.6 and 12.7 for the lowest quintile, and in Tables 12.8 and 12.9 for the second-lowest quintile. The effects of these policies on smoking-attributable deaths among both genders are shown in Table 12.10 for the first income quintile and Table 12.11 for the second income quintile. These tables reveal the effects of tax increases, universal adoption of smoke-free laws, enhanced mass media anti-tobacco campaigns, marketing restrictions, health warnings, cessation treatment policies, and youth access policies. Each tobacco control policy and data from Tables 12.6 to 12.11 are discussed in the subsections below.

Cigarette Taxes

Of the tobacco control policies, *SimSmoke* attributes the most pronounced effect on smoking prevalence trends between 1993 and 2003 to tax increases.[95] NCI Tobacco Control Monograph 21 concluded that "significantly increasing the excise tax and price of tobacco products is the single most consistently effective tool for reducing tobacco use."[97,p.151] However, the same absolute increase in taxes or price had a smaller effect in 2009 than in earlier years because prices were higher in 2009, and the same increases were smaller in relative terms. In both the lowest and second-lowest income quintile models, a $1.00/pack increase in the 2015 average tax rate was projected to result in a relative decline of about 3.5% in smoking prevalence for both men and women compared to the status quo tax rate in that year. By 2064, the tax rate increase is projected to lead to a much larger decline, about 8.2% in both income quintiles, compared to the status quo rate. In both the lowest and second-lowest income quintile models, an increase of $2.00 in the average tax rate is projected to result in a relative reduction of about 6.5% in both men's and women's smoking prevalence in 2015 compared to the status quo. By 2064, this increased rate is projected to lead to a reduction in prevalence of between 14.4% and 14.8% in males and females relative to the status quo. In 2015 in both the lowest and second-lowest income quintile models, an increase of $3.00 in the average tax rate was projected to result in about a 9.0% relative reduction in smoking prevalence for both men and women compared to the status quo. By 2064, smoking prevalence under the recommended policy scenario is projected to decrease to about 19.6% of the status quo smoking prevalence.

The largest effect of the price increases is seen among young people, particularly those younger than 18 years old. Price increases have a greater effect over time primarily because young people are more responsive to price increases than adults, and as those young people grow older, fewer of them smoke. As noted, the *SimSmoke* model assumes that taxes increase with the rate of inflation over time, but some of the effect of tax increases on smoking prevalence dissipates over time if the per-unit taxes are not indexed to inflation.[55]

Table 12.6 Comparison of Status Quo Policies With *SimSmoke*-Recommended Policies: Smoking Prevalence and Percentage Change Among Men Ages 18 to 85, Lowest Income Quintile (Percentages)

Policies and Effects	2014	2015*	2025*	2045*	2064*
Status quo policies – smoking prevalence	24.0	23.6	20.1	16.9	15.9
Recommended policies – independent effects on smoking prevalence					
1. Tax increases (per pack)					
By $1.00	24.0	22.8	19.1	15.7	14.6
By $2.00	24.0	22.1	18.3	14.7	13.6
By $3.00	24.0	21.5	17.7	13.9	12.8
2. Comprehensive, well-enforced smoke-free laws	24.0	23.1	19.6	16.5	15.5
3. High-intensity mass media anti-tobacco campaigns	24.0	23.0	19.3	16.0	15.0
4. Comprehensive, well-enforced marketing bans	24.0	22.8	19.3	16.1	15.1
5. Strong health warnings	24.0	22.8	19.1	16.0	15.0
6. Cessation treatment policies	24.0	23.3	19.4	16.2	15.3
7. Strong youth access enforcement	24.0	23.6	19.9	16.3	15.2
Combined policy effects on prevalence					
2–7 above, plus $1.00 tax increase	24.0	19.3	14.8	11.4	10.4
2–7 above, plus $2.00 tax increase	24.0	18.8	14.2	10.7	9.7
2–7 above, plus $3.00 tax increase	24.0	18.3	13.7	10.1	9.1
% Change in smoking prevalence with recommended policies vs. status quo					
Independent policy effects					
1. Tax increases					
By $1.00	0.0	–3.5	–4.9	–7.3	–8.2
By $2.00	0.0	–6.5	–8.9	–13.0	–14.5
By $3.00	0.0	–8.9	–12.1	–17.7	–19.6
2. Comprehensive, well-enforced smoke-free laws	0.0	–1.9	–2.3	–2.6	–2.6
3. High-intensity mass media anti-tobacco campaigns	0.0	–2.5	–3.9	–4.4	–4.4
4. Comprehensive, well-enforced marketing bans	0.0	–3.4	–3.9	–4.8	–5.1
5. Strong health warnings	0.0	–3.5	–4.8	–5.5	–5.7
6. Cessation treatment policies	0.0	–1.1	–3.5	–4.0	–3.9
7. Strong youth access enforcement	0.0	0.0	–1.1	–3.4	–4.7
% Change – combined policy effects					
2–7 above, plus $1.00 tax increase	0.0	–18.0	–26.3	–32.6	–34.4
2–7 above, plus $2.00 tax increase	0.0	–20.5	–29.5	–36.9	–39.0
2–7 above, plus $3.00 tax increase	0.0	–22.6	–32.1	–40.4	–42.8

*Predicted smoking prevalence or percentage change using the *SimSmoke* model.

Table 12.7 Comparison of Status Quo Policies With *SimSmoke*-Recommended Policies: Smoking Prevalence and Percentage Change Among Women Ages 18 to 85, Lowest Income Quintile (Percentages)

Policies and Effects	2014	2015*	2025*	2045*	2064*
Status quo policies – smoking prevalence	18.5	18.3	16.0	14.1	13.3
Recommended policies – independent effects on smoking prevalence					
1. Tax increases (per pack)					
By $1.00	18.5	17.6	15.2	13.1	12.2
By $2.00	18.5	17.1	14.5	12.3	11.4
By $3.00	18.5	16.6	14.0	11.6	10.7
2. Comprehensive, well-enforced smoke-free laws	18.5	17.9	15.6	13.7	12.9
3. High-intensity mass media anti-tobacco campaigns	18.5	17.8	15.3	13.4	12.7
4. Comprehensive, well-enforced marketing bans	18.5	17.6	15.3	13.4	12.6
5. Strong health warnings	18.5	17.6	15.2	13.3	12.5
6. Cessation treatment policies	18.5	18.1	15.3	13.4	12.7
7. Strong youth access enforcement	18.5	18.3	15.8	13.7	12.8
Combined policy effects on prevalence					
2–7 above, plus $1.00 tax increase	18.5	15.0	11.6	9.4	8.6
2–7 above, plus $2.00 tax increase	18.5	14.5	11.1	8.8	8.0
2–7 above, plus $3.00 tax increase	18.5	14.1	10.7	8.3	7.5
% Change in smoking prevalence with recommended policies vs. status quo					
Independent policy effects					
1. Tax increases (per pack)					
By $1.00	0.0	–3.6	–5.0	–7.3	–8.1
By $2.00	0.0	–6.5	–9.1	–13.0	–14.4
By $3.00	0.0	–9.0	–12.4	–17.6	–19.5
2. Comprehensive, well-enforced smoke-free laws	0.0	–1.9	–2.3	–2.6	–2.8
3. High-intensity mass media anti-tobacco campaigns	0.0	–2.5	–4.1	–4.8	–4.9
4. Comprehensive, well-enforced marketing bans	0.0	–3.4	–4.0	–4.9	–5.2
5. Strong health warnings	0.0	–3.5	–5.0	–5.8	–6.1
6. Cessation treatment policies	0.9	–1.1	–3.9	–4.7	–4.8
7. Strong youth access enforcement	0.0	0.0	–1.1	–3.1	–4.1
% Change – combined policy effects					
2–7 above, plus $1.00 tax increase	0.0	–18.0	–27.4	–33.4	–35.5
2–7 above, plus $2.00 tax increase	0.0	–20.5	–30.6	–37.7	–40.1
2–7 above, plus $3.00 tax increase	0.0	–22.6	–33.3	–41.1	–43.7

*Predicted smoking prevalence or percentage change using the *SimSmoke* model.

Table 12.8 Comparison of Status Quo Policies With *SimSmoke*-Recommended Policies: Smoking Prevalence and Percentage Change Among Men Ages 18 to 85, Second-Lowest Income Quintile (Percentages)

Policies and Effects	2014	2015*	2025*	2045*	2064*
Status quo policies – smoking prevalence	19.4	19.1	16.3	13.2	11.7
Recommended policies – independent effects on smoking prevalence					
1. Tax increases (per pack)					
By $1.00	19.4	18.4	15.5	12.2	10.8
By $2.00	19.4	17.8	14.8	11.4	10.0
By $3.00	19.4	17.4	14.2	10.8	9.4
2. Comprehensive, well-enforced smoke-free laws	19.4	18.7	15.9	12.8	11.4
3. High-intensity mass media anti-tobacco campaigns	19.4	18.6	15.7	12.6	11.2
4. Comprehensive, well-enforced marketing bans	19.4	18.5	15.7	12.5	11.1
5. Strong health warnings	19.4	18.5	15.5	12.4	11.0
6. Cessation treatment policies	19.4	19.0	15.9	12.7	11.3
7. Strong youth access enforcement	19.4	19.1	16.2	12.9	11.4
Combined policy effects on prevalence					
2–7 above, plus $1.00 tax increase	19.4	15.7	12.1	8.9	7.7
2–7 above, plus $2.00 tax increase	19.4	15.2	11.6	8.4	7.1
2–7 above, plus $3.00 tax increase	19.4	14.8	11.1	7.9	6.7
% Change in smoking prevalence with recommended policies vs. status quo					
Independent policy effects					
1. Tax increases (per pack)					
By $1.00	0.0	–3.6	–5.3	–7.4	–8.3
By $2.00	0.0	–6.7	–9.5	–13.2	–14.8
By $3.00	0.0	–9.2	–12.9	–17.9	–20.0
2. Comprehensive, well-enforced smoke-free laws	0.0	–1.9	–2.3	–2.8	–2.9
3. High-intensity mass media anti-tobacco campaigns	0.0	–2.6	–3.8	–4.5	–4.7
4. Comprehensive, well-enforced marketing bans	0.0	–3.4	–4.0	–5.0	–5.4
5. Strong health warnings	0.0	–3.5	–5.1	–6.2	–6.6
6. Cessation treatment policies	0.0	–0.7	–2.7	–3.7	–3.9
7. Strong youth access enforcement	0.0	0.0	–1.0	–2.3	–3.1
% Change – combined policy effects					
2–7 above, plus $1.00 tax increase	0.0	–17.7	–25.8	–32.3	–34.6
2–7 above, plus $2.00 tax increase	0.0	–20.3	–29.2	–36.7	–39.3
2–7 above, plus $3.00 tax increase	0.0	–22.5	–32.0	–40.2	–43.1

*Predicted smoking prevalence and percentage change using the *SimSmoke* model.

Table 12.9 Comparison of Status Quo Policies With *SimSmoke*-Recommended Policies: Smoking Prevalence and Percentage Change Among Women Ages 18 to 85, Second-Lowest Income Quintile (Percentages)

Policies and Effects	2014	2015*	2025*	2045*	2064*
Status quo policies	15.8	15.8	14.7	12.5	11.2
Recommended policies – independent effects on smoking prevalence					
1. Tax increases (per pack)					
By $1.00	15.8	15.2	14.0	11.6	10.3
By $2.00	15.8	14.8	13.4	11.0	9.6
By $3.00	15.8	14.4	13.0	10.4	9.0
2. Comprehensive, well-enforced smoke-free laws	15.8	15.5	14.3	12.2	10.9
3. High-intensity mass media anti-tobacco campaigns	15.8	15.4	14.2	12.0	10.8
4. Comprehensive, well-enforced marketing bans	15.8	15.2	14.1	11.9	10.7
5. Strong health warnings	15.8	15.2	14.0	11.8	10.6
6. Cessation treatment policies	15.8	15.7	14.4	12.1	10.9
7. Strong youth access enforcement	15.8	15.8	14.5	12.1	10.8
Combined policy effects on prevalence					
2–7 above, plus $1.00 tax increase	15.8	13.0	11.3	8.8	7.5
2–7 above, plus $2.00 tax increase	15.8	12.6	10.8	8.3	7.0
2–7 above, plus $3.00 tax increase	15.8	12.3	10.4	7.9	6.6
% Change in smoking prevalence with recommended policies vs. status quo					
Independent policy effects					
1. Tax increases (per pack)					
By $1.00	0.0	–3.5	–4.6	–6.8	–8.1
By $2.00	0.0	–6.4	–8.4	–12.1	–14.4
By $3.00	0.0	–8.8	–11.5	–16.5	–19.4
2. Comprehensive, well-enforced smoke-free laws	0.0	–1.9	–2.2	–2.5	–2.7
3. High-intensity mass media anti-tobacco campaigns	0.0	–2.6	–3.3	–3.9	–4.1
4. Comprehensive, well-enforced marketing bans	0.0	–3.4	–3.8	–4.6	–5.1
5. Strong health warnings	0.0	–3.5	–4.5	–5.4	–5.8
6. Cessation treatment policies	0.0	–0.7	–2.1	–2.7	–3.0
7. Strong youth access enforcement	0.0	0.0	–1.4	–3.2	–3.6
% Change – combined policy effects					
2–7 above, plus $1.00 tax increase	0.0	–17.6	–23.2	–29.4	–32.8
2–7 above, plus $2.00 tax increase	0.0	–20.1	–26.2	–33.5	–37.5
2–7 above, plus $3.00 tax increase	0.0	–22.2	–28.8	–36.9	–41.3

*Predicted smoking prevalence and percentage change using the *SimSmoke* model.

Table 12.10 Smoking-Attributable Deaths, from *SimSmoke* Model, Lowest Income Quintile

Policies and Effects	2014	2015*	2025*	2045*	2064*	2015–2064*
Status quo policies	119,526	119,151	111,280	74,671	62,207	4,382,226
Independent policy effects						
1. Tax increases (per pack)						
By $1.00	119,526	119,151	110,115	72,235	58,411	4,270,483
By $2.00	119,526	119,151	109,161	70,273	55,421	4,180,505
By $3.00	119,526	119,151	108,366	68,657	53,066	4,106,466
2. Comprehensive, well-enforced smoke-free laws	119,526	119,151	110,512	73,220	60,674	4,320,096
3. High-intensity mass media anti-tobacco campaigns	119,526	119,151	109,881	71,678	59,040	4,261,227
4. Comprehensive, well-enforced marketing bans	119,526	119,151	110,037	72,366	59,613	4,281,115
5. Strong health warnings	119,526	119,151	109,620	71,281	58,579	4,241,867
6. Cessation treatment policies	119,526	119,151	109,878	71,193	58,523	4,250,476
7. Strong youth access enforcement	119,526	119,151	111,280	74,467	61,152	4,369,917
Combined policy effects						
2–7 above, plus $1.00 tax increase	119,526	119,151	102,837	57,572	42,793	3,663,201
2–7 above, plus $2.00 tax increase	119,526	119,151	102,055	56,066	40,662	3,593,766
2–7 above, plus $3.00 tax increase	119,526	119,151	101,405	54,831	38,950	3,536,825
Attributable deaths with the status quo policies minus attributable deaths with recommended policies						
Independent policy effects						
1. Tax increases (per pack)						
By $1.00	—	—	1,166	2,436	3,797	111,743
By $2.00	—	—	2,120	4,399	6,786	201,721
By $3.00	—	—	2,915	6,015	9,201	275,760
2. Comprehensive, well-enforced smoke-free laws	—	—	768	1,451	1,534	62,130
3. High-intensity mass media anti-tobacco campaigns	—	—	1,400	2,993	3,167	120,999
4. Comprehensive, well-enforced marketing bans	—	—	1,244	2,305	2,595	101,111
5. Strong health warnings	—	—	1,660	3,391	3,629	140,359
6. Cessation treatment policies	—	—	1,402	3,479	3,685	131,750
7. Strong youth access enforcement	—	—	—	204	1,055	12,310
Combined policy effects						
2–7 above, plus $1.00 tax increase	—	—	8,444	17,100	19,414	719,025
2–7 above, plus $2.00 tax increase	—	—	9,223	18,606	21,545	788,461
2–7 above, plus $3.00 tax increase	—	—	9,875	19,840	23,258	845,401

*Predicted smoking-attributable deaths using the *SimSmoke* model.

Table 12.11 Smoking-Attributable Deaths, from *SimSmoke* Model, Second-Lowest Quintile

Policies and Effects	2014	2015*	2025*	2045*	2064*	2015–2064*
Status quo policies	95,986	96,366	95,629	67,723	54,395	3,842,548
Independent policy effects						
1. Tax increases (per pack)						
By $1.00	95,986	96,366	94,694	65,560	51,124	3,745,356
By $2.00	95,986	96,366	93,929	63,817	48,551	3,667,859
By $3.00	95,986	96,366	93,291	62,381	46,473	3,603,789
2. Comprehensive, well-enforced smoke-free laws	95,986	96,366	95,008	66,466	53,064	3,789,373
3. High-intensity mass media anti-tobacco campaigns	95,986	96,366	94,607	65,498	52,018	3,751,328
4. Comprehensive, well-enforced marketing bans	95,986	96,366	94,621	65,707	52,152	3,755,695
5. Strong health warnings	95,986	96,366	94,294	64,832	51,243	3,723,103
6. Cessation treatment policies	95,986	96,366	94,882	65,787	52,204	3,767,696
7. Strong youth access enforcement	95,986	96,366	95,629	67,574	53,680	3,833,878
Combined policy effects						
2–7 above, plus $1.00 tax increase	95,986	96,366	89,353	54,284	39,023	3,277,993
2–7 above, plus $2.00 tax increase	95,986	96,366	88,715	52,900	37,120	3,216,322
2–7 above, plus $3.00 tax increase	95,986	96,366	88,184	51,765	35,588	3,165,727
Attributable deaths with the status quo policies minus attributable deaths with recommended policies						
Independent policy effects						
1. Tax increases (per pack)						
By $1.00	—	—	935	2,163	3,270	96,792
By $2.00	—	—	1,700	3,907	5,844	174,689
By $3.00	—	—	2,339	5,342	7,922	238,759
2. Comprehensive, well-enforced smoke-free laws	—	—	621	1,257	1,331	53,175
3. High-intensity mass media anti-tobacco campaigns	—	—	1,022	2,225	2,377	91,220
4. Comprehensive, well-enforced marketing bans	—	—	1,008	2,017	2,243	86,853
5. Strong health warnings	—	—	1,335	2,891	3,151	119,445
6. Cessation treatment policies	—	—	747	1,936	2,190	74,852
7. Strong youth access enforcement	—	—	—	150	714	8,670
Combined policy effects						
2–7 above, plus $1.00 tax increase	—	—	6,276	13,439	15,371	564,555
2–7 above, plus $2.00 tax increase	—	—	6,975	14,823	17,275	626,226
2–7 above, plus $3.00 tax increase	—	—	7,445	15,959	18,806	676,821

*Predicted smoking-attributable deaths using the *SimSmoke* model.

In terms of lives saved, it is projected that in 2064, a $1.00 tax increase would avert 3,797 smoking-attributable deaths of men and women in the lowest income quintile and 3,270 deaths in the second-lowest quintile. A $1.00 tax increase in effect until 2064 would have averted a cumulative total of 111,743 smoking-attributable deaths in the lowest income quintile, and 96,792 deaths in the second-lowest quintile. A $2.00 tax increase is projected to avert 6,786 smoking-attributable deaths in 2064 in the lowest income quintile, and 5,844 deaths in the second-lowest quintile. Cumulatively, it is projected that 201,721 smoking-attributable deaths would be averted between 2015 and 2064 in the lowest income quintile, and 174,689 deaths in the second-lowest quintile with a $2.00 tax increase. A $3.00 tax increase would avert 9,201 smoking-attributable deaths in 2064 in the lowest income quintile, and 7,922 deaths in the second-lowest quintile. Over the 50-year period from 2015 to 2064, the model predicts that a $3.00 tax would avert a total of 275,760 smoking-attributable deaths in the lowest income quintile, and 238,759 deaths in the second-lowest quintile. These effects grow over time because individuals tend to reap the benefits of quitting smoking 2–10 years after quitting. In addition, those who are prevented from beginning to smoke between the ages of 10 and 24 also avoid smoking-attributable deaths, which generally occur at ages 35 and older.

Smoke-Free Laws

SimSmoke data support the conclusion that public health would be considerably improved if all states enacted and strongly enforced comprehensive laws that ban smoking in worksites, bars, restaurants, and other public places. These recommended measures are predicted to reduce male and female smoking prevalence in both the lowest and second-lowest income quintiles by 1.9% in 2015 relative to the status quo scenario. By 2045, smoking prevalence declines for men and women in the lowest income quintiles by 2.6%; in the second-lowest quintile it declines by 2.8% for men and 2.5% for women. By 2064, in the lowest income quintile smoking prevalence decreases by 2.6% for men and 2.8% for women; in the second-lowest quintile smoking prevalence by 2.9% for men and 2.7% for women. By 2064, comprehensive smoke-free laws would avert 1,534 smoking-attributable deaths (male and female) in the lowest income quintile and 1,331 deaths in the second-lowest quintile. From 2015 to 2064, comprehensive smoke-free laws would avert a total of 62,130 smoking-attributable deaths in the lowest income quintile and a total of 53,175 deaths in the second-lowest quintile.

Mass Media Anti-Tobacco Campaigns

A high-intensity mass media anti-tobacco campaign implemented in 2015 was projected to lead to a decline of 2.5% in adult smoking prevalence in the lowest income quintile compared to the status quo, and a 2.6% decline in the second-lowest income quintile. By 2064 the relative effect would increase to 4.4% among men and 4.9% among women, both in the lowest quintile, and to 4.7% among men and 4.1% among women in the second-lowest quintile. The model projects that in 2064, a strong campaign directed at all smokers would avert 3,167 smoking-attributable deaths in the lowest income quintile and 2,377 deaths in the second-lowest quintile. Between 2015 and 2064, the enhanced anti-tobacco media campaign would avert a total of 120,999 smoking-attributable deaths in the lowest income quintile and 91,220 deaths in the second-lowest quintile.

Marketing Restrictions

Strongly enforced restriction of both direct and indirect marketing is predicted to lead male and female smoking prevalence to decline 3.4% in 2015 for the lowest and second-lowest income quintiles, compared to status quo policies. By 2064, prevalence would decline by around 5.2% (in the lowest

quintile, 5.1% for men and 5.2% for women; in the second-lowest quintile, 5.4% for men and 5.1% for women). Strong marketing restrictions are estimated to avert 2,595 smoking-attributable deaths in 2064 in the lowest quintile, and 2,243 deaths in the second-lowest quintile. Over the 50-year period from 2015 to 2064, a total of 101,111 smoking-attributable deaths would be averted in the lowest income quintile and 86,853 deaths would be averted in the second-lowest quintile with well-enforced marketing restrictions.

Health Warnings

In 2015, a stronger health warning policy was predicted to reduce smoking prevalence by 3.5% among men and women in the lowest and second-lowest income quintiles relative to the status quo. By 2064 the stronger policy is projected to reduce smoking by a higher percentage compared to the status quo policy: in the lowest quintile, by 5.7% among men and 6.1% among women; in the second-lowest quintile, by 6.6% among men and 5.8% among women. It is projected that in 2064, a strong health warning policy would avert a total of 3,629 smoking-attributable deaths in the lowest quintile, and 3,151 deaths in the second-lowest quintile. The cumulative total number of smoking-attributable deaths averted by a strong health warning policy in the years 2015 through 2064 would be 140,359 in the lowest income quintile and 119,445 in the second-lowest quintile.

Cessation Treatment Policies

A policy requiring well-publicized, multi-session quitlines with free NRT would have relatively small effects in the earlier years of the projection compared to other policies, but over time would lead to higher rates of cessation, which reflects the tendency of people older than 24 to quit smoking at higher rates than younger people.[54] In 2015, enhanced cessation policies were projected to reduce smoking prevalence by 1.1% for men and women in the lowest income quintile compared to the status quo scenario, and by 0.7% for men and women in the second-lowest quintile. In 2064 these policies are expected to result in the following changes: in the lowest income quintile, a 3.9% relative reduction in prevalence rates among men and a 4.8% relative reduction among women; in the second-lowest quintile, these policies are expected to lead to a 3.9% relative reduction in men and a 3.0% reduction in women. It is projected that in 2064, a stronger cessation policy would avert a total of 3,685 smoking-attributable deaths in the lowest income quintile and 2,190 deaths in the second-lowest quintile. During the years 2015 through 2064 the comprehensive cessation policy is expected to avert a cumulative total of 131,750 smoking-attributable deaths in the lowest income quintile and 74,852 in the second-lowest quintile compared to the status quo policy. These effects are relatively small because the model takes into account that about 80% of states already provide free NRT, and 100% have active quitlines with follow-up.[105]

Youth Access

Strong enforcement of youth access policies is estimated to have no immediate effect on reducing smoking prevalence for men and women in the lowest and second-lowest income quintiles in 2015, since it is directed at youth, who make up a small percentage of the population; however, a stronger effect is predicted in later years. In 2064, stricter enforcement of youth access policies is projected to reduce smoking prevalence in comparison with status quo policies as follows: in the lowest quintile, by 4.7% among men and 4.1% among women; in the second-lowest quintile, by 3.1% among men and 4.0% among women. In 2064, strong enforcement of youth access is projected to prevent a total of 1,055 smoking-attributable deaths in the lowest income quintile and 714 deaths in the second-lowest income

quintile. During the years 2015 through 2064, it is estimated that a total of 12,310 smoking-attributable deaths would be averted in the lowest quintile and 8,670 in the second-lowest quintile.

Best-Case Scenario: A Comprehensive Set of Policies

Lastly, the combination of the individual policies described in previous sections—comprehensive smoke-free laws, a high-intensity mass media anti-tobacco campaign, enhanced marketing restrictions, strong health warnings, and strengthened cessation and youth access policies—with varying increases in cigarette taxes were considered. For 2015, these policies, combined with a tax increase of $1.00 per pack, would lead smoking prevalence among both men and women to decline to about 18.0% below what status quo policies would produce for the lowest income quintile and to decline by about 17.7% among men and 17.6% among women for the second-lowest quintile. Maintaining this set of policies is estimated to reduce the smoking rate in 2064 by 34.4% among men and 35.5% among women in the lowest income quintile relative to the status quo, and by 34.6% among men and 32.8% among women in the second-lowest quintile.

Similarly, a tax increase of $2.00 per pack in combination with the other policies was projected to reduce the smoking rate in 2015 by about 20.5% in men and women in the lowest income quintile, and by 20.3% in men and 20.1% in women in the second-lowest quintile, compared to the status quo. In 2064 this combination is projected to reduce the smoking rate by 39.0% among men and 40.1% among women in the lowest income quintile compared to the status quo, and by 39.3% among men and 37.5% among women in the second-lowest quintile. Increasing taxes by $3.00 per pack in combination with the other policies was projected to reduce the smoking rate in 2015 by 22.6% for men and women in the lowest income quintile, and by 22.5% for men and 22.2% for women in the second-lowest quintile. In 2064 this policy is projected to reduce the smoking rate by 42.8% among men and 43.7% among women in the lowest income quintile relative to the status quo, and by 43.1% among men and 41.3% among women in the second-lowest quintile.

In terms of smoking-attributable deaths averted, a comprehensive policy with a $1.00 per pack tax increase is projected to avert 19,414 deaths in the lowest income quintile and 15,371 deaths in the second-lowest quintile in the year 2064. The model projects that these combined policies will avert 719,025 smoking-attributable deaths between 2015 and 2064 for the lowest quintile and 564,555 for the second-lowest quintile. In 2064, a $2.00 tax increase combined with the other policies would prevent an estimated 21,545 smoking-attributable deaths in the lowest quintile and 17,275 deaths in the second-lowest quintile. Over the years 2015 through 2064 these policies are projected to avert 788,461 deaths in the lowest income quintile and 626,226 lives in the second-lowest quintile. A comprehensive policy that includes a $3.00 tax increase is projected in 2064 to prevent 23,258 smoking-attributable deaths in the lowest quintile, 18,806 deaths in the second-lowest quintile, and a cumulative total between 2015 and 2064 of 845,401 deaths in the lowest quintile and 676,821 deaths in the second-lowest quintile.

Of the seven policies in the comprehensive package, tax increases have the greatest effects overall in reducing smoking prevalence and smoking-attributable deaths. Some policies, such as cessation treatment programs, have a larger impact on adult smoking than on youth smoking. Others, such as taxes, have a greater effect on youth smoking prevalence than on adult smoking prevalence (especially those ages 35–64).

Conclusions

In 2006, smoking prevalence in the lowest income quintile was 30.2% for men and 22.7% for women, with rates for people ages 25 to 64 averaging 36.8% for men and 29.8% for women. Rates in the second-lowest income quintile were also high—25.3% for men and 18.4% for women, while rates for people ages 25 to 64 averaged 29.9% for men and 22.5% for women. Smoking prevalence was thus considerably higher in these income quintiles than the average for the population as a whole in 2006 (20.1% for males and 15.5% for females, based on the 2006-2007 TUS-CPS).[108] Based on current policies, *SimSmoke* predicts declining rates for both the lowest and second-lowest income quintiles, but it also predicts that smoking rates for these quintiles will be high for many years to come.

Through stronger tobacco control policies, smoking prevalence rates can be considerably reduced for the lowest two income quintiles. Raising average taxes by $3.00 per pack would lower prevalence rates by more than 19% by the year 2064. Health warnings, anti-tobacco media campaigns, and comprehensive marketing restrictions can also play an important role. With a $3.00 tax increase, comprehensive marketing restrictions, smoke-free laws, strong graphic health warnings, a higher intensity media campaign, broader cessation treatment coverage, and greater youth access enforcement, the model predicts that smoking prevalence will fall by about 23% in the first few years. By 2064, the recommended policies would reduce smoking prevalence by more than 41% compared to status quo policies. While cessation treatments did not appear to produce large effects in this model, other studies have shown that fully integrating cessation treatment policies into the health care system has strong potential to influence smoking prevalence, specifically through rewarding health care providers for conducting interventions with follow-up and providing low- or no-cost therapies.[103,104] Additionally, the Affordable Care Act emphasizes prevention of disease and expands access to tobacco cessation services.[109]

SimSmoke also estimated that in 2014, 119,526 people in the lowest income quintile and 95,986 people in the second-lowest income quintile would die prematurely from smoking. A stronger set of policies and a $3.00 tax increase is predicted to result in 42,064 fewer deaths in 2064 (23,258 in the lowest income quintile and 18,806 in the second-lowest) than with the status quo policies, and a cumulative total for the years 2015 through 2064 of 1,522,222 lives saved (845,401 in the lowest income quintile and 676,821 in the second-lowest). These figures do not include lives lost due to secondhand smoke or fires caused by smoking, nor are the savings in excess medical costs associated with smoking-related conditions taken into account. These results show that tobacco control policies can have a major effect in reducing health disparities in low-income populations.

This analysis was conducted at the national level, but disparities are also seen at the state level. Many of the states with the lowest median household income,[110] such as Alabama, Arkansas, Kentucky, Louisiana, South Carolina, and Tennessee, also have weak tobacco control policies.[111] Significantly increasing tobacco taxes, implementing comprehensive smoke-free laws, and conducting strong mass media campaigns in these states can go a long way toward reducing income-related health disparities.

Although this analysis focused on income disparities, disparities by education and race/ethnicity merit consideration both individually and as they interact with income. For example, chapter 11 discusses the potential for higher cigarette taxes and more rigorous marketing restrictions to reduce smoking by African Americans. A *SimSmoke* model developed by Levy and colleagues[112] examines how a ban on menthol cigarettes could affect both smoking prevalence and smoking-attributable deaths, considering three possible scenarios. The model projects that in the continued absence of a ban on menthol

cigarettes, smoking prevalence will decline slowly and the percentage of people smoking menthol cigarettes will increase. In contrast, a ban on menthol cigarettes is projected to lead to greater reductions in smoking prevalence and fewer smoking-attributable deaths; the largest proportion of benefits would accrue to African Americans. As the authors note, "our results suggest that somewhere between 323,000 and 633,000 deaths could be avoided under a [menthol] ban, almost one-third of which would be among Blacks."[112,p.1238] Similarly, a model could be developed specifically focused on the Hispanic population, for example, to distinguish the effects of policies on more acculturated versus less acculturated Hispanic smokers.

The income disparities model presented above did not consider the use of other tobacco products, such as smokeless tobacco and cigars, which are increasingly used with cigarettes.[113,114] Smokeless tobacco use has increased since the 1990s, especially among young, low-income white males, and much of this use is in conjunction with cigarettes.[115–119] Cigar use among young adults has also increased[120–122]; some brands are very similar to cigarettes in size and content but are taxed at lower rates. Use of e-cigarettes, a relatively new product, will be important to monitor in low-income populations.[123] In general, further research is needed on the different types of tobacco used by people of low SES, especially by youth and young adults.

Another limitation of the model is traceable to its method of evaluating initiation to smoking. Initiation generally takes place until the age of 24 in all models, but income varies over the life of the individual, and income until age 24 may be a poor indicator of later income and likely SES. For example, an individual may be in college through age 24 and receiving very low income. In addition, living circumstances vary, with some individuals living with their family of origin and others living independently with their own children. Therefore, it may be important to consider initiation at later ages, when income may more closely reflect eventual future income. Initially it appeared that TUS-CPS income data were missing for a disproportionate number in the lower age groups (< 24 years old), possibly because many in these younger groups were full-time students. However, using family income and analyzing missing income revealed that the proportion of those with missing income appeared to be roughly uniform across age groups. The model also might be extended to consider the steps in the progression to smoking initiation and to smoking cessation, rather than just considering simple initiation and cessation.

The results from *SimSmoke* are subject to the limitations of the existing data, which indicate the importance of better surveillance to a better understanding of disparities in relation to public health. The model applied variations in mortality by income from Canada, which did not distinguish by age. The model also did not incorporate variations in mortality rates by income as they apply to smoking status. Information on mortality rates by income and smoking status is needed to better estimate the number of smoking-attributable deaths. To the extent that there are greater variations in the United States than in Canada, smoking-attributable deaths are likely to have been underestimated.

In addition, the relatively high exposure to secondhand smoke among some racial/ethnic and low-SES groups (see chapter 9) is likely to impact the mortality rates of people of low SES more than those of high SES. More information is also needed on exposure to particular policies by SES. For example, compared to smokers of higher SES, lower SES smokers may pay lower prices for tobacco products on average, may be less subject to smoke-free laws, may be less likely to use quitlines and low- or no-cost pharmacotherapies, and may have less exposure to anti-tobacco media campaigns.

The *SimSmoke* results depend on a set of assumptions on effect sizes derived from the literature. The impact that an array of tobacco control policies have on different sectors of the population can be exceedingly complex, where the effect of an individual policy may depend on the array of policies already implemented or any policies implemented at the same time as the policy of interest. The strength of the evidence for each of the policies varies.[18,19,42,95,124] The evidence for taxes and smoke-free policies is stronger than the evidence for mass media campaigns, and the evidence for cessation policies is weaker and less consistent. The model allowed for some variations in the effects of tobacco control policies by SES, but these effects could be subject to greater uncertainty as they affect low-SES groups. With smoking increasingly concentrated in low-SES groups, better information is needed on the effects of policies by SES. Knowledge of the synergistic effect of policies is also limited. Although a small number of empirical studies simultaneously consider the effect of two tobacco control policies,[23,25,125] most studies examine the effect of only one policy, making it difficult to determine how multiple policies interact with one another.

The direct effect of policies on cessation in *SimSmoke* can be seen in a decrease in prevalence in the first year of the model. In future years, the effects of policy are maintained or increased through effects on initiation and cessation rates. The effects may also depend on relapse, although data on relapse rates in general and specifically relapse among lower SES individuals are limited.

Another simplifying assumption is that policies are modeled as having a unidirectional effect on smoking rates. *SimSmoke* does not explicitly model potential feedbacks through tobacco industry practices, social norms and attitudes, and peer and family behaviors. As policies are implemented, the tobacco industry might strategically respond and counteract some policies by changing pricing or marketing practices or by introducing new products. In particular, tobacco companies may increasingly target low-SES groups. Projections of the strongest case assume that actions of tobacco companies do not negate a set of strong regulations and treatment progress.

In addition to the validation conducted for this study, previous applications of *SimSmoke* to the United States, Arizona, California, Kentucky, and Minnesota[17–20,48–51] as well as to other countries[15,39–46,48] have accurately projected trends and turning points in smoking rates, confirming the validity of the parameters and assumptions underlying the income models. However, the income disparities models chart new territories for the *SimSmoke* models. It will be important to validate those models over time in future work. Other classifications of SES, such as by education level, might be considered, along with racial/ethnic interactions. By assessing the impact of policies on different sociodemographic groups, problem areas might be identified and policies evaluated so that future policies could be targeted to those areas.[27]

To summarize, smoking rates among the lowest and second-lowest income quintiles are considerably above the national average, leading to over half of the smoking-attributable deaths in the United States. *SimSmoke* projects that a stronger set of tobacco control policies, especially price policies, may reduce smoking prevalence in the two lowest income quintiles by 25% in the near term, increasing to almost 45% by 2065. These stronger policies will avert 850,000 smoking-attributable deaths in the lowest income quintile and 675,000 deaths in the second-lowest quintile by 2064. Modeling not only makes it possible to examine the potential role of policies in reducing smoking rates in disadvantaged populations, but also provides a framework for more systematically determining data and research needs.

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