Structural Drivers of Persistent Disparities in Tobacco Use and Secondhand Smoke Exposure

by

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List of Abbreviations

ACS	Acute coronary syndrome
AME	Average marginal effect
ANRF	American Nonsmokers' Rights Foundation
APC	Annual percent change
CI	Confidence interval
CDC	Centers for Disease Control and Prevention
DiD	Difference-in-difference
ERB	Ethics Review Board
HRC	Human Rights Campaign
IHD	Ischemic heart disease
LGBTQ	Lesbian, gay, bisexual, transgender, queer
LOD	Limit of detection
MRP	Multilevel regression and post-stratification
NATS	National Adult Tobacco Survey
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
OR	Odds ratio
PCA	Principal components analysis
PIR	Poverty income ratio
PSU	Primary sampling unit
RDC	Research data center
RR	Risk ratio
SAMMEC	Smoking-attributable mortality, morbidity, and economic costs
SD	Standard deviation
SES	Socioeconomic status
SHS	Secondhand smoke
SM	Sexual minority
STI	Sexually transmitted infection
TRHD	Tobacco-related health disparity
US	United States

Abstract

While the prevalence of tobacco use has declined substantially in recent decades, smoking remains the leading cause of preventable death in the United States. Moreover, patterns of tobacco use vary widely across socio-demographic groups. This dissertation explored structural drivers of persistent disparities in tobacco-related health outcomes, focusing on two examples in the U.S.: patterns of secondhand smoke (SHS) exposure among nonsmokers, and the high risk of smoking among sexual minority (SM) adults.

In Chapter 2, I examined the relationship between smoke-free law coverage of workplaces and hospitality venues (restaurants and bars) and disparities in SHS exposure between 1999 and 2014. I found that smoke-free law coverage was associated with narrowing the differentials in SHS exposure between males and females, however, workplace smoke-free laws may have exacerbated SHS exposure disparities across quartiles of poverty income ratio, particularly for younger adults.

In Chapter 3, I adopted attributable fraction and simulation modeling methods to quantify disparities in deaths attributable to SHS exposure between 2000 and 2016, and to project potential SHS exposure patterns through 2040. I found that Non-Hispanic Black adults have experienced a disproportionate burden of SHS-attributable mortality, compared to adults of other racial/ethnic backgrounds. In simulating the potential impacts of multiple intervention scenarios, I found that an intervention that weakened the association between smoking prevalence and SHS

exposure resulted in more substantial declines in SHS exposure compared to an intervention that increased smoking cessation.

Finally, in Chapter 4, I shifted focus to examine the high smoking prevalence among SM adults, including gay, lesbian, and bisexual individuals. I explored the relationship between smoking and exposure to state-level structural stigma, which encompasses societal norms and policies that constrain access to resources among stigmatized groups. Among SM adults, I found evidence of a curvilinear relationship between stigma and current smoking, where the probability of smoking was lowest at the lowest and highest levels of stigma.

In sum, this dissertation examined patterns of tobacco-related health outcomes through a health equity lens. By combining empirical methods and simulation-based approaches, these studies provide insight into potential levers for reducing tobacco use and tobacco-related health disparities in the United States.

CHAPTER 1. Introduction

Disparities in Tobacco-Related Health Outcomes

In 1964, the U.S. Surgeon General published a seminal report that first acknowledged smoking as a cause of lung cancer and chronic bronchitis.[1] Since the 1960s, a large body of research has established that the health effects of smoking and exposure to secondhand smoke (SHS) extend well beyond lung cancer to encompass numerous other conditions, including other cancers, cardiovascular diseases, and pregnancy and birth-related outcomes.[2] According to estimates from the Centers for Disease Control and Prevention (CDC), over 20 million premature deaths in the United States were attributable to smoking between 1965 and 2014, including over 2 million deaths caused by exposure to SHS.[2]

The recognition of the pernicious health impacts of cigarette smoking has led to a rapid decline in smoking rates in the United States. Tobacco control efforts – including education on tobacco's harmful effects, taxation of tobacco products, smoke-free air policies, media campaigns, restrictions on sales and marketing, lawsuits, and cessation support initiatives – have played a significant role in this decline, resulting in 8 million fewer premature deaths attributable to smoking between 1964 and 2012.[3] Yet, nearly 14% of U.S. adults were current smokers as of 2018, and smoking remains the leading cause of preventable death in the United States.[4] Increasingly, patterns of tobacco use are also characterized by socioeconomic gradients and disparities across population groups.[5] Individuals with less than a high school education and those who live below the federal poverty line are more likely to smoke, compared to individuals at higher levels of education or income.[6] There are differential smoking patterns by

race/ethnicity, with the prevalence of smoking among American Indians/Alaskan Natives over 5 times that of the prevalence among Asians.[7] Likewise, sexual minority (e.g., lesbian, gay, and bisexual) individuals are more likely to smoke compared to their heterosexual counterparts.[8] SHS exposure patterns are also characterized by disparities by race/ethnicity and socioeconomic status (SES), with the highest levels of exposure among non-Hispanic Black and lower SES-individuals.[9,10] Unsurprisingly, these disparities extend to downstream health outcomes, including smoking-related cancers.[5]

Conceptual Frameworks for Understanding Tobacco-Related Health Disparities

As noted in the 2014 National Cancer Institute monograph, A *Socioecological Approach to Addressing Tobacco-Related Health Disparities*, tobacco disparities are multifaceted, involving an interplay of societal norms and policies, community/neighborhood environments, interpersonal interactions, and individual/intrapersonal factors.[5] These factors interact with one another and produce heterogeneous patterns in outcomes that span the tobacco use continuum, which captures all phases of tobacco exposure, initiation, use, and downstream health outcomes.[5] In this dissertation, I focused on societal norms/policies and a number of outcomes related to current smoking and SHS exposure, with the understanding that these exposures and outcomes are part of a much broader context.

Broadly speaking, societal norms/policies can impact tobacco use disparities through two mechanisms: differential exposure and differential vulnerability.[11] Individuals may be differentially exposed to societal norms/policies based on where they live. For example, the patchwork implementation of smoke-free air laws over the past several decades in the United States has created substantial variation in smoke-free law coverage, by region, race/ethnicity, and SES.[12–14] Differential vulnerability implies that individuals may have different responses to

policies/societal norms based on their sociodemographic characteristics. While I focus primarily on differential vulnerability in this dissertation, both mechanisms are important contributors to population patterns of tobacco-related health outcomes.

In assessing differential vulnerability, I focus on several sociodemographic characteristics, including gender, race/ethnicity, education, poverty income ratio (PIR), and sexual minority status. I view these effect modification factors as representative of social conditions, rather than individual-level biological factors. For example, when examining effect modification by race/ethnicity, the mechanism of modification can be attributed to a deeply embedded system of race-based discrimination in the United States.[15] Likewise, effect modification by socioeconomic status encompasses the myriad ways by which differences in socioeconomic resources impact health, for example by creating differentials in access to quality health care.[16] This perspective builds on research that acknowledges social conditions as a fundamental cause of disease, as these conditions have the capacity to impact multiple disease states and health outcomes by mediating access to resources.[17,18]

Methods for Examining Differential Response to Social and Policy Factors

From a methodological perspective, examining differential vulnerability entails a statistical exploration of effect modification, where modifying variables include sociodemographic characteristics, such as race/ethnicity, education, poverty status, gender/sex, or sexual orientation. Effect modification analyses, which explore whether associations between an exposure and outcome vary across levels of the modifying variable, should be distinguished from interaction analyses, which focus on causal interactions between two exposures.[19] In this dissertation, I explore effect modification through the use of interaction terms (Chapter 2), as

well as stratified models (Chapters 3 and 4). The inclusion of interaction terms in regression models provides a statistical test of whether associations between exposure and outcome differ across sociodemographic groups. On the other hand, stratification provides point estimates and confidence intervals for each stratum of interest, but may not explicitly test for differences between groups. The choice between interaction terms and stratified models was largely based on the goal of each analysis in this dissertation. In all analyses of effect modification, I use statistical packages to compute and plot predicted probabilities of the outcome across effect modifying strata in order to aid interpretation.[20]

For models with interaction terms, I follow the recommended practice of examining the significance of interaction terms on both the multiplicative and additive scales.[19–21] However, it is widely known from analyses of interaction effects that if both exposures are associated with the outcome, interaction will be present on at least one scale, and that the absence of an interaction on one scale implies the presence of an interaction on the other.[21,22] Therefore, I focus primarily on the additive scale in interpreting results, given the relevance of the additive scale to public health interventions.[21]

National Trends in SHS Exposure

Biomarker data suggests that exposure to SHS in the United States has declined substantially over the past several decades. Studies using measures derived from National Health and Nutrition Examination Survey (NHANES) data have found that concentrations of serum cotinine (a metabolite of nicotine) among all U.S. nonsmokers declined by 70% between 1988 and 2002,[23] and that geometric means of serum cotinine levels declined 25% between 2001 and 2010 among nonsmoking workers.[24] While evidence is fairly conclusive that SHS exposure has declined for the U.S. population as a whole since the late 1980s, research on recent trends is

more mixed. Using NHANES data, Chen et al. (2010) found evidence of a leveling off of serum cotinine levels within the U.S. between 2002 and 2006, as compared to the period of 1988-2002.[25] More recently, Dai et al. (2017) reported a modest increase in serum cotinine levels among nonsmoking workers in the U.S. between 2010 and 2015.[26]

In addition to temporal variation in SHS exposure, SHS trends appear dissimilar across population subgroups, including across racial/ethnic and SES groups, and across genders. Prior studies using NHANES data have found that, historically, non-Hispanic Black (hereafter, Black) nonsmokers were more likely than individuals of other racial/ethnic groups to have detectable serum cotinine levels, while Mexican-Americans were the least likely to have detectable serum cotinine levels.[27,28] Likewise, in a study of serum cotinine among children using data from 2003-2006 NHANES surveys, Black children who were not exposed to SHS within their homes were more likely to have detectable SHS exposure as compared to children of other races.[29] Moreover, the gap in SHS exposure across racial/ethnic groups may have increased over time.[27] Lower SES is also associated with higher levels of SHS exposure, and declines in SHS between 1998 and 2010 were more pronounced among higher SES individuals than among lower SES individuals.[30] Higher levels of SHS exposure have also been observed among males and individuals working in blue-collar professions, relative to females and individuals working in white-collar professions.[24,26,25,27]

Smoke-free Polices and Patterns of SHS Exposure

Smoke-free laws may affect exposure to SHS by regulating smoking in public spaces, as well as by impacting norms around smoking behavior.[31] With regard to the direct regulation of smoking in public spaces, there is a substantial body of evidence that smoke-free laws are effective in reducing SHS exposure and improving air quality.[32–37] Multiple studies examining

smoke-free laws have found associations between the laws and reduced particulate concentrations in venues covered by the laws,[38] as well as reductions in measured exposure to SHS among workers employed in venues covered by the laws and in the general population.[39–45,36,46,47,48,34] Beyond their associations with measured SHS exposure, smoke-free laws have also been found to be associated with improved health outcomes, including reductions in the probability of low birth weight,[32] reductions in respiratory symptoms and inflammatory markers,[49] and reduced hospital admissions for acute coronary syndrome (ACS).[50]

The passage of smoke-free laws; however, is patterned by measures of SES and race/ethnicity. For example, communities with higher proportions of poorer households and households with less education have historically been less likely to be covered by smoke-free laws, particularly in workplaces.[12,51,52] Likewise, in an analysis of the diffusion of smoke-free law coverage between 2000 and 2009, Gonzalez et. al. (2013) found that Hispanic and Asian populations were more likely to be covered by smoke-free laws in restaurants and bars than Black and Non-Hispanic White (hereafter, White) populations.[13] Such patterns suggest that disparities in SHS exposure over time may be explained, at least in part, by disparities in smoke-free law coverage.

Smoke-free laws may also shape disparities in SHS exposure if there is effect modification of the relationship between the laws and SHS exposure. For example, in an analysis of smoke-free law coverage and serum cotinine levels among U.S. adults between 1999 and 2002, Pickett et al. (2006) found that in counties with less than extensive smoke-free law coverage, men were more likely than women to be exposed to SHS.[34] No evidence of effect modification was observed with regard to race/ethnicity, age, or education.[34] In McGeary et al.'s (2017) study of smoke-free law coverage and health outcomes among infants and children, the

greatest improvements in health associated with smoke-free laws were seen among mothers with the lowest levels of education, as compared to mothers with higher levels of education.[32]

Mechanisms of effect modification may include differential compliance, differential exposure to environments regulated by smoke-free laws (e.g. indoor workplaces), or differential changes in smoking behavior associated with the law's implementation. While compliance with smoke-free laws tends to be high in the United States,[53] qualitative research has suggested that instances of non-compliance may disproportionately affect lower-SES individuals. In response to smoke-free bar policies, for example, smokers may tend to gather in non-compliant bars, increasing SHS exposure for low-income workers in those environments.[54] Prior research also suggests that smoke-free laws may differentially affect home smoking behavior across sociodemographic groups, which could in turn impact SHS exposure disparities, although findings regarding the directions of these associations are not conclusive. Glantz et al. (2011) found that smoke-free law coverage was positively associated with home smoking bans, and that there were significant interactions between sociodemographic factors and household smoking status in predicting responsiveness to the smoke-free laws.[55] In particular, males, married individuals, and adults with a college education were more likely to implement a home smoking rule in response to a smoke-free law if they lived in smoking households, compared to nonsmoking households. In an analysis of smoke-free laws and SHS exposure, Adda and Cornaglia (2006) found that smoke-free laws were associated with reduced serum cotinine among higher-income smokers but were paradoxically associated with increased serum cotinine levels among lower-income nonsmokers because smoking behavior appeared to be displaced from public venues to home environments.[56] Complex relationships between smoke-free laws, smoking norms, and compensatory behavior suggest that the impact of smoke-free laws on

overall SHS exposure may vary according to sociodemographic characteristics, with important implications for health equity.

Motivation for Examining Drivers of Disparities in SHS Exposure

Trends in SHS exposure across sociodemographic groups highlight the need to better understand potential policy levers for reducing SHS exposure, while also considering the impact of these interventions on health equity. However, there has been surprisingly little literature that has systematically examined drivers of SHS exposure disparities in the United States, particularly in recent years. In Chapter 2, I exploited spatial and temporal heterogeneity in smoke-free laws between 1999 and 2014 to explore the contribution of these laws to patterns of SHS exposure. In Chapter 3, I adopted attributable fraction and simulation modeling methods to explore disparities in SHS-attributable mortality and project likely SHS exposure scenarios through 2040. Taken together, these chapters shed light on the relationship between smoke-free laws and disparities in SHS exposure, the temporal evolution of disparities in SHS-attributable mortality, and potential interventions to reduce the prevalence of SHS exposure across all population groups in the United States.

Smoking Patterns among Sexual Minorities in the United States

Smoking disparities by sexual orientation in the United States are well-documented. Data from the National Health Interview Survey (NHIS) suggests that 20.6% of SM adults were current smokers in 2018, compared to 13.5% of heterosexual adults.[57] Disparities appear to be particularly pronounced among females – according to data from the 2012-2013 National Adult Tobacco Survey (NATS), the prevalence of smoking was 36.0% among bisexual females, compared to 22.2% among lesbians and 14.3% among heterosexual females.[58]

A systematic review focused on the etiology of smoking disparities by sexual orientation found that many risk factors underlying the high smoking prevalence in SM communities are shared with the broader population, while some are unique to SM individuals. Factors that predispose both SM and heterosexual adults towards smoking, include younger age,[59–61] lower levels of educational attainment,[59,62–64] alcohol use,[59,65] depression or depressive symptoms,[59,60,66] stress,[61,67] and feelings of vicitimization.[68,69] Other risk factors that contribute to the disparity between SM and heterosexual individuals are more specific to SM communities, including experiences of stigmatization and discrimination on the basis of SM status,[70–72] as well as high levels of exposure to targeted tobacco industry marketing.[73–75]

Stigma and Smoking among Sexual Minorities

The work in this dissertation builds on the "minority stress hypothesis,"[70] which posits that exposure to stigma and discrimination associated with being a member of a marginalized group is causally associated with mental health disorders, as well as other related behaviors, including substance use. In recent years, researchers have extended the minority stress hypothesis to operationalize measures of stigma directed towards SM individuals on multiple levels – ranging from individual, to interpersonal, to structural stigma.[76] Individual-level stigma refers to the process in which stigma is internalized and expressed, for example through internalized heteronormativity, rejection sensitivity, or concealment of sexual identity. Interpersonal stigma encompasses stigmatizing interactions that occur between stigmatized and non-stigmatized individuals and include incidents of bullying or violence. Finally, structural stigma encompasses discriminatory policies and institutional practices – for example laws that ban same-sex marriage or permit employment discrimination on the basis of sexual orientation.[76]

A growing number of studies have begun to examine the relationship between stigma, minority stress, and health. At the individual level, studies on individual stigma have suggested that it is linked with a range of poor health outcomes. In a study of same-sex male couples, internalized heterosexism was found to be associated with a higher odds of self-reported smoking.[77] Likewise, in a study of both male and female SM adults, internalized heterosexism was found to be associated with higher rates of substance use.[78] A recently published meta-analysis concluded that there is a statistically significant association between internalized heterosexism and depression among SM individuals.[79] Studies of individual stigma appear sensitive to the operationalization of the stigma measure and the outcome. For example, Rendina et al. (2017) found that internalized heterosexism was associated with poorer mental health outcomes, but that there was no association between rejection sensitivity and mental health endpoints.[80]

Much work has also focused on the health effects of interpersonal and structural stigma, often through the lens of measuring perceived discrimination or victimization on the basis of sexual orientation. Self-reported past instances of sexual orientation discrimination and victimization have been tied to lower engagement in health care[81] and lower rates of uptake and awareness of HIV pre-prophylaxis,[82] as well as poorer self-rated health and functional status.[83] These exposures have also been found to be associated with higher risk of drinking,[84] cigarette smoking,[64,85] tobacco use disorder,[85] and STIs,[86] as well as increased suicidality among youth,[87] and suicidal ideation among LGBTQ (lesbian, gay, bisexual, transgender, queer) people of color.[88] On the other hand, other studies have failed to find consistent associations between perceived discrimination and poor mental health and substance use outcomes.[64,86,89] Variability in findings may be due in part to differences in the operationalization of exposure constructs or

outcome measures used, or differences in the underlying population being studied. For example, there is evidence of effect modification by sexual orientation category (e.g. gay/lesbian v. bisexual), both in terms of the quantity and type of discrimination perceived, and its relationship with health outcomes.[90–93] In particular, while bisexual individuals appear to be at particularly high risk for tobacco and alcohol use,[85,93] they may also be less likely to experience discrimination on the basis of their sexual orientation, compared to lesbian/gay individuals.[85] Other studies have explored effect modification of perceived discrimination by sex,[94] race/ethnicity,[93]and education status,[93] as well as interactions between perceived discrimination on the basis of sexual orientation and other instances of discrimination, including on the basis of gender or race/ethnicity.[95]

A final set of studies moves from individual stigma and measures of perceived interpersonal and structural stigma to explore contextual drivers of structural stigma and its relationship with health outcomes.[76] These studies use exogenous variables to capture the extent of structural stigma in an individual's area of residence. Papers examining the impact of structural stigma have found that living in areas with less structural stigma is associated with higher levels of satisfaction with health care,[96] reduced hate crime incidence,[97] and higher levels of wellbeing among SM adults.[98] Conversely, living in areas with higher levels of structural stigma has been associated with higher levels of depressive symptoms,[99] lower self-esteem,[99] increases in self-reported stress,[100] higher levels of smoking,[101,102(p2)] higher levels of psychiatric disorders,[103,104] higher levels of marijuana and illicit drug use,[105] decreases in life expectancy,[106] increased sexual risk behavior,[107] decreases in life satisfaction,[108,109] blunted cortisol responses to stress,[110] and worse mental health and overall health.[109] Also similar to the literature on perceived discrimination, there is evidence of interactions between structural stigma and both

race/ethnicity and education in predicting health outcomes.[111] In a study of sexual minority females, for example, Everett et al. (2016) found that legalizing civil unions in Illinois was associated with reduced rates of depressive symptoms, and that this relationship was strongest among racial/ethnic minorities and among females with the lowest levels of education.[111]

A range of structural stigma variables have been used in prior literature, often focusing on county- or state-level measures. These measures may be comprised of individual policies (e.g., same-sex marriage) or indices of policies in combination with data on the density of same-sex couples, public opinion, or other factors, including population composition by political affiliation.[76] Constructing a meaningful and internally reliable measure of structural stigma is characterized by a number of challenges. While single policy measures are appealing in that they bypass concerns about internal reliability, they may not be broad enough to capture the range of pathways through which SM adults may experience structural stigma. On the other hand, indices of structural stigma that attempt to reduce dimensionality in statistical models run the risk of conflating separate constructs. In developing composite measures of structural stigma, previous research has emphasized the importance of validating an index by examining internal reliability. Validation approaches including calculating the Cronbach's alpha between the individual components and/or examining eigenvalues associated with a principal components analysis (PCA).[102]

Motivation for Examining Structural Stigma and SM Smoking Patterns

While exposure to stigma has been found to be associated with health outcomes among sexual minorities, there are several gaps in the literature, particularly with regard to the association between structural stigma and smoking. We know particularly little about the relationship between structural stigma and smoking in recent years, as policies and discourse

towards SM individuals has changed substantively in the United States.[72] It is also unclear whether associations between stigma and smoking persist after adjusting for tobacco control policy environments, or whether these associations varies by sex. In Chapter 4, I adapted a previously validated index measure of structural stigma to combine information on state-level policies, density of same-sex couples, and public opinion towards same-sex marriage. I then explored whether this composite variable was associated with smoking among SM and heterosexual adults in a nationally representative sample, and furthermore whether the association appeared to vary for SM men and women. In doing so, this analysis sheds light on the extent to which structural stigma remains an important explanatory variable for the high risk of smoking in SM communities.

Specific Aims and Hypotheses

In sum, the chapters in this dissertation examine structural drivers of disparities in tobacco-related health outcomes, focusing on sociodemographic disparities in SHS exposure and disparities in smoking between SM and heterosexual adults. By combining a variety of approaches (e.g., regression analysis and simulation modeling), these analyses provide insight into the potential impacts of policy interventions on tobacco-related health outcomes and health equity. The specific aims and hypotheses associated with this dissertation are included below.

Specific Aim 1 (Chapter 2)

To determine the relationship between county-level smoke-free law coverage in workplaces and hospitality venues (restaurants and bars) and trends in secondhand smoke (SHS) exposure among nonsmokers, and to explore whether there is effect modification with regard to the relationship between smoke-free law coverage and SHS exposure by race/ethnicity, gender, education, and PIR, as well as the intersection of these variables with age.

Sub Aims

A. Examine whether county-level smoke-free law coverage of workplaces and hospitality venues was associated with SHS exposure within the U.S. population between 1999 and 2014.
B. Explore the potential for effect modification of the relationship between smoke-free laws and SHS exposure by race/ethnicity, gender, education, and PIR, as well as the intersections of these socio-demographic variables with age.

Hypotheses

A. Smoke-free law coverage in both workplaces and hospitality venues will be associated with lower levels of SHS exposure between 1999 and 2014.
B. The relationship between smoke-free law coverage and SHS exposure will be modified by race/ethnicity, gender, education, PIR, and age due to differences in employment, recreation patterns, and smoking norms among population subgroups. For example:
    - We hypothesize that the relationship between ecologic measures of smoke-free workplace law coverage and SHS exposure will be attenuated among Black and Hispanic adults, relative to White adults, given that White individuals are more likely to work in professions where they would be exposed to smoke-free laws.[112,113] Likewise, I hypothesize that the measured impact of hospitality laws will be attenuated in Black individuals, as there is some evidence that Black adults are less likely to report consuming commercially prepared meals, and thus may be less likely to spend time in hospitality environments, compared to other racial/ethnic groups.[114]

- With regard to measures of SES (i.e., education and PIR), prior evidence is mixed with regard to the relationship between comprehensive smoke-free laws and SHS exposure, as well as associated health outcomes.[115,116] Occupational patterns suggest that individuals with lower SES may be less likely to be exposed to smoke-free laws through their working environments,[113] which would imply that the relationship between ecologic measures of smoke-free workplace law coverage and SHS exposure would be attenuated among low-SES individuals, compared to high-SES individuals.
- We hypothesize that reductions in SHS exposure associated with smoke-free laws will be greater among males than females, consistent with prior research on smoke-free laws and SHS between 1999 and 2002.[34]
- Finally, I hypothesize that the impact of smoke-free workplace laws will be strongest for middle-aged individuals (between the ages of 40 and 59), as preliminary analyses of National Health and Nutrition Examination Survey (NHANES) data suggests that these individuals work more hours on average, compared to younger or older adults.[117] Similarly, I hypothesize that associations between smoke-free hospitality laws and SHS exposure will be strongest in younger adults, as evidence suggests that younger adults are more likely to be participate in social smoking, often in public environments.[118]

Specific Aim 2 (Chapter 3)

To quantify SHS-attributable mortality between 1999 and 2016 by race/ethnicity and sex, and to use simulation modeling to project potential patterns of SHS exposure disparities through 2040 under alternative intervention scenarios.

Sub-Aims

A. Use SHS exposure biomarker data and attributable fraction methods to calculate annual deaths attributable to SHS exposure between 1999 and 2016 for White males and females, Black males and females, Hispanic males and females, and males and females of other or multiple races.
B. Develop a simulation model based on difference equations to predict distributions of SHS exposure within population groups by race/ethnicity and sex through 2040 under a 1) baseline scenario, 2) a scenario with an intervention that increases smoking cessation, 3) a scenario with an intervention that weakens the association between smoking prevalence and the prevalence of SHS exposure, and 4) a scenario with a combined intervention focused on increasing smoking cessation and weakening the association between smoking prevalence and the prevalence of SHS exposure.

Hypotheses

A. SHS exposure will be associated with disparities in mortality due to differential SHS exposure patterns across racial/ethnic groups.[119] Specifically, Black adults will bear a disproportionately high burden of SHS-attributable mortality, whereas Hispanic adults will experience lower levels of SHS-attributable mortality.
B. The simulation model will predict that interventions focused on weakening the link between smoking prevalence and SHS exposure are more likely to reduce SHS

exposure disparities, compared to interventions focused on smoking cessation, given already steep declines in smoking prevalence in the U.S. population in recent years.[120]

Specific Aim 3 (Chapter 4)

To evaluate the association between a state-level index of structural stigma related to sexual orientation and disparities in current smoking between sexual minority (SM) and heterosexual adults.

Sub-aims

A. Adapt a previously validated measure of state-level structural stigma related to sexual orientation, including state-level policies linked to sexual minority discrimination, state-level density of same-sex couples, and public opinions towards same-sex marriage.
B. Evaluate whether smoking prevalence among SM and heterosexual adults is associated with state-level structural stigma, and further explore whether the relationship between stigma and smoking among SM adults is modified by sex (male v. female).

Hypotheses

A. State-level policies relating to SM discrimination, the proportion of the state household population comprised of same-sex couples, and public attitudes regarding SM relationships will have high levels of concordance and can be combined into a single index that is an internally reliable measure of structural stigma at the state level.
B. Exposure to structural stigma will be associated with higher levels of smoking among SM adults, but not among heterosexual adults. Developing a hypothesis with regard

to additional effect modification by sex is challenging as it requires considering both sexual orientation disparities in smoking across sex, as well as the role of stigma in shaping these disparities. On the one hand, data from national surveys suggests that sexual orientation disparities are more pronounced among females than males.[121] On the other hand, prior research from a nationally representative study has also found that SM males are more likely than SM females to "externalize" responses to stress (e.g. engage in coping behaviors, such as smoking).[94] Consequently, I hypothesize that while sexual orientation smoking disparities may be more pronounced among females in general, the impact of structural stigma on sexual orientation smoking patterns may be larger among males.

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CHAPTER 2: Smoke-free Laws and Disparities in Secondhand Smoke Exposure in the United States, 1999-2014

Introduction

Exposure to secondhand smoke (SHS) is associated with heart disease, lung cancer, sudden infant death syndrome, low birth weight, and asthma events in children, among other adverse health outcomes.[1] While research using both self-reported and biomarker data suggests that exposure to SHS among nonsmokers has declined significantly since the late 1980s,[2–4] disparities by race/ethnicity, gender, socioeconomic status (SES), gender, and occupation persist.[3,5–7] Black nonsmokers are more likely to be exposed to SHS compared to nonsmokers of other races and ethnicities,[5,7–9] and males have historically been more likely to be exposed, compared to females.[2] Higher levels of SHS exposure have also been associated with lower SES and with working in a blue-collar profession, compared to individuals with higher SES or those working in white-collar professions.[3,5,7,10,11]

Smoke-free laws, which restrict smoking in workplaces and/or public spaces, have been shown to be associated with improvements in air quality,[12–15] as well as reductions in SHS exposure among workers employed in venues covered by the laws and among the general nonsmoking population.[16–27] Yet despite evidence of their effectiveness on the population level, relatively little is known about whether associations between smoke-free laws and SHS exposure are modified by sociodemographic characteristics. One prior nationally representative study of U.S. adults between 1999 and 2002 found evidence that smoke-free laws were differentially

associated with SHS exposure by gender.[26] Whereas males were more likely than females to be exposed to SHS in counties without smoke-free law coverage, SHS exposure was nearly identical for males and females in counties with smoke-free law coverage.[26] No evidence of effect modification was observed with regard to race/ethnicity, age, or education.[26] Studies of smoke-free law coverage and other health outcomes—including asthma[28] and myocardial infarction[29,30]—have found evidence of effect modification by gender[29] and race/ethnicity,[28,30] though these analyses have often focused on evaluating a single local or state-level policy. A study examining the impact of smoke-free laws on birth-related outcomes using national data found that gains were most pronounced among mothers with lower levels of education, compared to mothers with higher levels of education.[31] While this body of research suggests that smoke-free laws may contribute to heterogeneous patterns of SHS exposure and related health outcomes, we lack systematic evidence of the equity effects of these policies, particularly on a national scale and in recent years.

In this study, we combined information on smoke-free laws with nationally representative biomarker data to explore the relationship between smoke-free laws and SHS exposure patterns in the United States between 1999 and 2014. We then systematically examined whether the association between smoke-free laws and SHS was modified by sociodemographic factors, including race/ethnicity, gender, education, poverty income ratio (PIR), and the intersection of these variables with age.

Methods

Analytic sample. The study sample was drawn from continuous National Health and Nutrition Examination Survey (NHANES) data, 1999-2014. NHANES combines interviews and physical examinations and is designed to be nationally representative of the health status of the civilian,

noninstitutionalized U.S. household population.[32] NHANES uses a complex, multistage probability sampling design which consists of first selecting primary sampling units (PSUs) consisting of single or multiple counties, then selecting segments within PSUs consisting of blocks or clusters of households, followed by households within segments and individuals within households. The survey is conducted in 2-year cycles, during which approximately 12,000 individuals from 30 U.S. counties are selected to participate.[32]

We restricted the analysis to adults (ages 25+) within the NHANES sample with information on serum cotinine, a metabolite of nicotine.[33] We further limited our analytic sample to nonsmokers by using previously established cut points of serum cotinine to distinguish smokers from nonsmokers.[33] These cut points were specific to each racial/ethnic group (6 ng/mL for Black, 5 ng/mL for White, and 1 ng/mL for Mexican-Americans).[33] We applied the cut-point for Mexican-Americans to all Hispanic participants in the study. For individuals who did not fall within the Black, White, or Hispanic categories (e.g., other non-Hispanic race or two or more racial categories), we used a recommended cut point for the overall adult population (3 ng/mL).[33] Cut-points were also available by gender (male and female), however, reliable cut-points by both race/ethnicity and gender (in combination) were not available.[33] We chose to use specific cut-points for racial/ethnic groups, given that the optimal cut-point values varied more substantially across racial/ethnic groups than between males and females. From the biomarker-defined nonsmoker population, we also excluded any individuals who reported using a product containing nicotine within the five days preceding the serum collection. The lower bound of the age range (age 25) allowed for completion of secondary and/or college education and has previously been used in studies examining the relationship between SES and health.[34,35]

Smoke-free law data. We derived data on county-level smoke-free law coverage from the American Nonsmokers' Rights Foundation (ANRF) Tobacco Control Laws Database.[36] We combined data on smoke-free laws passed at the state, county, or local level with population data from the Census Bureau's Cities and Towns Population Totals dataset.[37] Merging ANRF and Census population data, we calculated the percent of individuals in each county covered by laws passed at any jurisdictional level, for each month and year over the course of the study period, as described in previous literature.[38] We created separate variables to describe smoke-free law coverage for workplaces and hospitality venues (restaurants or bars). Data on restaurants and bars were combined into a single variable due to the high correlation between coverage levels for the individual venues. Continuous variables representing the percent of the county population covered by the laws were transformed into binary variables, where a county was considered "covered" by a smoke-free law if at least 50% of the population was covered. We merged this data with NHANES data based on county code, as well as month and year of the lab draw for the serum cotinine assessment. Because geographic variables including county are not publicly available in NHANES, county-level data were accessed through the Research Data Center (RDC) at the University of Michigan. Data collection for NHANES was approved by the National Center for Health Statistics (NCHS) Research Ethics Review Board (ERB). Analysis of de-identified data from NHANES is exempt from the federal regulations for the protection of human research participants. Analysis of restricted NHANES data through the NCHS RDC is also approved by the NCHS ERB. This analysis was reviewed by the University of Michigan Institutional Review Board and was deemed "not regulated".

SHS exposure data. Information on recent SHS exposure was derived from serum cotinine data. Exposed nonsmokers were respondents with measured serum cotinine above the limit of

detection (LOD). The limit of detection for cotinine changed over the study period, decreasing from .05 ng/mL in 1999-2000 to .015 ng/mL, beginning in 2001-2002.[39,40] To maintain consistency over the course of the study, individuals with measures of serum cotinine <.05 ng/mL were considered unexposed. For regression models and descriptive statistics using a continuous measure of serum cotinine, we replaced all values less than .05 ng/mL with .05 divided by the square root of 2.[39]

Socio-demographic variables. We incorporated information on several individual-level sociodemographic characteristics included in the NHANES survey. We explored effect modification by age category (25-39, 40-59, 60+); race/ethnicity (White, Hispanic, Black, and other non-Hispanic, including multi-racial); gender (male and female); education (less than high school, high school graduate or equivalent, some college, and college graduate or above); and quartiles of poverty income ratio (PIR), which represents a ratio of family income to the poverty threshold. While we are using "gender" to indicate whether a respondent was classified as male or female, it should be noted that the NHANES survey did not distinguish between gender and sex, and that the survey administrator was instructed to ask if an individual identified as male or female only "if not obvious."[41] PIR measures in NHANES are based on Health and Human Services poverty calculation guidelines to determine eligibility for means-tested federal programs.[42] The lowest PIR quartile in this study represented the lowest ratio of family income to the poverty threshold. Continuous age was also included in analytic models as a covariate.

State-level covariate data. Several state-level variables were included in statistical models to adjust for other tobacco control policies, as well as state-level demographic factors. Average price per pack of cigarettes at the state level was derived from "The Tax Burden on Tobacco, Volume 51, 1970-2016", published by the Centers for Disease Control and Prevention (CDC).[43]

A variable representing state tobacco control expenditures per capita was included in models to adjust for other tobacco control initiatives not captured by smoke-free laws or price measures. Both price and spending measures were inflation-adjusted to the year 2000. Because NHANES samples different counties from different states each year, a set of state-level demographic covariates were used in statistical models instead of state fixed effects. We derived the annual percent of the state population that was unemployed from the U.S. Bureau of Labor Local Area Unemployment Statistics,[44] and we included information on the percent of the state population below the federal poverty level from the U.S. Census Bureau Small Area Income and Poverty Estimates Program.[45] Information on the percent of the population with at least a Bachelor's degree or higher was derived from U.S. Census Bureau estimates and 5-year estimates from the American Community Survey, as compiled by the U.S. Department of Agriculture Economic Research Service.[46] Finally, we included Census Bureau information on each state's racial/ethnic composition (percent Black and percent Hispanic) using data on the resident population from intercensal estimates[47] and data downloaded from the American FactFinder database.[48] Continuous state-level variables were transformed into quintiles in order to preserve confidentiality of respondents within the restricted NHANES dataset. The value for each quintile was represented by the midpoint value within that category, and quintiles were included in regression models as continuous variables.

Statistical Analysis. Because workplace and hospitality smoke-free law variables were highly collinear (ρ=.74 among all U.S. counties during the study period), we examined associations between each law type and SHS exposure in separate models. We used logistic regression to explore associations in bivariate models and adjusted main effects models, controlling for individual-level sociodemographic variables and state-level variables, as well as a linear time

trend. We chose to use a continuous variable to represent secular time trends, instead of year fixed effects, due to concerns about positivity violations in smoke-free law exposure among nonsmokers across all waves of the NHANES sample. We then tested effect modification by each sociodemographic variable by including interaction terms between the smoke-free law exposure and the variable in separate models. While we reported both additive and multiplicative p-values associated with interaction terms, we focused on interpreting the additive p-values based on average marginal effects (AMEs).[49] In this study, marginal p-values from interaction models represented whether the average impact of a change in smoke-free law coverage on the probability of SHS exposure was statistically different across sociodemographic groups.[49] We examined associations within the full sample, as well as associations within three separate age strata: 25-39, 40-59, 60+. While all effect modification tests were specified a priori, we applied the Benjamini-Hochberg correction method[50] with a false discovery rate of .05 to all p-values within each age category for each exposure, due to the large number of models estimated. To aid interpretability, we also plotted predicted probabilities from main effects models and interaction models with significant additive p-values.[51]

In addition to the primary analysis, we conducted a number of sensitivity analyses. We explored several specifications of the exposure variable in order to examine associations with 1) any smoke-free law coverage (workplace OR hospitality smoke-free coverage) and 2) comprehensive smoke-free law coverage (workplace AND hospitality smoke-free coverage). We also stratified main effects models according to whether or not the respondent reported living with an individual who smoked inside the home. When we stratified by home smoking environment, we estimated two sets of models: one with a binary SHS exposure outcome and one with a continuous outcome (log-transformed serum cotinine). We conducted these sensitivity

analyses to assess whether there was any evidence that smoke-free laws led to displacement of smoking from public areas to home environments. Finally, we estimated models with an interaction between each smoke-free law variable and the linear time trend variable to explore whether associations between smoke-free laws and SHS exposure appeared to vary across the study period.

All analyses were conducted in Stata, version 15 using NHANES-provided survey weights and adjusting for the complex survey design.

Results

Table 2.1 includes weighted descriptive statistics for the analytic sample of nonsmoking adults. The total analytic sample included 25,444 nonsmokers. The majority of the sample was White (71.1%), followed by Hispanic (13.4%), Black (9.3%), and Other (6.2%). A slight majority of the sample was female (56.3%), and the mean age of the sample was 50.9 years. The weighted proportion of the sample that was exposed to SHS over the study period was 31.4%, while the geometric mean cotinine value was 0.058 ng/mL. When we computed average levels of exposure to smoke-free coverage across the study period, less than half of respondents lived in counties with ≥ 50% workplace smoke-free law coverage (31.4%) or hospitality smoke-free law coverage (46.0%).

Table 2.2 includes results from bivariate and adjusted main effects models, without interaction terms. In bivariate models, living in a county with ≥ 50% workplace smoke-free law coverage was significantly associated with a reduced odds of SHS exposure in the full sample and in the youngest age group. In adjusted models, higher levels of workplace smoke-free law coverage were only significantly associated with SHS exposure in the youngest age group (odds ratio (OR) = 0.81, 95% confidence interval (CI) = 0.65-0.99). Hospitality smoke-free law

coverage was associated with a lower odds of SHS exposure in all main effects model specifications. In adjusted models that included the full nonsmoker sample, living in a county with ≥50% smoke-free law coverage of hospitality venues was associated with 0.62 times the odds of SHS exposure (95% CI 0.51-0.76), compared to living in a county with <50% coverage. ORs for each age category suggested a similar effect size, with an OR of 0.58 (95% CI 0.44-0.77) for ages 25-39, 0.62 (95% CI 0.50-0.77) for ages 40-59, and 0.67 (95% CI 0.52-0.86) for ages 60+.

Table 2.3 includes additive p-values from all interaction models. Bold typeface indicates that the p-values remained significant at the 0.05 level after adjustment for multiple testing. In the full sample analyses, only the interaction between hospitality smoke-free law coverage and gender remained significant after the multiple testing correction. In age-stratified models, all significant interactions after adjustment occurred in the middle age group (ages 40-59). For workplace smoke-free laws, interactions with gender and PIR were significant; for hospitality smoke-free laws, interactions with gender were significant. Detailed results of interaction regression models are included in Supplementary Material Tables 2.1-2.8. Multiplicative interaction p-values, also adjusted for multiple testing, are included in Supplementary Table 2.9.

We graphed the probability of exposure associated with each type of smoke-free law coverage in main effects models. These graphs are included in Figure 2.1. In both sets of models, the probability of SHS exposure decreased with age. Within the full sample, the probability of SHS exposure decreased by approximately 10 percentage points in counties with ≥50% hospitality smoke-free law coverage, compared to counties with < 50% hospitality smoke-free law coverage.

We plotted results on the probability scale for additive interactions that remained significant after adjusting for multiple testing. Figure 2.2 includes probability plots associated with the interaction between hospitality smoke-free laws and gender in the full sample and for adults ages 40-59. The probability of SHS exposure among males was higher than among females, and the decline in SHS exposure associated with smoke-free law coverage was more pronounced among males than females.

Significant interactions between workplace smoke-free laws and sociodemographic variables within the middle age category (ages 40-55) are provided in Figure 2.3. For the gender interaction, females appeared to be slightly more likely to be exposed to SHS in counties with ≥50% workplace smoke-free law coverage compared to counties with <50% coverage; the association was in the opposite direction for males. For the PIR interactions, higher levels of workplace smoke-free law coverage were associated with a higher probability of SHS exposure for individuals in the lowest quartile, compared to flat or decreasing probabilities within the remaining quartiles.

As a sensitivity analysis, we examined results when alternative versions of the exposure variables were used. When "any" smoke-free law coverage was used as an exposure variable, main effects results closely resembled main effects associated with hospitality smoke-free laws, in magnitude and statistical significance. "Comprehensive" smoke-free law coverage was associated with a significantly lower probability of SHS exposure in bivariate models for ages 25-39 and ages 40-59, and in the full sample. There were no significant associations in adjusted models. Main effects results using these alternative exposure specifications are included in Supplementary Table 12. Additive and multiplicative p-values associated with interactions for "any" or "comprehensive" smoke-free law exposures are included in Supplementary Tables

2.13-2.14. For “any” coverage, there were statistically significant interactions between gender and the exposure for ages 40-59, 60+, and in the full sample, which suggested that any smoke-free law coverage was associated with narrowing the gap in exposure between males and females. For “comprehensive” coverage, statistically significant interactions all occurred within the middle age group and included interactions with gender, education, and PIR. Gaps between males and females narrowed, while interactions with education and PIR suggested that comprehensive coverage was associated with widening differentials between SES groups.

We also stratified models by whether or not nonsmokers reported living with an individual who smokes inside the home. In models using both binary and continuous outcomes, reductions in SHS exposure associated with law coverage appeared to be more pronounced among individuals who lived with smokers inside the home, although confidence intervals were quite wide, reflecting that only a small portion of the sample fell into this category. These results are included in Supplementary Tables 2.10-2.11. Finally, we explored whether associations between smoke-free laws and SHS exposure appeared to change over time by including an interaction between each smoke-free law exposure and a continuous variable representing the wave of the survey. This interaction was significant for hospitality smoke-free law coverage and suggested that the associations between coverage and a reduced probability of SHS exposure were stronger in the first wave than in the last wave. Plots of AMEs associated with the interaction between each type of smoke-free law and survey wave are included in Supplementary Figures 2.1-2.2.

Discussion

By exploiting temporal and spatial variation in the passage of smoke-free laws between 1999 and 2014, we explored the contribution of smoke-free laws to persistent SHS exposure

disparities. We found that higher levels of hospitality smoke-free law coverage were consistently associated with lower odds of SHS exposure for the full sample and across all age strata, whereas workplace smoke-free law coverage was only significantly associated with lower odds of exposure for younger adults (ages 25-39). We found several examples of potential effect modification by sociodemographic characteristics. In the full sample and in the middle age group (ages 40-59), higher levels of hospitality smoke-free law coverage appeared to be associated with a larger reduction in the probability of SHS exposure among males, compared to females. For nonsmokers in the middle age group, higher levels of workplace smoke-free law coverage were associated with an increase in SHS exposure for females and among nonsmokers in the lowest quartile of PIR, whereas associations were flat or declining among males and individuals in higher PIR quartiles.

Smoke-free laws can impact SHS exposure via multiple mechanisms, including through direct regulation of air quality in environments covered by the policies.[52] Smoke-free laws may also affect SHS exposure by impacting norms relating to smoking,[53–55] perhaps even leading to changes in smoking behavior outside of directly regulated environments.[56] For example, prior studies have found that smoke-free law coverage was associated with a higher likelihood of individuals instituting home smoking bans[57] and with decreased smoking in home environments.[31] On the other hand, Adda and Cornaglia (2006) found evidence of displacement of smoking to home environments following the introduction of a restaurant/bar smoking ban, as well as some evidence of higher cotinine levels among nonsmokers associated with smoking bans in workplaces.[58] However, associations were inconsistent and varied considerably across age groups.

While our findings regarding associations with hospitality smoke-free laws are in line with studies reporting that smoke-free laws are associated with reduced SHS exposure,[26,27] our findings regarding workplace laws suggest that these policies are not as consistently associated with a lower probability of SHS exposure, and in some cases, may be associated with higher levels of exposure. To explore a potential displacement hypothesis,[58] we repeated our main effects analyses, stratified by whether or not the respondent reported living with an individual who smokes inside the home. In logistic models with a binary SHS exposure outcome, as well as linear models with a continuous SHS exposure outcome, point estimates associated with smoke-free law coverage were generally lower among individuals who reported living with an indoor smoker. These findings are consistent with studies that have found that smoke-free laws are not associated with displacement into home environments and may even be associated with increases in voluntary home restrictions.[57,59] However, future studies should examine whether patterns of potential displacement are consistent across sociodemographic groups.

In examining potential effect modification by sociodemographic characteristics, we found evidence that workplace smoke-free laws were modified by age, as these laws were only significantly associated with SHS exposure for adults between the ages of 25 and 39. We are not aware of prior studies documenting disparate associations between smoke-free laws and SHS by age. However, these findings are in line with studies of smoking behavior, which have found that smoking bans may be particularly effective in reducing smoking among younger adults.[60–62] Variable associations by age could be due to differences in occupational patterns that would lead to differential exposure to environments affected by smoke-free laws, although age does not appear to strongly impact the likelihood of working in an indoor environment.[63] Instead, smoke-free laws may impact age-specific patterns of SHS exposure by differentially denormalizing

smoking practices,[64,65] which in turn impact the likelihood of SHS exposure. For example, denormalization of smoking has been found to be an important mediator of the association between smoking bans and smoking outcomes among young adults.[64] If younger adults are more likely to change their behavior as a result of the denormalization of smoking practices, this may help explain the relatively strong association between workplace smoke-free laws and SHS exposure among adults ages 25-39.

We also found that workplace and hospitality smoke-free laws were differentially associated with SHS exposure by gender. For hospitality laws, the reduction in SHS exposure associated with higher levels of coverage was more pronounced among males than females in the full sample, and among nonsmokers between the ages of 40 and 59. For workplace laws, higher levels of coverage were associated with higher levels of SHS exposure for females ages 40-59, whereas the association was relatively flat among males. Differential associations between smoke-free laws and SHS exposure by gender have been noted previously. In a prior study of NHANES data collected between 1999 and 2002, males were more likely than females to be exposed to SHS in counties without smoke-free laws, whereas the probability of exposure was similar in counties with extensive smoke-free law coverage.[26] Our findings are consistent with this prior study, in that they suggest that smoke-free laws may narrow exposure differentials between gender groups. One potential explanation, as has been noted previously, is that males may be more likely than females to be exposed to SHS at work, and consequently may be more likely to be impacted by smoke-free laws covering work environments.[66]

In addition, by examining effect modification by measures of SES, we contribute to the literature on the health equity impacts of tobacco control policies on tobacco-related health disparities. Specifically, we found evidence that workplace smoke-free laws were modified by

PIR for the middle age group (ages 40-59). Living in a county with ≥ 50% workplace smoke-free law coverage was associated with a higher probability of SHS exposure among individuals in the first quartile of the PIR distribution, while associations were flat or decreasing for individuals with higher PIR. These findings suggest that workplace smoke-free laws may exacerbate SHS exposure disparities within this age group. Few prior studies have examined effect modification of smoke-free laws on SHS exposure by SES. Using NHANES data from 1999-2002, Pickett et al. (2006) did not find any evidence of differential associations between smoke-free laws and SHS exposure by education.[26] However, a recent review raised concerns that lower-income women may not reap the same benefits from smoke-free policies as higher-income women, whether because the laws do not affect smoking behavior in the same way among lower-income populations or because lower-income individuals may be more likely to work in environments where smoke-free policies are not enforced.[67] On the other hand, studies of smoke-free laws and other health outcomes have suggested that these laws may have pro-equity effects. For example, McGeary et al. found that comprehensive smoke-free laws were associated with benefits in birth outcomes, particularly for lower-educated households, and that these benefits were primarily driven by reductions in SHS, rather than reductions in prenatal smoking.[31] While there is emerging evidence on potential differential associations across subpopulations, there has been a need for more direct analyses of the impact of smoke-free laws on disparities in SHS exposure.[68] The results of our study suggest that workplace smoke-free laws may contribute to SES disparities among middle-aged adults, although more evidence from longitudinal studies is needed.

In considering the implications of this study, it is also important to note that our results suggest that smoke-free laws may have had considerably weaker associations with SHS exposure

in recent years, compared to earlier years. In particular, we found that there was a significant association between a continuous variable representing survey wave and hospitality smoke-free law coverage. When we plotted the predicted change in the probability of SHS exposure associated with higher levels of law coverage in wave 1 and wave 8, point estimates were significantly below zero for all age groups and the full sample in wave 1, whereas they were statistically indistinguishable from zero in wave 8. It is possible that the impact of smoke-free laws on SHS exposure has weakened over time due to the widespread expansion of voluntary smoke-free policies.[69] In interpreting these findings, it is also important to note that we did not examine whether the effectiveness of these laws over time varied across different sociodemographic groups. This is an area for future research.

Strengths of this study include the use of nationally representative survey data over a 15-year period with significant variation in smoke-free law coverage. We distinguished between smoke-free laws in hospitality venues and workplaces, which allowed us to examine the potential for differential impacts on SHS exposure patterns associated with the regulation of different kinds of venues. By combining information on smoke-free laws with Census population data, we were able to construct smoke-free law variables that took into account laws passed at all jurisdictional levels. We controlled for a robust set of state-level factors that may confound associations between smoke-free laws and SHS exposure. Finally, by using biomarkers of SHS exposure, we avoided challenges associated with recall and self-reports of exposure to SHS.

There are also several limitations associated with this study. Data were cross-sectional, which precluded longitudinal examinations of smoke-free law passage and changes in SHS exposure. We were not able to account for the proliferation of voluntary smoke-free policies (e.g., smoking restrictions put in place by individual workplaces, bars, and restaurants) that may

have preceded smoke-free legislation in many areas.[69] We also were not able to measure compliance in this study; however, most studies have found high levels of compliance to smoke-free laws in the United States.[70] While we adjusted p-values to account for multiple comparisons, it is possible that some of our significant results were due to spurious correlations, particularly given the large number of models estimated in our analysis of effect modification.[71,72] On the other hand, there may also be instances of effect modification that were not uncovered in this study. For example, several p-values associated with the interaction between hospitality smoke-free laws and race/ethnicity were significant at the .05 level, although they did not remain significant after adjustment for multiple testing. Likewise, we observed a marginally significant p-value for the interaction between hospitality smoke-free laws and PIR within the youngest age group. These relationships deserve further scrutiny in future studies, given persistent disparities in SHS exposure by both SES and race/ethnicity.[9]

While we adjusted for a range of individual and state-level confounding variables, results may still be impacted by residual confounding. In our primary analysis, we did not examine associations with continuous serum cotinine levels, and instead focused on estimating associations with an indicator variable representing SHS exposure. It is possible that an analysis of levels of SHS exposure (versus presence of SHS exposure) may yield different findings. Finally, biomarker measures of SHS exposure are also subject to limitations. For example, individuals may exhibit variation in nicotine metabolism rates due to several factors not addressed in this study, including genetics, oral contraceptive use, and interactions with pharmaceutical drugs.[73] These factors may complicate efforts to compare cotinine levels across groups.[33,73,74] In addition, while we used cotinine cut-off points that were specific to racial/ethnic groups to distinguish smokers from nonsmokers, our sample may have been impacted by

misclassification of smoking status. Future research could explore the sensitivity of the sample and findings to alternative cotinine cut-points, such as those defined by gender, instead of race/ethnicity.[33]

Conclusions

As of January 2020, approximately 61.1% of the U.S. population was covered by comprehensive smoke-free laws in workplaces, restaurants, and bars.[75] This study suggests that there is considerable work to be done to expand smoke-free protections among nonsmokers and to eliminate disparities in SHS exposure. We found that hospitality smoke-free laws, in particular, were consistently associated with a lower likelihood of SHS exposure among nonsmokers between 1999 and 2014. Our results suggest that the association between smoke-free laws and SHS exposure may be heterogeneous across gender groups – narrowing SHS exposure differentials between males and females. We also found evidence that suggests that workplace smoke-free laws may exacerbate SHS exposure disparities among middle aged adults (ages 40-59) due to differential associations by PIR quartile. The results of this study highlight the importance of considering the equity impact of tobacco control interventions, including smoke-free laws.

Disclaimer: The findings and conclusions in this chapter are those of the authors and do not necessarily represent the views of the Research Data Center, the National Center for Health Statistics, or the Centers for Disease Control and Prevention.

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Tables

Table 2.1 Weighted descriptive statistics of analytic sample of nonsmokers, NHANES 1999-2014

	All	1999-2000	2001-2002	2003-2004	2005-2006	2007-2008	2009-2010	2011-2012	2013-2014
Total N	25,444	2,633	3,035	2,853	2,888	3,490	3,771	3,259	3,515
Sex									
Male	43.7%	42.8%	43.6%	43.0%	43.1%	44.0%	44.7%	43.6%	44.6%
Female	56.3%	57.2%	56.4%	57.0%	56.9%	56.0%	55.3%	56.4%	55.4%
Race/ethnicity									
Non-Hispanic White	71.1%	72.4%	75.3%	74.0%	73.0%	70.7%	70.3%	67.7%	66.9%
Non-Hispanic Black	9.3%	9.6%	8.6%	9.5%	10.3%	8.9%	9.1%	9.6%	9.3%
Hispanic	13.4%	14.0%	11.8%	11.3%	12.0%	13.6%	13.6%	15.0%	15.7%
Other Non-Hispanic	6.2%	4.0%	4.3%	5.3%	4.7%	6.9%	7.1%	7.7%	8.3%
Age Category									
25-39	28.6%	32.1%	28.9%	29.0%	29.2%	27.2%	27.9%	27.9%	27.9%
40-59	39.9%	37.2%	42.2%	40.4%	41.1%	41.3%	40.4%	39.0%	37.3%
60+	31.5%	30.8%	29.0%	30.6%	29.7%	31.5%	31.7%	33.1%	34.9%
Mean Age (S.D.)	50.9 (13.6)	50.1 (13.8)	50.4 (13.6)	50.8 (13.6)	50.7 (13.3)	51.2 (13.8)	51.0 (14.1)	51.3 (13.1)	51.6 (13.5)
Poverty Income Ratio Quartile									
1 (lowest)	16.3%	17.5%	16.5%	15.9%	14.0%	14.7%	16.1%	18.6%	17.5%
2	20.8%	19.7%	18.9%	21.6%	21.3%	22.6%	20.4%	20.2%	21.4%
3	28.2%	28.6%	29.2%	31.2%	29.9%	26.9%	28.4%	25.4%	27.0%
4 (highest)	34.6%	34.3%	35.4%	31.3%	34.8%	35.9%	35.1%	35.8%	34.2%
Education									
<High school	16.0%	20.7%	16.3%	16.7%	15.1%	16.8%	16.6%	14.1%	12.8%
High school or equivalent	21.1%	23.1%	22.1%	23.3%	22.5%	22.2%	19.9%	18.4%	18.3%
Some college	28.8%	26.7%	28.1%	30.1%	29.7%	28.4%	29.1%	28.4%	29.7%
College+	34.2%	29.6%	33.4%	29.9%	32.7%	32.6%	34.5%	39.1%	39.2%

Secondhand Smoke (SHS) Exposure									
Exposed	31.4%	45.9%	35.5%	41.2%	34.5%	34.5%	25.4%	19.2%	20.4%
Workplace Law Coverage (≥50%)									
Yes	31.4%	0.0%	2.5%	16.3%	15.0%	15.0%	58.8%	68.9%	58.9%
Hospitality Law Coverage (≥50%)									
Yes	46.0%	11.5%	21.0%	28.9%	19.7%	26.6%	69.4%	82.7%	89.6%
Mean ng/mL SHS (95% CI)	0.058 (0.056-0.060)	0.074 (0.068-0.081)	0.063 (0.058-0.070)	0.070 (0.061-0.081)	0.060 (0.056-0.065)	0.061 (0.056-0.067)	0.053 (0.049-0.056)	0.047 (0.045-0.049)	0.049 (0.046-0.052)

Table 1.2 Odds ratios for SHS exposure associated with smoke-free law coverage in the full sample and in age-stratified models[a,b]

Bivariate[a]	**Full Sample**	**Ages 25-39**	**Ages 40-59**	**Ages 60+**
Workplace Law (≥50%)	0.84 (0.70,0.99)	0.80 (0.65,0.99)	0.84 (0.69,1.02)	0.84 (0.66,1.07)
Hospitality Law (≥50%)	0.49 (0.42,0.58)	0.46 (0.37,0.57)	0.48 (0.40,0.57)	0.51 (0.42,0.62)
Adjusted[b]				
Workplace Law (≥50%)	0.90 (0.77,1.05)	0.81 (0.65,0.99)	0.95 (0.79,1.15)	0.92 (0.72,1.17)
Hospitality Law (≥50%)	0.62 (0.51,0.76)	0.58 (0.44,0.77)	0.62 (0.50,0.77)	0.67 (0.52,0.86)

a) Bivariate models control for survey wave (continuous variable)

b) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender); state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic); and survey wave (continuous)

Table 2.2 Additive p-values associated with interaction terms in adjusted models[a,b]

	Full Sample	**Age Category 1**	**Age Category 2**	**Age Category 3**
Workplace Laws				
Law*Race/ethnicity	0.3462	0.3722	0.1447	0.1128
Law*Gender	0.0267	0.7614	**0.0039**	0.607
Law*Education	0.5336	0.2761	0.0758	0.6376
Law*PIR	0.1023	0.6357	**0.0142**	0.0999
Hospitality Laws				
Law*Race/ethnicity	0.0274	0.1863	0.0374	0.1923
Law*Gender	**<0.0001**	0.1002	**0.0014**	0.0274
Law*Education	0.815	0.3083	0.1962	0.9106
Law*PIR	0.7647	0.0330	0.1415	0.3182

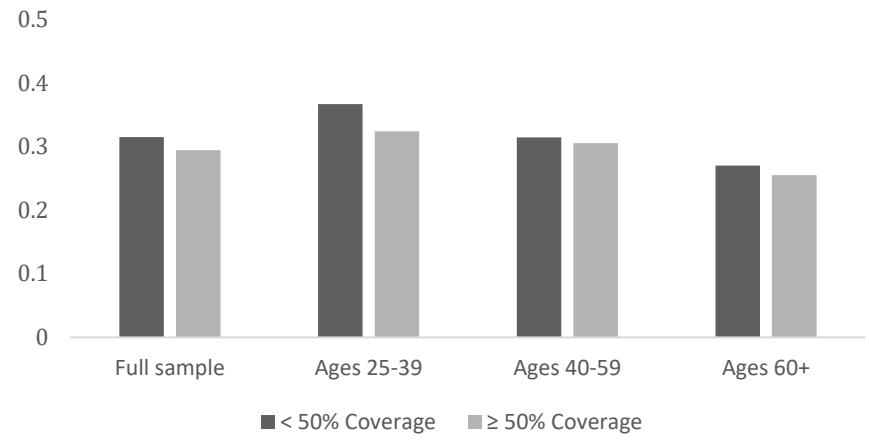
a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

b) Bold p-values represent statistical significance after multiple testing adjustment

Figures

Figure 2.1 Predicted probability of SHS exposure based on average marginal effects of county-level smoke-free law coverage of workplaces (1) and hospitality venues (2), adjusted models[a]

1)



2)

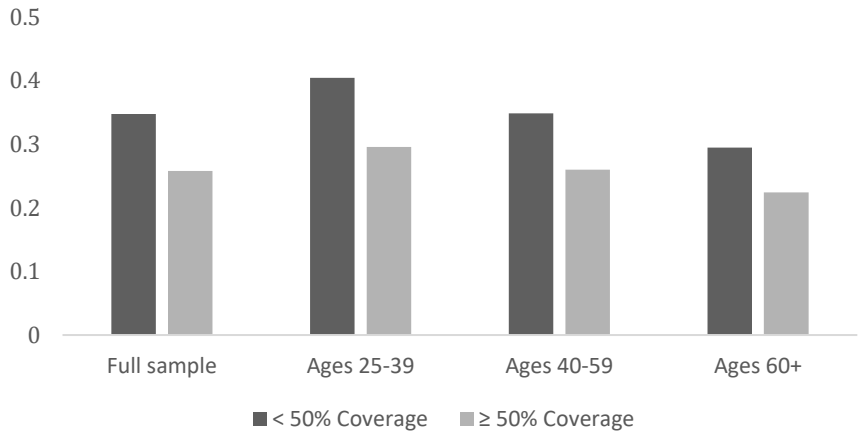
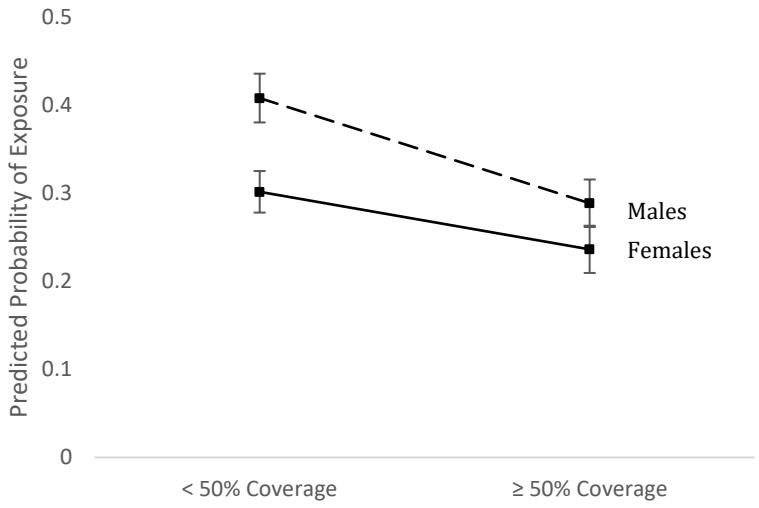


Figure 2.2 Predicted probability of SHS exposure associated with county-level hospitality smoke-free law coverage by gender for the full sample (1), and among adults ages 40-59 (2)

1)



2)

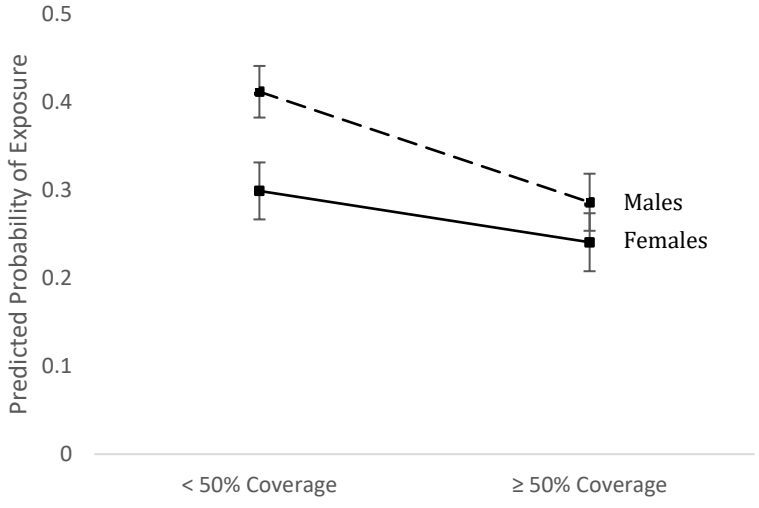
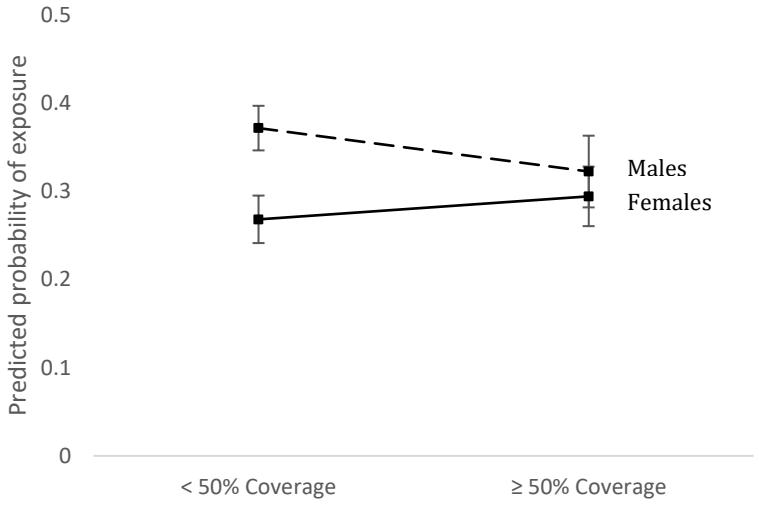
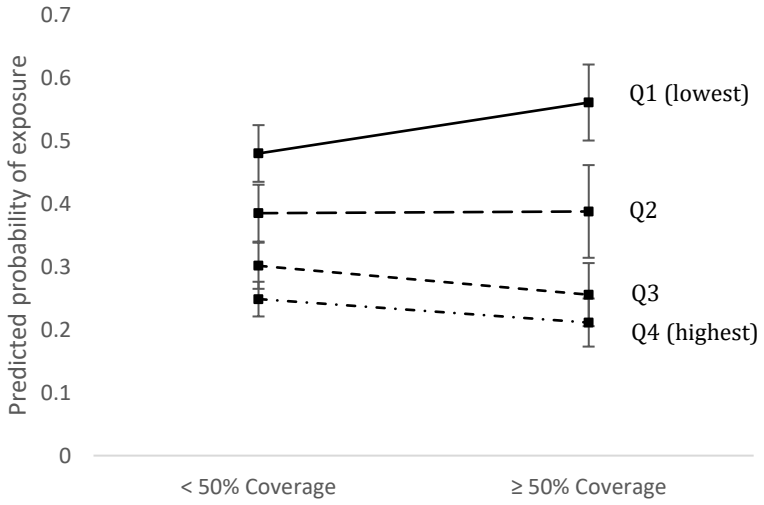


Figure 2.3 Predicted probability of SHS exposure associated with county-level workplace smoke-free law coverage by gender (1), and poverty-income-ratio (PIR) (2), ages 40-59

1)



2)



Supplementary Material

Table SM 2.1 Odds ratios for SHS exposure associated with workplace smoke-free law coverage, including interaction between coverage and race/ethnicity[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Workplace Law (≥50%)	0.84		0.73		0.88		0.89	
	[0.69,1.02]		[0.55,0.96]		[0.69,1.12]		[0.67,1.19]	
Race/ethnicity (White Ref.)								
Hispanic	0.65		0.55		0.65		0.83	
	[0.53,0.78]		[0.42,0.72]		[0.52,0.81]		[0.60,1.15]	
Black	2.02		1.91		2.04		2.11	
	[1.72,2.37]		[1.44,2.53]		[1.61,2.58]		[1.74,2.55]	
Other	1.29		1.26		1.28		1.25	
	[1.03,1.62]		[0.92,1.73]		[0.89,1.84]		[0.87,1.79]	
Workplace Law*Race/ethnicity								
Law*Hispanic	1.14		1.13		1.38		0.77	
	[0.87,1.48]		[0.74,1.70]		[0.98,1.93]		[0.49,1.21]	
Law*Black	1.19	0.3462	1.45	0.3722	1.01	0.1447	1.24	0.1128
	[0.91,1.55]		[0.96,2.20]		[0.68,1.49]		[0.84,1.85]	
Law*Other	1.37		1.29		1.5		1.51	
	[0.96,1.95]		[0.80,2.09]		[0.86,2.60]		[0.86,2.65]	

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.2 Odds ratios for SHS exposure associated with hospitality smoke-free law coverage, including interaction between coverage and race/ethnicity[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Hospitality Law (≥50%)	0.59		0.56		0.56		0.64	
	[0.47,0.74]		[0.40,0.79]		[0.43,0.72]		[0.49,0.85]	
Race/ethnicity (White Ref.)								
Hispanic	0.68		0.65		0.62		0.82	
	[0.55,0.85]		[0.48,0.89]		[0.48,0.80]		[0.56,1.21]	
Black	1.78		1.83		1.65		1.89	
	[1.50,2.11]		[1.35,2.49]		[1.27,2.14]		[1.56,2.29]	
Other	1.32		1.18		1.45		1.28	
	[0.96,1.82]		[0.75,1.83]		[0.89,2.36]		[0.75,2.18]	
Hospitality Law*Race/ethnicity								
Law*Hispanic	1.01		0.84		1.38		0.9	
	[0.78,1.30]		[0.58,1.21]		[1.00,1.90]		[0.58,1.38]	
Law*Black	1.57	0.0274	1.51	0.1863	1.68	0.0374	1.57	0.1923
	[1.21,2.03]		[1.01,2.25]		[1.19,2.39]		[1.08,2.28]	
Law*Other	1.24		1.4		1.11		1.29	
	[0.84,1.83]		[0.81,2.43]		[0.61,2.05]		[0.69,2.39]	

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.3 Odds ratios for SHS exposure associated with workplace smoke-free law coverage, including interaction between coverage and education[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Workplace Law (≥50%)	0.82		0.88		0.75		0.80	
	[0.67,1.01]		[0.65,1.18]		[0.54,1.05]		[0.55,1.15]	
Education (College+ Ref.)								
Less than High School	3.48		3.60		4.22		2.48	
	[2.97,4.08]		[2.73,4.76]		[3.31,5.39]		[2.01,3.05]	
High School or Equivalent	2.77		3.62		2.90		1.87	
	[2.37,3.24]		[2.88,4.56]		[2.32,3.62]		[1.54,2.28]	
Some College	2.02		2.49		2.13		1.32*	
	[1.74,2.34]		[2.01,3.08]		[1.73,2.64]		[1.04,1.67]	
Workplace Law*Education								
Law*Less than High School	1.25		1.07		1.67		1.2	
	[0.97,1.63]		[0.69,1.65]		[1.10,2.53]		[0.80,1.79]	
Law* High School or Equivalent	1.13	0.5336	0.98	0.2761	1.2	0.0758	1.28	0.6376
	[0.88,1.46]		[0.68,1.40]		[0.77,1.88]		[0.93,1.78]	
Law*Some College	1.05		0.75		1.38		1.11	
	[0.81,1.37]		[0.51,1.11]		[0.86,2.23]		[0.76,1.64]	

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.4 Odds ratios for SHS exposure associated with hospitality smoke-free law coverage, including interaction between coverage and education[a]

	Full Sample	**p-value**	**Ages 25-39**	**p-value**	**Ages 40-59**	**p-value**	**Ages 60+**	**p-value**
Hospitality Law (≥50%)	0.53		0.61		0.43		0.57	
	[0.41,0.70]		[0.42,0.89]		[0.32,0.59]		[0.39,0.81]	
Education (College+ Ref.)								
Less than High School	3.33		3.65		3.87		2.39	
	[2.78,4.00]		[2.62,5.09]		[2.89,5.18]		[1.90,3.00]	
High School or Equivalent	2.71		3.75		2.79		1.80	
	[2.27,3.23]		[2.91,4.84]		[2.17,3.60]		[1.49,2.18]	
Some College	1.88		2.32		1.95		1.29	
	[1.59,2.24]		[1.81,2.98]		[1.52,2.49]		[1.01,1.64]	
Hospitality Law*Education								
Law*Less than High School	1.32		1.00		1.88		1.25	
	[1.02,1.70]		[0.65,1.53]		[1.28,2.75]		[0.90,1.74]	
Law* High School or Equivalent	1.16	0.8150	0.87	0.3083	1.33	0.1962	1.32	0.9106
	[0.89,1.50]		[0.60,1.25]		[0.90,1.97]		[0.93,1.87]	
Law*Some College	1.25		0.94		1.73		1.16	
	[0.99,1.57]		[0.66,1.34]		[1.18,2.53]		[0.79,1.70]	

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.5 Odds ratios for SHS exposure associated with workplace smoke-free law coverage, including interaction between coverage and gender[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Workplace Law (≥50%)	0.83 [0.70,0.99]		0.83 [0.65,1.08]		0.78 [0.61,0.98]		0.89 [0.67,1.19]	
Gender (Male Ref.)								
Female	0.63 [0.58,0.67]		0.66 [0.57,0.77]		0.58 [0.51,0.65]		0.68 [0.60,0.77]	
Workplace Law*Gender								
Law*Female	1.16 [1.01,1.34]	0.0267	0.94 [0.70,1.24]	0.7614	1.49 [1.13,1.96]	0.0039	1.06 [0.82,1.36]	0.6070

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.6 Odds ratios for SHS exposure associated with hospitality smoke-free law coverage, including interaction between coverage and gender[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Hospitality Law (≥50%)	0.56 [0.46,0.68]		0.54 [0.39,0.74]		0.53 [0.43,0.66]		0.60 [0.44,0.81]	
Gender (Male Ref.)								
Female	0.60 [0.55,0.64]		0.60 [0.51,0.70]		0.57 [0.50,0.66]		0.64 [0.56,0.72]	
Hospitality Law*Gender								
Law*Female	1.25 [1.10,1.42]	<0.0001	1.16 [0.90,1.49]	0.1002	1.34 [1.07,1.69]	0.0014	1.23 [0.98,1.56]	0.0274

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.7 Odds ratios for SHS exposure associated with workplace smoke-free law coverage, including interaction between coverage and PIR[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Workplace Law (≥50%)	0.81		0.89		0.8		0.82	
	[0.65,1.00]		[0.62,1.27]		[0.60,1.05]		[0.56,1.19]	
PIR Quartile (Q4 Ref.)								
Q1 (lowest)	2.92		3.32		3.09		2.96	
	[2.50,3.42]		[2.56,4.31]		[2.42,3.96]		[2.31,3.79]	
Q2	1.80		1.95		2.01		1.87	
	[1.53,2.11]		[1.46,2.60]		[1.62,2.50]		[1.50,2.34]	
Q3	1.40		1.41		1.34		1.67	
	[1.20,1.63]		[1.11,1.79]		[1.08,1.65]		[1.33,2.11]	
Workplace Law*PIR Quartile								
Law*Q1 (lowest)	1.29		0.81		1.80		1.31	
	[0.98,1.70]		[0.52,1.26]		[1.21,2.69]		[0.89,1.94]	
Law*Q2	1.25	0.1023	0.94	0.6357	1.27	0.0142	1.36	0.0999
	[0.92,1.68]		[0.58,1.54]		[0.82,1.98]		[0.83,2.22]	
Law*Q3	0.93		0.88		0.98		0.85	
	[0.70,1.22]		[0.56,1.39]		[0.67,1.44]		[0.51,1.42]	

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.8 Odds ratios for SHS exposure associated with hospitality smoke-free law coverage, including interaction between coverage and PIR[a]

	Full Sample	p-value	Ages 25-39	p-value	Ages 40-59	p-value	Ages 60+	p-value
Hospitality Law (≥50%)	0.53		0.64		0.50		0.51	
	[0.40,0.69]		[0.42,0.95]		[0.36,0.69]		[0.35,0.76]	
PIR Quartile (Q4 Ref.)								
Q1 (lowest)	2.90		3.74		2.99		2.75	
	[2.47,3.40]		[2.78,5.02]		[2.29,3.90]		[2.11,3.58]	
Q2	1.74		2.09		1.84		1.74	
	[1.47,2.06]		[1.51,2.90]		[1.45,2.33]		[1.38,2.18]	
Q3	1.32		1.3		1.30*		1.57	
	[1.12,1.56]		[0.98,1.73]		[1.05,1.62]		[1.26,1.97]	
Hospitality Law*PIR Quartile								
Law*Q1 (lowest)	1.29		0.7		1.74		1.54	
	[1.00,1.66]		[0.47,1.03]		[1.16,2.61]		[1.05,2.27]	
Law*Q2	1.31	0.7647	0.81	0.0330	1.54	0.1415	1.56	0.3182
	[1.00,1.72]		[0.54,1.22]		[1.03,2.28]		[0.99,2.45]	
Law*Q3	1.11		1.06		1.11		1.05	
	[0.85,1.43]		[0.70,1.63]		[0.73,1.69]		[0.66,1.68]	

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous); p-values represent interactions on additive scale

Table SM 2.9 Multiplicative p-values associated with interaction terms in adjusted models[a,b]

	Full Sample	**Age Category 1**	**Age Category 2**	**Age Category 3**
Workplace Laws				
Law*Race/ethnicity	0.3275	0.3355	0.1388	0.0643
Law*Gender	0.0420	0.6420	**0.0050**	0.6730
Law*Education	0.3432	0.3413	0.0668	0.5111
Law*PIR	0.0524	0.7446	**0.0109**	0.0666
Hospitality Laws				
Law*Race/ethnicity	0.0025	0.0456	**0.0297**	**0.0616**
Law*Gender	0.0010	0.238	**0.0110**	**0.0750**
Law*Education	0.1871	0.8503	**0.0068**	0.3761
Law*PIR	0.1149	0.1304	**0.0164**	0.0548

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

b) Bold p-values represent statistical significance after multiple testing adjustment

Table SM 2.10 Odds ratios of SHS exposure associated with smoke-free law coverage, stratified by living with a smoker[a]

Living with an indoor smoker	Full sample	Ages 25-39	Ages 40-59	Ages 60+
Workplace Law (≥50%)	0.65	0.30	0.59	1.07
	[0.32,1.35]	[0.10,0.92]	[0.15,2.36]	[0.26,4.44]
Hospitality Law (≥50%)	0.48	0.23	0.23	0.89
	[0.19,1.21]	[0.065,0.85]	[0.045,1.20]	[0.25,3.19]
Not living with an indoor smoker				
Workplace Law (≥50%)	0.90	0.82	0.94	0.91
	[0.77,1.05]	[0.67,1.02]	[0.78,1.14]	[0.71,1.17]
Hospitality Law (≥50%)	0.60	0.57	0.60	0.64
	[0.49,0.74]	[0.43,0.76]	[0.48,0.75]	[0.49,0.83]

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

Table SM 2.11 Change in geometric mean SHS exposure associated with smoke-free law coverage, stratified by living with a smoker[a,b]

Living with an indoor smoker	Full sample	Ages 25-39	Ages 40-59	Ages 60+
Workplace Law (≥50%)	0.86	0.68	0.92	0.9
	[0.62,1.20]	[0.40,1.18]	[0.56,1.54]	[0.52,1.55]
Hospitality Law (≥50%)	0.80	0.67	0.72	1.07
	[0.60,1.07]	[0.44,1.01]	[0.46,1.14]	[0.63,1.80]
Not living with an indoor smoker				
Workplace Law (≥50%)	0.96	0.94	0.96	0.98
	[0.92,1.01]	[0.87,1.02]	[0.92,1.00]	[0.93,1.04]
Hospitality Law (≥50%)	0.88	0.84	0.88	0.93
	[0.83,0.93]	[0.76,0.93]	[0.83,0.93]	[0.87,1.00]

a) Models estimated using linear regression with log-transformed outcome; serum cotinine values less than limit of detection (.05 ng/mL) replaced by $.05/\sqrt{2}$

b) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

Table SM 2.12 Odds ratios for SHS exposure associated with "any" and "comprehensive" smoke-free law coverage in the full sample and in age-stratified models[a,b]

Bivariate[a]	**Full Sample**	**Ages 25-39**	**Ages 40-59**	**Ages 60+**
Any Law (≥50%)	0.48 (0.40,0.56)	0.43 (0.34,0.54)	0.49 (0.40,0.59)	0.47 (0.39,0.58)
Comprehensive Law (≥50%)	0.83 (0.70,0.99)	0.82 (0.62,1.02)	0.78 (0.63,0.95)	0.87 (0.68,1.10)
Adjusted[b]				
Any Law (≥50%)	0.59 (0.49,0.72)	0.54 (0.41,0.71)	0.63 (0.51,0.78)	0.60 (0.46,0.78)
Comprehensive Law (≥50%)	0.94 (0.80,1.10)	0.86 (0.70,1.07)	0.93 (0.77,1.13)	1.01 (0.81,1.27)

a) Bivariate models control for survey wave (continuous variable)

b) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

Table SM 2.13 Additive p-values associated with interaction terms in adjusted models for "any" and "comprehensive" law exposures[a,b]

	Full Sample	Age Category 1	Age Category 2	Age Category 3
Any Law				
Law*Race/ethnicity	0.0653	0.1474	0.2481	0.178
Law*Gender	**<0.0001**	0.1309	**0.0041**	**0.0093**
Law*Education	0.3931	0.1458	0.4483	0.8316
Law*PIR	0.8871	0.0345	0.2715	0.2647
Comprehensive Law				
Law*Race/ethnicity	0.1762	0.5052	0.0638	0.0231
Law*Gender	0.0144	0.8945	**0.0007**	0.7527
Law*Education	0.3802	0.5114	**0.0027**	0.2323
Law*PIR	0.0805	0.4755	**0.0015**	0.1445

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

b) Bold p-values represent statistical significance after multiple testing adjustment

Table SM 2.14 Multiplicative p-values associated with interaction terms in adjusted models for "any" and "comprehensive" law exposures[a,b]

	Full Sample	Age Category 1	Age Category 2	Age Category 3
Any Law				
Law*Race/ethnicity	**0.0070**	0.0367	0.2493	0.0448
Law*Gender	**0.0010**	0.3220	0.0210	0.0480
Law*Education	0.1591	0.5586	0.1250	0.1869
Law*PIR	0.1517	0.1400	0.0674	0.0184
Comprehensive Law				
Law*Race/ethnicity	0.1657	0.4664	0.0631	0.0159
Law*Gender	0.0190	0.812	**0.0010**	0.7350
Law*Education	0.2126	0.5685	**0.0008**	0.2222
Law*PIR	0.0469	0.5382	**0.0008**	0.1405

a) Adjusted models control for individual-level covariates (age, race/ethnicity, education, gender), state-level covariates (average cost per pack of cigarettes, spending per capita on tobacco control initiatives, percent unemployed, percent with at least a Bachelor's degree, percent below the federal poverty line, percent Black, and percent Hispanic), and survey wave (continuous)

b) Bold p-values represent statistical significance after multiple testing adjustment

Figure SM 2.1 Predicted change in the probability of SHS exposure associated with ≥ 50% county-level workplace law coverage versus < 50 % coverage, at wave 1 and wave 8, among the full sample (1), ages 25-39 (2), ages 40-59 (3), and 60+ (4)

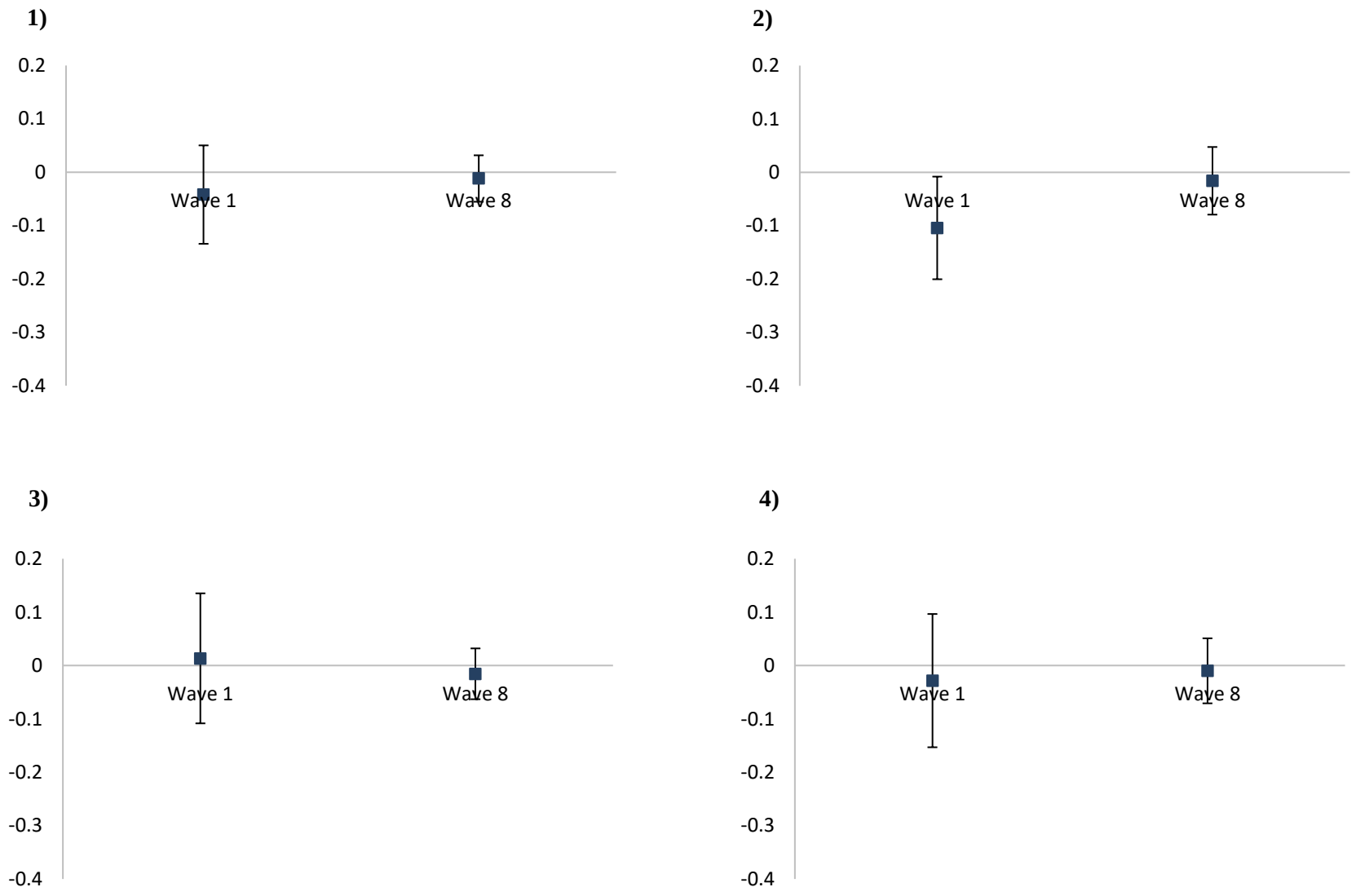
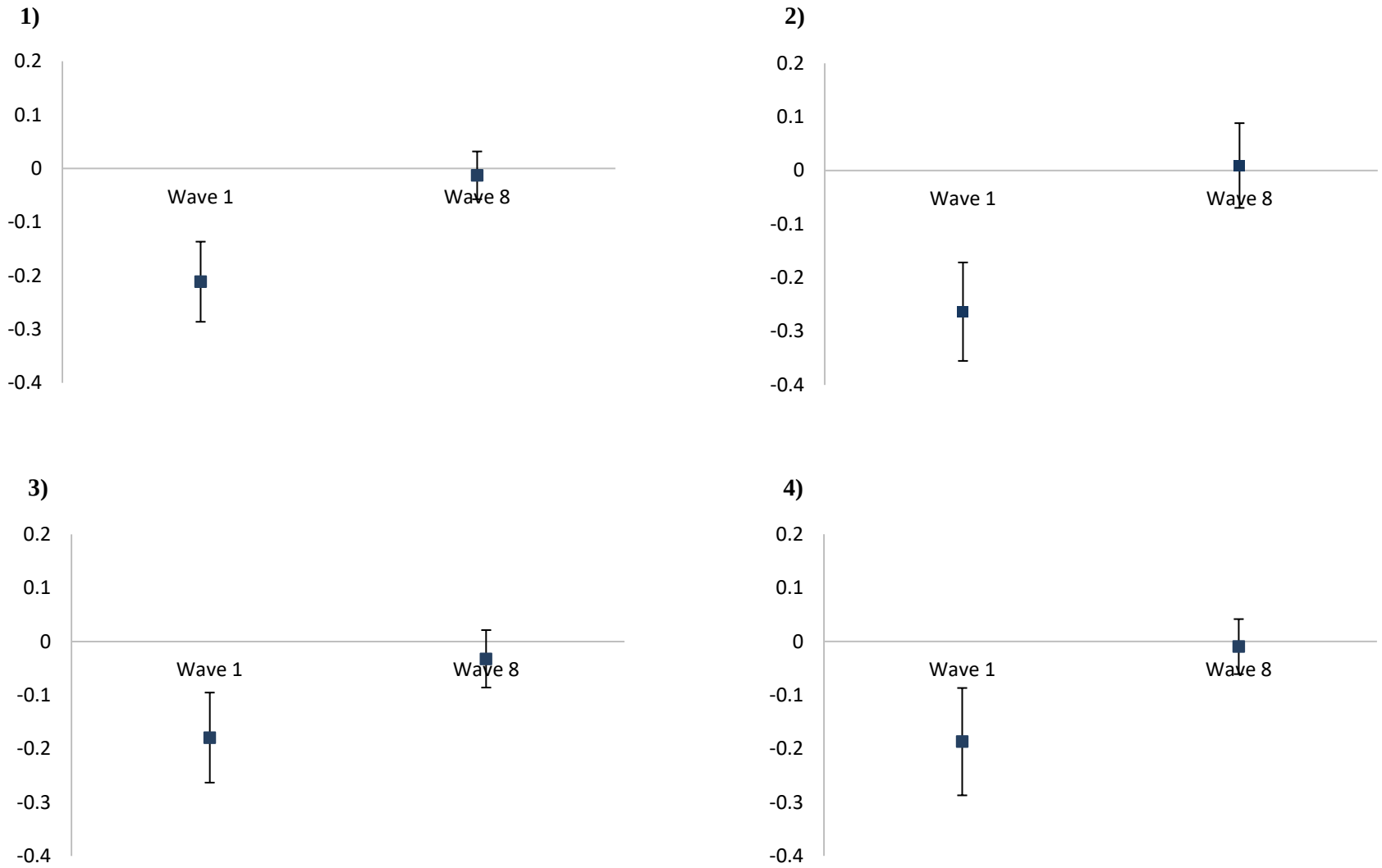


Figure SM 2.2 Predicted change in the probability of SHS exposure associated with ≥ 50% county-level hospitality law coverage versus < 50 % coverage, at wave 1 and wave 8, among the full sample (1), ages 25-39 (2), ages 40-59 (3), and 60+ (4)



CHAPTER 3. Disparities in Exposure and Mortality Attributable to Secondhand Smoke: A Simulation Study

Introduction

The prevalence of secondhand smoke (SHS) exposure among nonsmokers in the United States (U.S.) varies widely by race/ethnicity and sex.[1–3] In 2013-2014, 50.3% of Black nonsmokers 3 years of age and older were exposed to SHS, compared to 21.4% of White nonsmokers, and 20.0% of Mexican Americans.[1] While differences between males and females have narrowed over time, males remain more likely to experience higher levels of SHS exposure compared to females.[1] Heterogeneous patterns of SHS exposure translate into disparities in health outcomes, as SHS exposure has been causally linked to numerous health conditions, including cardiovascular disease and lung cancer.[4]

Relatively few estimates of SHS-attributable morbidity and mortality have focused on health disparities, but those that have suggest that the burden of disease is particularly concentrated among non-White individuals. For example, Max et al. (2012) found that the burden of lost productivity (lifetime earnings) due to SHS exposure in 2006 was particularly high among Black and Hispanic adults, compared to White adults. While these findings provide a snapshot of SHS exposure and downstream health outcomes, there has been little research to date that dynamically assesses how changing patterns of SHS exposure over time, particularly in recent years, impact attributable mortality and disparities. Moreover, we have limited insight into how persistent disparities in SHS exposure and related health outcomes may be impacted by a

changing landscape of tobacco control interventions, including policies focused on reducing smoking prevalence and/or banning smoking from public environments.

In this context, simulation modeling can be a useful tool for producing dynamic estimates of attributable burdens and projecting the potential impact of policy scenarios. Simulation modeling has been widely adopted in tobacco control research to explore the potential impact of counterfactual scenarios on smoking[5–8] and SHS exposure.[9–11] However, only a small number of models address tobacco-related health disparities. For example, the *SimSmoke* model has been utilized to examine the potential impacts of tobacco control polices on smoking disparities by income quantiles.[12] Racial/ethnic disparities have also been addressed in models of menthol use,[13] given the disproportionate burden of menthol smoking concentrated in Black communities.[14] We are not aware of simulation modeling studies that have explicitly been used to examine disparities in SHS exposure.

In this study, we employed a simulation modeling approach to: (a) quantify the burden of cause-specific mortality due to SHS exposure in recent decades, (b) project trends in SHS exposure through 2040, and (c) explore the potential impact of intervention scenarios on SHS exposure distributions for White, Black, Hispanic, and other non-Hispanic (hereafter, Other) males and females. We examine the relative impact of interventions of comparable magnitude that focus on smoking cessation versus interventions that weaken the association between smoking prevalence and SHS exposure prevalence. Finally, we discuss the implications of our findings for future tobacco control interventions aimed at reducing SHS exposure and improving health equity.

Methods

Modeling framework. A conceptual diagram of the model is provided in Figure 3.1. At each time point (year) a cohort of adults enters the model as current smokers, former smokers, or never smokers. A series of difference equations then guide transitions between model compartments. Table 3.1 includes a description of all model parameters, along with data sources. Current smokers could transition to a former smoker compartment by quitting smoking. Current, former, and never smokers could be removed from the model each year according to compartment-specific probabilities of death. We further separated out nonsmokers (never and former smokers) to model SHS exposure prevalence. Nonsmokers could be unexposed or exposed to SHS, and we assumed that the likelihood of exposure among nonsmokers was a function of the overall smoking prevalence in the population (see equations 3.8 -3.10). We fit separate models, with separate parameters (as indicated in Table 3.1), for White males, White females, Black males, Black females, Hispanic males, Hispanic females, Other males, and Other females. We simulated the general population, current smokers, former smokers, and never smokers, according to the following set of equations (3.1-3.5):

$$(3.1) \quad \mathrm{GP}_{a,t} = \mathrm{GP}_{a-1,t-1} * \left(1 - \mathrm{p(dGP)}_{a-1,t-1}\right)$$

$$(3.2) \quad \mathrm{CS}_{a,t} = \mathrm{CS}_{a-1,t-1} * (1 - \mathrm{p(dCS)}_{a-1,t-1} - \mathrm{p(qCS)}_{a-1})$$

$$(3.3) \quad \mathrm{FS}_{a,t} = \mathrm{FS}_{a-1,t-1} * \left(1 - \mathrm{p(dFS)}_{a-1,t-1}\right) + \mathrm{CS}_{a-1,t-1} * \mathrm{p(cCS)}_{a-1}$$

$$(3.4) \quad \mathrm{NS}_{a,t} = \mathrm{GP}_{a,t} - \mathrm{CS}_{a,t} - \mathrm{FS}_{a,t}$$

$$(3.5) \quad \mathrm{p(CS)}_{t} = \frac{\sum_{a=18}^{84} \mathrm{CS}_{t}}{\sum_{a=18}^{84} \mathrm{GP}_{t}}$$

where $\mathrm{GP}_{a,t}$ represents the general population, $\mathrm{CS}_{a,t}$ represents the current smoker population, $\mathrm{FS}_{a,t}$ represents former smokers, and $\mathrm{NS}_{a,t}$ represents never smokers, for each age and year. Compartment-specific death probabilities are represented by $\mathrm{p(dGP)}$, $\mathrm{p(dCS)}$, and $\mathrm{p(dFS)}$.

$p(qCS)$ represents the probability of a current smoker quitting smoking and moving into the former smoker compartment. The prevalence of smoking in each year, $p(CS)_t$, can be expressed as a fraction of current smokers over the general population. We used the equations to fill out tables containing the total population (GP), as well as the number of never (NS), current (CS) and former smoking (FS) individuals for each age (a) and year (t). The model was developed in Python v. 3.7.1.

Calibrating the smoking prevalence model. We calibrated the model of smoking prevalence to observed data by race/ethnicity and sex. The age-specific number of individuals in the year 2000, as well as the number of 18-year olds in each year between 2000 and 2018, were used as starting conditions for the modeled table of the general population. Population data by age, race/ethnicity, and sex was derived from U.S. Census Bureau intercensal estimates of the resident population,[15] as well as data downloaded from the American FactFinder database.[16] The starting condition for the table of current smokers consisted of the starting data for the general population multiplied by the age-specific observed smoking prevalence in 2000, and the year-specific smoking prevalence among 18-year olds between 2000 and 2018. We generated estimates of observed smoking prevalence using 2000-2018 data from the National Health Interview Survey (NHIS).[17] We estimated a logit regression of current smoking status (binary) on age, age-squared, and year of the survey, modeled using linear splines with a knot in 2010. We chose to place the knot in 2010 based on visual inspection of the observed data. We then predicted the probability of current smoking for each individual in the dataset and averaged these probabilities across age and year. We performed a similar process to generate starting probabilities for the former smoker table, based on observed probabilities in the NHIS dataset.

All empirical analyses using NHIS data were performed in Stata v. 15 and incorporated survey design parameters, as well as population weights.

The model also required an age-year table of mortality probabilities by race/ethnicity and sex. We derived these probabilities from the CDC WONDER Underlying Cause of Death database.[18] Mortality data is released in 10-year age groups; we estimated age-specific mortality by assuming that each mortality probability represented the mid-point of the corresponding age group and linearly interpolating between these points. While the CDC WONDER database provides mortality data for the general population, the database does not provide mortality probability estimates for current or former smokers. Instead, we approximated probabilities of death according to smoking status by deconstructing the probability of death in the general population into an average weighted by the prevalence of current smokers, former smokers, and never smokers in the U.S. population. We then re-arranged this equation to solve for the probability of death among never smokers as shown in equations 3.6 and 3.7.

$$(3.6) \quad p(dGP)_{a,t} =$$

$$p(dNS)_{a,t} * p(NS)_{a,t} + p(dNS)_{a,t} * RR_{FS} * p(FS)_{a,t} + p(dNS)_{a,t} * RR_{CS} * p(CS)_{a,t}$$

$$(3.7) \quad p(dNS)_{a,t} = \frac{p(dGP)_{a,t}}{p(NS)_{a,t} + RR_{FS} * p(FS)_{a,t} + RR_{CS} * p(CS)_{a,t}}$$

RR_{FS} and RR_{CS} represent the unadjusted relative risks of mortality associated with being a former smoker and current smoker, respectively. We derived these relative risks from Thun et al. (2013),[19] and applied them to the population 35 years and older, as we assumed that smoking-attributable mortality would be concentrated among middle- and older-aged adults. After solving for $p(dNS)_{a,t}$, we then calculated the probability of death among current smokers as $p(dNS)_{a,t}$*RR_{CS} and the probability of death among former smokers as $p(dNS)_{a,t}$*RR_{FS}. These

mortality probabilities were dynamically re-calculated each year by deconstructing the probability of death in the general population (by race/ethnicity and sex) as described above.

Following the approach by Mendez et al,[20] we estimated cessation probabilities by comparing the model output to a table of observed smoking probabilities from NHIS data (2000-2018). We used the scipy.optimize.minimize function in Python to choose cessation parameters that minimized a sum-of-squares goodness of fit function, comparing the table of observed smoking probabilities to modeled smoking probabilities. We numerically solved for the optimal values using the "Nelder-Mead" simplex algorithm.[21] We estimated six cessation probabilities for each population group (i.e., White, Black, Hispanic, and Other males and females), corresponding to the three age strata across two separate time periods (2000-2010 and 2011-2018). Our optimization algorithm did not constrain probabilities to be positive; therefore, negative cessation probabilities reflected net initiation.

Simulating SHS exposure. In order to simulate the relationship between smoking prevalence and the likelihood of SHS exposure, we first empirically estimated the relationship between overall smoking prevalence and SHS exposure by race/ethnicity and sex using data on smoking prevalence from NHIS and data on SHS exposure from the National Health and Nutrition Examination Survey (NHANES), 1999-2016.[22] The NHANES survey uses a multi-stage sampling design and oversamples racial/ethnic minorities.[22] Data on SHS exposure was derived from laboratory measures of serum cotinine, a metabolite of nicotine and an indicator of recent exposure to tobacco smoke.[23] First, we defined a sample of nonsmokers based on previously established serum cotinine cutoff points to distinguish smokers from nonsmokers, using cut-off points specific to racial/ethnic groups where possible (6 ng/mL for White males and females; 5 ng/mL for Black males and females; and 1 ng/mL for Mexican-American males and females).[24]

We applied the cut point for Mexican-Americans to all Hispanics in our study. We used the recommended cut point for U.S. adults as a whole for the Other racial/ethnic group (3 ng/mL).[24] From this biomarker-defined population of nonsmokers, we then excluded any individuals who reported having used a product containing nicotine within the past five days. Nonsmokers were considered exposed to SHS if their serum cotinine levels were above the detection limit (>.05 ng/mL). Within the population of nonsmokers, we estimated a log-binomial regression model of binary SHS exposure as a function of overall year-specific smoking prevalence. We estimated separate models for each age category (18-34, 34-64, 65+), by race/ethnicity and sex. These three age categories were chosen based on the age-stratification of relative risks for cardiovascular disease associated with current and former smoking published by the U.S. Surgeon General,[25] which were used to estimate attributable deaths due to smoking and SHS exposure. All models were estimated in Stata and accounted for complex survey design and incorporated survey weights.

Using the estimated intercept and coefficient from the regression model, we modeled SHS exposure according to equations 3.8-3.10, separately by race/ethnicity and sex.

$$\text{NonS}_{a,t} = \text{GP}_{a,t} - \text{CS}_{a,t} \tag{3.8}$$

$$\text{SHS}_{a,t} = \text{NonS}_{a,t} * e^{\beta_{0a} + \beta_{1a} * p(\text{CS})_t} \tag{3.9}$$

$$p(\text{SHS})_t = \frac{\sum_{a=18}^{84} \text{SHS}_t}{\sum_{a=18}^{84} \text{NonS}_t} \tag{3.10}$$

$\text{NonS}_{a,t}$ represents the total population of nonsmokers (inclusive of never and former smokers). β_0 and β_1 represent age-stratified parameters from the log-binomial regression models (the intercept and coefficient associated with smoking prevalence, respectively). In the above equation, the β_1 coefficient relates smoking prevalence to the probability of SHS exposure. We theorized that the value of β_1 could be impacted by policy interventions that seek to weaken the

relationship between smoking prevalence and SHS exposure, i.e., by limiting contact between nonsmokers and active smokers. The prevalence of SHS exposure at any time point was the number of exposed nonsmokers over the total nonsmoking population. To assess model fit, we visually compared the modeled SHS exposure prevalence to the observed SHS exposure prevalence in each year.

Calculating deaths attributable to SHS exposure. We used the model to calculate deaths attributable to SHS exposure in each year for two conditions with a strong evidence base supporting a causal relationship between SHS exposure and mortality: ischemic heart disease and lung cancer.[26] We gathered data on total deaths from each cause from CDC WONDER based on ICD-10 codes for the relevant conditions. We estimated attributable deaths for each disease following a process outlined in prior literature.[26–28] First, we calculated the smoking-attributable fraction and the total attributable deaths among smokers according to equation 3.11.

$$\text{SmAF}[c]_{a,t} = \frac{p(CS)_{a,t} * RR[c]_{CS} - 1}{p(NS)_{a,t} + p(FS)_{a,t} * RR[c]_{FS} + p(CS)_{a,t} * RR[c]_{CS}} \quad (3.11)$$

$\text{SmAF}[c]_{a,t}$ is the smoking-attributable fraction for each cause; $RR[c]_{CS}$ and $RR[c]_{FS}$ represent the cause-specific relative risks associated with current smoking and former smoking, respectively. We derived relative risks for IHD and lung cancer from the U.S. Surgeon General's estimates of smoking-attributable morbidity, mortality, and economic costs (SAMMEC).[25] After subtracting the smoking-attributable deaths from the total deaths, we then weighted the remainder by the proportion of nonsmokers in the population in order to calculate the number of deaths among nonsmokers, as shown in equation 3.12.

$$\text{deaths}[c]_{NonS,a,t} = \left(\text{deaths}[c]_{a,t} - \text{deaths}[c]_{a,t} * \text{SmAF}[c]_{a,t}\right) * (1 - p(CS)_{a,t}) \quad (3.12)$$

Once we had calculated the total number of deaths among nonsmokers, we then calculated the fraction of these deaths attributable to SHS exposure, using the formula in equation 3.13, which is also equivalent to the formula in equation 3.14.

$$\text{ShAF}[c]_{a,t} = \frac{(1 - p(\text{SHS})_{a,t}) + p(\text{SHS})_{a,t} * \text{RR}[c]_{\text{SHS}} - 1}{(1 - p(\text{SHS})_{a,t}) + p(\text{SHS})_{a,t} * \text{RR}[c]_{\text{SHS}}} \quad (3.13)$$

$$\text{ShAF}[c]_{a,t} = \frac{p(\text{SHS})_{a,t} * (\text{RR}[c]_{\text{SHS}} - 1)}{p(\text{SHS})_{a,t} * (\text{RR}[c]_{\text{SHS}} - 1) + 1} \quad (3.14)$$

$\text{RR}[c]_{\text{SHS}}$ is the relative risk of cause-specific death associated with SHS exposure among exposed nonsmokers compared unexposed nonsmokers. Following on examples in previous literature, we derived relative risks associated with SHS exposure for IHD from Whincup et al. (2004)[29] and for lung cancer from a report of the California Environmental Protection Agency (2005).[30] In each year, we summed the total number of attributable deaths across all ages to generate the mortality burden associated with SHS exposure for each population group (by race/ethnicity and sex) across the entire United States.

Simulating smoking prevalence and SHS exposure through 2040. Once the smoking prevalence model was calibrated to NHIS data through 2018, we simulated the smoking prevalence and SHS exposure prevalence through 2040. To ensure that all-cause and cause-specific mortality probabilities reflected changes in the prevalence of smoking in the U.S. population, we dynamically re-calculated all-cause and cause-specific death probabilities for each year, based on the formula provided in equation 3.6. In practice, this ensured that mortality probabilities decreased over time as the overall proportion of smokers decreased over time. For years after 2018 (the last year of observed mortality data), we assumed that the death probability among never-smokers remained at 2018 values.

Also in the years following 2018, we adjusted the starting smoking prevalence among 18-year olds to incorporate information on recent trends in smoking initiation in the United States. We did this by estimating the average annual percent change (APC) in smoking prevalence among 18-year olds between 2010 and 2018 and decreasing the smoking prevalence by the APC in future years, de-trending every five years. This approach is based on prior literature describing methods for modeling future cancer incidence trends.[31,32] The APCs were specific to each group by race/ethnicity and sex and were multiplied by .75 in 2023, .5 in 2028, and .25 in 2033, before assuming a constant smoking prevalence among 18-year olds for 2038-2040.

Intervention scenarios. We evaluated three intervention scenarios to assess their potential impact on SHS exposure and disparities in exposure by race/ethnicity and sex. We compared the potential impacts of an intervention on cessation versus an intervention on the β_1 parameter in equation 3.9 representing the association in log-binomial models between overall smoking prevalence and SHS exposure. We projected the prevalence of SHS exposure corresponding to a 20% increase in the probability of cessation versus a 20% decrease in the β_1 parameter. We also examined the impact of a combined intervention consisting of both a 20% increase in cessation and a 20% decrease in β_1. We applied the 20% increase in cessation to the absolute value of each cessation probability, as some optimized cessation probabilities were less than zero (reflecting net initiation). We implemented each intervention in 2019.

While the interventions on smoking cessation and the β_1 parameter explored in this study are hypothetical, they also reflect potential tobacco control policies that are frequently implemented. For example, tobacco taxation has been found to be associated with higher levels of smoking cessation.[33,34] We chose the magnitude of the cessation intervention to be in line with prior work examining the price elasticity of cessation, which is estimated to be between 0.3 and

0.5.[34] Interventions on the parameter that related smoking prevalence to the probability of SHS exposure (β_1) are more aligned with interventions that limit interactions between nonsmokers and active smokers, including smoke-free air laws, which restrict smoking in public spaces (including workplaces, restaurants, and bars).[35] In calibrating this parameter, we were not aware of other studies that have attempted to examine how the specific relationship between smoking prevalence and SHS exposure may be impacted by the introduction of a policy. However, we chose an intervention magnitude that produced declines in SHS exposure that were in line with prior estimates of the association between smoke-free laws and SHS exposure. For example, Pickett et al. (2006) found that adults in counties with extensive smoke-free law coverage were ~73% less likely to be exposed to SHS compared to adults in counties with no smoke-free law coverage, whereas adults in counties with limited smoke-free law coverage were ~24% less likely to be exposed.[36] We compared the output of our intervention scenario to these estimates to ensure that percent reductions in SHS exposure fell within a range of feasibility.

Assessing impacts of interventions on SHS exposure. We plotted the effects of different intervention scenarios to explore the impact of each scenario on projected SHS exposure disparities. We also compared model results in 2016 (the last year of observed SHS exposure data) to projected SHS exposure in 2040 under all scenarios. We chose to use modeled prevalence instead of observed prevalence in 2016 for these comparisons, as the model provided smoothed estimates of SHS exposure that were not subject to random variability associated with annual NHANES estimates. We assessed changes in SHS exposure by examining the percent change in SHS exposure between 2016 and 2040, as well as the absolute change in percentage points.

Modeling assumptions. We assumed that cessation probabilities and the relationship between smoking prevalence and SHS exposure was constant within age and time ranges. We also assumed constant cessation probabilities in future years (except under the cessation intervention scenario), equal to the age-specific cessation probabilities in 2018. Similar to some prior simulation studies,[9] we assumed that the majority of smokers would initiate smoking prior to the age of 18, so we did not explicitly model initiation. However, we did not constrain cessation parameters to be positive. Finally, the modeling strategy assumed that the relationship between prevalence and SHS exposure could be estimated empirically, and that SHS exposure was a function of overall population smoking prevalence, not of prevalence specific to racial/ethnic or sex subgroups.

Results

Plots of the model output versus observed data are included in Supplementary Figure 3.1 (smoking prevalence) and Supplementary Figure 3.2 (SHS exposure prevalence among nonsmokers) for the eight race/ethnicity and sex subgroups. Optimized cessation parameters for 2000-2010 and for 2011-2018, by age group, sex, and race/ethnicity are in Supplementary Table 3.1. Between 2000 and 2010, Black males and females, White males and females, and Hispanic females experienced negative cessation probabilities in the youngest age group, which reflects net initiation in these groups. Supplementary Table 3.2 contains estimated all-cause mortality probabilities for the general population for 2015-2040, taking into account the projected composition of current smokers, former smokers, and never smokers in the U.S. population in future years.

Figure 3.2 includes plots of attributable deaths due to IHD and lung cancer between 2000 and 2016 (the last year of observed data) by race/ethnicity. Total attributable IHD deaths

decreased across the time period, from 30,782 deaths in 2000 to 8,469 deaths in 2016. Likewise, SHS-attributable lung cancer deaths decreased from 7,348 to 2,888 over the same period. The majority of deaths occurred among White nonsmokers. Annual deaths by sex and race/ethnicity for each condition are included in Supplementary Table 3.3.

We then calculated a ratio measure to more accurately compare the burden of SHS-attributable deaths across each racial/ethnic group. In the numerator of the ratio, we calculated the proportion of all SHS-attributable deaths occurring within the racial/ethnic group. In the denominator, we calculated the proportion of all nonsmokers within the group. This measure essentially scaled SHS-attributable deaths within each group by the size of their nonsmoker population. We plotted the ratios for each racial/ethnic group in each year in Figure 3.3. Ratios greater than 1 suggest that the group bears a disproportionate burden of SHS-attributable mortality, relative to the size of their nonsmoker population. For both IHD and lung cancer, White and Black adults appeared to experience particularly high levels of SHS-attributable mortality throughout the study period, however, the relative burden has been decreasing for White adults, whereas it has been increasing for Black adults.

Figures 3.4 and 3.5 include projections of smoking prevalence and SHS exposure from 2000 through 2040. Both smoking prevalence and SHS exposure decreased across the time period. Among males, smoking prevalence was highest among Black males, followed by White, Other, and Hispanic males. Among females, smoking prevalence was highest among White females and lowest among Hispanic females. The model suggests that differentials between groups will persist through 2040 under a baseline scenario, but that absolute differentials will narrow over time. With regard to SHS exposure, Black males and females experienced

considerably higher levels of SHS exposure compared to other racial/ethnic groups across the simulation period.

Figures 3.6 and 3.7 contains plots of projected SHS exposure under the baseline and intervention scenarios for males and females. These plots suggest that an intervention on the β_1 parameter that weakens the association between smoking prevalence and SHS exposure results in a more substantial decrease in SHS compared to an intervention of comparable magnitude focused on smoking cessation, although differences between the two interventions are modest. In Table 3.2, we included estimates of SHS exposure prevalence in 2016 and in 2040 under each scenario. We also computed the prevalence ratio and the prevalence difference, comparing each group's prevalence in 2040 with their modeled prevalence in 2016. Under all scenarios, relative (%) decreases were largest among Hispanic males and smallest among Black females. On the other hand, Black males and females were projected to experience the most substantial decreases on the absolute scale under all scenarios. Across all racial/ethnic groups, decreases were more substantial among males than females, on the both relative and absolute scales. Finally, comparing the range of SHS exposure prevalence across scenarios suggests that the combined intervention scenario yielded the smallest range in absolute terms (2.8%-16.1%), followed by the intervention weakening the association between smoking prevalence and SHS exposure (3.1%-16.7%), the cessation intervention (3.3%-17.2%), and the baseline scenario (3.6%-18.0%).

Discussion

In this study, we used simulation modeling as a framework for examining patterns of SHS-attributable health outcomes and potential future trends in SHS exposure disparities across population groups. We found that deaths from SHS exposure are decreasing for the U.S. population as a whole, but that Black populations continue to bear a disproportionate burden of

exposure and attributable mortality outcomes, compared to other racial/ethnic groups. Within each racial/ethnic group, we found that differences in SHS exposure between males and females have largely narrowed over time. Our simulations suggest that racial/ethnic disparities in SHS exposure are likely to persist into future decades; however, high-prevalence groups are likely to experience the most substantial absolute declines in prevalence, even under a baseline scenario. Finally, we find that interventions that weaken the association between smoking prevalence and SHS exposure are modestly more likely to reduce SHS exposure prevalence among all groups, compared to interventions that focus on smoking cessation alone.

Our observation that SHS exposure prevalence has dramatically declined in recent decades across all sociodemographic groups is in line with findings from surveillance studies.[1,2] Accordingly, we also find that SHS-attributable deaths from IHD and lung cancer have decreased substantially over time. Our estimates suggest that SHS exposure caused fewer than 12,000 deaths from IHD and lung cancer in 2016. This is well below commonly cited estimates of SHS-attributable mortality, which suggest that SHS exposure is a cause of over 41,000 deaths from heart disease and lung cancer in the U.S. annually.[37] These published estimates, however, are based on estimated SHS exposure prevalence in the early to mid-2000s.[26,38] Our estimations of SHS-attributable mortality may also be lower than previously published estimates because we account for the age distribution of SHS exposure. Younger adults are more likely to experience higher levels of SHS exposure, but are less likely to experience SHS-attributable mortality. One implication of our study is that commonly cited estimates of attributable mortality likely need to be updated to reflect substantial decreases in SHS exposure in recent years.

Similar to our study, prior research using single year data has suggested that Black adults face a disproportionate burden of years of productive life lost due to SHS exposure, compared to

other racial/ethnic groups.[26] This analysis adds to the existing literature by quantifying attributable deaths over nearly two decades, allowing us to examine dynamic changes in the share of the attributable burden across racial/ethnic groups over time. We also contextualize this calculation by scaling by the size of each group's nonsmoker population over time. Our results suggest that both White and Black populations face a high burden of mortality from SHS exposure given the size of the nonsmoker population within each of these groups. However, unlike for White adults, the mortality burden among Black adults relative to the number of Black nonsmokers is becoming increasingly disproportionate over time.

In projecting future trends in SHS exposure, we find that SHS exposure prevalence is likely to continue to decline in future years for all groups, even under the baseline scenario. However, our model also suggests that SHS exposure prevalence will remain above 15% for Black males and females by 2040 under the baseline scenario, while the prevalence estimates among all other racial/ethnic groups will be concentrated between 3.6% (Hispanic males) and 7.6% (Other females). These results suggest that targeted interventions are likely necessary to narrow differentials in SHS exposure across racial/ethnic groups.

Examining trends in SHS exposure prevalence highlights the importance of considering both the additive and multiplicative scales in assessing disparities across population groups.[39] Often, surveillance studies focus on percent change in SHS exposure, which may lead to the conclusion that declines in SHS exposure in recent years have not been as pronounced among high-prevalence groups as low-prevalence groups.[40,41] However, this conclusion is in part an artifact of examining relative, rather than absolute, changes in SHS exposure. In our projections, we find that disparities between racial/ethnic groups on the multiplicative scale do not improve, and may even be worsened over time. However, absolute differences in SHS exposure are likely

to narrow, even under the baseline scenario. As prior research has noted, quantifying differences on the absolute scale may be most relevant when assessing whether disparities have increased or decreased across population groups.[39]

The findings from this study suggest that interventions that weaken the relationship between smoking prevalence and SHS exposure are modestly more likely to reduce SHS exposure among nonsmokers than interventions of comparable magnitude that target smoking cessation alone. There are considerable opportunities in the United States to expand interventions that focus on limiting nonsmokers' exposure to environments with active smokers, whether legislatively or through the adoption of voluntary measures. As of January 2020, approximately 61.1% of the U.S. population was covered by comprehensive smoke-free laws covering all non-hospitality workplaces, restaurants, and bars.[42] Likewise, voluntary smoke-free home policies are becoming increasingly prevalent[43] and newly enacted rules requiring housing agencies to implement smoke-free policies in public housing units[44] may also limit involuntary exposure to cigarette smoke, particularly among low-income nonsmokers. The long-term effects of these interventions on SHS exposure disparities remains to be seen.

While we attempted to choose feasible intervention scenarios for this study, there is significant ambiguity surrounding the potential impact of tobacco control interventions. A 20% increase in cessation yields relatively muted impacts in our study, however, it may still represent a very optimistic scenario. Previously published estimates of the price elasticity of cessation would imply that an increase of this magnitude would require at least a 40% increase in price.[34] Outside of taxation, it is possible that achieving such an increase in cessation levels would require interventions not currently employed in the U.S., such as plain packaging laws or graphic health warning labels. For example, preliminary evidence of plain packaging and graphic health

warning labels from Australia has documented a substantial increase in Quitline calls, as well as quit attempts, following the introduction of these policies,[45–47] though evidence on long-term cessation effects is lacking. Furthermore, without examples of these policies in the U.S., it is not known whether they might have similar effects on U.S. smokers.

Likewise, the impact of the intervention targeting the association between smoking prevalence and SHS exposure may also be over-estimated. As noted previously, we calibrated this intervention by comparing the output from the model to published estimates of the relationship between smoke-free laws and reductions in biomarker-measured SHS exposure.[36] However, these published estimates were based on data collected between 1999 and 2002, and it is possible that the relationship between smoke-free policies and SHS exposure has changed over time, as is suggested in Chapter 2 of this dissertation.

Future studies should explore the sensitivity of SHS exposure models to varying the assumptions underlying the impact of policy scenarios, including the potential for differential policy impacts across sociodemographic groups. A growing body of literature has documented variability in the associations between smoke-free laws and health outcomes by age, sex, and poverty status.[48,49] In addition, a nationally representative study of SHS exposure among U.S. adults reported differential associations between smoke-free laws and SHS exposure for males and females.[36] Intervention scenarios may also differentially impact population subgroups by closing existing gaps in policy coverage. For example, previous studies suggest that the likelihood of smoke-free law coverage varies by race/ethnicity.[50] An intervention that assumed 100% smoke-free law coverage in the U.S., therefore, could disproportionately impact groups with lower levels of coverage. By assuming comparable effects across population groups, the

policy scenarios explored in this Chapter ignore potential heterogeneity in both exposure and response to policy interventions.

Strengths of this study include quantifying the attributable mortality burden of SHS exposure within sociodemographic groups across a long time frame, which helps elucidate how disparities in attributable outcomes have changed over time. With the exception of relative risk estimates for IHD and lung cancer, we utilized parameters that were specific to population subgroups, by race/ethnicity, sex, and age. This allowed detailed exploration into the impact of potential interventions on SHS exposure disparities. By comparing across multiple intervention scenarios, policy makers can gain insight into tobacco control strategies that are relevant to reducing both absolute levels and disparities in SHS exposure.

This study also has a number of limitations. Although intervention scenarios implicitly assumed a causal relationship between smoking prevalence and SHS exposure, our empirical estimates of this relationship may not capture a true causal parameter. Simplifying assumptions, including the assumption that several parameters are constant over time, may have weakened the validity of the model projections. We assumed that the relative risks associated with SHS exposure were the same for males and females. While this is in line with approaches in prior research,[26,38] several studies have also suggested potential effect modification of the SHS-CVD relationship by sex, with higher RRs seen in females compared to males.[51,52] There is also evidence of potential effect modification of the SHS-lung cancer relationship by sex, although confidence intervals between males and females overlap.[53,54] This study is limited in terms of the intervention scenarios examined. Moreover, we did not examine SES disparities, despite well-documented differences between SES groups in the likelihood of SHS exposure.[1] While SES disparities would be of interest, particularly given recently implemented rules regarding smoke-

free policies in public housing developments,[44] the CDC does not currently provide differential mortality estimates by SES, which limits our ability to incorporate SES into our model. However, other models of smoking behavior, including the *SimSmoke* Disparity model, have tackled this problem by applying adjustment factors to general population mortality rates by income quintile.[12] As with any simulation study focused on predicting future trends, results should be interpreted cautiously. The landscape of tobacco use and tobacco products in the United States is changing rapidly, which complicates efforts to project trends associated with combustible cigarettes.

Several limitations regarding the measure of SHS exposure also warrant attention. While cotinine is a reliable biomarker of SHS exposure, the presence of serum cotinine is only indicative of recent exposure to nicotine, with a plasma half-life of approximately 16 hours.[55] By modeling binary exposure to SHS, we failed to account for the relationship between levels of SHS exposure and mortality risk. This is an area for future research, as several studies have suggested a potential dose-response relationship between SHS exposure and health outcomes.[56,57] Finally, there is evidence of differential metabolism of nicotine across racial/ethnic groups, which is not addressed in this study. In particular, prior studies suggest that nicotine is metabolized more slowly among Black individuals, compared to White individuals.[55] Therefore some of the differential in exposure to SHS between racial/ethnic groups in this study may be explained by differential metabolism, versus differential exposure to SHS.

Conclusion

Reducing exposure to SHS remains a public health priority.[58] In this study, we used simulation modeling to explore disparities in smoking prevalence and SHS exposure, calculate deaths attributable to SHS, and predict future trends in SHS exposure under different

intervention scenarios to give insights into the potential consequences for health equity. We found that while SHS exposure disparities between racial/ethnic groups are decreasing on the additive scale, Black adults still bear a disproportionate burden of exposure and associated health outcomes. Our simulation model also suggests that focusing interventions on weakening the association between smoking prevalence and SHS exposure may result in greater decreases in SHS exposure compared to interventions focused on increasing smoking cessation alone, although differences between the scenarios were modest. The results from this study may help guide future decisions regarding the relative impact of potential interventions on health equity. This analysis also contributes to a small but growing body of literature that incorporates a disparities lens into simulation modeling in tobacco control. Future studies can build on the approach outlined here to examine other aspects of persistent sociodemographic and economic gradients in tobacco use and associated health outcomes.

References

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Tables

Table 3.1 Inputs and data sources for smoking and SHS models[a]

Parameter	Description	Subgroups	Data source
$GP_{a,t}$	Age/year-specific estimates of general population	Race/ethnicity and sex	U.S. Census Bureau annual estimates of the resident population
$CS_{a,t}$	Age-year specific number of current smokers	Race/ethnicity and sex	Estimated from National Health Interview Survey (NHIS) data
$FS_{a,t}$	Age/year-specific estimates of former-smoking population	Race/ethnicity and sex	Estimated from National Health Interview Survey (NHIS) data
$NS_{a,t}$	Age/year-specific estimates of never-smoking population	Race/ethnicity and sex	Estimated from model
$p(dGP)_{a,t}$	Age/year-specific probability of death (all-cause) for the general population	Race/ethnicity and sex	CDC WONDER database[25]
$p(dNS)_{a,t}$	Age/year-specific probability of death (all-cause) for the never-smoking population	Race/ethnicity and sex	Estimated empirically (equation 7)
RR_{CS}	Risk ratio for all-cause mortality comparing current smokers to never smokers	Sex	Thun et al. (2013)[19]
RR_{FS}	Risk ratio for all-cause mortality comparing former smokers to never smokers	Sex	Thun et al. (2013)[19]
$p(dCS)_{a,t}$	Age/year specific probability of death (all-cause) among current smokers	Race/ethnicity and sex	Derived as the p(dNS) multiplied by RR_{CS}
$p(dFS)_{a,t}$	Age/year specific probability of death (all-cause) among former smokers	Race/ethnicity and sex	Derived as the p(dNS) multiplied by RR_{FS}
$p(qCS)_{a}$	Probability of cessation for each age group (18-34, 35-64, >64)	Race/ethnicity and sex	Estimated from model
$NonS_{a,t}$	Age/year specific estimates of nonsmoker population	Race/ethnicity and sex	Estimated from model
$SHS_{a,t}$	Age/year specific estimates of nonsmoker population exposed to SHS	Race/ethnicity and sex	Estimated from model
β_{0a}	Intercept associated with regression of SHS exposure on smoking prevalence for each age group (18-34, 34-64, >64)	Race/ethnicity and sex	Empirically estimated from National Health and Nutrition Examination Survey (NHANES) data
β_{1a}	Coefficient associated with smoking prevalence from regression of SHS exposure on smoking prevalence for each age group (18-34, 34-64, >64)	Race/ethnicity and sex	Empirically estimated from NHANES data
$SmAF[c]_{a,t}$	Age/year specific smoking attributable fraction of cause-specific mortality	Race/ethnicity and sex	Estimated from model
$deaths[c]_{NonS,a,t}$	Age/year specific total number of cause-specific deaths among nonsmokers	Race/ethnicity and sex	Estimated from model
$deaths[c]_{a,t}$	Age/year-specific total cause-specific deaths	Race/ethnicity and sex	CDC WONDER database[19]
$ShAF[c]_{a,t}$	Age/year specific SHS attributable fraction of cause-specific mortality among nonsmokers	Race/ethnicity and sex	Estimated from model

$RR[c]_{CS}$	Risk ratio for cause-specific mortality comparing current smokers to never smokers	Sex	CDC (2014)[18]
$RR[c]_{FS}$	Risk ratio for cause-specific mortality comparing former smokers to never smokers	Sex	CDC (2014)[18]
$RR[c]_{SHS}$	Risk ratio for cause-specific mortality comparing SHS-exposed nonsmokers to unexposed nonsmokers	None	Whincup et al. (2004)[28], CA EPA (2005)[29]

a) Subscript legend : a=age, t=time

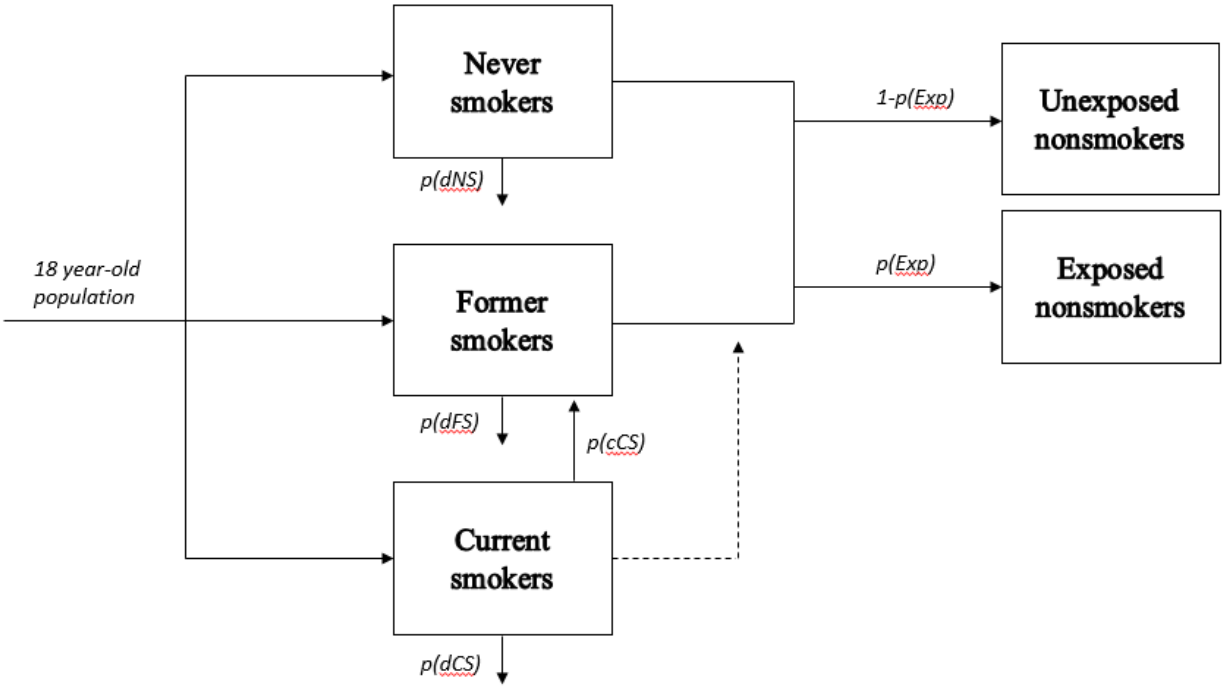
Table 3.2 Relative and absolute changes in SHS exposure prevalence associated with intervention scenarios[a]

		2016 (model)	2040 baseline	2040 cessation[b]	2040 β_1[c]	2040 combined[d]
White males	**Prevalence**	16.5%	5.1%	4.7%	4.5%	4.5%
	% change	Ref.	69.1%	71.5%	72.7%	72.7%
	Absolute change	Ref.	11.4pp	11.8pp	12.0pp	12.0pp
White females	**Prevalence**	15.6%	5.7%	5.3%	5.1%	4.8%
	% change	Ref.	63.5%	66.0%	67.3%	69.2%
	Absolute change	Ref.	9.9pp	10.3pp	10.5pp	10.8pp
Black males	**Prevalence**	36.4%	16.1%	15.2%	14.7%	14.0%
	% change	Ref.	55.8%	58.2%	59.6%	61.5%
	Absolute change	Ref.	20.3pp	21.2pp	21.7pp	22.4pp
Black females	**Prevalence**	36.1%	18.0%	17.2%	16.7%	16.1%
	% change	Ref.	50.1%	52.4%	53.7%	55.4%
	Absolute change	Ref.	18.1pp	18.9pp	19.4pp	20.0pp
Hispanic males	**Prevalence**	15.6%	3.6%	3.3%	3.1%	2.8%
	% change	Ref.	76.9%	78.8%	80.1%	82.1%
	Absolute change	Ref.	12.0pp	12.3pp	12.5pp	12.8pp
Hispanic females	**Prevalence**	13.8%	4.2%	3.9%	3.7%	3.5%
	% change	Ref.	69.6%	71.7%	73.2%	74.6%
	Absolute change	Ref.	9.6pp	9.9pp	10.1pp	10.3pp
Other males	**Prevalence**	20.1%	6.2%	5.8%	5.6%	5.2%
	% change	Ref.	69.2%	71.1%	72.1%	74.1%
	Absolute change	Ref.	13.9pp	14.3pp	14.5pp	14.9pp
Other females	**Prevalence**	18.3%	7.6%	7.1%	6.8%	6.6%
	% change	Ref.	58.5%	61.2%	62.8%	63.9%
	Absolute change	Ref.	10.7pp	11.2pp	11.5pp	11.7pp

a) Percent and absolute changes calculated relative to 2016 prevalence
b) Intervention represents a 20% increase in smoking cessation
c) Intervention represents a 20% decrease in parameter (β_1) relating smoking prevalence to SHS exposure
d) Intervention represents a combination of "cessation" and "β_1" interventions

Figures

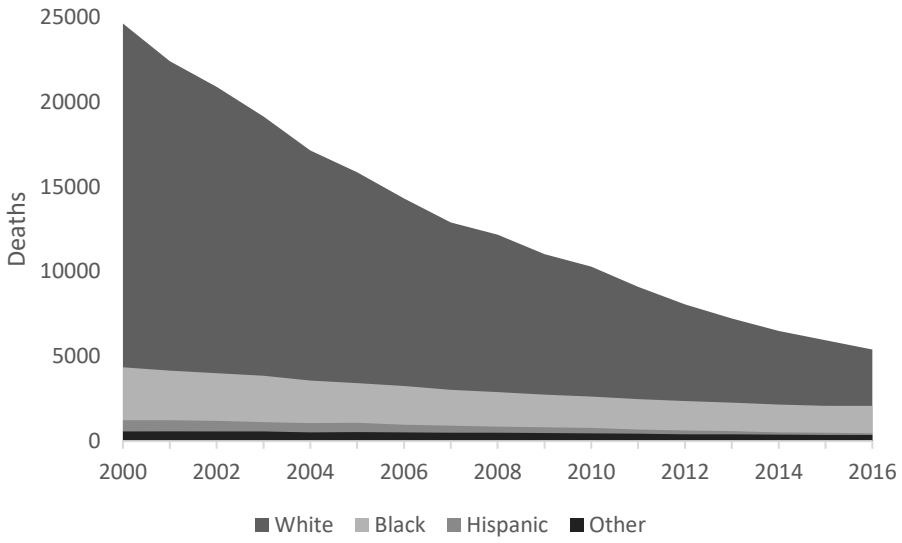
Figure 3.1 Conceptual diagram of simulation model[a]



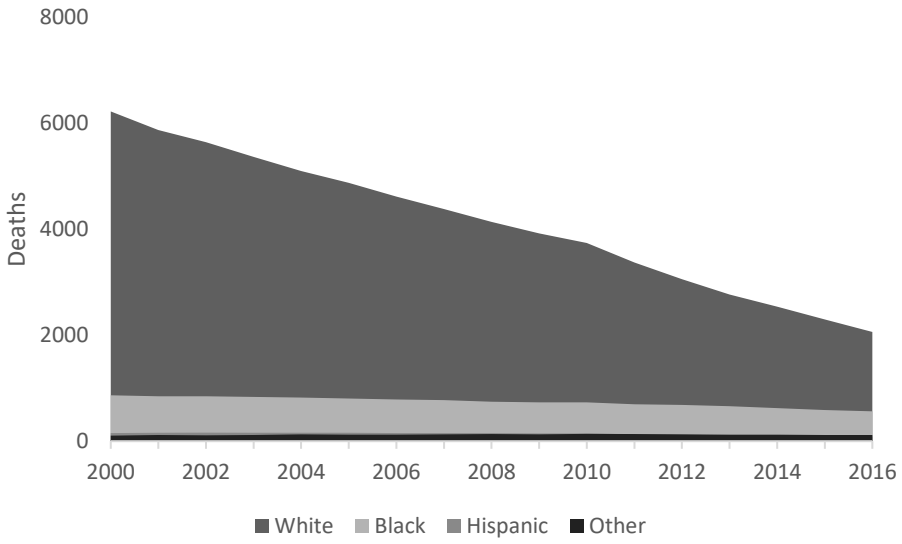
a) Compartments and parameters are age-year specific

Figure 3.2 Total IHD deaths (1) and lung cancer deaths (2) attributable to SHS exposure by race/ethnicity, 2002-2016[a]

1)



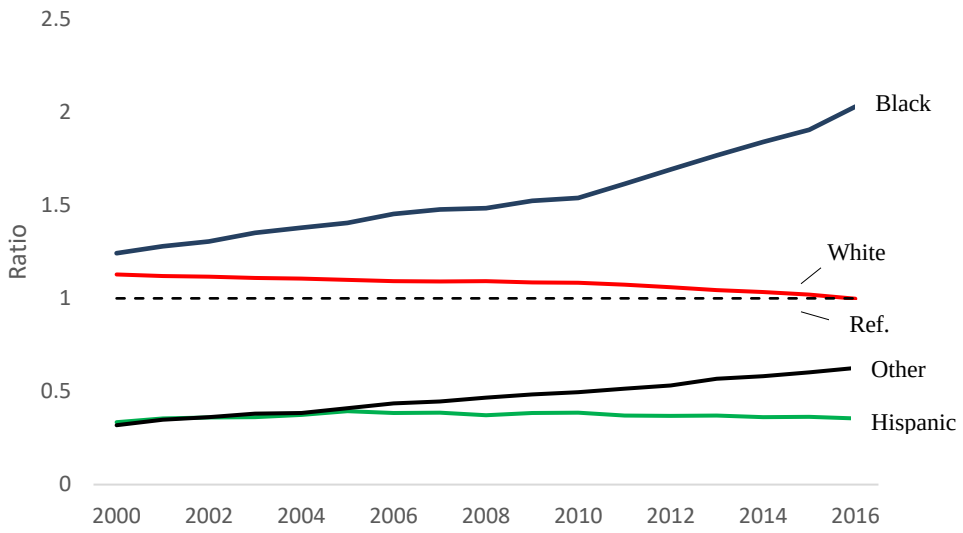
2)



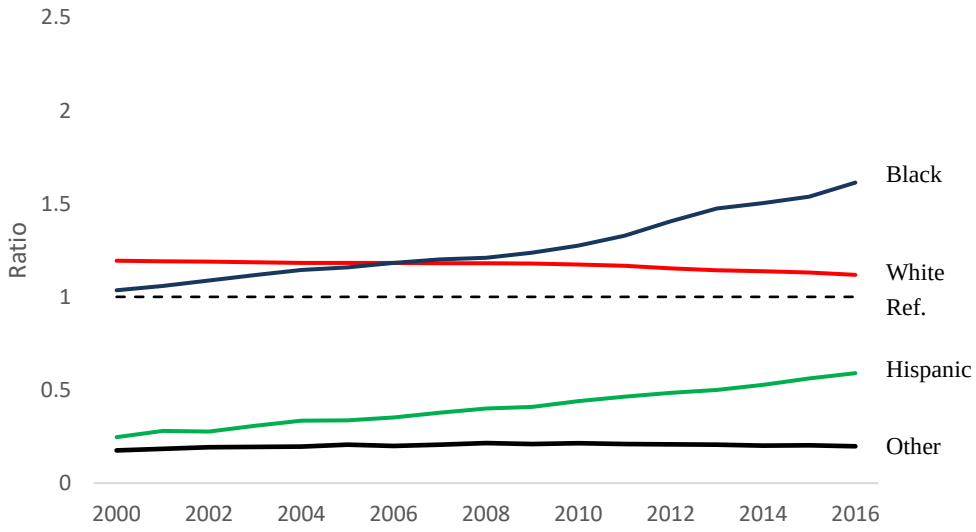
a) Y-axes scaled differently to highlight trends

Figure 3.3 Ratio representing proportion of SHS attributable deaths relative to proportion of nonsmoker population for IHD (1) and lung cancer (2), by race/ethnicity[a]

1)



2)

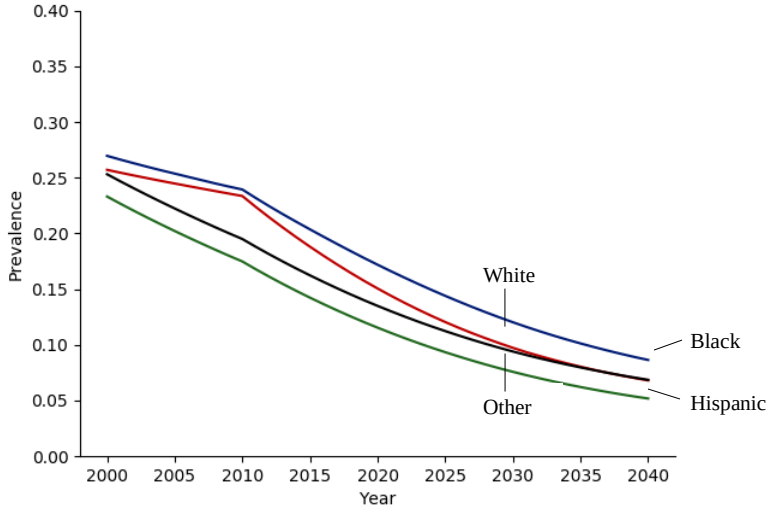


a) Plots represent (proportion of deaths)/(proportion of nonsmoker population) for each groups, e.g., for White adults:

$$Ratio = \frac{\frac{attributable\ deaths\ among\ Whites}{total\ attributable\ deaths}}{\frac{White\ nonsmokers}{total\ nonsmokers}}$$

Figure 3.4 Simulated smoking prevalence for males (1) and females (2), stratified by race/ethnicity, 2000-2040

1)



2)

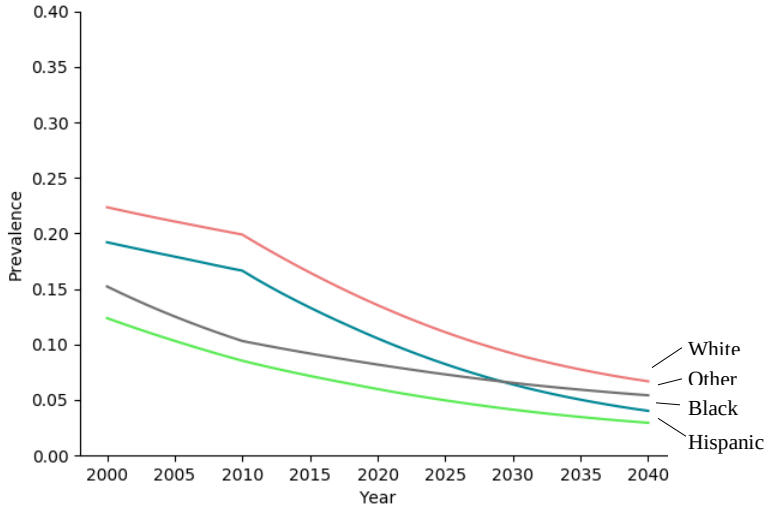
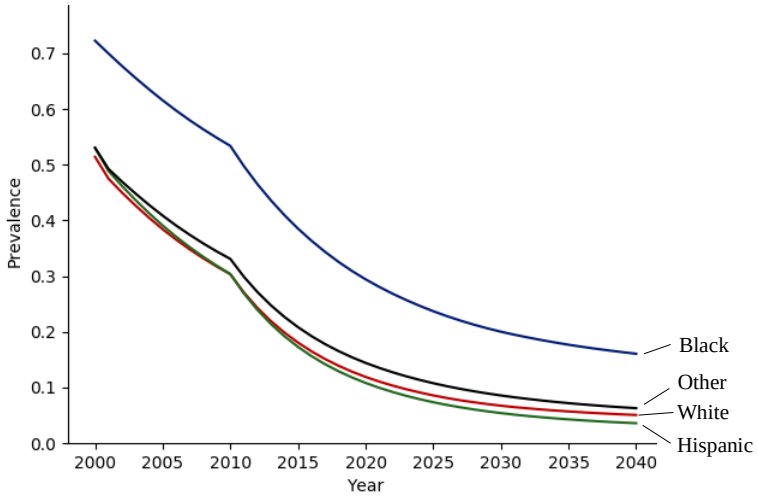


Figure 3.5 Simulated SHS prevalence for males (1) and females (2), stratified by race/ethnicity, 2000-2040

1)



2)

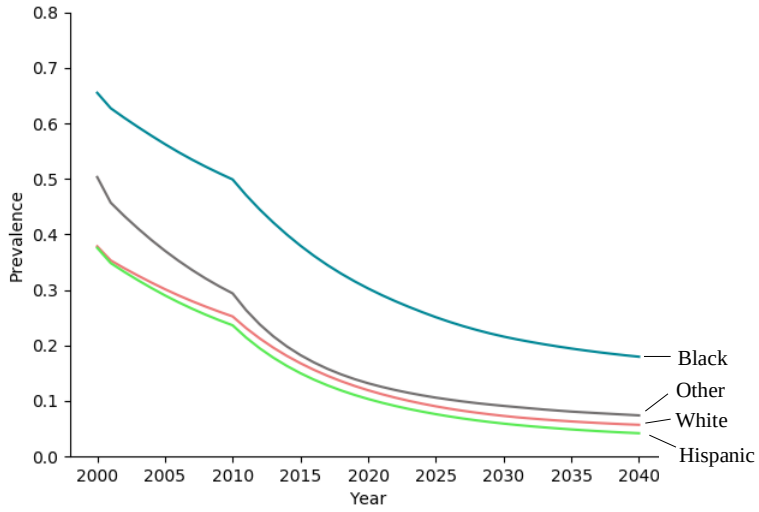
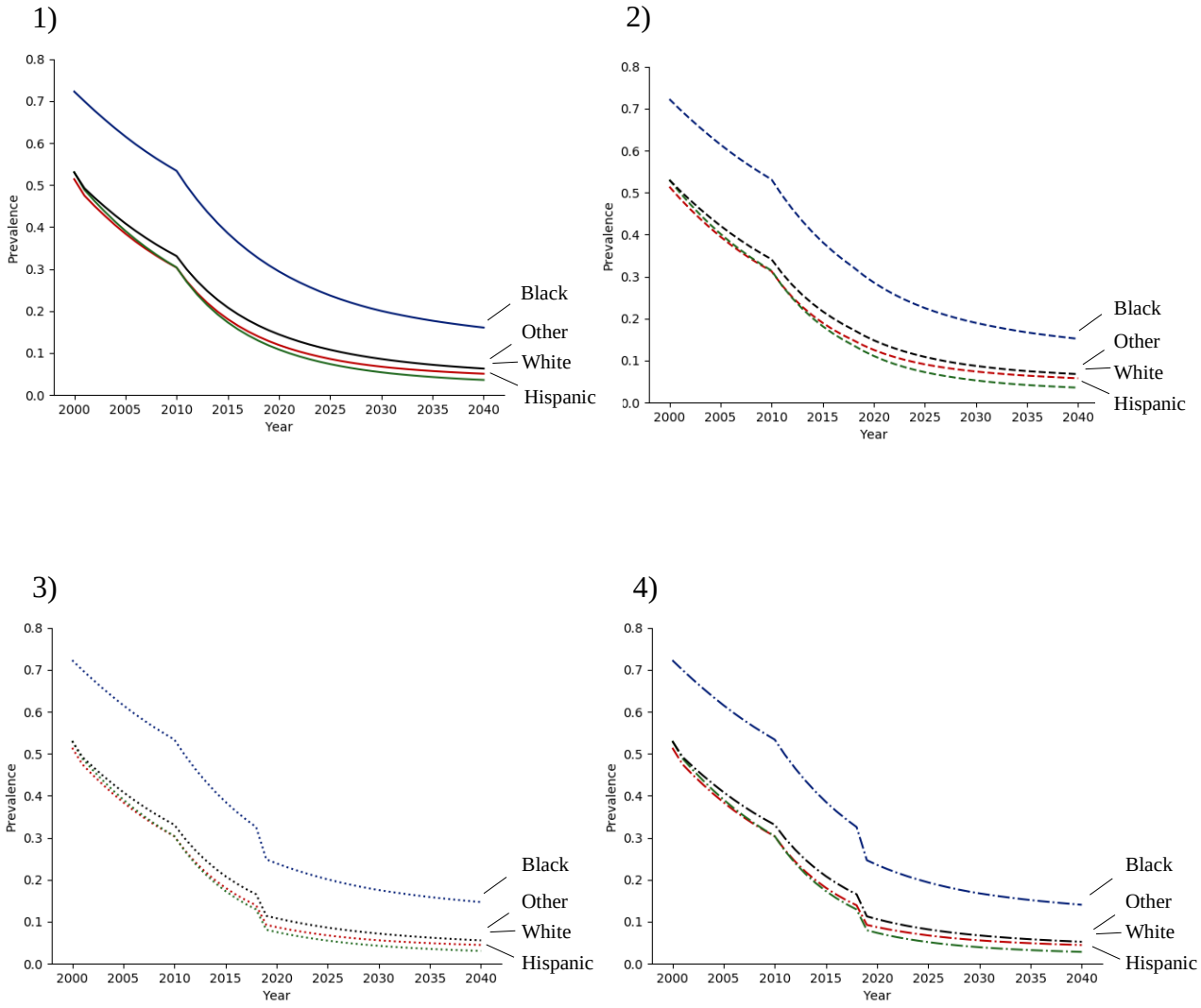
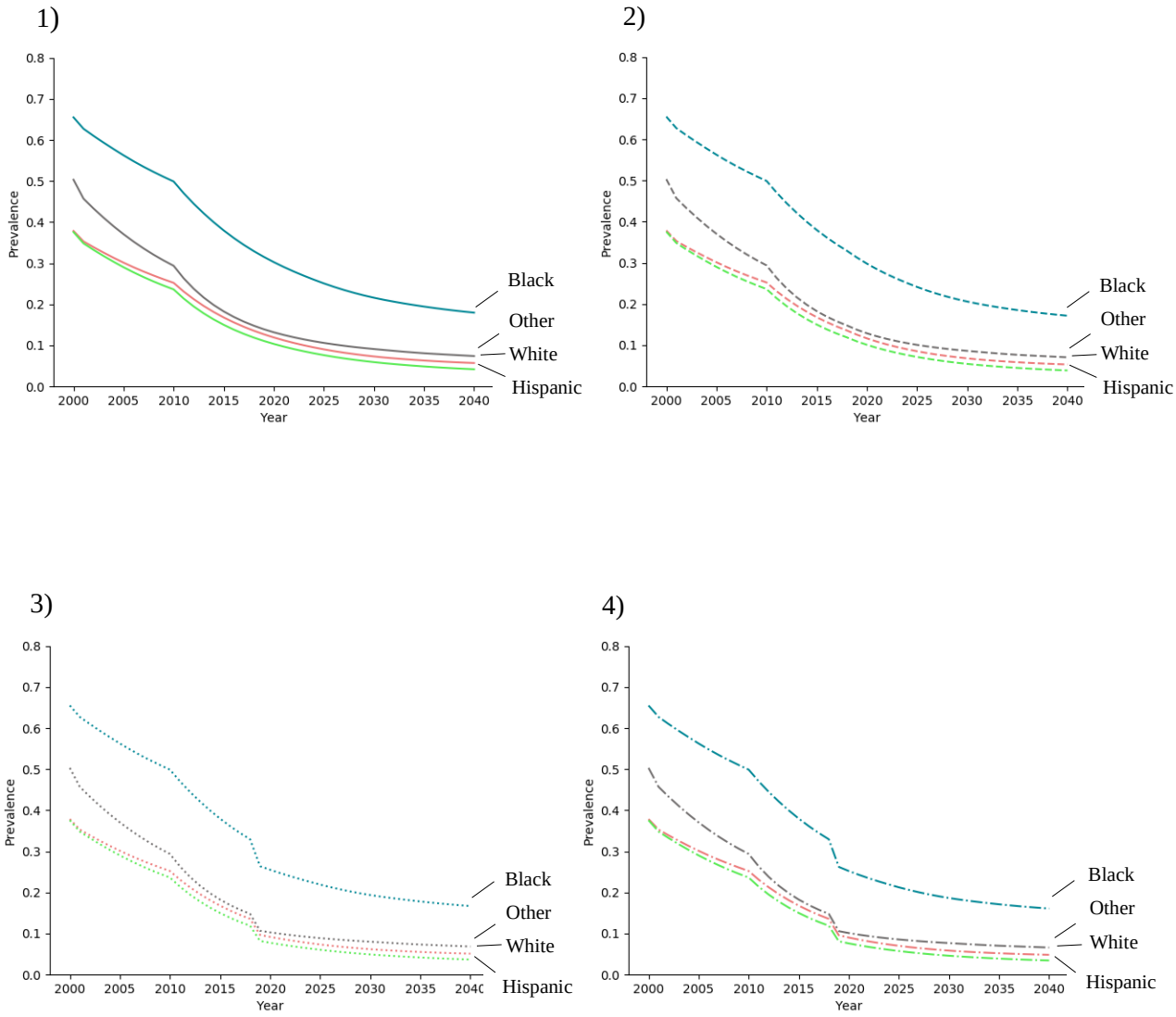


Figure 3.6 Simulated SHS exposure prevalence under intervention scenarios among males[a]



a) Intervention scenarios:
   1) Baseline
   2) 20% increase in cessation
   3) 20% decrease in parameter (β_1) relating smoking prevalence to SHS exposure
   4) Combined (2 and 3)

Figure 3.7 Simulated SHS exposure prevalence under intervention scenarios among females[a]



b) Intervention scenarios:
   1) Baseline
   2) 20% increase in cessation
   3) 20% decrease in parameter (β_1) relating smoking prevalence to SHS exposure
   4) Combined (2 and 3)

Supplementary Material

Table SM 3.1 Optimized cessation probabilities using Nelder-Mead algorithm

	Ages 18-34	Ages 35-64	Ages 65+
		2000-2010	
White males	-0.52%	1.94%	3.92%
White females	-0.59%	2.29%	4.33%
Black males	-2.06%	0.61%	3.66%
Black females	-3.38%	1.26%	5.29%
Hispanic males	1.10%	3.18%	6.23%
Hispanic females	-0.02%	3.80%	6.77%
Other males	0.36%	3.07%	6.11%
Other females	2.75%	4.32%	4.90%
		2011-2040	
White males	2.63%	4.95%	8.97%
White females	1.74%	4.42%	9.27%
Black males	0.17%	2.68%	5.84%
Black females	0.26%	3.88%	10.55%
Hispanic males	2.29%	4.24%	7.56%
Hispanic females	0.30%	3.21%	8.64%
Other males	1.37%	3.90%	8.47%
Other females	1.12%	2.59%	6.00%

Table SM 3.2 Estimated probability of all-cause mortality in the general population, by age group, 2015-2040

	2015	2020	2025	2030	2035	2040
Ages 28-34	0.00074	0.00076	0.00076	0.00076	0.00076	0.00076
Ages 25-34	0.00115	0.00126	0.00127	0.00126	0.00127	0.00127
Ages 35-44	0.00194	0.00199	0.00193	0.00186	0.00182	0.00176
Ages 45-54	0.00421	0.00410	0.00399	0.00372	0.00362	0.00355
Ages 55-64	0.00881	0.00882	0.00865	0.00827	0.00798	0.00763
Ages 65-74	0.01847	0.01833	0.01806	0.01772	0.01750	0.01702
Ages 75-84	0.04588	0.04346	0.04277	0.04296	0.04254	0.04247

Table SM 3.3 Estimated number of SHS-attributable deaths due to (1) IHD and (2) lung cancer, 2000-2016

1)

	Males				Females			
	White	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**
2000	15895	2252	722	344	8738	2102	509	220
2001	14268	2157	733	344	8146	1993	491	231
2002	13362	2096	721	343	7537	1908	472	229
2003	12237	2063	676	340	6909	1794	452	224
2004	10967	1901	657	307	6170	1670	413	214
2005	10166	1849	658	311	5687	1577	418	215
2006	9259	1798	594	307	5051	1452	380	211
2007	8389	1672	562	288	4504	1351	343	200
2008	7923	1614	525	293	4251	1283	320	197
2009	7251	1545	518	286	3778	1206	297	187
2010	6815	1487	495	275	3468	1140	289	187
2011	6033	1418	434	254	3061	1067	251	180
2012	5333	1373	395	244	2726	987	226	164
2013	4816	1315	374	239	2421	955	207	163
2014	4328	1267	336	220	2180	896	188	158
2015	3925	1233	323	213	2019	853	172	154
2016	3591	1238	298	205	1817	847	161	154

2)

	Males				Females			
	White	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**
2000	4049	502	100	65	2175	364	54	39
2001	3757	490	102	69	2116	357	54	45
2002	3551	479	105	67	2090	367	56	43
2003	3353	469	103	72	2015	368	57	48
2004	3160	460	101	76	1941	365	55	50
2005	3013	444	104	72	1866	363	57	52
2006	2823	435	95	74	1795	353	55	51
2007	2638	425	94	74	1742	347	59	56
2008	2483	410	96	77	1659	334	57	55
2009	2330	403	91	74	1593	329	54	57
2010	2211	401	91	78	1528	330	55	60
2011	1970	381	82	74	1400	317	50	60
2012	1764	373	74	71	1296	312	48	58
2013	1574	359	68	66	1193	303	44	58
2014	1424	338	61	65	1114	287	43	57
2015	1276	321	57	64	1021	267	40	55
2016	1134	304	51	61	926	259	36	54

Figure SM 3.1 Observed v. simulated smoking prevalence for White males (1) and females (2), Black males (3) and females (4), Hispanic males (5) and females (6), other males (7) and females (8)

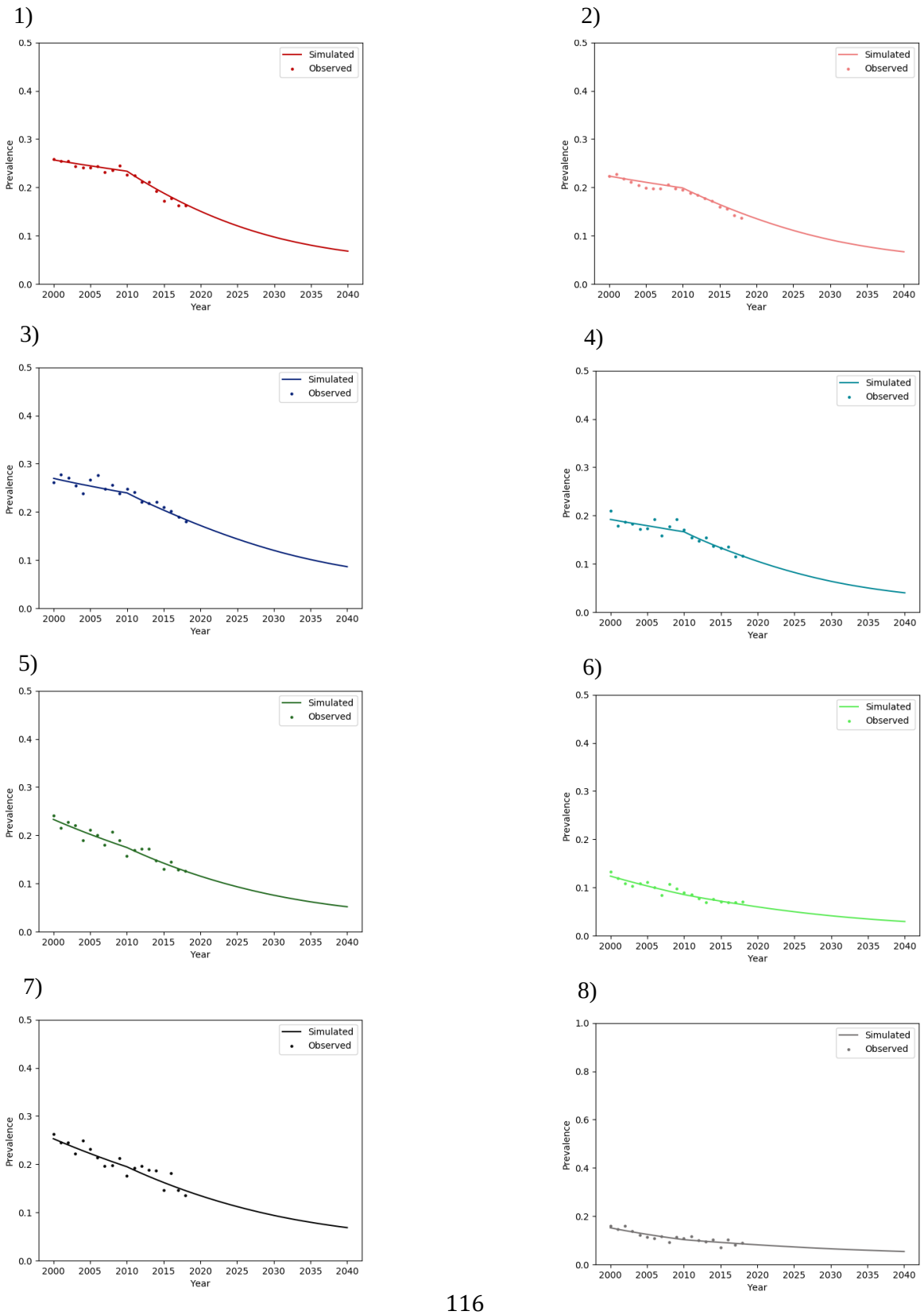
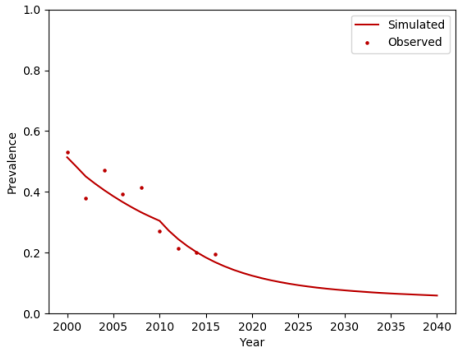
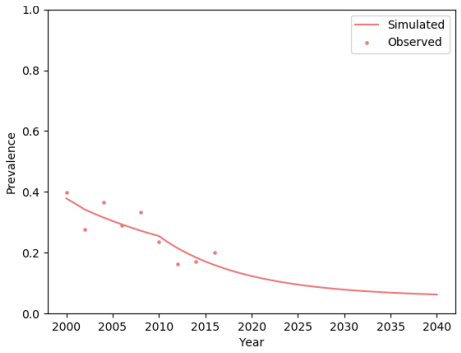


Figure SM 3.2 Observed v. simulated SHS exposure prevalence for White males (1) and females (2), Black males (3) and females (4), Hispanic males (5) and females (6), other males (7) and females (8)

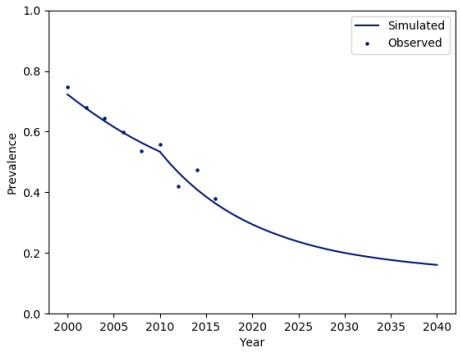
1)



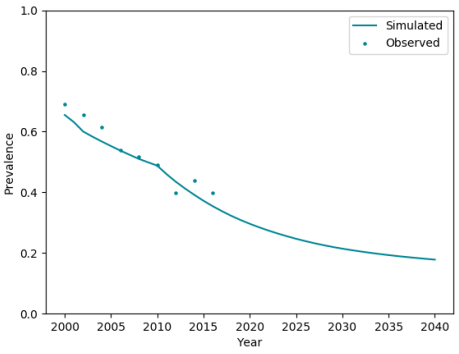
2)



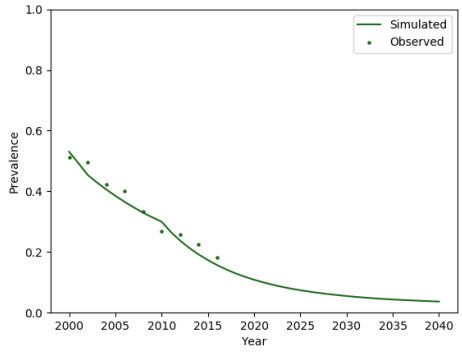
3)



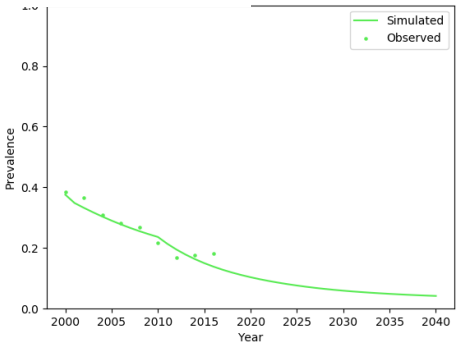
4)



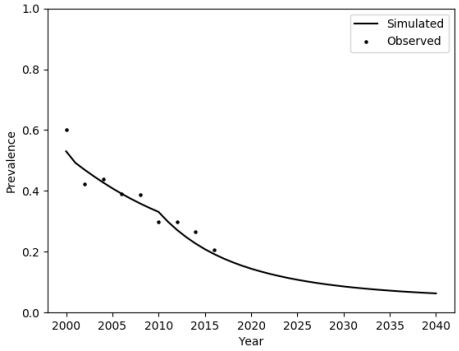
5)



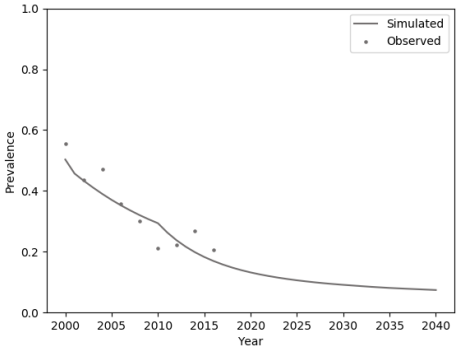
6)



7)



8)



CHAPTER 4. State-Level Structural Stigma and Smoking among Sexual Minority Adults in the United States, 2012-2014

Introduction

Despite reductions in smoking in the United States over the past several decades, the prevalence of smoking among sexual minority (SM)—lesbian, gay, and bisexual—adults remains higher than among heterosexual adults.[1] Data from the National Health Interview Survey (NHIS) suggests that 20.6% of SM adults smoked in 2018, compared to 13.5% of heterosexual adults.[2] This disparity may be even more pronounced among females: according to data from the 2012-2013 National Adult Tobacco Survey (NATS), the prevalence of smoking was 36.0% among bisexual females, compared to 22.2% among lesbians and 14.3% among heterosexual females.[1] SM females are also less likely to quit smoking and more likely to use alternative tobacco products, compared to heterosexual females.[1,3]

Disparities in smoking by sexual orientation have been well documented; however, less is known about the drivers underlying the high prevalence of smoking among SM individuals. One potential explanation rests on the "minority stress hypothesis,"[4] which suggests that SM individuals may experience stress associated with membership in a stigmatized group, and that these stress processes contribute to negative health outcomes, including smoking or other substance use.[4–6] Minority stress has been described as a type of social stress, where external stigmatizing factors affect individual psychological processes.[4] In developing a taxonomy to link minority stress to types of stigma faced by SM individuals, researchers have conceptualized

stigma as a multi-level construct, ranging from individual-level experiences of internalized stigma, to interpersonal experiences of discrimination, bullying or violence, to structural factors, encompassing policies and attitudes towards SM individuals.[5]

While stigma has been operationalized in a multitude of ways, higher levels of exposure to these stigmatizing factors are associated with numerous adverse health outcomes among SMs. These outcomes include higher levels of substance use,[7–10] poorer mental health outcomes,[11–16] increases in self-reported stress,[17] lower levels of engagement and satisfaction in health care,[18,19] higher prevalence of sexually transmitted infections (STIs),[20] lower uptake of HIV prevention strategies,[21] worse self-rated health and life satisfaction,[12,22,23] and blunted cortisol responses to stress.[24] On the other hand, several studies have found mixed or no associations between exposure to sexual orientation stigma and some mental health and substance use outcomes in studies of young SM men,[20] and young adults experiencing homelessness.[25]

With regard to smoking in particular, the minority stress hypothesis provides a theoretical foundation for linking stress associated with stigma to tobacco use, as indicators of stress have been implicated in smoking initiation and transition to regular smoking.[26] Empirical research on SM populations also supports the connection between exposure to stigma and smoking behavior. Among SM individuals, higher levels of internalized heterosexism (internalization of negative societal attitudes about sexual minorities) were associated with a higher odds of smoking among men,[7] and have also been hypothesized to explain differences in tobacco use between butch and femme SM women.[10] Likewise, in a nationally representative sample of adults, experiencing self-reported past-year discrimination on the basis of sexual orientation was associated with a higher likelihood of past-year cigarette smoking, any tobacco/nicotine use, and tobacco use disorders among SMs.[27] Studies of exposure to stigma on the structural level have revealed

similar patterns. In a study of county-level indicators of support for same-sex marriage, higher levels of support were associated with a lower likelihood of smoking among SM adults.[28] Likewise, living in states with lower levels of structural stigma was found to be associated with a lower risk of smoking among a sample of SM youth, after controlling for individual risk factors, and state-level inequality, median household income, and smoking prevalence.[29]

While a growing number of studies have examined exposure to structural stigma and smoking, there are several gaps in the literature that remain unfilled. For example, it is challenging to disentangle structural stigma from other correlated structural factors that may also be associated with smoking. In some prior research, associations between exposure to structural stigma and smoking were similar among both SM adults and heterosexual adults,[28] which could be due to residual confounding, potentially by sociodemographic variables and/or tobacco control policies. In particular, we are not aware of prior studies that attempt to disentangle stigma factors from correlated tobacco control policies, including smoke-free laws and taxation policies. In addition, it is not known whether the relationship between structural stigma and smoking among SM adults varies by sex. Given observed difference in smoking patterns between SM males and SM females,[1] the potential for effect modification by sex is an important consideration. Finally, the changing environment of policies and social attitudes towards same-sex relationships in recent years has led researchers to call for a renewed research agenda focused on examining the dynamics of the minority stress hypothesis using contemporary datasets, as it is not clear that findings from earlier studies still apply today.[6] Spatial and temporal heterogeneity in structural stigma indicators over the past decade provides a rich opportunity for probing the continuing relevance of the minority stress hypothesis in explaining persistent disparities in smoking by sexual orientation.

In this study, we examined the relationship between structural stigma and smoking among SM adults using a nationally representative dataset collected between 2012 and 2014. We focused on structural stigma, as opposed to interpersonal or individual stigma, to help elucidate potential policy drivers of SM health outcomes. In addition to exploring structural stigma and smoking among SM adults, we also examined whether structural stigma was associated with smoking among heterosexuals within the same dataset. While we did not hypothesize that stigma against SM individuals would predict smoking among heterosexuals, a significant association could signal residual confounding or could indicate other potential mechanisms linking structural stigma to smoking patterns among heterosexual adults. We employed a robust set of control variables to attempt to disentangle structural stigma from sociodemographic factors and state-level tobacco control policy environments. Finally, we examined whether the association between stigma and smoking among SM adults was modified by sex.

Methods

Sample. To characterize smoking outcomes, we used data from the National Adult Tobacco Survey (NATS), 2012-2014. NATS is a landline and cell phone survey on tobacco use behaviors, which is representative of the U.S. adult non-institutionalized adults (ages 18+).[30] The NATS dataset was chosen for this analysis for three reasons. First, it contains questions on sexual orientation as well as detailed questions regarding tobacco use behaviors. Second, it is a large, nationally representative sample with publicly available indicators on state of residence, which allows for the assignment of state-level exposures. Third, the NATS survey data were available over two waves, spanning 2012-2014, which is a critical period for studying structural stigma indicators among SM populations, given the changing landscape of policies and social attitudes during these years. This period was also critical for observing dynamic changes in state-level

stigma environments, as our study period directly preceded *Obergefell v. Hodges* (2015), the Supreme Court case that required all states to license same-sex marriages and recognize same-sex marriages performed out of state.[31] The analytic sample for the primary analysis consisted of all U.S. adults (ages 25+) with nonmissing observations on smoking status, sexual orientation, state of residence, and control variables. The lower age limit was chosen to restrict the analysis to adults who had likely completed their educational attainment, since education was used as a control variable in the analysis. We also excluded individuals from Washington, D.C., as we did not have full information to construct a structural stigma score for this area.

Tobacco use measures. Current smoking status was assessed from a derived variable based on the NATS survey question, "Do you now smoke cigarettes every day, some days, or not at all?" This question was asked of all individuals ages 18-29 and of individuals at least 30 years old who reported that they had smoked at least 100 cigarettes in their entire life. Respondents indicating that they smoke "every day" or "some days" were considered current smokers.

Sexual orientation and sex measures. Sexual orientation and sex were also assessed via questions in the NATS survey. SM status was coded as a dichotomous variable (yes v. no), where individuals who responded that they considered themselves as gay, lesbian, or bisexual were considered sexual minorities. Heterosexuals encompassed any respondent who indicated that they considered themselves to be "straight, that is, not lesbian or gay." For the purposes of this analysis, respondents for whom sexual orientation data was not ascertained, who refused the question, who did not understand the question, or who responded "something else" or "don't know", were not included in the analytic sample.

Ascertainment of sex in the NATS survey changed over the course of the study period. In

the first survey wave, the questionnaire asked individuals to specify whether they were male or female, whereas in the second survey wave, the questionnaire asked respondents to indicate their sex at birth and whether they currently considered themselves to be male or female. To address this discrepancy, we included only cisgender individuals from wave 2 in our sample, i.e., individuals whose identity as "male" or "female" aligned with their reported sex at birth. Other responses were not included in the analytic sample.

Structural stigma data. We adapted a previously developed index of structural stigma as the exposure variable for our analysis.[8,11,32] Following methods described in prior literature, the measure was comprised of three primary components: state-level policies relating to sexual minorities, the state-level density of same-sex couple households, and public opinion towards same-sex marriage. State-level policies included laws regarding same-sex marriage (permitted v. not), laws regarding the inclusion of sexual orientation as a protected category under hate crime laws (included v. not), and laws prohibiting discrimination on the basis of sexual orientation in employment, housing, and public accommodations (prohibit discrimination in all three venues v. not). For each state in each month, a value of 0 or 1 was assigned for each policy area, with the value of 1 representing the presence of an inclusive policy. Policies were considered present based on when laws were implemented (e.g., when a state began to issue marriage licenses to same-sex couples). The policy components were then summed, creating a summary measure ranging from 0 to 3. Policy data were derived from the Human Rights Campaign (HRC) "State Maps of Laws & Policies," the Center for American Progress Action Fund, and the Anti-Defamation League.[33–35] Estimates were dynamically updated throughout the study period based on month and year.

Information on the density of same-sex couple households was derived from the 2010

decennial U.S. Census and compiled by the Williams Institute at the UCLA School of Law.[36] While there is no direct measure of sexual orientation in the U.S. Census, the distribution of same-sex couple households can be inferred based on the sex of the household owner/renter and the sex of their spouse or unmarried partner.[37] Initial estimates released by the Census Bureau over-counted the number of same-sex couple households due to coding errors in recording the sex of respondents. The estimates of same-sex couple households used in this analysis have accounted for these errors and represent the Census Bureau's "preferred" estimates of same-sex couple households at the state level.[36]

Annual measures of public opinions towards same-sex marriage were derived from work by Lewis and Jacobsmeier using multi-level regression and post-stratification (MRP) methods to develop state-level measures of public opinion.[38] Briefly, the MRP approach combined polling data from the Roper Center's iPOLL archive with Census data to post-stratify and weight individual responses such that they become more accurate representations of overall state-level opinion. Previously derived MRP measures of same-sex marriage opinion were only available for 2012-2013; consequently, we extended the method outlined by Lewis and Jacobsmeier to generate a state-level measure of attitudes towards same-sex marriage in 2014. Indicators of state-level average support for same-sex marriage were generated from individual responses to the following question, administered via Pew Research Organization polls: "Do you strongly favor, favor, oppose, or strongly oppose allowing gays and lesbians to marry legally?"[38] The proportion of respondents in a state who supported same-sex marriage were all individuals who responded "strongly favor" or "favor" divided by the total number of respondents in each state, incorporating the post-stratification weights.

Previous literature has demonstrated the validity of combining these three factors—

policies, density of same-sex couples, and public opinion—into a single variable to represent a multi-faceted construct of structural stigma.[29] However, we similarly validated this approach by evaluating the Cronbach's alpha and performing a principal components analysis (PCA) across the three individual variables.[29] The alpha value was 0.83, which implied that all components could be combined into one variable representing a common underlying construct.[29] When we standardized each variable and performed a PCA, the eigenvalue associated with the first component was 2.44, whereas the second component was 0.33. The coefficients associated with the first component were also of the same direction and of similar magnitude (between .56 and .59), which provided further support for combining the three measures into a single variable. Following an approach outlined in prior literature, we constructed this variable by summing the standardized component measures.[11] The resulting index was reverse-coded such that higher values indicated higher levels of structural stigma.

Other covariate data. A set of individual-level covariates were employed as control variables in the statistical analysis, including race/ethnicity (*non-Hispanic White, non-Hispanic Black, Hispanic, Other/multiracial*); age (continuous); education (*less than high school, high school/equivalent, some college, college +*); and sex (*male, female)*. Models also controlled for state-level tobacco control policies, including smoke-free laws, tobacco taxation, and state-level spending on anti-tobacco initiatives. A variable representing the average proportion of the state population covered by smoke-free laws in workplaces and hospitality venues was derived from the American Nonsmokers' Rights Foundation (ANRF) and Census Bureau population data.[39] The CDC's "Tax Burden on Tobacco, Vol. 51" was used to assess average price per pack of cigarettes in each state.[40] We adjusted for per capita state-level tobacco control expenditures using data from the Campaign for Tobacco-Free Kids.[41] Because it is possible that other state-

level factors could be associated with measures of structural stigma and smoking patterns, models also contained a set of state-level sociodemographic controls representing percent of the state population with at least a Bachelor's degree, percent Black, percent Hispanic, and a variable representing household income inequality (gini index).[42] These variables were derived from 1-year estimates from the American Community Survey.[43–45] We also explored including two additional state-level demographic variables representing the percent of the population unemployed and the percent below the federal poverty line. These variables were highly collinear with other state-level variables and so were not included in the final models. Finally, all models included year fixed effects to control for secular time trends.

Statistical analysis. We used modified Poisson regression models with robust standard errors to estimate the relative risk of smoking as a function of exposure to structural stigma. We stratified models by sexual minority status to examine whether there was a significant association between stigma and smoking among both SM adults and heterosexual adults. We first fit bivariate models to examine unadjusted associations, and then fit fully adjusted models including individual-level covariates, state-level sociodemographic factors, state-level tobacco control variables, and year fixed effects. We also explored whether the association between structural stigma and smoking among SM adults appeared to differ by sex by estimating models that included an interaction between sex and structural stigma and examining the *p*-value associated with the interaction. In all models, we included a squared term for the structural stigma measure to capture potential non-linearities in the association between exposure to structural stigma and smoking. All analyses adjusted for the complex survey design, using variables included in the NATS dataset representing strata and primary sampling units, as well as survey weights.

We also undertook several sensitivity analyses. We explored a number of model

specifications, including controlling for additional state-level covariates and disaggregating the structural stigma score to explore associations between individual components of the score and smoking status. We also revised our analytic sample definition to include all adults (ages 18+), instead of restricting our sample to adults ages 25+. Because younger adults may not have completed their educational attainment, we controlled for household income in these models, instead of education. We constructed a three-level variable for sexual minority status that included a category for individuals with missing data, and stratified by this variable to explore the potential dynamics of stigma among individuals missing information on sexual minority status. Finally, we estimated multinomial logistic regression models to examine the relationship between structural stigma and a three-level outcome variable: no current smoking (including former and never smokers), current some-day smoking, and current every-day smoking. All analyses were completed in Stata SE, version 15.

Results

The pooled sample across the two waves of data included 3,174 SM adults and 105,803 heterosexual adults. A flow chart of the exclusion criteria applied to the full NATS sample is included in Supplementary Figure 4.1. Almost 15,000 respondents were excluded due to missing or unused data on sexual orientation, including 6,938 individuals for whom the data was "not ascertained" and 4,674 individuals who "refused" the question. A detailed breakdown of the characteristics of individuals who were excluded from the analysis based on sexual orientation information is provided in Supplementary Table 4.1. Excluded individuals were more likely to be female, Hispanic, have lower levels of education, and have lower levels of income compared to the heterosexual and SM samples. An additional 8,495 respondents were excluded due to the age restriction; 1,250 due to missing covariate information; 1,230 due to residence in

Washington, D.C.; 392 due to missing outcome data; and 233 due to exclusions based on response to questionnaire items regarding gender identity. Table 4.1 contains descriptive details of the final analytic sample. The average state-based structural stigma score was lower among SM individuals (-0.37) compared to heterosexual individuals (0.07), indicating that SMs tended to live in states with lower exposure to structural stigma. The weighted prevalence of current smoking was 28% among SMs and 17% among heterosexuals.

Table 4.2 contains results from unadjusted and adjusted models examining the association between state-level structural stigma and the relative risk of current smoking. In bivariate models that included only year fixed effects, the linear term for structural stigma was positively associated with smoking for both SM adults (relative risk (RR) = 1.05, 95% confidence interval (CI) = 1.02-1.09) and heterosexual adults (RR = 1.05, 95% CI = 1.04-1.06); that is, higher levels of structural stigma were associated with a higher relative risk of smoking. In adjusted models, the associations appear to be curvilinear, and there was a significant association between the squared stigma term and risk of smoking in each model. Relative risks for SM adults were 1.03 (95% CI = 0.97-1.08) for the linear structural stigma term and 0.98 (95% CI = 0.97-1.00) for the squared term. For heterosexual adults the RR estimates were 1.00 (95% CI = 0.99-1.01) for the linear term and 0.99 (95% CI = 0.988-0.995) for the squared term. The squared terms for both the SM and heterosexual samples were statistically significant ($p<0.05$) in adjusted models. When we included an interaction between sex and the structural stigma score for the SM sample, a joint test of interaction with the linear and squared stigma terms was not significant, which suggests that the relationship between structural stigma and smoking did not significantly vary by sex in this sample.

To aid interpretation, we transformed the results from the adjusted Poisson models to the

probability scale and plotted the average marginal effects of structural stigma on smoking, ranging from the 10th to the 90th percentile of the structural stigma score. Plots for adjusted models without interactions by sex are included in Figure 4.1. For SM adults, the lowest probabilities of smoking occurred at the lowest and highest levels of exposure to structural stigma, and the highest probability of smoking occurred in the middle of the stigma distribution. The shape of the relationship was similar for heterosexuals, but the magnitude of the association was far less pronounced. Figure 4.2 contains a probability plot for the analysis that included an interaction between structural stigma and sex, estimated among SM adults included in the sample. This interaction was not statistically significant, and the shapes of the probability curves do not suggest that the relationship between structural stigma and smoking varied by sex.

We also conducted a number of sensitivity analyses to explore the robustness of results to variations in model specification. Results were similar in terms of magnitude and significance when we controlled for additional state-level sociodemographic variables, including measures of unemployment and poverty. We disaggregated the structural stigma index to examine relationships between each component of the index and smoking, and we observed a similar curvilinear association for each component. When we expanded the analytic sample to include all adults ages 18+ (rather than limiting the sample to ages 25+), there was a disproportionate increase in the number of SM adults in the sample, compared to heterosexuals, which suggests that younger adults were more likely to identify as sexual minorities than older adults. However, the addition of these individuals to the analytic sample produced similar results to those from models where age was restricted to 25+. When we estimated the relationship between structural stigma and smoking among individuals with missing data on sexual minority status, results closely resembled results among heterosexuals. Finally, we explored the use of multinomial

models with a three-level variable to capture every-day smoking, some-day smoking, and no current smoking outcomes among sexual minority adults. Regression estimates and predicted probability plots are included in Supplementary Table 4.2 and Supplementary Figure 4.2. The results of this sensitivity analysis suggest that the significant curvilinear relationship was driven entirely by the relationship between structural stigma and every-day smoking.

Discussion

We found that structural stigma was independently associated with smoking among SM adults, but that the relationship appeared to be non-linear. Exposure to structural stigma was similarly associated with smoking among heterosexuals, but the magnitude of the association was less pronounced. We did not find evidence that sex modified the association between exposure to structural stigma and smoking among SM adults.

Our study builds on a handful of analyses that have established exposure to structural stigma as a risk factor for smoking among SM adults as well as youth.[28,29] Importantly, we find that the relationship between structural stigma and smoking persisted after we incorporated a robust set of control variables, including state-level sociodemographic variables as well as indicators of the tobacco control environment, which we hypothesized could be confounders of the relationship between structural stigma and smoking. For example, it is well established that tobacco control environments vary widely by state and that tobacco control policies impact population-level smoking rates.[46] To address potential confounding by the tobacco control environment, we included variables representing state-level average price per pack of cigarettes, percent of the population covered by smoke-free laws, and per capita spending on tobacco control initiatives.

Our finding that structural stigma is associated with smoking in a curvilinear fashion departs from previous studies of structural stigma and smoking behavior, which tend to report linear associations.[28,29] In our study, the highest and lowest levels of stigma were associated with the lowest probabilities of smoking for both SM and heterosexual adults in adjusted models. There is some precedent for finding that the lowest risk of poor health outcomes may occur at both tails of the distribution of exposure to discrimination or at the highest levels of discrimination. Among a sample of gay and bisexual men in New York City, for example, a combined measure of the frequency of discriminatory experiences across a number of domains (race/ethnicity, gender, age, sexual orientation, income or socioeconomic position, HIV status) was found to be associated with psychological distress in a curvilinear fashion, where moderate levels of discrimination were associated with the highest depressive and anxious symptom scores.[47] Likewise, a prior study found that individuals who reported exposure to sexual orientation discrimination were more likely to have lower systolic blood pressure, compared to individuals who did not report exposure to discrimination, although the sample size for this study was limited.[48] These studies tend to focus on interpersonal forms of stigma, specifically perceived/self-reported discrimination.

While it is not clear what may drive curvilinear relationships between interpersonal stigma and poor health outcomes, one potential explanation is that individuals in highly stigmatizing environments are more likely to attribute negative events to discrimination, and that such external attributions may be protective for individual self-esteem.[49,50] In addition, the curvilinear relationship may be in part explained by variations in social support that accompany living in high or low stigmatizing environments, as there is strong evidence that the relationship between stigma and health outcomes is modified by social support.[4,51,52] If SM individuals in

high-stigma states also lived in areas with more social support—for example, urban areas[53]—this could help mitigate the effects of exposure to structural stigma on smoking. The observed curvilinear relationship may also be explained by migration patterns within SM communities, as evidence suggests that SM individuals are likely to selectively migrate to lower-stigma environments and urban areas.[54] Because our primary sample was cross-sectional and limited to adults ages 25 and older, we did not directly observe exposure to structural stigma during critical windows for initiating smoking behavior, such as adolescence and early adulthood.[55] Longitudinal studies of SM populations that incorporate dynamic information on exposure to structural stigma are needed to further elucidate the relationship between stigma and smoking trajectories, particularly as smoking is mostly initiated at younger ages and persists because of its addictive character. Additional studies of youth and young adult populations are also warranted.

While we did not find any evidence that the association between exposure to structural stigma and smoking varied by sex, we had limited power to detect the statistical significance of interactions between the sex and structural stigma variables. Data from national surveys suggest that sexual orientation smoking disparities are more pronounced among females than males.[56] On the other hand, while the prevalence of smoking tends to be highest among SM females, prior research also suggests that SM males may be more likely than SM females to "externalize" responses to stress (e.g., engage in coping behaviors, such as substance use).[9,57] Future work should examine the potential for effect modification by sex in a larger sample with more statistical power.

Strengths of this study include the use of a nationally representative dataset with substantial heterogeneity in state-level structural stigma environments. Our models accounted for numerous potential confounding variables at the individual and state level. Importantly, we

adjusted for aspects of the tobacco control environment that may be confounders of the association between structural stigma and smoking behavior, as there may be unobserved factors that affect both the tobacco control environment and other policies and attitudes at the state level. We also examined the functional form of the association between structural stigma and smoking by including a squared term for structural stigma. In doing so, we highlighted potential non-linearities in the relationship between exposure to structural stigma and smoking.

There were also a number of limitations in this study. While there are potential causal explanations for nonlinear response functions, the associations between structural stigma and smoking in this study should be interpreted with caution for several reasons. While we controlled for a number of individual and state-level factors, it is possible that our results were impacted by residual confounding. The significant, although small, association between exposure to structural stigma directed towards SM individuals and smoking among heterosexuals could indicate that stigma towards SM individuals is harmful for all populations. Alternatively, it is plausible that there are unmeasured aspects of the environment, including other correlated forms of stigma (e.g., other forms of discrimination), which also contribute to associations with smoking behavior. As we were limited to examining state-level variables, we were also not able to incorporate important sources of sub-state level variability in the environment. This limitation extends to both measures of structural stigma as well as variables accounting for sociodemographic characteristics and tobacco control policies. In addition, while point estimates suggested a curvilinear association between stigma and smoking among SM individuals, the confidence intervals associated with the estimation of smoking among sexual minorities at high levels of stigma were quite wide, which reflects that a smaller portion of the SM sample resided in areas with high levels of stigma.

An additional limitation to the current study was our modest sample size, which precluded us from exploring other avenues of potential effect modification of the association between exposure to structural stigma and smoking among SM adults, aside from sex. Several studies on nonlinear relationships between interpersonal stigma and health outcomes have documented important differences by socioeconomic status (SES), namely that the associations for individuals of lower SES were characterized by nonlinear dynamics, whereas nonlinear associations for higher-SES individuals were not as apparent.[58,59] Also due to sample size limitations, we were not able to examine the potential for a differential association between exposure to structural stigma and smoking across sexual orientation subgroups, despite evidence that smoking behavior and experiences of stigma vary across sexual orientation identities.[27,60–62] We also excluded a substantial number of individuals due to missing or unusable sexual orientation data. Additional analysis of this excluded sub-sample suggested that these data were not missing at random, a finding that has been previously noted with regard to other nationally representative datasets.[63] Missing data could have introduced bias into our statistical analysis, however, results from a sensitivity analysis suggested that the relationship between structural stigma and smoking among individuals excluded due to missing SM data strongly resembled associations among heterosexual adults. The ascertainment of sex and gender identity also changed across the survey waves. While not explored in this study, the incorporation of questions on both sex at birth and gender identity into the latter wave of the NATS is promising in that it could facilitate future work on gender differences in smoking. The use of cross-sectional data limited the causal assertions we are able to draw regarding the association between exposure to structural stigma and smoking. These limitations point to the need for more data—

particularly longitudinal data—to explore the dynamics of structural stigma and its association with smoking behavior.

In this study, we found that exposure to high levels of a variable capturing multiple aspects of the structurally stigmatizing environment towards SM individuals—including laws/policies, attitudes towards same-sex relationships, and same-sex couple density—was associated with smoking among SM adults in a curvilinear fashion. Exposure to structural stigma was also associated with smoking among heterosexual adults, although the relationship was far less pronounced. We did not find any evidence of effect modification by sex, though sample size was limited. This study highlights the potential role of structural stigma in helping to explain patterns of smoking among SM adults.

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Tables

Table 4.1 Descriptive characteristics of the NATS analytic sample[a]

	Sexual minority	**Heterosexual**
	N=3,174	N=105,803
Individual-level covariates	**N (%)[b]**	**N (%)[b]**
Sex		
Male	1,602 (52%)	44,536 (48%)
Female	1,572 (48%)	61,267 (52%)
Race/ethnicity		
Non-Hispanic White	2,372 (65%)	83,970 (70%)
Non-Hispanic Black	231 (11%)	7,582 (11%)
Hispanic	269 (13%)	6,471 (11%)
Other Non-Hispanic	302 (11%)	7,780 (8%)
Education		
<High school	131 (8%)	6,756 (12%)
High school graduate	508 (22%)	22,462 (26%)
Some college	1,342 (44%)	44,790 (41%)
College+	1,193 (26%)	31,795 (21%)
Annual household income		
<$30k	596 (23%)	17,838 (21%)
$30k - $49,999	635 (22%)	19,498 (23%)
$50k - $69,999	459 (16%)	14,658 (16%)
$70k - $99,999	484 (14%)	15,593 (17%)
$100k+	730 (24%)	21,964 (24%)
Age (mean & s.d.)	44.50 (14.19)	50.69 (16.50)
State-level covariates	**Mean (s.d.)**	**Mean (s.d.)**
% with at least a Bachelor's degree	19% (2%)	18% (2%)
% Hispanic	18% (13%)	17% (13%)
% Black	12% (8%)	12% (8%)
Gini index	47.42 (.02)	47.33 (.02)
Price per pack of cigarettes, USD	4.76 (1.11)	4.69 (1.11)
Tobacco control spending, USD[c]	1.42 (1.53)	1.44 (1.70)
% covered by smoke-free laws[d]	73% (31%)	71% (35%)
Structural stigma	**Mean (s.d.)**	**Mean (s.d.)**
Average structural stigma score	-.37 (2.62)	.07 (2.72)
Smoking status	**N(%)**	**N(%)**
Current smoker	643 (28%)	13,408 (17%)

a) Estimates incorporate survey weights
b) Estimates of N(%), except where noted otherwise; % weighted
c) Per capita
d) Average state-level coverage of workplaces and hospitality venues (restaurants or bars)

Table 4.2 Relative risk estimates from bivariate and adjusted models stratified by SM status relating structural stigma to current smoking

	Model 1[a] (SM)	**Model 1[a] (Heterosexual)**	**Model 2[b] (SM)**	**Model 2[b] (Heterosexual)**	**Model 3[c] (SM)**	**Interaction p-value**
Structural stigma	1.05** (1.02-1.09)	1.05*** (1.04-1.06)	1.03 (0.97-1.08)	1.00 (0.99-1.02)	1.04 (0.98-1.10)	
Structural stigma^2	0.99 (0.97-1.00)	1.00 (0.996-1.002)	0.98* (0.97-1.00)	0.99*** (0.988-0.995)	0.98 (0.96-1.00)	
Male					0.88 (0.69-1.12)	
Structural stigma*male					0.98 (0.92-1.05)	
Structural stigma^2*male					1.00 (0.98-1.03)	p=0.744[d]

*p< 0.05, **p<0.01, ***p<0.001

a) Bivariate models with year fixed effects

b) Models adjust for individual level covariates (sex, age, race/ethnicity, educational attainment), state-level characteristics (percent with at least a Bachelor's degree, percent Black, percent Hispanic, Gini index), state-level measures of tobacco control policies (spending per capita on tobacco control, percent covered by smoke-free laws, average price per pack of cigarettes), and year fixed effects

c) Models adjust for all covariates listed in (b), and include interactions between structural stigma, structural stigma squared, and sex

d) p-value represents joint test of interaction with linear and quadratic structural stigma terms

Figures

Figure 4.1 Average marginal effects of structural stigma on smoking for SM and heterosexual adults

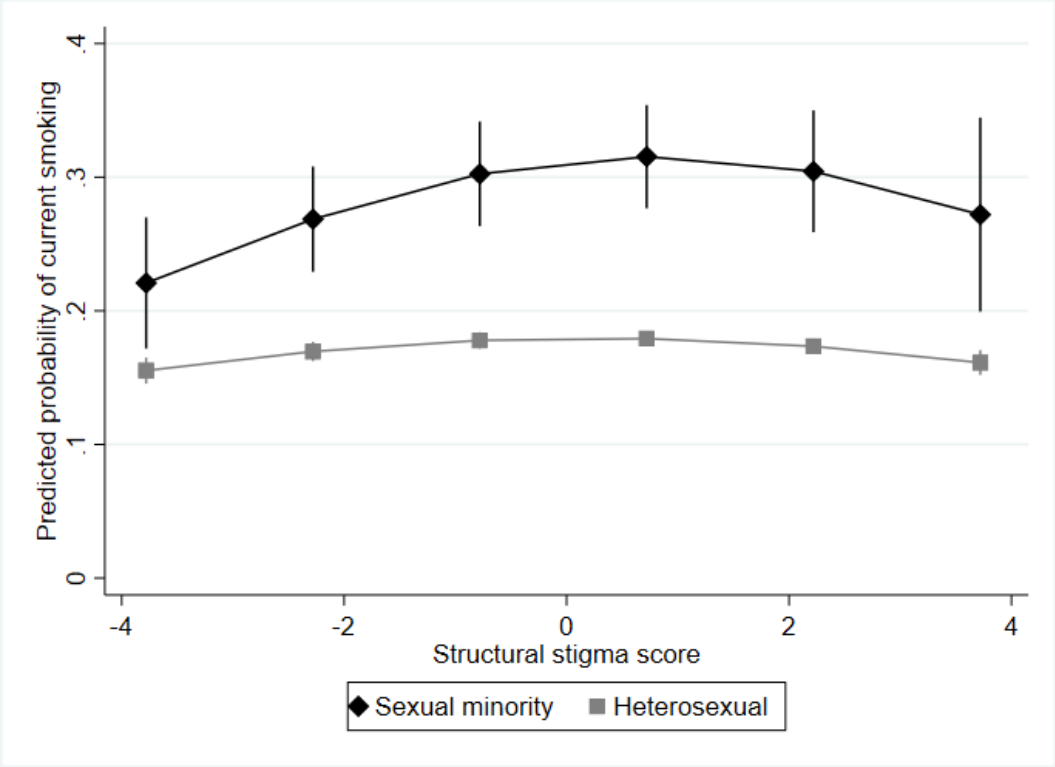
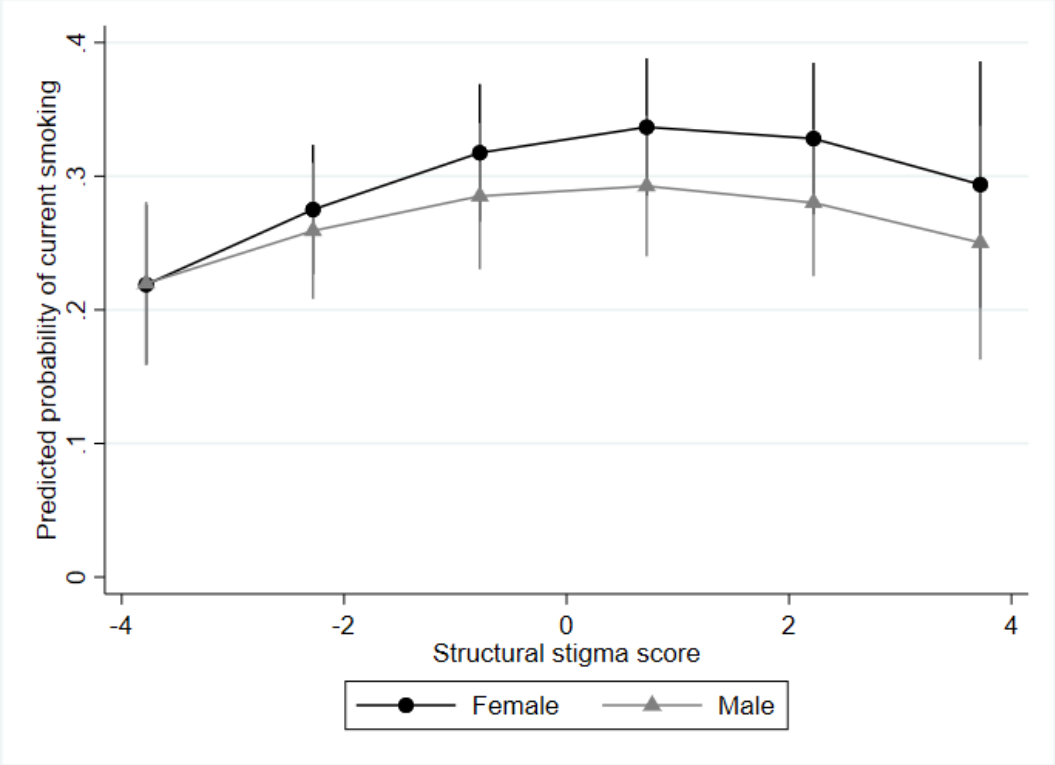


Figure 4.2 Average marginal effects of structural stigma on smoking for males and females, among SM adults



Supplementary Material

Table SM 4.1 Descriptive characteristics of analytic sample versus individuals excluded based on sexual orientation data[a]

	Sexual minority	**Heterosexual**	**Excluded**
	N=3,174	N=105,803	N=10,198
Individual-level covariates	**N (%)[b]**	**N (%)[b]**	**N(%)[b,c]**
Sex			
Male	1,602 (52%)	44,536 (48%)	6,130 (46%)
Female	1,572 (48%)	61,267 (52%)	4,068 (54%)
Race/ethnicity			
Non-Hispanic White	2,372 (65%)	83,970 (70%)	6,547 (46%)
Non-Hispanic Black	231 (11%)	7,582 (11%)	1,065 (13%)
Hispanic	269 (13%)	6,471 (11%)	1,639 (33%)
Other Non-Hispanic	302 (11%)	7,780 (8%)	947 (8%)
Education			
<High school	131 (8%)	6,756 (11%)	1,860 (32%)
High school graduate	508 (22%)	22,462 (26%)	2,743 (28%)
Some college	1,342 (44%)	44,790 (41%)	3,834 (31%)
College+	1,193 (26%)	31,795 (21%)	1,761 (10%)
Annual household income			
<$30k	596 (23%)	17,838 (21%)	1,366 (47%)
$30k - $49,999	635 (22%)	19,498 (23%)	775 (25%)
$50k - $69,999	459 (16%)	14,658 (16%)	414 (12%)
$70k - $99,999	484 (14%)	15,593 (17%)	296 (7%)
$100k+	730 (24%)	21,964 (24%)	354 (9%)
Age (mean & s.d.)	44.50 (14.19)	50.69 (16.50)	51.68 (17.21)
State-level covariates	**Mean (s.d.)**	**Mean (s.d.)**	**Mean (s.d.)**
% with at least a Bachelor's degree	19% (3%)	18% (2%)	19% (2%)
% Hispanic	18% (13%)	17% (13%)	18% (13%)
% Black	12% (8%)	12% (8%)	12% (8%)
Gini index	47.42 (0.02)	47.33 (0.02)	47.58 (0.02)
Price per pack of cigarettes ($)	4.76 (1.11)	4.69 (1.11)	4.76 (1.11)
Tobacco control spending ($)[d]	1.42 (1.53)	1.44 (1.70)	1.42 (1.52)
% covered by smoke-free laws[e]	73% (31%)	71% (35%)	73% (31%)
Structural stigma	**Mean (s.d.)**	**Mean (s.d.)**	**Mean (s.d.)**
Average structural stigma score	-.37 (2.62)	.07 (2.72)	-0.25 (2.59)
Smoking status	**N(%)**	**N(%)**	**N(%)**
Current smoker	643 (28%)	13,408 (17%)	1,446 (18%)

a) Estimates incorporate survey weights; "excluded" individuals include respondents for whom sexual orientation data was not ascertained, or who responded "don't know" or "something else"
b) Estimates of N(%), except where noted otherwise; % weighted
c) N includes all individuals who were only excluded from the analytic sample based on sexual orientation information
d) Reported per capita
e) Average state-level coverage of workplaces and hospitality venues

Table SM 4.2 Odds ratios for every-day smoking and some-day smoking versus no current smoking among SM respondents[a]

	Model 1[b]	Model 2[c]
Every-day smoking v. no current smoking		
Structural stigma	1.09** (1.04-1.15)	1.03 (0.94-1.13)
Structural stigma^2	0.98 (0.96-1.00)	0.97* (0.95-1.00)
Some-day smoking v. no current smoking		
Structural stigma	0.97 (0.90-1.06)	0.99 (0.85-1.15)
Structural stigma^2	0.99 (0.96-1.02)	0.99 (0.95-1.03)

*p< 0.05, **p<0.01, ***p<0.001

a) Estimates generated from multinomial logistic regression models

b) Bivariate models with year fixed effects

c) Models adjust for individual level covariates (sex, age, race/ethnicity, educational attainment), state-level characteristics (percent with at least a Bachelor's degree, percent Black, percent Hispanic, Gini index), state-level measures of tobacco control policies (spending per capita on tobacco control, percent covered by smoke-free laws, average price per pack of cigarettes), and year fixed effects

Figure SM 4.1 Analytic sample flow diagram

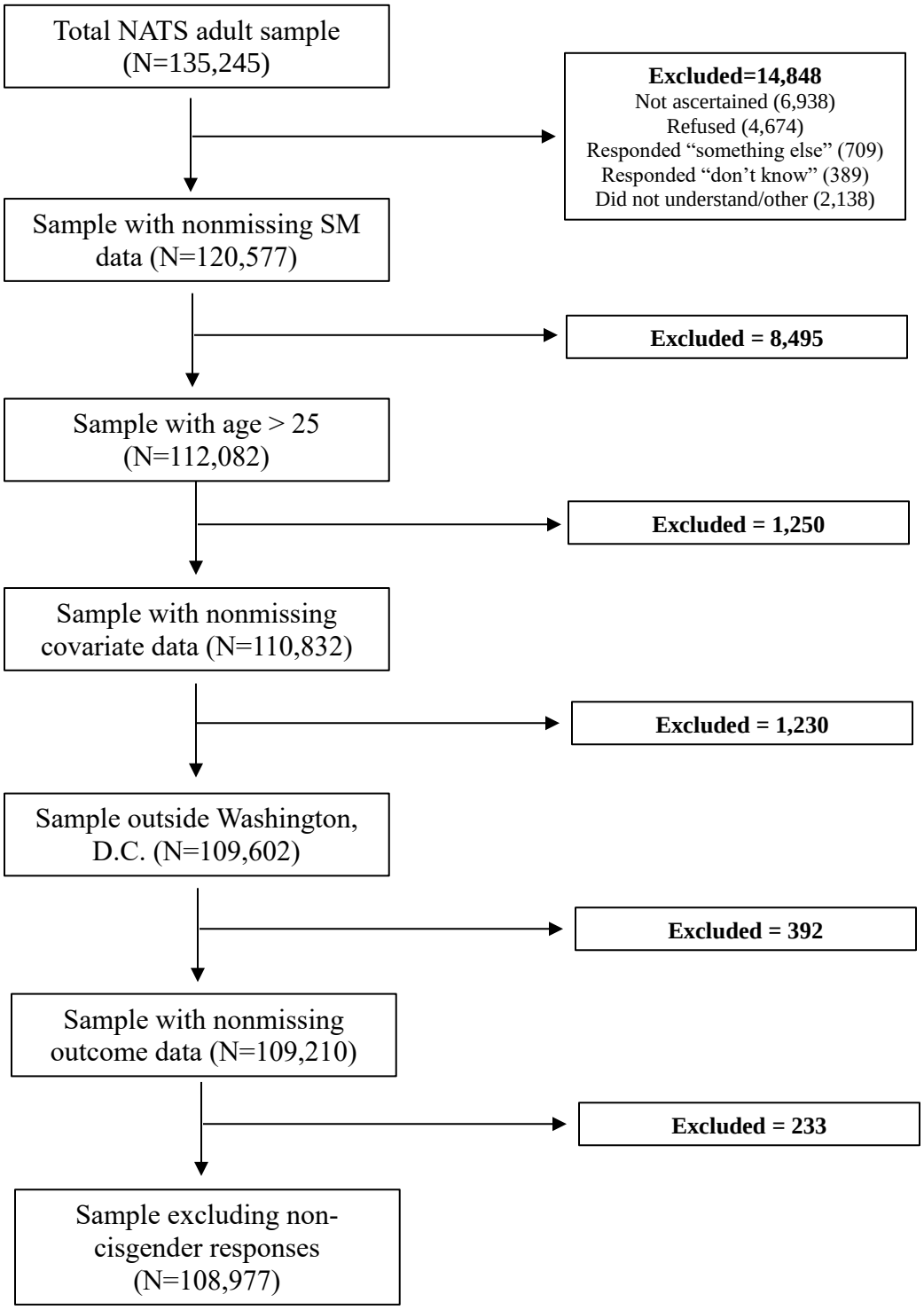
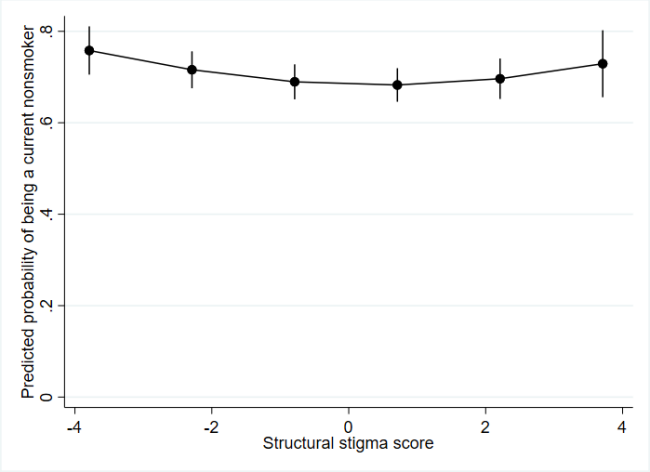
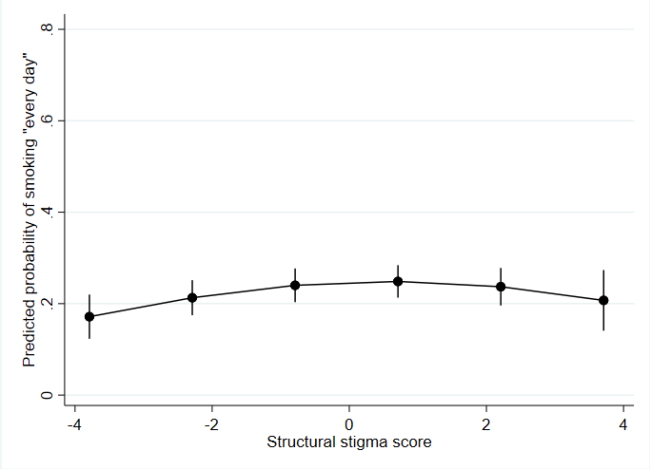


Figure SM 4.2 Average marginal effects of structural stigma on being a current nonsmoker (1), smoking "every day" (2), and smoking "some days" (3) among SM adults

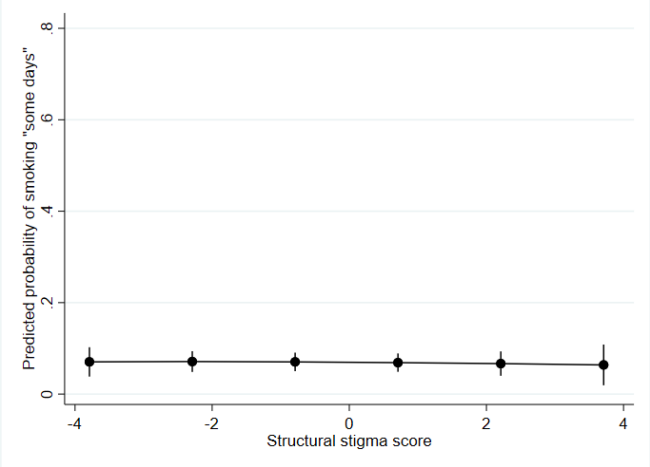
1)



2)



3)



CHAPTER 5: Discussion

Summary and Implications of Main Findings

Despite significant gains in reducing the burden of tobacco use, smoking remains the leading cause of preventable mortality in the United States.[1] Moreover, patterns of tobacco use in the U.S. are increasingly characterized by sociodemographic disparities, which in turn translate into disparities in tobacco-related health outcomes.[2]

Local environments can significantly impact disparate patterns of tobacco use by exposing individuals to policies and/or risk factors that are associated with tobacco use outcomes. In the case of tobacco control policies, for example, there is a large body of evidence that suggests that commonly implemented tobacco control policies, including taxation of tobacco products and smoke-free air laws, are associated with decreased smoking.[3] Yet, there is also debate as to the effects of these policies on health equity, which requires considerations of differential exposure as well as differential response (i.e., through effect modification) to policies based on sociodemographic characteristics. Understanding the structural drivers of persistent disparities in tobacco use is critical to designing effective interventions that target high rates of smoking in the most vulnerable groups.

In this dissertation, we explored two examples of tobacco-related health disparities, relating to 1) heterogeneous patterns of exposure to secondhand smoke (SHS) and 2) the differential smoking risk in heterosexual and SM communities. Despite substantial declines in SHS exposure over recent decades, Black children and adults, as well as individuals with lower

socioeconomic status (SES), remain significantly more likely to be exposed to SHS, compared to individuals of other races/ethnicities or with higher SES.[4] Likewise, SM adults, including gay, lesbian, and bisexual adults, are considerably more likely to smoke cigarettes, compared to heterosexual adults.[5]

In Chapter 2, we combined information on county-level smoke-free law coverage in workplaces and hospitality venues (restaurants and bars) with repeated cross-sectional biomarker data on SHS exposure in the NHANES survey, collected between 1999 and 2014. We examined the relationship between each type of smoke-free law coverage and the odds of SHS exposure in the full population, as well as in regression models stratified by age. We explored the potential contribution of smoke-free laws to disparities in SHS exposure among nonsmokers by examining effect modification by race/ethnicity, gender, education, and poverty-income-ratio (PIR). We found that smoke-free laws in hospitality venues were associated with lower probability of SHS exposure in the full NHANES sample, as well as in each age group (i.e., 25-39, 40-59, 60+). Workplace smoke-free laws, on the other hand, were associated with lower probability of SHS exposure for the youngest age group only (ages 25-39). Gender modified the association between both hospitality smoke-free laws (in the full sample and for ages 40-54) and workplace smoke-free laws (for ages 40-54) and SHS exposure, with stronger declines among males than females. Among adults ages 40-54, workplace smoke-free laws were associated with increased SHS exposure for individuals in the lowest PIR group, whereas associations were flat or declining for other PIR groups. We also found evidence of significant effect modification by survey wave, where associations between hospitality laws and lower levels of SHS exposure appeared to wane over time.

The results from this study suggest that hospitality smoke-free laws may have been an important driver of reduced SHS exposure among nonsmoking adults in the early 2000s. Our findings also suggest that both types of laws may have contributed to narrowing the differentials in SHS exposure between males and females. While the cross-sectional nature of this study limits our ability to make causal assertions about the effects of smoke-free laws on SHS exposure, differential associations between workplace laws and SHS exposure across levels of PIR warrant additional consideration in future research. If workplace smoke-free law coverage is indeed associated with higher levels of SHS exposure in low-PIR groups, this would be evidence that these laws have a negative equity impact. The significant interaction between hospitality laws and survey wave is also concerning, as it implies that these laws may not be as effective in the contemporary era as they have been in prior years. Taken in total, these results suggest that additional strategies, beyond smoke-free laws, may be needed to continue to reduce both levels of SHS exposure and disparities in SHS exposure by SES. For example, relatively new requirements surrounding smoke-free policies in public housing developments may more effectively address SES disparities in SHS exposure, though the impacts of these policies remain to be seen.[13]

In Chapter 3, we adopted a simulation modeling approach to estimate deaths attributable to SHS exposure between 2000 and 2016 and to project future trends in SHS exposure under different intervention scenarios. We calibrated models to nationally representative survey data and incorporated parameters specific to racial/ethnic and sex groups in order to examine disparities in SHS-attributable health outcomes and in SHS exposure. We found that SHS-attributable deaths from IHD and lung cancer have decreased substantially in the U.S. over time, from over 30,000 deaths in 2000, to less than 12,000 deaths in 2016. However, we also found

that Black adults have borne an increasingly disproportionate burden of SHS-attributable mortality, compared to other racial/ethnic groups. In projecting future trends, our model suggests that disparities between racial/ethnic groups will continue to decrease over time under a baseline scenario; however, Black adults are likely to remain at particularly high levels of exposure compared to other groups. Interventions weakening the association between smoking prevalence and SHS exposure appeared to be slightly more effective in reducing SHS exposure across all groups, compared to interventions targeting smoking cessation.

The implications of this study are multifold. First, our calculations of attributable mortality suggest that while SHS exposure remains a major public health issue in the U.S., widely cited estimates of the burden of SHS-attributable mortality are likely outdated given current levels of SHS exposure in the U.S. population.[6] This simulation study, which incorporates parameters specific to race/ethnicity and sex, serves as one case study among a small but growing number of simulation models that explicitly consider disparities in modeling tobacco-related health outcomes.[7]

Finally, in Chapter 4, we shift to examining the role of structural stigma in explaining the high risk of smoking among sexual minority adults. We adapted a previously validated measure of structural stigma that includes state-level information on policies related to discrimination on the basis of sexual orientation, the density of same-sex couple households, and public opinion towards same-sex marriage. We combined this index of structural stigma with individual-level data from the National Adult Tobacco Survey (NATS), 2012-2014. In adjusted models, we found that structural stigma was associated with smoking risk in a curvilinear fashion, with the lowest risk of smoking at the lowest and highest levels of structural stigma. This association was significant for SM and heterosexual adults, but the magnitude of the association was larger

among SM adults. Additional analysis of effect modification by sex suggested that the shape of the association was similar for SM males and females.

This study adds to prior work that has linked exposure to structural stigma to a range of poor health outcomes among sexual minorities; however, we build on this body of literature in a number of ways. First, we show that structural stigma remains significantly associated with smoking patterns among SM adults even after adjusting for correlated tobacco control environments. Second, we show that the association between structural stigma and smoking appears to be similar between SM males and females, which has not been previously explored. Finally, while prior studies have documented linear associations between structural stigma and health outcomes, we found evidence of a more nuanced, non-linear relationship. These results suggest that structural stigma continues to be an important explanatory factor underlying the high smoking prevalence in SM communities. They highlight the importance of considering stigma at all levels (individual, interpersonal, and structural) as a public health priority for reducing health disparities that affect SM populations.

Strengths and Limitations

Strengths. Strengths common to all studies included in this dissertation include the use of large, nationally representative datasets, which is advantageous for multiple reasons. First, the large samples provide adequate power to test effect modification by numerous sociodemographic characteristics. This is essential for a systematic examination of the relationship between policies/societal norms and disparities in tobacco-related health outcomes. Second, combining multiple waves of large, nationally representative surveys also allowed us to exploit substantial spatial and temporal heterogeneity in our exposures over a critical period encompassing

substantial changes to tobacco control environments in Chapter 2 (1999-2014) and structural stigma towards SM individuals in Chapter 4 (2012-2014).

There are a number of strengths associated with the analytic methods used in this dissertation as well. In Chapter 2, we reported results from effect modification analyses on both the additive and multiplicative scales, which is the recommended approach for nonlinear models.[8] Similarly, in Chapter 3, we reported trends in disparities on both relative and absolute scales, which provides a more complete picture of temporal changes disparities in SHS exposure, compared to studies that focus only on one scale (often the multiplicative).[9] Finally, the combination of methods employed in Chapters 2 and 3 (regression-based estimation and simulation modeling) provides an example of how policymakers can utilize complementary methodologies to gain insights into potential policy outcomes.

Limitations. There are also several limitations associated with the analyses included in this dissertation. The empirical studies in Chapters 2 and 4 were conducted with repeated cross-sectional data. Longitudinal survey data would provide a better foundation for establishing the temporal sequence of exposure and outcome and for making assertions regarding causal relationships. The exposures explored in this dissertation are exogenous measures, which bypass concerns about self-reported data but do not directly measure individual-level exposures. For example, we relied on summary measures of county-level smoke-free law coverage; however, each individual's exposure to these laws may depend on occupational and recreational patterns. Likewise, measures of structural stigma at the state-level may obscure sub-state level variability in environments. As with all observational studies, residual confounding could have affected results of empirical analyses. Additional limitations specific to each study are outlined in the relevant chapter.

Future Directions

In examining structural drivers of tobacco-related health outcomes, this dissertation focuses on one component in a multifactorial landscape of tobacco use disparities. We highlight the contributions of smoke-free laws and structural stigma in helping to explain disparities in SHS exposure and sexual orientation smoking disparities, respectively. Future research can build on this work, by exploring related substantive questions and employing alternative study designs and analytic strategies.

With regard to SHS exposure disparities, longitudinal studies of smoke-free laws and SHS exposure would help strengthen causal assertions regarding the contribution of these laws to heterogeneous patterns of SHS exposure in the U.S. This would require the use of panel data, which is not available in the NHANES survey. We focused on SHS exposure among adults, however, this research could easily be extended to include children. While smoke-free laws have been shown to be associated with decreases in SHS exposure among youth,[10] sociodemographic disparities in SHS exposure among children are particularly pronounced,[4] and the contribution of smoke-free laws to these disparities remains unclear. In addition, the results from our analysis should be compared with alternative regression modeling methods based on causal inference approaches. For example, a prior study comparing a repeated cross-sectional analysis with a difference-in-difference (DiD) design for examining associations between smoke-free laws and household smoking behavior found that the two methods yielded substantively different results.[11] Future studies should consider incorporating DiD approaches into evaluations of the effects of smoke-free laws or other tobacco control policies on smoking and SHS exposure outcomes.

The simulation model described in Chapter 3 also provides a foundation for exploring a number of additional questions related to disparities in SHS exposure. While our study focused

on race/ethnicity, future work could also incorporate parameters specific to SES, given well-documented disparities in SHS exposure by poverty level and occupation.[4,12] Incorporating measures of SES into simulation models of tobacco-related health outcomes is rare; however, *SimSmoke* models provide an example of modeling smoking disparities by income.[7] In addition, it would be helpful to consider the potential impact of additional intervention scenarios, including interventions targeted to high-prevalence groups. The modeled scenarios in Chapter 3 assume interventions are equally effective across the entire population. The simulations suggest that under these scenarios, racial/ethnic disparities in SHS exposure will persist. Future iterations of SHS exposure modeling could explore the impact of more targeted interventions. One example of such an intervention is the recently implemented Housing and Urban Development rule that requires public housing developments to implement smoke-free policies.[13] As this policy primarily affects lower-SES populations residing in public housing units, it has the potential to reduce disparities in SHS exposure by SES. In exploring different interventions, it is important to note that comprehensive frameworks for regulatory evaluation extend beyond the outcomes explored in this study to also include considerations of economic costs, burdens to individuals, and the distributional impacts of interventions across population groups.[14]

Our examination of structural stigma and smoking among SM adults points to several avenues of future research. A recent commentary argued that studies of structural stigma are extremely sensitive to measurement and analytic decisions, and that research must move beyond descriptive analysis to explore the mechanisms underlying the associations between structural stigma and health outcomes.[15] In line with this observation, the observed nonlinear association between stigma and smoking points to opportunities to explore nuances in the relationship between stigma and health outcomes, particularly related to resiliency in high-stigma

environments. These factors may include measures of social support, which have the potential to modify the effects of living in high-stigma states.[16–18] Given the high smoking prevalence among SM adults, studies that explore factors related to smoking cessation are also needed. As with other empirical analyses in this dissertation, longitudinal analyses would provide stronger evidence of causal relationships between structural stigma and smoking outcomes.

While the studies in this dissertation tend to focus on disparities characterized by a single sociodemographic factor (e.g., race/ethnicity or sexual minority status), there is a great deal of work to be done to incorporate an intersectional perspective into research on tobacco-related health disparities. Intersectional approaches acknowledge that individuals exist beyond an additive sum of their identities,[19,20] and that failing to examine behaviors and health outcomes at the intersections of sociodemographic characteristics may obscure important patterns of disparities. For example, simply examining smoking disparities between SM and heterosexual adults would fail to reveal important variations at the intersection of sexual minority status and sex, namely that SM females have the highest risk of smoking, compared to SM males and heterosexual males and females.[21] Work that considers drivers of tobacco-related health outcomes at the intersection of multiple sociodemographic factors is sorely needed.

Finally, throughout this dissertation we have alluded to a framework for understanding the contribution of societal/policy factors to tobacco-related health outcomes: through the lens of "differential exposure" and "differential vulnerability."[22] In our empirical analyses, we focus primarily on differential vulnerability, exploring effect modification of structural environmental factors by sociodemographic characteristics. However, recent research has focused more formally on decomposing the relative contributions of both differential exposure and differential vulnerability on chronic disease risk.[22] Adopting this analytic framework is an important next

step in more fully understanding the relationship between policies/societal norms and disparities in tobacco-related health outcomes, as it could inform the extent to which future efforts should focus on expanding access to existing policies versus developing strategies to bolster the effectiveness of interventions in vulnerable groups.

Conclusion

Collectively, the research in this dissertation provides evidence to policymakers regarding the relationship between policies/societal norms and persistent disparities in tobacco-related health outcomes. We find that smoke-free laws, while important tools for reducing SHS exposure, may be differentially associated with the probability of SHS exposure, depending on age, gender, and PIR. Furthermore, we find that structural stigma is a potential explanatory factor for the high rates of smoking among SM adults, but that the shape of this relationship may be more nuanced than previously described. During an era of unprecedented declines in smoking prevalence, results from these studies underscore the importance of incorporating considerations of health equity into analyses of tobacco-related health outcomes.

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