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THE SOCIAL PRODUCTION OF HEALTH INEQUALITIES ACROSS STATE AND REGIONAL CONTEXTS

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ABSTRACT

Our chances of living a long and healthy life are influenced by our position in intersecting social hierarchies, but also by our exposure to social policies. My research explores the policy mechanisms by which social inequalities in health are modifiable. In this dissertation, I draw on the Panel Study of Income Dynamics, the longest-running panel study in the U.S., and the National Social Life Health and Aging Project, a nationally representative, longitudinal survey of community-dwelling older adults. I combine these surveys with data on contextual factors to reveal that educational and regional inequalities in health and mortality can transform with shifts in state policy and regional context. The first chapter focuses on the sharp contrast between the South and the rest of the U.S. in health and mortality and examines regional trajectories to sort out when and how region influences health over the life course. The second chapter clarifies the potential for state cigarette taxes to moderate the educational gradient in mortality. In the third chapter, I make the case that advancing the study of health inequalities will require explicit study of the dynamics in fundamental causes through population comparison. I draw on examples from the literature to demonstrate how reframing fundamental causes as systems of exposure can reveal the dynamic nature of health inequality. By studying the extent to which health and mortality inequalities do indeed vary over state and regional contexts, I aim to provide evidence that health inequality is not an intractable problem, but rather something that we produce and maintain through social policy and social structures.

CHAPTER ONE

INTRODUCTION

Health embodies social inequality. It makes visible some of the most problematic, painful truths of social exclusion and exploitation in our society. Studies that document social disparities in health have been fundamental in the movement toward health equity. Evidence abounds showing that health exhibits a gradient across socioeconomic status. Similarly, there are dramatic and persistent gaps in health according to regional inequality in the U.S. Thanks to the work of many epidemiologists, sociologists, and demographers, talk of the "social determinants of health" has become mainstream. Physicians now acknowledge the roles that residential segregation and poverty play in exacerbating risk for death due to cancer or heart disease. So today's urgent questions are not *if* poverty and racism impact health negatively, but *how* we can intervene to protect opportunities for maximal health in the face of persistent socioeconomic and racial inequality. The present moment needs research that goes beyond demonstrating the existence of social disparities in health. It is time for researchers to investigate the modifiability of the disparities in health.

At the core of my dissertation research is the recognition that socioeconomic gradients and racial gaps in health are socially produced and maintained. Social stratification is a major driver of trends in health inequality. Social policies interact with the influence of stratification, sometimes maintaining or exacerbating health inequalities and sometimes dismantling them. My dissertation research takes up the challenge of investigating variation in the effects of the social causes of health inequality by looking across policy regimes and residential contexts. First, I consider how exposure to contextual disadvantages at the regional level can have lasting influence on health and mortality. Then, by studying how one of the most stubborn social

disparities in health (the educational gradient in mortality) varies over state context, I provide insight into the potential for state policy to reduce population health inequalities. Finally, I develop a theoretical intervention to advance research on the modifiability of social inequalities in health. In this introductory chapter, I review the literature that provides a basis for my research. Then I summarize the substantive chapters of the dissertation.

Contemporary Theories of Health Inequalities

Today, much of the research on the social determinants of health inequality remains atheoretical, ahistorical, and individualistic (Krieger 2008; Smedley and Myers 2014). Without historical context for health inequalities and without a population-level perspective, researchers continue to overestimate the role of individual behavior in generating health inequality (LaVeist 1994; Wilkinson and Pickett 2006). This is why we need more than just theories of the social determinants of health. Theories of the social production of health inequalities are also necessary (Mechanic 2002).

In this introductory chapter, I briefly review several theories of the social production of health inequalities. This is in no way an exhaustive review, but a starting point for thinking about the social processes that generate population health inequalities. My discussion is organized around four theoretical orientations cited in the literature on social disparities in health to which I refer as: a) susceptibility hypotheses; b) resource-distribution hypotheses; c) macro-structural hypotheses, and d) the life-course perspective. It is important to note that just because I consider the Weathering Hypothesis to be a susceptibility hypothesis and Fundamental Cause Theory to be a resource-distribution hypothesis does not mean they are incompatible with each other. To the contrary, I think most of these perspectives complement each other well and differ mainly in the factors that they prioritize. Overall, I think these theories all support the idea that the social

production of health inequalities involves a combination of macro-structural forces that shape the distribution of key social resources, which in turn influences susceptibility to disease and also exposure to illness and injury.

Susceptibility Hypotheses

Scientific study of the social production of health inequalities blossomed as an interdisciplinary field in the 1980s and 1990s (Muntaner and Navarro 2004) following the publication the Black Report in the UK in 1980 (Black 1982). But its roots go back well before that, at least to the dawn of social medicine in the mid-1800s (House 2016; Waitzkin 2005). Despite the long-held view that health inequalities mirror social stratification, skepticism persists in the broader medical community over the relative importance of social processes in generating patterns in health (House 2016). Perhaps it is the difficulty of conceptualizing pathways that link distal social processes to health that compels us to continue searching for biological and behavioral explanations for health inequalities.

In recent decades, theories of psychosocial causation of disease have developed as a way to counter biomedical explanations for health inequalities (Krieger 2005). The concept of differential susceptibility to disease is a common thread running through Barker's Fetal Origins Hypothesis (1990), Geronimus's Weathering Hypothesis (1992), and Engel's Biopsychosocial Model (1980), as well as foundational work by Cassel (1976) and Syme and Berkman (1976) on the psychosocial causation of susceptibility through stress pathways. Much scholarship has developed around the core idea that social stratification, marginalization, exclusion, discrimination, and racism generate health disparities by making some individuals more susceptible to poor health outcomes than others (Williams and Collins 1995). Some think this happens through exposures during critical periods of development such as pregnancy (Barker

1990) and early childhood (Hayward and Sheehan 2016); some think it results from insults and imbalances to the interconnections between the social, the psychological, and the physiological (Engel 1980); and, some think it happens through cumulative exposure to stress due to racial inequality (Geronimus 1992, 2000).

Barker (1990, 1995) put forward the hypothesis that there are critical periods for development, particularly the in-utero period from conception to birth, during which time the developing fetus is sensitive to environmental exposures, and extreme exposures can be consequential well into later life. Engel (1980) proposed a theory that integrates the social and psychological experiences, including stress, with the physiological health of the body. While foundational, Barker's and Engel's hypotheses do not specifically address health inequalities. So I will focus here on Geronimus's Weathering Hypothesis (1992). This hypothesis is related to other susceptibility hypotheses in its proposal of a mechanism of accelerated aging by which psychological and social stress due to racism can have negative health consequences.

Geronimus's Weathering Hypothesis (1992) conceptualizes a kind of susceptibility to poor health that results from cumulative stress due to racism and socioeconomic disadvantage. I say a "kind of susceptibility" because for Geronimus (1992), the result of cumulative socioeconomic disadvantage due to racial inequality is not so much susceptibility as it is accelerated aging. She frames her finding that the Black-White infant mortality gap varies by maternal age as evidence of accelerated health deterioration among Black women (Geronimus 1992). Still, because the weathering process is based on mechanisms of psychosocial stress, I think it fits as a theory of general susceptibility. References to the weathering hypothesis are pervasive (Hicken et al. 2012; Lukachko et al. 2014; Murray et al. 2006; Schulz et al. 2002; Wallace et al. 2015), but it has not been engaged and explicitly tested nearly as much as Link and

Phelan's (1995) Fundamental Cause Theory as an explanation for racial inequalities in health (Clouston et al. 2016; Daw 2015; Gee et al. 2012; Krieger 2013; Lutfey and Freese 2005; Masters et al. 2015; Montez et al. 2016; Phelan et al. 2004; Schulz et al. 2002). Perhaps this is because the mechanisms of embodiment and the biology of inequality are still controversial (Krieger 2005), whereas theories of differential exposure and response based in the unequal distribution of material resources impacting health are less so (Wilkinson and Picket 2006). Or, on the other hand, perhaps it is because there are so many variations of psychosocial stress-susceptibility hypotheses, and so much evidence emerging to support stress pathways to susceptibility that weathering has been subsumed into a more general cumulative stress hypothesis (Hayward et al. 2000). Regardless, Geronimus has made a major contribution through her emphasis on the *cumulative* health effects of social disadvantage (Geronimus 2000).

It is illuminating to remember the context in which many hypotheses of general susceptibility and cumulative stress mechanisms were developed—one in which cultural and behavioral explanations for racial health disparities were widespread (Kitano et al. 1992; LaVeist 1994). Thus, it is heartening to see that social science research has advanced in recent decades to avoid notions of cultural superiority and to acknowledge influence from structural inequality on culture (Allard and Small 2013; Desmond 2014; Lamont et al. 2014; Wacquant 2015), and also that mainstream research on health disparities now pursues the study of social structure and social equity (Diez Roux 2016; House 2016; LaVeist and Isaac 2013; Viruell-Fuentes et al. 2012).

Resource-Distribution Hypotheses

It is intuitive that social inequality and health inequality are linked, but why is this such a robust association? Fundamental Cause Theory (Link and Phelan 1995) provides a middle range

theory of *how* that link transcends specific disease mechanisms. Fundamental Cause Theory (FCT) is based on the idea that because social resources can be used to avoid disease and death, the unequal distribution of resources will exacerbate health inequalities in the face of efforts to improve health (Link and Phelan 1995). FCT posits that advantaged members of society have access to resources that they can direct to avoid disease and prolong life regardless of the specific threat. In contrast, individuals of lower socioeconomic position are not able to escape toxic or infectious exposures, work injuries, or the general toll of life in poverty or marginalization.

Fundamental Cause Theory (FCT) was so named by Link and Phelan in 1995, but it incorporated ideas presented by Lieberson (1985) and House and colleagues (1990) of basic causes that transcend specificity or the causes of causes (Rose 1992). The puzzle addressed by FCT is not simply, why do the poor live shorter and sicker lives than the rich, but why is this the case despite the work of health professionals to eliminate the main causes of sickness and death that disadvantage the poor? FCT stands out from other theories for its explicit puzzle and careful logic, which makes it highly generative of future research and application.

Despite the dramatic reduction or elimination of common causes of death that occurred over the Epidemiologic Transition, the marked socioeconomic gradient in mortality has been stubborn to change. Neither the introduction of vaccines and treatment for infectious causes of death, nor major improvements to infrastructure for sanitation, nor the steady increases in average life expectancy have resulted in any erasure of this simple fact: the poor live shorter, sicker lives than the rich. This is a social fact, in the Durkheimian sense, in that it is real only as a collective phenomenon that emerges from a society's norms, values, and social structural.

FCT critiques the tendency in medical sociology and social epidemiology to focus too much on specific mechanisms, which can become obsolete while the underlying relationship

with health persists. Instead it searches for fundamental causes that influence population health in ways that are non-disease-specific. FCT calls for attention to key social resources, including money, knowledge, power and prestige, and beneficial social connections, as they matter for health. Access to flexible social resources appears to be important at both the individual level and the contextual level (Daw 2015; Schulz et al. 2002). Using one's individual resources to secure residence in a high SES neighborhood then conveys a suite of additional contextual resources, from neighborhood safety to high quality infrastructure to low exposure to toxicity to social supports for healthy lifestyles. FCT posits that the deployment of social resources is critical to maintaining a health advantage. This assumes that the resources possessed by individuals of lower socioeconomic position are fewer and less flexible such that they cannot be used to secure health advantages in the face of changing lifestyles and changing risk factors.

Clouston and colleagues (2016) extended FCT by articulating a four-stage process through which the emergence of new diseases prompts medical innovations, which in turn lead to the creation of new health inequalities, and eventually, to the reduction of those health inequalities. According to the predictions under their "unnatural history of disease" model, any given disease will fall somewhere along the continuum of the four stages, and thus, will be either in a phase of expanding inequality, reducing inequality, or neutral (without inequality). And it is the compounding of this process across all diseases that leads to the apparent persistence of the socioeconomic gradient in health and mortality despite the demise of specific mechanisms (e.g., polio). They make the compelling point that when causes of a disease are unknown (e.g., primary brain cancer), access to resources to avoid disease are futile, and thus, we should not see socioeconomic status (SES) differences in incidence of death due to the disease.

It is important to note that FCT brings resources into the causal chain prior to susceptibility. Flexible social resources are thought to determine general susceptibility to disease incidence and death. In this sense, FCT is compatible with the susceptibility theories of health inequality.

Macro-Structural Hypotheses

Several scholars have developed theories that emphasize the role of macro-structural factors in shaping health inequalities. In what has become known as "The Gardener's Tale," Camara Jones (2000) uses allegory to reveal the structural injustice of racial inequality based in white supremacy. Jones connects this story of flowers trying to grow in soil of different quality and with differential treatment by the gardener, to a three-level framework for conceptualizing the influence of racism on health. She describes the potential for each level, internalized racism (i.e., acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth), personally-mediated racism (i.e., prejudice and discrimination), and institutional racism, to have detrimental health effects. She also goes further than most scholars of racial health disparities to say that "once institutionalized racism is addressed, the other levels may cure themselves over time" (2000:1214). In this sense, Jones emphasizes the primacy of structural/institutional racism in shaping racial health inequalities.

Drawing similarities to Fundamental Cause Theory (FCT), Jones (2000) discusses differential access to information as consequential for health, but she extends the scope of what is meant by differential access to information beyond FCT's narrow focus on access to medically-relevant information to include information about one's own history. Jones (2000) makes clear that she thinks the figurative gardener in her tale is government. Even asking the metaphoric question of "who is the gardener?" is a radical step, for few scholars of racial

inequalities go so far as to identify the powerful White actors who make the decisions (Feagin and Bennefield 2014).

Krieger (2005), in her Ecosocial Theory, agrees that government policy is a highly influential structural factor, but does not specify it as the most important one. Rather, Ecosocial Theory feels all-encompassing; it is both multileveled and intersectional, acknowledging spatial variation and life course variation. Despite its complexity, Krieger's theoretical work fits best as a macro-structural perspective because of statements like this: "Ever more evidence shows that the shorter, sicker lives of people burdened by economic deprivation, discrimination, noxious jobs, and environmental pollution result from injurious political priorities, not individual failure" (Krieger 2008:1102). This excerpt of a core proposition of Ecosocial Theory clarifies it even further: "Macro-level phenomena are more likely to drive and constrain meso- and microlevel phenomena than vice versa" (Krieger 2012:937). So Krieger and Jones seem to indicate that political priorities matter most for the work to eliminate health inequalities.

Metaphor is important for scientific thought—it is what makes theory stick. Krieger's Ecosocial Theory is so broad and complex that it is not well captured through metaphor, and thus is not easily applied by empirical researchers. But one of its constructs is metaphorical enough to be compelling and that is "embodiment." According to Krieger (2005:104), embodiment is the process by which we literally incorporate, biologically, in societal and ecological context, the material and social world we experience over the life course. Thus, social inequalities become translated into corporeal inequalities and population health inequalities. The embodiment construct is multilevel and not attached to any specific biological mechanism, but Krieger proposes five pathways through which people embody social inequality: 1) economic and social deprivation, including not having basic needs met; 2) toxic substances, pathogens, and hazardous

conditions at work, in the neighborhoods, and more generally; 3) social trauma, including institutional and interpersonal discrimination and violence, plus additional psychosocial stressors; 4) targeted marketing of commodities that can harm health (e.g., junk food, alcohol, and drugs); and 5) inadequate or degrading medical care (Krieger 2005:2).

Also built into current elaborations of Ecosocial Theory is the concept of susceptibility. According to Ecosocial Theory, there is a cumulative interplay of exposure, susceptibility, and resistance across the life course. In this sense, Ecosocial Theory is the most integrative of any of the perspectives I discuss here because it incorporates classical thinking on susceptibility and psychosocial mechanisms as well as structural influences based in material relationships. But it is not testable in the way that FCT is, and perhaps that is the reason why, despite both models being introduced in the mid-1990s, FCT seems to have a more general audience than Ecosocial Theory as an explanation of the social production of population health inequalities (Clouston et al. 2016; Daw 2015; Gee et al. 2012; Krieger 2013; Lutfey and Freese 2005; Masters et al. 2015; Montez et al. 2016; Phelan et al. 2004; Schulz et al. 2002).

Life Course Perspective

The life course perspective emphasizes timing of exposures, turning points in a lifetime, latency, stress proliferation, human agency, and linked lives—all sources of variation in individual health trajectories (Elder 1994, 1998; Gee et al. 2012). My theoretical approach in this dissertation aims to integrate the Life Course Perspective and Fundamental Cause Theory. From conception to death, individuals are exposed to the fundamental causes of health inequality. As Hayward and Sheehan (2016) concluded in a recent review, the body does not appear to forget. This intuition that exposures may be embodied through a process that takes time is central to various hypotheses of the social production of health inequality (Barker 1990; Geronimus 1992;

Kreiger 1994, 2005), but scholars are just beginning to connect it to Fundamental Cause Theory (Daw 2015). This is likely because few life course studies of health have investigated influence on multiple health domains (Hayward and Sheehan 2016). And while the health effects of social conditions are indeed multiple, what if there are life course trends such that early life exposures matter more than later life exposures?

Research suggests that early life (pregnancy, infancy, and the first two years) is a critical period during which the effects of poverty and poor health become embodied by the next generation in a way that may have lasting consequences (Langley-Evans 2004). But it is unclear whether early life health is causal in its effect on later life health disadvantages, or if it is associated with other social processes that maintain and reinforce health disadvantages over the life course. An alternative to the critical period hypotheses, such as Barker's fetal origins hypothesis, is the explanation that early life circumstances, such as family poverty, condition future access to opportunities and exposure to risks in ways that cascade to magnify the initial differential. Cumulative advantage/disadvantage theory posits that initial relative advantages in life, due to social location, are magnified through time in a way that leads to divergent trajectories (Palloni 2006). Still, that is only part of the story. Just as we are more than our present circumstances, we are also more than our circumstances at birth. Scholars are beginning to ask when contextual exposures matter most for health and mortality. New research suggests that while early life exposures matter more for chronic disease risk and general physical health in older adulthood, contemporaneous local context matters more for mortality risk (Montez et al. 2017).

In summary, there are dynamics in the effects of fundamental causes on health over the life course. Whether the vaccine for polio was administered before or after a particular cohort

reached adulthood will shape that cohort's distribution of health into later life. There are also dynamics in the formation and persistence of fundamental cause associations over the life course (Gee et al. 2012). For instance, Jackson and colleagues (1996) found the highest levels of physical and mental well-being experienced by Blacks during the period from 1979 to 1992 coincided with Jesse Jackson's 1988 presidential campaign. This as an example of how changes in America's racial climate can impact health through period effects. My dissertation considers these two kinds of variations in fundamental cause associations: 1) social change that disrupts the influence of a fundamental cause on health; and 2) social change that alters the fundamental social gradient itself.

The Dissertation

My dissertation research builds on the work of sociologists and epidemiologists cited above to study how the fundamental causes of health inequality vary over geographic context and over time in their influence on mortality. I work from the belief that we can gain insight into how to reduce and eliminate population health inequities by studying how seemingly fixed social differentials in health vary over policy environment. My aim is to contribute to understanding the modifiability of fundamental social causes of population health inequalities.

Chapter Two: The Differential Influence of Regional Context on Later Life Health and Mortality

Chapter Two examines regional disparities in later life health from a life course perspective. To sort out when and how region influences health over the life course, I focus on the sharp contrast between the South and the rest of the U.S. in health and mortality. I draw on data from the National Life Health and Aging Project (NSHAP), a nationally representative sample of community-dwelling older adults in the U.S., to estimate the differential risk of multiple health outcomes and mortality by regional trajectory. I find that older adults who leave

the South are worse off in multiple outcomes than those who stay. I also find evidence of a protective health effect of community cohesion and dense social networks for Southerners who stay in the South. My results suggest that regional trajectory influences health in later life through its associations with socioeconomic status, access to healthcare, and social rootedness.

Chapter Three: State Cigarette Taxes, Smoking, and Implications for the Educational Gradient in Mortality

Chapter Three of my dissertation explores the potential for state cigarette taxes to moderate the educational gradients in smoking and mortality. It is taken as a social fact that less educated people live sicker, shorter lives. But educational disparities in mortality in the U.S. have widened in recent decades. Concurrent with these changes, the U.S. has seen a divergence in state policies. In this evolving political landscape, state-level policy is increasingly consequential for population health. I explore variation over time and across states in cigarette taxes to demonstrate how dynamics in state policy can magnify or shrink disparities in smoking and mortality. My results suggest that higher state cigarette taxes weaken the educational gradient in mortality. While higher state cigarette tax is associated with a reduction in the magnitude of educational disparities in mortality, this appears to be driven, in part, by a direct mechanism through smoking behavior and, in part, by the indirect effect of overall progress in tobacco control.

Chapter Four: Advancing the Study of Health Inequality: Fundamental Causes as Systems of Exposure

Researchers agree that an individual's positions in systems of social stratification convey health benefits or burdens due to mechanisms that go beyond individual risk factors. There is, however, much still to uncover about these population-level mechanisms. In this chapter, I propose reframing fundamental causes as "systems of exposure" that exist as attributes of

populations defined at various spatiotemporal scales. By documenting the extent to which social gradients in health do indeed vary over spatiotemporal contexts, I offer an approach for studying how health inequalities are produced and maintained through electoral politics, local policy, and social structures.

Finally, in Chapter Five, I conclude by integrating the contributions of the three substantive chapters and providing recommendations for future research on the social production of health inequalities. Taken as a whole, my dissertation emphasizes that the social mechanisms that produce health inequalities depend on historical context, local culture and stratification, and local policy to have their effects. With these chapters, I aim to contribute to sociological research that uses trends in population health to demonstrate not only the consequences of social inequality, but also the possibility of transforming those consequences.

CHAPTER TWO: THE DIFFERENTIAL INFLUENCE OF REGIONAL CONTEXT ON LATER LIFE HEALTH AND MORTALITY

Introduction

The U.S. is characterized by marked regional disparities in health. The American South has worse general health status than other regions of the U.S., a trend that has held for decades. While the majority of the concentrated morbidity and mortality in the South presents in later life, research suggests that exposures earlier in life shape later life health (Glymour et al. 2008; Hoynes et al. 2016; Roseboom et al. 2006). Accordingly, the patterning of regional health disparities we see today may be shaped by regional contexts of the past. Regional context at birth is especially important for study because fetal and infant exposures may have lasting effects on adult health (Barker 1990; Roseboom et al. 2006). This paper examines regional disparities in later life health from a life course perspective. I aim to clarify when exposure to the American South is more influential for later life health and mortality: at birth or in later life. While I expect that an older adult's current region of residence constrains or supports their health behaviors, their socioeconomic position, their access to healthcare, and their social context, research suggests that regional context in early life may be just as influential. Building on findings about the importance of early life exposures for later life health, I ask: What can a comparison of regional trajectories reveal about when and how regional context shapes health and mortality?

To sort out when and how region influences health over the life course, I focus on the sharp contrast between the South and the rest of the U.S. in health and life expectancy. Studies using cross-sectional population data generally find the South is heavily burdened with sicker people living shorter lives. In the current study, I draw on survey data to compare regional trajectory groups. These groups approximate regional trajectories by linking region of birth to

region of residence in later life. I compare Southern Stayers (Born South, Live South) to Former Southerners (Born South, Live Non-South), Snowbirds (Born Non-South, Live South), and Non-Southern Stayers (Born Non-South, Live Non-South). I look for patterns in the consequences of these regional trajectories on the health of older adults. In doing so, I offer novel insights about the social mechanisms linking regional context and later life health.

Background

Regions of the U.S. differ in their economic base, their political system, their climate and topography, their demographic composition, and their cultural values, all of which have consequences for population health (Montez and Berkman 2013). Estimating the influence of region on later life health is complicated by the interplay of composition and context. For instance, the West has led the nation in high school completion, while the South has the lowest rates of high school completion and college attendance (Montez and Berkman 2013). Is this a difference of composition or context? It is challenging to say whether a regional trend results more from composition (aggregated characteristics of individual residents) or context (ecological effects of institutions, policy, and environment). It is usually some combination of both. Some regional trends that impact health are more clearly contextual effects. For example, the Northeast has the most progressive tax policy, including taxes that impact health behavior such as high cigarette taxes (Montez and Berkman 2013). Other examples of regional context that impact health are that the South has the lowest public expenditure per capita, and the South has lagged behind the Non-Southern states in Medicaid expansion (Montez and Berkman 2013). This study theorizes several contextual effects of region on later life health and mortality while acknowledging that these contextual influences have a basis in the history of a region, particularly in its demographic composition.

The American South

Look at any map of chronic disease prevalence or life expectancy in the U.S. and you will see it—a striking geographic disparity: the American South is bad for health. From obesity to cancer, from infant mortality to lack of health insurance, the South is burdened with higher rates of disease and premature death and less access to healthcare than the rest of the U.S. Southern health disadvantages are so persistent that they are among the factors that have shaped the distinctiveness of the region.

It must be said that the American South has been incomparably worse for the health of Black Southerners and Native Americans due to slavery, forced segregation and Jim Crow laws, forced relocations, lynchings and racialized violence, and ongoing structural racism. This is reflected in the health trends of generations of people of color. However, this paper focuses on White Southerners, a distinct group that may reveal lessons about regional influences on health and mortality.

The health disparities between Non-Hispanic Whites in the South and the rest of the U.S. cannot be understood without attention to historical context. When today's older adults were in early childhood, the South was plagued by disease. Malnutrition resulting from endemic malaria, hookworm, and pellagra had catastrophic influence on child development and worker productivity in the South up until the post-Depression period (Humphreys 2009). In fact, scholars have suggested that the heightened disease burden of the South helps explain why Southerners are stereotyped as lazy and stupid (Martin and Humphreys 2006).

Malaria, hookworm, and pellagra acted as a trifecta of debilitating illness in the South during the birth years of today's older adults. Due to the combination of climate, rural poverty, and the legacy of slavery, malaria ravaged the South until the 1920s. By the time the National

Social Life Health and Aging Project (NSHAP) cohort was born (on average in 1938), conditions had begun to improve. In the mid-1940s, the Centers for Disease Control successfully eradicated malaria by spraying DDT, but exposed 1 million Southern households to the insecticide. Hookworm was endemic in the American South in the first decades of the 1900s, though not in any other region (Brinkley 1995). Hookworm interferes with nutrient intake and causes anemia, fatigue, and often death. It affected nearly half of all Southerners in 1910 and declined subsequently, but certainly influenced the Southern born in the NSHAP cohort. For instance, 37 percent of schoolchildren in Alabama were positive for hookworm in 1937, and 17 percent were positive in 1954 (Humphreys 2009). Niacin deficiency and its manifestation as pellagra was another threat to healthy child development. Food fortification to combat the niacin deficiency that causes pellagra began in the South in 1938 (Humphreys 2009). The South subsequently saw a major drop in nutrient deficiency between 1946 and 1947 with the advent of food enrichment laws. (This coincides with the birth of the youngest respondents in the NSHAP cohort.) Even well-off Southern Whites were impacted by hookworm, pellagra, and malaria.

During the 1920s–40s, most White Southerners were rural farmers with little money, an inadequate diet, and no access to formal healthcare (Humphreys 2009). The South was far behind the rest of the nation in access to basic amenities, such as electricity. This was the backdrop of the Great Migration of White Southerners out of the South. Many White Southerners left farming for work opportunities outside of the South or to join the military. Much changed between 1935 and 1946 because of the New Deal and World War II. Eventually, with interventions in sanitation (flush toilets), food fortification, insecticide use, road building, and the general movement of people away from farmland to towns (Humphreys 2009), infectious disease decreased.

The Southerners in the NSHAP cohort lived through the transition from an agrarian economy dominated by cotton, to manufacturing, and then to service and tourism. The American South of 2010 is more urbanized and its material infrastructure has improved, but the relative poverty of the South persists, and the overall health profile is still worse than other regions in the U.S. (Goldhagen et al. 2005; Montez and Berkman 2014).

Regional Disparities in Health

It is not a surprise that the American South has the highest rates of obesity, hypertension, and diabetes among older adults of any region of the U.S. States. The lowest average life expectancy is concentrated in the South, with life expectancies two to four years less than the national average in 2010 (Montez et al. 2016). Analyses with cross-sectional data show that a robust negative association exists between Southern residence and morbidity and mortality (Bandeen-Roche 2015). Taking a longitudinal perspective, Ziembroski and Brieding (2006) found that the health risk of long-term Southern residence is cumulative and detectable in later life. In other words, the negative effects of Southern residence on health appear to build over the life course.

How we explain regional disparities in morbidity and mortality depends on our understanding of what causes chronic disease. Despite nearly a century of research linking early life exposures to chronic disease in later life, the dominant model used to explain chronic disease risk remains the lifestyle model, known for its emphasis on health behaviors. The lifestyle model posits that lifestyle factors throughout adulthood, such as eating habits, exercise habits, smoking and drinking, have the potential to increase or decrease one's risk of developing chronic diseases. By extension, the lifestyle model assumes that the greater burden of heart disease, obesity, and overall mortality that distinguishes the South from other regions of the U.S. stems from choices

made in adulthood. And it is hard to argue against such a model—how we live as adults surely influences our risk of developing certain diseases—but evidence is mounting that chronic diseases in adulthood are also spurred by exposures in early life (Ben-Shlomo and Kuh 2002; Langely-Evans 2004). According to the early-life origins hypothesis, hardship in early life is positively associated with heart disease in adulthood (Barker and Osmond 1986). This early-life origins hypothesis stresses the importance of nutrition and environmental exposures during fetal development and infancy. Thus, it may actually be regional context at birth that initiates geographic disparities in chronic conditions and, ultimately, mortality (Howard et al. 2010).

Questions remain as to whether the health disadvantages associated with the South emerge through cumulative exposures over time or through exposures during sensitive periods in early life or later life. The evidence to date is mixed. Some prior comparisons by regional trajectory suggest that the health disadvantages associated with exposure to the South are cumulative and uniform over the life course, while others point to the salience of early life exposures. Glymour and colleagues (2009) found that stroke mortality was independently associated with both birth and adult residence in the Southern states known as the "Stroke Belt," but the risk of stroke mortality was highest among those who lived in the Stroke Belt at both birth and adulthood (Glymour et al. 2009). These findings are consistent with the idea that exposure to the South has a uniform influence over the life course, but a follow-up study by Glymour and colleagues (2011) found that birth in a Stroke Belt state was significantly associated with increased risk of dementia mortality. They concluded that childhood exposure to geographic context is especially consequential for dementia mortality (Glymour et al. 2011). Looking at obesity, Zheng and Tumin (2015) found evidence that the influence of Southern exposure is patterned differently for men and women. Specifically, they found that women's

obesity is influenced by birth region, while men's obesity risk is not (Zheng and Tumin 2015). Finally, Lauderdale and colleagues (1998) found that geographic variation in hip fracture rates is best explained by regional context in early life, while region of current residence has little influence. Thus, there is motivation from the literature to consider the lasting influence of early life exposure on health outcomes, but it is likely that the way exposure to the South influences health varies by outcome.

Prior studies of the early-life origins hypothesis have used interregional migration to isolate the effect of regional context at particular stages in the life course (Glymour et al. 2011; Lauderdale et al. 1998; Valkonen 1987). I build on this tradition by comparing four regional trajectory groups and looking for patterns in the consequences of exposure to the South on multiple measures of health in later life.

Interregional Migration

There are two main migration flows that shaped the regional trajectories of the cohort of older Americans born between 1920 and 1947. First is the Great Migration from the South to cities in the Midwest and the West. Second is the amenity moves made by retirees from the North to destinations in the Sun Belt, particularly to Florida. I will briefly discuss each and then describe the common migration flows observable in the NSHAP sample.

The Great Migration of Whites from the South was a massive exodus of farmers from the rural South that began in the early 1900s. It had multiple phases. Initially, some families moved to escape the South, pushed out by extreme poverty and material deprivation. Others, particularly young men, were lured out of the South by working-class manufacturing jobs in the Non-South or military service. The second phase of the Great Migration peaked in the postwar period of the 1940s and 1950s (Gregory 1998). California was the primary destination of these Southern

White migrants (Gregory 1998). Labor force migration tends to happen during the transition to adulthood and during working ages. Hence, the White Former Southerners in the NSHAP sample may have made their South to Non-South moves with their families in childhood or during early adulthood as part of the Great Migration. An alternative possibility is that they moved around retirement age for amenities or to be closer to family, as I describe next.

Interregional migration in later life, around retirement, tends to be "amenity moves" made by healthy older adults from all over the nation to a few popular destinations that are often already well-known to the mover from previous visits. The migration of "Snowbirds" from the Northeast to the Sunbelt upon retirement is the classic amenity move. Florida is the leading destination for retired migrants from across the nation and has been since the 1960s (Bradley and Longino 2009). The older adults who make interregional amenity moves are healthier, better educated, and of higher SES than their counterparts who stay.

According to the life course model of later life migration, interregional migration in the latest stages of life tends to be "assistance-seeking moves" or "institutional moves" (Bradley and Longino 2009). Assistance-seeking moves are made in anticipation of or in response to a health concern, functional limitation, or lack of income, which results in the preference to be closer to caregiver support (Bradley and Longino 2009). Assistance-seeking moves are often made as part of a counterstream back to one's birth region. Institutional moves occur when an older adult can no longer live independently. However, this is not likely to be an important migration flow in this study. Because this study focuses on community-dwelling older adults, individuals living in institutions, such as nursing homes, are excluded from the sample. Rather, it is likely that Snowbirds are the majority of the Non-South→South regional trajectory group and that their amenity moves were made around retirement age.

I inspected the migration flows within the NSHAP sample. It is important to note that these flows are constructed from retrospective data that have been shaped by prior mortality selection. California is the top Non-Southern receiving state for Former Southerners, and the six most common South→Non-South migration flows are: AL to WA, KY to IN, MS to CA, OK to CA, TX to CA, and WV to MD. Florida is the top Southern state with Non-Southerners. The most common migration flow is NY to FL. PA to FL and MA to FL are also common, but there is migration to Florida from nearly all of the birth states represented in the NSHAP sample. There are geographic differences in the proportion of the sample that is living in their birth state. For example, in the NSHAP sample, only 6 percent of Florida residents were born in Florida, but 82 percent of Mississippi residents were born in Mississippi. The birth state/current state combinations in NSHAP show that the most common interstate trend is migration to a neighboring state.

Without more information, it is difficult to draw up hypotheses about the relative health status of the regional trajectory groups. Migrants tend to be healthier, better educated, and of higher socioeconomic status (SES) than their nonmigrant counterparts in the sending location (Markides and Coreil 1986). But this expectation of migrant health selectivity does not account for the unique historical circumstances that shaped interregional migration in the U.S. for this cohort. While the Non-South→South "Snowbirds" have a socially privileged profile, the South→Non-South "Former Southerners" may be a more heterogeneous group in terms of socioeconomic background.

Regional identity and Rootedness

Regions contribute to the construction of a person's identity in early life (Cuba and Hummon 1993). Regional identity can provide a sense of belonging through shared interests,

lifestyles, and values (Hernandez et al. 2007). In this sense, health behaviors can be closely tied to regional identity. Long-term residence and local social participation foster stronger place identity (Cuba and Hummon 1993). The South, in particular, has a strong place identity, not to mention a lasting marker: the Southern accent. The South "endows many southern residents with a suitable identity and a sense of attachment" (Cuba and Hummon 1993:114). Bradley and Longino acknowledge that "moving oneself physically to another community does not necessarily mean that one also moves emotionally…. There are some migrants who never put down roots but remain emotionally tied to their former communities" (Bradley and Longino 2009:326). This may be especially true of Former Southerners both because of the ways they maintain their Southern identity through lifestyle choices and because of the ways Southerness is stigmatized outside of the South.

Most White Former Southerners of the Great Migration achieved economic stability in their new destinations quickly. Examining data on household incomes and poverty status in 1969, Gregory (1998) provides evidence that White Former Southerners "were anything but a distressed, marginal population." Their poverty rate was the same as that of other Whites (11 percent) as was their average household income ($10,000) (Gregory 1998). If anything distinguished the Former Southerners from other Whites it was their slight overrepresentation in blue-collar jobs and in suburban communities instead of core cities or rural areas (Gregory 1998). Publications in the 1950s and 1960s reflect a consensus that the Former Southerners of the Great Migration assimilated into Non-Southern working-class culture (Gregory 1998; Killian 1985).

The cultural distinctiveness of the South may have diminished over time, but a recent study comparing attitudes of Southerners and Non-Southerners concluded that "the South

remains a distinctive cultural region" (Hurlbert 2016). Scholars have even argued that White Southern identity is more like an ethnic identity (Killian 1985). The cultural distinctiveness of White Southerners is based on shared values around family and community, religion, violence, and race relations. There is a sense of unity in White Southern racism, a central theme of the region's history. An additional sense of unity comes from the stigma that surrounds Southern identity. White Southerners see themselves as a marginalized group and hold a defiant attitude toward Non-Southern culture and its moral high ground (Killian 1985). Place identities, whether the identity of being a White Southerner or the identity of being a public housing resident, can be valorized or stigmatized depending on the cultural setting (Cuba and Hummon 1993). Former Southerners face cultural stigma in the Non-South where they are stereotyped as backward, racist, lazy, and trashy (Reed 1982). In this sense, White Southern identity carries a class penalty outside of the South. This may result from the "Southernization" of working-class jobs and, in turn, "Southernization" of working-class lifestyle and identity (Gregory 1998). The marginalization that Former Southerners face outside of the South may cause them to double down on their Southern ways (Cuba and Hummon 1993). This could encourage negative health behaviors. Applying Fundamental Cause Theory to this case, the class penalty experienced by Former Southerners may restrict flexible social resources, thus resulting in worse health across a range of outcomes (Link and Phelan 1995).

Regional trajectories have consequences for regional identity but also for social rootedness. Social rootedness appears to become more salient in later life and can be protective for health (York Cornwell and Cagney 2014). The influence of social rootedness has generally been studied using the concept of community social cohesion (York Cornwell and Cagney 2014). A large body of research shows that strong social networks, high community social cohesion,

and participation in one's residential community reduce loneliness and depression and are, in turn, protective for health (York Cornwell and Behler 2015; York Cornwell and Cagney 2014; Glymour et al. 2008). Research also shows that social isolation is associated with increased risk of mortality and chronic disease (Ertel et al. 2009). It follows that being uprooted from one's birth region may have negative health consequences in later life due to lower community social cohesion and increased social isolation. It is likely social rootedness influences multiple health outcomes through behavioral, psychosocial, and physiological mechanisms (Umberson and Karas Montez 2010). Further, to the extent that regional culture includes traditions around social interaction that promote strong social relationships, it should benefit health in later life. The ways that traditional Southern culture prioritizes family and local ties are likely to be health promoting. Also likely to be health promoting is living in a small town with low social turnover. Southern older adults are more likely to live in small towns, whereas Former Southerners and Non-Southerners are more likely to live in urban communities where there is less social stability. Thus, beyond the potential social advantages specific to Southern culture, the social rootedness that results from living in the same area one's whole life may convey health advantages, especially in later life.

Figure 1. Regional Trajectory Profiles in the NSHAP Sample

The Southern Stayers (South→South)
Southerners who stay do not experience southern identity as a class penalty the way the Former Southerners do. While they have more economic disadvantage in absolute terms and worse access to medical coverage and care (as evidenced by low medication usage despite medical need), they enjoy other advantages in terms of social context. They enjoy a consistency of regional culture as part of their identity. They enjoy the stability of the White South (though there was so much migration, it was common for White migrants to return—it was circulation more than migration). There is a magnetism to the South that draws its people back home to their communities and their way of life. All this is reflected in the high scores of perceived community support, and it contributes to lower rates of depression and loneliness in the Southern group.

The Former Southerners (South→Non-South)
The Southern-born Whites who end up in the Non-South live in more urban and diverse neighborhoods than the other trajectory groups. In addition, their neighbors are better educated and have higher earnings than the other trajectory groups. The Former Southerners themselves come from slightly disadvantaged backgrounds in terms of parental education, but are not socioeconomically disadvantaged as older adults. Still, it is possible that their White Southern identity persists and results in social disadvantages in terms of a class penalty that results in a more negative health behavior profile. Also, this group has the lowest levels of community social cohesion reflecting their regional uprootedness.

The Snowbirds (Non-South→South)
There is a distinctiveness of the Snowbird migrant flow, especially from New York to Florida. They tend to be very privileged. Their socioeconomic privilege is reflected in their health status. But they grew up in a different cultural environment than their Southern neighbors. The Snowbirds are later life movers. Their habits around health behaviors were set outside of the South. We see this reflected in different smoking histories and different hygiene levels.

The Non-Southern Stayers (Non-South→Non-South)
This is the largest and most heterogeneous group, including people from the Northeast, the Midwest, and the West. This group also includes regional migrants between the Non-Southern regions. Thus, it is very difficult to make any generalizations about this group.

Conceptual Framework

Health in older adulthood is shaped by health behaviors, socioeconomic status, social support, access to healthcare, local policy, and more. But these influences are primarily studied and theorized as contemporaneous exposure relationships. A life course perspective guides us to

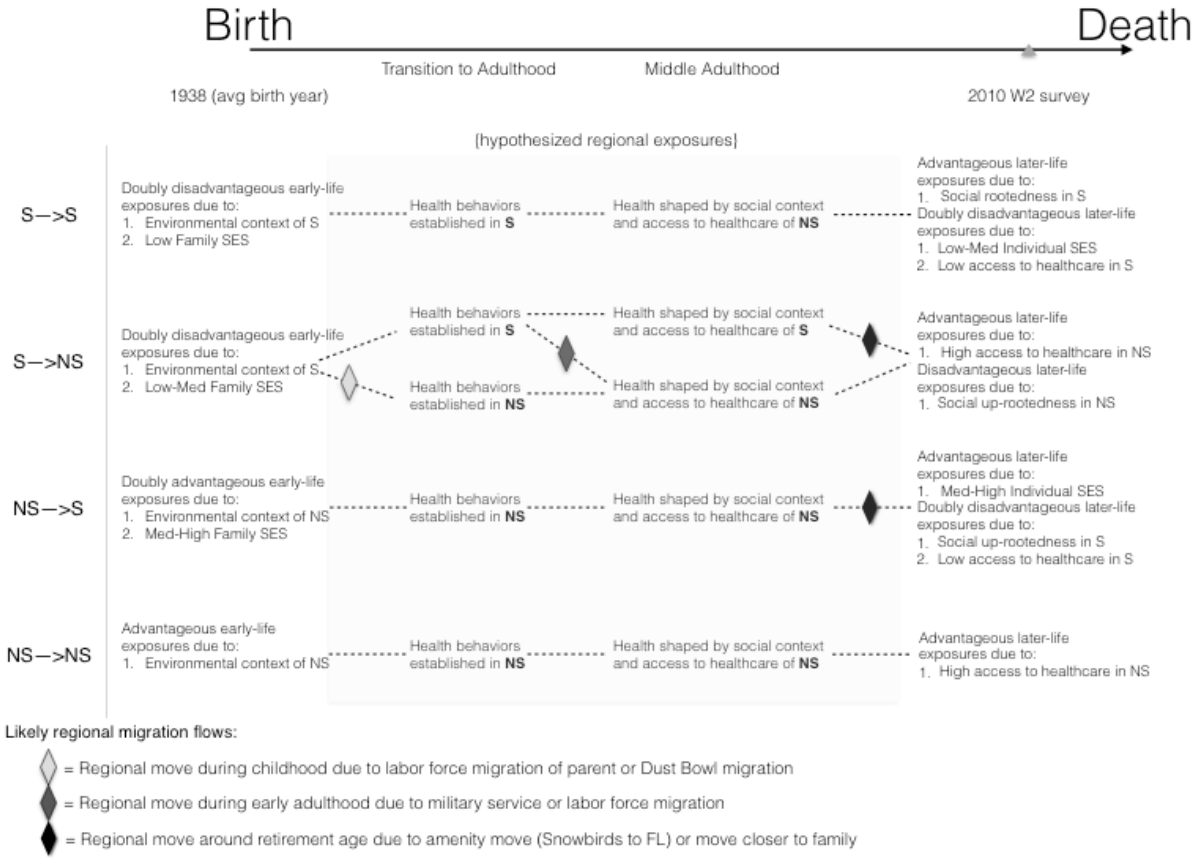
consider how later life health is part of a lifelong process of exposures and behaviors and access to health care. Older adult health can be conceptualized as a trajectory of exposures experienced over the life course that in turn correspond to an individual's health over the life course, often with long lags between an exposure and its detectable health consequences. Thus, by the time a particular birth cohort turns sixty-five, their health has been shaped by sixty-five years (plus nine months of gestation), each of which influenced them at a particular age.

This study is motivated by the novel idea that regional context may influence health and risk of death in varied ways over the life course and depending on regional trajectory. Certain aspects of regional context, particularly community social cohesion, may become more salient for health in later life. For example, the literature suggests that adults become increasingly sensitive to their residential environment as they age (Cagney and York Cornwell 2010; Glass and Balfour 2003). Later life is a time of increasing dependence on one's immediate local environment and social network. Other aspects of regional context, such as poverty and nutritional deprivation, will be more consequential when exposures occur in early life. In this sense, both later life (approximated by the last five years for older adults in this study) and the period from birth into early infancy can be thought of as "critical periods"—windows of sensitivity during which exposures can have undue influence on health trajectories.

The conceptual framework I developed for this study (See Figure 2) uses a life course perspective to integrate both the early-life origins hypothesis and the lifestyle model as plausible mechanisms by which regional context shapes health for the cohorts of Americans who are older adults today. While the lifestyle model leads us to focus on the ways that Southern culture and norms may constrain health-promoting behaviors during adulthood, the early-life origins hypothesis redirects us to consider how material conditions in the South during the 1920s, 1930s,

and 1940s may have constrained infant nutrition and development in ways that surface as health disadvantages in later life.

Figure 2. Conceptual Framework: Hypothesized Regional Trajectories and Exposures



This conceptual framework is not intended to be comprehensive.[1] Rather, it is tailored to relevant associations testable within the scope of the current study. I consider five domains of regional context likely to differentiate the health of White older adults:

[1] There are certainly other highly influential domains of regional context not included in this framework, such as state and local policy, and the framework could be extended to include additional domains in a future study.

1. Material conditions/Environmental context (Poverty, infrastructure, exposure to disease agents)
2. Family/Individual socioeconomic status
3. Norms and conditions constraining health behaviors
4. Access to healthcare
5. Social rootedness

Informed by the early-life origins hypothesis and the lifestyle model of chronic disease, I conceptualize influence from these five domains of regional context on health at four stages of the life course: 1) Early life; 2) Transition to adulthood; 3) Middle adulthood; and 4) Later life. The conceptual framework is organized around four possible regional trajectories: Born South−Live South; Born South−Live Non-South; Born Non-South−Live South; and Born Non-South−Live Non-South. The hypothesized associations with health (advantaged or disadvantaged) presented in the conceptual framework refer to expectations for the analytic sample of White, community-dwelling older adults with an average birth year of 1938 and are informed by historical trends and social science theory, as well as by descriptive statistics from the analytic sample (See Table 1).

Here are a few points about the hypothesized exposures to regional context listed in the conceptual framework. The South was the poorest region of the U.S. when the NSHAP cohort was born and is still. The South had the worst disease ecology and does still, despite the epidemiologic transition from infectious diseases to chronic diseases. And the South lacked infrastructure for sanitation, electricity, transportation, and even radios or air conditioning. These disease exposures and material conditions would have influenced nutrition and development of Southern-born individuals in line with the early-life origins hypothesis. These effects are most

likely to be detectable in global measures of health and mortality. There is also evidence that the geographic clustering of cardiovascular disease in the South, what is referred to as "the Stroke Belt," is driven in large part by exposures at birth and into adolescence (Howard et al. 2010). The early-life origins hypothesis suggests that early-life nutrition, and especially nutritional deprivation, can increase the risk of cardiovascular disease and obesity in later life (Barker and Osmond 1986; Langley-Evans 2004; Zheng and Tumin 2015).

In the realm of health behaviors, food and eating habits are a feature of regional culture, and this is particularly true in the American South. To the extent that regional norms promote smoking or consumption of high fat, high sugar, high salt, nutrient-poor foods, the South may pose a health disadvantage. Habits around health behaviors are often set during the transition to adulthood. For example, the average age of smoking initiation in the cohorts sampled for NSHAP was 17.4 to 18 years.

Finally, much of the South remains rural. Older adult Southerners are twice as likely to live outside of an urban center than older adults in the Northeast or the West. This is one way that living in the South may restrict access to quality health care. Further, the policy environment of Southern states has tended to provide reduced public benefits for health and less generous Medicaid and Medicare coverage relative to states in other regions (Montez and Berkman 2014).

It is important to note that the study does not observe respondents prior to older adulthood. Birth region is assessed through self-report. The conceptual framework reflects this lack of information about respondents' regional exposures between birth and later life, as well as the inability to ascribe directionality to the associations for some regional trends. This is why the Former Southerner trajectory includes multiple alternate pathways. The Snowbird trajectory does not include alternate pathways because there is enough information in the literature to

hypothesize that this migrant flow is dominated by amenity moves made around retirement age. Where there was not enough information to rule out an exposure or hypothesize the direction of an association, I omitted it from the conceptual framework.

The result is a conceptual framework in which regional trajectory's influence on health begins with regional variation in material conditions and family SES in early life and concludes with the influence of regional trajectory on individual SES, access to healthcare, and social rootedness in later life. In between, regional trends influence health behaviors set during the transition to adulthood, and regional context determines access to healthcare during adulthood. This conceptual tool proves useful for making sense of a very dynamic process of contextual influence on health.

Summary

This study compares regional trajectories to ask whether living in the South is more likely to be associated with worse health when the exposure happens in early life or in later life or both. The underlying motivation is to understand how the health disadvantages that characterize the South develop over the life course. I aim to advance our understanding of regional health disparities in the U.S. by integrating a life course perspective of exposure to regional context.

Data and Methods

The data come from the National Life Health and Aging Project (NSHAP), a nationally representative sample of community-dwelling older adults in the U.S. It is difficult to find complete data on place trajectories linked to health outcomes, but the NSHAP provides a good starting point to differentiate the effects of region of birth and current region of residence on later life health. I compared four regional trajectory groups in this nationally representative sample of older adults. The sample was restricted to non-Hispanic White respondents present in Wave 2 of

the survey in 2010 who had complete data on birth state and state of residence. My analytic sample consisted of 2,049 White older adults. I focused only on non-Hispanic Whites for three reasons. First, there was not enough regional variation in birthplace for Blacks and Latinos of this age cohort to study the differential influence of regional context on health at birth and in later life. Second, the regional experiences of this generation of Asian Americans and Native Americans are too different from those of White Americans to be grouped together, and yet there were too few older adults in the NSHAP sample to treat them as a distinct group. Third, regional context is likely experienced differently by non-Hispanic Whites because of their positioning as the dominant racial group.

Data were linked to the 2005 American Community Survey for census-tract characteristics. One of the unique features of the NSHAP data set is that it includes self-reports of health conditions, such as hypertension and diabetes, and it also includes biomeasures taken at the time of survey, such as blood pressure. NSHAP also collects a medication inventory for each respondent.

Outcomes of Interest

This study tested the effects of regional trajectory on three domains of health: Group A) Global Measures of Health and Mortality; Group B) Lifestyle and Health Behaviors; and Group C) Chronic Illness and Treatment.

Group A included global measures of health and well-being: 1) self-rated physical health (dichotomous; poor/fair health vs. good/very good/excellent); 2) functional limitation (dichotomous; at least one limitation in an Activity of Daily Living); 3) broken bone in the last five years (dichotomous); 4) pain in the past four weeks (dichotomous); and 5) 5-year mortality (dichotomous).

Group B included measures of health behaviors: 1) hygiene (continuous; interviewer-assessed with a scale from hygienic [1] to not hygienic [5]); 2) smoking (dichotomous; current smoker; and a three-category variable for never smoker, former smoker, or current smoker); 3) obesity (dichotomous; body mass index of 30 or greater); and 4) waist circumference (continuous).

Group C included chronic illness outcomes and measures of treatment: 1) hypertension (dichotomous; ever-diagnosed, from self-report); 2) uncontrolled hypertension (dichotomous; indicator for hypertension based on two blood pressure measurements at time of survey); 3) use of antihypertensive medication (dichotomous; from medication log); 4) diabetes (dichotomous; ever-diagnosed, from self-report); 5) dichotomous; indicator for high hemoglobin A1c, a biomeasure for diabetes measured from blood taken during survey); 6) use of an antidiabetic medication (dichotomous; from medication log); 6) depression (dichotomous; indicator based on Center for Epidemiologic Studies Depression scale); and 7) use of antidepressant medication (dichotomous; from medication log).

I also tested the effects of regional trajectory on measures related to social integration: 1) loneliness; 2) social network size; and 3) social network density. The social network variables come from data in NSHAP's social network roster in which respondents list the people they confide in and the connections of those named people to each other.

Primary Predictor Variable

The primary predictor variable used in the regression models is a four-category variable intended to capture consequential dimensions of regional context over the life course. While the U.S. is generally divided into four administrative regions (Northeast, Midwest, South, and West), this study explores what the literature suggests is the most salient regional pattern in U.S.

population health: the distinctiveness of the South from the other regions (Gregory 2006; Howard et al. 2010; Obisesan et al. 2000). The novelty of this paper comes not from its focus on the South per se, but from its combination of data on region of birth and region of current residences in order to construct a measure that approximates an older adult's regional trajectory over the life course. I use place of birth as an indirect measure of exposure and then can compare individuals based on their place of residence in later life. The regional trajectory variable is designed to facilitate comparison across the four possible birth region–current region pairings: 1) Southern birth and Southern current residence (S–S); 2) Southern birth and Non-Southern current residence (S–NS); 3) Non-Southern birth and Southern current residence (NS–S); 4) Non-Southern birth and Non-Southern current residence (NS–NS). In order to compare the relative influence of Southern birth and Southern current residence, the reference category in the regression models is always either Category 1–Individuals with Southern birth and Southern current residence or Category 2–Individuals with Southern birth and Non-Southern current residence.

This study design provides a general indication of the independent effects of regional context in early life on chronic disease in later life but is not appropriate for estimating specific causal effects. Rather, it is useful for hypothesis generation. Also, the study design makes a strong assumption that current residence appropriately classifies older adult exposure to regional context based on sufficient exposure to their current regional context. The data set used does not measure region of residence in the period between birth and the time of study (2005–2006). The residential mobility literature indicates that after a period of high mobility in early adulthood, residential mobility becomes much less frequent, and when moves do occur, they are most often local (Riley et al. 2016). The likelihood of a move increases slightly around retirement (Litwak

and Longino 1987). Bearing this in mind, I conducted sensitivity analyses restricting the analytic sample to respondents who had lived in their neighborhoods for at least five, ten, and twenty years. Using separate models for each sample, I did not find that conditioning on residential stability significantly altered the main results nor did introducing a dummy variable for retirement status to the models.

Of course, just as timing of exposure is consequential for health, so is duration of exposure. This study is not designed to test the health effects of cumulative exposure to living in the South. A challenge inherent to this study's design is that I do not observe residential trajectories between birth and later life. Still, we can safely assume that individuals born in the South and living in the South as older adults have had longer exposure to the South than those who were born elsewhere or who live elsewhere in later life.

Analytic Strategy

Depending on the health outcome measure modeled, I used either ordinary least squares regression or logistic regression to estimate the association between regional trajectory and the health outcomes of interest. I include dummies for the Former Southerner, Snowbird, and Non-Southerner regional trajectory groups and omit the Southerners (Born South, Live South) as the reference group in all models. All models adjusted for age (centered at 65), college education, and gender. All models applied Wave 2 survey weights to account for the complex survey design.

In addition to modeling the effect of regional trajectory using dummy variables for each group, I also ran the same models with an alternative specification. I estimated the independent effects of Born South and Live South and the interaction between Born South and Live South. Models from this alternative specification are shown in Appendix A (see Tables A-1 to A-4).

Testing the Conceptual Framework

To shed light on the plausible mechanisms linking regional trajectory to health and mortality, I inspected sample statistics for indicators of exposure that corresponded to the conceptual framework. These hypothesized exposures are grouped into early life exposures and late life exposures. For measures related to material conditions/environmental context and family socioeconomic status (SES) in early life, I looked at father's education and self-reports of health in childhood (ages 6 to 16) and family finances in childhood (ages 6 to 16). For insight into experiences of SES/social class in late life, I looked at self-reported income relative to other Americans, as well as two measures based on Census data: neighborhood poverty and neighborhood educational attainment. For insight into social rootedness in late life, I looked at network size, network density, and perceived community cohesion. I constructed the "perceived community cohesion" scale (adapted from Sampson et al. 1999) based on eight items (*1. How often do people in this area visit? 2. How often do people in this area do favors? 3. How often do people in this area ask for advice? 4. This is a close-knit area; 5. People in this area don't get along [inverse]; 6. People in this area don't share the same values [inverse]; 7. People in this area can be trusted; 8. People in this area are afraid at night [inverse]*.) A higher score indicates greater collective efficacy, trust, and interconnectedness—stronger social fabric. Finally, to approximate access to healthcare in late life, I looked at self-reports of the number of visits to a doctor in the past year.

Robustness Checks

I did several robustness checks to confirm the consistency of my findings across different model specifications. I ran all of the models without survey weights, but still with robust standard errors. I also inspected gender-stratified versions of all of the models. Finally, I ran all

of the models using OLS regression instead of logistic regression. There were no substantive differences in the conclusions.

A limitation of my study design is that I constructed the regional trajectory groups retrospectively, based on the regions of birth of individuals who have survived to older adulthood. Although I employ a nationally representative sample of older adults in the U.S., the sample suffers from mortality selection. In other words, there was unobserved mortality prior to 2010 which shapes membership in each of the regional trajectory groups, and this could bias the health profiles of each group. Of particular concern is the likelihood of higher mortality selection action on the Southerners group (Born South, Live South) such that some of the sickest individuals may have died prior to reaching older adulthood. To assess the potential magnitude of this mortality selection on my results, I conducted two separate analyses. First, I used individual characteristics measured very early in life—in theory, prior to any interregional migration—to predict membership in the regional trajectory groups. Multinomial logistic regression comparing the four regional trajectory groups shows that the Southern Stayer and the Former Southerner groups are less likely to have completed high school than the Non-Southerners. In general, though, these models did not tell a clear story of how background characteristics predict regional trajectory membership. Second, I drew on the Panel Study of Income Dynamics to estimate the potential magnitude of the premature mortality differential between the Southerners (S-S) and the Former Southerners (S-NS). I started with the original nationally representative sample of U.S. families in 1968 and restricted my sample to White, male, household heads aged 21-48 years in 1968 to match my analytic sample. I created S-S and S-NS regional trajectory groups based on "region grew up" and current region in 1968. I ran regression models to predict the differential odds of dying between 1968 and 2009. I found that

18.6 percent of the Southerners (S-S) and 13.5 percent of the Former Southerners (S-NS) died in the 1969-2009 window. Applying these numbers to my analytic NSHAP sample, I estimate that the equivalent of 22.7 percent of my analytic sample of White Southerner men are likely missing due to premature mortality, while the equivalent of 15.1 percent of my analytic sample of Former Southerners are likely missing due to premature mortality. This could make the Southern Stayers group healthier, on average, than they would have been without the effect of selective mortality. Bias due to selective mortality among women is much less of concern. The effect of mortality selection is not so much a threat to inference about the health of the current population of older adults, but it should be considered when interpreting the results on the relative influence of regional context on health.

Results

Despite expecting to find that longer exposure to the South translated into worse health across measures, I found that older adults who leave the South are worse off in multiple outcomes than those who stay (broken bones, pain, hygiene, obesity, antihypertensive medication, and depression). Some trajectory differences are only present for men and not for women (i.e., waist circumference) or for women and not for men (e.g., antidepressant medications), but there is an overall tendency for the Former Southerners (S-NS) to be at least as disadvantaged in terms of health outcomes as their Southern-born counterparts, often more so.

Table 1. Descriptive Statistics of Estimation Sample

		Born South Lives South	Born South Lives Non-South	Born Non-South Lives South	Born Non-South Lives Non-South
	Range	Mean / Prop	Mean / Prop	Mean / Prop	Mean / Prop
Total (n=2049)*		.20	.04	.10	.66
Female	0 or 1	.553	.465	.490	.541
Age	46 to 93	72.3	72.6	72.0	72.7
Post-High School Education	0 or 1	.511	.600	.657	.594
Father's Education Category (1=no schooling, 6=BA+)	1 to 6	2.68	2.99	3.03	3.05
Childhood SES Category (1=not well off, 5=very well off)	1 to 5	2.44	2.43	2.66	2.50
Childhood Health (1=poor, 5=excellent)	1 to 5	4.04	4.04	4.08	4.08
Marital Status					
Married/Cohabiting	0 or 1	.715	.837	.789	.748
Separated	0 or 1	.055	.023	.078	.063
Widowed	0 or 1	.215	.128	.118	.117
Never Married	0 or 1	.015	.012	.015	.019
Living Alone	0 or 1	.228	.140	.158	.218
Military Service (Males only)	0 or 1	.635	.778	.677	.617
Rural	0 or 1	.438	.186	.343	.237
Medicaid Coverage	0 or 1	.058	.133	.082	.097
Household Earnings	$0 to $1,500,000	$56,748	$75,923	$74,056	$67,368
Relative Household Income (1=Far below average, 5=Far above average)	1 to 5	2.78	3.04	3.18	2.96
Residential Tenure (1=Less than 1 year, 4= 11 to 15 years, 8=More than 50 years)	1 to 8	5.48	4.73	4.37	5.61
Moved in last 5 years	0 or 1	.202	.296	.326	.240
Relative Status of Home (Interviewer assessed: 1=Far below average, 5=Far above average)	1 to 5	3.28	3.09	3.31	3.24
Local Poverty (Census Tract)	.003 to .75	.168	.095	.120	.091

* Sample only includes Wave 2 respondents who self-identified as White and non-Hispanic, and who were not missing data on birth state and state of current residence.

Looking across multiple health outcomes and backed up by descriptive statistics, the regression coefficients tell a clear story of health disadvantages associated with Southern origins, and particularly with residence outside of the South in later life. The generally poor health profile of the Former Southerners is paradoxical because the Former Southerners live in better neighborhoods and are of higher socioeconomic status than the Southern Stayers (S-S) and the Non-Southern Stayers (NS-NS).

I present the multiple regression results in three parts reflecting the three groups of health outcomes evaluated in this study. First, as shown in Table 2, I tested for differences by regional trajectory in the odds of several well-studied global measures of health and well-being: self-rated physical health (SRPH), functional limitation in the activities of daily living (ADL), broken bones, pain, and 5-year mortality. A primary aim of this study was to leverage the consistent health disparities between the South and other regions of the U.S. to compare the relative influence of being born in the South to residing in the South in later life. I found no significant differences by regional trajectory for poor self-rated health or functional limitation. The Former Southerners are more likely to have broken a bone in the last five years. This difference is most pronounced in comparison to the Snowbirds. The Former Southerners are also more likely than Southern Stayers to have experienced pain in the past four weeks as indicated by the significant interaction term between Born South and Live South. Interestingly, both Southern birth and Southern residence in later life have independent positive associations with pain, but remaining in the South from birth weakens those associations. When it comes to mortality, there are no statistically significant differences by regional trajectory. Looking at just the coefficients, it appears that the Former Southerners may suffer a mortality disadvantage, but the sample size is too small to confirm the trend.

Table 3 shows results for outcomes closely linked to health behavior. Hygiene is an important and understudied health behavior with consequences for later life mortality. In fact, in this sample, interviewer-assessed hygiene is more predictive of 5-year mortality than self-rated physical health. Here, a lower value indicates better hygiene. The two trajectory groups living currently in the Non-South have worse hygiene, as perceived by the interviewers, than the Southern group (S-S). This may be an example of regional culture shaping norms and, in turn, shaping health.

Southerners are more likely to be never smokers than any other group, but they also have the highest prevalence of current smoking. While smoking prevalence is also high among Former Southerners, they are nearly twice as likely to be a former smoker than the Southerners. These results may reflect the way today's older adults experienced changes in regional trends in smoking behavior over the last several decades. When the cohort under study was young, smoking initiation was high in the U.S., but smoking prevalence decreased in the Non-South at a faster pace than it did in the South (CDC 2014). The South today has fewer policy incentives to quit smoking. The low likelihood of being a former smoker in the Southern group may also reflect mortality selection that occurred prior to entry into the sample.

Surprisingly, Former Southerners and Non-Southerners are more likely to be obese than Southerners. The Snowbirds as a group are indistinguishable from their Southern-born neighbors in terms of obesity. Also contrary to regional stereotypes, waist circumference singles out the Former Southerner men from men in the other groups. Among men only, the Former Southerners have a 2.5 inches larger waist circumference than the Southerners. Higher waist circumference is strongly associated with functional limitations in this older adult sample, such that a two-inch difference in waist circumference for white men is associated with a 20 percent increase in the

odds of having at least one ADL limitation. There are no differences in waist circumference by regional trajectory for women.

As shown in Table 4, I also looked at three chronic illness outcomes: diabetes, hypertension, and depression. Data from self-report of diagnoses shows no difference in hypertension by regional trajectory. Data from direct measurements of blood pressure show that Former Southerners are significantly less likely to have uncontrolled hypertension than Southerners. In fact, Southern (S-S) men were 2.2 times more likely and Southern (S-S) women 2.8 times more likely to have uncontrolled hypertension than their Former Southerner (S-NS) counterparts. The low, uncontrolled hypertension among Former Southerners appears to be due to their increased utilization of antihypertensive medications, but the differences are not statistically significant. Snowbirds are less likely than Southerners to have been diagnosed with diabetes, but this may indicate some under-diagnosis because they appear more likely to have elevated hemoglobin A1c levels (a biomeasure for diabetes) and less likely to be using an antidiabetic medication. Former Southerners also appear to have higher utilization of an antidiabetic medication than Southerners, though not statistically significant. These results are consistent with the expectation of better access to health care and medication coverage in the Non-South. The triangulation of diagnosis, uncontrolled disease, and treatment can be seen clearly in Figure 3. In Figure 3, a negative coefficient suggests a protective effect of staying in the South and a positive coefficient suggests a protective effect of leaving the South.

The results for depression highlight a strong disadvantage experienced by the Former Southerners. Former Southerners are over twice as likely as Southerners to be depressed, as measured with the Center for Epidemiologic Studies Depression scale. Risk of depression is also increased for Non-Southerners, but it is not nearly as high as the risk for Former Southerners.

This result is in spite of the fact that Former Southerners are significantly more likely than the Snowbirds to be taking antidepressant medication. Even with their higher levels of antidepressant use, the Former Southerners are much more likely to be depressed than the Southerners.

Table 2. Global Measures of Health and Mortality

	(1)	(2)	(3)	(4)	(5)
	Poor health (self-rated physical health)	Any ADL Disability	Broken bone in past 5 years	Pain in past 4 weeks	5-year Mortality
Born South, Live South	ref	ref	ref	ref	ref
Born South, Live Non-South	0.753	0.774	1.577	1.456	1.087
	(0.428)	(0.486)	(0.284)	(0.185)	(0.816)
Born Non-South, Live South	0.686	0.924	0.720	1.389	0.700
	(0.206)	(0.790)	(0.276)	(0.107)	(0.197)
Born Non-South, Live Non-South	0.818	0.950	1.007	0.884	0.886
	(0.259)	(0.744)	(0.971)	(0.416)	(0.510)
Observations	3017	3019	2896	2793	2997

Models control for age, gender, and college education. Estimated with survey weights. Coefficients reported as odds ratios. Standard errors in parentheses. *^ p<0.10 * p<0.05 **p<0.01 ***p<0.001*

Table 3. Health Behaviors

	(1) Hygiene	(2) Current Smoker	(3) Obese	(4) Waist Circumference
Born South, Live South	ref	ref	ref	ref
Born South, Live Non-South	**1.351^**	0.531	1.471	2.013
	(0.059)	(0.245)	(0.264)	(0.425)
Born Non-South, Live South	0.919	0.846	0.791	0.587
	(0.287)	(0.542)	(0.373)	(0.428)
Born Non-South, Live Non-South	**1.142^**	0.832	1.050	0.860
	(0.077)	(0.277)	(0.810)	(0.786)
Observations	3014	3020	2921	2980

Models control for age, gender, and college education. Estimated with survey weights. Coefficients reported as odds ratios in Models 2 and 3. Standard errors in parentheses. *^p<0.10 * p<0.05 **p<0.01 ***p<0.001*

Table 4. Chronic Illness and Treatment

	(1) Hypertension Diagnosed	(2) Hypertension Uncontrolled	(3) Anti-hypertensive Medication	(4) Diabetes Diagnosed	(5) Diabetes Uncontrolled	(6) Antidiabetic Medication	(7) Depression	(8) Antidepressant Medication
Born South, Live South	ref	ref	ref	ref	ref	ref	ref	ref
Born South, Live Non-South	1.134	**0.399*** **	1.409	0.782	1.138	0.681	**2.290**	1.456
	(0.592)	(0.000)	(0.400)	(0.314)	(0.736)	(0.338)	(0.007)	(0.323)
Born Non-South, Live South	1.190	0.740	0.907	0.644	**1.882^**	0.606	1.234	**0.615***
	(0.394)	(0.149)	(0.730)	(0.156)	(0.058)	(0.118)	(0.550)	(0.046)
Born Non-South, Live Non-South	1.081	0.835	0.813	0.832	1.147	**0.671***	**1.380***	**0.754+**
	(0.581)	(0.166)	(0.416)	(0.186)	(0.499)	(0.033)	(0.033)	(0.058)
Observations	3015	2947	2995	3016	3020	2995	3007	2995

Models control for age, gender, and college education. Estimated with survey weights. Coefficients reported as odds ratios.
Standard errors in parentheses. ^ p<0.10 * p<0.05 **p<0.01

Figure 3. Chronic Illness and Treatment Interaction Terms Plot

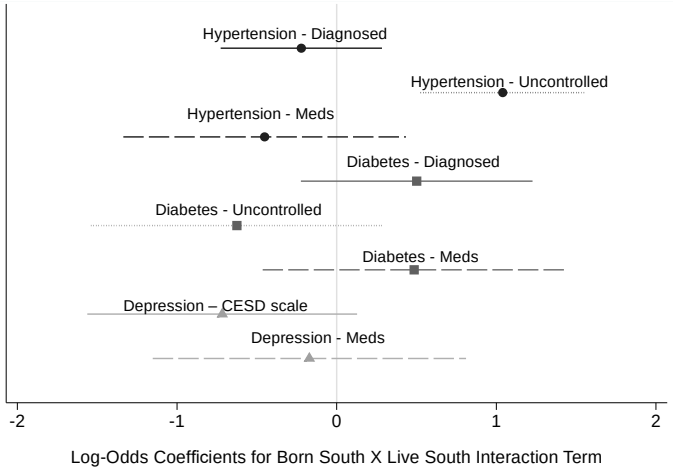


Table 5. Social Integration

	(1)	(2)	(3)
	Loneliness	Network Size	Network Density
Born South, Live South	ref	ref	ref
Born South, Live Non-South	0.118	0.088	**-0.043**^
	(0.225)	(0.688)	(0.094)
Born Non-South, Live South	0.033	0.107	**-0.052**^
	(0.615)	(0.536)	(0.058)
Born Non-South, Live Non-South	**0.131****	**0.287***	**-0.042***
	(0.002)	(0.048)	(0.019)
Observations	2976	3016	2933

Models control for age, gender, and college education. Estimated with survey weights. Standard errors in parentheses. *^p<0.10 * p<0.05 **p<0.01 ***p<0.001*

Table 6. Early and Late Life Exposures by Regional Trajectory

Regional Trajectory Group	Early Life Exposures			Later Life Exposures						
	Childhood Health: Poor/Fair *(proportion)*	Below Avg Family Finances in Childhood *(proportion)*	Father's Education: HS or less *(proportion)*	Below Avg Income Relative to Other Americans *(proportion)*	Network Size *(mean)*	Network Density *(proportion)*	Perceived Community Cohesion *(proportion)*	Local Population in Poverty *(proportion)*	Local Population with BA or more *(proportion)*	Number of Visits to Doctor in Past Year *(mean)*
S-->S	.06	.47	.80	.34	4.60	.73	.51	.17	.29	3.13
S-->NS	.05	.53	.72	.26	4.58	.70	.41	.09	.43	3.28
NS-->S	.07	.40	.71	.22	4.69	.67	.48	.12	.37	2.86
NS-->NS	.06	.45	.72	.28	4.77	.68	.52	.09	.39	3.02

The Southern Stayer group appears to have less depression and loneliness than others, though only marginally statistically significant. This is consistent with previously cited evidence that particularly in later life, social integration matters. Could it be that the social fabric of communities in the South buffers the negative health consequences of lower healthcare access and socioeconomic disadvantage? To investigate this further, I modeled social network size and density by regional trajectory. I found that Southerners have the smallest networks in terms of size, but their social networks are the most dense. This high social network density may contribute to their relatively low levels of loneliness. Next, I compared the average level of community cohesion by regional trajectory group and found that the Former Southerners are indeed disadvantaged in terms of community cohesion. Average community cohesion scores for the Southerner and Non-Southerner groups were 0.51 and 0.52, respectively. The Snowbird group was close with 0.48, but the Former Southerner had an average community support score of 0.41.

As shown in Table 6, I inspected additional sample statistics for measures of exposure that corresponded to the conceptual framework. Former Southerners in the sample were more likely to report below-average family finances in childhood, yet they look more like the Non-Southern born in terms of father's education. The data on relative income, neighborhood poverty, and neighborhood educational attainment do not suggest that Former Southerners are disadvantaged in terms of SES. Additionally, Former Southerners reported the highest access to healthcare.

Discussion

This study employed data from a nationally representative sample of older adults to make two main contributions to research on regional contextual influences on health in the U.S. The two major contributions are theoretical. First, my study urges consideration of the ways that regional trajectory is more than the aggregation of regional exposures over the life course. Especially for older adults, regional trajectory is an important part of one's personal identity. Regional trajectory may influence health indirectly by acting as an indicator for social class, thus affecting health behaviors, lifestyle, and social integration. Second, I connect regional trajectory to the concept of social rootedness—the way that residential stability facilitates social integration. Regional movers may miss out on the health benefits of social rootedness that become salient in later life. Further, aspects of regional culture, in this case Southern culture, may promote social rootedness in later life. In the remainder of this section, I discuss these theoretical contributions in more detail as they relate to my findings.

My results suggest that older adults born in the South and living outside of the South in later life experience multiple health disadvantages compared to both Non-Southern Stayers and Southern Stayers. Contrary to the expectation that interregional migrants would be healthier than stayers, I found that compared to the Southern Stayers, the Former Southerners are more likely to be obese, have broken a bone in the last five years, have pain, be taking antihypertensive medication, have worse hygiene, and suffer from depression and loneliness. Based on trends in population health, I expected the Southern Stayers to be the least healthy trajectory group. This was the case for smoking and uncontrolled hypertension, but for the many other outcomes tested, the Former Southerners were as disadvantaged or more disadvantaged than the Southern Stayers. While the early-life origins hypothesis helps explain why Southern Stayers and Former

Southerners suffer similar health disadvantages and have worse health than migrants who moved to the South, it does not explain why Former Southerners would have worse health across multiple outcomes than Southerners.

There are three possible explanations for the puzzling poor health profile of Former Southerners. First, mortality selection has shaped the composition of the regional trajectory groups I compare in this study. The Southern Stayers may appear healthier in part because the sickest members of this group died prior to older adulthood. While this helps us understand why the Southern Stayer trajectory group might appear healthier than regional statistics on disease prevalence would suggest, this does not fully explain why the Former Southerners would be worse off than the Non-Southern Stayers or the Snowbirds. To understand this further, we can draw on the conceptual framework.

Southern origins may have a negative effect on health through nonbiological mechanisms, mainly through the way Southerness is stigmatized in the Non-South. Thus, the second possible explanation is that the health disadvantages experienced by Former Southerners living in the Non-South reflect a class penalty associated with a White Southern rural identity. In this sense, regional context in early life may be more consequential for health in later life not only through its impact on fetal development, but because of the way it shapes individual identity, particularly social class. For the Former Southerners, a class penalty from growing up Southern is activated when they leave the South and move to a Northern or Western city, particularly to more diverse, urban settings. I propose that White Southern identity impacts health by acting as an indicator of lower social class outside of the South. As such, it may constrain lifestyle and health behaviors and hinder a sense of belonging. Even for college-educated Former Southerners, an educational credential may not convey the same social class

benefit in cities outside of the South because a greater percentage of their neighbors have a bachelor's degree or higher.

If we do not consider regional trajectory as a dimension of social class, it is difficult to make sense of why the Former Southerners have a lower perception of community cohesion in light of their neighborhood characteristics. According to the Census data, the Former Southerners are living in neighborhoods that have less poverty, more people with college degrees, higher home values, and higher median household incomes than respondents in the other regional trajectory groups. These neighborhood characteristics are generally thought to be desirable and health promoting. The idea that Former Southerners carry a class penalty for White Former Southerners helps explain why Former Southerners may experience loneliness in their residential destinations outside of the South. The Former Southerners are living in more urban communities, with less poverty and more educated neighbors than they would have experienced had they stayed in the South. Paradoxically, these signs of community-level privilege may exacerbate their individual-level socioeconomic disadvantage as well as their sense of isolation.

It may also be the case that neighborhood socioeconomic status is not a worthy substitute for a strong local social fabric. Southern Stayers seem to enjoy a social rootedness, whereas the Former Southerners have been uprooted and seem to suffer the consequences of this social uprootedness in their health. Thus, a third conceivable contributor to the disadvantaged health profile of the Former Southerners is that they missed out on health benefits of social rootedness. This relates to the Repotting Hypothesis, which proposes that residential mobility reduces the ability to plant deep enough roots to nurture social integration in the new community (Putnam 2000). The Former Southerners have been "repotted" in the Non-South, and as a result, they forfeit the benefits of provincial Southern culture and White social privilege.

The concept of social rootedness helps us make sense of the poor health profile of the Former Southerners. It is also helpful for understanding the relatively good health profile of the Southern Stayers. When we compare by regional trajectory, the group of Southerners who are living in the South in later life enjoy more health advantages than the literature would suggest. For example, when we think of the "Stroke Belt," we tend to assume these health disadvantages are a result of exposure to contemporaneous regional culture and policy environment. Yet I found Southern Stayers are less obese, less disadvantaged in certain global measures of health, and suffer from less depression and loneliness than other regional trajectory groups.

Why might staying in the South be protective for those born in the South? Again, it seems social rootedness is key. We know that as adults experience physical and cognitive declines, they tend to rely more on family and friends for caregiving and support. Further, older adulthood is a time when weaker social ties fall away (Umberson and Karas Montez 2010). Myriad studies have concluded that social integration is protective for health in later life. Indeed, the literature on social capital has shown that living in the same place or close to where one was born is protective to the extent that hometown social ties are long-lasting and hometown social networks are dense (Putnam 2000). Hence, we would expect that Southern Stayers and Non-Southern Stayers should experience a health benefit from to their deep regional roots.

I found evidence of a protective health effect of regional rootedness via local community cohesion and dense social networks in later life for the Southern Stayers, but not for Non-Southern Stayers. Beyond any effects of regional stability, there is a known social rootedness associated with Southern culture. This Southern social rootedness may act as a buffer to mitigate the health disadvantages of Southern poverty, policy, and limited access to healthcare. Consequentially, Former Southerners may suffer a health penalty from being uprooted not just

from their hometown, but specifically from their White, rural, Southern community and its connection to their identity. This kind of disconnect from one's hometown is captured by the concept "place identity mismatch" (Bradley and Longino 2009). The concept of place identity mismatch, along with the repotted hypothesis, could explain why the Former Southerners have the lowest perceived community cohesion. It is possible that the social uprootedness of being a regional transplant becomes more consequential for health in later life when individuals retreat from work-related activities and ongoing social integration requires new effort. While the Snowbird group also experiences a regional uprootedness, they tend to be a more advantaged group and also a more socially integrated group as many of them live in retirement communities with others like them. Further, the literature on regional mobility in later life indicates that a significant portion of the Snowbirds will return to their home communities when they face major health declines (Bradley and Longino 2009).

Returning to my conceptual framework, I considered the potential for regional context in early life to have a more pronounced effect on health than regional context in later life. Such effects are most likely to be detectable in global measures of health and mortality, yet I did not find evidence that exposure in early life to the detrimental material conditions of the South led to any additional risk of poor health (as assessed by the global measures) or mortality. In the realm of health behaviors, to the extent that regional cultural norms and policies in the South make it difficult to quit smoking or eat nutritiously, regional context during the transition to adulthood and during adulthood should contribute to disparities in chronic disease via health behaviors. My results showed the disadvantageous effect of the South on smoking in the Stayed Southerners, but surprisingly, I found no disadvantageous effect of prolonged exposure to the South on obesity or waist circumference. As for chronic illnesses, I found an important pattern in

hypertension that supports both the early-life origins hypothesis and my expectation that the worse access to healthcare in the South would have a negative impact on health. Southern birth is associated with increased risk of diagnosed or uncontrolled hypertension, but the Former Southerners are significantly less likely to have uncontrolled hypertension, and this is because they are much more likely to be taking antihypertensive medication. I found further evidence that the Former Southerners have better access to healthcare than the Southern Stayers in their usage of antidepressant medication and their frequency of doctor visits in the past year.

As mentioned previously, Ziembroski and Brieding (2006) found that the health risk of long-term Southern residence is cumulative and is detectable in later life. But other studies have suggested early life may be a critical period during which exposure to regional context has lasting influence on health (Glymour et al. 2011; Lauderdale et al. 1998; Zheng and Tumin 2005). My results lead to a conclusion that the ways that regional context influences health over the life course, whether intensely during critical periods or steadily over time, varies by health outcome. My results also raise two additional points for consideration. First, my results suggest that regional trajectory may be consequential for health in ways that are distinct from the effects of regional context. To the extent that regional trajectory shapes identity, social class, health behaviors, and social isolation, it may have independent effects on health. Second, regional context in later life may also be a critical period for exposure, in addition to early life. To the extent that regional culture promotes social integration, it may buffer other negative influences of regional context on health. This may be especially important in later life when individuals depend more on their family and local community for social support (Umberson and Karas Montez 2010).

Limitations

These findings raise a number of questions regarding the health consequences of exposure to regional context over the life course that cannot be addressed with these data. First, as discussed in the Methods section, mortality selection into the NSHAP sample may differ by regional trajectory. Second, the sample size is too small to do more nuanced analyses looking at subgroup differences, especially in rare outcomes such as mortality or smoking reduction. Third, the Former Southern trajectory is the least common and thus the numbers are small. While this limits statistical power for detecting differences between trajectory groups, a different concern is that the results could reflect uniqueness of a few individuals.

Conclusion

Regional health disparities are not as straightforward as "the South is bad for health." This study presents evidence that regional trajectory impacts later life health in a way that combines the influence of socioeconomic status, access to healthcare, community cohesion, and social rootedness. Certain aspects of regional context, particularly community cohesion, may become more salient for health in later life. In the end, to understand the puzzle of the Former Southerners, we encounter the question of context vs. composition. How much of their less optimal health profile is a reflection of the social disadvantage they carry with them in their place identity, and how much of it is shaped by contextual exposures, either in early life in the South or in adulthood in another region? The Former Southerner group may have missed out on the health protective aspects of continued Southern residence (e.g., Southern culture and social rootedness). Further, they may have encountered the stigma of a class penalty in their destinations. The data seem to suggest that it is composition in context that matters. It is not just who you are, but *where* you are that shapes how *who* you are matters for health.

CHAPTER THREE: STATE CIGARETTE TAXES, SMOKING, AND IMPLICATIONS FOR THE EDUCATIONAL GRADIENT IN MORTALITY

Introduction

It is taken as a social fact that less educated people live shorter lives. But the association between educational attainment and mortality is not static. Educational disparities in mortality in the U.S. have widened in the past three decades (Montez et al. 2011). In fact, the educational gap in life expectancy at age twenty-five among Whites has doubled for men and tripled for women since 1990. There has also been a dramatic divergence in mortality trends between states due in large part to the premature mortality of individuals with low education (Montez et al. 2016). Concurrent with these changes, the U.S. has seen a rise in state's rights, divergence in state policies, and widespread use of state preemption. In this evolving political landscape, state-level policy is increasingly consequential for population health.

New research suggests that state policy is most consequential for individuals with low levels of education (Beckfield and Bambra 2016; Krieger et al. 2014; Montez et al. 2016, 2017). This is because college graduates have personal resources that make them less dependent on their local context for health. Rich, educated people can "buy" their way into the determinants of health regardless of where they live (Chetty et al. 2016). This idea is consistent with Fundamental Cause Theory (FCT) (Link and Phelan 1995). According to FCT, increased educational attainment affords flexible social resources, which can be marshaled to avoid health risks and access medical technology. Individuals with less education, on the other hand, are more dependent on their local context. This is where variation between states becomes consequential for health. For example, an individual with less than a high school education has limited employment options and is less likely to receive adequate health insurance through an employer.

For this reason, under the American model of employer-based health insurance, not finishing high school is associated with less access to health insurance (Farber and Levy 2000). But states differ in the extent to which they will offer public health insurance to low-income individuals. Thus, the same individual might qualify for health insurance through Medicaid in Illinois, but not in South Carolina. This is an example of a social mechanism by which state policy influences the strength of the low education→no health insurance association.

With this chapter, I contribute to a growing body of research that suggests state policy can disrupt the extent that educational attainment maps onto health resources, even when there is no change in the underlying social stratification (Beckfield and Bambra 2016; Cylus et al. 2015; Montez et al. 2019). I conceptualize educational attainment as a component of socioeconomic status and as a factor that has differential influence on mortality depending on state policy. I propose a Tobacco Control Transition model to conceptualize the multistage process of tobacco control policy and its corresponding impacts on smoking, smoking-related mortality, and the educational gradient in mortality. I draw on data from two nationally representative longitudinal surveys (The Panel Study of Income Dynamics and The National Social Life Health and Aging Project) to explore the potential for a specific state policy—the excise tax on cigarettes—to reduce smoking and moderate the educational gradient in mortality. Drawing on my conceptual framework, I argue that cigarette taxes act as an indicator for progression along the Tobacco Control Transition. Thus, in addition to their direct effect on smoking, cigarette taxes reflect states' overall progress in moving through the Tobacco Control Transition. State cigarette taxes may also be representative of a state's policy regime, which facilitates or inhibits educational disparities in health via multiple policy domains. My research here examines the contributions of these three possible mechanisms. With this study, I highlight one way that the association of a

fundamental cause—in this case educational attainment—with health inequality is contingent on state policy.

Background

Education differentiates social resources such that less educated people, on average, experience less healthcare, more toxic exposures, more instability, more stress, worse nutrition, and worse health behaviors (Crimmins and Saito 2001; Kubanksy et al. 1999; Ross and Wu 1995). But what if people were assured quality healthcare, financial stability, and protection from toxic exposures regardless of their social resources?

In today's America, states have unprecedented control over access to the social determinants of health (Nathan 2005). This is because decisions about public health insurance, public economic benefits, regulation of health risks, and environmental standards are made at the state level, and increasingly so. The American Federalist system has seesawed between giving more or less autonomy to states (Nathan 2005). Since the 1980s, more discretion to expand or limit public access to everything from education to clean air rests in the hands of the state government (Kondratas et al. 1998; Nathan 2005). State policies determine who is exposed to what and for how long and who gets access to protective resources ranging from healthcare to stable housing to paid sick leave. I should note that I follow convention in conceptualizing educational attainment not as a measurement of an individual's cognitive abilities, but as a component of and proxy for socioeconomic status (House 2002). More education is power; it is agency; it is the privilege of highly-educated individuals to move to any state and know that their health will be minimally impacted.

States vary in the degree that educational attainment predicts health. If we imagine a spectrum with complete decoupling of the educational inequality/health association on one end and complete coupling of the educational inequality/health association on the other end, states are distributed at different positions along this spectrum. Two general strategies will, in theory, reduce the educational gradient in mortality: 1) reduce educational inequality (e.g., compulsory schooling laws, mandated school desegregation); or 2) decouple educational inequality from health and mortality (House 2002; Phelan et al. 2010). The second strategy, decoupling educational inequality from health and mortality, requires investment in non-education sectors. It may seem counterintuitive, but it takes investment in policy domains other than education to decouple educational inequality from health. For example, mandatory seat belt laws reduce motor vehicle fatalities (Rivera et al. 1999), and mandatory seat belt laws have a stronger effect on seat belt use among those with less education (Harper et al. 2014). Interestingly, the differential impact of seat belt laws by education was larger for states that transitioned from no law directly to primary enforcement (i.e., drivers can be stopped and ticketed for failing to use seat belts alone), instead of upgrading from secondary (i.e., drivers can be ticketed for no seat belt when stopped for something else) to primary enforcement (Harper et al. 2014).

There are various theoretical perspectives on the educational/health inequality association. This chapter draws heavily on Fundamental Cause Theory. According to Fundamental Cause Theory, educational/SES gradients in health and mortality emerge as a result of advances in scientific knowledge and medical interventions (Clouston et al. 2016; Phelan et al. 2010). When we first learn how to intervene in a disease process, people with more education, money, and social privilege will benefit disproportionately. As the better educated use their social resources to avoid a particular disease outcome, a gap in health emerges (Clouston et al.

2016). In the time it takes for less educated, poorer people to gain access to the new treatment or knowledge for disease prevention, the gap widens. When this inequality-producing process occurs across multiple health outcomes, the result is a steep educational gradient in mortality.

State policies with the best chance of flattening educational gradients in health and mortality are those that ensure public access to things that highly educated people have in abundance or that protect populations against harmful exposures to materials, toxins, or lifestyle factors (e.g., zoning to prevent toxic exposures, occupational safety regulations, investment in public transportation, etc.). Policies that ensure universal or uniform access to a determinant of health should contribute to a decoupling of educational inequality and health (Phelan et al. 2010). For example, laws mandating universal access to vaccination, fluoride, clean indoor air, and minimum wages reduce the channels through which educational inequality can shape health (Gostin and Gostin 2009; Hodge and Gostin 2001). Investment in roads, more generous unemployment insurance, and increased regulations to protect worker safety will also help decouple the link between low education and poor health/shorter life (Cylus et al. 2015; McKinlay 1979; Viscusi 1986).

While it is relatively easy to list policies that should, in theory, result in a weakening of the educational gradient in health and mortality, it is much more difficult to find empirical evidence of this effect. Here are some of main challenges to empirical work of this sort. First, and most obvious, social policies constrain health in complex ways, so it is difficult to isolate the effect of a single policy on the educational gradient in mortality. Yet, standard methods of causal inference in quantitative research rely on isolating treatment effects and minimizing bias. Second, policy changes can have gradual influence that is detectable only at the aggregate level, often many years after exposure. The lagged nature of most social policy effects on disease and

mortality means that there are countless opportunities for intervening mechanisms to transform the treatment effects over the life course. Third, although social policies initiate causal chains, they are often several causal steps away from measurable health outcomes. Because medical research traditionally focuses on proximal, visible causes at the individual level, it is easy to underestimate the role of social policy variables in producing health inequalities. Their distal or upstream position in a causal relationship makes their effects "invisible" with standard research methods. Worse yet, we erroneously attribute their effects to other downstream variables. Fourth, exposure to policies is often uneven across the population. Of particular concern are situations where privileged individuals are exempted from regulations or laws or can opt out of exposure to a social policy. Fifth, social policies hang together and often have their effect in concert with each other. In this sense, efforts to isolate the effect of a single social policy on health may be misguided because social policy (particularly state policy) may have the majority of its influence as a "package deal." These are just some of the challenges to studies that aim to demonstrate the causal influence of social policies on trends in population health.

Despite these challenges, several recent studies have demonstrated that when localities or states adopt policies that promote universal access to the social determinants of health, educational inequalities in health are reduced. This evidence has come from studies that have attempted to isolate and quantify the impact of the Earned Income Tax Credit, paid family leave, access to WIC and food stamps, and racial integration of schools on health inequalities (Alvarez et al. 2015; Cylus et al. 2015; Hamad et al. 2018a; Hamad et al. 2018b; Liu et al. 2012).

Other scholars have taken a more holistic approach to show that regional context or state political regime can influence health and moderate health inequalities. A team of researchers led by Jennifer Karas Montez has identified five domains of state context that are consequential for

mortality disparities between states: economic output, income inequality, adoption of the Earned Income Tax Credit, Medicaid program quality and expansion, and tobacco policy environment (Montez et al. 2016).

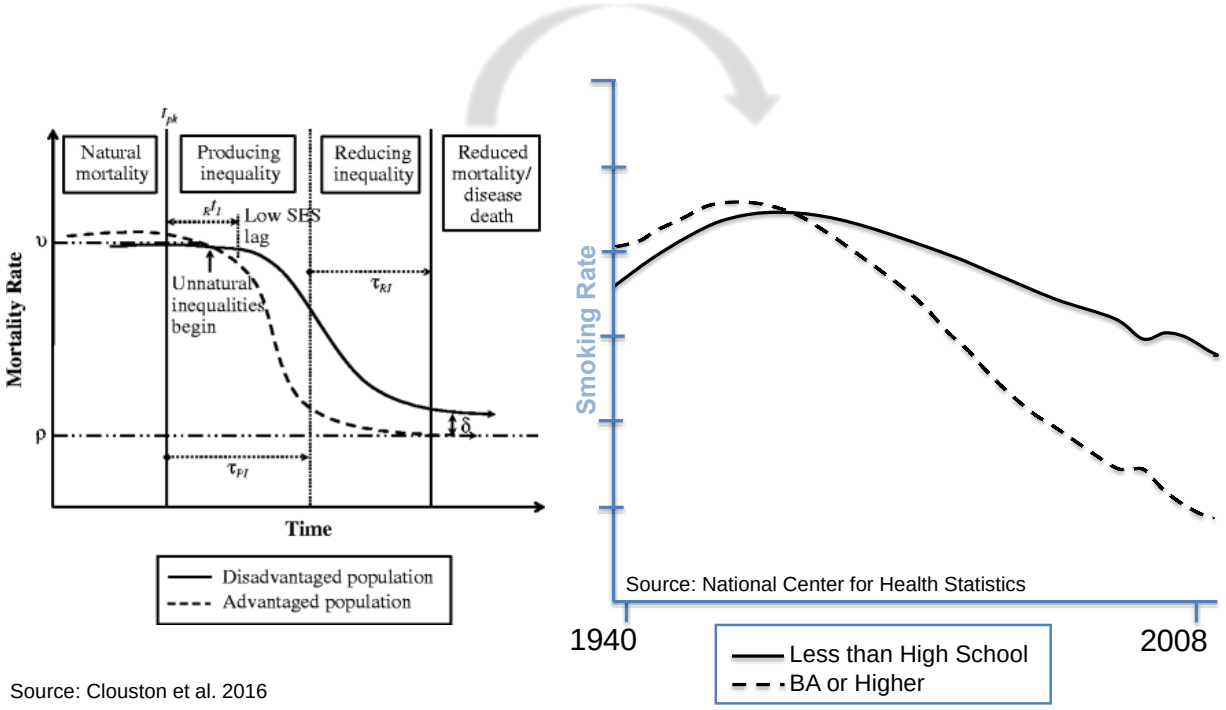
In this chapter, I chose to focus on cigarette taxes as a case for studying how state-level policy can moderate the educational gradient in mortality. The general consensus in the literature is that cigarette taxes reduce smoking prevalence, with the strongest effects seen in young people. This reduction in smoking is achieved through deterring smoking initiation and through encouraging smoking cessation. Because we know so much about the effect of cigarette taxes on smoking behavior (Bush et al. 2012; Chaloupka et al. 2012; Hill et al. 2014; MacLean et al. 2015), it is a useful case for testing the potential for state policy to modify educational disparities in mortality. Additional reasons why cigarette tax policy is ideal for exploring the potential for state policy to moderate the educational gradient in mortality are that: 1) Unlike policies such as paid family leave, state cigarette taxes have been implemented since the 1920s, so there are many years of data, which allow for the possibility of looking between and within states for effects; 2) smoking accounts for half of the recent increase in the educational gap in mortality for White women and much of it for White men (Ho and Fenelon 2015); and 3) smoking-related mortality explains 60 percent of the mortality disadvantage of Southern states compared with other regions (Fenelon 2013). Smoking is an especially important driver of the educational gradient in later-life mortality because over half of today's older adults were smokers at some point, but consistent with FCT, the more educated individuals quit (Fenelon and Preston 2012; Phelan et al. 2010).

Indeed, smoking is a compelling example of FCT in action (Link and Phelan 2009). Before the health risks of smoking were publicized in the 1964 Surgeon General's Report on

Smoking and Health, there was no educational gap in smoking. Then, as knowledge spread about the health risks of smoking, a large educational gap in smoking emerged (See Figure 3). College educated people quit or never started smoking. But without the same access to knowledge or resources to quit, low-educated people kept smoking. This resulted in the large educational disparities in smoking and smoking-related mortality that we still see today. Among today's older adults, the college-educated are more likely to be former smokers instead of current smokers (Link and Phelan 2009). It is important to note that although cigarette taxes are thought to be most effective at preventing and reducing smoking among young people (Lewit and Coate 1982), studies have also shown significant reductions in smoking among older adult smokers in response to cigarette taxes (DiCicca and McLeod 2008; MacLean et al. 2015; Stevens et al. 2017). This tax-induced smoking cessation in later life may be especially likely to result in detectable reductions in mortality in the short-term since a large literature suggests smoking cessation even later in life reduces morbidity and increases longevity (DiCicca and McLeod 2008).

The public health crusade to reduce smoking is now over a half century underway in the U.S. State cigarette excise taxes have become a key strategy for tobacco control. The causal effect of cigarette tax increases on smoking has been thoroughly studied by economists. The general consensus in the literature is that cigarette taxes reduce smoking, with the strongest effects being on young people. Because we know so much about this mechanism, it is useful for testing the potential for state policy to modify educational disparities in mortality.

Figure 4. Smoking Trends as Example of Fundamental Cause Theory



Source: Clouston et al. 2016

Tobacco control efforts shape trends in smoking initiation, smoking prevalence, and smoking-related mortality in patterned ways in line with what I refer to as the "Tobacco Control Transition." In the following section, I propose a model (the TCT) that I use as a conceptual framework to guide my expectations about the interplay between cigarette taxes, smoking, death, and disparities.

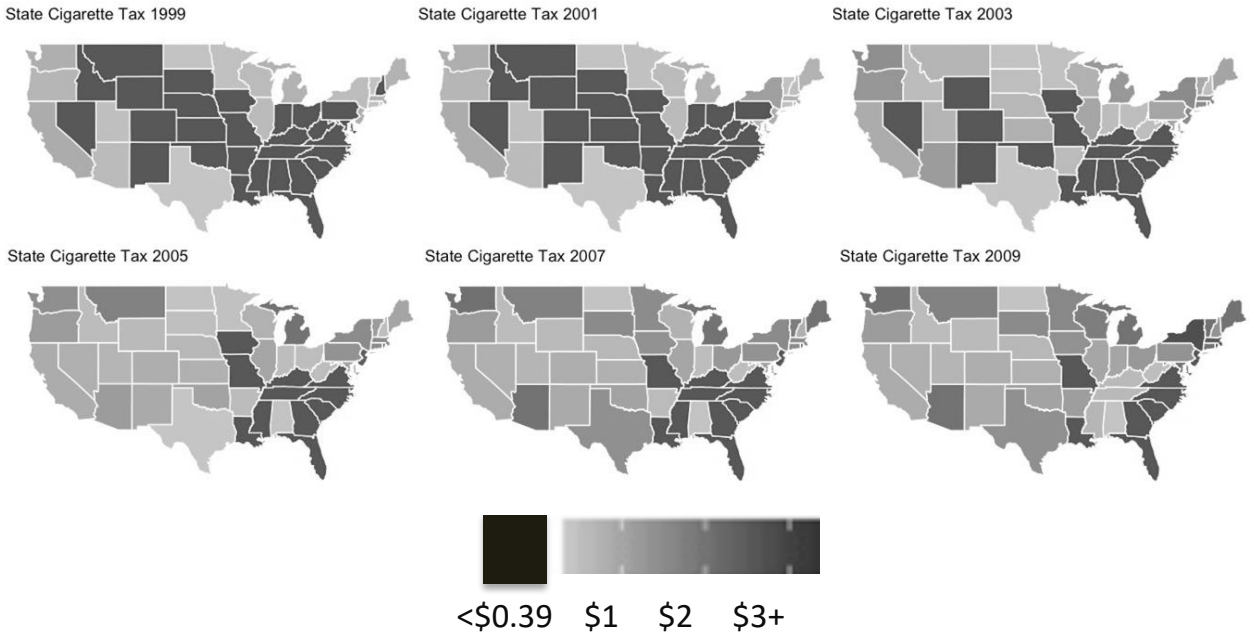
Conceptual Framework: The Tobacco Control Transition

To make sense of the complex dynamics between tobacco-control strategies such as cigarette excise taxes, and smoking behavior, mortality, and mortality inequalities, I propose a model I call the "Tobacco Control Transition" (TCT). The Tobacco Control Transition describes the population health consequences of the adoption of a suite of strategies intended to reduce

smoking and prevent tobacco use. In the model, tobacco control refers to taxation of cigarettes, laws to regulate indoor air, laws to restrict tobacco advertising, and campaigns to educate the public about the health risks of smoking. It also involves the contemporaneous shifts in culture and norms around smoking in public and private spaces. All of these variables shift at a contextual level (e.g., state), and through their mass influence on individual behavior, they have consequences for population trends in smoking and mortality.

States have been taxing cigarettes since 1921. Cigarette taxes were used to generate revenue for states. Cigarette taxes remained relatively low until the late-1990s. There has been a divergence in state-level cigarette taxes since 1999 (see Figure 5), with the range between the highest and lowest state tax widening from $2.43 in 2005 to $4.18 in 2015. States in the Northeast are the leaders in high cigarette taxes, while the Southern states consistently have the lowest cigarette taxes. On average, states in the South saw a $0.35 increase in cigarette tax levels from the 2001–2005 average to the 2011–15 average, whereas states in the Northeast saw a $1.58 increase over the same period. Few states increased cigarette taxes in the 2011–15 period. This may be because there was a $0.62 increase in the federal excise tax in April 2009, which is something I do not explore in this analysis but may consider in future work.

Figure 5. State Cigarette Taxes 1999 through 2009



Although they were not initially part of tobacco control efforts during the 1970s and 1980s, cigarette excise taxes are now a central pillar of a state's tobacco control program (Gorovitz et al. 1998). In every state during my study period, smoking prevalence is highest among individuals with low education (Farrelly et al. 2012). This means that every state has progressed to at least Stage 2 of the TCT. And of great relevance to my current investigation, cigarette taxes have been shown effective at not just furthering reductions in smoking prevalence and initiation, but at reducing the educational gap in smoking (Chaloupka et al. 2012). Much of the controversy surrounding cigarette taxes stems from concern that they punish poor smokers economically. But because cigarette taxes effectively deter smoking and promote smoking cessation, there is a compelling argument that by having a stronger effect on the poor and less educated, they promote equity in smoking-related mortality (Chaloupka et al. 2012).

Some studies have shown that individuals with lower socioeconomic status are more responsive to cigarette tax increases (Siapush et al. 2009), although other studies have shown no increased responsiveness to tax among low-income or low-educated individuals (Borren and Sutton 1992). Regardless of differential sensitivity to cigarette taxes, as long as low-educated individuals have a higher prevalence of smoking, smoking contributes more to mortality for the low educated. It follows that any reductions in smoking due to taxes should have a greater impact on mortality among the low educated. As my TCT models show, there need not be a differential responsiveness to cigarette taxes by education for cigarette taxes to reduce the educational gradient in mortality.

One of the challenges in studying the effects of cigarette taxes on smoking disparities is that states have increased cigarette taxes concurrently with other tobacco control strategies, such as labeling requirements for cigarette packaging, restrictions on tobacco advertising, and clean indoor air regulations. Some studies of cigarette taxes have tried to address this by controlling for the passage of smoking bans (see, for example, MacLean et al. 2015). This helps somewhat, but the problem of confounding by general progression through the Tobacco Control Transition remains. The Tobacco Control Transition involves multiple strategies that impact smoking behavior as well as social norms around smoking. Cigarette taxes and clean indoor air regulations are just two of those strategies. Further, there may be feedback between cigarette taxes and smoking behavior such that as norms around smoking change, it becomes easier to pass larger increases in cigarette taxes. Because of this complexity, I consider the possibility that cigarette taxes act as a signal for progression through the TCT. In this way, any detectable effect of base cigarette tax level on educational disparities in smoking and mortality may reflect the aggregate effect of all of the tobacco control efforts undertaken in addition to taxing cigarettes.

In other words, cigarette taxes may have their effects, in part, from the direct price increase, and in part, indirectly from what tax level signals about the progression along the Tobacco Control Transition.

The TCT depicts the expected consequences of tobacco-control strategies for population health, but there is great variation in the pace that states progress through the five stages of the transition (See Figure 6). State differences in demographics, culture, economy, and politics make it such that certain states (e.g., California) have progressed quickly through the Tobacco Control Transition, while others (e.g., North Carolina) have stalled. For nearly three decades, California led the way in tobacco control (Rogers 2010). In 1989, California launched a $0.25 per pack tax on cigarettes as part of the state's larger strategy of tobacco control. Revenue generated through the tax was funneled back into other strategies for tobacco control (Roeseler and Burns 2010). In line with the National Cancer Institute's *Standards for Comprehensive Smoking Prevention and Control*, California's tobacco control program aimed to achieve comprehensive social norm change, which was believed to be more effective for smoking reduction than focusing on individual smokers (Roeseler and Burns 2010). This model of social norm change was disseminated widely and influenced tobacco control efforts in other states. But not all states have been receptive. Former tobacco-producing states, such as North Carolina, have trailed behind in raising cigarette taxes and regulating smoking in public spaces. The lobbying against anti-smoking legislation and state preemption to block tobacco control by lower jurisdictions are also factors that have slowed the pace at which states proceed through the TCT.

While this policy variation across states is indicative of deeper differences in state political culture, it is useful for studying the effects of cigarette taxes on the educational gradient in mortality. Here, I conceptualize each state as a population with its own educational gradient in

mortality that is dynamic over time in response to state policy and other factors that differ between states. I make the assumption that the meaning of educational attainment for social stratification is stable over my study period and across states. Thus, I interpret any variation in the educational gradient in mortality as indicative of changes in the extent to which education maps onto health, not changes in the extent to which education stratifies social resources. With that said, a recent study showed that the functional form of the educational gradient in mortality varies by region in the U.S. In the South, the form is consistent with the Human Capital Hypothesis (a linear association between years of schooling and mortality), whereas the other regions reflect the Credentialism Hypothesis (high school completion and college completion are marked by stepwise decreases in mortality) (Sheehan et al. 2018).[1] Put simply, this suggests that a college degree does not mean the same thing for social status in one region as it does in another. It is possible that some of this variation is because states within these regions are at different stages of the TCT, but investigating the causes of interregional variation in the functional form of the educational gradient in mortality is beyond the scope of this study. I acknowledge it here as a reminder that my study observes states at different stages in the TCT and, in turn, with different educational gradients in mortality at the study baseline in 1999.

There are four population-level measures that show patterned trends as a consequence of the Tobacco Control Transition and its influence on individual smoking behavior within a

[1] The human capital hypothesis and credentialism are the two main theories of how years of schooling relates to health and mortality (Sheehan et al. 2018). According to the human capital hypothesis, each additional year of schooling enhances human capital that manifests as reductions in mortality (Mirowsky and Ross 1998). According to credentialism, the relationship between educational attainment and health is not linear, but an incremental trichotomy with mortality reductions resulting from the earning of educational credentials, specifically, a high school diploma and a college degree (Backlund et al. 1999).

population: 1) smoking initiation; 2) smoking prevalence; 3) smoking-related mortality; and 4) contribution from smoking to SES-inequalities in mortality such as the educational gradient in mortality. Similar to the Demographic Transition (Lee 2003), the Tobacco Control Transition can be conceptualized with five stages. The TCT model I introduce here is not the first attempt at modeling the complex relationship between smoking prevalence and smoking-related mortality. In 1994, Lopez and colleagues proposed a multistage model of the cigarette epidemic where they conceived of four stages and depicted trends in smoking prevalence by gender and smoking-related deaths by gender (Lopez et al. 1994). Thun and colleagues extended the Lopez model in 2012, updating it with recent data and extending it to the year 2020 (Thun et al. 2012). The aim of these models was to depict the long delay between widespread uptake of smoking in a population and its effects on mortality. The multistage TCT model I propose builds on these models, but is distinct in that the TCT also considers influence from tobacco-control efforts, including cigarette taxes, and it depicts the consequences for the educational gradient in mortality as predicted by Fundamental Cause Theory.

Stage 1. Pre-Transition. Populations (in this study, states) which have yet to undergo the Tobacco Environment Transition look like this:

1. HIGH Smoking Initiation
2. HIGH Smoking Prevalence
3. HIGH Smoking-related Mortality
4. LOW Educational gradient in mortality (contribution from smoking)

Stage 2. Initiation. As a population initiates the Tobacco Control Transition, public awareness about the health risks of smoking grows through the publicizing of research and through anti-smoking campaigns. This causes the more educated to use their social resources to quit. At the same time, the context (e.g., state government) begins to restrict ease of smoking through regulations and cigarette excise taxes.

1. DECLINING Smoking Initiation
2. DECLINING Smoking Prevalence
3. HIGH Smoking-related Mortality
4. LOW Educational gradient in mortality (contribution from smoking)

Stage 3. Saturation. As a population proceeds through the Tobacco Control Transition, public awareness about the health risks of smoking reaches saturation through the publicizing of research, anti-smoking campaigns, and tobacco product-labeling requirements. It is no longer just the most educated who are aware of the health risks of smoking. High cigarette taxes provide a strong incentive to quit or reduce smoking and a disincentive to initiate smoking. These influences combine to prevent smoking initiation among young people of all education levels. They also spur reduction in smoking among people of all education levels, though the highly educated people are more successful at quitting because they have better access to medical and cultural resources to support their decision to quit. With reductions in smoking, smoking-related mortality begins to decline, but primarily among the highly educated since they were the first to quit smoking. Reflecting the early reductions in smoking among the highly educated, the educational gradient in mortality is increasing. This is consistent with evidence that educational disparities in mortality in the U.S. have widened in recent decades (Montez 2012).

1. LOW Smoking Initiation
2. DECLINING Smoking Prevalence
3. DECLINING Smoking-related Mortality
4. INCREASING Educational gradient in mortality (contribution from smoking)

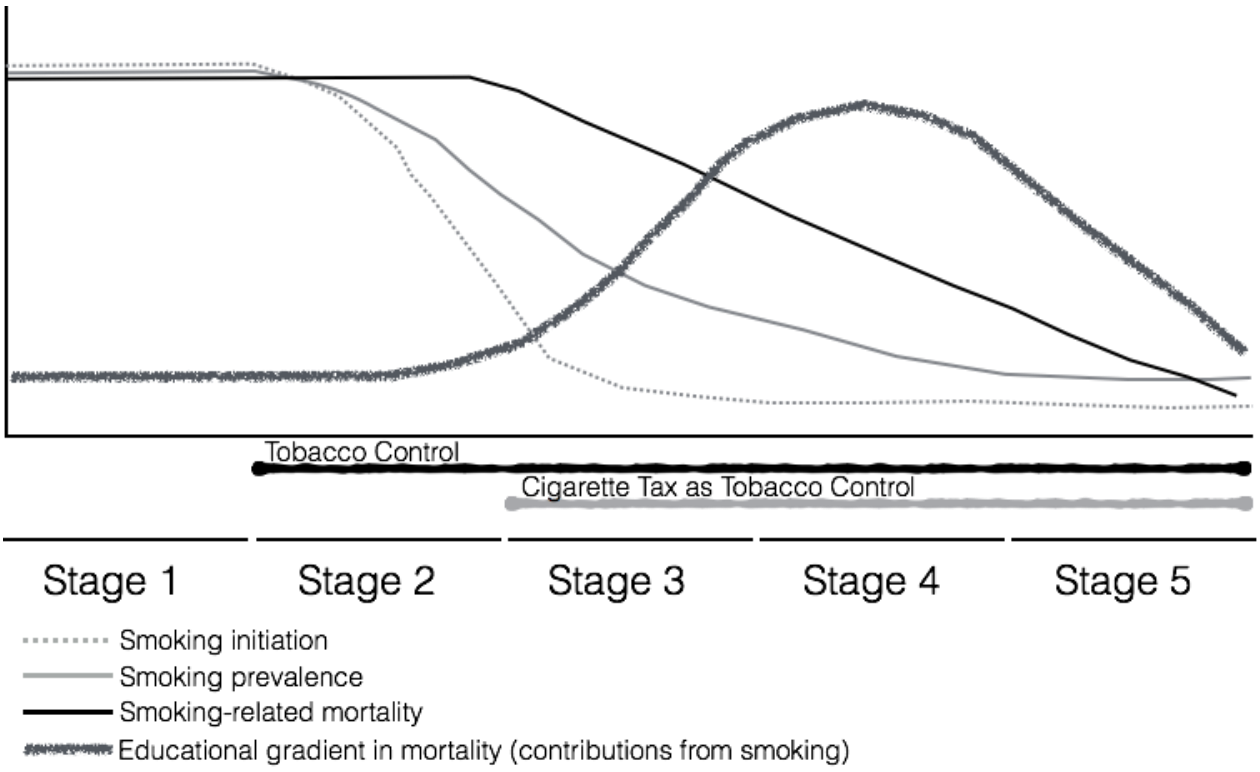
Stage 4. Completion. As a population completes the Tobacco Control Transition, public awareness about the health risks of smoking reaches saturation through the publicizing of research and through anti-smoking campaigns. It is no longer just the most educated who are aware of the health risks of smoking. High cigarette taxes provide a strong incentive to quit or reduce smoking and a disincentive to initiate smoking. These influences combine to prevent smoking initiation among young people of all education levels. While these influences also spur reduction in smoking among people of all education levels, the highly educated people are more successful at quitting because they have better access to medical and cultural resources to support their decision to quit. With reductions in smoking, smoking-related mortality begins to decline, but primarily among the highly educated because they were the first to quit smoking. Reflecting the early reductions in smoking among the highly educated, the educational gradient in mortality is high.

1. LOW Smoking Initiation
2. LOW Smoking Prevalence
3. DECLINING Smoking-related Mortality
4. HIGH Educational gradient in mortality (contribution from smoking)

Stage 5. Maintenance. As a population sustains the Tobacco Control Transition, the effects of clean indoor air regulations and cigarette taxes on smoking prevalence materialize as gains in life expectancy among former smokers. Smoking-related mortality continues to decline, with life expectancy gains strongest among those with less education because of the burden of smoking-related mortality in this subsection of the population. As declines in smoking-related mortality among those with less education catch up to those with more education, the educational gap in mortality stabilizes at a new low.

1. LOW Smoking Initiation
2. LOW Smoking Prevalence
3. DECLINING Smoking-related Mortality
4. DECLINING Educational gradient in mortality (contribution from smoking)

Figure 6. The Tobacco Control Transition Model



Situating cigarette taxes within the Tobacco Control Transition helps us predict how increases in cigarette taxes will impact smoking prevalence, smoking-related mortality, and, in turn, the contributions from smoking to the educational gradient in mortality. For states in Stages 2 and 3 of the Tobacco Control Transition, an increase in cigarette taxes should result in a reduction in smoking prevalence through increased quitting and deceased initiation. But there is a lag between the effects of a cigarette tax increase on smoking-related mortality and, eventually, on the educational gradient in smoking. A recent study found that it takes ten years for the health consequences of cigarette tax increases to materialize as gains in life expectancy (Baum et al. 2019). Thus, cigarette tax increases enacted by states in the late 1990s and early 2000s may not have any noticeable effect on the educational gradient in mortality until 2010 or later.

Based on the tobacco policies enacted and population trends in smoking, I estimate that most U.S. states were in Stages 2 through 5 of the Tobacco Control Transition over my study period, 1999 to 2015. For most states, the educational gradient in mortality had already increased as a result of the educational inequalities in smoking cessation and initiation that emerged in response to scientific evidence of the health risks of smoking. During the late 1990s and early 2000s, large cigarette tax increases occurred contemporaneously with anti-smoking media campaigns, clean air regulations, and shifts in public consciousness and norms around smoking. Cigarette taxes complimented the efforts to raise awareness of the harms of smoking because they provide an additional incentive to not smoke. In this sense, any detectable impacts of cigarette tax level on an individual smoker's likelihood of quitting during my study period likely reflect more than just a response to a cost increase in smoking. Mindful of this, and mindful that cigarette taxes have been a central pillar of tobacco control policy since the late 1990s, I expect that the level of a state's cigarette taxes will act as an indicator of that state's progression along the TCT during my study period.

Disentangling State Policy Effects

Finally, it is important to acknowledge a major challenge with this kind of research is isolating the effect of state cigarette tax level from other state characteristics that vary with it. State policies tend to hang together. For example, Massachusetts and Alabama sit at opposite ends of the spectrum of state cigarette tax levels, but they also are at opposite ends of the spectrum of progressive social and economic policies (e.g., access to Medicaid, duration of unemployment benefits, and environmental regulations). This poses a challenge to estimating the effects of a single state policy with observational data. To account for this, I estimated models with and without state fixed effects. Others have also used state fixed effects to prevent bias from

omitted state characteristics in studying the effect of cigarette taxes (For examples, see Bishop 2015, and DiCicca and McLeod 2008). In this paper, I considered to what extent the effect of cigarette tax policy on educational disparities in mortality reflects a direct effect via changes in smoking behavior, or a confounded association driven by other unobserved state characteristics.

Summary

This study explores dynamics between state cigarette taxes, smoking, and the educational gradient in mortality. Figure 7 depicts the hypothesized associations I test in this study. Educational attainment exhibits a negative gradient in mortality. This study investigates whether state cigarette taxes have an equalizing effect on the education-mortality association and whether it is plausible that this effect occurs by reducing smoking. First, I ask: Does state cigarette tax moderate the effect of education on mortality? Second, I ask: Does state cigarette tax reduce smoking? Finally, I consider to what extent the effect of cigarette tax policy on educational disparities in mortality is a direct effect via smoking, an indirect effect of the Tobacco Control Transition, or a confounded association driven by unobserved state characteristics. To answer these questions, I test the following hypotheses:

H1: Higher cigarette taxes will have a stronger protective effect against mortality for the low educated.

H2: Higher cigarette taxes will weaken the effect of years of schooling on time to death.

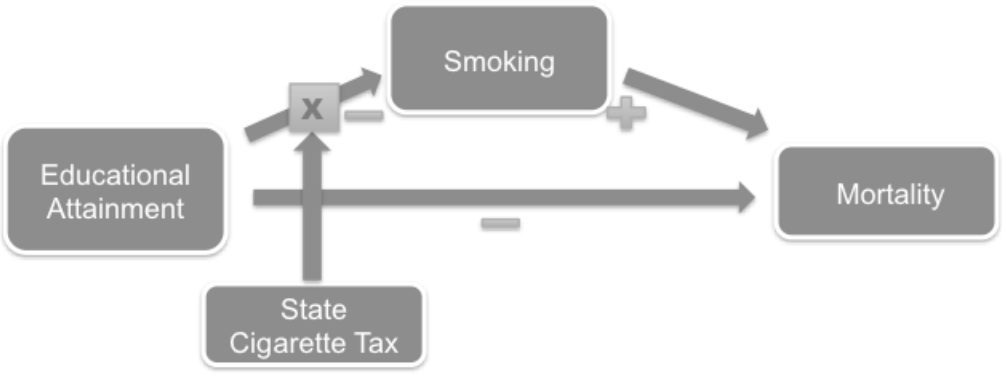
H3: Higher cigarette taxes will be positively associated with smoking cessation.

H4: A large increase ($0.50 or greater) in cigarette taxes will increase the likelihood of smoking cessation, above and beyond the effect of base tax level.

There are two reasons why higher cigarette taxes should have a stronger protective effect against mortality for the less educated (H1 and H2). First, cigarette taxes incentivize quitting more for individuals with limited financial resources. Second, cigarette taxes began to be used as a

strategy for tobacco control at a time when educational disparities in smoking had already widened. As long as the burden of smoking is higher among the lower educated, I expect cigarette taxes will have a greater impact on mortality for the lower educated. This will result in a weakening of contributions from smoking to the educational gradient in mortality. As part of my efforts to test the potential for increases in state cigarette taxes to weaken the educational gradient in mortality, I first look for a positive association between higher cigarette taxes and smoking cessation (H3). But a key question is whether the positive association hypothesized in H3 results from a direct effect on smoking behavior, from an indirect effect of general progression through the Tobacco Control Transition, or from confounding due to unobserved state characteristics. To clarify causality, I test H4 with longitudinal panel data that links dynamics in cigarette tax exposure to changes in smoking behavior. I estimate the effect of a large increase in tax on smoking in the subsequent survey wave, allowing for an interaction with base tax level and adding state fixed effects to control for unobserved confounding at the state level.

Figure 7. Directed Acyclic Graph Depicting Hypothesized Associations



Data and Method

Data

I drew on two survey data sets to carry out this study. The first is the National Social Life Health and Aging Project (NSHAP). The NSHAP is a nationally representative survey of community-dwelling older adults born between 1920 and 1947. I set up two analytic samples for cross-sectional analysis with 5-year mortality as the outcome. Because 58 percent of the sample smoked at one point in their lifetime and 17 percent of respondents died between waves, NSHAP is a useful data set despite its small sample size. My analytic samples consisted of the 3,005 respondents from Wave 1 and 3,377 respondents from Wave 2. At Wave 1, 14.8 percent are smokers, and 13.3 percent are smokers at Wave 2. I used the NSHAP primarily for descriptive analyses, which informed the regression models I ran using a second data set, the Panel Study of Income Dynamics.

The majority of the analyses I present in this chapter drew on this second data set, the Panel Study of Income Dynamics (PSID). This is the longest running panel study in the U.S. Questions about smoking status were asked in 1986 and then in every biennial wave from 1999 onward. I treated 1999 as the baseline year and defined my analytic sample as household heads and spouses, aged 25-97 in 1999 (n=10,949). I followed these respondents through eight subsequent biennial waves to 2015. I set up the PSID data for longitudinal analysis with person-years nested within persons, nested within states. Of the respondents, 2,372 were smokers at baseline in 1999.

The data on cigarette taxes came from the Tax Burden on Tobacco data set **(**Orzechowski and Walker 2017). I merged this publicly available data on state cigarette taxes with the NSHAP and PSID analytic samples.

Dependent Variables

I measured mortality cross-sectionally as 5-year mortality with the NSHAP (death between 2005 and 2010) and as 15-year mortality with the PSID (death between 2000 and 2015). I also measured time trends in death over the nine PSID survey waves using survival analysis methods. My measures of smoking behavior were based on self-report. I distinguished between current smokers, former smokers, and never smokers. I defined smoking cessation as the transition from being a current smoker to a nonsmoker. I also constructed a categorical measure of smoking intensity (nonsmoker, light smoker, moderate smoker, or heavy smoker) based on number of cigarettes smoked per day. I used this to define an alternative outcome variable: reduction in smoking. I counted any transition from an increased level of smoking to a lower level of smoking as a reduction (i.e., Heavy→Light and Light→Nonsmoker both count as a reduction in smoking).

Independent Variables

I measured state cigarette tax in multiple ways. First, I constructed a categorical measure of cigarette taxes with substantive cut points. I defined low cigarette taxes as less than $0.20 in 1999, medium taxes as $0.20-$0.59 in 1999, and high taxes as greater than $0.59 in 1999. But the distribution of state cigarette taxes in 1999 was such that many states fell in the low tax bin. Thus, I also constructed a 4-category variable based on quartiles of the distribution of person-year exposure to state cigarette taxes in 1999 experienced by the PSID sample. I constructed a similar 4-category variables of tax quartile for the 2001–2005 average and the 2006–2010 average based on the tax distribution in the NSHAP sample. I also used a continuous measure of state tax (in dollars) by year, and averaged over 2001–2005 and 2006–2010. The measures I have described so far are measures of base state tax level.

I also measured changes in base cigarette tax level, or tax increases. Studies of the effects of cigarette taxes on smoking behavior tend to model tax increases dichotomously, defining a large tax increase as $0.50 or greater (see Baum et al. 2019). I follow this approach.

I measured educational attainment with a continuous measure of years of schooling completed and a 3-category measure of educational attainment: <High School, High School, or College or more.

Analytic Strategy

Unless otherwise noted, the models described below drew on the analytic sample from the PSID. Thus, when I refer to the "study period," I am referring to the period from 1999 to 2015 during which my analytic sample from PSID was observed. The exploratory analyses I conducted using the two waves of the NSHAP are included as Appendix B. Ultimately, the larger sample size and the longer time period covered by the PSID data made it more useful than the NSHAP data for testing my hypotheses.

Mortality

I explored the impact of cigarette taxes on the odds of 5-year mortality and 15-year mortality, as well as time to death.

> Testing H1: Higher cigarette taxes will have a stronger protective effect against mortality for the low educated.

First, to test H1, I used logistic regression with dummy variables for each year of schooling to inspect the functional form of the educational gradient in mortality. The specific outcome modeled here is odds of 15-year mortality—death between 2000 and 2015. I ran separate regressions for each quartile of the 1999 cigarette tax distribution to see whether the educational gradient is steeper when cigarette taxes are lower. Second, drawing on the NSHAP sample, I

used multiple logistic regression to test the effect of 2001–2005 average cigarette taxes on the odds of 5-year mortality among smokers. I estimated this same model for never-smokers as a robustness check.

Testing H2: Higher cigarette taxes will weaken the effect of years of schooling on time to death.

To test H2, I first used unadjusted Kaplan-Meier survival curves to compare the magnitude of educational disparities in survival time across low, medium, and high cigarette taxes. I compared survival time between the three educational subgroups: less than high school, high school completed, and college or more. Kaplan-Meier survival curves are a common way of depicting differences in survival time (Rich et al. 2010). Kaplan-Meier curves provide more information about differences in time-to-death than a simple comparison of mean survival time. Nevertheless, they are univariate analyses, so I proceeded to multivariate analyses.

Next, I modeled time to death in two ways. I used Cox proportional hazards regression with person random effects and state fixed effects to estimate the influence of cigarette tax on educational disparities in the relative hazard of death over the study period. I ran a series of these models, varying the subpopulation of observations by level of cigarette taxes. The first model estimates the hazard ratio for years of schooling from a Cox proportional hazards model not conditional on tax. The subsequent stratified models estimate the hazard ratio for years of schooling if cigarette tax is: less than $0.39, less than $0.50, less than $1.00, between $1.00 and $1.99, and more than $2.00. Hazard ratios can be interpreted as the risk of dying at time (t). In the case of Cox proportional hazards regression, a unit increase in the covariate of interest is multiplicative of the hazard rate. Thus, a covariate with a hazard ratio greater than 1 is interpreted as a good prognostic factor while a hazard ratio of less than 1 indicates a bad

prognostic factor. Cox models make no assumption about the baseline hazard function. These models control for age, gender, and race.

I also analyzed educational disparities in time to death by cigarette tax level with parametric survival analysis models using the command `mestreg` in Stata 14. These survival models allowed me to add a state-level random effect to account for the correlation of observations from the same state. I tested models that assume a hazard function with a Weibull distribution and models that assume a Gompertz distribution. The results shown come from models with a Weibull distribution. I stratified the models by gender and included person-level and state-level random effects. I used these mixed-effects Weibull regression models to estimate the hazard ratio of the interaction between years of schooling and state cigarette tax. The models control for age, gender, race, and total family income.

Smoking

Testing H3: Higher cigarette taxes will be positively associated with smoking cessation.

To test H3, I first inspected trends in smoking prevalence by cigarette tax quartile in 1999. Second, I fit unadjusted Kaplan-Meier survival curves again to understand the effects of cigarette taxes on time to smoking reduction. Third, I used multiple logistic regression with the 1999 baseline sample to estimate the differential odds of smoking by education and cigarette tax quartile in 1999.

Testing H4: A large increase ($0.50 or greater) in cigarette taxes will increase the likelihood of smoking cessation, above and beyond the effect of base tax level.

Next, I turned to longitudinal analysis to test the hypothesized causal association between state cigarette tax increases and smoking cessation. I used random-effects logistic regression set up for panel data with person-years nested within persons from my 1999 baseline sample. With nine

waves of biennial data, I was able to test both between- and within-state effects of cigarette taxes on smoking cessation. I began by testing the effects of base state cigarette tax (continuous) on smoking cessation among smokers during the study period from 1999 to 2015. Exposure to cigarette tax is lagged (*t-1*) and used to predict odds of staying a smoker in the subsequent survey wave.

Then I turned to the additive effect of an increase in state cigarette tax, beyond the effect of base cigarette tax. I modeled the state tax increase continuously and inspected plots of its interaction with base tax. I also tested the effect of a large increase of cigarette taxes ($0.50 or greater =1, <$0.50 =0) on smoking cessation, and I allowed for an interaction between a large tax increase (0 or 1) and base state tax at the previous wave (continuous). My preferred model, Model 4, included a random intercept for subject and state fixed effects (as dummies). Finally, using mixed-effects logistic regression with random intercepts for persons and states, I tested whether education level (categorical) moderates the effect of cigarette tax on smoking cessation. These models included age, gender, and years of education as controls.

Robustness Check

The primary way I evaluated the robustness of my results was by treating never-smokers as a negative control. Where possible, I reran all of the models estimating an effect of cigarette taxes on smoking for never-smokers to check that the effects of cigarette tax increases were minimal for never-smokers.

Results

Descriptive Statistics

Table 7 displays descriptive statistics for the PSID analytic sample, the primary sample used for the analyses presented here. While the majority of respondents had completed high school, 16.5

percent had not completed high school and 22 percent had some college or more. In all, 21.8 percent of the PSID sample smoked at baseline, and 28.8 percent of the person-years over the study period were contributed by smokers. The average state cigarette tax level experienced by the PSID sample in 1999 was $0.40, and the average cigarette tax increase experienced over the study period was $0.84. The average age for the PSID sample was 44 years at baseline in 1999. The NSHAP is a much older sample with an average age of 73 years in 2005. Descriptive statistics for the NSHAP samples can be found in Appendix B (see Tables B-1 and B-2).

Table 7. PSID Sample Statistics (baseline = 1999)

	Possible Range	Mean (SD) Number (%)
15-year mortality 1999-2015	0 or 1	614 (5.6)
Current Smoker (longitudinal)	0 or 1	3,145 (28.8)
Baseline Smoker	0 or 1	2,372 (21.8)
State cigarette tax in $ (longitudinal)	0.025 to 4.35	0.84 (0.44)
Baseline state cigarette tax in $	0.025 to 1.00	0.40 (0.25)
Increase in experienced cigarette tax from baseline in $ (longitudinal)	-0.80 to 4.18	0.44 (0.35)
Large (>$0.50) increase in cigarette tax (longitudinal)	0 or 1	5,860 (55.3)
Cigarette tax in 1999:		
Low < $0.20	0 or 1	2,448 (22.5)
Medium $0.20-$0.59	0 or 1	6,124 (56.2)
High > $0.59	0 or 1	2,318 (21.3)
Years of schooling	1 to 17	12.9 (2.6)
Educational attainment:		
Less than High School	0 or 1	1,693 (16.5)
High School	0 or 1	6,329 (61.6)
College or more	0 or 1	2,248 (21.9)
Age	15 to 97	43.8 (15.1)
Female	0 or 1	6,039 (55.2)
White	0 or 1	6,980 (64.5)

Educational Gradients in Mortality

An observable educational gradient in mortality was present in the sample data from both the NSHAP and the PSID. Figure 7 shows the functional form of the educational gradient in mortality by 1999 cigarette tax quartile in the PSID sample. Comparing these plots, I see there was already evidence of differentiation of the gradient by cigarette tax level in 1999. There is a steep negative linear association between years of schooling and odds of death when cigarette taxes are low (See Figure 8, Quartile 1) and that transforms to a nearly flat association in the higher quartiles of cigarette taxes. A discontinuity in odds of death remains visible around the completion of high school when cigarette taxes are at medium levels. The highest quartile of taxes shows a flat gradient with the exception of a mortality disadvantage for individuals with nine years of education.

Figure 8. Educational Gradient in Mortality by 1999 Cigarette Tax Quartile

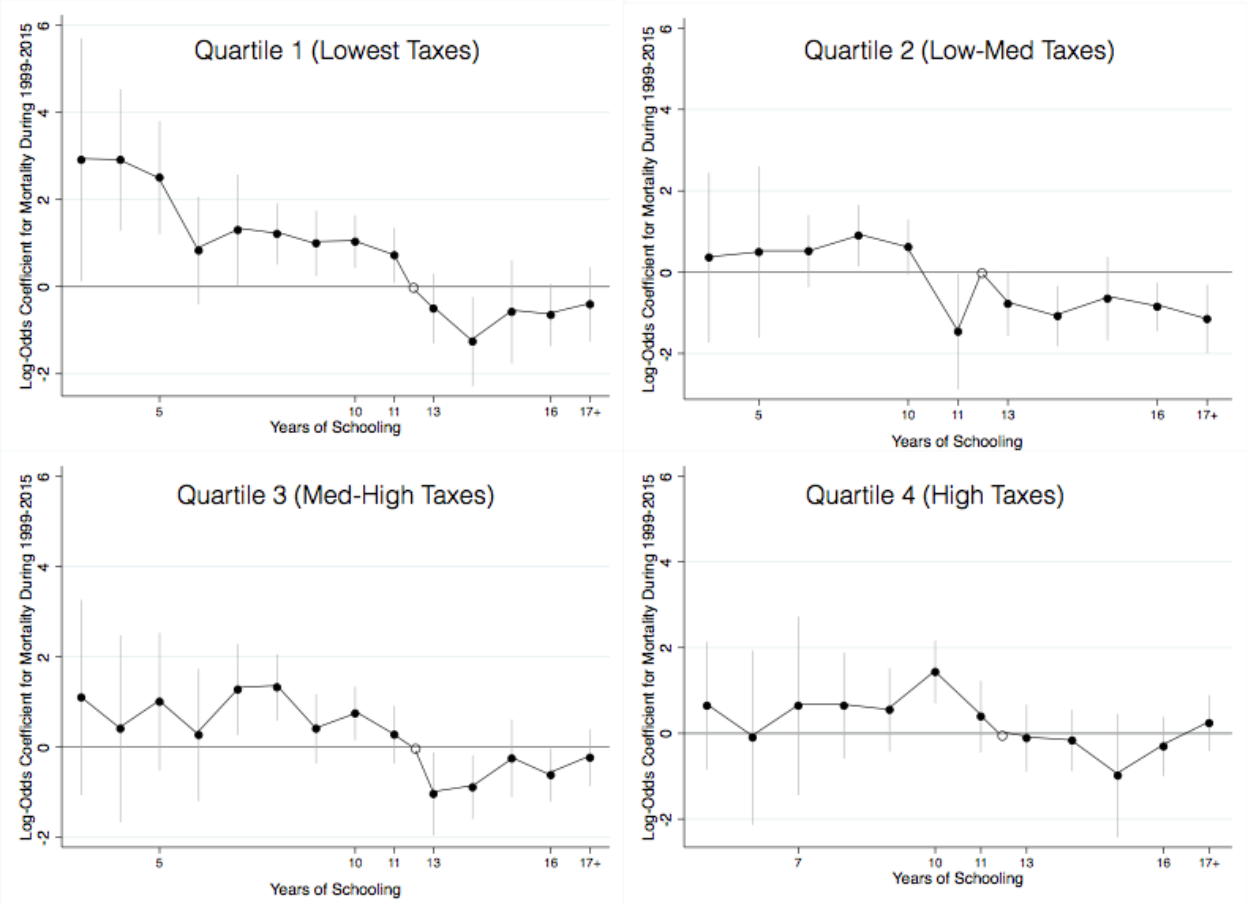
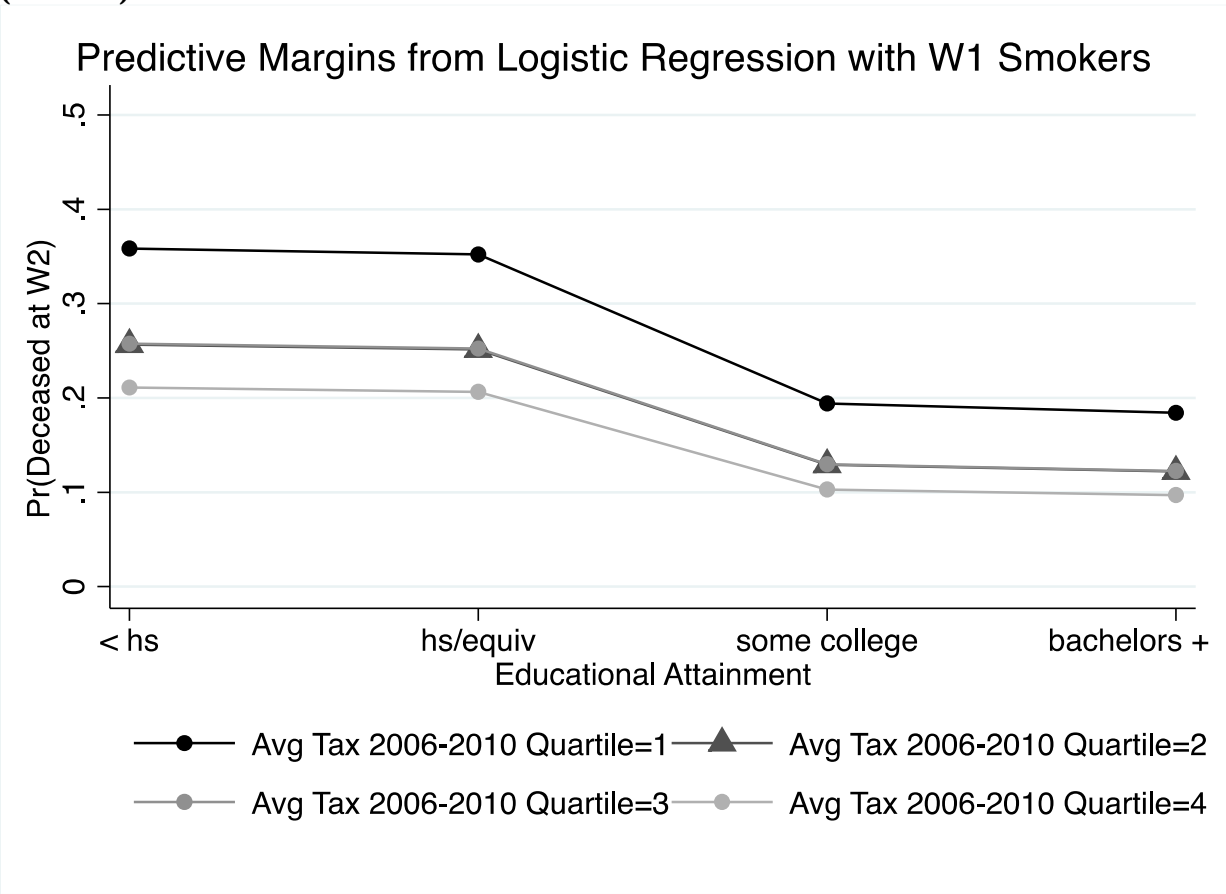


Figure 9 shows results from logistic regression predicting 5-year mortality with the interaction of cigarette taxes and education category, adjusting for age. Older adult smokers exposed to higher state cigarette taxes are less likely to have died between 2006 and 2010. Further, the relative mortality disadvantages associated with lower education are largest in the lowest quartile of cigarette taxes, smaller in quartiles 2 and 3, and again smaller in the higher quartile of taxes. It appears that the educational gap in mortality is reduced in the shift from tax quartile 1 to tax quartile 2 (see Figure B-1 in Appendix B). This same regression model with never-smokers does not show any moderation of education by cigarette taxes. Thus, there is

support for Hypothesis 1: higher cigarette taxes have a stronger protective effect against mortality for the low educated.

Figure 9. Probability of Death Among Smokers by Tax Quartile and Education Level (NSHAP)



Finally, Figures 10 and 11 display predicted survival curves for a 65-year-old with 11 years of schooling, by tax level ($0.39, $1, $2), stratified by gender. Tax has a graded, inverse effect on time to death. Results from this model did not show differences by gender, but there was an opposite effect of tax level at the highest levels of education. I have also run the same model with a Gompertz distribution and the results are similar.

Figure 10. Predicted Survival Function by Tax Level for Female Smokers with Low Education

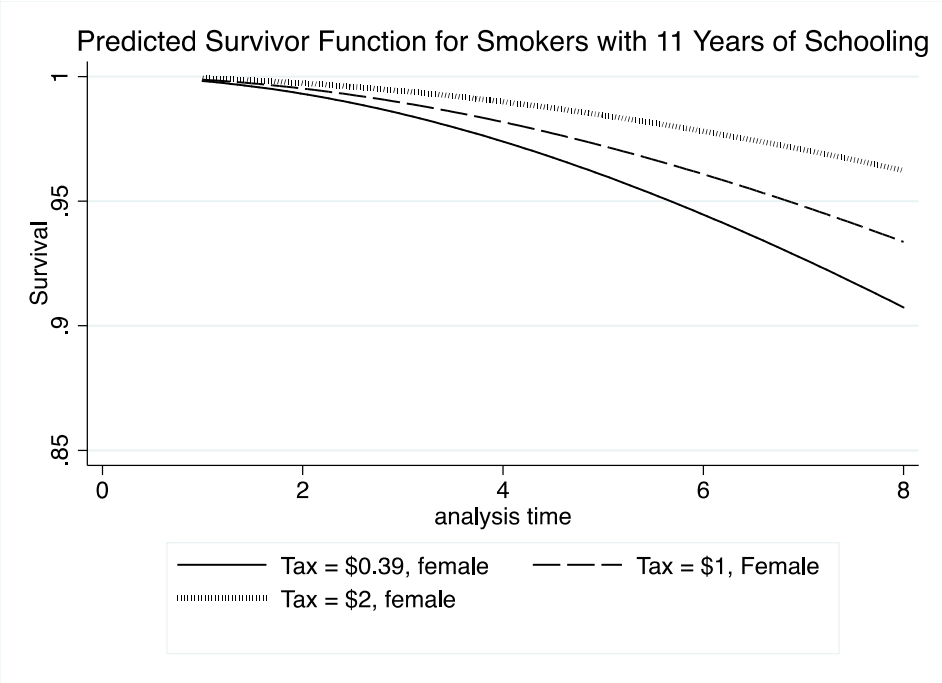
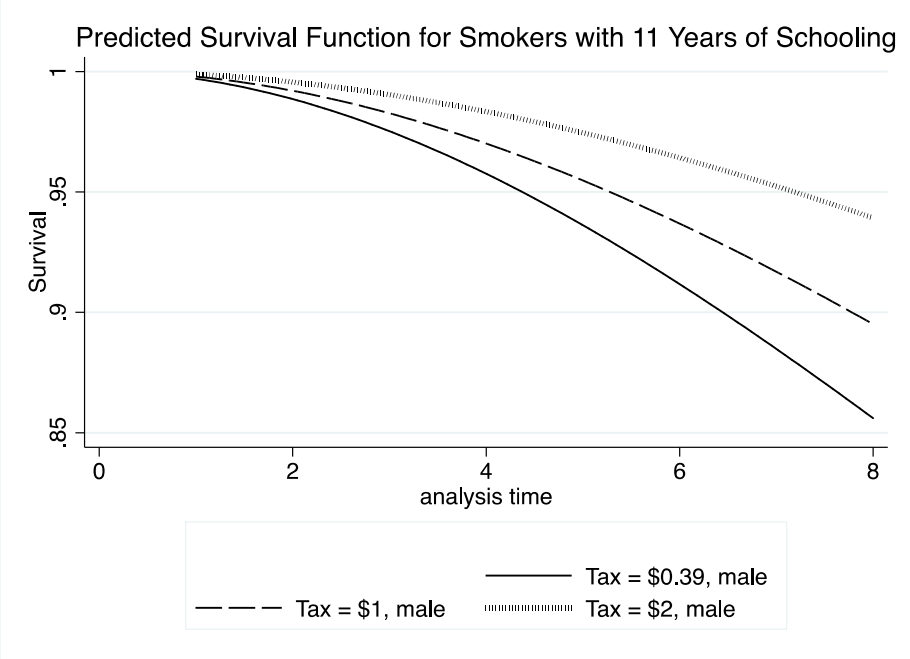


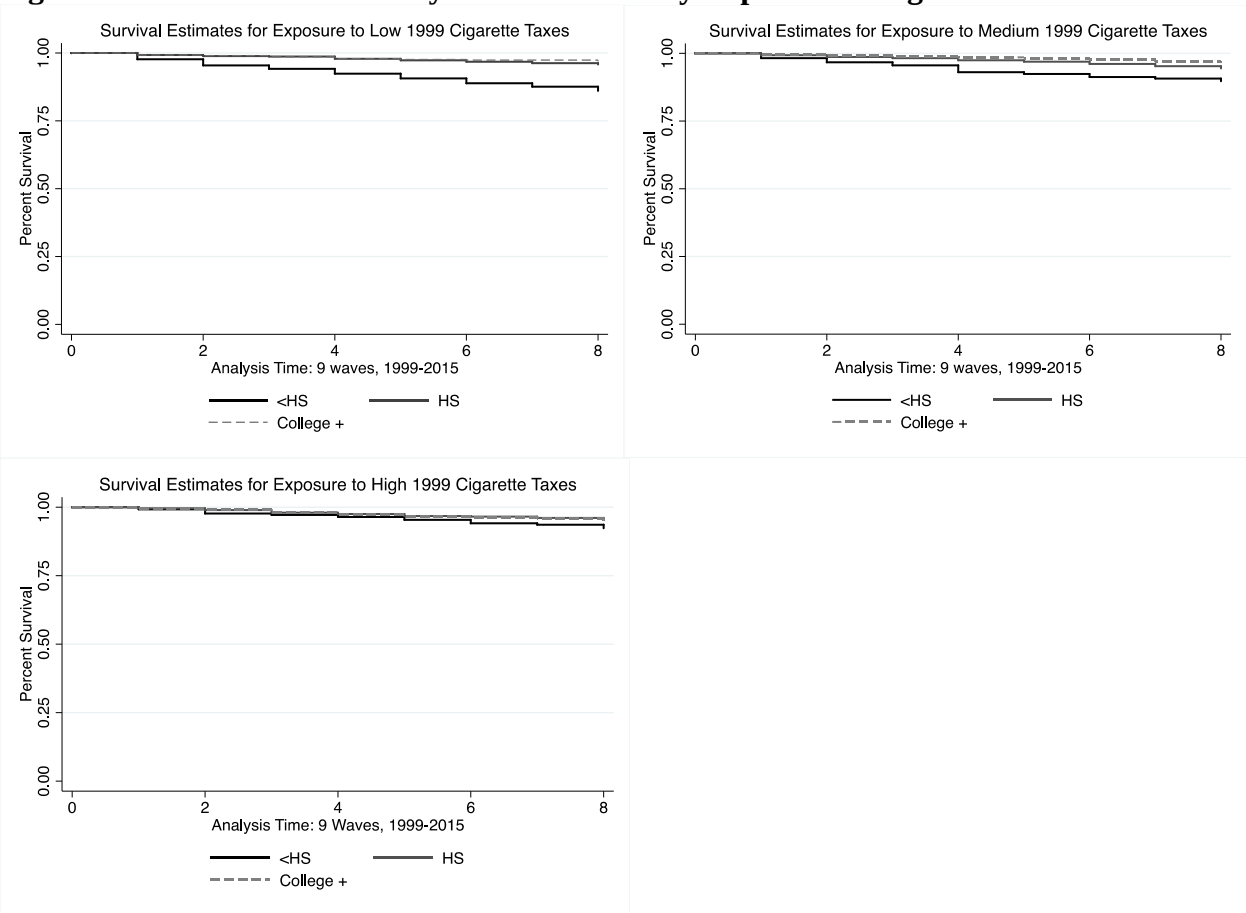
Figure 11. Predicted Survival Function by Tax Level for Male Smokers with Low Education



Effects of State Cigarette Taxes on Time to Death

Moving next to the effects of cigarette taxes on time to death, Figure 12 shows educational differences in survival curves, by substantive tax level in 1999 (low <$0.20, medium $0.20-$0.59, and high >$0.59). These plots reveal that, particularly for the lowest educated individuals, cigarette tax level differentiates their survival time. State cigarette taxes greater than $0.59 close the gap in survival between respondents with less than high school and those with a high school or college education.

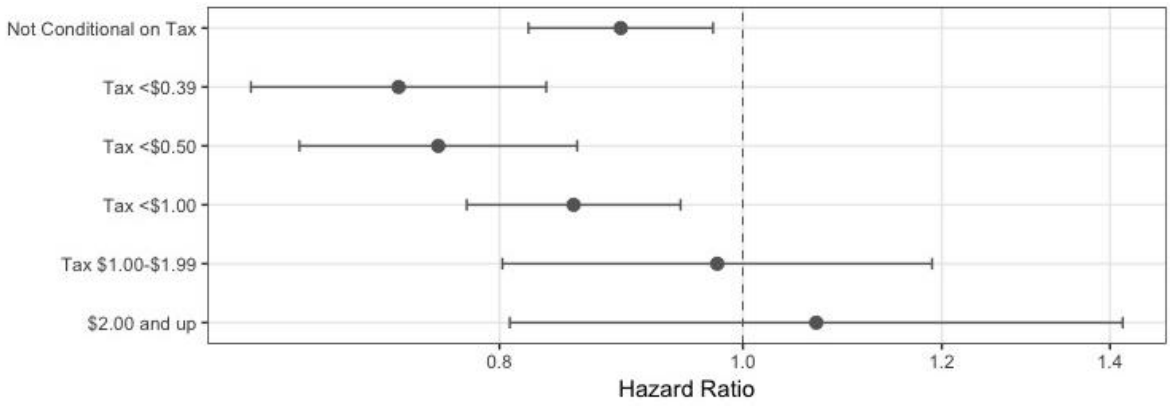
Figure 12. Survival Estimates by Education and by Exposure to Cigarette Taxes in 1999



I find that there is a protective effect of higher taxes on the relative hazard of death for individuals with less than a high school diploma (See Appendix B, Figure B-2). The educational gap in the predicted hazard of death is large among individuals exposed to state taxes, <$0.20 in 1999, but disappears among individuals exposed to tax states of $0.20 to $0.59. Taken together, the results from logistic regression and survival analysis suggest that variation in state cigarette taxes in 1999 was sufficient to moderate educational disparities in risk of death and time to death. This evidence supports Hypothesis 2, which supposes that higher cigarette taxes weaken the effect of years of schooling on time to death.

I explored this education-tax interaction further as displayed in Figure 13. The first line reports the hazard ratio for years of schooling from a Cox proportional hazards model not conditional on tax. Then I ran a series of stratified models that varied the subpopulation of observations. The resulting trend shows that the years of schooling effect on time to death is strongest when taxes are low and then gets weaker when taxes are higher. This suggests that cigarette taxes reduce educational disparities in mortality, providing further support for H2.

Figure 13. Hazard Ratios for Years of Schooling from Models Stratified by Tax Level



Additional models revealed that the educational gradient in time to death is weakened with each $1 increase in state cigarette taxes (see Appendix B, Table B-3). This interaction effect is strong among men, but not statistically significant among women.

Effects of State Cigarette Taxes on Smoking

Figures 14-16 display time trends in smoking prevalence by education under exposure to relatively low, medium, or high state cigarette taxes. The quartiles correspond to the distribution of person-year exposure to cigarette taxes at each survey wave. There were declines in smoking among the less educated (<HS and HS only) under exposure to low-to-medium taxes over the study period. The results also reveal a puzzling increase in smoking prevalence among individuals with less than a high school diploma under exposure to the highest taxes. There is minimal reduction in smoking prevalence among the college-educated over the study period. This is likely because smoking prevalence is already quite low in this group at the start of the study period in 1999, regardless of exposure to cigarette taxes.

Figure 14. Time Trend in Smoking Prevalence in Tax Quartile 1 (Low Tax), by Survey Wave

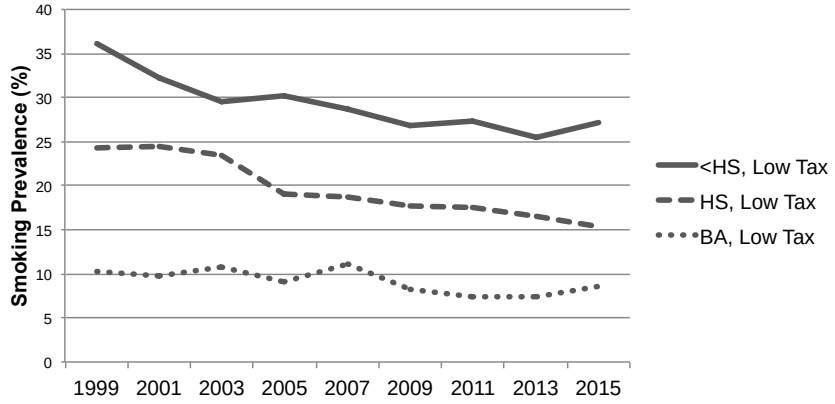


Figure 15. Time Trend in Smoking Prevalence in Tax Quartiles 2 and 3, by Survey Wave

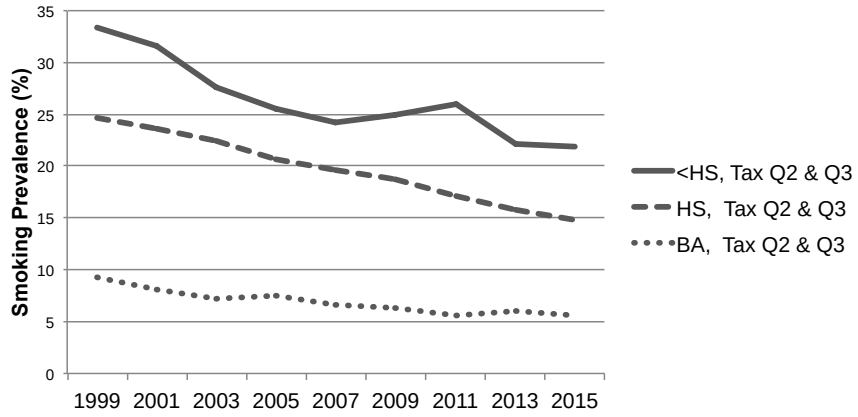
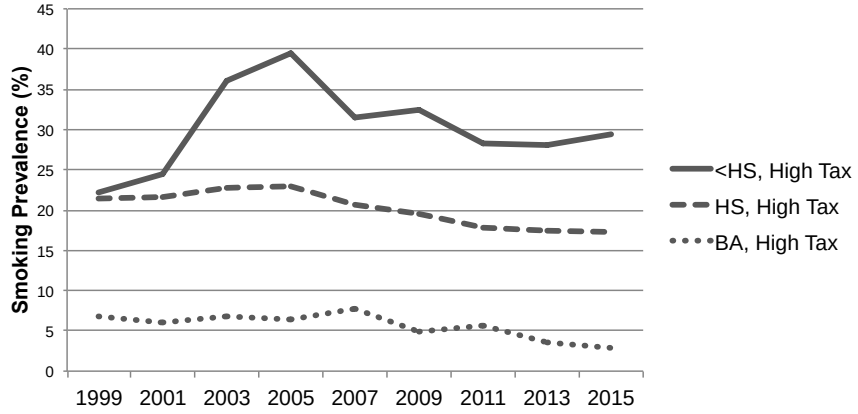


Figure 16. Time Trend in Smoking Prevalence in Tax Quartile 4 (High Tax), by Survey Wave



Studies have shown that many smokers who attempt to quit are unsuccessful. In light of this, I also looked at the effects of cigarette tax increases on any reduction in smoking intensity. I found that there were reductions in smoking intensity over the study period, regardless of exposure to cigarette taxes (see Figure B-3 in Appendix B). In fact, the trend in smoking reduction for exposure to a decrease or no change in cigarette tax was similar to the trends for exposure to large proportional increases in cigarette taxes. Only exposure to a ten-fold increase or greater led to a distinguishably stronger trend in smoking reduction.

Multiple logistic regression with the baseline sample (see Appendix B, Figure B-4) confirmed the existence of an educational gap in smoking prevalence between individuals with high school or more, and individuals with less than a high school diploma at all levels of cigarette taxes. But there is evidence that already in 1999, cigarette taxes moderated the educational gap in smoking such that the <HS vs. HS+ gap was largest in the lowest quartile of taxes and smallest in the highest quartile of taxes. Interestingly, this moderation by cigarette taxes of the educational gap in smoking was not detectable in the <College vs. College+ comparison.

So far, I have described results that support Hypothesis 3: higher cigarette taxes are positively associated with smoking cessation. But the question remains as to whether this positive association results from a direct effect on smoking behavior, an indirect effect of general tobacco control, or confounding due to unobserved state characteristics. Table 8 displays the results from random effects logistic regression models testing for a causal relationship between cigarette tax increases and smoking cessation. I follow the example of previous cigarette tax studies and estimate the effects of a large increase in cigarette taxes (>$0.50) as a binary variable (see Baum et al. 2019). I also inspected the linear effect of tax increases (not shown), but because states increase cigarette taxes in discrete steps and not one cent at a time, it is preferable to estimate the effect of a large tax increase on smoking. Model 1 estimates the effect of base state cigarette tax at time *t-1* on the odds of staying a smoker at time *t*. Exposure to $1 higher state cigarette tax is associated with 27 percent lower odds of staying a smoker (OR: 0.724). The addition of state fixed effects only strengthens the effect of base cigarette tax (OR: 0.593). Models 3-5 tested the effect of a large increase in tax (>$0.50) on smoking in the subsequent survey wave, allowing for an interaction with base tax level. While the negative effect of base

cigarette tax on likelihood of staying a smoker remains unchanged (OR: 0.710), there is a nonsignificant negative effect of a large tax increase on staying a smoker that interacts with base tax such that the effect of a large tax increase is negative (OR: 0.767) when base tax is 0 and becomes positive as base tax increases. This significant interaction likely reflects the ceiling in smoking cessation that is reached once all of the smokers sensitive to price changes have already quit smoking. Some smokers are simply resistant to tobacco control. It is likely that the smokers who remain in states with high cigarette taxes are more likely to be these resistant smokers. Thus, it makes sense that the interaction term for large increase X base tax level is positive. Model 4, the preferred model, adds state fixed effects to control for any unobserved confounding at the state level. The addition of state fixed effect strengthens the negative effect of base tax on continued smoking, bumps the negative effect of a large tax increase into marginal statistical significance, and weakens the interaction term. Finally, Model 5 adds a random intercept for state instead of fixed effects. This increases the statistical significance of the interaction term, but the overall story told by the coefficients remains unchanged. The results of Model 4 are presented visually in Figures 17 and 18. Figure 17 provides a graphical representation of the interaction of base state cigarette tax and large increase in tax, estimated as the marginal odds of continued smoking for 10, 12, and 16 years of schooling. Figure 18 displays the marginal odds ratio for a large increase in cigarette tax at various levels of base state tax estimated for 10, 12, and 16 years of schooling. We see that a large increase in cigarette tax makes quitting more likely in the subsequent two years when the base tax is less than $1.00. When the base tax is greater than $1.00, the odds of quitting without a large tax increase are already quite high so a large increase does not further enhance the likelihood of quitting.

Table 8. Random-Effects Logistic Regression Predicting Stayed Smoker

Person-years	11,747	11,746	11,734	11,733	11,734
Persons	2,692	2,692	2,689	2,689	2,933
States		51		51	50
MODELS:	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**
Coefficients reported as Odds Ratios.					
State Tax (Lagged t-1)	.724***	.593***	.710***	.574***	.672***
	(.041)	(.042)	(..041)	(.043)	(.044)
Large Tax Increase ($0.50 or greater)	--	--	.767	.735°	.757°
			(.127)	(.123)	(.127)
Large Increase X **State Tax**	--	--	1.572*	1.341	1.486***
			(.324)	(.279)	(.309)
Age	.993*	.993*	.993*	.992*	.992*
	(.003)	(.003)	(.003)	(.003)	(.003)
Female	.996	.986	.997	.987	.987
	(.071)	(.071)	(.072)	(.072)	(.070)
Years of Education	.935***	.936***	.931***	.931***	.932***
	(.016)	(.016)	(.016)	(.016)	(.016)
Constant	18.708***	16.317***	20.173***	20.185***	21.859***
	(4.703)	(6.452)	(5.108)	(8.172)	(5.467)
Person Random Intercept	YES	YES	YES	YES	YES
State Random Intercept					YES
State Fixed Effects		YES		YES	

°p<0.10 * p<0.05; **p<0.01; *** p<0.001

Data Source: Panel Study of Income Dynamics, Waves 1999-2015

Figure 17. Odds of continued smoking during any 2-year period from the preferred model. Dashed lines are for a recent tax increase of $0.50 or more. Solid lines are for a recent increase of $0.49 or less (including no change or a decrease).

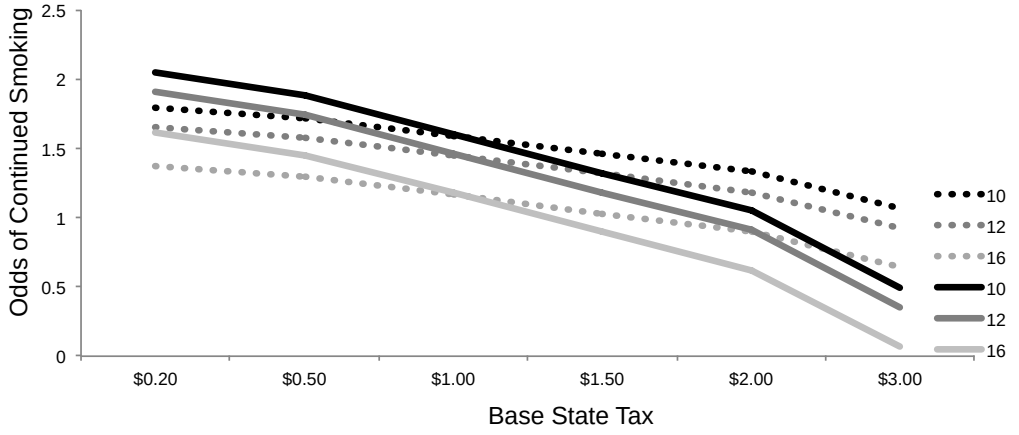
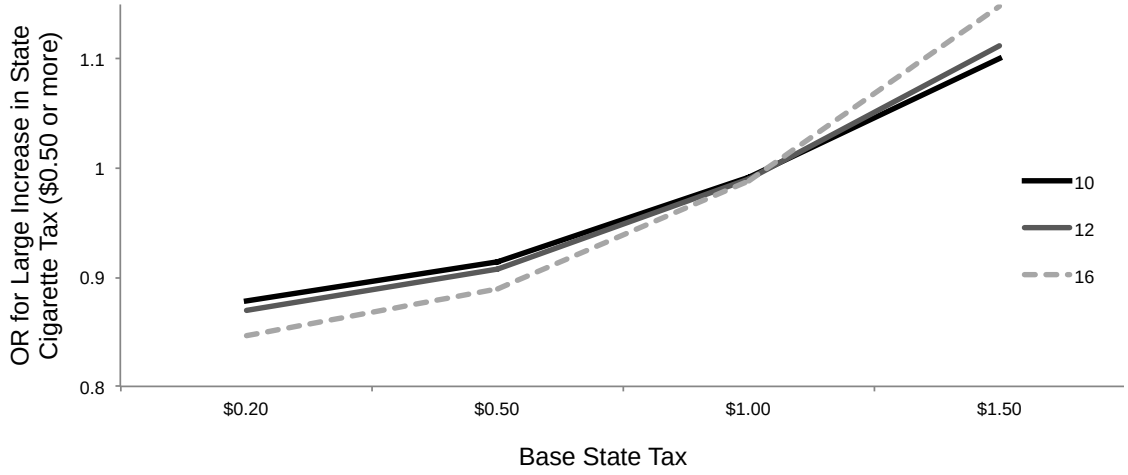


Figure 18. Odds Ratios showing the effect of a $0.50 or more tax increase (dichotomous variable) on the odds of continued smoking by base state tax.



To summarize, I find that smokers are more likely to have quit smoking within two years when they are exposed to a large increase in state cigarette tax compared to a smaller increase or no increase. These results support H4 and suggest that the effect of cigarette taxes on smoking behavior reflects both direct effects through tax increases, but also indirect effects. The effects of cigarette taxes on smoking do not appear to be driven by unobserved variation between states. Instead, it is likely that the strong negative effect of base cigarette taxes on smoking reflects the tendency for cigarette taxes to act as an indicator or signal of general progress in tobacco control.

Discussion

This chapter set out to explore the potential for a state policy, namely, cigarette taxes, to moderate the educational gradient in mortality. I found evidence that higher state cigarette taxes weaken the educational gradient in mortality. It appears that higher taxes have a stronger protective effect against mortality for the less educated, particularly for men. While it is difficult to determine causality, I find a positive association between state cigarette tax and the odds of

quitting smoking, even after controlling for unobserved differences between states. Furthermore, I find evidence that when cigarette taxes in a state are still approximately $1.00 or less, an increase in taxes of $0.50 or more will increase the likelihood of quitting within the next two years. I interpret these results as evidence that state cigarette taxes can and do influence the educational gradient in mortality in an equalizing way.

It is helpful to situate these findings within the Tobacco Control Transition model I described earlier in this chapter. My study period, 1999 to 2015, includes the largest increases in state cigarette taxes to date. My study period also begins 30 years after the first major public report on the health risks of smoking. Thus, the formation of educational disparities in smoking and in smoking-related mortality had already occurred across the U.S. prior to my study period. This is important to note because it is only once educational disparities in the prevalence of smoking exist that state cigarette taxes should have an equalizing effect on the educational gradient in mortality. Further, any causal effect of cigarette taxes on mortality takes time to materialize. A recent study estimated that it takes ten years for cigarette taxes to show up at the population level as detectable gains in life expectancy (Baum et al. 2019). If this is an accurate estimate, tax rates in 1999 should influence mortality rates beginning around 2009.

While it is plausible that my mortality models detect causal effects of cigarette tax increases that occurred since 1999, it is also likely that a state's cigarette tax level at baseline in 1999 accounts for additional unobserved variation between states. For example, Alabama has very low cigarette taxes and Massachusetts has high cigarette taxes placing them at opposite ends of the distribution of state cigarette taxes. But they also sit at opposite ends of the distributions of Medicaid inclusivity, unemployment insurance benefits, public health regulations, and many other political and economic policy variables. Thus, it should be quite easy to show that state

cigarette taxes are merely associated with a weakening of the educational gradient in mortality. It is much more difficult to provide causal evidence that specific increases in state cigarette taxes reduce smoking and, in turn, reduce mortality rates among those with lower education disproportionately such that the educational gradient in mortality lessens.

I have presented evidence in parts: first demonstrating that higher state cigarette taxes are associated with a weaker educational gradient in mortality; then showing a positive association between state cigarette tax and the odds of quitting smoking; and finally showing mixed evidence from longitudinal data that exposure to a large tax increase increases the likelihood of quitting smoking within two years when base taxes are low. We are left to interpret to what extent these findings convince us that cigarette taxes are indeed an example of a single state policy moderating the educational gradient in mortality.

Taken broadly, my results provide compelling evidence that states do indeed vary in the extent to which educational attainment maps onto health and, in turn, onto mortality. How exactly state cigarette taxes disrupt the effects of educational inequality on mortality is less clear. I have considered three possible mechanisms: 1) the direct effect of cigarette taxes on smoking; 2) the indirect effect of state's progress in tobacco control for which cigarette taxes may act as a proxy or indicator; and 3) the effect of a package or clustering of state policies that distinguish states and of which cigarette tax is representative. While I find some evidence for the direct effect of cigarette tax increases on smoking cessation when the base tax is low, this may not be the whole story. The next obvious explanation for my results is that the effect of cigarette taxes is confounded by a myriad of other state policies and state characteristics; however, the effects of cigarette taxes persisted even in the models that explored only variation within states. This leaves

the possibility of an indirect effect due to cigarette taxes acting as an indicator of general progress in tobacco control.

My results are consistent with the idea that cigarette taxes have their equity-promoting effect in part because they signal progress in tobacco control. Considering results from all of the analyses, but particularly the models of continued smoking that controlled for state fixed effects, it is clear that a state's base cigarette tax level is more influential than a tax increase. Cigarette tax increases are thought to have their effect on smoking cessation within the first six months of implementation (Chaloupka et al. 2012). Thus, it is not likely that the strength of the base tax association is due to the lagged effects of previous cigarette tax increases. This suggests there is something about base tax that is influential beyond the effect of a tax increase. Moreover, already in 1999, cigarette taxes moderated the educational gap in smoking such that the gap between individuals with less than high school and individuals with high school or more was largest in the lowest quartile of taxes and smallest in the highest quartile of taxes (see Appendix B, Figure B-4). These trends are detectable prior to the period of large increases in state cigarette taxes across the country between 2001 and 2005. The fact that the magnitude of educational disparities in smoking prevalence are already differentiated by cigarette tax quartile in 1999 may reflect the way that state cigarette taxes can be a signal for general progress in tobacco control.

An alternative interpretation is that the differentiation of educational disparities in smoking by cigarette tax in 1999 is due to cigarette tax increases having their equity-promoting effects only when base tax is still relatively low (e.g., <$1.00). If this is the case, states that raised cigarette taxes early (e.g., California) would have seen a reduction in the educational gap in smoking in the late 1990s, whereas states that raised cigarette taxes later (e.g., Texas) would see the educational gap in smoking reduce in the mid-2000s. This alternative interpretation has

support in the literature. Bishop (2015) found that "the lowest-tax states are those with the greatest power to reduce the national smoking rate." By extension, state cigarette tax increases may be most effective at reducing smoking disparities when base tax is still relatively low. This could be the case for two reasons. First is because the remaining smokers in states with an already high base tax are compositionally distinct from the smokers in the low tax states: a higher proportion of the smokers in high tax states will be "die-hard" smokers who continue to smoke regardless of the price of cigarettes. Thus, the low-tax states have a higher proportion of smokers who are likely to quit in response to a tax increase. The second reason is the possibility of a ceiling effect such that as base tax gets higher, large tax increases become more rare. While there is some evidence of a ceiling effect in models of the *base tax-by-tax increase* interaction, there are several examples of states with relatively high base taxes in 1999 that continue to adopt large tax increases during the study period (e.g., Connecticut, DC, Delaware, Hawaii, Massachusetts, and Washington) (see Appendix B, Figures B-5 and B-6).

My ability to further tease apart the relative contribution of each of the three mechanisms is limited in several ways by my study design. First, many of the sample respondents quit smoking prior to the study period. This is particularly true of the respondents from states with the highest taxes, so I only guess retrospectively based on the mortality trends that they quit in response to increasing cigarette taxes. But it may be that they quit for other reasons as well. Second, my current analysis does not take into account local taxes, which may be higher than state taxes. Still, local taxes are a much more recent trend, and they are blocked in many states by preemption laws. Where they are legal, they are generally very small (<$0.20). Third, I draw on the publicly available PSID data, so my outcome of death is not verified as in the restricted National Death Index data files. In future work, I intend to use the restricted data to compare

trends in cause-specific mortality. Finally, although controlling for state fixed effects helps reduce some of the concern around confounding by other unobserved state characteristics, I was unable to control for state-level confounding in my cross-sectional mortality models.

In summary, higher state cigarette taxes are associated with a weaker educational gradient in mortality, as well as a weaker educational gradient in smoking prevalence. I conclude that cigarette taxes weaken educational disparities in smoking, in part, through a direct effect on smoking cessation when state tax is low and, in part, by acting as a signal of a state's overall progress in tobacco control.

Conclusion

This study uses state cigarette taxes as a case to demonstrate how a specific state policy can shift educational disparities in smoking and mortality. My research begins to reframe fundamental causes, such as educational inequality, as contingent on state policy. While there has been progress in theorizing the processes by which social policies moderate health inequalities (Geronimus 2000; House 2016; Mechanic 2002), much more research into specific policy mechanisms is needed to generate an evidence base that can inform intervention. With its focus on cigarette taxes, my study demonstrates how a specific policy change can modify the educational gradient in mortality by intervening on a single risk factor: smoking. Future research should look for similar effects from state policies in the social and economic domains that influence health through mechanisms that go beyond a single risk factor.

CHAPTER FOUR: ADVANCING THE STUDY OF HEALTH INEQUALITY: FUNDAMENTAL CAUSES AS SYSTEMS OF EXPOSURE

Introduction

Persistent social inequalities in health are a compelling puzzle for health researchers because they seem to defy explanation. For example, there is a strong socioeconomic gradient in heart disease mortality that cannot be fully explained by smoking, obesity, and the other known risk factors for heart disease. And while researchers agree that an individual's socioeconomic status (SES) conveys health benefits in ways that go beyond individual risk factors, there is a tendency to assume that the association is fixed. But even in the stubborn SES-health association, there is variation. This variation can reveal insights about how health inequality is socially produced, but it can only be seen if we zoom out to a broader perspective. Despite calls in the literature for population-level analysis and systems thinking, our scope of investigation rarely goes beyond the boundaries of a single population. This chapter argues that advancing the study of health inequalities will require explicit study of the dynamics in fundamental causes through population comparison. By reframing the fundamental causes of health inequality as *systems of exposure***,** we can bring a comparative lens to the study of health inequalities.

Health inequalities are differentials in disease and death that exist *within* a population. But the most important cause of health inequality is social stratification, and social stratification only varies *between* populations. Thus, as scholars of health inequality, we encounter a mismatch between the level of analysis we think about and the level of analysis we must study to reveal the mutability of fundamental cause associations. This chapter highlights a way forward in the study of health inequality that resolves this dilemma. Here I argue that the explicit study of variation in social stratification is the next frontier in research on fundamental causes of health inequality.

The chapter proceeds in the following way. First, I consider Fundamental Cause Theory as the theoretical basis for the approach I advocate. I highlight two common pitfalls in the application of Fundamental Cause Theory that limit insight into the mutability of health inequalities. Second, I introduce the system of exposure concept. Then, I briefly review some key lessons from the past that inform my call to study systems of exposure. Next, I highlight four studies that succeed in using population-level comparison to reveal the influence of social stratification on health. Finally, I discuss the benefits of this approach for future research.

Fundamental Cause Theory

Fundamental Cause Theory (FCT) is a theoretical attempt to resolve the puzzle of why social inequalities in health (e.g., the socioeconomic gradient in mortality) persist despite medical innovation and disease elimination (Link and Phelan 1995). FCT points to the ways that flexible social resources can be marshaled to protect against illness even in the face of changing times (Link and Phelan 1995). According to FCT, it is social inequality in access to flexible social resources (in particular wealth, income, education, and racial privilege) that drives population health inequalities. The unequal distribution of social resources, in turn, determines general susceptibility. Individuals with high SES can deploy their resources to avoid disease, seek treatment, and adopt healthy behaviors. Further, regardless of whatever the disease or cure of the moment may be, those with higher SES are ensured access to the social determinants of health (Link and Phelan 1995).

FCT critiques the tendency in medical sociology and epidemiology to focus too much on specific mechanisms, which can become obsolete while the underlying relationship with health persists. For example, infectious diseases once accounted for a large SES gap in life expectancy. Despite the transition from infectious diseases to chronic diseases as the most important

contributors to mortality, there is still a large SES gap in life expectancy. In light of this, FCT advocates are searching for fundamental causes that influence population health in ways that are non-disease-specific. FCT specifies four criteria of a fundamental cause: 1) influences multiple disease outcomes; 2) influences disease through multiple risk factors; 3) involves access to resources that can be used to avoid risk or minimize the consequences of disease once it occurs; 4) is associated with health through intervening mechanisms that are replaced over time. Socioeconomic position (Masters et al. 2015; Phelan et al. 2004) and position in racial hierarchy (Williams and Collins 1995) meet these criteria and are the most studied fundamental causes of health inequalities. Other fundamental causes mentioned in the original articulation of FCT are gender stratification, social support, crime victimization, death of a loved one, and job loss (Link and Phelan 1995). Studies of fundamental causes have succeeded in illuminating the ways that social inequality has a remarkably stable influence on health. But the success of efforts to document the existence and persistence of fundamental cause associations, particularly the SES-health association, may have an unintended consequence.

Since its introduction in 1995, Fundamental Cause Theory has grown into, arguably, the most developed theoretical model of how social inequality produces health inequality. FCT has been further articulated by the original authors and others. And while Fundamental Cause Theory has given us an excellent model for how social conditions map onto health, it has fallen short of its original mission to disrupt notions that health inequality is inevitable. Instead, FCT has been used by scholars to frame health inequalities with a new kind of inevitability—one based in the assumed stability of social stratification rather than biological determinism—but it is an inevitability, nonetheless.

Scholars of health inequality are seemingly stuck comparing subgroups and documenting disparities attributable to SES or race or gender. Perhaps this has resulted from the presumptive permanence of the word "fundamental." Perhaps it simply reflects the difficulty of imagining other worlds, but few of the many studies that apply FCT explore variation in the fundamental causes themselves (i.e., variation in racial hierarchy). Instead, the literature continues to confirm the existence of the fundamental cause associations. On the one hand, it is great progress to show that fundamental cause associations exist and drive health inequalities. On the other hand, the more we show they exist and persist, the easier it is to assume fundamental cause associations cannot change. As the evidence documenting health inequalities that result from socioeconomic inequality and racism and residential segregation accumulates, these fundamental cause associations can seem fixed and intractable. Thus, in its emphasis on the persistence of socioeconomic inequalities in health, research on fundamental causes perpetuates the assumption that social stratification is static.

Goal: Study Dynamics in Fundamental Cause Associations

FCT has inspired an exciting area of research on the ways that social policy can intervene to disrupt the social production of health inequality. This research is focused on social policies that weaken fundamental cause associations by ensuring universal access to the social determinants of health regardless of an individual's social resources, power, or privilege. For example, studies have shed light on the ways that school desegregation (Liu et al. 2012), anti-immigrant laws (Torche and Sirois 2019), unemployment insurance (Cylus et al. 2014), and state policy regimes (Montez et al. 2019) can prevent or exacerbate social stratification from turning into health inequalities, even without any direct intervention on the system of social stratification itself.

But this focus on policies that disrupt the extent to which individuals can "buy" their way into good health with flexible social resources is only one side of the equation. The associations between a fundamental cause and health inequalities can change in not one, but two main ways: either what a particular position within the stratification system means for health can change, or the patterning of the underlying stratification system itself can change. While research on the first type of change is advancing, there has been limited attention to the second type of change. The ways that the patterning of social stratification itself can change has been a more elusive area of study for health researchers.

Why has there been limited attention among health researchers to social policies and unplanned social changes that flatten systems of social hierarchy? I think there are two reasons for the neglect that have their basis in how we conceptualize fundamental causes. First is the tendency to accept the existence of fundamental causes, such as socioeconomic stratification, as natural. Second is the usual mismatch between our level of analysis in studying fundamental causes and the level at which stratification systems are defined. These common practices restrict the potential for studies to reveal the mutability of health inequalities. In this chapter, I propose reframing fundamental causes as systems of exposure in order to resolve these two common pitfalls in the application of FCT.

Pitfall 1: Assuming Social Inequality is Static

There is an enduring tendency among medical professionals to look for the basis of health disparities in biology. The study of fundamental causes has helped change thinking in the medical and public health communities away from biological determinism. Instead of searching for the roots of health disparities in genetics, proponents of FCT generally locate the roots of health disparities in social inequality. This is indeed progress that should be celebrated. Yet the

tendency to naturalize health disparities persists. FCT has failed to overcome the assumption that there is something immutable about disparities by gender, race, and social class in health. Perhaps this is because the concept of fundamental causes allows health researchers to merely transfer their assumptions about the basis for the fixed nature of inequalities in health from biology to social stratification. And among many health professionals, social stratification is assumed to be just as immutable as genetics.

Like draping a cloth over a table, health trends reveal the shape of even hidden systems of social stratification. Indeed, health can make visible social inequalities assumed to be natural in our society. Herein lies an opportunity. Population health data can be used to study dynamics in systems of social stratification. But rarely do researchers consider the ways that fundamental cause gradients are context-specific and dynamic. Instead, current theory and methods in epidemiology and medical sociology make it easy to accept that there is a "natural" order to social inequalities in health. This is evident in the way health disparities researchers make comparisons to a White reference group unquestioningly and in the way that the persistence of Black/White or Native American/White disparities in health and life expectancy are taken as the way things are. It is also evident in the tendency to view any divergence from White advantages in health as a paradox (see Palloni and Morenoff's 2001 critique of the "Hispanic Mortality Paradox") or puzzle (see Navarro's 2019 critique of Case and Deaton's "deaths of despair" puzzle) or to ignore them completely (e.g., Asian American and Pacific Islander health, see Chen and Hawks 1995). Although the public health literature does show increasing comfort with the idea that race is a social construct, race is still conceptualized as a fixed individual-level trait. Health researchers lack models that reject notions of a natural social order and instead take the social construction of racial stratification—and other forms of stratification—as a starting point.

Omi and Winant (2014) state it plainly, “Race is a way of ‘making up people.’” Racial identities are not stable concepts. Rather, they are constantly being renegotiated and reformulated with consequences for population-level rules (de facto and de jure) of social inclusion and exclusion. For instance, following World War II, political organizing and mobilization transformed racial stratification in the U.S. (Omi and Winant 2014). Uprisings in the 1950s and 1960s challenged the Jim Crow-based system of white supremacy and succeeded in achieving partial reforms (Omi and Winant 2014). But these political movements were met with a backlash in the 1970s and 1980s based in protection of a white supremacist racial hierarchy. Despite some progress, hopes of total transformation of American racial hierarchy waned.

Nonetheless, health is surprisingly sensitive to even subtle shifts in racial order. So while white supremacy continues in the U.S., there are both acute and gradual shifts in racial and ethnic stratification that have occurred in recent decades that have consequences for health inequality. For example, a change in ethnic stratification occurred during the postwar period with the absorption of hyphenated Whites into an undifferentiated White majority. This is an example of a gradual transformation that likely has had gradual consequences for population health. Just as ethnic distinctions can lose their power to differentiate and exclude, ethnic distinctions can emerge as a new basis for social exclusion and, in turn, for health. The attacks of September 11, 2001, major immigration raids, and anti-immigrant laws are each examples of events that transformed the links between ethnic hierarchy and health, or between immigration status and health. Thus, studies of these dynamics in stratification systems can be very instructive for understanding the social production of health inequality. While less common, there is research being done that avoids the pitfall of assuming social stratification is static. Later in this essay, I

highlight four examples from the literature that explore the health consequences of a change in the patterning of a system of social stratification.

Pitfall 2: Mismatched Levels of Analysis

In order to study dynamics in stratification systems, we have to zoom out enough to detect variation. This is impossible with research designs that compare individuals, yet the individual-level is the most common level of analysis for studying the fundamental causes of health inequality. We tend to study the health consequences of socioeconomic inequality or racial hierarchy as differences between individuals (e.g., rich and poor, or White and Black). This is because we mistakenly conceptualize fundamental causes as individual attributes. This kind of conceptual slippage away from population-level thinking is so common it has its own name in the literature: "lifestyle drift" (Hunter et al. 2009). Herein lies the pitfall of mismatched levels of analysis, which I argue has limited our study of fundamental cause associations.

Health inequalities are differentials in disease distribution among subgroups or between different social positions that exist *within* a population. Yet the most salient cause of health inequality is social stratification, which only varies *between* populations. This mismatch of levels of analysis has confused efforts by researchers committed to understanding social inequalities in health. Our research suffers from "the streetlight effect"—an observational bias to look for things where there is light, not where we are most likely to find what we are looking for. This is why the study of fundamental causes has been limited up to this point. But an opening can be found in Rose's (1985) call to study ubiquitous exposures across populations and in a related call for systems thinking in health research (Diez Roux 2007). In order to advance research that reveals how health inequalities within a population are modifiable, we actually must look across populations.

Solution: Reframe Fundamental Causes as Systems of Exposure

So how do we move beyond documenting the influence of fundamental causes to exploring their mutability? I propose we build on FCT to reframe fundamental causes as *systems of exposure* to the determinants of health that vary across populations.

We can resolve the two common pitfalls in applications of FCT by recognizing a special case of fundamental causes that are actually ubiquitous exposures: stratification systems. The patterning that stratification systems exhibit within a population can trick us into thinking that their influence on health can be studied by comparing subgroups within a population. Indeed, it is tempting to think of fundamental causes as we do any other risk factor or exposure: as a determinant of health risk that is either present or absent. But some fundamental causes, such as racism, are not merely exposures. Rather, they are systems of exposure—not everyone is exposed to the same disease agents, but everyone is included in the system that shapes exposure to the determinants of health.

Accordingly, my proposition is this: to the extent that a particular fundamental cause stratifies exposure to the determinants of health, we should think of it as a system of exposure, not an individual trait. Stratification systems are actually population-level attributes, so they must be studied across populations or population moments. This approach offers ways to address the constraints that come from an intense focus on fundamental causes without an equally intense focus on population-level analysis.

As I mentioned previously, there is growing interest in the potential for policy (national, state, and local) to modify the influence of social inequalities on health. This exciting direction for research requires theoretical grounding. A systems of exposure reframing makes FCT more relevant to research on local policy environments and health. Thinking about fundamental causes

as systems of exposure helps us bring FCT along as we study variation in the educational gradient in mortality across states or variation in the effects of cigarette taxes on health. Further, we need an approach that is accessible to health researchers. It can be intimidating to try to think about social stratification systems. This is why I propose thinking instead about systems of exposure, a concept that emerges directly from the epidemiology and medical sociology literatures. It invites health scholars to acknowledge that stratification systems are dynamic and to think explicitly about variation in stratification systems without asking that they incorporate theory from other disciplines.

Origins: How the Systems of Exposure Concept Integrates Insights from the Past

In 1985, sociologist Stanley Lieberson introduced the idea of basic causes, which he distinguished from superficial causes. A change in a basic cause will actually transform the dependent variable—for example, the racial gap in income. In contrast, a change in a superficial variable will have no direct consequence for the income gap. Lieberson cautioned, "It is rarely possible to distinguish between basic and superficial causes if the research is working exclusively with data for a single point in time" (Lieberson 1985: 186). He also explained that basic causes can have their influence through multiple mechanisms, but the effect on the income gap will be the same.

House and colleagues (1994) picked up on Lieberson's idea of basic causes in their paper on socioeconomic stratification in health and aging. House and colleagues concluded that "the social stratification of aging and health is arguably a fundamental outcome of our social stratification system itself." They even called for "comparative and historical research on variation in social stratification," which is precisely what I advocate for in this essay. A year later, Link and Phelan (1995) introduced Fundamental Cause Theory. Building on Lierberson

(1985), they explained how individuals with higher SES can translate their social privilege into better health regardless of changes in the disease landscape.

Meanwhile, in the same year that Lieberson introduced his idea of basic causes, epidemiologist Geoffrey Rose (1985) published a now classic piece, "Sick Individuals, Sick Populations." In the piece he described the challenge of studying the influence of ubiquitous exposures with our tendency for individual-level comparison. Rose (1985) distinguished between the causes of individual cases of disease and the causes of population incidence rates. He used the example of a population distribution of hypertension in England and Kenya to point out that simply comparing two Englanders, one case and one control, and asking why one has hypertension and the other does not will not reveal why England has much more hypertension than Kenya. Generally speaking, ubiquitous exposures that have uniform population distributions do not shape within-population health inequalities. Rather, they drive inequalities between populations. Uniformly distributed ubiquitous exposures are the most difficult to detect because they have no influence on the distribution of cases, and yet the main thing we know how to study with our methods is the distribution of cases. Our methods for causal inference leverage heterogeneity of exposure. Rose argues that to understand the causes of population-level attributes such as ubiquitous exposures, we must compare across populations. In 2004, Diez Roux re-issued Rose's call for the comparison of different populations to investigate population-level attributes (Diez Roux 2004).

While there is similarity in the motivation behind Rose's conceptualization of ubiquitous exposures and Link and Phelan's fundamental causes, they are distinct concepts. According to Rose (1985), ubiquitous exposures explain the incidence rate of disease in a population, and they can be used to understand the basis for differences in disease incidence between populations.

Link and Phelan's fundamental causes explain the differential incidence of disease between population subgroups (Link and Phelan 1995). But what I want to emphasize is that there is a special case of fundamental causes that can and should be studied as ubiquitous exposures, namely, stratification systems.

It may be helpful to clarify here what I mean by stratification. Massey defines stratification as "the unequal distribution of people across social categories that are characterized by differential access to scarce resources" (Massey 2007: 1). Stratification systems order social categories such that higher positions get more access to social resources and power at the expense of the lower order positions. This results in social inequality. Stratification systems are defined at various social scales, and they vary greatly across populations and over time.

My proposal to reframe fundamental causes as systems of exposure draws on Rose's insights about the importance of population-level comparison. As I depict in Figure 19, stratification systems sit at the intersection of ubiquitous exposures and fundamental causes. Rose explained that the study of ubiquitous exposures requires comparison at the scale at which the exposure varies. For example, we cannot effectively study the effect of American culture on premature mortality by comparing one American who died prematurely to another who did not. As Rose reminds us, in order to see the effect of uniformly-distributed ubiquitous exposures, we have to zoom out to compare between populations with differential exposure to the ubiquitous cause. For example, to see the effect of "England" on hypertension, we need to compare to a population distribution that does not share the uniform exposure to England.

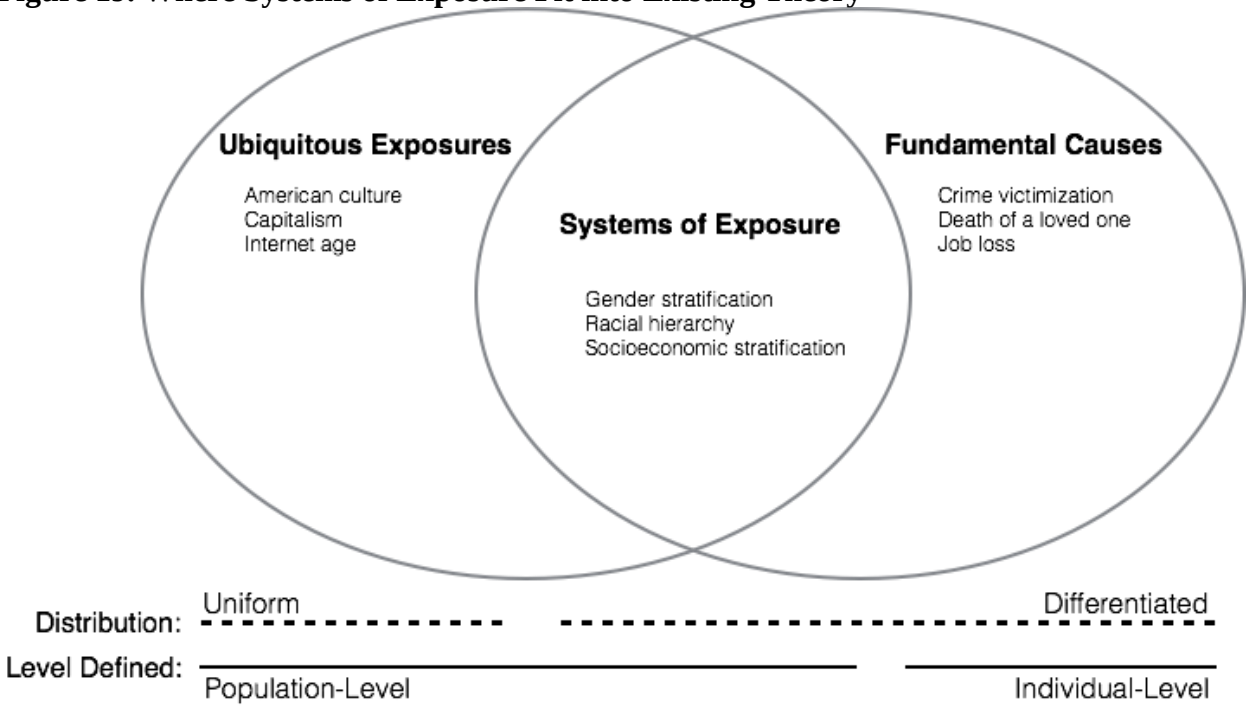
Stratification systems are special because they can be conceptualized as both ubiquitous exposures and fundamental causes. As such, they can help us uncover both causes of population incidence rates (which satisfies Rose 1985) and causes of health inequality (which satisfies Link

and Phelan 1995). For our research on health inequality, it is helpful to conceptualize stratification systems as systems of exposure because it reminds us that we are looking to compare these stratification systems across populations. Local systems of racial hierarchy are systems of exposure. National systems of gender stratification are systems of exposure. The stratification of regions within the U.S. is a system of exposure. We find them in a population well-circumscribed in time and space. Ultimately, they are the systems that determine the patterning of social resources and power.

Stratification systems are distinct from other ubiquitous exposures in that they do not have a uniform distribution across a population. Their differentiated patterning is precisely what is dynamic and thus extremely useful for health researchers if studied explicitly using systems thinking. The term "systems of exposure" reminds us we cannot study these exposures as we do individual-level risk factors. All of us hold positions in stratification systems. There is no absence of exposure.

Not all fundamental causes are systems of exposure that only vary between populations. Job loss is an example of a fundamental cause cited by Link and Phelan (1995), which is not a ubiquitous exposure. Some individuals are exposed to job loss and spells of unemployment while others are not. While the social meaning of job loss certainly varies across populations, job loss is not usually conceptualized as a stratification system. Similarly, crime victimization and death of a loved one (also among the examples of fundamental causes provided by Link and Phelan 1995) are not ubiquitous exposures. These in themselves are not population-level attributes that can be compared across populations.

Figure 19. Where Systems of Exposure Fit into Existing Theory



How: Articulating Systems of Exposure

The s*ystem of exposure* concept invokes systems thinking; this is on purpose. There has been growing interest in systems theory among health scholars (Diez Roux 2007, 2008, 2015). General systems theory involves intentionally studying a system's dynamics and evolution. Systems theory takes a holistic approach and does not reduce systems to simply the sum of their parts. In these ways, systems thinking is both "a healthy antidote to the obfuscation that can result from too much simplification" (Diez Roux 2015), but also a way to handle greater complexity.

By thinking of a fundamental cause, such as structural racism, as a system, we can more easily think of its properties: a) What is the system's patterning (e.g., continuous, threshold, dichotomous, ordinal)? b) At what scale is the system defined (e.g., national, regional, state,

local)? c) How is the system maintained or enforced? d) How do we theorize the system's influence on susceptibility (e.g., cumulative, fixed, reversible, critical periods, dynamic over the life course)? This simple reframing of fundamental causes as systems of exposure urges us to theorize more clearly how we expect fundamental causes to vary, and then to conduct studies designed to detect that variation.

Scale

To study fundamental causes as systems of exposure, we must locate them in populations. We must find the scale at which the fundamental cause is assigned its relative meaning. Most systems of exposure can be defined at multiple possible population scales (Matthews and Yang 2013). The scale can be any social unit large enough to have its own norms, rules, and patterning, as well as its own distribution of disease.

When we think about the scale at which to study a system of exposure, we should ask: 1) At what level is the system assigned meaning? 2) At what level can this meaning be transformed or disrupted? 3) At what level could we intervene? Adapting Matthew's concept of "spatial polygamy," people belong to multiple nested and non-nested, social and geographic, past and present contexts (Matthews 2011). Each of these can be studied as a population. For example, racial hierarchy as a system of ubiquitous exposure can be studied at the scale of New Orleans, Louisiana, the South, or the U.S. Each of these levels represents a distinct population unit with its own system of racial hierarchy that exerts an inescapable influence on its members. Granted the nesting of the population scales results in tremendous similarity and influence between each system of racial hierarchy, but there are also subtle distinctions that can be studied to reveal important insights about power and health.

Place-Time Population Coordinates

Variation in systems of exposure tends to be either spatial or temporal. To study this variation, it helps to draw comparisons across populations with different place-time coordinates. Since they are population-specific, we can think of each particular fundamental cause association as having a place-time coordinate. This is just a systems-thinking way of expressing that the fundamental cause association we observe is context-dependent. The place coordinate locates the spatial scale at which they are defined and given meaning. Distinct systems of educational inequality can be defined and compared at the national, regional, state, county, and school-district levels. For example, a recent study by Sheehan and colleagues showed differentiation in the functional form of the educational gradient in mortality by region in the U.S. (Sheehan et al. 2018).

The time coordinate corresponds to the temporal scale, the time period during which the fundamental cause as a system of exposure is stable. For example, racial disparities in teen pregnancy can be studied immediately before and immediately after the transitions away from de jure racial segregation (Liu et al. 2012). This particular study found that the temporal change in the intersecting systems of racial and educational stratification reduced health inequality (Liu et al 2012). Another recent study compared the educational gradient in chronic disease across four time periods in Brazil and found evidence that a gradual weakening of the educational gradient resulted in smaller educational inequalities in chronic disease (Beltran Sanchez and Andrade 2016). In this example, the place time coordinates compared were: Brazil 1998, Brazil 2003, Brazil 2008, and Brazil 2013.

Patterning

Our recognition of fundamental causes is possible precisely because of the existence of social gradients in health. The gradation or stratification in a social condition is what creates the possibility for detectable variation in health or mortality. To borrow a classic example of ubiquitous exposure from Rose (1985): if everyone in a population smoked a pack of cigarettes a day from age 18 onward, we would likely assume that lung cancer was a genetic disease because the main source of variation would be biological. Under this ubiquitous exposure, it would be nearly impossible to detect the influence of smoking on lung cancer. Unless we compare temporally across birth cohorts or spatially across populations or find experimental ways to vary the cause's patterning, we will not detect a ubiquitous cause. It is easier to detect fundamental causes with a dichotomous or categorical patterning because this is more visible to us and fits the human proclivity toward categorization. Gradients (i.e., continuous distributions of resources) are much harder to see if we are not explicitly looking for them. Similarly, stratification systems that influence susceptibility dynamically or gradually over time rather than in fixed ways or acutely during critical periods are harder to study with current methods.

Socioeconomic status is both a fundamental cause and a ubiquitous cause. The reason socioeconomic status is detectable is because it exhibits a patterned variation at the population level. The same is true for the effects of racial hierarchy or residential segregation. Those of us interested in disrupting the social production of health inequalities can, therefore, use theory to hypothesize what the pattern of variation should look like, and then compare populations that should differ in the system of exposure to see if the pattern changes.

System Enforcement

Another important way conceptualizing systems of exposure counters the tendency to naturalize stratification is by emphasizing how stratification systems are produced and maintained through laws, culture, institutions, and social interaction. We can use the legal terminology of "de facto" and "de jure" to specify whether a system of exposure is enforced formally through laws and policy (de jure enforcement), or informally through norms and culture (de facto enforcement). It is helpful to specify system enforcement to our best ability because, as we see in studies of school desegregation (Liu et al. 2012) and anti-immigrant laws (Torche and Sirois 2019), changes in system enforcement, from de facto to de jure or the reverse, can alter fundamental cause associations with health outcomes.

In summary, stratification systems are dynamic. By ignoring their dynamism, we miss an opportunity to learn about the influence of fundamental causes on health. Studying dynamics in social structures, such as stratification systems, can reveal the contingent nature of fundamental cause associations. It can uncover how these seemingly intractable health disparities are created and maintained through social policy, institutions, and culture.

Examples: Articulating Systems of Exposure in Existing Research

Next I highlight four studies that succeed in doing comparisons across temporal or spatial populations. These studies leverage a sharp shift in social meaning of relative positions with a stratification system to show that social stratification has consequences for health.
Though they are not framed by the authors in these terms, I hope that by reframing the investigations as studies of systems of exposure, I can both demonstrate the ease of applicability of the approach and hold up these studies as models for future work. (See Table 9 on next page.)

Table 9. Articulating Systems of Exposure with Four Examples from the Health Inequalities Literature

	System of Exposure	Scale	System Patterning	Patterning of the theorized fundamental association over the life-course	System Enforce-ment	Comparison of Place–Time Population Coordinates	Outcome(s)	Found variation in system of exposure?
Beltran-Sanchez et al. 2016	SES Gradient **X** Ethnic Hierarchy	Nation	*Intersectional:* Continuous SES gradient **X** Ordinal Ethnic Hierarchy	Cumulative, with discontinuity in exposure upon immigration	De Facto	Spatial: (Mexico, Mexican-born, 2006); (U.S., Mexican-born 1999-2010); (U.S., U.S.-born Mexican American, 1999-2010); (U.S., U.S.-born Non-Hispanic White 1999-2010)	Prevalence of Metabolic Syndrome	Yes
Lauderdale 2006	Ethnic Hierarchy	Nation	Ordinal Ethnic Hierarchy	Critical period	De Facto	Temporal: (California, 2000-01) (California, 2001-02)	Incidence of low birth weight and pre-term birth	Yes
Krieger et al. 2013	Racial Hierarchy	Region	Gradient	Critical period	De Jure and De Facto	Temporal: (Jim Crow polity, 1940-64); (Jim Crow polity 1965-2006) vs. (Non-Jim Crow polity 1940-64); (Non-Jim Crow polity 1965-2006)	Incidence of infant death	Yes
Novak et al. 2017	Immigration Status **X** Ethnic Hierarchy	State	*Intersectional:* Dichotomous Immigration Status **X** Ordinal Ethnic Hierarchy	Critical period	De Jure and De Facto	Temporal: (Iowa, 2007-08); (Iowa, 2008-09)	Incidence of low birth weight	Yes

Lauderdale, D. S. (2006). Birth outcomes for Arabic-named women in California before and after September 11. *Demography*, *43*(1), 185–201.

In this study, Lauderdale used a temporal comparison to demonstrate the effects of 9/11 on adverse birth outcomes among babies born to mothers with Arab-sounding last names in California. Lauderdale found that the surge in anti-Arab sentiment that followed 9/11 was associated with increased risk of preterm birth and low birth weight among babies born to Arab American mothers. Lauderdale acknowledged a key strength of her study: "By focusing on a period effect at the population level, this study circumvents the complexities and ambiguities of subjective reports of discrimination experiences."

Lauderdale hypothesized the biological mechanism for the health effects of 9/11 was exposure to acute stress during fetal development. Applying system-of-exposure thinking, 9/11 can be conceptualized as a mass disruption of the existing system of ethnic stratification in the U.S. such that Arab Americans became excluded from social resources in a new way. Arab Americans went from being "White, but not quite White" prior to 9/11, to being distinctly-racialized "others" after 9/11 (Jamal et al. 2008). Thus, the system of exposure under study was ethnic stratification in California 2000–2001, contrasted with California 2001–2002. Prior to 9/11, Arab Americans were positioned along the periphery of the White majority. After 9/11, Arab Americans dropped to a lower status in the ethnic hierarchy, making them targets of ethnic discrimination. Lauderdale defined the system of exposure at the national scale and studied it at the state scale. The system's enforcement is de facto, and Lauderdale detailed how increased violence and workplace discrimination against Arab Americans as an ethnic group influenced Arab American pregnant women across California. Integrating a Life Course Perspective, Lauderdale tested for effects during a critical period (sensitive period) of exposure: fetal development. Birth outcomes are like the canary in the coal mine for population health research

because of their sensitivity to even subtle shifts in systems of exposure. With its population-level comparative design and careful attention to dynamics in racial/ethnic discrimination, this study avoided the two pitfalls I discussed previously and made a major advance in research on how racism/ethnic discrimination impacts health.

Novak, N. L., Geronimus, A. T., & Martinez-Cardoso, A. M. (2017). Change in birth outcomes among infants born to Latina mothers after a major immigration raid. *International Journal of Epidemiology*.

Novak and colleagues use a temporal comparison of birth outcomes at the scale of state population to test the effects of a major immigration raid. Applying the system of exposure concept, we can reframe their exposure of interest as the intersecting stratification systems of immigration status and ethnic hierarchy. A major immigration raid in 2008 plausibly altered the social meaning of immigration status and Latino ethnicity in the state of Iowa. While the immigration raid was a sharp change in de jure stratification system enforcement, the larger system of ethnic hierarchy persists with de facto enforcement. Novak and colleagues compared the incidence of low birth rate from Iowa 2007–2008 to Iowa 2008–2009 across Latino ethnicity-by-immigration status subgroups. Like Lauderdale, the authors leveraged the critical period of fetal development to detect the system change.

Krieger, N., Chen, J. T., Coull, B., Waterman, P. D., & Beckfield, J. (2013). The unique impact of abolition of Jim Crow laws on reducing inequities in infant death rates and implications for choice of comparison groups in analyzing societal determinants of health. *American Journal of Public Health*, *103*(12), 2234–2244.

Krieger's work exemplifies the spirit of my call to study systems of exposure. In this 2013 study, Krieger and colleagues intentionally studied dynamics in race relations, stating that "conceptualizing Jim Crow legislation as a political determinant of health shifts the focus from 'race/ethnicity' to race relations as a causal exposure." The end of de jure racial stratification

through Jim Crow legislation marked a dramatic transformation of the system of racial stratification in Jim Crow polities and in the U.S. as a whole. While Lauderdale and Novak and colleagues focused on a sharp disruption in a system of exposure and looked at the immediate consequences, Krieger and colleagues took a longer view. But they still leveraged the sensitivity of the critical period in early life in their choice of infant death as the health outcome. By comparing populations distinguished by region (i.e., states with Jim Crow legislation vs. states with no Jim Crow legislation) and time scales (i.e., years pre- and years post-1964 Civil Rights Act), Krieger and colleagues found a convergence in infant death rates between Blacks in the Jim Crow states and Blacks in non-Jim Crow states in the period immediately after the 1964 US Civil Rights Act, which ended de jure racial segregation through Jim Crow laws. A major strength of this study is that it moves beyond the default of Black-White subgroup comparison to compare Blacks across population coordinates. Krieger and colleagues' careful attention to racial stratification as a dynamic system helped them design a study that revealed how the influence of race on infant mortality is changed by our laws.

Beltrán-Sánchez, H., Palloni, A., Riosmena, F., & Wong, R. (2016). SES gradients among Mexicans in the United States and in Mexico: A new twist to the Hispanic paradox? *Demography*, *53*(5), 1555–1581.

Beltran Sanchez and colleagues' (2016) paper on the Hispanic paradox is an example of the kind of theory-informed, population-level comparison approach that systems-of-exposure thinking can prompt. Beltran Sanchez and colleagues compared educational gradients in metabolic syndrome between four populations: Mexicans in Mexico, Mexican immigrants to the U.S., U.S.-born Mexican Americans, and U.S.-born Non-Hispanic Whites. They explicitly theorized schooling levels as a proxy for SES and as "markers of the opportunity structure available to people and their actual social position" (Beltran Sanchez et al. 2016). With this

comprehensive population-level comparison, they were able to test multiple hypotheses related to the influence of SES gradients and ethnic stratification on health. They dismissed the possibility that weaker SES gradients in health among Mexican immigrants are due to low SES gradients in Mexico (Beltran Sanchez et al. 2016). In addition, they found that Mexican immigrants in the U.S. experience significantly weaker SES gradients than the Non-Hispanic White population (Beltran Sanchez et al. 2016).

The four studies I highlighted found variation in fundamental cause associations by looking at the effects of natural system perturbations on a specific health outcome. But applying the logic of FCT, the fundamental cause associations demonstrated in these four studies almost certainly influence multiple health outcomes. So each of these studies can be viewed as a springboard for further work to document influence of these same systems of exposure on additional health outcomes. Much more research on the social processes that create and maintain health inequality is needed to generate an evidence base that can inform policy interventions (Geronimus 2000; House 2016; Mechanic 2002).

These studies show the value of turning our search for fundamental causes of health inequality to the study of differences between contexts, and specifically, to the study of dynamics in systems of exposure. There are three key benefits to reframing the fundamental causes of health inequality as systems of exposure. First is flexiblility of social scale. Second is an intersectional perspective. Third is a relational perspective. I discuss these benefits to future research briefly in the section that follows.

Benefit 1: Flexibility of Social Scale

Although health researchers tend to study "populations" at the national scale, stratification systems also exist at smaller scales of aggregation. An approach that reframes

fundamental causes as systems of exposure urges us to think flexibly about the social scale at which the system will be defined. To illustrate this, consider racism. Racism has been theorized as a fundamental cause (Phelan and Link 2015). Racism or racial hierarchy tends to be thought of as a national stratification system, which it is. But racism *as a system of exposure* also varies greatly by region within the U.S., by state within the U.S., and even by city. Once we reframe fundamental causes as systems of exposure to the determinants of health, we see that the system is actually context-specific. Its patterning varies depending on the social scale at which it is defined and maintained. For example, racial segregation in the American South in 1950 was a distinct system of exposure from racial segregation in Louisiana in 1950, and distinct still from racial segregation in New Orleans in 1950. As I argued previously, systems of exposure should be studied across population moments—a glimpse of a population in a particular spatiotemporal context. But we need not look for systems of exposure only in national population moments. Rather, with systems of social stratification, the meaningful scale can be a much smaller area such as a city or even a neighborhood. The scale need only be a social context with an intact system of social stratification that distributes access to the determinants of health. This flexibility is important because we can and should be studying the ways that social stratification varies at subnational levels.

The system of exposure concept is also flexible beyond geography. We can apply it to social conditions that are not formal stratification systems, but that can differentiate stigma and power nonetheless. Whether a specific variable works as a system of exposure depends on how we theorize it. The key is to articulate the social condition as a system that differentiates social resources and power. For example, to the extent that body size (i.e., fatness) carries varying degrees of stigma in different places, it could be studied as a system of exposure. Obesity has a

different social meaning in the U.S. than it does in France, or than it does in the poorer countries in the Global South. Here is an instance where system of exposure framing can be used as an insight-generating practice. If we think about the ways that being fat carries stigma that differentiates social resources, we may unlock new insights about the obesity epidemic.

Ultimately, the practice of articulating systems of exposure advances how we study the social production of health inequality. It matters little if a social condition truly warrants being classified as a "system of exposure" or not. More important is using the concept to locate, imagine, and articulate to the fullest extent possible the systems of exposure that shape access to the determinants of health. Doing this helps clarify how far we need to broaden the scope of our research and what an effect might look like. In this sense, reframing fundamental causes as systems of exposure helps guide our research so that we can identify social processes that modify health inequalities.

Benefit 2: Intersectional Perspective

So far, I have only discussed systems of exposure that involve a single system of stratification, a single fundamental cause association (e.g., racism). However, we can also think of places as systems of exposure (e.g., the American South). Places with meaningful social and political boundaries act as containers for intersecting systems of exposure to the resources needed to live a long, healthy life. Thus, in the same way that the American South could be theorized as a fundamental cause, it can be framed as a system of exposure. In this case, the system of exposure is intersectional—it involves multiple overlapping and intersecting stratification systems. Intersectional systems of exposure are shaped by a multitude of intervening factors: demographics, culture, history, policy, and geography. Distinct combinations of these factors create variation in social hierarchies and in the extent to which overlapping

social hierarchies determine health. Attention to this variation can reveal how social policies change that distribution of power and resources, and how in turn, they change health inequalities.

As mentioned previously, contextual variation in health inequalities is due in part to differences in social hierarchies themselves, and in part to differences in the extent to which health can be "bought" with social resources. In places where policy, culture, and even geography make it harder to buy one's way into good health, we might expect to see a flatter social gradient in health than in places where social position is rewarded by way of health. Likewise, in spatiotemporal contexts averse to government regulation that lack initiatives to ensure access to the determinants of health, stratification by SES and race will be more deterministic in its influence on health trajectories and mortality. The result is that in some places poverty is more likely to condemn someone to a shorter, sicker life because of the ways it is further stigmatized by an intersecting social hierarchy. States in the U.S. are a good example of intersectional systems of exposure. They encompass multiple intersecting stratification systems, such as socioeconomic stratification, racial hierarchy, and residential segregation. Further, with the rise of state's rights in recent decades, states are increasingly able to shape the distribution of social resources and control access to the social determinants of health (Montez et al. 2017; Nathan 2005). Thus, research that compares systems of exposure between states or over time is a promising direction for future research.

Benefit 3: Relational Perspective

Fundamental Cause Theory tells us that social inequality is linked to health inequality, not just because of the constraints placed on people with low status but also because of the health advantages enjoyed by those with high status (Link and Phelan 1995; Clouston et al. 2016). So a focus on resource distribution is productive because it moves us away from "theorizing health

deficits," a common pitfall of health disparities research, and instead gives us traction to think relationally about how health inequalities are socially produced (Wilkinson and Picket 2006). This is one of the major contributions of FCT. We come to see fundamental causes not merely as risk factors that are present or absent, but as positions within a system of resource distribution—a stratification system. Every position is relational.

However, in the application of FCT, there is a strong tendency to lose the relational perspective and to think of fundamental causes as just other individual risk factors. Reframing fundamental causes as systems of exposure emphasizes the relational nature of health inequality. For example, racism is a system of exposure to the determinants of health. Everyone holds a position in the system of racial hierarchy. Although the health effects of racism on White people are rarely studied explicitly or conceptualized as dynamic, they should be. White people are not just the reference category; they are the beneficiaries of racism as a dynamic system of exposure. Studying racism and other forms of stratification as systems of exposure helps bring out the way that "unmarked" privileged categories may convert their power and privilege into health through the exploitation of others.

In studies of health inequality, the dominant group is often assumed to be unmarked by health disadvantage. For example, in studies of the negative health effects of irregular work schedules among low-wage workers, the beneficiaries of their labor are ignored. Studies demonstrate that graveyard shifts and highly variable shift work are disruptive for sleep and worsen physical and mental health among the workers. But what about the people who benefit from this labor? What about the health benefits that come from the convenience of being able to buy anything at any hour of the night? To what extent does the adage "my loss is your gain" apply to health? These are the kinds of questions we can explore with a relational perspective. A

focus on the relationships between positions within a system of exposure moves us to consider exploitation. Health disadvantages may not be linked to health advantages in a way that is outcome-specific. As FCT reminds us, fundamental cause associations persist by influencing multiple health outcomes. Therefore, it is possible that a disadvantage in one health outcome is connected to an advantage in another health outcome through a relationship of exploitation. In general, the relational aspects of fundamental cause associations are undertheorized. Hopefully, thinking about systems of exposure will generate new hypotheses about the relational processes that produce health inequalities.

Conclusion

In this chapter, I have highlighted an opportunity to advance how we study the association between systems of social stratification and health inequalities. The goal in reframing fundamental causes as systems of exposure is to produce research that illuminates dynamics in the influence of social stratification on health and also to reveal the potential for context to modify social gradients in health. Articulating systems of exposure and their variation across contexts can make visible the connections between the policies and institutions we support or oppose and the health disparities we tolerate. Until the general public sees the influence of social inequality on health as something that is both real and modifiable, we will not have the new thinking needed for change.

CHAPTER FIVE: CONCLUSION

This dissertation is focused on the social production of health inequalities in and across state and regional contexts. In the previous chapters, I considered the ways that population health is shaped by regional context, state policy, and social stratification. These chapters underscored that health inequalities cannot be understood without attention to social context.

My findings from Chapter Two highlight the potential for regional context to shape health through its influence on identity and social integration. This alerts us to the ways that the timing of contextual exposures matters for health because of the ways that the context in which we grow up becomes integrated into our social identity. While Chapter Two focuses specifically on regional context, the findings could likely be applied to other contextual effects such as the effect of growing up in a big city or a rural area.

Chapter Three helps us understand why growing up in the South might encourage smoking and other deleterious health behaviors. It is easy to blame individuals for their lifestyle choices, but as we see from Chapter Three's exploration of state cigarette taxes, structural factors such as state policy constrain these lifestyle choices. In the case of state cigarette taxes, the influence of subtle differences in state policy can result in a weakening of the educational gradient in mortality.

In Chapter Four, I mention that there are two main ways that a social inequality in health, such as the educational gradient in morality, can change. The first way is that the underlying system of social stratification changes. The second way is that the influence of social stratification on health is somehow blocked, usually through a public health intervention or social policy. State cigarette taxes are an example of the second kind of change. By dis-incentivizing smoking (especially for low-income people), state cigarette taxes make it less

likely that only the more educated individuals will quit smoking. High cigarette taxes intervene on the existing educational disparity in smoking by giving current smokers, who are more likely to have fewer years of schooling, more incentive to quit. But the mechanisms by which cigarette taxes can reduce educational disparities in smoking and mortality are complicated. The equity-promoting effect of cigarette taxes is contingent on existing disparities in smoking. It is an effective strategy under current conditions, but its effectiveness is limited. Some smokers are resistant to tax dis-incentives. Some smokers do not have the resources to quit successfully. And with the rise of e-cigarettes and vaping, norms around smoking are being transformed yet again. This case illustrates the need for more durable ways to block the influence of social inequality on health.

Policy approaches that can shield health from the influence of social inequality in more lasting ways are mainly laws that guarantee universal access to and adoption of a key determinant of health. Examples of such policies are vaccination requirements in schools, fluoridation of public water, mandatory seatbelt laws, and bans on the use of certain chemicals in food production. These policy approaches have something important in common, which is that they treat health as a human right rather than a privilege. Policies based on the recognition that health is a human right will reduce the potential for social status to determine health status.

As Chapter Four urges, scholars should also direct attention to the other way inequalities in health can be modified: through changes in the underlying systems of social stratification. This kind of social change is generally considered to be beyond the scope of population health research. But, as I argued in Chapter Four, it is a necessary area of research for scholars interested in the modifiability of health inequalities.

Looking back over the past several decades can illuminate the ways that American commitments to freedom, individualism and capitalism, and recent political partisanship pose challenges to the right to health and, in turn, to health equity. A historical perspective can also help us see that some of the things we assume as immutable are relatively recent developments—such as the War on Drugs, the power of the gun lobby, the rise of income inequality, and the aversion toward public social services and government regulation.

In this dissertation, I have considered ways in which social context influences health, both directly through regional exposures and indirectly through social inequality. I have presented the results of various quantitative analyses of survey data. I have argued that the study of contextual influence on health inequality requires population-level thinking—something that is not always intuitive, but that is necessary for seeing the root causes and the ways that social contexts can vary, over time and place, in their influence on health. And while I do think this high-level, comparative perspective is key to advancing health equity, there is an aspect of quantitative population health research that concerns me. I worry that the language scholars use to discuss health inequality (e.g., "social stratification," "fundamental cause," even "systems of exposure") permits us to distance ourselves from the realities of oppression and suffering that is the true basis of our work. This raises questions: Can academics be partners in the struggle for health equity if they are not connected to community-based movements? Is scholarship enough or should it be paired with activism? These are questions that I have wrestled with during my work on this dissertation and throughout my time in graduate school. For all of the advances made possible through quantitative research on population health, there may be costs to such impersonal methods. Social distance makes it possible for our fears to be used for stigmatizing, for "othering," and for denying the full agency and humanity of those among us with less power.

Academics are not immune to this. This is why I think it is important for scholars to balance population-level analysis with empathy-building community engagement, community partnership, and community exposure.

In the coming decade, we will contend with the health inequalities that are being produced by climate change, mass incarceration, the criminalization of migration, and more. We will encounter new health inequalities yet to be produced by personalized medicine and machine learning applications to health. A key challenge as we confront these health inequities will be humanizing our research. Much attention is given to the "translation" of scientific research to the general public. Indeed, translational science is an important endeavor. But just as important, if not more, is learning from community-based efforts to reduce health inequities, to redistribute power, to defend health as a human right. Just as important is practicing empathy in our privileged role as scholars of health inequality, which ultimately is a study of human suffering and injustice. Therefore, in addition to honing our theoretical tools for research, population health scholars can improve how we partner with the public, how we learn from community organizations, how we engage experts in community health and organizing who may not have formal credentials but who are experts nonetheless. This combination of population-level thinking and community engagement will get us closer to the research we need to advance health equity—research that does not diminish the complexity and uniqueness of others; research that exposes the larger power structures that influence our relationships with each other and our access to health-promoting resources; research that shines a light toward social justice.

APPENDIX A:

FIGURES AND TABLES TO CHAPTER 2

Tables with Alternative Specification (Born South X South Interaction)

Table A-1. Global Measures of Health and Mortality, Alternative Specification

	(1) health_A	(2) ADL_A	(3) brokebone_A	(4) pain_A	(5) mortality_A
bornsouth	0.921	0.814	1.567	1.647*	1.227
	(0.800)	(0.551)	(0.277)	(0.043)	(0.487)
south_w2	0.839	0.972	0.715	1.571*	0.790
	(0.579)	(0.906)	(0.211)	(0.015)	(0.258)
bornsouthXsouth	1.583	1.329	0.886	0.437**	1.164
	(0.301)	(0.551)	(0.816)	(0.009)	(0.705)
N	3017	3019	2896	2793	2997

Exponentiated coefficients; p-values in parentheses
+ p<0.10, * p<0.05, ** p<0.01, *** p<0.001

Table A-2. Health Behaviors, Alternative Specification

	(1) hygiene_A	(2) smoking_A	(3) obesity_A	(4) waist_A
bornsouth	1.183	0.639	1.401	2.341
	(0.276)	(0.414)	(0.214)	(0.254)
south_w2	0.805**	1.017	0.754	0.682
	(0.002)	(0.949)	(0.146)	(0.482)
bornsouthXsouth	0.920	1.850	0.902	0.728
	(0.626)	(0.293)	(0.796)	(0.762)
N	3014	3020	2921	2980

Exponentiated coefficients; p-values in parentheses
+ p<0.10, * p<0.05, ** p<0.01, *** p<0.001

Table A-3. Chronic Illness and Treatment, Alternative Specification

	(1) hypertensi~A	(2) hypertensi~A	(3) antihypert~A	(4) diabetes_d~A	(5) diabetes_h~A	(6) antidiabet~A	(7) depression_A	(8) antidepres~A
bornsouth	1.049	0.477***	1.732	0.940	0.993	1.015	1.660*	1.930
	(0.794)	(0.000)	(0.100)	(0.794)	(0.982)	(0.968)	(0.043)	(0.122)
south_w2	1.101	0.886	1.115	0.775	1.641+	0.904	0.894	0.815
	(0.597)	(0.530)	(0.713)	(0.378)	(0.079)	(0.728)	(0.747)	(0.438)
bornsouthXsouth	0.801	2.830***	0.637	1.650	0.535	1.625	0.488+	0.843
	(0.382)	(0.000)	(0.311)	(0.171)	(0.176)	(0.309)	(0.095)	(0.728)
N	3015	2947	2995	3016	3020	2995	3007	2995

Exponentiated coefficients; p-values in parentheses
+ p<0.10, * p<0.05, ** p<0.01, *** p<0.001

Table A-4. Social Integration, Alternative Specification

	(1) loneliness_A	(2) netsize_A	(3) density_A
bornsouth	-0.0127	-0.199	-0.00119
	(0.894)	(0.302)	(0.959)
south_w2	-0.0978	-0.180	-0.00970
	(0.109)	(0.167)	(0.704)
bornsouthXsouth	-0.0202	0.0921	0.0527
	(0.855)	(0.727)	(0.129)
N	2976	3016	2933

p-values in parentheses
+ p<0.10, * p<0.05, ** p<0.01, *** p<0.001

APPENDIX B:
FIGURES AND TABLES TO CHAPTER 3

Table B-1. NSHAP Wave 1 Sample Statistics

	Possible Range	Mean (SD) Number (%)
5-Year Mortality 2006-2010	0 or 1	430 (14.4)
Current Smoker	0 or 1	445 (14.8)
Former Smoker	0 or 1	1288 (42.9)
Average Cigarette Tax 2001-2005 in cents	3 to 165	70.2 (44.5)
Average Cigarette Tax 2006-2010 in cents	7 to 259	113.2 (63.9)
Years of Schooling	0 to 32	12.7 (4.1)
Educational Attainment:		
Less than High School	0 or 1	699 (23.3)
High School	0 or 1	793 (26.4)
Some College	0 or 1	856 (28.5)
Bachelors or more	0 or 1	657 (21.9)
Age	57 to 85	69.3 (7.9)
Female	0 or 1	1551 (51.6)

Table B-2. NSHAP Wave 2 Sample Statistics

	Possible Range	Mean (SD) Number (%)
5-Year Mortality 2006-2010	0 or 1	624 (18.1)
Current Smoker	0 or 1	450 (13.3)
Former Smoker	0 or 1	1502 (44.5)
Average Cigarette Tax 2001-2005 in cents	3 to 165	69.6 (49.6)
Average Cigarette Tax 2006-2010 in cents	7 to 259	112.9 (62.3)
Years of Schooling	0 to 32	13.1 (3.9)
Educational Attainment:		
Less than High School	0 or 1	645 (19.1)
High School	0 or 1	833 (24.7)
Some College	0 or 1	1072 (31.7)
Bachelors or more	0 or 1	827 (24.5)
Age	36 to 99	72.4 (8.1)
Female	0 or 1	1839 (54.5)

Figure B-1. Probability of 5-Year Mortality Among Smokers by 2006-2010 Cigarette Tax Level

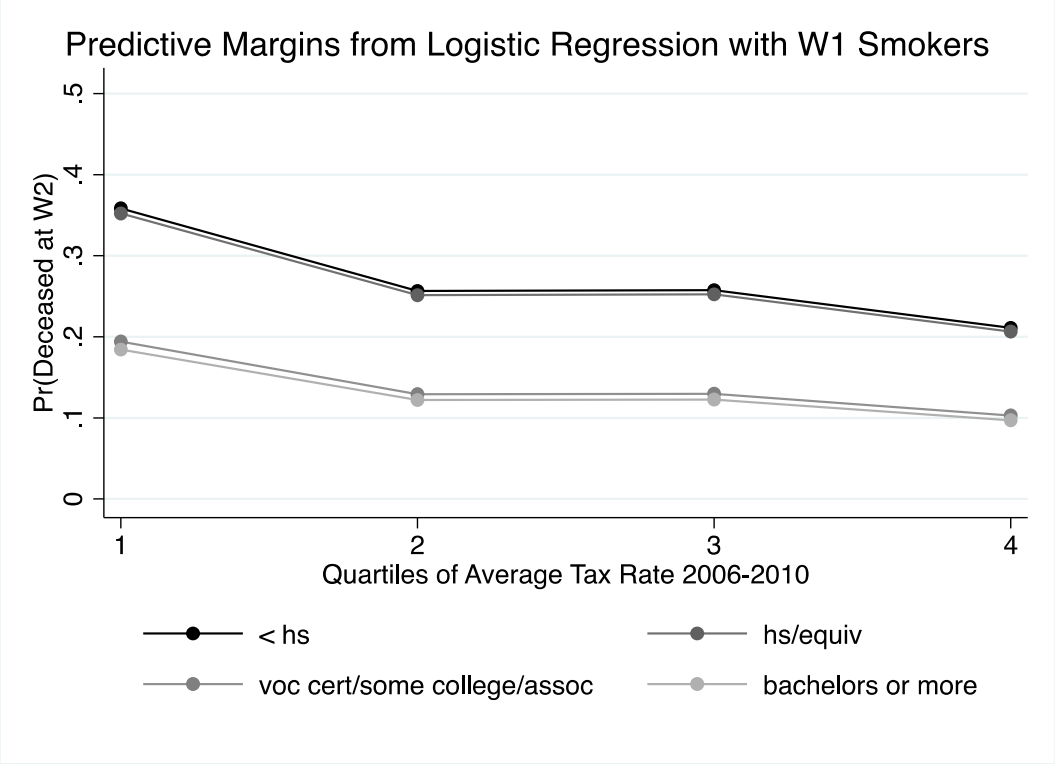


Figure B-2. Hazard of Death Among Smokers by 1999 Cigarette Tax Level

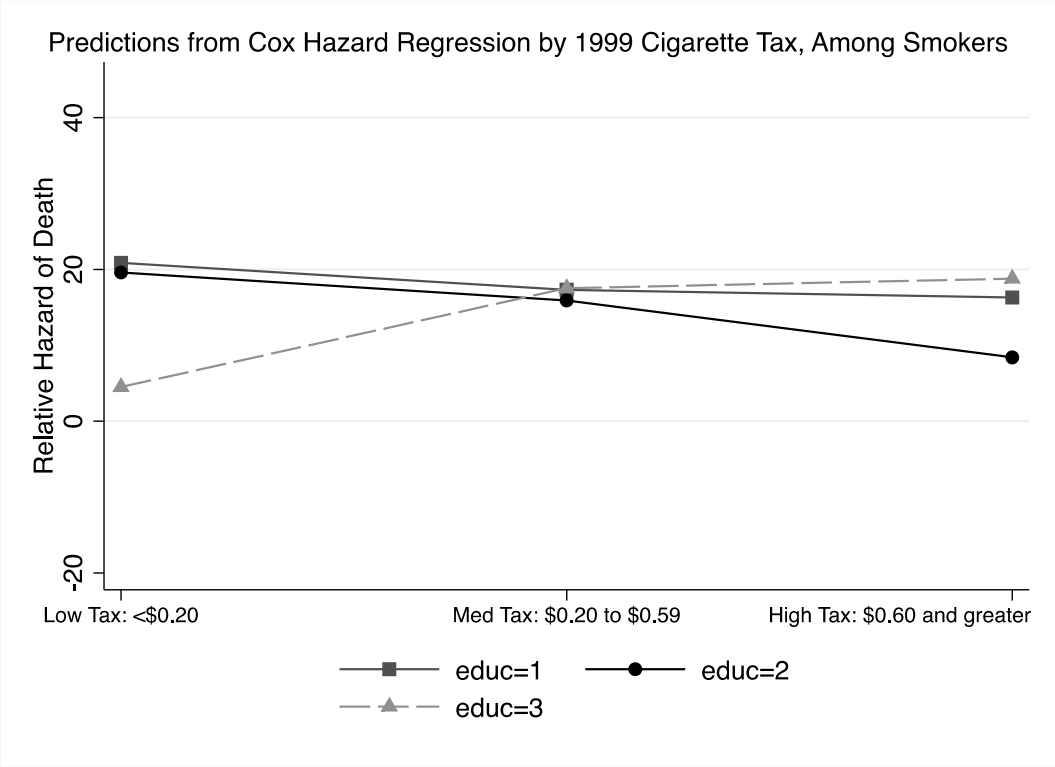


Table B-3. Cox Proportional Hazard Regression Predicting Death During 1999-2015

Hazard of Death	**Model 1**	**Model 1.1 Stratified: Men**	**Model 1.2 Stratified: Women**
Predictors of Interest			
Years of Schooling	.761***	.748***	.793
	(.054)	(.059)	(.114)
State Cig Tax	.097**	.034**	.554
	(.081)	(.035)	(.776)
Years of Schooling	1.200**	1.284**	1.052
X State Cig Tax	(.080)	(.100)	(.127)
N Subjects	1,945	980	965

Legend: * p<0.05; ** p<0.01; ***p<0.001
Note: All models control for white, age, total family income. Model 1 also controls for female.
Data Source: Panel Study of Income Dynamics, Waves 1999-2015.

Figure B-3. Reduction in Smoking Intensity by Proportional Tax Increase

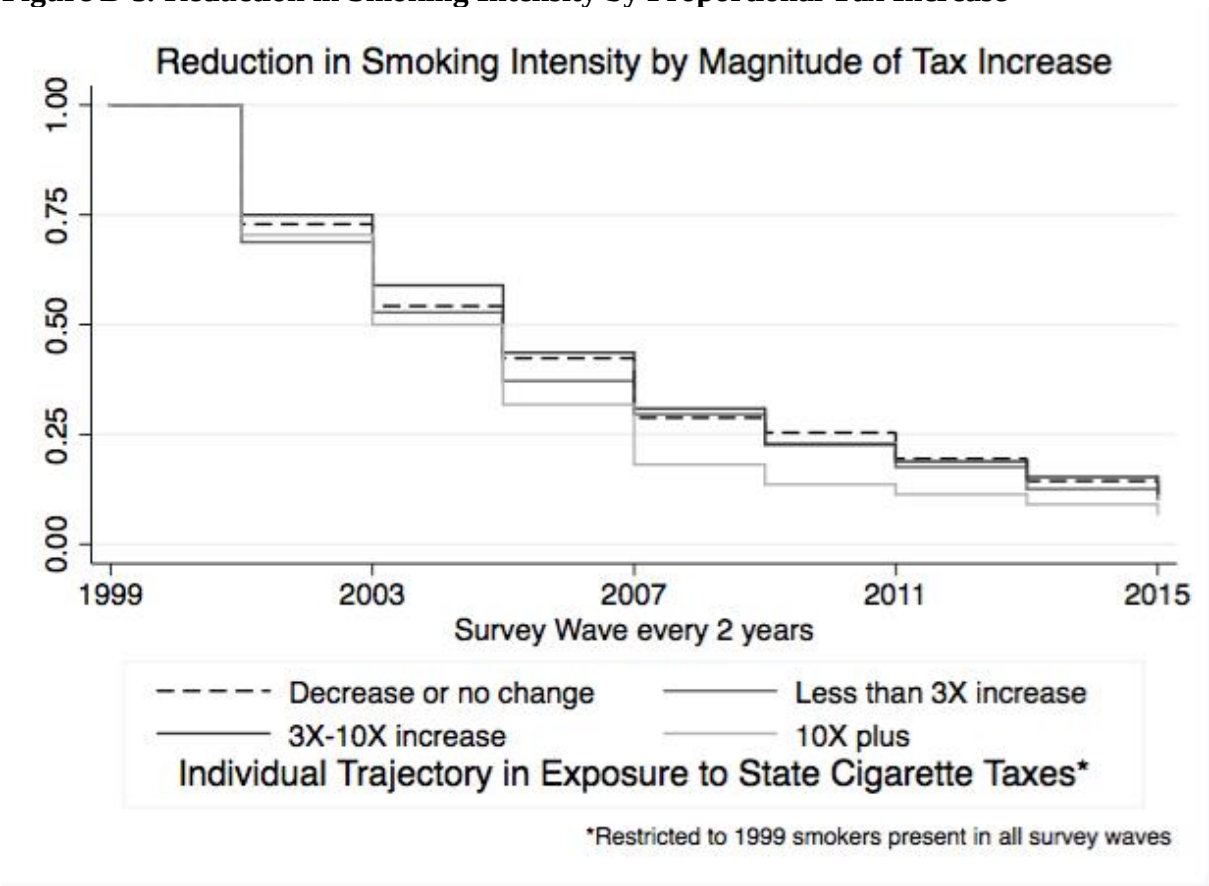


Figure B-4. Smoking Prevalence by 1999 Cigarette Tax Quartile

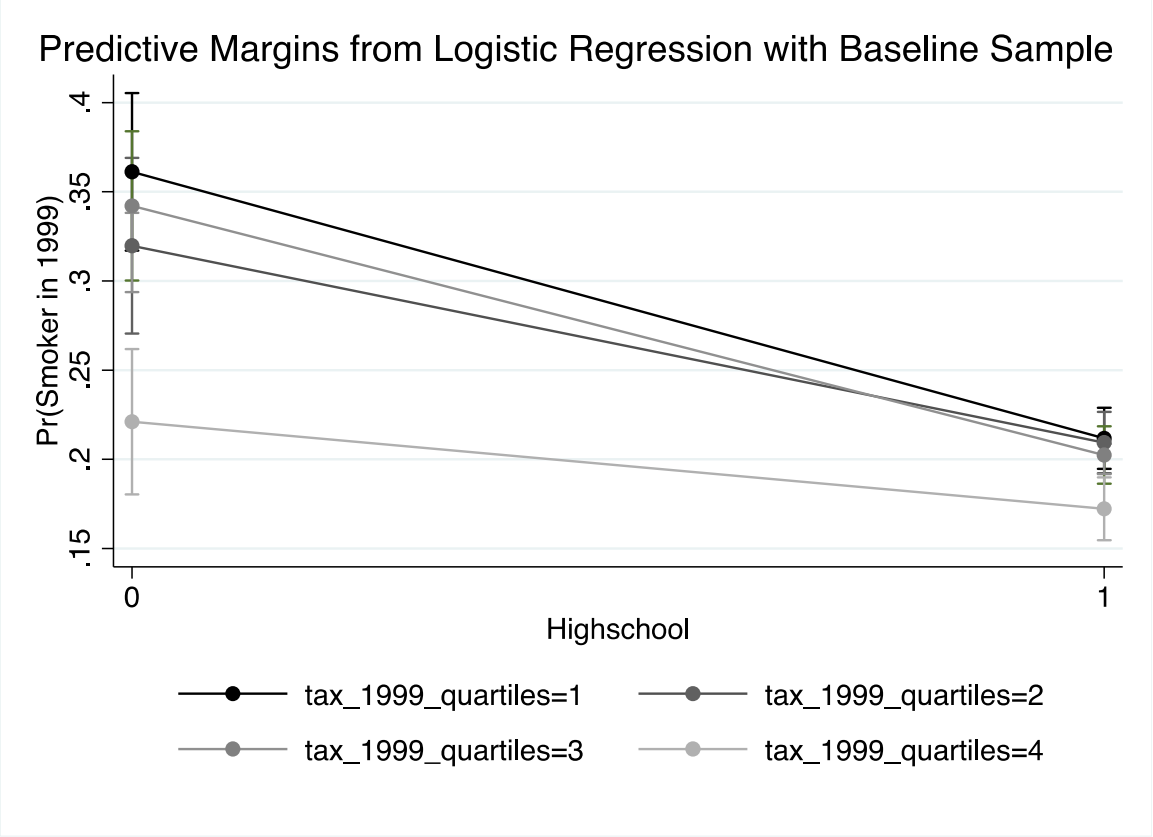


Figure B-5. State Cigarette Tax Dynamics 1999 to 2015

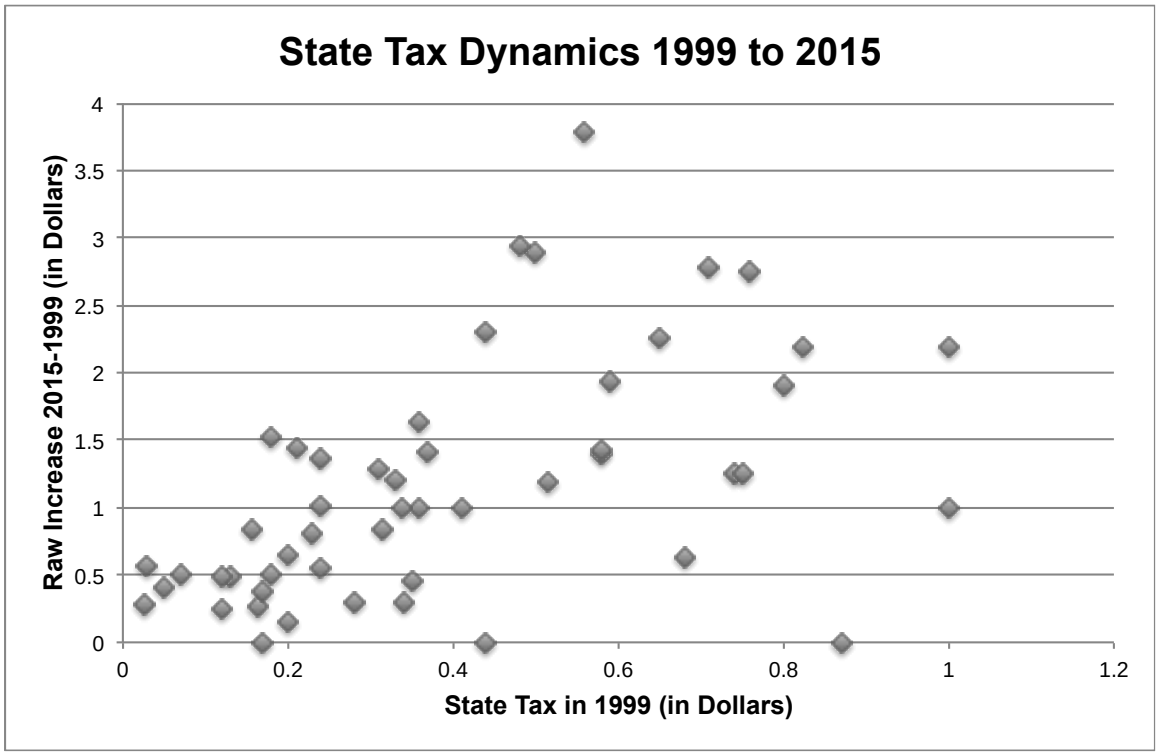


Figure B-6. State Cigarette Taxes and Cigarette Tax Increases 1999 to 2015

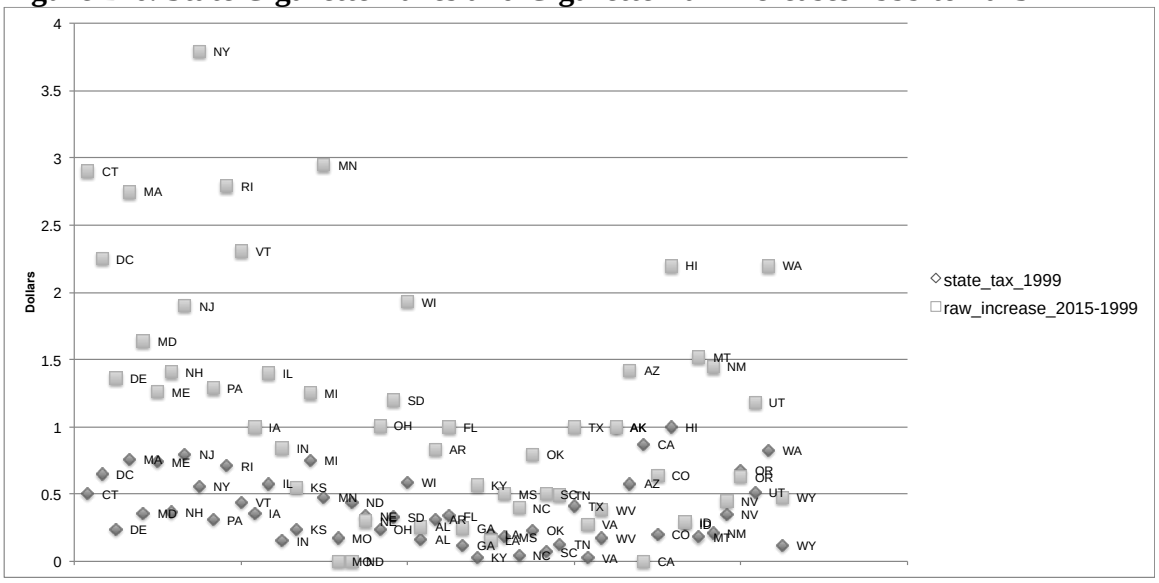
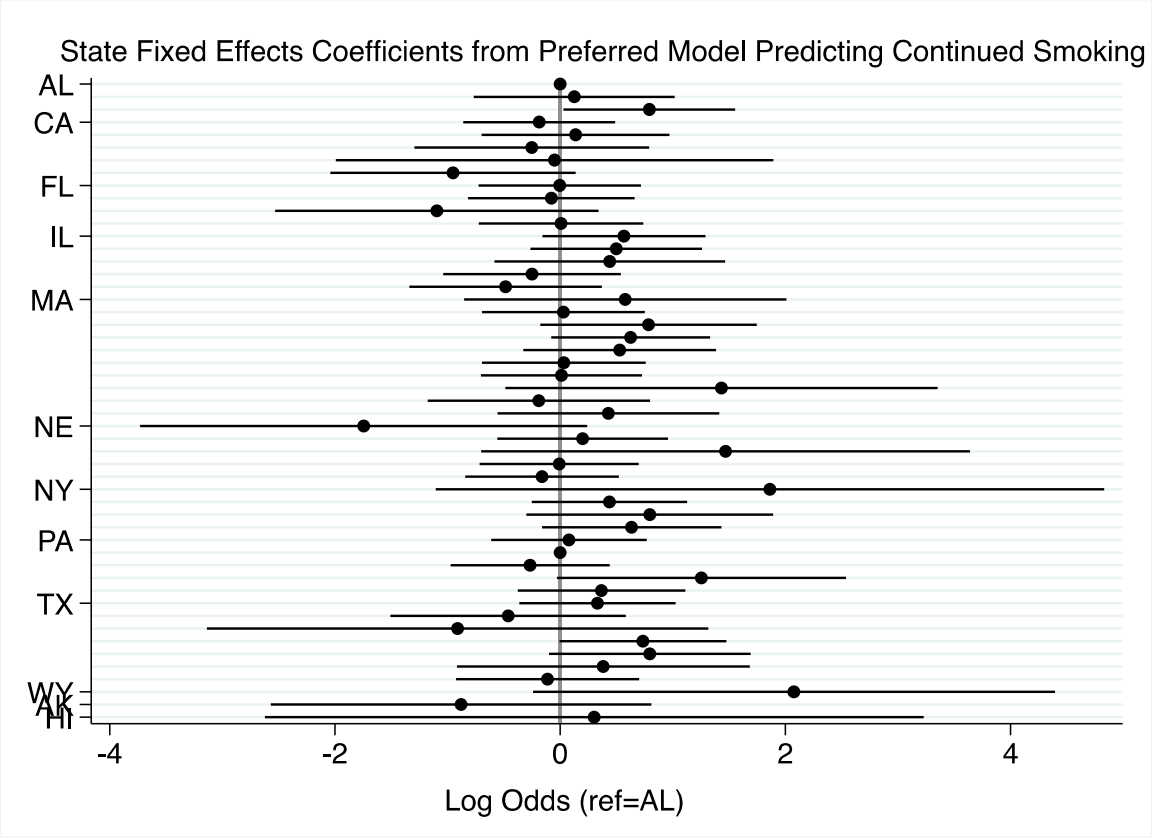


Figure B-7. State Fixed Effects Coefficients from Preferred Model Predicting Continued Smoking



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